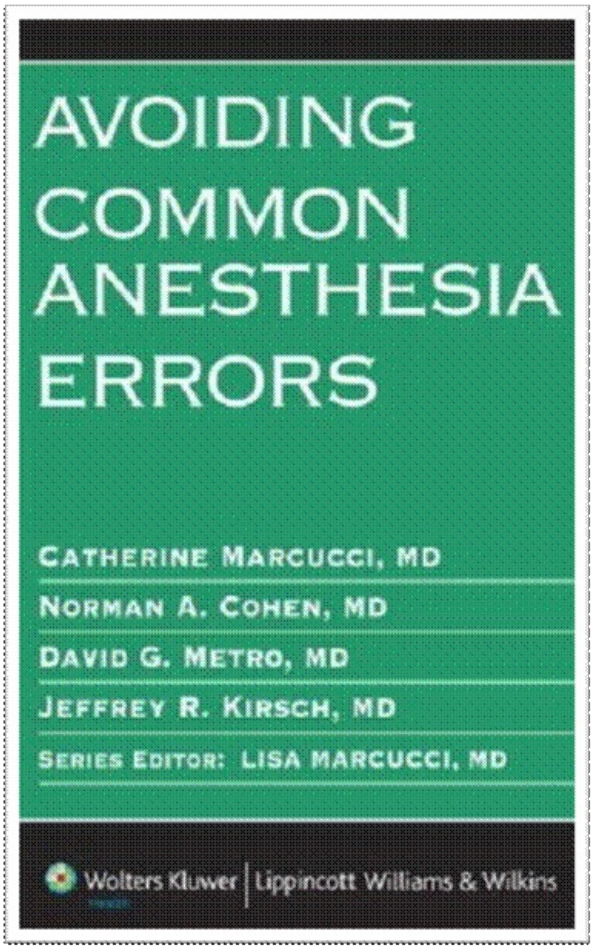
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Editors: **Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.**
Title: ***Avoiding Common Anesthesia Errors, 1st Edition***

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2008

Lippincott Williams & Wilkins
Philadelphia
530 Walnut Street, Philadelphia, PA19106 USA

978-0-7817-8847-2

0-7817-8847-1

Acquisitions Editor: Brian Brown

Managing Editor: Nicole Dernoski

Marketing Manager: Angela Panetta

Production Editor: Bridgett Dougherty

Senior Manufacturing Manager: Benjamin Rivera

Design Coordinator: Risa Clow

Compositor: Aptara, Inc.

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Printed in the United States of America

Library of Congress Cataloging-in-Publication Data

Avoiding common anesthesia errors/editor, Cathy Marcucci.

p.; cm.

Includes bibliographical references and index.

ISBN-13: 978-0-7817-8847-2 (case)

ISBN-10: 0-7817-8847-1 (case)

1. Anesthesia. 2. Medical errors—Prevention. I. Marcucci, Cathy.

[DNLM: 1. Anesthesia—methods. 2. Medical Errors—Prevention & control. WO 200 A961 2007]

RD81.A96 2007

617.9'6—dc22

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FOREWORD

Anesthesiology has evolved to a place in medicine far beyond the traditional provision of intraoperative care, solo or small group practices, and one-office billing processes. The scope of practices in the specialty now encompasses the entire perioperative spectrum, ranging from preoperative assessment and management through postoperative intensive care and acute pain management. Pain medicine also has transitioned from “mom and pop” injection shops to expansive, multidisciplinary practices and hospice care. Even the business processes of anesthesiology, from billing to call responsibilities to exclusive contracting, have become increasingly complex. Thus, medical students, residents, fellows, and other anesthesia students, plus a growing proportion of us older folks who see our practices expanding or who are involved in maintenance of certification, need concise, accurate, and useful information that covers the broad range of topics that we encounter in practice and the business of anesthesiology.

Drs. Cathy Marcucci, Norm Cohen, David Metro, and Jeff Kirsch have provided all anesthesia trainees and many of our practicing anesthesiologists with a book that is terrific. It provides practical advice on many of the most important and often perplexing issues that we see in everyday practice. I am not aware of any other anesthesiology text that is so useful and, to be honest, fun to read. It is not—and was not designed to be—a comprehensive text. Instead, it provides common sense advice for many of our specialty's most important clinical issues in small, easily digestible bites. In addition, many trainees will find the chapters on practice selection and management to be very worthwhile.

The editors have performed a great service for those young men and women who are attracted to our specialty, plus for those of us who have been blessed with the opportunity to care for anesthetized patients, those who are critically ill, and those who require relief from acute or chronic pain. They deserve our thanks for producing such a useful contribution to anesthesia education.

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PREFACE

The pressures facing the anesthesiologist or anesthetist are in some ways unique. As always, the patients say it best. Recently, one of the editors of this book was asked the following question, “You mean to tell me that it takes three surgical doctors to take care of just my ear drum, but you and that young fellow over there can take care of all of the rest of me all by yourselves?”

As part and parcel of taking care of “all of the rest” of the patient, the anesthesia provider must fully understand physiology, pathophysiology, and pharmacology; become experts in anesthesia equipment and associated specialized technology; and master skills in airway management and the performance of many procedures. And we must do this in a complex, noisy, and often hostile clinical setting, all while trying as hard as possible to do the right thing and not do the wrong thing! Unfortunately, in spite of everyone's best efforts, mistakes will happen. The editors and authors of this book have had the goal to convey their knowledge from *personally* dealing with and learning from the vast majority of situations in this book, either as a trainee, provider, or a supervisor. We hope that we can impart the knowledge of our pooled experience to the reader who may have limitations due to the nature of their clinical setting or the stage of their career.

It has been suggested facetiously that this book might also be titled “Conversations from the Anesthesiology Break Room,” which is exactly what we intended from the outset of this project. There are many excellent standard comprehensive and reference texts available to anesthesia providers and trainees; this book is intended to complement rather than supersede them. Our goal is to provide our collective wisdom in readable, conversational bites, exactly as if you are sitting in the anesthesia office with your supervising faculty or attending and you have about ten minutes before the case starts. Literally, when pondering points for the book, two of our main criteria were “What do we hear the senior staff saying to the junior staff over and over? What was said to us when we were in training that had lasting impact on our practices?” It's also okay with us if the readers laugh a little while reading this book.

In developing the content, we owe a tremendous debt to both our contributing editors and section editors. These individuals contributed expertise, enthusiasm, energy, and ideas and shouldered huge amounts of the work as well. Special thanks to Randal O. Dull, MD, PhD for believing in and “signing onto” the project in its very earliest stages and F. Jacob Seagull,
PhD for helping us with his special expertise in the field of human factors and errors psychology.

We also thank several others for their unique contributions: Katey Millet and Renee Redding for their advice and assistance in preparing the manuscript; the anesthesia residents at the Johns Hopkins Hospital, including Chauncey T. Jones, MD, Andrew M. Gross, MD, and Hassan Ahmad, MD as well as the anesthesia residents at the University of Utah for writing the first chapters and showing us it could be done; Charles W. Emala, MD for his help in finding us authors and editors at Columbia; John L. Cameron, MD for giving us permission to quote him; Sharon Merrick, CCS-P, Coding Manager at ASA's Washington Office, for her technical review of the coding chapters; Michael J. Moritz, MD for his review of the initial chapters of the book; Michael W. Barts, CRNA, for reviewing the topics list and sending us input “from the field;” Deborah Dlugose, CRNA, for her advice, suggestions, and encouragement; and Denis L. Bourke, MD, for his ongoing support.

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The authors and editors have unanimously donated their royalties to the Foundation for Anesthesia Education and Research in hopes of further improving the foundation of knowledge in our specialty.

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ACKNOWLEDGMENT

Many thanks to Surjya Sen, MD, who carried full editorial duties for this project, in spite of being a busy resident and fellow, and whose feedback and advice were invaluable. C.M. I'd like to thank Sharon Merrick, CCS-P, Coding Manager at ASA's Washington Office, for her technical review of the coding chapters. N.A.C.

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1

Never Neglect the Basics of Airway Management

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Establishing an adequate airway is an important task for every anesthesia provider, as is having a contingency plan in place for a “lost airway.” In general, most airway management occurs in the operating room. Before any operation, the anesthesiologist or anesthetist must evaluate the patient's airway and focus on predictors of a difficult airway to minimize the stress and surprise if the airway cannot be secured via the originally planned intervention. Absence of such preparedness could have disastrous consequences.

PATIENT-RELATED CAUSES OF INABILITY TO VENTILATE

The most frequent complications of premature tracheal extubation are hypoventilation, apnea, or obstructive breathing, all frequently associated with hypoxemia and hypercarbia. Immediate postoperative apnea or hypoventilation is most often caused by residual inhaled anesthetic and opioid effects, incomplete reversal of neuromuscular blockade, or the presence of redundant oral tissues. Some patients may have increased sensitivity to anesthetic agents (e.g., elderly, children, patients with obstructive sleep apnea). All this can contribute to loss of airway. Postextubation apnea or hypopnea generally can be resolved with positive-pressure bag-mask ventilation, which may be aided with the placement of an oral or nasopharyngeal airway. However, if ventilation is not achieved, airway patency must be re-established by reintubation or placement of an alternative airway device, such as a laryngeal mask airway (LMA).

Before tracheal extubation, the risk of apnea or hypopnea can be minimized by achieving spontaneous ventilation with adequate tidal volumes and respiratory rate. In addition, anesthetic levels should be low enough to ensure that the patient is able to follow simple commands (e.g., opening eyes, squeezing hands, head lift sustained >5 s). For every patient, the anesthesia provider must check for elegibility for reversal of muscle relaxation by using the twitch monitor, and the reversal agent should be administered well before the end of the surgery to allow enough time for maximal inhibition of pseudocholinesterase. We believe that every patient who has received nondepolarizing muscle relaxants during surgery should receive reversal agents, even if the train-of-four ratio appears visually normal. Exception to that practice should be rare and well considered. P.3

Another possible cause of a lost airway after tracheal extubation is laryngospasm. The highest risk for laryngospasm occurs after induction, just before intubation, or upon emergence from general anesthesia after tracheal extubation as the patient passes through the excitatory phase of anesthesia. Certain patients are at higher risk, such as children and those with a history of smoking, asthma, bronchitis, or bronchiectasis. In addition, certain anesthetic agents, such as desflurane, are more likely to be associated with laryngospasm. If use of bag-mask positive-pressure ventilation with 100% oxygen is unsuccessful in breaking the laryngospasm and maintaining oxygenation, neuromuscular blockade with succinylcholine may be indicated.

MECHANICAL CAUSES OF INABILITY TO VENTILATE

Occlusion of the endotracheal tube by the teeth can occur at virtually any time during anesthesia. It may be due to the patient approximating his or her teeth during emergence or light anesthesia, or to surgical personnel inadvertently applying pressure on the patient's face, causing closure of the teeth. Attempting to breathe spontaneously through an occluded or near-occluded endotracheal tube can cause negative-pressure pulmonary edema, especially in athletic (muscular) patients. In these situations, positive-pressure ventilation could be difficult or impossible. This can be remedied by increasing depth of anesthesia with intravenous agents or administration of muscle relaxants (propofol, lidocaine, or succinylcholine). Typically, after the patient is anesthetized, the endotracheal tube will recoil and airway patency will be re-established. However, when using a wire spiral endotracheal tube, there may be less elastic recoil and the tube may remain partially occluded, requiring emergency tracheal extubation and reintubation with a new tube. This situation is best avoided by placing a “bite block” device, such as an oral airway or other premanufactured hard plastic device, immediately after intubation (especially if muscle relaxants are not used during the operation). Alternatively, a soft bite block may be made from tightly rolled and taped gauze pads placed between the molars.

SURGICAL SITUATIONS CONTRIBUTING TO THE DIFFICULT AIRWAY

Anesthesiologists must exercise special caution when a double-lumen endotracheal tube is changed to a single-lumen tube. The anesthesiologist should critically assess the need for tube exchange because it may be risky in certain patients, such as those who

- Were initially difficult to intubate
- Are morbidly obese
- May be difficult to ventilate via bag-mask device in case reintubation is unsuccessful (beard, obese, facial disfiguration, edentulous) P.4
- Have a compromised airway as a result of the just-performed oral surgery or edema from fluid overload

For these patients it may be safer to keep the double-lumen tracheal tube in place. Alternatively, the anesthesiologist can pull the bronchial lumen into the trachea (after properly deflating both bronchial and tracheal cuffs). After such repositioning, only the tracheal cuff should be reinflated. Another approach is to use a single-lumen tube from the beginning of the case and use a bronchial blocker to perform one-lung ventilation when necessary. However, exchanging the endotracheal tube with “tube exchangers” may not guarantee success.

Although the situation is rare today, special caution should be exercised before tracheal extubation in a patient whose mouth has been wired shut after oral surgery. For these patients, a wire cutter must be readily available at the bedside at all times to facilitate access to the oropharynx in case of emergency. To avoid a potential airway emergency, these patients should be fully awake and responding appropriately to commands before tracheal extubation. All preventive measures should be taken to decrease or eliminate potential postoperative nausea and vomiting, including placing a nasogastric tube and suctioning of gastric contents, administering antiemetic agents, and using a total intravenous anesthetic technique.

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PREPARING FOR TRACHEAL EXTUBATION

The scenarios described above clearly illustrate the necessity for always having a backup plan in mind at the time of tracheal extubation. Necessities that always must be available include

1. 100% oxygen source and face mask with bag-mask device
2. Oral suction system
3. Laryngoscope with various blades, endotracheal tube, intubating stylet, and alternative airway device (e.g., LMA)
4. Oral and/or nasopharyngeal airway
5. Induction drugs drawn up and ready, especially succinylcholine

AVOIDING UNEXPECTED LOSS OF THE AIRWAY

After tracheal intubation of every patient, it is crucial to properly secure the endotracheal tube at the appropriate length. In addition, the endotracheal tube should be manually secured (guarded) during any change of the patient's position, as well as during endoscopic procedures. The endotracheal tube should be briefly disconnected from the circuit for transport of the intubated patient from the operating-room bed to the hospital bed. The same caution should be exercised even if simply turning the operating-room table, because tension from the breathing circuit can pull out a previously well-secured endotracheal tube. The endotracheal tube position should also be checked intermittently throughout the operation.

Although the above recommendations may reduce the risk, airway mishaps can still occur. In this emergency situation, members of the surgical team must have clear communication (stop the surgery, prioritize regaining airway access) and the anesthesiologist must take decisive actions. Asking for help from another anesthesia provider may be prudent.

ESTABLISHING AN AIRWAY IN AN EMERGENCY SITUATION

Emergency resuscitation situations can present many difficult airway situations, often in areas not as familiar to the anesthesiologist or not as well equipped as the operating room. This emphasizes the importance of anesthesiologist input when stocking an emergency airway cart. In addition to the traditional tools such as face mask, oral/nasopharyngeal airway, laryngoscope, multiple blade sizes and styles, appropriate-sized endotracheal tubes, and intubating stylet, the cart should also include alternative airway devices (e.g., LMA, intubating LMA, fiber-optic bronchoscope, Bullard scope, light wand, retrograde guidewire intubation, cricothyrotomy kit, and transtracheal jet ventilation). In certain situations, such as a profusely bleeding upper airway, however, a fiber-optic bronchoscope may not be of much help. Well-functioning suction must be immediately at hand. The anesthesia provider should be familiar with alternative devices and be comfortable in using them in an emergency; an emergency airway situation is not a good time to attempt unfamiliar techniques. Consider calling for help early, including from a surgeon in case a surgical airway (emergency tracheotomy, cricothyroidectomy) is needed.

TAKE HOME POINTS

- Lack of airway preparedness can have disastrous consequences when planning extubation of the patient.
- The most frequent complications of premature tracheal extubation are hypoventilation, apnea, or obstructive breathing. Immediate postoperative apnea or hypoventilation is most often caused by residual inhaled anesthetic and opioid effects, incomplete reversal of neuromuscular blockade, or the presence of redundant oral tissues.
- Maintain vigilance for laryngospasm. Be ready to move quickly to positive-pressure ventilation with a mask, or if that fails, succinylcholine.
- Be aware of the special risks involved with changing a double-lumen endotracheal tube to a single-lumen endotracheal tube. Remember that in almost every instance, this maneuver, though advantageous, is optional.
- Do not extubate without being prepared to reintubate if necessary.

SUGGESTED READINGS

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Editors: **Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.**
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Basics of Airway Management—Part II (Tips and Tidbits)

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Airway management is both a science and an art, and the wise anesthesia provider will make himself a lifelong student. An informal review of the peer-reviewed anesthesia literature shows that there are at least 25 new manuscripts, case reports, and letters on airway management each month. Similarly, even the “old” drugs in airway management are under ongoing evaluation—the literature for the first months of 2007 contains dozens of reports on the use of succinylcholine, a drug that has been in clinical use for about 60 years. The authors feel strongly that there is also ample opportunity to learn from the spoken tradition as well.

- Try to become expert in each new airway device and technique as it comes out, but remember that the most valuable airway techniques are sometimes the simplest and most basic—a nasal trumpet, a headstrap, or even a jaw thrust. It is best to become proficient with a new airway device on patients with a normal airway before trying to use the new airway device on a complicated patient or in an emergency situation.
- Control your airway situation by controlling your airway drugs. One of the authors saw a case of significant bradycardia probably caused by repeated doses of succinylcholine given to facilitate intubation. The clinical situation then focused on dealing with the bradycardia rather than securing the airway.
- One of the most valuable drugs in airway management is oxygen (the authors had a professor of anesthesia who used to say to put the patient on oxygen first and then think). If difficulty is suspected, have the patient breathe 100% oxygen for 5 minutes. Remember that oxygen utilization by the (nonpregnant) human adult is approximately 3 mL/kg/min. If you have fully denitrogenated the lungs, the chances of having “the oxygen out last the succinylcholine” are increased.
- When you are just learning airway skills, try to do as many mask cases as possible. The successful mask airway is the foundation on which all airway management is based.
- Do not underestimate the amount of airway edema and/or bleeding that can occur with even one or two attempts at laryngoscopy.
- Remember that for an intubated patient, the tip of the endotracheal tube (ETT) follows the chin. Flexing the patient's head toward the chest seats the ETT more deeply in the trachea and vice versa. P.8
- Excessive tape on the ETT can be as troublesome as undertaping the ETT. Many senior anesthesia providers have anecdotes involving skin damage to patients' faces or accidentally extubating too soon when “trying to loosen the tape” in advance of being asked to do a planned extubation, such as in a tracheostomy.
- Asking a patient to “squeeze my hand” is not a great pre-extubation test; it really only demonstrates the ability to follow verbal commands. Remember that hand strength is not a standard indication of reversal of neuromuscular blockade. It is best to use standardized methods to assess strength (e.g. head lift, leg lift) before progressing to extubation. It is also suggested that the anesthesia provider ask the patient to do things that are linked functionally to your needs during the first few seconds after extubation: “open your mouth,” “take a deep breath,” or “try to swallow.” This will also increase your confidence that the patient will be able to take a deep enough breath to cough, open their mouth for suction, and be able to control their airway enough to handle secretions.
- A surprising number of serious airway events include the fact that the ETT was actually in the trachea at some point and then was accidentally or purposely removed due to delayed taping, an attempt to change the ETT to a more advantageous size, or the like. A corollary to this is to never feel oversecure when using a tube changer to change the ETT even if the initial airway management was not especially difficult.
- Always be very vigilant when doing airway management in remote locations. The authors feel that the best adjunct intubating aid to take with you is another experienced anesthesia provider. After that, a simple intubating stylet and a laryngeal mask airway are very valuable tools.
- It is surprisingly easy to forget the cricoid pressure and to have working suction when intubating in the intensive care units and other remote locations.
- Always remember to wear your mask and eye protection when intubating in the intensive care units. This is commonly where anesthesia providers incur eye exposures to body fluids (including one of the authors).
- When called to a remote location to do airway management, you, as the expert airway consultant, should expect to assume control of the airway at the time of your arrival.
- One of the most dangerous requests is the call to the intensive care unit to “change the tube,” either due to secretions or a ruptured ETT cuff. Exhaust all options before taking out that ETT. Be sure that high airway pressures are not due to a mainstem intubation. Call for a bronchoscope to confirm that there truly are secretions in the ETT. Try a wet throat pack in the supraglottic area if a cuff leak is suspected as the cause of an inability to deliver an appropriate ventilator volume. Do a direct laryngoscopy with the tube in place to make sure that a “leaking ETT cuff” is not just a cuff that has herniated out from the trachea. Be sure “excessive leak” around an ETT isn't due to the combination of the patient requiring high airway pressures (poor pulmonary compliance) and low cuff pressure. When those options are exhausted, call for a colleague. P.9

TAKE HOME POINT

- Although the authors talk about airway management being an “art,” it does not mean that there are not right and wrong ways of doing things. The American Society of Anesthesiologists has developed a difficult airway algorithm that has become a standard that anesthesia providers need to be aware of. The best advice a new practitioner can receive is to always err on the side of caution and to have many possible options in their armamentarium of airway management techniques.

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3

Consider PEEP

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Positive end-expiratory pressure (PEEP) is defined as an elevation of alveolar pressure at the conclusion of passive expiration that exceeds atmospheric pressure. PEEP is categorized as applied/extrinsic PEEP or auto/intrinsic PEEP. Extrinsic PEEP is delivered directly through a mechanical ventilator or through a tight-fitting face mask at the end of exhalation. PEEP maintains the patient's airway pressure above atmospheric level by exerting pressure to oppose passive emptying of the lung. Intrinsic PEEP occurs when the lungs fail to deflate fully prior to the next breath, which maintains the alveolar pressure above the atmospheric pressure. If otherwise unspecified, PEEP usually refers to extrinsic PEEP. Both extrinsic PEEP and intrinsic PEEP are covered in this chapter.

EXTRINSIC PEEP

Why Use PEEP?

PEEP is used to completely or partially replace the function of the respiratory muscles and therefore correct hypoxemia secondary to alveolar hypoventilation. PEEP is effective in improving arterial oxygenation simply through increasing functional residual capacity (FRC), reducing venous admixture, shifting tidal volume, preventing loss of pulmonary compliance, and decreasing the work of breathing.

Effects of PEEP

Respiratory System.

Extrinsic PEEP improves oxygenation by directly affecting alveolar gas exchange and pulmonary mechanics of the respiratory system. Overall, alveolar gas exchange improves through redistribution of fluid within the alveoli. As a result, there is a decrease in intrapulmonary shunting and an improvement in arterial oxygenation. With the addition of PEEP, arterial oxygenation goals may be achieved with a lower fraction of inspired oxygen (FIO_2) and therefore a decreased risk of oxygen toxicity. In terms of lung mechanics, FRC increases and lung compliance improves. PEEP is also responsible for prevention of alveolar collapse, which is of critical importance because the repetitive collapse of lung units at the end of expiration may lead to ventilator-induced lung injury (VILI).

Cardiovascular System.

From the cardiovascular standpoint, the addition of PEEP causes a significant increase in intrathoracic pressure. As a result, there is a decrease in venous return and decrease in right and left ventricular preload. This leads to a decline in cardiac output. Biondi et al. showed that at low levels of PEEP the predominant effect is right ventricular (RV) preload reduction. Above 15 cm H_2O PEEP, RV volumes increase and RV ejection fraction (EF) decreases, consistent with increased RV afterload and declines in RV contractility, presumably as a result of increased RV wall stress.

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Several possible factors can lead to decrease in cardiac output during application of PEEP. In addition to changes in loading conditions, the leftward septal displacement, systolic and diastolic ventricular interdependence and altered cardiac geometry can all affect cardiac output during the application of PEEP. In any case, the decline in cardiac output may progress toward hypotension and subsequent hypoperfusion of critical organs.

The adverse effects of PEEP on the cardiovascular systems cannot be overlooked. One study has revealed that PEEP may cause tricuspid regurgitation. The combination of increased pulmonary artery pressures, increased vascular resistance, and altered RV geometry may all affect tricuspid valve function. This is critical because the presence of undiagnosed tricuspid regurgitation during mechanical ventilation may invalidate cardiac output values as well as the derived parameters that are determined through thermodilution.

Liver.

Effects of PEEP on the hepatic system are also noticeable. The drop in cardiac output causes a decrease in splanchnic blood flow and subsequent decrease in hepatic and portal blood flow. However, an increased in hepatic outflow resistance as a result of impaired venous drainage can lead to the observed liver congestion and hinder the hepatic perfusion. The use of PEEP causes a downward shift of the diaphragm, which further compresses the liver and worsens the hepatic perfusion.

Renal.

PEEP causes a decrease in urinary output, sodium excretion, and creatinine clearance. The mechanisms responsible for the decreased urine output are most likely multifactorial, including a decrease in cardiac output, renal blood flow, intravascular volume, and reflex sympathetic nerve activation. There is also an altered release of various hormones including catecholamines, renin-angiotensin-aldosterone, antidiuretic hormone, and atrial natriuretic factor, which contributes to the effects of PEEP on renal function. The patient's volume status and the amount of applied PEEP both contribute to alterations in renal function.

Central Nervous System.

The effects of PEEP on cerebral mechanics are numerous. As PEEP is added, there is a notable rise in right atrial pressure. This further translates to an increase in superior vena cava pressure and a decrease in cerebral venous return, resulting in an increase in intracranial pressure (ICP). Other influencing variables include intracranial compliance and body positioning. When the head is elevated above the chest, the transmitted intrathoracic

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pressure to the intracranial venous sinuses is decreased.

As a result, ICP is minimally affected. In patients with normal ICP, the effect of PEEP can be easily overcome by maintaining normal intravascular volume and systemic blood pressure. P.12

Bronchial Circulation and Thoracic Lymph Drainage.

Because it increases the intrathoracic pressure, PEEP affects both bronchial blood flow and thoracic lymph drainage. Lymph drainage from the thoracic duct may be obstructed as a result of PEEP, and edema fluid removal from the lungs may be impaired. Bronchial blood flow may also be affected and interfere with lung repair.

To PEEP or Not to PEEP?

Typically, only a small amount of PEEP is needed. About 3 to 5 cm of water is considered the physiologic amount required to overcome the decrease in FRC from bypassing the glottic opening with an endotracheal tube. Use of supraphysiologic PEEP (>5 cm H_2O) may be indicated in certain settings.

Specific indications for use of PEEP include acute lung injury, acute respiratory distress syndrome (ARDS), cardiogenic pulmonary edema, diffuse pneumonia requiring mechanical ventilation, and atelectasis associated with severe hypoxemia.

Patients with acute lung injury and ARDS should be managed using lung-protective strategies including (a) low tidal volumes (6 mL/kg), (b) high-frequency ventilation, and (c) PEEP; the main goal is to maintain an acceptable level of PaO2but not necessarily a normocarbia (permissive hypercarbia may be allowed). The PEEP may be initiated at 5 cm H_2O and gradually increased to a maximum of 20 cm H_2O. This range allows optimization of arterial oxygenation as well as minimization of adverse effects. These strategies are critical for avoiding ventilator-induced lung injury. Volutrauma, which includes lung overinflation and overstretching of alveoli, barotraumas due to high inflation pressure and high PEEP, and atelectrauma, the repetitive opening and closing of recruitable alveoli, may be avoided. The decrease in VILI will certainly decrease production and release of inflammatory mediators, such as cytokines, TNFα, and interleukin-6, which may progress toward systemic inflammatory response and eventual multiple organ dysfunction.

Contraindications to the Use of PEEP.

PEEP is not a benign therapy and may be associated with adverse hemodynamic consequences that cannot be overlooked. There are potentially two absolute contraindications to the use of PEEP: hypovolemic shock and undrained high-pressure pneumothorax. Other relative contraindications include hypotension, hypovolemia, intracranial hypertension, unilateral or focal pulmonary disease, pulmonary embolism, and bronchopleural fistula.

Hypotension.

Positive-pressure ventilation has been shown to decrease venous return, and this effect is amplified by the addition of PEEP. Furthermore, if the patient is in a hypovolemic state, the hypotension is further accentuated.

P.13

Intracranial Hypertension.

PEEP should be used cautiously in these cases, because it may decrease the cerebral perfusion pressure (CPP) (the difference between mean arterial pressure and ICP) by either increasing ICP or decreasing mean arterial pressure (MAP). If it is necessary to use PEEP as in the cases listed above, then appropriate monitoring should be obtained to accurately measure both intracranial pressure and MAP. The use of a vasopressor agent may be indicated to maintain the MAP in these circumstances.

Unilateral/Focal Lung Disease.

This category includes patients who have severe pneumonia requiring mechanical ventilation. In this case the use of PEEP may worsen hypoxemia, because further ventilation of lung units already under compensatory hypoxic vasoconstriction will only exacerbate ventilation-perfusion mismatch.

Pulmonary Embolism.

PEEP may worsen hypoxemia secondary to pulmonary embolism. Increasing the alveolar pressure with PEEP may compress patent pulmonary vessels.

INTRINSIC PEEP

What Is It?

Intrinsic PEEP occurs when the alveolar pressure is greater than the atmospheric pressure at the end of normal passive exhalation. It is the end result when there is inadequate time for the lungs to empty secondary to high minute-volume ventilation, restricted expiratory flow, and/or high expiratory resistance.

What Causes It?

There are three scenarios in which intrinsic PEEP typically occurs.

1. The first scenario is referred to as high minute-volume ventilation secondary to a high respiratory rate and/or a large tidal volume. An increase in either variable has the same end result, the new breath being initiated before completion of the last exhaled breath, leading to intrinsic PEEP and air trapping.
2. Intrinsic PEEP is also commonly seen when expiratory flow is restricted. This occurs most frequently in patients with chronic lung disease and obstructive airway disease. The airways are constricted from limited expiratory flow as in bronchospasm, inflammation, or severely decreased pulmonary compliance such as occurs in ARDS.
3. The third type of intrinsic PEEP is seen with expiratory resistance. This may occur because of a kinked endotracheal tube or one with a narrowed diameter, secondary to accumulation of secretions. It may also present in cases in which the patient is agitated and/or in pain.

Complications of Intrinsic PEEP.

It is important for the clinician to determine the underlying cause of intrinsic PEEP because there are potentially devastating sequelae. Intrinsic PEEP may result in a significantly increased work of breathing, which may make it more difficult to wean a patient off a

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ventilator. Alveolar gas exchange may decrease, resulting in a lower arterial oxygen tension. This may be explained by an uneven distribution of intrinsic PEEP among lung units compared to when extrinsic PEEP is used. Finally, hemodynamic instability may occur. Intrinsic PEEP creates an increase in intrathoracic pressure, which decreases preload of both ventricles and decreases left ventricular compliance. Once this occurs, there is a resultant increase in right ventricular afterload and pulmonary vascular resistance, which leads to a decrease in cardiac output. P.14

Management of Intrinsic PEEP.

Various techniques may be utilized to minimize intrinsic PEEP. These include aggressive bronchodilator therapy, adequate pain control and/or sedation, and minimization of the inspiratory-to-expiratory ratio. In patients who are actively breathing, continuous positive airway pressure (CPAP) or extrinsic PEEP may be used to counter intrinsic PEEP. However extrinsic PEEP should be used with caution in this setting, as it may potentially worsen hyperinflation and compromise the patient's hemodynamic status.

TAKE HOME POINTS

- PEEP comes in two varieties: applied/extrinsic PEEP and auto/intrinsic PEEP.
- Positive pulmonary effects associated with PEEP include improvements in arterial oxygenation (PaO_2), recruitment of lung units, lung compliance, and increased functional residual capacity.
- The hemodynamic effects of PEEP include decreased venous return and biventricular preload, and subsequent decreased cardiac output.
- PEEP also has widespread multiorgan effects, including:
  - Liver: decreased splanchnic blood flow, resulting in liver congestion and hypoperfusion
  - Kidneys: decreased renal blood flow, resulting in decreased urine output, decreased sodium excretion, decreased creatinine clearance, and altered hormonal release
  - Brain: decreased cerebral venous return, resulting in increased ICP
  - Bronchial circulation: decreased bronchial blood flow, resulting in impaired lung repair
  - Thoracic lymph drainage: thoracic duct obstruction, resulting in impaired removal of edema from the lungs
- Key indications for use of PEEP include treatment of acute lung injury, acute respiratory distress syndrome (ARDS), cardiogenic pulmonary edema, diffuse pneumonia, and atelectasis with associated severe hypoxemia.
- Relative contraindications to use of PEEP include hypotension, hypovolemia, intracranial hypertension, unilateral or focal pulmonary disease, pulmonary embolism, and bronchopleural fistula. P.15
- Intrinsic PEEP occurs when there is inadequate time for the lungs to empty secondary to high minute-volume ventilation, restricted expiratory flow, and/or expiratory resistance.
- Negative sequelae associated with intrinsic PEEP include difficulty weaning patient from the ventilator, decreased alveolar gas exchange, and hemodynamic instability.
- Techniques to minimize intrinsic PEEP include bronchodilator therapy, administration of analgesics and sedatives, and addition of CPAP or extrinsic PEEP.

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4

A Variety of Techniques Provide Acceptable Anesthesia for Awake Intubation of the Airway; Ultimately, the Most Important Factors Are Operator Experience and Adequate Time

Chauncey T. Jones MD

Awake fiber-optic intubations are an integral component of known difficult airway management. Operator experience in managing these airways is the most important variable. However, preparation of the patient and the airway weigh heavily in achieving a successful awake intubation.

Preparation of the patient involves discussion of what to expect and how the patient can help the process go smoothly, and then mild sedation, as respiratory status permits. However, sedation should not be to the point of airway obstruction or disinhibition of the patient.

Preparation of the airway involves administering an antisialagogue (such as 0.2 to 0.4 mg glycopyrrolate) to block the secretory reflex and anesthetizing the entire route that the intubation will take, whether nasal or oral. This course can be divided into nasal and oral cavies; nasopharyngeal, oropharyngeal, and hypopharyngeal regions of the pharynx; or subglottic regions, which include the larynx and trachea. The innervation of these regions must be blocked for successful awake intubation.

INNERVATION

Nasal cavity innervation is derived from two branches of the trigeminal nerve (V), the ophthalmic (V1) and the maxillary (V2). The anterior ethmoid nerve (from V1) innervates the anterior aspects of the nasal cavity and septum. The posterior nasal cavity and septum are innervated primarily by the lateral posterior superior, lateral inferior posterior, and nasopalatine nerves of V2.

The sensory innervation of the oral cavity and muscles of mastication derive from the mandibular branch of the trigeminal nerve (V3). Muscles of the tongue are supplied primarily by the hypoglossal, CN XII. Its general sensory path is from the lingual nerve, a branch of V3, for the anterior two thirds, and by the glossopharyngeal nerve, CN IX, for the posterior one third. The hard and soft palate are innervated by the greater and lesser palatine nerves, respectively, which are branches from V2.

Sensory innervation of the entire pharynx and epiglottis is supplied primarily by the glossopharyngeal nerve, CN IX, and motor nerve supply is from the vegas nerve, CN X. The superior laryngeal nerve, derived from the vegas nerve, CN X, supplies sensory to the larynx above the true vocal cords as well as to the cricothyroid muscle. The recurrent laryngeal nerve, also from the vegas, CN X, supplies all other muscles of the larynx and sensation at the level of vocal cords and below. Recurrent laryngeal nerve also supplies sensory and motor components to the trachea. P.17

Several protective reflexes can be elicited with airway manipulation, including gag, glottic closure (laryngospasm), and cough. These reflexes can be blocked by either their afferent (sensory) and/or efferent (motor) components. Input for the gag reflex is the glossopharyngeal nerve, CN IX, and output is the vegas, CN X. Fibers for glottic closure reflex are superior laryngeal for afferent and both superior and recurrent laryngeal for efferent. Other reflexes that may also be encountered are bronchospasm reflex, secretory reflex, vomiting reflex, and cardiovascular reflex.

ANESTHETIZATION OF THE AIRWAY

Anesthetization of the airway for awake fiber-optic intubation can be achieved by a number of techniques and can be adjusted to the patient, the operator preference, and availability of supplies (Table 4.1). These often include a mixture of topicalization and nerve blocks. Topicalization methods include direct application of lidocaine or viscous lidocaine, aerosol spray, atomization, or nebulization (Table 4.2). Common nerve blocks include glossopharyngeal, superior laryngeal, and translaryngeal. Other blocks may be performed, such as maxillary and mandibular nerve blocks. However, these are more invasive with a lower risk/benefit ratio, and their relevant terminal nerves can easily be blocked topically.

TABLE 4.1 COMMONLY USED TOPICAL METHODS FOR AIRWAY BLOCKS

METHOD	LOCAL ANESTHETIC	NOTES
Nasal topical	1. 4% cocaine 2. 2% to 4% lidocaine or viscous lidocaine with 1% phenylephrine	Cocaine and phenylephrine cause vasoconstriction and decrease chance of epistaxis
Aerosol spray	Benzocaine	Risk of methemoglobinemia if maximum dose exceeded
Atomizer	4% lidocaine	Usually takes longer to work than direct topicalization
Nebulizer	4% lidocaine	Usually takes longer to work than direct topicalization

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Syringe	2% to 4% lidocaine	Can be injected through nasal trumpet or slowly deposited onto the posterior tongue and run inferiorly to reach the hypopharynx

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TABLE 4.2 COMMONLY USED LOCAL ANESTHETICS FOR AIRWAY BLOCKS

DRUG	CLINICAL USE	ONSET	DURATION (MIN)	MAXIMUM DOSE
Cocaine	Topical (nasal)	Slow	30-60	1.5 mg/kg
Benzocaine	Topical	Fast	5-10	200 mg
Lidocaine	Topical/nerve blocks	Medium/fast	30-60/60-180	4 mg/kg no epi; 7 mg/kg with epi

Nasal Cavity/Nasopharynx.

Anesthetization of the V1 and V2 branches of the nasal cavity can be achieved with cocaine or lidocaine. Cocaine also provides vasoconstriction to decrease the incidence of epistaxis. Phenylephrine can be added to lidocaine to provide the same result. A3:1 combination of 4% lidocaine with 1% phenylephrine will yield a 3%/0.25% solution that can be applied by inserting a cotton-tipped applicator. Alternatively, 2% viscous lidocaine can be mixed with phenylephrine to make a similar suspension and applied with a cotton-tipped applicator as well as serially increasing sizes of nasal airways. This technique not only anesthetizes and vasoconstricts the nasal cavity, it also lubricates and dilates it to the desired-sized endotracheal tube, usually 7.0. A nasal trumpet usually extends farther into the nasopharynx and provides better nasopharyngeal block than a cotton-tipped applicator. In addition, a slip-tip syringe can be used to inject several milliliters of 2% to 4% lidocaine through the nasal trumpet to further anesthetize the nasopharynx, oropharynx, and hypopharynx as the solution travels down the pharynx.

Acontraindication to this technique is trauma to the nasal cavity. Relative contraindications include uncontrolled hypertension and coagulopathy.

Oral Cavity/Oropharynx.

The oral cavity (mandibular nerve V3) is not usually affected by pain during oral awake fiber-optic intubation and thus does not have to be blocked, but usually it is at least partially blocked by several of the methods utilized for blocking the oropharynx. The oropharynx (glossopharyngeal nerve, CN IX) does need to be blocked. This can be achieved with any number of procedures: gargling several milliliters of 2% to 4% lidocaine and then expectorating the solution; using an aerosol spray such as benzocaine spray; atomization; nebulization; or applying lidocaine-soaked pledgets directly to the oropharynx. If oral awake intubation is planned, prevention of the patient biting the endotracheal tube or scope is needed. The muscles of mastication usually are not blocked, but rather, a bite block or oral airway is inserted.

Adirect glossopharyngeal nerve block can be performed by either an intraoral or peristyloid approach. For the intraoral approach, adequate mouth opening and topicalization for the patient to tolerate the block are needed. The tongue is distracted in an anteroinferior direction, which can be done with a laryngoscope blade or tongue depressor. The base of the tonsillar pillar is identified and a 25-gauge spinal needle is advanced submucosally at the caudal-most portion of the pillar. Aspiration before injection to rule out intravascular location is important, given the close proximity of the internal carotid artery. Then 5 mL of local anesthetic can be injected. The same process is repeated on the other side. The glossopharyngeal block is sometimes avoided by junior practitioners, however, there are clinicians who have used it routinely for many years with great success.

P.19

The peristyloid approach requires access to the lateral neck and ability to identify bony landmarks. The midpoint of a line between the mastoid process and the angle of the mandible is identified. Deep to this should be the styloid process, which can sometimes be felt with deep palpation. The overlying skin is injected with a wheel of local anesthetic and a 22-guage needle is inserted perpendicular to the skin. This is advanced until the styloid process is contacted, usually in 1 to 2 cm. The tip of the needle is then walked off the process posteriorly. Once contact is lost, and after negative aspiration for blood, 5 to 7 mL of local anesthetic is injected. The process is repeated on the contralateral side.

Contraindications for glossopharyngeal nerve block include overlying infection and coagulopathy.

Hypopharynx.

The hypopharynx is usually anesthetized by the methods used for the oropharynx as the local anesthetic migrates inferiorly. In addition, a syringe can be used to trickle anesthetic down the posterior tongue and onto the hypopharynx. If an oral airway is in place, it can be used as a conduit to direct local anesthetic to the hypopharynx. Local anesthetic can also be injected under direct visualization through the port of the fiber-optic scope.

Larynx.

The larynx may be reached with local anesthetic using the same methods as for anesthetizing the oropharynx and hypopharynx, such as an atomizer, a nebulizer, or by trickling anesthetic down the posterior tongue. Local anesthetic can be deposited on the larynx through the fiber-optic scope as well.

Asuperior laryngeal nerve block can be used to block the nerve directly. The patient is placed in a supine position and the neck fully extended. The hyoid bone is identified by careful palpation. Once identified, it is displaced toward the ipsilateral side of injection and a 25-gauge needle is advanced until it makes contact with the greater cornu of the hyoid bone. The needle is then walked off inferiorly and advanced 2 to 3 mm to place the tip between the thyrohyoid membrane laterally and the laryngeal mucosa medially. After negative aspiration for air and blood, 2 to 3 mL of local anesthetic is injected. The process is repeated on the contralateral side. Alternatively, the superior laryngeal nerve can be blocked topically by applying 4% lidocaine on a pledget to the bilateral piriform fossa for several minutes.

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Contraindications include coagulopathy and an inability to extend the neck.

Vocal Cords and Trachea.

The vocal cords and trachea may be anesthetized by several of the procedures mentioned previously as anesthetic is aspirated or inhaled. However, they are more reliable and completely blocked by direct injection of local anesthetic through a fiber-optic scope or translaryngeal block.

Atranslaryngeal or transtracheal nerve block is used to anesthetize the true vocal cords and the trachea. It can also block some supraglottic structures as it is coughed out through the vocal cords. The patient is placed in a supine position and the neck extended. The cricothryoid membrane is identified in its midline location. The overlying skin is injected with local anesthetic. A22-gauge needle on a 10-mL syringe with 3 mL of 4% lidocaine is introduced perpendicular to the skin and advanced while continuously aspirating for air. Positive air aspiration indicates that the needle tip is in the trachea. Care must be taken not to advance into the posterior wall of the trachea. Once correct position is confirmed, the local anesthetic is injected as the patient inhales and the needle is quickly removed, as injection will elicit a cough. Some operators prefer to use a catheter-over-needle technique, with only the catheter left in place while injecting.

Performing a series of topical and/or regional nerve blocks to anesthetize the involved branches of the V1, V2, V3, glossopharyngeal, and vegas nerves as described above will make the experience of awake fiber-optic intubation more tolerable for the patient and significantly increase the chance of a successful procedure.

TAKE HOME POINTS

- Watch as many of these awake airway procedures as possible—operator experience is one of the most important variables in providing anesthesia for the airway smoothly and efficiency, and every experienced anesthesia provider has his or her own favorite techniques.
- Always extend as much empathy and patience to the patient as possible, remember that even when everything goes smoothly, this is not the easiest thing in the world for anybody, most of all the patient. If he asks to stop a minute to catch his breath, stop a minute.
- A variety of sedation techniques will work, but don't try to substitute heavy sedation for inadequate topical anesthesia. Be aware that droperidol will make the patient appear sedated and compliant, but the anesthesia provider has to be prepared for the patient to later describe feeling like a zombie that couldn't run away.
- Most anesthesia providers use a lidocaine nebulizer as well as blocks.
- The other important "ingredient" in providing anesthesia to the airway is *time*!
- Check with the surgeons (particularly the ENT surgeons) before doing a transtracheal block; they will sometimes ask you not to broach the trachea with a needle in advance of their procedure.

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5

An Awake Intubation Should Not Be a Traumatic Experience for the Patient

Chauncey T. Jones MD

To some, the phrase “awake intubation” may conjure up thoughts of medieval torture tactics. Indeed, intubations are commonly performed without sedation during code situations on semiconscious patients. However, in the operating room and in other controlled situations, awake intubations should not be a traumatic experience for the patient.

Anesthesiologists must possess sound management skills with difficult airways. Most American Society of Anesthesiologists (ASA) closed claims studies are related to inadequate oxygenation or ventilation. Part of the preoperative evaluation by the anesthesiologist is to determine how endotracheal intubation can be safely executed. In most instances, induction of anesthesia followed by asleep intubation is reasonable. In certain patients, however, awake intubation may be safest. Indeed, awake intubation is part of the ASA Difficult Airway Algorithm.

INDICATIONS FOR AWAKE INTUBATION

The decision for an awake intubation may be based on a history of difficult ventilation and/or intubation during prior anesthesia. The indication for surgery may require awake intubation. For example, you may be asked to anesthetize a patient with severe cervical spine instability, requiring awake intubation and awake positioning; or a patient might have a head and neck mass that is partially obstructing the airway or compressing the trachea. Information obtained from history and physical examination is also useful and may reveal congenital syndromes, abnormal facial structure such as micrognathia, morbid obesity, obstructive sleep apnea with poor respiratory reserve, severe aspiration risk, or significant hemodynamic instability.

NASAL VERSUS ORAL

Most intubations are performed orally, unless there is a contraindication. Nasal intubations are used for oral surgeries in which an endotracheal tube would hinder the operation, or if the mandible is to be wired closed. Nasal intubation can be performed on patients with poor mouth opening and to aid patient comfort and oral hygiene if immediate postoperative extubation is not planned. Fiber-optic nasal intubation is often technically easier than oral fiber-optic intubation, and a blind nasal intubation can also be employed. P.23

Contraindications to nasal intubation include coagulopathy, abnormal nasal anatomy, sinusitis, and facial trauma where there may be a basilar skull fracture. Complications of nasal intubation include epistaxis, trauma to nasal structures, sinusitis with possible bacteremia, and cribriform plate perforation resulting in cerebral injury/death.

TECHNIQUES

There are multiple ways to perform an awake intubation. Common to all awake techniques is preparation of the patient and the airway. Letting the patient know what to expect is a key component to success. Mild sedation, as respiratory status permits, is also helpful. Preparation of the airway includes administration of antisialagogues and anesthetization of the airway with topical and regional techniques (see Chapter 4).

Currently, the most commonly employed technique is oral or nasal fiberoptic intubation. Keep in mind that fiber-optic scopes are not always available and are not a requirement for awake intubation. Other options include blind nasal intubation, laryngoscopy, or some combination of techniques. Keep in mind that an ultimate option may be a surgical airway.

FIBER-OPTIC INTUBATION

First, become familiar with and test the equipment. Verify adequate light source, focus, and white balance if applicable. Scope manipulation is most efficient when the shaft is devoid of slack and held taught. All scope maneuvers are controlled by the dominant hand with the up/down lever, by rotating the wrist, and scope advancement or withdrawal. Obtain suction for the scope. Test the cuff on the endotracheal tube (ETT), usually 7.0 or smaller, and soften it in warm solution. Lightly lubricate the shaft of the scope to facilitate easy passage of the ETT. To prevent fogging, antifog solution can be applied to the scope tip, or the tip can simply be placed in warm saline. Patient position can be sitting, semirecumbent, or supine with the head in neutral position.

For nasal intubation, insert the endotracheal tube into the previously anesthetized, dilated, and vasoconstricted nasal passage (see Chapter 4) until the tip is in the oropharynx. This serves as a conduit to introduce the fiber-optic scope and guide it toward its destination. Alternatively, some anesthesiologists prefer to have the scope tip threaded through the ETT, but introduce the scope tip first and let it serve as a guide for the ETT—similar to the Seldinger technique for inserting central venous catheters.

Once the scope tip is in the oropharynx, the key is to stay midline, maneuver the scope anteriorly, and identify relevant structures. Anterior displacement of the tongue is helpful. This can be done by having an assistant grasp the tongue with gauze. Ideally, with advancement of the scope, one should see posterior tongue, epiglottis, arytenoids, vocal cords, trachea, and carina. Ask the patient to take a deep breath or pant just before traversing the vocal cords. The ETT can then be advanced into the trachea over the scope while maintaining visualization of the carina. The ETT may sometimes meet resistance as it contacts the arytenoids. If this is encountered, rotate the ETT 90 degrees. Distal ETT location can be confirmed with the scope. With the scope tip at the carina, use the nondominant hand to hold the scope at the proximal end of the ETT. Withdraw the scope until the distal end of the ETT can just be seen through the scope and stop. The distance from the nondominant hand to the proximal end of the ETT is the height of the ETT tip above the carina. P.24

Oral fiber-optic intubations are similar to nasal fiberoptic bronchoscopy (FOB) except that an oral airway or bite block should be used to prevent operator injury or scope damage. The technique is often more technically difficult because a more acute angle must be managed. A jaw thrust may help.

BLIND NASAL

The blind nasal technique can be used when blood or secretions impede visualization with the fiber-optic scope. Prepare the airway as described in

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Chapter 4. During the procedure, have the patient pant. Using a 7.0 ETT, introduce it through the nasal passage and slowly advance. Patient respirations can be heard through the ETT. Advance toward the respirations as a guide. If respiratory sounds through the ETT disappear, advancement into the esophagus is likely and the ETT should be withdrawn and redirected. The ETT can be rotated to maintain a midline position. If there is no a concern for cervical spine instability, the neck can be extended or flexed to help facilitate intubation. Special endotracheal tubes are available that have a drawstring to help manipulate the tip of the endotracheal tube.

If blind nasal intubation is unsuccessful and there is no contraindication to oral instrumentation, the technique can be converted from blind to oral by using laryngoscopy and Magill forceps to help aid the nasal intubation.

AWAKE LARYNGOSCOPY

Awake direct laryngoscopy and intubation can be performed on patients after anesthetization of the airway, which will also attenuate gag and cough reflexes (see Chapter 4). Once the vocal cords are identified, intubate as the patient inhales. Awake indirect laryngoscopy and intubation can be done in a similar manner with the Bullard scope. The Bullard scope can be helpful for patients with a small mouth opening and anterior airways.

TAKE HOME POINTS

- Awake intubation is part of the ASA Difficult Airway Algorithm.
- Indications for awake intubation include cervical spine instability, head and neck mass, congenital syndromes, morbid obesity, or severe aspiration risk.
- Proper preparation of the patient and airway is paramount.
- Remember that awake intubations do not have to be via fiber-optic scope—other techniques include blind nasal placement of the ETT and direct laryngoscopy.
- Do *not* "torture" the patient—if he or she is crying or asking for you to stop, *stop* and reassess your technique and airway preparation.

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6

A High Inspired Concentration of Oxygen is Contraindicated in Certain Circumstances

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The concept of oxygen toxicity in certain circumstances is well known among medical professionals. To anesthesia providers, however, the most common errors concerning oxygen toxicity are in the following assumptions: (a) the benefits of oxygenation will outweigh its potential toxic effects; (b) it is irrelevant in the emergency department (ED) or the operating room (OR), where long-term exposure to high concentration oxygen is rare; and (c) damage from high-concentration oxygen is mild to moderate.

Indeed, the importance of oxygenation cannot be exaggerated, and the most well-known toxicities take days to develop clinical signs after exposure to high-concentration oxygen (hyperbaric oxygen is not discussed in this chapter). Certainly, a high inspired concentration of oxygen is warranted in some situations—for instance, if a patient who is being turned prone and placed in pins becomes extubated, the anesthesia providers have several minutes longer to turn him back over and get him reintubated if he is being ventilated with 100% oxygen as opposed to an intentionally lowered FiO2. In the past decades, however, we have come to realize that oxygen toxicity can have very acute and significant effects in multiple clinical scenarios such as cardiopulmonary resuscitation (CPR), cardiopulmonary bypass (CPB), and organ transplantation. It is clearly an inevitable issue confronting every anesthesia provider in the intensive care unit (ICU), the OR, or the ED. Avoiding oxygen toxicity may not only avoid tissue damage, it may also reduce mortality.

We will briefly discuss several clinical scenarios of oxygen toxicities. Among them, pulmonary injuries secondary to prolonged exposure and retinopathy of prematurity might be viewed as “chronic” toxicity of high concentration oxygen, whereas the others represent acute detrimental effects, happening minutes to hours after exposure to high oxygen concentrations.

PROLONGED EXPOSURE (ICU VENTILATION)

The damage resulting from prolonged exposure to high oxygen concentrations involves several organ systems, including the lungs (fibrosis, hemorrhage, atelectasis, edema, and hyaline membrane damage), the central nervous system (CNS) (convulsion, paralysis, and death), the testes, and blood cells (hemolysis). Pulmonary injuries are the most common sequelae in human beings. The mechanisms are still not fully understood. It is generally held P.27 that oxygen toxicity in human beings can be avoided if 100% O_2 is taken for less than 12 hours, or 80% for less than 24 hours, or 60% for less than 36 hours. This is relevant to patients with respiratory failure who need prolonged respiratory support. As a general rule, oxygen concentration should be reduced to the lowest possible level. Unfortunately, this often is a challenging task when patients need a higher FiO2 for oxygenation. It is important to optimize other factors, such as positive end-expiratory pressure (PEEP) and ventilation modes before increasing oxygen concentration is considered.

RETINOPATHY OF PREMATURITY

High oxygen concentration was associated with increased incidences of retinopathy of prematurity (ROP) in the 1940s. During the 1950s and 1960s, the oxygen tension of incubators was decreased. The incidence of ROP decreased as expected, but mortality seemed to increase. Based on recent studies, the 2002 American Association for Respiratory Care (AARC) guideline recommended that for infants less than 37 weeks' gestation, PaO_2 should be kept lower than 80 mm Hg.

CONGENITAL HEART DISEASES

Inhalation of oxygen may substantially reduce pulmonary vascular resistance in infants with pulmonary hypertension. If there is an intracardiac shunt, such as in hypoplastic left heart or a single ventricle, the balance between pulmonary and systemic circulations may be disrupted, causing left-to-right shunting and potentially devastating consequences within minutes. This is extremely important in the induction of anesthesia when denitrogenation with 100% oxygen would otherwise be routine.

ACCENTUATION OF HYPERCAPNIA

Giving oxygen to patients with compensated respiratory failure should be done cautiously, as the treatment may worsen hypercapnia. This rapid consequence is more a “physiologic” than a “toxic” effect of high-concentration oxygen. Its mechanisms include decreased stimuli on the peripheral chemoceptors, worsened V/Q mismatch, and increased CO2-hemoglobin dissociation.

ABSORPTION ATELECTASIS

In patients with a low regional V/Q ratio in the lungs, the involved alveoli are kept open mainly by nitrogen. Inhalation of high-concentration oxygen maximizes the nitrogen partial pressure gradients between alveoli and palmary capillaries, enhances absorption of nitrogen in the alveoli, and completely collapses the poorly ventilated alveoli. Most often in the elderly, the resultant increased shunting may be as high as 11% and take place as rapidly as 30 P.28 min after inhalation of pure oxygen.

CARDIOPULMONARY RESUSCITATION

Numerous animal experiments and clinical studies suggest that 100% oxygen used during CPR may be detrimental, especially in neonates. Davis et al. analyzed the outcomes in five separate clinical trials (1,302 cases total) that compared resuscitations with either 100% oxygen or room air in neonates with asphyxia. Although no original study found any difference in mortality rates between groups, the pooled data revealed a significant benefit of room air resuscitation for infants (relative risk of 0.71). It is estimated that one death would be prevented for 20 patients if air, instead of 100% oxygen, were used

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for resuscitation. During the 2005 International Consensus Conference on Cardiopulmonary Resuscitation, it was acknowledged that excessive oxygen given during resuscitation may cause oxidant injury and should be avoided, especially in premature infants. However, the current evidence was weighed as insufficient to specify the concentration of oxygen to be used initially, or to indicate a change in the oxygen concentration afterward. In the 2005 American Heart Association (AHA) guidelines, the standard approach to resuscitation is still to use 100% oxygen. However, it accepts use of an oxygen concentration of less than 100% (or room air) for neonatal resuscitation.

In contrast, the existing evidence of oxygen toxicity in adult CPR is much less compelling. The major concern is still focusing on airway, breathing, and circulation (ABC), and giving 100% oxygen during resuscitation remains preferred.

CARDIOPULMONARY BYPASS

High oxygen concentration may be toxic during CPB. Pizov et al. reported in a clinical study that ventilation with 100% oxygen during surgery and insuflation of the lungs with 100% oxygen during CPB was associated with a lower PaO_2/FiO_2 ratio and a higher TNF level in bronchoalveolar lavage, as compared to the control group that was treated with air instead of 100% oxygen. Patients with pre-existing hypoxia/cyanosis may be even more vulnerable to oxygen damage. In animal models, Morita et al. demonstrated that CPB primed and run with the conventional hyperoxic perfusate was associated with more reperfusion injuries and poorer myocardial functions compared with those with hypoxic or normoxic perfusate. However, there are no further studies exploring whether avoiding hyperoxic ventilation and lung insuflation has any significant effects on clinical outcome. Aconsensus on this issue has not been reached in clinical practice.

ORGAN TRANSPLANTATION

Research data supports the thought that hyperoxic reperfusion may exacerbate ischemia/reperfusion injury in organ transplantation. Here are a couple of examples. Detterbeck et al. demonstrated that hyperoxic reperfusion in lung transplantation in animals decreased PO_2 and shortened survival time. In another animal experiment, hyperoxic reperfusion after 30 minutes of complete renal ischemia was associated with sustained elevation of creatinine and blood urea nitrogen (BUN), while normoxic reperfusion caused only transient elevation. However, so far there is no established guideline that defines an optimal oxygen tension for initiation of reperfusion in organ transplantation.

OTHER ISSUES

It has been reported that inhalation of high-concentration oxygen causes vasoconstriction of coronary and retinal arteries. In patients who have received bleomycin, the risks for oxygen toxicity are significantly elevated.

The mechanisms for oxygen toxicity are associated with excessive production of free oxygen radicals. Many antioxidants have been shown to have promising effects in the laboratory, but none has been used successfully in the clinical realm to attenuate oxygen toxicity from prolonged exposure or during ischemia/reperfusion.

TAKE HOME POINTS

- Prolonged exposure to high-concentration oxygen may cause significant tissue injuries, such as pulmonary fibrosis, hemorrhage, edema, hyaline membrane, and ROP.
- Acute and devastating oxygen toxicity should be anticipated in the following situations: (a) patients with chronic obstructive pulmonary disease (COPD), (b) patients with regional decreased V/Q ratio in the lungs, (c) patients with pulmonary hypertension and congenital heart diseases, and (d) neonatal CPR patients.
- High oxygen concentration may be toxic in the following situations: adult CPR, lung injuries in CPB, aortic cross-clamping/declamping, and organ transplantation.
- Antioxidant treatments are still experimental. Therefore, identifying patients with increased risks and avoiding oxygen toxicity is important.

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7

Understand and Take Advantage of the Unique Properties of Helium for the Management of the Compromised Airway

J. Mauricio Del Rio MD

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A 22-month-old girl began coughing while eating some sunflower seeds. Shortly thereafter, she appeared to be gasping for air. Her mother performed back blows and called for an ambulance. In the emergency department she was awake, alert, and sitting forward with obvious stridor, increased work of breathing, frequent coughing, and drooling. The patient was transported emergently to the operating room for airway management. When you arrive, her vital signs are blood pressure 119/76 mm Hg, respiratory rate 66 breaths/min, heart rate 160 beats/min, and pulse oximetry 90% with some blow-by oxygen. Examination of the chest reveals mild retractions and faint bilateral wheezing. While awaiting the arrival of the ENT surgeon, the patient has intermittent episodes of perioral cyanosis, repetitive coughing, and worsening stridor. How would you manage this patient?

Since 1934, when helium was first described as therapy for upper-airway obstruction and asthma exacerbation, it has been advocated for the treatment of a wide variety of respiratory conditions. However, the optimal application and effectiveness of the helium—oxygen mixture (heliox) for managing air flow obstructive disorders has not been clearly supported by the evidence. It is important to understand the mechanism of action and the scenarios in which this therapy is and is not warranted.

PHYSICAL PROPERTIES OF HELIUM

Helium is a colorless, odorless, tasteless, monatomic element. It is the first noble gas in the periodic table, with an atomic number of 2 and an atomic weight of 4 g/mol. Helium is the second most abundant element in the universe but relatively rare on earth, representing 0.000524% of the atmosphere. With the lowest boiling and melting points of the elements, helium exists as a gas except in extreme conditions. In natural conditions, most helium is created by the radioactive decay of heavier elements and is trapped within natural gas in concentrations up to 7%. Helium is extracted from natural gas by fractional distillation. At a temperature near absolute zero the liquid helium has virtually no viscosity, a property called superfluidity.

As a consequence of its low atomic weight, the density of helium is the lowest of any gas except hydrogen; unlike hydrogen, it is nonflammable. Helium's P.32
low density allows CO_2 to diffuse five times more rapidly through a helium—oxygen mixture than through air or oxygen. Being biologically inert, helium has no direct pharmacologic effects and has no intrinsic bronchodilatory or anti-inflammatory properties. For the same reasons, it has no known toxic effects, even when used for extended periods of time.

HELIOX

Heliox is a mixture of helium and oxygen gases. Standard heliox cylinders contain 80:20 heliox, though heliox also is also available in 70:30 and 60:40 mixtures. Commercial-grade heliox is available in H-size cylinders, which contain approximately 1,200 L of gas, at approximately 2,200 psi. If the patient requires supplemental oxygen, this limits the helium concentration that can be administered.

HISTORICAL USES OF HELIOX

Helium was discovered in 1868 by a French astronomer, Janssen, as a bright yellow line in the spectrum of the sun during a solar eclipse. The new element was first isolated on Earth in 1895 by a British chemist, Ramsay. At the time of its discovery, helium had no practical use. Heliox was first used physiologically to create breathing environments for diving. It reduced the formation of nitrogen bubbles responsible for decompression illness in divers and allowed them to work at depth for extended periods of time. In 1934, Barach noted the biologic inertness of heliox; he found no ill effects in mice exposed to a high helium concentration for 2 months. Those experiments were followed by clinical investigations of its potential benefits for various disorders, including asthma and infant airway obstruction. Barach noted evidence of relief within 6 to 10 breaths, with dyspnea returning in as little as 3 to 4 breaths after discontinuation of heliox. After the late 1930s, little was reported about clinical applications of heliox until 1979. Since then, there has been a resurgence in the investigation of its potential uses.

PHYSICS OF FLUID FLOW WITH HELIOX

The behavior of a fluid in flow is related to two intrinsic properties of the fluid: density and viscosity. The density of a substance is its mass per unit volume. Because 1 gram molecular weight of a gas occupies a volume of 22.4L under standard conditions, gas density is its gram molecular weight divided by 22.4 L. The density of helium is thus 0.179 g/L. Oxygen's molecular weight is 32 g, so its density is 1.43 g/L. Viscosity is an internal property of a fluid. Viscosity causes resistance to flow. A fluid with a high viscosity strongly resists flow.

Gas flow can vary from laminar at one end of the spectrum to turbulent at the other. Under normal circumstances, gas flow in many parts of the respiratory P.33
tract is largely laminar and follows the Hagen—Poiseuille law. This law states that the fluid flow rate (Q) through a straight tube of uniform bore is proportional to the pressure gradient (ΔP) and the fourth power of the radius (r), and is related inversely to the viscosity of the gas (μ), and the length (l) of the tube:

$$\dot{Q} = \frac{\Delta P \pi r^4}{8 \mu l}$$

Flow varies directly with pressure, so quadrupling the pressure will quadruple the flow; it also varies as the fourth power of the radius, so doubling the radius will increase flow by a factor of 16. Note that laminar flow is viscosity-dependent and density-independent. Because the viscosity of helium, oxygen,

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and air is similar and laminar flow velocity is independent of density, heliox has no effect on areas of laminar flow.

Turbulent flow occurs in constricted passages and follows a different law:

$$\dot{Q} = k\sqrt{\frac{\Delta P}{\rho}}$$

Compared with laminar flow, turbulent flow is less efficient. Flow increases with the square root of the pressure gradient, so quadrupling the pressure gradient merely doubles the flow. Decreasing the density will also increase the flow. As flow velocity decreases and/or airway resistance increases, there is a critical level at which the flow pattern changes from laminar to turbulent. This transitional zone is defined by Reynolds number (Re):

$$Re = \frac{VD\rho}{\mu}$$

where *V* represents the mean velocity of the gas, *D* represents airway diameter, π represents the gas density, and μ is the viscosity of the gas. The Reynolds number is the ratio of inertial forces (density-dependent, viscosityindependent) to viscous forces (viscosity-dependent, density-independent). Laminar flow occurs at low Reynolds number (Re <2,100) when viscous forces are dominant and is characterized by smooth, constant fluid motion. Turbulent flow occurs at high Reynolds number (Re >4,000) and is dominated by inertial forces, producing random eddies, vortices, and other flow fluctuations. Transitional flow lies between these values and displays qualities of both types of flow.

While breathing air, there is an insignificant difference in relative gas density at various oxygen concentrations. However, by replacing nitrogen with helium, there is a wide variability in the density between the different oxygen concentrations. As a result, the Reynolds number is decreased, making flow more likely to be laminar. With extremely turbulent flow patterns, flow rate becomes dependent on gas density according to the Bernoulli equation: P.34

$Q = (2\,\delta\, p/\pi)^{1/2}$

Therefore, by using helium as the carrier gas, heliox will reduce the density of the inspired gas and will provide a higher flow rate even if the flow remains turbulent. This results in a lower resistance to gas flow, allowing for increased bulk flow through high-resistance airways and converting some or all of the turbulent flow to laminar flow. In the end, there is increased oxygen flow and decreased work of breathing.

CLINICAL USE OF HELIOX

Air flow patterns in the pulmonary system are products of the physical conditions in the airway (e.g., diameter, anatomic shape, branching, and smoothness of airway lining) and the composition of the inhaled gas. Air flow in the lung periphery is primarily laminar because of the large cross-sectional surface area through which the gas flows. Conversely, air flow in the larger upper airways is turbulent, with relatively high flow and relatively small cross-sectional surface area. The flow characteristics in the airways may also vary depending on the inspiratory and expiratory flow rates. During normal breathing, a transition from turbulent to laminar flow occurs from the trachea to around the second generation of bronchi. However, in patients with airway obstruction, turbulent flow occurs randomly and more frequently, even at low respiratory and flow rates. This leads to increased work of breathing. In patients with increased airway resistance, heliox administration will reduce resistance to flow, which will lead to decreased work of breathing.

ADMINISTRATION

Helium has a very high diffusion coefficient, which requires it to be stored in special, tightly sealed containers. In order to achieve a sufficient helium concentration (>50%) to gain maximal advantage from the helium, the administration system needs to be “helium-tight”. Therefore, the most effective heliox systems are closed systems. The system should be also high-flow, with sufficient flow to meet or exceed the patient's minute volume and peak inspiratory flow, and to minimize dilution with ambient air. Flushing a high flow of heliox into the patient's airway is expensive and can be wasteful. Hence, the delivery method should include a reservoir and an on-demand delivery system that minimize total flow and helium requirements.

Heliox can be used in patients who are breathing spontaneously, but also in the settings of both invasive mechanical ventilation (e.g., with tracheal intubation) and noninvasive mechanical ventilation. P.35

BENEFICIAL RESPIRATORY EFFECTS

- Reduces transpulmonary pressure requirement
- Increases tidal volume
- Improves homogeneity of gas distribution
- Improves elimination of CO_2
- Improves delivery of aerosolized medications

POSSIBLE CLINICAL APPLICATIONS

- Upper-airway obstruction
- Postextubation stridor
- Croup
- Bronchiolitis
- Asthma exacerbation
- Chronic obstructive pulmonary disease (COPD) exacerbation
- Pulmonary function testing

Being used for these purposes, in different clinical trials, heliox therapy has been shown to:

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- Relieve stridor, reduce work of breathing, and reduce respiratory distress
- Allow time to assess patient's airway and clinical status
- Allow time to prepare for further interventions
- Act as a therapeutic bridge to allow time for definitive treatments to become effective
- Act as an alternative to effective treatments when they are contraindicated
- Prevent intubation in patients with impending respiratory failure
- Decrease the need for intubation or reintubation in perioperative settings
- Be clearly useful for upper-airway obstruction
- Provide greater benefit in patients with a greater degree of respiratory distress
- Shorten the length of perinatal ICU stay in infants with bronchiolitis
- In croup, improves respiratory distress, though not better than conventional therapies

FOR ASTHMA EXACERBATION

- Conventional treatment is sufficient for the majority of patients.
- First-line use of heliox is not warranted for most patients.
- Heliox is more effective for patients with the most severe exacerbations and air flow obstruction.
- The beneficial effects seem to be most efficacious when heliox is used early (before 24 h).
- Early use may decrease dyspnea and improve gas exchange, especially when implementing conventional therapies that require hours for full effect (e.g., steroids). P.36
- Heliox can be used as a temporizing measure to prevent intubation.
- Heliox has a relatively safe treatment profile, and clinical benefits should be rapid.

FOR SEVERE COPD

- Heliox may improve alveolar ventilation and gas transfer (decreasing $PaCO_2$).
- Heliox may reduce the need for intubation.
- Heliox should be considered in individual cases when intubation is required but would be undesirable.
- Wide-scale use of heliox cannot be recommended based on clinical evidence.

RISKS OF HELIOX USE

Hypoxemia/Anoxia.

There is a possibility of delivering a gas mixture that contains <21% oxygen. This risk is reduced by administering only heliox that contains at least 20% oxygen. The cylinder should be mixed and tested before use. The use of an oxygen analyzer in line with the gas output provides monitoring to prevent hypoxemia. There have been reports of hypoxia during heliox administration in preterm infants who have a history of bronchopulmonary dysplasia and subglottic stenosis. This could be related to the reduction of lung volume and the increased intrapulmonary shunt.

Volutrauma/Barotrauma.

Heliox's flow is faster than that of air or oxygen. Consequently, when using a flow meter calibrated for oxygen or air, a correction factor (based on the helium concentration) must be applied to correct for the difference in flow rate. The heliox correction factors are generally rounded off to 1.4 for 60:40, 1.6 for 70:30, and 1.8 for 80:20. Thus, when an oxygen flow meter delivering 80:20 heliox reads 10 L/min, it is actually delivering 18 L/min. If a system delivers more than the set volume, there is a risk of volume-induced injury, pressure-induced injury, or hypocarbia. This is of particular concern with closed systems that are not designed for heliox administration.

Inadequate Delivery of Aerosolized Medications.

Poorly adjusted flow of heliox in a nebulizer can result in a subtherapeutic dose or can increase the dose delivered to the lungs above the intended levels.

Hypothermia.

Hypothermia has been associated with hood administration of heliox to infants. Heliox has a high thermal conductivity with consequent risk of hypothermia when the gas temperature is <36°C, especially when it is administered for long periods. The risk of hypothermia can be avoided with adequate warming and humidification of the inhaled gas.

TAKE HOME POINTS P.37

- Heliox is clearly useful in patients with upper-airway obstruction.
- It can relieve stridor, reduce work of breathing, and reduce respiratory distress.
- It may decrease the need for intubation or reintubation in perioperative settings.
- It can allow time for other interventions or therapies to become effective.
- Heliox must be administered with devices designed for that purpose.
- Definitive clinical evidence is needed to elucidate its role.

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Editors: **Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.**
Title: ***Avoiding Common Anesthesia Errors, 1st Edition***

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8

Remember that There Are Special Considerations Involved with both Intubation and Chronic Airway Management of Burn Patients

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Burns can result from thermal, chemical, electrical, or radiation injury. Thermal injury is most common, accounting for up to 90% of all burn insults. Carbon monoxide poisoning is the most frequent cause of death during and immediately after a fire. If the patient survives the first few hours, morbidity and mortality are related to the total body surface area involved, the depth of the burn, and the patient's age. The likelihood of airway compromise increases with more serious burns.

Characteristics of major burns include:

1. Partial-thickness burns involving >25% of total body surface area (TBSA) in adults
2. Full-thickness burns involving >10% of TBSA
3. Presence of inhalational injury
4. Involvement of the face, eyes, ears, hands, feet, or perineum causing impairment
5. Caustic chemical burn etiology
6. High-voltage electrical burns
7. Burns in patients with coexisting debilitating disease

Characteristics of moderate burns include:

1. Partial-thickness burns involving 15% to 25% of TBSAin adults
2. Full-thickness burns involving 2% to 10% TBSA

Burns range from first- to fourth-degree, depending on the depth of the destroyed skin. First-degree burns are limited to the epidermis. Second-degree burns extend into the dermis and are divided into superficial and deep partial-thickness burns based on upper or lower dermis involvement, respectively. Third-degree burns include the entire epidermis and dermis, and fourth-degree burns extend into muscle, fascia, or bone.

Total body surface area of burned skin is estimated using the “rule of nines.” Body parts are divided into allotments or multiples of 9% (Table 8.1).

ACUTE MANAGEMENT

Burn patients with inhalational injury may require endotracheal intubation and ventilatory support at the site of the fire. If the patient has an endotracheal tube (ETT) on arrival to the hospital, do not change the tube; it may be difficult to get another one in place. It is not unusual that burn patients present asymptomatically. These patients have the potential to become problematic because of subsequent sequelae of burn injury that lead to a challenging airway. Burn patients develop excessive amounts of upper- and lower-airway edema as a result of mucosal destruction and capillary leakage that is further exacerbated by fluid resuscitation. Airway edema and narrowing continues for 12 to 24 hours after the initial burn insult. Athorough history and physical are helpful in determining which patients are candidates for early intubation. While assessing the patient, administer oxygen by face mask and monitor oxygen saturation with pulse oximetry.

P.39

TABLE 8.1 RULE OF NINES

Entire head and neck	9%
Each arm	9%
Anterior upper/lower trunk	18%
Posterior upper/lower trunk	18%
Each lower extremity	18%
Genitals and perineum	1%

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Each hand	1%

History.

A burn injury that occurs in an enclosed space with highly combustible material is very suspicious for inhalation injury. Patients who report coughing, drooling, hoarseness, dysphonia, and dysphagia are at risk of airway swelling.

Physical Exam.

Facial burns and the presence of oral soot correlate highly with airway edema and impending airway compromise. Other signs include singed facial hair, soot in the nose, stridor, and respiratory distress.

Diagnosis.

The diagnosis of inhalation injury is made primarily by history and physical examination. Patients who demonstrate the above signs and symptoms should undergo endotracheal intubation. In addition, patients with a partial pressure of arterial oxygen (PaO_2) < 60 mm Hg, $PaCO_2$ >50 mm Hg (acutely) on room air, or a PaO_2 <200 mm Hg on 100% oxygen also require intubation. Computed tomography (CT) scan and radiographs of the chest are not helpful in the acute setting. Some facilities advise using fiberoptic bronchoscopy every 3 to 4 hours to diagnose impending airway obstruction; however, this recommendation may not be clinically practical.

Management.

Initial management includes securing the airway, breathing, and circulation (ABC) as in any other form of injury. If the patient's history and physical are indicative of airway compromise, perform endotracheal intubation early, before it becomes a technical challenge. Inform the entire team of physicians of the primary approach and alternative plans for managing the airway. Difficult-airway equipment should be readily available.

As per the American Society of Anesthesiologists (ASA) Difficult Airway Algorithm, elective measures are appropriate when the physician is able to ventilate the patient (www.asahq.org/Newsletters/2005/11-05/2003TraumaAlgorithm.html). P.40

If a “can't ventilate, can't intubate” situation arises, the physician should approach the airway based on the Difficult Airway Algorithm.

The most suitable elective methods of securing the airway in a burn patient are discussed below.

1. Direct vision laryngoscopy is the most commonly used technique for endotracheal intubation. However, if the patient has inadequate mouth opening, limited range of neck motion, and distorted postburn airway anatomy, consider alternate routes of intubation.
2. Flexible fiberoptic laryngoscopy is the alternative method of choice because it can overcome the problems of direct-vision laryngoscopy.
3. Retrograde wire technique is useful when bleeding prevents adequate visualization of the airway. This is best performed by a provider with experience.
4. Tracheostomy is done electively in a sterile environment, usually by a surgeon. Avoid surgical tracheostomy if possible because of the high incidence of pulmonary sepsis, subglottic stenosis, fistula formation, and subsequent death.

Succinylcholine, a depolarizing muscle relaxant, should be avoided when intubating burn patients. Succinylcholine can produce dangerous levels of hyperkalemia, leading to cardiac arrest. The greatest risk of hyperkalemia occurs within 2 weeks to 6 months after the burn injury.

Be careful when using non-depolarizing muscle relaxants initially. Because there is a possibility of failed intubation, the patient may need to have spontaneous ventilation reestablished. Interestingly, patients are generally resistant to the effects of non-depolarizing muscle relaxants. This desensitization appears by post-injury day 7 and may last up to 70 days.

Nasotracheal intubation is suggested if facial trauma or burns do not preclude its use. Patients tolerate this method better, especially those who need intubation for an extended period of time. The risk of sinusitis increases after 4 days of nasotracheal intubation. Do not extubate patients who require multiple repeat surgeries until the planned surgical course of treatment is completed. If extubation occurs, the provider may run into the same problems faced acutely as well as complications that occur in the chronic setting.

Dressing changes and irrigation and débridement of the wound are common procedures done in the early days after burn injury. If the patient does not require airway support with intubation, these procedures are done under monitored anesthesia care with mask ventilation. Short-acting medications such as midazolam, propofol, and ketamine are the anesthetic agents of choice. P.41

High-frequency positive-pressure ventilation (HFPV) is beneficial in preventing barotrauma and pneumonia in patients with inhalational injury. Tidal volumes of 1 to 3 mL/kg are given at frequencies of 100 to 3,000 cycles per minute. This method of ventilation allows adequate recruitment of collapsed alveoli while avoiding high peak airway pressures. Proper gas humidification is required to avoid severe necrotizing tracheobronchitis.

CHRONIC MANAGEMENT

Chronic complications in burn patients include wound contracture and tracheal stenosis, which make late or subsequent attempts at intubation difficult.

History.

Patients with third- and fourth-degree burns are likely to develop scar contracture. Face and neck contractures make intubation challenging by preventing adequate oral opening, neck flexion, and head extension. Contractures in patients with circumferential full-thickness chest wall burns have problems with chest wall motion, decreasing the ability to ventilate the lungs adequately.

Direct inhalational injury, scar contracture, and/or mechanical damage from the endotracheal tube, tracheostomy tube, or their respective cuffs put burn patients at risk for tracheal stenosis. Stenosis is caused by pressure necrosis with subsequent healing and scar formation. Prolonged intubation and repeated intubation are additional factors that play a role in stenosis formation. Patients may report dyspnea or may have a history of requiring smaller endotracheal tubes for repeated intubations.

Physical.

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Findings of stenosis are made by fiberoptic bronchoscopy. Most stenoses occur in the subglottic region, but they can also involve the upper and lower trachea or a combination of these. Stridor is indicative of stenosis.

Management.

Tracheal stenosis is difficult to treat once it occurs. Prevention is the key to management. Although inhalational injury causes tracheal stenoses in and of itself, the risk can be decreased by using low-pressure cuffs (<20 cm H_2O), ensuring tube stability, and minimizing the number and length of intubations. Early tracheal resection should be avoided because of high restenosis rates.

The approach to airway management in the chronic setting is similar to that in the acute setting. However, there are key differences. In the none-mergency pathway, successful intubation is more likely with fiberoptic bronchoscopy because it allows the provider to detect the presence of subglottic and tracheal stenoses. As stated previously, maintain open communication with the surgical team. When endotracheal intubation is needed emergently, their surgical skills will be required to release wound contractures to make

head and neck manipulation less difficult, or they may be needed to secure a surgical airway.

TAKE HOME POINTS

- A detailed history and physical examination are essential in predicting airway compromise.
- It is safer to intubate sooner rather than later.
- Avoid succinylcholine.
- Elective tracheostomy in burn patients is associated with a high incidence of complications.
- Utilize a team approach. Maintain open communication with the surgical team.
- Have the appropriate equipment and personnel to manage a difficult airway readily available.

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Consider the Use of Lidocaine in the Cuff of the Endotracheal Tube, But Be Aware of the Risks and Alternatives

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Anesthesia providers must mitigate the undesired hemodynamic and neuromuscular responses to intubation. Usually (but of course, not always!), large doses of induction drugs and neuromuscular blockade smooth the way during intubation. Intraoperatively, an adequate level of inhalational anesthetic plus opioids will help the patient tolerate the endotracheal tube (ETT). On extubation, however, the dissipating anesthetic effects and re-emerging pharyngotracheal reflexes may lead to coughing and adverse symptoms, including tachyarrhythmias and increased intracranial, intraocular, or intravascular pressures, via the airway-circulatory reflex.

The incidence of coughing at extubation varies between 6.6% and 96%. Mitigating circulatory-airway reflex symptoms is especially important in patients with cardiovascular and reactive airway disease, neurosurgery, and certain head, neck, or ophthalmologic conditions. Multiple anesthetic strategies to prevent ETT-stimulated coughing have been described, including:

1. Elimination of the ETT completely during general anesthesia (i.e., mask anesthetics) or extubation during profound anesthesia when the ETT is no longer necessary (known universally as a “deep extubation”)
2. Use of local anesthetic via systemic intravenous (IV) or topical endotracheal administration

Anesthesia providers must consider patient history, co-morbidities, and surgical needs to select appropriate strategies.

INTRACUFF LIDOCAINE (ICL)

Nonionized lidocaine has been shown to diffuse readily along a concentration gradient through the lipophilic plastic of an ETT cuff sufficiently to anesthetize adjacent mucosa, typically sparing the vocal cords (which are not in contact with the cuff). The cuff serves as a reservoir of local anesthetic. Comparing equal volumes of intracuff saline versus lidocaine, the incidence of postextubation coughing has been shown to be 59% versus 38%, respectively. Saline-filled cuffs functioned as the control and were shown to have comparable efficacy to air-filled cuffs. Estebe et al. extensively studied various concentrations of lidocaine (2% to 10%), as well as alkalinization of the lidocaine solution. Higher lidocaine concentrations and alkaline solution pH effectively increase the available nonionized, lipophilic, and diffusible lidocaine. Alkalinization increases the available nonionized and diffusible lidocaine at any given concentration. P.44

Risks.

Although the use of lidocaine in the ETT cuff is appealing, serious potential risks deserve consideration. Plain or alkalinized lidocaine solutions (2% to 4%) are clinically effective while limiting exposure to potentially toxic (gram) amounts of intratracheal lidocaine from 10% solutions, should the approximately 10-mL-volume cuff rupture. Filling and deflating the cuff with noncompressible aqueous solutions typically occur much more slowly than with air, whereas similarly, complete elimination of intracuff air and the pressure-volume effects from N_2O can be difficult. Many studies of intracuff lidocaine employed direct manometry to avoid excessive pressure; others inflated cuffs until positive-pressure ventilation at 20 cm H_2O was confirmed without air leak. Prolonged cuff pressure >20 cm H_2O can tamponade mucosal perfusion and result in tissue necrosis. Fluid-filled intracuff pressures may be difficult to assess, with slow fluid flow between pilot and tracheal balloons. Anesthesia providers who are unfamiliar with or unaware of fluid-filled cuffs may not completely empty them at extubation during the 1- to 2-second deflation attempt that is typical for air. Thus, the noncompressible fluid-filled ETT balloon may lead to vocal cord trauma when it is inadvertently pulled through the glottic opening. Although extubation with air-filled balloons was not found to cause arytenoid subluxation in several cadavers, the effect of fluid-filled balloons is unknown.

Albeit rare, cuff rupture has been reported, and lidocaine can be rapidly absorbed through the lung. Although diffusion from 10% lidocaine via intact ETT cuffs did not reach toxic plasma levels, systemic toxicity could occur if the cuff ruptures and releases >1 gram of lidocaine into circulation. Cuffs with pinhole-sized defects filled at minimal pressure typically leak slowly, thus limiting peak and rapid uptake and potential lidocaine toxicity. Knight et al. studied high-dose administration of lidocaine in a patient population to assess systemic toxicity. After induction with 6 mg/kg of lidocaine and maintenance anesthesia of 0.6 mg/kg diazepam and 50% N_2O, a total of 21 mg/kg lidocaine via controlled intravenous infusion was administered up to the point of cardiac bypass without toxicity. Peak plasma concentrations of 9.5 mcg/mL were noted at sternotomy. Indeed, rapid parenteral injections carry a higher risk of toxicity, but based on this study, systemic toxicity should not be clinically evident in adults with doses smaller than 100 mg intravenous push or 160 mg via tracheal instillation.

Time-dependent diffusion of lidocaine from the cuff effectively limits toxicity, as hepatic metabolism occurs with a $T_{1/2}$ of 90 minutes. With excessive pressure and resulting tracheal mucosa ischemia, systemic absorption may be further curtailed. In vitro studies demonstrated that higher concentrations of lidocaine (4% vs. 2%), prolonged duration for diffusion (360 minutes vs. 60 minutes), and “priming” cuff membranes (by prefilling them) resulted in a maximum diffusion of only 17.49 mcg lidocaine. A similar independent study noted as much as 1% of a 4% lidocaine solution diffusing after 6 hours, whereas alkalinized lidocaine resulted in 65% diffusion of the stock solution (2 mL of 2% lidocaine) after 6 hours. Sufficient time must elapse for an effective topical accumulation of mucosal lidocaine, making intracuff lidocaine questionably effective in surgeries lasting <1 hour. Typically, cuffs are also prefilled significantly in advance to saturate the cuff membrane and to eliminate air, which may limit clinical utility in high-volume and rapid-turnover practices. Two separate in vivo studies comparing control to lidocaine-filled cuffs showed very significant cough suppression (5% vs. 70% and 16% vs. 38% to 44%, respectively). The primary goal of intracuff lidocaine is to prevent adverse hemodynamic and oxygenation changes. Unfortunately, this occurred with limited success: “The emergent hemodynamic and oxygenation saturation data were similar for all three groups [air, saline, lidocaine]” —Fagan et al. P.45

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ALTERNATIVES

The minimal anesthetic concentration for suppression of the airway-circulatory reflex from the ETT at extubation (MAC extubation on MAC_{ex}) is comparable to the stimulus of skin incision and is effectively managed at 1.2 MAC. Extubation at MAC_{ex} can effectively mitigate hemodynamic and hyperreactive airway responses from tracheal stimulation once the ETT is no longer required. “Deep extubation” involves removing the ETT while the patient is in a surgical plane of anesthesia. Removing the endotracheal tube can subsequently reduce anesthetic depth requirements, as surgical stimulation abates and dressings are applied, and can thus expedite emergence and transfer to the postoperative acute care unit. The anesthesia provider must, however, demonstrate airway skills to ensure adequate ventilation throughout emergence. Deep extubation is best performed after spontaneous respiration becomes evident. Removing an unnecessary ETT results in a situation equivalent to anesthesia via mask ventilation and with similar contraindications (i.e., high aspiration risk, difficult airway/high ventilation pressure requirements). Daley et al. showed that most anesthesia providers are hesitant to extubate in a deep plane of anesthesia because of aspiration risk. Thus, placement of a Salem sump (naso-gastric tube) after intubation (with removal immediately before extubation) maximizes stomach decompression and helps identify particulate stomach content.

Lidocaine, 1 to 2 mg/kg intravenous push 1 minute before extubation, effectively suppresses cough. Other intravenous medications that can also reduce coughing at extubation include opiates, propofol, succinylcholine,
beta-adrenergic blockers, and dexmedetomidine. However, most systemically administered medications, including lidocaine, also increase anesthetic depth, increase sedation, and/or decrease respiratory drive, and thereby unnecessarily prolong emergence from inhalation anesthetics. Lidocaine plasma concentrations of 3 mcg/mL are required to suppress the cough reflex, while contributing 0.15 MAC of anesthetic effects. Animal studies also confirm that as little as 1 mcg/mL of lidocaine contributes significantly to inhalational anesthetic effects. Because many IV medications have antitussive properties, it is clinically difficult to attribute specific mitigation of cough alone to acutely injected lidocaine, without invoking general anesthetic effects per se.

P.46

Topical tracheal lidocaine has been effectively demonstrated to reduce the incidence of coughing. Patients undergoing awake fiber-optic intubation are routinely anesthetized topically and are without reaction upon tracheal contact. ETT lubrication with nonaqueous lidocaine jelly may be irritating to the mucosa and is therefore discouraged. Available instillation methods of aqueous solutions include LTA (Hospira, Chicago, IL) at intubation, spraying lidocaine down the ETT tube lumen, or via the LITAtube (Sheridan Catheter Corp., Argyle, NY). The LITAtube has an integrated injection port to allow simultaneous injection above and below the cuff via a separate injection port. Although Gonzalez et al. demonstrated the LITA's effective in decreasing cough, LITAs are twice as expensive as regular ETTs, can fail when fenestrations become plugged, and must be inserted at induction for routine use at extubation. An inflated cuff may limit lidocaine from quickly reaching underlying mucosa, while mucosal innervation may be interrupted from above and below the cuff. Likewise, lidocaine may flow beneath the cuff via the film of tracheal secretions. Complete topical effects may require time, whereas systemic transpulmonary effects develop rapidly.

Spraying lidocaine solution down the ETT is a practical and effective alternative to intracuff application. Patients often cough briefly, thus distributing lidocaine throughout the airway and facilitating systemic effects via lung absorption. More effectively, the lidocaine can be trickled in over 10 to 20 seconds via a Luer-Lok port on the airway elbow. With the cuff completely deflated, a sustained insufflation/Valsalva maneuver directs the lidocaine upward to the vocal cords and mucosa adjacent the cuff. This is best performed under anesthesia as ventilatory drive returns in response to normalization of the CO_2 levels and before complete reversal of neuromuscular blockade, thus facilitating air and lidocaine “bubbling up” through relaxed vocal cords. Upon completion of the injection, neuromuscular blockade is reversed. After typically 20 to 30 seconds, maximal topicalization and a return of regular spontaneous respiration develops. At this point, a rapid deflation of the cuff resulting in no reaction or change in respiration
(i.e., as evidenced on ETCO2 tracing) tests to identify adequate topical anesthesia to allow uneventful extubation. This topical effect lasts about 15 to 20 minutes and allows removal of the ETT without significant hemodynamic or respiratory changes. Topically eliminating tracheal stimulus appears to be protective against laryngospasm at all depths of extubation. Although Burton at al. original description recommends 4 mL of 4% lidocaine solution, 5 mL of 2% lidocaine solution is equally effective in our practice. The airway-circulatory reflex is effectively suppressed with 2% lidocaine dosed at 1 mg/kg via intratracheal instillation. The lowest effective dose via intratracheal instillation that effectively mitigates hemodynamic changes immediately before extubation and that has consistent duration of such effects remains to be shown definitively.

P.47

TAKE HOME POINTS

- Intracuff lidocaine is effective for cases >1 hour in duration but may carry significant risks and requires significant preparation.
- Lower concentrations of lidocaine are more effective when alkalinized and are less likely to result in systemic toxicity should the cuff rupture.
- Use of manometry is useful to prevent mucosal ischemia but is underutilized.
- Endotracheal instillation appears to be a safe, easy, effective, and widely available technique.
- Clinical skill and experience are required to safely match available techniques to appropriate patients, and the use of intratracheal local anesthetics to remove the ETT is typically contraindicated in patients “at risk” for aspiration.

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10

Always Troubleshoot An Increase in Peak Airway Pressure

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Juraj Sprung MD, PhD

Occasionally, the anesthesiologist needs to troubleshoot elevated peak airway pressure in an intubated patient. Although each patient has a different baseline peak airway pressure, the anesthesiologist must investigate possible causes of a sudden increase in ventilating pressure. For the most part, these pressure increases can be attributed to problems related to the patient, to airway/ventilation equipment, or even to positioning of surgical staff and equipment.

CONSEQUENCES OF ELEVATED VENTILATORY PRESSURE

Any sudden increase in peak airway pressures requires immediate attention. Barotrauma (trauma resulting from high peak pressure) or volutrauma (trauma to the lungs resulting from overstretched alveoli caused by increased tidal volume) can result from prolonged positive-pressure ventilation at high pressures and/or unrestricted lung expansions. Overstretch of alveoli may cause disruption of the alveolar-capillary membrane, thereby causing an increase in pulmonary vascular permeability and pulmonary edema. High peak inspiratory pressure (PIP) may also cause pneumothorax and pneumomediastinum. As a consequence, an acute respiratory distress syndrome-like picture, referred to as ventilator-induced lung injury, may develop. To prevent ventilator-induced lung injury, excessive PIP should be avoided and alveolar plateau pressures should be maintained below 30 cm H_2O.

In addition to injuring lungs, high airway pressures may also affect hemodynamics. If excessive positive end-expiratory pressure (PEEP) is used, or if auto-PEEP is generated by breath stacking, cardiac output may be adversely affected through increase in intrathoracic pressure, which can impede venous return to the right ventricle. In addition, compression of pulmonary vessels may increase right ventricular afterload, which in turn decreases right ventricular stroke volume and causes right ventricular dilatation. This subsequently shifts the interventricular septum toward the left ventricle, further reducing left ventricular preload and stroke volume. If breath stacking is prolonged and severe, cardiovascular collapse may ensue, appearing as pulseless electrical activity.

P.50

AIRWAY/VENTILATION EQUIPMENT PROBLEMS

One of the most important strategies in preventing high airway pressure-induced lung injury is to thoroughly check the anesthesia machine and breathing circuit system (including inspiratory and expiratory valves) before each use. In this way, mechanical problems can be remedied early. However, the fact that the machine was checked does not rule it out as the source of a sudden problem. Some examples include a suddenly stuck manual PEEP valve, a PEEP valve placed incorrectly into the inspiratory limb of the breathing circuit (this rarely happens because contemporary anesthesia machines have a built-in PEEP valve), a stuck expiratory valve causing breath stacking, or a faulty pressure-relief (pop-off) valve during spontaneous breathing. Other causes of high circuit pressures have been described, such as malfunctioning of ventilator relief and control valves, scavenger system blockage, or occlusion of the muffler on the ventilator. Any of these can result in elevated peak airway pressures and should be considered in determining the underlying cause of elevated pressures in the breathing circuit.

After initiation of mechanical ventilation, the pop-off valve should be set to the “open” position. With resumption of spontaneous breathing at the end of the case, if this valve were closed and this remained unnoticed, high pressure (50 to 60 mm Hg) could develop in the breathing circuit and in the patient's lungs. This high pressure can be further aggravated by flushing bypass oxygen into the system. The same applies when, after the circuit has been disconnected and then reconnected to a tracheal tube, in order to fill the ventilator bellows, an anesthesiologist pushes the oxygen bypass button simultaneously with mechanical inspiration. During this maneuver, all gas delivered to the circuit is directed toward the patient's lungs, potentially causing barotrauma.

PATIENT-RELATED PROBLEMS

One of the most clinically apparent causes of increased peak airway pressures in the practice of anesthesia is the patient coughing or bucking during light anesthesia. This is often resolved simply by increasing the depth of anesthesia by increasing inspiratory concentration of inhaled anesthetic, or administration of intravenous (IV) anesthetic (e.g., propofol, lidocaine) or IV opioid.

Bronchospasm is a frequent cause of increased PIP. Auscultation of the lung fields can confirm this diagnosis if expiratory wheezes are present. Extreme cases of bronchospasm may be associated with no wheezing because air may not be moving. Bronchospasm can also be a part of intraoperative allergic reactions (anaphylactic or anaphylactoid reactions), but rash, tachycardia, and hypotension frequently will be associated. Treatment with albuterol, epinephrine, or diphenhydramine, deepening of inhalational anesthesia, and discontinuing or avoiding the offending agent can help relieve this type of bronchospasm. Reviewing the patient's allergies and avoiding those drugs and other similar compounds is the easiest way to prevent such anaphylactic reactions.

P.51

Wheezing.

Intraoperative wheezing with high PIP can also be caused by endotracheal tube narrowing (tube kinking, the patient biting the tube, overinflation of the tube cuff causing occlusion of the lumen) or increased airway secretions. Even a dislodged nasal turbinate has been described to occlude the endotracheal tube, causing inability to ventilate the lungs. Passing a flexible suction catheter through the endotracheal tube can help confirm patency and, in the case of secretions, can help remedy the problem. However, direct visualization with a fiber-optic bronchoscope is the “gold standard” for determining the definitive cause of endotracheal tube obstruction. Secretions distal in the tracheobronchial tree or foreign-body aspiration can produce similar signs and are unlikely to be identified by the suction catheter test, requiring further exploration with fiber-optic bronchoscopy or possibly chest radiography in the case of a foreign body. External compression of the airway by surgical equipment or personnel also can cause an acute increase in PIP, and if recognized, can be easily remedied by communicating the problem and possible solutions to the surgical staff.

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Positioning.

When a patient is placed in a steep Trendelenburg position, increased peak airway pressures may be encountered, especially if the patient is morbidly obese. This position causes the abdominal contents and overlying chest structures (large breast) to shift in the cranial direction. In that case, an endotracheal tube placed in the distal part of the trachea may move farther toward the carina tracheae and may even result in endobronchial intubation. This may result in increased peak airway pressure; however, a sudden decrease in end-tidal CO_2 concentration on capnography may be suggestive of endobronchial migration of the tracheal tube. Furthermore, endobronchial intubation can be detected by auscultation for bilateral breath sounds—if they are absent, withdrawing the endotracheal tube is mandatory. The anesthesiologist should always observe the tracheal tube depth markings (labeled in centimeters) to prevent accidental tracheal extubation, which in a patient with a difficult airway can be particularly catastrophic.

Decreased Pulmonary and Chest Wall Compliance.

Agradual decrease in pulmonary compliance with an increase in PIP during massive fluid administrations may be indicative of pulmonary edema. Furthermore, continuous monotonous ventilation without PEEP may result in gradual atelectasis, which will increase overall stiffness of the lungs. Occasional intraoperative performance of the vital capacity (recruitment) maneuver may be therapeutic. Rarely, decreased chest wall compliance can be encountered with use of opioids. Centrally mediated muscle contraction can be seen usually

after quick and large boluses of opioids, particularly fentanyl, sufentanil, and alfentanil. Chest wall rigidity caused by opioids can be severe enough to impede ventilation. Fortunately, treatment with neuromuscular blockade is an effective method for reversing chest wall rigidity and allowing adequate ventilation. Asimilar situation with decreased chest wall compliance may occur if additional force is applied to the chest (e.g., surgical equipment, surgical staff leaning on chest).

Acute Decreases in Lung Parenchymal Volume.

Anything that increases the intraabdominal volume (e.g., insufflation of the abdomen for laparoscopic surgery, ascites, abdominal packing) may also elevate PIP. Similarly, compression of the lung parenchyma by pleural effusion or tension pneumothorax can affect airway pressures. Evacuation of pleural fluid by needle aspiration is helpful but should be done before anesthesia and mechanical ventilation; even then, signs of pneumothorax must be excluded before mechanical ventilation is initiated. In the case of pneumothorax, even a simple pneumothorax can become a tension pneumothorax when positive-pressure ventilation is used. If a tension pneumothorax is suspected intraoperatively, immediate needle decompression is indicated. This is performed by inserting a 14-gauge over-the-needle catheter into the second intercostal space at the midclavicular line on the affected side. This will convert a tension pneumothorax to an open pneumothorax, buying time until a chest tube can be placed.

CONCLUSION

Every anesthesiologist must be able to quickly troubleshoot increased ventilatory pressure during anesthesia. Ignoring it may result in severe consequences and induce serious injury to the patient's lungs.

TAKE HOME POINTS

- A sudden increase in peak airway pressure requires immediate investigation.
- Barotrauma or volutrauma can lead to ventilator-induced lung injury.
- High airway pressures can also affect hemodynamics, including increases in thoracic pressures, compression of pulmonary vasculature, and a decrease in biventricular output.
- A thorough machine check-out does not eliminate the possibility of an intraoperative machine failure as the cause of high airway pressures.
- Consider patient-related problems as well, including bronchospasm, decreases in lung compliance, and acute decreases in lung volume.
- Fiber-optic bronchoscopy is the gold standard to rule out an obstruction of the endotracheal tube as a source of increased airway pressures.

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11

Avoid Common Airway and Ventilation Errors in Morbidly Obese Patients

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Over the past 20 years, the incidence of obesity has increased significantly in both adults and children. Obesity is defined as a body mass index (BMI) of 30 kg/m^2 or greater. The latest data from the U.S. National Center for Health Statistics show that 32% of adults aged 20 years and older—more than 60 million people—are obese, up from 22% in the 1988-1994 data. This increase is not limited to adults; the percentage of young people who are overweight has more than tripled since 1980.

For the anesthesiologist, this means that greater numbers of morbidly obese patients will require care. Concerns with regard to morbid obesity include:

- Airway and ventilatory management
- Drug dosing
- Injuries related to positioning
- Coronary artery disease, hypertension, pulmonary hypertension, and cor pulmonale
- Glucose control, hyperlipidemia
- Increased incidence of various postoperative complications
- Sleep-disordered breathing (central or obstructive sleep apnea).

This chapter focuses on airway management and ventilatory issues in morbidly obese patients, although morbid obesity affects virtually every organ system.

Several studies have compared the difficulties in intubating the trachea in morbidly obese and normal-weight patients. Intubation is more difficult in morbidly obese persons, although BMI alone has not been shown to be an independent predictor of difficult intubation. Neck circumference and Mallampati score have been shown to be more accurate indicators of a difficult airway: neck circumference of 40 cm was associated with a 5% incidence of a difficult airway, and a 60-cm circumference was associated with a 35% incidence of difficulty.

Morbidly obese patients have a lower functional residual capacity, larger alveolar-to-arterial oxygen gradient, and a higher tendency for hypoventilation during sedation; all these factors make them more prone to oxyhemoglobin desaturation during tracheal intubation. Positioning of morbidly obese patients in the reverse Trendelenburg position has been shown to allow a longer period of apnea without oxyhemoglobin desaturation, as well as lower respiratory system compliance once mechanical ventilation has resumed. Therefore, the reverse Trendelenburg position may give the operator more time for airway management and also may reduce the incidence of pulmonary aspiration. P.55

ESTABLISHING THE AIRWAY

In morbidly obese patients with a high Mallampati score or large neck circumference, awake fiber-optic intubation should be considered a first-choice technique for tracheal intubation. Other possibilities include performing the tracheal intubation with rigid intubating devices designed for a “difficult airway,” such as a Glidescope, Bullard laryngoscope, or Wuscope. After being sedated, morbidly obese patients, especially those with sleep apnea, are prone to upper-airway collapse, which thus contributes to increased resistance and obstructive apnea. This situation may convert an elective airway to an emergency procedure. Spontaneous breathing will provide oxygenation and the airway will open to a greater degree, thus providing a better view. Therefore, all attempts should be made to preserve spontaneous ventilation during management of a difficult airway. Alternative airway management, such as translaryngeal illumination with a lighted stylette (e.g., Lightwand), may not be effective in these patients because of increased soft tissue over the anterior neck or redundant tissue in the posterior pharynx.

If endotracheal intubation is difficult and/or mask ventilation is suboptimal, alternative strategies must be available. A laryngeal mask airway (LMA), intubating LMA, or Combitube can be used to establish ventilation and maintain oxygenation. Cricothyroidotomy or tracheostomy are the last options, although these can be difficult in obese patients because of lack of recognizable landmarks and excessive tissue. Some reports have noted using ultrasonography-guided placement of a tracheostomy in morbidly obese patients.

Use of the LMA in morbidly obese patients is plagued by the fear of gastric aspiration. However, no reports have noted increased pulmonary aspiration of gastric contents in obese patients, and contrary to popular belief, retrieval of gastric contents in these patients revealed low residuals. When placed electively after induction of anesthesia in morbidly obese patients, the LMA is an effective airway tool. The intubating LMA has been used effectively in obese patients with a difficult airway. This device provides adequate ventilation, creates additional time for airway management, and assists in placement of an endotracheal tube.

ASSOCIATED PROBLEMS

Sleep-disordered breathing is associated with morbid obesity; however, the vast majority of these patients are not diagnosed before the perioperative period. Additional predictors for obstructive sleep apnea include neck circumference in men and BMI in women. Patients with obstructive sleep apnea are more sensitive to respiratory depression from anesthetics and sedatives. They are at increased risk of airway obstruction or apnea postoperatively, which may precipitate a situation that requires urgent establishment of an airway. Timely intervention with noninvasive ventilation in these patients in a monitored setting may be life-saving. Specifically, if the patient has a prescribed level of continuous or biphasic positive airway pressure preoperatively, these treatments should be initiated immediately postoperatively. P.56

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VENTILATION AFTER THE AIRWAY IS ESTABLISHED

Morbidly obese patients have lower functional residual capacity when supine, which leads to an increased alveolar-to-arterial oxygen gradient and may contribute to faster development of hypoxemia. A ventilation strategy that uses a larger tidal volume will not improve oxygenation, and data are emerging that high tidal volumes may actually worsen postoperative outcome as a result of lung injury. Isolated effects of positive end-expiratory pressure (PEEP) of 10 cm H_2O used intraoperatively only modestly improve oxygenation in morbidly obese anesthetized patients. During induction of anesthesia, the application of 10 cm PEEP has been shown to decrease atelectasis and improve PaO_2 in morbidly obese patients. Preoxygenation with an FIO_2 of 60% versus 100% may be advantageous because it does not contribute to the development of postinduction (absorption) atelectasis.

TAKE HOME POINTS

- Tracheal intubation may be more difficult in morbidly obese patients. Therefore, careful assessment of the airway, precise management, and development of alternative intubating strategies are mandatory.
- These patients are at higher risk for airway compromise because of obstructive sleep apnea; therefore, sedation should be gradual, and spontaneous breathing must be maintained.
- When establishing a difficult airway in a morbidly obese patient, awake fiber-optic intubation is the technique of choice because it decreases the risk of a "lost airway."
- Appropriate positioning in the reverse Trendelenburg position with a wedge placed below the shoulders and maximal "sniffing position" will facilitate control of the airway.
- Finally, full readiness with alternative intubating devices will help in avoiding potential disasters.

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12

Plan for An Airway Fire with Every Head and Neck Case

Julie Marshall MD

A 67-year-old woman with a history of chronic obstructive pulmonary disease (COPD) with a home oxygen requirement of 3 L/min by nasal cannula and coronary artery disease was admitted to the burn unit. She had been involved in a house fire after falling asleep while smoking and dropping her cigarette. She sustained an inhalation injury and was intubated in the intensive care unit (ICU) for 2 weeks before coming to the operating room (OR) for a tracheostomy. The procedure was done using the in situ *polyvinyl chloride endotracheal tube. During the procedure, you had trouble maintaining her blood pressure under anesthesia and were reluctant to use pressors due to recent skin grafts. Because she was tolerating only about 0.6% isoflurane and not much midazolam, you added 20% nitrous oxide to her inspired oxygen.*

Unfortunately, she suffered desaturation of her SpO_2 and ST segment changes with attempts to further lower the FiO_2. As the airway was entered with electrocautery at the tracheostomy site, a flame was noted that flashed from the tracheostomy. What might have been done to decrease this risk? Now that a fire has occurred, what should be done?

One of the primary responsibilities of the anesthesia provider is the management of the airway, including anticipating the airway problems that may arise during surgery. Airway fires are complications that are both dramatic and critical when they occur. Selecting an anesthetic plan that decreases the risk of fire is one of the first priorities in airway management. If an airway fire does occur, an anesthesia provider must know what to do to minimize harm to the patient.

The risk of fire occurs whenever the fire triad is present. The fire triad consists of an ignition source, fuel, and oxidizer. It must be recognized that these elements are nearly always present in the OR! During head and neck surgery, these elements are not only present but in close proximity. The oxidizer, oxygen or nitrous oxide, is provided by the anesthesiologist through the endotracheal tube into the surgical site. The ignition source is provided by the surgeon, usually in the form of an electrocautery device or laser. The fuel in airway fires may be the endotracheal tube, packing in the airway, and necrotic or charred tissue. If an endotracheal tube is ignited in a patient,
the flow of gas through the tube can create a “blowtorch”-type flame that can injure more distal structures. Fires that occur at tracheostomy sites as the P.59
airway is entered have been reported to cause more local injury and flames that enter the OR environment instead of the distal lungs. Oral and pharyngeal procedures performed in children with uncuffed tubes may result in airway fire because of the leak of oxygen-rich gas into the site on surgery.

The endotracheal tube is the fuel source that is most consistently present in the airway. It has been shown that red rubber, polyvinyl chloride, and silicone endotracheal tubes are all flammable at <26% oxygen. To decrease the risk of combustion, anesthesiologists historically wrapped the endotracheal tubes in metal tape. This practice is not often used today because the tape may loosen, thereby exposing areas of the tubing, it does not cover the cuff, and the tubes may kink easily. Commercially available “laser-resistant” tubes are flexible metal tubes that are more resistant to combustion and may be a better choice for airway surgery. However, these tubes are more bulky and may be more difficult to place. The cuff of the endotracheal tube may be filled with methylene blue; in the event of a cuff perforation, the color change will notify the surgeon. The necessity for an endotracheal tube should be discussed with the surgeon as well. Some procedures may be done with intermittent apnea, although this may require a higher FiO_2 during episodes of ventilation.

Common procedures that carry a high risk of airway fire include tracheostomy, tonsillectomy, adenoidectomy, airway tumor debulking, oral surgery, and tracheal reconstructions. Because it is inevitable that the fire triad will be present and in close proximity during ENT procedures, steps must be taken to minimize the risk.

The surgeon must be in communication with the anesthesiologist about the surgical plan including any planned airway or intraoral procedures. If there is a portion of the surgery that precedes the high-risk portion of the case, the anesthesia provider should be notified as the airway portion approaches so that adjustment may be made to lower the FiO_2 if needed.

Minimizing the FiO_2 is one of the trickiest aspects of managing the fire triad, even for experienced anesthesia providers. It is especially difficult in patients who have poor lung function or tenuous cardiovascular status. The risk of using a higher FiO_2 must always be weighed against the risk of fire and the alternative options available to the surgeon. Remember that both oxygen and nitrous oxide are flammable to equal degrees, so a mixture of a low oxygen flow with air may be a safer option. If there is difficulty in maintaining the patient at a low FiO_2, the surgeon must be notified so that he or she can use non-heat-generating surgical tools (scalpel instead of electrocautery) or modify the surgical plan. Also, during intraoral or pharyngeal procedures,
wet packing may be placed to decrease the leakage of oxygen-rich gas into the surgical site. Care must be taken that these stay moist, because dry P.60
packing will act as an additional source for ignition.

If an airway fire occurs, because of poor planning or in spite of good planning, care must be taken to prevent further harm. The oxidizer should be removed by stopping ventilation. The endotracheal tube is removed to remove the source of fuel and prevent further thermal burns. The patient should be ventilated and reintubated. After the patient has stabilized, the extent of damage should be determined with bronchoscopy and any additional relevant workup or treatment (arterial blood gas (ABG), steroids, tracheotomy) considered immediately. The management should be tailored to the specific clinical situation.

So what could have been done differently for the patient above, who sustained an airway fire during tracheostomy? Changing the polyvinyl tube to a metal tube would not be warranted for this procedure. Using a gas mixture of oxygen and air, instead of oxygen and nitrous oxide, would have decreased the amount of oxidizer present and should have been done. The surgeons should have been notified of the difficulty in decreasing the FiO_2 so an alternative method of entering the airway could be utilized, such as a scalpel. (*Actually*, the surgeons should have known not to use electrocautery to enter the airway in the first place, and should have been reminded by the anesthesia provider, who should have been on his or her feet, hanging over the drapes and watching every move). Once the fire occurred, the steps of disconnecting from the circuit, extubation, extinguishing the flame, ventilation, and reintubation would have been appropriate.

Finally, there have been some reports of keeping patients intubated after airway fires during tracheotomy if the fire has been extinguished. This is because of the risk of extubating patients who usually have prior lung disease, as does the patient in our example. Following stabilization, appropriate measures

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include bronchoscopy, ABG, chest radiography, and continuing with the tracheotomy.

TAKE HOME POINTS

- The fire triad is both present and in close proximity in the airway—develop a method to remind yourself of that for every case.
- Minimize oxidizer FiO_2 and nitrous oxide concentration.
- Minimize ignition generators—electrocautery or laser time.
- Metal tubes are harder to ignite, but all tubes can combust.
- If a fire occurs, disconnect from the circuit, extubate, extinguish the flame, ventilate, reintubate, then do diagnostic tests and treatment.

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13

Know How to Perform a Cricothyroidotomy

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Hilary Koprowski II MD

Although instances when management of an airway has progressed down the algorithm to the need for an emergency surgical airway are fortunately rare, it is sometimes necessary to perform this procedure to gain control of the airway. In the past, there was most likely "a surgeon within earshot," but with the advent of more restricted residency work hours and the need to do more cases to generate the same billing income, surgeons are "spread more thinly" than in the past.

A cricothyroidotomy is a variant approach to tracheostomy and is considered by surgeons to be the preferred technique for initial emergent surgical airway management. The cricothyroid membrane is generally easy to locate and is fairly avascular. Because cricoid cartilage chondritis with the unfortunate sequelae of subglottic stenosis can develop after cricothyroidotomy, revision to a conventional tracheostomy, usually at the second or third tracheal ring (which involves exposing and separating the strap muscles and dividing the thyroid isthmus) is generally performed within 24 hours.

The most important step in performing a cricothyroidotomy successfully is making the decision to do one. The second most important step is remaining calm; it is the authors' opinion that most anesthesiologists have the technical skills necessary to facilitate an "emergency surgical airway" via cricothyroidotomy.

The steps used by general surgeons in performing emergent cricothyroidotomy are as follows.

1. Position yourself on the side of the bed where your dominant hand is cephalad (i.e., if you are right-handed you will be standing by the patient's left shoulder).
2. Do not prep the neck—this is an emergency procedure and seconds count.
3. Palpate the cricoid membrane—this lies above the cricoid cartilage and below the thyroid cartilage. It is the exact location where transtracheal lidocaine is injected for anesthesia for an awake airway technique.
4. Ask for a no. 10 blade.
5. With your dominant hand moving from *foot to head,* use the belly of the blade to make a *longitudinal* incision in the midline 4 cm long superficial to the cricoid membrane.
6. Take care to stay in the midline. Large anterior thyroid veins run on either side of the midline and can cause bleeding sufficient to completely obscure the field. P.63
7. Do not be stingy on the length of the incision. A long incision allows the tissues to "gap open" and will make visualization of the trachea easier.
8. Apply enough pressure to the blade to reach the level of the cricoid membrane with two passes.
9. If the person is obese, ask for instruments to retract the tissue. You will need two (one for each side). Army-Navy, thyroid rectors, or vein retractors are all acceptable choices.
10. Once at the cartilage level, palpate again for the cricoid membrane.
11. When the cricoid membrane is located, use the no. 10 blade to "poke through" the cricoid cartilage.
12. You will know you are in the airway when there is "sputtering and splashing." Take care not to have your face too close to the operative field when performing this step so as not to have a splash exposure.
13. Without removing the blade, turn it 180 degrees several times to widen the opening.
14. Still without removing the blade, move it from your dominant hand to your nondominant hand and retract the lower edge of the incision you have just made toward the feet with the side of the blade. Do not take the blade out of the airway at any time before the endotracheal tube is placed. The incision will "close shut" and you will have difficulty relocating the site where you entered the airway.
15. With your now free dominant hand, take a 6.0 cuffed styletted endotracheal tube; have an assistant bend the tip to an approximate angle of 75 to 90 degrees.
16. Insert the tube with a curving motion.
17. Remove the stylet and check for good air flow and return of CO_2 per standard anesthesia protocol.
18. Do not let go of the tube until it has been sewn into place. Securing the tube and closing the incision is probably best done by your hopefully soon to be arriving surgical colleagues.
19. If at any stage bleeding is experienced, do not use cautery. This risks igniting the surrounding oxygen and putting the patient and staff at extreme risk.
20. Do not be concerned about the esthetics of the incision or where the tube actually entered the airway or the comments of colleagues. Remember, there is almost no incision that a plastic surgeon cannot revise and almost no tracheal or laryngeal injury that an otolaryngologist cannot repair. Your colleagues will undoubtedly be polite as they know that an anesthesiologist performing a cricothyroidotomy is the very definition of an emergency situation.

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Do Not Overinflate the Cuff of the Endotracheal Tube

J. Todd Hobelmann MD

You are supervising a junior resident for the first time and are proceeding nicely through the start of a femoral-dorsal pedis bypass. You are helping your CA-1 place an arterial line when two senior anesthesiologists make a surprise visit to your room. You have a slight sinking feeling when you notice that one of them is holding a small manometer. He applies it to the balloon of the endotracheal tube (ETT) and you find that the measured pressure in the cuff of the ETT is 65 cm H_2O. This particular colleague happens to have a PhD in biomedical engineering (and the other person has an interest in tracheal injuries), so you know that there is not even a chance of arguing about it. You have been busted by the cuff police.

The cuff of an endotracheal tube is an essential piece of modern anesthesia. It serves as a seal of the airway—decreasing the likelihood of aspirating pharyngeal secretions into the trachea and lungs (debatable) and allowing adequate positive-pressure ventilation. It also serves to anchor the ETT in place, thus allowing position changes during surgery with less risk of mainstem intubation or inadvertent extubation. Traditionally, we learn in anesthesiology that the cuff pressures of the ETT should be within the range of 20 to 30 cm H_2O. Although these numbers are very strict in pediatric anesthesia (and a component of every pediatric anesthesiology text), cuff pressure in adults is of key importance as well.

The pediatric airway differs in many ways from that of an adult. One of the key differences is the location of the narrowest portion of the airway. Remember that the adult airway is cylindrical-shaped, with the tightest area being between the vocal cords or glottis. However, in neonates, infants, and young children, the airway is more funnel-shaped and becomes tightest at the level just below the glottic aperture (the subglottis). Because the pediatric subglottic region can be very narrow, anesthesiologists often do not use cuffed ETTs unless the patient is around age 8 to 10 years or older. In many circumstances, without a cuff, the diameter of the ETT itself may often be too wide for the airway, even when it passes easily beyond the cords. It is not uncommon in pediatric cases that the anesthesiologist changes to a smaller ETT for this reason. Above 30 cm H_2O, the pressure exerted by the tube on the subglottis for a prolonged operation may cause mucosal ischemia,
resulting in sloughing of the mucosal lining and potentially resulting in subglottic stenosis or worse.

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With modern single-lumen ETTs (those that are low-pressure, high-volume), catastrophic consequences are relatively rare in the operating room, especially for adult cases. With older ETTs (high-pressure, low-volume), overinflation of the cuff could result in severe morbidity such as rupture of the trachea, tracheal-innominate artery fistulas, and tracheo-carotid artery erosions. Luckily, the biggest complication of having an ETT inflated in the airway during modern anesthesia is postoperative sore throat. Nonethe-less, animal data imply that overinflation of modern ETT cuffs may result in serious ciliary damage and reduced tracheal blood flow (more severe in hypotensive states).

Of note, another operating room population of concern with respect to ETT cuff pressure is patients undergoing thoracic cases involving lung isolation with a double-lumen ETT. The cuff on the bronchial lumen can easily be overinflated and result in bronchial edema and stenosis (as this cuff is often of lower volume and higher pressure than the standard tracheal cuff). Proper practice requires the provider inflate this cuff with only the minimal pressure required to offer adequate lung isolation. Once isolation is no longer required, the cuff should be deflated.

It has long been known that there are limited ways of detecting true cuff pressure in the operating room during adult cases. Previous studies have demonstrated that manual palpation of the cuff generally underestimated cuff pressure. A well-performed study from 2004 suggested the use of manometry in gathering accurate data regarding ETT cuff pressure. This suggestion may be taken under stronger consideration in the intensive care unit (ICU) setting, where patients remain intubated for prolonged periods of time. However, utilization of manometry could well be warranted for longer cases, such as transplants, and cardiac and neurosurgery. This has not gained wide acceptance in clinical practice at this time.

TAKE HOME POINTS

- ETT cuff pressure has implications in anesthesia.
- ETT cuff pressure is of vital importance in the pediatric population to avoid serious airway consequences.
- Maximum cuff pressure should not exceed 20 to 30 cm H_2O.
- Be especially careful not to overinflate the blue cuff on the bronchial side of a double-lumen tube.
- After intubation, inflate the cuff just enough to prevent an air leak.
- Manual palpation of the balloon is not reliable in determining cuff pressures.

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Don't Underrepresent the Risks Associated with the Use of a Laryngeal Mask Airway

Surjya Sen MD

A 57-year-old, otherwise healthy professional concert singer presents for an elective outpatient prostate biopsy for suspected adenocarcinoma. He has a healthy body mass index (BMI), and physical exam indicates that he would be a grade 1 intubation. He reports that 10 years ago he experienced hoarseness and prolonged throat and mouth pain after intubation for an emergency appendectomy. He concludes this bit of history by volunteering, "I almost sued the guy that jammed that tube down my throat; I couldn't do my concert dates for two months." He had surgery for a meningomyocele as an infant (wouldn't you just know), so a spinal is out. He inquires whether there are any other options. When you suggest a laryngeal mask airway, he replies, "Well, that sounds great, Doc, but are there any drawbacks to having anesthesia with this 'ellemae' thing?"

In 1983, Dr. Archie Brain introduced the laryngeal mask airway (LMA) as a device to secure the airway without the use of direct laryngoscopy. By 1988 the LMA became commercially available worldwide, and by 1991 the U.S. Food and Drug Administration had approved its use in the United States. Soon thereafter, the device gained rapid popularity because of its advantages over other options for airway control—namely, direct laryngoscopy with endotracheal intubation or bag-mask ventilation (Table 15.1). These days, the LMA is an essential piece of equipment on every emergency airway cart and has become nearly ubiquitous in outpatient anesthesia. Despite its many advantages, however, the use of an LMA does carry a small but significant set of risks. Every anesthesia provider should be familiar with these risks—not only to recognize potential complications if they occur, but also to counsel patients properly when requesting informed consent.

DISADVANTAGES AND COMPLICATIONS

LMA Misplacement.

The first requirement to placing an LMA successfully is proper positioning. Problems such as a folded tip, supraglottal placement, and an improper seal over the upper esophagus can usually all be resolved with repositioning. Just as with endotracheal intubation, verification of proper LMA placement immediately after insertion is imperative.

End-tidal capnography, bilateral breath sounds, and auscultating over the laryngopharynx for evidence of leaks should be essential parts of a preliminary check after initial positioning. The placement should be periodically verified throughout the case and after any repositioning of the patient. P.68

TABLE 15.1 SOME ADVANTAGES OF THE LMA

- Use in an ACLS algorithm for difficult airway.
- Use in the absence of trained personnel to perform tracheal intubation or surgical airway.
- Use when direct laryngoscopy equipment malfunctions.
- Use in reactive airway disease when endotracheal intubation may be overstimulating.
- Less intraocular pressure increases when compared to endotracheal intubation.
- Less local trauma and better hemodynamic stability during insertion than with direct laryngoscopy and endotracheal intubation.
- Less incidence of "sore throat" and "hoarse voice" when compared to endotracheal intubation.
- Less gastric insufflation when compared to face-mask ventilation.
- Better air-tight seal, less hand fatigue, and more stability than a face mask.
- Less manipulation of the patient's head, neck, and jaw.

Mucosal Injury and Sore Throat.

With a mean incidence of approximately 10%, mucosal injury is usually recognized by blood on the LMA after removal. The main causes are usually cuff

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pressure on the pharyngeal mucosa (though cuff pressures are typically less than that required for mucosal perfusion) and manipulation during insertion. Sore throats are more common, with a mean incidence of 18%, but are still less common than are typically seen with endotracheal intubation.

Gastric Inflation.

Though certainly not as effective as an endotracheal tube in preventing gastric inflation, the LMA is superior to mask ventilation. The risk of gastric inflation is usually present because most patients may require some assisted ventilations during the anesthetic. Adherence to strict nothing-by-mouth (NPO) guidelines and careful patient selection (i.e., not using an LMA electively in patients with severe reflux or in patients who are obese) can help minimize the risks of regurgitation and aspiration that can come with overinflation of the stomach. It is worthwhile noting that the Proseal brand of LMA has a separate esophageal drainage tube that can help reduce the risk of gastric inflation and subsequent regurgitation.

Regurgitation and Aspiration.

In an elective situation, if there is any definable preoperative risk of regurgitation or aspiration in a patient, the anesthesia provider should not chose an LMA. Endotracheal tubes are reliably better at minimizing this risk. In cases when aspiration does occur with an LMA (incidence rates are reported at approximately 0.02%), malposition and/or gastric inflation are often the cause. In a review of the literature, Keller et al.

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found several other factors that may increase the risk: *inadequate depth of anesthesia, intra-abdominal surgery, upper gastrointestinal disease, lithotomy position, patient movement, exchanging the LMA for an endotracheal tube, multiple insertion attempts, opioids, diabetes with gastroparesis, obesity, and cuff deflation.* Catastrophic outcomes including brain injury and death have also been reported.

Hyperinflation of Cuff .

Multiple studies have demonstrated that LMA cuff pressures increase over the course of an operation when nitrous oxide is used. The increase in cuff pressures correlated directly with the length of the procedures (as high as 42 mm Hg in procedures that lasted from 2 to 5 hours). Cuffs that were constructed from polyvinyl chloride (PVC) seemed less susceptible to hyperinflation. The clinical correlate to these increased pressures is not clear (though some authors have reported cases of nerve injuries thought to be related to high pressures), but anesthesia providers will be well advised to check the inflation pressures of the LMA cuff periodically over the course of a long procedure when nitrous oxide is being used.

Nerve Injury.

Various nerve injuries have been reported with the use of an LMA. Injuries to the recurrent laryngeal, superior laryngeal, hypoglossal, and lingual nerves have all been reported. Implicated causes have ranged from high cuff pressures to malpositioning. Most are reported to have resolved within 6 months.

Mediastinitis and Retropharyngeal Abscess Formation.

In a recent "case and commentary" discussion of laryngeal mask airways posted on Agency for Healthcare Research and Quality (ahrq.gov), the occurrence of mediastinitis and retropharyngeal abscess formation was reported. This complication is worth noting because systemic manifestations such as fevers and myalgias combined with "a fullness in the neck" may warrant an emergency evaluation of possible retropharyngeal perforation, abscess formation, and mediastinal involvement. Definitive therapy with antibiotics and surgical drainage were reported to be effective for the complication.

TAKE HOME POINTS

- LMAs may offer an advantage over endotracheal intubation or prolonged mask anesthesia in certain well-selected cases.
- Despite certain advantages, however, patients should be appropriately counseled regarding the associated complications that may occur.
- Never compromise the aspiration risk of a patient when selecting an LMA; endotracheal anesthesia is decidedly better.
- Beware of high cuff inflation pressures with prolonged nitrous anesthesia—nerve injuries may occur.

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Do Not Be Intimidated By the Placement and Use of Double-Lumen Endotracheal Tubes

Jay K. Levin MD

The use of double-lumen endotracheal tubes (DLTs) is standard practice for thoracic and other surgical procedures requiring one-lung ventilation, as well as in life-threatening conditions requiring lung isolation, such as hemoptysis. Although the techniques for accurate placement and ventilation with DLTs are more complex than for placement of a standard endotracheal tube, practitioners should be no more intimidated by them than by any other (slightly advanced) airway procedure. The trick to the placement and use of a DLT is to take it step by step. Remember that appropriate caution in placing DLTs is always warranted (never force "the square peg in a round hole"), but no more than in any other area of anesthetic practice.

DLTs are designed to ventilate one lung while isolating the contralateral side. There are design differences depending on the manufacturer, but all DLTs consist of a tracheal lumen and endobronchial lumen. Sizes of DLTs vary from 26 to 41 French (one French equals 1/3 mm and is a measurement of diameter). A 39 F DLT is equivalent to a 9.5-mm-internal-diameter endotracheal tube. Choosing the "proper size" has been a topic of many investigations, including the use of chest radiographs and computerized tomography (CT) scans to measure tracheal and bronchial diameter. No one method has proven an absolute predictor of the "optimal" size of DLT or, in other words, the size DLT that results in the fewest minor and major complications for any given patient but that still functions reliably in the intraoperative period. Traumatic airway injuries are common with double-lumen endotracheal tubes, with a high incidence of hoarseness and sore throat, and presumably an oversized tube will contribute to this unnecessarily. However, undersizing a DLT could lead to distal migration and possible pneumothorax.

The typical method of placing a left DLT is to use direct laryngoscopy to pass the bronchial tip through the vocal cords, remove the stylet, and then advance the tube until the "double" portion passes through the larynx. At this point, for proper placement in the left bronchus, a "blind" or fiberoptic technique may be used. In the "blind" technique, the tracheal cuff is placed past the vocal cords, the tube is rotated counterclockwise, and the

tube is advanced until it meets resistance (approximately 28 to 30 cm at the teeth). Using the fiber-optic method, the bronchial tip is placed past the vocal P.72
cords, the scope is placed through the bronchial lumen and driven into the left mainstem bronchus, and the tube is advanced over the scope into the left side.

There are advantages and disadvantages to each technique for initial placement of DLTs. The fiber-optic scope technique is favored by some senior practitioners and is often described as the technique of choice. However, a fiber-optic scope may not be available or may not be helpful in situations of significant hemorrhage or secretions, and one must rely on clinical skills for proper positioning.

The "blind" technique allows for rapid placement without the need for excess equipment other than a stethoscope for auscultation to confirm correct placement. Unfortunately, malposition requiring further manipulation has been reported as from 30% to 78%. Leaving the stylet in place for the entire "blind" placement has demonstrated improved success, although the potential risk for airway trauma may outweigh this benefit. An additional technique reported increased success by insufflating 2 mL of air into the bronchial cuff after placement, withdrawing while holding the pilot balloon until it collapsed, deflating the bronchial cuff, and advancing 1.5 cm. Proper placement occurred in 26 of 29 left-sided DLT attempts. One major disadvantage with the "blind" method beyond lower success rates is the inability to visualize anatomic problems. An example is thoracic aortic aneurysms, which can compress the left main bronchus, impairing placement and potentially leading to aneurysm rupture by "blind" DLT placement.

Left-sided double-lumen placement can be confirmed by auscultation or by fiber-optic visualization. With proper technique, clinical confirmation can be performed successfully with a stethoscope. First, inflate the tracheal cuff and listen for bilateral breath sounds. Clamp the tracheal side, open the tracheal vent, and listen for a leak on the tracheal side. Inflate the bronchial cuff until the leak disappears, usually less than 2 mL and never more than 3 mL. Isolation is confirmed by the loss of breath sounds on the tracheal side and preserved sounds on the bronchial side. Bilateral breath sounds should return after the clamp is removed and the vent is closed.

If the auscultation technique does not confirm placement, fiber-optic confirmation is warranted, if available. One should always find the right upper-lobe bronchus to confirm accurate placement in the left side. The bronchial cuff should be barely seen or, with BronchCath Mallinckrodt DLTs, a black line marks 4 cm from the distal tip of the bronchial lumen. Because the left main bronchus is longer than 5 cm in both men and women, this should provide ideal placement.

Insufflation of the bronchial cuff is an important part of final placement of the DLT. If the patient is to be placed in the lateral decubitus position, it may be P.73
best to insufflate the bronchial cuff after final positioning to prevent the DLT from being displaced as well as to avoid overinflating the cuff. There are alternative methods to auscultation to determine the minimal amount of air needed for lung isolation to prevent mucosal ischemia. While ventilating, 0.5 mL of air at a time (not to exceed 3 mL) is injected into the bronchial cuff, a bare hand or arm is placed over the bronchial vent, and the bronchial side is clamped. The leak will disappear when isolation is achieved. Another technique involves placing a nasogastric tube tip in the bronchial lumen and the other end in a bottle of saline while slowly injecting air into the cuff until bubbles are no longer apparent. *Always remember to deflate the bronchial cuff when lung isolation is no longer necessary.*

The choice of the appropriate tidal volume for one-lung ventilation (OLV) is an important area of evolving practice. Recent evidence suggests that lower lung volumes during OLV (5 mL/kg) with positive end-expiratory pressure (PEEP) during esophagectomy, a "protective ventilation strategy" similar to that used in acute lung injury (ALI) patients, resulted in decreased inflammatory response, improved lung function, and earlier extubation. In patients with obstructive lung disease, there is the possibility for auto-PEEP with larger tidal volumes (TVs) if adequate time is not allowed for exhalation. Maintaining peak pressures >40 cm H_2O and higher tidal volumes led to a higher incidence of postpneumonectomy ALI and respiratory failure. These considerations suggest that the higher traditional TVs of 9 to 12 mL/kg should be reduced during OLV.

The use of PEEP is sometimes considered controversial because of concern that capillary compression will cause shunting of blood flow away from the ventilated lung. However, given the prevalence of atelectasis contributing to hypoxemia and the body of data showing the protective role of PEEP in reducing ventilator-associated lung injury, the use of moderate PEEP (4 to 8 cm H_2O) is recommended. In general, if a manual recruitment maneuver improves oxygenation, then PEEP will help maintain that effect.

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TAKE HOME POINTS

- Double-lumen endotracheal tubes provide a simple means for one-lung ventilation and lung isolation.
- The ability to properly place and confirm DLT position needs to be part of every anesthesia practitioner's repertoire.
- Seek out these cases and take the airway management one step at a time: “Number one, have we intubated the trachea? Number two, what information do we have that the bronchial lumen is where we want it?” and then proceed down the algorithm.
- The editor recommends that junior practitioners take a try at confirming placement by auscultation before using the fiber-optic method. It is a great way to really get a sense of how the DLT will function later in the case.
- If the DLT is “not working,” remember that unless you have turned the whole thing 180 degrees, it is either in too far or not in far enough. If you can't figure out which is the case, consider withdrawing the bronchial lumen into the trachea and starting over.
- Once placement has been confirmed, avoid taping it to what will be the dependent side of the face, and don't “overtape” it. Occasionally, the DLT will have to be moved several millimeters during the case, and many practitioners have had to struggle with peeling a wad of tape off the side of the patient's face that has been positioned in the foam head ring.
- With careful placement and a lung-protective strategy with lower TVs, and the use of PEEP, proper use of DLTs may provide postoperative benefits and better surgical outcomes.

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Do Not Underestimate the Difficulty of Reintubating a Patient Who Has Undergone Carotid Endarterectomy or Cervical Spine Surgery

Heath R. Diel MD

Randal O. Dull MD, PhD

Anesthesia providers do not always appreciate the potential difficulties in reintubating patients who have undergone carotid endarterectomy and cervical spine surgery. Because these patients do not have tumors and usually do not initially present with stridor or signs of vocal cord paralysis (unless there has been a previous stroke), the initial intubation often is uncomplicated. During the operative phase there are many important physiologic issues, such as management of hemodynamics and maintenance of organ perfusion, that compete with the anesthesia provider's attention to airway issues. Prompt emergence and extubation is desired to facilitate postoperative neurologic checks and to avoid hypertension and coughing at the time of emergence. However, at all times, anesthesia providers must be alert for postextubation airway issues. It can be extremely difficult to get the airway secured after failed extubation even if the initial intubation was uncomplicated. This is a situation to consider very carefully before planning a “trial of extubation” or a “let's see how he does without the tube” maneuver.

The causes of postoperative respiratory dysfunction after carotid endarterectomy or cervical spine fusion include laryngeal or pharyngeal edema, hematoma, cerebrospinal fluid (CSF) leak, recurrent laryngeal nerve dysfunction, carotid body dysfunction, cervical fusion, malalignment, and improperly applied bandages.

EDEMA/HEMATOMA

Postoperative cervical edema is present in every anterior neck procedure to some degree and occurs in cervical laminectomy because of prone positioning. In carotid endarterectomy patients, computed tomographic studies have shown a 25% to 60% reduction in airway volume and a 200% to 250% increase in retropharyngeal mass as a result of edema alone. Edema of the larynx and pharynx is thought to be caused by venous and lymphatic disruption, as well as direct tissue trauma with increased capillary permeability secondary to release of local inflammatory mediators. Tissue edema can be difficult to gauge clinically if the patient has remained in the supine position. There can be significant internal compression of airway structure with little or no change in neck circumference, and stridor may not be heard until the airway has narrowed to 4 mm. Also, unilateral tissue disruption can cause bilateral edema. Edema is rarely the sole cause of respiratory distress leading to reintubation, but it compounds the effects of other problems that might arise (such as hematoma) and can make visualization of the vocal cords for reintubation extremely difficult. If the edema has occurred because of prone positioning, elevating the head of the bed to 30 degrees during emergence can reduce soft tissue edema.

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Significant hematoma occurs in about 1.4% to 10.0% of carotid endarterectomy patients, with higher frequency in cases involving coagulopathies and heparin use without reversal. Frequently, hematomas do not develop until several hours into the postoperative phase, and the “carotid take-back” patient is often hypertensive, hypercarbic, and partially obtunded as well. Reintubation in this situation can be one of the most difficult airway situations to manage, and the airway can quickly become a truly emergency situation. Surgical evacuation of the hematoma without regard for aseptic technique may be sufficient to relieve the obstruction until oral direct laryngoscopy can be performed. The authors recommend that anesthesia providers have available maximum airway support in terms of personnel and equipment when reintubating any patient who has developed significant hematoma after carotid or cervical neck surgery.

CEREBROSPINAL FLUID LEAK

Cerebrospinal fluid leak is a possible complication of anterior cervical vertebral fusion and occurs when the integrity of the dura mater and arachnoid tissue is disrupted. Collection of CSF can cause impaired ventilation as a result of mass effect, very similar to hematoma formation.

RECURRENT LARYNGEAL NERVE INJURY

Recurrent laryngeal nerve injury happens most often after thyroid surgery but is a recognized complication of carotid endarterectomy and anterior cervical spine surgery as well. Acute dysfunctions of the recurrent laryngeal nerve occur in about 0.2% to 10.0% of carotid surgery cases, but, fortunately, less than 1.0% involve permanent injuries.

The recurrent laryngeal nerve runs in the groove between the trachea and esophagus and innervates all the muscles of the larynx except the cricothyroid muscle. Injury is usually the result of ischemia, surgical manipulation, dissection, stretching, or compression. Unilateral dysfunction results in unilateral vocal cord adduction and hoarse voice. Bilateral dysfunction causes airway obstruction from bilateral cord adduction. The anesthesiologist should be aware of the preoperative function of the recurrent laryngeal nerves and patients with hoarse voice related to stroke or prior surgery. These patients may benefit from preoperative evaluation. Upon emergence and extubation, bilateral laryngeal nerve dysfunction presents as immediate airway obstruction requiring intervention.

P.77

CAROTID BODY DYSFUNCTION

The carotid body functions primarily as a chemoreceptor for blood oxygen and carbon dioxide content. Surgical denervation secondary to carotid endarterectomy impairs the patient's physiologic response to hypoxia. Patients treated with respiratory depressants such as opiates are at greater risk for postoperative hypoxia

CERVICAL MISALIGNMENT

Cervical misalignment is an unusual but possible complication of cervical spinal fusion. Misalignment can cause structural impingement of the airway, which, coupled with edema, can lead to ventilation deficiencies. Also, cervical fusion limits neck extension, and this could contribute to difficult

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postoperative reintubation.

SURGICAL DRESSINGS

Supportive dressings after neck surgery can cause airway obstruction by impeding venous and lymphatic drainage and worsening edema. Large dressings also obscure visualization of the operative site and may cover signs of bleeding that would alert the anesthesia and postoperative acute-care staff to a developing hematoma. Cervical collars should be sized appropriately and allow for some postoperative expansion due to edema. It should be obvious but bears stating that excessively tight or circumferential neck bandages should never be used. The authors are aware of several cases of apparent airway obstruction after carotid endarectomy that were relieved when the neck dressing was removed.

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Do Not Start the Airway Management of a Ludwig Angina Patient Until Personnel and Equipment for a Definitive (Surgical) Airway Are Assembled

Anne L. Lemak DMD

Todd M. Oravitz MD

Ludwig angina is more formally known as septic cellulitis of the submandibular, submental, or sublingual spaces. Ludwig angina is most often the result of a dental abscess of the second or third mandibular molar, before or after tooth extraction, and presents as diffuse swelling of the floor of the mouth and airway. Symptoms can arise quickly and may consist of neck swelling, redness and pain, fever, chills, fatigue, earache, drooling, confusion, and eventually, airway collapse. Prompt treatment is compulsory to prevent the spread of infection and asphyxiation associated with subsequent airway edema.

Ludwig angina is typically caused by hemolytic streptococci from normal oral flora, but it may be caused by a combination of both aerobic and anaerobic bacteria resulting in the infectious process. The swelling in the floor of the mouth may be so extensive that the tongue is displaced upward and posterior, occluding the mouth and oropharynx, and if left untreated, the infection may spread caudally into the thoracic cavity and result in an abscess of the pericardium and lungs or throughout the body as septic shock. Patients may present with extreme lethargy, dehydration, and shortness of breath, and require immediate medical attention.

Treatment of the Ludwig angina patient depends on the extent of airway involvement. Aless extensive case may simply require incision and drainage, surgical decompression, and a full course of broad-spectrum antibiotics. However, to determine the degree of inflammation, a computerized tomographic (CT) scan of the head and neck may be warranted as the first step to treatment. Athorough history and physical should also be obtained, and the patient's anesthesia team should work in conjunction with the surgeons to devise a comprehensive treatment plan. Managing the Ludwig patient may be complex, as he or she may be unable to talk or open his or her mouth adequately for incision and drainage of the affected area. Difficulty opening the mouth in combination with airway edema and displacement of the tongue presents the potential for serious anesthetic risk and complication. Adefinitive anesthetic strategy, along with backup preparations, is highly recommended for any Ludwig patient.

The anesthesia provider(s) should carefully examine the airway, evaluating all the usual components including Mallampati class, oral opening, cervical range of motion, thyromental distance, and presence/absence of teeth. Additionally, in the Ludwig angina patient, the degree of airway edema, both clinically and radiographically, ability to swallow, amount of secretions, and mobility of the tongue need to be assessed. Tongue mobility is a fairly unique consideration in the Ludwig patient, as the disease process itself involves the submandibular space, which may become edematous and hardened. This is important with respect to airway management because during routine laryngoscopy the tongue is displaced into the submandibular space. In the Ludwig patient, if that space is diminished or unavailable, it can make visualization of the vocal cords and subsequent intubation quite difficult. The anesthesiologist and surgeon may proceed only after this checklist has been carefully evaluated and a clear plan has been devised. P.80

The airway of a Ludwig patient may be deceiving in that it may appear to be unobstructed when the patient is awake and breathing spontaneously, but that may change dramatically after induction and paralyzation. Topically anesthetizing a patient's larynx and trachea in order to take an “awake look” may not be beneficial, as visualization of laryngeal anatomy postinduction may be greatly reduced compared to that of the awake patient, because of decreased laryngeal muscle tone. Muscle relaxants may allow the edematous tissues adjacent to the trachea to relax after induction and occlude an airway that was patent in an awake Ludwig patient, possibly resulting in difficult, if not impossible, ventilation and/or intubation. Multiple intubation attempts or manipulations of a Ludwig patient's airway can be especially harmful, as it may cause the accumulation of blood and secretions in the pharynx and can potentiate further complications in already dire circumstances. For these reasons, direct laryngoscopy after topicalization of the airway is not advised in the Ludwig angina patient.

For the mildest cases of Ludwig angina, traditional direct laryngoscopy after routine induction may be sufficient. If this route is undertaken, it is advisable to have emergency airway equipment in the operating room should either ventilation or intubation prove more difficult than anticipated. For example, a laryngeal mask airway (LMA) may prove more effective than bag-mask ventilation if soft tissue collapse is a problem. Even more important than emergency airway equipment, however, is the presence in the operating room during induction of an experienced surgeon ready to provide a surgical airway should ventilation and intubation prove impossible.

For moderate Ludwig angina patients, a safer means of obtaining a secure airway is to carry out an awake intubation, in which the patient retains consciousness and spontaneous respirations until the endotracheal tube is in place. Before beginning an awake intubation, the patient must be fully informed of what he or she is about to experience, and he or she must be willing and able to cooperate with the anesthesiologist in attaining adequate airway anesthesia. To overcome the laryngeal reflexes and anesthetize the airway, a variety of methods may be used. Supraglottic anesthesia can be obtained via nebulized topical anesthetic followed by lidocaine-soaked pledgets, gargles, or sprays. Bilateral superior laryngeal nerve blocks may also be performed to anesthetize the cricothyroid muscle and to ameliorate sensation from the base of the tongue to the vocal cords. Patients with a full stomach, evidence of a tumor at the site of block, and those with an active infection at the site of injection are contraindications to the use of this nerve block. Because tracheal nerve blocks can be somewhat uncomfortable, benzodiazepines and/or opioids may be used to offset the stress associated with this procedure. Caution must be taken to ensure that the patient maintains spontaneous respirations and does not become oversedated. Infraglottic anesthesia may also be warranted and can be achieved via a transtracheal nerve block by injecting via the cricothyroid membrane with 2% lidocaine. Once full airway anesthesia is accomplished, the anesthesiologist can proceed with intubation. The endotracheal tube can be passed by means of a flexible fiber-optic bronchoscope, a direct-vision, lighted stylet (Shikani optical stylet or retromolar scope), or via an intubating laryngeal mask airway (Fastrach LMA). Care must be taken to avoid contacting abscessed areas in the mouth and oropharynx with airway instruments to prevent purulent discharge from migrating into the lungs. Once the trachea has been successfully intubated and endotracheal tube (ETT) position confirmed with positive end-tidal CO_2 and auscultation, the anesthesiologist can induce general anesthesia and the surgical procedure may begin. An experienced surgeon should be present in the operating room, ready to do an emergency tracheostomy, during any awake intubation attempt. P.81

One important point to remember when considering awake intubation in a patient with Ludwig angina is that airway topical anesthetization itself may be

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difficult. The infected laryngeal tissues will likely be acidotic, which means that a greater percentage than normal of the local anesthetic will remain in its ionized form. Because the nonionized form traverses the neuronal membrane, the effectiveness of the local anesthetic may be reduced and the degree of airway anesthesia may be suboptimal.

Ludwig angina patients who present with advanced disease, evidenced by labored respirations, drooling, or limited mouth opening, are candidates for awake tracheostomy. An experienced surgeon should always be present in the operating room, ready to perform an emergency tracheostomy, regardless of the apparent level of airway involvement.

The decision to extubate the patient upon conclusion of the surgical procedure is based on the extent of the infection and the level of edema present. For more serious infections, a timely course of antibiotics may be necessary to alleviate the swelling and allow for an unimpeded airway before extubation. For patients of this type, who were preoperatively classified as having a “difficult” airway, a decision to remain intubated should be made, along with arrangements for postoperative sedation. All patients with considerable edema of the pharynx, submandibular space, and tongue should remain intubated indefinitely, until it is certain that a natural airway can be maintained. It is not uncommon for a Ludwig patient to remain intubated and mechanically ventilated for several days postoperatively, until extubation criteria have been met. It is mandatory that any patient with Ludwig angina who was intubated orally be able to breathe around the endotracheal tube with the cuff deflated before extubation. This helps to ensure that the edema has subsided and a patent airway is present without the endotracheal tube in place. In patients who have required a tracheostomy, maintaining the tracheostomy site is inevitable; however, whether the patient is kept on mechanical ventilation or allowed to breathe spontaneously depends on the patient's prior medical history (presence or absence of lung disease, localized versus systemic infection, etc). P.82

In any Ludwig angina case, meticulous attention must be paid to devising and executing the safest and most thorough anesthetic and surgical plan. A thorough understanding of the “difficult” airway algorithm is paramount, as well as a close collaboration among all involved parties on the surgical and anesthesia teams. A prompt and comprehensive strategy is the key to enduring the most serious of circumstances challenging a Ludwig angina patient.

TAKE HOME POINTS

- Ludwig angina is most commonly seen after dental work. A typical scenario is for the patient to present in the evening or on a weekend after an urgent call to the dentist or endodontist that resulted in referral to the emergency department.
- Airway assessment for these patients incorporates all the basics, plus an assessment of tongue mobility, drooling and/or pooling of secretions, and ability to swallow.
- *Do not “spray the patient up for a quick look” while you are waiting for the surgeons to arrive.* This can and has resulted in patient mortality.
- Depending on the severity of the airway obstruction, either very careful awake or anesthetized techniques are appropriate. Err on the side of conservative treatment.
- Do not attempt an airway maneuver unless you can rapidly progress to a definitive, surgically secured airway. This mandates the presence of an experienced surgeon, ready to do a surgical airway. P.83

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Remember that the IV Start is Your First Chance to Make a Favorable Impression on the Patient

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Catherine Marcucci MD

Intravenous access is a crucial part of anesthesia care, and placement and management of lines are important skills for an anesthesiologist. Virtually all anesthetics require some degree of intravenous (IV) access, whether it is for induction of general anesthesia, administration of medications, fluid resuscitation, or blood sampling.

The majority of adult surgical cases start with a peripheral IV for induction of general anesthesia, initiation of conduction blockade, or conscious sedation (for pediatric cases, it is usually appropriate to induce via an inhalational agent; however, endotracheal intubation should not be attempted until IV access is obtained). When performing regional anesthesia, it is also important to have an IV, not only for the administration of sedative and anxiolytic medications, but also to respond to potential hemodynamic changes related to neuraxial or peripheral blocks.

Peripheral IV placement can be one of the most challenging procedures in anesthesia, especially for beginners. When placing a peripheral IV, always make sure to:

- The initial needle stick can be very unpleasant for some people and a startled patient may move suddenly, making the procedure more difficult.
- If your operating room has a practice that allows IVs to be started in seated patients, always ask about and watch for vasovagal reactions. It seems counterintuitive, but often the patients who have the worst vasovagal reactions are the youngest and healthiest. One of the authors once saw a strapping young ex-marine slide right out of his chair onto the ground. Of course, there was a huge ruckus in the preoperative area. His family said, “oh, we forgot to tell you, he does that all the time.”
- Choose a location that is convenient for you and the surgeon. For example, don't place an IV in the right hand if the patient is having a right-sided carpal tunnel release. Consider carefully before starting an IV in the foot of a patient with diabetes or significant soft tissue changes in the lower extremities. Some anesthesia providers will do it, but the podiatrists tend to advise against it.
- Use a tourniquet to engorge veins. A great technique before placing the tourniquet is to have the patient exercise the extremity against resistance for 20 seconds and then “hang” the extremity in a dependent position. This will provide a manyfold increase in blood flow to the extremity. To avoid cutting off arterial flow, do not make the tourniquet too tight. Put the tourniquet as close to the location of the vein that is being accessed as possible, so you don't forget to remove it. Consider the use of a forcing function as well—the person who puts the tourniquet on the patient is responsible for removing it—to make sure this happens, put a tourniquet on yourself or your pen or your stethoscope at the same time. Be especially careful if you have put a tourniquet on the lower leg, for some reason, these seem to be “forgotten” more often than tourniquets placed on the upper extremity. P.87
- Flush your IV tubing before connecting it to the catheter to avoid letting air into the patient's circulation.
- Have gauze, tape, and dressings readily available and within your reach before you begin placing the IV. You don't want to lose a newly placed IV simply because you are fumbling with supplies. Have someone assist you whenever possible.
- If the patient shows signs of being a difficult stick, ask him if he can recommend a site.
- Only one person sticks the patient at a time!
- Advance your needle far enough that the catheter is within the vein. Remember that a flash of blood in the angiocath indicates that the *needle* has punctured the wall of a vein, but the catheter might still be outside. This is especially true in larger-bore IVs, in which there is 2 to 3 mm between the tip of the needle and the tip of the catheter.
- Give the patient reliable information. If you tell him that you will try only one more time before having someone else try, keep your word.
- Don't be greedy, the truism about the 18-gauge IV that runs versus the 16-gauge hematoma may be annoying when someone says it to you, but it is true.
- Recognize when it's “not your day” and don't be shy about having another anesthesia provider do the IV if you can't after about two (or so) tries.
- There are many individual styles of securing the IV once it is inserted, and this has actually been the subject of scientific inquiry. One study found that the “double-chevron” method, using two pieces of tape for the hub and the barrel of the needle, gave superior results (Fig. 19.1). Flat transparent dressings are generally superior to gauze dressings. Take care not to wrap the IV tubing circumferentially around the arm or hand, and don't make “purse handles” with extra long loops of unsecured tubing.

Although a single IV is adequate for some anesthetics, sometimes it is necessary to place additional ones. This is typically done after induction, because not only does it avoid patient discomfort, the vasodilating effects of general anesthesia will often make peripheral veins more obvious. Some indications for starting a second IV are P.88

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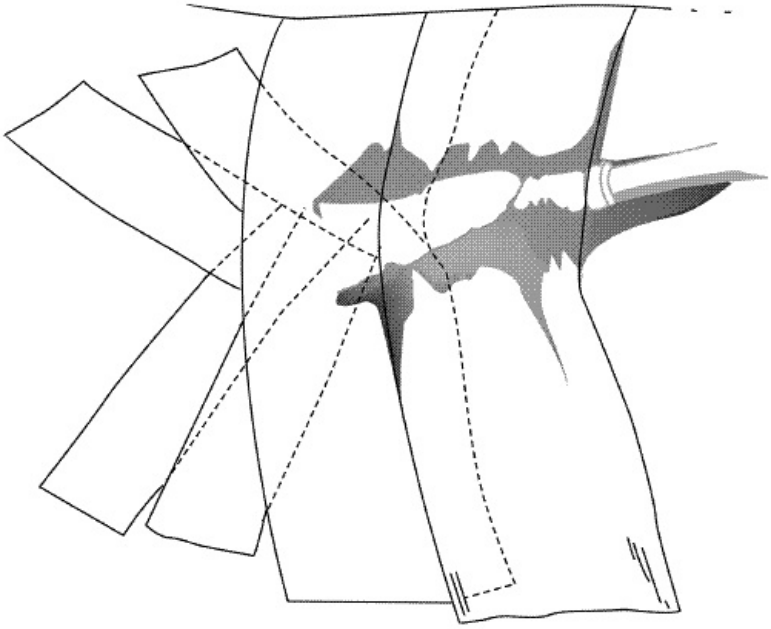


FIGURE 19.1. Preferred taping method. [Modified from Patel N, Smith CE, Pinchak AC, et al. Evaluation of different methods of securing intravenous catheters: measurement of forces during simulated accidental pullout. *Can J Anaesth* . 1995;42(6):504-510.]

- The potential for large-volume blood loss. Although administration of medications can be done with small-caliber IVs, replacement of fluids and blood products is best done through large peripheral IVs, typically 18 gauge or larger. The larger the diameter of the catheter, the lower the resistance to flow, and the more rapidly you can push fluids. Always make sure to discuss the expected blood loss with the surgeons before the operation, and when in doubt, play it safe! Also, remember to obtain informed consent from your patient for all blood products.
- When patient positioning is such that obtaining additional IV access during surgery will be difficult. For example, if both arms will be tucked to the sides, it will be wise to start a second IV just in case one fails during the procedure.
- If you are planning to use a continuous infusion. Although it is not entirely necessary, it is sometimes preferred to use a dedicated IV at a constant rate for infusions, so that fluid and medication boluses do not disrupt the flow rate.

TAKE HOME POINTS

- Be confident and calm when starting an IV on an awake patient.
- Remember that peripheral IV skills can atrophy a bit even during a two-week vacation.
- Never argue with patients about their veins—if they insist they will be difficult, agree and tell them you will use the techniques used for children.
- Control the tourniquet!
- Exercise the extremity before placing the tourniquet.
- Make sure you are in the vein before trying to advance the catheter.
- Remember that the IV start is a chance to make a good impression, but unfortunately, it is also a chance to make an unfavorable impression.

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Never Use An Intravenous Line Without Palpating and Inspecting It Visually

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Juraj Sprung MD, PhD

Intravenous (IV) infiltration is the unintentional extravascular leakage of IV fluid. Signs of infiltration include tissue edema, skin blanching or erythema, and skin temperature change. Patients may report pain and tightness around the IV site. Sites that are commonly infiltrated include the hand, forearm, and antecubital fossa, which are the most frequent sites of IV placement. Patient populations associated with higher rates of infiltration are critical care and oncology patients, owing primarily to the increased number of IV lines placed at a given time and the number of times that veins need to be accessed (e.g., repeated treatments with cytotoxic agents). Age groups at highest risk include the elderly and neonates. Neonates have small vessels and immature skin, whereas elderly persons have fragile vessels and skin. Placement of the IV outside the vessel in loose subcutaneous skin may mimic proper IV placement. Before use, every IV should be inspected visually for IV infiltration.

PERIOPERATIVE IV FLUID CONCERNS

Perioperative IV fluid therapies include crystalloids and colloids. Crystalloids are water-based solutions, usually with low-molecular-weight salts (vs. ions), whereas colloids contain high-molecular-weight proteins or glucose polymers. Another type of infusing material, called vesicants, has greater potential for substantial cellular injury. Vesicants include radiocontrast dyes, pressor agents, and chemotherapy solutions. In theory, colloid solutions may increase extravascular fluid content after infiltration by drawing fluid out of the cells, whereas any hypotonic solution may lead to cellular rupture after fluid absorption. Dyes act more like hypotonic solutions and include methylene blue and indocyanine green. Infiltration with these dyes may result in severe skin discoloration, but this effect is not permanent. The duration of discoloration is dependent on the rate of dye (fluid) reabsorption from the extracellular space, usually 12 to 24 hours. Hyaluronidase injected in multiple sites around the infiltration site has been used to treat mannitol infiltration.

INTRAOPERATIVE MONITORING

Before anesthetic induction, initial palpation of the IV site is recommended. If fluids (or medications) are administered by syringe extravascularly, they may elicit high pressure on injection, causing pain in awake patients. Relying on a free-flowing IV may lead to false positives, particularly in the elderly, for whom loose skin may allow easy infiltration. For catheters that are not located in an extremity, such as central lines, be mindful of which vein the catheter is expected to enter because infiltration will depend on its position in the body; extravasated fluid may leak into the arm, chest wall, or neck. Positive aspiration of blood must be achieved, providing the IV was placed in the major central vein. Transducing the catheter should be done to confirm venous placement of a central line.

P.91

If the extremity is exposed during surgery, continuous monitoring of the IV site for edema or blanching are necessary. Jewelry such as rings should be removed before surgery, especially for arms that are tucked in (placed along the body and covered with drapes) and arms in which the IV catheter has been placed. However, the frequent practice of tucking in arms requires careful IV monitoring. Should the IV stop flowing intraoperatively, full inspection of the IV site must be made. Facilitation of a sluggish IV by using “pressure bags” should not be done until the site is properly inspected for signs of possible extravascular infiltration.

CONSEQUENCES

Consequences of infiltration can be divided into pressure-related injuries and cellular irritant-related injuries. Pressure-related injuries may expose tissue to arterial ischemia, and irritant-related injuries may expose tissue to pH differences, osmotic differences, and direct toxicity. Severe consequences of either may include compartment syndrome, tissue necrosis, ulcers, and complex regional pain syndrome. Perioperatively, vigilance is required, particularly when administering antibiotics, vasopressors, cytotoxic drugs, osmotically active substances (total parenteral nutrition, hypertonic dextrose solutions), and cationic solutions (potassium, calcium, or bicarbonate salts).

TREATMENT OPTIONS

Simple maneuvers should be done initially. First, discontinue the IV infusion. Second, elevate the affected extremity, providing this elevation does not interfere with extremity perfusion (pulse oximetry can be placed on the extremity for perfusion assessment). Application of warm compresses, to decrease pain and swelling, may help for all fluids except vesicants. Warming a site extravasated with vesicant fluid may increase induration of the area and slow reabsorption. Ice compresses should be applied to a vesicant extravasation site for the first 24 hours, with subsequent moist heat thereafter until inflammation decreases.

Certain vesicants have specific antidotes. For example, norepinephrine extravasation can be treated with local infiltration of phentolamine. Chemotherapeutic agents have specific antidotes depending on the agent infiltrated. Invasive techniques such as saline flushout and liposuction have been described. The worst cases may be treated by fasciotomy, débridement, and split skin grafting. However, a fairly recent study examining extravasation injuries in three hospitals recommended conservative treatment. A more recent study suggested prompt action and appropriate intervention should irritant injuries occur with vasopressors, hyperosmolar solutions, and concentrated electrolyte solutions in the perioperative period. If a substance is known to be an irritant, it should be used in a stable vein and in a vein without prior intravenous attempts.

P.92

CONCLUSIONS

After an injury occurs, management should fall into two categories, operative or nonoperative. Postinfiltration management should include frequent assessment and, most important, perfusion of the affected extremity. Ultimately, education of all perioperative staff on safe practices regarding intravenous access is the best approach to decrease the incidence of these complications.

TAKE HOME POINTS

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- Be aware of which patients are at high risk for infiltration of the IV—the very young and elderly, ICU patients, and chemotherapy patients.
- Do *not* depend on the rate the IV is flowing to ensure that fluid will not extravasate—always inspect visually and palpate an IV before using it.
- Do *not* put a pressure bag on an IV that is suddenly running sluggishly—it is infiltrated until proven otherwise.
- Injuries are categorized as pressure-related or irritant-related. Some hospitals have policies involving peripheral infusion of soft tissue irritants such as potassium—know them.
- Treatment options range from conservative to invasive, depending partially on distal perfusion.
- Call the hospital pharmacy to see if there is a specific antidote.

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Use of Ultrasound Guidance for Cannulation of the Central Veins Improves Success Rates, Decreases Number of Attempts, and Lowers Complication Rates

Michael Aziz MD

Ultrasound guidance for placement of central venous catheters is becoming the standard of care across medical disciplines. Doppler localization of vascular structures has been used for many years, and the literature has slowly come to prove the utility of ultrasound. Newer portable equipment has made ultrasound useful and practical at the bedside. The widespread use of ultrasound has been slower to occur than might be anticipated. The success rate for internal jugular cannulation by the landmark technique is impressive at 95% to 99%. Many clinicians with skilled hands do not feel that they need assistance in localizing a central vein because they have had such dramatic success with the landmark method. However, in reviewing the studies that document such widespread success for internal jugular cannulation, we find that they often require multiple attempts and have a significant complication rate. A review of the literature demonstrates that two-dimensional ultrasound guidance for central venous access improves the success rate, reduces the complication rate, and reduces the number of attempts when compared to landmark techniques.

Ultrasound guidance improves cannulation success rates. For the internal jugular vein there is a relative risk reduction of 86% for failed cannulation in adults, and risk reduction of 85% in infants. For the subclavian vein, data demonstrate a relative risk reduction of 86% for failed cannulation. For the femoral vein, there is a relative risk reduction of 73%. These data are compiled from meta-analysis of large randomized controlled trials and are statistically significant.

Ultrasound guidance also reduces the likelihood of complication during central venous cannulation. There is a relative risk reduction of 57%. With a complication rate of 5% in experienced hands and 11% in inexperienced hands, these findings stand to improve patient safety and outcome dramatically.

Ultrasound guidance reduces the number of attempts for venous cannulation. The relative risk reduction for successful cannulation on the first attempt is 41%. Ultrasound also decreases the time to successful cannulation.

Some clinicians believe that ultrasound guidance adds time and burden to their procedures, but the evidence demonstrates fewer attempts and shorter times to successful venous cannulation. P.95

The data presented above demonstrate a favored approach for ultrasound over landmark methods for central venous cannulation. Other metaanalyses have pooled data that include Doppler localization into the ultrasound group and could not demonstrate results as favorable. The Hind study demonstrates that Doppler localization (as opposed to two-dimensional ultrasound) is less successful and more time-consuming than even landmark methods. These data, therefore, support the use of two-dimensional ultrasound over Doppler localization and the landmark method.

There are several reasons why this author believes that the literature has come to prove that ultrasound guidance improves safety and success with venous cannulation. When the landmark method is used and a cannulation becomes difficult, subsequent ultrasound often demonstrates venous thrombosis or absence of a patent vein. This difficulty is completely avoided by using ultrasound first and guiding the choice of central vein based on visualized venous patency. I think direct visualization of a needle compressing a vein avoids carotid and pleural puncture. With the landmark method, at times the needle is passed directly through the vein and into the carotid artery without venous aspiration, because the vein becomes compressed from needle pressure. Ultrasound shows this compression occurring as the needle is passing and keeps one from passing the needle into deeper structures such as arteries, pleura, or nerves. Often, central lines are placed during states of hypovolemia, when veins are especially prone to collapse.

Widespread use of ultrasound guidance for central venous access remains limited for several reasons. Some institutions cannot justify the cost of high-resolution ultrasound equipment. I argue, however, that the health care system stands to save money from reduced complication costs. Other clinicians fear that the use of ultrasound will impair their ability to cannulate a vein in an emergency situation using the landmark method. Ultrasound does not affect our ability to use landmarks as well. As I teach these methods, I like to identify the landmarks and expected course of the vein before ultrasound identification. In this way, students gain skill in both methods. Also, in an emergency situation, an available ultrasound probe is likely to expedite venous cannulation, not slow it down. However, if a probe is not available in an emergency situation, the landmark methods become necessary.

Practice habits have varied to accommodate the above considerations. Some authors advocate the use of ultrasound only in situations that predict difficult central vein access, such as previous neck surgery, multiple previous cannulations, hypovolemia, existing catheters or pacemaker, and obesity. Other clinicians use ultrasound to confirm the landmark anatomy,

then remove the probe from the field. Others use ultrasound only after failed attempts using the landmark method. In such scenarios, the landmark method skill is retained for emergency situations. Although these practices have merit, evidence favors a prepuncture ultrasound scan with ultrasound-directed needle placement and cannulation. P.96

TAKE HOME POINTS

- Ultrasound guidance is becoming the standard of care in placement of central venous access.
- Even in studies in which success rate was extremely high using the landmark technique, ultrasound guidance decreased the number of attempts and complication rates.
- Ultrasound guidance for access can be used for infants as well as adults.
- Two-dimensional ultrasound guidance is a superior technique for central line placement over Doppler localization.
- Resistance to adopting ultrasound guidance as a standard technique is still prevalent—concerns that it slows line placement time are not supported by the literature.
- Maintain skill in the landmark technique by appreciating and outlining the pertinent anatomic landmarks before placing the ultrasound probe.

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Central Line Placement: Never Neglect the Basics

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Intravenous (IV) access is a crucial part of anesthesia care, and placement and management of lines are important skills for both the anesthetist and anesthesiologist. Virtually all anesthetics require some degree of IV access, whether it is for induction of general anesthesia, administration of medications, fluid resuscitation, or blood sampling.

Before attempting to place a central line, be sure you are well versed on anatomic landmarks and appropriate techniques. This is never a benign procedure, so always make sure to weigh the risks and benefits and discuss them with the patient as part of your informed-consent process. Central line placement has been associated with several complications, including:

- Accidental arterial puncture with pseudoaneurysm or hematoma
- Pneumo- or hemothorax
- Venous air embolism
- Infection and sepsis
- Cardiac arrhythmias

The decision to place a central line is multifactorial. Always assess the overall clinical picture, and discuss with the surgeon and intensive care unit (ICU) staff if possible. Some of the indications for obtaining central venous access are

- Inability to obtain peripheral venous access.
- Cardiac arrest or “code” situation. In this case, a femoral approach is best, as it does not interfere with chest compressions or endotracheal intubation.
- Administration of certain medications. Generally, any medication that can cause direct damage to peripheral veins must be given centrally, for example, 3% saline. Also, highly concentrated vasopressors can cause vasospasm if given through a peripheral vein. Check with the pharmacy for other medications that need to be given centrally.
- Central venous pressure monitoring. Although not considered to be a highly accurate measure of volume status, it can be used in conjunction with other clinical signs to guide fluid management.
- Massive blood transfusion. As mentioned above, large-diameter catheters are preferred for rapid transfusion, but remember that the *length* of the catheter also contributes to resistance to flow. For example, a short 16-gauge peripheral IV has much less resistance than a long 16-gauge central line. For rapid infusion of large volumes, a large-bore, short catheter, such as a Cordis or introducer sheath, is best. P.98
- Potential need for a pulmonary artery catheter placement. A right-sided internal jugular or left-sided subclavian introducer is preferred.
- High risk of venous air embolism (VAE) during surgery. Be familiar with the clinical signs of VAE and potentially high-risk procedures. In addition to placement in the left-lateral decubitus position, flooding the surgical field with fluid, increasing FiO_2, and discontinuing nitrous oxide, aspirating through a central line may remove air from the right side of the heart. In this case, a multilumen catheter is preferred.

Some basic things *always* to remember when placing a central line:

- Be aware of coagulopathy, thrombocytopenia, or any other contraindication to a particular line (thrombosis, existing hardware, etc).
- Choose your site wisely. For example, in a patient with a right-sided pneumothorax, choose a right IJ or SC to avoid the risk of bilateral pneumothoraces.
- Place your patient in Trendelenburg position, or at least flat, when placing an internal jugular or subclavian line. This minimizes the risk of venous air embolism.
- Create a sterile field. Studies have shown that infection risk decreases significantly when proper sterile techniques, including a full body drape, are used.
- Be familiar with your supplies and have them ready and within your reach before you begin the procedure. Get an experienced nurse to help you whenever possible.
- Be aware of your patient's condition during the procedure. Draping can make an awake patient very anxious, and a head-down position can exacerbate orthopnea or gastric reflux. Use monitoring and inhaled oxygen whenever possible.
- Always have control of the guidewire!
- Confirm venous (versus arterial) placement. There are several ways to do this. The most accurate is ultrasound-guided placement. However, if you do not have access to that equipment and the clinical situation warrants central line placement, the options include a simple “drop-test,” transducing a pressure tracing, or obtaining a radiograph. Do not use the line until you are convinced it is in the right place.
- Secure the line with sutures and a sterile dressing.

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Approach the Use of a Pulmonary Artery Catheter with Caution

Amy V. Isenberg MD

The pulmonary artery (PA) catheter is an invasive hemodynamic monitor utilized to both diagnose and manage certain conditions. Optimally placed catheters give direct measurements of central venous, pulmonary artery, and pulmonary artery wedge pressures.

Features of the PA catheter (PAC) include a thermistor at the tip to estimate cardiac output (CO) and systemic and pulmonary vascular resistance, the ability to perform mixed venous oximetry, the option of pacing, and the measurement of right-heart cardiac output in order to infer left-heart output.

USES FOR THE PAC

The pulmonary artery catheter, although controversial, has demonstrated continued usefulness in differentiating shock states, defining the etiologies of respiratory and cardiac failure, and assessing intracardiac shunts and regurgitation of mitral and tricuspid valves. The PAC also has use in several specific surgical situations involving large fluid shifts or potential cardiac damage, such as liver transplantation, aortic cross-clamp cases, and large operating room (OR) cases involving renal-failure patients. Although PACs were traditionally used to guide therapy in patients with acute myocardial infarct, recent studies have shown that PAC tracings are marginally beneficial in sensing ischemia, except in the instance of intraoperative right ventricular ischemia.

Perioperative use of the pulmonary artery catheter is common in the intensive care unit (ICU) setting. Many patients in the ICU suffer multiple organ dysfunctions, and data from PACs are useful in detecting and defining various hemodynamic disturbances for therapy implementation. Infection becomes a greater risk with ICU use, particularly for more than 6 days.

RISKS OF THE PAC

Pulmonary artery catheterization may be complicated by adverse events that occur during access of the central venous system, positioning of the PA catheter, and catheter "residence". According to American Society of Anesthesiologists (ASA) 2003 data (Table 23.1), the most common complication in obtaining central venous access is postoperative neuropathy, followed by arterial puncture, air embolism, and pneumothorax. Placement of the PAC commonly causes minor dysrhythmias (4.7% to 68.9% reported incidence), followed by ventricular tachycardia or fibrillation, right bundle branch block, and complete heart block preceded by left bundle branch block; the catheter may also coil or knot. Once it is in place, the PAC can rupture the pulmonary artery (0.1% to 1.5% reported incidence), become infected and lead to sepsis, or cause thrombophlebitis, venous thrombosis, pulmonary infarction, mural thrombus, or valvular or endocardial vegetations. Deaths attributed to pulmonary artery catheters have a reported incidence of 0.02% to 1.5%.

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TABLE 23.1 ADVERSE EFFECTS ASSOCIATED WITH PULMONARY ARTERY MONITORING

COMPLICATION	REPORTED INCIDENCE (%)
Central venous access	
Arterial puncture	1.1-13
Postoperative neuropathy	5.3
Pneumothorax	0.3-1.1
Air embolism	0.3-4.5
Positioning of the PAC	
Minor dysrhythmias	4-68.9
Ventricular tachycardia or fibrillation	0.3-62.7
Right bundle branch block	0.1-4.3

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Complete heart block (prior LBBB)	0-8.5
Complications associated with catheter residence	
Pulmonary artery rupture	0.1-1.5
Positive catheter tip cultures	1.4-34.8
Sepsis secondary to catheter residence	0.7-11.4
Thrombophlebitis	6.5
Venous thrombosis	0.5-66.7
Pulmonary infarction	0.1-5.6
Mural thrombus	28-61
Valvular or endocardial vegetations	2.2-100
Deaths attributed to PAC	0.02-1.5

Source: ASA Task Force on Pulmonary Artery Catherization. Practice guidelines for pulmonary artery catherization: an updated report by The American Society of Anesthesiologists Task Force on Pulmonary Catherization. *Anesthesiology*. 2003;99:998-1014.)

SPECIAL ISSUES IN PAC INSERTION

Sterile technique, including use of mask, cap, sterile gloves and gown, and large sterile drape, must be applied to avoid complications with infection. Use of 2% chlorhexidine is recommended for skin preparation. Remember that insertion requires two people—one sterile person to "float" the catheter and a knowledgeable nonsterile person to assist with the transducers, flush,

and balloon inflation. Also, a surprisingly common error is to forget to place the sterile sleeve on the catheter before insertion or to put in on backwards. P.102

The right atrium is most easily accessed via the right internal jugular vein. The left internal jugular approach may be complicated by damage to the thoracic duct as well as by variable anatomy such as may exist in patients with congenital heart disease. Additionally, the left internal jugular vein may drain into a persistent left superior vena cava. Approach from the subclavian veins (higher risk of pneumothorax) or from various peripheral veins (higher risk of venospasm, which increases placement difficulty or thrombosis) may be sought as well.

The electrocardiograph (EKG) should be monitored continuously during placement of the PAC, and a defibrillator should be available. When the PAC has been inserted through the introducer, the balloon should be inflated at approximately 20 cm and should always been inflated as the catheter is advancing (and the balloon should always be deflated for withdrawal). Avoid coiling and knotting of the catheter (which can result in the need for surgical or radiologic intervention) by not inserting excessive lengths of catheter. The wave pattern should be monitored for its characteristic form as the catheter tip passes through the right atrium, right ventricle, pulmonary artery, and the wedge (Fig. 23.1). Persistent premature ventricular contractions may be suppressed with lidocaine. The right ventricular trace is commonly measured

at the 30- to 35-cm mark on the catheter, the PA trace at 40 to 50 cm, and the pulmonary capillary wedge pressure trace at 50+ cm. If the characteristic tracings are not readily observed, the balloon should be deflated and the catheter withdrawn to 20 cm. P.103

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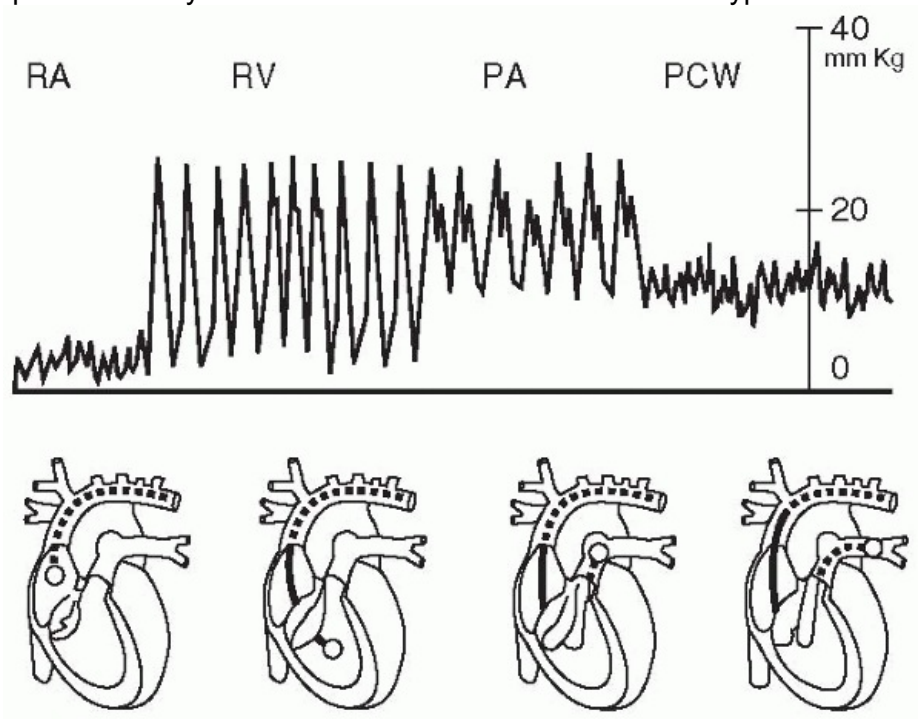


FIGURE 23.1. Picture of waveform change as catheter tip progresses. (From Dizon CT, Barash PG: The value of monitoring pulmonary artery pressure in clinical practice. *Conn Med*. 1977;41:622-625.)

The proximal port should reside in the right atrium in order to transduce right atrial pressure and to inject thermodilution fluid. The distal lumen, when properly seated, will transduce pulmonary artery pressure and pulmonary capillary wedge pressure.

The risk of pulmonary artery perforation can be minimized by not "overwedging" the balloon, by minimizing the number of balloon inflations, and by using proper balloon technique (inflate slowly and deflate passively). Do not leave the balloon inflated continuously. As balloon rupture can devastate patients with right-to-left intracardiac shunts, the anesthesiologist may use CO_2 in the balloon instead of air. Remember that PA rupture is more common in patients with pulmonary hypertension, partly because the PAC is more difficult to place.

In patients with known dysrhythmia, especially bundle branch blocks, external pacing/defibrillator pads placed before placing the catheter may help manage dysrhythmias induced by line insertion. Patients with left bundle branch block may be candidates for fluoroscopic placement of the PAC to expedite passage of the catheter through the right ventricle to reduce the chances of instigating a right heart block (which would leave the patient in complete heart block with the need for immediate transthoracic or transvenous cardiac pacing). Fluoroscopic guidance of insertion is also useful in patients with right atrial or ventricular dilatation, and in those with severe tricuspid regurgitation. Transesophageal echo may be useful in intubated patients in the OR or ICU setting to ascertain correct placement.

SPECIAL ISSUES IN PAC DATA INTERPRETATION

Unfortunately, intraobserver variation in data interpretation has been reported among critical care specialists and anesthesiologists. Proper utilization of the PAC necessitates knowing how best to position the catheter to optimize data generation as well as understanding what the data signify in different situations.

The transducer must be "zeroed" with atmospheric air. Correct equipment calibration requires that the transducer be level with the right atrium (approximately 5 cm below the sternal angle of Louis). Remember that wave damping may result from clot formation over the catheter tip, air bubbles, or tip against the pulmonary artery wall. Cardiac contractions causing catheter motion may lead to "fling" or "catheter whip." High-frequency components of the pressure-wave signals matching the resonating frequency of the fluid-filled monitoring system can result in harmonic resonance. The resonating frequency can be altered by changing the length of the tubing or using tubing made of a different material, preferably of a rigid, noncompliant type.

Spontaneous respiration may result in negative and positive pressure artifacts being transmitted to the PAC during inspiration and exhalation, respectively. Positive-pressure artifacts may be seen during mechanical ventilation. The degree of artifact is affected by lung and chest wall compliance, PAC position, and by the use of positive end-expiratory pressure (PEEP).

Although left atrial pressure is generally a good estimate of left ventricular end diastolic pressure (LVEDP), in mitral stenosis, left atrial pressure (LAP) is greater than LVEDP. LAP is also greater than LVEDP in mitral regurgitation, with systolic retrograde flow resulting in a large V wave on the PCWP tracing. Conversely, LVEDP is greater than LAP when there is retrograde flow from the aorta into the ventricle, as with aortic regurgitation and premature closure of the mitral valve.

Preload/left ventricular end-diastolic volume (LVEDV)/LVEDP measurements may be inaccurate with changes in compliance. Factors that decrease compliance include myocardial ischemia, restrictive cardiomyopathy/myocardial fibrosis, right-to-left interventricular septal shift, aortic stenosis, cardiac tamponade, effusion, inotropic drugs, and hypertension. Factors that increase compliance include vasodilators, congestive myopathies, left-to-right interventricular septal shift, and mitral and aortic regurgitation.

Problems with data interpretation occur when pulmonary vascular resistance is increased, reducing pulmonary blood flow and changing the relationship between PCWP and LAEDP. This relationship can also be modified in conditions such as acute/chronic lung disease, pulmonary emboli, hypoxia, acidosis, and hypoxemia. Drugs that increase pulmonary vascular resistance, and tachycardia (which shortens ventricular diastole), will increase pulmonary vascular resistance and affect the accuracy of the PAC data.

Finally, the PAC tip must be located in Zone 3 (PAP > PA < PVP), where there is an open fluid channel between the catheter tip and the left atrium. Supine

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patients will generally have the catheter at this level. However, PEEP and hypovolemia can alter the pressure relationships in this zone.

OUTCOMES ASSOCIATED WITH THE PAC

The effect of PAC monitoring remains controversial. The 1997 Pulmonary Artery Consensus Conference produced a statement concerning PAC use in cardiac and noncardiac surgery. PAC use demonstrated no benefit or uncertain benefit in patients undergoing cardiac surgery, low-risk aortic surgery, neurosurgery, or in surgical patients >65 years old. The experts supported use of PACs to reduce complications in patients undergoing peripheral vascular surgery and high-risk aortic valve surgery. It was recommended

that in the absence of severe lung disease, pulmonary hypertension, or impaired right ventricular function, central venous pressure monitoring alone may be adequate to guide fluid management. The 2003 ASA guidelines offer suggestions for appropriate use of the PAC in the current OR and perioperative environment. New technology offering continuous cardiac output monitoring allows the ability to detect changes in ventricular performance as alterations occur; how this will change current clinical practice is still being discussed. Controversy continues over anesthesiologist preference for PAC versus transesophageal echocardiography (TEE) as well as the timing of PAC placement (before or after induction of anesthesia). As such, there is considerable variation in how the use of a PAC is "taught" to anesthesia students.

TAKE HOME POINTS

- The risk/benefit ratio for use of a PAC favors certain specific indications, e.g., sepsis, persistent hypotension of unknown etiology, fluid management in the anuric patient, and "special" cases (acute anterior myocardial infarction, high aortic cross-clamp, liver transplantation).
- Insertion requires sterile technique and two people.
- Do not overwedge the catheter, and *always inflate the balloon carefully!*
- Remember that as experience increases, anesthesia providers tend to have increasing respect for the possible morbidity and mortality incurred with the use of these catheters.
- Expect that anesthesiologists will use the measured and calculated values differently in managing care—do not be shy about asking why some practitioners may feel the cardiac output is the single most important value and others may more carefully follow left ventricular stroke work index.

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Avoid Technique-Related Central Venous Catheter Complications By Using Modern Tools

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Karen Hand MD

Placement of central venous catheters is a routine procedure in modern anesthesia care. The somewhat routine nature of this procedure should not lull the anesthesia provider into complacency regarding the importance of meticulous attention to technique. Of the many thousands of central venous catheters placed each year, the U.S. Food and Drug Administration (FDA) estimates that approximately 10% will be associated with a complication, 52% of which are related to practitioner technique. The cost of these complications to the U.S. health care system exceeds $1 billion annually.

In anesthesia practice, right internal jugular vein cannulation is most commonly selected. This is because of the relatively straight path to the superior vena cava, increased distance from the cupola of the lung (vs. left side), and absence of the thoracic duct on the right side. The potential for nerve, vessel, or lung injury exists for any approach to central venous catheterization, including the right internal jugular approach. The subclavian approach increases the risk of pneumothorax because of the proximity of the pleura to the subclavian vein. In addition, if the subclavian artery is punctured, it is not possible to control hemorrhage with direct pressure. Femoral vein cannulation is associated with increased risk of infection, but can be a good alternative if the line is intended for rapid volume infusion. Peripherally inserted central catheters can also be used. Catheter length hinders this option for the purpose of rapid volume infusion.

Although infections are responsible for approximately 75% of catheter complications, American Society of Anesthesiologists (ASA) closed claims data indicate that technical complications during placement are the most deadly. Pneumothorax, wire or catheter embolization, air embolism, extravasation of fluid or blood into the neck, and a variety of cardiac and vascular injuries may occur. Of these, direct arterial injury and vascular injury resulting in cardiac tamponade or hemo/hydrothorax have the worst mortality (Table 24.1). In addition to site of catheter placement, the risk of pneumothorax can be diminished by using ultrasonic guidance and properly angling the needle away from the pleura on initial approach to the vessel. Wire embolization is avoided by using a technique of catheter insertion that permits one hand to continuously control the wire until it is removed. Catheter embolization is prevented by taking care to avoid withdrawing the catheter at any time over a needle with a cutting bevel, which can shear the tip. Air embolization is of particular concern when the patient is breathing spontaneously or when the insertion site is above the level of the heart. Avoid this complication by using the Trendelenburg position (which improves identification of neck vessels as well), occluding the catheter/introducer with a gloved finger, and taking care to eliminate air from the catheter itself with aspiration and flushing either before or after insertion. The risk of fluid or blood extravasation is avoided by ensuring that the skin nick for the introducer does not lacerate the vein being cannulated, and by suturing the catheter securely to avoid the tip slipping. Cardiac tamponade usually results from a catheter with the tip inside the right atrium or angled against the wall of the superior vena cava, which can erode through the thin-walled atrium or vessel. This usually occurs after hours or days. The most serious direct vascular injury is caused by the inadvertent insertion of the catheter or introducer sheath directly into an artery that was mistaken for a vein.

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TABLE 24.1 OVERALL COMPLICATIONS AND FATALITIES FOR THE PERIODS 1978-1989 AND 1990 AND LATER

COMPLICATIONS	1978-1989	1978-1989 FATALITIES	1990 & LATER	1990 & LATER FATALITIES
Cardiac tamponade	10	8	2	2
Wire or catheter embolism	10	0	0	0
Vascular injuries	16	9	16	7
Hemothorax	6	6	5	3
Hydrothorax	3	1	1	1
Carotid artery injury	5	1	9	3
Subclavian artery injury	2	1	1	0
Pulmonary artery rupture	4	4	1	1
Pneumothorax	7	1	3	0

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Air embolism	2	1	1	1
Fluid extravasation in neck	0	0	3	1
Total	49	23	26	12

Eliminating life-threatening vascular complications entails correct positioning of the introducer and catheter within the appropriate central vein. Fortunately, modern tools are available to assist the anesthesia provider in correct placement and definitive confirmation that the catheter will be inserted into a vein and not an artery. Subjective methods of ascertaining correct placement such as evaluation of the pulsatile quality or color of the blood are unreliable. This is because they can depend on variable factors such as oxygen saturation of the blood or arterial/venous pressure. Use of two-dimensional ultrasound as a guide to initial venous puncture is the best means of avoiding insertion complications. A 2001 review of the literature by the Agency for Healthcare Research and Quality revealed that use of ultrasound for placement of central venous catheters improved catheter insertion success rate, diminished the number of venipuncture attempts, and reduced the number of overall line complications. The best means of confirming correct line placement is a two-step process: step one is to tranduce the pressure in the vessel before placing the introducer (ensure that it is not an arterial waveform), and step two is to confirm proper placement via a chest radiograph either intraoperatively or at the end of the procedure. A retrospective study of 1,021 central lines by Jobes et al. revealed that among 43 cases of arterial puncture, five resulted in inadvertent placement of 8.5-French catheters in the carotid artery and one patient subsequently died. Conversely, in a prospective study of 1,284 central line placements in which the line was transduced after insertion, unrecognized arterial puncture was detected in 10 patients and there were no inadvertent arterial cannulations.

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TAKE HOME POINTS

- Beside infections, the majority of central venous catheter complications are secondary to technical problems.
- Minimizing serious complications related to central venous catheter insertion can be accomplished by using the techniques discussed above.
- Strict aseptic technique and avoiding confusion between artery and vein are paramount.
- Using modern tools such as two-dimensional ultrasound and pressure transducers has been shown to reduce the number of life-threatening complications.
- Keep in mind the primary dictum of medical practice when inserting central lines: First, do no harm!

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Don't Overflush Lines

Julie Marshall MD

Peter Rock MD, MBA

Anesthesiologists become experts at the placement of lines for access and monitoring, but the routine care and maintenance of these lines may be less familiar. It is important to remember that unintentional overflushing of arterial and central lines happens commonly (especially during the busier intervals in a procedure) and can lead to significant complications. Also, patients not infrequently come to the operating room (OR) or intensive care unit (ICU) with indwelling devices already in place. These may under certain circumstances be accessed for use in the OR, provided that the anesthesia providers have a plan and proceed carefully.

Overzealous flushing of arterial lines may lead to retrograde embolization of air or clot into the central arterial circulation. It has been shown that the saline volume needed to reach the subclavian-vertebral artery junction from a radial artery averages just 6.6 mL, with a range from 3 to 12 mL. Arterial catheter continuous-flush devices deliver 3 mL/hour of saline, often with heparin, at a pressure of 300 mm Hg. Flushing the line through the flush valve or with a syringe can increase the volume and rate of delivery. Continuous-flush devices have been reported to deliver 0.8 to 4.7 mL/second when flushing an arterial line by opening the flush valve (commonly referred to as the "pigtail"). An increased rate of delivery, smaller patient size (infants), and increased flush volume all increase the potential for embolization of air bubbles into the central arterial circulation. To decrease the risk of embolization, all air should be removed from the arterial line. The drip chamber in the flush bag should be completely filled with fluid to decrease the chance of entraining air. Volume and rate of flush should be limited by opening the flush valve for only 2 to 3 seconds at a time. When flushing an arterial line with a syringe, the volumes should be small, 1 to 3 mL, and the flush should be done slowly. Both the injection port and the syringe should be free of air. Emboli may also occur from a clot in the arterial line or from arterial thrombi at the end of the catheter. If the line is not cleared following blood draws, small clots may be found in arterial lines or stopcocks. These small clots can be embolized with a forceful flush. Thrombi may form on the tip of the arterial catheter, which may also embolize with forceful flushing. These emboli have the possibility of entering the central circulation or embolizing peripherally to cause tissue ischemia. Care should be taken to monitor for clots in the line and avoid forceful flushing.

Central lines and vascular access devices are common in the OR and ICU population. Lumens not connected to continuous infusions require intermittent P.110
flushing to remain patent or before being put into use. Every line that remains patent is one less line you will have to replace. As with arterial lines, there is risk of air and clot embolization with excessive flushing of lines and/or lack of vigilance in eliminating air and clot from circuits and injections. Infection from indwelling catheters is a serious and frequent occurrence. Maintaining aseptic technique by using alcohol to clean the access port before flushing or accessing lines is required to decrease this risk. *Before flushing, the line should be aspirated and the old blood that has been sitting in the line should be discarded.* There are two reasons to do this—first, there is no reason to flush into the circulation blood that may have formed clots or be colonized with bacteria that found a nice culture medium of old blood. Second, large-bore indwelling catheters such as the double-lumen Groshong or Shiley catheters that are used for dialysis access will have between 2,500 and 5,000 units of heparin in each lumen. The frequency and volume of flush varies among institutions and depends on the type of catheter. Typically, central lines list the volume on the packaging or on the lumen of the catheter. Groshong catheters need to be flushed weekly; other central lines often need to be flushed more than once per day to remain patent. Saline or heparinized flush may be used, depending on institution preference. When in doubt, consult the ICU or dialysis services at your practicing institution; they will be able to advise you.

Patients may come to the operating room with vascular access devices, such as Port-A-Cath, Q-Port, or Infuse-A-Port, that may be used for infusions. These can be useful devices for anesthesiologists in patients with poor venous access. However, there are also several caveats to accessing these ports. First, why was the device placed? How has it been maintained, and when was it last accessed? Is it currently in use for ongoing chemotherapy treatments? Second, you must know how to access the device. Again, maintaining aseptic technique is necessary, using sterile gloves and cleaning the port site with alcohol. Palpate the skin to find the port septum, which may be on the top or the side of the device. To avoid damage to the septum, a noncoring needle (usually a right-angle Huber needle) is used to access the port. The needle is pushed through the septum until it hits the bottom of the reservoir. Aspirate for blood and discard the old blood, flush with saline, and apply a sterile dressing. The needle can be connected to an infusion or an extension set and flushed as your institution directs. Before deaccessing, the port is flushed with a heparinized solution to maintain patency. It is important to use the right amount of heparin, as an excessive amount may lead to unintentional systemic anticoagulation. If you are unsure about whether the port can or should be used and how to deaccess it, contact the oncology
service. Usually, the oncology or chemotherapy nurses will be able to advise you. Remember that these needles and ports are designed for slow drips, P.111
not for boluses, because chemotherapy is generally given over several hours. They will not look or infuse the way a normal "induction IV" does, and caution must be taken when using them to induce a patient.

Indwelling lines for access and monitoring are necessary and useful devices in anesthesiology. However, these lines have the potential for complications. Knowing the proper methods of care and access will decrease these risks and improve perioperative patient care.

TAKE HOME POINTS

- The volume of the arterial tree from radial artery to vertebral artery is only about 6 cm!
- Never "power flush" the arterial line either by hand or via a prolonged flush through the valve. This can fill the fill the 6-cm volume in 1 to 2 seconds.
- Don't flush a line if there are visible air bubbles or clots in either the central or arterial line.
- Remember that there is a *significant* amount of heparin in the lumens of the common in-dwelling catheters—always withdraw from them before you flush.
- Vascular access ports must be accessed with a noncoring needle to avoid damaging the diaphragm.

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Do Not Use the Subclavian Vein for Central Access of Any Type in a Patient Planned for Dialysis

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Catherine Marcucci MD

There are about 300,000 hemodialysis patients in the United States today, and the number is increasing by about 4% to 5% annually. The increasing incidences of diabetes mellitus and hypertension, and the relative scarcity of renal transplants (still only about 10,000 annually in the United States) mean that hemodialysis will continue to be required. For many patients who will never receive a transplant, hemodialysis must be considered a lifelong treatment. Because the critical nature of vascular access for hemodialysis is amplified by length of time on treatment, provision and maintenance of vascular access will remain one of the greatest problems in dialysis medicine.

In the most optimal situation, permanent dialysis access is placed in advance of dialysis. However, more commonly, patients present with an acute need for dialysis that requires temporary dialysis access via a percutaneous catheter. Anesthesiologists will usually be confronted with this situation as part of the intensive care management of a patient. If there is any thought that the patient may go on to require chronic dialysis, it is imperative that the temporary access not compromise the vasculature. The preferred sites for temporary access catheter placement are the internal jugular veins or the femoral veins —*not* the subclavian veins.

Temporary percutaneous dialysis catheters are associated with significant damage to the cannulated vein, and there is a very real risk of acute or delayed thrombosis or stenosis of the vein. Dialysis catheters in subclavian veins have been associated with a rate of thrombosis/stenosis of 50% to 70%, versus 0 to 10% for catheters placed in the internal jugular veins. Patients with subclavian vein occlusion may have spontaneous recanalization after 3 to 6 months and can also be treated with angioplasty and stent placement, but the vein will never be completely normal. Remember that permanent hemodialysis access involves an arterial-to-venous connection, either as a fistula (direct artery-to-vein connection) or with a prosthetic graft between the artery and vein. The longest-lasting and most successful access is placed in the upper extremity because of lower infection rates and better patient comfort. Permanent hemodialysis access will create high flow through the vessels of the upper extremity, and the success and function of the site is highly dependent on adequate venous outflow from the access site to the right atrium. Decreased venous outflow can also create profound edema that can be limb-threatening. P.113

Although the risk of injury to the subclavian vein is less with smaller catheters such as sheaths for pulmonary artery catheters and triple- or singlelumen central lines, subclavian vein cannulation with these lines should be avoided for the same reasons. Remember that subclavian thrombosis or stenosis (even if clinically silent) results in loss of all potential access sites in the ipsilateral extremity.

TAKE HOME POINTS

- Many renal failure patients will never receive a kidney transplant and will require lifelong dialysis.
- Temporary access secured in the perioperative period for dialysis and other reasons must not compromise the vasculature of the upper extremity.
- For this reason, catheters (especially dialysis cathers) should be preferentially placed in the internal jugular veins.

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27

Remember that Inadvertent Intra-Arterial Injection is Not Rare

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The estimated incidence of iatrogenic complication from accidental intra-arterial (IA) injection of drugs is between 1 in 3,440 and 1 in 56,000 anesthetics administered. The potential complications range from none to severe tissue ischemia and necrosis necessitating amputation. All anesthesia providers should be aware of risk factors, signs and symptoms, available therapeutic modalities, and preventive measures for IA drug injection during anesthesia. Commonly used anesthetic drugs and inadvertent IA injection effects are shown in Table 27.1.

RISK FACTORS FOR IA INJECTION

- Patients who are unable to report pain on injection. Patients during general anesthesia, comatose patients, or patients with altered mental status, and very young pediatric patients.
- Pre-existing vascular anomalies of the forearm. The most common arterial anomaly of the upper limb is a high-rising radial artery resulting in a superficial branch (prevalence, 1% to 14%). This anomaly results in the radial artery ending in a thin superficial palmar branch that can be mistakenly cannulated *(*Fig. 27.1*, Left).* Another common anomaly (1% prevalence) is the antebrachialis superficialis dorsalis artery *(*Fig. 27.1*, Right).* The radial artery bifurcates in the forearm, resulting in an anomalous superficial branch between the index finger and thumb. Often, this branch will cross underneath a terminal branch of the cephalic vein, just superficial to the radial styloid process—a common site for insertion of intravenous (IV) catheters (called the “intern vein”).
- High-risk anatomic locations, where arteries and veins lie in close proximity.
  - Antecubital fossa: brachial artery may be cannulated rather than the median basilic vein.
  - Groin: femoral artery cannulation rather than femoral vein.
- Multiple infusions through several IV lines with numerous ports (patients in the intensive care unit).
- Miscellaneous factors. Morbid obesity, darkly pigmented skin, multiple attempts at line placement, placement of lines under nonideal situations (central line during operations and/or without ultrasonographic guidance), those with a pre-existing arterial or venous catheter who present for urgent resuscitation (trauma).

TABLE 27.1 EFFECTS OF IA INJECTION OF COMMONLY USED ANESTHETIC AGENTS

DRUG	EFFECT OF IA INJECTION
Midazolam	Initial discoloration but no long-term effect
Promethazine, chlorpromazine	Gangrene
Thiopental	Chemical endarteritis, immediate vasoconstriction, thrombosis, tissue necrosis, endothelial cell destruction
Etomidate	No reports of necrosis
Ketamine	Proximal skin necrosis
Propofol	Pain, cutaneous hyperemia
Lidocaine	Used therapeutically, no adverse effects
Penicillin	Gangrene
Cefazolin, ceftazidime	Arteriospasm and distal necrosis

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Meperidine	Gangrene
Sodium bicarbonate	Edema, erythema, pain; tissue necrosis
Metoclopramide	Discoloration but no long-term effect
Phenytoin	Cyanosis, digital artery occlusion, gangrene
Atracurium	Marked ischemia but full recovery
Dextrose solution	Gangrene
Calcium chloride	No adverse effects reported Used therapeutically to localize insulinoma
Ephedrine, phenylephrine, epinephrine, succinylcholine, vecuronium, rocuronium, fentanyl, oxymorphone, hydromorphone, ketorolac, ondansetron, atropine, glycopyrrolate, neostigmine	No reports available

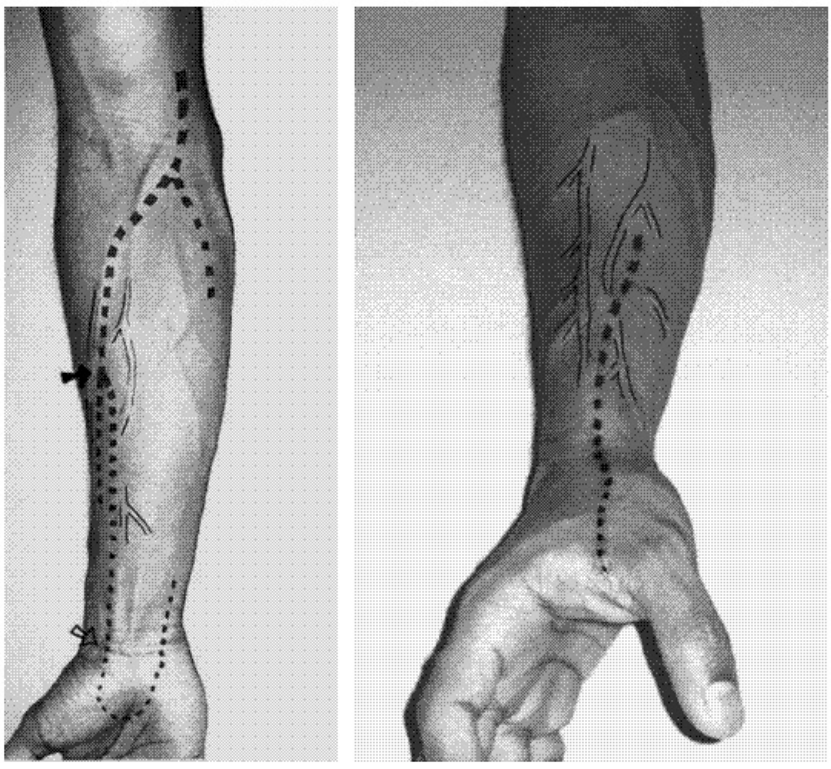
IA, intra-arterial.

SIGNS AND SYMPTOMS OF ARTERIAL CANNULATION

- Bright red backflow and/or pulsatile blood into an IV catheter
- Backflow of blood into the IV tubing, even with the fluid bag at a higher level than the catheter insertion site
- Increase in blood pressure on an indwelling arterial catheter with ipsilateral “IV” bolus
- Distal (e.g., nail bed) signs of ischemia (pallor) and skin mottling with “IV” fluid bolus
- Greater-than-expected pain with injection

PATHOPHYSIOLOGY

Although the underlying pathophysiologic mechanisms to explain the sequelae of an IA drug injection remain unclear, a few concrete conclusions can be drawn.



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FIGURE 27.1. Vascular anomalies of the forearm. **Left:** Thick dotted line shows abnormal artery. *Open arrow,* normal branching point of radial artery; *closed arrow,* anomalous high-rising radial artery. **Right:** Thick dotted line shows anomalous antebrachialis superficialis dorsalis radial artery. Thin black lines show typical venous pattern. The abnormal artery course is in close proximity to commonly cannulated veins. (From Sen S, Chini EN, Brown MJ. Complications after unintentional intra-arterial injection of drugs: risks, outcomes, and management strategies. *Mayo Clin Proc.* 2005;80:783-795. Used with permission of Mayo Foundation for Medical Education and Research.)

- Not all medications cause ischemia by the same mechanism. Some may cause crystallization and mechanical blockage of blood flow, whereas others may be directly toxic to the endothelium.
- Regardless of the mechanisms involved, thrombosis seems to be the common end point.

TREATMENT OPTIONS

Most treatment modalities are based on maintaining distal perfusion to the involved extremity. Management should be based on the following principles:

- Immediately stopping administration of the offending agent
- Cessation or reversal of arterial spasm
- Maintaining or re-establishing blood flow to distal extremities
- Treating any sequelae of vascular injury or ischemia (edema, compartment syndrome, infection, necrosis, gangrene)
- Symptomatic relief

Step 1: Maintain the IA Catheter in Situ.

When an inadvertent IAinjection occurs, the first instinct may be to remove the catheter, but maintaining the arterial catheter has several advantages. First, the catheter should be cleared of any remaining drug, either by opening the catheter to atmosphere to allow backflow of blood or by simply aspirating with a syringe. This should be followed by slow infusion of heparinized saline to help maintain catheter patency. If there is any doubt regarding the catheter site (i.e., arterial vs. venous), confirm placement by drawing a sample for arterial blood gas analysis or by transducing the line. Maintaining the IAcatheter will also allow immediate delivery of specific medications to the site of injury, including contrast dye for angiography. If the larger-bore IAcatheter was placed inadvertently, removing the catheter may also require surgical intervention to suture the large hole in the artery.

Step 2: Identify Potential for Injury.

Certain clinical indicators can be roughly correlated with progressive tissue injury. Treiman et al. developed a "tissue ischemia score" based on signs and symptoms at presentation of 48 patients with IAinjections. Patients were assigned a 0 for the absence or a 1 for the presence of four symptoms: cyanosis, cool extremity, delayed capillary refill, and sensory deficit. Of patients who had a tissue ischemia score of 2 or lower, 92% had a normal outcome. Of patients who had a score higher than 2, only 41% had a normal outcome in the involved extremity; the others had tissue necrosis or permanent neurologic dysfunction.

Step 3: Initiate Anticoagulation.

Administration of heparin seems to be broadly accepted as the initial treatment of IAinjections. There is no consensus on the initial bolus amounts to use, appropriate target ranges for the activated partial thromboplastin time (aPTT), or duration of therapy, but an initial loading dose of 60 U/kg with subsequent aPTT goals of 1.5 to 2.3 times higher than normal seem prudent. Of course, this must be balanced against the risk of hemorrhage in a postoperative patient. The duration of therapy can then be guided by resolution of symptoms, angiographic evidence of clot resolution, or the need for surgical intervention.

Step 4: Symptomatic Relief.

Increased sympathetic vascular tone, edema distal to the site of injury, muscle injury or forced flexion contractures, temperature hypersensitivity, and paresthesias may all contribute to ongoing pain. Multiple case reports suggest that oral analgesics, elevation, massage, and passive motion devices can be critical to treatment and recovery.

Step 5: Specific Interventions.

After implementing the first four steps, it is important to determine whether any other specific intervention is needed. The following interventions have been used with varying degrees of success:

- Local anesthetic injection: IAlidocaine (without epinephrine) to prevent or treat reflex vasospasm
- Extremity sympatholysis: stellate ganglion/axillary plexus blocks
- Arterial vasodilators: calcium-channel blockers (nicardipine)
- Thromboxane synthase inhibitors: topical aloe vera, methimazole, aspirin, methylprednisolone
- Iloprost: a prostacyclin analog used for various ischemic conditions
- IApapaverine: induces vascular smooth muscle relaxation by increasing intracellular cyclic adenosine monophosphate levels
- IAthrombolytics
- Hyperbaric oxygen therapy
- Corticosteroids: mainstay of treatment for any condition involving inflammation.

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PREVENTION OF IA INJECTION

- Be aware of the risk and maintain a high index of suspicion when placing any intravascular catheter, particularly in antecubital and cephalic veins.
- Look for signs of arterial cannulation as outlined above.
- Use caution and maintain an index of suspicion with all pre-existing lines.
- Palpate target veins before IV placement.
- Do not tie a tourniquet tight enough to occlude arterial flow.
- Use clear labeling of arterial line at all injection ports.
- Color-code lines and injection ports.
- Remove unnecessary injection ports or stopcocks. If an injection port is necessary, keep it as close to the patient as possible.
- Trace each extension line back to the site of the cannula before injecting any medications.

Despite recognition of accidental IA injections for many decades, these cases still commonly occur. As always, the best solutions are prevention and continuous vigilance. Clinicians should be aware of risk factors for IA injection, the associated signs and symptoms, the underlying pathophysiology, and available treatment options. Such awareness will help decrease the incidence, delays in diagnosis, and resulting complications of this medical error.

TAKE HOME POINTS

- Remember that inadvertent IA injection is not rare.
- IA happens because you mistake an artery for a vein line on insertion of the cannula or because you mistakenly inject into the wrong stopcock.
- Develop techniques for making sure you do not get venous and arterial lines and/or stopcocks mixed up—red and blue tape at injection ports, etc. Minimize the number of ports.
- Do not take out the catheter if there is an inadvertent injection.
- Inform the necessary people (surgeons, etc.) and start treatment as soon as possible.

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Avoid Errors in Invasive Blood Pressure Measurement

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Monitoring of a patient's blood pressure (BP) is crucial to the safe conduct of any surgical procedure. This chapter focuses on invasive arterial-line BP monitoring and some of the common errors associated with inaccurate readings.

For an arterial monitoring system to provide an accurate reading of the measured waveform, it must have an appropriate natural frequency and damping *(*Fig. 28.1A*)*. Briefly, each measuring system has a natural frequency about which it oscillates. This frequency is proportional to the stiffness of the tubing and the transducer diaphragm and to the cross-sectional area of the catheter. It is inversely proportional to the catheter length and fluid density. If the frequency of the pressure wave being measured approaches the natural frequency of the system, the system distorts the measurements by excessive amplification (ringing or resonance) of the incoming waveform. This may overestimate the systolic BP by as much as 25% and underestimate the diastolic BP by 10% (the mean pressure is not affected). Damping counteracts this phenomenon.

Several problems can occur in this interaction between the natural frequency and damping parameters. Overdamping decreases the frequency response too much, underestimating the systolic BP and overestimating the diastolic BP *(*Fig. 28.1B*)*. Blood clots, air bubbles in the tubing, and kinked catheters are common causes of an overdamped system. Underdamping, or hyperresonance, occurs when long connecting lines (>1.4 m) or smalldiameter tubing (<1.5 mm internal diameter) are used or when the catheter is too large for the vessel (e.g., 18-gauge catheter in a small radial artery). This overestimates the systolic BP and underestimates the diastolic BP. Underdamping may also cause the appearance of additional, small, nonphysiologic pressure waves on the tracing *(*Fig. 28.1C*)*. Slow degradation in the dynamic response also may occur over time (decrease in the natural frequency with increase in the damping coefficients), causing underdamping. Fortunately, this problem can be easily rectified with periodic manual flushing of the system.

Problems within the system that lead to inaccurate measurements are usually due to problems with the mechanical connection components (i.e., the catheter, fluid-filled tubing, or the stopcock). To prevent these mechanical problems, several steps should be followed.

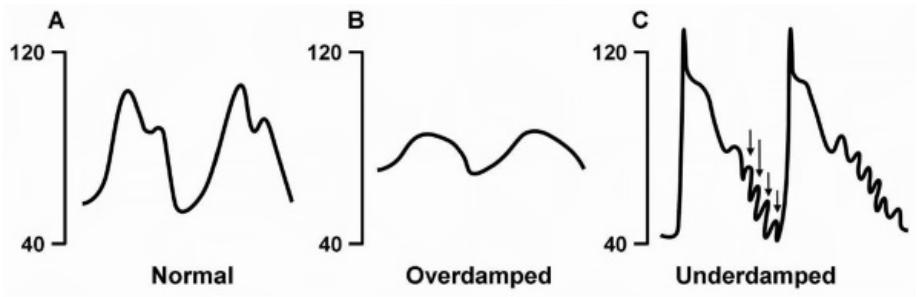


FIGURE 28.1. Arterial blood pressure (BP) waveform tracings. **A:** Normal. **B:** Overdamped tracing, causing underestimated systolic BP and overestimated diastolic BP. **C:** Underdamped tracing, causing overestimated systolic BP and underestimated diastolic BP. Extra nonphysiologic waveforms on the underdamped tracing reflect hyperresonance of the measuring system *(arrows)*.

- Choose the proper size arterial cannula (20 gauge for radial or brachial, 18 gauge for femoral).
- Optimize the frequency response of the measuring system by using tubing that is short (maximum length, 120 cm), wide (1.5 to 3.0 mm internal diameter), and rigid.
- Use only one stopcock per line. Keep the tubing, stopcocks, and domes free of bubbles and clots. Avoid kinks in the tubing.
- If arterial BP is overdamped:
  - Try flushing the line.
  - Keep the entire mechanical coupling system flushed with heparinized saline to prevent development of clots (1 U of heparin per milliliter of normal saline) at 1 to 3 mL/hour.
  - Be sure that the flushing system of the arterial line is adequately pressurized.
- If arterial BP is underdamped:
  - Use a resonance overshoot eliminator (commercial product).
  - Temporarily put a small air bubble in the system (do not forget to withdraw it immediately after measurement is accomplished). This can help to

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estimate the “real” BP, but this is a controversial technique that not all authors recommend.

- Use a transducer with the highest possible frequency response.
- Zero the arterial line to atmospheric pressure to provide a reference point. By convention, the transducer is placed level with the right atrium or midaxillary line. For a sitting patient, the transducer is placed at the level of the brain (ear). Check frequently for improper zeroing and drift. Every time the measured BP is “out of expected range,” quickly check that the transducer is zeroed (open the transducer to air and see if the baseline goes to zero; waiting for the digital reading to confirm the proper “zeroing” numerically is not necessary. Readjust the transducer after every change in patient positioning.

Anesthesiologists often have no control over the inherent natural frequency and damping coefficients present in monitoring systems. Furthermore, clinical circumstances sometimes require longer extension tubing or extra stopcocks for blood sampling and flushing. In these circumstances, the fast-flush test, described in Fig. 28.2, is a convenient bedside method for determining system performance parameters. The damping coefficient may be determined as described in the legend of Fig. 28.2.

In the clinical situation, if there are two different arterial BP readings (e.g., noninvasive vs. invasive, left arm vs. right arm) and/or two different waveforms (e.g., femoral vs. radial), it may not be clear which pressure to “trust.” The phenomenon of distal pulse amplification can affect the morphology and detail of the arterial waveform and can often give important diagnostic information. In short, pressure waveforms recorded simultaneously from different arterial sites will have different morphologies. This difference is due to the physical characteristics of the vascular tree—specifically, impedance and harmonic resonance. As the arterial pressure wave travels toward the periphery, the arterial upstroke steepens, the systolic peak increases, the dicrotic notch appears later, the diastolic wave becomes more prominent, and the end-diastolic pressure decreases. This results in higher systolic pressure, lower diastolic pressure, and wider pulse pressure. In addition, a substantial difference between peripherally measured BP (radial artery) and more centrally measured BP may temporarily exist in specific conditions such as after cardiopulmonary bypass surgery. However, despite these differences, the mean arterial pressure between central and peripheral readings should be roughly the same. Ultimately, the pressure that can more adversely affect outcome (e.g., low systolic BP in a patient with critical aortic stenosis) is the one that should be “believed.”

During the course of a surgery, if significant hypotension occurs, the following steps should be performed:

- Look for other obvious signs of hypotension:
  - Sudden decrease in end-tidal CO_2 (indicates low cardiac output and poor perfusion of pulmonary vasculature)
  - Poorly palpable radial pulse (can indicate systolic BP <55 to 65 mm Hg)
- Check the result against a cuff pressure. If a substantial discrepancy exists:
  - Check the transducer position. Having the patient higher than the transducer produces falsely high readings and vice versa. The magnitude of the error is exactly equal to the difference in hydrostatic pressure between the patient and the transducer (1 mm Hg = 1.3 cm H_2O).

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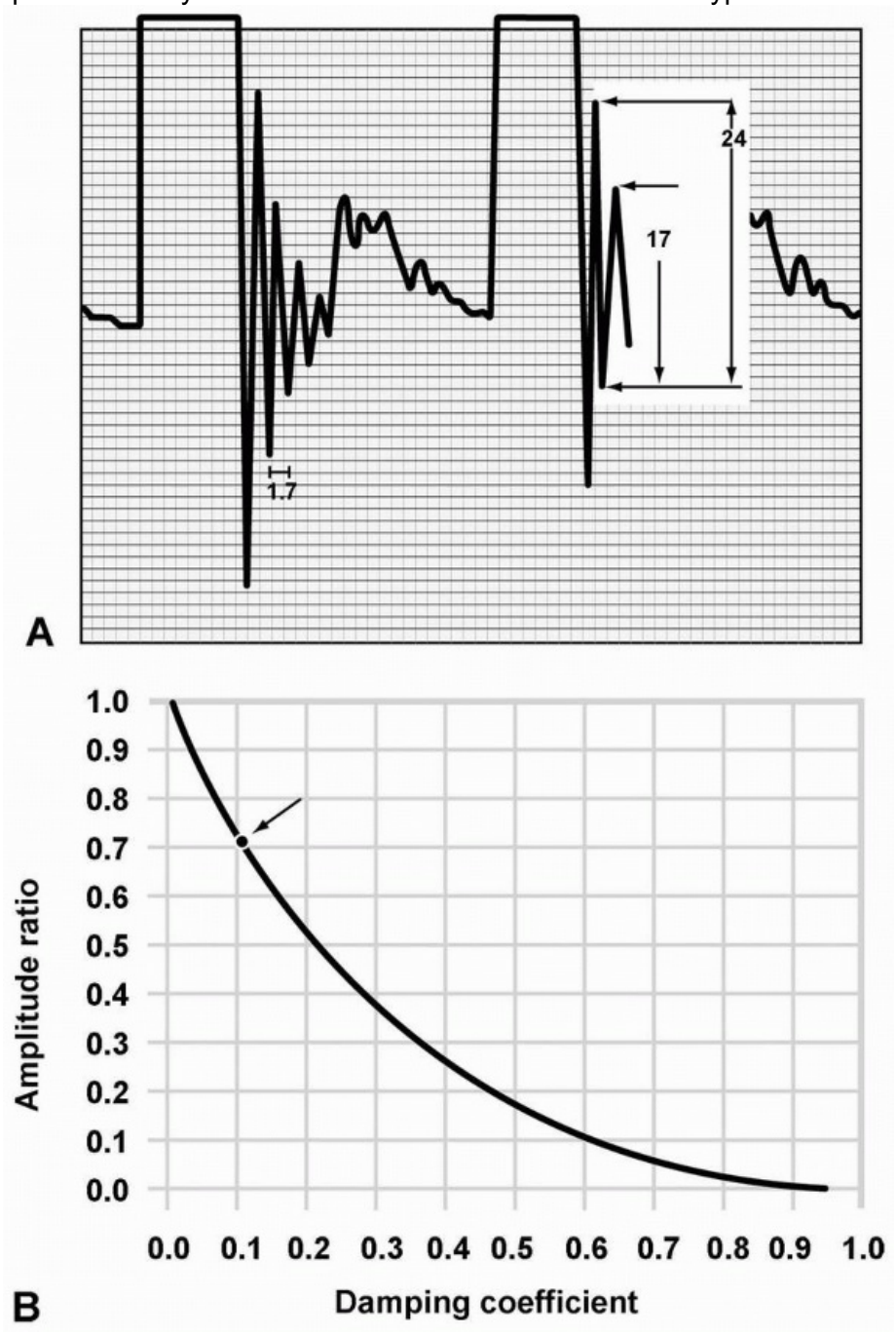


FIGURE 28.2. Clinical measurement of natural frequency and damping coefficient with the fast-flush test. **A:** Two square-wave fast-flush artifacts interrupt an arterial pressure waveform recorded on standard 1-mm grid paper at a speed of 25 mm/second. Natural frequency is determined by measuring the period of one cycle of adjacent oscillation peaks (1.7 mm). The damping coefficient is determined by measuring the heights of adjacent oscillation peaks (17 and 24 mm). From these measurements, a natural frequency of 14.7 Hz (1 cycle/1.7 mm × 25 mm/s = 14.7 cycle/second) and an amplitude ratio of 0.71 may be calculated. **B:** The amplitude ratio corresponds to a damping coefficient of 0.11, suggesting overdamping (an optimally damped system has a damping coefficient of 0.6 to 0.7). (From Mark JB. *Atlas of Cardiovascular Monitoring*. New York: Churchill Livingstone; 1998:112-113. Used with permission.)

- Zero the reference value on the monitor. Open the stopcock to air and inspect the monitor screen to ensure that the pressure trace overlies the zero pressure line on the screen and that the digital pressure value equals zero. (Note that checking the zero value is not the same as "zeroing the arterial line," which is done at the start of the procedure.) If the pressure is not equal to zero, baseline drift of the electrical circuit's transducer has occurred.
- Double-check all the components of the system (electrical plug-ins, loose attachments, etc.).
- Interrupt the surgical course until the problem is identified. In extreme cases, ask the surgeon to palpate some of the abdominal or thoracic vessels if any are accessible.
- Do not assume an overdamped waveform, because the "overdamped waveform" may be true hypotension.
- Be aware that severe "hypotension" on one extremity may reflect severe peripheral vascular disease (subclavian stenosis), and that in these patients BP measurements should be made on both extremities (noninvasively, or by palpating the radial pulse) before deciding which side will be used for placing the invasive arterial line.

TAKE HOME POINTS

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- Measuring systems oscillate around a natural frequency, which can amplify a measured waveform, if the frequencies are close. Damping can counteract this, but be aware that a variety of factors can cause either underdamping or overdamping.
- Problems within the system that lead to inaccurate measurements are usually due to problems with the mechanical connection components.
- Pressure waveforms recorded simultaneously from different arterial sites will have different morphologies. This difference is due to the physical characteristics of the vascular tree—specifically, impedance and harmonic resonance.
- The mean arterial pressure between central and peripheral readings should be roughly the same.
- If the blood pressure measurement decreases suddenly, do not assume an overdamped waveform, because the "overdamped waveform" may be true hypotension.

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Remember that Loss of a Patent Hemodialysis Fistula in the Perioperative Period is a Serious Event for the Patient and Requires Immediate Communication with the Surgeons

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Chronic renal insufficiency/failure (CRI/F) is a leading cause of morbidity and mortality in a growing percentage of the U.S. population. Most causes of end-stage renal disease stem from two major comorbid conditions—diabetes mellitus and hypertension. The ultimate end result of CRF is reliance on hemodialysis for maintenance of renal function. The use of hemodialysis fistulas has both improved the quality and prolonged the lives of CRF patients, although there are associated complications *(*Table 29.1*).*

HEMODIALYSIS FISTULAS

Hemodialysis fistulas or ports for dialysis are surgically created communications between the native artery and vein in an extremity. Direct communications between artery and vein are called native arteriovenous fistulas (AVFs). Polytetrafluoroethylene (PTFE) and other materials (Dacron, polyurethane) are used or have been used as a communication medium between the artery and the vein and are termed prosthetic hemodialysis access arteriovenous grafts (AVGs). The patency rates for AVFs are about four times greater than for AVGs. The access that is created is routinely used for hemodialysis two to five times per week. Preservation of a patent well-functioning dialysis fistula is one of the most challenging issues for the dialysis patient. As many as 25% of hospital admissions in the dialysis population have been attributed to vascular access problems, including fistula malfunction and thrombosis.

In fact, only 15% of dialysis fistulas remain patent and can function without problems during the entire period of a patient's dependence on hemodialysis. The majority of patients who have native fistulas placed will be problem free with their fistulas for a mean of 3 years after creation, whereas prosthetic PTFE grafts usually last only about a year before indications of failure or thrombosis are noted.

Long-term secondary patency rates are reportedly 7 years in the forearm, 3 to 5 years in the upper arm for native fistulas, and about 1 year for prosthetic grafts after multiple interventions to treat the underlying stenosis and thrombosis.

TABLE 29.1 COMPLICATIONS OF HEMODIALYSIS FISTULAS

- Hemodynamic complications—congestive heart failure
- Arterial steal syndrome
- Carpal tunnel syndrome
- Infection
- Noninfectious fluid collections—seroma, lymphocele, hematoma
- Aneurysm, pseudo-aneurysm

ARTERIOVENOUS FISTULA FAILURE

The underlying cause of AVF failure in the nonacute setting or outpatient setting is invariably thrombosis due to venous anastomosis in prosthetic grafts or anastomosis of the outflow vein in native fistulas *(*Table 29.2*).* The pathophysiology behind this failure is the eventual intimal hyperplasia at the anastomosis site. Future therapy is directed at halting the intimal growth of anastomotic vessels, using technology similar to that employed with drug-eluting cardiac stents.

The main cause of AVF thrombosis in the acute care setting, particularly the perioperative period, is almost always low flow, usually caused by hypotension and/or poor cardiac output. A sudden decrease in blood pressure can lead to platelet aggregation, sledging, and eventual thrombosis. An important secondary cause for the loss of a patent fistula is excessive external pressure on the AVF, usually as a result of monitoring or positioning. Remember also that surgical patients are in a hypercoagulable state—this may also predispose to thrombotic events.

It is imperative that anesthesia providers recognize the clinical signs of impending AVF thrombosis. It is wise to evaluate the fistula as thoroughly as possible *before* starting the anesthetic. Remember that dialysis patients are often the best source for information on their personal fistula histories.

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Clinical signs of impending AVF thrombosis include the following:

- Loss of thrill
- Increase pulsatility (water-hammer pulse)
- Direct palpation of stenosis

TABLE 29.2 CAUSES OF AVF THROMBOSIS

- Poor cardiac outflow (hypotension)—sudden decrease in blood pressure can lead to platelet aggregation, sledging, and eventual thrombosis.
- Excessive pressure on AVF (blood pressure measurement)
- Hypercoagulable state
- Progressive inflow stenosis
- Venous outflow stenosis

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- Insufficient inflow, such as stenosis in the supplying native artery or proximally in the subclavian or brachiocephalic artery
- Identification of high venous pressures or low flow according to the protocol provided by the specific type of hemodialysis machine—try to ascertain whether there were problems with the duration of treatment or volume of fluid removed in the last dialysis run before surgery.
- Ipsilateral arm edema and/or collateral venous pathways suggestive of a central venous stenosis

Similarly, the care of AVFs during anesthesia requires the very close attention of the practitioner:

- Maintain normotension—hypotension may lead to clot formation and thrombosis.
- Maintain heart rate—symptomatic bradycardia may decrease coronary output and lead to thrombosis.
- Do *not* place the noninvasive blood pressure cuff on the AVF arm—compression can limit inflow/outflow and promote thrombosis.
- Do *not* perform venipuncture on the AVF arm—local clotting factors may favor thrombosis. If you get stuck for sites for line placements, consult with the surgeons and/or vascular surgeons before the start of the operation. Emergency situations may involve broaching the integrity of the vasculature of the extremity that has the fistula—if so, expect that the fistula may be lost and make proactive plans.
- Actively feel and *document* presence of a thrill at regular intervals during the procedure—presence confirms a functioning AVF.
- Consider regional anesthesia when appropriate to minimize fluctuations in blood pressuer and heart rate.
- Document the thrill when signing the patient out in the PACU or ICU.
- Communicate immediately with the surgeon if there is a change in AVF status (this usually means the loss of the thrill)

TREATMENT OF AVF THROMBOSIS

Unfortunately, thrombosis of dialysis access is not an uncommon event during the perioperative period. Historically, treatment of AVF thrombosis involved surgical thrombectomy with ongoing hospital admission. Typically, surgical treatment resulted in sacrificing a portion of the outflow vein.

Multiple treatment options are now available and depend on the patient's co-morbid conditions, the availability of the appropriate surgical personnel, the availability of radiologic support, and previous fistula treatment *(*Table 29.3*)*. Often, a radiologic intervention is considered most favorable if possible. Patients may thus sometimes go directly from the PACU to interventional radiology. Alternatively, other dialysis access, such as a double-lumen Shiley, may be placed in the immediate postoperative period. Occasionally,
treatment is deferred until the patient is clinically and hemodynamically stable.

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TABLE 29.3 TREATMENTS

- Thrombolysis
  - With angioplasty
  - Without angioplasty
- Percutaneous thrombectomy

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- With angioplasty
- Without angioplasty

- Surgical thrombectomy
  - With revision
  - Without revision

TAKE HOME POINTS

- Native AVFs are less prone to thrombosis than grafts.
- Loss of the graft is a serious perioperative event for the patient.
- Low flow states caused by hypotension, low cardiac output, and external compression are the most significant causes of fistula thrombosis. If there is a new or existing hemodialysis fistula, think carefully before lowering the blood pressure aggressively with propofol, labetalol, or hydralazine.
- Maintain high vigilance for hemodynamics and positioning.
- Don't be afraid to consult the surgeons about the preferred sites for both venous and arterial line placement.
- Check for the thrill at regular intervals and let the surgeons know immediately if the thrill is lost.
- One final point—what you feel when lightly palpating the fistula is *not* a bruit—*by definition,* a bruit is an abnormal sound heard when placing a stethoscope over an artery.

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Hypertonic Saline: the "Solution" to the Solution Problem?

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Analogous to the variety of beverages in your supermarket's soda aisle, there exist numerous choices for fluids for patients. What is best for your patient's needs? What fluid is appropriate for specific situations? How much fluid should be administered? Although these questions may seem trivial, the kneejerk reflex for most residents is to use a fluid that they feel most comfortable administering. Let us wade out of the "normal saline" pool and explore the clinical uses of hypertonic saline.

Fluids became the focus of prehospital resuscitation research more than 75 years ago. The same clinical problems encountered with fluid resuscitation efforts then remain the focus of today's research. The search continues for an "ideal" fluid, i.e., one that is inexpensive, generates sustained hemodynamic effects with minimal peripheral or pulmonary edema, and is effective even in small volumes.

Traditionally, isotonic crystalloids such as lactated Ringer's solution and normal saline have been considered first-line treatment for trauma patients. Although these fluids are inexpensive, readily available, nonallergenic, noninfectious, and efficacious in restoring total body fluid, they lack oxygen-carrying capacity and coagulation capability, and they have limited intravascular half-life. A proposed solution to the "solution problem" is to augment the osmotic properties of traditional resuscitation fluids. In theory, this would extend the intravascular half-life and reduce the amount of third spacing. The "ideal" fluid search then focused on osmotic properties, and hypertonic saline (with and without colloid) became a prime subject of research.

Hypertonic saline already had a role in the trauma setting, as it was used for patients with increased intracranial pressure secondary to traumatic head injury. Of note, mannitol (an osmotic diuretic and cerebral arteriolar vasoconstrictor) was traditionally used as first-line therapy in this patient population, but it was associated with rebound intracranial hypertension. It was believed that this effect was due to intravascular dehydration and an inability to maintain cerebral perfusion pressure when diuresis was not matched by appropriate fluid administration. Hypertonic saline became the agent of choice, as it served a dual purpose as a diuretic and a resuscitative agent.

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TABLE 30.1 HYPERTONIC SALINE: BEYOND PLASMA VOLUME EXPANSION

Vasoregulatory effects	Counteracts vasospasm occurring after traumatic brain injury through its vasodilatory effects. Improves microvascular perfusion by attenuating leukocyte-endothelial interactions.
Immunologic effects	Changes in extracellular sodium that occur after injury may be attenuated by hypertonic saline, which is thought to depress leukocyte adherence and neutrophil margination.
Neurochemical effects	Changes in excitatory neurotransmitters that occur after injury may be attenuated by hypertonic saline, which stimulates adrenocorticotropin and cortisol release.

Further research in the use of hypertonic saline in the trauma setting revealed that it had vasoregulator, immunologic, and neurochemical effects (Table 30.1). Hypertonic saline increases blood pressure and cardiac output secondary to plasma volume expansion, and it is also thought to stimulate adrenocorticotropin and cortisol release. Hypertonic saline is also believed to counteract vasospasm occurring after traumatic brain injury through its vasodilatory effects. On a molecular level, hypertonic saline has been shown to improve microvascular perfusion by attenuating leukocyte-endothelial interactions. It has also been suggested that hypertonic saline can provide protection from bacterial illness by attenuating changes in extracellular sodium and excitatory neurotransmitters that occur after injury. These changes are thought to depress leukocyte adherence and neutrophil margination.

Although the most common concentration of hypertonic saline used clinically is 3%, the concentrations studied in clinical trials ranges from 1.7% to 29.2%. In spite of concern for hypertonicity, acute increases in serum sodium levels to 155 to 160 mEq have not been shown to be harmful to most patients.

Another clinical problem with hypertonic saline is the transient nature of the resulting hemodynamic improvement (30 to 60 minutes) when it is used as a resuscitative fluid, much like traditional resuscitative fluids. Proposed strategies to lengthen the hemodynamic effects of hypertonic saline include longer infusion times, subsequent or concomitant infusion of blood or conventional fluids, and adding 6% dextran to the hypertonic saline. Despite the claims surrounding the addition of colloid, there remains no clear answer as to the best strategy to lengthen hemodynamic effects of hypertonic saline.

The reality is that the clinical efficacy of hypertonic saline resuscitation versus conventional fluids remains unclear because many prehospital studies compared only single boluses of experimental and control fluids. Furthermore, misleading conclusions exist about the clinical responses to different types of fluid resuscitation (including hypertonic saline with and without colloid additives). This is likely the result of studies comparing two or more regimens that alter more than one variable.

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In addition to the use of hypertonic saline in the prehospital and trauma setting, it has several well-established roles. These include the care of patients with severe, symptomatic hyponatremia (<115 to 120 mEq) and in the fluid management of critical burn injuries. Nontraditional uses of hypertonic saline

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include stimulation of expectoration, which has been implemented in the care of patients with cystic fibrosis. Hypertonic saline has also been used in the treatment of tricyclic antidepressant (TCA) toxicity. Rapid, cataclysmic clinical deterioration may occur shortly after TCA overdose. Traditionally, sodium bicarbonate is used to reverse TCA cardiotoxicity (dysrhythmias), but hypertonic saline has also been shown to do this. Furthermore, in the setting of hemodynamic instability generated by TCA toxicity, hypertonic saline may be especially useful for a hypotensive patient.

Support for hypertonic saline is growing in the prehospital and trauma setting based on recent research into its beneficial effects at the cellular and molecular levels. Hypertonic saline may come to be viewed as a "drug" instead of a simple fluid, which will bring about more research into the adequate "dose" and "timing of administration." This may ultimately clarify its clinical role and clear up the misleading conclusions that currently exist about its clinical efficacy in the trauma setting.

TAKE HOME POINTS

- Hypertonic saline has documented vasoregulatory, immunologic, and neurochemical effects—it may come to be viewed more as a drug than a simple fluid.
- The use of hypertonic saline has been driven by the resuscitation issues encountered with burn, trauma, and critically ill patients. Seek out senior providers in these areas to gain clinical expertise.
- From her personal use of 3% hypertonic saline, the editor recommends establishing a baseline serum sodium before initiating therapy. An infusion (typically 1 to 2 mL/hour) is then started via central access, with an initial serum sodium value checked at 30 minutes and then at regular intervals (usually 60 minutes). Consider discontinuation if the serum sodium increases 10 to 15 mEq above baseline or at 155 mEq, whichever is reached first.

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31

Remember that the Synthetic Colloid Solutions Have Distinct Properties and Risk/Benefit Ratios

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Before an anesthesia provider can consider the colloid solutions in general and the hetastarch products in particular, he or she must first have an understanding of the basics of perioperative fluid therapy. Perioperative fluid therapy includes the replacement of pre-existing fluid deficits, maintenance requirements, and surgical losses.

Normal maintenance requirements (Table 31.1) are what the body needs to keep up with urine production, losses from the respiratory tract or skin, and gastrointestinal (GI) secretions. Because these losses are hypotonic meaning that the fluids lost have a lower osmolality than the cells in the body.

Fluid deficits may come from a variety of sources. First, the great majority of patients who come to the operating room have fasted for at least 6 hours. For the average 70-kg man this calculates to a deficit of (110 mL/hour × 6) or 660 mL. For many GI procedures, patients may also get a preoperative bowel cleansing preparation. This may increase fluid losses by 1 to 2 L. Increased insensible losses also occur as a result of increased sweating, respiratory losses from fevers, as well as in any patient who has been breathing nonhumidified gases. Finally, patients who have diarrhea, vomiting, diuresis, or bleeding will have fluid depletion as well as electrolyte disturbances.

For surgical losses, the anesthesia provider has to be very vigilant. Underestimating surgical blood loss can have devastating consequences. The first obvious place to look is in the suction canister or at the sponges. Remember that a 4 × 4 sponge may hold 10 mL of blood and a full-sized, soaked laparotomy pad may represent 100 mL of blood loss. In addition, one must take into account the irrigation solution used. Good communication with the scrub nurse for estimating this fluid solution use is essential. With the increased use of cell salvage techniques, a second suction canister may be present. However quick visual estimation of blood losses may account for only one third of the “real blood loss.” There may be a significant amount not accounted for in the tubing, on the drapes/floor, or on the surgeon/scrub nurse. Once an estimate of surgical blood loss has been made, a good rule for replacement volume for surgical blood loss (when not using blood products) is 1:3 for crystalloid solutions and 1:1 for colloid solutions. This means that if there is a 300-mL blood loss, one could correct this loss by giving 900 mL of crystalloid or 300 mL of colloid. P.137

TABLE 31.1 FLUID MAINTENANCE ESTIMATES

WEIGHT (KG)	RATE (ML/KG/H)
0-10	4
Next 10-20	2
Each kg >20	1

Fluid Estimates Calculation

Example 1. 100-kg person.

(10 × 4) + (10 × 2) + (80 × 1) = 140 mL/h

Example 2. 23-kg child

(10 × 4) + (10 × 2) + 3 = 63 mL/h

What are colloid solutions? Basically, they are solutions of highmolecular-weight substances administered to maintain intravascular volume. They last intravascularly from 3 to 6 hours. Some indications for use are for fluid resuscitation before blood products are available or in patients with severe hypoalbuminemia.

There are many different types of colloid solutions. One of the most familiar to anesthesia providers is albumin, which is available in 5% and 25% solutions. The albumin protein is relatively stable and is heated to 60°C for 10 hours. It is heated to potentially remove HIV, hepatitis, etc. Albumin is one of the most expensive solutions because it is derived from blood donations. Recently, Pentastarch and Hespan (Hetastarch) have been increasingly popular because of the lower cost. Both are composed of chains of glucose molecules to which hydroxylated ethyl ether groups have been added to resist

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degradation. The average molecular weight of Hespan is 450,000. Hetastarch comes in a 6% solution of either NaC1 (Hespan) or LR (Hextend) (Table 31.2). Pentastarch, which has a slightly smaller molecular size, was developed secondary to the fact that the large Hespan molecules are sequestered in the reticuloendothelial system (RES), the kidney, and the liver. Although this has not been shown clinically, it may impair the RES. For a comparison of Hetastarch to Pentastarch (both in NaC1 solution) see Table 31.3.

TABLE 31.2 HESPAN VERSUS HEXTEND

	HES/BS (HEXTEND)	HES/NS (HESPAN)
HES (g/dL)	60	60
Na (mEq/L)	143	154
Cl (mEq/L)	124	154
Lactate (mEq/L)	28	—
Ca (mEq/L)	5	—
K (mEq/L)	4	—
Mg (mEq/L)	0.9	—
Dextrose (mg/L)	99	—

TABLE 31.3 HETASTARCH VERSUS PENTASTARCH

	HETASTARCH	PENTASTARCH
pH	5.5	5
Avg. MW	450,000	264,000
Intravascular half-life	25.5 h	2.5 h
Elimination	Renal	Renal
Coagulation effects	Increases PT, PTT, bleeding time	Increases PT, PTT, bleeding time
Liver effects	Temporarily increases amylase, increases indirect bilirubin	Temporarily increases amylase
Maximum dose	15-20 mL/kg, max. 1 L	15-20 mL/kg, max. 1 L

Avg. MW, average molecular weight; PT, prothrombin time; PTT, partial thromboplastin time.

The polyhydroxylated starches have relative contraindications in patients with coagulopathies, fluid overload, or renal impairments, and both produce rare hypersensitivity reactions. Hespan has been shown to have a decreased inflammatory response compared to albumin preparations. However, there was evidence that showed possible impaired coagulation with Hespan as compared to albumin, particularly in cardiac surgery. The question was raised: If one put the Hetastarch in a balanced salt (BS) solution (i.e., Hextend), can the coagulation issues be avoided? One must also be aware that Hespan/normal saline (HES/NS) solution can cause a hyperchloremic metabolic acidosis because it is not a balanced salt solution (similar to giving too much normal saline). In June 2002 the U.S. Food and Drug Administration (FDA) Blood Products Advisory Committee recommended that Hespan/balanced salt solution (HES/BS) (a) should not have a warning label for bleeding, (b) is pharmacologically different from HES/NS, (c) is equivalent to 5% albumin, and (d) is superior to HES/NS.

Gan and colleagues studied 120 patients undergoing major elective surgery who received HES/NS or HES/BS intraoperatively. Table 31.4 compares these solutions in patients who needed blood transfusions. No statistical differences between groups were found for volume of HES, crystalloid, packed red blood cells (PRBC), fresh frozen plasma (FFP), platelets, or cryoprecipitate infused, or estimated blood loss. However, in the subgroup of patients who

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received intraoperative blood transfusions, there were significant differences in the mean heart rate and estimated blood loss. In addition, the thromboelastogram (TEG) *r*-time was measured. Patients who received ≥20 mL/kg of HES/NS had a statistically significant change in *r*-time from baseline to the end of surgery ($p = 0.01$). Patients who received HES/BS or <20 mL/kg of HES/NS did not have a significant change in *r*-time.

TABLE 31.4 HESPAN VERSUS HEXTEND IN PATIENTS NEEDING BLOOD TRANSFUSIONS

	HES/NS (n = 25)	HES/BS (n = 31)	p VALUE
Heart rate, beats/min	85	78	0.049
EBL, mL	2,516 ± 1,856	1,560 ± 99	0.02
PRBC, mL	1,516 ± 1,397	1,040 ± 639	
FFP, mL	288 ± 697	73 ± 202	
Platelets, mL	83 ± 205	7 ± 38	
Cryoprecipitate, mL	4 ± 20	0	

HES/NS, Hespan/normal saline; HES/BS, Hespan/balanced salt solution; EBL, estimated blood loss; PRBC, packed red blood cells; FFP, fresh frozen plasma.

Bennett-Guerro and colleagues included 200 perioperative (intraoperative plus 24 hours postoperative) coronary artery bypass graft and/or valvular heart surgery patients in a study comparing 5% albumin, HES/NS, and HES/BS administration. No differences between the albumin and HES/BS groups were identified. There was a statistically significant difference between the HES/BS and HES/NS groups for the number of units of PRBC and FFP transfused (Table 31.5). Serum creatinine measured 1 week postoperatively was also significantly different between the two groups (Table 31.5). Finally, Table 31.6 includes several additional studies that support the FDABlood Product Advisory Committee's recommendations on the use of HES/BS.

TABLE 31.5 CORONARY ARTERY BYPASS GRAFT AND/OR VALVULAR HEART SURGERY PATIENTS

	5% ALBUMIN	HES/NS	HES/BS
PRBC transfused, U	2 (0-2)	4 (2-6)[a]	2 (0-2)
FFP transfused, U	0 (0-4)	3 (0-6)[a]	0 (0-4.5)
Platelets transfused, U	0 (0-6)	6 (0-9)	0 (0-6)
Preoperative Scr, mg/dL		1.0 ± 0.3	1.0 ± 0.2
Postoperative Scr, mg/dL		1.5 ± 0.7[a]	0.9 ± 0.2

HES/NS, Hespan/normal saline; HES/BS, Hespan/balanced salt solution; PRBC, packed red blood cells; FFP, fresh frozen plasma; Scr, serum creatinine.

[a] $p < 0.05$ between HES/NS and HES/BS groups.

TABLE 31.6 COMPARISON OF 5%ALBUMIN, HES/NS, AND HES/BS

	PRODUCTS COMPARED	PATIENT POPULATION	RESULTS
Petroni et al.	HES/BS, 5% albumin/LR	Perioperative cardiac surgery patients ($n = 28$)	No difference in chest tube output, pre/postoperative Hct, or blood transfusions

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Gan et al.	HES/BS, 5% albumin/NS	Perioperative GU surgery patients ($n = 25$)	No significant difference in PT, aPTT, Factor VIII, vWF, or platelet function
Roche AM	HES/BS, HES/NS, LR	In vitro TEG *r*-time	75% dilution of human blood: HES/BS-diluted blood samples had similar *r*-time as fresh blood; HES/NS-diluted samples failed to clot.

HES/NS, Hespan/normal saline; HES/BS, Hespan/balanced salt solution; Hct, hematocrit; GU, genitourinary; PT, prothrombin time; PRBC, packed red blood cells; FFP, fresh frozen plasma; aPTT, activated partial thromboplastin time; TEG, thromboeslastogram.

There are several possible adverse reactions one should be aware of when giving Hetastarch products: volume overload, pulmonary edema, congestive heart failure, anaphylactic or anaphylactoid reactions (particularly with a corn allergy), hemodilution, nausea/vomiting, peripheral edema, submaxillary and parotid glandular enlargement, mild influenzalike symptoms, headaches, muscle pain, and pruritus. When giving Hetastarch in a NaC1 solution, there is direct inhibition of Factor VIII, prolongation of activated partial thromboplastin time (aPTT), prothrombin time (PT), clotting and bleeding times, as well as increased risk of hyperchloremic metabolic acidosis.

Hetastarch products are available in 500-mL bags. The dosage and rate of administration depend on the clinical situation as well as the dynamics of the patient. Amaximum of 1,000 mL of the HES/NS product should be used in a 24-hour period. There is no recommended maximum amount of Hextend®.

TAKE HOME POINTS

- Hespan is a colloid that can last intravascularly from 3 to 6 hours.
- Hespan has relative contraindications in patients with coagulopathies, fluid overload, or renal impairments and may produce hypersensitivity reactions.
- Hetastarch comes in a 6% solution of either NaCl (Hespan or HES/NS) or LR (Hextend or HES/BS).
- In June 2002 the FDA Blood Products Advisory Committee recommended that HES/BS (a) should not have a warning label for bleeding, (b) is pharmacologically different from HES/NS, (c) is equivalent to 5% albumin, and (d) is superior to HES/NS.

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Protect the Kidneys, Not the "UOP"

Michael P. Hutchens MD, MA

It is axiomatic in anesthesiology that abundant, or at least physiologically normal, intraoperative urine output (UOP) is essential to good postoperative renal outcome. Although it is true that the best monitor of intraoperative renal function is UOP, this is unfortunately true only in the sense that there is no other monitor of even passable use. One should not confuse poor data from a bad monitor with excellent data (such as from end-tidal capnography) from a monitor that demands action. Unfortunately, the differential diagnosis of intraoperative oliguria is not hypovolemia alone.

Clearly, slavish devotion to producing a UOP of 3 mL/kg/hour in an anuric renal failure patient during a lengthy bowel procedure will predictably produce pulmonary edema and ultimately congestive heart failure. A more probable situation is that in which large volumes of fluid are administered to a physiologically normal patient in an attempt to correct presumed hypovolemia in an oliguric patient who in fact has a kinked Foley catheter or an unsuspected urinary tract injury in the surgical field. In the first case, delayed diagnosis of the kinked catheter and volume resuscitation will result in an overdistended bladder and ultimately obstructive renal failure. In the second, renal function may be unaffected.

It has been frequently observed that patients undergoing laparoscopic surgery have lower-than-expected intraoperative UOP. Aggressive volume resuscitation in these patients may or may not increase their UOP. Euvolemia or slight relative hypervolemia may be necessary to ensure adequate blood pressure in this setting (because of increased intrathoracic pressure reducing return to the right heart). But does the reduced UOP reflect a danger to the kidney? Early laparoscopists thought this was the case—that pneumoperitoneum caused renal vein and parenchymal compression, which resulted in oliguria and eventually renal injury. There is now good evidence to show otherwise. In multiple studies in animal models, pneumoperitoneum of 15 mm Hg or less (the upper limit for most laparoscopic procedures is 15 mm Hg) predictably reduces UOP but does not result in significant changes in ultrasound, pathologic, or chemical indicators of renal function. This question has also been evaluated in humans. As laparoscopic gastric bypass was being developed, Nguyen et al. reported on renal physiology in more than 100 patients assigned randomly to either laparoscopic or open gastric bypass. Despite longer operative time and 64% lower intraoperative UOP in
the laparoscopic group, there was no significant difference in postoperative blood urea nitrogen (BUN), creatinine, antidiuretic hormone (ADH), aldosterone, or renin levels, strongly suggesting that the reduced UOP reflects a reversible noninjurious physiologic consequence of pneumoperitoneum.

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Similarly, the development of the laparoscopic donor nephrectomy offered an opportunity to study the effect of pneumoperitoneum directly in both the retained and the transplanted (hence stressed) kidney. Hawasli et al. studied the effect on donated and retained kidneys of two different levels of pneumoperitoneum (10 and 15 mm Hg). They found no difference in UOP or other indices of renal function between the groups. Importantly, the transplanted kidneys functioned equally well whether harvested at high or low pressure. Intraoperative UOP is generally controlled aggressively with fluids, hypervolemia, and diuretics during donor nephrectomy, so it is impossible to assess relative oliguria in this population, but studies have shown that the laparoscopic donor operation produces allograft function that is equivalent to that of the open operation.

Laparoscopic surgery is not the only setting in which relative oliguria may not be harmful. Patients undergoing radical head and neck surgery have also been observed to have relative oliguria intraoperatively. Priano et al. studied "dry" versus "wet" resuscitation strategies in head/neck surgery patients with normal preoperative renal function, finding that resuscitation of more than 1 L/hour produced a UOP of 1.3 mL/kg/hour; half dose resuscitation was associated with significantly less UOP (.4 mL/kg/hour). Both groups had normal intraoperative and postoperative hemodynamics, and normal postoperative renal function.

The anesthetic doctrine of "plentiful pee," then, has important exceptions. This is the case because a "normal" UOP is a second-line surrogate marker for instantaneous renal function, analogous to depending on the surgeons' assessment of blood color to measure adequacy of oxygenation. New technologies such as continuous renal ultrasound and near-infrared regional oximetry offer some hope of improving the monitoring of intraoperative renal function. In the meantime, we must use the monitor we have with intelligence, sophistication, and a high level of skepticism.

TAKE HOME POINTS

- Urine output is a poor monitor of renal function, but it's the only one we have in the operating room.
- Laparoscopic surgery patients can have low intraop UOP without detriment to postoperative renal function.
- Radical head and neck surgery patients can have low intraoperative UOP without detriment to postoperative renal function.

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Do Not Treat Lactic Acidosis with Bicarbonate

Prasert Sawasdiwipachai MD

Lactic acidosis is one type of anion-gap metabolic acidosis (AGMA). It occurs when the tissue metabolism has to undergo anaerobic metabolism, which produces significant amounts of lactic acid.

Lactic acidosis occurs at the cellular level (mitochondria) when the oxygen delivery to the tissue is inadequate or the cells cannot utilize the oxygen. Many causes of lactic acidosis are recognized, the most common being a result of low oxygen delivery because of shock (septic, hypovolemic, cardiogenic, or neurogenic), severe anemia, or severe hypoxemia. It also can occur from exposure to drugs (e.g., metformin, antiretroviral therapy, acetaminophen, salicylates, cocaine, valproic acid, sulfasalazine, isoniazid [INH], 5-fluorouracil [5-FU]), toxins, and sugar alcohols (sorbitol, xylitol, fructose).

Many systemic diseases can also be complicated by lactic acidosis, including diabetes mellitus, alcoholism, pancreatitis, cancer, infections, vitamin B_1 deficiency, short-gut syndrome, malabsorption syndrome, etc. Certain rare inborn errors in metabolism (e.g., Von Gierke disease, methylmalonic aciduria, pyruvate dehydrogenase deficiency) can also be responsible for lactic acidosis.

The best treatment of lactic acidosis is to treat the underlying causes. This often includes volume expansion, correcting anemia, increasing cardiac performance, and correcting hypoxemia. If the cause is detected and treated promptly, the lactic acidosis should gradually subside. The severity of acidemia (arterial pH) can be used to follow the effectiveness of the intervention.

Alkali therapy (especially sodium bicarbonate) should not be given in lactic acidosis. Although it may initially correct the deranged laboratory value, it actually worsens the clinical condition. The exogenous bicarbonate will result in a significant amount of CO_2, which can move freely between the compartments (including across the blood-brain barrier), whereas the bicarbonate does not. This creates disequilibrium of strong ions. Many studies in both animal and human cells have confirmed the increase in intracellular acidosis after bicarbonate therapy. Bicarbonate administration is widely accepted in acidosis associated with chronic loss of bicarbonate (e.g., renal tubular acidosis, pancreatic transplantation, diarrhea) but not in lactic acidosis.

One final thought is that mild to moderate systemic acidosis is not as deleterious as was once thought. The growing use of permissive hypercapnia in acute respiratory distress syndrome (ARDS) patients has shown that many adult patient have no change in cardiac contractility even when systemic pH is as low as 7.15.

TAKE HOME POINTS

- Lactic acidosis is the final common pathway resulting from a number of conditions and diseases, such as shock, severe anemia, severe hypoxemia, drug exposure, diabetes mellitus, and inborn errors of metabolism
- Treat the underlying cause, not the numbers!
- Sodium bicarbonate will “fix the number” on a short-term basis, but it actually worsens the clinical situation by increasing intracellular acidosis.
- Mild to moderate systemic acidosis may not be as deleterious as was once thought.

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Consider the Use of Tris-Hydroxymethyl Aminomethane (THAM) to Treat Refractory or Life-Threatening Metabolic Acidosis

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Elliott R. Haut MD, FACS

You just assumed care of a 35-year-old man with severe septic shock and acute respiratory distress syndrome (ARDS) resulting from necrotizing pancreatitis and aspiration pneumonia. He has a combined metabolic and respiratory acidosis secondary to his underlying pancreatitis and permissive hypercapnia from the appropriate ventilatory strategy necessary for treatment of ARDS. His arterial pH remains <7.15 and mean arterial pressure (MAP) is 50 mm Hg despite being on a sodium bicarbonate ($NaHCO_3$) drip and multiple vasoactive agents. His serum sodium is 165 mEq/L and his serum creatinine is 1.0 mg/L. Your critical care attending physician suggests stopping the bicarbonate infusion and switching to tris-hydroxymethyl aminomethane (THAM) instead.

Acidemia, in critically ill patients, can have devastating consequences if it is not corrected in a timely fashion. These major adverse consequences often include impairment of cardiac contractility, cardiac arrhythmias, elevated pulmonary artery pressures, decreased responsiveness to catecholamines, hyperventilation, insulin resistance, hyperkalemia, increased metabolic demands, and changes in mental status. The role of buffers in treating severe acidosis (i.e., arterial pH < 7.20) remains controversial. Sodium bicarbonate is the most commonly used buffer used to treat severe acidosis in North America, but the use of sodium bicarbonate is not without consequences. Its use can be associated with hypernatremia, hyperosmolality, volume overload, and exacerbation of acidosis through an increase in CO_2 production.

THAM is a biologically inert weak base that has a greater buffering capacity than sodium bicarbonate (pK_a 7.8 vs. pK_a 6.8, respectively). THAM is effective in buffering severe metabolic acidosis, permissive hypercapnia associated with Acute Lung Injury/Acute Respiratory Distress Syndrome (ALI/ARDS), and mixed acidoses. THAM can buffer both metabolic acids ($THAM + H^+ = THAM^+$) and respiratory acids ($THAM + H_2CO_3 = THAM^+ + HCO^-_3$). Unlike sodium bicarbonate, THAM does not generate CO_2 but uses CO_2 to generate bicarbonate and also buffers other sources of protons in body fluids, such as lactic acid.

Currently, there are few clinical trials regarding the efficacy of THAM in treating severe acidosis in critically ill patients. The literature supports consideration for the use of THAM in patients with the following acid-base disturbances: severe metabolic acidosis and hypernatremia, extreme levels of permissive hypercapnia in patients with ALI/ARDS at maximum ventilatory support, treatment of dilutional acidosis intraoperatively, and failure of sodium bicarbonate to correct metabolic acidosis. The usefulness of THAM has been limited because of its serious side effects, including hyperkalemia, hypoglycemia, ventilatory depression, local injury with intravenous extravasation, and hepatic necrosis in neonates. P.148

ADMINISTRATION OF THAM

THAM is available in several preparations. THAM base solution has a pH = 10.4 and should be administered only through a central venous catheter. THAM acetate solution (0.3 mol/L concentration is titrated to pH = 8.4 with acetic acid) can be administrated peripherally because it does not cause venous irritation. THAM also comes as a citrate syrup. It is indicated for use in patients with life-threatening acidemia (defined as pH 7.1 to 7.2, $paCO_2$ < 35 mm Hg, and lactic acid > 10 mEq/L) to temporarily or permanently normalize the acid-base status. THAM should be administered as a loading dose of 2 to 4 mmol/kg over 20 minutes, followed by a constant infusion of 0.5 to 1 mmol/kg/hour for 4 to 10 hours. The estimated dose of THAM is calculated as follows:

$$\text{Volume of 0.3 mol/L THAM (mL)} = \text{lean body weight (kg)} \times \text{base deficit (mEq/L)}$$

THAM is excreted by the kidneys, as $THAM^+$ (the protonated form), through glomerular filtration as a nonreabsorbable cation paired with an anion by the kidneys. It has an osmotic diuretic effect, increasing the excretion of urinary sodium and chloride. THAM can accumulate in patients with renal failure and produce an "osmolar gap" with pseudohyponatremia. Renal clearance of THAM is 2% greater than that of creatinine. At steady state, if no drug accumulates, the rate of THAM elimination must equal the rate of THAM infusion. It is recommended that the THAM plasma concentration be maintained ≤6 mmol/L. To calculate maximum infusion rate,

$$\text{Infusion rate of THAM (mmol/hr)} = 0.06 \times \text{creatinine clearance (ml/min)} \times \text{THAM plasma level (desired)}$$

THAM should generally be avoided in evolving renal failure, but if it is used, it must be monitored closely and stopped if hyperkalemia and an osmolar gap begin to develop.

TAKE HOME POINTS P.149

- The primary goal in the treatment of metabolic acidosis is reversing the underlying cause.
- THAM may represent a viable alternative to sodium bicarbonate in treating severe metabolic acidosis.
- THAM should be used with extreme caution in patients with renal insufficiency.

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- Hyperkalemia, hypoglycemia, and ventilatory depression should be actively watched for in patients receiving THAM.

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Use the Principles of "Damage Control Anesthesia" in the Care of the Massively Bleeding Patient and Ask the Surgeons to Implement "Damage Control Surgery" if Necessary

T. Miko Enomoto MD

Michael P. Hutchens MD, MA

Intraoperative life-threatening hemorrhage must be controlled with teamwork between the surgeons and anesthesiologist. With the realization that ongoing hemorrhage after injury leads to derangements in the body's ability to maintain hemostasis and homeostasis, trauma surgeons have evolved the concept of "damage control surgery," which proposes that definitive repair or fixation can in many cases be delayed until the patient has been resuscitated to the goals of appropriate blood composition, normalization of acid-base status, and normothermia. Among the anesthesia provider's roles in the care of the massively bleeding patient is to implement the accompanying concept of "damage control anesthesia" and help the nontrauma surgical team recognize that the magnitude of hemorrhage may require altering the surgical goals and plan.

At its most basic, damage control anesthesia seeks to mitigate the effects of the lethal triad of acidosis, hypothermia, and coagulopathy. Hypothermia results from infusion of intravenous (IV) fluids, and convective and conductive heat losses. Coagulopathy, resulting from depletion of clotting factors by ongoing hemorrhage, is exacerbated by hypothermia and acidosis, leading to further bleeding and hypotension. Resulting poor perfusion furthers acidosis, and the classic "downward spiral" ensues.

Dutton identifies the essentials of damage control anesthesia as: (a) control of the airway and ventilation, (b) control of bleeding, (c) preservation of homeostasis, and (d) analgesia and sedation. Indeed, these are similar to routine anesthetic care, but there are a few key differences.

Fluid resuscitation needs to focus not only on replacing circulating volume, but also on maintaining the appropriate blood composition in terms of oxygen-carrying capacity, clotting potential, and chemistry. This requires large-bore IV access, usually considered to be a minimum of two 18-gauge or larger IVs, and frequent laboratory analysis of blood. The desired end points or goals of volume resuscitation are currently being re-examined in light of recent evidence suggesting that hypotensive resuscitation may be beneficial. Strategies for hypotensive resuscitation include delayed resuscitation, in which fluid resuscitation is delayed until after control of the site of bleeding, and permissive hypotension, in which resuscitation is continuous but with a goal of a subnormal blood pressure. In a landmark study, Bickell et al. demonstrated a survival advantage to limited volume replacement until the time of operative intervention in the setting of penetrating torso trauma. A proposed mechanism for the advantage of hypotensive resuscitation is that, by limiting fluid volume, regional vasoconstriction and lower blood pressure allow the adhesion of the fragile platelet-fibrin clot. In addition, the composition of the blood, including coagulation factors, is minimally diluted. Limited infusion of fluids reduces the development of hypothermia.

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Of course, the potential benefit of hypotensive resuscitation in the multiply injured trauma patient must be weighed against the risk of worsening hypoperfusion, resulting in increased acidosis and ischemic organ damage. Hypotension resulting from hemorrhagic shock is thought to be a significant mechanism for the "second hit hypothesis," which adds ischemic injury to the initial insult in the setting of traumatic brain injury. In practice, blood pressure is typically used as one of the main parameters guiding resuscitation, but there is abundant animal and clinical data that blood pressure is not an accurate gauge of tissue oxygenation.

Diligent attention must be paid to maintaining normothermia: Keep the ambient temperature warm despite medical team discomfort, warm all IV fluids using a rapid infuser with inline air detection and heat exchanger, place a convective warmer where possible, and minimize the length of exposure of internal organs and raw surfaces.

Blood products should be replaced in the appropriate proportions, guided by cell counts and coagulation studies. Active repletion of calcium and other electrolytes must be pursued, guided by frequent chemistries. In the setting of active bleeding, the platelet count should generally be maintained > 100K, and the international normalized ratio (INR) kept < 1.4. These goals should be adjusted based on the clinical situation.

In addition to direct control of a bleeding vessel, there are many methods of achieving hemostasis. Packing of venous bleeding or diffuse oozing, or clamping of arterial bleeding, can be used as a temporizing measure while the patient is warmed, resuscitated, and stabilized. In the setting of some types of bleeding, such as that resulting from pelvic trauma, angiographic embolization may be the safest and fastest way to control the bleeding without causing further damage. For diffuse bleeding from raw surfaces, novel topical hemostatic agents, such as fibrin glue, chitosan (HemCon), modified rapid deployment hemostat (MRDH), or QuikClot, continue to become available. In the setting of diffuse coagulopathic bleeding not controlled by replacement with blood products, administration of recombinant factor VIIa (rFVIIa) should be considered. rFVIIa is postulated to jump-start the initiation phase of coagulation in a site-specific manor, and has been shown to be of benefit in trauma patients who fail standard methods of correcting coagulopathy. Significant drawbacks include great expense and a small risk of thrombotic complications.

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Perhaps the most important thing that the anesthesiologist can do to prevent lethal hemorrhage is to communicate with the surgical team. If the patient is unstable, the surgeons must be made aware of this.

TAKE HOME POINTS

- Damage control surgery was developed for the case of massively bleeding patients whose medical status is compromised by the hemorrhage. It is based on delay of the definitive repair.
- Damage control anesthesia involves correction of coagulopathy, acidosis, and establishment of normothermia.
- Become familiar with the concept of "limited resuscitation" and "limited infusion" techniques as applied to trauma patients.
- Remember that blood pressure is not necessarily an accurate indicator of tissue oxygenation.

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Learn from the Care of the Combat Victim: Ask the Surgeons to Consider Damage Control Surgery for the Bleeding Patient

Surjya Sen MD

Damage control surgery is defined as operative management that employs a staged approach to the severely injured patient. It seeks to avoid "physiologic burnout" seen with the lethal triad of hypothermia, acidosis, and coagulopathy. Definitive therapy for the patient's problem or injury is deferred to give preference to the stopping of hemorrhage and the control of contamination by using the simplest and most rapid means possible. Temporary wound closure methods are employed initially, with a plan to return the patient to the operating room after physiologic abnormalities have been corrected (or at least stabilized) in an intensive care setting.

HISTORICAL PERSPECTIVE

Traditionally, it was believed that the operative management of a patient should provide definitive care even if the physiologic condition of a patient was deteriorating (especially if the physiologic deterioration was due to a problem that was the focus of the operation). In the early 1900s, this dogma was challenged in management of battlefield victims with exsanguinating injuries. J. H. Pringle introduced the techniques for the temporization of hepatic hemorrhage for trauma by using liver packs and vascular compression. However, because of poor outcomes, these techniques fell out of favor until the 1970s and 1980s, when they were reintroduced with "abbreviated laparotomies." These procedures were used to temporize the source of bleeding in trauma victims so that they could return later to the operating room, after their coagulopathies had been corrected. By the 1990s, damage control surgery (coined by Rotondo in 1993) had gained popularity not only for application toward soldiers in combat settings, but also for trauma victims in civilian settings.

APPLICABILITY TO NONCOMBAT SITUATIONS

Although damage control surgery offers evident advantages in a combat situation (faster evacuation times, better use of limited resources, and more efficient management of a large number of casualty victims who may present simultaneously), it also has applicability in civilian traumas. Abbreviated surgeries allow the surgical, anesthesia, and intensive care teams to steer clear of irreversible physiologic end points such as uncontrolled hemorrhage from disseminated intravascular coagulopathy. Avoiding life-threatening abnormalities such as hypothermia, coagulopathy, and acidosis are the primary end points for the care providers. Once these have been met, the patient is returned to the operating room for definitive management.

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BASICS OF THE PROCEDURE (THREE STAGES)

The following outlines the damage control procedure. Initial Management

- Initiation of advanced trauma life support (ATLS) protocols
- Proper patient selection
  - Patients with injuries that are technically difficult
  - Patients who will require extensive, multistep surgical procedures
  - Patients with exsanguinating blood loss
  - Patient in whom delaying definitive repair will not result in death

Stage 1 (the damage control operation)

- Abdominal injury
  - The abdomen is usually packed with a goal to tamponade bleeding while maintaining organ perfusion.
  - Vascular injuries may be treated with ligation, temporary intraluminal shunts, or intravascular balloon catheters.
  - Hollow viscus injuries are often resected (reanastomosis is postponed).
  - Biliopancreatic injuries can be temporized with closed suction drainage.
  - Renal damage is often best treated with radical nephrectomy.
  - Closure: Temporary closure is employed.
- Thoracic injury
  - Usually involves the placement of chest tubes.
  - Management of cardiac tamponade.
  - Lobectomies or pneumonectomies are often performed.

Stage 2 (resuscitation)

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- Patients are returned to the intensive care unit (ICU) for correction and management of
  - Acidosis
  - Hypothermia
  - Coagulopathy

Stage 3 (definitive operation)

- Exact timing of the reoperation has not been standardized.
- Patients who return in less than 72 hours tend to have lower morbidity and mortality.
- Athorough exploration is performed to ensure that no injuries have been missed.

COMPLICATIONS

Complications following damage control surgery tend to not be specific to this type of procedure, but rather to the population of surgical and critically ill patients. Local complications include abscesses, fistulas, necrosis, and pseudocysts; systemic complications include acute respiratory distress syndrome (ARDS), abdominal compartment syndrome, and multiple organ failure.

OUTCOMES

The reported incidence of ARDS and multiple organ failure in this patient population ranges from 14% to 53%. The best predictors of mortality tend to be the *initial pH* upon return to the ICU (for stage 2) and the *worst partial thromboplastin time PTT* from hospital admission to ICU admission. Overall mortality following damage control surgery varies, but the trend has shown a progressive decrease, from as high as 67% (in 1983) to as low as 10% (in 2001).

TAKE HOME POINTS

- Damage control surgery has applicability in both the combat and noncombat situation. It can be employed in the treatment of trauma victims and even in elective operations when unforeseen complications lead to the destabilization of normal physiologic homeostasis.
- The central focus of damage control surgery attempts to delay definitive therapy while prioritizing the management of coagulopathies, maintaining normothermia, and correcting acid-base abnormalities.
- Patients who undergo definitive therapy within 72 hours tend to have better outcomes.

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Know What Screening Tests are Performed on Volunteer Donor Blood

Andrew M. Gross MD

Donated blood is subjected to a rigorous series of tests before it is deemed safe for transfusion. The list of tests performed by the Red Cross seems ever expanding, and this has resulted in an overall safer blood supply and fewer transfusion-associated risks. Blood testing consists of screening for infectious disease as well as ABO screening, Rh screening, and testing for various red-cell antibodies.

Infectious disease screening is accomplished two ways. An initial level of screening is by self-report of previous diseases and living or travel situations. Since March 2005, all blood donors in the United States must complete a 54-question survey called the Universal Donor Survey. For example, donors are asked if they have had oral surgery in the last 3 days (to eliminate the possibility of transient bacteremia of mouth pathogens) or have ever had certain blood-borne infections such as malaria or babesiosis (both intracellular erythrocyte parasites). They are also queried about residence in Europe during the interval that may increase the epidemiologic risk for bovine spongiform encephalopathy. Affirmative answers on the test can disqualify the donor temporarily or permanently.

Serum tests for infectious disease entities include HIV-1 and -2, hepatitis C (HCV), hepatitis B (HBV), human T-lymphocyte virus (HTLV)-I and-II, West Nile virus, and *Treponema palladium* (syphilis). Alanine aminotransferase is also tested as a laboratory marker of infectious disease. All viruses are initially tested for by enzyme-linked immunoassay (ELISA). An initial positive result is then rechecked in two separate ELISA tests, and if one of those two subsequent tests is also positive, the blood is discarded. Only if both are negative is the blood taken out of special quarantine and subjected to further testing.

Anew method of testing blood was started in 1999. Nucleic acid testing or NAT is used to screen blood for the presence of HIV, HCV, and, as of 2003, West Nile virus. Nucleic acid testing is done by polymerase chain reaction (PCR) or transcription-mediated amplification (TMA). As both of these are fairly expensive tests, most commonly nucleic acid testing is done on a minipool sampling. This involves pooling multiple donations, and if an infection is present, then all of the individual donations in the pool are tested. This supersensitive assay is responsible for detecting HIV an average of 11 days earlier than other forms of testing, and in concert with ELISA and Western blot tests for anti-HIV antibodies, has decreased the risk of transfusion-related infection to 1 in 2 million in the United States, 1 in 5 million in Germany, and 1 in 10 million in Canada. HCV infection has been dramatically reduced using nucleic acid testing, from 1 in 100,000 in 1996 to 1 in 2 million in the United States in 2005. Nucleic acid testing results in a decrease from an average of a 70-day window for undetectable infection using ELISA screening for HCV antibodies to an 8- to 10-day window of undetectable infection.

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Blood infected with HBV is screened for by hepatitis B surface antigen and core antigen testing. The latency period of about 60 days results in a higher amount of undetected virus in the donor pool and thus HBV infection risk remains the highest among the diseases tested for—estimates vary from 1 in 60,000 to 1 in 270,000. HTLV-I and -II are screened for by ELISA and confirmed by Western blot or PCR. The resulting infection rate is estimated at 1 in 2 million for HTLV. Syphilis is also screened for by a number of serologic assays varying from lab to lab using highly sensitive specific antigens for *T. palladium*. Finally, cytomegalovirus (CMV) is screened for only in a select number of cases, because of the need for CMV-negative blood for transplant recipients and the immunosuppressed population, especially HIV-infected individuals.

In addition to infectious agents, blood type testing is still of critical importance, and all blood donations are screened for ABO type using anti-A and anti-B antibodies. Each unit is also tested for the presence of a D antigen. Those who have the D antigen are said to be Rh-positive, and those without, Rh-negative. Blood types and Rh type are rechecked in individual hospital blood banks before dispensing units for transfusion, as the result of a mislabeled or improperly tested unit could be catastrophic or even fatal. Some but not all donated blood is subsequently screened for red-cell antigens. These antigens include the remainder of the Rh system in addition to the D antigen and the more minor groups (e.g., Duffy, Kell, Big E, Kidd). Special units that are negative for all of these antigens are stored for donation to people who have preformed antibodies to these groups. Cross-matching is done in the blood bank at individual hospitals to determine which antigens are present in donated blood versus which antigens and antibodies are present in the recipient. Donated blood is itself screened for antibodies to the minor blood groups by exposing it to control red cells that have sed antigens present on their surface. The components that have preformed antibodies cannot be given to a recipient with the corresponding antigen, as major complications can arise.

Blood transfusions are a crucial part of practicing anesthesiology. Many critically ill patients' lives will depend on receiving transfusions that are safe and properly administered. In order for an informed decision to be made by both physician and patient, it is important to understand the risks of blood administration. Transfusions are still a source of anxiety for patients, and knowing how to alleviate that fear is of the utmost importance. Most anxiety surrounding blood transfusion stems from patients' fears of contracting an infectious disease, and although the risk is low, it is not zero. Of understandable concern to patients is the risk of acquiring a disease from an “emerging” condition such as Lyme, ehrlichiosis, or Chagas disease; all conditions that are well established in the United States. These and other conditions with known or probable blood transmission can be debilitating or fatal if acquired and are not tested for in the blood supply in the United States. When discussing the risks of acquiring these diseases (or yet-unknown diseases; thousands of patients died from AIDS acquired through blood transfusions), it is incumbent to tell patients that self-report measures have never been proven to be fail-safe screening procedures for preventing transmission of blood-borne pathogens.

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TAKE HOME POINTS

- Screening of volunteer donor blood starts with the Universal Donor Survery—this is strictly a “self-report” instrument that attempts to indirectly screen for such blood-borne diseases as malaria and babesiosis. For a true positive diagnosis, both these disease require a thin-smear erythrocyte analysis, which is not performed on donated blood. In Lyme-endemic areas, such as Connecticut, it has been determined that a certain percentage of volunteers presenting for blood donation are actually asymptomatic carriers of the babesia parasite. Transfusion-related transmission of the disease has been reported.
- The first level of serologic testing is by ELISA—this is intended to screen against viral and spirochetal organisms.
- Nucleic acid testing is usually done on pooled samples and has increased the sensitivity of the serologic testing process.

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- The blood supply has increased in safety in recent years, but of course is not completely safe, and the risks should not be minimized or "glossed over" in discussions with the patient. A good approach is to discuss the relative risks of transfusion vs. significant perioperative events linked to anemia, such as retinal ischemia and visual loss.

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Transfusion of Packed Red Blood Cells Requires a Careful Risk-Benefit Analysis

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Michael P. Hutchens MD, MA

Transfusing allogenic red blood cells is an intervention that has both life-saving and life-threatening potential. Anesthesia providers are familiar with the deleterious effects of acute hemorrhagic anemia: Reduced cardiac output associated with severe blood loss can result in tissue hypoxia and eventually organ dysfunction and failure. Consequences of anemia depend on the patient's ability to compensate for these physiologic changes. Because hemoglobin values are readily determinable in most intraoperative settings, it is tempting to transfuse packed red blood cells based on a number. However, the down sides to transfusion are more complex than just acute transfusion reaction alone. In order to fully assess the risks and benefits of transfusion, anesthesiologists must have a thorough understanding of their patient's current medical condition and co-morbidities as well as the current literature on transfusion.

Laboratory experiments indicate that extreme hemodilution is well tolerated in healthy animals. Animals subjected to acute hemodilution tolerate decreasing hemoglobin concentrations down to around 5 g/dL, with ischemic electrocardiographic changes and depressed ventricular function noted at these levels. However, acute hemodilution is tolerated less well in animal models of coronary stenosis—ischemic electrocardiographic changes and depressed cardiac function are seen at hemoglobin concentrations between 7 and 10 g/dL. Human data regarding the limits of anemia tolerance are inadequate and often conflicting.

Guidelines regarding red cell transfusion have been presented by the American Society of Anesthesiologists Task Force on Blood Component Therapy. The authors strongly recommend reading the document in its entirety. The Task Force consensus states that transfusion is rarely indicated when hemoglobin concentration is greater than 10 g/dL and is almost always indicated when it is less than 6 g/dL, especially when the anemia is acute. Determining the need to transfuse for levels in between these numbers should take into account the individual's risks of potential damage from possible inadequate oxygen delivery.

A patient's likelihood of complications from anemia must be weighed against the risks of red cell transfusion. Some of the known risks of transfusion include threat of infection such as HIV, hepatitis B or C, or cytomegalovirus (CMV). Other, unquantified infectious risks almost certainly exist. There is also a danger of hemolytic or nonhemolytic reactions, allergic reactions, transfusion-related acute lung injury, and anaphylactic shock with any blood product transfusion. Despite numerous safeguards, transfusion mishaps due to human (often clerical) error remain the most common cause of serious transfusion reaction. P.161

Numerous studies suggest that patients who are transfused more liberally have worse outcomes, in both the short and long term. Specific perioperative hazards associated with liberal transfusion include increased delirium and worsened pulmonary function. Some studies have shown increased postoperative infection, mortality, and length of stay. The seminal TRICC trial of 1998 concluded that intensive care unit (ICU) patients who received transfusions to maintain their hemoglobin greater than 9 g/dL (rather than greater than 7 g/dL) had increased mortality. These effects are thought to be secondary to immunomodulation from leukocytes in stored blood. There is speculation that routine transfusion of leukodepleted blood may reduce some of these adverse outcomes. Another important sequela of red blood cell transfusion is decreased long-term survival of some cancer patients, again possibly mediated by an alteration of immunologic status, although this is not borne out in large-scale studies.

Note: An interesting aspect to this discussion is whether anesthesiologists can and/or should use ICU studies as guideline for intraoperative transfusion practices. The question is do ICU patients comprise a similar cohort for the average same-day-admit patient (ASA 1-3) that we routinely care for? It is the senior author's opinion that although the data on transfusions are complicated, confusing, and occasionally contradictory, the power of the aggregate data is quite large. The author feels that a combination of this power and what can reasonably be taken as “expert consensus” justifies a more restrictive approach to transfusion.

It is still true that the best therapy for the perioperative anemic patient who is exsanguinating is blood transfusion. This is a clinical situation that is usually readily recognized by anesthesia providers. However, it is the patient with a relatively stable hemoglobin in the range between 6 and 10 g/dL that presents a clinical conundrum. Patients with risk factors for organ dysfunction, such as severe coronary artery disease, known carotid artery stenosis, a history of transient ischemic attack (TIA), or renal insufficiency will least tolerate anemia and are therefore thought to be most likely to benefit from a more liberal transfusion strategy, although slim evidence supports this conclusion. As the risk of anemia to the patient is that of oxygen supply-demand imbalance, a rational transfusion strategy should include the full armamentarium of strategies to optimize oxygen balance. If clinically appropriate, pharmacologic reducers of oxygen demand, such as optimal anesthesia, opioids, and beta-blockers, should be used. It is reasonable to deliver high concentrations of inspired oxygen for short periods of increased oxygen demand. Nontransfusion strategies to increase red cell mass, such as intraoperative autotransfusion, acute normovolemic hemodilution, or preoperative autologous donation and retransfusion, should be considered. Antifibrinolytics such as aminocaproic acid, aprotinin, or tranexamic acid may be considered in cases in which exuberant fibrinolysis is a known consequence, such as liver transplant or cases involving cardiopulmonary bypass. P.162

Remember also that patients with coronary or cerebrovascular disease are not the only ones who may not tolerate sudden decreases in hematocrit. Patients undergoing spinal surgery with prolonged intraoperative hypotension, perioperative anemia, and facial swelling are at risk of developing posterior ischemic optic neuropathy (PION). PION follows infarction of the posterior portion of the optic nerve and can result in the devastating complication of postoperative visual loss. This list of risk factors is based on retrospective analyses and a very small number of actual cases; it is not clear that transfusion alone can prevent PION, and experts are in disagreement on this topic. Nonetheless, the best available data suggest that it is prudent to closely monitor these patients and consider a different threshold for red cell blood transfusion.

Generally, experienced anesthesia providers do not transfuse packed red blood cells without discussing the ongoing clinical situation (as possible) with the surgeons and notifying them that transfusion is planned. Because the intraoperative period can be busy, one way to handle the situation is to talk with the surgeons in the preoperative period and outline the parameters that would trigger a transfusion. This is a situation in which it is necessary to consider closely the surgeons' opinions; however, it is unusual for a surgeon to disagree with a thought-out and well-articulated plan.

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TAKE HOME POINTS

- It is no longer axiomatic that blood is the best intravenous fluid.
- Although it is not possible to state with certainty that transfusion is hurtful, the preponderance of evidence suggests that the outcomes of transfusion are complex and deleterious and may outweigh the advantages of simple resolution of perioperative anemia.
- Don't forget about the important consideration of postoperative visual loss and the role that anemia may plan in its etiology.
- Talk to the surgeons!

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Jehovah's Witnesses and Transfusion: Ethical Issues

Rose Christopherson MD, PhD

The issues surrounding transfusion of Jehovah's Witnesses should be examined in the broader context of medical ethics. Two aspects of medical ethics are important in this discussion: informed consent, and Do Not Resuscitate (DNR) orders. Patients are normally informed about their treatment plans. They have the right to refuse any care, as long as they are both competent and have capacity to make health care decisions. Many Jehovah's Witnesses refuse transfusion of blood or some blood products because of their interpretation of certain passages in the Bible. Because other patients have the same right to refuse blood transfusion, it is not appropriate to argue with patients about their reasons for refusal. If they are competent, they have the right to refuse.

It is important to determine whether the patient is competent and has the capacity to make medical decisions, and to determine exactly what the patient refuses. Some patients are willing to accept reinfusion of their own blood, or transfusion of some blood components such as fresh frozen plasma. Jehovah's Witnesses are as diverse a group as any other religious group. It is wrong to assume from the fact that a patient is a Jehovah's Witness that she or he refuses blood transfusion. It is also important that there is no coercion of the patient, intended or unintended, because of the presence of family members or clergy. This is best done by speaking with the patient alone. It is important, in obtaining informed consent, to make it very clear that patients do not necessarily die as a result of withholding of transfusion. They may have strokes, myocardial infarction, or other organ damage. Thus the patient needs to understand that, if transfusion is withheld, she or he may emerge from the anesthetic with permanent impairment.

All participating caregivers must understand the patient's wishes and agree to the same plan. Surgeons have been known to refuse to perform surgery on patients who refuse transfusion. The type of surgery to be performed, urgency of the surgery, and likelihood of blood loss are all important. Participating nurses must agree with the plan. Even though the anesthesiologist will give or withhold blood products, if the patient dies or has some other adverse outcome related to anemia, all on the care team will share the pain of this bad, and in a sense avoidable, outcome.

Often, Do Not Resuscitate (DNR) orders are suspended in the perioperative period. Transfusion of blood or blood products is often part of resuscitation. P.165
DNR orders are on most patients' charts because of some terminal disease the patient has. When these patients need surgery for palliation or for some unrelated problem, they are generally willing to have their DNR orders cancelled for the perioperative period. They may need airway management or administration of resuscitation medicines to maintain appropriate vital signs during surgery. Their DNR status will resume after surgery.

Jehovah's Witnesses, on the other hand, are often quite healthy, and they may want every form of resuscitation other than blood transfusion. The situation can be even more troubling if the Jehovah's Witness wants transfusion withheld from a child or an elderly parent. Thus the refusal of a Jehovah's Witness, or of any other patient, of perioperative transfusion is somewhat contrary to accepted hospital procedures related to perioperative resuscitation.

It is not surprising if anesthesiologists feel uncomfortable about caring for Jehovah's Witnesses. We are asked to agree to things that we do not otherwise agree to. We are asked, potentially, to allow a patient to bleed to death while withholding the red cells, platelets, or fresh frozen plasma that would deliver oxygen to their tissues or even help stop the bleeding. Physicians or other caregivers may feel that they are asked to compromise their own religious or other deeply held moral beliefs. In fact, this is true. The patient's wishes have some priority over the physician's wishes, simply because treating patients against their will is battery and is not permitted.

However, it is not clear that a caregiver who refuses to anesthetize a patient because of the patient's unwillingness to receive blood is necessarily doing wrong. In some cases it is obvious that the patient is very unlikely to survive a procedure without transfusion. If at all possible, the caregiver should find another anesthetist, who is willing to agree to the patient's wishes. Lists of colleagues who are willing to withhold transfusion are helpful. It is never possible to avoid this conflict completely. Sometimes anesthesiologists will have to decide whether their duty to give the anesthetic and do their best to keep patients in good condition without transfusion outweighs their personal religious or moral beliefs.

Finally, we turn to patients who cannot make health care decisions. This includes unconscious patients; incompetent patients; and children. Some of these patients may have a document stating what interventions may be made on their behalf, e.g., a living will. Some Jehovah's Witnesses may carry a wallet card, which may state that they refuse blood. However, if such documentation cannot be found, given the diversity of beliefs among Jehovah's Witnesses,
it should be assumed that transfusion is permissible. In the case of children, there is legal controversy (www.virtualmentor.org). If surgery is elective, it is P.166
probably best to obtain consultation from both an ethics committee and a hospital attorney.

TAKE HOME POINTS

- The issues involved in the care of Jehovah's Witness patients are rooted in the medical ethics problems of informed consent and DNR orders.
- Do not argue with a Jehovah's Witness patient about his or her reasons for refusal of blood or blood products.
- Jehovah's Witnesses are as diverse a group as any other religious group. It is wrong to assume from the fact that a patient is a Jehovah's Witness that she or he refuses blood transfusion.
- Always speak to the Jehovah's Witness in a confidential location.
- Accept that there may be varying levels of discomfort among the anesthesia and operating room teams. It is never possible to avoid this conflict.
- Unconscious, incompetent, and pediatric Jehovah's Witness patients are a special situation—consult the ethics and legal staffs.

MANAGEMENT STRATEGIES FOR JEHOVAH'S WITNESS PATIENTS—THE BASIC 3 PART APPROACH

1. Minimize Losses

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- Consider staging complex procedures so that time between operations can allow some recovery for patients
- Apply damage control surgery principles to minimize bleeding as a first objective, then return later for definitive treatment
- Minimize blood draws
- Avoid anticoagulants and antiplatelet agents as much as possible
- Consider Recombinant Factor VIIa to decrease the need for further transfusions and improve hemostasis
- Employ the use of hemostatic surgical devices like electrocautery, ultrasonic scalpels and tourniquets
- Utilize pharmacologic agents such as fibrin glues as blood vessel sealants and antifibrinolytics
- Consider using strategies such as normovolemic hemodilution, controlled hypotension and continuous recovery cell salvage devices

2. Increase Production

- For planned elective cases, use recombinant human erythropoetin to improve reticulocytosis and increase normboblast production
- Supplement with IV iron, folate and vitamin B12

3. Stabilization and substitutions

- When bleeding is suspected, take decisive interventional steps early to quickly control blood loss
- Supplement oxygen and resuscitate fluid losses aggressively to improve oxygen delivery
- Manage coagulopathies early
- In select cases, consider blood substitutes such as Polyheme and Hemospan (still in clinical trials)

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Beware of the Mechanical Bowel Prep

James C. Opton MD

Mechanical bowel preparation (MBP) is commonly used for colonoscopy and elective colorectal surgery. Its goal is to rid the colon of solid fecal material. It is believed that the "clean" bowel has a lower bacterial load and has a lower risk of contaminating the wound and peritoneal cavity during surgery. The prepped bowel is much easier to manipulate during surgery and enables the surgeon to perform intraoperative colonoscopy. Disruption of the anastomosis and the sequela of an anastomotic leak may be attenuated by the MBP as well. Other surgeries in which the MBP is commonly used include abdominal and pelvic operations involving the aorta, kidneys, bladder, and reproductive structures. Absolute contraindications to the mechanical bowel prep include complete bowel obstruction and free perforation.

For colonoscopy and elective colorectal surgery, MBP is the standard of care worldwide. Historically, agents such as castor oil, senna, bisacodyl, and magnesium citrate have all been used in conjunction with a low-residue diet. Whole-gut lavage with large volumes of isotonic solutions via a nasogastric tube has also been used. However, this technique has been associated with side effects including electrolyte abnormalities, abdominal distention, nausea, and vomiting. Whole-gut lavage with mannitol has also been used, but this practice has been shown to be associated with catastrophic intraoperative explosions when gases produced from fermentation of mannitol by *Escherichia coli* were ignited by electrocautery. Currently, the two most widely used agents for MBP are polyethylene glycol and sodium phosphate.

Polyethylene glycol (PEG) was developed in the 1980s as an oral agent for MBP. It is an iso-osmotic, nonabsorbable sodium sulfate-based solution that cleans the bowel by washout of ingested material without significant fluid and electrolyte shifts. Patients are required to drink at least 2 to 4 L of solution along with additional fluids. Abdominal cramping, nausea, and vomiting are common side effects, and patients are typically prescribed prophylactic antiemetics.

Sodium phosphate (NaP) was developed in response to patient dissatisfaction with PEG and has generally been found to be more tolerable and associated with a higher rate of compliance. NaP is a hyperosmotic electrolyte solution that provides high-quality bowel cleansing while avoiding the need to ingest large volumes of solution. It can be taken in two 45-mL doses or in a series of 40 tablets. Abdominal cramping, nausea, and vomiting are common side effects. P.171

Because NaP is a hyperosmolar solution, a watery diarrhea is induced with its administration. A negative fluid balance can ensue, which can lead to hypovolemia that may become especially evident at the induction of anesthesia if adequate fluid replacement has not taken place. PEG, on the other hand, leaves the fluid balance virtually unaffected. Water and electrolytes are neither absorbed nor excreted from the bowel. Therefore, patients do not usually exhibit hemodynamic instability during the induction of anesthesia. For these reasons NaP may have a narrower therapeutic index than PEG, especially in patients who may be vulnerable to shifts in intravascular volume. NaP has been linked more frequently than PEG to serious electrolyte and metabolic derangements. Most commonly reported are transient decreases in pH, magnesium, calcium, and potassium. Hypernatremia and hyperphosphatemia can also occur, because these solutions contain 48 g of monobasic sodium phosphate and 18 g of dibasic sodium phosphate per 100 mL of solution. Although most patients are not clinically affected by these electrolyte abnormalities, NaP should be avoided in patients with renal disease, congestive heart failure, ascites, and in those who cannot adequately hydrate themselves. Furthermore, severe hyperphosphatemia and associated electrolyte and metabolic derangements following administration of NaP have been reported, as well as at least one case report of fatal hypocalcemic, hyperphosphatemic, metabolic acidosis following sequential NaP enema administration.

Cleansing the bowel before colorectal surgery with some form of MBP is a major surgical dogma with a long-standing history. However, this view has recently come into question. Several prospective, randomized trials comparing preprocedural MBP (with NaP and/or PEG) to no bowel prep have shown no difference in rates of postoperative wound infection, abscess formation, or anastomotic leakage. In fact, some of the studies have shown an increased rate of anastomotic complications in the MBP group. It has also been repeatedly demonstrated that the frequency of anastomotic leakage is no higher in patients with penetrating trauma to the bowel who do not undergo MBP due to the urgency of surgery than it is for scheduled patients who do undergo preoperative MBP. The same comment holds true for patients with obstructive lesions in the colon, in whom preoperative MBP is contraindicated. As more such data become available, the use of the MBP may evolve into an "as indicated" procedure. However, when a "clean view" of the colon is necessary, such as for colonoscopy (whether intraoperative or in the gastrointestinal [GI] laboratory), MBP will likely remain a standard practice.

TAKE HOME POINTS P.172

- Remember that bowel preps are not used just for abdominal surgeries; ask all patients having surgery on internal viscera if they have done a prep.
- The polyethylene glycol bowel preps have trade names such as Golytely and Nulytely.
- The sodium phosphate bowel preps have trade names such as Fleets Phospha-Soda.
- Polyethylene preps can cause significant upper GI distress and vomiting; look for upper GI loss of potassium and disruptions in acid/base status and consider preinduction antiemetics and cricoid pressure.
- Sodium phosphate preps can cause significant electrolyte depletion from lower GI losses; consider checking preinduction potassium levels, especially if the patient is also on a diuretic or digoxin.
- Patients who have had a NaP prep may also need extra intravenous fluid replacement at the time of induction.

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Beware the Antibiotic Bowel Prep

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Bowel preparation for elective colorectal surgery typically consists of mechanical bowel prep, oral antibiotics, and intravenous antibiotics. Mechanical bowel prep is discussed elsewhere; this chapter focuses on perioperative antibiotics for colorectal surgery. Other surgeries for which antibiotic bowel preparation is commonly used include abdominal and pelvic operations involving the aorta, kidneys, bladder, and reproductive structures.

Infectious complications are the leading cause of morbidity and mortality in colorectal surgery. The most common infectious complications include wound infection, intra-abdominal or pelvic abscess, and anastamotic leaks. Offending micro-organisms are typically endogenous colonic flora and include *Bacteroides fragilis*, *Clostridia*, *Escherichia coli*, *Klebsiella*, *Proteus*, and *Pseudomonas.* The logical goals of perioperative antibiotics include decreasing fecal load and bacterial count in the colonic lumen and achieving therapeutic tissue levels of antibiotics in the event that contamination occurs.

Oral antibiotics act on colonic flora intralumenally to decrease bacterial load when the lumen is opened, promoting less contamination and fewer infectious complications. Although there are many conflicting studies on the utility of oral antibiotics in this setting, >70% of surgeons use them as part of their bowel preparation. A combination of neomycin plus erythromycin or metronidazole are the most commonly used agents. The oral antibiotics used are typically given the day before surgery. At the senior author's hospital, the regimen includes three doses of erythromycin of 1 g each on the day before surgery. Although some oral antibiotics are not well absorbed from the gut, there is significant systemic absorption of erythromycin following oral administration.

One underappreciated problem that may arise with this protocol is the occurrence of unanticipated drug-drug interactions. Through two distinct mechanisms, erythromycin is likely to raise the blood levels of a large number of other drugs. First, erythromycin is a potent inhibitor of both intestinal and hepatic forms of the most important of the cytochrome P450 enzymes, CYP3A4. As such, it inhibits the metabolism of the broad array of CYP3A4 substrates, leading to increases in the blood levels of such drugs. Significant examples of CYP3A4 substrates include alprazolam, carbamazepine, cyclosporine, methadone, and quinidine, to name just a few. Second, erythromycin also inhibits the functioning of the extruding P-glycoprotein transporter. P.174
Thus, inhibition of this transporter also leads to increased levels of P-glycoprotein substrates, such as carbamazepine, cyclosporine, digoxin, morphine, phenytoin, tacrolimus, and tricyclic antidepressants. Drug toxicities are a clear concern when a patient is taking any of these drugs and they also receive a bowel preparation that includes erythromycin. Additionally, inferential reasoning also implicates metronidazole as a P-glycoprotein inhibitor, so the same toxicity concerns would be present when P-glycoprotein substrates are co-administered with metronidazole.

The goal of intravenous antibiotics is to provide therapeutic tissue levels at the time of exposure if contamination occurs. Agents traditionally used have covered colonic microflora. In the past, the triad of an aminoglycoside, a penicillin, and clindamycin or metronidazole has been used to cover Gram-negative bacteria, enterococci, and anaerobes, respectively. However, with the development of cephalosporins, aminoglycosides are no longer commonly used, avoiding the potential toxicity of this class of antibiotics. The majority of surgeons administer a second-generation cephalosporin that is active against both aerobic and anaerobic colonic bacteria within 1 hour of skin incision and continue it for up to two doses for the first 24 hours postoperatively. Continuing prophylaxis beyond the first 24 hours postoperatively has not been shown to be of any additional benefit and increases the risk of side effects, bacterial resistance, and *Clostridium difficile* colitis.

TAKE HOME POINTS

- There are many conflicting studies on the utility of oral antibiotics in this setting, however, >70% of surgeons use them as part of their bowel preparation.
- Erythromycin is a potent inhibitor of both intestinal and hepatic forms of the most important of the cytochrome P450 enzymes, CYP3A4.
- It will inhibit the metabolism of CYP3A4 substrates, such as alprazolam, carbamazepine, cyclosporine, methadone, and quinidine, leading to increases in the blood levels of such drugs.
- Erythromycin also inhibits the functioning of the extruding P-glycoprotein transporter. Thus, inhibition of this transporter also leads to increased levels of P-glycoprotein substrates, such as carbamazepine, cyclosporine, digoxin, morphine, phenytoin, tacrolimus, and tricyclic antidepressants.
- Cephalosporins do not inhibit or induce the metabolism of other drugs.

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Be Aware of the Drugs that Require Slow Intravenous Administration

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The speed at which intravenous (IV) medications can be administered is often overlooked and can have devastating consequences. The drug package inserts contain information on administration, but these are frequently unavailable to anesthesiologists and the information included is not always appropriate for anesthetized patients. We often rely on pharmacists to relay important information regarding the IV administration of particular medications. As anesthesia providers, however, we frequently prepare medications for administration without pharmacy intervention. The purpose of this chapter is to review common medications that need to be administered slowly or that have other special considerations. The chapter is not intended to be used for dosing of medications. This is by no means an all-inclusive list; our intention is to cover commonly used medications that are at high risk for being administered inappropriately.

The following medications *must* be given slowly when they are administered intravenously: Phenytoin, protamine, vancomycin, potassium chloride, clindamycin, thymoglobulin, furosemide, gentamicin, and oxytocin.

PHENYTOIN (DILANTIN)

Phenytoin is a neurologic and cardiac depressant and is one of the most dangerous medications we administer intraoperatively. For status epilepticus, the maximum rate of IV administration is 50 mg/min (for children, 1 to 3 mg/kg/min up to a max of 50 mg/min). Because we generally give the medication prophylactically in the operating room, it is recommended to administer phenytoin much more slowly in anesthetized patients (e.g., max. 10 to 20 mg/min). Elderly patients and patients with cardiovascular disease should also receive the medication more slowly. If phenytoin is given rapidly IV, asystole and cardiovascular collapse is possible, but more commonly hypotension, bradycardia, and cardiac dysrhythmias are seen. These effects may be related to the diluent (propylene glycol) and may be minimized by using fosphenytoin. The dose must also be adjusted in those with hypoalbuminemia and liver disease. It is advised that the medication be diluted in *normal saline* to a final concentration of 1 to 10 mg/mL and be administered with an infusion pump. Extravasation of this medication can cause severe soft tissue injury, and it is therefore recommended to inject phenytoin into a large vein through a large-gauge IV catheter. Be sure to flush the line before and after administration with normal saline. This medication may be piggybacked, but there is a high potential for precipitation in the presence of other medications, so a 0.22-µm filter should be used. P.177

PROTAMINE

Protamine is administered frequently in cardiac and vascular anesthesia to reverse the anticoagulant effects of heparin. Administer protamine slowly, no faster than 50 mg over 10 minutes. Severe hypotension, bradycardia, pulmonary hypertension, and an anaphylactic reaction can result if protamine is given by IV push. Patients at increased risk of severe reactions include diabetics taking insulin, patients with fish sensitivities, men who have undergone vasectomy, and patients who have been previously exposed to protamine. Dilution of the medication is not necessary.

VANCOMYCIN

Administration of vancomycin should be no faster than 10 mg/min and should be administered with the use of an infusion pump. “Red man” syndrome (erythematous rash on face and body) may occur, and if so, the infusion rate should be reduced. Rapid IV infusion has been reported to cause hypotension and, rarely, cardiac arrest. Vancomycin may cause soft tissue injury if it extravasates. When possible, a large-gauge IV catheter or central line should be used for administration.

POTASSIUM

Potassium may precipitate cardiac arrhythmias if serum concentrations increase too rapidly. In general, the maximum speed of administration is 40 mEq/h. The patient should have electrocardiogram (EKG) monitoring in place before administration. This medication causes venous irritation, so infusion via large IV catheters is preferred. If it is not prepackaged, potassium must be diluted.

CLINDAMYCIN

Clindamycin should be diluted before administration to a concentration of no more than 18 mg/mL. Do not “push” clindamycin intravenously, because it can cause profound hypotension. Infuse over 10 to 60 minutes at a rate no greater than 30 mg/min. This medication can cause thrombophlebitis at the injection site.

THYMOGLOBULIN

Thymoglobulin (lymphocyte immune globulin or antithymocyte globulin rabbit) is often used in renal transplant patients at increased risk for rejection. Administration is through a 0.22-µm filter and is infused over at least 6 hours for the first infusion to decrease the development of fever and/or chills. Hypertension is a known side effect, but hypotension may be noted intraoperatively. Administration through a central line or other high-flow vein is preferred to decrease the risk of thrombosis or thrombophlebitis. Patients may be premedicated with corticosteroids, Tylenol, and/or antihistamines to reduce side effects. P.178

FUROSEMIDE (LASIX)

Lasix should also be administered slowly over 1 to 2 minutes. Administer no faster than 4 mg/min if large doses are to be given. There have been reports of acute hypotension and sudden cardiac arrest after IV administration. Ototoxicity has been associated with rapid IV infusion. Renal impairment, concurrent administration of other ototoxic medications, and excessive doses increase the risk of ototoxicity.

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GENTAMICIN

Gentamicin has a small therapeutic window, which can make dosing challenging. Dosing adjustments in situations of renal impairment are particularly important. Aminoglycosides in general are associated with both nephrotoxicity and ototoxicity. It is thought that toxicity is related to the dose given and the duration of therapy. There have been reports of hypotension after administration of an aminoglycoside, and it is therefore recommended that gentamicin be diluted and administered slowly. Gentamicin can potentiate neuromuscular blockade.

OXYTOCIN (PITOCIN)

There are multiple uses of Pitocin for obstetric patients. Infusions of Pitocin for induction of labor are managed by the obstetric staff, but anesthesia staff are often asked to administer this medication during caesarian sections and in hemorrhagic emergencies. Dilute Pitocin in either a 0.9% normal saline or lactated Ringer's solution and administer slowly. Rapid administration may cause arrhythmias and/or hypertension in the mother. Uterine hyperactivity as severe as uterine hypertonicity, tetanic contraction, and uterine rupture is also possible. Be aware that Pitocin has an antidiuretic effect that may result in water intoxication, with convulsions, coma, or death.

OTHER PEARLS TO AVOID COMMON ERRORS INVOLVING IV ADMINISTRATION IN THE OPERATING ROOM

- The following IV-administered products must be given through a filter: *All* blood products—packed red blood cells, platelets, fresh frozen plasma, cryoprecipitate, etc.
- Pressors that require dilution for administration include the following (with recommended final concentration): Phenylephrine (100 mcg/mL), ephedrine (5 mg/mL), and epinephrine (non-cardiac arrest situation, 10 and 100 mcg/mL). P.179
- Label all medications carefully!

TAKE HOME POINTS

- One of the most important tasks for the anesthesia provider is to give dangerous drugs in a precision—this includes not only giving the correct dose of a drug, but giving it at an appropriate rate
- Rapid administration of certain drugs can have fatal consequences
- Do not accept information from the neurosurgeons as to the appropriate rate of phenytoin infusion—know the limiting rate yourself and personally insure it is not being exceeded. If the surgeons insist they cannot continue on with the case until the infusion is complete, then advise them they may have to adjust their expectations about how rapidly the case will proceed, but do not speed up the rate of the infusion. The editors are personally aware of several cases with fatal outcomes due to excessively rapid administration of phenytoin. In one case, it effectively ended the career of the anesthesia provider.
- Protamine is used in the heart rooms, where it is commonly given via central access and invasive monitoring allows for close inspection of the hemodynamic effects. Somewhat more dangerous is a request by the surgeons for “50 of protamine” in a peripheral vascular case at the end of surgery, when the high-vigilance times of the case (placement and removal of the crossclamp are over). The request is typically made because the patient “is oozy” and so that the surgeons can close. Often the anesthesia provider is requested to give it via a peripheral IV.
- The key to controlling the rate of potassium chloride infusion is to have a policy to always use an infusion pump (NOT just an infusion set with a piece of tape over the Cair clamp).

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Remember that Smoking Cessation and Re-Initiation are Important Variables in the Perioperative Period

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It has been estimated that at least one third (and probably closer to one half) of all patients presenting for surgery are smokers, which predisposes them to significant postoperative respiratory complications. As such, patients are usually counseled to stop smoking before surgery and may even be prescribed a nicotine patch as therapy.

Certainly, a few patients achieve the recommended goal of smoking cessation 4 to 6 weeks before surgery, but by far the greater number do not manage to stop smoking until the day before or even the morning of surgery ("No, I don't smoke, I quit two hours ago"). Acute smoking cessation has the paradoxic action of transiently increasing airway secretions due to improved mucociliary transport, but the balance of effects on the pulmonary and cardiovascular systems is beneficial. These include a decrease in blood carbon monoxide levels as well as a rightward shift in the oxygen-hemoglobin dissociation curve and an increase in tissue oxygenation. However, anesthesia providers must be aware of the unintended consequences of smoking cessation —balanced against the obvious benefits is the fact that the polycyclic aromatic hydrocarbons in smoked tobacco act as strong inducers of the P450 1A2 enzyme, moderate inducers of P450 2E1, and inducers of some not yet well characterized phase II enzymes. Abrupt cessation of smoking may cause alterations in the blood levels and/or end organ effects of various medications, possibly in ways that could produce inconvenience or even harm to patients.

Only smoked tobacco acts as an enzymatic inducer. Rather, it is the polycyclic aromatic hydrocarbons that must be smoked in order to induce metabolic enzymes. The P450 1A2 enzyme catalyzes the metabolism of a number of substrates that are of clinical relevance in the perioperative period. These include cyclobenzaprine, flecainide, propranolol, theophylline, and R-warfarin (Table 43.1). In the days following smoking cessation, the decreased availability of P450 1A2 decreases the metabolic clearance of these 1A2 substrates, leading to significantly increased blood levels. These increases in substrate levels can produce frank toxicity (such as with theophylline) and otherwise complicate perioperative management. There have been cases of increased bleeding attributable to smoking cessation in patients who take warfarin. The effects of smoking cessation may be mimicked by the abrupt introduction of inhibitors of P450 1A2, such as caffeine, cimetidine, ethinylestradiol, and many of the fluoroquinolones (Table 43.1). Other inducers of 1A2 (besides tobacco smoking) include carbamazepine, rifampin, and modafinil (Table 43.1).

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TABLE 43.1 DRUGS THAT INTERACT WITH CYTOCHROME P450 1A2

1A2 SUBSTRATES	1A2 INHIBITORS	1A2 INDUCERS
Caffeine		
Clozapine	Caffeine	Carbamazepine
Cyclobenzaprine	Cimetidine	Modafinil
Flecainide	Ciprofloxacin	Rifampin
Fluvoxamine	Ethinylestradiol	Tobacco (smoked)
Haloperidol	Fluvoxamine	
Mexiletine	Grapefruit juice	
Olanzapine	Mexiletine	
Propranolol	Norfloxacin	
Tacrine	Ofloxacin	
Theophylline	Ticlopidine	

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(R-)Warfarin

Although smoked tobacco produces a less potent induction effect at P450 2E1 than at 1A2, smoking cessation can still lead to decreased metabolism and increased blood levels of notable 2E1 substrates, including acetaminophen, ethanol, and the “flurane” inhalational anesthetics.

Awareness of the pharmacokinetic issues arising from abrupt smoking cessation can help the careful clinician avoid drug toxicity that might otherwise develop in the perioperative period.

TAKE HOME POINTS

- For the majority of smokers who present for surgery, the perioperative period is characterized by abrupt smoking cessation.
- The polycyclic aromatic hydrocarbons in smoked tobacco act as strong inducers of the P450 1A2 enzyme, moderate inducers of P450 2E1, and inducers of some not yet well characterized phase II enzymes.
- Abrupt cessation of smoking may cause alterations in the blood levels and/or end organ effects of various medications, especially substrates of P450 1A2, such as cyclobenzaprine, flecainide, propranolol, theophylline, and R-warfarin.
- Re-initiation of smoking after discharge from the hospital will re-induce P450 1A2, leading to increased clearance of 1A2 substrates and a corresponding decrease in blood levels. In the days following re-initiation of smoking, dosages of 1A2 substrates that were decreased following smoking cessation will need to be increased to their original levels.

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Stop Metformin before Elective Surgery or Intravascular Contrast Dye Study to Decrease the Risk of Lactic Acidosis

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Michael J. Moritz MD

Metformin is an insulin-sensitizing agent that has been in use for more than 50 years. Chemically, metformin is a biguanide. Its main action is to increase peripheral glucose utilization and decrease hepatic glucose release. In overweight patients with Type 2 diabetes, metformin has been shown to lower cardiovascular and diabetes-related deaths and has been considered the oral hypoglycemic agent of choice. It is also indicated to treat the insulin resistance of polycystic ovary syndrome. Metformin is excreted renally and has a half-life of 1.5 to 5 hours. Metformin is available as the branded drugs Glucophage, Glucophage XR tablets, Fortamet tablets, and Riomet liquid. It is also a component of three combination drugs: Glucovance, Metaglip, and Avandamet.

Lactic acidosis is a high-anion-gap acidosis. Lactate is produced by anaerobic glycolysis, and the development of lactic acidosis requires over-production, slowed breakdown, or both. At high blood levels, metformin produces severe refractory lactic acidosis by the uncoupling of oxidative glycolysis, thus driving cellular mechanisms toward anaerobic metabolism.

Lactic acidosis associated with metformin is rare but has a mortality of 50%. The risk with chronic use in the outpatient setting ranges from 1/1,000 to 1/30,000 patient-years. Patients in the acute care setting, however, incur an additional risk because they are exposed to situations, such as transient or ongoing renal insufficiency, that may lead to drug accumulation and increases in drug levels. As such, surgery or exposure to iodinated radiologic contrast dye (seen with angiography and intravenous contrast for computed tomography scan) must prompt discontinuation of metformin. Other clinical situations that may precipitate lactic acidosis in patients taking metformin include advanced age, dehydration, liver disease, congestive heart failure, chronic alcohol abuse or binge drinking of alcohol, shock with tissue hypoperfusion (septic, cardiogenic, etc.), and hypoxia.

At present, there are no outcome studies pertaining to the administration of metformin in the perioperative period. Generally, endocrinology consultants recommend that metformin be withheld on the day of surgery
or contrast dye study only or at most for 24 hours. This represents a change in current opinion from as recently as 5 years ago and is based on both pharmacodynamic studies and the very important competing issue of improved perioperative outcomes due to tighter perioperative glucose control. For both surgical and radiology patients, the drug should be held for at least 48 hours and restarted only after renal function is known to be normal. Additionally, surgical patients should have resumption of adequate oral caloric intake.

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Remember that Administration of Angiotensin System Inhibitors within 10 Hours Before Surgery Is A Significant Independent Risk Factor for Hypotension in the Postinduction Period*

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Juraj Sprung MD, PhD

In general, the decision to withdraw medications with cardiovascular effects before surgery depends on the risk balance between the deleterious interaction of the drug with anesthetics and possible morbidity resulting from hemodynamic effects that may occur in the absence of these medications.

Drugs that affect the renin-angiotensin system, such as angiotensinconverting enzyme inhibitors (ACEIs) and angiotensin II receptor subtype 1 antagonists (ARAs), are often used in the management of hypertension, congestive heart failure, and chronic renal failure. These drugs may interfere with the regulation of arterial blood pressure by several different mechanisms, including sympathetic blockade, decrease in responsiveness to α_1-adrenergic agonists, impaired degradation of bradykinin (which promotes vasodilation), and inhibition of the receptor binding of angiotensin II.

Current practice guidelines recommend the perioperative continuation of therapies that have potential for myocardial protection, such as β-adrenoceptor-blocking drugs and calcium-channel blockers. Other drugs that are generally recommended to be continued are those with a potential for rebound hypertension with withdrawal, such as α_2-adrenoceptor agonists.

In the case of perioperative ACEI/ARA therapy, clear guidelines are not available. Severe intraoperative hypotension has been observed in patients treated with preoperative ACEIs and ARAs, and adequate perioperative management of antihypertensive therapy in patients receiving angiotensinsystem blockers has been debated.

Preoperative withdrawal of ACEI/ARA therapy has been proposed on the basis of several reports of intraoperative hypotension, which has been reported to be refractory to common measures such as fluid boluses or intravenous ephedrine and phenylephrine. Several small, controlled, randomized studies showed an increased frequency of hypotension after the induction of anesthesia when ACEI/ARA therapy was continued through the morning of surgery compared with discontinuation of therapy the night before surgery. P.189

Angiotensin-system inhibition has also been shown to increase vasopressor requirements after cardiopulmonary bypass. Vasopressin or vasopressin analogs such as terlipressin have been advocated in patients on ACEI/ARA therapy to treat hypotension that is refractory to other measures.

Despite reports of intraoperative hypotension, some authors have recommended continued perioperative ACEI/ARA therapy for its potentially beneficial effects. Possible benefits are a decrease in ischemia-related myocardial cell damage in cardiac surgery and improved renal function in patients undergoing cardiopulmonary bypass, as measured by an improvement in urinary excretion of sodium.

In a recent large clinical trial by Comfere et al., the timing of the last ACEI or ARA dose was a major determinant of the frequency of postinduction hypotension. During the first 30 minutes after anesthetic induction, moderate hypotension (systolic blood pressure <85 mm Hg) was more frequent in patients whose most recent ACEI/ARA dose was taken within 10 hours (60%) compared with those whose last dose was taken more than 10 hours before induction (46%).

These findings can be explained by the elimination half-lives of ACEI/ARA drugs (Table 46.1). Specifically, a 10-hour interval between the last dose and anesthetic induction corresponds to the average half-life of an ACEI or ARA and appears to be sufficient to decrease the incidence of hypotensive episodes after anesthetic induction. In humans, most ACEIs are eliminated renally via glomerular filtration or tubular secretion. Renal insufficiency may have a substantial effect on the half-life of certain ACEIs, and the altered pharmacokinetics of ACEIs in chronic renal failure may be a potential hazard; thus, abstinence for longer than 10 hours before surgery may be considered in patients with renal insufficiency. P.190

TABLE 46.1 ELIMINATION HALF-LIVES OF ANGIOTENSIN-CONVERTING ENZYME INHIBITORS (ACEIS) AND ANGIOTENSIN-RECEPTOR ANTAGONISTS (ARAS)

DRUG	ACTIVE METABOLITE	HALF-LIFE, H
ACEI		
Benazepril	Benzaprilat	≈ 11
Enalapril	Enalaprilat	≈ 11
Lisinopril		≈ 12
Quinapril	Quinaprilat	≈ 2

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	Fosinopril	Fosinoprilat	≈ 12
	Ramipril	Ramiprilat	Range, 9-18
ARA			
	Candesartan		Range, 5.1-10.5
	Losartan	E-3174	Range, 6-9
	Valsartan		Range, 6-9

Source: Modified from Comfere T, Sprung J, Kumar MM, et al. Angiotensin system inhibitors in a general surgical population. *Anesth Analg.* 2005;100:636-644. Used with permission.

In the study by Comfere et al., there was no clinically significant effect of timing of the last ACEI/ARA dose on preanesthetic arterial blood pressures, and both study groups had moderate preoperative hypertension regardless of when the last ACEI/ARA dose was taken.

TAKE HOME POINTS

- In patients receiving chronic ACEI/ARA therapy who receive general anesthesia, the administration of these drugs within 10 hours before anesthesia is a significant independent risk factor for the development of moderate hypotension within 30 minutes after induction in a common clinical setting.
- Anecdotally, hypotension in a setting of ACEI/ARA premedication was described in several case reports as being refractory to the usual therapeutic interventions. However, in a large clinical trial, all episodes of hypotension responded to conventional therapy, such as fluid boluses and intravenous ephedrine or phenylephrine, and thus seemed to be of little clinical consequence.
- Preoperative withholding of ACEI/ARA therapy should be considered for patients who may be especially prone to hypotension-induced complications (e.g., patients with severe aortic stenosis or cerebrovascular disease).
- No trial to date has demonstrated any deleterious effects of a short interruption of ACEI/ARA therapy in the immediate perioperative period.

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Editors: **Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.**
Title: ***Avoiding Common Anesthesia Errors, 1st Edition***

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Be Aware that Many Drugs Commonly Given in the Perioperative Period Have Significant P-Glycoprotein Transport Pump Activity

Neil B. Sandson MD

In today's medical climate, the anesthesiologist must be prepared to function as a specialist in perioperative medicine. This includes (among many other tasks) a thorough knowledge of the drugs commonly given not just in the operating room (OR), but in the perioperative period, including routes of administration, dosing, and the specifics of drug elimination. Although discussions of pharmacokinetic drug handling most often focus on issues of metabolism and excretion, the emerging importance of the P-glycoprotein transporter highlights the relevance of absorption and distribution as well. The P-glycoprotein transporter is an ATP-dependent pump that extrudes various compounds (substrates) from protected intracellular domains. For instance, P-glycoprotein lines the lumen of the small intestine. When a P-glycoprotein substrate is absorbed across its concentration gradient into the cytosol of an enterocyte, this transporter acts to then extrude that drug out of the enterocyte and back into the gut lumen. Insofar as this process moves the drug against its concentration gradient, it requires ATP. P-glycoprotein also lines the blood-brain barrier, acting as one of the constituents that minimizes access of xenobiotic substances to the central nervous system. In a manner analogous to the small intestine, P-glycoprotein substrates diffuse from the vasculature into the cytosol of blood-brain barrier capillary endothelial cells, but the pump then acts to extrude them back into the vasculature. Significant P-glycoprotein substrates include carbamazepine, corticosteroids, cyclosporine, dexamethasone, digoxin, morphine, ondansetron, phenytoin, risperidone, tacrolimus, and tricyclic antidepressants. Dose-finding studies to determine effective doses of these and other P-glycoprotein substrates have already incorporated these effects.

Many compounds act to inhibit the action of the P-glycoprotein. Thus, P-glycoprotein inhibitors decrease the efflux of P-glycoprotein substrates from enterocytes into the gut lumen. The net effect of this process is that in the presence of a P-glycoprotein inhibitor, there will be greater systemic absorption of substrates at given doses. Similarly, at given intravascular

concentrations of P-glycoprotein substrates, there will be more influx past the blood-brain barrier and into the central nervous system. In these instances, drug toxicity may be a concern. Significant P-glycoprotein inhibitors include atorvastatin, erythromycin, fluoxetine, lidocaine, lovastatin, midazolam, omeprazole, propranolol, simvastatin, tricyclic antidepressants, and verapamil. For instance, there are reports that co-administration of digoxin with a number of P-glycoprotein inhibitors, including atorvastatin, fluoxetine, and verapamil, among others, will reliably raise serum digoxin levels.

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Asmaller number of compounds act as inducers of the production of the P-glycoprotein transporter. When an inducer causes increased presence of the transporter, this leads to enhancement of the pump's action on substrates. Thus, in the presence of P-glycoprotein inducers there will be a decrease in substrate blood levels and less influx from the systemic vasculature into the central nervous system. In these instances, loss of drug efficacy may be a concern. Significant P-glycoprotein inducers include aspirin, rifampin, St. John's wort, and trazodone. It has been established that co-administration of rifampin and/or St. John's wort with digoxin will decrease serum digoxin levels through this mechanism.

These lists include a fraction of the known P-glycoprotein substrates, inhibitors, and inducers, and each month a growing number of drugs are found to have a functional relationship with this transporter system. When a patient is taking a P-glycoprotein substrate with a narrow therapeutic index, such as digoxin or tacrolimus, it is prudent to attend to the presence, addition, or deletion of P-glycoprotein inhibitors or inducers from the patient's regimen. Alist of known P-glycoprotein substrates, inhibitors, and inducers may be found at www.mhc.com/Cytochromes.

TAKE HOME POINTS

- Today's anesthesia provider must expand her knowledge base beyond that of the basic anesthetic drugs.
- The P-glycoprotein transporter is an ATP-dependent pump that is relevant in the absorption and distribution of a number of important drugs in the perioperative period.
- There are both inducers and inhibitors of the P-glycoprotein transport pump.
- Be aware of the co-administration of both inhibitors and inducers of the P-glycoprotein pump when the patient is on a drug with a narrow therapeutic window, such as digoxin.

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Acknowledge the Complex Medical and Legal Issues Surrounding Off-Label Drug Use

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Imagine this scene: Your healthy, 5-year-old patient has just emerged from anesthesia after a routine, 30-minute case for PE tube placement. She is ventilating spontaneously and her vital signs are stable. As you leave the operating room (OR), she becomes agitated, screaming and thrashing on the stretcher and inconsolable. Pondering your options for treating her emergence delirium, you acknowledge that she does not have an intravenous (IV) line. Quickly, you administer 2 mcg/kg of intranasal clonidine. She becomes calm and quiet and you are thankful she is no longer a risk to herself. Her parents are reassured, the postanesthesia care unit (PACU) nurses are able to carefor their new patient efficiently, and you move on to your next case without interruption. This was a successful and safe intervention, wasn't it? Perhaps so, but intranasal clonidine use is not described in the U.S. Food and Drug Administration (FDA) package insert.

The practice of anesthesiology reflects a mastery of pharmacology. Not only is the proper indication for and administration of a particular drug important to the anesthesia provider, but also the ability to co-administer agents or even to use them in novel ways. The most responsible and safest approach to this practice is to acknowledge drug labels created by the FDAand then strive to apply those guidelines to clinical practice. If a drug is used beyond those conditions described on its label, it is considered off-label.

The FDAregulates many aspects of drug development, research, safety, marketing, and labeling. The drug label is a summary of data collected by clinical trials. Therefore, in order to achieve approval for specific uses of a drug there must be evidence-based data to support it. Obviously, we cannot anticipate every detail of our patient population. So, based on medical literature, drugs are frequently used beyond the specific guidelines listed in the drug label for dose, route, indication, regimen, or patient population. How common is this practice? Sources site up to 23% of prescriptions being written for off-label use. This trend is not necessarily harmful, but, rather, reflects a lack of well-controlled study data held by the FDA(the study has not been done or there is no financial incentive to develop new indications,
e.g., isobaric spinal bupivacaine). Further, if there is a known adverse effect of an off-label drug use, it will be listed in the Contraindications, Warnings, or Precautions section of the label. P.196

According to the FDA, "once a product has been approved for marketing, a physician may prescribe it for uses in treatment regimens or patient populations that are not included in approved labeling. Valid new uses for drugs already on the market are often first discovered through serendipitous observations and therapeutic interventions." It is important to realize that the FDAdoes not seek to restrict a physician's decision to use a drug off-label and the *Physicians' Desk Reference* (PDR) also acknowledges this use. Interestingly, the publisher of the PDR, Medical Economics, also publishes an "off-label treatment guide." So, there is acknowledgement that this use has an important place in patient care. Currently in the United States, the physician has no ethical or legal obligation to educate the patient on FDAregulatory status when a drug is used. However, it is the physician's responsibility to be aware of details in the drug label, including pharmacology, toxicology, and chemistry (buffers, preservatives, antioxidants, and incompatibility with other drugs).

Anesthesiologists must also consider the unfortunate litigious climate of medicine. Drug manufacturers, the FDA, and the PDR all recognize the clinically observed safe and effective uses of a variety of medicines although they may not be supported by well-controlled, evidence-based data. However, courts and judges may have the ultimate authority over a physician's ability to practice off-label pharmacology and the liability for doing so (e.g., gabapentin and suicide risk). To be found liable for off-label drug use, the patient must prove that the anesthesiologist committed a breach of duty of care, that the breach resulted from a failure to reach the standard of care required by the law, and that the breach of duty resulted in injury. Based on the importance and well-recognized practice of off-label drug use per a physician's discretion, for a drug manufacturer to assert that it warned against unapproved uses is disingenuous. However, harm incurred to a patient related to an off-label use of a medication may ultimately be the prescriber's responsibility.

In general, there is a growing feeling that off-label drug use is the newly discovered gold mine of malpractice attorneys. The "learned intermediary" doctrine used by pharmaceutical manufacturers to protect them from liability asserts that they have provided the physician with warnings about a drug via the drug label. Hence, the physician now has the duty to warn the patient about that drug's hazards. Of course, this issue is really very complex. Ultimately, the FDApackage insert must list the side effect of which the patient complains and there must be a way to prove that the patient was not adequately warned or was misled by the physician about those side effects. And,
in countries such as England, cases are being decided in favor of patients who claim that they were given inadequate information about a drug and, therefore, did not give informed consent. In situations in which there are alternative, FDA-approved medications to a drug being used off-label and purportedly resulting in harm, it might be difficult to convince a jury that the decision of the physician was sound. Thus, in England, physicians must now obtain informed consent for prescribed medicines, especially those used off-label. This includes risks and benefits, information about "approved" alternatives, education about the meaning of off-label drug use, and the lack of testing existing for that use. In England, to use a drug off-label is then justified only if there is no approved alternative and not because the use is more convenient or less costly. P.197

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TABLE 48.1 EIGHT DRUGS AND THEIR APPROVED AND OFF-LABEL USES

	PROPOFOL	FENTANYL	BUPIVACAINE	CLONIDINE	KETAMINE	LORAZEPAM	SUFENTANIL	NEURONTIN
Description	Propofol is a hindered phenol compound with IV general anesthetic properties. The drug is unrelated to any of the currently used barbiturate, opioid, benzodiazepine, arylcyclohexylamine, or imidazole IV anesthetic agents.	Binds stereospecific receptors at multiple CNS sites; increases pain threshold; alters pain perception; inhibits ascending pain pathways.	Blocks both the initiation and conduction of nerve impulses by decreasing the neuronal membrane's permeability to sodium ions, which results in inhibition of depolarization with resultant blockade of conduction.	Stimulates α_2-adrenoreceptors in brainstem to activate inhibitory neuron-decreased sympathetic outflow from CNS; decreased PVR, renal vascular resistance, HR, BP. Relief of epidural pain at spinal presynaptic, postjunctional α_2-adrenoreceptors by preventing transmission of pain signals.	Blockade of neuronal postsynaptic NMDAreceptor. Direct action on cortex and limbic system. Stimulates release of endogenous catecholamines that maintain BP and HR. Reduces polysynaptic spinal reflexes.	Benzodiazepine—Binds to GABA receptors, increasing permeability to Cl^- ions and enhancing inhibitory effects on neuronal excitation.	Binds opioid receptors in CNS to open K^+ channels and inhibiting Ca^{2+} channels; increases pain threshold; alters pain perception; inhibits ascending pain pathways; short-acting narcotic.	Mechanism of action unknown; similar properties to other anticonvulsants; structurally similar to GABA.

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Approved uses	Induction of anesthesia for inpatient or outpatient surgery in patients aged ≥3 y; maintenance of anesthesia for inpatient or outpatient surgery in patients aged >2 mo; in adults, for the induction and maintenance of monitored anesthesia care sedation during diagnostic procedures; treatment of agitation in intubated, mechanically ventilated ICU patients.	Injection: Sedation, relief of pain, preoperative medication, adjunct to general or regional anesthesia. Transdermal: Management of moderate-to-severe chronic pain. Transmucosal (Actiq): Management of breakthrough cancer pain. Sedation for minor procedures/analgesia: For children aged 1-12 y, for children aged >12 y, refer to adult dosing. Continuous sedation/analgesia: For children aged 1-12 y. Chronic pain management: For children aged ≥2 y (opioid-tolerant patients). Transdermal: Refer to adult dosing.	Local anesthesia: Infiltration: 0.25% infiltrated locally; maximum: 175 mg. Caudal block (preservative-free): 15-30 mL of 0.25% or 0.5%. Epidural block (other than caudal block; preservative-free): Peripheral nerve block: 5 mL of 0.25% or 0.5%; max.: 400 mg/d Sympathetic nerve block. Retrobulbar anesthesia. Spinal anesthesia: Preservative-free solution of 0.75% bupivacaine in 8.25% dextrose.	Acute hypertension, hypertension. Pain management: Epidural infusion: Starting dose: 30 mcg/h; titrate as required for relief of pain or presence of side effects; minimal experience with doses >40 mcg/h; should be considered an adjunct to intraspinal opiate therapy. Warning against using as epidural for perioperative, obstetric, or postpartum pain because of possible bradycardia or hypotension. ADHD in children	Induction and maintenance of general anesthesia; sedation; analgesia.	Nausea/vomiting w/chemotherapy; status epilepticus; anxiety; preanesthesia for amnesic effect.	10-50 mcg/kg for induction w/10-50 mcg PRN. Pediatrics: ages 2-12 y, 10-25 mcg/kg induction; 1-2 mcg/kg maintenance (total dose).	Adjunct for partial seizures with and without secondary generalized seizures in patients aged >12 y. Adjunct therapy for partial seizures in pediatric patients aged 3-12 y. Management of postherpetic neuralgia in adults.
Off-Label Uses	Postoperative antiemetic; refractory delirium tremens (case reports); conscious sedation. Infusions faster than 40 mg q 10 s or 20 mg q 10 s for elderly, debilitated, ASAIII or IV patients.	Epidural or intrathecal. Children aged <2 y.	Isobaric intrathecal. Pediatric patients aged <12 y.	Pediatric emergence delirium. Heroin or nicotine withdrawal; severe pain; dysmenorrhea; vasomotor symptoms associated with menopause; ethanol dependence; prophylaxis of migraines; glaucoma; diabetes-associated diarrhea; impulse-control disorder; ADHD; clozapine-induced sialorrhea.	Obstetric patients. Pediatric patients aged <16 y.	Agitation-IV. Insomia: short-term therapy. Ethanol detoxification. Psychogenic catatonia. Partial complex seizures.	Intrathecal.	Chronic pain. Bipolar disorder. Social phobia. Postoperative pain.

IV, intravenous; CNS, central nervous system; PVR, pulmonary vascular resistance; HR, heart rate; BP, blood pressure; ICU, intensive care unit; ADHD, attention-deficit/hyperactivity disorder.

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Of note, it is illegal for drug manufacturers to market off-label uses of their products. An interesting example of this involves Neurontin (gabapentin), originally manufactured by Parke-Davis (now controlled by Pfizer). This drug underwent scrutiny beginning in 2005 when concerns were raised about increased suicide rates among patients taking the drug not for seizures, but for other medical conditions including chronic pain. The FDA has asked Pfizer to analyze its study data for evidence of suicide risk. Attorneys are requesting that the FDA attach a black-box warning addressing suicide risk. To date, there is no clear evidence of increased suicide risk in those using Neurontin outside its FDA-approved treatment of seizure disorder. The final decision is expected in 2007. It should be recognized that it is also illegal for drug representatives to discuss off-label use, and this information should be obtained from the medical literature. The ultimate decision to use a drug off-label is appropriately made by an individual physician for an individual patient.

In general, off-label drug use by a licensed physician is a common and widely recognized practice. The decision to do so should be based on current medical literature and with the acknowledgment that there is not a better, FDA-approved drug available. In addition, consideration should be given to current legal action surrounding the drug. For anesthesiologists who primarily administer drugs in the hospital setting, it may also be wise to educate and inform the patient via literature included with the consent for care received upon admission. Table 48.1 describes eight drugs and their approved and off-label uses.

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Editors' Note: We recommend strict on-label use for drugs administered in the intrathecal space, and advise against the use of certain lidocaine preparations and isobaric bupivacaine for spinals. Remember, it is easy to establish that isobaric bupivacaine is not only off-label but "against-label", as the vials are plainly marked as "Not for Spinal Anesthesia"—do you want to be forced to read that in a deposition or court of law? Another good example of the off-label controversy concerns the use of certain lidocaine spinals. There has been quite a bit of fallout at very respected institutions regarding off-label use of lidocaine, even with IRB approval. One of the many articles that appeared about this can be found at Anesth Analg. 2002 Sep;95(3):757-9.

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Remember that the Unthinkable *is* Possible—Follow These Principles in the Evaluation and Treatment of Patients Suffering from Nerve Agent Poisoning

Daniel J. Bochicchio MD, FCCP

It's rush hour on a warm June afternoon. While walking past a television in the operating room (OR) lounge, a news flash breaks into today's soap opera. A reporter at the baseball stadium approximately 7 miles away from your hospital is describing a plane crash. A small crop-dusting aircraft flew low over the grandstands and crashed into the press box. Apparently, some form of toxic chemical was being released by the aircraft at the time of the crash. The scene on television is chaotic. The news camera pans toward the stadium exits, where there are numerous dead bodies. Several individuals are staggering about. Several collapse onto the sidewalk. One victim vomits, collapses, and begins seizing. The chemical is tentatively identified as a "nerve agent." Absolute chaos is an appropriate description for the scene at the trauma center closest to the disaster. The press is estimating as many as 1,000 causalities, with >100 dead at the scene. You hear over the public address system that the hospital is implementing its mass casualty disaster plan. You have just been informed that your hospital can expect >100 casualties. Among the casualties are numerous police, fire, and emergency medical personnel. In an effort to evacuate the site rapidly, some victims may not have received adequate decontamination.

Thirty minutes later you receive your first victim from the scene. A 28-year-old woman, while attempting to flee, fell and was trampled by the crowd. She now presents with multiple contusions and abrasions for a laparotomy and possible splenectomy. She is awake and alert but appears very anxious, tremulous and tearful. Her blood pressure is 119/74 mm Hg, pulse 118 beats/min, respirations 32/min with mild dyspnea. While the staff in the emergency department is performing a brief focused physical exam, she vomits and voids on the stretcher. The patient's respiratory condition begins to deteriorate. She is promptly brought to the OR. You are informed that the agent has been identified as Sarin. You consider that the luckiest moment in your life is when the anesthesiologist who is covering the intensive care unit (ICU) this week comes over to help you start your case. He is a colonel in the Army Reserves with a specialty in chemical warfare.

He induces general endotracheal anesthesia. You notice that the airway pressure at 7-mL/kg tidal volume is 52 cm H_2O. There are copious P.203 *secretions noted in the no. 8 endotracheal tube. The surgeons are asking to begin. The patient's blood pressure is 134/67, pulse is 110 beats/min, SaO_2 is 92% by pulse oximetry on 100% inspired oxygen. Approximately 30 minutes after induction, your anesthesia technician begins to complain of blurry vision and rhinorrhea.*

The surgical procedure, an exploratory laparotomy and splenectomy, requires 1 hour to complete. It is without significant surgical complication. The patient is brought to the postanesthesia care unit (PACU). In the PACU there are again copious secretions noted in the endotracheal tube. Your ICU colleague says he will give more antidote (in the chaos, you were not completely clear that "antidote" had been given in the first place) and asks you to resuction the airway. After a few more minutes the spontaneous respirations appear much less labored and the secretions are improving. Your senior colleague decides not to extubate at this time and leaves you to care for the patient while he goes downstairs to help there. The nurse in pre-op holding calls you to come quickly; your next patient is apneic and seizing. She also confirms that there are hundreds of patients downstairs who need care.

How do you approach this mass casualty situation? What is meant by decontamination? What is meant by "the antidote?" How will you do the anesthesia for the next patient? Can you keep yourself safe?

DISCUSSION

Chemical warfare agents are chemicals that have direct toxic effects on mammalian tissue. The modern era of chemical warfare began on the French battlefields of World War I. On April 22, 1915, the German Army attacked the Allied positions with chlorine gas. The bombardment caused 15,000 Allied casualties, with 5,000 dead. In 1936, Dr. Gerhard Schrader discovered the highly toxic effects of the pesticide Tabun on mammalian tissue. This was quickly followed by the discovery of several more nerve agents. Although these highly toxic compounds have been in existence for 70 years, the vast majority of civilian health professionals have received little if any education or training on the medical management of victims of nerve agent poisoning.

Accordingly, most physicians and other health care providers caring for chemical casualties in recent conflicts lacked any formal training in this area. There are case reports of casualties contaminated with mustard agent in the Iraq-Iran war having been evacuated as far as hospitals in France without adequate decontamination. Lack of adequate knowledge to deal with the problem under the stress of a mass casualty situation will have a serious negative effect on survivability. There is nothing unique about the pathophysiology of chemical injury that is beyond the understanding of properly trained clinicians. The principles of rapid, thorough decontamination, self-protection, and an understanding of the actions of antidotes are the keys to effective chemical casualty care. P.204

Nerve agents are the most toxic chemical weapons known. They exert their biologic effect by inhibiting the enzyme acetylcholinesterase (AChE), thereby precipitating a cholinergic crisis. There are two major classes, the carbamates and the organophosphates (OPs). Among the former are neostigmine, physostigmine, pyridostigmine, and several commercially available insecticides. The OPs include the "military" nerve agents and several insecticides. Five OP AChE inhibitors are recognized as military nerve agents (NAs). They are commonly known as Tabun (North Atlantic Treaty Organization [NATO] designation: GA), Sarin (NATO: GB), Soman (GD), GF, and VX. The agents GF and VX do not have common names. The "G" agents were developed in Nazi Germany by Gerhard Schrader and his associates while researching insecticides for the conglomerate IG Farben between 1936 and 1944. VX was developed by the British in 1954 while searching for a replacement for the insecticide DDT. A nerve agent, given adequate time, will irreversibly bind all three forms of AChE.

The binding of NAs to AChE prevents the degradation of acetylcholine (ACh). The toxic effects of NAs are a result of massive excess of ACh. The symptoms are related to both the dose and the route of exposure. Inhalation of vapor causes immediate symptoms. If the vapor concentration (mg/m^3) and exposure time are significantly high, then the onset will be immediate and death may occur in minutes. Nerve agents are readily absorbed through intact skin and will penetrate clothing. The onset of symptoms after skin exposure to liquid agent can be delayed. Onset of systems depends on the

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amount of exposure, the promptness of decontamination, and temperature, moisture, and location on the body. Time of onset can range from several minutes up to 12 to 18 hours (Sidel et al., 1997).

All NAs are liquid at standard atmospheric conditions. The G-series agents are more volatile than VX and constitute a significant vapor hazard, whereas VX presents predominately a liquid hazard. Sarin (GB) is the most volatile of the G-agents and has a vapor pressure (2.1 mm Hg. at 20°C) that is close to that of water. Nerve agents are essentially colorless, odorless, and tasteless liquids. The G-agents are less persistent than VX, with $t_{1/2}$ of several hours to a few days. The $t_{1/2}$ of VX can be a week or longer. Nerve agents may be dispersed via several types of delivery systems. Identification of NAs can be accomplished using several detection systems. These detectors may not be immediately available to the initial civilian responders. Initial identification will likely be made by recognition of the clinical symptoms of toxicity until the arrival of civilian or military hazardous materials (HAZMAT) teams.

The toxic effects of cholinergic poisoning occur in the central, autonomic, and peripheral nervous systems. The most immediately lifethreatening are the P.205
respiratory compromise resulting from increased airway resistance, central nervous system (CNS)-mediated respiration depression, and paralysis. At higher exposures, apnea, seizures, and coma occur. Of particular concern to health care providers are the ophthalmic symptoms of miosis, blurred vision, diminished visual acuity, and loss of accommodation. These symptoms can occur after minimal exposure to vapor and can severely affect patient care. In the 1995 Sarin attack on the Tokyo subway, miosis was the most common symptom among victims and health care providers. Miosis was reported by >90% of victims.

If the toxic agent is not immediately identified, secondary contamination may pose a significant risk to emergency response personnel. Decontamination should occur as close to the site of exposure as possible to limit the spread of contamination and reduce the time to initial treatment. A hospital receiving potentially contaminated casualties should establish a “hot zone,” where casualties can receive adequate decontamination before being permitted entry into the facility. This area is best located in a well-ventilated open area outside the hospital. A “dirty dump” site must be established downwind from the treatment facility.

Decontamination begins with removal of the victim's clothing. The goal is the physical removal and chemical degradation of the chemical agent. Casualties should be decontaminated with copious amounts of tepid soap and water. The victim's hair may become a source of contamination and may require cutting to facilitate decontamination. Avoid hot water, harsh chemicals, strong soaps, and stiff brushes. Care should be taken to avoid causing vasodilatation or mechanical damage to the skin, as this may enhance absorption of nerve agents. Mild alkaline solutions and chlorine releasing agents such as diluted (0.5% hypochlorite) household bleach will neutralize NAs more quickly that plain soap and water. This solution is contraindicated in open injuries of the eye, abdomen, or brain.

In spite of decontamination stations, contaminated patients may find their way into the emergency department, operating room, intensive care unit, or any other hospital location if not controlled. Contaminated medical electronic equipment may not be recoverable. Secondary injury to health care providers will seriously degrade medical capabilities and add to the casualty numbers. *Institutional and individual self-protection is of critical importance. Latex gloves, surgical masks, and gowns are not protective.* Butyl rubber gloves and gowns or chemical-protective overgarments must be worn by all personnel caring for all casualties whose contamination status is unknown. “Off-gassing” of volatile nerve agent from clothing and, to a much lesser extent, skin poses a vapor threat, and appropriate respiratory protection must be worn. P.206

For chemically injured patients, the fundamental principles of resuscitation do not change. Establish an airway and provide adequate gas exchange and circulatory support. However, to this must be added: Terminate the exposure and decontaminate the victim. The exact sequence in which these occur can be greatly influenced by the situation. The cornerstones of pharmacologic management consist of three classes of drugs: Atropine, an anticholinergic; pralidoxime (2-PAM), a pyridinium oxime; and the benzodiazepines. The actions of atropine and the oximes may be synergistic. Atropine is extremely effective at blocking the effects of excess ACh at peripheral muscarinic sites. Atropine will improve the bronchoconstriction, bronchorrhea, nausea, vomiting, diarrhea, and bradycardia. Atropine will not alleviate many of the ocular disturbances or prevent skeletal muscle paralysis. The oxime 2-PAM reactivates AChE by breaking the AChE-NA bond. The NA bound to 2-PAM is unable to attack and bind AChE. The 2-PAM reactivation of AChE is most apparent in improved skeletal muscle strength. It should be noted that reactivation of AChE by oximes will occur only if this treatment is given before the chemical process of aging of the AChE-NA bond. Once the AChE-NA bond has aged, the oxime cannot reactivate the enzyme. Thereafter, recovery of function depends on synthesis of new AChE. The aging half-time varies from a few minutes for Soman to 48 hours for VX. The aging half-time for Sarin is 5 hours. Benzodiazepines are used to decrease convulsant activity associated with severe exposure.

Simplicity should be the goal for any mass-casualty treatment protocol. The U.S. Army Chemical Casualty Care Division of the Medical Research Institute of Chemical Defense offers a practical approach to medical management initially developed for battlefield use. Remember that exposure to NAs generally occurs via one of three routes—inhalation of vapor, liquid on skin, or ingestion. Because of the large absorptive capacity of the respiratory system, exposure to vapor rapidly produces symptoms. Exposed patients present with acute-onset miosis, copious rhinorrhea, dyspnea, apnea, and ultimately, convulsions and death. Depending on the severity of the exposure, the time course of events may vary from a few seconds to a few minutes. Victims exposed to high concentrations of vapor are unlikely to survive, so you probably won't be seeing these patients in the surgical suite. The time to onset of symptoms following dermal exposure may be highly variable.

Exposures are characterized by the severity of the intoxication as well as the route. Intoxication can be mild to moderate or severe. A mild vapor exposure might present with miosis, dim vision, rhinorrhea, salivation, anxiety, and/or mild dyspnea. The presence of severe respiratory distress, loss of bowel or bladder control, generalized muscle fasciculation, convulsions,
or apnea implies severe poisoning. Mild to moderate dermal exposure may present with localized muscle twitching and sweating at the exposure site, P.207
anxiety, nausea, vomiting, and a feeling of weakness. Severe liquid exposure would include all of the aforementioned symptoms, plus severe dyspnea, generalized muscle twitching, seizures, loss of consciousness, apnea, and loss of bowel and bladder control. For mild to moderate intoxication in an adult, 2 to 4 mg of atropine by intravenous (IV) bolus plus 600 to 1200 mg of pralidoxime IV over 30 minutes would be the initial therapy. For severe intoxication, 6 mg of atropine with 1,800 mg pralidoxime plus 10 mg Diazepam IV is the recommended initial therapy. Atropine is titrated to an improvement in symptoms as generally indicated by a reduction in secretions and the ability to oxygenate and ventilate the victim. Nebulized [beta]-agonists should be administered to help treat NA-induced bronchoconstriction. It should be noted that nerve agent-induced miosis is resistant to parenterally administered atropine and therefore is not a good indicator of full atropinization. If intravenous access is unavailable, then both antidotes may be given intramuscularly. Civilian physicians may see military-style, U.S. Food and Drug Administration-approved, Atro-Pen MARK I kits used in a NA attack. Many municipalities are acquiring them for emergency responders and other providers. The kits contain two autoinjectors: One containing 2 mg of atropine and another containing 600 mg of pralidoxime chloride. Autoinjectors are a superior delivery method for intramuscular antidotes and can quickly achieve high serum concentrations. Using this dosing method, casualties would receive one, two, or three MARK I kits intramuscularly (IM), with or without diazepam, based on the severity of the exposure. Additional atropine would then be titrated to clinical response at 10- to 15-minute intervals. Children are more susceptible to the effects of NAs. Resuscitation of pediatric victims of NA exposure poses a greater challenge. Dosing is weight-based, and intravascular access may be difficult. The current recommendations are atropine 0.05 to 0.1 mg/kg, pralidoxime 25 to 50 mg/kg, and Diazepam 0.05 to 0.3 mg/kg IV/IM.

Victims with combined injuries are at higher risk of mortality. Nerve agent intoxication reduces the cardiopulmonary response to physical trauma. Penetrating trauma provides a direct portal of entry for NAs. Examination and evaluation of a toxic, traumatized patient may be a particular challenge. The

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anesthetic management of the NA-intoxicated, traumatized patient is based on a very small number of cases and is therefore largely unknown. General endotracheal anesthesia may be the safest anesthetic technique. Inhaled volatile anesthetics are potent bronchodilators and relax skeletal muscle. This should facilitate ventilation and provide acceptable surgical conditions. The high risk of nausea and vomiting will require airway protection during the induction of anesthesia. Selection of induction

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agents can be a challenge. Sodium thiopental or propofol may worsen the hemodynamic instability of the NA-intoxicated patient. Ketamine may offer some advantages. Ketamine is commonly used to induce anesthesia in the hemodynamically compromised patient and is a potent analgesic. The bronchodilation and mild increase in heart rate may be beneficial. Atropine should be given before the ketamine to avoid central apnea. The skeletal muscle relaxation choices may also be restricted. NA-induced inhibition of plasma cholinesterase and the unique structure of succinylcholine may cause its duration of action to become unpredictable. The positive chronotropic effect of pancuronium may make it a better choice. Using neostigmine to reverse the effects of the neuromuscular blockade may prove problematic. Careful monitoring of the neuromuscular blockade is recommended. Perioperative pain management may require opioids. The histamine-releasing potential of morphine may make it a poor selection. Histamine release can exacerbate the bronchoconstriction and hypotension associated with NAs. Fentanyl has potential vagotonic effects and may worsen the bradycardia. The use of meperidine as an analgesic has been suggested because of its lack of histamine release and its tendency to cause an increase in heart rate.

TAKE HOME POINTS

- The medical management of mass-casualty events poses some of the greatest challenges to the health care delivery system.
- Mass-casualty situations involving chemical, biologic, or radiologic weapons present an even greater threat.
- One fact is certainly not debatable: Lack of preparedness on the part of the health care community will not improve the outcome of such an event. With proper training and preparation, the human costs of a chemical attack can be mitigated.
- Nerve agents are odorless, tasteless, colorless liquids—the term “nerve gas agent” is a common misnomer.
- Individual self-protection is critically important—standard OR garb *does not* provide an adequate barrier to contamination.
- Ask what the nerve agent mass-casualty plans are for your location—do not assume it can't happen!

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Use Bicarbonate as A Buffer to Local Anesthetics, Especially for Skin Infiltration

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Local anesthetics reversibly block impulse conduction along nerve axons and other excitable membranes that utilize sodium channels as the primary means of action potential generation. The local anesthetics used clinically consist of a lipid-soluble, substituted benzene ring linked to an amine group via an alkyl chain containing either an amide or an ester linkage.

Local anesthetics are weak bases (pK_a 7.6 to 9). These agents are poorly soluble in water and therefore are usually marketed in acidic hydrochloride salt solutions (pH 3 to 6). In this form, the local anesthetic rapidly becomes reduced to a cationic form. This process is readily reversible, and the relative proportions of neutral base and ionized form will equilibrate as described by the Henderson-Hasselbach equation:

Log (cationic form/uncharged form) = pK_a - pH

The proportions of the drug that exist in each form depend on the pH of the solution and the pK_a, or dissociation constant, of the particular drug. This dissociation constant (pK_a) denotes the pH at which the ionized and neutral forms of the molecule are present in equal amounts. Since the pK_a of most local anesthetics is in the range of 7.6 to 9.0, the larger fraction in the body fluids at physiologic pH will be the charged, cationic form.

Local anesthetics reversibly block conduction of action potentials by interacting with the intracellular portion of the voltage-gated sodium channels. It is the charged form of the local anesthetic molecule that appears to interact with this portion of the channel. The local anesthetic must first penetrate the lipid-rich neural cell membrane as the uncharged, lipid-soluble form.

BUFFERING LOCAL ANESTHETICS TO DECREASE ONSET TIME

Local anesthetics are administered in an acidic solution, so most of the agent is in the ionized form, which is lipophobic. Therefore, the drug must first be converted to the nonionized form in sufficient quantity to enter the nerve cell. This depends on the pK_a of the local anesthetic and the pH of the tissue. Once inside the nerve cell, the lower pH converts the drug back into the ionized form, which is then able to block the sodium channels.

Increasing the pH of the carrier solution of local anesthetics with sodium bicarbonate will favor the formation of a more neutral base form of the drug. This uncharged molecule is more capable of diffusing through the cell membrane and will theoretically result in more rapid nerve blockade. P.211

The addition of sodium bicarbonate to local anesthetic solutions has been reported to decrease the time of onset of conduction blockade. Alkalinization of solutions of bupivacaine or lidocaine accelerates the onset of brachial plexus and epidural blockade in some studies but not others. In addition, a recent animal study suggests that alkalinization of lidocaine decreases the duration of peripheral nerve blocks if the solution does not also contain epinephrine.

BUFFERING LOCAL ANESTHETICS TO DECREASE PAIN ON INJECTION

The acidity of the commercial preparations of local anesthetics makes them painful to inject into the skin or subcutaneous tissues. There are several studies that show adding bicarbonate to lidocaine attenuates pain on skin infiltration.

The mechanism of pain reduction is unknown but probably relates to an increase in pH. If H^+ ions themselves cause pain, sodium bicarbonate would decrease pain merely by decreasing H^+ concentration. Consistent with this idea, commercial lidocaine with epinephrine (pH 4.15) has a low pH and is more painful than is lidocaine with added epinephrine (pH 6.4). However, the cause of pain on skin infiltration is probably more complex and depends on factors other than pH alone. For example, procaine (pH 4.3) is more acidic but less painful than lidocaine (pH 6.3). Alternatively, the pain-reducing effect of sodium bicarbonate may represent a shift of the equilibrium between the ionized and nonionized forms of lidocaine, favoring formation of the nonionized form. The more rapid diffusion of the nonionized form may result in rapid inhibition of pain transmission, thereby preventing nociceptive impulses from being fully appreciated.

How to Prepare Buffered Lidocaine for Skin Infiltration

- Add 1 mL of sodium bicarbonate at a concentration of 1 mEq/mL to 9 mL plain lidocaine 1%.
- Add 1 mL of sodium bicarbonate at a concentration of 1 mEq/mL to 4 mL plain lidocaine 2% (will increase the pH from approximately 6.2 to 7.26).

Buffering local anesthetic solutions decreases their shelf life. Most amides and amines are chemically unstable in the buffered, uncharged form and are subject to photodegradation and aldehyde formation. Buffered lidocaine remains active for only 1 week when stored at room temperature, and for a few weeks when refrigerated.

TAKE HOME POINTS P.212

- Local anesthetics are weak bases that are poorly soluble in water. They are marketed in acidic salt solutions to improve stability and shelf life.
- Buffering with bicarbonate solution increases the neutral (nonionized) fraction, which can cross the lipid-rich neural cell membrane and then be converted to the ionized form to act on the sodium channel.
- Some (but not all) studies have shown that buffering local anesthetic decreases the onset time for brachial plexus and epidural blocks.
- Buffering local anesthetics *will* decrease “pain of injection.” Palmon et al. found that the addition of bicarbonate had a greater overall effect than needle size in decreasing the pain associated with the intradermal injection of lidocaine. It is hoped that increased use of buffered

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lidocaine for intravenous starts and needle placement for nerve blocks will decrease the necessity of the warning, “A little stick and a burn here, Mr. Johnson.”

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Consider Perioperative Clonidine Administration—it Has Anxiolytic, Antiemetic, and Analgesic Properties

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Shashank Saxena MD

Clonidine, originally introduced clinically as a nasal decongestant and subsequently used as a centrally acting antihypertensive, has become increasingly utilized for its anesthetic properties. The sedative effects of α_2-agonists have been known since their introduction. In fact, a volunteer once slept for an entire day following a dose of intranasal clonidine. Human and animal studies performed in the late 1960s demonstrated a profound reduction in minimum alveolar concentration (MAC) when inhalation agents were administered concurrently with α_2-receptor agonists such as clonidine. α-Receptor agonists have been utilized in veterinary medicine for many years as a regional anesthetic, but only relatively recently have they been employed in human patients. Tamsen and Gordh injected a parenteral preparation of clonidine epidurally into two patients in 1984, after they first established its safety in animals. Since that time a large body of research has explored both the safety and the clinical benefits of clonidine as an analgesic; however, it is only approved by the U.S. Food and Drug Administration (FDA) to treat hypertension and in the treatment of cancer pain. Though it is more popular in Europe, clonidine is used off-label in the United States for many other benefits.

CHEMICAL PROPERTIES/PHARMACOLOGY

Clonidine, an imidazoline, is a partial α_2-aerometric receptor agonist that acts primarily presynaptically and inhibits norepinephrine release in the nucleus tractus solitarii of the medulla oblongata. It is not highly selective at α-receptors, as it has a selectivity ratio of approximately 200:1 for α_2- to α_1-receptors, respectively. Furthermore, clonidine is able to interact with nonadrenergic imidazoline receptors found in the brain, kidney, and pancreas. α_2-Adrenergic receptors function through G-protein mechanisms. Four subtypes exist, and all activate well-defined intracellular cascades. Clonidine was first thought to exhibit its effects primarily through presynaptic receptors in the medulla, though it is now known to act both directly in spinal preganglionic sympathetic neurons as well as in the dorsal horn by pre- and postsynaptic mechanisms.

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PHYSIOLOGIC EFFECTS

The primary affect of clonidine is sympatholytic, but—possibly because it binds with many different receptors—the drug has various actions beyond antihypertensive properties, such as sedation, anxiolysis, analgesia, and as an adjunct with other anesthetics. Clonidine causes a decrease in central sympathetic outflow, thereby lowering arterial pressure. At low doses, clonidine has an anxiolytic effect on the central nervous system (CNS), though at higher doses it can be anxiogenic and cause hypertension, most likely as a result of α_1-activity. Little effect is exerted on the respiratory system, save a small reduction in minute ventilation. α_2-Receptors of β-cells in the pancreas are stimulated and cause a temporary inhibition of insulin release. This has not been proven to be problematic in a clinical setting. Clonidine has the added benefit for anesthesia of an antisialagogue effect.

DOSING/DURATION OF ACTION

The high lipid solubility of clonidine allows rapid and complete absorption after oral administration. Less than 50% is metabolized hepatically to inactive metabolites, with the remaining drug excreted unchanged by the kidneys. Orally, the peak plasma level is reached in 1 to 1.5 hours. It readily crosses the blood-brain barrier with an elimination half-life of 30 minutes after epidural injection of 150 mcg. In general, hemodynamic effects peak at around 1 to 2 hours and last approximately 6 to 8 hours. Sedation occurs in a dose-dependent fashion within 20 minutes regardless of the route of administration. These effects can last 4 to 6 hours. Dosing information on clonidine is shown in Table 51.1.

ANTIHYPERTENSIVE EFFECTS

Hypertensive patients usually respond to clonidine with a more profound drop in blood pressure than do normotensive patients. Though clonidine reduces heart rate, it seems to have little effect on the baroreceptor reflex, thus accompanying orthostatic hypotension and profound bradycardia are produced less frequently than with other antihypertensive drugs. Researchers have shown an attenuation of the stress response to direct laryngoscopy when a minimum dose of 4 mcg/kg clonidine is administered intravenously (IV) preoperatively. Further studies have shown a possible benefit in reducing perioperative cardiac ischemia, though conflicting reports exist. Rapid discontinuation of clonidine can precipitate a hypertensive crisis, so clonidine should be continued throughout the perioperative period.

SEDATION/REDUCTION IN ANESTHETIC REQUIREMENTS

Although clonidine is not used as a sole anesthetic agent, it has been used as a premedication in numerous studies. It is especially useful in pediatrics. In

comparison to benzodiazepines, clonidine produces a state of sedation similar to sleepiness rather than amnesia. Subjects are more easily roused when asked to perform tasks. Clonidine has also been shown to be an effective anxiolytic. A reduction in halothane MAC by as much as 50% has been demonstrated experimentally with clonidine. This effect has largely borne out clinically with other inhaled anesthetics. Other studies have demonstrated reductions in opioid, benzodiazepine, barbiturate, and propofol requirements.

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TABLE 51.1 DOSING INFORMATION FOR CLONIDINE

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Hypertension	Oral (adults): Initially, 0.1 mg PO twice daily; increase by 0.1-0.2 mg/d PO until desired effect is achieved (usual dosage range 0.2-0.6 mg/d PO). Children: Initially, 5-10 mcg/kg/d PO in divided doses every 8-12 h. Increase gradually (every 5-7 d) to 5-25 mcg/kg/day PO in divided doses every 6 h. Maximum dosage is 0.9 mg/d. Transdermal (adults only): Initially, apply one patch (delivers 0.1 mg/24 h) patch to an intact area of hairless skin on the upper arm or torso, once every 7 d. Adjust dosage every 1-2 wk.
Neuraxial pain control	Adults: Initially, 30 mcg/h by continuous epidural infusion in combination with opioid analgesics. Dosage titration is based on pain relief and adverse events; max. rate is 40 mcg/h. In clinical trials, bolus doses of epidural clonidine range from 100 to 900 mcg/dose. Intrathecal: Clonidine in doses of 25-40 mcg/h (600-960 mcg/d) has been used to manage chronic-pain patients.
Pediatric anesthesia	Given orally preoperatively (4 mcg/kg), decreases intraoperative anesthetic requirements and postoperative opioid consumption. The epidural or caudal dose is 1-2 mcg/kg, which may be followed by an infusion of 0.05-0.33 mcg/kg/h. It may cause sedation and hypotension when given via the epidural route.
Regional anesthesia	Dose of 0.5 mcg/kg or more enhances and prolongs the effect of local anesthetics used for brachial plexus block, peribulbar and retrobulbar blocks, and IV regional anesthesia. An intra-articular dose of 150 mcg enhanced postoperative analgesia in patients undergoing knee arthroscopy. Hemodynamic effects, namely, bradycardia and hypotension, increase with doses of 1.5 mcg/kg or more.

ANALGESIA

Oral, epidural, intrathecal, and parenteral administration of clonidine all produce analgesia and potentiate the action of other agents, thus enhancing motor and sensory blockade. This corresponds to a reduced side-effect profile in most instances. Epidural administration is most common, as a number of studies have demonstrated epidural clonidine to be an efficacious adjunct to opioid and local anesthetic injection in the management of acute postoperative pain. Studies have also demonstrated epidural clonidine to be superior to IV use for pain control after orthopedic surgery. Intrathecal clonidine seems to provide no additional benefit to epidural clonidine, as it provides no additional analgesia but does cause a greater incidence of hypotension. Also popular for use as an adjunct in regional anesthesia, clonidine prolongs duration of peripheral nerve blocks at small doses but does run the risk of prolonging motor blockade. Chronic pain treatment with clonidine has several benefits, including the avoidance of opioids in patients who may become dependent or addicted with long-term use. Clonidine is approved in the United States for the treatment of intractable cancer pain. P.216

OTHER USES

Additional off-label uses are particularly interesting to the anesthesiologist. Clonidine has been shown to decrease postoperative nausea and vomiting. It is an effective antisialagog. Some studies have shown clonidine to be an efficacious alternative to meperidine for postoperative shivering. A decrease in intraocular pressure has been reported with the use of clonidine during ophthalmic surgery. Hyperactive children with manic symptoms have been treated with the drug. Clonidine has also been used in the treatment of opioid, benzodiazepine, and alcohol withdrawal, as well in smoking cessation.

SIDE EFFECTS/CONTRAINDICATIONS

Clonidine's side-effect profile makes it a useful adjunct in anesthesiology. Common side effects that are often desirable perioperatively include dry mouth and sedation. Abrupt discontinuation of clonidine can produce a withdrawal syndrome possibly resulting in nausea, insomnia, headaches, and restlessness. Severe sudden cessation can result in profound hypertension and tachycardia. As stated above, clonidine is a very mild respiratory depressant at indicated doses and does not appear to potentiate opioid-induced respiratory depression. In the event of an overdose, clonidine has been reported to act as a more significant respiratory depressant. Furthermore, in high doses, α_1-activation can cause anxiety and this limits its use as a sedative. Although it is well established that intrathecal or epidural administration of clonidine is not neurotoxic, these routes are not recommended in pregnancy because they can cause hypotension. Clonidine is classified as a pregnancy category C drug. The contraindication includes hypersensitivity to clonidine hydrochloride or any component of the formulation. It should be avoided in patients with perioperative hypovolemia, spontaneous bradycardia, atrioventricular block, or prolonged P-R interval. Transdermal patch may contain conducting metal (e.g., aluminum); therefore it should be removed prior to MRI. Due to the potential for altered electrical conductivity, the transdermal patch should be removed before cardioversion or defibrillation. P.217

TAKE HOME POINTS

- Clonidine serves as a useful drug to the anesthesiologist beyond its use as an antihypertensive.
- It is useful as a sedative, especially in pediatric anesthesia.
- Although it is not used as a sole anesthetic, clonidine reduces the dose requirements in epidural, intrathecal, and peripheral nerve blocks.
- Clonidine is approved for cancer pain and serves as an off-label adjunct in the treatment of other chronic pain syndromes. It is also used in the treatment of opioid and nicotine withdrawal.
- Clonidine has additional benefits as an antisialagogue, antiemetic, and in reducing postoperative shivering.
- When the recommended dose is used, clonidine has very few side effects or contraindications.
- Newer, more selective α_2-agonists such as dexmedetomidine are likely to find more widespread use by the anesthesiologist.

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Consider Chloroprocaine for Regional Blockade When Appropriate—It Is a Rapid-Onset Local Anesthetic with Low Systemic Toxicity

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Randal O. Dull MD, PhD

2-Chloroprocaine is an ester local anesthetic introduced into clinical practice in 1951; the initial paper described >200 successful spinal anesthetics. Despite its initial use in spinal anesthetics, 2-chloroprocaine found its greatest use in labor epidurals because of its rapid metabolism and low toxicity to both mother and fetus. The widespread use and availability of intrathecal lidocaine, however, apparently prevented most anesthesiologists from adopting 2-chloroprocaine as their spinal drug of choice. After decades of use, a report of 8 patients who suffered neurologic deficits after "epidural" anesthesia with 2-chloroprocaine was published. It appears that at least half of these patients had inadvertently received intrathecal administration, and this raised the question of neurotoxicity. It was not clear at the time whether the etiology was the local anesthetic or the sodium bisulfite preservative. A flurry of publications have attempted to address this issue of toxicity, establish a dose-response curve, and evaluate various adjuncts to 2-chloroprocaine as an effective spinal anesthetic and as a replacement for intrathecal lidocaine.

CHEMICAL PROPERTIES

2-Chloroprocaine contains a lipophilic aromatic ring bound to a hydrophilic tertiary amine by an ester linkage.

CLINICAL PHARMACOLOGY

Like all local anesthetics, chloroprocaine inhibits the generation and propagation of nerve impulses by increasing the threshold for electrical excitation, slowing conduction, and reducing the rate of rise of the action potential. Being an ester, 2-chloroprocaine is metabolized by plasma cholinesterase (and red cell esterase, to a lesser extent) into an alcohol and a *para*-aminobenzoic acid. Chloroprocaine is metabolized much more quickly than procaine or tetracaine, contributing to its low systemic toxicity. Its plasma half-life is approximately 21 seconds in adults and 43 seconds in neonates. Any condition, however, that inhibits the activity or quantity of pseudocholinesterase will prolong action and increase toxicity (e.g., liver disease, atypical cholinesterase). The kidney is the primary route of excretion for chloroprocaine metabolites. P.219

CLINICAL USE

Intrathecal Use.

Though chloroprocaine was initially described as a spinal anesthetic, it has fallen into disfavor in the past two decades because of concerns about neurotoxicity. This notion has recently been challenged by a series of studies. The future of 2-chloroprocaine as a medication for intrathecal use is currently in flux, and further studies are underway to further define its use. It was initially shown that 2-chloroprocaine was an appropriate substitute for intrathecal lidocaine, providing similar quality, height, and duration of block when compared to equal doses of lidocaine. A later study showed that 30, 45, and 60 mg of 2-chloroprocaine yielded mean maximal block heights of T7, T5, and T2 and block duration of 98, 116, and 132 minutes, respectively. When 20 mcg of fentanyl was added, the duration of sensory but not motor blockade was lengthened (by 25 minutes). When epinephrine was added to 2-chloroprocaine, a transient flulike syndrome was reported, leading to the recommendation to avoid this combination. When compared to other local anesthetics for ambulatory surgery, 2-chloroprocaine provides adequate duration and quality of blockade, with significantly faster resolution than bupivacaine and less risk of transient neurological syndrome (TNS) than lidocaine. Considerable controversy still surrounds the issue of potential neurotoxicity of spinal 2-chloroprocaine. The prevailing conclusion of a series of animal studies was that the preservative sodium bisulfite, possibly in combination with low pH, was responsible for several early episodes of irreversible blockade. However, at least one study suggests that 2-chloroprocaine may itself be neurotoxic, and that sodium bisulfite may actually be neuroprotective.

Epidural Use.

Because of its low toxicity and rapid onset, chloroprocaine has been used effectively for years as an epidural anesthetic. When a 3% solution is used, an epidural dose of 15 mL will provide analgesia to T10, and 20 mL will yield a level to T4 with a rapidity that approaches that of subarachnoid anesthesia. The duration of action is short, however, with chloroprocaine lasting only 30 to 50 minutes even when epinephrine is added. These properties can be used to good effect in the delivery suite—chloroprocaine can be used to "repaint" an epidural block after delivery when the labor block has worn off and episiotomy repair will be extensive or when a small surgical procedure, such as tubal ligation, is planned after delivery.

Formulations of 2-chloroprocaine containing ethylenediamminetetraacetic acid (EDTA) were associated with severe low back pain on injection. It was hypothesized that the EDTA chelated calcium in the paraspinous muscle, inducing local muscle spasm. Formulations are now available that do not contain EDTA. P.220

Topical Use.

Although it is not an "on-label" use, 2-chloroprocaine provides rapid topical anesthesia for skin incisions. Because of its rapid plasma clearance, direct administration of 2-chloroprocaine into incisions appears to be safe and effective. This is the "splash block" that anesthesiologists with many years of experience may refer to.

TAKE HOME POINTS

- Chloroprocaine is an ester local anesthetic with a plasma half-life of 21 seconds in adults and 43 seconds in neonates.

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- Whether chloroprocaine should be used as a local anesthetic for intrathecal purposes is still debatable (it may actually be neurotoxic).
- Using chloroprocaine with epinephrine as an additive is not recommended—especially with spinal anesthesia.
- For epidural use, chloroprocaine provides an excellent safety index. Further, it has a rapid onset that comes close to that seen with intrathecal use of local anesthetics.
- Typical anesthetic levels with a 3% epidural solution are T10 with 15 mL and T4 with 20 mL.
- Chloroprocaine can be useful to supplement an epidural when short-acting anesthesia is necessary for an episiotomy repair.
- Because of its rapid plasma clearance, it can even be administered directly into an incision (as a "splash block").

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Editors: Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.
Title: *Avoiding Common Anesthesia Errors, 1st Edition*

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Consider Using Ketamine When Appropriate—If Managed Carefully, the Benefits will Usually Outweigh the Potential Side Effects

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In 1970, 8 years after it was first synthesized, ketamine was released for clinical use in the United States. Initially ketamine was believed to be the “ideal” anesthetic, providing amnesia, analgesia, immobility, and loss of consciousness. Shortly after its use became widespread, however, there were many reports of patients emerging from anesthesia with hallucinations and recalling vivid dreams. The discovery of “emergence reactions” led many practitioners to avoid using ketamine for routine cases. However, ketamine remains an important tool in the anesthesiologist's armamentarium, as it is a versatile drug that can be given by almost any route and can provide analgesia, sedation, or general anesthesia depending on the dose administered. Originally, ketamine was indicated as an anesthetic as well as an analgesic for cardiac surgery, trauma, obstetric analgesia, as well as for dressing changes in burns patients.

Ketamine is different from other anesthetics in that it induces a state of dissociate anesthesia, whereby higher centers in the brain are unable to perceive auditory, visual, or painful stimuli. There is a dose-related loss of consciousness and profound analgesia. Patients' eyes remain open and they often maintain reflexes, although corneal, cough, and swallow reflexes cannot be assumed to be protective. In addition, patients have anterograde amnesia and have no recall of surgical procedures. There is a rapid onset of action, as the drug crosses the blood-brain barrier quickly secondary to low molecular weight, high lipid solubility, and a pK_a near physiologic pH. Following intravenous (IV) administration, onset is seen in 30 seconds, with peak action in 60 seconds. The anesthetic effects of ketamine following a usual induction dose (1 to 2 mg/kg) remain for 10 to 15 minutes only. Drug plasma levels required for anesthesia and amnesia are 0.7 to 2.2 mcg/mL, with awakening occurring at levels of 0.5 mcg/mL. Although the anesthetic duration of action of ketamine is short, the analgesic effects last much longer, as a plasma level of only 0.1 mcg/mL is required for analgesic effects to be seen.

Ketamine interacts with N-methyl-D-aspartate (NMDA), opioid, nicotinic, muscarinic and calcium-channel receptors. It is the antagonistic action at NMDA receptors that produces the majority of ketamine's effects, including analgesia, amnesia, and the psychomimetic side effects. It also has effects on both central and spinal opioid receptors, with μ-receptors contributing to analgesia and κ-receptors to the psychomimetic effects. On a larger scale, ketamine leads to inhibition of thalamocortical pathways and stimulation of the limbic system. P.223

Ketamine acts differently from other anesthetic drugs in relation to both the respiratory and cardiac systems. Ketamine does not depress respiration unless it is given in a large rapid bolus, and CO_2 responsiveness is maintained at or close to normal levels. The major respiratory advantage of ketamine is that it causes profound bronchodilation, to the same degree as inhalation agents. This makes ketamine an excellent choice of induction agent in asthmatic patients. It has even been used to treat refractory cases of status asthmaticus.

Unlike other anesthetic agents, ketamine does not depress the cardiac system. On induction with ketamine, increases in heart rate, blood pressure, and cardiac output are seen. The mechanism of the cardiovascular effects is sympathetic stimulation and inhibition of both intraneuronal and extraneuronal uptake of catecholamines. Because it is indirect stimulation that leads to these effects, in the catecholamine-depleted patient, ketamine can have the opposite effect, as the drug also has a direct myocardial depressant effect that is normally masked by its sympathetic activity. Ketamine's effects on the heart lead to an increase in cardiac oxygen consumption, thus making it a poor choice for patients with ischemic cardiac disease. Patients with pulmonary hypertension are also poor candidates for ketamine, as the drug causes a greater increase in pulmonary vascular resistance than systemic vascular resistance.

A variety of patients can benefit from the use of ketamine as an induction agent, especially ASA class 4 patients with respiratory and cardiac dysfunction, excluding cardiac ischemia. The bronchodilating effects of ketamine make it particularly helpful for patients with severe bronchospastic disease. The other niche where ketamine has been found to be interesting is with patients with hemodynamic compromise secondary to hypovolemia or cardiomyopathy. This includes such diagnoses as sepsis, trauma, cardiac tamponade, and restrictive pericarditis. The only caveat is that if these patients are catecholamine-depleted, the direct myocardial depressant effects may be seen. In tamponade, the benefit of ketamine is that it maintains the heart rate and right atrial filling pressures through its sympathetic stimulation. Patients who require frequent, brief procedures, such as burn patients undergoing dressing changes, can benefit from the use of ketamine as a sedative, because subanesthetic doses can be used with good analgesia and rapid return to normal function. It can be used as an adjunct to regional anesthesia, as a sedative prior to painful blocks, or to position patients already in pain. The main advantage is the profound analgesia without respiratory depression or hypotension. Because of these properties, ketamine is useful in emergency medicine, war zones, entrapment situations, and high-altitude anesthesia. P.224

Pediatric anesthesia includes another subset of patients who benefit from the use of ketamine. Intramuscular ketamine (7 to 9 mg/kg) remains in use for induction of anesthesia in uncooperative pediatric patients. Children with neuromuscular disorders also benefit from the use of ketamine as a maintenance agent, as volatile agents should be avoided in these children, who are at increased risk of malignant hyperthermia. Children with congenital heart defects leading to right-to-left shunt do well with ketamine as an induction agent for cardiac surgery, as it increases systemic vascular resistance and does not reverse the shunt. Finally, ketamine is useful for pediatric sedation, as it produces analgesia and sedation with a low incidence of complications. Emergence reactions are much less common in the pediatric population than in adults.

A more controversial area for ketamine is its use in neurosurgical patients. It had previously been thought that ketamine increased intracranial pressure and cerebral blood flow and was therefore contraindicated in patients at risk for elevated intracranial pressure. However, recent studies have shown that if benzodiazepines are given with ketamine and normocapnea is maintained, there is no elevation in intracranial pressure and so ketamine may have a role in neurosurgery. Additionally, blockade of NMDA receptors may be neuroprotective, as it can decrease cell destruction and necrosis following cerebral ischemia. The role of ketamine in neurosurgery has not yet been decided, and further research will determine its utility.

Recently, ketamine has received much attention for its use as an analgesic. It has been used as an adjunct in both acute postoperative pain management as well as in chronic pain, including both neuropathic and refractory cancer pain. The analgesic effects of ketamine are due to several mechanisms. The

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primary mechanism is NMDA-receptor antagonism leading to suppression of pain transmission to higher centers in the brain. Ketamine also prevents the development of tolerance to acute opioid administration and development of increased pain sensitivity secondary to opioid-induced hyperalgesia by its NMDA antagonism. By this mechanism it also prevents the "windup" phenomenon, which is an increase in dorsal horn activity due to repetitive and constant C-fiber stimulation. There is some evidence that ketamine also acts directly on opioid receptors in the brain and spinal cord, contributing to its analgesic effect.

For postoperative pain control, ketamine can be given preincision, postincision, or as a part of the postoperative pain regimen. Subanesthetic doses of ketamine (0.1 to 0.3 mg/kg) produce analgesia, and the analgesic effects of an induction dose of ketamine are seen for several hours following an initial bolus dose. The goal of pre-emptive analgesia is to decrease postoperative pain by interrupting nociceptive pathways. NMDA receptors are responsible for pain memory and "windup," so ketamine, an NMDA antagonist, can prevent the massive nociceptive afferent impulses from reaching the brain. However, at the present time there is some controversy regarding the efficacy of pre-emptive ketamine and whether it is able to reduce total opioid requirements postoperatively. If it is given intraoperatively, it has been shown that ketamine is a much more effective analgesic when given as an infusion rather than as a single bolus dose at the time of induction. Anesthesia providers who have a lot of sedation cases report that a ketamine infusion coupled with a low- to moderate-dose propofol infusion (known sometimes in the vernacular as "P-K anesthesia") has good efficacy in a wide range of surgical cases. Postoperatively, ketamine has been added to Patient Controlled Analgesic (PCAs) (1 to 2 mcg/kg/h) and has been shown to decrease the total amount of morphine required as well as to decrease side effects. P.225

Ketamine has been used as an additive with local anesthetics for epidural or caudal use, and patients receiving ketamine in this form require less postoperative opioid than those receiving only local anesthetics. However, racemic ketamine is not preservative-free, and the preservative benzethonium chloride may be neurotoxic. S(+)-ketamine is an isolated stereoisomer that is preservative-free and shows potential for future neuraxial use. However, in the United States, ketamine is available only in racemic form. In Europe, S(+)-ketamine has gained in popularity and is a more potent anesthetic associated with more rapid emergence and a lower incidence of emergence reactions.

Although it has many advantageous properties, no drug is without its adverse reactions. Ketamine has a fairly notorious side-effect profile, which may lead to hesitance in using this drug. The emergence reactions seen with ketamine include visual, auditory, and proprioceptive illusions that can illicit fear or excitement in patients. An illustrative clinical anecdote was related by one of the Certified Registered Nurse Anesthetist (CRNA) advisers to this book: The anesthetist was using ketamine as a partial induction agent, and just as the patient lost consciousness, one of the operating room (OR) staff mentioned that he had killed a large black snake in his yard that morning. On emergence, the patient related that he had had vivid and severely unpleasant "dreams" under anesthesia about black snakes. The anesthetist now uses the power of suggestion in a positive way before the administration of ketamine—she tells her patients they may have vivid dreams and asks them to imagine their favorite or a very pleasant "brightly colored place"—e.g., a sunny tropical beach or brilliant fall foliage. She also requests that there be no excessive chatter or loud noises at induction. It is also well recognized that the concomitant use of benzodiazepines reduces the risk of emergence reactions, and virtually all practitioners who use ketamine preadminister a benzodiazepine. P.226

Ketamine also causes profuse salivation in some patients, which can be a problem in the unprotected airway with the possibilities of laryngospasm and aspirations. It is recommended to pretreat these patients with an antisialagogue such as glycopyrrolate. Atropine is to be avoided, as it can cause an increased risk of delirium on emergence. However, with appropriate use and proper patient selection, ketamine has proven to be beneficial for a variety of procedures and patient populations.

TAKE HOME POINTS

- Although ketamine went through a period of "unpopularity," it is actually a valuable drug that every anesthesia provider should be familiar with and comfortable using.
- It can be given by a number of routes and acts at a number of receptors. It can be used for sedation, amnesia, anesthesia, and pain relief.
- Consider it especially in cases when sedation/anesthesia is needed for short periods of intense surgical or procedural stimulus, such as dressing changes, burn treatments, or removal of K-wires.
- It is valuable as an adjunctive agent in both general anesthetic and sedation cases.
- Actively manage the potential side effects, particularly the possibilities of hallucinations—reassure and discuss beforehand with the patient, always give a benzodiazepine, and ensure a quiet, calm anesthetizing location.
- Contraindications to ketamine include:
  - Elevated ICP
  - Open eye injury - ketamine increases IOP
  - As sole anesthetic in ischemic cardiac disease
  - Vascular aneurysm - don't want sudden change in arterial pressure
  - Psychiatric disorders like schizophrenia
  - History of adverse reaction to ketamine or pcp
  - If post op delirium could be from other cause-delirium tremens, head trauma
- Factors that increase risk of delirium include:
  - Age >16 years
  - Dose >2 mg/kg
  - Pre-existing personality problems

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Editors: Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.
Title: *Avoiding Common Anesthesia Errors, 1st Edition*

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Dexmedetomidine can be a Useful Drug, But Will It Be Universally Applicable?

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Sedative-hypnotic and analgesic agents (benzodiazepines, propofol, and opioids) form an integral part of anesthesia to provide patients' comfort and safety in the operating room (OR) and the intensive care unit (ICU). a_2-Adrenergic receptor agonists have been used increasingly as a new armamentarium to provide sedative/hypnotic, analgesic, anxiolytic, and sympatholytic effects in the perioperative and critical care settings.

WHAT IS DEXMEDETOMIDINE?

Dexmedetomidine (Precedex; Abbott Labs, Abbott Park, IL, USA) is a relatively selective α_2-adrenergic receptor agonist (eight times more specific for the α_2-adrenoreceptor compared to clonidine). Compared to other α_2-adrenergic agonists such as clonidine, dexmedetomidine has a shorter half-life (2 hours for intravenous dexmedetomidine versus 8 to 12 hours for peroral clonidine).

HOW DOES DEXMEDETOMIDINE WORK?

Dexmedetomidine stimulates a_2-adrenoreceptors in the locus ceruleus of the brainstem to provide sedation. It also provides analgesia by stimulating a_2-adrenoreceptor in the central and peripheral nervous systems. Dexmedetomidine causes sympatholysis via central and peripheral mechanisms. Following intravenous (IV) administration, dexmedetomidine undergoes rapid redistribution, with a distribution half-life of 6 minutes and an elimination half-life of 2 hours. Dexmedetomidine exhibits linear kinetics in the dosing range of 0.2 to 0.7 mcg/kg/h when administered through IV infusion for 24 hours. Dexmedetomidine is 94% protein-bound and undergoes nearly complete biotransformation in the liver to inactive metabolites that are excreted in the urine.

POTENTIAL BENEFITS OF DEXMEDETOMIDINE

During a continuous infusion within its therapeutic level, dexmedetomidine provides unique sedation (patients appear to be asleep but are readily aroused), analgesic-sparing effect, and minimal depression of respiratory function. Dexmedetomidine has no pharmacokinetic or cytochrome P450 enzyme drug-drug interactions.

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COMPLICATIONS/CONTRAINDICATIONS

Hypotension, hypertension, nausea, bradycardia, fever, and vomiting are most frequently observed adverse events associated with dexmedetomidine. The net effect of α_2-adrenergic agonists, exerted via central and spinal receptors, is sympatholytic (or "provagal"). Therefore, caution should be exercised in patients with pre-existing severe bradycardia disorders or severe ventricular dysfunction, in whom sympathetic tone is critical for maintaining hemodynamic balance. Clinical events of bradycardia and sinus arrest have been associated with dexmedetomidine administration in young, healthy volunteers with high vagal tone or with different routes of administration including rapid IV/bolus administration (www.fda.gov/cder/foi/label/1999/210381bl.pdf). Transient hypertension has also been observed, primarily during the loading infusion, associated with initial peripheral vasoconstriction effects of dexmedetomidine prior to its central nervous system (CNS)-mediated vasodilatory effect.

Recent clinical studies, however, demonstrated more reliable control of heart rate and blood pressure in patients undergoing surgery with appropriate doses of dexmedetomidine. Dose reduction should be considered in patients with impaired liver and renal function. Dexmedetomidine is contraindicated in patients with a known hypersensitivity to the drug, which has not been reported so far.

FDA-APPROVED USES

In 1999, dexmedetomidine was approved by the U.S. Food and Drug Administration (FDA) for sedation of adult patients who are intubated and mechanically ventilated in the intensive care setting. Dexmedetomidine should be administered by continuous infusion not to exceed 24 hours.

HOW TO ADMINISTER DEXMEDETOMIDINE

Dexmedetomidine is supplied in 2-mL vials each containing 100 mcg of drug/mL (200 mcg total) and should be diluted with 48 mL of sterile water or 0.9% sodium chloride to a final concentration of 4 mcg/mL. Dexmedetomidine is recommended to be administered as a loading dose of 1 mcg/kg over 10 to 20 minutes, followed by a maintenance infusion of 0.2 to 0.7 mcg/kg/h, titrated to the desired sedation scale.

CLINICAL APPLICATIONS OF DEXMEDETOMIDINE

Intensive Care Unit.

Because of its nonopioid mechanism of analgesia and lack of respiratory depression, dexmedetomidine has been used in the critical care setting for sedation. It is not necessary to discontinue dexmedetomidine infusion before extubation. The use of dexmedetomidine is not limited to the ICU, and a growing body of literature has described the effectiveness of the drug in perioperative settings.

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Operating Room.

Compared to placebo or propofol infusion, dexmedetomidine infusion was reported to provide more stable hemodynamics for patients who underwent

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cardiac and vascular surgeries. Dexmedetomidine seems to attenuate the stress-induced sympathoadrenal response seen with laryngoscopy, intubation, and surgery. It potentiates the anesthetic effects of varieties of intraoperative anesthetic drugs. This anesthetic-sparing effect of dexmedetomidine can be translated into a more rapid recovery from anesthesia. Dexmedetomidine may also be useful as an anesthetic adjunct in patients who are susceptible to narcotic-induced respiratory depression, such as morbidly obese patients, in whom the opioid-sparing effects of dexmedetomidine have proven extremely useful. Also, in the difficult-airway realm, dexmedetomidine has been used as a single-agent sedative along with topical anesthesia for awake fiber-optic intubation. It has been used as an adjunct in monitored anesthesia care. Some studies have demonstrated the effective use of dexmedetomidine in awake craniotomy, or even as a sole anesthetic for minor procedures.

Pediatrics.

Dexmedetomidine has gained significant attention for the treatment of emergence delirium, which is a frequent phenomenon in children recovering from general anesthesia. Dexmedetomidine has been shown to reduce the incidence of emergence delirium when administered as a single dose of 1 mcg/kg intraoperatively approximately 30 minutes before the end of the procedure. Treatment of existing emergence delirium is also effective, with 0.125 mcg/kg bolus and repeated doses as needed, which avoids unnecessary oversedation. Continuous infusions have also been studied, and both treatment options have not shown any increase in prolonging time to extubation or time to discharge from the postanesthesia recovery unit. Dexmedetomidine may prove to be very useful in children with underlying neurologic disorders, who often develop agitation or adverse hemodynamic or respiratory reactions with opioids or benzodiazepines. Dexmedetomidine has also become a viable option for pediatric ICU sedation, much as in the adult population. This has allowed for avoiding the prolonged use of propofol for sedation, minimizing the possibilities of "propofol infusion syndrome." Though dexmedetomidine is not currently approved by the FDA for use in the pediatric population, it may offer an additional choice for the sedation of children both postoperatively and in the ICU. The efficacy of non-IV administration (intramuscular, per-oral, and buccal) of the drug has also been suggested.

CLINICAL CONTROVERSY

Many investigators have demonstrated a variety of off-label clinical applications of dexmedetomidine as described above. A few studies also suggest caution in the usage of dexmedetomidine. The provagal effect of

dexmedetomidine was demonstrated by Ebert and associates, with augmentation of baroreceptor response to phenylephrine and preservation of baroreceptor response to nitroprusside. This "provagal" effect of dexmedetomidine was considered to be a contributory factor in cardiac arrest of an adult patient, reported by Ingersoll-Weng and others, who underwent sternotomy with a thoracic epidural anesthesia and preoperative pyridostigmine. We had a pediatric patient who developed hypotension without bradycardia with low-dose dexmedetomidine in the presence of baclofen (unpublished observation). The delayed recovery can be a problem with dexmedetomidine. Jalowiecki and colleagues counseled against the use of dexmedetomidine as a sole agent for procedural sedation, in light of hemodynamic instability and the need for prolonged recovery. Koroglu and associates compared the sedative, hemodynamic, and respiratory effects of dexmedetomidine and propofol in children undergoing magnetic resonance imaging. In the study, they found the onset of sedation, recovery, and discharge time were significantly longer in the dexmedetomidine group, although the decrease of mean blood pressure, heart rate, and respiratory rate from the baseline were smaller in the dexmedetomidine group compared to the propofol group.

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There is significant synergism of opioids with dexmedetomidine. Therefore, one should be cautious against overdosing with dexmedetomidine in the presence of opioids, which can result in delayed emergence. In practice terms, the use of dexmedetomidine may be cumbersome because of the need for a loading dose, controlled continuous infusion, and titration of the dosage to prevent overdosing.

Dexmedetomidine is, at the present time, approved by the FDA only for sedation in mechanically ventilated adult patients who are being monitored in the ICU. Large prospective clinical studies are needed to further elucidate and confirm the effectiveness of the off-label uses of dexmedetomidine.

TAKE HOME POINTS

- Dexmedetomidine, a newer selective a_2-adrenergic receptor agonist, decreases sympathetic tone with attenuation of the neuroendocrine and hemodynamic responses to anesthesia and surgery, reduces anesthetic and opioid requirements, and provides sedation and analgesia without respiratory depression.
- Dexmedetomidine is recommended to be given as a loading dose of 1 mcg/kg over 10 to 20 minutes, followed by a maintenance infusion of 0.2 to 0.7 mcg/kg/h for a total duration of not more than 24 hours.
- Dexmedetomidine is currently approved for intubated and mechanically ventilated adult patients in the ICU. However, it has demonstrated its effectiveness in other surgical settings.

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Check for History of Migraine before Giving Ondansetron, Especially in Children

Michael J. Moritz MD

Serotonin, or 5-hydroxytryptamine (5-HT) is a neurotransmitter derived from the amino acid tryptophan. Systemic 5-HT affects the cardiovascular, respiratory, and gastrointestinal systems, with vasoconstriction being the typical vascular response. Thus, 5-HT antagonists will cause vasodilation. In the gastrointestinal system, most serotonin receptors are of the 5-HT3 type. Thus, 5-HT antagonists will cause vasodilation. In the gastrointestinal system, most serotonin receptors are of the 5-HT3 type. The strongest stimulus for emesis from both chemotherapy and postoperatively is serotonin release from the gut enterochromaffin cells. This release stimulates afferent vagal fibers via their 5-HT3 receptors that activate the vomiting center in the brainstem (chemoreceptor trigger zone). Thus, serotonin antagonists decrease nausea and cause vasodilation.

The three 5-HT3 antagonist drugs available in the United States are ondansetron (Zofran), granisetron (Kytril), and dolasetron (Anzemet). These 5-HT3 receptor antagonists are the most effective antiemetic drugs available. All three drugs are similar in effectiveness, cost, and side-effect profiles.

The 5-HT3 antagonists have relatively few side effects. The most common side effect is headache, occurring in 10% to 20% of patients receiving doses to prevent chemotherapy-induced emesis and in 10% of patients receiving the lower doses used for postoperative nausea and vomiting. In children in particular, a personal or family history of migraine headache leads to a much higher risk of ondansetron-related migraine at the antiemetic dosing for chemotherapy.

The interplay of the 5-HT3 receptor, vasodilation, and vasoconstriction can be seen in the management of migraine. The treatment of migraine blocks the vasodilation that causes the headache, typically with serotonin agonists. For example, sumatriptan succinate (Imitrex) is a 5-HT1 agonist that causes vasoconstriction. Because the 5-HT3 antagonists cause vasodilation, their use should be considered carefully in patients susceptible to migraines.

P.234

TAKE HOME POINTS

- The strongest stimulus for emesis from both chemotherapy and postoperatively is serotonin release from the gut enterochromaffin cells.
- In the gastrointestinal system, most serotonin receptors are of the 5-HT3 subtype.
- Serotonin has multiple systemic effects; the most typical vascular response is vasoconstriction.
- The 5-HT3 antagonists generally have relatively few side effects; however, anesthesia providers should be aware of the interplay of the 5-HT3 receptor and vasodilation, as it plays a role in the pathogenesis of migraine-type headaches, especially in children.

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Remember that Not All Blue-Colored Compounds are the Same

Chauncey T. Jones MD

There are many blue dye compounds, and each is used for a variety of purposes. Their uses include food coloring, dye for materials, antiseptics, tissue stains, chemical reaction indicators, and to treat diseased aquarium life. Blue dyes are also used in humans for both diagnostic and therapeutic purposes. These dyes include methylene blue, patent blue, isosulfan blue, and indigo carmine. While these substances are all blue in color, they are distinct compounds and have widely varying properties, uses, and adverse effects (Table 56.1).

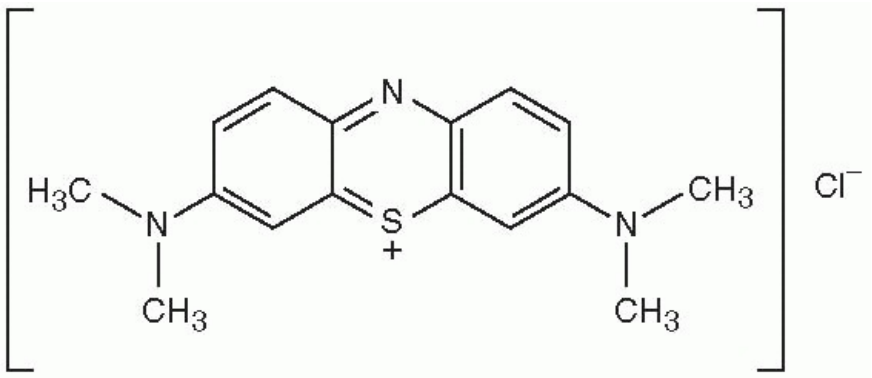
METHYLENE BLUE

Synonyms: Methylthionium chloride, tetramethylthionine chloride, urolene blue, swiss blue, solvent blue

Molecular formula: $C_{16}H_{18}ClN_3S$

Molecular weight: 319.85

Structure:



Methylene blue has been used in many clinical situations in humans. It is used primarily as an antidote for both chronic methemoglobinemia (e.g., occurring with dapsone therapy) and acute methemoglobinemia. At low concentrations it promotes the reduction of methemoglobin (the ferric form of hemoglobin) to hemoglobin by acting as a cofactor for the enzyme NADPH-methemoglobin reductase. The recommended dose is 1 to 2 mg/kg administered intravenously (IV). The dose may be repeated after 1 hour if symptoms persist.

Methylene blue has been used as a sentinel lymph node tracer for breast cancer and may be as efficacious and have fewer side effects than other blue dyes more traditionally used for this purpose. It has also been used as a genitourinary antiseptic and for topical virus therapy. The antimicrobial effect is presumed to be secondary to its oxidative/reduction properties.

TABLE 56.1 BLUE DYE COMPOUNDS

BLUE COMPOUND	COMMON USES	ADVERSE EFFECTS
Methylene blue	▪ Antidote for metHgb ▪ Sentinel lymph node tracing ▪ Genitourinary antiseptic ▪ Septic shock ▪ Cyanide toxicity	▪ Hypertension ▪ Chest pain ▪ Anemia ▪ Bladder irritation ▪ Transient, false SpO_2 decrease ▪ Headaches, diaphoresis, abdominal pain
Patent blue	▪ Sentinel lymph node tracing • Breast cancer • Melanoma • Endometrial cancer • Colon cancer	▪ Severe anaphylaxis ▪ Prolonged, false SpO_2 decrease
Isosulphan blue	▪ Tumor marking ▪ Lymphangiography	▪ Urticarial reactions ▪ Anaphylaxis ▪ Transient, false SpO_2 decrease

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Indigo carmine	▪ Urology • Locate ureteral orifices during cystoscopy • Test anastomoses • Identify transected ureters intraoperatively	▪ Mld elevation of blood pressure ▪ Relatively low side-effect profile ▪ Transient, false SpO_2 decrease
Indocyanine green	▪ Ophthalmology • Macular surgery • Cataract surgery ▪ Blood volume, cardiac output determinations ▪ Infrared therapy for colon cancer	▪ Can be toxic to eye unless thoroughly washed out ▪ Transient, false SpO_2 decrease
Blue #1	FDA black box warning against use in enteral feeds for detecting aspiration	▪ Refractory hypotension ▪ Metabolic acidosis (acts as a mitochondrial toxin) ▪ Worse in patient with increased gastrointestinal permeability ▪ Death

Although it is not the treatment of first choice, at high concentrations methylene blue can be used to treat cyanide toxicity by converting ferrous hemoglobin to ferric methemoglobin. Methemoglobin combines with cyanide to form cyanmethemoglobin. This serves as an alternative pathway for cyanide and prevents it from combining with cytochrome oxidase, which results in cyanide toxicity. P.237

Possible side effects of methylene blue include hypertension; chest pain; headache; confusion; dizziness; diaphoresis; anemia; discoloration of skin, urine, and feces; nausea; vomiting; abdominal pain; and bladder irritation. The increase in blood pressure caused by methylene blue is so pronounced that it was historically used as a treatment for septic shock, before the era of modern-day vasopressors.

Methylene blue can also transiently decrease pulse oximetry readings by causing the oximeter to interpret methylene blue as deoxygenated blood. It is sometimes used by anesthesia personnel as an indicator dye or "marker" to denote a special situation in the operating room, such as an IV bag that has had succinylcholine added to it or to provide a visual cue to diagnose an endotracheal cuff leak. Be aware, however, that the high side-effect profile of methylene blue mandates careful consideration of this type of use.

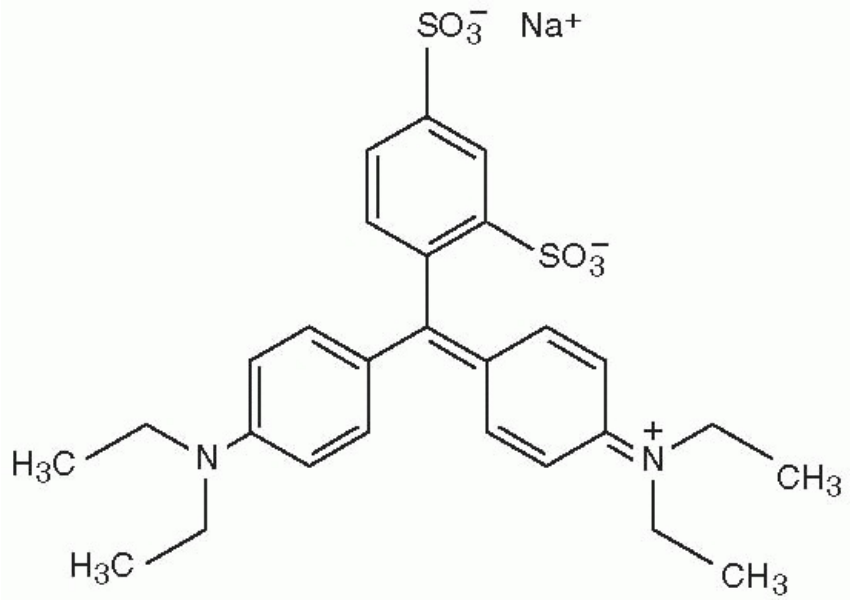
PATENT BLUE

Synonyms: Sulphan blue, blue v, disulphine blue, food blue 3, acid blue 1

Molecular formula: $C_{27}H_{31}N_2NaO_6S_2$

Molecular weight: 566.67

Structure:



In humans, patent blue is used primarily for lymphangiography and sentinal node biopsy. When a tumor is identified, dye is injected into the tumor and peritumoral tissue. The dye is then taken up by the lymphatics that drain that region. The first lymph node that absorbs dye is the sentinal lymph node. This node is then biopsied and checked for malignancy. This information is used for staging, to decide whether more extensive lymph node dissection is needed, and to determine the need for other ajuvant therapies. This modality is most commonly used in breast cancer but is also used for other cancers, such as melanoma, endometrial cancer, and colon cancer. Before the development of sentinal node biopsies, radical lymph node dissections were routinely performed for breast cancer. Patent blue can also be used in conjunction with radiolabeled materials as an alternative method of identifying sentinal nodes. P.238

The side-effect profile of patent blue is of considerable concern to users. It has been cited as causing severe anaphylaxis in humans and its use is limited in certain countries, including the United States. In addition, pulse oximetry readings can be artificially reduced for prolonged periods with higher doses of patent blue. This is likely because the dye is being injected subcutaneously versus intravascularly as with methylene blue.

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ISOSULPHAN BLUE

Isosulfan blue (also called lymphazurin) is the 2,5-disulfophenyl isomer of patent (or sulphan) blue and is also used for tumor marking and lymphangiography for sentinal node biopsies.

Though it is generally thought to be safer than sulfan blue, it has been cited as causing an array of allergic reactions ranging from mild urticaria to severe anaphylaxis. Preoperative prophylaxis appears to reduce the severity but not the incidence of these reactions. Isosulfan blue has similar affects as patent blue on transiently decreasing pulse oximetry readings.

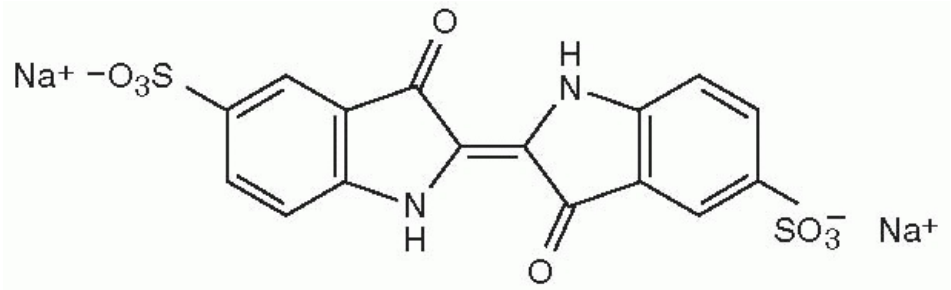
INDIGO CARMINE

Synonyms: Sodium indigotin disulfonate, soluble indigo blue, indigotine, Acid Blue 74, FD&C Blue No. 2

Molecular formula: $C_{16}H_8N_2Na_2O_8S_2$

Molecular weight: 466.35

Structure:



Indigo carmine is excreted unaltered by the kidneys. This excretion is relatively fast, and blue urine can be seen as quickly as 5 to 10 minutes after administration. It was once used as a kidney test to rule out obstruction before more sophisticated modalities became available. It currently is used to locate ureteral orifices during cystoscopy, to test cystourethral anastomosis, and to identify transected ureters intraoperatively. Typically, a 5-mL one-time dose of indigo carmine is administered intravenously. P.239

This compound has a relatively low side-effect profile. Mild elevations in blood pressure have been reported. Indigo carmine can cause a transient decrease in pulse oximetry readings.

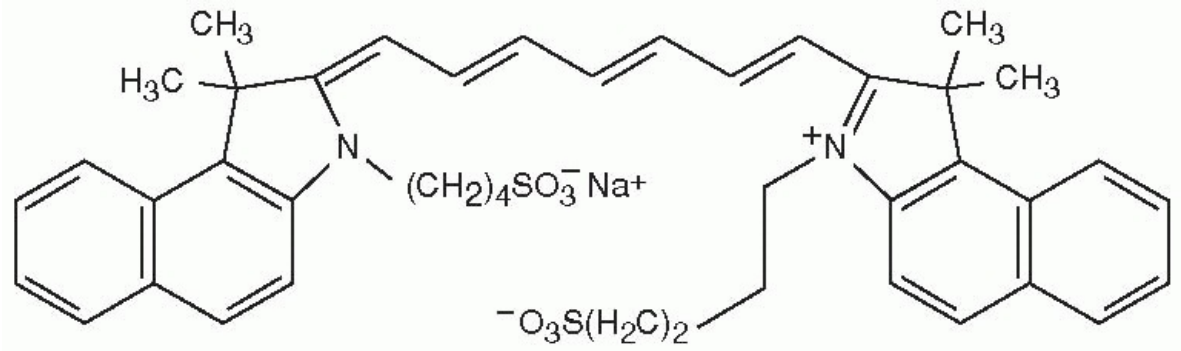
INDOCYANINE GREEN

Synonyms: Fox Green, Cario-Green

Molecular formula: $C_{43}H_{47}N_2NaO_6S_2$

Molecular weight: 774.98

Structure:



Despite its name, indocyanine green is often grouped with the blue dyes. In humans, indocyanine green is most commonly used for eye surgery, specifically for macular hole and cataract surgery. It can be toxic to the eye if not thoroughly removed at the end of surgery. Its tissue-toxic effects have been used for therapeutic purposes in the photo-oxidative destruction of colon cancer cells with infrared light. It can also be used as a diagnostic aid in blood volume determination and cardiac output via its absorption properties. Indocyanine green may cause a transient decrease in pulse oximetry if it is injected intravascularly.

FDA BLACK BOX WARNING!

In September 2003, the U.S. Food and Drug Administration (FDA) issued a Public Health Advisory ("Black Box Warning") against the use of blue dye to color enteral feeds as a means to detect pulmonary aspiration. Though this practice had been in effect for about 30 years, it had been previously uninvestigated by the FDA. The report specifically targets FD&C Blue No. 1, which is used in much lower concentrations in foods, cosmetics, and drugs. This compound is also known as Blue #1 and Steri-Blue. Blue #1 was temporally associated with several effects. These included discoloration of skin, urine, feces, serum, refractory hypotension, metabolic acidosis, and death. The most susceptible patient population was the critically ill and those with increased gut permeability (i.e., patients with sepsis, trauma, burns, inflammatory bowel disease). The exact reason for severe side effects is not known, but Blue No. 1 is known to be a mitochondrial toxin, which could lead to severe metabolic acidosis. Finally, the FDA states that other blue dyes such as methylene blue and FD&C Blue No. 2 (indigo carmine) may have similar or even worse toxicity than Blue No. 1 and are not appropriate replacements. Some experienced hospital pharmacists are now recommending that the above-described blue dyes not be used when contact with the gastrointestinal tract is possible. P.240

TAKE HOME POINTS

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- These compounds are all blue in color, but they have a wide variety of properties.
- Most blue compounds can affect pulse oximetry readings.
- Methylene blue has a high side-effect profile (under general anesthesia, hypertension is likely to be the most significant one).
- Patent blue can cause severe anaphylaxis in humans.
- Preoperative prophylaxis can reduce the severity of reactions seen with isosulphan blue.
- Indigo carmine has a relatively low side-effect profile.
- Indocyanine green must be thoroughly washed out after eye surgery.
- Blue compounds should not be used as markers in enteral feeds.

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Do Not Forget that Linezolid is a Monoamine Oxidase Inhibitor (MAOI) as Well as an Antibiotic

Neil B. Sandson MD

Linezolid is one of the more recently developed antibiotics, and it is gaining increasing use in the treatment of infections caused by multiply resistant gram-positive organisms. An important, often underappreciated, feature of linezolid is its action as a weak, reversible inhibitor of monoamine oxidase. Lack of recognition of the possibility of significant MAOI-based interactions has led to unfortunate results in hospitalized patients.

To review briefly, the enzyme monoamine oxidase is critically important in the metabolism of the monoamines, which include all of the catecholamines deriving from tyrosine (epinephrine, norepinephrine, and dopamine) as well as serotonin. Inhibition of monoamine oxidase via pharmacologic agents can result in greatly increased catecholamine levels. Co-administration of an MAOI with a sympathomimetic agent, or with any medication that increases the activity or availability of catecholamines, creates the danger of a hypertensive crisis. Similarly, combining MAOIs with serotonergically active compounds may lead to a central serotonin syndrome, characterized by flushing, diarrhea, myoclonus, fever, delirium, seizures, and possibly autonomic collapse and death. Patients who take MAOIs must also adhere to a tyramine-free diet (no aged cheeses, aged or fermented meats, broad bean pods, chianti wine, sauerkraut, or most soybean products) in order to avoid a potential hypertensive crisis.

The most commonly prescribed monoamine oxidase inhibitors (MAOIs) are the antidepressant medications phenelzine (Nardil) and tranylcypromine (Parnate), both potent and irreversible inhibitors of monoamine oxidase. These drugs have been in clinical use for decades and are generally readily recognized by anesthesia providers. Clinical practice concerning the administration of phenelzine and tranylcypromine in the perioperative period has undergone some evolution over the years—generally, most practitioners do not withhold these drugs in the perioperative period, but in order to minimize the danger of a hypertensive crisis instead advocate careful and titrated administration of sympathomimetics.

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Because linezolid is a reversible and less potent inhibitor of monoamine oxidase than phenelzine or tranylcypromine, it was hoped that the same pharmacodynamic interactions would not be seen. Unfortunately, such has not been the case. Since its introduction, there have been numerous reports of central serotonin syndrome when linezolid has been combined with selective serotonin reuptake inhibitors. There has also been one report of intractable intraoperative hypertension occurring in a patient on a combination of linezolid and bupropion (an antidepressant that inhibits the reuptake of both norepinephrine and dopamine).

Therefore, the same caveats that we apply to the administration of adrenergic and serotonergic drugs to patients receiving the traditional MAOIs should also apply to patients on linezolid. For patients who are receiving linezolid in the perioperative period, it is likely that pressor agents would produce exaggerated and possibly dangerous responses. These agents include ephedrine, isoproterenol, and phenylephrine. It is important to note that rapid increases in desflurane concentration have been described as causing a paradoxical increase in sympathetic activity, including increases in plasma norepinephrine as well as the clinical effects of hypertension and tachycardia. Therefore, the author recommends that desflurane be avoided as well. Serotonergic drugs that are contraindicated for patients on linezolid include meperidine and dextromethorphan.

If the patient is on linezolid or has been in the previous 2 weeks, it is important to either craft an anesthetic plan that minimizes the use of agents that would interact with an MAOI, or to communicate with the treatment team as to possible alternative antibiotic agents that might be used. It is important to review the patient's medication regimen before surgery, as an interaction between monoaminergically active medications and linezolid may become apparent only during the sympathetic activity that may accompany painful surgical maneuvers. Although there have been no case reports of linezolid-tyramine interactions, avoidance of high-tyramine foods while taking linezolid is prudent.

Remember also that there are actually several drugs with a degree of MAOI activity. One such drug is a new antidepressant in the form of transdermal selegiline (trade name Emsam), approved by the U.S. Food and Drug Administration (FDA) in March 2006. This antidepressant is likely to find widespread clinical use, so anesthesia providers will probably be encountering this drug in patients' preoperative medication panels. Traditionally, selegiline is an oral drug that is used to treat Parkinson disease. At low doses, neither of these forms of selegiline requires a tyramine-free diet. However, use of these drugs at any dose dictates the same perioperative concerns as with linezolid, phenelzine, or tranylcypromine.

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TAKE HOME POINTS

- Linezolid is frequently used in the perioperative setting for the treatment of resistant gram-positive organisms.
- Anesthesia providers must be aware that linezolid is an inhibitor of monoamine oxidase.
- Although linezolid is a weak, reversible inhibitor of MAO, it has the same potential for deleterious interactions as phenelzine (Nardil) and tranylcypromine (Parnate).
- Other drugs with a degree of MAOI activity have recently been approved, such as transdermal selegiline (trade name Emsam), an antidepressant that is likely to find widespread use.

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Be Alert for the Signs and Symptoms of Perioperative Digoxin Toxicity, Especially if the Patient is at Risk for Electrolyte Depletion

Grace L. Chien MD

Digoxin is a semisynthetic steroid glycoside derived from the plant *Digitalis lanata* (common name: Grecian foxglove). It is used for the treatment of systolic dysfunction and some arrhythmias. By reversibly inhibiting the Na-KATPase pump, digoxin increases the availability of intracellular calcium and subsequently increases cardiac contractility. This increase shifts the Frank-Starling curve to the left in both healthy and failing myocardium. Digoxin also reduces circulating levels of norepinephrine and thus increases parasympathetic tone. This in turn reduces sinoatrial nodal automaticity and atrioventricular nodal conduction. With its neurohumoral-modulating effects, digoxin has been used to treat some re-entrant paroxysmal supraventricular tachycardias.

Digoxin toxicity develops when there is an overload of intracellular calcium in both systolic and diastolic states, and this may cause significant arrhythmias. According to Mahdyoon et al., digoxin toxicity occurred in 0.8% and possible toxicity occurred in another 4% of the population. Factors that increase the risk of digitalis intoxication are renal insufficiency, electrolyte abnormalities (hypokalemia, hypomagnesemia, and hypercalcemia), acute myocardial ischemia, advanced age, hypothyroidism, and pulmonary disease. Pharmacokinetic interactions with other drugs can also affect digoxin levels. Remember that digoxin is a substrate of the P-glycoprotein transporter, so inhibitors and inducers of this transporter will raise and lower serum digoxin levels respectively (see Chapter 47).

Signs of digoxin toxicity may appear at a level of 2 ng/mL, and the incidence of toxicity increases with plasma concentration. Plasma concentration reaches steady state between 6 and 12 hours after the last dose, and thus digoxin levels are best measured within this time. Signs of digoxin toxicity may be very nonspecific. Patients may present with confusion, dizziness, ventricular ectopy, tachycardia, visual disturbances, depression, anxiety, apathy, gynecomastia, or simply a rash.

Dangerous signs of digoxin toxicity include atrioventricular (AV) nodal block, bradycardia, ventricular arrhythmia, thrombocytopenia, delirium, and hallucinations. For patients under general anesthesia, the main warning signs of toxicity are electrocardiogram (EKG) changes. Digoxin toxicity can cause many types of dysrhythmias but does not usually cause atrial flutter, atrial fibrillation, or Mobitz type II second-degree AV block. However, digoxin toxicity can superimpose on the underlying cardiac pathology. For example, digoxin may create an unusually slow ventricular response to atrial fibrillation. Digoxin can also increase both atrial and ventricular ectopic activity and induce junctional tachycardia. Premature ventricular contraction is often the first sign of digoxin toxicity, progressing to ventricular bigeminy, fascicular tachycardia, ventricular tachycardia, and fatal ventricular fibrillation.

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One arrhythmia that is almost pathognomonic for digoxin toxicity is bidirectional ventricular tachycardia (Fig. 58.1). The pattern resembles bigeminy except that the R-R interval is regular because all of the beats arise from a single ventricular focus. Patients with the rare disorder of familial catecholaminergic polymorphic ventricular tachycardia may also show this EKG pattern. Digoxin toxicity may cause AV junctional rhythms with escape rates of 30 to 50 beats/min. When the patient's heart rate is 50 to 60 beats/min, an escape rhythm may be in play. With an accelerated junctional rhythm of 60 to 120 beats/min, nonparoxysmal junctional tachycardia is seen. The enhanced vagal tone created by digoxin may generate first-degree, second-degree type I, and third-degree heart blocks.



FIGURE 58.1. Bidirectional ventricular tachycardia. (Reproduced with permission, © UpToDate at www.uptodate.com.)

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Of course, the first-line treatment for digoxin toxicity is to avoid it. Anesthesia providers should pay special attention to both digoxin levels and serum electrolytes in a patient who has had a bowel prep. Remember that a patient who has even low-normal serum potassium may be at increased risk of digoxin toxicity, even at "therapeutic" digoxin levels. Most clinicians aim for a serum potassium of around 4.0 mEq/L at the time of induction; this is supported by a classic scattergraph in Opie's *Drugs for the Heart.* Also, be aware that a common time to see digoxin-induced arrhythmias is at induction or just after induction. Keep close surveillance of the EKG during this time and do not disrupt the patient's acid-base status with untoward adjustments in ventilation.

Other treatment of digoxin toxicity depends on the symptoms of the patient. Impending cardiac arrest necessitates the ABCs (airway, breathing, and circulation). Bradycardia can be treated with atropine. Transvenous cardiac pacing and β-receptor agonists should be avoided because they may precipitate other cardiac arrhythmias. Administering activated charcoal within 6 to 8 hours postingestion may ameliorate digoxin toxicity. If hyperkalemia coexists with digoxin toxicity, calcium should not be given in the usual fashion for hyperkalemia, because it may potentiate digoxin toxicity. Hypomagnesemia should be treated promptly, because it also potentiates cardiac glycoside toxicity.

The most specific and effective treatment for digoxin toxicity is digoxin-specific Fab fragments (Digibind). Digibind is indicated in patients with life-threatening arrhythmias associated with digoxin, patients with hemodynamic instability, patients with severe bradycardia, patients whose plasma potassium levels are >5 mEq/L, patients with plasma concentration >10 ng/mL, ingestion of >10 mg of digoxin in adults or >4 mg in children, or digoxin-toxic rhythm in the setting of an elevated digoxin level. Digibind binds to intravascular and interstitial digoxin, causing diffusion of digoxin from the intracellular to the extracellular space. Bound digoxin cannot reassociate with the inhibitory site on the α-subunit of Na-K-ATPase, rendering it ineffective. Dosing of Digibind is as follows:

$$\text{Vials of digoxin-specific Fab fragments} = \text{digoxin level (ng/mL)} \times \text{mass (kg)} / 100.$$

The digoxin and digoxin-specific Fab fragment complex is rapidly excreted in urine with an elimination half-life of 15 to 20 hours. In patients with renal failure, dialysis can effectively eliminate Fab fragment-digoxin complexes. In contrast, digoxin has not been shown to be effectively eliminated by dialysis.

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TAKE HOME POINTS

- Digoxin is a steroid glycoside with multiple cardiac and neurohumoral actions.
- Digoxin toxicity is not rare—known risk factors include advanced age, acute myocardial infarction, renal insufficiency, and electrolyte abnormalities, all of which are factors commonly seen in surgical patients.
- Digoxin serum levels are mediated by the P-glycoprotein transporter.
- There are multiple signs of digoxin toxicity in the unanesthetized patient; in patients under anesthesia, the first sign is usually an EKG abnormality, often PVCs.
- If the patient is on digoxin and has had a bowel prep, ensure electrolyte repletion before the start of the anesthetic.
- Do not give calcium to treat hyperkalemia in a digoxin-toxic patient.
- The most specific and effective treatment for digoxin toxicity is Digibind.

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Exercise Care in the Use of Amiodarone and Alternative Antiarrhythmics for the Treatment of Atrial Fibrillation

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Atrial fibrillation is a common arrhythmia in both the intensive care unit (ICU) and perioperative cardiothoracic surgery settings. The mainstay of therapy is rate control, with beta-blockers being the first-line agents. In more acute clinical situations that involve significant hemodynamic deterioration, synchronized, direct-current cardioversion is immediately indicated. Frequently, however, the decision is made to use rhythm-converting agents, such as amiodarone. Amiodarone is a complex antiarrhythmic agent (predominantly class III) that shares at least some of the properties of each of the other three Vaughn-Williams classes of antiarrhythmics. Amiodarone is commonly used for the treatment and prevention of persistent atrial and ventricular tachyarrhythmias, although it is approved by the U.S. Food and Drug Administration (FDA) only for management of ventricular arrhythmias. It is one of the few agents that can be used safely in individuals with congestive heart failure. Contraindications to amiodarone include severe sinus node dysfunction with marked sinus bradycardia or syncope, second- or third-degree heart block, known hypersensitivity to its contents, cardiogenic shock, and probably severe chronic lung disease.

Amiodarone is highly lipid-soluble, extensively distributed in the body, and highly concentrated in many tissues, especially in the liver and lungs. After variable (30% to 50%) and slow gastrointestinal (GI) absorption, amiodarone is very slowly eliminated with a half-life of about 25 to 110 days. The onset of action after oral administration is delayed, and a steady-state drug effect may not be established for several months unless large loading doses are used. Amiodarone undergoes extensive hepatic metabolism to the pharmacologically active metabolite, desethylamiodarone (DEA). Amiodarone is excreted not by the kidneys but rather by the lacrimal glands, the skin, and the biliary tract. Neither amiodarone nor DEA is dialyzable.

Amiodarone is both an antiarrhythmic and a potent vasodilator. Amiodarone lengthens the effective refractory period by prolonging the actionpotential duration in all cardiac muscles, including bypass tracts (class III activity). It also has a powerful class I antiarrhtythmic effect that works by inhibiting inactivated sodium channels at high stimulation frequencies.

Amiodarone slows phase 4 depolarization of the sinus node as well as conduction through the atrioventricular node. It also decreases Ca2+ current (class IV effect) and transient outward delayed rectifier and inward rectifier K+ currents. Amiodarone noncompetitively blocks α- and β-adrenergic receptors (class II effect); this effect is additive to competitive receptor inhibition by beta-blockers. P.250

There is a risk of hypotension with intravenous amiodarone administration, more common with rapid administration. Other notable acute effects include bradycardia, hypokalemia, interactions with medications such as Coumadin and digoxin, and, rarely, torsades de pointe. There is a risk of pulmonary toxicity with high doses, starting with pneumonitis and leading to pulmonary fibrosis. Other organ systems affected by amiodarone therapy include the thyroid (hypothyroidism or hyperthyroidism), the central nervous system (proximal muscle weakness, peripheral neuropathy and neural symptoms), and the gastrointestinal tract (nausea 25%, elevated liver functions), and it may cause testicular dysfunction, corneal microdeposition, and photosensitive slate-gray or bluish skin discoloration.

Agents that can be considered as alternatives to amiodarone for the treatment of atrial fibrillation depend on the patient's cardiac history, as individuals with reduced left ventricular systolic function are especially prone to the proarrhythmic effects of certain antiarrhythmic agents. Some commonly used alternatives include ibutilide, dofetilide, and sotalol. Strong consideration should be given to obtaining consultation with a cardiac electrophysiologist before initiating these agents.

Ibutilide prolongs repolarization by inhibition of the delayed rectifier potassium current (/kr) and by selective enhancement of the slow inward sodium current. This drug is efficacious in the termination of atrial fibrillation and flutter with both single and repeated intravenous infusions. It is as effective as amiodarone in cardioversion of atrial fibrillation. The proarrhythmic effect resulting in torsades de pointe is higher in individuals with heart failure, those with bradycardia, nonwhite subjects, women, and in those given the drug for atrial flutter rather than atrial fibrillation. The risk of this is greatest during or shortly after the infusion of the drug (within 1 hour) and wanes rapidly after administration because the half-life (2 to 12 hours) of this agent is short. The patient should be monitored for at least 4 hours after the start of the infusion.

Dofetilide prolongs the action potential and QTC in a concentrationrelated manner. Dofetilide exerts its effects solely by inhibition of the rapid component of the delayed rectifier potassium current /kr. Dofetilide has stronger evidence in its favor for acute cardioversion of atrial fibrillation than for maintenance thereafter, according to a meta-analysis. It can be given to patients with depressed function but needs to be initiated while being continuously monitored on telemetry for the first 3 days of therapy because this too, like ibutilide, carries a risk of proarrhythmia. The risk of torsades de pointes can be reduced by establishing or maintaining normal serum potassium and magnesium levels, predose adjustment of renal function, and postdose reduction based on QT_C (ideally, baseline QT_C < 429 milliseconds). Administration of dofetilide requires that the hospital and the prescriber be trained as confirmed administrators. Further information can be found at www.tikosyn.com. P.251

Sotalol has combined class II and class III properties, is active against a variety of arrhythmias, and has the ability to produce profound bradycardia or prolongation of the QT interval. Of the many indications, sotalol is most commonly used for maintenance of sinus rhythm after cardioversion for atrial fibrillation and for reducing ventricular tachyarrhythmias. Despite its ability to prevent tachyarrhythmias, sotalol (like any other antiarrhythmic) can also be proarrhythmic because of its ability to profoundly prolong the QT interval. As a result, initiation of this drug should occur while the patient is closely monitored. Sotalol is contraindicated in patients with reduced creatinine clearance (<40 mL/min) and asthma. It should be avoided in patients with serious conduction defects, in patients with bronchospastic disease, and when there are evident risks of proarrhythmia.

The use of these antiarrhythmic medications must be undertaken in a clinical environment with the appropriate resources to deal with the complications that may accompany their use. Specifically, these drugs should be used only in care settings with immediately available emergency resuscitation equipment (e.g., a code cart with emergency drugs, airway mechanisms, and pacing devices) as well as personnel appropriately trained (e.g., anesthesiologists, intensivists, or cardiologists) to use this equipment.

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Use Extreme Caution When Administering Milrinone in Renal Failure

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MECHANISM OF ACTION

Milrinone is a selective inhibitor of peak cAMP phosphodiesterase III isozyme in cardiac and vascular smooth muscle. Its inhibitory action on phosphodiesterase results in increased cAMP levels, which in turn increases contractility in cardiac muscle and stimulates vasodilation in blood vessels. This causes an increase in cardiac output and a decrease in pulmonary wedge pressure. These hemodynamic changes are obtained without excessive changes in heart rate or increase in myocardial oxygen consumption.

INDICATIONS

Milrinone's use has been best studied in patients with congestive heart failure. It appears to be very efficacious in nonhypotensive patients with acute nonischemic cardiomyopathy despite treatment with diuretics. These patients benefit from an enhancement in contractility and afterload reduction. Additionally, milrinone is known to provide enhanced diastolic function. Duration of therapy should last for 48 to 72 hours. There are no studies to date that support its use for a longer period. Long-term oral therapy with milrinone has been associated with increased mortality.

DOSE

The recommended dose in patients with normal renal function is a 50-mcg/kg bolus followed by a continuous infusion at 0.375 to 0.75 mcg/kg/min. Because milrinone is excreted mainly through the kidneys, its dose in patients with renal impairment should be adjusted accordingly (by surface area) (CrCl denotes creatinine clearance):

CrCl 50 mL/min/1.73 m^2: Administer 0.43 mcg/kg/min.

CrCl 40 mL/min/1.73 m^2: Administer 0.38 mcg/kg/min.

CrCl 30 mL/min/1.73 m^2: Administer 0.33 mcg/kg/min.

CrCl 20 mL/min/1.73 m^2: Administer 0.28 mcg/kg/min.

CrCl 10 mL/min/1.73 m^2: Administer 0.23 mcg/kg/min.

CrCl 5 mL/min/1.73 m^2: Administer 0.2 mcg/kg/min.

An immediate improvement in hemodynamics is seen within 5 to 15 minutes after initiation of therapy. The mean half-life of milrinone is approximately 2.4 hours, and patients reach a steady-state plasma milrinone concentration (200 ng/mL) within 6 to 12 hours of a continuous maintenance infusion of 0.50 mcg/kg/min. The impact of the half-life of milrinone is clinically important, as the effects of the drug cannot be "turned off" rapidly as can those of many of the other commonly used inotropic agents, such as epinephrine.

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CAUTION

Milrinone decreases atrioventricular nodal conduction time, allowing a potential for increased ventricular response rates (up to 3.8%) for patients with supraventricular arrhythmias. Ventricular arrhythmias (ventricular ectopy, sustained and nonsustained ventricular tachycardia) have also been reported in up to 12% of patients. Life-threatening ventricular arrhythmias appear to be related to the presence of other underlying factors such as a pre-existing arrhythmia and/or metabolic abnormalities. Extreme caution must be used in patients with renal compromise. Fatal ventricular arrhythmias developed in six of nine patients in a recent study of the pharmacokinetics of milrinone in patients on continuous venovenous hemofiltration (CVVHD). These patients were oliguric (<400 mL/24 h) and had a serum creatinine of >2.0 mg/dL. All patients received 0.25 mcg/kg/min of milrinone and developed a mean steady-state concentration of 845 ng/mL; four times higher than that of patients with normal renal function. The high protein-binding affinity of milrinone and decreased urinary excretion could have contributed to this high concentration, thereby leading to the increased incidence of ventricular arrhythmias.

TAKE HOME POINTS

- Milrinone is a phosphodiesterase inhibitor that acts primarily on cardiac and vascular smooth muscle. Its effects are to enhance diastolic function, increase cardiac output, and decrease pulmonary wedge pressure.
- Milrinone is given intravenously for a period of 48 to 72 hours. It has a mean half-life of 2.4 hours—it cannot be turned off as most inotropes can.
- Extreme caution must be used in patients with renal failure because of the potential development of life-threatening arrhythmias.
- For patients with renal compromise, the milrinone dose must be adjusted on a sliding scale.

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Use Meperidine with Caution

Neil B. Sandson MD

Meperidine (Demerol) was the first phenylpiperidine analgesic to come onto the market in the United States. Through the years it has been used extensively as a narcotic analgesic as well as a favored drug for sickle-cell crises, shaking chills, and various surgical uses. However, in more recent years, a growing number of clinicians are recognizing that the benefits of meperidine probably do not justify the risks.

Meperidine has disadvantages compared to other narcotics, even when given as monotherapy. It will provide adequate analgesia for the surgical patient on initial dosing, but the risk-benefit ratio then shifts somewhat. Meperidine has a half-life of only 3 to 5 hours. The primary metabolite, normeperidine, has a half-life of 15 to 20 hours, but it is only half as effective an analgesic as meperidine. A significant problem is that normeperidine has neuroexcitatory properties. So, when meperidine is given in repeated doses, which is often required given the relatively short-lived nature of its analgesic effect (for postoperative pain), there is a tendency for patients to experience irritability, tremors, muscle twitching, and new-onset seizures. Normeperidine toxicity can even prove fatal, especially in individuals with hepatic and/or renal impairment, because of their greater susceptibility to normeperidine accumulation.

Other problematic issues with the use of meperidine involve the likelihood of troublesome drug-drug interactions (DDIs). The first set of DDI concerns that must be considered by the anesthesia provider are pharmacodynamic in nature. As a serotonergically active agent, meperidine can act synergistically with other serotonergic drugs (such as fluoxetine, paroxetine, sertraline, and other selective serotonin reuptake inhibitors) or with monoamine oxidase inhibitors (phenelzine, tranylcypromine, selegiline, and linezolid) to produce a potentially lethal central serotonin syndrome (Table 61.1). This was the primary cause of the death of Libby Zion in the 1984 case that generated national attention for a number of years. She visited a New York emergency department and received meperidine for her shaking chills, even though she was known to be taking phenelzine. She developed a fever of 107.6°F in the course of her serotonin syndrome and eventually expired. Her death then became a rallying point around the issue of house staff work hours, as it was suspected that the resident who ordered the meperidine would have known better had he been better rested.

TABLE 61.1 MEPERIDINE DRUG-DRUG INTERACTIONS (DDIs)

MECHANISM OF DDI	INTERACTING DRUGS	CLINICAL CONSEQUENCES
Pharmacodynamically synergistic serotonergic effects	Selective serotonin reuptake inhibitors (citalopram, escitalopram, fluoxetine, fluvoxamine, paroxetine, sertraline)	Central serotonin syndrome (nausea, diarrhea, fever, flushing, autonomic instability, diaphoresis, myoclonus, seizures, coma, death)
	Monoamine oxidase inhibitors (linezolid, phenelzine, selegiline, tranylcypromine)	
	Theoretically, other serotonergically active drugs (dextromethorphan, tricyclic antidepressants, venlafaxine, etc.)	
Inhibition of P450 2B6 and/or 3A4	Azole antifungal agents (fluconazole, ketoconazole, etc.)	Excessive narcotic effects (sedation, respiratory depression, delirium)
	Cimetidine	
	Ciprofloxacin	
	Diltiazem	
	Erythromycin	
	Fluoxetine	
	Fluvoxamine	
	Paroxetine	

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	Protease inhibitors (acute ritonavir, saquinavir, etc.)	
	Sertraline	
	Verapamil	
Induction of P450 2B6 and/or 3A4	Barbiturates Carbamazepine Phenytoin Prednisone Rifampin Ritonavir (chronic) St. John's wort	(a) Decreased analgesia; (b) increased production of neurotoxic normeperidine metabolite

Pharmacokinetic DDIs are also quite problematic when using meperidine. Meperidine is metabolized (demethylated) into normeperidine primarily by the cytochrome P450 enzymes 2B6 and 3A4. This gives rise to two main concerns. First, any of the numerous *inhibitors* of these enzymes are likely to yield excessive narcotic effects, such as oversedation, respiratory depression, and delirium. Inhibitors of these enzymes include such drugs as azole antifungals, ciprofloxacin, diltiazem, erythromycin, fluoxetine, paroxetine, protease inhibitors, sertraline, and verapamil, to name a few (Table 61.1).

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The second of these pharmacokinetic concerns involves interactions with enzymatic *inducers* of these P450 enzymes. These inducers generate both decreases in meperidine levels, with associated loss of analgesic efficacy, as well as increased production of the normeperidine metabolite with its associated neurotoxicity. Inducers of these P450 enzymes include barbiturates, carbamazepine, phenytoin, prednisone, rifampin, ritonavir (chronically administered), and St. John's wort.

There are anesthesia providers who “like” meperidine, have used it often, and feel they have never had a patient with a significant interaction. There is also the occasional patient who claims that only meperidine helps him or her with an acute or chronic pain syndrome. Finally, it is still not infrequent that an anesthesia provider will get a call from the postanesthesia care unit (PACU) asking for a verbal meperidine order for a patient who is shivering. *In any of these situations, it is incumbent that the provider ascertain that there is no possibility of a significant pharmacodynamic or pharmacokinetic DDI.* Failing to do so will both endanger the patient and leave the practitioner open to medical and legal censure, should an interaction arise.

One final note: In late 2006, this author's hospital removed meperidine from its formulary, as have many other hospitals. With the availability of safe, effective warming devices to combat patient shivering and newer-generation analgesic drugs, it was the consensus opinion of the staff physicians that meperidine was no longer a necessary agent in the pharmacologic armamentarium.

TAKE HOME POINTS

- Meperidine has a short half-life (3 to 5 hours). Its primary metabolite, normeperidine, has much less therapeutic value and a half-life of 15 to 20 hours.
- Even with monotherapy, neurotoxicity is a concern with repeated doses.
- Patients on multidrug therapy including meperidine are at risk for both pharmacodynamic and pharmacokinetic DDIs.
- *Never* give meperidine to a patient who is taking a serotonergic drug or a monoamine oxidase inhibitor.
- Meperidine is metabolized by the P450 3A4 and 2B6 enzyme subsystems.
- Pharmacokinetic DDIs involve the interaction of both inhibitors and inducers of these enzymes.
- Do not give a verbal order for meperidine unless you have verified that the patient is not being co-administered any of the drugs in Table 61.1.

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Wash Your Hands!

James W. Ibinson MD, PhD

David G. Metro MD

You've heard it since you were too young to remember: *"Wash your hands!"* Despite that, many anesthesia providers practice poor habits in this area. Although most anesthesiologists on direct questioning will "know" the benefits of good hand hygiene, their habits may leave something to be desired. We must remember that hand hygiene (handwashing *and* glove use) benefits not only you but also your patients.

Although the absolute indications for ideal handwashing practice is not known (because of the lack of controlled studies), the Centers for Disease Control and Prevention (CDC) state that health care workers (HCWs) should "decontaminate hands before having direct contact with patients," after "contact with body fluids or excretions, mucous membranes, non-intact skin, and wound dressings," and even after "contact with a patient's intact skin" (Table 62.1). These recommendations are based on their findings that "Handwashing is the single most important procedure for preventing nosocomial infections" (Boyce and Pittet, 2002).

The American Society of Anesthesiologists (ASA) has also published guidelines for hand hygiene using the above findings as justification. Stating that "strict adherence to handwashing and aseptic technique remains the cornerstone of prevention of catheter-related infections," the ASA recommends that hands be washed before and after any patient contact, as indicated in Table 62.2. Their statement details the use of both hand cleansing and glove use. For this discussion, both will be included in the term "hand hygiene."

As obvious as the above standards may seem, anesthesiologists have repeatedly been shown to be among the worst of physicians for adherence to hand hygiene standards. Pittet et al. (2004) demonstrated that anesthesiologists showed proper hand hygiene only 23% of the time. In a survey of anesthesiologists in the United Kingdom, El Mikatti et al. stated that only 36.4% washed their hands between cases. Studies show that anesthesiologist hand hygiene rates increase, however, when they perceive an infectious threat. Tait and Tuttle (1995) showed that when treating patients thought to be carrying HIV or HBV, 95% of HCWs washed their hands, versus only 58% of HCWs for the exact same patients when knowledge of HIV or HBV status was not known. This suggests the HCWs understand the importance of hand hygiene in preventing the transmission of disease but fail to appreciate its importance in routine contact. Although it is noted that these studies may be somewhat dated, clear evidence that hand hygiene has improved within anesthesiology is not available.

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TABLE 62.1 CDC RECOMMENDATIONS FOR ACTIVITIES THAT REQUIRE HANDWASHING

1)	Decontaminate hands before having direct contact with patients.
2)	Decontaminate hands before donning sterile gloves when inserting a central intravascular catheter.
3)	Decontaminate hands before inserting indwelling urinary catheters, peripheral vascular catheters, or other invasive devices that do not require a surgical procedure.
4)	Decontaminate hands after contact with a patient's intact skin (e.g., when taking a pulse or blood pressure, or lifting a patient).
5)	Decontaminate hands after contact with body fluids or excretions, mucous membranes, nonintact skin, and wound dressings even if hands are not visibly soiled.
6)	Decontaminate hands if moving from a contaminated-body site to a clean-body site during patient care.
7)	Decontaminate hands after contact with inanimate objects (including medical equipment) in the immediate vicinity of the patient.
8)	Decontaminate hands after removing gloves.
9)	Before eating and after using a restroom, wash hands with a nonantimicrobial soap and water or with an antimicrobial soap and water.

The reasons for this poor compliance have been studied extensively and include logistical barriers (sink/antiseptic hand-rub location) and a heavy workload resulting from overcrowding and understaffing of patient care areas. These issues need to be addressed on an administrative as well as an individual level. The open layout of most postanesthesia care units (PACUs) and many intensive care units (ICUs) also seems to lower hand hygiene compliance. Pittet et al. (2003) found that only 19.6% of HCWs cleaned their

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hands properly when admitting a new patient to the PACU.

TABLE 62.2 SUMMARY OF RECOMMENDATIONS OF THE ASA FOR HAND HYGIENE

1) Wash hands before performing invasive procedures.

2) Wash hands after touching blood, body fluids, secretions, excretions, and contaminated items, whether or not gloves are worn.

3) Wash hands immediately after gloves are removed, between patient contacts, and when otherwise indicated to avoid transfer of micro-organisms to other patients or environments.

4) It may be necessary to wash hands between tasks and procedures on the same patient to prevent cross-contamination of different body sites.

5) Waterless handwashing solutions may be considered when the anesthesiologist cannot leave the room.

6) Appropriate barrier precautions such as gloves, fluid-resistant masks, face shields, and gowns must be used routinely with all patients.

7) Remove gloves and gowns promptly after use, before touching noncontaminated items and environmental surfaces, and wash hands before seeing another patient.

Anesthesiologists who do not follow recommended hand hygiene practice in terms of glove use most commonly cite a loss of touch as the reason. Several studies have investigated this claim. Tiefenthaler et al. (2006) showed that touch sensitivity as tested by filament force on the fingertip required for sensation is indeed reduced with either standard single-use protective gloves or sterile surgical gloves. Two-point discrimination, on the other hand was not significantly different. No difference was found between the two glove types. Kopka et al. (2005) examined single-use protective gloves, standard sterile surgical gloves, and extrathin surgical gloves in terms of skin-pressure sensation. They found no significant difference between standard protective and surgical gloves but did note an improvement when using extrathin surgical gloves. Although these studies may support the case against glove use in terms of sensation, arguments for routine use are strengthened by the finding that approximately 10% of all patients undergoing surgery can be infected with infectious viruses or methicillin-resistant *Staphylococcus aureus*, and that *glove use can prevent up to 98% of blood-borne pathogens*. CDC and ASA recommendations on this are clear, and the Occupational Safety and Health Administration (OSHA) requires gloves as part of its standard precautions.

TAKE HOME POINTS

- Both the CDC and the ASA recommend that hands be cleaned before and after all contact with patients and potentially when touching two areas of the same patient.
- Anesthesiologists are not exempt from these recommendations but have been shown to be among the least compliant practitioners.
- Although glove use has been shown to decrease touch sensitivity, the benefits of their use should outweigh this concern.
- Mom was right: Wash your hands! (Even when you don't know that the patient has an infection.)

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Never Rush Through A Sign-Out

John T. Bryant IV MD

As symbolized by the seal of the American Society of Anesthesiologists (ASA), the practice of anesthesia is a profession based on vigilance. Attention to detail and continuous situational awareness are essential to anticipate and avoid preventable complications. Similar to other professions requiring constant vigilance, it is generally accepted that periodic relief of anesthesia providers is necessary to promote patient safety. Extrapolating from data in their landmark 1978 study regarding preventable mishaps in the practice of anesthesia, Cooper et al. found that the provision of periodic breaks to anesthesia personnel during long procedures had a favorable effect on preventing critical incidents by allowing their (earlier) detection by the relieving anesthetist. This finding has not been universal, as some evidence shows that continuity of care throughout the course of an anesthetic case decreases the risk of perioperative morbidity and mortality. Though examples have been observed of critical incidents attributed to, or perpetuated by, the exchange of personnel, most of these can be avoided by a detailed and inclusive personnel exchange protocol. This chapter presents a stepwise approach to organizing essential data during personnel exchanges to enhance patient care focusing on three critical categories: patient factors, surgical procedure factors, and key elements of the anesthetic plan.

Effective July 1, 2004, all Joint Commission on Accreditation of Healthcare Organizations (JCAHO)-accredited surgical facilities became required to adopt the Universal Protocol to Prevent Wrong Site, Wrong Procedure, and Wrong Person Surgery. This “time-out” protocol provides a point of embarkation in the initiation of sign-out protocol between anesthesia providers. Confirming demographic information including age, gender, race, and name establishes the context in which all further information can be understood. Included in the introduction is mention of any medical allergies unique to the patient. Furthermore, it is essential that the replacing anesthetist be versed in what surgical procedure is being performed and its indications in order to anticipate anesthetic needs.

Anesthetic technique is dictated not only by surgical requirements but also largely by co-morbid conditions. Consequently, the next logical piece of information to cover deals with the patient's relevant past medical history. The relieving anesthetist should assume care of the patient only after acquiring a thorough grasp of his or her medical problems, concentrating
on cardiovascular, pulmonary, renal, and neurologic status. Attention should be paid to objective measures of physiologic organ system function such as P.265
pertinent laboratory values, radiologic studies, and baseline vital signs. Anticipating and avoiding preventable morbidity hinges on planning around pitfalls inherent to the patient's physiologic state. Knowledge of these co-morbidities helps explain the medications that the patient chronically takes that may compliment or complicate anesthetic management. Discovering that a patient is inadequately treated for a certain medical condition may help to modulate the anesthetic plan and thereby avoid morbidity.

The logic of the anesthetic plan should be apparent after establishing what is being done, to whom it is being done, and what special challenges are presented by the patient. To ensure that critical information is not overlooked, it is useful to conduct the discussion of anesthetic technique in chronological order. Beginning with induction, it is essential that the replacing anesthetist have a thorough understanding of what technique was used. Critical items to note include the mode (e.g., mask induction, intravenous induction, or rapid sequence induction) and the medications used.

Airway management is the next critical factor to discuss. If a mask airway was utilized during induction, it is important to note the ease of ventilation and the need for airway adjuncts. The relieving anesthesia provider should be informed of the specifics of the definitive airway plan: The device, its size, depth, and ease of placement are all critical factors. If direct laryngoscopy was conducted, the outgoing anesthetist should highlight what kind of blade was utilized and what his or her view was of the glottic inlet.

The relieving anesthetist will need to have an understanding of the type and indication for the monitors that are utilized. If any indicated monitors were omitted, the original anesthetist should be prepared to articulate the reasoning behind the omission. The relieving anesthetist should be aware of the size and location of invasive devices such as venous access lines, arterial lines, and central venous cannulae. It is incumbent on both anesthetists to confirm that all lines are patent so that fluids, blood products, and medications reach the vasculature as intended. Closely related to the issue of invasive devices is a summary of the amount and type of fluids given, estimated blood loss, and urine output up to the point of personnel exchange.

Athoughtful sign-out should include a summary of important medications administered during the procedure thus far. Important medications to note include premedications given before induction, as these may later have an effect during emergence. Reviewing the dose and timing of antibiotics given may not only be a cue to administer an overlooked medication but also prompts the relieving anesthetist of the possibility that antibiotics may need to be readministered before the close of a lengthy procedure. The relieving anesthetist will need to be aware of the dose and timing of narcotics
and antiemetics, as these also have effects at the time of emergence. Viewed in light of the patient's co-morbidities, the administration of any vasoactive P.266
medications is of primary importance. Finally, the use and continued need for neuromuscular junction blockers is critical because they can have significant effects on surgical conditions and emergence.

Afinal topic of discussion pertaining to the anesthetic plan relates to preparing for the end of the procedure. The relieving anesthetist should know the estimated time and major events remaining in the procedure. He or she will need to continue or modify the existing plan for postoperative pain and nausea. Also, he or she will need to plan for disposition of the patient. Whether the patient will be extubated is a key planning factor. If the patient is to be extubated, does the nature of the surgery require a deep extubation, or do patient-specific factors necessitate an awake extubation? Finally, the relieving anesthetist should understand where the patient is to go following the procedure. If the patient is to be admitted, the relieving anesthetist should ensure that a bed in an appropriate ward or unit has been procured.

After gaining a firm understanding of the patient, the procedure, and the anesthetic plan, it is essential that the relieving anesthetist confirm the presence of emergency medications, equipment, and contacts. Inappropriately labeled syringes have been shown to be a common source of preventable error during exchanges of personnel. Such errors have been attributed to the lack of standardization in labels and doses of medications. It is incumbent on the relieving anesthetist to check the identity and concentration of prepared medications and correct any discrepancies. Similarly, the relieving anesthetist will want to confirm the location and functionality of the laryngoscope and accompanying blades. Additional endotracheal tubes of appropriate sizes with stylets should be readily available. If blood products are anticipated to be required, the relieving anesthetist should inquire about their availability. Afinal essential piece of information is an understanding of who is available for assistance. The relieving anesthetist should not allow his or her predecessor to

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leave the room without handing down the names and contact numbers for the attending anesthesiologist and anesthesia technician.

TAKE HOME POINTS

- Vigilance is the hallmark of the practice of anesthesia.
- Periodic short breaks during prolonged procedures can enhance patient safety by giving the anesthetist an opportunity to rest and renew focus. They also afford a new perspective on a case and help to discourage complacency.
- To enhance rather than hinder patient care, exchanges in anesthesia personnel must be accompanied by a thorough and thoughtful sign-out. Exchanges of information between providers can be highly variable.
- To promote a thorough, efficient, and retainable exchange, anesthetists should make an effort to give and receive their report free from interruption.
- Other elements that may improve sign-out effectiveness include using a checklist, taking notes, and having the relieving anesthetist read back information to the outgoing anesthetist to ensure comprehension.
- Any successful sign-out protocol must include pertinent discussions of the patient, the procedure, and the plan.
- Approaching the sign-out in a logical manner can assist the anesthetist in assimilating a large body of critical information in an effective, efficient, and safe manner.

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Preoperative Anxiolysis: It's Not Just "Two of Midaz"

Michael P. Hutchens MD, MA

William Thomas Green Morton did not meet his patient before their encounter in the Ether Dome on October 16, 1846, as he was running late. Robert Hinckley's painting of the scene depicts an anxiety-producing environment—the patient strapped to a chair, the focus of attention in an auditorium full of surgeons. Gilbert Abbott might have benefited from 2 mg of midazolam, had it been available; but perhaps he would then have been unable to tell the audience, as he did immediately postoperatively, that the operation had been painless. As far as history records, his perioperative experience was satisfactory to him, entirely without premedication or a preanesthetic interview. Nonetheless, the preoperative anxiety of Gilbert Abbott and that of modern patients has implications for patient satisfaction and for patient care and physiology. Preoperative anxiety is common—conservatively 25% and up to 80% in some studies. Concerns associated with anxiety include postoperative pain, incapacitation, and death. Preoperative anxiety is correlated with delayed gastric emptying, with increased intraoperative heart rate and anesthetic requirements, and with increased postoperative pain scores.

It remains unclear what preoperative strategy is most effective, in most patients, in reducing perioperative anxiety. Based on a 1997 survey, most U.S. anesthesiologists give anxiolytic medication as part of this therapy, but there are significant geographic, patient age, and hospital size variations. There is no consensus in the literature, and the number of regimens investigated is large and diverse. What is most clear is that patients have increased anxiety before operations, and that interventions performed by anesthesiologists can reduce that anxiety.

An oft-cited study purporting to show that the anesthetic interview is more effective than pentobarbital premedication was performed in 1963, but this study was methodologically flawed. The most anxious patients (those who kept the interviewer past the allotted time) were dropped from the study, but only in the interview arm. There was no assessment of baseline (prehospital) anxiety and no objective measure of anxiety. All patients received intramuscular atropine before assessment, patients were not aware they were part of a study despite being administered study medications, and it is unlikely that it is possible to blind an observer to whether a patient has
received pentobarbital, as investigators claimed to have done. The literature on premedication and preoperative anxiolysis is rife with similar P.269
methodologic problems (although, thankfully, not the absence of consent). However, at least one well-designed study shows that a visit from an anesthesiologist can play a significant role. In a 1977 study conducted in Britain, Leigh et al. used an objective psychometric questionnaire to assess baseline preoperative anxiety and that after no intervention, a preoperative visit, or viewing a 10-page booklet on anesthesia. All patients had more than normal anxiety preoperatively. The preoperative visit was significantly more effective in reducing anxiety than the booklet or no intervention.

Although the content and tone of the preoperative visit have not been objectively evaluated, several basic principles of physician-patient interaction apply. There is no other physician-patient interaction in which the conscious, competent patient has greater reason for anxiety and is meeting a physician for the very first time. Patient concerns and anxiety must be taken seriously. Although some of these concerns may seem trivial or unusual, they are foremost in the patient's mind and need to be addressed with compassion. An example is fear of intraoperative awareness. Anesthesiologists know that intraoperative awareness is an extremely rare event, that it is almost never complete awareness, and that it is associated with certain situations and kinds of anesthetics. Laypersons do not have access to this knowledge, and the only information to which they may have access (perhaps a television program or a magazine article) will have presented a population in which 100% of subjects are affected ("victims" or "survivors") with the most extreme awareness and are hurt or disabled as a result. It may be a challenge to encourage patients to believe an unknown, new authority rather than one they trust and with whom they are familiar. No pharmacologic agent can substitute for this process; one must simply pay serious attention to patients, gain their trust, and state clearly that it is the anesthesiologist's central concern to assure their safety and comfort. Indeed, amnesia produced by preoperative medication may erode some of the benefit of such a conversation if it is not remembered postoperatively.

The obligation to take patient concerns seriously does not end on leaving the preop area. Patients en route to the operating room often express fears they may have been unwilling to voice in the presence of family or friends. These "corridor concerns" may be trivialized by the setting, but to the patient they may have the gravity of "last wishes," as they know their next experience will be anesthetic induction. If necessary, one should stop patient transportation long enough to confront these fears. In the operating room, in the bustle of activity before induction, patients may again express fear or discomfort resulting from cold, monitoring equipment (tightness of a blood pressure cuff, for example), or claustrophobia from the mask. Patients
will express anxiety about "misbehaving under anesthesia" or not having their dentures. Again, these concerns must be taken seriously. Each anesthesia P.270
provider eventually develops an individual style, but the fact that the patient's concern is taken seriously may have more effect than alleviating the discomfort that provoked it and probably is not dependent on any one thing the provider says. Tell patients that warm blankets are on the way, allow patients to hold their own mask, let them know that the blood pressure cuff is the cause of their arm discomfort and that the discomfort will go away. Ask if there is anything the staff can do to make them more comfortable before getting started, then tell them you are getting started, and that they are doing well. Children who have access to their parents during induction may have less anxiety, although at least one study has shown no difference between patients given premedicants and those with parental presence at induction. It is most likely that this is a function of population and that some children benefit from parental presence more than others.

Although the presence of the anesthesiologist is a significant factor in reducing preoperative anxiety, many other interventions may be useful. Most commonly, preoperative medication is employed. Midazolam is commonly used because it is relatively short-acting, has amnestic properties, is anxiolytic, and is available in an oral form that can be given to children. Other benzodiazepines are used, but their properties as preoperative medications differ. Lorazepam has the specific disadvantage of producing prolonged (up to 24 hours) cognitive dysfunction, most commonly in the elderly. Two studies show *increased* postoperative pain scores in patients who received preoperative diazepam. Many other classes of medication are employed for preoperative anxiolysis. Clonidine has been shown to reduce postoperative pain scores and intraoperative heart rate and blood pressure. Anticholinergics, butyrophenones, opioids, barbiturates, antihistamines, and antiseizure medications have all been used to reduce preoperative anxiety and have specific roles.

Finally, there are many other preoperative interventions that may reduce patient anxiety. For children, video games and even the presence of a clowndressed anesthesiologist are reported to reduce anxiety. These interventions, unfortunately, have not been studied in adults. Music, either of the patient's choosing, or of a "soothing" nature, has been shown to reduce preoperative anxiety as reported by the patient, as well as intraoperative bispectral index scores and anesthetic requirements. Physical correlates of anxiety such as plasma catecholamine levels and blood pressure are unchanged by

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music, but it certainly does not hurt. Hypnosis has also been shown in one study to reduce preoperative anxiety.

It is clear, certainly to patients, that preoperative anxiety is an important component of the perioperative experience. There are many effective interventions that may reduce this anxiety and lead to improved patient satisfaction. Most important of these is the compassionate understanding of a concerned anesthesiologist. Many medication regimens may be employed to augment the anxiolysis of physician counseling, and other interventions such as music, hypnosis, or parental presence for children may be helpful.

TAKE HOME POINTS

- Preoperative anxiety is common and distressing to patients.
- Preoperative anxiety may have deleterious physiologic effects intraoperatively.
- Patient concerns must be taken seriously and assuaged compassionately.
- A visit from an anesthesiologist is an effective way to reduce anxiety.
- Preoperative medication, music, hypnosis, and other interventions may all be effective in reducing preoperative anxiety.

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Recognize that Transport Is One of the Most Hazardous Intervals in the Perioperative Period and Prepare Accordingly

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Charles D. Boucek MD

Anesthesiology management includes the transport of patients (from the preanesthesia area or intensive care unit to the operating room, from the operating room to the intensive care unit or recovery room, and from many areas outside of the operating room where anesthesia is administered to the recovery room or the intensive care unit). During transportation, many avoidable pitfalls can arise.

Transportation of patients in the immediate perioperative period carries hazards due to both patient and equipment factors. Human factors and task loading also add to the risk. The anesthesia provider must manage a patient undergoing the transition from the anesthetized to the conscious state while also giving report, charting, removing stationary and applying portable monitors, and physically helping to move the patient. A transportation plan including the destination and route should be established and the patient, the anesthesia personnel, and all necessary equipment should be ready before transportation begins. A telephone call should confirm that the recovery destination is ready to receive the patient. The anticipated distance partially determines the equipment needed. All patients recovering from anesthesia should have supplemental oxygen. Monitoring devices, manual ventilators, intubation equipment, intravenous (IV) poles, portable infusion pumps, and medications may be needed for longer distances or critically ill patients. Useful medications include antiarrhythmics, agents to raise and lower blood pressure, sedation agents, and muscle relaxants. The added equipment and supplies complicate the move and may require more time or additional personnel.

During emergence from anesthesia, patients experience discomfort from both the surgical wound and airway devices. Nausea due to medications or movement, and gagging from pharyngeal stimulation, both can result in emesis and possible aspiration. Teeth clenching may limit the effectiveness of suctioning. Before the full return of situational awareness by the patient, attempts at self-extubation may occur. Self-induced corneal injury may occur when patients who have not yet regained full coordination attempt to rub their eyes. Patients may try to sit up or roll off the table. An individual located at the patient's head is at a mechanical disadvantage in trying to restrain a large patient. Available “safety straps” may also fail. P.273

Residual effects of anesthetics can lead to hypoxia and hypotension. Opioids and muscle relaxants decrease respiratory reserve, whereas antihypertensives, intravenous and inhalational anesthetics decrease blood pressure. The transition from controlled to spontaneous ventilation may be accompanied by heightened airway reflexes. Functional residual capacity and respiratory drives are reduced. Release of dissolved anesthetic gasses, especially nitrous oxide, from the blood may displace oxygen from the alveoli and lead to diffusion hypoxemia. For patients being transported to the intensive care unit while still anesthetized, it is essential that ventilation is controlled and that perfusion is adequate.

One of the most effective ways to avoid hypoxemia is to provide supplemental oxygen. Oxygen delivery systems include nasal cannulae, face masks, manual ventilation bags with face mask, endotracheal tubes or laryngeal mask airways. It has been shown that healthy patients (ASAI and II patients) transported with oxygen after undergoing general anesthesia were less likely to desaturate during transport as compared to healthy patients who did not receive oxygen. For healthy extubated patients, oxygen provided via nasal cannula or face mask may be sufficient. For the transportation of seriously ill, intubated, or pediatric patients (pediatric patients are more prone to desaturate, as they have higher ratios of minute ventilation to functional residual capacity compared to adults), the ability to provide positive-pressure ventilation is important. Before transport begins, the adequacy of the supply of oxygen (usually provided in an e-cylinder) should be checked *personally* by the anesthesia provider because unexpected events may prolong the transport. (Editor's note: Every single anesthesia provider with more than 2 minutes of experience can tell a story of asking the OR nurse to open the oxygen cylinder to 10 L/min, only to realize when the patient starts to desaturate going out the door that an empty tank has been put on the bed.)

Vital signs monitored routinely during the transportation of critically ill intubated patients include invasive or noninvasive blood pressure, pulse oximetry, and the electrocardiogram. Routine end-tidal carbon dioxide level is not essential except in situations in which this value is to be maintained within a narrow range. A portable transport monitor that is adequately charged is needed.

Moving the patient from table to stretcher requires personnel; a minimum of four individuals should be available to help move the patient, one at the patient's side opposite the stretcher, one lifting the feet; the third across the receiving stretcher. The forth supports the patient's head and controls the airway. For large or boisterous patients, additional help may be needed along with rollers or sliding devices. Confirmation that both the table and stretcher wheels are locked is important, but stretcher locks are notoriously unreliable. Safety straps should be removed just before transferring the patient to the stretcher. The move should be called by the person at the head using a “three count” to avoid inadvertent extubation or neck injury. The receiving stretcher should be of appropriate size and the height should be even with the operating table to avoid painful impacts from drops to a lower elevation. Dislodgement of endotracheal tubes, vascular access, and chest, gastric, wound, or bladder drains may occur. Many hospital beds used as stretchers have electric motors and short power cords. Often the most easily available outlet is located on the anesthesia gas machine. The bed may trip the circuit breaker, temporarily inactivating the gas machine's ventilator and monitors. P.274

Hallway obstructions, ramps, and elevators complicate transportation. Supplies should be sufficient to maintain the patient in the event of temporary elevator malfunction. Battery life for monitors and infusion pumps can become an issue. Elevators should be entered patient's feet first so that the patient's head is accessible to the elevator door. This facilitates reintubation, and cardiopulmonary resuscitation without needing to remove the patient from the elevator.

The stretcher is ordinarily pulled by the individual leading from the patient's feet. The speed should permit timely arrival; it should be controlled by the individual at the patient's head so that monitoring and manual ventilation can be provided. Top-heavy IV poles can fall on the patient and may require additional personnel. Portable suction is often inefficient when moving ventilated patient with chest tubes and large air leaks.

On arrival, rapidly depleting battery-powered devices should be connected to wall utilities and oxygen should be changed from tank to stationary supplies. Portable monitors and vasoactive infusions are replaced by those of the receiving unit. Avoid the temptation to remove all monitors simultaneously. The patient remains in the care of the transporting team until report is given and care assumed by the receiving personnel. Report should include identification

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of the patient and surgeon, the procedure, medications and allergies, complications, fluids, ventilator settings, and analgesic plan.

In anticipation of events that can occur during transport, one needs to have available resources to resolve the situation. In the transport of healthy patients, one may need to provide analgesics for pain. In the transport of critically ill intubated patients, one should be ready to provide for immediate stabilization of anticipated problems.

TAKE HOME POINTS

- Skills to accomplish safe and efficient transport are as critical to an anesthesia provider as airway and access skills.
- Start packing the transport bag or basket well before the end of the procedure. Err on the side of extra equipment and resuscitation drugs. Pack your kit the same way for each type of transport (PACU kit vs. ICU kit).
- Check personally to make sure that a report has been called, ventilator settings have been given, and there is enough oxygen in the tank to get the patient to the destination.
- Transfer from the operating table to the gurney is a prime time for losing lines. For big cases with a lot of invasive lines, one anesthesia provider transfers the head and airway and a second anesthesia provider transfers the lines (somebody else has to do the legs and feet, so call for lifting help if necessary). After transfer onto the gurney, check to make sure that stopcocks, transducers, PA catheter, etc., are intact.
- Sort out the IVs, i.e., “untangle the spaghetti,” before leaving the room. Always have ready access to a stopcock in case drugs have to be given during transport or immediately on arrival. Know which IV bag the stopcock is hooked up to and be able to get to the Cair clamp easily. Start the transport with a full fluid bag (you wouldn't start an important car trip with an empty tank of gas, would you?).
- Confirm respiratory effort in the hallways and elevators by whatever is your maneuver of choice. For example, ask the patient to cough or take a deep breath and show you “the fog” in the nonrebreather mask.
- Remember that the surgeons that are helping you transport will probably be walking too fast (especially if they are residents).
- After giving report in the postanesthesia care unit or the intensive care unit, always let the receiving team know you are leaving and ask if they have any issues they would like you to deal with before reporting back to the operating room.

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The Same Simple Mistakes at Induction (and Emergence) Happen Over and Over Again—So Develop a Checklist, and Make it Ironclad

Brandon C. Dial MD

Randal O. Dull MD, PhD

For the airplane pilot, the most critical moments of a successful flight are takeoff and landing. So it is with anesthesia—the most critical moments occur at induction and emergence of general anesthesia. Like the pilot with a preflight checklist, the successful anesthesiologist has a checklist of important items that are reviewed prior to every induction to avoid many of the common pitfalls that may occur at these crucial times. Preventable anesthetic mishaps are often caused by a lack of familiarity with anesthetic equipment and a failure to check the anesthesia machine for proper function.

INDUCTION

Although there are many such checklists, the following mnemonic has been used at our institution to help ensure that the anesthesia machine and other equipment are ready for use by the prepared practitioner.

S—Suction

O—Oxygen

A—Airway

P—Positive-pressure ventilation

M—Medications

M—Monitors

S is for suction, probably the most easily and hence most commonly overlooked piece of equipment necessary for a successful induction. Although it is not always used, its presence is crucial for patients at risk for emesis and aspiration of gastric contents. It should be turned on and readily available with an appropriate tip and suction strength (-125 to -200 cm H_2O). If you have checked your suction but then left the operating room (OR) for something, you must recheck it again right before induction.

O is for oxygen. The anesthesia machine should be checked to ensure that oxygen is being delivered from the wall outlets and that the flow meters for oxygen and air are working appropriately. A secondary source of oxygen should be available. This is accomplished most commonly by an auxiliary oxygen E-cylinder located at the back of the anesthesia machine. The valve

should be opened and the pressure checked to ensure that it is full and ready for emergency use. P.277

A is for airway equipment. Laryngoscope handles should be connected to blades to ensure that the light source is working. If it is not, the batteries or light bulb may need to be replaced. Several sizes of laryngoscope blades should be available, including both straight and curved blades. Despite the thorough preoperative airway exam, many difficult airways are realized *after* induction. Other airway devices such as oral and nasal airways for mask ventilation should be a close reach away. The Eschmann stylet (or Bougie) is a helpful airway tool for the unexpected difficult laryngoscopy. Assorted laryngeal mask airways should be ready for use. Other devices for the difficult airway are noted in the American Society of Anesthesiologists (ASA) emergency airway algorithm. These should be available and the practitioner familiar with their use, including supplies necessary to create a surgical airway. Preparation is paramount in the “can't intubate, can't ventilate” clinical emergency.

P is for positive-pressure ventilation. Failure to check the anesthesia machine for potential malfunction is a significant cause of anesthesia accidents. The anesthesia machine should undergo a leak test to ensure that positive-pressure ventilation can be provided reliably. Many newer machines have automated leak tests that require several minutes but minimal effort. In many older machines, a rapid leak test may be accomplished by turning the ventilator switch to the bag position, closing the APL (“pop-off”) valve and occluding the circuit at the Y-piece, then filling the breathing bag and circuit with the oxygen flush button to a level of 30 mm Hg. Maintaining a constant pressure with no leak is a rapid and reliable test that ensures your ability to deliver positive-pressure ventilation.

M is for medications. Induction drugs should be drawn up in sterile syringes. Double doses should be drawn of key induction agents in case they get dropped on the floor, the patient requires more, or a difficult intubation creates a prolonged induction process. *Never use anything that has been dropped on the floor.* Multidose vials should be cleaned with alcohol before needle insertion. Most induction drugs and muscle relaxants are stable in syringes for 24 hours, but use beyond that period should be discouraged because the risk of bacterial contamination increases. Obvious exceptions include propofol, which should be used within 6 hours after accessing the sterile vial. Emergency drugs for unexpected but common events such as hypotension should be drawn up. Ephedrine, phenylephrine, and epinephrine diluted to appropriate concentrations are common examples. Many practitioners consider succinylcholine to be an emergency drug for rapid treatment of laryngospasm.

M is for monitors. Standard ASA monitors including blood pressure, pulse oximetry, capnography, and temperature monitoring are mandatory for every general anesthetic. Another key monitor during induction is the twitch monitor. Its use after administration of a nondepolarizing neuromuscular blocker will ensure optimal intubating conditions. P.278

EMERGENCE

Avoiding common pitfalls during emergence from general anesthesia can be accomplished with a similar checklist.

Once again, having suction at the ready can prevent many problems during extubation. One of the most common causes of laryngospasm during

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emergence is pooling of secretions on the vocal cords. Patient transport at the end of a general anesthetic should include oxygen, whether or not extubation is planned. Check the oxygen E-cylinder to ensure that it has enough oxygen for the transport. An E-cylinder with 1,000 psig is half-full, and contains about 330 L of oxygen. If it is exhausted at a rate of 6 L/min, this amount will last 55 minutes.

After extubation, airway equipment including a mask and oral airway should be readily available. When transporting an intubated patient to the intensive care unit (ICU), a laryngoscope and endotracheal tube should be brought for emergency reintubation if an unexpected extubation occurs. Positive-pressure ventilation can be given by way of a portable breathing circuit. Common examples used for patient transport include Jackson Rees circuits (Mapleson Class A) and Ambu bags that allow the delivery of positive-pressure ventilation.

Medications, including crystalloid fluids, are especially critical for the transport of an intubated patient to the ICU. Sedation and muscle relaxation may be important. Pressor agents and additional intravenous (IV) fluids should be available for patients with cardiovascular instability. Clinical circumstances may require the need for beta-blockade or other antihypertensive treatment, or narcotics for treatment of surgical pain.

Once again, the twitch monitor can be crucial to avoiding airway compromise from continued neuromuscular blockade if muscle relaxation was administered and extubation is planned. Its use can apprise you of whether reversal agents are needed, and the effectiveness of the reversal agent after it has been given to ensure the patient wakes up without muscular weakness and airway compromise.

One last point for a successful takeoff and landing: know your patient's name. Your safe navigation of these critical tasks requires skill. Knowing your patient's name is part of the professionalism required of a perioperative physician.

TAKE HOME POINTS

- Suction, suction, suction!
- Always have multiple laryngoscopes available.
- Double-draw the crucial induction drugs—including, at a minimum, the induction agent and succinylcholine (if it is not contraindicated).
- Don't pick up anything from the floor and use it on the patient.
- Remember that emergence, in a sense, is induction in reverse. Redundancy of your critical components is just as important during this phase.

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Editors: **Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.**
Title: ***Avoiding Common Anesthesia Errors, 1st Edition***

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Never Fail to Report a Needlestick Injury

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According to the World Health Organization (WHO), an estimated 3 million people worldwide suffer from accidental needlestick injuries each year. The Centers for Disease Control (CDC) and the National Institute for Occupational Safety and Health (NIOSH) report that an estimated 600,000 to 800,000 needlestick injuries occur annually among health care workers in the United States. There are limited data regarding specific occupational hazards to anesthesia personnel. However, Greene et al., in a multicenter study, reported 1.35 contaminated needlestick injuries per 1,000 anesthetics administered, or 0.54 per 1,000 hours of anesthesia.

Accidental needlestick injuries in the health care setting have been associated with the transmission of up to 20 blood-borne pathogens, most commonly human immunodeficiency virus (HIV), hepatitis C virus, and hepatitis B virus. Although significant data exist regarding transmission rates based on documented needlestick injuries in the health care setting, exact information is unavailable because of the underreporting of needlestick injuries by health care workers.

Despite well-publicized institutional protocols for reporting needlestick injuries and the ready availability of postexposure prophylaxis and medical follow-up for health care workers, recent research demonstrates that underreporting continues to be a significant problem. In fact, in one report a surprising 60% to 95% of house staff failed to report needlestick injuries! In the United States, only 45% of anesthesia personnel who suffered a needlestick injury sought treatment! Surveys of anesthesiology residents and attending physicians revealed significant underreporting as well. Residents and attending physicians reported only 29% and 19% of needlestick and percutaneous injuries, respectively. Although needlestick injuries may be individually detrimental and potentially life-threatening, the underreporting of these injuries has the secondary effect of preventing precise calculations of disease transmission rates.

COMMON SCENARIOS ASSOCIATED WITH NEEDLESTICK INJURIES

Needlestick injuries may occur any time a needle is exposed. Engineering and technology have dramatically increased the safety of needle use, but these advances represent only one component of accidental needlestick prevention. Examples of safety features include built-in safety devices, such as needle shields, retractable needles, safety catheter encasement devices, and needleless intravenous (IV) delivery systems. P.281

However, accidental needlestick injuries continue to occur despite these advances in technology. These injuries most commonly occur during certain situations:

- During clean-up after use (such as starting an IV, phlebotomy)
- When sharps are disposed of improperly
- When a needle is being recapped
- While manipulating the needle in the patient
- While handling a device or passing a device to another health care worker during or after use
- While handling or transferring specimens
- During collision with a sharp or with a health care worker handling the sharp
- Between steps of a multistep process

Some needlestick injuries are considered to be “high risk.” These include injuries from hollow-bore needles, needles that are contaminated with blood, and needles that are blood-filled. Anesthesiology personnel are especially susceptible to the above high-risk situations. Prevention of needlestick injuries involves vigilance on the part of personnel handling sharps. Most injuries have been shown to arise from the use of needles attached to syringes, suture needles, hollow-bore needles, IV catheter-needle stylets, and epidural needles. The majority of contaminated needlestick injuries in anesthesia personnel are preventable. One multicenter study reported that 59% of all injuries, 68% of injuries from the use of hollow-bore needles, and 78% of highrisk contaminated percutaneous injuries were preventable.

HUMAN IMMUNODEFICIENCY VIRUS (HIV)

The risk of HIV transmission to health care workers after percutaneous exposure to HIV-infected blood has been estimated at approximately 0.3% percent, and among anesthesia personnel, an estimated 0.56 HIV infections will occur per year to anesthesia personnel in the United States. The transmission rate varies based on the seroprevalence of the patient population. Projections by Greene et al. cite the 1-year risk of an anesthesia provider contracting HIV from occupational exposure at 0.00013% to 0.3%, and the 30-year risk to be from 0.0038% to 0.94%.

Several factors have been associated with an increased risk of HIV transmission from a patient to a health care worker as a result of a needlestick injury. These factors include an increased quantity of blood transferred during the exposure, use of a larger-bore needle (gauge smaller than 18), deep injury, a device visibly contaminated with the patient's blood, exposure during a procedure that involved placing a needle into a source patient's vasculature, or exposure to a source patient in the terminal stage of acquired immunodeficiency syndrome (AIDS). It is likely that the immunologic status of the injured health care worker also plays a role in determining risk. P.282

Symptoms of primary or acute HIV infection are generally nonspecific, resemble flu-like symptoms, and can occur within days or weeks of the initial exposure. They typically include fever, rash, malaise, pharyngitis, lymphadenopathy, and headache. Some people experience severe symptoms after contracting HIV, whereas others experience no symptoms whatsoever. Given the non-specificity of symptoms, the only reliable method of diagnosing HIV infection is antibody testing, with subsequent confirmation by Western blot. Historically, antibody testing was effective after the “window period,” which

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ranges from 2 weeks to 6 months following exposure. Current enzyme immunoassays, however, are able to detect the HIV antibody within several days.

There are two primary approaches in managing contaminated needlestick injuries with regard to HIV. One approach involves immediate postexposure prophylaxis with antiretroviral therapy unless or until the source patient tests HIV-negative or circumstances surrounding the injury indicate a low possibility of transmission. The second approach involves the initiation of antiretroviral therapy if analysis of the exposure indicates a risk of HIV transmission, such as a positive HIV test in the source patient.

There is some evidence to suggest that immediate postexposure prophylaxis is associated with decreased risk of transmission, specifically in animal models. Factors influencing the risk of transmission in those models include viral load, interval between exposure and the start of therapy, duration of therapy, and selection of antiretroviral regimen. The data available on human subjects is far less extensive. A retrospective case-control study conducted by the CDC, specifically on health care workers, indicated an 81% reduction in HIV transmission risk with postexposure treatment with zidovudine. Definitive protection against HIV transmission, however, is not guaranteed with immediate antiretroviral therapy, as limited information exists regarding the optimal treatment regimen, period, or duration.

Current postexposure prophylaxis regimens include two antiretroviral agents or, based on the circumstances of the exposure, an expanded antiretroviral regimen involving a third drug. Although chemoprophylaxis may provide benefit, it is not without risk or side effects. Adverse effects range from mild symptoms such as nausea, diarrhea, and headache to more severe effects such as neutropenia, neuropathy, and hepatotoxicity. Two-drug regimens resulted in increased compliance and fewer adverse effects than expanded three-drug regimens. Additionally, antiretroviral drug resistance has become increasingly problematic, resulting in the use of combination or expanded antiretroviral therapy. P.283

HEPATITIS B VIRUS (HBV)

Hepatitis B virus is much more commonly transmitted via needlestick injuries than HIV, with transmission rates ranging from 2% to 40%, depending on the hepatitis B antigen status of the source patient. Interestingly, the transmission rate has actually experienced a significant decline over the last 20 years, due largely to more widespread hepatitis B immunization among health care workers. There are a number of other factors associated with the risk of transmission, including the patient's hepatitis B surface antigen titers and degree of contact with the patient's blood.

History of previous hepatitis B immunization should not be interpreted as absolute protection against transmission. The duration of immunity conferred by the vaccination has not been determined, and it may not develop after a primary series. Additionally, the effectiveness of the hepatitis B immunization may be reduced by a number of factors, including immunocompromise, obesity, advanced age, smoking history, improper vaccination storage, and location of vaccine administration. Health care workers with a history of vaccination but without measurement of anti-HBsAg titers during the previous 2 years should be evaluated for immunity after a needlestick injury or any other accidental exposure.

Symptoms of acute hepatitis B infection occur in one third to one half of infected people. These symptoms include jaundice, fever, nausea, and abdominal pain. The majority of acute infections resolve, but chronic infection may develop in 5% to 10% of infected people. One in five patients who develop chronic hepatitis B infections has a risk of developing liver cirrhosis, while 6% of chronically infected people may die from liver cancer.

Unlike the questionable effectiveness of postexposure prophylaxis in the prevention of HIV transmission, prophylaxis for hepatitis B virus with hepatitis B hyperimmune globulin and hepatitis B vaccine is >90% effective in preventing HBV transmission. Exposed health care workers should be tested for anti-HBsAg after exposure. Postexposure prophylaxis with HBV hyperimmune globulin and HBV vaccinations are based on the exposed health care worker's vaccination status, response to previous vaccinations, and serum anti-HBsAg titers. HBV hyperimmune globulin, if needed, should be provided within 24 hours of exposure, as efficacy may decline if treatment is postponed. HBV hyperimmune globulin should be followed within 7 days by HBV vaccine.

Health care workers who are vaccinated against HBV should be tested 6 months following primary vaccination to evaluate immunity. If they receive hyperimmune globulin, immunity testing may need to be postponed until 4 to 6 months later. If health care workers follow the recommended regimens following exposure, they are unlikely to become infected and may continue to participate in their normal professional and personal activities. P.284

HEPATITIS C VIRUS (HCV)

The rate of transmission of hepatitis C virus (HCV) has not been well established, but the prevalence of HCV in health care workers is slightly higher than in the general population. The annual incidence of acute infections of HCV is approximately 2% to 4% in health care workers who have a history of occupational exposure to blood, such as with a history of needlestick injuries. The 1-year risk of an anesthesia provider contracting HCV via occupational exposure ranges from 0.00084% to 0.21%, and the 30-year risk ranges from 0.025% to 6.12% based on HCV seroprevalence in the patient population. Among anesthesia personnel in the United States, an estimated 5.18 HCV infections are expected to occur per year. The incidence of anti-HCV seroconversion after accidental percutaneous exposure to HCV is approximately 1.8%, with transmission occurring primarily with the use of hollow-bore needles.

Symptoms of acute hepatitis C infection range from fever, jaundice, and malaise to fulminant hepatic failure. Many patients are asymptomatic during the acute phase. Patients may have elevated liver transaminases. Up to 75% to 85% of acute hepatitis C cases will progress to chronic hepatitis, with a smaller proportion of chronic patients developing cirrhosis or hepatic cancer. Given the nonspecificity of symptoms, HCV antibody testing must be performed to confirm the diagnosis.

Unlike HIV and HBV, there is no vaccine to prevent HCV and no recommended postexposure prophylactic regimens at the present time. There are limited data that support the use of interferon monotherapy early in the course of acute hepatitis C, specifically in the setting of elevated liver transaminases and viremia. This evidence indicates that use of interferon early in the course of acute hepatitis C may increase the rate of resolution of acute infection, potentially preventing progression to chronic infection. Unlike HBV, there is no evidence to support the use of immune globulin as part of postexposure prophylaxis.

If a health care worker is concerned about possible exposure to HCV, he or she should be tested immediately and then again after 6 to 9 months. If HCV is detected, or the individual develops any symptoms of hepatitis, liver function tests should be performed to evaluate for chronic hepatitis. Once the diagnosis of HCV is established, interferon therapy may be considered. Potential side effects of interferon therapy include mild to severe flulike symptoms, tissue damage at the site of injection, depression, diarrhea, pancytopenia, elevated transaminases, skin rash, hair loss, and edema.

Despite the potentially life-threatening consequences of developing HCV infection, at this time, health care workers with accidental exposures need not change their personal or professional activities. However, any health care worker who has experienced a needlestick injury or any percutaneous exposure to the blood of a patient with an uncertain or suspected HCV infection should undergo postexposure surveillance so that seroconversion can be detected early and managed appropriately. P.285

TAKE HOME POINTS

- Needlestick injuries expose health care workers to blood-borne pathogens that can cause potentially fatal diseases. Three of the most

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pathogens of most concern are HIV, HBV, and HCV.

- Advances in technology and the engineering of safety devices has dramatically reduced the incidence of needlestick injuries.
- Prevention is the best defense against needlestick injuries. Avoid risky behaviors such as needle recapping and transferring of specimens, pay increased attention to proper sharps disposal, and avoid using needles if an alternative is available.
- Needlestick injuries are grossly underreported by physicians, especially house staff. Unreported needlestick injuries could have devastating personal consequences and also prevent the precise calculation of transmission and seroconversion rates.
- *Always* report needlestick injuries! Although postexposure prophylaxis does not provide absolute protection against pathogen transmission, early detection and proper medical follow-up could save your life!

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Understand the Utility of Preoperative Stress Testing in Suspected Heart Disease

Matthew V. DeCaro MD, FACC

Although stress testing is quite useful in preoperative cardiac risk stratification, it is a flawed modality.

There are several serious cardiac complications that can result from non-cardiac surgery. These include ischemic events, arrhythmias—both bradyand tachyarrhythmias—and heart failure. Although stress testing can give some insight into an individual's propensity for arrhythmias and heart failure, its role is quite limited. By and large its main use is in the diagnosis of clinically significant coronary artery disease (CAD). Much attention has therefore been focused on stress testing, because the most catastrophic cardiac complication of surgery, myocardial infarction (MI), with the attendant permanent loss of cardiac muscle and thus function, is caused by CAD.

The sensitivity and specificity of exercise stress testing for diagnosing coronary disease varies with the severity of this disease. Table 68.1 gives average results for various severity subsets across studies.

As can be seen, the test is more useful in severe forms of CAD. It is in these patients that prior intervention (regardless of the proposed surgery) may improve survival. Remember that the greater the sensitivity of a test, the more useful it is for excluding a diagnosis when the test is negative. More specific tests help in establishing a diagnosis when the test is positive. The main utility of the stress test in the context of risk stratification for surgery is in excluding severe CAD in a patient with a negative test. To add to the confusion, many of these statistics are from specialized centers under controlled circumstances. Depending on the population studied, the accuracy of stress testing may be much poorer. In several community hospital studies in a general medical population, the sensitivity was substantially worse (<50%).

The posttest likelihood of CAD is highly dependent on the pretest likelihood. If the pretest probability of CAD is either very low or high, the test provides little additional information. Anegative test in a 70-year-old hypertensive diabetic with typical anginal chest pain is not helpful for excluding the diagnosis of CAD. It can be argued that it decreases the statistical chance of severe three-vessel or left main disease, but recall that one of the causes of a false-negative nuclear scan is global ischemia, usually caused by critical left main stenosis.

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TABLE 68.1 AVERAGE RESULTS FOR VARIOUS SEVERITY SUBSETS

	SENSITIVITY	SPECIFICITY
Single-vessel CAD	68	77
Multivessel CAD	81	66
Three-vessel/left main CAD	86	53

CAD, coronary artery disease.

Unfortunately, although there is a general correlation between severity of CAD and operative morbidity and mortality, it is an imprecise predictor of acute coronary syndromes (ACSs). These include unstable angina, non-ST-segment-elevation MIs (NSTEMI) and ST-segment-elevation MIs (STEMI). It should be recalled that one is just as likely to experience an ACS from a plaque that results in a stenosis ≤50% as it is from a stenosis >50%. This is due to several factors. The primary factor that determines plaque instability is not the severity of the narrowing. Although the physical bulk of the plaque plays a role, other factors such as erosion of the fibrous cap overlying the plaque are more important. Vessel inflammation, of both the humoral and cellular components, are primarily responsible for the thinning, erosion, and ultimately rupture of this cap. This is the proximate cause of the ACSs. Hence, stress testing is an imprecise indicator of an imprecise predictor for ACS! Stress testing can be expected to be much better at predicting what it measures —namely, coronary stenoses of >70% that would produce flow-limiting ischemia, i.e., angina pectoris triggered by physiologic stress.

Pharmacologic testing is only slightly less sensitive and specific than exercise modalities, so the same analysis as above applies here as well. The major disadvantage of these techniques is the loss of clinical information related to the assessment of exercise capacity. This is actually quite meaningful information. The single best prognosticator in some studies on stress testing is the number of minutes the individual can exercise on a symptomlimited test, even more useful in one study than the coronary anatomy at angiography.

TAKE HOME POINTS

- Although stress testing yields some valuable anatomic and physiologic data, it cannot be relied on as the sole, accurate predictor of cardiac risk for noncardiac surgery.
- Many factors need to be factored together to arrive at an individuals risk. The intrinsic risk of the procedure to be performed and the urgency of this procedure are two important issues. In addition, as simple an intervention as the use of beta-blockers in the perioperative period has been shown to reduce morbidity and mortality in certain at-risk patients.

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- The risk assessment of an experienced clinician, combining clinical data obtained from a careful history and physical exam, determination of the traditional risk factors for CAD, exercise capacity in daily living, analysis of left ventricular function, supplemented where appropriate by noninvasive and invasive testing for coronary disease, should be considered the “gold standard” for preoperative evaluation.

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Perioperative Beta-Blocker Therapy is Indicated for High-Risk Patients Who Are Having Noncardiac Surgery, But Specific Questions Remain Unanswered

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Perioperative adverse cardiovascular events among patients who are having noncardiac surgery are relatively common, confer additional risk of other serious complications including death, and add significantly to health care costs. Relatively simple methods exist for risk stratification of these patients, including indices such as Goldman's original cardiac risk index and Lee's revised Cardiac Risk Index. However, few effective interventions exist for reducing the incidence of adverse perioperative cardiovascular occurrences and decreasing the associated morbidity and mortality. The risk-stratification schemes mentioned above are based on clinical history and symptoms. In addition to these, noninvasive testing, cardiac catheterization, and, for some subgroups, subsequent myocardial revascularization, together with careful perioperative monitoring, are often cited as reducing the risk of adverse cardiovascular events. In terms of available pharmacologic interventions, the perioperative use of beta-adrenergic-receptor blockers is often advocated as a safe, effective, and relatively inexpensive method of reducing adverse cardiovascular events after noncardiac surgery.

Although the majority of evidence suggests that beta-blockade is effective in reducing cardiovascular events after noncardiac surgery, especially for high-risk surgical patients, many questions currently remain unanswered. For which patients is the perioperative use of beta-blockade actually indicated? Are there patients who could be harmed by such treatment? Does it matter which beta-blocker is used? How early should beta-blockade be started, and how long should therapy continue postoperatively? What is the target heart rate? Is the protective effect mediated by blockade of the beta-adrenergic receptor or is it a heart-rate control effect?

Among the earliest reports of the beneficial effect of perioperative beta-blockade was the randomized trial of atenolol reported by Mangano et al. In this study, a relatively brief exposure to atenolol in the perioperative period was associated with morbidity and mortality improvement measured as late as 2 years after the surgery. Since that publication, numerous studies have been published using various beta-blocking medications in varying doses for varying time intervals in varying patient groups. Most of these show outcome

improvements associated with the use of beta-blockade in the perioperative time frame. Another interesting study, with bearing on the heart-rate control versus beta-adrenergic-receptor-blockade question, was carried out by Wallace et al. using clonidine as a heart-rate control intervention in place of beta-blockade. In this study, 190 patients were randomly assigned to either clonidine therapy or placebo; treatment was begun preoperatively and continued for 4 days after surgery. Thirty-day mortality was 6.2% with placebo, compared with just 0.8% with clonidine; 2-year mortalities were 29.2% and 15.2%, respectively. Most recently, Lindenauer published the results of a large-scale retrospective analysis of patients who underwent noncardiac surgery at one of 329 small to mid-size nonteaching hospitals throughout the United States. Among 122,338 patients who received perioperative beta-blockade there was an apparent relationship between underlying health status and benefit or not from the treatment. Using the revised cardiac risk index (rCRI), patients who had no or only minor cardiovascular risk (rCRI score of 0 or 1) had no benefit, and possibly a worsened outcome, associated with beta-blockade. However, patients with an rCRI score of 2, 3, or 4 had benefit (odds ratio for in-hospital death less than 1) associated with beta-blocker treatment. And perhaps more interesting, this risk reduction improved as the rCRI score increased: the higher the risk, the greater the apparent risk reduction from beta-blockade.

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There have also been several larger-scale meta-analyses of the individual publications that appear to show clinical outcome advantage to the use of beta-blockers. Auerbach and Goldman, in 2002, assessed five randomized, controlled trials and reached the conclusion that beta-blockade was likely beneficial in preventing cardiac morbidity. Stevens et al. performed a quantitative systematic review in 2003 of pharmacologic myocardial protection for patients undergoing noncardiac surgery and found that the 11 beta-blockade trials reviewed showed decreased ischemic episodes, reduced risk of myocardial infarction, and reduced risk of cardiac death in the beta-blockade group compared to placebo. They also observed that higher-risk patients received greater benefit from beta-blocker therapy. In 2005, McGory et al. performed a formal meta-analysis of six randomized, controlled trials and concluded that beta-blockade therapy was not only associated with reduced perioperative myocardial ischemia, myocardial infarction, and cardiac mortality but also conferred a reduction long-term cardiac mortality and overall mortality.

Meanwhile, the Agency for Healthcare Research and Quality and others have issued statements strongly supportive of the perioperative use of beta-blockers for patients who are having noncardiac surgery, especially those with increased risks of adverse perioperative cardiovascular events. The American College of Cardiology/American Heart Association (ACC/AHA) Task

Force on Practice Guidelines has recently released an updated guideline specific to perioperative beta-blocker usage. According to this statement, beta-blockers should be continued in surgical patients who are already receiving them for angina, arrhythmias, hypertension, or other ACC/AHA Class I guideline indications. Beta-blockers should be given to patients who are undergoing vascular surgery when ischemia has been noted on preoperative testing. Beta-blockers are probably indicated for patients undergoing vascular, high-risk, or intermediate-risk surgeries who have coronary heart disease or multiple clinical risk factors for increased perioperative cardiovascular risk. Beta-blockers may be considered for patients who are undergoing vascular high-risk or intermediate-risk surgeries who have a single clinical risk factor and for patients who are undergoing vascular surgery with low cardiac risk who are not already on beta-blockers. Finally, beta-blockers should not be given to patients undergoing surgery who have absolute contraindication to beta-blockade. The ACC/AHA recommends that beta-blockers be started several days or weeks before elective surgery with a target resting heart rate of 50 to 60 beats/min—an indication that the patient is receiving the benefit of beta-blockade but not yet evidence-based except for one recent study showing that "tight" heart rate control with beta-blockers was beneficial in patients having vascular surgery procedures (Feringa et al.). According to AHA recommendations, therapy should continue intra- and postoperatively to keep heart rates below 80 beats/min.

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Some studies and meta-analyses debate the efficacy of beta-blockade in reducing major cardiovascular events or challenge the strength of evidence, but the weight of evidence suggests that perioperative beta-blockade is an inexpensive and effective means of reducing the incidence and associated morbidity and mortality of adverse cardiovascular events. Despite the observations of the current randomized, controlled trials, retrospective studies, and meta-analyses, the current literature is still limited in that several of the trials performed may be inadequately powered and few assess issues such as titration of therapy to effect, optimal beta-blocker type, or ideal target population. Practical matters such as route of administration and length of

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administration still need to be addressed. Furthermore, there is a lack of data regarding the potential harm of perioperative beta-blockade, especially in low-risk patients, or in patients who experience rebound phenomena after postoperative cessation of beta-blockade. Currently, there are prospective, randomized trials underway or emerging that may shed light on many of these questions.

Lastly, a common and vexing problem for the anesthesia provider is what to do when a patient presents who was intended for preoperative beta-blockade but did not initiate therapy for whatever reason. Or perhaps the patient has had a cardiology consult suggesting a specific heart-rate range but the patient's heart rate is significantly higher, despite being compliant with the medication as ordered. It is the opinion of the senior author of this chapter as well as the cardiology consultant to this book that there are insufficient data to warrant holding the surgery to implement oral beta-blockade before induction. However, for high-risk patients who are having high-risk or moderate-risk surgery (e.g., requiring a general anesthetic with or without planned invasive monitoring), who do not have contraindications, then it is prudent to give some beta-blocker before induction. A reasonable choice is 10 to 50 mg of atenolol PO or intravenous (IV) metoprolol titrated up to 0.1 mg/kg, and then aggressive control of heart rate with an IV agent such as metoprolol or esmolol during the surgery. In addition, it would be prudent to continue the beta-blocker therapy with oral agents (or IV, depending on the circumstances) postoperatively for 72 to 96 hours. This is opinion; it must be stated that there is little or no class 1 or 2 evidence that addresses this specific clinical scenario.

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TAKE HOME POINTS

- Perioperative cardiovascular events after noncardiac surgery are common and a source of significant morbidity and mortality.
- The perioperative use of beta-blockade agents is advocated as a safe, effective, and relatively inexpensive method of reducing adverse events.
- The majority of evidence suggests that beta-blockade is effective in reducing cardiovascular events after noncardiac surgery, especially for high-risk surgical patients; however, many questions remain unanswered.
- A reasonable approach for an at-risk patient who has not been beta-blocked is to administer both PO and IV therapy before induction and to control heart rate intraoperatively with IV agents (esmolol or metoprolol).

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A Positive Troponin Is Not Necessarily a Myocardial Infarction

Michael P. Hutchens MD, MA

Bradford D. Winters MD, PhD

A common scenario in the preoperative evaluation of an acutely ill patient is the discovery of a positive troponin. Although this is commonly interpreted as a sign of myocardial ischemia, this may or may not be the case. Positive troponins occur without myocardial infarction (MI) in several situations that have implications for perioperative care very different than those of active MI.

Troponins I, T, and C form a complex with actin and myosin and control contraction in cardiac and skeletal muscle. A small amount of cardiac troponin is cytoplasmic, whereas the rest is sarcomeric. Although cardiac and skeletal muscle share isoforms of troponin C, cardiac troponins I and T are distinct from skeletal isoforms.

Since the year 2000, the international consensus definition of MI has included positive troponins with gradual rise and fall, and clinical evidence of MI (characteristic electrocardiogram changes or ischemic symptoms). Within 3 years of the introduction of this definition, the incidence of emergency department-diagnosed acute MI increased nearly 200%. Clearly, troponin values are a sensitive tool with which to diagnose myocardial ischemia, but this sensitivity has posed some problems. The value of this test led to the marketing of a large number of diagnostic assays, which have improved significantly since their introduction; early assays were less specific for cardiac troponins. The variety of assays means that clinical labs at different institutions must promulgate their own norms; values must be interpreted relative to the local reference values. It is not unheard of for neighboring institutions to have upper-limit-of-normal values that differ by an order of magnitude.

The earlier, nonspecific assays created confusion when emergency departments began using them in patients with low pretest probability of MI. What appeared to be spurious positives were found to be quite common. Although the newer assays are more specific, there are still disease states other than acute MI that can elevate blood levels of cardiac troponins T and I. Familiarity with these states can prevent embarrassing mistakes, complications, and unnecessary case cancellations.

Chronic renal failure patients represent the largest group of patients who may have elevated troponins in the absence of acute myocardial ischemia or infarction. With newer assays, up to 50% of chronic renal failure patients without acute myocardial ischemia (based on clinical signs and electrocardiogram) have elevated troponin T. Troponin T is more frequently elevated than troponin I in this population, leading to speculation that troponin I is more specific; this speculation is not evidence-vetted, however. The mechanism for this finding is not yet clear. Troponins are large molecules that are cleared by the reticuloendothelial system and not the kidney, and troponin levels are not related to creatinine, blood urea nitrogen (BUN), or calcium phosphate product. Most evidence points to a cardiac source, whether that be clinically silent microinfarction, myocardial strain from heart failure, or alteration in expression of troponin molecules by renal failure. Regardless, what is known is that even in renal failure patients without active MI or coronary artery disease by angiography, positive troponins are a risk factor for intermediate and long-term mortality. The perioperative implications of this finding are not clear but are likely different from those of a patient undergoing surgery in the midst of an acute MI. P.295

Other disease states that have been observed to elevate troponins without other evidence of acute myocardial infarction include congestive heart failure (CHF), left ventricular hypertrophy, supraventricular tachycardia, pulmonary embolism (PE), cardiac surgery, cardiac trauma, defibrillation and cardioversion, sepsis, subarachnoid hemorrhage, cocaine use, methamphetamine use, and endurance running. This is by no means an all-inclusive list. What these things have in common is direct oxidative stress to myocardiocytes or overt necrosis and apoptosis—without occlusion of a large coronary artery. With the exception of marathon running, all of these states are known to have the potential for lasting cardiac effects, and in the case of PE, CHF, and sepsis, studies have shown that elevated troponins portend worse outcome.

So, unless your patient has recently run a marathon or been cardioverted, if the troponin is positive, it's probably a bad sign. Is the patient having an MI though? The diagnosis of MI by troponins requires the characteristic rising and falling of troponins; this usually occurs over a 24-hour period and thus sequential values are needed. They are generally drawn every 8 hours. This definition also requires other evidence of MI: ST changes, new Q wave, or ischemic symptoms. Patients who have positive troponins with appropriate kinetics and electrocardiogram changes or ischemic symptoms are having an acute MI. These patients have very high perioperative risk, and all but the most critical emergency surgical intervention should be postponed. Patients with chronic renal failure and chronically elevated troponins, or critically ill patients with elevated troponins with no new evidence of myocardial ischemia, may not be having an acute MI. Although a positive troponin in this setting has adverse intermediate and long-term prognostic impact, it probably does not carry the same level of perioperative risk as an active, acute myocardial infarction. P.296

TAKE HOME POINTS

- Elevated cardiac troponin is present in acute MI.
- Elevated cardiac troponin is present in disease states other than MI.
- Chronic renal failure patients often have elevated troponin without other evidence of acute MI.
- The diagnosis of MI by troponin requires a rise and fall as well as clinical evidence of ischemia.

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Do Not Disregard an Elevated Partial Thromboplastin Time When the Prothrombin Time Is Normal

Lisa Marcucci MD

The consensus on preoperative laboratory screening in moving toward fewer tests being performed in the perioperative setting. Although the yield is low, preoperative coagulation studies can be vitally important. When they are ordered, these tests must be correctly interpreted for good care. Any abnormality of the partial thromboplastin time (PTT) in the setting of a normal prothrombin time (PT) cannot be dismissed as a lab error, as it can be a marker for an underlying coagulation factor disorder that can cause fatal bleeding.

PTT measures the coagulation ability of the intrinsic pathway, and to a lesser extent, the common pathway in the clotting cascade. Factors in the intrinsic pathway include VIII, IX, XI, XII, and prekallikrein. Factors in the common pathway include fibrinogen, II, V, VII, and X. Anticoagulation with heparin (including the low-molecular-weight heparins if used in sufficiently high doses), hirudins, danaparoid, and argatroban cause a prolonged PTT in the setting of a normal PT. However, a prolonged PTT with normal PT in the absence of these drugs must prompt further laboratory investigation using mixing studies.

Virtually every laboratory has a protocol for performing a PTT mixing study. For PTT mixing studies, the patient's serum is mixed with pooled serum of normal controls and then incubated for 1 to 2 hours at an elevated temperature. The PTT is measured twice after mixing—before and after incubation. There are three possible outcomes of this test:

1. The PTT of the mixture is normal on initial mixing and remains normal after incubation. This suggests that the patient's abnormal PTT is due to a deficiency of factor VIII, IX, XI, XII, or prekallikrein, because the pooled serum provides the missing factor. Specific assays for factors VIII, IX, XI, and XII are performed to find out which factor is deficient. Assays for prekallikrein are not performed, because deficiency of this factor causes an increased PTT but is extremely rare and does not cause abnormal bleeding. Treatment is repletion of the missing factor before a surgery or other invasive procedure.
2. The PTT is immediately prolonged on mixing and remains prolonged after incubation. This suggests the presence of a coagulation inhibitor in the patient's serum. The most common inhibitor that causes this mixing study outcome is lupus anticoagulant, and assays for this can be performed. If this assay is negative, specific assays for inhibitors for factors of IX, XI, and XII are done. Inhibitors are antibodies against the individual factors and are detected by testing for their unique molecular composition. The presence of lupus anticoagulant often causes an increased PTT but is paradoxically associated with thrombosis, not bleeding, and may require preoperative anticoagulation. P.298
3. The PTT of the mixture is initially normal (or significantly shorter than the patient's PTT) and then becomes prolonged after incubation. This strongly suggests the presence of a factor VIII inhibitor. A factor VIII assay should be performed and, if this factor is decreased, then a factor VIII inhibitor assay (Bethesda assay) should be performed. The larger the amount of inhibitor present, the higher is the risk of severe bleeding. Each Bethesda unit connotes reduction in functional factor VIII of 50% (i.e., 3 Bethesda units contain only 12.5% functional factor VIII).

Treatment of acquired factor VIII inhibitor is difficult and is aimed at reversing the coagulopathy and eliminating the inhibitor. Substances used to stop active bleeding include desmopressin acetate (DDAVP), factor VIII bolus/infusion, prothrombin complex concentrates (which bypass factor VIII), recombinant factor VIIa (very expensive), and repletion of ongoing loss of blood components. Treatment protocols aimed at eliminating the underlying inhibitor use corticosteroids, plasmapheresis, intravenous immunoglobulin, and (in resistant cases) cyclophosphamide.

Clinically, acquired factor VIII inhibitor can present as spontaneous muscle and soft tissue hematoma, hematuria, severe epistaxis, or fatal bleeding with even mildly invasive procedures such as cystoscopy or bronchoscopy. As these inhibitors are acquired, lack of a previous bleeding diathesis does not rule out their presence at the time of the prolonged PTT. Most often their etiology is idiopathic, but factor inhibitors are associated with several conditions as listed below:

Autoimmune—systemic lupus erythematosis, rheumatoid arthritis, ulcerative colitis, psoriasis, and pehphigus vulgaris.

Pregnancy—can appear at term or just after delivery, sometimes with spontaneous disappearance 12 to 18 months postpartum; most likely to occur in the first pregnancy.

Malignancy—lymphoproliferative disorders, plasma cell dyscrasias, and solid tumors.

Drugs—sulfa, penicillin, phenytoin, and especially animal-derived, nonautologous fibrin glue. This type of fibrin glue is made from the mixing of several components of bovine serum, of which remnants of bovine factor VIII may survive. Two percent of patients who receive fibrin glue develop acquired factor VIII inhibitor because of this exposure to bovine factor VIII. P.299

TAKE HOME POINTS

- Always obtain mixing studies if the PTT is prolonged.
- Lack of previous bleeding diathesis does not rule out the presence of an **acquired** factor 8 deficiency.

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Avoid a 70% Mortality Rate: Do Everything You Can to Prevent Perioperative Renal Failure

Michael P. Hutchens MD, MA

Acute renal failure in the postoperative period is appallingly common and has abysmal outcomes. In high-risk populations the incidence may be as high as 25%, and patients who require critical care services and renal replacement therapy have repeatedly been shown to have a mortality rate >70%. Despite this, perioperative resuscitation strategies are frequently formed solely with cardiac and pulmonary outcomes in mind. An early extubation is a pyrrhic victory if it condemns the patient to a slow death or lifetime dialysis. Despite decades of research, the risk factors, etiology, prevention, and treatment of perioperative acute renal failure all remain murky. Numerous interventions have been assessed, with few promising results. There are a few tools at hand, though, and the devastating effect of acute renal failure obligates anesthesiologists to use the tools available with the best possible dexterity.

The most consistent risk factor for perioperative renal failure is preoperative renal dysfunction. Elevated creatinine or decreased creatinine clearance, or a preoperative diagnosis of renal insufficiency, all significantly increase the risk of postoperative renal failure. Preoperative heart failure and diabetes mellitus are also significant predictors, as are the acute comorbidities rhabdomyolysis, fulminant liver failure, abdominal compartment syndrome, and sepsis. Surgical procedures involving cardiopulmonary bypass or aortic cross-clamp (any aortic cross-clamp, not just suprarenal clamping) increase risk as well. Perioperative diagnostic studies involving nephrotoxic radiocontrast and medical management of concurrent nonsurgical disease (chemotherapy, aminoglycosides, nonsteroidal anti-inflammatory drugs [NSAIDs]) can further elevate the risk.

Anesthetic management directed at preventing acute renal failure ranges from banal to complex. If possible, stop nephrotoxic medications preoperatively. Certainly do not give preoperative NSAIDs to improve analgesia in at-risk patients. For procedures that will take 2 hours or more, a Foley catheter should be placed. Ensure that urine enters the Foley catheter when it is placed, as a misplaced Foley and subsequent obstruction can *cause* renal failure. In current practice there is no longer a fixed "goal" for hourly urine output; the objective is to monitor urine output diligently, and if it drops, intervene—increase perfusion pressure, give a fluid challenge, and inform the surgeon. If need be, place a central venous pressure (CVP) monitor to assure adequate intravascular volume. Eschew nephrotoxic agents in your anesthetic plan: ketorolac, dextran, and aminoglycosides should be avoided. It is clear that hypoperfusion contributes to perioperative renal failure; in at-risk patients, maintaining perfusion pressure is a high priority. Have a low threshold for placing an intra-arterial monitoring line, and use vasoactive agents, if necessary, to treat hypotension.

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No therapeutic agent has shown broad renal-protective effect in the perioperative setting. Although there is lasting enthusiasm for low-dose dopamine, extensive study has shown it to have no effect on need for dialysis or mortality. Use of dopamine predictably causes tachycardia, which may be deleterious in a patient with concurrent coronary artery disease and increases the incidence of perioperative atrial fibrillation. Mannitol is commonly used in aortic cross-clamp cases; however, evidence to support this use is slim at best. Mannitol has been shown to be effective in preventing acute renal failure when used properly during renal transplant procedures. Although furosemide is popularly used to increase urine output, there is 30 years of evidence that furosemide can cause acute renal failure, and its use in the hypoperfused patient in the operating room (OR) is anathema. Other agents that have been used as putative intraoperative renal protectants include atrial natriuretic peptides, *n*-acetylcysteine, and fenoldopam. All have shown benefits in small studies that have not been replicable in large studies, suggesting that each may have use in a confined population, but what population remains unclear. A tantalizing study by Gandhi et al. in 2005 showed a significant association between intraoperative hyperglycemia and postoperative renal failure (defined as a doubling of creatinine, a creatinine >2, or new requirement for dialysis) in cardiac surgical patients. As intensive insulin therapy in the intensive care unit (ICU) reduces the need for dialysis, and as diabetes mellitus is a known risk factor, this raises the possibility that intraoperative insulin treatment may be renoprotective.

TAKE HOME POINTS

- Although the anesthesiologist may not see it happening in the OR, acute renal failure is an anesthetic outcome with gargantuan impact on patient morbidity and mortality. Remember that we can see only oliguric acute renal failure, because typically we don't check the BUN and creatinine.
- The alert anesthesiologist must consider renal outcome in the anesthetic plan. Recognize the patient who is at risk because of preoperative disease or medical interventions.
- Maintain adequate circulating volume and perfusion pressure, and at all costs avoid doing harm with nephrotoxic agents.
- Consider tight intraoperative glucose control with insulin in diabetic patients.
- Finally, if you are concerned that the patient may be at risk for postoperative renal failure, communicate this to the surgeons, because early recognition and intervention in the postoperative period may improve the outcome.

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"Renal Dose" Dopamine Must Die

Todd J. Smaka MD

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Dopamine (DA) is an endogenous catecholamine that influences receptors in a concentration-dependent manner. For >20 years, low-dose or "renal-dose" DA(LDD) (1 to 5 mcg/kg/min) has been used in an attempt to preserve renal function in critically ill patients. This use stems from studies that demonstrated increased renal blood flow and diuresis in healthy experimental animals and human volunteers. However, there have never been studies demonstrating benefit in critically ill patients, and recently, several high-quality studies have shown that LDD does not confer meaningful renal protection and indeed may cause harm.

LDD causes renal vasodilatation, predominately by stimulating DA-1 receptors in the renal vasculature. It causes natriuresis by inhibiting Na^+/K^+-ATPase activity in the proximal tubule, thick ascending limb of the loop of Henle, and the cortical collecting ducts. It causes diuresis by stimulating prostaglandin E_2 (PGE_2) production, which antagonizes the effects of antidiuretic hormone. These effects would appear to indicate improved renal function, but consideration of the patient and not the laboratory values suggests otherwise.

In a 1998 study of critically ill patients without renal or hepatic dysfunction, investigators found no correlation between DAinfusion rate and DAserum concentration; serum concentrations in patients were significantly higher than in healthy controls. Thus the notion that LDD in healthy volunteers is the same as LDD in critically ill patients is highly questionable. The definitive study of LDD is a large multicenter, randomized, double-blind, placebo-controlled study performed by the Australian and New Zealand Intensive Care Society (ANZICS) Clinical Trials Group and published in 2000. It looked at 328 patients in 23 intensive care units (ICUs) and concluded that LDD does not benefit critically ill patients who are at risk for renal failure. The study demonstrated that LDD did not differ from placebo in resultant peak serum creatinine concentration, creatinine increase from baseline, number of patients requiring renal replacement therapy, duration of ICU stay, duration of hospital stay, and number of deaths. In another large trial, NORASEPT II study investigators performed a retrospective analysis of 395 oliguric patients with sepsis in the placebo arm of a TNF-α antibody sepsis trial and showed that LDD did not reduce the incidence of acute renal failure, the need for dialysis, or the 28-day mortality in patients with oliguria due to septic shock. These same conclusions have been reached in several meta-analyses. The study data therefore do not support the hypothesis that LDD benefits patients with respect to the meaningful outcomes of death or dialysis.

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Although there is physiologic rationale suggesting benefit from LDD, there is also abundant physiologic rationale against it. LDD directly vasodilates vessels in the cortex and indirectly vasodilates vessels in the inner medulla. This causes a redistribution of blood away from the outer medulla, which contains the energy-dependent Na^+/K^+-ATPase channels of the medullary thick ascending limb. Furthermore, the natriuretic effect of LDD causes an increased solute load to the distal tubular cells, which may actually increase oxygen consumption and thereby increase the risk of ischemia. Therefore, although LDD does increase blood flow to the kidney as a whole, it does so at the expense of blood flow to the metabolically active outer medulla, resulting in an increased susceptibility to focal ischemia and eventually acute renal failure.

The increased blood flow and glomerular filtration rate induced by LDD in healthy volunteers has not been demonstrated in patients with early renal failure. Even so, such effects are likely due to LDD's cardiac effects. In fact, there is no evidence that drug-induced diuresis protects renal function in the setting of decreased cardiac output or hypovolemia.

LDD may in fact cause harm. LDD can induce tachycardia, dysrhythmias, and myocardial ischemia. Attenuation of ventilatory response to hypoxia and increased intrapulmonary shunting are known complications. Mesenteric ischemia has been observed in patients receiving LDD. This is particularly harmful in the critical care and intraoperative setting, because it compromises the mucosal lining and reduces perfusion to anastamoses. LDD also causes panhypopituitarisms, including suppression of prolactin, growth hormone, and thyrotropin. Growth-hormone suppression contributes to a negative nitrogen balance in critical illness, and prolactin suppression induces a transient decrease in T-cell function. Other immunologic effects of LDD include inhibition of stimulated lymphocyte proliferation, immunoglobulin synthesis, and cytokine production. It also promotes lymphocyte apoptosis.

Studies showing a renal protective benefit of DAin healthy volunteers either have insufficient power to draw conclusions, are confounded by simultaneous administration of other putative nephroprotective agents, or are associated with doses higher than true LDD. Very strong evidence now shows no meaningful benefit from LDD. At higher doses of DA, the effect on renal physiology is likely due to its cardiac effects, and these effects can be induced with other agents. Although increased blood flow may be advantageous in preventing renal failure, DAshould not be used solely for its renal effects. Rather, if renal perfusion is felt to be insufficient, the appropriate drug should be chosen for the clinical situation and should be used in the appropriate *cardiac* dose.

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TAKE HOME POINTS

- Renal-dose DA (1 to 5 mcg/kg/min) does not have beneficial effects on the kidney even though urine output may be increased.
- Renal-dose DA does not prevent renal failure.
- Renal-dose DA does not prevent death or the need for dialysis in critically ill patients.
- Renal-dose DA has potential to cause harm.

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Remember that There Are at Least Seven Modalities for Treating Hyperkalemia in the Perioperative Period

Grace L. Chien MD

Hyperkalemia means that serum potassium levels are elevated beyond normal levels (the upper limit is usually reported as 5 to 5.5 mmol/L). Hyperkalemia has few clinical manifestations. In awake patients, severe muscle weakness beginning with the lower extremities and progressing cephalad, usually sparing cranial nerves and respiratory muscles, is sometimes evident. In patients under general anesthesia, the primary abnormality manifests as electrocardiogram (EKG) changes starting with peaked T waves, shortened QT interval, progressive lengthening of the PR interval, and QRS duration. The P wave may disappear, and ultimately, wide, complex ventricular tachycardia presents, eventually progressing to ventricular standstill. Hyperkalemia may also cause a variety of heart blocks, including bundle branch block and atrioventricular (AV) blocks. The manifestations of hyperkalemia depend on the rate of potassium rise—they are better tolerated by patients with chronic hyperkalemia. EKG changes with hyperkalemia are affected by concomitant hypocalcemia, academia, and hyponatremia.

PATHOGENESIS OF HYPERKALEMIA

Although hyperkalemia has many etiologies (Table 74.1), there are three distinct pathways: (i) ineffective elimination of potassium during any interval in the perioperative period, almost always due to renal failure; (ii) acute potassium load from a medication error or massive transfusion of stored blood; (iii) movement of potassium ions from the intracellular space to the extracellular space. This last can occur with the use of succinylcholine, especially for burn patients or paralyzed patients. It can also be seen with changes in ventilation and/or acid-base status in patients on certain drug combinations (spironolactone/beta-blockers) or significant physiologic derangements such as diabetic ketoacidosis. At the cellular level, hyperkalemia interferes with neuromuscular transmission and thus produces skeletal and cardiac muscle abnormalities. Neuromuscular transmission depends on membrane excitability. Increased extracellular potassium depolarizes the cell membrane, making the cell more excitable, and requiring less of a stimulus to generate an action potential. The hyperexcitability eventually

inactivates sodium channels and leads to cardiac conduction abnormalities and muscle paralysis.

TABLE 74.1 DISORDERS THAT CAUSE HYPERKALEMIA

DISORDERS THAT LEAD TO HYPERKALEMIA CAUSED BY IMPAIRED RENAL EXCRETION OF POTASSIUM	DISORDERS THAT LEAD TO HYPERKALEMIA CAUSED BY SHIFT OF POTASSIUM INTO THE EXTRACELLULAR SPACE
Acquired hyporeninemic hypoaldosteronism Addison disease Congenital adrenal hyperplasia (recessive or autosomal dominant) Mineralocorticoid deficiency Primary hypoaldosteronism of hyporeninemia Pseudohypoaldosteronism Renal insufficiency or failure Systemic lupus erythematosus Type IV renal tubular acidosis	Damage to tissue from rhabdomyolysis, burns, or trauma Familial hyperkalemic periodic paralysis Hyperosmolar states (e.g., uncontrolled diabetes, glucose infusions) Insulin deficiency or resistance Tumor lysis syndrome

TREATMENT OF HYPERKALEMIA

A valuable resource is the Cochrane Database of Systematic Reviews, which has published recommendations for emergency interventions for hyperkalemia. Current evidence suggests that intravenous (IV) insulin and glucose combined with nebulized beta-adrenergic receptor agonists were more effective than each treatment alone.

Calcium.

Calcium stabilizes cardiac muscle and directly antagonizes the hyperexcitability induced by hyperkalemia. Calcium starts acting within minutes and can be used as an infusion over 2 to 3 minutes. The dose is 500 mg to 1 g and may be repeated after 5 minutes. Calcium should not be given with bicarbonate, as calcium carbonate will precipitate. Patients who are taking digitalis are more vulnerable to toxicity with hypercalcemia, so calcium should be used with great caution in these patients.

Insulin and Glucose.

Insulin drives potassium ions into the cell by enhancing the sodium/potassium ATPase pump in skeletal muscle. Ten units plus 50 mL of 50% glucose as a bolus, followed by a glucose infusion, had been found to be effective in decreasing serum potassium. Alternatively, 50 mL of 50% glucose may be given to rapidly induce hyperinsulinemia.

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Sodium Bicarbonate.

Sodium bicarbonate raises the pH of serum and, via the H^+/K^+ exchanger, drives hydrogen-ion release from inside the cell. Potassium in turn moves into the cell to maintain electroneutrality and thus reduces serum potassium levels. Ngugi et al. demonstrated that bicarbonate infusion lowered serum potassium levels by 0.47 ± 0.31 mmol/L at

30 minutes and thereafter. In addition, bicarbonate appears to reduce serum potassium levels by an unknown mechanism. However, the only placebo-controlled study, by Allon, found that bicarbonate did not lower serum potassium levels compared to placebo. P.308

Beta-Adrenergic Agonists.

Beta-adrenergic agonists are effective in reducing serum potassium levels by 30 minutes after administration. The effect appears to be dose-responsive. Allon et al. demonstrated that 20 mg of nebulized albuterol reduced serum potassium levels. There does not seem to be a significant difference in effect between IV-administered or inhaled albuterol. With either route of delivery, further reduction in serum potassium levels may be attained by readministration of albuterol at 120 minutes. Of note, epinephrine can be used for its mixed effects if beta2-adrenergic agents are not available. However, α-receptor stimulation causes potassium release from cells, especially in patients with renal failure.

Loop or Thiazide Diuretics.

Loop diuretics induce diuresis through sodium excretion, in the process activating the Na/K exchanger and inducing potassium wasting. Loop diuretics may be effective in mild renal impairment because loop diuretics are high-ceiling diuretics, meaning they have dosedependent effects. Administering Lasix via IV produces effects that depend on the patient's renal function, prior Lasix exposure, and many intraoperative factors such as hydration status. Therapy using diuretics to lower potassium should be guided by frequent serum chemistry, patient symptoms, and expected potassium trend.

Cation-Exchange Resin.

Sodium polystyrene sulfonate, administered orally or as a retention enema, binds potassium and releases sodium in the gastrointestinal (GI) tract. The oral dose is 15 to 30 g in 20% sorbitol solution. Enema doses are usually 50 g mixed with 50 mL of 70% sorbitol plus 100 to 150 mL of tap water. One dose can potentially lower plasma potassium by 0.5 to 1 mg/L. Resin seems to be less effective in renal failure patients. The routes of administration and slow onset render resins less effective in the acute rescue of intraoperative hyperkalemia.

Dialysis.

Dialysis may be performed in the perioperative period (including in the operating room!) in the event that more conservative treatments fail, if the hyperkalemia is severe, or if the patient has such massive tissue injuries that conservative treatment cannot keep pace with the amount of potassium released from cells. More potassium may be removed with increasing blood flow during dialysis.

Blood Products.

Although blood products are not a treatment for hyperkalemia, in cases in which massive transfusion is ongoing, especially if there is a degree of renal insufficiency, the anesthesia provider should request that the blood bank send recently donated blood and/or “washed” packed

red blood cells to try to reduce the levels of exogenously administered potassium. P.309

TAKE HOME POINTS

- Be aware of the situations in which hyperkalemia commonly occurs—succinylcholine “mishaps”, crush injuries, reperfusion of transplanted organs or ischemic limbs, and patients on spironolactone and beta-blockers.
- Consider hyperkalemia if the patient develops a new bundle branch or atrioventricular block in the perioperative period.
- Calcium stabilizes cardiac muscle membranes, but it must be used cautiously in patients on digitalis.
- Other therapies use the Na^+/K^+ and H^+/K^+ exchangers to move potassium intracellularly.
- Ask the blood bank to send packed red cells <5 days “old” or washed units.
- For severe or refractory cases, dialysis may be warranted.

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Editors: Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.
Title: *Avoiding Common Anesthesia Errors, 1st Edition*

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Manage Obstructive Sleep Apnea Patients Conservatively

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Obstructive sleep apnea (OSA) is defined as air flow cessation during sleep for at least 10 seconds, caused by the collapse of the airway. Hypopnea is defined as a moderate reduction in air flow that is associated with arousal diagnosed by electroencephalogram. Hypopnea represents a >50% reduction in air flow or a <50% reduction in air flow that is associated with a desaturation of >3% from the baseline reading.

EPIDEMIOLOGY

The exact incidence of OSAin the adult population is unknown. It was previously thought to be between 5% and 10%, but a recent study showed that one in four U.S. adults appears to be at high risk for OSA. This fact underscores the increased chance of having a patient with undiagnosed OSAas a candidate for general anesthesia.

RISK FACTORS

The most clearly recognized risk factor for OSAis a narrow upper airway. The most common etiologies involve congenital abnormalities of the airway (e.g., Pierre-Robin syndrome, midface hypoplasia), hypertrophy of the adenoids and tonsils (especially in children), macroglossia (e.g., Down syndrome), and increased uvula size. Other important risk factors are obesity (body mass index >30 kg/m^2), nasal polyps, deviated nasal septum, and chronic rhinitis. Neck circumference of >17 in for men and >16 in for women also represents an additional risk for OSA. Obesity by itself can lead to OSAor can worsen a pre-existing OSA. In the adult population the male-to-female ratio is 2:1, but this ratio does not apply to children or postmenopausal females. In women over the age of 65 years, the risk for OSAincreases threefold. Other predisposing factors for OSAinclude a family history of OSA, smoking, and use of alcohol, tranquilizers, or sedatives.

PATHOLOGY

The patient with OSAhas a smaller and more easily collapsible upper airway. The risk of complete collapse of the airway is accentuated at the end of expiration, when the tissue pressure overcomes intraluminal pressure. The critical collapsible area is usually seen in the velopharynx, as evidenced by imaging studies (computerized tomography, magnetic resonance, or fluoroscopy). Recurrent apnea will lead to intermittent hypoxemia, hypercapnia, P.312
and sleep fragmentation, with secondary increase of the sympathetic tone. OSArepresents an independent risk factor for cardiovascular morbidity and mortality. Aplausible explanation seems to be the increased oxidative stress secondary to alternate hypoxia/reoxygenation. The oxidative stress causes systemic inflammatory response with activation of endothelial cells, leukocytes, and platelets that culminates in early signs of atherosclerosis. OSAis one of the leading causes of systemic hypertension, left ventricular hypertrophy, pulmonary hypertension with right ventricular failure and congestive heart failure, cardiac dysrhythmias, ischemic heart disease, and stroke.

Sleep fragmentation leads to chronic fatigue, excessive daytime somnolence that markedly increases the risk of motor vehicle accidents, memory problems, anxiety, and depression.

DIAGNOSIS

The diagnosis of OSAis based on patient history, clinical presentation, physical examination, and sleep study (polysomnography). The classical symptoms of OSAare snoring, excessive daytime sleepiness, and witnessed episodes of breathing cessation during sleep. Usually, the patient with OSA is a man, over age 60 years, obese, with a short and thick neck. The "gold standard" for diagnosing OSAremains the polysomnography or sleep study performed overnight in a sleep laboratory. An accurate measurement requires 12 physiologic signals (Table 75.1) with a computer-based automated sleep analysis.

The final result of the analysis is the apnea-hypopnea index (AHI), which is the total number of apnea and hypopnea events per hour of total sleep time. According to the AHI, the severity of OSAcan be stratified as shown in Table 75.2.

TREATMENT

The mainstay of treatment for OSAis continuous positive airway pressure (CPAP) applied during sleep via a tight nasal or facial mask. Indications for CPAP therapy are an AHI >10 accompanied by symptoms or an AHI >30 regardless of symptoms. This technique has been proven to be efficient in preventing the collapse of the airway and improving breathing during sleep. CPAP therapy improves concentration, alertness, neurocognitive function, and mood. The CPAP has favorable effects on cardiovascular outcome but necessitates a strict commitment from the patient. The addition of humidification, bi-level positive airway pressure (BiPAP), and auto-titration of positive airway pressure has been tried to improve compliance and patient comfort. Mandibular repositioning appliances represent a simple and noninvasive alternative method of treatment for mild OSA(AHI 5 to 15) and are used in patients who are unable to tolerate CPAP. The mechanism of action is similar to the jaw-thrust technique. Surgery is reserved for severe OSAor lack of P.313
response to CPAP. The main objective of any surgical technique is to enlarge the upper airway. Adenoidectomy, tonsillectomy, nasal polypectomy, septoplasty, uvulopalatopharyngoplasty, maxillomandibular advancement and hyoid expansion are all procedures used to alleviate the symptoms of OSA surgically. If these are unsuccessful and the obstruction is severe, the last option is tracheostomy. As adjuvant therapy, the following may be helpful: losing P.314
weight, position therapy, topical application of soft tissue lubricant, and the use of acetazolamide to increase respiratory drive to compensate for the metabolic acidosis caused by hypoxia.

TABLE 75.1 SIGNALS REQUIRED FOR POLYSOMNOGRAPHY (HYPOPNEIC AND APNEIC EVENTS ARE ASSOCIATED WITH

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AROUSAL FROM REM SLEEP, OXYHEMOGLOBIN DESATURATION, POSSIBLE ARRHYTHMIAS AND HIGH BLOOD PRESSURE.)

FUNCTION	SIGNAL	RESPONSE IN OBSTRUCTIVE SLEEP APNEA
Sleep	Electroencephalogram (EEG)	Arousal episodes
	Electrooculogram (EOG)	Rapid eye movements (REM)
	Electromyography submentalis (EMG)	Decrease of chin muscle tone
Respiration	Oronasal air flow	Decrease or cessation of air flow
	Ribcage and abdominal movement	Increased respiratory effort
	Oxygen saturation	Desaturation
	End-tidal CO_2	Increased value after apnea
Cardiovascular	Electrocardiogram (EKG)	Cyclical variation of bradycardia episode associated to apnea followed by tachycardia Other arrhythmias
	Blood pressure	Possible elevated values linking to cardiovascular consequences of OSA
Movement	Electromyography tibialis	Limb movement
Position	Body position	Correlation with occurrence of OSA
Behavior	Video, audio	Snoring, sleep talking, sleep apnea events, movement disorder, seizures

TABLE 75.2 APNEA-HYPOPNEA INDEX (AHI) AND SEVERITY OF OBSTRUCTIVE SLEEP APNEA (OSA)

SEVERITY OF OSA	ADULT AHI	PEDIATRIC AHI
None	0-5	0
Mild OSA	6-20	1-5
Moderate OSA	21-40	6-10
Severe OSA	>40	>10

OUTCOME

The natural history of sleep apnea is not yet fully described, but the mortality is increased with the development of cardiovascular complications, especially in the middle-aged population. CPAP, surgery, and dieting can improve the outcome and lower the risk of death from cardiovascular complications.

IMPLICATIONS FOR ANESTHESIA

In October 2005 the American Society of Anesthesiologists (ASA) developed practice guidelines for perioperative management of patients with obstructive sleep apnea. This document should be considered required reading for every anesthesia provider.

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Preoperative Evaluation.

Patients with an established diagnosis of OSAwith the severity of the OSAgraded from the results of the sleep study (AHI) (Table 75.1) should be managed according to certain practice guidelines that are recommended by the ASA. Otherwise, all surgical patients should be evaluated for OSArisk factors. Multiple screening methods are available, such as the Berlin Questionnaire, which has been proven to be specific and sensitive for OSA screening. The Berlin Questionnaire focuses primarily on symptoms of snoring, apnea, fatigue, hypertension, and body mass index (BMI).

The ASAhas come up with two models, one for identification and assessment of OSAand a second to be used as a scoring system (Tables 75.3 and 75.4) to predicate the possibility of OSAfrom the presence of various risk factors. The ASAmodules need clinical validation and proof of their usefulness in the perioperative management of OSA.

In patients who are evaluated as being at high risk for OSAand/or who are undergoing a major surgical procedure with a possible requirement for high doses of opioids for postoperative pain, it is advisable to postpone an elective surgery until further evaluation and proper management of their OSAcan be undertaken.

During the preoperative evaluation of surgical patients with OSA, it is vital to perform a comprehensive history and physical examination to learn of any comorbidities or previous anesthesia-related complications and to document the settings of the patient's CPAP/BiPAP. One should also make sure that the patient's equipment will be available for postoperative use.

The use of sedatives and analgesics should be avoided in the preoperative setting because of the increased sensitivity to any respiratory depression. The consequences of upper-airway muscle tone relaxation could lead to devastating effects before the start of surgery and anesthesia. It is important not to underestimate the challenge of mask ventilation and/or intubation in these patients and to remember to have a clear plan for airway management before going ahead with a general anesthetic.

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TABLE 75.3 ASA PROPOSED EXAMPLE FOR IDENTIFICATION AND ASSESSMENT OF OSA

Clinical signs and symptoms suggesting the possibility of OSA

1) Predisposing physical characteristics	BMI >35 kg/m^2 (95th percentile for age and gender) Neck circumference >17 in (men); >16 in (women) Craniofacial abnormalities affecting the airway Anatomical nasal obstruction Tonsils touching or nearly touching in the midline
2) History of apparent airway obstruction during sleep	Snoring (loud enough to be heard through closed door) Frequent snoring Observed pauses in breathing during sleep Awakens from sleep with choking sensation Frequent arousals from sleep Intermittent vocalization during sleep Parental report of restless sleep, difficult breathing, or struggling respiratory efforts during sleep
3) Somnolence	Frequent somnolence or fatigue despite adequate “sleep” Falls asleep easily in a nonstimulating environment Parent or teacher comments that the child appears sleepy during the day, is easily distracted, overly aggressive, or has difficulty concentrating Child often difficult to arouse at usual awaking time

If patient has signs or symptoms in two or more of the above categories, there is a significant probability that OSAis present.

Sleep study done

SEVERITY OF OSA	ADULT AHI	PEDIATRIC AHI
None	0-5	0
Mild OSA	6-20	1-5
Moderate OSA	21-40	6-10
Severe OSA	>40	>10

ASA, American Society of Anesthesiologists; OSA, obstructive sleep apnea; BMI, body mass index; AHI, apnea-hypopnea index.

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TABLE 75.4 ASA PROPOSED EXAMPLE FOR OSA SCORING SYSTEM

a) Severity of sleep apnea based on sleep study or clinical indicators if sleep study not available; point score (0-3)	None	0
	Mild	1
	Moderate	2
	Severe	3
b) Invasiveness of surgery and anesthesia; point score (0-3)	Superficial surgery under local or peripheral nerve block anesthesia without sedation	0
	Superficial surgery with moderate sedation or general anesthesia	1
	Peripheral surgery with spinal/epidural anesthesia (no more than moderate sedation)	1
	Peripheral surgery with general anesthesia	2
	Airway surgery with moderate sedation	2
	Major surgery with general anesthesia	3
	Airway surgery with general anesthesia	3
c) Requirement for postoperative opioids; point score (0-3)	None	0
	Low-dose oral opioids	1
	High-dose oral opioids, parenteral or neuraxial opioids	3
Estimation of perioperative risk: overall score = score from A plus the greater of the score for either B or C; point score (0-6)		

ASA, American Society of Anesthesiologists; OSA, obstructive sleep apnea

Intraoperative Management.

OSA patients should be considered potential difficult airway management patients, and the anesthesia team should be prepared in advance for this possibility.

As a general rule, whenever it is possible, a regional anesthesia technique should be used instead of general anesthesia. General anesthesia with a secured airway is a better choice than monitored anesthesia care (MAC) with spontaneous breathing and an unsecured airway. Short-acting anesthetic drugs represent an attractive choice for general anesthesia.

For monitoring, standard noninvasive monitoring such as pulse oximeter, capnography, electrocardiography (EKG), and blood pressure are critical, especially for patients who are breathing spontaneously under MAC.

Extubation should be done in the semiupright position when the patient is fully awake and completely recovered after muscular blockade. P.317

Postoperative Care.

The main objectives in postoperative care are oxygenation and maintenance of a patent airway using supplemental oxygen and CPAP/BiPAP support. This is especially true for patients who are already on these treatments or high-risk patients.

Adequate analgesia with minimal depression of respiratory drive provided by continuous analgesia through regional techniques such as continuous nerve blocks, paravertebral blocks, or epidurals are the best choice for postoperative pain control. When appropriate, regional techniques should be used, preferably without opioids. Nonsteroidal anti-inflammatory drugs or other adjuvants should be used whenever possible to decrease opioid requirements. The use of patient-controlled analgesia (PCA) without the continuous-infusion mode represents a better alternative than opioids administered on a regular or as-required basis. The antidote for opioid overdose should be readily available to rescue the OSA patient with respiratory compromise. Supine position should be avoided, and lateral, prone, or sitting position should be used.

Continuous monitoring of oxygenation, breathing, blood pressure, and heart rhythm is required because of the possibility of serious complications such

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as hypertension, dysrhythmias, hypoxia, and airway obstruction, the latter potentially requiring urgent reintubation.

Discharge Criteria.

In addition to the standard discharge criteria used routinely in the postanesthesia care unit (PACU), patients with OSA should be able to maintain adequate oxygenation on room air with no hypoxemia or critical airway obstruction when they are asleep. OSA patients should stay at least 3 hours longer than non-OSA patients to ensure the required discharge criteria. OSA patients who develop an episode of hypoxemia or critical airway obstruction while in the PACU require an extended monitoring period of 7 hours (median time recommended by ASA guidelines in this circumstance) and the most careful evaluation thereafter (many anesthesia providers simply secure a postoperative bed in this instance). Patients with mild OSA who have undergone minor surgery (superficial plastic surgery, eye procedures, and superficial orthopedics) under local or regional anesthesia can be discharged home on the same day. Patients with moderate OSA and intermediate co-morbidities who have undergone intermediate-risk surgery (does not include abdominal, faciomaxillary, thoracic, or intracranial) should be hospitalized in a standard surgical unit. Patients with severe OSA, necessitating CPAP at home and/or who have had major surgical procedures, should be admitted to a step-down unit or intensive care unit until the threat of respiratory complications is no longer present.

TABLE 75.5 CONSULTANT OPINIONS REGARDING PROCEDURES THAT MAY BE PERFORMED SAFELY ON AN OUTPATIENT BASIS FOR PATIENTS AT INCREASED PERIOPERATIVE RISK FOR OBSTRUCTIVE SLEEP APNEA

TYPE OF SURGERY/ANESTHESIA	CONSULTANT OPINION
Superficial surgery/local or regional anesthesia	Agree
Superficial surgery/general anesthesia	Equivocal
Airway surgery (e.g., UPPP)	Disagree
Tonsillectomy in children <3 years old	Disagree
Tonsillectomy in children >3 years old	Equivocal
Minor orthopedic surgery/local or regional anesthesia	Agree
Minor orthopedic surgery/general anesthesia	Equivocal
Gynecologic laparoscopy	Equivocal
Laparoscopic surgery, abdominal surgery	Disagree
Lithotripsy	Agree

UPPP, uvulopalatopharyngoplasty.

Source: American Society of Anesthesiologists. Practice guidelines for the perioperative management of patients with obstructive sleep apnea. *Anesthesiology.* 2006;104(5):1087.

Outpatient Surgery.

Outpatient surgery for OSA patients remains controversial. According to Sabers et al., the presence of OSA does not increase the risk of readmission to the hospital after ambulatory surgery. However, this view is not shared by many same-day surgery anesthesia providers, and it is the practice in some ambulatory surgery centers to refuse care to sleep apnea patients (and sometimes even suspected sleep apnea patients). Risk must be assessed for each individual patient, because there are a large number of undiagnosed cases of OSA (Table 75.5).

OSA CHARACTERISTICS FOR CHILDREN

The peak period for OSA in the pediatric population is preschool age, with equal boy/girl distribution. The common causes are adenotonsillar hypertrophy, obesity, and craniofacial anomalies. Usually, children with OSA suffer growth retardation, hyperactivity disorders, developmental delay, or attention deficit disorders. Excessive daytime somnolence is uncommon in children with OSA. Sleep studies show normal sleep architecture and a <50% cortical arousal with apnea, but results should be interpreted according to the age of the child (Table 75.1). The treatment is primarily surgical (adenotonsillectomy), and CPAP is used as required.

Anesthetic management of children with OSA should not include premedication with sedatives and respiratory depressant medication, and a difficult airway should always be anticipated. Induction of general anesthesia is achieved by inhalational agents. The application of an artificial oral airway and CPAP will improve airway patency and mask ventilation. Administration of

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antisialagogue (anticholinergic) medications can be beneficial during both the intraoperative and postoperative periods. Neuromuscular blockade should either be avoided or full recovery from its effects ensured before extubation. Doses of opioids should be decreased, and it is preferable to use short-acting, noncumulative agents. The incidence of airway obstruction in the immediate postoperative period is higher in children with OSA, especially in those who undergo adenotonsillectomy. Because of the increased risk for postobstructive negative-pressure pulmonary edema that necessitates reintubation and mechanical ventilation, children with moderate to severe OSA should always be hospitalized overnight after surgery.

TAKE HOME POINTS

- Snoring, excessive daytime somnolence, and witnessed apnea episodes together with an AHI >5 are positive for OSA diagnosis.
- In the absence of sleep study, have a high index of suspicion to the diagnosis of OSA in the presence of risk factors.
- Avoid preoperative benzodiazepines or opioids.
- Use regional anesthesia whenever appropriate or general anesthesia with secured airway (an endotracheal tube or, controversially, a laryngeal mask airway).
- Extubate in semiupright position, when the patient is awake and fully recovered from neuromuscular blockade.
- Use supplemental oxygen with or without CPAP and monitor oxygen saturation.
- Avoid the supine position.
- Use adjuncts to decrease opioid requirement.
- Discharge adult patients when they reach their baseline room-air oxygen saturation and have no hypoxemia or critical airway obstruction when not stimulated.
- Pediatric patients with moderate to severe OSA should be hospitalized overnight after surgery.

SUGGESTED READINGS

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Have a High Index of Suspicion for Perioperative Pulmonary Embolism in Patients Who Have Traveled to Your Hospital By Air*

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Prolonged immobility associated with long-distance air travel predisposes patients to deep vein thrombosis (DVT) and venous thromboembolism (VTE). A recent systematic review found that long-haul flights of 8 hours or more increased the risk of thrombosis, with asymptomatic DVT developing in up to 10% of such travelers. The prolonged immobility and hypercoagulable state associated with major surgery (especially lower-extremity orthopedic or pelvic surgery) may add to this risk. Therefore, patients who travel long distances to have surgery may be at high risk for perioperative venothrombotic events. Indeed, in a large retrospective review at Mayo Clinic, patients traveling more than 5,000 km had a higher rate (≈30×) of perioperative VTE than those traveling a shorter distance before surgery. In that study, those in whom VTE developed also were younger, had VTE development significantly earlier in the postoperative course, had a higher American Society of Anesthesiologists (ASA) physical status classification, and were more likely to be smokers. Therefore, all perioperative physicians should be aware of this risk and must be ready to recognize even the subtle signs and symptoms of DVT and pulmonary embolism (PE) to avoid catastrophic perioperative complications of PE.

Several mechanisms have been proposed by which long-haul air travel may contribute to the risk of VTE. Long periods of relative immobility (also called "economy class syndrome"), especially in patients sitting in nonaisle seats; obstruction of venous return as a result of compression of popliteal veins at the edge of the seat; exposure to hypobaric, low-humidity air; the stress of travel; hypercoagulability (seen even in healthy volunteers exposed to a simulated airplane cabin environment); and possibly dehydration resulting from decreased fluid intake or excessive use of alcohol during the trip all may be additive risk factors.

Morbidity and mortality from VTE is attributable primarily to PE, which is sometimes difficult to diagnose in the perioperative period because clinical manifestations are often nonspecific. Manifestations include dyspnea, substernal chest pain, syncope, tachycardia or even cardiac dysrhythmias, and worsening of pre-existing congestive heart failure. Pleuritic chest pain and hemoptysis are seen only when pulmonary infarction has occurred. Physical examination may indicate lower-extremity swelling resulting from obstruction of venous outflow by thrombus in large vessels. P.322

Results of electrocardiography are altered in most cases, although abnormalities are often subtle and nonspecific. T-wave inversion in the precordial leads ("anterior ischemic pattern") is the most common finding in massive PE. Tachycardia and signs of right ventricular strain (right-axis deviation, peaked P waves, and ST-T-segment abnormalities) may be observed. When seen together, an S wave in lead I, a Q wave in lead III, and an inverted T wave in lead III are suggestive of PE. This "S1, Q3, T3" pattern has a sensitivity of about 50% for both massive and nonmassive PE.

Arterial blood gas analysis generally shows arterial hypoxemia with a widened alveolar-to-arterial O2 gradient and respiratory alkalosis. Because of its high sensitivity (>95%), testing for elevated D-dimers may potentially be a useful preoperative screen for patients suspected of having VTE. However, because D-dimer levels can fluctuate widely in the postoperative period, this test is not likely to be of great utility immediately after surgery without corroborative evidence. Definitive testing for DVT or VTE can only be accomplished with venous angiography, computed tomography, or pulmonary angiography (for PE only). Definitive testing may be merited in patients presenting with any of the above signs or symptoms, especially in long-distance travelers, a group known to have a higher probability of VTE.

Patients traveling long distances (especially overseas) before surgery should be made aware of the potential for increased risk of perioperative thrombotic complications. Preoperative in-flight prevention could include maintaining hydration, exercise, and elastic compression stockings. In addition, high-risk patients may benefit from perioperative pharmacologic prophylaxis for DVT and VTE. Because VTE is sometimes a difficult diagnosis to make in the perioperative period and its potential morbidity is so high, these patients should have careful perioperative surveillance. Unexplained hypoxemia, leg swelling, and electrocardiographic abnormalities are all alarming findings in a patient traveling a long distance for surgery.

TAKE HOME POINTS

- Venous thromboembolism is not rare in long-haul air travelers—ask your patients how they traveled to the hospital, especially if you are at a tertiary referral center.
- VTE is more common in patients who are younger, have higher ASA status, and who are smokers. P.323
- Consider examining your patients specifically for signs of VTE in the immediate preoperative period. Also consider a repeat electrocardiogram to look for new changes that might be indicative of PE.
- One final note—many referral hospitals in the Midwest and Western states (including those in the public health system) draw patients long distance by car or bus. Patients traveling more than 500 miles by car on the day before surgery may warrant similar consideration.

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Be Aware that Schizophrenic Patients Have Greater Perioperative Risks Than Age-Matched Controls

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A large body of evidence has demonstrated that schizophrenic individuals are afflicted with a wide array of medical problems. Some of these issues are likely due to genetic abnormalities that are associated with schizophrenia. Other medical concerns develop as a result of poor attention to self-care and inconsistent medical follow-up that arise from the hypofrontality/negative symptoms that actually generate most of the longitudinal morbidity of schizophrenia. Schizophrenic patients are also more likely to engage in lifestyle behaviors that affect their health status adversely, such as poor compliance with medications and/or perioperative instructions, smoking, poor dietary intake, and use of drugs and alcohol. It is also likely that antipsychotic medications contribute to this greater prevalence of medical co-morbidities. Taken together, these medical issues significantly increase operative risk in schizophrenic patients.

It has been estimated that schizophrenic patients have a 20% shorter life expectancy than age-matched controls. Another study found that schizophrenia is associated with a greater prevalence of heart disease, chronic obstructive pulmonary disease, hypertension, and diabetes. Some research suggests that there are increased rates of hyperglycemia and diabetes, hyperlipidemia, hypercholesterolemia, and obesity that occur as intrinsic, associated features of this illness. Obstructive sleep apnea is especially prevalent among schizophrenic patients. The operative risks posed by these problems are only exacerbated by the tendency for schizophrenic patients to neglect their own care. These metabolic abnormalities are allowed to persist without medical intervention far more frequently than is the case in the general population. Thus, progression to significant cardiac, pulmonary, and renal pathology is more frequent, leading to greater operative risk.

Although antipsychotic drugs are essential in the management of schizophrenia, the vast majority of these agents have been implicated in generating all of these metabolic abnormalities. Thus, these drugs can exacerbate the risks that already arise from the illness itself. Additionally, antipsychotic agents often increase the length of the QT interval, yielding a greater risk of malignant arrhythmias. One drug in particular, clozapine, has been associated with myocarditis as well as significant lowering of the seizure threshold. P.325

Although it may seem counterintuitive or irrelevant to the core symptoms that define schizophrenia, it is important to keep the greater prevalence of medical co-morbidities in mind when assessing operative risk in this patient population.

TAKE HOME POINTS

- Besides the obvious mental health issues, schizophrenic patients warrant careful attention to their medical status.
- The increase in perioperative risk is multifactorial.
- *Be aware that sleep apnea is especially prevalent in schizophrenic patients.* An anecdote from the author: It was a great relief for everybody (patients and staff alike) when the "dorm rooms" housing six patients were eliminated from the modern psychiatric hospital layout. "In the old days," it was not unheard of to have six large, male, schizophrenic patients, all of whom had gained 30 pounds from their atypical antipsychotic medications, all suffering from a degree of sleep apnea, disrupting each others' sleep, and making it difficult for anyone to get rested and/or well.

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Use Extra Care in Positioning Patients Who Have Had Amputations

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All anesthesia providers must take extra care with patients with amputations to avoid positioning injuries. Be aware that limbs that have sustained partial amputation have undergone subtle but very real changes in musculoskeletal structure, skin integrity, and somatic and autonomic innervation.

The joints proximal to an amputation are often affected on both the contralateral and ipsilateral sides. For example, because of increased weight bearing in the contralateral leg, it is very common after lower-limb amputation for the contralateral hip to develop significant osteoarthritis. This is true even for patients who use a prosthesis. On the ipsilateral side in above-knee amputations, a hip flexure contracture results from immobility (more time sitting) and a hip abduction contracture from loss of the insertions of the adductors to the distal femur. On the ipsilateral side in below-knee amputations, a knee flexion contracture develops as a result of impaired mobility and loss of quadriceps extension strength. This is very common even in relatively young and active patients and will predispose a patient to injury in any position involving extension at the knee, such as the simple supine position. A pad below the distal thigh allows mild knee flexion and protects the integrity of the skin at the end of the stump.

In all types of amputation, there can be subcutaneous fibrosis at the amputation site that puts the overlying skin at risk because of increased tension and decreased blood flow. In many patients with amputations, the underlying medical reason for the amputation (diabetes or vascular disease) may mean that the skin of the residual limb is at high risk for skin breakdown independent of amputation. Even in patients whose limb loss was due to trauma, there is a higher risk of skin breakdown because of concomitant skin disorders such as contact dermatitis and verrucous hyperplasia. Patients who show skin breakdown or ulceration on a residual limb should be positioned so that there is minimal chance of further pressure injuries. Perturbations in both the somatic and autonomic nervous systems can cause patients who have had amputations to experience a variety of hypersensivity and chronic pain syndromes, including causalgia and phantom limb pain. Positioning injuries therefore can also cause an acute-on-chronic pain syndrome that can be especially distressing to the patient.

It is now understood that the perception of pain involves cortical processing of afferent input to the nervous system. There is also a tremendously complex interplay among all types of pain and psychologic and emotional resilience. Amputation patients have an array of psychological “defenses” pertaining to their amputations, ranging from World War II veterans who say simply, “I got my leg shot off in Germany,” as if they were ordering coffee, to patients who carefully guard their residual limbs. Patients who have had recent amputations may also be prone to depression. Ask if there is pain in the limb and discuss the plan for careful positioning with the patient. This can lessen anxiety, which is a good start to preventing pain. It is also appropriate to check for changes in the usual pain pattern during the postoperative check. P.327

TAKE HOME POINTS

- A new generation of war-wounded patients will be entering care in both civilian and veterans hospital—a significant percentage of whom will have had one or more amputations.
- Be attentive to changes in musculoskeletal structure, skin, and innervation.
- Above-knee amputees can have hip flexion and abduction contractures requiring positioning that must account for this.
- Below-knee amputations can result in knee flexion contractures.
- Subcutaneous fibrosis can put the overlying skin at increased tension with decreased underlying blood flow.
- Positioning injuries can result in acute-on-chronic pain in amputated limbs.
- If possible, involve the patient in the positioning before induction of anesthesia.

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Positioning Patients for Spine Surgery: How to Minimize the Risks

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The obligatory positions required for spine surgery subject patients to significant risk above and beyond the inherent risk of the procedure itself. These risks include, but are not limited to, injury to the eyes, ears, nose, breasts, penis, extremities, and peripheral nerves. Peripheral nerve injury is a well-recognized anesthetic complication and is the second largest cause of malpractice actions in anesthesiology, accounting for 16% of claims. Minimizing potential injury to peripheral nerves through proper positioning and monitoring is extremely important.

Monitoring of somatosensory evoked potentials (SSEP) is frequently utilized during spine surgery (75%) and is available in most institutions in the United States (94%). In addition to monitoring spinal cord function, SSEP can detect peripheral nerve injury during spine surgery. Conduction changes, such as a decrease in amplitude or an increase in latency of the signal, are believed to indicate impending upper-extremity nerve injury. Modification of the arm position by the anesthesiologist often improves the SSEP signal and may return it to baseline. Reversal of position-related SSEP changes can influence impending nerve injury and prevent postoperative peripheral nerve injury.

The prone position is a dangerous position for patients. Attention to securing the airway is very important, as it is difficult to re-establish an airway in the prone position. Though we must always be cautious regarding protection of the eyes, this position raises additional concerns. Both ophthalmic ointment and occlusive eye tape may be appropriate if the procedure involves the cervical spine; prep solution that comes into direct contact with the eyes can lead to corneal injury. Additionally, we must assure that there is no direct pressure on the eye, ears, or nose. Pressure could lead to loss of function and/or a disfiguring ischemic injury. For the same reason, it is important when positioning chest rolls to assure yourself that the breasts in women and the genitalia in men are free from compression of any sort.

With regard to the patients' extremities, the mechanisms of nerve injury associated with surgical positioning and anesthesia are not completely understood. Peripheral nerve injury may be due to direct trauma or more commonly due to ischemia of the intraneural capillaries. Compression of peripheral nerves or stretch beyond 15% of original length may also cause peripheral nerve injury. Diabetes, hypertension, and uremia are known to affect peripheral nerves and predispose them to injury; intraoperative conditions such as prolonged hypotension and anemia are believed to facilitate ischemic injury. Finally, certain operative positions are known to place patients at increased risk for nerve damage. During spine surgery the overall incidence of impending upper-extremity peripheral nerve injury, as defined by changes in the SSEP, is >6%. P.329

The ulnar nerve and the brachial plexus are the most commonly injured neural structures during this type of surgery. Risk factors for ulnar nerve injury include male gender, very thin or very obese patients, and prolonged hospitalization for >14 days. Risk factors for brachial plexus injury include the use of shoulder braces, the prone head-down position, and some regional anesthetic techniques such as interscalene and axillary blocks.

There are five positions commonly employed during spine surgery: supine, arms out; supine, arms tucked; lateral decubitus; prone “Superman” position; and prone, arms tucked. The prone “Superman” position and the lateral decubitus position have been identified as high-risk positions for upper-extremity nerve injury, especially if the surgical procedure is prolonged. Techniques for minimizing injury in these positions are reviewed below.

The incidence of impending upper-extremity nerve injury during spine surgery in the supine arms-out position is 3.2%. Overstretch of the brachial plexus can occur as a result of abduction of the shoulder >90 degrees and should be consistently avoided. Direct compression of the ulnar nerve against the medial epicondyle may occur, especially if the forearm is in the prone position. Placing the forearm in the supine or neutral position decreases pressure over the ulnar nerve at the elbow. Elbows should be padded to further protect the nerve from compression. Direct compression of the radial nerve in the spiral groove of the humerus can be avoided with proper padding. Overextension of the elbow should be avoided because it may stretch the median nerve. The patient's head and neck should ideally be maintained in a midline position. Tilting the head and neck laterally may stretch the contralateral brachial plexus.

The incidence of impending upper-extremity nerve injury during spine surgery in the supine arms-tucked position during spine surgery is 1.8%. The tucked arms should be placed in neutral positions to decrease the pressure over the ulnar nerve. The ulnar nerve should be padded at the elbow for additional protection. Shoulder tape, which is often applied by the surgeon to pull the shoulders down and maximize the lateral radiographic view of the spine, may compress the brachial plexus. The duration of application of this shoulder tape should be minimized. The head and neck should be maintained in midline position. Placement of the patient's forearms on the lower abdomen should be avoided, as excessive flexion at the elbow joint can simultaneously stretch and compress the ulnar nerve. P.330

The lateral decubitus position is a challenging position for the anesthesiologist. The incidence of impending upper-extremity peripheral nerve injury during spine surgery in the lateral decubitus position is 7.5%. Compression of the brachial plexus is the most common mechanism of injury. The dependent brachial plexus can be compressed between the clavicle and first rib as well as against the humeral head. Obese patients and patients placed in a head-down position are at higher risk, as compressive forces are augmented. A chest roll should be placed to elevate the dependent chest and decrease the likelihood of compression. The chest roll should not be placed in the axilla, but under the lateral, superior chest; the goal is to prevent compression of the contents of the axillae. The dependent arm should be padded at the elbow and maintained in the supinated position to avoid compression of the ulnar nerve. To minimize the likelihood of brachial plexus stretch, abduction of the dependent and nondependent arm should be less than 90 degrees. Excessive flexion or extension at the elbow should be avoided. The elbow in the nondependent forearm should be padded, and lateral rotation of the shoulder should be avoided. The patient's head and neck should be maintained in a midline neutral position without excessive flexion or extension.

The prone “Superman” position is another challenging position with a relatively higher incidence of impending upper-extremity nerve injury (7%). Stretching forces on the brachial plexus is the commonest mechanism of injury. Again shoulder abduction beyond 90 degrees should be avoided, and the patient's head and neck should be in a neutral midline position. Elbows should be padded and the forearm and hand placed in a neutral position. Placing patients in steep head-down position should be avoided or limited if possible. Pressure from chest rolls should not be directly on the shoulders and the clavicle, as they may compress the brachial plexus, especially in obese patients. In women, the position of the chest rolls should be checked to ensure

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that there is no possibility of ischemic injury to the breasts. In men, the position of the lower roll should be checked to confirm that the genitalia are free from direct compression.

The prone arms-tucked position is relatively lower-risk compared to the prone "Superman" and the lateral decubitus positions. The incidence of impending nerve injury in the prone arms-tucked position during spine surgery is 2.1%. Special attention should be given to positioning the head and neck as described in the previous paragraph. The forearms should be in neutral positions. Shoulder tape, applied to maximize the lateral radiographic view of the neck, should be minimized. The elbows should be padded to avoid compression by arm sleds. Direct compression by the chest rolls is of concern as in the prone "Superman" position.

Attention to detail is one of the reasons for our specialty's superior safety record. When caring for patients undergoing spinal surgery, we must assume an integral role in positioning, monitoring, and interceding as appropriate.

TAKE HOME POINTS

- The prone position can be dangerous for patients. Attention must be given to preventing injury to eyes, ears, nose, breasts, penis, extremities, and peripheral nerves.
- Somatosensory evoked potential monitoring can be used to detect and prevent peripheral nerve injury during spine surgery.
- The lateral decubitus position and the prone "Superman" position are high-risk positions for nerve injury during spine surgery.
- Abduction of the shoulder beyond 90 degrees should be avoided because it stretches and may injure the brachial plexus.
- Excessive flexion of the elbow should be avoided; it stretches and compresses the ulnar nerve.
- Excessive extension of the elbow should be avoided because it stretches the median nerve.
- The forearm in the supinated position may be safest because of the decreased pressure on the ulnar nerve; the neutral position is a good second choice.
- Appropriate padding can minimize excessive pressure on the radial nerve in the spiral groove.

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Be Vigilant During Placement of the Camera in Laparoscopic Procedures and Always Watch Carefully for the Physiologic Effects of Carbon Dioxide (CO_2) Insufflation

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Laparoscopic surgery presents challenging issues for the anesthesiologist. It is one of the few procedures in which the surgical "incision" has resulted in life-threatening events. It is also in the category of surgical procedures (much like the methylmethacrylate cases in orthopedic surgery) in which the *surgeons* administer a substance with wide-ranging physiologic sequelae.

The first task for the anesthesiologist is to ascertain by query or observation the planned technique for initial entry into the abdomen. Surgical practice has tended to evolve away from the Veress needle technique, which involves picking up the skin on either side of the umbilicus and using a "blind" technique to first pass a needle followed by a trochar. This technique has resulted in the uncommon but devastating complications of aorta and bowel injury. Much more common today is the Hassan cannula technique, which involves a microlaparotomy and placement of the initial intra-abdominal instrument (which is blunt-tipped) under direct observation. Subsequent trochars are then placed under camera visualization after insufflation.

Try to observe the placement of the trochars personally. It is not necessarily true that several attempts to place trochars and/or failure to initially insufflate to a proper pressure means trouble, especially with the Hassan technique. The trochars have a "cocking" mechanism that allows the sharp point to protrude out of the sheath, and this may need to be reset if activated when the surgeon attempts to place the trochar through the layers of the abdominal wall. Also, there may be leakage around the Hassan cannula, and the surgeon may ask for Xeroform to wrap around the cannula to get a seal. In general, however, placement of the trochars should go fairly smoothly without an excessive number of attempts.

Of course, it is the insufflation of the abdomen or pelvis with gas (usually CO_2) to improve visualization of the surgical field that results in the physiologic changes that are of interest to us. The high solubility of CO_2 allows rapid diffusion into the blood compartment and causes predictable changes in blood gas chemistries and hypercapnia. It must be remembered at all times
that the physiologic effects of insufflation and the resulting hypercapnia are multisystemic and quite complex. P.333

CARDIOVASCULAR EFFECTS

The potential cardiovascular effects are the ones most pronounced at the time of and just after insufflation. They require that a range of resuscitative drugs be available. Hypercarbia and the associated acidosis cause direct cardiac depression and peripheral vasodilatation, which can lead to hypotension. However, hypercarbia also promotes a strong sympathetic reflex and increases in plasma catecholamine levels may offset this depressant activity. The result is a hyperkinetic circulation with hypercarbia causing a tachycardia, increased cardiac output, increased contractility, and increased systemic blood pressure. These changes may increase myocardial O_2 demand, and patients with known or suspected coronary artery disease will require appropriate interventions. In addition, arrhythmias are a commonly encountered problem during laparoscopic procedures. Hypercarbia, acidosis, and the associated increase in catecholamines may sensitize the myocardium and induce a variety of arrhythmias. Vagally mediated reflexes caused by insufflation can induce a variety of bradyarrhythmias, including asystole. Lastly, increases in intra-abdominal pressure can inhibit venous return, resulting in decreased cardiac output and hypotension; adjustments to intravascular volume may be required to normalize venous return and maintain cardiac output.

INTRACRANIAL EFFECTS

The intracranial effects of insufflation are the easiest to overlook, as it is extremely rare to have direct measurement of intracranial pressure (ICP) during laparoscopic surgery.

CO_2 is the primary regulator of cerebral blood flow through its effect of inducing cerebral vasodilatation. Cerebral blood flow increases linearly with changes in P_{CO2} between 20 and 100 mm Hg. ICP is a function of brain mass, cerebrospinal fluid volume, and blood volume. Although the relationship between cerebral blood flow and ICP is complex, a general assumption should be that increases in P_{CO2} will increase ICP. In the absence of direct ICP monitoring, prudence suggests that any change in CO_2 above normal may increase ICP. It is not uncommon for a senior practitioner to hear the question, "How high can we let the CO_2 go?" A reasonable answer is that acceptable limits to the increase in P_{CO2} during laparoscopic procedures should be directly related to concerns regarding elevated ICP. Thus a patient who should be exposed to only moderate increases in ICP should undergo only moderate increases in P_{CO2}.

PULMONARY EFFECTS

Insufflation of the abdomen is generally considered among the stronger indications for endotracheal intubation because the pulmonary changes that occur during CO_2 insufflation are usually related to increased intra-abdominal pressure. The upward displacement of the diaphragm can predispose to P.334
passive or active regurgitation, cause atelectasis, reduce functional residual capacity (FRC), create an intrapulmonary shunt, and produce ventilation/perfusion (V/Q) mismatching and hypoxemia. Higher P_{CO2} levels as the case progresses may also cause the resumption of spontaneous ventilation. To offset these changes, administration of higher doses of neuromuscular blockade, adjustments to the rate of mechanical ventilation, tidal volume, inspiratory flow rate, and the implementation of lung volume recruitment efforts are all part of intraoperative management during laparoscopic procedures. In addition, the anesthesiologist must always be vigilant for increases in airway pressure caused by a pneumothorax, which can occur when laparoscopic procedures are performed close to the diaphragm (e.g., gastric fundoplication).

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SOFT TISSUE EFFECTS

It is common for patients to develop subcutaneous emphysema during long laparoscopic procedures. Because of the high solubility of CO_2, considerable quantities can accumulate within body tissues during long procedures. Consider carefully and on an individual basis before extubating patients who have undergone extended laparoscopic procedures. The large majority of patients can be and are extubated at the end of the procedure, but there are several issues complicating extubation. Subcutaneous emphysema involving the head and neck can be ascertained by visual inspection or by palpation—the patient may have a "crackling" under the skin. This may require postoperative intubation to prevent airway obstruction and while tissue stores are removed. Also, always be aware that subcutaneous emphysema can be a sign of pneumothorax. Additionally, elderly patients and/or those with pre-existing pulmonary disease may be unable to sustain the P_{CO2}-induced high minute ventilation following extubation and are at higher risk for reintubation.

One final note regarding the management of the sequelae of insufflation and hypercapnia is that in patients with comorbidities, those with impaired CO_2 excretion capacity (e.g., patients with chronic obstructive pulmonary disease), and patients with acute cardiopulmonary disturbances, strongly consider an arterial line for hemodynamic and blood gas monitoring.

TAKE HOME POINTS

- Personally observe placement of the trochar—make sure the surgeons are using the Hassan technique.
- The physiologic effects of insufflation and hypercapnia are complex and can result in significant sequelae in cardiovascular, intracranial, and pulmonary status.
- Check every patient for significant subcutaneous emphysema in the chest and neck—look for "crackling" under the skin. Subcutaneous emphysema can indicate a clinically significant pneumothorax or predict a failed extubation.

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81

Do Not Use Urine Output as an Indicator of Volume Status in Hypothermic Patients

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Daniel R. Brown MD, PhD

Hypothermia is a clinical entity defined as a core body temperature less than 35°C (95°F) and is classified in four stages depending on the temperature, symptomatology, and effect on specific organ physiology (Table 81.1).

All organs are ultimately affected by hypothermia, including the kidneys. The renal response to cold is rapid and varies with the different stages of hypothermia. Initially, peripheral vasoconstriction results in relative central hypervolemia producing an increase in urine output. This response, termed "cold diuresis," has been described even in patients with mild to moderate hypothermia. The etiology of this phenomenon is multifactorial and includes an initial increase in cardiac output and renal blood flow resulting from hypothermia-induced changes in vascular capacitance. Other important contributors are nonosmotic suppression of antidiuretic hormone (ADH) release by the hypothalamus and subsequent decreased renal tubular reabsorption. These responses usually begin as soon as the core body temperature reaches 35°C and become more pronounced until moderate hypothermia, when decreased renal blood flow and glomerular filtration rate (reduced 50% at 27°C to 30°C) may lead to renal failure.

Even in the setting of a large diuresis (the urine is usually dilute, with osmolarity <300mOsm/L and specific gravity <1.003), the kidneys are unable to handle nitrogenous waste because of tubular dysfunction. Although they are uncommon, electrolyte disturbances including hypernatremia, hyperchloremia, and hyperkalemia can occur and are more frequent as hypothermia progresses in duration and/or severity. "Cold diuresis" is exacerbated by ethanol ingestion and water submersion, which may coexist with hypothermia and can potentiate inappropriate diuresis by inhibiting ADH secretion.

It is important to understand the pathophysiology of this phenomenon when making clinical decisions regarding fluid management in hypothermic patients. The "cold diuresis" can be massive and generally creates a hypovolemic state that worsens with rewarming because of the reverse changes in vascular tone as core body temperature is raised. If it is overlooked or underappreciated, this phenomenon can exacerbate electrolyte disturbances, contribute to hypotension, and result in prerenal stress.

P.337

TABLE 81.1

STAGE	CORE TEMPERATURE, °C (°F)	CHARACTERISTICS
Mild	32 (89.6) to 35 (95)	Increased metabolic rate, hypertension, tachycardia, shivering, *cold diuresis,* CNS hyperexcitability, coagulopathy
Moderate	28 (82.4) to 32 (89.6)	Decreased cardiac output, hypoventilation, CNS depression, atrial arrhythmias, ↓ O_2 consumption (~ 25-50%)
Severe	22 (71.6) to 28 (82.4)	Progressive hypotension and bradycardia, ventricular arrhythmias, VF, decreased CBF, areflexia, loss of bulbar reflexes, decreased O_2 consumption (<50% of baseline)
Profound	<22 (71.6)	Asystole, EEG burst suppression

CNS, central nervous system; VF, ventricular fibrillation; CBF, cerebral blood flow; EEG, electroencephalogram.

Intravascular volume status should be closely monitored to avoid complications of this "physiologically inappropriate" renal response. Initially, it should be assumed that the patient is significantly dehydrated. Frequent measurements of electrolytes and hematocrit will help guide fluid therapy and electrolyte replacement, and help monitor for dehydration. Central venous access should be considered to allow for safer electrolyte replacement and rapid-volume administration, though electrolyte abnormalities may increase cardiac irritability and arrhythmia risk during catheter placement. Invasive arterial blood pressure monitoring should also be considered to facilitate laboratory determinations and to evaluate fluid responsiveness.

TAKE HOME POINTS

- The kidneys respond rapidly to hypothermia.
- Cold diuresis will occur even in patients with mild to moderate hypothermia.
- The etiology is multifactorial, including changes in cardiac output, organ perfusion, and suppression of ADH.
- Cold diuresis is exacerbated by ethanol ingestion and water submersion.
- Assume that cold patients are significantly dehydrated—consider central venous access and invasive arterial blood pressure monitoring.

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82

Anesthesia for Eye Surgery—The Innate Culture of "1-N-1"

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"Indolence is a delightful but distressing state; we must be doing something to be happy"

—Mahatma Gandhi

"What are your concerns during an eye surgery?" we asked the resident. "My attending might walk into the room and catch me snoozing!" was his reply. "What about your anesthesia?" we tried challenging him. "Oh! I will just use my one-and-one," he said cautiously.

This benign scenario is disturbingly common across the anesthesia community. With the advancement and increasing acceptance of topical and regional techniques, general anesthesia is uncommonly needed for ophthalmic surgeries. Even with regional techniques, the onus of block performance has shifted toward the surgeons, further impeding our involvement in patient care. Under these circumstances, it is not surprising that more and more anesthesia providers dislike working on the "eye surgery" cases, because they all "enjoy" doing the "stuff" and hate *just sitting there* . Interestingly, there is a lot more to anesthetic care for eye surgeries than just providing 1 mL of midazolam and fentanyl. The purpose of this chapter is to highlight some of these vital issues.

Topical and regional anesthesia seems to be adequate for the majority of eye cases. Most ophthalmic surgeons prefer the quicker recovery with these techniques and do not request "general anesthesia" unless it is necessary. For some cases, however, it is necessary (Table 82.1). It is also occasionally necessary to convert to a general anesthetic in the middle of the procedure. When faced with a request for general anesthesia, junior anesthesia providers sometimes have a tendency to both "oversedate" and "overdebate" before complying with the request. There are several ways to avoid this.

The first step is to make sure that a complete set of intubating and resuscitative drugs is available and readily accessible as well as laryngoscopes and endotracheal tubes.

After that, a little understanding about ocular anatomy helps in predicting cases that may not be effectively covered by available regional techniques. For most ocular surgeries, blocking five cranial nerves (CN II through CN VI) is usually sufficient. CN II (optic nerve) conveys vision; CN III (oculomotor), IV (trochlear) and VI (abducens) supply the extra-ocular muscles; while the sensory supply of the globe (or eyeball) is mainly from the ophthalmic division of CN V (trigeminal). The periorbital skin (eyelids, eyebrow, forehead, and nose) is supplied by branches of the ophthalmic division of trigeminal nerve and the intraorbital nerve (a division of V_2). Most of the extraocular muscles arise from the annulus of Zinn at the orbital apex and insert onto the globe near the corneal limbus. Together they form a conical potential space (annulus or muscle cone) behind the globe that encases the ophthalmic artery, cranial nerves II, III, and the nasociliary branch of the trigeminal nerve. CN IV (the trochlear nerve), which supplies the superior oblique muscle, lies outside the muscle cone.

P.340

TABLE 82.1 INDICATIONS FOR GENERAL ANESTHESIA IN OCULAR SURGERIES

Absolute	
1)	Pediatric patients
2)	Uncooperative patients (e.g., those with altered mental status, anxiety, or severe claustrophobia)
3)	Lengthy procedures (>3 hours)
4)	Surgical procedures not adequately covered by regional anesthetic techniques
Relative	
1)	Patients with penetrating eye injuries
2)	Patients on anticoagulant therapy
3)	Surgical cases requiring complete ocular akinesia
4)	Patients with a history of nystagmus

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5) Anatomic abnormalities making regional techniques challenging or unsafe

6) Patient's or surgeon's preference

Source: Navaleza JS, Pendse SJ, Blecher MH. Choosing anesthesia for cataract surgery.

Ophthalmol Clin North Am . 2006;19(2):233-237.

As expected, injection of local anesthetic solution within or around the muscle cone reliably anesthetizes the globe. Anesthetizing the orbit (the eye socket and all its contents), in contrast, can be more challenging due to anatomical consideration its conspicuous sensory nerve supply (Fig. 82.1). Thus, oculoplastic cases involving surgical manipulation of the orbit are less well tolerated under regional anesthesia. For instance, the senior author of this chapter estimates that up to 40% of his oculoplastics cases require general anesthesia (similarly, expect that enucleation of a blind, painful eye will often require general anesthesia for patients' psychological welfare). Frequently employed ocular regional techniques for most other cases include retrobulbar, peribulbar, and subtenon block. When the block needle is inserted within the muscle cone, the regional technique is called a retrobulbar block. To reduce the complication rates associated with this technique, peribulbar block was later described, whereby anesthetic solution is injected around (but not within) the muscle cone. Pertinent differences between these techniques are outlined in Table 82.2. Despite providing adequate anesthesia to the P.341
globe, these techniques fail to provide complete akinesia of the eyelids. The muscles involved in eyelid movements are the orbicularis oculi (innervated by the facial nerve, CN VII) and the levator palpebrae (innervated by the oculomotor nerve). For prolonged surgeries, squeezing by the eyelids is prevented by separate facial nerve blocks by Van Lint, O'Brien, Atkinson, or Nadbath technique. Various other regional anesthetic techniques such as subtenon, infraorbital, supraorbital, infratrochlear, and supratrochlear nerve blocks are often utilized by ophthalmic surgeons, but their discussion is beyond the scope of this chapter.

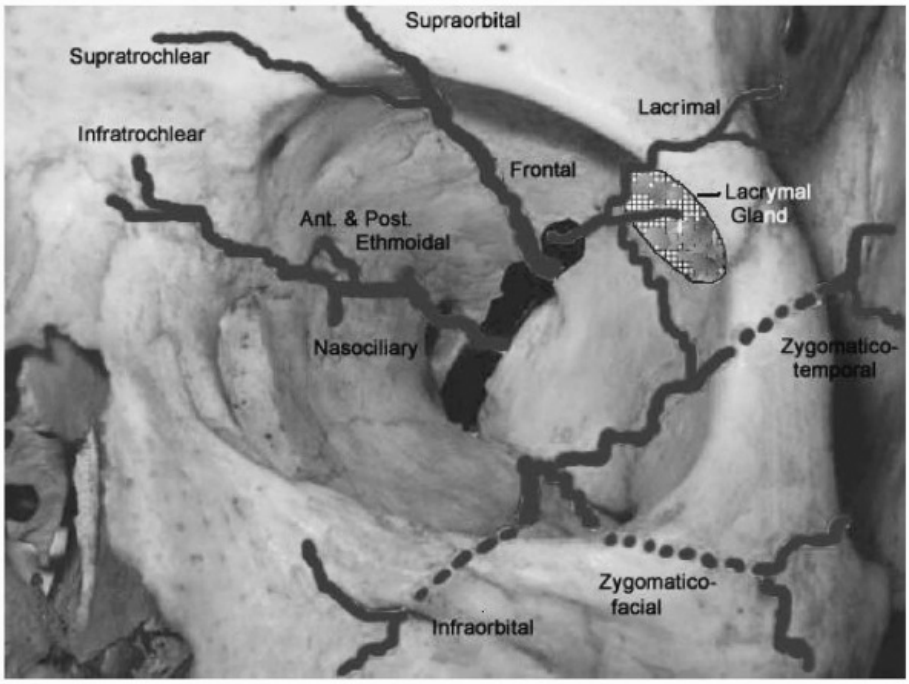


FIGURE 82.1. Sensory nerve supply to the orbit. [Modified from Rene C. Update on orbital anatomy. *Eye.* 2006;20(10):1119-1129.]

In this changing era, most ocular regional techniques are performed by the surgeons. Each technique comes with its own set of complications (Table 82.3). It is important for anesthesiologists to know how to detect and manage these problems. Communication with the surgeon will help in knowing the specific technique(s) planned and the anticipated issues. Retrobulbar block is often known to be associated with more frequent and worrisome problems that require close monitoring during and after the block. Retrobulbar hemorrhage is the most common ocular complication (incidence ~1% to 2%) and presents clinically as increasing proptosis, and subcutaneous and/or subconjunctival ecchymosis. Depending on the severity, treatment may vary from intermittent digital pressure application to immediate decompressive lateral canthotomy by the ophthalmologist. P.342

TABLE 82.2 RETROBULBAR VERSUS PERIBULBAR BLOCK

RETROBULBAR BLOCK	PERIBULBAR BLOCK
▪ Also known as the “intraconal block” because the needle tip lies within the muscle cone (formed by four recti, the superior and inferior oblique muscles)	▪ Considered as a safer alternative to retrobulbar block ▪ Also termed the “extraconal” block because the needle tip remains outside the muscle cone

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▪ Low-volume block, requires 4 mL of anesthetic solution ▪ Rapid onset of 3 to 5 minutes ▪ Provides reliable anesthesia ▪ Provides reliable, but not complete, akinesia of the eyelids ▪ Carries a higher risk of complications	▪ High-volume block (~8 mL), which depends on the diffusion of anesthetic solution into the muscle cone across connective tissue ▪ Slower onset time, typically 8 to 12 minutes ▪ Supplemental injections are required in up to 20% of cases ▪ Lesser risk of inadvertent globe perforation ▪ Does not provide reliable akinesia of the eyelids

Source: Troll GF. Regional ophthalmic anesthesia: safe techniques and avoidance of complications. *J Clin Anesth* . 1995;7(2):163-172.

Systemic complications are often quicker to present and normally require intubation and/or full cardiopulmonary resuscitation. It is recommended to prepare airway equipment and emergency drugs before initiation of any periocular block. Two of the systemic complications, retrobulbar apnea syndrome and oculocardiac reflex, are worth mentioning. Retrobulbar apnea syndrome (RAS) occurs as a result of inadvertent injection of local anesthetic P.343
solution into the optic nerve sheath. The anesthetic solution presumably spreads along the nerve sheath to subdural or subarachnoid areas, leading to brainstem anesthesia. Signs and symptoms include initial drowsiness, mild agitation, or confusion, progressing to respiratory depression or arrest within a few minutes of injection. The presence of ptosis, mydriasis, loss of vision, and contralateral ophthalmoplegia often help in formulating an early diagnosis. Treatment is often supportive, but requires cardiopulmonary resuscitation. The oculocardiac reflex (OCR), frequently seen in the context of strabismus cases in children, has also been described following retrobulbar block. It often manifests as cardiac arrhythmia, such as sinus bradycardia, nodal rhythm, ectopies, or even asystole. Treatment is mainly supportive.

TABLE 82.3 COMPLICATIONS OF PERIOCULAR REGIONAL TECHNIQUES RELEVANT TO ANESTHESIOLOGISTS

OCULAR COMPLICATIONS	SYSTEMIC COMPLICATIONS
1. Retrobulbar hemorrhage 2. Perforation of globe 3. Penetration of optic nerve 4. Optic atrophy and retinal vascular occlusion 5. Postoperative ptosis	1. Allergic reaction to local anesthetics 2. Local anesthetic toxicity 3. Arterial injection 4. Retrobulbar apnea syndrome (secondary to optic nerve sheath injection) 5. Oculocardiac reflex

Sources: Troll GF. Regional ophthalmic anesthesia: safe techniques and avoidance of complications. *J Clin Anesth* . 1995;7(2):163-172.
Ben-David B. Complications of regional anesthesia: an overview. *Anesthesiol Clin North Am* . 2002;20(3):665-667, ix.

Given the facts, it is evident that anesthesia for eye surgery requires a fair amount of preparation and constant vigilance during and after the regional block. Any indolence on the part of the anesthesia provider could potentially turn out to be a life-threatening situation for the patient.

TAKE HOME POINTS

- Anesthesia for eye surgery requires fair understanding of periocular regional techniques, especially the complications associated with them.
- Constant watchfulness is crucial in early detection and treatment of complications associated with these techniques.
- General anesthesia is not infrequently required for ocular surgeries.

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83

Remember that Fires in the Operating Room Can Be Prevented By Minimizing Just One Component of the Fire Triad

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Catherine Marcucci MD

You are on call and are notified that a patient is coming to the operating room (OR) urgently for a stab wound to the neck. You make your way to the room that has been set up for cases from the emergency room, the standard OR table is already made up with a draw sheet. You know that this is actually a polyurethane foam mattress, covered with a synthetic fabric, covered with a cotton sheet, but of course, you are not really thinking about that right now. The patient arrives shortly in a cloth hospital gown and a paper hat. She is a young woman who ran into trouble while out clubbing; she is wearing both hair gel and perfume. After induction, the patient's skin is quickly cleaned with an alcohol-based prep solution, with little or no time to place pads to catch drips.

Her head and neck are draped as quickly as possible with four cotton towels and a clear incise drape. Dry sponges are placed on the field. The patient is breathing high-concentration oxygen or perhaps a combination of oxygen and nitrous oxide. Of course, there was not time to check for frayed power cords or current inspection stickers, and that's not something the anesthesia provider typically does anyway. You notice there are multiple foot pedals at the surgeon's feet and wonder what the cases had been in that room during the day. The surgical case starts and you notice several times that the electrosurgical unit (ESU) pencil is not holstered when not in use; instead it is allowed to rest on the drapes. You see the ESU spark a bit the next time the surgeon picks it up, then suddenly, it "flashes" on you—this is the perfect setup for an OR fire.

Fire is the result of a chemical reaction between a fuel and oxygen. It will occur in any setting where the three elements that form the fire triad (fuel, oxygen, and heat) come together. Approximately 100 surgical fires are reported each year, resulting in about 20 serious injuries and one to two patient deaths, although these numbers may represent only a fraction of actual fires. Besides the devastating consequences of physical injury and death, OR fires can cause great psychological trauma to patients, family members, and staff. Unfortunately, there are also unfavorable public relations as well as potential legal ramifications.

The vast majority of fires ignite on or in the patient—34% in the airway (see Chapter 12), 28% on the head or face, and 38% elsewhere on or inside the patient. Thus, the most common injuries are airway burns, skin or internal burns, and burns to the patient's unprotected eyes during laser surgery. Also, the volatile gases and toxic fumes and by-products that are produced as synthetic materials melt are caustic to both the skin and eyes and can be lethal if inhaled. Most flames have a temperature that is approximately 1,200°C, with ambient temperatures ranging from 90°F near the floor to 600°F. Smoke from an uncontrolled fire can fill a room in approximately 3 minutes. P.345

A fuel is anything that will burn. Remember that the OR environment has abundant fuel sources. These include bone cement, cloth and paper items (drapes, dressings, sponges, patient gowns), plastic and rubber items (anesthesia circuit, blood pressure cuffs, endotracheal tubes, mattresses, masks, positioning devices, and tourniquets), solutions and ointments (petroleum-based ointments, prep solutions, suture pack solutions, tincture of benzoin), and the patient (head, facial, and body hair, gastrointestinal methane gas, hairspray, and perfume). Items that would not normally burn in room air (21% oxygen) most likely will burn in an oxygen-enriched environment. The higher the oxygen concentration, the less energy is required to ignite the materials. It has been found that an oxygen-enriched environment was present during 74% of fires. Nitrous oxide is also an oxidizer and is capable of supporting combustion.

High-energy ignition sources are also plentiful in today's ORs, including ESUs, lasers, and fiber-optic light cords. Other heat sources include high-speed burrs and drills, embers from charred tissue, and sparks produced from activating the ESU pencil on the end of a hemostat.

Prevention of OR fires is widely recognized to be based on the team concept. It is the responsibility of *all* OR team members to be cognizant of the factors that predispose to fire (including human complacency!) and to understand how the various OR team members will function in the event of a fire. Communication and cooperation are vital. The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) recommends a number of steps to reduce the risk of fire and fire injuries, including staff education; developing, implementing, and testing procedures; understanding laser basics, principles of electricity, and mode of operation of the ESU; the location of emergency equipment; and the location of gas shut-off valves and who is authorized to turn them off.

In the event of a "surgical" fire (i.e., other than an airway fire), the goal is to remove the drapes from the patient as quickly as possible to protect against burns and inhalation of toxic gases and then stabilize and treat the patient as necessary. In some cases, it will be necessary to first douse and then remove the drapes. The scrub nurse will assist with wet towels and water. Drapes should be removed horizontally to prevent spread of the fire up the drapes. Remember that paper drapes are water-repellant, so a fire burning under these drapes still may not be completely extinguished. The scrub nurse will also help pack open incisions with saline-soaked laps, and then move away from the table to assemble the instruments necessary to close the patient. The circulating nurse will call for help, notify the charge nurse, activate the alarm, and assist the anesthesia provider in preparing to move the patient to another room if required. P.346

The anesthesia provider must replace wall anesthetic gases with tank gases (in case the charge nurse or fire chief gives the order to discontinue wall gases) and get ready to move patient elsewhere so the procedure can be completed. OR personnel should *not* try to contain fires that extend beyond the patient—the building and sprinkler systems are designed to do this. Rather, the goal is to "put the patient out" and then get him or her moved as quickly as possible to safety. Keep in mind that hospital sprinkler systems are flushed regularly, but the 300 gallons of water that will be dumped in the OR are far from sterile, so the patient should be covered as much as possible.

Every modern OR will have a fire and fire evacuation plan. The authors strongly encourage anesthesia providers who are new to an operating room theater to obtain a copy and review it thoroughly. This is truly an area of clinical practice that deserves the utmost vigilance.

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TAKE HOME POINTS

- The fire triad is oxygen, fuel, and heat.
- All are plentiful in the OR, so instead of viewing OR fires as unlikely or rare events, consider that OR conditions are actually very favorable for the creation of fires.
- Develop a method to maintain vigilance with respect to fires. One of the authors used to keep an empty matchbook cover in her tackle box with her airway equipment as a "reminder."
- If the fire is not an airway fire, the anesthesia provider must be ready to switch from wall gases to tank gases and immediately prepare to transport the patient to safety.
- Actively seek out the fire and fire evacuation plan when joining a new OR staff. Attend all fire drills given by your operating room department.

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Remember that Elevated Temperature is a Late Finding in Malignant Hyperthermia

James C. Opton MD

Malignant hyperthermia (MH) is a clinical syndrome characterized by uncontrolled skeletal muscle metabolism. First defined in 1960 by Denborough and Lovell, it is classically "triggered" by volatile anesthetics and the depolarizing muscle relaxant succinylcholine in genetically susceptible individuals. MH is caused by uncontrolled calcium release in skeletal muscle cells as a result of abnormal function of the ryanodine receptor (RYR1). Diagnosis can be made clinically, by caffeine-halothane contracture testing of a muscle biopsy specimen, or by genetic testing. The caffeine-halothane test is very sensitive. However, it is invasive and expensive (about $800). Genetic testing for one of several known mutations of the RYR-1 genes is done via a simple blood test. Although it is not very sensitive, it is very specific and much less costly (about $200).

The overall incidence of MH-susceptible patients in the general population is hard to estimate, because it is a "cluster" disease. However, in 2005, there were 12 confirmed cases and an additional 10 probable cases in the United States, according to an official at the Malignant Hyperthermia Association of the United States (MHAUS).

MH can occur at any time during anesthesia or in the immediate postoperative period. It is the standard of care to ask every patient exposed to a triggering agent if he or she has a family history suspicious for MH ("Has a family member ever had a dangerous or unusual reaction to anesthesia? etc."). Although it is a rare disease, it is an important one in terms of anesthetic practice—anesthesiologists are expected from a medical/legal standpoint to be able to recognize and treat MH even if they are seeing it for the first time.

The most consistent indicator of potential MH is a large increase in end-tidal carbon dioxide. End tidal CO_2 can double or triple in minutes (although it may increase over hours). Masseter rigidity is another early sign, and tachycardia, arrhythmias, unstable or rising blood pressure, cyanosis and mottling, myoglobinuria, and tachypnea follow. Whole-body rigidity is a specific sign of MH and is seen in the limbs, abdomen, and chest if muscle relaxants have not been used. Respiratory and metabolic acidosis indicate fulminant MH and are typically followed by temperature elevation, a late sign. The temperature rise can be rapid and may exceed 43°C. Rhabdomyolysis
and disseminated intravascular coagulation (DIC) may also occur. Death is usually from cardiac arrest secondary to acidosis or hyperkalemia. In the dantrolene era, mortality from MH is less than 10%. P.349

TREATMENT OF ACUTE MH

- Call for help! Get dantrolene!
- Discontinue volatile anesthetics and succinylcholine.
- Hyperventilate with 100% oxygen at high fresh gas flows.
- Give dantrolene 2.5 mg/kg by intravenous (IV) bolus. Repeat dose as needed until signs of MH are controlled.
- Place two large-bore peripheral IV lines and an arterial line.
- Treat acidosis with bicarbonate as guided by blood gas analysis.
- Actively cool patient with IV cold saline, nasogastric lavage, rectal lavage, and surface cooling. Discontinue cooling when temperature has fallen to 38°C.
- Treat hyperkalemia with hyperventilation, bicarbonate, insulin with glucose. Life-threatening hyperkalemia should be treated with calcium.
- Dysrhythmias usually respond to treatment of acidosis and hyperkalemia. If not, use appropriate antiarrhythmics with the exception of calcium-channel blockers.
- Serially monitor end-tidal CO_2, arterial blood gases (ABG), serum potassium and other electrolytes, urine output, and international normalized ratio (INR)/prothrombin time (PTT).
- Ensure urine output of at least 2 mL/kg/h by hydration and/or diuretics. Consider central venous or pulmonary artery monitoring to guide fluid management.
- Call the MH Hotline.

TREATMENT OF POSTACUTE MH

- Observe in intensive care unit (ICU) for at least 24 hours, as recrudescence may occur.
- Give dantrolene 1 mg/kg IV every 4 to 6 hours for 24 to 48 hours after the episode.
- Follow arterial blood gases, creatine kinase (CK), potassium, coagulation factors, urine myoglobin, and temperature until they return to normal. CK may stay elevated for up to 2 weeks.
- Report patients to the North American MH Registry of MHAUS and refer patients to MHAUS for information.

WHAT TO STOCK IN THE MALIGNANT HYPERTHERMIA CART

The MHAUS is very specific about what needs to be stocked in the MH cart. In addition to the standard American Society of Anesthesiologists (ASA)
monitors, all locations where general anesthesia is administered should have a posted plan to treat MH; a means to continuously monitor end-tidal CO_2, P.350

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blood oxygen saturation, and core body temperature; and a means to actively cool a patient. A malignant hyperthermia cart stocked with the following should be immediately available wherever general anesthesia is administered:

Drugs

1. Dantrolene sodium for injection: 36 vials (each able to be diluted at the time of use with 60 mL sterile water).
2. Sterile water for injection USP (without a bacteriostatic agent) to reconstitute dantrolene: 1,000 mL × 2.
3. Sodium bicarbonate (8.4%): 50 mL × 5.
4. Furosemide 40 mg/ampule × 4 ampules.
5. Dextrose 50%: 50-mL vial × 2.
6. Calcium chloride (10%): 10-mL vial × 2.
7. Regular insulin 100 U/mL × 1 (refrigerated).
8. Lidocaine for injection, 100 mg/5 mL or 100 mg/10 mL in preloaded syringes (×3). Amiodarone is also acceptable. Advanced cardiac life support (ACLS) protocols, as proscribed by the American Heart Association (AHA), should be followed when treating all cardiac derangements caused by MH.

General Equipment

1. Syringes (60 mL × 5) to dilute dantrolene.
2. Mini-spike IV additive pins × 2 and Multi-Ad fluid transfer sets × 2 (to reconstitute dantrolene). Call MHAUS for ordering info.
3. Angiocaths: 16G, 18G, 20G, 2-in; 22G, 1-in; 24G, ¾-in (4 each) for IV access and arterial line.
4. Nasogastric (NG) tubes: sizes appropriate for your patient population.
5. Blood pump.
6. Irrigation tray with piston syringe (× 1) for NG irrigation.
7. Toomey irrigation syringes (60 mL × 2) for NG irrigation.
8. Microdrip IV set (× 1).

Monitoring Equipment

1. Esophageal or other core temperature probes.
2. Central venous pressure (CVP) kits (sizes appropriate for your patient population).
3. Transducer kits for arterial and central venous cannulation.

Nursing Supplies

1. A minimum of 3,000 mL of refrigerated cold saline solution.
2. Large sterile Steri-Drape (for rapid drape of wound).
3. Three-way irrigating Foley catheters (sizes appropriate for your patient population).
4. Urine meter × 1.

5. Irrigation tray with piston syringe.
6. Large clear plastic bags for ice × 4.
7. Small plastic bags for ice × 4.
8. Bucket for ice.

Laboratory Testing Supplies

1. Syringes (3-mL) for blood gas analysis for ABG kits × 6.
2. Blood specimen tubes (each test should have 2 pediatric and 2 large tubes): (A) for CK, myoglobin, SMA19 (LDH, electrolytes, thyroid studies); (B) for PT/PTT, fibrinogen, fibrin split products; (C) complete blood count (CBC), platelets; (D) blood gas syringe (lactic acid level).
3. Urine-collection container for myoglobin level. Pigmenturia indicates that renal protection is mandated; unless the centrifuged or settled sample shows supernatant, i.e., the coloration is due to ret cells in the sample.
4. Urine dipstick: hemoglobin.

Forms

1. Laboratory request forms: ABG form × 6; hematology form × 2; chemistry form × 2; coagulation form × 2; urinalysis form × 2; physician order form × 2.
2. Adverse Metabolic Reaction to Anesthesia (AMRA) Report form (obtained from MH Registry).
3. Consult form, if needed, for requesting a consultation from another physician.

PREPARING THE ANESTHESIA MACHINE FOR MH-SUSCEPTIBLE PATIENTS

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- Ensure that vaporizers are disabled by removing, draining, or taping in the “OFF” position.
- Some consultants recommend changing the CO_2 absorbent.
- Flow 10 L/min O_2 through circuits via ventilator for at least 20 minutes.
- Place new, disposable breathing circuit and breathing bag.
- Use expired-gas analyzer to confirm absence of volatile gases.

CONTACT INFORMATION

- MH Hotline: 1-800-644-9737 or 1-315-464-7079 (outside U.S.).
- North American MH Registry of MHAUS registers information about specific families and patients. Registry is located at Children's Hospital at the University of Pittsburgh. See www.mhreg.org or call 1-888-274-7899.
- MHAUS provides educational and technical information to patients and health care providers at www.mhaus.org. They generally do not register cases in other countries, but do field calls from all over the world.

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American Society of Anesthesiologists website. www.asahq.org.

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Consider Methemoglobinemia After Ruling Out the Common Causes for a Low Pulse Oximeter Reading

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Amorbidly obese 39-year-old man presented for open gastric bypass surgery. He had a history of obstructive sleep apnea, requiring continuous positive airway pressure (CPAP) at night. He had no known allergies to medications. The anesthetic plan was to proceed with an awake fiber-optic intubation for general endotracheal anesthesia. He was given a treatment of aerosolized 4% lidocaine for 20 minutes before entering the operating room (OR). In the OR, Cetacaine (benzocaine 14%, tetracaine 2%, and butyl amino benzoate 2%) spray was applied to the posterior oropharynx.

Atranstracheal block was performed using 3 mL of 2% lidocaine. An awake nasal fiber-optic intubation was completed without difficulty. Within 15 minutes of intubation, the patient's pulse oximetry registered a progressive decline to 90% on a FiO_2 of 100%. As the surgeon made the incision he commented that the blood on the field looked "dark like chocolate." When an arterial blood sample was taken, it was also noted to be dark chocolate-colored. Apresumptive diagnosis of methemoglobinemia was entertained. The arterial blood sample was sent for blood gas analysis and methemoglobin (MHb) levels. Because of technical difficulties in the laboratory, there was a delay in processing the blood gas sample. At this point the decision was made to treat empirically with methylene blue; accordingly, 70 mg of methylene blue diluted in 50 mL of normal saline was given intravenously over 10 minutes. After an initial drop in the pulse oximetry reading to 88%, the reading rose progressively to the high 90s. Ten minutes after the entire dose, pulse oximetry read steadily at 99% on 100% oxygen. The patient was extubated without difficulty at the end of the procedure and discharged to home several days later. The result of the MHb level, available on postoperative day 1, was 24.6%.

Anesthesiologists commonly use local anesthetics to anesthetize the airway and pharynx in preparation for awake fiber-optic intubations or endoscopies. Arare though potentially serious toxicity associated with the use of local anesthetics is methemoglobinemia (MHbemia). Anesthesiologists should understand the pathophysiology, presentation, diagnosis, and treatment of MHbemia to prevent and/or manage potential problems.

In the case above, the patient experienced arterial desaturation to 90% on a FiO_2 of 100% oxygen. The differential diagnosis for a low pulse oximetry measurement (SpO_2) included: pulse oximetry artifact, hypoxemia, abnormal hemoglobin variants, sulfhemoglobinemia, and MHbemia. Pulse oximetry relies on measurement of absorbed light from two diodes, one emitting light at 660 nm (red) and another emitting light at 940 nm (near-infrared). It is well known that pulse oximetry is distorted by artifacts arising from motion, ambient light, poor perfusion, and injected dyes including indigo carmine and methylene blue.

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Hypoxemia is the most critical cause for low SpO_2 and must be investigated urgently. Hypoxemia may result from hypoxia, hypoventilation, shunting, ventilation/perfusion mismatching, decreased partial pressure of oxygen in mixed venous blood, and rarely, diffusion abnormalities. Quickly ruling out these causes of a low SpO_2 is paramount to caring for any patient. In this case, the patient was ventilated manually with 100% FiO_2. The tube placement was verified to be correct with a fiber-optic scope; auscultation, and presence of end-tidal CO_2. The nasotracheal endotracheal tube was suctioned and the breathing circuit checked for kinks. New-onset diffusion abnormality was highly unlikely.

METHEMOGLOBINEMIA

Only after ruling out the most common and critical causes for a low SpO_2 should one consider the possibility of MHbemia as the cause. Methemoglobin is the oxidized form of hemoglobin in which the iron moiety of MHb is in a trivalent (Fe^{3+}) ferric state rather than a divalent (Fe^{2+}) ferrous state. MHb is unable to bind oxygen and thus reduces the oxygen-carrying capacity of blood. In addition, MHb shifts the oxygen dissociation curve to the left, thus impairing release of oxygen from the heme molecule to tissue. Significant levels of MHbemia can place patients in a state of functional anemia.

METHEMOGLOBIN FORMATION AND REDUCTION

Normally there is a steady-state equilibrium between the levels of hemoglobin and MHb. The auto-oxidation of hemoglobin to MHb occurs at a rate of 0.5% to 3% per day. Levels of MHb remain below 2% by a mechanism of constant reduction (addition of an electron). The primary mechanism of MHb reduction to ferrous hemoglobin is through the nicotinamide adenine dinucleotide (NADH)-cytochrome b5 reductase pathway. An alternate enzymatic pathway for the reduction of MHb is mediated by NADPH MHb reductase. This pathway generates electrons from glucose-6-phosphate dehydrogenase in the hexose monophosphate shunt, which requires methylene blue and/or flavin to function. Other minor pathways that reduce MHb include ascorbic acid, reduced glutathione, reduced flavin, tetrahydropterin, cysteine, cysteamine, 3-hydroxyanthranilic acid, and 3-hydroxykynurenine.

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CAUSES OF METHEMOGLOBINEMIA

Significant MHbemia occurs when there is an imbalance between hemoglobin oxidation and reduction. The causes of MHbemia may be categorized as congenital or acquired. The rare congenital causes include hemoglobin M and cytochrome b5 deficiency.

Hemoglobin M is a variant of hemoglobin that has an amino acid substitution rendering it resistant to reduction. It is inherited in an autosomal dominant pattern. Patients with this form of hemoglobin usually present with a long-standing history of cyanosis and a positive family history.

NADH-cytochrome b5 reductase deficiency is the other congenital cause of MHbemia. Inheritance of this deficiency occurs following an autosomal recessive pattern. This deficiency is subdivided into types 1 and 2. Type 1 NADH-cytochrome b5 reductase deficiency is limited to the soluble form in erythrocytes. Cyanosis is often the only symptom, and patients with this deficiency are not usually treated. Type 2 deficiency involves both the soluble and

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bound isoforms of the enzyme. As opposed to the type 1 deficiency, patients with type 2 deficiency may have severe progressive neurologic symptoms which are untreatable.

The acquired causes of MHbemia are more common than the congenital causes. Acquired causes mostly involve medication/drug administration. Drugs known to cause MHbemia include antimalarials (chloroquine, primaquine), nitrites or nitrates, nitroprusside, inhaled nitric oxide, sulfonamides, acetanilide, metoclopromide, phenacetin, phenytoin, probenecid, chlorates, phenazopyridine hydrochloride, and local anesthetics including prilocaine, benzocaine, and lidocaine.

SIGNS AND SYMPTOMS

The most common sign of MHb is cyanosis. In addition, patients with MHbenemia have been noted to have chocolate-brownish-colored blood. Our patient's blood was described as being such by the surgeon, and the anesthesia team observed “chocolate”-colored blood on obtaining the arterial blood gas.

DIAGNOSIS

The diagnosis of MHbemia requires co-oximetry. Co-oximeters are spectrophotometers able to emit at least four different wavelengths of light, thus allowing for the measurement of four hemoglobin species: hemoglobin, oxyhemoglobin, carboxyhemoglobin, and MHb. An arterial blood gas sample should be drawn and sent to the laboratory specifically requesting co-oximetry for methemoglobin levels.

TREATMENT

Not everyone with MHbemia requires treatment. MHblevels <30% usually resolve spontaneously over 15 to 20 hours without serious consequences. Those patients with higher MHb levels or patients limited by symptoms of MHb may be treated with methylene blue. The recommended dosage of methylene blue is 1 mg/kg over 5 minutes, and the dose may be repeated in 1 hour if the first dose is not ineffective. Methylene blue accelerates the action of the NADPH-MHb reductase, which is the body's alternate pathway for MHb reduction.

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Methylene blue acts as an electron acceptor of NADPH-dependent MHbemia reduction, which under normal circumstances does not have an electron acceptor. Because treatment with methylene blue depends on NADPH as an electron donator, there must be adequate levels of NADPH available via the glucose-6-phosphate dehydrogenase (G-6-P-D) pathway. Therefore, methylene blue needs an intact pentose phosphate pathway to regenerate NADPH for effectiveness.

For this reason, methylene blue would not be the treatment of choice in patients with G-6-P-D deficiency. In fact, giving these patients methylene blue may increase levels of MHbemia and cause hemolysis, because high concentrations of methylene blue can act as an oxidant. In patients with a G-6-P-D deficiency, treatment is available through the administration of ascorbic acid and riboflavin. Finally, in refractory or life-threatening cases, exchange transfusion and the use of a hyperbaric oxygen chamber may be necessary to treat MHb.

TAKE HOME POINTS

- Anesthesia providers should be aware of the complication of acquired methemoglobinemia resulting from the use of local anesthetics.
- The topicalization of a patient's airway for fiber-optic intubation is an especially vulnerable time for both the development of local anesthetic toxicity and MHbemia. This has been corroborated by multiple case reports in the gastrointestinal literature of MHbemia secondary to Cetacaine spray topicalization of oral mucosa for endoscopic gastroduodenoscopies.
- In addition, there have been case reports in the cardiology literature of MHbemia from Cetacaine spray preparation of the oral mucosa for trans-esophageal echocardiogram.
- The early recognition and treatment of methemoglobinemia in patients can prevent adverse outcomes in these patients and avoid unnecessary interventions that may contribute to morbidity.

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Pulse Oximetry: Even a Simple Device Requires User Understanding

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Pulse oximetry has become an invaluable tool in the current practice of perioperative medicine. It offers dynamic assessment of oxygenation status, which allows for rapid recognition and evaluation of interventions in cases during times of hypoxia on a nearly moment-to-moment basis. Advantages of this monitor include its noninvasiveness, wide availability, and simplicity. However, studies have shown a lack of knowledge concerning the limitations and interpretation of pulse oximetry saturation among both physicians and medical staff.

Pulse oximetry is based on three principles. First, every substance has a unique absorbance spectrum. Different types of hemoglobin have different absorption spectra. The two light-emitting diodes (LEDs) of the pulse oximeter each emit a different, specific wavelength of light within the red to near-infrared range, and a photo detector measures the amount of light transmitted through the tissue. The second principle is based on the Beer-Lambert Law which states that the absorption of light as it passes through a clear nonabsorbing solvent is proportional to the concentration of the solute and the length of the path along which the light travels in that solvent. Finally, the third principle states that the presence of a pulsatile signal generated by arterial blood is relatively independent of the nonpulsatile venous blood in the tissues. By measuring transmitted light several hundred times per second, the pulse oximeter can distinguish the variable, pulsatile component of arterial blood from the unchanging component of the signal made up of the tissue, venous blood, and nonpulsatile arterial blood. The pulsatile or alternating component, which generally comprises 1% to 5% of the total signal, can then be isolated by canceling out the static component.

ACCURACY

Pulse oximetry, or the determination of SpO_2 level, is derived from calibration curves of oxygen saturation that were experimentally measured in healthy volunteers. Values obtained with coincident determination of oxygen saturation by the pulse oximeter were compared with values obtained in *in vitro* laboratory co-oximetry during normal oxygenation and induced hypoxia. Researchers were limited in the degree of hypoxemia inducible in these volunteers to an arterial saturation (SaO_2) of approximately 75% to 80%. Thus, the shape of the curve below these levels must be extrapolated. P.361

For peripheral arterial saturation, most manufacturers report accuracy to within ±2% for SpO_2 of 70% to 100% and ±3% for SpO_2 of 50% to 70%. There is no reported accuracy below 50% saturation. These claims are largely supported by several review studies addressing accuracy by correlating SaO_2 with SpO_2. As an SaO_2 level of 75% represents a PaO_2 level of 40 mmHg, reduced accuracy in this low range does not have any meaningful clinical implications.

There is a delay between when alveolar oxygen delivery changes and when the SpO_2 level shows a change. In general, finger probes are slower (24 to 35 seconds) when compared with earlobe probes (10 to 20 seconds). Changing ventilator management to improve oxygenation requires allowing time for the alveolar oxygen concentration to rise, for the blood with higher oxygen content to reach the site of the pulse oximeter probe, for signal processing to occur, and then for display of the processed signal on the monitor as an SpO_2 value. Sufficient time should be allowed when interventions are made based on pulse oximetry readings.

SIGNAL ARTIFACTS

The major limitations of pulse oximetry can be divided into two major categories: those arising from optical signal artifact and those resulting from optical interference. Most problems with pulse oximetry arise from signal artifact. Signal artifact can result from false sources of signal or from a low signal-to-noise ratio. The presence of a sharp pulsatile waveform on the oximeter does not guarantee no signal artifact.

Sources of false signal include detection of nontransmitted light (ambient light or optical shunt) and nonarterial sources of alternating or pulsatile signal. Because light can be a potentially major source of interference, designers divided the LED and photo detector activities into three sensing periods, which can cycle at hundreds of times per second. Of these three periods, two use light emitted by the LEDs at each of the two incident wavelengths. During the third period, neither LED is activated and the photo detector measures only ambient light. This measurement is then eliminated from the LED-illuminated sensing periods. Despite this, ambient light still interferes, and implicated sources include fluorescent lights, surgical lamps, fiberoptic instruments, and sunlight. A simple solution is covering the probe with an opaque shield.

Optical shunt occurs when light emitted from an LED reaches the photo detector without passing through an arterial bed. This typically occurs when the probe is positioned poorly or when an inappropriate probe is used (for example, a finger probe placed on the ear). Nonarterial sources of alternating signal are typically produced by patient movement and resulting motion artifact. Shivering, movement during cardiopulmonary resuscitation, repetitive cough, and the cycling of ventilators are common potential sources of motion artifact. P.362

OPTICAL INTERFERENCE

Optical interference can limit the value of pulse oximetry. Several substances in the blood can interfere optically with pulse oximetry. These substances absorb light within the red and near-infrared wavelengths used in pulse oximetry. Carboxyhemoglobin (COHb) and methemoglobin (MetHb) are the most significant potential false absorbers. By conventional two-wavelength pulse oximetry, COHb will appear much like O_2Hb in the red range, with virtually no effect in the infrared range. The net effect on SpO_2 is an overestimation of true oxygen saturation. What occurs in the instance of elevated MetHb level is that the MetHb contributes greatly to the perceived absorption of both deoxyHb and oxyHb, driving the ratio of relative absorbances calculated by the pulse oximeter to one. When this ratio is one, the calibrated saturation is approximately 85% leading to an underestimation of true oxygen saturation.

Intravenous dyes can produce falsely low pulse oximetry readings. Changes begin 30 to 45 seconds after dye injection, and recovery to baseline occurs within 3 minutes. Methylene blue causes a profound false decrease in SpO_2. Five mL of methylene blue was reported to decrease oxygen saturation in

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one patient to as little as 1%. Oxygen saturation can decrease less significantly after intravenous administration of indocyanine green and indigo carmine. False desaturations with these dyes are also noted on the multiwavelength cooximeter.

Skin pigmentation has been shown to affect pulse oximetry. Signal detection failures are more common in patients with dark skin. Darker skin appears to make light penetration more difficult. Nail polish may also affect accuracy of readings and, when problems occur, they appear to arise more commonly from blue or black polish.

CLINICAL APPLICATION

In most cases, a normal SPO_2 is a reassuring result, whereas a low SPO_2 is an indication for further investigation. Unfortunately, a low SpO_2 is a very nonspecific finding. Assuming the value is accurate, all aspects of oxygen delivery, including the basic aspects of resuscitation, must be addressed. The clinician must ensure that the airway is patent; airway obstruction, esophageal intubation, or even endobronchial intubation can cause significant hypoxia or anoxia. The clinician must ensure that the patient has adequate ventilation, oxygen concentration, circulation, and tissue perfusion, and, in many cases, can do so by quickly inspecting the patient, the anesthesia machine, and the patient monitors. In other cases, determining the cause of the low SpO_2 value can be difficult. Depending on the clinical circumstances, the clinician may consider, among other things, atelectasis, pneumothorax, low cardiac output, and anemia. The clinician must consider auscultation, and a chest x-ray may be indicated. Occasionally, measuring arterial blood gas levels to determine the partial pressure of carbon dioxide ($PaCO_2$) and checking the acid-base status to validate the partial pressure of oxygen (PaO_2) may be appropriate.

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Pulse oximetry is a measure of arterial oxygen concentration at the site being measured. A normal value obtained at the finger tip does not always permit conclusions to be drawn regarding adequate oxygen delivery to tissues. Adequate organ and tissue perfusion requires both an adequate perfusion pressure and blood flow to achieve sufficient oxygen delivery. Tissues or organs distal to tourniquets or other arterial obstruction can suffer significant ischemic injury.

SaO_2 and PaO_2 have a curvilinear relationship. For SpO_2 values in the mid 90s and lower, SpO_2 is a reasonable predictor of PaO_2. At the highest SpO_2 values (99% or 100%), predicting PaO_2 is not possible. At PaO_2 values from 150 to 600 mmHg, the SpO_2 should be 100%. SpO_2 values can be unreliable in predicting PaO_2 values when PaO_2 is high but dropping or is simply lower than expected. For example, during an episode of apnea, PaO_2 levels decline; however, a corresponding change in SpO_2 level may not show until response time is limited. Other monitors, such as capnography or apnea alarms, generally provide an earlier warning.

Healthy patients receiving 100% inspired oxygen with an endobronchial intubation or lobar atelectasis often have SpO_2 values of 100%. A stable but lower PaO_2 value could be a symptom of an underlying problem that a normal SpO_2 value is masking.

In cases in which the oximeter cannot find a pulse, potential underlying cardiopulmonary problems should be addressed first. If use of a finger probe is causing the problem, changing the probe location may prove successful. Earlobe probes have been shown to produce better signal than do digit probes in some cases. Studies have shown that the earlobe is less vasoactive than the finger pad or nail bed and, so, is less susceptible to vasoconstrictive effects. Forehead probes also exist. Other solutions include using topical nitroglycerin, local heat, or massage. Even digital blocks and intra-arterial vasodilators via ipsilateral radial artery lines have proven successful without much systemic effect, although many would regard them as extreme interventions. Placement of the sensor on the same extremity as a blood pressure cuff can also cause erroneously low readings while the cuff is inflating and should be avoided when possible.

Most currently used pulse oximeters use complex algorithms to distinguish signal artifact (noise) from actual signal and will only accept a certain signal-to-noise ratio. However, sometimes noise is processed and presented as signal. A low signal-to-noise ratio results from absent or low amplitude pulses. Causes include hypotension; hypovolemia; hypothermia; peripheral vascular disease; and iatrogenic causes, such as noninvasive blood pressure cuff inflation and vasoconstrictor administration. A low-pulse state would cause a drop out of signal in older models, whereas newer, more sensitive models can detect low signals but may display a false low saturation.

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TAKE HOME POINTS

- Pulse oximetry is a widely available, noninvasive, and reliable monitor that provides an early warning of hypoxemia that frequently is not detected upon subjective observation.
- Pulse oximetry uses the differential absorbance of light by oxyhemoglobin and deoxyhemoglobin to estimate oxygen saturation.
- Pulse oximetry monitors oxygenation, not ventilation.
- Understanding both the physiologic and technical limitations of pulse oximetry is necessary to use it appropriately. Major aspects to be considered include accuracy, response time, the issue of hypoperfusion or “the low-pulse problem,” and problems caused by motion artifact.

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Noninvasive Blood Pressure Management—It's Not Just a Piece of Nylon Around the Arm

Jorge Pineda JR MD

Stephen T. Robinson MD

The automated, noninvasive, blood-pressure cuff is a safe, commonly used monitoring device. It provides consistent, reliable values for systolic, diastolic, and mean arterial pressure (MAP). One great advantage of such a monitor is that it frees the anesthesiologist for other tasks. Blood-pressure-cuff cycling intervals can be set and the machine can store data to provide trends. Automated devices also provide alarm systems to draw attention to extremes in blood-pressure values. Disadvantages include frequent failure with profoundly hypotensive patients, patients with arrhythmias, and during periods of movement artifact (resulting, for instance, from shivering or tremors).

OSCILLOMETRY

Most noninvasive devices used in current practice measure blood pressure by oscillometry. In this method, variations in cuff pressure resulting from arterial pulsations during cuff deflation are sensed by the monitor and used to determine pulsations corresponding closely to true mean arterial pressure. The cuff is inflated to a pressure above the previous systolic pressure and then it is deflated incrementally. A transducer senses the pressure changes, which are then processed by the microprocessor. This technique has an accuracy of ±2%. The mean arterial pressure corresponds to the maximum oscillation (maximum amplitude = A_{max}) at the lowest cuff pressure (Fig. 88.1).

Systolic and diastolic pressure readings are derived indirectly from formulas that examine the rate of change of the pressure pulsations. It has been determined that systolic and diastolic pressure occur when the amplitudes of oscillation ($A_{systolic}$ and $A_{diastolic}$) are a certain fraction of A_{max}. The systolic pressure corresponds to the onset of rapidly increasing oscillations. Algorithms used by different manufacturers vary and are never publicly disclosed, making it impossible for investigators to verify the accuracy of their underlying physiologic principals. In general, the systolic pressure is chosen as the pressure above the mean pressure at which oscillations are increasing in amplitude and are at 25% to 50% of the maximum ($A_{systolic}/A_{max}$ = 0.25 to 0.5). Diastolic pressure corresponds to the onset of rapidly decreasing oscillations and is more difficult to determine. It is commonly labeled as the point below the mean pressure at which the pulse amplitude has declined to 60% of A_{max} ($A_{diastolic}/A_{max}$ = 0.6), or it is calculated from the systolic and mean arterial pressure, using the following formula: MAP = diastolic + one-third pulse pressure. main problem with this technique is that the amplitude of the oscillations depends on several factors other than blood pressure, such as the stiffness of the arteries. Elderly subjects and patients with "stiff," atherosclerotic arteries have a loss of arterial wall compliance, which is associated with higher systolic and diastolic blood pressure readings using blood-pressure cuffs as compared with values obtained from direct arterial measurements.

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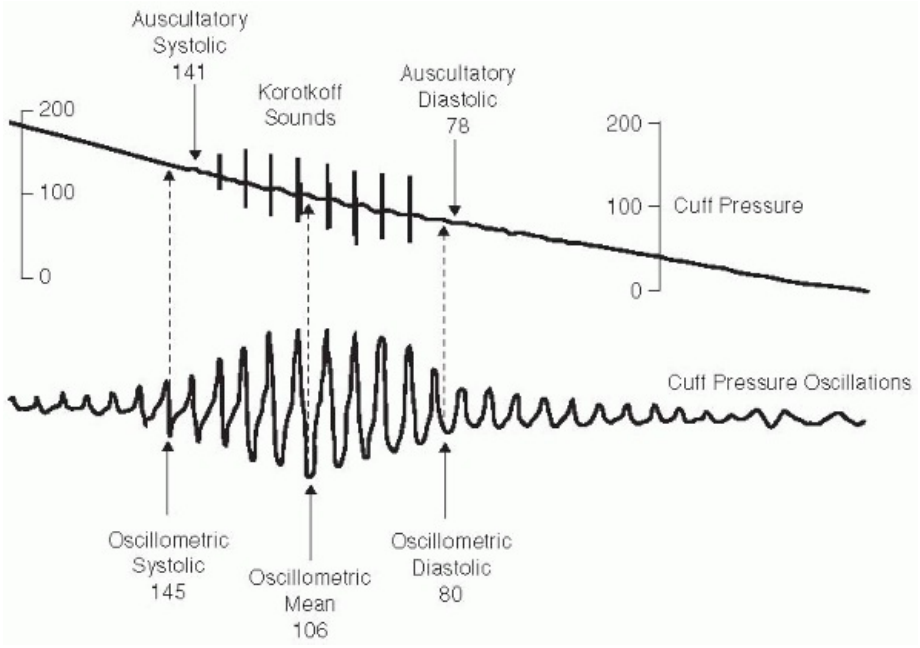


FIGURE 88.1. Blood pressure from Karotkoff sounds (*top*) compared to oscillometry (*bottom*). From Geddes LA. *Cardiovascular Devices and their Applications*. New York: John Wiley; 1984: *Fig. 34.2.*

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Appropriate sizing and positioning of the blood-pressure cuff are important in measuring pressure accurately. Cuff width should be 20% greater than arm diameter, and the cuff should be applied snugly after any residual air has been squeezed out. The pneumatic bladder inside the cuff should span at least half the circumference of the arm and should be centered over the artery. In general, a cuff that is too large works well and produces little error; however, a cuffs that is too narrow yields falsely elevated values for blood pressure because the pressure within the cuff is incompletely transmitted to the underlying artery.

In certain situations, noninvasive blood-pressure cuffs may be used on one extremity but not on another. Noninvasive blood-pressure cuffs should not be used on an extremity with deep-vein thrombosis, ischemic changes, arteriovenous fistula, or arteriovenous graft. In addition, such cuffs should not be applied directly over a peripherally inserted central catheter. If a patient has had a mastectomy or lumpectomy, avoid using noninvasive blood-pressure cuffs on the involved arm or arms to avoid worsening lymphedema. Use this monitor cautiously in patients with depressed consciousness or pre-existing peripheral neuropathies. Arrhythmias may lead to unreliable measurements from oscillometric devices. P.373

In some situations, noninvasive blood-pressure monitoring is inadequate, and placement of an arterial line is indicated. Situations that require beat-to-beat, blood-pressure monitoring; frequent cuff cycling for prolonged periods; titration of vasoactive infusions; or frequent laboratory draws of arterial blood gas are better suited to invasive monitoring with an arterial line. Keep in mind that the peripheral arterial line is not without its limitations. For example, in a patient with substantial vasodilation during the period immediately after a cardiopulmonary bypass procedure, blood-pressure measurements obtained using a noninvasive blood-pressure cuff on the arm may be more accurate than a radial-artery invasive line secondary to the cuff's more central location.

ERRORS OF MEASUREMENT

Atherosclerotic lesions in the subclavian or axillary arteries, causing obstruction of flow, may lead to misleadingly low pressure distally in the affected arm. Thus, elderly patients or patients with known peripheral atherosclerosis should have blood pressure verified in both arms, and the arm with the higher blood pressure should be monitored to avoid this "pseudohypotension." If both arms are diseased, resulting in bilateral falsely low pressures, the femoral artery may be the best option for monitoring. Alternatively, if the risks of invasive monitoring are too substantial, relative to its benefit, the noninvasive pressure may be followed as a trend relative to its baseline.

Besides cuff size and bladder placement, other potential sources of error include arrhythmias causing pulse irregularities; small wrinkles or folds in the cuff expanding and changing the cuff's volume suddenly during data collection; and small movements of the patient, which may create excessive artifact. When the heart rate is irregular, the cardiac output and blood pressure vary greatly from beat to beat. Performance of the oscillatory cuff depends on all measurements made during a cycle. Cuff bladders are set to deflate at a manufacturer-specific "bleed rate," which assumes a regular pulse between bleed steps as part of the algorithms used to determine systolic and diastolic pressure. Therefore, any error caused by irregular beats or by motion of the patient may affect the accuracy of the blood-pressure device. P.374

Data that include significant artifacts should be identified and discarded. The source of the artifact, such as poor positioning of the cuff, tubing occlusion, patient movement, and intermittent compression by the surgeon, should be removed and the affected data should be discarded. The entire cycle should be repeated if measurements are determined to be unreliable. If it is not possible to remove the source of the artifact, consider moving the cuff to an alternative site.

COMPLICATIONS

Complications with use of the noninvasive blood-pressure cuff may occur on rare occasions despite its being noninvasive and relatively safe. These complications include skin irritation and bruising, infection, neuropathy, thrombophlebitis, venous stasis, petechiae, ecchymoses, and even compartment syndrome and skin necrosis. Compartment syndrome is a serious complication and requires prompt surgical intervention. Patients taking anticoagulants are at an increased risk for formation of hematoma and development of compartment syndrome with use of noninvasive blood-pressure cuffs. These events typically occur during prolonged periods of use with excessively frequent cuff cycling, which results in local trauma or impaired distal limb perfusion. Other contributing factors include poor positioning of the cuff across a joint, the continuous pressure of a firmly applied deflated cuff, or repeated attempts to determine blood pressure in the presence of a malfunctioning cuff or an artifact-producing condition, such as involuntary tremors.

TAKE HOME POINTS

- Automated noninvasive blood-pressure devices are reliable and sufficient in most clinical settings.
- The blood-pressure cuff should be properly sized and placed to be effective.
- Problems with accuracy of noninvasive readings may arise in those patients with severe hypotension, arrhythmias, or severe atherosclerosis.
- Use an arterial line when beat-to-beat monitoring of blood pressure, use of vasoactive infusions to control blood pressure, or frequent laboratory draws of arterial blood gas are anticipated.
- Although relatively safe, this device has been associated with development of complications, including skin irritation, bruising, infection, neuropathy, thrombophlebitis, venous stasis, petechiae, ecchymoses, compartment syndrome, and skin necrosis.

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Editors: **Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.**
Title: ***Avoiding Common Anesthesia Errors, 1st Edition***

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Don't Let the Tourniquet Time Run Long

Byron D. Fergerson MD
Randal O. Dull MD, Phd

Asenior anesthesiologist once facetiously remarked to the authors that tourniquet use requires its own little anesthetic plan—discussion of risks and benefits, inflation (equals induction?), maintenance, deflation (equals emergence?), and post-tourniquet (equals postoperative?) pain management. There is a kernel of truth in this.

Tourniquets are used by surgeons during surgery on extremities to decrease overall blood loss and to facilitate operating conditions by keeping the surgical field clear of blood. To deliver effective care to patients, anesthesia providers must carefully maintain a state of vigilance over all aspects of tourniquet use. Surgeons inform patients of plans to use a tourniquet during the procedure, and plans for tourniquet use may be noted in consents as well. However, planned tourniquet use may necessitate a separate discussion between the anesthesia provider and the patient, especially if a regional block is planned to counteract pain resulting from tourniquet use.

Application of the tourniquet is often preceded by exsanguination of the limb by circumferential wrapping of the limb in an Esmarch bandage. An alternative to using an Esmarch bandage is elevating the limb for 5 minutes (at 90°- and 45°-angles for upper and lower extremities, respectively). The cuff should be from 7 to 15 cm greater that the circumference of the limb and should be placed at the site of maximal circumference. The padding placed under the cuff should have no obvious folds. It is recommended that the cuff be inflated to a level 50 mmHg above the level at which distal pulse is lost, as assessed by Doppler, or to a level 50 mmHg above the patient's systolic-pressure level for upper-limb procedures and 100 mmHg above the patient's systolic-pressure level for lower-limb procedures. Most authors agree that from 1.5 to 2 hours is the outer limit for safe use of a tourniquet. This period can be extended by cooling the limb or allowing for 10 minutes of reperfusion for every hour of cuff inflation. Paying vigilant attention to the length of tourniquet use, inflation pressure, and proper cuff fit is important in preventing complications. Communicating with the surgeons also helps; do not be shy about asking questions and expressing concerns, especially if the length of tourniquet use is running long. Establishing open communication before a crisis arises will facilitate resolution of any problem, so discuss plans
and target goals for the length of tourniquet use with the surgeons before beginning the procedure. P.377

Tourniquets are among the oldest medical devices in history. There is a considerable body of literature on the consequences of tourniquet application, and the physiologic effects are protean.

MUSCLE

Predictably, tourniquet use affects muscle tissue. During tourniquet inflation, the combination of acute ischemia and pressure may cause increased microvascular permeability. Cellular hypoxia occurs within minutes of inflation. Ischemic cells release lactic acid, lysozymes, myoglobin, proteolytic enzymes, and inflammatory mediators, including histamine, leukotrienes, platelet-activating factors, and oxygen radicals. All of these substances are redistributed into the systemic circulation immediately after tourniquet deflation and may cause significant vasodilatation and hypotension. In addition, venous stasis distal to tourniquets allows the accumulation of high levels of CO_2 and metabolic byproducts, including potassium, which may cause arrhythmias. After tourniquet deflation, reperfusion hyperemia can lead to compartment syndrome, rhabdomyolysis, and the post-tourniquet syndrome of stiffness, pallor, and weakness without paralysis or numbness.

NERVES

Nerve conduction stops approximately 30 minutes after inflation of the tourniquet, possibly due to axonal hypoxia. Nerve injuries, from simple paresthesia to paralysis, have been reported following tourniquet use. Nerves are most commonly injured at the edges of the tourniquet where the applied pressure is greatest. The radial nerve is the nerve most commonly injured by tourniquets, followed by the ulnar and median nerves. The overall incidence of tourniquet-induced nerve injury in the upper extremity is 1 in 11,000 procedures. In the lower extremity, the sciatic nerve is the most susceptible, with injuries occurring in approximately 1 in 250,000 procedures. The use of Esmarch bandages to exsanguinate the upper limb increases the risk of injury, because it can generate pressures of as much as 1000 mmHg.

Tourniquet use is frequently painful and patients who are awake during tourniquet use often show signs of increasing distress. The patient may deny incisional or operative pain but complain bitterly of an aching or burning pain in the distal extremity. Slow, unmyelinated, C-nerve fibers are thought to be responsible. Under normal circumstances, pain impulses from fast, myelinated Afibers inhibit C-fiber conduction, but these fast fibers are more susceptible to compression and are thus blocked earlier, leaving C-fiber conduction uninhibited.

The anesthesia provider must manage pain from tourniquet use on an ongoing intraoperative basis. Because pain may be experienced as soon as
30 minutes after inflation, the patient may say that “the block is not working.” Sedation is often called for, but, unfortunately, pain from tourniquet use does P.378
not respond to narcotics or sedation in a predictable manner. Most experienced anesthesia providers, however, have and are willing to use an informal algorithm for the intravenous treatment of pain from tourniquet use. Some feel that ketamine, given in sedation doses with an appropriate benzodiazepine, may provide help; others initiate a low-dose propofol infusion.

Using a very dense sensory block with a long-acting anesthetic as the primary technique may provide the best chance of avoiding pain from tourniquet use. For the upper extremity, an adjunctive “ring” or musculocutaneous nerve block may also be done with 10 cc of local anesthetic, often an equalparts mixture of lidocaine and bupivacaine. Remember that, in the limb bearing the tourniquet, epidural and intrathecal sensory levels must be assessed by touch instead of pinprick. Blockade of the nerve fibers associated with touch occurs more slowly and regresses more quickly than blockade of those associated with pinprick sensation. Use of epinephrine, morphine, and clonidine should be considered, because these drugs increase the intensity of the neuraxial sensory block.

Occasionally, asking the surgeons to consider deflation after 120 minutes of tourniquet use or at some other point during surgery is necessary. This request can cause considerable contention in the operating room, as surgeons rarely plan for premature tourniquet deflation. The final option is to begin

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using a general anesthetic at that point.

CARDIOVASCULAR

Significant blood volume shifts can be associated with inflation and deflation of the cuff. Exsanguination of and inflation of the cuff on a lower limb can lead to a 15% increase in circulating blood volume, thus increasing the pulmonary-artery, central-venous, and systemic arterial pressures. These effects can be exaggerated in patients with severe varicose veins or poor left ventricular compliance. Patients under general anesthesia will show an increase in blood pressure and heart rate about 30 minutes after cuff inflation. Deepening the level of anesthesia does not always effectively resolve this problem, and use of vasoactive substances may be necessary. Caution is advised, however, because the hemodynamic effects of inflation are reversed at deflation and reperfusion. Anesthesia practitioners at all levels are usually prepared for an initial decrease in the hemodynamic state; what sometimes surprises less-experienced practitioners is how persistent it can be. Previous administration of a significant dose of hydralazine can compound the problem.

HEMATOLOGIC

The major hematologic concern is the incidence of thrombosis, although study results regarding whether tourniquet use actually increases the incidence of deep-vein thrombosis and subsequent pulmonary embolism are inconclusive. Several studies using transesophageal echocardiography have documented emboli in the right atrium following cuff deflation in up to 100% of patients having total-knee arthroplasty. Emboli tend to form in a large portion of lower-extremity surgeries, regardless of whether a tourniquet is used, but tourniquets are associated with larger, more hemodynamically significant emboli. Sickle-cell disease is a relative contraindication to tourniquet use as there is a theoretical risk that the hypoxic and acidotic environment distal to the cuff may induce a vaso-occlusive crisis. However, several studies suggest that if normothermia, normocarbia, and normoxia are maintained and the limb is properly exsanguinated, tourniquets can be used safely in these patients.

PHARMACOKINETICS

Tourniquet inflation and deflation also alter drug kinetics. Drugs given before inflation may be sequestered in the limb, although this is of uncertain clinical significance. Antibiotics should be given at least 5 minutes before inflation to ensure adequate concentrations at the surgical site. Drugs with large volumes of distribution, such as fentanyl and midazolam, have prolonged durations of action when given after inflation, particularly in the elderly.

TAKE HOME POINTS

- Watch the tourniquet time.
- Tourniquets have predictable physiological effects.
- Tourniquets cause pain in awake patients and elicit sympathetic activation in patients under general anesthesia.

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Editors: Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.
Title: *Avoiding Common Anesthesia Errors, 1st Edition*

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Brain Function Monitors Attempt to Link an Intraoperative Measurement with Postoperative Recall—Much Is Known and More is Unknown as Definitive Practice Parameters and "Best Use" Guidelines for These Monitors

Stephen T. Robinson MD

Catherine Marcucci MD

BACKGROUND

In October, 2005, the American Society of Anesthesiologists (ASA) published a Practice Advisory for Intraoperative Awareness and Brain Function Monitoring (Practice Advisory). A task force composed of anesthesiologists, methodologists, and consultants, some of whom disclosed financial relationships with the brain-monitoring device companies, prepared the Practice Advisory. The primary issue addressed in the Practice Advisory is whether use of clinical techniques, conventional monitoring systems, or brain-function monitors to assess the depth of anesthesia will reduce the occurrence of intraoperative awareness. The Practice Advisory summarizes the most pertinent information on brain-function monitoring and provides an in-depth review of the literature. We recommend that all anesthesia providers review this document in its entirety.

One of the key points of the Practice Advisory is that "intraoperative awareness cannot be measured during the intraoperative phase of general anesthesia, since the recall component of awareness can only be determined postoperatively by obtaining information directly from the patient." Essentially, the term "intraoperative awareness" is commonly used to mean explicit recall. Brain-function monitors attempt only to monitor depth of anesthesia, defined by the ASA as "a continuum of progressive central nervous system depression and decreased responsiveness to stimulation." Calling such monitors, which monitor brain function or brain activity, "awareness monitors" is misleading.

AWARENESS MATTERS

Published studies have indicated that the incidence of intraoperative recall may be as high as 1 to 2 per 1,000 patients receiving general anesthetics. One study concluded that the use of brain-function monitors could reduce this incidence by as much as 80%. P.381

Intraoperative recall is an important event. Some patients who have had unplanned intraoperative awareness have suffered from posttraumatic stress disorder, primarily relating to the circumstance of being aware during surgery, and possibly in pain, without the ability, because of paralysis, to convey their predicament. Unfortunately, the ability to measure the presence and impact of implicit memory is beyond our current technology.

TRADITIONAL SIGNS OF ANESTHETIC DEPTH

Traditionally, anesthesia providers evaluated heart rate, blood pressure, purposeful movement, papillary responses, lid reflex, sweating, and tearing and monitored the patient with an end-tidal analyzer to assess whether anesthesia depth was adequate. At present, no clinical trials or other comparative studies have examined the effect of clinical assessment or conventional monitoring on the incidence of intraoperative awareness; however, some cases have been documented in which patients had intraoperative awareness in the absence of observed increases in heart rate and blood pressure. In a consensus opinion, the Practice Advisory recommends continued use of these physiologic parameters to assess anesthetic depth in all patients.

CURRENT TECHNOLOGY AND APPLICATION

The major devices currently being marketed to assess anesthetic depth use forehead electrodes to record electroencephalographic (EEG) activity with or without electromyographic (EMG) information, or they use auditory evoked potentials (AEP). An analog EEG signal is processed via various public or proprietary algorithms applied to one or more of the following categories of data: frequency, amplitude, latency, and phase-relationship. A unitless number, often referred to as an "index," is generated. It typically is scaled from 0 to 100, and it represents the spectrum of states of consciousness. Zero corresponds with an isoelectric EEG or absent middle-latency AEP, and 100 corresponds with a state of complete wakefulness.

The bispectral (BIS) monitor (made by Aspect Medical Systems, Inc.) is the best known device and is the device most frequently cited in published studies. It uses a BIS-analysis approach to a single channel of frontal-lobe EEG signal processing. A linearly decreasing BIS number is generated from the nonlinear EEG changes as plasma concentrations of anesthetics increase. The manufacturer posits that a BIS monitor number of from 40 to 60 reflects a low probability of consciousness under anesthesia, and the literature supports this to an extent. One randomized controlled clinical trial found lower explicit recall rates when BIS monitors were used than when they were not used. Other studies have shown shorter periods for emergence from anesthesia, first response, and eye opening as well as lower consumption of anesthetic drugs and shorter post anesthesia care unit (PACU) times with use of BIS monitors. P.382

LIMITATIONS AND CAVEATS

Several caveats govern use of BIS and other brain-activity monitors, and anesthesia providers using these devices must be aware of these limitations. First, the index values generated by individual devices are not standardized across the various monitoring technologies. Second, the measured values do not have uniform sensitivity across a cross-section of patients. For example, mean preinduction values for the BIS monitor have been reported to range from 80 to 98. Third, although the monitors provide numeric values that *generally* correlate with depth of anesthesia, equipotent doses of drugs may produce different values, depending on the use of opioids and nitrous oxide. Fourth, the monitors require time to collect and process sufficient information to provide accurate results. If there is a rapid change in the dosage of anesthetics or the level of surgical stimulation, other signs of inadequate anesthesia may precede indication of a change by the brain-activity monitor.

Some case reports describe additional difficulties with brain-activity monitors. Both routine and untoward intraoperative events may interfere with monitor function. These events include administration of muscle relaxants, use of the electrocautery, patient warming or cooling, hemorrhage, and cerebral

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ischemia. Of great interest to the clinical anesthesiologist, several reports have been made of patient awareness despite a generated BIS index value consistent with a depth of anesthesia sufficient to produce minimal risk of recall. These cases serve as an important reminder that, even with a low BIS number, all conversation near the patient should be conducted as if the patient were awake.

WHEN TO USE

Practice standards for brain-function monitors are far from complete. Except for situations in which anesthesia providers cannot place the electrodes, there are no situations in which brain-function monitoring is absolutely indicated or contraindicated. In the Practice Advisory, the ASA task force distinguishes among situations in which such monitoring likely would have differing degrees of clinical usefulness. The circumstances that tended to increase support for monitoring were those in which the patient was likely to have very light general anesthesia as a result of patient condition or surgical procedure.

The ASA task force does *not* agree with the statement that a brain-electrical-activity monitor should be used to assess intraoperative depth of anesthesia for all patients. They agree that an electrical activity monitor may

or should be used to gauge depth of anesthesia for patients with certain conditions requiring smaller doses of anesthetics, but they also state that "there is insufficient evidence to justify a standard, guideline, or absolute requirement that these devices be used to reduce the occurrence of intraoperative awareness in high-risk patients undergoing general anesthesia." Further, the ASA task force is equivocal about basing use of brain-activity monitors on the type of surgery (Cesarean section, cardiac surgery, trauma surgery, etc.).

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In an editorial accompanying the release of the Practice Advisory, ASA president Eugene Sinclair, MD, writes that the entire area of brain-function monitoring likely will require continuing and frequent review, both by the ASA and the individual anesthesia provider. He suggests that, much like pulse-oximetry monitoring, the clinical-practice issues pertaining to the use of the BIS monitor (and other monitors) eventually may not be driven by outcome studies, but by an evolving legal climate. One can hope that good science will ultimately determine the use of these monitors.

TAKE HOME POINTS

- Explicit memory is the only reliable end point that can be used to determine whether intraoperative awareness has occurred.
- Awareness monitors are more accurately characterized as brain-activity or brain-function monitors.
- The primary goal of using such monitors is providing intraoperative values that reflect the likelihood of explicit recall.
- Several devices, of which the best known and most commonly used is the BIS monitor, are used to measure anesthetic depth. The BIS monitor uses a bispectral-analysis (or BIS-analysis) approach to a single channel of frontal-lobe EEG signal processing.
- In October, 2005, the American Society of Anesthesiologists (ASA) published a Practice Advisory for Intraoperative Awareness and Brain Function Monitoring, which contains an overview of the subject of brain-function monitoring and elucidates several clinical situations in which brain-function monitoring may be sufficiently useful to recommend its use.
- Using a brain-function monitor is not a substitute for using traditional techniques to determine depth of anesthesia; nor does it guarantee that the patient will not have explicit or implicit memory. Maintaining proper decorum in the presence of patients is a must.
- Many anesthesia providers use brain-function monitors regularly and feel that such monitors are an important tool in clinical care; however, practice parameters are still evolving.

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Do Not Improvise Techniques to Warm Patients—Use Warming Devices *only* as Per Manufacturers' Recommendations

Jeff T. Mueller MD

NORMAL THERMOREGULATION AND MONITORING STANDARDS

In human beings, thermoregulatory mechanisms maintain a core body temperature of approximately 37°C, with a "normal" range of only 0.2°C. (circadian and menstrual influences also cause up to a 1.0°C variation of the core temperature). The body-temperature control system operates via an elegant feedback loop. Widely distributed, distinct heat and cold receptors send afferent thermal input via nerve fibers to the central nervous system and more specifically to the hypothalamus, which is the primary thermoregulatory control center. Hyperthermia results in efferent hypothalamic output that causes cutaneous vasodilation and sweating. Hypothermia generates hypothalamic outputs leading to vasoconstriction, shivering, and, in infants, nonshivering thermogenesis. *Overall*, the most important mechanism for maintaining normal human body temperature is behavior. The obvious inability of anesthetized patients to add clothing and blankets or adjust the thermostat removes their most important defense against hypothermia.

It has long been recognized that metabolic functions deteriorate when internal temperatures are abnormal and also that both regional and general anesthesia impair thermoregulatory mechanisms. This knowledge is reflected in the American Society of Anesthesiologists' Standards for Basic Anesthetic Monitoring, which state that "during all anesthetics, the patient's oxygenation, ventilation, circulation, and *temperature* should be continually evaluated" and that "every patient receiving anesthesia shall have temperature monitored when clinically significant changes in body temperature are intended, anticipated or suspected."

Core-temperature monitoring sites include the tympanic membrane, pulmonary artery, nasopharynx, and the distal portion of the esophagus. Whether the bladder is a site permitting accurate measurement of core temperature depends upon urine flow. Core temperature may be measured in the bladder if urine flow is high; however, if urine flow is low, the measurement made may reflect peripheral temperature.

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COMPLICATIONS OF HYPOTHERMIA

Evidence suggests that perioperative hypothermia contributes to increases in surgical-wound infections, intraoperative blood loss, transfusion requirements, myocardial ischemia, and arrhythmias. The cardiac risks are highlighted in perioperative guidelines published by the American College of Cardiology and American Heart Association. Hypothermia has also been shown to slow the metabolism of anesthetic drugs and to increase the duration of postanesthetic recovery. The postoperative sensation of cold and shivering is unpleasant for patients and is recalled by some as causing more discomfort than was caused by surgery.

THE PATHOPHYSIOLOGY AND PHYSICS OF HYPOTHERMIA

Perioperative hypothermia is caused when a patient is in a relatively hypothermic environment while simultaneously experiencing a loss of thermoregulatory control.

The difference between the patient temperature and the operating room ambient temperature drives heat transfer from the patient to the surrounding environment. There are four modes of heat transfer: radiation, convection, evaporation, and conduction. All play some role in heat loss in surgical patients; the greatest amount occurs by radiation.

Both general anesthesia and regional anesthesia impair thermoregulation. General anesthesia inhibits thermoregulation in a dose-dependent manner. Cold-response thresholds are significantly reduced, and warm-response thresholds are slightly elevated. Anesthesia increases the above-mentioned threshold range from 0.2°C to 2° to 4°C. The initial phase of anesthesia is dominated by a rapid redistribution of heat from the core to the periphery due to vasodilation. During the next 2 to 4 hours, core temperature decreases, albeit at a slower rate, primarily because heat loss exceeds metabolic heat production. Neuraxial anesthesia disrupts thermoregulatory mechanisms by blocking sensation and neural transmission, causing vasodilation without inducing any compensatory mechanisms. Thus, a combination of anesthesia-induced thermoregulatory dysfunction and an unfavorable thermal environment result in heat loss and hypothermia in surgical patients.

PREVENTING HYPOTHERMIA

All members of the patient's surgical care team are responsible for preventing perioperative hypothermia, beginning with care in the preoperative area and extending into the operating room by maintaining the ambient room temperature at a reasonable level. A normal and comfortable room temperature is required during patient transport into and out of the operating room. For the comfort of the care team, it may be reasonable to lower the ambient temperature once the patient is covered, the surgical site is prepped, and

warming devices have been applied. The operating room ambient temperature is the most important determining factor of heat loss. In addition to ambient temperature management, warming devices can be used to prevent hypothermia. Less than 10% of metabolic heat is lost through the respiratory tract. Therefore, active heating and humidification of ventilator gases has minimal impact on core-temperature maintenance. The temperature of intravenous fluids is also a consideration. Since the temperature of intravenous fluids and blood products cannot significantly exceed body temperature, actively heating patients with fluid warmers is not possible. However, heating of fluids is required to prevent heat loss when large volumes are administered. Preventing cutaneous heat loss is also necessary. Passive insulation, such as using blankets, may be adequate for small operations. More extensive operations require active cutaneous warming, using forced-air or circulating-water devices.

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PREVENTING PATIENT INJURY WHILE PREVENTING HYPOTHERMIA

Warming devices should always be operated according to the manufacturer's instructions. Improper use can result in excessive heat and burns. The use of forced-air blankets should be avoided in areas affected by peripheral vascular disease or other causes of significantly impaired circulation, especially vascular cross clamping. These warming devices should be used only with the appropriate, specified blanket. The heated forced-air output should not be

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used in any other way since improper use has been documented to cause thermal injury to the patient. In addition, operating-room warming cabinets must be operated at a safe temperature. Burns can also occur when heated intravenous fluid bags or blankets are used as positioning aids. Items pressed against the patient may reduce perfusion in the contact area and cause burns at temperatures that would otherwise be safe. All operating rooms should follow a rigorous plan for monitoring and maintaining safe temperatures in warming cabinets. An analysis of the American Society of Anesthesiologists closed claims database revealed that intravenous fluid bags or bottles were the most common cause of claims related to operating-room burns; the second most common cause was warming devices, such as heating pads and blankets. Remember that any warming device can potentially harm anesthetized patients, who cannot sense excessive heat.

TAKE HOME POINTS

- Regional and general anesthesia impair thermoregulation.
- Hypothermia can harm patients by impairing coagulation, increasing infection rates, and contributing to myocardial stress.
- Patients can be kept normothermic when appropriate measures are instituted throughout the perioperative period.
- Misuse of warming devices can cause thermal injury.
- Do *not* use heated bags of crystalloid solution to warm patients!

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Title: *Avoiding Common Anesthesia Errors, 1st Edition*

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Remember that the Effects of Prone Positioning Depend on Frames

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Prone positioning is necessary for surgical access to the posterior spine, the posterior fossa, and the posterior aspect of the lower extremities. Spine procedures commonly using the prone position are discectomy, laminectomy, and spinal fusion. Recent studies have shown that the cardiopulmonary effects of prone positioning depend on which surgical frame is used, rather than on patient body mass index. Knowing the various frames therefore will greatly assist anesthesiologists as they care for patients in the prone position.

Many surgical frames are used for posterior spine procedures, and the differences between them can confuse the anesthesiologist. Keep in mind that the surgical frame selected should help expose the surgical field and decrease blood loss, which commonly results from prone positioning. Spine surgeons choose surgical frames based on whether the procedure requires preserving or decreasing lordosis of the cervical and lumbar spines. Preserving lordosis maintains alignment of the spine for fusion procedures. Decreasing lordosis allows better access to the intervertebral space and is commonly used for discectomy after herniation. Understanding the commonly used surgical frames will help the anesthesiologist understand the cardiopulmonary physiology encountered during procedures done on patients in the prone position.

The two frames that maintain lordosis involve using longitudinal bolsters or the Jackson table. Longitudinal bolsters, consisting of two parallel chest rolls commonly made from foam or rolled sheets, are inexpensive and effective. The bolsters extend from just caudad to the clavicle to just beyond the inguinal area, bilaterally, and are used on a conventional surgical table. The Jackson table is an elaborate, free-standing surgical table with 360-degree rotational capabilities and pads to support the thighs, pelvis, and chest, which allows the abdomen to hang freely. Using the Jackson table has been shown to affect ventilatory mechanics and hemodynamics less than does using longitudinal bolsters.

The frames that decrease lordosis are the Wilson frame, the Relton-Hall frame, and various kneeling frames. The Wilson frame involves using two longitudinal, curved pads to provide continuous support to the chest
and pelvis. This frame is used on a conventional surgical table. With space between the two pads, the abdomen is not fully compressed using this frame. P.390
The curve in the Wilson frame pads can also be decreased to restore lordosis to permit a fusion to be done. The Relton-Hall frame, consisting of two pairs of V-shaped pads, one pair in the upper thoracic region and one pair along the pelvis, leaves the abdomen hanging free. Kneeling frames come in many varieties including the knee-chest, the modified knee-chest (Andrews), the tuck, and the modified tuck frames. Use of these frames optimizes pulmonary physiology, especially in obese patients, but increases the risk for nerve palsies and hypotension, because these frames cause blood to pool in the lower extremities. Use of the kneeling frames is also contraindicated in patients whose vertebral columns are unstable.

Prone positioning alters pulmonary mechanics and hemodynamics no matter which frame is used. Specifically, prone positioning decreases pulmonary compliance. The magnitude of this decrease varies with the frame used. The main cause of such reduced pulmonary compliance is elevated intra-abdominal pressure resulting from the surgical table or frame compressing the abdominal wall and abdominal viscera. This increased pressure is referred to the diaphragm and, in turn, to the lungs, resulting in a decrease in pulmonary compliance. These changes are manifested clinically as high peak airway pressures during positive pressure ventilation. Pressure control ventilation can be used to limit the high airway pressures. Palmon et al. demonstrated that pulmonary mechanics in the prone position depend on which frame is used and not on body habitus. This study concluded that use of the Jackson table disturbed ventilatory mechanics, as measured by peak airway pressure, less than did use of the Wilson frame or longitudinal bolsters. Several studies have assessed the efficacy of different surgical frames for prone positioning. Use of a surgical frame that allows the abdomen to hang freely in the prone position has repeatedly been shown to decrease pulmonary compliance less than does use of other surgical frames.

Hemodynamic changes in patients in the prone position also stem from elevated pressure on the abdominal wall, which exerts increased pressure on the abdominal viscera. The pressure is transmitted throughout the abdomen and can compress blood vessels, specifically the inferior vena cava (IVC). Venous return is forced to follow collateral pathways to the right heart. One such pathway is the epidural plexus, and engorgement of epidural veins can significantly increase blood loss during and otherwise complicate spine procedures. In addition to increasing blood loss, compression of the IVC can also decrease preload, which, in turn, decreases cardiac output and systemic blood pressure. Use of an appropriate surgical frame is intended to decrease abdominal compression and, consequently, to decrease surgical blood loss and systemic hypotension. Dharmavaram et al. studied hemodynamic and
cardiac function in prone patients using transesophageal echocardiogram. The changes in hemodynamics noted in this study with use of various frames P.391
were not statistically significant. Despite the lack of statistical significance, the data reflected that prone positioning affects hemodynamic function. A trend in the data suggested that the Jackson table may produce the least amount of hemodynamic variation with prone positioning. Note that the patients in this study were healthy; hemodynamic changes may become clinically and statistically significant in patients with decreased cardiac reserve.

Prone positioning poses other unique risks to the patient, and the anesthesia provider must consider these risks in the preoperative assessment and discuss them with the patient. Before the procedure, patients should be informed of the possibility of vision loss, neuropathies, and need for blood transfusion due to surgical blood loss. Perioperative vision loss, according to The American Society of Anesthesiologists (ASA) Closed Claims Database, is becoming more frequent in spine surgery. The Post Operative Vision Loss (POVL) Registry was formed to permit study of these relatively rare complications. A recent retrospective case analysis of cases involving POVL demonstrated that 83% of cases of POVL resulted from ischemic optic neuropathy (ION) and were not related to pressure on the globe during prone positioning. The development of ION positively correlates with intraoperative blood loss of more than 1 liter or anesthesia lasting more than 6 hours; the etiology of this correlation is unknown. Central retinal artery occlusion (CRAO), a less frequent cause of POVL, however, is thought to be related to pressure on the globe, is usually unilateral, and typically is accompanied by ipsilateral periorbital erythema, corneal abrasion, ptosis, or decreased supraorbital sensation. Development of POVL can devastate patients, and informing patients in advance of this rare risk is prudent.

Any surgery may result in development of neuropathies; however, brachial plexus injury and lateral femoral cutaneous nerve palsy are specific risks in procedures done on patients in the prone position. Lateral femoral cutaneous nerve palsy is thought to result in as many as 20% of patients who have spine surgery while in the prone position. It is manifested by decreased sensation over the lateral thigh and has been shown to resolve in as many as

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90% of patients. Brachial plexus injury, although rarer, is thought to result from turning the patient's neck and overstretching the nerves of the brachial plexus while the patient is in the prone position. The patient's head must be left in a neutral position, using a foam prone positioning pillow or cradle.

TAKE HOME POINTS

- Increased intra-abdominal pressure decreases pulmonary compliance in patients in the prone position.
- Using a surgical frame that allows the abdomen to hang freely disturbs pulmonary compliance less than does use of other frames.
- Prone positioning can also decrease systemic blood pressure and increase blood loss.
- Use of particular frames in surgery on healthy patients has not been shown to change hemodynamic variables significantly; however, it may do so in patients with decreased cardiac reserve.
- Use of the Jackson table affects ventilatory mechanics and hemodynamics less than does use of other frames.
- Prone positioning also increases the risk for development of peripheral neuropathies and postoperative vision loss (POVL). Discussing these complications with patients before surgery is important.

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Cardiac Output Measurement: Do You Really Understand the Underlying Principles?

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Matthew Caldwell MD

Fegler introduced the thermodilution method for measuring blood flow in 1954. The introduction of the flow-directed pulmonary artery catheter (PAC) in the 1970s permitted clinicians to measure intracardiac pressures directly. Hemodynamic data gathered from PAC use are widely used to diagnose and to help guide medical therapy in critically ill patients in the operating room, cardiac catheterization laboratory, and intensive care unit. Such data can be helpful in differentiating between cardiogenic and noncardiogenic shock and in guiding decision-making about fluid, vasoactive, and inotropic drug therapy over time. For the patient to benefit from PAC use, the clinician must thoroughly understand the interpretation and use of hemodynamic data and must be well aware of the limitations of data obtained from PAC use. Although pulmonary artery catheterization has not been proven to improve outcome, expert opinion continues to support its judicious use after evaluation of the potential risks and benefits for each patient (Pulmonary Artery Catheter Consensus Conference 1997).

HOW PAC TECHNOLOGY WORKS

The PAC is 100 cm long, with proximal and distal ports that enable the measurement of intravascular and intracardiac pressures and the sampling of blood as well as the infusion of vasoactive and inotropic drugs and fluids. At the very tip of the PAC lies the thermistor. The thermistor, when appropriately positioned in the pulmonary artery, continuously measures the temperature of the blood passing by the tip of the PAC in the pulmonary artery. Relatively cold (iced or room-temperature) fluid is injected into the right atrium via the proximal port of the PAC and decreases the temperature of the blood passing distally by the thermistor into the pulmonary circulation. A thermodilution curve is generated by plotting the decline in the pulmonary artery temperature against time in seconds. The area under the curve is integrated and then incorporated into the Stewart-Hamilton equation to yield the cardiac output (CO):

$$CO = [V \times (T_b - T_i) \times K_1 \times K_2]/[\int \Delta T_b(t)dt]$$

where V = volume of injectate, T_b = temperature of blood, T_i = temperature of injectate, K_1 = density factor, K_2 = computation constant, and $\int \Delta T_b(t)dt$ = integral of blood temperature over time. The computation constant is specific to the brand of catheter, and it incorporates specific variations in catheter dead space, approximate injection temperature, injection rate, heat exchange, and unit conversion. The greater the cardiac output the smaller the temperature change over time. Matching the constant with the correct volume (5 or 10 cc) and temperature (iced or room temperature) of injectate to be used is imperative. Dissection of this equation exposes a number of data points that, if inaccurately obtained, will result in a miscalculated CO, which may lead to an inaccurate diagnosis and a misguided therapy. P.394

PLACEMENT AND RISK

Assessing the overall risks and benefits of placing a PAC is important. Placing a PAC not only permits calculation of CO but also permits direct measurement of pulmonary-artery systolic, diastolic, and mean pressures and right heart filling pressures as well as indirect measurement of left ventricular filling pressures. Figure 93.1 depicts the correct path of the PAC through the right heart. An extra port in the PAC permits direct infusion of vasoactive and inotropic drugs into the central venous circulation.

Placing the large bore catheter that allows the introduction of the PAC into the central venous circulation poses risks for infection, pneumothorax, hemothorax, and bleeding at the puncture site or from disconnection of a line. Placing the PAC itself increases the risk for arrhythmia while the PAC floats through the right atrium and ventricle to the pulmonary artery. The most devastating complication associated with PAC use is pulmonary artery rupture. Although pulmonary artery rupture with hemorrhage is infrequent, with an incidence of 0.2%, it is associated with a mortality rate of up to 50%. The right pulmonary artery is involved in 93% of such cases, which usually affect the right lower or middle lobe branches.

ERRORS OF MANAGEMENT

Keep in mind that the thermodilution method measures pulmonary blood flow. Under normal circumstances, pulmonary blood flow equals systemic blood flow. The presence of intracardiac shunts, tricuspid regurgitation (TR), and cardiac arrhythmias, and rapid infusion of intravascular fluid can affect the accuracy of thermodilution CO measurements. The presence of a left-to-right shunt will yield a falsely high CO, because the pulmonary blood flow exceeds the systemic blood flow by an amount equal to the shunt from left to right. The presence of significant TR makes the CO calculations unreliable; it invalidates the thermodilution method, because a portion of the cold indicator continues to stay in the right atrium and right ventricle, therefore not producing a significant change in blood temperature. The
presence of cardiac dysrhythmias alters the beat-to-beat cardiac ejection, thereby yielding an inaccurate CO. P.395

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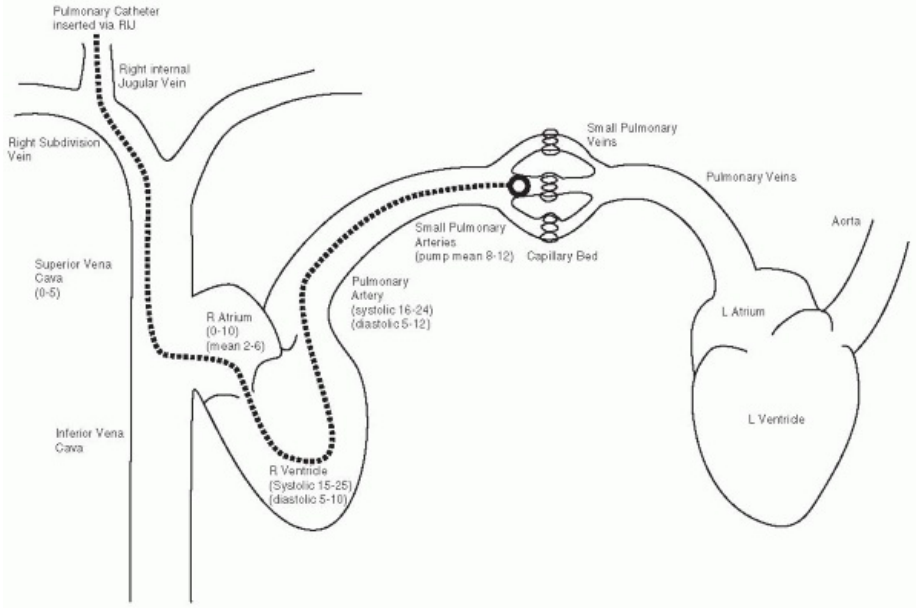


FIGURE 93.1. The path of a PAC through the right heart chambers to the pulmonary artery and the relation of PCWP to left atrium pressure. Normal pressures are given in mmHg. RIJ, right internal jugular vein.

The accuracy of the CO calculation depends highly on operator consistency. Averaging at least three measurements made by the same operator yields the most reliable results, eliminating interoperator inconsistency and improving the precision of the CO calculation. The number of measurements averaged for each determination greatly affects the standard error of the mean (SEM), which is the basis for predicting reproducibility. There is a 2% to 5% variance in SEM when three measurements are averaged to calculate a single CO, and there is a 3% to 9% variance in SEM when only one measurement is used. When comparing CO over time and using three measurements per CO determination, a difference of 15% or more is needed to indicate that the results are actually different. If only one measurement per CO determination is used, then a difference of 26% or more is needed to indicate that the results are actually different. Therefore, at least three measurements should be done by the same operator and the average should be used to calculate CO. P.396

Five or 10 cc of room-temperature or colder injectate should be selected. The volume and temperature must remain consistent. Use of room-temperature and use of colder injectate both have produced accurate and reproducible results. Variations in injectate volume alter results. Excess volume yields falsely low CO calculations, whereas inadequate volume yields falsely high CO calculations. Injectate should be administered steadily over 2 to 4 seconds. Deviation from this rate may alter heat transfer with the surrounding tissues, reducing the accuracy of the computation constant. A faster rate of injection may affect filling volume for the cardiac cycles being measured. Measurements used to calculate CO should be done at end expiration because, at that time, the baseline pulmonary artery blood temperature varies less.

ERRORS OF INTERPRETATION

Whether the risk-to-benefit ratio favors use of the PAC in critically ill patients remains unclear; however, it remains one of the procedures most commonly done on critically ill patients around the world. For more than three decades, the PAC has been used primarily to monitor cardiac function via the thermodilution method, and it also is used to indicate left-ventricular end-diastolic pressure (LVEDP) and left-ventricular end-diastolic volume (LVEDV) status by measuring pulmonary artery occlusion pressure (PAOP). Measuring PAOP and thereby indicating LVEDP in a critically ill patient permits more accurate prediction of the change in cardiac output in response to a fluid challenge. To use PAOP as an accurate surrogate for LVEDP or LVEDV, the PAOP tracing must be valid, accurate, and correctly interpreted; the PAOP must reflect accurately left-ventricular end-diastolic pressure; and, most importantly, the relation between LVEDP and LVEDV must be predictable. P.397

Normally the relation between LVEDP and LVEDV depends on ventricular compliance and, when graphed, has a characteristic curvilinear appearance. The criteria noted in the previous paragraph for use of PAOP as an accurate surrogate for LVEDP or LVEDV are not likely to be met simultaneously in a clinical setting; in addition, the presence of other clinical factors, such as mechanical ventilation or positive end-expiratory pressure, may reduce accuracy. The change in PAOP after a fluid challenge appears to indicate better left-ventricular compliance than it does left-ventricular filling. However, the change in CO may indicate the position of the left ventricle on the Frank-Starling curve. Many studies have shown that PAOP does not indicate LVEDP reliably and predicts recruitable CO poorly.

ALTERNATIVE OR SUPPLEMENTAL TECHNIQUES

Assessing patients using data derived from the PAC may no longer be the gold standard. In critically ill patients, clinician assessment of left-ventricular dysfunction and ventricular preload status showed lower predictive probability than did assessment by transesophageal echocardiography (TEE). Unfortunately, extensive training in and experience with TEE are necessary for its use in assessing ventricular function, volume status, and cardiac output, and such training and experience are not universal among intensive care unit and operating room personnel. Physicians have been shown to acquire skill rapidly in using transthoracic echocardiography (TTE) to assess volume only. Potential use of TTE to estimate LVEDV directly, using CO, is quite promising.

Additional ways to measure cardiac output exist and have limitations of their own. Esophageal Doppler has been used to guide fluid therapy by optimizing stroke volume in cases in which the patient has lost a large amount of blood, and its use has been shown to reduce the length of hospital stay. By integrating the velocity of blood flow through the aorta over time and multiplying it by the cross-sectional area of the aorta, a stroke volume is determined, conferring information on contractility, preload, and vascular resistance. This method has technical limitations. Frequent adjustment of the Doppler probe is necessary for accurate measurements, and calculation of stroke volume depends on the cross-sectional area of the aorta being cylindrical and not changing over time. The Doppler method's advantage is that it is much less invasive than other methods; however; the learning curve associated with its use is steep. Its use is limited to intubated patients.

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Pulse contour analysis assumes that the elasticity and impedance of the aorta remain constant and that the shape of arterial pulsation is proportional to stroke volume. To determine the elasticity and impedance of the aorta,

an independent cardiac output determination by indicator dilution must be made. Some systems for doing so exist, have been validated, and are slowly emerging in the market place. The most reliable systems tend to be the most invasive. For example, one reliable system requires use of a central line and a femoral arterial line for optimal calibration. Thoracic bioimpedance, carbon dioxide rebreathing, and perfusion surrogates are other methods, but are clumsy and clinically impractical. P.398

TABLE 93.1 CERTAIN VARIABLES' EFFECT ON CARDIAC OUTPUT

TECHNICAL ERRORS	MEASUREMENT ERROR CONSEQUENCE	COMMENT
Rate of injection too slow >4 seconds	Inaccurate CO	
Rate of injection too fast <2 seconds	Inaccurate CO	
Falsely high computation constant	Falsely high CO	May occur if entered constant for room-temperature injection when using iced injection
Falsely low computation constant	Falsely low CO	May occur if entered constant for iced injection when using room-temperature injection
Excess volume injected	Falsely low CO	Will cause larger temperature change at catheter tip
Inadequate volume injected	Falsely high CO	Will cause smaller temperature change at catheter tip
L → R shunt	Falsely elevated CO	Pulmonary CO > systemic CO
Tricuspid regurgitation	Falsely elevated CO	
Cardiac dysrhythmias	Less precise result	Greater beat-to-beat variability

Table 93.1 illustrates certain variables' effects on cardiac output accuracy.

TAKE HOME POINTS

- The risks of inserting a PAC should be weighed carefully against the value of the data that will be obtained.
- Operator knowledge and experience influence the reliability of cardiac output measurements.
- Taking at least three measurements and using the average is important because of the variability among measurements.
- Inputting the correct catheter constant is essential to obtaining a valid cardiac output measurement.
- Operator consistency increases the accuracy of the cardiac output measurement. P.399
- Understanding the limitations of PAOP as a surrogate of LVEDV is important.
- Using TEE or TTE along with CO may provide the most complete picture of a patient's cardiovascular status.

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Infusion Pumps: Great Technology When It Works

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Infusion pumps are invaluable devices for delivering drugs and fluids at reliable and controlled rates. They are especially valuable for establishing stable blood levels of short-acting drugs. The three basic infusion delivery methods are gravity, peristalsis or metered delivery, and piston (syringe pump).

GRAVITY

Before infusion devices were widely used, infusion rates were regulated manually, and the rate was estimated by counting drops (gtts) in the intravenous (IV) drip chamber (Fig. 94.1). Adjustments were made by moving the roller ball on the IV tubing. Typical chambers delivered an estimated 10 to 60 gtts/ml. Depending on the chamber design, 10 to 60 gtts/min were required to deliver 60 ml/hr. Although manual regulation is still used for simple IV fluid infusions, it is generally not appropriate for most drug administration unless there is a wide tolerance for error, close user observation and regulation, or an emergency situation.

Relying on gravity poses many challenges. No alarms or other safety features alert the user of problems or prevent errors in flow rate. Since the flow rate depends on gravity and on the patency of the IV line, the flow rate may change as the hydrostatic pressure from the bag decreases as the fluid is consumed. IV bags commonly empty unnoticed, requiring flushing of air from the IV line before continuation of infusion. Adjusting the wrong line can cause delivery of an inadequate amount of drug or cause inadvertent delivery of a bolus of a high-risk medication, such as potassium, with disastrous results. Labeling each line near the regulator and taping the roller ball may help reduce this risk.

Gravity devices are essentially manual systems that use a calibrated constrictor on the IV tubing. Using these devices, when they are working as intended, provides a more reliable infusion rate than does using the roller ball. Using gravity devices also reduces the risk for inadvertent delivery of a bolus of drugs, but poses the same risks as do standard gravity setups for obstruction and reduced delivery. In short, gravity devices are easy to set up and simple to use, but require close user monitoring.

d = desired infusion rate in mcg/kg/min

w = weight in kg

c = concentration in mg/ml

g = drops per milliliter (gtts/ml) for that tubing

k_1 = 1000 mcg/1mg

k_2 = 60 min/hr

$$\text{gtts/min} = \frac{d \cdot w \cdot g}{c \cdot k_1}$$

$$\text{ml/hr} = \frac{d \cdot w \cdot k_2}{c \cdot k_1}$$

FIGURE 94.1. Calculating infusion rates in gtts/min (a) or ml/hr (b) for doses in mcg/kg/min.

EARLY ELECTRONIC INFUSION DEVICES

There were many early infusion devices, all of them crude by today's standards and prone to substantial errors. One early device counted drops and adjusted the flow to match the desired count per minute. It was critical that the operator knew the volume/gtt. Converting the desired dose to the corresponding rate was a nontrivial exercise. If the sensor was partially dislodged, the device was prone to administer a large bolus because of the faulty feedback. Another device used special tubing that was tightly wound around three occluding rollers. Based on the bore of the selected tubing and the revolutions per minute of the device, one could estimate the delivery rate from a table provided by the manufacturer. Different models had different rate profiles. Of course, the wrong tubing or confusion of units (for instance, ml/min vs. ml/hr) posed some threat to the patient. Both of these systems could deliver wide-open flow when disconnected from the device without first manually occluding the IV tubing.

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Early syringe pumps used a screw and piston to advance the plunger into the barrel of the syringe. The rate was determined from a table that calculated the rate in ml/min or ml/hr, based on syringe bore and screw revolutions per minute. All of the complications described below with current devices as well as additional risks existed. These pumps had no high pressure alarm, and the risk of error in calculating the rate was substantial.

When using any early infusion system, infusing drugs in some forms, such as mcg/kg/min, required using a relatively complex formula, and the potential for error was great.

P.402

COMMON PRINCIPLES

Current devices primarily provide peristaltic or similar, metered delivery or are syringe pumps. They can be programmed to deliver fluids or drugs in a wide array of units, such as ml/hr or mcg/kg/min. Many allow the entry of a drug name to provide default settings, including concentration, rate parameters, bolus permission with parameters, and even differing protocols, for that drug.

Infusion devices must be set up and operated properly. Fluid connection must be done appropriately and aseptically. The drug and its concentration must be checked carefully to ensure that they correspond to the values entered on the pump. Carefully inspecting the fluid path, including all connections, can help prevent obstruction of flow and inadvertent infusion of fluid into the atmosphere as well as inadvertent administration of air into the vein. Labeling each set of tubing at both the site of rate regulation and the site of insertion into the common carrier can avoid trouble downstream.

When administering drugs, a carrier should be used. To ensure reliable delivery, the carrier should be placed on a pump. Lines with drugs should be placed close to the distal end of the carrier tubing to limit dead space, to reduce the risk for inadvertent delivery of a bolus, and to reduce changing flow rates' effect on the rate of drug administration.

Newer pumps have safety features, which usually make the device safer. These features may include dosing recommendations, alarms, and automated stopping features. Standardized protocols, especially for drug concentration, are useful to avoid large errors of drug administration. Never assume that a drug has been prepared with the recommended concentration because a protocol exists. Each use requires doing the appropriate checks.

Alarms alert the user that a device has failed and assist in troubleshooting the cause. An obstruction-to-flow alarm is probably the most useful type of alarm. Some alarms, like high-pressure alarms, sound unnecessarily when set too sensitively and thus distract the user. However, setting such alarms too permissively may delay recognition of an obstruction for a prolonged period, especially when a lower flow rate is used. Automated stopping is most typically for volume regulation or air identification. Proper de-airing of the line is critical to avoid pump failure. When clearing air or when decompressing an obstruction, avoid inadvertent delivery of a bolus of drug by ensuring that the tubing is disconnected from the patient or that the drug has an alternative pathway for elimination from the system.

Multiple infusions pose special problems. One must ensure reasonable drug compatibility if using a common carrier line. Spaghetti tangles can cause obstruction, disconnects, and administration errors. Neatness counts.

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PERISTALTIC AND METERED DEVICES

Peristaltic devices are the most commonly used infusion devices in hospitals and are primarily used to administer drugs and fluids from a bottle or bag. They have been manufactured to handle from one to four simultaneous infusions. The portion of tubing that passes through the device must be matched to the device. Although some devices can be modified to accommodate syringes, the higher resistance associated with pulling the plunger into the barrel tends to limit such use. Inadvertent delivery of a bolus of fluid is possible when connecting or disconnecting these devices. To prevent this, the manufacturers routinely provide a device that automatically obstructs flow when removing the tubing from the device. They also, wisely, recommend that a manual regulator on the IV tubing be fully closed when loading or unloading tubing.

SYRINGE PUMPS

Syringe pumps tend to be smaller and lighter than standard peristaltic pumps. They work by loading a syringe into an array of clamps and plungers. A rotating screw functions as a piston to push the syringe plunger at the desired rate. The faster the screw spins, the faster the plunger is advanced into the barrel of the syringe. To match the actual flow rate of the syringe with the programmed rate of flow, the correct brand and size of syringe must be entered into the pump. If the syringe's actual bore is larger than the size programmed into the pump, the rate of fluid administration will be greater than expected. Most devices have sensors attached to the barrel fastener to prevent large errors in bore size from being entered. Such sensors would prevent, for example, a 10 cc syringe from being programmed as a 20 cc syringe. However, differences in bore size among syringes of a given size produced by different manufacturers are too small for these devices to distinguish accurately yet are large enough to create errors in the range of 5% to 10% if there is a mismatch between the actual and programmed brands.

Syringe pumps can fail in several ways to deliver fluids. Obvious causes include programming the device erroneously, triggering safety features and preventing activation; failing to turn on the device; clamping the tubing; and depleting the battery instead of using AC power. Improperly loading the syringe can cause problems. Failing to secure properly the barrel of the syringe can cause the whole syringe to advance rather than advance the plunger into the barrel. Even when a user follows the loading process reasonably well, additional sometimes more minor loading errors may prevent the system from working. Improper priming of the system may cause delays in delivering fluids at very low rates (<10ml/hr), because the entire screw, piston, plunger axis has not been fully engaged. Manually advancing the piston is not priming, because some slack may remain in the screw-piston connection. At higher rates, this is less of a concern. Use the purge function to prime the pump, but do not purge into the patient's IV.

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TAKE HOME POINTS

- Be properly trained on the device before you use it on a patient.
- Generally, peristaltic pumps are more versatile, but syringe pumps can be simpler to use in some locations.
- Carefully load, program, and label each drug and fluid.
- When initiating the administration of a drug, ensure that the device is properly connected and that flow is not obstructed.
- Use a carrier that is also on a pump.
- Use standardized concentrations where possible to avoid errors when changing bags or syringes.

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Scavenging Waste Gases Benefits the Staff But May Harm the Patient

Terrence L. Trentman MD

Why scavenge waste anesthetic gases? Since the early days of anesthesia, anesthesia providers have been both concerned with and challenged by minimizing the risks associated with volatile anesthetics. Ether and cyclopropane were known to cause explosions, and, by the 1960s, serious questions had also arisen with regard to possible mutagenicity, carcinogenicity, and teratogenicity of the volatile anesthetics. Because the volatile anesthetics are likely to remain ubiquitous in clinical practice, the anesthesia provider must be aware of the following issues: the history behind current anesthetic waste gas scavenging practice; the recommended limits for trace gas levels issued by the National Institute for Occupational Safety and Health (NIOSH); the real risks of teratogenicity; the mechanics of scavenging systems, including open versus closed interfaces and active versus passive gas-disposal assemblies; sources of exposure to waste gas; and the risk of obstructions in the scavenging system.

CHRONIC EXPOSURE TO WASTE GASES

Studies done in the 1960s on the risks of chronic exposure to waste anesthetic gases yielded conflicting results. A human study suggested an increased incidence of spontaneous abortion among female anesthesiologists, and an animal study showed that high concentrations of nitrous oxide could cause skeletal deformities in offspring. Subsequent publications supported the idea that waste anesthetic gases put anesthesia providers at risk for adverse health effects. In the 1970s, NIOSH recommended that waste anesthetic gases be scavenged and set recommended acceptable waste gas levels (Table 95.1). However, both prospective and epidemiologic studies conducted in the 1980s and 1990s have shown that, when scavenging is used to reduce amounts of volatile anesthetics to trace amounts, the waste gases pose no risk of adverse health effects. Also, health risks are not associated with short-term clinical (or unscavenged) exposure to potent volatile agents, such as isoflurane or sevoflurane. Experimental nitrous oxide exposure has been associated with animal reproductive abnormalities; however, these conditions do not exist for workers in the clinical environment in which scavenging is present. Of note, NIOSH transmitted its recommendation to the Occupational Safety and Health Administration (OSHA) in 1977. Since that time, OSHA has not taken the necessary steps to promulgate the standards, but it has published technical instructions regarding waste gases. The Joint Commission on Accreditation of Healthcare Organizations has recommended that each anesthesia machine be equipped with a scavenging system and that monitoring be carried out.

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TABLE 95.1 NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH TRACE GAS RECOMMENDATIONS, 1977

ANESTHETIC GAS	MAXIMUM CONCENTRATION (PPM)
Agent alone	
Halogenated	2
Nitrous oxide	25
Combined halogenated and nitrous oxide	
Halogenated agent	0.5
Nitrous oxide	25

Adapted from US Department of Health, Education, and Welfare: Criteria for a Recommended Standard: Occupational Exposure to Waste Anesthetic Gases and Vapors. Washington, DC, US Department of Health, Education, and Welfare, March, 1977.

SCAVENGING SYSTEMS

An anesthesia provider can use several methods to minimize the concentrations of anesthetic gases in the operating room environment. These methods include ensuring a tight mask fit, flushing into the waste gas system rather than the room, turning gas flow off at the end of each case, carefully filling vaporizers using a keyed filler rather than a funnel, and using a cuffed endotracheal tube when possible. In addition, a leak test should be done routinely on both the high- and low-pressure components of the anesthesia machine. *Most important is the presence of a properly functioning waste-gas-scavenging system.*

A waste-gas-scavenging system has five components (Figure 95.1), including the gas-collecting assembly, the transfer means, the scavenging interface (open versus closed), the gas-disposal assembly tubing, and the gas-disposal assembly (active versus passive). Excess gas from the patient is collected via the ventilator relief valve or the adjustable pressure limiting (APL) valve. Collected gas is transferred to the scavenging interface via short, rigid (nonkinking) tubing. If the transfer causes obstruction of the tubing, *patient barotrauma can result as this portion of the scavenging system is*

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proximal to the pressure relief capability of the scavenging interface.

The scavenging interface can be of two types, open or closed. An open system is valveless and is essentially a canister open to the atmosphere. Gas from the transfer tubing is delivered to the bottom of the canister which serves as a reservoir. This arrangement prevents excessive negative- and positive-pressure build-up within the system; however, an active gas-disposal assembly (central vacuum) is necessary to ensure that waste gases stored in the reservoir (canister) do not escape into the room. The reservoir should be large enough to handle a variety of gas flow rates and gas surges. Some open systems rely upon the user to regulate the vacuum by adjusting a vacuum control valve. P.407

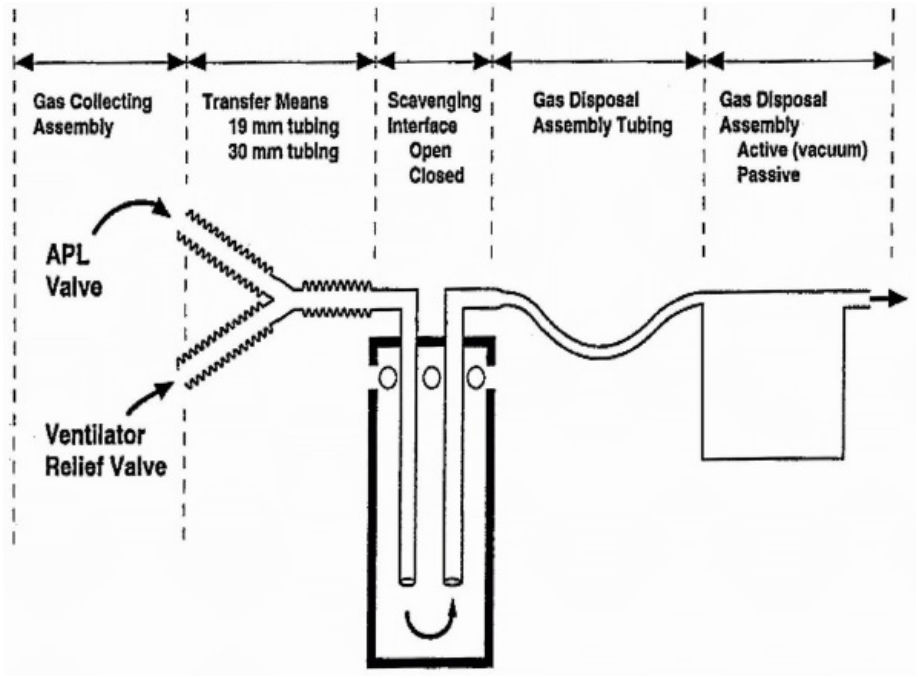


FIGURE 95.1. Components of a scavenging system. (Reproduced with permission from Barash PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia.* 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2006:589.) APL, adjustable pressure limiting valve.

Closed scavenging interfaces use valves to communicate with the atmosphere. A positive-pressure relief valve is necessary to protect the system from excess downstream pressure. The positive-pressure relief valve will open to room air if an obstruction occurs between the scavenging interface and the gas-disposal assembly. If the closed scavenging interface uses a passive gas-disposal assembly, only a positive-pressure relief valve is necessary. In this arrangement, the pressure from the waste gas itself moves the gas toward the disposal assembly, and neither a reservoir bag nor a negative-pressure relief valve is required.

When an active (e.g., central-vacuum) gas-disposal assembly is in place, a negative-pressure relief valve is necessary in the closed scavenging interface. This prevents excessive negative-pressure build-up within the system by entraining room air as needed. A reservoir bag is also necessary to store, briefly, excess gases during the ventilator cycle. The vacuum control valve is adjusted so that the bag is neither under- nor over-inflated. The bag will be seen to expand during expiration and deflate during inspiration. P.408

The gas-disposal tubing transfers waste gases to the disposal assembly. The appearance of such tubing should differ from that of the breathing system hoses and should resist kinking. The tubing is ideally run overhead to minimize the risk of the anesthesia machine or other equipment rolling on top of it. The gas-disposal assembly vents waste gases to a point outdoors that is remote from air intakes and people. The disposal assembly can employ a piped central-vacuum system or an active-duct system, in which fans or blowers move gases outside.

AVOIDING MISHAPS

Anesthesia providers can avoid mishaps by first understanding the mechanics of the scavenging system. Although trace waste anesthetic gases do not pose health risks, the scavenging system should be visually inspected to ensure tight connections and unobstructed tubing. As noted in Figure 95.1, the scavenging system uses 19- or 30-mm tubing that can be distinguished from the 15- and 22-mm tubing of the breathing system. Unfortunately, misconnections are still possible. The use of adapters or tape to make connections should warn the anesthesia provider of a possible misconnection.

One-hundred percent oxygen should be administered at the end of each case to wash excess gases into the disposal system. The vaporizers should be filled using a keyed filler, and the connections should be securely tightened. Use of low gas flows also reduces waste gas, as does use of regional and intravenous anesthetic techniques. However, minimizing waste gas should not supersede clinical considerations in choice of anesthetic technique.

Invariably, all anesthesia machines leak somewhat. Testing machines daily for leakage and servicing machines regularly minimizes leakage. Room ventilation systems should also be inspected and serviced by the institution's facilities department. Scavenged gases should be vented to the outside and not to other rooms in the hospital in which personnel may be exposed or the gases may contribute to fire risk (e.g., machine rooms).

TAKE HOME POINTS

- Short-term clinical (unscavenged) or long-term trace (scavenged) exposure to potent volatile agents does not cause adverse health effects.
- When scavenging systems are in place, nitrous oxide exposure has not been shown to increase reproductive risks.
- The anesthesia provider can use various techniques to minimize exposure to trace anesthetic gases, including inspecting the components of the scavenging system to ensure proper connections and absence of obstruction. P.409
- The scavenging system protects health care workers, but malfunction of the system can lead to patient injury, including barotrauma or

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negative-pressure injury.

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Carbon Dioxide Absorbers Save Gas and Moisture But Create the Potential for Mechanical Hazards, Chemical Soup, or A Thermal Disaster

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Stephen T. Robinson MD

The purpose of anesthesia circuits is to provide an efficient configuration to deliver oxygen and anesthetic gases and vapors to the patient. If only fresh gas is going to be delivered, the required gas flows may need to be as high as two and one half times the patient's minute ventilation (MV) to avoid rebreathing of carbon dioxide (CO_2). A patient with an MV of 6 L/min would require a fresh gas flow of 15 L/min. Using a CO_2 absorber, fresh gas flows of 500 ml/min or lower can be achieved.

BASIC MANAGEMENT OF ABSORBERS

Currently available CO_2 absorbents consist of soda lime (sodium hydroxide lime), barium hydroxide lime, potassium-hydroxide-free lime, calcium hydroxide lime, and non-caustic lime. These absorbents all contain hydroxide bases as their active component. The main reactant in these absorbents (except for in barium hydroxide lime) is calcium hydroxide. Since it is a slow reactant, other constituents are needed to allow the conversion to occur at a sufficient rate in vivo.

Soda Lime Reaction:

$$1)\ CO_2 + H_2O \rightarrow H_2CO_3\ (\text{fast})$$
$$2)\ H_2CO_3 + 2NaOH\ (\text{or } KOH) \rightarrow Na_2CO_3\ (\text{or } K_2CO_3) + 2H_2O + \text{Energy}\ (\text{fast})$$
$$3)\ Na_2CO_3\ (\text{or } K_2CO_3) + Ca(OH)_2 \rightarrow CaCO_3 + 2\,NaOH\ (\text{or } KOH)\ (\text{slow})$$

The CO_2 absorbent is typically stored in two containers that operate in sequence on the expiratory limb of the anesthetic circuit. They lie distal to the ventilator relief valve and the adjusting pressure limiting (APL) valve. Hence, exhaled gas is preferentially routed to the scavenging system before it is cleared of CO_2. The lower the fresh gas flow, the more rapid the consumption of absorbent.

Because the flow of expired gas is unidirectional, most of the reaction occurs in the proximal chamber. As the reactant is consumed, the pH changes and the indicator in the white granules turns blue. Generally, the first chamber shows substantial change in color before the second chamber shows any change in color. The second chamber begins to change in color when the amount of available reactant becomes insufficient to eliminate fully the CO_2 at lower flows. It is then necessary to change the reactant or to use higher fresh gas flows.

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Caustic water and residue can build up at the base of the absorber and along the distal tubing. To avoid inadvertent obstruction to gas flow, draining the fluid periodically and following routine maintenance recommendations, as provided by the anesthesia machine manufacturer, are necessary.

MECHANICAL COMPLICATIONS

Channeling is a condition that occurs when the gas flow through the absorbent is diverted through areas of low resistance, causing a 'channel' to be formed. Gas flowing through a channel does not undergo the reaction clearing CO_2—hence, absorbents that form channels are less effective. More ominously, absorbent that is exhausted or in which channeling has occurred may permit rebreathing of CO_2 through the circuit, with subsequent patient morbidity.

The issue of channeling is related to that of absorbent pellet size. Air flow through a mass of pellets varies inversely with pellet size; that is, larger pellets generate less resistance through the circuit. At the same time, large pellets expose less total surface area to air flow, decreasing the total surface area available to participate in the reaction that removes CO_2. The most commonly available pellet size for CO_2 absorbents is 4 to 8 mesh. “Mesh” indicates the number of openings per linear inch in a sieve used to measure the pellet particles.

Not all CO_2 rebreathing is related to consumption of CO_2 absorbent. Inspiratory and expiratory valves stuck in the open position can cause rebreathing. Kinked anesthesia hoses can also play a role.

Improperly sealing the CO_2 canister can cause a leak. The leak may be caused by failing to close the canister or by a loose granule obstructing the seal. Prepackaged granules, although more expensive, may reduce this risk. Improperly placed soda lime can also obstruct flow. This has been reported to occur with prepackaged granules when the packaging was not removed before insertion into the circuit.

ADVERSE CHEMICAL REACTIONS

The hydroxide bases in CO_2 absorbents are caustic and can cause tissue damage with skin exposure or inhalation. Persons handling these materials must take care to avoid splashing or spilling the new or used chemical. Gloves and eyewear should be worn when the canisters are moved or replaced.

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In addition to removing CO_2 from the gas-and-vapor mixture, these strong bases break down or degrade the potent inhaled anesthetic agents.

The breakdown products differ among the various agents. Sevoflurane, upon degradation, generates a potentially nephrotoxic product called Compound A (2,2-difluoro-1-(trifluoromethyl)vinyl ether). Using fresh gas flows of 2 L/min or greater can prevent this reaction. Lower flow rates are acceptable for moderate periods of time. Isoflurane, desflurane and enflurane, under circumstances in which the CO_2 absorbent is dehydrated, can be broken down into carbon monoxide (CO), with subsequent reported cases of CO poisoning. P.412

Dehydration is an important component of breakdown—the reaction of anesthetics increases as the water content of the CO_2 absorbent decreases. Complete desiccation of absorbent requires exposure to dry gas at room temperature for a number of hours, i.e., a fresh gas flow of 5 L/minute for 24 hours.

In addition to CO or Compound A, the reaction of inhalational anesthetics with desiccated CO_2 absorbent can produce flammable organic compounds. In the case of sevoflurane, these by-products include methanol and formaldehyde. Further, these breakdown reactions are exothermic. The combination of exothermic reaction and flammable breakdown products can accelerate the overall cycle to the point where spontaneous combustion may occur. Use of sevoflurane, in particular, has been reported to cause acute respiratory distress syndrome and spontaneous ignition and fire when combined with barium hydroxide lime absorbent. The combustion reaction may also consume sufficient anesthetic gas as to make maintenance of anesthesia problematic.

The desiccation of absorbent and the subsequent production of CO cannot be detected with routine anesthetic monitors. Traditional absorbents typically turn a blue-violet color when approaching exhaustion—this does not occur with desiccation. Diagnosing CO toxicity can be quite difficult when a general anesthetic has been used; symptoms such as confusion, nausea, shortness of breath, and dizziness might also be attributed to emergence from anesthesia. A pulse oximeter can measure arterial saturation of CO, but its efficacy in the operating room setting has not been well demonstrated.

Some experts have advocated monitoring the temperature of absorbents as a way to avoid potentially lethal outcomes. Such monitoring can be done, for example, by placing a temperature probe in the center of each canister of absorbent and sealing the edge of the canister with foam tape. Yet what temperature cut-off signals an impending event is unclear, as the temperature of the absorbent is elevated during normal use.

Although the capnogram is not a foolproof method of determining absorbent exhaustion, the presence of an elevated inspired CO_2 baseline may indicate absorbent exhaustion and or desiccation.

Of note, not all CO_2 absorbents are equal in terms of their propensity to react with anesthetic agents. Barium hydroxide lime appears to be significantly more reactive than soda lime, which is more reactive than potassium hydroxide-free lime, which is more reactive than calcium hydroxide lime and non-caustic lime. P.413

Absorbent manufacturers have developed products, some moist and some desiccated, that produce an insignificant amount of or no compound A or CO. These products also produce minimal heat and minimally adsorb volatile agents.

The Anesthesia Patient Safety Foundation has issued recommendations for the use of absorbents, including use of CO_2 absorbents "whose composition is such that exposure to volatile anesthetics does not result in significant degradation of the volatile anesthetic." The foundation also recommends implementing policies at the institutional and departmental level "regarding steps to prevent desiccation of the carbon dioxide absorbent should they choose conventional carbon dioxide absorbents that may degrade volatile anesthetics when absorbent desiccation occurs." It further recommends adopting some simple approaches, including turning off gas flow when the machine is not in use, changing the absorbent at regular intervals, changing both canisters, and changing the canisters if they have been exposed to long-term fresh gas flow.

TAKE HOME POINTS

- CO_2 absorbers allow for safe, economical use of anesthetic gases.
- Anesthesia machines must be maintained to avoid leaks and obstruction of flow.
- The breakdown of anesthetic gases, particularly of sevoflurane, can lead to production of toxic byproducts and of CO.
- Desiccated CO_2 absorbent tends to promote adverse reactions.
- Breakdown reactions produce heat, and operating room fires can ensue under the right conditions.
- Use of more reactive formulations, such as barium hydroxide and those using potassium hydroxide, is being replaced by use of less reactive formulations. Note: Baralyme (Allied Health Care/Chemtron) brand of barium hydroxide lime has been removed from the market.

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Do Not Use Your Cell Phone in the Operating Room

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Electronic medical devices have become a mainstay of modern health care, and their complexity and usefulness continue to grow. Similarly, cellular telephones and other wireless communications devices have become commonplace in the United States and around the world, and their use also continues to increase with time. As part of this trend, many hospitals now use cellular telephones (cell phones), two-way pagers, and other wireless communications devices to assist with health-care delivery. Since the advent of cell phone technology, numerous and reproducible reports have documented cell phone use interfering with the operation of medical devices. These reports have prompted hospitals to enact various restrictions on cell phone use within hospital grounds, and they raise the question of whether cell phones can be safely used in proximity to medical devices. This question is of particular importance to anesthesia providers, who work with complex medical devices daily.

MECHANISMS OF INTERFERENCE

Radio waves can induce currents within electrical circuits. Some electrical circuits are designed for this purpose (e.g., an antenna), which is the basis for the use of radio waves as communication signals. Unfortunately, radio waves can sometimes induce unwanted electrical currents within the circuitry of medical devices, thereby causing interference. This phenomenon is known as electromagnetic interference (EMI). Cell phones operate on radio frequencies that are reserved for their use, so even medical devices that are designed to receive data via radio waves are unlikely to have interference by mistaking a cell phone transmission for an appropriate incoming signal. Rather, cell phone radio waves can induce currents in circuits that were not designed to receive radio waves at all. Other wireless communications devices, such as some personal digital assistants (PDAs) and two-way pagers, transmit data in the same way that cell phones do and are theoretically capable of producing EMI.

The risk of cell phone use causing EMI with a medical device depends on the distance between the devices, telephone transmission power, and device construction. Risk decreases with distance, and increases with higher transmission power. A cell phone's transmission power varies inversely with the strength of the signal it receives from its telephone tower. A cell phone receiving a strong signal will decrease its output to conserve battery life; a phone receiving a weak signal will increase its output to ensure more reliable reception. However, power output is always at a maximum when the cell phone is ringing. Characteristics of the medical device, such as outer metal shielding, can decrease vulnerability to EMI.

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EFFECTS OF ELECTROMAGNETIC INTERFERENCE ON MEDICAL DEVICES

The devices most commonly affected by EMI are electrocardiographic (ECG) monitors, which often display signal noise and baseline movement as a result of EMI. Interference with ECG monitors is worrisome because of possible effects on defibrillator function. One European study tested automated external defibrillator response to nearby cell phone use and demonstrated that monitor display and voice commands were often distorted; however, at no time did an automated external defibrillator fail to deliver or incorrectly deliver a defibrillator shock because of EMI. Loss of synchronization with QRS complexes has been demonstrated in a defibrillator as a result of nearby cell phone use.

Mechanical ventilators have also shown vulnerability to EMI. Effects include change of readouts, variation of operation (including changes in rate, tidal volume, and positive end-expiratory pressure), inappropriate sounding of alarms, and shutdown. Death has even occurred because of ventilator shutdown resulting from EMI caused by nearby cell phone use.

Implantable cardiac devices seem relatively immune to EMI effects. Permanent pacemakers have demonstrated overpacing and underpacing but never at a distance greater than 2 cm between the cell phone and the pacemaker pocket. Also, newer pacemakers that include electromagnetic filters have not shown any vulnerability to EMI. Implantable cardioverterdefibrillators also have shown no vulnerability to EMI resulting from nearby cell phone use; however, one report documented an incident in which an antitheft scanner in a bookstore produced EMI that caused an implantable cardioverter-defibrillator to deliver inappropriate shocks.

Many other devices, including dialysis machines, drug infusion pumps, ultrasonographic probes, anesthesia machines, and heart-lung bypass machines, have shown vulnerability to EMI.

RISK OF ELECTROMAGNETIC INTERFERENCE

The probability of EMI with medical devices resulting from nearby cell phone use is difficult to quantify. Many studies have examined EMI but without rigorous standardization of testing protocols or of devices tested. A recent study indicated that 44% of medical devices showed at least some form of EMI from nearby cell phone use, whereas other studies have found that as few as 15% and as many as 60% show such EMI. Sending or receiving a cell phone call may cause EMI. All cellular telephone transmission technologies in use in the United States have been shown to cause EMI. In general, newer medical devices are less vulnerable to EMI. EMI has been shown at distances of as much as 4 m between the phone and the device, but most EMI occurs when this distance is 30 cm or less.

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Although the number of medical devices vulnerable to EMI is substantial, clinically relevant EMI is less common. For example, many of the interference effects noted in studies (such as ECG signal noise and inappropriate sounding of ventilator alarms) are a nuisance but do not ultimately alter patient care. When defined as EMI that hinders data interpretation or alters patient treatment, clinically relevant interference has been shown in 10% or fewer of devices. One review found this proportion to be less than 1% when the distance between the phone and the device is 1 m or more. A recent report of the use of cell phones in the hospital environment reported no clinically important interference in 300 testings performed. Cell phones were used in a “typical manner,” but the authors did not specify proximity of the cell phone to the medical device tested. Finally, this report is from a single institution (Mayo Clinic) with relatively new medical equipment that may be less prone to interference.

REGULATION AND STANDARDIZATION

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At present, the US Food and Drug Administration uses EMI vulnerability standards set by the International Electrotechnical Commission when it evaluates medical devices for approval. Devices compliant with these standards should be vulnerable to EMI only at distances of 50 cm or less. This practice has been in place since 1998 and helps explain why newer devices seem to be more resistant to EMI. However, a device is not legally required to meet these or any other standards to attain approval.

TAKE HOME POINTS

- EMI with medical devices resulting from nearby cell phone use is a documented problem and has harmed patients.
- The exact incidence of EMI resulting from nearby cell phone use is difficult to quantify; however, the risk increases as the distance between the cell phone and the device decreases.
- Likewise, the risk of EMI resulting from nearby cell phone use increases as the phone's power output increases; a phone's power output is always greatest when it is ringing.
- To provide the safest possible care, anesthesia providers should be aware of EMI, especially with older medical devices, resulting from nearby cell phone use and should consider it as a possible cause in cases of equipment malfunction.

SUGGESTED READINGS

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Remember that the Line Isolation Monitor is Based on a Simple Principle of Electrical Safety: Make Sure the Patient Does Not Become Part of A Grounded Circuit

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The issue of electrical safety in the operating room has several factors. Anesthesiologists must manage the risk for patient injury caused by electrical currents several orders of magnitude apart in amperage, and they must use complex equipment-laden electrical setups. In addition, the monitor primarily used by anesthesiologists to indicate electrical issues, the line isolation monitor, rarely sounds an alarm and, in reality, does not guarantee electrical safety. This subject can provoke anxiety in anesthesiologists and merits frequent review.

The presence of any electrical device in the operating room creates the possibility of an unintended completed current pathway or circuit. Basically, the goal is to prevent the patient (or staff member) from becoming part of that circuit. An analogy to household electrical circuits is helpful—the safety features used in operating rooms do not differ in purpose from those used in houses (ground fault circuit interrupters) and appliances (three-pronged cords).

Electrical current involves the flow of electrons down a potential (i.e., *voltage*) gradient, following the path of least *resistance*. Ohm's Law defines the relationship between voltage (V), current (I), and resistance (R):

$V = I R$

For a shock to occur, the flow of electrons must complete a circuit. In most cases of shock, in both household and operating room situations, the circuit is completed by the flow of electrons from a source (usually an energized power line or faulty equipment), through the affected person, who is in contact with the *ground*, and then back to the original power source.

Electrical current harms a person at several current levels and in several ways. In general, electricity alters the electrochemical activity of the body and the organs and cells that it flows through. Exogenous electrical current causes nerve excitation and muscle contraction (exploited for clinical use with the nerve stimulator). Exogenous current also induces cardiac dysrhythmias

when it flows through the heart (exploited for clinical use with defibrillators). As electrical current encounters resistance as it flows through the tissues of P.420
the body, the electrical current loses energy in the form of heat, which can cause both external and internal burns. The amount of tissue damage relates to the amount of current, the duration of the exposure, the current's frequency (i.e., frequencies that exceed roughly 10 kHz do not cause ventricular fibrillation), and the area over which the current is delivered (current density). Which specific injury occurs depends on the amount of current and the resistance in the electrical pathway. Microshock results when a current is applied internally in the body and bypasses the high resistance of the skin. Central venous catheters, pulmonary artery catheters, and pacemakers have all been known to conduct electricity internally and cause microshock. Currents as low as 100 µA have been reported to induce ventricular fibrillation. The second category of electrical injury is called macroshock, in which a large amount of current is applied externally to the skin; the patient usually perceives such shock. The amount of current, applied externally to dry skin, required to produce ventricular fibrillation is about 100 mA. Sensation of electrical current will occur around 1 mA and electrical burns occur at about 6 A.

Modern operating rooms are designed to lessen the probability of completing a circuit through a patient. Keeping the patient ungrounded in a grounded operating room, which contains metal tables, fluid pathways, etc., is not practical; accordingly, keeping the entire operating room electrically isolated or ungrounded is the goal. Using a transformer to create an isolated power system accomplishes this goal. One side of the transformer is grounded and receives a direct source of power. This current flow creates an electromagnetic field that induces current flow in two separate lines (Line 1 and Line 2) that provide current to the operating room. Since Line 1 and Line 2 are not grounded, they theoretically cannot complete a circuit, thus eliminating the risk of shock.

However, in reality, the isolated power system does not completely prevent the risk of shock. If a person were to come in contact with both Line 1 and Line 2, the circuit could be completed and a shock could occur. In addition, the isolated power system could become grounded accidentally if faulty equipment that permits a current leak to ground is connected to the power system. Then, if a person were to come in contact with one of the power lines, a circuit could be completed through the grounding created by the faulty equipment. Capacitive coupling can also result in leakage currents to ground. If enough equipment is plugged into an isolated power supply, the resultant cumulative leakage current can also transform an isolated power supply to a grounded system.

Because a faulty piece of equipment that converts the isolated power supply back to a grounded system may continue to function normally, a line isolation P.421
monitor is used to identify when the isolated power system has become accidentally grounded. The line isolation monitor assesses the resistance from Line 1 to ground and Line 2 to ground. Should the resistance from either line of the isolated power system to ground become sufficiently reduced, an audible and visual alarm activates to alert operating room staff to a fault and the power system's loss of isolation, or connection to ground. Activation of the line isolation monitor's alarm does *not* mean that a shock has occurred; rather, it indicates that the power system in the operating room has become grounded in some way and that a second fault in the system could harm a patient or a staff member. To identify the cause of the ground fault, the last piece of equipment that was plugged in should be unplugged, and then the second to last, and so on, until the alarm turns off. The magnitude of the current change shown on the line isolation monitor indicates whether a piece of equipment is faulty or too many pieces of equipment were plugged in. If a piece of equipment is faulty, the displayed leakage current would show a large change. Alternatively, if too many pieces of equipment were plugged in, the displayed leakage current would show a much smaller change. However, there may be circumstances in which the magnitude of the leakage current cannot be quantified (e.g., if the line isolation monitor does not have a meter). In that case, if the equipment may be moved (i.e., if it is not necessary for life support), it can be taken to a different location and plugged in. In the new location, if the equipment is faulty, the line isolation monitor's alarm should activate; however, if too many pieces of equipment being plugged in activated the alarm, the line isolation monitor's alarm should not activate. If an item is found to have a ground fault, then it should be removed from service and should not be used again until it can be properly inspected and repaired.

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Like the isolated power system, the line isolation monitor does not completely protect the patient or staff from electrical hazards in the operating room. Line isolation monitors cannot detect all faults. For instance, they do not protect against the capacitance between two wires in proximity, which can cause microshock.

To reduce the risk posed by faulty equipment, the casing ("chassis") is connected to ground via the third prong on a standard plug, which is known as the grounding prong. If the equipment's casing connects accidentally to the internal electrical circuitry (fault), a low-resistance pathway is provided along which current may flow to ground, rather than through a person. Hence the adage that the equipment, not the person, should be grounded.

Finally, although sometimes referred to as the "grounding pad," the gelcoated blue pad that nurses often put on the patient's thigh is *not* a grounding pad. P.422
It could more correctly be called the "dispersive electrode." It provides a large area, low-resistance pathway along which applied current may leave the body, and it prevents current from a cautery from leaving the body via pacemaker wires, electrocardiogram electrodes, or contact points between the patient and the metal table.

TAKE HOME POINTS

- Current, voltage, and resistance are related by Ohm's Law:
  V = IR.
- The magnitude of macroshock current required to induce ventricular fibrillation is 100 mA.
- Activation of the line isolation monitor's alarm does *not* mean that a shock has occurred; rather, it means that the power system in the operating room has become grounded in some way and that a second fault in the system could harm a person.
- Upon such activation, unplug pieces of equipment sequentially, starting with the last piece of equipment that was plugged in before activation of the alarm, and, if it has a ground fault, do not use it until it can be repaired.

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Complications of Regional Anesthesia: Don't Touch the Needle Until You Know Them

David A. Burns MD

INTRODUCTION

With the ever-growing number of older persons that need joint replacements and the never-ending orthopedic trauma of the entire population, the practice of regional anesthesia is a growing wave of the future. Regional anesthesia, however, is not without risks or complications. This chapter discusses these risks and and how to manage them.

INFECTION

Whenever the integrity of the integument is violated, the risk of introducing infection is present, and, when a foreign body (like a peripheral nerve catheter) is left in situ, that increases the risk by maintaining a conduit for skin flora to bypass the protective barriers of the skin. Further, the location of the regional technique is important as an epidural abscess or spinal meningitis can cause significant morbidity or mortality. Infection localized to the insertion site would contaminate the needle as it passes through, allowing direct access of the microorganisms to the nerve plexus. Finally, systemic infection or bacteremia can allow infection to be spread hematogenously to the foreign body.

- Factors predisposing toward infection:
  - Immunocompromised state
    - Immunosuppression
    - Steroid use
    - Diabetes
    - Sepsis or concurrent infection
  - Extended duration of use
  - Sterile technique not employed
- Techniques for avoiding infection:
  - Sterile preparation of the site
  - Aseptic technique
  - Sterile occlusive dressings
- Techniques for avoiding or limiting gross contamination:
  - Limit duration or tunnel the catheter (or both).
  - Avoid placement through sites of infection.
  - Administer a dose of antibiotics before placement of invasive lines or nerve-block catheters in patients with sepsis. Studies of placement of such lines and catheters have shown the risk of infection with bacteremia is reduced to baseline if administration of antibiotics has been started before placement. Therefore, you should carefully weigh the risks and benefits of the technique and make sure an appropriate antibiotic has been given at least 1 hour before the procedure. P.427
  - Limit breaks in tubing for bag changes. Every time the integrity of the tubing system is violated by reservoir changes, contamination of the local anesthetic solution is possible-limit such opportunities for contamination.
  - Follow the patient daily and inspect the patient for signs of infection, including erythema, pus, tenderness at the site, and fever.
- Treatments for infection:
  - If infection is suspected, remove the catheter. Usually such removal leads to avoidance or resolution of infection, unless the patient is immunocompromised.
  - Ensure that the patient is receiving appropriate antibiotics.
  - Continue to follow the patient's case to ensure resolution of infection.
  - In the case of suspected epidural abscess, speed of diagnosis and treatment is of the essence to avoid permanent neurologic injury.

BLEEDING

The anatomic locations where many peripheral nerves are blocked contain major blood vessels (for example, axillary, infraclavicular, supraclavicular, femoral, popliteal fossa, and sciatic). Many are deep locations at which applying direct pressure would be difficult or impossible (for example, epidural,

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spinal, classic paravertebral, infraclavicular, supraclavicular, lumbar plexus). Because the epidural space has an extensive venous plexus, bleeding can easily be caused by epidural or spinal anesthesia, inducing an expanding hematoma in a fixed and enclosed space that would cause spinal cord compression and paralysis.

- Techniques for avoiding bleeding:
  - While placing the block, maintain continuous aspiration for blood.
  - If you are proficient in ultrasound techniques, use visualization of the vessels in real-time with the passage of the needle to avoid vascular puncture and to place the local anesthetic more precisely.
  - If anticoagulant drugs are being used before or during surgery (the incidence of such use has grown tremendously), understand the drugs being used, know their duration of effect, and closely monitor the patient during their use. In addition, know which anticoagulant drugs the patient received before surgery and which the patient is receiving
    upon placement of the block and upon removal of the continuous catheter. P.428
  - American Society of Regional Anesthesia (ASRA) guidelines for anticoagulants are followed for all neuraxial anesthetics and for placement of deep peripheral-nerve blocks at our institution (Table 99.1).
  - In the patient with normal coagulation, bleeding complications rarely cause clinical morbidity.

TABLE 99.1 ASRA GUIDELINES IN A NUTSHELL

- Warfarin:
  - Avoid patients taking coumadin if the International Normalized Ratio (INR) is greater than 1.5. Also, the INR should be < 1.5 for removal of an epidural.
- Heparins:
  - Use of subcutaneous, unfractionated heparin at a prophylactic dosage presents no contraindication.
  - For patients receiving heparin infusions, the infusion needs to be stopped 4 hours before the block and the Partial Thromboplastin Time (PTT) drawn showing return to < 1.5 times normal. The catheter may be removed after heparin administration has been discontinued for 2 to 4 hours. Heparin should not be re-administered until 1 hour after placement or after catheter removal.
  - For patients taking subcutaneous heparin or Low Molecular Weight Heparins (LMWH) at therapeutic (bid) dosages for Deep Venous Thrombosis/Pulmonary Embolus (DVT/PE) or Acute Myocardial Infarction (AMI), if the patient has received one dose, wait 12 hours before doing the block. If the patient has received more than one dose, wait 24 hours.
  - For patients taking LMWH at a prophylactic (q day) dosage, wait 12 hours after the last administration of a dose to place and to remove the catheter, and wait 2 hours after removal before administering the next dose.
- Antiplatelet medications:
  - Use of aspirin, nonsteroidal anti-inflammatory drugs, or COX-2 inhibitors presents no contraindication.
  - Patients taking clopidogrel (Plavix) should discontinue use for 7 days.
  - Patients taking ticlopidine (Ticlid) should discontinue use for 14 days.
  - Patients taking GP IIb/IIIa inhibitors (abciximab [ReoPro]) should discontinue use for 48 hours, and patients taking eptifibatide (Integrilin) or tirofiban (Aggrastat) should discontinue use for 8 hours.
- Co-morbid conditions:
  - Thrombocytopenic patients with platelet counts of less than 80K, or 100K if platelets are impaired as in uremia.
  - Hemophiliac patients should not receive a block unless they have had their factor concentrates infused to provide adequate levels for surgery.

Horlocker TT, Wedel DJ, Benzon H, et al. Regional anesthesia in the anticoagulated patient: defining the risks (The Second ASRA Consensus Conference on Neuraxial Anesthesia and Anticoagulation). *Reg Anesth Pain Med.* 2003;28:172-197. This table is my own summary of the recommendations from this reference.

NERVE DAMAGE

The incidence of nerve damage after use of a regional block is reported to be between 0.04% and 5%. The vast majority of these are dysesthesias
that last less than a week. It is more common to damage a nerve by many means that are unrelated to the regional anesthetic but are coincidental in their P.429
timing. Nerve damage may result from surgical trauma, retractor use, excessive pressure from tourniquet use, or excessively long tourniquet use. Poor positioning, leading to tension, stretch, or compression, not only in the operating room but also elsewhere, caused by casts, bandages, bed rails, or failure to protected pressure points, may damage nerves. Although use of a regional anesthetic often is initially blamed for nerve damage, only careful

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investigation will lead to the true cause.

Theoretically, use of a regional block can damage the nerve in the following ways: the needle may cut the nerve; an intraneural injection may mechanically disrupt the nerve; an intraneural injection with high pressure may cause ischemia; additives like epinephrine may cause intense vasoconstriction; and, finally, neurotoxicity of the solutions may damage the nerve.

Techniques for avoiding nerve damage:

- Avoid using deep sedation and general anesthesia during placement. If the patient cannot tell you reliably whether he has pain upon injection or has a paresthesia, you have lost that safety monitor.
  - Stop if the patient has pain upon injection. Intraneural injection may cause pain in the distribution of that nerve.
    - By the same logic, consider avoiding “rescue” or supplemental blocks because the patient's ability to feel pain of paresthesia is diminished.
  - Avoid paresthesias. The majority of cases of nerve damage also coincided with a paresthesia upon placement.
- Avoid using currents of less than 0.2 mA. Because current density is proportional to the distance squared, avoiding needle placement that results in a motor response with a current of less than 0.2 mAis widely believed to prevent intraneural injection. However, this belief is currently being widely challenged as ultrasound techniques have shown intraneural needle placement without any motor response.
- Use short-beveled needles. Such use is widely thought to reduce the likelihood of cutting the nerve.
- Use low pressure to inject. If there is resistance to injection, the needle may be placed intraneurally. In fact, a group of anesthesia providers located in New York uses a pressure manometer in doing injections.
- Remember that the neurotoxicity of all local anesthetics increases with increasing concentration. Such increases are nominal at clinically used concentrations unless injected intraneurally.
- Use ultrasound guidance. Ultrasonographic visualization of the nerves and the needle allows you to avoid poor positioning.

ALLERGIC REACTIONS

Allergic reactions to regional anesthetics are extremely rare and are often misdiagnosed as an iatrogenic reaction or an immunologic response to a different antigen, like the preservatives methylparaben or metabisulfite. The ester local anesthetics are hydrolyzed to PABA(para-aminobenzoic acid), which can be allergenic.

BLOCKADE OF OTHER NERVES

Examples of such blockades include blockade of the ipsilateral phrenic nerve with nearly every interscalene block as well as blockade of the recurrent laryngeal nerve. It sounds like the recurrent laryngeal nerve is also blocked every time so I clarified. Such blockades may cause significant reduction in respiratory function and hoarseness and therefore may be contraindicated in patients with poor respiratory reserve or contralateral, recurrent laryngealnerve palsy. Other examples include epidural spread from paraneuraxial techniques like the interscalene, paravertebral, or lumbar plexus blocks. In addition, the femoral nerve can become blocked with a field block for an inguinal hernia.

INTRAVASCULAR INJECTION AND LOCAL ANESTHETIC TOXICITY

This complication and its treatment are addressed in Chapter 110.

SUPPLIES AND EQUIPMENT THAT SHOULD BE AVAILABLE WHEN DOING REGIONAL ANESTHESIA

- Airway equipment:
  - Laryngoscope blades, endotracheal tubes, a suction device, a self-inflating bag, a mask, and a source of oxygen.
- Emergency drugs:
  - Epinephrine, atropine, propofol, succinylcholine, benzodiazepines.
- Cardiopulmonary bypass capabilities and 100 ml of 20% Intralipid, especially if using bupivacaine.

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Know the Facts and Be Ready with an Answer When A Patient Who Will Receive Spinal Anesthesia Asks "Can This Paralyze Me?"

Angela M. Pennell MD

Despite ongoing efforts to educate patients, reluctance to accept spinal anesthesia is still fairly common. Patients in all demographic groups ask about the toxicity of spinal anesthetic agents, the possibility of paralysis, or both, even when they have already consented to neuraxial blockade. Patients also commonly refer to troubles experienced by a family member or acquaintance after spinal anesthesia. "My buddy had the needle in his back and it messed him up good" has been heard by just about every practicing anesthesiologist. Periodically reviewing both the basic knowledge base and the recent literature on the neurotoxicity of spinal agents and other general risks is helpful as new reports on the incidence of the adverse effects of spinal and epidural anesthesia continue to be published.

CAUDA EQUINA SYNDROME

The incidence of cauda equine syndrome is 32 in 1.2 million subarachnoid blocks and 32 in 450,000 epidurals. The syndrome is a combination of bowel and bladder dysfunction with paresis of the lower extremities and sensory dysfunction in a patchy distribution. It is often associated with hyperbaric lidocaine (lidocaine combined with dextrose) or highly concentrated lidocaine (5%) and suggests neurotoxicity caused by the agent itself. However, this syndrome has also been observed with other local anesthetics, such as 0.5% hyperbaric bupivacaine. Therefore, the etiology *may* be related to insufficient mixing of local anesthetic with cerebrospinal fluid, with resulting neurotoxicity. Cauda equina syndrome has also been linked to the use of microcatheters for repeated or continuous dosing where concentrated, local anesthetic pools around the sacral nerve roots (microcatheters are now prohibited by the FDA).

TRANSIENT NEUROLOGIC SYMPTOMS (TNS)

Transient neurologic symptoms consist of back pain radiating to the lower extremities, without sensory or motor deficits, occurring after resolution of the spinal block and subsiding after several days. These symptoms are

most commonly seen with lidocaine use and in outpatients having surgery in the lithotomy position and are thought to be secondary to neurotoxicity (probably not related to concentrations of <5%). Whether the true etiology is neurotoxicity or soreness from muscle strain is unclear. P.432

OTHER NEUROLOGIC DEFICITS

The incidence of other neurologic deficits is 20 in 1.2 million subarachnoid blocks and 20 in 450,000 epidurals. Permanent or transient neurologic deficits probably represent direct damage to the nerve roots. These deficits are commonly associated with multiple attempts at accessing the intrathecal space. Paraplegia can result if there has been direct contact between spinal cord and needle. Frequently observed are loss of bowel and bladder function; paralysis of the biceps femoris muscles; and sensory loss in the dorsal thigh, saddle region, or great toes as often seen with damage to the conus medullaris. Deficits may also be due to neurotoxicity of the local anesthetic agent.

MENINGITIS AND ARACHNOIDITIS

The incidence of meningitis or arachnoiditis is 29 in 1.2 million subarachnoid blocks and 29 in 450,000 epidurals. Meningitis and arachnoiditis may be infectious or noninfectious; present as pain coupled with other neurologic symptoms; and are identified using radiography, which shows clumped nerve roots. Meningitis and arachnoiditis have been previously attributed to detergents used in procaine or solutions used to clean reusable spinal needles.

SPINAL HEMATOMA

The incidence of spinal hematoma is 33 in 1.2 million subarachnoid blocks and 32 in 450,000 epidurals. It is secondary to trauma to an epidural vein caused by a needle or catheter and is most often seen in patients with abnormal coagulation or bleeding disorders. It is also associated with technically difficult or bloody blocks. In addition, a spinal hematoma may occur immediately after removal of an epidural catheter. The hematoma behaves as a mass directing pressure to the neural tissue of the spinal cord. Symptoms initiate as sharp back and leg pain that progress to numbness and motor dysfunction (including loss of bowel and bladder control).

BUPIVACAINE

Bupivacaine is an amide local anesthetic commonly used for spinal anesthesia. Having a higher pKa of 8.1, it has a slower onset but a longer duration of action than some other amide anesthetics. Concentrations range from 0.25% to 0.75%. Hyperbaric formulations in 8.25% dextrose are available. It affects sensory-nerve fibers more readily than motor-nerve fibers. The central nervous system toxicity of bupivacaine is relatively low compared with its cardiac toxicity. However, cauda equina syndrome has been associated

with intrathecal bupivacaine use (although not as frequently as with lidocaine use). The toxic intravenous dose of bupivacaine is considered to be 1.6 mg/kg. At this dosage, cardiac complications, including dysrhythmias, hypotension, and depression of cardiac output, have been observed. The maximum total dose of bupivacaine is 3 mg/kg. P.433

LIDOCAINE

Lidocaine is an amide local anesthetic. Concentrations range from 0.5% to 5%. Compared with other amide anesthetics, lidocaine has a more rapid onset and a shorter duration of action, due to its lower pKa (7.7), and lower lipid solubility. Its maximum dosage is 4.5 mg/kg without epinephrine and 7 mg/kg with epinephrine. Cauda equina syndrome and TNS are consistently linked to intrathecal lidocaine (especially 5% lidocaine and hyperbaric

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lidocaine). Therefore, lidocaine concentration should not exceed 2.5%, a lidocaine dose should not exceed 60 mg, and combining lidocaine use with epinephrine use should be avoided.

ROPIVACAINE

Ropivacaine is an ester local anesthetic with a pKa of 8.1, a long duration of action, and high lipid solubility. Ropivacaine has been used less than some other local anesthetics in spinal anesthesia, and it does not seem to have an advantage over bupivacaine for intrathecal use. The maximum recommended dose is 3 mg/kg.

TETRACAINE

Tetracaine is an ester local anesthetic with a pKa of 8.2, a long duration of action, and high lipid solubility. Tetracaine use provides a higher degree of motor blockade than does bupivicaine use. Combined use of epinephrine and tetracaine may prolong tetracaine's duration of action by 50%. Tetracaine use, at a concentration of 0.5%, with repeated doses or via small-bore catheters (or both), has been associated with cauda equina syndrome. Of note, tetracaine is the only local anesthetic that does not contain epinephrine or glucose but also does not carry the “Not for Spinal Anesthesia” warning on its label. It requires reconstitution of its crystals with sterile, normal saline to result in an isobaric solution. The maximum recommended total dose is 3 mg/kg.

CHLOROPROCAINE

Chloroprocaine is an ester local anesthetic with a pKa of 9.0, higher than those of other local anesthetics. Chloroprocaine use is associated with prolonged neurologic deficit attributed to neurotoxicity. This neurotoxicity is presumed to result from chloroprocaine's low pH when it is combined with preservative. The former preparation used sodium bisulfate as a preservative; the current preparation uses EDTA as a preservative. A preservative-free formulation

also is available. Another etiology of neurotoxicity may be the use of large volumes (>40 ml) of chloroprocaine. The maximum recommended dose is 12 mg/kg.

TAKE HOME POINTS

- Know the complications that can result from spinal anesthesia.
- Know the incidence of each complication:
  - Cauda equina syndrome: 32 in 1.2 million intrathecal injections or 450,000 epidurals;
  - Transient neurologic symptoms: the incidence is unclear and these symptoms resolve by themselves;
  - Other neurologic injury: 20 in 1.2 million intrathecal injections or 450,000 epidurals;
  - Meningitis: 29 in 1.2 million intrathecal injections or 450,000 epidurals; and
  - Hematoma: 33 in 1.2 million intrathecal injections or 450,000 epidurals.

Know the maximum allowable dose of each local anesthetic.

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Remember the Low-Risk and High-Yield Blocks

Jennifer Vookles MD, MA

You are in the midst of a busy day in the orthopedic operating rooms. You dash through the postanesthesia care unit (PACU) and happen to notice a patient reading the paper. The next day, you notice another patient in the PACU reading the paper. The PACU nurse tells you that Dr. Doe's patients are so comfortable, they usually ask to read or watch TV while waiting for their floor beds. The following day, when you take your total-knee arthroplasty patient to the PACU, the nurse asks if you did a femoral-nerve block. When you reply that there wasn't time, she frowns a little and says, “Well, I guess we won't be needing any newspapers today.”

Peripheral nerve blocks provide a variety of perioperative benefits. The most obvious benefit is analgesia extending for many hours postoperatively, both from direct action of the local anesthetic but also potentially from pre-emptive analgesic mechanisms if the block is established prior to incision. In addition, they can help reduce the need for intraoperative doses of volatile agents and opioids; this may reduce recovery times by decreasing postoperative sedation and nausea. Finally, peripheral-nerve blocks can also reduce the need for muscle relaxants by blocking motor as well as sensory fibers.

Despite these benefits, peripheral-nerve blocks are often omitted when a general anesthetic or spinal is planned, because they can be time consuming to perform and carry their own procedural risks. However, even recognizing these limitations, there are a few “low-risk and high-yield” blocks that are relatively quick to perform and should always be considered as an adjunct to primary anesthetic. These blocks include the superficial-cervical-plexus block, femoral-nerve block, and obturator-nerve block.

SUPERFICIAL-CERVICAL-PLEXUS BLOCK

The cervical plexus has both superficial and deep components. The superficial plexus contains cutaneous branches from the ventral rami of C2-4 providing innervation from the posterior cranium to the shoulder via the lesser occipital, greater auricular, transverse cervical, and supraclavicular nerves. The superficial plexus can be very easily blocked and utilized as supplementary analgesia for carotid endarterectomy (CEA) and other neck surgeries. As the sole anesthetic, one study even found equivalent benefits of the superficial-cervical-plexus block compared to combined superficial- and deep-cervical-plexus blocks for CEA. It can also be used to ensure cutaneous coverage of the shoulder following interscalene block.

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Infiltration of local anesthetic deep to the posterior border of the sternocleidomastoid muscle will block the superficial cervical plexus. At the midpoint of the muscle's posterior border, a 22G 4-cm needle is inserted just below to the muscle and 5 mL of local anesthetic is injected. The needle is then redirected cephalad and caudad along the muscle border, with a total of 10 mL injected along these paths. The external jugular vein often overlies this area and should be avoided. When this block is properly performed, deeper structures should not be affected; however, one should be aware of the proximity of the phrenic nerve, the internal jugular vein, and the carotid artery if the needle is inserted too deeply.

FEMORAL NERVE BLOCK

The femoral nerve is formed from branches of L2-L4; it crosses the pelvis in a groove between the psoas and iliacus muscles to emerge beneath the inguinal ligament lateral to the femoral artery. The femoral nerve can begin to divide into its branches at or above the inguinal ligament. This nerve provides both deep and superficial innervation of the anterior thigh extending to the knee with a distal branch, the saphenous nerve, continuing along the anteromedial shin to the ankle and occasionally into the dorsum of the foot.

Although a femoral-nerve block must usually be combined with a sciatic nerve block or a lateral-femoral-cutaneous-nerve block (or both) to provide adequate anesthesia for surgery on the leg and foot, a femoral-nerve block on its own can provide significant postoperative analgesia for surgery involving the anterior thigh, femur, and knee. The patient may still have some discomfort in the posterior knee, but this is usually easily managed.

Landmarks for the femoral-nerve block are the pubic tubercle and anterior superior iliac spine. A line is drawn connecting these points, and the femoral artery is palpated along this line. The needle insertion site is on this line immediately lateral to the femoral artery. Using a nerve-stimulation technique, the injection site can be determined by observing quadriceps twitch; do not be misled by stimulation of the sartorius muscle.

A femoral-nerve block can often be completed as a field block. Beginning at the same entry site, the needle is initially inserted perpendicular to the artery and then redirected laterally in a progressive, fan-like manner. Approximately 20 mL of local anesthetic should be injected incrementally in this process. It can also be helpful to displace the needle site 1 cm lateral and, aiming medially, place 2 to 5 mL of local anesthetic posterior to the femoral artery. If available, ultrasound can be useful in better localizing the artery, in experienced hands, the nerves themselves can also be visualized.

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OBTURATOR NERVE BLOCK

The obturator nerve is formed by the ventral branches of the L2-L4 anterior primary rami and emerges from the medial border of the psoas at the pelvic brim. It follows the lateral wall of the pelvis to exit via the obturator foramen, emerging into the anteromedial thigh. The anterior branch supplies the hip joint and anterior adductor muscles and sends sensory branches to the skin over the medial thigh. The posterior branch supplies the deep adductor muscles and a sensory branch, which extend to the posterior knee joint.

The obturator-nerve block can be used in combination with the sciatic, femoral, and lateral-femoral-cutaneous blocks to allow complete surgical anesthesia for the lower extremity. It can also be useful in treating hip-joint pain and in relieving adductor-muscle spasticity. The obturator-nerve block can also be used to suppress a reflex-adductor contraction not uncommonly seen during transurethral resection of the lateral bladder wall lesion; this reflex is often not abolished by spinal anesthesia.

There are several approaches to performing this block. For the “classic” approach, the patient is supine with legs slightly adducted. The entry site is 1.5 cm both caudal and lateral to the pubic tubercle. A needle is inserted perpendicular to the skin and advanced 2 to 4 cm until the inferior edge of the superior pubic ramus is contacted. The needle is then withdrawn slightly and redirected laterally about 45% to a depth 2 to 3 cm deeper. Fifteen mL of

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local anesthetic should be injected while advancing and withdrawing slightly. If a nerve stimulator is utilized, thigh adductor contractions should be observed.

The newer "inguinal" approach is simpler and less uncomfortable for the patient; but it usually does not block the articular branches to the hip. A nerve stimulator must be used for this approach. With the hip abducted, the insertion site is found by marking the midpoint between the pubic tubercle and pulsation of the femoral artery, as it emerges beneath the inguinal ligament. The needle is directed roughly 30% cephalad. Once stimulation of the gracilis and adductor longus muscles are obtained, the needle is directed slightly lateral and advanced another 0.5 to 1.5 cm. After the adductor magnus is stimulated, 5 to 7 mL of local anesthetic is injected.

TAKE HOME POINTS

- These three blocks can be performed relatively quickly and have relatively low risk associated with them.
- As with any peripheral-nerve blocks, care should be taken to avoid local-anesthetic toxicity. Always inject slowly and incrementally, attempting to aspirate after every 3 to 5 mL to detect intravascular needle placement.
- Consider adding low-dose epinephrine to the local anesthetic to further aid detection of intravascular injection and to slow absorption.
- A more precise description of the techniques for performing these blocks can be found in many regional anesthesia texts or in online resources.

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Consider the Paramedian Approach for Spinal Anesthetic Placement if the Patient is in the Lateral Position

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Aspinal anesthetic may be placed on a patient in the lateral position for several reasons: The patient may require significant sedation; be at risk for a vasovagal reaction; have pain in the sitting position due to trauma or disease process, such as a fractured hip or perirectal abscess; or may be difficult to turn quickly after placement of the spinal due to morbid obesity. The lateral position is also very typically used if a hyperbaric or hypobaric one-sided spinal is planned. However, the advantages of having a patient in the lateral position are often offset by increased difficulty in correctly placing the spinal needle, especially if a midline approach is attempted. The issues to be aware of are the loss of visual anatomic landmarks, particularly the longitudinal midline bony landmarks, and the relative inability of patients to curl into the correct position to open the interspinous spaces.

Gravity can be blamed for increasing the chances of unintended deviation from the interspinous spaces in patients who are in the lateral position. Typically, less experienced anesthesia providers correctly palpate and appreciate the bony structures and spaces of the spinal column during the preparatory and draping phases but then use the visual cue of the longitudinal “dimple” in the soft tissue and skin in placing the introducer and spinal needles. Particularly in older or overweight patients, there can be as much as 1 to 2 cm of “sag” of the soft tissues towards the dependent side and it is extremely easy to get off the midline. A resident who is consistently contacting bone during a midline approach is usually tapping the dependent lamina. On repeated palpation of the bony structures and redirection of the needle, the true midline of these patients is almost always found to be “more towards the ceiling.”

Patients in the lateral position may have difficulty in flexing both the knees and hips actively to assume and maintain a kyphotic position. Psychological and muscular relaxation due to sedation or a startle reflex will cause the patient to extend at both joints, narrowing the interspinous spaces. Patients who aggressively curl up will also tend to roll anteriorly, which shifts
the midline axis away from the perpendicular plane of the operating room table or floor. P.440

The paramedian approach, which does not depend on maintaining an open interspinous space and is much more forgiving of unintended deviations in the lateral traverse of the needle, should be considered if the midline approach on a patient in the lateral position is unsuccessful after one or two attempts. To review briefly, first establish the deviation of the patient's back from the plane perpendicular to the bed by lightly resting your palm flat against the patient's back—it is easier for you to determine whether your hand is off that plane than to gauge visually whether the patient is off that plane. Second, place the spinal needle about 1 to 2 cm lateral to the interspace. Most practitioners choose the dependent side of the spinal column and place the needle slightly towards the “bottom” of the interspace. Then, direct the needle 15° medial and cephalad to the plane of the back and advance until the ligamentum flavum is transversed and the spinal fluid is entered. An alternative technique is to place the needle 1 cm lateral to the midline, but first to advance perpendicular to the back, expecting to contact the lamina. The needle is then partially withdrawn and the medial and cephalad angle is increased in increments until the needle is “walked off” the edge of the lamina and the spinal canal is entered.

TAKE HOME POINTS

- Spinal anesthetics are frequently done in the lateral position due to disease process or need for deeper sedation.
- The lateral position can make it difficult for patients to curl into the fetal position and for anesthesia providers to appreciate the bony landmarks.
- It is very common for practitioners attempting the midline approach to be as much as one centimeter off the midline.
- Placement of intrathecal anesthetics can usually be easily and safely accomplished using the paramedian approach.

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Consider the Paramedian Approach for Thoracic Epidural Placement, Especially at the Midthoracic Level

Amit Sharma MD

You are scheduled to do the anesthesia for a lung resection for a 68-year-old man with severe chronic obstructive pulmonary disease. You usually use intrathecal preservative-free morphine for this type of case. Today, however, you hear from the surgical resident, surgical fellow, and surgical attending, all within a 15-minute interval and all requesting a thoracic epidural. It isn't your favorite type of catheter to place, but you decide to give it a try. Unfortunately, all you and your junior resident seem to hit is “bone everywhere.” You and your patient are both getting a bit uncomfortable and discouraged. You call your favorite senior pain attending, who is doing cases in the outpatient center and, fortunately, he can come within 5 minutes. He arrives and does one of the “Mr. Miyagi” moves he is famous for—and the catheter is in, taped, and test dosed within 2 minutes. You wonder to yourself, “How the heck does he do that?”

Neuraxial (spinal and epidural) techniques are a vital component of anesthesia and pain-management practice. Since August Bier first described spinal anesthesia in 1898, neuraxial techniques have been in constant use and have been the subject of a vast amount of clinical research and inquiry directed at maximizing both safety and efficacy. Today, epidural analgesia is widely used for postoperative pain control after thoracoadominal and lower-extremity procedures. Insertion of a thoracic epidural catheter is often considered more challenging than placement of its lumbar counterpart. Fortunately, a thorough understanding of the anatomy of the thoracic spine and adherence to a few simple principles will alleviate trepidation and facilitate placement.

The spinal cord is divided into eight cervical (C), twelve thoracic (T), five lumbar (L), five sacral (S), and a few coccygeal (Co) segments. Each spinal segment (except C1) involves a dorsal (sensory) and a ventral (motor) nerve root on either side that join to form a spinal nerve. Each spinal nerve subsequently exits through an intervertebral foramen of the vertebral column. The bony vertebral column surrounds and protects the spinal cord and the spinal nerves encased by arachnoid membrane filled with cerebrospinal fluid. The epidural space is a potential area just outside the dura mater, bound anteriorly by the posterior longitudinal ligament; posteriorly by the vertebral laminae and the ligamentum flavum; and laterally by the vertebral pedicles and intervertebral foramina. At the thoracic level, the epidural space is around 3 to 5 mm in width and contains fat, veins, arteries, lymphatics, and connective tissue. Local anesthetic solution administered in the epidural space bathes the spinal nerves, leading to sensory and motor blockade. Similarly, opioid medications are either absorbed by the venous plexus and act systemically or they cross the dura mater and bind to the opioid receptors on the spinal cord.

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Thoracic dermatomes are organized in circumferential bands from axilla to iliac crest. The T4 dermatome (sensory distribution of T4 spinal nerve) is often located at the level of nipples and T10 at the level of umbilicus (these are universally used reference points). There is a degree of dermatomal overlap, which can cause difficulties with insufficient block for all areas of the surgical site. This problem has been overcome by introduction of newer multibore epidural catheters that enhance the local anesthetic spread and therefore are expected to cover a wider dermatomal distribution.

Technically, both the upper (T1-T2) and the lower thoracic (T10-T12) vertebral interspaces are functionally equivalent to lumbar area since spinous processes at these levels are almost angled horizontally. In contrast, placing epidural catheters at the T3 to T9 level is challenging because of the acute downward angulation of these respective spinous processes (Fig. 103.1). Since successive spinous processes are angled less acutely, the interspinous space is narrower at the distal end (superficial), making the needle entrance harder.

Most anesthesia providers who do not have experience in treating pain start with the midline approach, even in the midthoracic region and that is perhaps why they often have such difficulty in placing the catheter. A senior pain anesthesiologist or an experienced regional anesthesia provider would probably suggest using the paramedian approach to facilitate placing the thoracic epidural catheter at these middle thoracic interspaces. With this approach, the needle is entered slightly inferolateral to the spinous process and angled cephalomedial, thus avoiding the crowded interspinous space. With this technique, the needle tip travels through the skin, subcutaneous tissue, and ligamentum flavum before entering the epidural space, unlike the classic *interspinous* technique in which supraspinous and interspinous ligaments are also encountered in needle trajectory.

The epidural catheter should be placed on the unilateral side, approximately at the mid-dermatomal level to the corresponding surgical incision. For instance, if the intended thoracotomy incision is planned at the right T5-T7 dermatomes, the epidural catheter should be inserted at the right T5-T6 interspace. The unilateral catheter placement is perhaps necessary since Blomberg described midline epidural septa or dorsomedian connective tissue bands in 1986 and are believed to be one of the causes of unilateral anesthesia after an adequate catheter placement.

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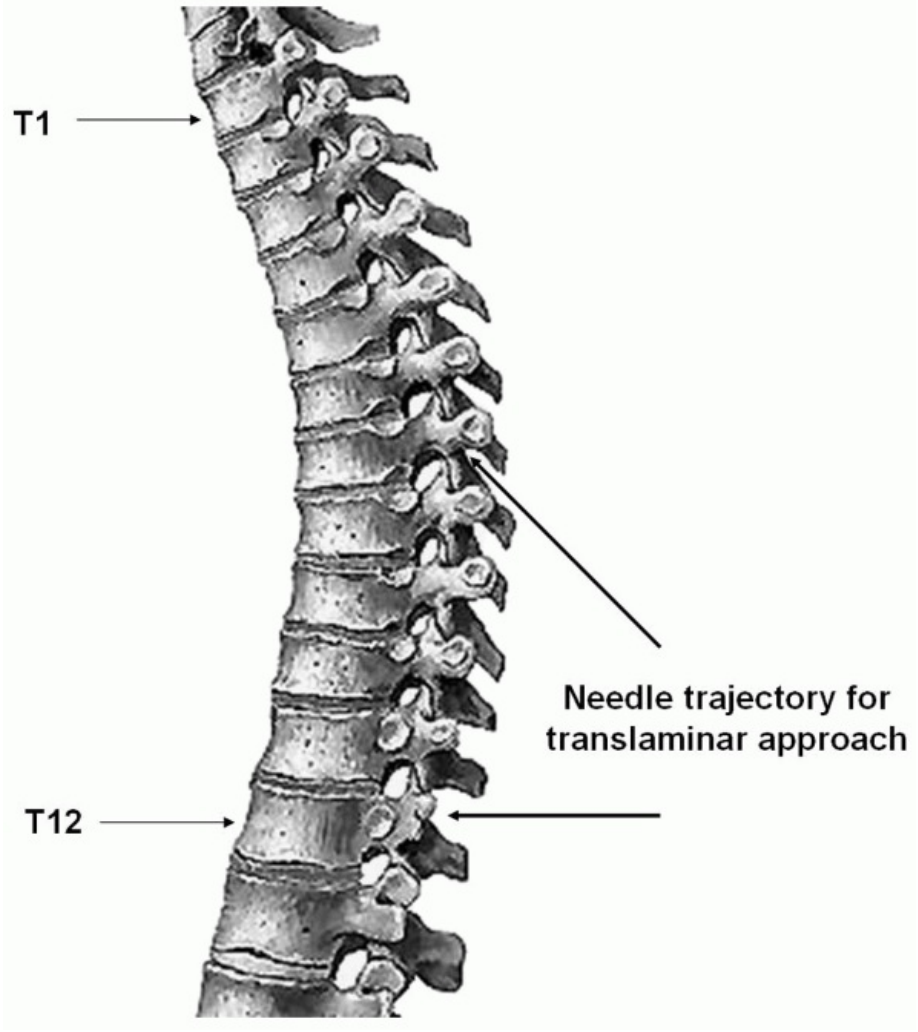


FIGURE 103.1. Difference in needle angulation needed to access epidural space at midthoracic level.

Modified from Netter FH. *Atlas of Human Anatomy.* 3rd ed., 2003. *Icon Learning Systems* , Teterboro, NJ. Plate 146.

Midthoracic epidural technique using the paramedian approach is optimally carried out with the patient in the sitting position. The patient is asked to flex at the thoracic spine by bending forward, and his (or her) forehead is placed on a padded bedside table. A commercially available epidural positioning device (EPD) can make this process simple and can also obviate the need for an assistant to hold the patient (Fig. 103.2). Before beginning the procedure, adequate landmarks should be identified and marked on the patient's skin. The most important landmark is the spinous process of C7 vertebral body, which is very prominent at the midline of the posterior and inferior margin of neck. Subsequent spinous processes can be identified by palpating downwards along the spine. This method is recommended even for the inferior thoracic epidural placement, since a parallel line connecting the superior borders of the iliac crest is an unreliable method of identifying the

L4-5 inter-laminar space. After the target inter-laminar space is identified, the inferior tip of the superior spinous process is marked. The site of needle entry is 1 cm inferior and 1 cm lateral from this point.

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FIGURE 103.2. Epidural positioning device (EPD).

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After adequate skin preparation and draping, the skin is anesthetized using 1% lidocaine. After raising a skin wheal, local anesthesia should be infiltrated along the path of probable needle trajectory (30° to 45° cephalomedian). The 17-gauge, 3½-inch Hustead or Tuohy needle is then inserted perpendicular to the skin into the subcutaneous tissues and advanced until it touches the lamina of inferior vertebral body. This step gives the anesthesia provider an estimation of the depth of epidural space. The needle is now withdrawn to the skin and redirected 30° to 45° cephalomedian almost to the initial depth, as noted in the previous step. Once the needle is positioned at the specified angulation and depth, the stylet is removed and further advances are cautiously made, using the loss-of-resistance technique with either the saline or the dual-chamber (air-saline) method. The mean depth of thoracic epidural space using the paramedian technique is 5.11+/-0.94 cm and varies with body weight, body mass index, and anatomic levels. The greater the patient's weight and the higher the puncture level, the deeper the thoracic epidural space from the body surface.

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TABLE 103.1 TROUBLESHOOTING STEPS FOR THORACIC EPIDURAL TECHNIQUE

RESPONSE OBTAINED	INTERPRETATION	PROBLEM	ACTION
Bony contact is made after the initial needle pass.	Contact with lamina of interior vertebral body or articular process of facet joint is made.	None; needle depth should be noted.	Needle should be withdrawn to skin and redirected 30° to 45° cephalomedial.
Bony contact is made after the second needle pass.	Contact with lamina of interior or superior vertebral body or	Needle is angled	Needle should be withdrawn to skin and redirected 5° to 10° cephalomedial.

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	articular process of facet joint is made.	excessively in either direction.	
If similar response is encountered, a 5° to 10° change should be made in either direction and needle should be advanced until loss of resistance is observed.			
Clear fluid is observed to drip from the needle hub after epidural space is entered and glass syringe is removed.	If the flow stops after few drops, it is probably the saline used with loss-of-resistance technique. If the flow is constant, it is indeed cerebrospinal fluid.	Dura is penetrated.	The needle should be withdrawn and the epidural technique may be repeated at a different interspace. Drug dosages should be adjusted since subarachnoid migration of drugs through the dural rent can occur.
Blood is observed to drip from the needle hub after epidural space is entered and glass syringe is removed.	Needle tip is positioned in the lumen of an epidural blood vessel.	Needle penetrated the dense epidural venous plexus.	The needle bevel is rotated slightly until blood flow stops. If this maneuver fails, the needle tip be withdrawn 1 to 2 centimeters and redirected into the epidural space using loss-of-resistance technique.
Resistance is encountered during advancement of epidural catheter.	False loss of resistance or catheter tip is stuck against dura.	Improper needle tip position.	Loss of resistance should be rechecked after withdrawing the catheter. If present, needle bevel is rotated 30° to 45° and catheter is reinserted.

After the epidural space is identified, the syringe is gently removed and the epidural catheter is inserted approximately 3 to 5 cm into the epidural space. There should be very little resistance to passage of the catheter, once it has exited the needle. The catheter should be tested for intrathecal or intravascular placement, using 3 mL of 1.5% lidocaine mixed with 1:200,000 epinephrine and tunneled, taped, or both, as planned. The correct epidural placement of the catheter can also be confirmed using fluoroscopy, a lowcurrent, electrical-stimulation test, or a Tsui test. The needle should be withdrawn immediately if the patient reports sudden pain in a radicular fashion at any time during the procedure. The troubleshooting process for each step of this procedure is described in Table 103.1.

TAKE HOME POINTS

- Neuraxial techniques are valuable tools in the hands of anesthesiologists and pain physicians.
- Thoracic epidural placement is often difficult and time consuming. Understanding spinal anatomy well and using the paramedian technique can facilitate such placement.
- This chapter briefly describes certain important technical steps as basic principles. Consistently following these guidelines can make thoracic epidural placement a pleasant and an organized venture.

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Incorporate Ultrasound Guidance for Peripheral Nerve Blockade Into Your Practice

Michael Aziz MD

Jean-Louis Horn MD

Use of ultrasound-guided, peripheral-nerve blockade has become more popular in the last 5 years. Historically, peripheral-nerve blockade was facilitated by contacting the nerve with the block needle to elicit a parasthesia or by feeling a “pop” with fascial perforation (or both). In the 1970s, nerve stimulation allowed for more precise location of peripheral nerves based on motorstimulation patterns. Despite the growing popularity of regional anesthesia, it remains burdened with failures and occasional complications, including nerve injury and parasthesia, local anesthetic toxicity, pneumothorax, painful muscle stimulation, and neuroaxial anesthesia. With the decrease in cost and improved portability of high-resolution ultrasound devices, we now have access to devices that visualize peripheral nerves and surrounding structures to guide needle and catheter placement for peripheral-nerve blockade. Using ultrasound-guided, peripheral-nerve block decreases the length of procedures and the period during which the block takes effect; improves the quality of the block; improves the success rate; and reduces the risk for complications.

Understanding the basic physics of ultrasound is necessary to safely master its clinical uses. The echo is a sound wave generated by the vibration of piezoelectric crystals aligned at the tip of a probe. The received sound wave vibrates the piezoelectric crystals to generate an electric current. Structures are visualized depending on their specific acoustic impedance or echogenicity. Depth is measured by the delay of the echo wave's return to the probe, assuming a speed of sound of 1540 m/sec in human soft tissue. The echo pulse can be absorbed, reflected, refracted, or scattered, creating artifacts. Image resolution and echo penetration relates to the frequency of the sent signal. High frequency signals (from 8 MHz to 15 MHz) produces high-resolution images at the expense of penetration and is suited for superficial structures only. As the frequency decreases (from 2MHz to 8 MHz), penetration increases, but resolution decreases. To maintain proper orientation, each probe has a mark on its side that corresponds to a mark on the screen.

Simultaneous use of a nerve stimulator is valuable to those learning to use ultrasound guidance. Attention to sterility is crucial as adding a probe to the field complicates sterile technique. The identification of nerves requires detailed knowledge of trans-sectional anatomy and the characteristic P.448 echogenicity of the adjacent structures and nerves in question. Scanning a nerve in a transverse plane, as opposed to a longitudinal plan, facilitates its identification. Neurons appear to be hypoechoic or black, and connective tissue appears to be hyperechoic or grey or white. Each nerve has its own characteristic pattern of echogenicity, depending on its location: The trunks of the brachial plexus appear to be hypoechoic, whereas distal nerves are relatively hyperechoic. Nerves are anisotropic meaning their echogenicity changes depending on the angle of incidence of the echo beam, a property that can be utilized to differentiate peripheral nerves from other structures less anisotropic (tendons) or not anisotropic (muscle, fat, bone), which do not change echogenicity. Standardizing the approach by developing a specific pattern of recognition for each peripheral nerve is useful. First, specific surrounding structures are identified (bones, vessels, muscles), and then nerves are located. When learning the technique, the nerve, the needle shaft, and the tip of the needle should be visualized using the long axis of the probe to maintain constant visualization of the needle. A motor pattern can be elicited and the practitioner can observe appropriate local anesthetic spread in the tissue or the advancement of a catheter for continuous perineural infusion. Some authors have identified preferable patterns of local anesthetic spread along the nerve. Ideally, the local anesthetic should be seen bathing the entire nerve as the local anesthetic is injected to ensure reliable anesthesia of the nerve (donut sign). When dealing with separated nerves, as in the axillary approach of the brachial plexus, local anesthetic should be seen bathing all desirable nerves or the needle can be moved appropriately if only partial local anesthetic spread is visualized.

Using ultrasound guidance presents several important potential advantages. It takes much of the guessing out of the regional technique and relieves the anxiety of not being able to find a nerve, as anatomic variations are frequent. It minimizes the number of needle passes, with a subsequent decrease in procedural pain and trauma. Preblock scanning precisely maps the topography of the targeted area, including the depth of structures, and defines the best angle of approach (Fig. 104.1). Real-time tracking of the needle can avoid injury to vascular structures, pleura, and spinal cord. Visualizing local anesthetic spread and adjusting the needle to bathe the targeted nerves thoroughly with local anesthetic can improve success rate and efficiency (Fig. 104.2). Using a nerve-stimulator technique alone, current is dispersed immediately after an initial injection of local anesthetic and nerve relocalization becomes difficult with subsequent needle repositioning. Experienced users of ultrasound guidance may not need to use a nerve stimulator, because they have better control of needle positioning near the nerve. Using ultrasound guidance may reduce the incidence of neuropathy by avoiding direct nerve puncture and nerve injury. P.449

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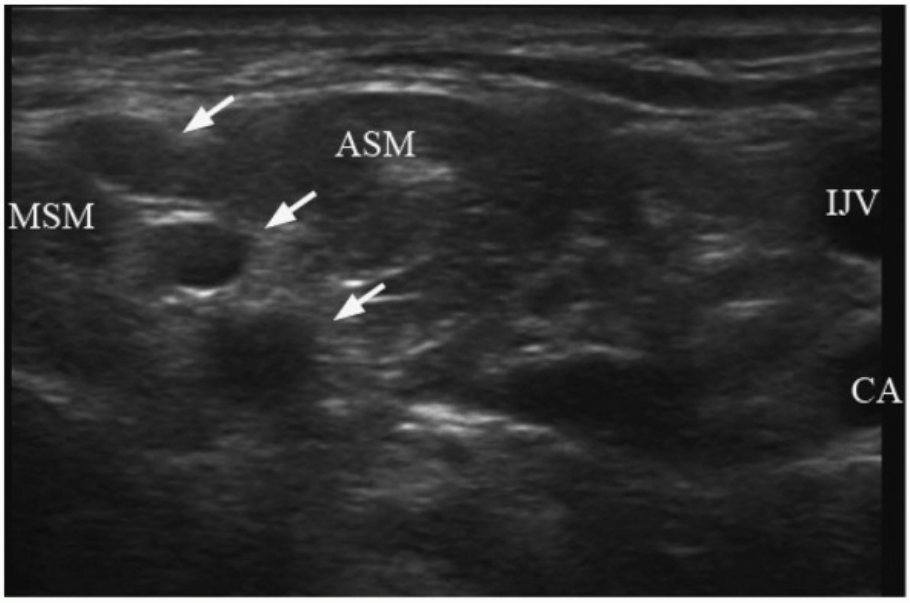


FIGURE 104.1. Interscalene brachial plexus. Arrows indicate nerve roots; ASM, anterior scalene muscle; MSM, middle scalene muscle; IJV, internal jugular vein; CA, carotid artery.

Ultrasound guidance has some limitations. Ultrasound machines with high resolution are costly and complicated to use. Mastering the techniques and learning the detailed trans-sectional anatomy takes months of education and practice. Needle guidance under ultrasound represents another challenge, because the beam is very narrow (about 1 mm thick) and a sharp angle of approach limits needle visualization. To date, no formal education program with certification exists to facilitate training. Those well trained in regional anesthesia have a strong understanding of the involved anatomy and can perform fast and efficient nerve blocks without ultrasound. For those clinicians, the extra time required to learn this new approach seems like a burden on an already-efficient practice; however, no technique is free from failure or complication, and we should always look for ways to improve patient care. The literature is beginning to show the benefits of using ultrasound guidance. Several prospective, randomized, controlled trials have shown use of ultrasound guidance to reduce the length of procedures and the period during which the block takes effect; to extend the duration of the block; and to reduce the amount of local anesthetic needed to obtain a complete block. The literature is beginning to demonstrate increased success rates and reduced complication rates. Most of those anesthesiologists who provide regional anesthesia and who have embraced ultrasound in their practice believe that the literature will ultimately support its widespread use for improved patient care and safety.

P.450

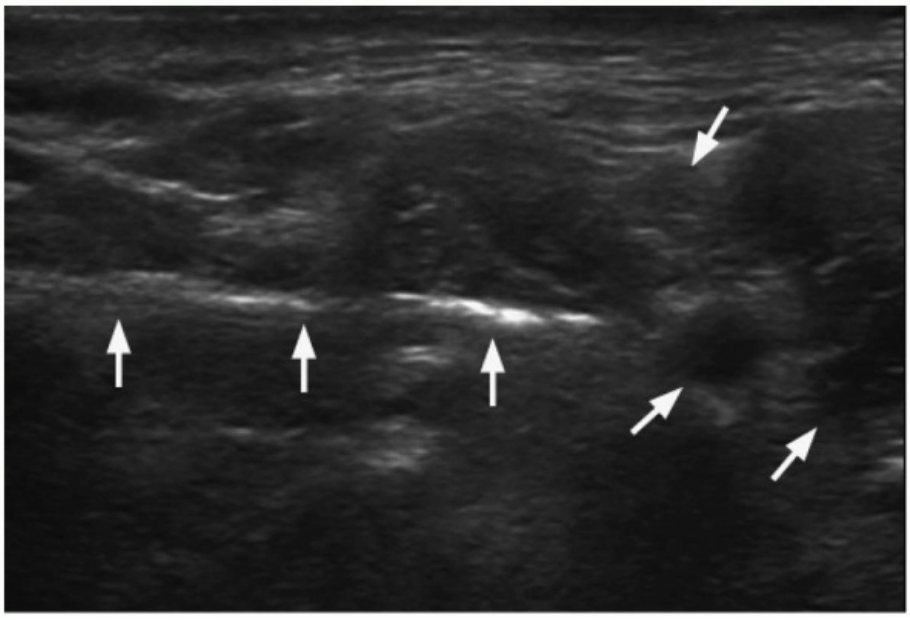


FIGURE 104.2. Interscalene brachial plexus with local anesthetic. Local anesthetic fills the interscalene groove. Arrows indicate needle with tip in close proximity to nerve.

TAKE HOME POINTS

- Ultrasound technology has become a useful adjunct to peripheral-nerve blockade.
- Becoming familiar with the trans-sectional anatomy and appearance of nerves on ultrasound takes time and practice.

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- Ultrasound guidance adds certainty and comfort to needle placement and visualizes nearby structures to be avoided.
- Visualization of local anesthetic spread around nerves assures a reliable block and can be used to guide relocation of a needle.
- Look for future evidence to validate that using ultrasound guidance in placing nerve blockades increases the success rate and reduces the complication rate more than does using nerve-stimulation technique alone.

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Do Not Overlook the "Old-Fashioned" Bier Block, but Beware of the Speedy Surgeon!

Surjya Sen MD

Michael W. Barts CRNA, BSAnesth, AAAPM

A 26-year-old woman with no significant past medical history presents for removal of a ganglion cyst on the dorsum of her wrist. The surgeon predicts a quick procedure, and an intravenous regional anesthetic with lidocaine is chosen. As promised, from incision to closure, the operation is completed within 12 minutes. Planning for a rapid turnover and quick recovery, the tourniquet is released and the patient is taken to the postanesthesia recovery unit. Within seconds after arriving, the patient begins to complain of severe pain in her arm, then dizziness, and, ultimately, she begins to seize. What happened? Could such an outcome have been predicted? If so, what could have been done to prevent it?

INTRODUCTION AND HISTORY

Described by August Bier in 1908, "vein anesthesia" was originally proposed for surgery of the elbow and amputations of the feet. It was noted to use a "new avenue" for getting the anesthetic agent "to the end apparatus of the nerves as well to the nerve trunks": the blood vessel. Though Bier reported his method in at least five journals over the course of 2 years, the technique did not rapidly gain popularity. It involved special equipment (Esmarch bandages were not widely available at the time), meticulous exsanguinations of the limb, and a cutdown to locate the vein. With the introduction of brachial plexus blocks in 1911, interest in "vein anesthesia" quickly faded.

Nearly three decades after its introduction, use of the technique surged when it became particularly useful on the battlefields of World War II. The introduction of safer amide local anesthetics (i.e., lidocaine), the use of percutaneous needles to cannulate veins, and the introduction of a commercially available double-cuff tourniquet helped what was then known as the Bier block gain popularity.

ADVANTAGES AND DISADVANTAGES

With some of the initial disadvantages being overcome by advancements in the field of anesthesia, the advantages of the block are more apparent. From a technical standpoint, all that is needed is successful cannulation of a vein in the involved extremity. Anesthesia can be set up quickly and easily, the length of anesthesia is predictable, recovery is rapid, and the block itself is extremely reliable. P.452

Any clinician planning to use the block technique must also be aware of the disadvantages of the procedure. As originally described by Bier himself, upon release of the tourniquet, the local anesthetic may cause systemic toxicity. Second, pain from tourniquet use is the primary limitation on the duration of anesthesia. To combat this, double tourniquets and subcutaneous infiltration anesthesia have been proposed but have not yet eliminated this limitation. It is also important to note that postoperative pain relief is virtually nonexistent with this method. Unlike brachial and lumbar plexus blocks, once the tourniquet is released, surgical anesthesia quickly dissipates and the patient is left without the benefit of residual analgesia.

Two other limitations involve patient selection. First, the block cannot be used for a patient in whom movement of the operative extremity causes pain. The process of exsanguination with an Esmarch bandage can generate significant pressures that can be quite painful. Second, the venous system of the involved extremity must be intact. Traumatic hematomas, open fractures, and the like are contraindications to performing the block.

TECHNIQUE

1. Place a small-gauge intravenous catheter in the distal portion of the extremity to be blocked. A smaller gauge helps decrease the area through which the injected local anesthetic can ooze out after exsanguination and tourniquet inflation
2. Wrap soft cloth over the proximal portion of the extremity to be blocked. This wrap should be free of wrinkles and have a smooth circumferential fit, since the tourniquet will compress it.
3. Place the tourniquet over the cloth padding. Raise the limb to allow venous drainage via gravity. To exsanguinate the forearm and particularly the hand for the purpose of finger, hand or wrist short procedures, one of the authors (MWB) has the patient squeeze a tennis ball in his hand. The ball has the correct shape, size, and degree of hardness to force the palmar blood out during Esmarch exsanguination. The structures of the hand remain in anatomic alignment, so there is less discomfort with compression. If cross contamination of the ball is a concern, first place the ball in a disposable glove for protection.
4. Next, exsanguinate the extremity by wrapping an Esmarch bandage in a distal to proximal manner.
5. Inflate the tourniquet to 300 mmHg. If using a double tourniquet, inflate the distal cuff first. Then, inflate the proximal cuff and deflate the distal cuff.
6. Inject local anesthetic into the vein, and raise the extremity to allow "downward" flow via gravity. P.453
7. The block is significantly better tolerated with concomitant intravenous sedation (via an intravenous catheter in the other arm, of course!). Sedation can specifically help a patient tolerate pain from tourniquet use for longer durations and may raise the patient's seizure threshold. If a double tourniquet is used, inflation of the distal cuff followed by deflation of the painful proximal cuff can add 15 to 30 minutes of operative time. Alerting the surgeon of this "window" of time when the cuff is changed is important.

AGENTS AND ADJUNCTS

Most commonly, the local anesthetic agent of choice is lidocaine. Large volume, dilute concentrations (i.e., 50 mL of 0.5% lidocaine) have been advocated, but smaller, more concentrated amounts (12 to 15 mL of 2% lidocaine) can also serve the same purpose. Mepivacaine provides a well tolerated alternative to lidocaine, one that has some longer anesthetic and analgesic effects after the tourniquet is released. Typical dosing involves 1.5% 30 cc diluted with 20

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cc of NaCl (preservative free). This renders a final concentration of 0.9% in 50 cc, which can be used in 35-cc to 50-cc aliquots for small to large arms. Other local anesthetics, such as ropivacaine (0.2%) and even bupivacaine (0.25%), have been described for use in the Bier block. However, since the block is usually limited by pain from tourniquet use and not by local anesthetic duration, these choices do not offer major advantages. Use of ropivacaine or bupivacaine poses higher risks for significant cardiac toxicity in the event of inadvertent tourniquet failure and resulting release of drug, which sometimes leads to resuscitative difficulty. Most anesthesia providers feel that the benefits of using these anesthetics do not justify the risks of using them.

Various adjuncts to the use of local anesthetics have also been studied. In a systematic review, Choyce et al. noted minimal positive benefits for most adjuncts. Opiates, such as meperidine and morphine, showed slightly faster onset and somewhat slower recovery. Fentanyl, however, did not provide any detectable advantage.

The primary benefit of using nonsteroidal anti-inflammatory drugs as an adjunct to use of local anesthetics is prolonged postoperative analgesia. Alkalinization helped to decrease the discomfort of injection but failed to provide a statistically significant advantage in terms of anesthesia or postoperative pain relief. Additionally, neuromuscular blockers, such as atracurium, were noted to help with muscle relaxation (particularly helpful for fracture reduction) but carried the additional risk of residual blockade.

Overall, nonsteroidal anti-inflammatory drugs and clonidine were the adjunctive agents noted to supplement the primary properties of the local anesthetic best. P.454

CAUTIONS

The most common complication of the use of intravenous regional anesthesia is that of systemic toxicity of local anesthetic. The clinician should be aware of both central nervous system and cardiac symptoms of toxicity-especially premonitory ones, such as circumoral numbness, dizziness, and tinnitus. To avoid this complication, the tourniquet should always be deflated in a gradual, step-wise fashion. Most texts recommend that tourniquets be left inflated for a minimum of 20 minutes, even for procedures that are shorter in duration.

Other complications include hematomas (especially at the site of intravenous catheter insertion), ecchymoses, and subcutaneous hemorrhage—all of which require vigilance and careful padding with prolonged direct pressure for treatment.

Lastly, insufficient tourniquet pressure can be of significant concern with this method of regional anesthesia. If the tourniquet pressure is not carefully maintained above systolic pressure, arterial inflow with lack of venous outflow can cause engorgement of the extremity. This can be particularly common with lower-extremity procedures in which arteries can be harder to compress (either due to calcifications or location deep within the leg musculature).

TAKE HOME POINTS

- Wrap the extremity in soft cloth before placing the tourniquet.
- Use a small intravenous catheter for introducing the local anesthetic.
- Keep an eye on the pressure gauge of the pneumatic tourniquet (or use a constant-pressure automated tourniquet).
- When deflating the proximal cuff and inflating the distal cuff, double check the valves and lines of each cuff to make sure that the proper one is being deflated or inflated. An error in this part of the procedure can introduce a bolus of the local anesthetic into the patient's system with potential risk for toxicity.
- Have a plan for postoperative pain control in place before terminating the block. Peri-incisional infiltration, intravenous opiates, and appropriate amounts of anxiolytics and amnestics can be very useful adjuncts.
- Pay attention to postprocedure tourniquet deflation. If the operation is completed quickly (i.e., in less than 45 minutes when lidocaine is used),

  multi-stage deflation to allow gradual washout of the local anesthetic is recommended. P.455

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Consider Epidural Anesthetic as an Adjunctive or Primary Technique for Patients Having Mastectomy

Jennifer Vookles MD, MA

Mastectomies are commonly performed under general anesthesia; however, various regional anesthetic techniques for breast surgery have been evaluated to serve as the primary anesthetic, the adjunctive anesthesia, or the postoperative analgesia, or to serve as a combination of these roles. Examples of these techniques include field local infiltration, brachial-plexus blocks, paravertebral blocks, intercostal nerve blocks, and thoracic epidural anesthesia.

These different approaches have a wide range of utility. At one extreme, local infiltration is very limited and could only be used as the sole anesthetic choice for more confined surgeries. Also, the benefits of local anesthesia as well as of some of the peripheral nerve blocks cannot be extended beyond the duration of the local anesthetic effect. At the other extreme, thoracic epidural analgesia (TEA) can be used as the sole anesthetic for extensive breast procedures and can provide prolonged postoperative pain control via continuous infusion. Use of TEA obviates the need for using volatile anesthetics and opioid analgesics, and, as a result, less postoperative nausea and vomiting and shorter recovery times have been reported with TEA use. Even using TEA as an adjunct to a general anesthetic can provide substantial benefits. Using TEA and a local anesthetic selectively blocks cardiac sympathetic fibers, resulting in greater hemodynamic stability, improved myocardial oxygen balance, and an attenuated stress response. Pre-emptive analgesia may also play a role in decreasing postoperative pain and opioid requirements. Greater patient satisfaction has been reported.

Yeh and Doss both used TEA for their primary anesthetic plus sedation. They placed catheters at a T5-6 or T6-7 level and threaded catheters 3 to 5 cm into the epidural space. One reports maintaining a block between C5-T6 with 2% lidocaine; the other reports maintaining a block from roughly 2 cm below the clavicle and to the costal arch inferiorly with 0.2% ropivacaine. Both approaches were successful, although, in some patients, the surgeons needed to supplement with local anesthetic during the axillary-node dissection.

Using various epidural infusions can maintain postoperative analgesia. Doss recommends infusing 0.2% ropivacaine, beginning with 4 to 6 mL/hr. Systemic opioids may be needed if the patient experiences inadequate analgesia. At my institution, an infusion containing a lower concentration of local anesthetic in combination with a low dose of opioid is more commonly used (usually 0.1% ropivacaine, hydromorphone 10 mcg/mL); our initial infusion rate is 10 mL/hr, but this can titrate as indicated by either pain or side effects. P.457

Either technique can be effective, but each has unique precautions. With higher doses of local anesthetic, upper-extremity weakness and subjective shortness of breath should be monitored. Subjective shortness of breath generally results from chest-wall numbness, and patients generally tolerate this well with education. Diaphragmatic weakness would not be expected in the absence of proximal upper-extremity weakness. Such weakness is less common using lower concentrations of local anesthetic; however, patients receiving such lower doses usually need some opioid for adequate analgesia. The opioid can be provided as part of the epidural infusion, as is done at my institution, or systemically. The usual side effects from opioid use can occur with either approach.

TAKE HOME POINTS

- Epidural anesthesia is a well-described and efficacious technique for patients having mastectomy.
- Greater patient satisfaction has been reported with use of epidural anesthesia in patients having mastectomy.
- Various strategies for catheter management and dosing give satisfactory results. Also, if you are using epidural anesthesia as your primary anesthetic, ask the surgeons to supplement with local anesthesia for axillary-node dissection.
- This author uses a low-dose local anesthetic and a low-dose opioid infusion for postoperative analgesia.

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Consider Continuous Paravertebral Block as Your Primary Analgesic Technique

Bruce Ben-David MD

SCENARIO

Your colleagues who are surgeons and your hospital administration are increasingly pushing the limits on what can be done as an ambulatory surgical procedure. Plans are now taking shape to perform mastectomies as same-day surgery. You are asked for your input into realizing this goal.

CONSIDERATIONS

Although the anesthetic can be done appropriately to achieve rapid recovery, the two primary problems to be expected in discharging these patients are pain and postoperative nausea and vomiting (PONV). These problems are not unrelated. Postoperative pain can itself lead to PONV, and so can its treatment with opiates. In fact, we now know that opiate side effects are linearly correlated with opiate consumption. Since this is a very painful procedure, we can anticipate significant need for opiate analgesics postoperatively.

We also know that opiates may adequately control pain at rest but, because of limited mu-agonist-receptor participation in the relevant pathways, they are less effective in controlling pain with movement. Opiates alone therefore may be insufficient to get patients mobilized and discharged home. Moreover, we cannot ignore the significant correlation between severe postoperative pain and the development of persistent or chronic postoperative pain. This is a serious concern in mastectomy surgery; a high percentage of patients having mastectomy develop chronic pain known as “postmastectomy syndrome.” Trying to shuffle our patients quickly out of the hospital without aggressively addressing pain management beyond the immediate hours after surgery clearly is not only likely to fail in the short term but also in the long term.

OPTIONS

Multiple intercostal blocks or local-anesthetic wound infiltration can be expected to provide analgesia of only limited duration. A “soaker” catheter infusing local anesthetic into the wound may add modestly to the analgesia, but the technique generally has not been shown to provide the same level of analgesia as do neural-blockade techniques.

The most commonly used neural-blockade technique today for procedures of the thorax and abdomen is thoracic epidural analgesia (TEA). While TEA can be expected to provide analgesia far superior to that possible with patient controlled analgesia (PCA) opiate, it is not a practicable solution here. TEA use is associated with several side effects, some of them quite common, which render the technique unacceptable for the ambulatory surgical patient. These side effects include pruritis, urinary retention, nausea and vomiting, respiratory depression, and hypotension. Though probably not relevant here, TEAuse is a particular concern in the patient who will be administered anticoagulants postoperatively. In the United States, epidural hematoma has rendered dozens of patients paraplegic when, postoperatively, low-molecular-weight heparin was administered in conjunction with the use of epidural analgesia. P.459

An alternative technique of neural blockade, continuous paravertebral block (CPVB), is at least as effective an analgesic as TEAbut is nearly devoid of TEA's side effects. Originally described more than 100 years ago, paravertebral block has found renewed popularity in recent years and has proven most useful as a continuous technique. Its advantages include its ability to achieve a high degree of sensory and analgesic blockade without causing urinary retention, respiratory depression, pruritis, or hypotension. In addition, CPVB better preserves forced vital capacity (FVC) following thoracotomy. It preserves lower-limb strength, thus facilitating early mobilization of patients. It affords the possibility of unilateral or bilateral blockade, as needed. Lastly, its offset from the midline promises less risk of spinal cord injury as a result of either needle trauma or hematoma. In fact, a second and newer technique of CPVB, the lateral intercostal approach, is even feasible in situations of coagulopathy, spine abnormality, or spinal trauma.

ANATOMY OF THE PARAVERTEBRAL SPACE

The thoracic paravertebral space is a triangular space bounded anteriorly by the parietal pleura, posteriorly by the superior costotransverse ligament, and medially by the vertebral body, intervertebral disc, and intervertebral neural foramen. The apex of the triangle laterally is continuous with the intercostal space. The space is bisected by the very thin endothoracic fascia, which effectively creates two “compartments.” The anterior compartment contains the sympathetic chain, and the posterior compartment contains the intercostal nerve, dorsal ramus, intercostal blood vessels, and rami communicants. Spinal nerves in the paravertebral space are relatively devoid of fascial covering, making them uniquely and exceptionally sensitive to local anesthetic blockade. The paravertebral space openly communicates cephalocaudad, thereby making it possible to effect multiple-dermatomal blockade via a single catheter positioned in the space.

P.460

TECHNIQUE OF CPBV

Placing the continuous paravertebral catheter preoperatively is recommended, because it is technically easier, it affords the chance to use the block as part of the anesthetic, and it allows recovery room nurses immediate access for postoperative pain control. The patient is positioned sitting, with feet dangling over the side of the bed. An intravenous infusion is established, standard monitors applied, and mild sedation administered. The relevant spinal process is identified (note that the steep angulation of thoracic spinous processes brings them opposite the transverse processes of the adjacent more caudad vertebra) and the needle-entry point is marked 2.5 cm lateral to the spinous process. Catheter placement should be at a dermatomal level that represents the midpoint of the surgical wound. Patients may exhibit a vagal response during performance of the block. Aprepared syringe of an anticholinergic drug (e.g., glycopyrrolate) and a syringe with a pressor agent (e.g., ephedrine) therefore should be immediately at hand.

The skin is disinfected and local anesthesia is injected subcutaneously at the needle-entry point. A9-cm 18-gauge Tuohy needle with 1-cm graduated markings is introduced and walked caudally off the transverse process to a depth 1 cm beyond the transverse process. Often one feels a confirmatory “pop” upon penetration of the costotransverse ligament. Adrop of fluid is placed in the needle hub, and the patient asked to inspire deeply. Correct placement is confirmed by lack of movement of the fluid bubble. Adrawing inward of the fluid indicates intrapleural needle placement, in which case the

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needle should be immediately withdrawn. After negative aspiration for blood, 5 ml of 0.5% ropivacaine is injected. Having an assistant inject through an extension tube is helpful, as this helps avoid significant movement of the needle. Following the injection, the extension tube is disconnected and a polyamide, 20-gauge, closed-tip, multiport catheter is inserted to a depth of 3 to 5 cm beyond the tip of the needle. The catheter is affixed in standard fashion.

Postoperatively, a pump containing 0.2% ropivacaine is attached to the CPVB catheter and is infused at a rate of between 6 and 10 mL/h. Adisposable infusion pump can be used for this purpose just as for other continuous peripheral-nerve blocks in patients having ambulatory surgery. Before discharge, patients should receive a set of written instructions and a phone number to call with questions or concerns. Catheters can be easily removed by the patients themselves at home and may be discarded along with the disposable pumps.

MULTIMODAL ANALGESIA: MAKING THE MOST OF YOUR NERVE BLOCK

CPVB and all other nerve blocks, for that matter, are only a temporary and a very incomplete treatment of postoperative pain. Even complete neural blockade does not inhibit the accumulation of inflammatory mediators in the peripheral tissues and their influence on nociceptors. Nor will it block the access of inflammatory mediators to the central nervous system where nociceptive processing will be altered as well. Therefore, once the neural blockade is lifted, the patient is still left in a hyperalgesic state. In this sense, neural blockade can be viewed as simply a window of opportunity. It allows us a several-day window during which to mobilize the patient rapidly, control her pain effectively, and minimize opiate consumption. But neural blockade can provide only a partial answer to postoperative pain.

By combining the nerve block with other analgesic agents, one can achieve better short-term and long-term pain relief. These agents include nonsteroidal anti-inflammatory drugs, COX-2 antagonists, alpha-2-delta calcium-channel blockade (gabapentin or pregabalin), N-methy-l-Daspartase (NMDA) blockade (e.g., intraoperative low-dose ketamine, dextromethorphan, magnesium), single-dose dexamethasone, alpha-2 agonists (clonidine, dexmedetomidine), and, of course, short-acting opioids for breakthrough pain. Use of multimodal analgesia not only enhances the success of early analgesia and discharge of patients but also profoundly reduces patients' chances of developing a chronic pain state.

TAKE HOME POINTS

- CPVB is a remarkably effective analgesic technique, with many advantages over TEA.
- For bilateral breast surgery, as for surgery of the abdomen or retroperitoneum, one needs to place bilateral CPVB catheters.
- Heavy reliance on opioid analgesia is contrary to many of our modern goals of postoperative care.
- Do not rely solely on neural blockade for postoperative analgesia. The best results, in the short and long terms, are achieved by using a multimodal approach.

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Wet Tap? What Now?

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When a puncture occurs in the course of placing an epidural, causing the patient to have headache after the procedure, such headache is called postdural puncture headache (PDPH). The puncture may be accidental or intentional (for example, an intentional puncture is made for lumbar drains for abdominal aortic aneurysm surgery). APDPH may also result from a spinal anesthetic; however, this is much less common due to the smaller size of the needle puncturing the dura mater of the spinal cord.

PDPH is defined by the International Headache Society as follows: "Bilateral headache that develops within 7 days after lumbar puncture and disappears within 14 days after the lumbar puncture. Headache worsens within 15 minutes of upright position and disappears or lessens within 30 minutes of supine position. Usually is frontal, occipital, or both and may involve the neck and upper shoulders."

Note that the differential diagnosis for an acute headache after dural puncture must include cortical venous thrombosis, meningitis, and intracranial hematomas (intracerebral and subdural). PDPH characteristically depends on position and is usually noted within 48 hours after dural puncture; however, PDPD may develop in approximately one third of cases more than 72 hours after dural puncture and, in rare instances, several weeks after dural puncture. Symptoms associated with this type of headache are nausea, emesis, back pain, and visual and hearing alterations.

WHY IS A "SIMPLE HEADACHE" SO IMPORTANT?

Unintentional dural puncture in the obstetric patient is a complication. In an Obstetric Anesthesiology closed-claim study published in the American Society of Anesthesiologists newsletter in 1999, PDPH was the third most common reason for claim (15% of obstetric claims). Such headaches are more common than nerve damage, pain during anesthesia, and maternal brain damage. The median duration of untreated PDPH is 5 days, with a range of 1 to 12 days.

Note that other complications may result from neuraxial anesthesia for obstetrics, and always consider the possibility of concomitant complications. Some of the other complications of epidurals include epidural catheter complications, unintentional subarachnoid injection, intravascular injection of local anesthetic, direct spinal cord injury, bloody tap, epidural abscess, local anesthetic toxicity, and epidural hematoma. P.464

WHAT CAUSES THIS TYPE OF HEADACHE?

Two proposed pathophysiologic mechanisms for PDPH are (a) continued leak of cerebrospinal fluid (CSF) leads to loss of brain support and results in traction on the meninges of the brain, which are pain-sensitive structures; and (b) loss of CSF results in intracranial hypotension, causing compensatory cerebral vasodilatation and dilatation of intracerebral veins, which, in turn, results in a downward sagging of the brain and subsequent tension on the meninges. Activation of adenosine receptors is another, less widely accepted, postulated mechanism.

Significant risk factors include age, gender, needle diameter, needletip design, orientation of the tip during puncture, previous PDPH, history of migraine, and repeated attempts to achieve puncture. Younger patients are more prone to PDPH. Premenopausal women are twice as likely as men to have PDPH. The risk for PDPH relates directly to the diameter of the needle used. The smaller the gauge of the needle used, the lower the incidence of PDPH is. Needle-tip design is also important. Pencil-point needles (Sprotte, Whitacre) have a side orifice on the needle and result in a lower incidence of PDPH than do those with a cutting-point (Quincke). Needle-bevel orientation during insertion is also important. The frequency nearly doubles when the bevel of the needle is inserted perpendicularly instead of parallel to the longitudinal dura fibers. Lastly, the more attempts that are made at a neuraxial technique, the greater the risk for PDPH is, and patients with a history of migraine headaches and previous PDPH are more prone to PDPH.

SO WHAT DO I DO IF I AM PLACING AN EPIDURAL AND CAUSE AN UNINTENTIONAL DURAL PUNCTURE?

Most anesthesiologists recommend removing the needle and placing the epidural in a new interspace, either above or below the previous attempt. Some anesthesiologists recommend placing an intrathecal catheter and using this for obstetrical anesthesia.

Historically, conservative treatment modalities for PDPH were bed rest; hydration; lying in a prone position; abdominal binders; caffeine, oral, or intravenous; theophylline; serotonin agonists; and corticosteroids.

WHICH OF THESE TREATMENTS HAVE EFFICACY AND WHICH HAVE BEEN DISCOUNTED?

Continued bed rest and lying in the prone position often result in a "trend toward increased headache." Allowing early ambulation does not increase the risk for PDPH; on the contrary, early ambulation is recommended.

Additional hydration does not decrease incidence of PDPH. Anecdotal evidence for abdominal binders has been theorized to work by increasing intra-abdominal pressure, thereby equalizing CSF compartment pressures with epidural space pressures. Caffeine (500 mg, intravenously) resolved the symptoms in over two thirds of subjects tested in one trial. Oral dosing of 300 mg of caffeine resulted in only brief relief of symptoms in most patients. A single study using oral theophylline was done, but benefits have not been confirmed, and most anesthesiologists would say the risks outweigh the possible benefits. Trials of serotonin agonists have been done but have not had reproducible results. Lastly, there is only anecdotal evidence for use of corticosteroids. P.465

More aggressive treatments for PDPH include the placement of intrathecal catheters with saline infusion; epidural saline infusions; epidural blood patch (EBP); and epidural colloid infusions.

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Studies of continuous epidural infusions of saline have had inconsistent results. Some trials have shown that a saline bolus infused into the intrathecal space does not reduce the risk for PDPH but may decrease the need for an EBP. Several studies have shown that placement of an intrathecal catheter and continuous infusion of local anesthetic for labor analgesia may decrease the incidence of PDPH. Many practitioners would consider using Epidural Dextran 40 as nonstandard therapy.

An EBP should be reserved for patients for whom conservative treatment fails, and the primary indication for doing an EBP is moderate to severe PDPH. A previous EBP is not a contraindication to epidural anesthesia, and human immunodeficiency virus infections are not a contraindication to doing an EBP. In addition, as with any procedure, an EBP has its own subset of complications, the most common of which are back pain, bradycardia, cauda equina syndrome, pneumocephalus, arachnoiditis, abdominal pain and diarrhea, and cerebral ischemia.

HOW AND WHEN DO I DO AN EPIDURAL BLOOD PATCH?

An EBP is a sterile procedure in which a new or existing epidural is used to inject sterile blood into the epidural space. Using 15 to 20 cc of sterile blood, with the patient remaining recumbent for 1 to 2 hours after the EBP, is the standard of care. The most widely accepted etiology for relief of symptoms from use of an EBP is equalization of compartment pressures and normalization of physiologic compartment pressures.

Early studies noted significant benefits in prophylactic use of an EBP. Unfortunately, the majority of the studies were not prospective. More recent reports suggest that the success rate of EBP may be as low as 65%. Studies indicate that the best time to place a prophylactic EBP is 48 hours after a postdural puncture. The authors recommend using conservative treatment first and using an EBP after failure of conservative treatment for moderate to severe PDPH or for recurrent headaches after a postdural puncture.

TAKE HOME POINTS

- Note the general contraindications for epidural placement: Patient refusal, coagulopathy, local infection, systemic sepsis, and anatomic abnormalities. Avoid dealing with PDPH in someone who should not have had an epidural at all.
- PDPH is fairly rigorously defined. Remember that not all headaches occurring after dural puncture are “spinal headaches.” Always evaluate the patient for other conditions.
- Closed-claim analyses show that malpractice claims relating to PDPH are quite common, especially in obstetrics cases.
- Several mechanisms are proposed for the pathophysiology of PDPH.
- Significant risk factors for PDPH include age, gender, needle diameter, needle-tip design, orientation of the tip during puncture, previous PDPH, history of migraine, and repeated attempts to achieve puncture.
- The authors recommend reserving use of an EBP, which carries its own significant risk, for patients for whom conservative treatment has failed.

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Editors: **Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.**
Title: ***Avoiding Common Anesthesia Errors, 1st Edition***

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A Patient under Regional Anesthesia Who Suddenly Cannot Speak Above A Whisper Has A High Block Until Proven Otherwise

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Use of regional anesthesia has grown in recent years, due in large part to the benefits of improved postoperative pain management. Regional anesthesia also continues to be the preferred technique for management of pain during labor and is considered the safest approach for elective cesarean section. Despite adoption of safer regional anesthetic dosing practices, catastrophic complications from inadvertent intrathecal or intravascular injection are still reported.

INADVERTENT INTRATHECAL INJECTION

High spinal or "total" spinal block is one of the most feared complications of neuraxial anesthesia and other regional techniques. Total spinal block is typically caused by excessive cephalad spread of local anesthetic. Severity of block is variable and depends primarily on baricity and duration of local anesthetic action. Patient characteristics, such as age, height, and anatomic considerations (e.g., pregnancy) also have a role in the level of spinal block. The most common cause of excessive anesthetic spread is unintentional dural puncture and subsequent injection of a large volume of local anesthetic intrathecally. Mechanisms of total spinal block include the following:

1. Migration of the epidural catheter into the intrathecal space after proper placement;
2. Repeated spinal anesthetic administration;
3. Large volume expansion of the epidural space, leading to compression of the dura;
4. Intrathecal spread of an epidural bolus after a previous dural puncture;
5. Unrecognized subdural placement of the epidural catheter; and
6. Inadvertent injection or catheter placement into the dural sleeve surrounding the nerve root.

The onset of total spinal block may be quick or delayed, depending on the type, volume, rate of injection, and location of injection of local anesthetic. In a patient who is awake, the first signs of high spinal block may include dyspnea, loss of speech, feeling of impending doom, or restlessness. These signs may then be followed by hypotension, bradycardia, unconsciousness, and, ultimately, circulatory (cardiac) arrest.

Anesthesiologists must keep in mind that a total spinal block may occur in patients under general anesthesia who have had epidural or peripheral nerve blocks for perioperative pain management. In these patients, a total spinal block presents only as hypotension or bradycardia intraoperatively. In the postoperative setting, these patients remain unconscious with fixed, dilated pupils and require full ventilatory support until brainstem and respiratory functions return. P.469

INTRAVASCULAR INJECTION

In general, local-anesthetic toxicity results in benign transient symptoms; however, with large doses of anesthetic, central nervous system (CNS) and cardiovascular toxicity can lead to catastrophic consequences. The most common cause of local-anesthetic toxicity is unrecognized intravascular injection of bolus doses of local anesthetics from a misplaced catheter or needle during a regional anesthetic block. The surest sign of intravascular injection is aspiration of blood, but this test is far more sensitive than specific (resulting in a higher false-negative rate). Several case reports have described intravascular injection despite negative aspiration from the catheter. Most authors of these case reports suspect migration of the needle or catheter tip intravascularly as the primary cause. Others theorize that a catheter tip flush against an intravessel wall may also give a falsely negative aspirate. Less commonly, local anesthetic uptake by highly vascular tissues can lead to toxic plasma levels. Understanding the relationship between anesthetic dose, body weight, and speed of systemic absorption is important in clinical practice to help avoid high plasma concentrations of local anesthetic.

The initial symptoms of CNS toxicity may be excitatory and include tinnitus, perioral numbness, metallic taste, visual disturbances, peripheral motor twitching, and, eventually, grand mal seizures. As plasma levels of anesthetic increase, CNS depressant effects predominate, with disorientation, drowsiness, or unconsciousness being most common.

The cardiovascular effects of local-anesthetic toxicity are seen at serum concentrations of local anesthetic higher than those necessary to elicit CNS toxicity. All local anesthetics directly depress cardiac contractility through their inhibitory action on voltage-gated sodium channels in cardiac muscle. Local anesthetics have a variable effect on the fast-conducting tissues that predispose the heart to dysrhythmias. Although all anesthetics at high plasma levels cause dose-dependent cardiac depression, bupivacaine and tetracaine are the most potent inhibitors and are associated most frequently with severe cardiac collapse. Bupivacaine-associated cardiac arrest is often refractory to normal resuscitative efforts; prolonged cardiopulmonary support is often required.

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PREVENTION AND TREATMENT

Focusing on prevention is of the utmost importance for the anesthesiologist. No single test can guarantee proper needle or catheter placement. Several case reports involving total spinal anesthesia or large bolus intravascular injection have been reported despite use of proper preventive techniques. Possible causes of false-negative aspiration include obstruction of the catheter tip (by tissue or vascular wall), partial dural puncture, or subdural placement. Most false-negative test doses are the result of insufficient wait time or inadequate concentration of epinephrine in solution.

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The use of dilute anesthetic solutions and the practice of slow, incremental dosing are both very effective methods of preventing serious complications of local anesthetics. The use of dilute anesthetic solutions for epidurals has been clearly supported in the obstetric literature and is most likely responsible for the sharp decrease in morbidity and mortality associated with epidurals placed on patients in labor. Similarly, the practice of fractional dosing, typically 5 mL, has very effectively avoided these complications. The ideal incremental dose is one sufficient to elicit mild toxicity symptoms while avoiding the life-threatening sequelae from a large-dose intravascular or intrathecal injection. The addition of epinephrine to local anesthetic provides a means to identify reliably an intravascular injection-namely, if the systolic blood pressure increases by more than 15 mm Hg in healthy patients, such an injection is deemed to have occurred. The efficacy of this method may be less reliable in certain subsets of patients, such as those taking β-blockers and those in active labor.

The American Society of Regional Anesthesia and Pain Medicine recommends taking the following steps to enhance safety:

1. Gentle aspiration at the needle or catheter;
2. Slow, incremental injection;
3. Dose limitation based on established per-kg guidelines (Table 109.1); and
4. Use of intravascular markers.

Even with strict adherence to these guidelines, the anesthesia provider should always be prepared to treat the most severe complications of regional anesthesia. The treatment for total spinal block is always supportive and often requires ventilatory assistance with 100% oxygen via mask or endotracheal intubation and circulatory support with intravenous fluids and vasopressors. In the laboring patient, reducing aortocaval compression by positioning the patient in the lateral (tilt) position is necessary.

The treatment for intravascular injection is also mainly supportive, with the use of benzodiazepines, thiopental, or propofol for cessation of seizures and prolonged cardiopulmonary resuscitation for severe cardiotoxicity. Anesthesiologists must remain vigilant for signs of toxicity and must rely on safe practice guidelines to avoid these untoward effects of local anesthetics.

TABLE 109.1 DOSE LIMITATION BASED ON ESTABLISHED PER-KG GUIDELINES

DRUG	PLAIN (MG/KG)	WITH EPI (MG/KG)
Amides		
Bupivacaine	2.5	3.0
Dibucaine	1.0	—
Etidocaine	4.0	5.0
Lidocaine	4.5	7.0
Mepivacaine	4.5	7.0
Prilocaine	6.0	9.0
Ropivacaine	2.5	2.5
Esters		
Chloroprocaine	12.0	15.0
Cocaine	3.0	—
Procaine	7.0	8.0
Tetracaine	1.5	2.5

TAKE HOME POINTS

- Know the situations in which total spinal blocks typically occur-in laboring patients, in elderly patients, etc.
- Don't do a block without having pharmacologic intervention to treat both CNS and cardiac toxicity immediately available.
- A patient who suddenly starts whispering is deemed to have a high block until proven otherwise.
- Remember that high blocks and intravascular injections can and do occur in patients under general anesthesia-consider this as a possible

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etiology if the patient suddenly becomes unstable or fails to emerge as expected.

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Consider Lipid Emulsion Rescue for Local Anesthetic Overdose

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Regional anesthesia—the injection of local anesthetics to block or treat pain—continues to grow in popularity. Regional techniques can provide complete nerve blockade, leading to superior analgesia with relatively few side effects. In rare cases, however, use of regional techniques leads to an overdose of local anesthetic through unintentional intravascular injection of anesthetic or through systemic absorption following local anesthetic infiltration. Systemic overdose is a potentially devastating complication characterized by seizures, cardiovascular collapse, and, possibly, death. Until recently, the standard treatment available to treat local anesthetic overdose was the standard advanced cardiac life support (ACLS) protocol for cardiopulmonary resuscitation (CPR) and, especially in the case of bupivacaine toxicity, these measures were often not enough and cardiopulmonary bypass was necessary. Now, however, new evidence supports using lipid infusion to treat local anesthetic overdose.

BACKGROUND

Local anesthetics produce their effects by blocking sodium channels. The effect is desirable during regional anesthesia when local anesthetics are infiltrated around nerves to provide analgesia and, if desired, motor blockade. If a local anesthetic is absorbed intravenously, however, the same sodiumchannel blockade can occur in all tissues, including the brain and the heart, producing similar dose-dependent attenuation of action potentials throughout these structures.

In the central nervous system (CNS), local anesthetics first depress the inhibitory pathways in the amygdala and hypocampus. Symptoms of such depression begin with visual, auditory, or sensory hallucinations (including circumoral numbness, metallic taste, and tinnitus); progress to irritability, lightheadedness, tremors and muscle twitching; and finally culminate in generalized tonic-clonic seizures. As blood levels increase, the excitatory pathways are also suppressed, leading to loss of consciousness, coma, and eventual respiratory arrest. In general, CNS changes are an early indicator of local anesthetic toxicity and are seen before cardiac toxicity develops.

Local anesthetics also cause direct vasodilation and cardiac depression. The cardiac tissue response to local anesthetics is similar to the nerve tissue response: Blockade of sodium channels inhibits the cardiac action potential, leading to myocardial depression. Lidocaine is frequently used to decrease P.474
myocardial irritability through this mechanism. More potent anesthetics, such as bupivacaine and etidocaine, however, can actually act as proarrhythmic agents, since these anesthetics bind to sodium channels in a statedependent fashion, leading to bradycardia and conduction delay through the myocardium, thereby allowing re-entrant arrhythmias, ventricular fibrillation, or asystole. These cardiac complications resist normal resuscitative measures, and multiple deaths from systemic local anesthetic toxicity, especially with bupivacaine, have been reported.

PREVENTION/PREVENTION

So how do we treat local anesthetic overdose? The primary method of treatment is prevention. Use the least potent and least toxic drug available to achieve the desired affect, and alter the dose based on patient age, weight, and comorbidities to prevent systemic toxicity. Bupivacaine is very cardiotoxic, and the dextro enatiomer seems to possess the most potent effects. This is why the levobupivacaine enatiomer was developed as a separate preparation. Bupivicaine, particularly in high doses or high concentrations, should be avoided. Premedication of patients with benzodiazepines, such as midazolam or lorazepam, may facilitate patient cooperation and can raise the seizure threshold and the level of local anesthetic required to cause CNS symptoms. Remember, however, that these symptoms usually occur early with local anesthetic toxicity and benzodiazepine use may mask these early warning signs without raising the cardiac toxicity threshold.

Before administering local anesthetic, ensure adequate intravenous (IV) access is available, standard monitors are in place, and resuscitative drugs and equipment are readily available. Airway equipment must also be readily available in the event of an overdose, as rapid intubation may be required.

Consider using the following techniques while placing a block to prevent overdose. Avoid transvascular approaches, if possible, and use ultrasound imaging, if available, to confirm needle position before injection. Aspirate before injection and frequently during block placement, and remember to allow adequate time for blood to become visible in the hub of the needle or catheter. Inject a small test dose after aspiration, and evaluate the patient for symptoms before giving the full dose to the patient. Fractionation, the splitting of the total dose into smaller aliquots with aspiration between them, helps ensure that the needle or catheter has not migrated into a nearby vessel during the block placement. Finally, monitor the patient throughout the block, and maintain verbal communication, if possible, to ascertain early signs of intravascular injection (tinnitus, metallic taste in mouth, anxiety or faintness).

To prevent systemic absorption, you may mix a vasoconstrictor with the anesthetic to slow systemic absorption. Epinephrine is a popular choice, although P.475
other vasoactive medications are also used. Epinephrine has the added advantage of causing a rapid increase in heart rate during test-dose administration if it is absorbed systemically.

TREATMENT

Despite the best techniques, however, systemic injection or toxic absorption of local anesthetics still occurs, perhaps in as many as 20 of every 10,000 peripheral nerve blocks. In the case of overdose, treatment measures are largely supportive and follow established treatment protocols for the symptoms experienced. First and foremost, evaluate the airway. Apply supplemental oxygen, ventilatory support, or intubation, if necessary. Ventilate the seizing patient to limit acidosis and to ensure adequate tissue oxygenation. Treat seizures with thiopental, 50 mg IV, or midazolam, 2 mg IV, and titrate up, as necessary, until seizures cease. Succinylcholine may help relax the seizing patient, but remember that muscle blockade only stops the muscular symptoms of seizures; the CNS will have continued seizure activity along with increased metabolic and oxygen demands.

If cardiovascular symptoms occur, begin CPR and ACLS resuscitation measures immediately. Local anesthetic overdose may lead to refractory cardiovascular symptoms, and prolonged CPR may be necessary—CPR of 1 hour or more may be required until clearance of the anesthetic occurs and the heart can begin to beat again; do not give up too quickly! Use a cardioverter to treat malignant ventricular arrhythmias, as required, and provide

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pharmacological support as necessary. Atropine for bradycardia; ephedrine for hypotension; and epinephrine, as required, for cardiovascular collapse. Multiple doses of these drugs may be required and are indicated in local anesthetic overdose. If available, consider instituting cardiac bypass to allow the patient time to clear the drug from the circulatory system and regain normal cardiac function.

TWENTY-PERCENT LIPID EMULSION AS A POTENTIAL TREATMENT

In many cases, however, all of these methods have failed to restore a normal cardiac rhythm, and multiple patients have died secondary to local anesthetic overdose. Recently, a new treatment has emerged, which is showing great promise. Twenty-percent lipid emulsion may provide a "magic bullet" for cardiac collapse secondary to local anesthetic overdose. This milky mix of soybean oil and water (similar to the emulsion in which propofol is mixed) has been in use for many years for total parenteral nutrition (TPN). (Note that propofol is currently supplied in a 10% soybean emulsion, equivalent to 10% lipid emulsion.) Some authors have therefore suggested propofol

infusion as a lipid rescue agent. However, using a dose of 3 mL/kg of propofol (equivalent to the lipid content of 1.5 mL/kg of 20% Intralipid), a 70-kg patient would require injection of 100 cc of propofol-a dose of 1 g (1,000 mg) of propofol! Clearly, propofol is not a suitable substitute for 20% lipid emulsion for treatment of local anesthetic overdose and is contraindicated in cardiovascular collapse; see comment in Weinberg (Suggested Reading, item 16). Sold under the trade names Intralipid and Liposyn, the emulsion is used in thousands of patients daily as a lipid nutritional replacement. Its short-term side effects are minimal, and long-term effects, such as pancreatitis and hypertriglyceridemia, would be expected due to the high fat load in the bloodstream. The drug is inexpensive and readily available in most hospital pharmacies (20% Intralipid retails for approximately $20.00 for a 500 mL bag [in bulk] and bags may be stored at room temperature for approximately 2 years according to the product label). While the required dosage for local anesthetic toxicity is not fully established, a currently suggested protocol uses a loading dose of 1.5 mL/kg IV of 20% lipid emulsion followed by infusion of 0.5 mL/kg/min until cardiac recovery. The patient should be closely monitored until all symptoms resolve. P.476

The above dosing protocol has been used successfully in case reports of adult patients and in studies of adult animals. No evidence exists to suggest a safe dosing regimen in special populations such as pediatric or pregnant patients. However, intravenous lipid emulsions are an established treatment for patients in all stages of life and, in general, the daily dose of lipid for TPN is much greater than the lipid bolus described above.

In pediatric patients, daily lipid doses of 1 to 2 g /kg have been used for decades, well in excess of the above-proposed bolus of 1.5 mL/kg of 20% Intralipid (equivalent to 300 mg/kg). Even in low-birthweight neonates, starting dosages of 1 to 1.5 g/kg/d are well tolerated and levels are often titrated to above 2 g/kg/d without side effects.

In pregnant patients, the safety of lipid infusion has recently become slightly less clear. A 2004 retrospective study suggested that pregnant patients with hyperemesis gravidarum undergoing total parenteral nutrition therapy may have much higher rates of complications directly related to TPN therapy. However, this study is limited to a retrospective analysis of a small number of patients at a single hospital. Multiple other studies and decades of clinical experience have demonstrated the safety of total parenteral nutrition in pregnant patients. Parenteral infusions, including lipid dosing of 1 to 2 g/kg/d or more, have been demonstrated as safe for both the mother and the fetus, particularly since soybean emulsions have replaced older cottonseed-oil emulsions. In light of this evidence, and the potentially catastrophic effects of local anesthetic overdose, there appears to be little to

contraindicate lipid infusion as a last resort in a pregnant patient, and no adjustment in dosing is suggested by existing data. P.477

POTENTIAL MECHANISM

The action of the lipid infusion is unknown, but at least two mechanisms are proposed. The first theory is that the lipid infusion provides a lipid phase in the plasma that absorbs the lipophilic local anesthetic and sequesters it away from body tissues. Indeed, in a study by Weinberg et al., lipid infusion sped the efflux of bupivacaine from isolated rat hearts, supporting this "lipid sink" hypothesis. Other authors have suggested that a different mechanism may be responsible: Since the heart derives most of its energy from lipids and since bupivacaine interferes with lipid metabolism, some have suggested that a massive lipid load simply overwhelms the bupivacaine interference and provides the heart with ample energy supplies to function. Of course, neither of these hypotheses is exclusionary; both could be correct or perhaps another reason lies behind the success of lipid infusion for local anesthetic overdose.

EVIDENCE

While further investigation into the exact mechanism of action proceeds, the limited evidence so far is that it does work. Besides the study mentioned above, additional studies have shown lipid infusion to be potentially lifesaving in the event of local anesthetic overdose. In a study with dogs, Weinberg et al. once again demonstrated the efficacy of lipid infusion. Twelve dogs were given toxic overdoses of bupivacaine, the local anesthetic notorious for refractory cardiac collapse. In the study, all 12 dogs experienced cardiac collapse. All dogs were aggressively resuscitated; half were given lipid infusions, and half were given saline boluses. Remarkably, all six of the lipid-infused dogs survived, whereas the non-lipid-infused animals all died. These are striking results even in a small study.

Now, too, case reports of lipid infusions reversing local anesthetic overdose in human beings are starting to appear. In July of 2006, Rosenblatt et al. reported the first use of lipid infusion to treat bupivacaine cardiac arrest successfully, and, in August of 2006, Litz et al. reported a similar success after ropivicaine-induced asystole. These case reports are promising and support the use of lipid infusion to reverse cardiovascular symptoms of local anesthetic toxicity.

Of course, the use of lipid infusion is not yet a proven, established method for treating local anesthetic overdose. Until more data becomes available, its use must still be considered experimental. The mainstay of safe local anesthetic use in regional anesthesia remains prevention of overdose, and, in the case of toxicity, to follow established ACLS protocols, as needed to

resuscitate patients. However, given the promising track record of lipid infusion and the extremely limited downside of its use in the event of refractory local anesthetic-induced cardiac arrest, its use should be considered early on in the event of an overdose, and a 500-ml bag of 20% lipid emulsion should be readily available in areas in which regional blocks are performed. After all, the drug is relatively harmless, and it just might provide the "magic bullet" required to rescue a patient from a devastating complication. P.478

TAKE HOME POINTS

- Intravascular injection or absorption of local anesthetics can be a catastrophic complication of regional anesthesia.
- Bupivacaine overdose is particularly dangerous because of its cardiac toxicity profile.
- The primary method of treatment of local anesthetic overdose is preventative.
- Supportive measures, including the possibility of cardiopulmonary bypass, are the mainstays of therapy once local anesthetic toxicity occurs.
- 20% lipid emulsion is a promising treatment for local anesthetic toxicity.

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Remember that Postoperative Pain Management Should Be Started Preoperatively

J. Todd Hobelmann MD

Postoperative pain is a significant issue in the anesthesia provider's management of patients. A survey mailed to 56 anesthesiologists stated that the principal endpoints they believed to be the most important to patients were postoperative incisional pain, nausea, vomiting, preoperative anxiety, and pain related to intravenous access placement. A key part of the recovery period is appropriate control of both pain during rest and pain with activity (incident pain). With the continued advances in anesthesia practice, anesthesiologists now have a growing armament to combat postoperative pain, including use of neuraxial anesthesia, nerve blocks, and perioperative analgesic medications. We will discuss some of the pharmaceutical interventions at the anesthesiologists' disposal for the time after surgery.

Planning is an integral, and arguably the most important, component of anesthesia management. As anesthesiologists, our job frequently demands preventing an undesirable situation, like a postinduction hypotension, a difficult airway, patient's movement at the time of skin incision, or postoperative nausea and vomiting. Similarly, postoperative pain management starts with pre-emptive analgesia. Pre-emptive analgesia is an antinociceptive therapy aimed at preventing both peripheral and central sensitization, thereby attenuating the postoperative amplification of pain. Regional anesthesia (both neuraxial and peripheral-nerve blockade) and even local infiltration for superficial procedures are excellent examples of pre-emptive analgesia. Preoperative use of certain nonsteroidal anti-inflammatory drugs (NSAIDs) (piroxicam, 20 mg, taken orally 2 hours before the procedure; intravenous ketorolac), antiepileptic drugs (gabapentin, 600 mg, taken orally 2 hours before the procedure) as well as intravenous opioids and receptor antagonists (ketamine) at the time of induction have all been shown to reduce postoperative analgesic requirements. The pre-emptive analgesic plan should be titrated according to the type of surgery and patients' characteristics. Whenever possible, a multimodal approach should be used.

After adequate preventative steps have been taken, the next step is often dealing with postoperative pain. Opioids are the most commonly used medications under these circumstances. Titrations of morphine, fentanyl, dilaudid, etc. are often used as a first-line agent. Ethnicity (white race), emergency surgery, major surgery, surgery lasting more than 100 minutes, and high pain score on arrival to the PACU are factors predictive of increased opioid requirements. Titration to effect is the goal; however, it is important to realize that the presence of pain does not prevent narcotic-induced respiratory depression, the most worrisome side effect of narcotics. Opioids also have other potential adverse effects, such as pruritis, nausea, vomiting, and constipation. The PACU anesthesiologist must always remain vigilant when administering any opioid dose. Patient-controlled analgesia (PCA) allows patients to determine the timing of analgesic doses and allows for improved titration of analgesia while minimizing patient anxiety. When used correctly, pain management may occur with less risk of side effects. Common intravenous (IV) PCA orders used at the Johns Hopkins Hospital are listed in Table 111.1.

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NSAIDs can be used as part of an effective multimodal analgesia regimen. NSAIDS are beneficial postoperatively, because surgery causes both pain and inflammation. These medications can be divided into three groups: NSAIDs with predominantly analgesic effects (Naproxen, Ketorolac), NSAIDs that are mainly anti-inflammatory (oxicams), and those that confer both benefits (Diclofenac, Ketoprofen, Indomethacin). The one most often used in the PACU is Ketorolac (Toradol). It has been estimated that the use of Toradol in pain management may reduce opioid requirements by up to a third (range of 0% to 79%, depending on the type of surgery) and may improve the patient's pain relief. Nonsteroidal medications have their own set of risk factors, including bleeding from platelet dysfunction, gastrointestinal (GI) upset, and renal impairment. These risks increase with higher dosages, prolonged therapy (more than 3 to 5 days), and, in susceptible patients (i.e., those with a history of GI bleeding or renal insufficiency). The newer class of selective COX-2 inhibitor nonsteroidals (celecoxib, rofecoxib, valdecoxib) was believed to have fewer side effects; however only celecoxib is still on the market as the other two have been show to cause cardiac events in susceptible patients.

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TABLE 111.1 OPIOID NAÏVE PATIENTS

DRUG NAME	DEMAND DOSE	LOCKOUT	LOADING DOSE
Hydromorphone	0.1-0.3 mg	5-10 min	0.4 mg prn
Fentanyl	5-20 mcg	5-8 min	25 mcg prn
Morphine	0.6-2 mg	5-10 min	2-5 mg prn

Demand dose, the dose administered whenever the patient presses the dosing button; lockout, the timeframe in which no medication will be injected, regardless of how many time the patient presses the button; loading dose, the initial bolus given before beginning the PCA. Advanced pain specialists may also use a continuous infusion that runs at a set rate (low dose) throughout. With permission from Jeffrey Richman, MD.

TABLE 111.2 OPIOID CONVERSION CHART

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DRUG NAME	IV DOSE (MG)	PO DOSE (MG)	DURATION (HRS) IV	DURATION (HRS) PO
Morphine	10	30	4-5	4-7
Hydromorphone	1.5	7.5	4-5	4-6
Methadone	10	20	4-5	4-8
Meperidine	75	300	3-5	4-6
Fentanyl	0.1	-	1-2	-
Codeine	130	200	4-6	4-6
Hydrocodone	-	30	-	4-5
Oxycodone	-	20	-	4-5
Propoxyphene	-	65	-	4-6
Nalbuphine	10	-	4-6	-
Butorphanol	2	-	4-6	-

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After discharge, patients may take pain medications for various reasons. In addition, hospital pharmacies may stock certain medications, and not others, limiting physicians' choice of drugs. Understanding how opioids are converted thus is important in the perioperative setting, in the postoperative setting, and upon patient discharge from the hospital. The opioid conversion chart is outlined in Table 111.2. The steps for opioid conversion are discussed in another chapter of this book.

In addition, those patients who are seen on an outpatient basis may take drugs included in other families of pain medications, which must be considered in the perioperative setting. Whether the patient has Complex Regional Pain Syndrome (CRPS), low-back pain, or postherpetic neuralgia, many other classes of pharmaceuticals may be seen. The groups most likely to be encountered (besides narcotics and NSAIDs) include the tricyclic antidepressants (TCAs: Amitriptyline, nortripyline, desipramine); anticonvulsants and membrane stabilizers (gabapentin, pregabalin); and NMDA receptor antagonists (dextromethorphan). TCAs are used in “subclinical” doses to improve overall pain control. The older forms of the drugs, like amytriptyline, carry significant side effects, ranging from anicholinergic phenomenon (dry mouth, sedation) to orthostatic hypotension. The anticonvulsants also have untoward side effects of sedation and potential liver damage (it is essential for the primary provider to follow liver function tests on patients using this class of medication). They are believed to relieve pain by decreasing the overall electrical activity in the brain. The NMDA receptor antagonists are believed to exert their effects by decreasing the sensitization of the central nervous system to pain. The main side effect of this class of drugs is sedation.

TAKE HOME POINTS

- Postoperative pain control plays a vital role in patient satisfaction.
- Postoperative pain management is an integral part of anesthetic management and should be started pre-emptively. Whenever feasible, a multimodal approach should be chosen.
- Some of the patients are already taking pain medications preoperatively, and such medications should be taken into account when planning postoperative pain management. Administration of such medications should not be discontinued before surgery, unless side effects are unmasked during the interview or laboratory values demonstrate areas of concern.
- As our understanding of the pain pathways emerges, our tools for treating pain perioperatively will grow, and patient satisfaction may increase as well.

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Seek Out Hypercapnea in the PACU and Remember that an Acceptable Pulse Oximeter Reading is *not* Assurance of Adequate Ventilation

Michael P. Hutchens MD, MA

"Global warming is not the only downside of elevated CO_2 concentration"

Respiratory complications are common events in the postanesthesia care unit (PACU) — as many as 7% of patients require upper-airway support of some kind during their PACU course. Although pulse oximetry provides excellent monitoring of arterial oxygen saturation, it is an anesthesia truism that reduced arterial oxygen saturation due to hypoventilation in patients receiving supplemental oxygen is a late finding. The physiologic manifestations of hypercapnea are truly protean, and although central nervous system effects are noticeable in patients who are awake, these findings, and others, may be masked by the shifting perioperative milieu. Hypercapnea both results from and causes respiratory arrest and is usually easily reversible in the PACU patient if caught in time.

Carbon dioxide (CO_2) is an odorless, colorless, heavier-than-air gas, which, apart from oxygen and water, may be the most ubiquitous drug in medicine. Anesthesiologists routinely manipulate the CO_2 content of blood to achieve physiologic goals. It is endogenously produced as a byproduct of energy production by degradation of carbohydrates in the Krebs cycle. Although physiologic investigation of the effects of CO_2 began during the Enlightenment, its toxic effects were known in antiquity from the occasional (sometimes intentional) lethal encounter between living beings and caves with elevated CO_2 concentration. Indeed, CO_2 is itself a general anesthetic; it is currently used for laboratory animal euthanasia and sedation of livestock before slaughter. Experimentation in the 1960s using monkeys and cats breathing a 50:50 mix of CO_2 and oxygen demonstrated electroencephalogram (EEG) slowing to isoelectricity after initial activation. Unlike the mechanism of conventional inhalational anesthetics, the mechanism of CO_2 anesthesia is thought to be an effect of locally induced brain acidosis. In an elegant paper published in 1967, Eisele et al. measured the $PaCO_2$ at which response to surgical stimulus was abolished in 50% of experimental dogs as 222 mmHg; this is, in essence, the MAC in dogs of CO_2. Thirtypercent CO_2, in oxygen administered by mask to human beings, produces
anesthesia, but produces a foul acidic taste in the mouth and feelings of anxiety and dyspnea. As $PaCO_2$ rises above 90 mmHg, human beings exhibit stupor and, ultimately, loss of consciousness. P.487

CO_2, whether exogenously or endogenously sourced, has significant physiologic effects apart from those on consciousness. Hypercapnea increases cerebral blood flow and, thus, intracranial pressure. CO_2 is a pulmonary vasoconstrictor, however, and causes a predictable increase in pulmonary-artery pressures at elevated levels. Because of the Bohr effect, hypercapnea decreases hemoglobin affinity for oxygen. An increased CO_2 level also causes systemic hypertension, both from increased cardiac output and arteriolar vasoconstriction. An elevated $PaCO_2$ is proarrythmic, particularly in the presence of halothane. Hyperkalemia can result from release of cellular potassium stores, and at a very high $PaCO_2$, glomerular filtration rate is reduced by afferent arteriolar vasoconstriction. Interestingly, a brief review of the literature reveals multiple cases of $PaCO_2$ levels of more than 200; these patients (mostly children) were stuporous or comatose but, in the absence of hypoxemia, recovered without permanent deficit.

In the immediate perioperative period, it is not unusual to encounter a stuporous (or anxious) patient with abnormal vital signs. While these aberrations are most commonly caused by residual anesthetic, pain, or medication, they can all be caused by hypercapnea as well. Since extubated patients in the PACU do not usually have their end-tidal CO_2 displayed on the monitor, it is up to the clinician to suspect and diagnose hypercapnea from clinical data. Patients in the PACU have two excellent reasons to become hypercapneic. They almost universally receive medications that depress their respiratory drive, and they therefore frequently hypoventilate. Secondly, as patients emerge from anesthesia, their basal metabolic rate increases, and, as a result, they may have increased production of CO_2, thus increasing the relative impact of hypoventilation. Shivering increases this effect, as do agitation and fever. Malignant hyperthermia causes a sustained increase in CO_2 production, but other systemic signs are usually present.

While reduced respiratory rate is one mechanism for hypoventilation, patients who are splinting may hypoventilate while breathing rapidly and taking small breaths. Patients with obstructive sleep apnea or postanesthetic airway obstruction may have a normal respiratory rate but only transport gas during a short part of the respiratory cycle, thus reducing their alveolar ventilation. Regardless of the etiology, the clinician must have a high index of suspicion for hypercapnea in the postoperative patient. Since it often results in end-organ dysfunction, it must be treated quickly. Patients who have decreased respiratory drive from opioid administration should receive naloxone, 40 mcg every 1 to 2 minutes, intravenously, until their respiratory rate and level of consciousness increase. Patients who are apneic from
opioids, or imminently apneic, should of course receive bag-mask ventilation, and the clinician should consider administering a larger dose of naloxone intravenously (the maximum single dose is 400 mcg). Patients with obstructive sleep apnea may benefit from the application of noninvasive ventilation. Patients with splinting can be the most challenging; their hypercapnea paradoxically improves with opioid administration. The clinician must be confident of this etiology before treating it, and, ultimately, these patients may benefit most from regional analgesia. As always, endotracheal intubation and mechanical ventilation are the definitive treatments and should be considered early, while they are still optional, rather than late, when control of the airway becomes emergent. P.488

CO_2 is a powerful drug that affects the physiology and care of the postoperative patient. Regardless of the etiology, the clinician must have a high index of suspicion for hypercapnea. Since it often results in end-organ dysfunction, it must be diagnosed and treated quickly to prevent such dramatic physiologic consequences as intracranial hypertension, confusion, stupor, coma, respiratory arrest, and death.

TAKE HOME POINTS

- Respiratory events in the PACU are common.
- Patients in the PACU are at high risk of hypercapnea.

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- The onset of hypercapnea may be masked by other perioperative conditions.
- CO_2 is a general anesthetic and loss of consciousness occurs above a $PaCO_2$ level of about 90.
- CO_2 is a powerful drug that affects intracranial pressure, hemodynamics, oxygen transport, renal function, and other physiologic parameters.

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Consider Acupuncture as an Adjunct for the Prophylaxis and Treatment of Postoperative Nausea and Vomiting

Leena Mathew MD

Unfortunately, postoperative nausea and vomiting (PONV) is still a common side effect of anesthesia despite the availability of newer-generation antiemetics and shorter-acting anesthetics and opioids. PONV is one of the two most significant factors leading to patient dissatisfaction (the other, of course, being pain). The incidence of PONV is approximately 70% in those patients who are at high risk for this complication and 30% in the general population. Even though postoperative vomiting is self-limited, it can cause significant morbidity, including dehydration, wound dehiscence, aspiration, and electrolyte imbalance. PONV also increases costs since each episode of emesis in the postanesthesia care unit (PACU) delays discharge by an average of 20 minutes.

The following factors place a patient at high risk for PONV:

1. Patient-related factors: Female, nonsmoking, dehydrated, and with prior episodes of PONV.
2. Procedure-related factors: Ear, nose, or throat surgery, intra-abdominal surgery, laparoscopic surgery, strabismus repair, and obstetric or gynecologic surgery. A 30-minute increase in the duration of surgery increases the baseline risk for PONV by 60%. Any surgical procedure needing large fluid administration can cause gastrointestinal edema, which can also increase the incidence of PONV.
3. *Anesthesia-related factors* : The use of volatile agents, nitrous oxide, neostigmine (more than 2.5 mg) and long-acting opioids.

At present, the most common modalities for the prophylaxis and treatment of PONV include ensuring adequate, but not excessive, hydration; avoidance of risk factors; and the use of pharmacologic agents, such as metoclopromide, dexamethasone, and 5 HT 3 antagonists. Acupuncture is an additional approach that shows great promise in prophylaxis and treatment of PONV. In 1997, a National Institutes of Health Consensus Development Panel on acupuncture reviewed the available evidence from randomized controlled trials and concluded that clear evidence indicates that needle acupuncture is efficacious for postoperative and chemotherapy-related nausea and vomiting and probably also for pregnancy-related nausea.

Americans are now spending approximately $27 billion annually on complementary and alternative medical treatments. Statistical reports published as early as 1997 suggest that visits to complementary practitioners outnumber those to conventional practitioners by 12 to 1. Acupuncture is only one of many complementary and alternative modalities, but it is one that is already somewhat familiar to patients. A great degree of integration exists between “standard” medical techniques and acupuncture in the East, especially in China, and there is now a growing recognition of the need for such integration in the Western health system. P.490

To review briefly, acupuncture is accomplished by stimulating acupoints. There are 14 major meridians corresponding to internal organs, along which there are a total of 365 acupoints. Acupuncture involves the placement of fine, disposable, stainless-steel needles (Fig. 113.1) at select acupuncture points. Acupuncture needles range from 1/4 inch to several inches in length and a few thousandths to several thousandths of an inch in diameter. One inch and 1.5 inch are the most commonly used lengths of needle. The vast majority of needles used in the United States are of stainless steel; however, copper, gold, and silver also are used. The placement of needles is followed by a stimulation technique done to the needle to elicit a characteristic sensation called *De Qi*. Stimulation is usually done by manual twirling or electrical stimulation but can also be done by laser or by moxibustion. Moxibustion involves the burning of fine herbs to apply gentle heat at the free end of the needle. The choice of specific needle stimulation technique depends upon the therapist's experience, preference, and patients' concerns and condition. P.491

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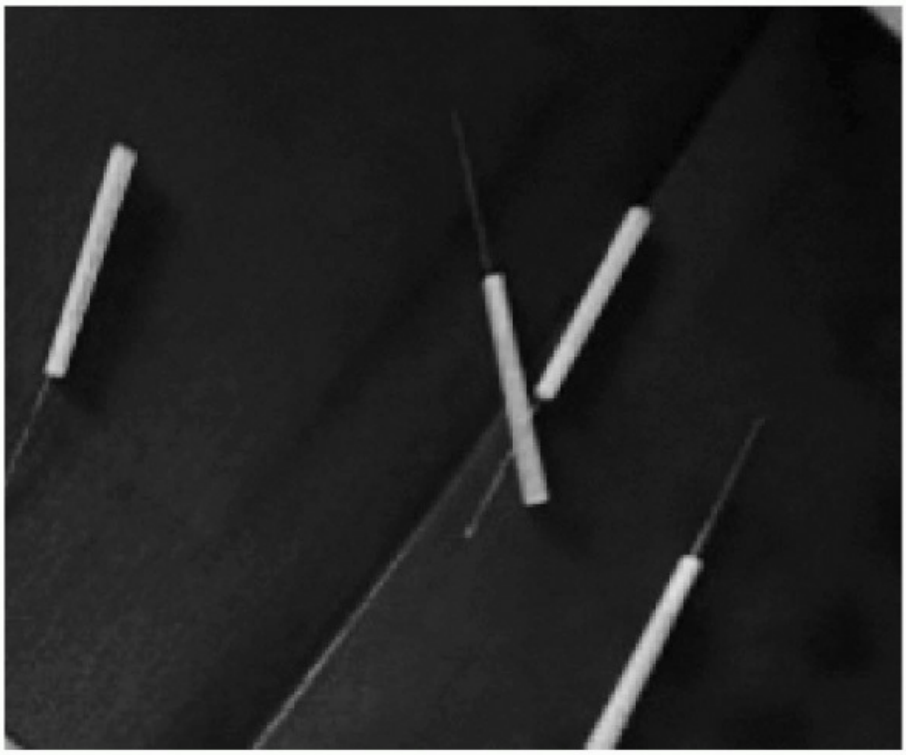


FIGURE 113.1. Stainless-steel acupuncture needles.

Manual acupuncture involves manipulating the inserted needles by lifting, thrusting, twisting, twirling, or doing other complex combinations. It is a traditional method of acupuncture and is most commonly used in clinical practice.

Electroacupuncture (EA) is achieved by attaching the acupuncture needles to an electrical-pulse generator and stimulating the acupoints with electrical pulses. EA appears to be more consistent and to generate more reproducible results, and it is more efficacious than manual acupuncture. The procedure for EA is to insert the acupuncture needle as would normally be done, attain the *De Qi* reaction by hand manipulation, and then attach an electrode to the needle to provide continued stimulation. The benefits of using electrical stimulation are absence of prolonged hand maneuvering, reduction of total treatment time by providing continued stimulus, and better control of the frequency of the stimulus. It can also produce a stronger stimulation, without causing the tissue damage associated with twirling and lifting the needle.

Transcutaneous electrical acustimulation (TEAS) or transcutaneous electrical nerve stimulation (TENS) refers to electrical stimulation performed via skin-surface electrodes placed on the acupoints or nerve dermatomes. TEAS and TENS are very similar because acupoints are very likely distributed along the nerve dermatomes.

The mechanism of action of acupuncture is still unknown. It is hypothesized that, with low-frequency stimulation at certain acupoints, there is activation of A-β and A-alpha fibers, which may influence neurotransmission in the dorsal horn or higher centers. Increased concentrations of β-endorphins have been reported in human cerebrospinal fluid after acupuncture in patients with chronic pain. Different frequencies of stimulation have been shown to be associated with the release of specific opioids. Stimulation at a level of 2 Hz releases enkephalin and β-endorphin, whereas stimulation at a level of 100 Hz releases dynorphin. Acupuncture also decreases gastric acid secretion and promotes forward peristalsis. It is well understood that the analgesic effect of acupuncture can be reversed completely by administration of naloxone.

Many studies indicate that acupuncture at the P6 acupuncture point is effective in ameliorating postoperative nausea and also nausea and vomiting associated with chemotherapy and pregnancy. Acupressure at P6 has also been shown to be antiemetic, and there is fairly extensive literature on the use of P6 acupressure for treating pregnancy-related nausea and vomiting. This point is specifically designated in traditional Chinese medicine for the treatment of vomiting.

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FIGURE 113.2. P 6 point.

The Neiguan or P6 acupoint is located along the pericardial meridian on the volar aspect of the forearm, 2 Cun proximal to the distal wrist crease and approximately 1 to 1.5 cm below the surface of the skin. It lies in between the tendons of the flexor carpi radialis and palmaris longus (Fig. 113.2). A Cun is a unit of traditional Chinese medicine measurement-1 Cun is equal to the middle finger's middle interphalangeal distance and 2 Cun is equal to three times the diameter of the patient's little finger.

Prophylaxis of PONV by acupuncture is more efficacious than treatment of PONV by acupuncture and involves the placement of bilateral P6 needles. These needles are inserted in a sterile fashion immediately after induction of anesthesia and are kept in place for 15 to 30 minutes. If patient positioning or operation timing precludes this, the acupuncture may be done in the postoperative recovery unit after surgery. The needles can then be manually or electrically stimulated.

Acupressure may also be applied by the application of "sea bands," which do not have the same degree of efficacy as acupuncture; however, placement is easily done and does not require professional training. Other techniques of stimulation include TEAS38 and even laser stimulation, although these are not yet well-described techniques.

Studies done of pediatric patients having tonsillectomy have shown that acupuncture is unsuccessful in preventing or treating postoperative nausea and vomiting. However, in these studies, only a unilateral P6 point was used without uniformity in the exact location of P6 as 2 Cun above the wrist. In adult patients, Dundee performed a series of well-designed studies, including prospective, randomized, sham-controlled trials. These studies provided reliable evidence on the efficacy of acupuncture in treating postoperative nausea and vomiting by TEAS at PC6 as an adjuvant to antiemetic use in more than 100 patients in whom chemotherapy-induced sickness was not adequately controlled by antiemetic use alone. In a randomized, controlled trial with concealed allocation, sham control, and careful blinding, EA was found to control emesis effectively in 104 patients with breast cancer receiving chemotherapy. In a recent single-blinded study involving 593 women with early pregnancy, nausea was significantly reduced with acupuncture at P6 alone. No side effects have been reported to date, other than local soreness at the site of needle placement.

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The mechanism of acupressure or acupuncture at P6 to prevent PONV is not clearly understood. Because the P6 point is located near the median nerve, stimulation of this point has been postulated to release neurotransmitters that desensitize the chemo trigger receptor zone (CTZ) and thus prevent nausea and vomiting. However, once the CTZ is sensitized, it is difficult to desensitize it. Accordingly, for prophylaxis of PONV, it may be better for acupuncture to be done after induction but before emergence.

TAKE HOME POINTS

- Acupuncture is an excellent adjunct with minimal side effects in the treatment and prophylaxis of postoperative nausea and vomiting. It is more effective in the prophylaxis than in the treatment of PONV.
- The P6 point is stimulated bilaterally by acupressure or acupuncture with manual or electrical stimulation.
- Placement of the needles can be done after induction or in the PACU.
- The mechanism of action of acupressure or acupuncture at P6 is not completely understood.
- Some providers have had good results with "sea bands," which can be applied by the nursing staff.

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Editors: **Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.**
Title: ***Avoiding Common Anesthesia Errors, 1st Edition***

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Never Delay in Responding to a Call from the PACU about an Eye Complaint

Anagh Vora MD

INCIDENCE AND ETIOLOGY

Eye complications during the perioperative period are actually quite common. In one study, the incidence was 44% after general anesthesia when appropriate protective measures for the eye were not taken. Although ophthalmic complications after anesthesia are often less dramatic than the airway and cardiopulmonary issues that receive so much focus, the importance of promptly recognizing and appropriately managing ophthalmic complications in the perioperative period cannot be overestimated.

Remember that the American Society of Anesthesiologists (ASA) Closed Claim Study demonstrates that there is a significant cost to ignoring or trivializing the patients' risk of perioperative eye injuries. *Of the total claims against anesthesia providers for ocular injuries, an astonishing 81% of cases did not meet the standard of care and could have been prevented.*

Common anesthesia-related eye injuries include corneal abrasions, exposure keratitis, chemical keratitis, movement during ophthalmic surgery, or improper positioning with direct pressure on the eyes. Other less common, but much more serious, ocular complications include postoperative visual loss (POVL) from ischemic optic neuropathy or central retinal artery occlusion.

The underlying etiology of perioperative eye injuries, as with most complications in medicine, is often multifactorial. General anesthesia causes changes in ocular physiology that predispose the eye to injury. These changes include a decrease in both tear production and tear-film stability, suppression of the Bell's phenomenon (normal upward gaze upon closure of the eyelids), inactivation of the blink reflex, and an increase in lagophthalmos (incomplete closure of the eyes). The suppression of these reflexes abolishes the innate protective mechanism of the eye that guards against direct trauma and corneal desiccation and can lead to an increased incidence of corneal abrasions and exposure keratitis.

CORNEAL ABRASIONS AND KERATITIS

Corneal Abrasions.

Corneal abrasions result from disruption of the corneal epithelium, usually as the result of direct trauma to the cornea. Common scenarios include direct injury from stethoscopes, identification badges, and watches during intubation; improper face-mask positioning or surgical draping; and even the patient himself rubbing his eyes.

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Exposure Keratitis.

Exposure keratitis (EK) is inflammation of the cornea from exposure and drying of the corneal epithelial surface. The most common cause of EK during anesthesia is lagophthalmos or incomplete closure of the eyelids for prolonged periods. Partial exposure of the eye, coupled with decreased tear-film production and stability, leads to breakdown of the corneal epithelium. Sequelae of EK include epithelial defects, corneal infiltrates, ulceration, perforation, and even endopthalmitis.

Chemical Keratitis.

Chemical keratitis is serious eye condition, resulting from exposure of the eye socket and globe to potentially toxic substances, such as surgical preparatory solutions. A commonly used agent for surgical scrubs is Hibiclens, a preparation of 4% chlorhexidine antiseptic solution and detergent. Remember that in *very dilute* concentrations (0.005%), chlorhexidine is a valuable ophthalmologic agent and has been used as a preservative for soft and gas-permeable contact-lens solutions. Also, concentrations of 0.02% can be used with propamide to treat *Acanthamoeba* keratitis. However, the 4% solution should be considered toxic to the eye. Injuries usually involve epithelial defects associated with epithelial-cell death and desquamation, which is potentiated by the presence of the detergent. Other described injuries include conjunctival hyperemia, chemosis, hypotony, anterior-chamber flare, and corneal edema. Rarely, exposure to large amounts of chlorhexidine has necessitated corneal transplant.

SIGNS AND SYMPTOMS

Symptoms

- Foreign-body sensation (usually described as a gritty sensation);
- Pain, burning, itching;
- Decreased vision; and
- Photophobia.

Signs

- Dye uptake of the cornea on fluroscein staining;
- Specific pattern of staining related to etiology;
- Circumcorneal injection (limbal reaction); and
- Severe cases may manifest as corneal epithelial defects or ulcerations.

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Management

- Prompt recognition of corneal abrasion and keratitis;
- For mild symptoms, administration of preservative-free drops during the day and ointment at night;
- Reassurance that condition is temporary and should resolve in 2 to 3 days; and
- For moderate to severe symptoms, early ophthalmic consultation (possible treatments by the ophthalmologist include addition of antibiotics to lubricating regimen, temporary tarsorrhaphy, and definitive surgical therapy). P.497

Prevention

- Patients' eyes should be taped shut upon loss of consciousness, except in cases in which securing the airway should take precedence (e.g., patients with obstructive sleep apnea, morbidly obese patients with rapid desaturation, and patients requiring rapid-sequence intubation);
- Prevent direct pressure on the eyes in patients having surgery in the prone position (consider using molded eye shields if they are available at your hospital);
- Maintain neutral position of the head as a head in the lateral position increases intraocular pressure in the dependent eye and increases the likelihood of corneal edema and epithelial breakdown; and
- Anticipate possible eye injuries during administration of anesthesia and remain vigilant in the PACU for signs and symptoms of postoperative ocular complications.

CASE SCENARIOS

Scenario 1.

Your parents are in town, and your spouse has just paged you to tell you that they will meet you at the restaurant. You are happily changing out of scrubs when you are paged by the postanesthesia care unit (PACU). The nurse tells you that your last patient of the day had been dozing but is now awake and complaining of right-eye pain and foreign-body sensation. She is a 27-year-old woman who had repair of a compound fracture of the left tibia and fibula. She has minimal pain in her leg after the excellent epidural you placed earlier. You ask the nurse how bad the eye pain is, and she says, “The patient's crying pretty hard and the light hurts her eyes.”

Do:

- See the patient yourself as soon as possible-your mom and dad will have to wait! Understand that this is actually a fairly common clinical scenario, one that you will encounter intermittently in your practice. The patient is not experiencing incisional or surgical pain because of the epidural; therefore, she may have a heightened perception of the eye pain. Remember also that people in general are usually very careful and anxious about their eyes and eyesight and the fear of being blinded is a universal one—it is not unheard of for a patient to describe for you an eye injury that caused them great distress that they suffered years ago.
- Review the basic history of the patient, including the ophthalmologic history, and try to establish the temporal associations of the eye symptoms as well as descriptive characteristics and alleviating and exacerbating stimuli.
- Try assessing visual acuity (counting fingers, reading name badge, etc.). You do not need a Snellen card (the little pocket card with the black letters), unless a medical student happens to have one handy. P.498
- Reassure the patient that, most likely, this is a benign process (probably a corneal abrasion), but you will consult ophthalmology to make sure. Inform the patient that the ophthalmology consultant is likely to repeat the exam you just did as well as to do some additional testing after adding some drops to the eye.

Don't:

- Never avoid the complaint or wait for the patient to leave the PACU.
- Don't tell the PACU nurses that a significant percent of corneal abrasions happen after the patient has left the operating room and that, therefore, this is not a true anesthetic complication. This *is* a perioperative complication, and, as the anesthesia provider, you are responsible for medical care in the PACU.
- Don't call an ophthalmology consultant before seeing the patient yourself. The ophthalmology consultant will rightfully expect you to give a brief synopsis of the current situation.

Scenario 2.

You are on call, and, at about midnight, you receive a page from the intensive care unit (ICU), asking you to see the patient whose anesthesia you provided earlier that day. The patient is a 70-year-old man with a history of diabetes and hypertension who had lumbar-spine fusion and instrumentation and who is now complaining of decreased vision in his right eye. During the procedure (which lasted about 8 hours), the patient had a significant blood loss of 3200 mL and, despite transfusion during the procedure, had an initial hematocrit in the ICU of 27%. The patient was not placed in Mayfield pins; instead, the patient's head was placed in a foam headrest. In your anesthesia record, you were careful to make sure that you wrote “the face was padded without direct pressure on the eyes” (which it was). After completion of the surgery, the anesthesia team agreed that there was significant facial edema, and the patient was left intubated. After a short course of postoperative ventilation, the patient was extubated. He had no initial complaints, until the patient's daughter mentioned that the patient told her he could not see her standing by the side of the bed.

Do:

- Recognize that this is potentially a much more serious situation than a corneal abrasion.
- See this patient immediately. Document your assessment as above.
- Call the surgeons immediately to discuss the risks inherent in cases in doing a lengthy procedure on a patient in the prone position, with significant

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blood loss, and with a decreased mean arterial pressure (MAP) from baseline. Recommend that the surgeons obtain an ophthalmology consultation immediately. Document your conversation. P.499

- Openly express your concern about postoperative visual loss (POVL) with the patient and family.
- Reassure the patient that you will get all the "specialists" involved in conjunction with the surgeons.
- Explain to the patient that POVL is very unusual, with the highest complication rate reported in the literature of just 0.2% of all spinal cases.
- Review your anesthetic record for periods of hypotension, documented eye exams, and transfusions. Give your legal department an early notice of incidents and possible medical and legal issues.
- Register the case with the POVL registry (www.asa.closedclaims.org).

Don't:

- Do not accept responsibility for negligence of care.
- Do not change any anesthetic or other patient record ever.

In the future:

- Always discuss possible catastrophic complications in high-risk surgeries.
- Discuss possible eye-related complications with surgeons intraoperatively when doing a procedure on a patient in the prone position if the patient has a decreased MAP or a decreased hematocrit. Suggest that the surgeon stage the procedure and come back a second time. Document your suggestion.
- Discuss POVL complications with the surgeons prior to speaking with the patient.

TAKE HOME POINTS

- Understand the resources available in your practice location with respect to managing perioperative eye injuries. In private practices or smaller hospitals, there may not always be an ophthalmologist immediately available to see the patient in the PACU. In that case, it is usually both reasonable and feasible to apply saline eye drops and patch the eye while awaiting the consultant's input.
- Mention possible eye-related complications in your preanesthetic evaluation, especially when a procedure is to be done on a patient in the prone position or coronary bypass is to be done.
- Tape the patient's eyes shut as early in the procedure as the patient's medical condition allows.
- ***Watch your watch*** ; better still, don't wear a watch (the anesthesia machine states the time).
- Be cognizant of your personal paraphernalia (e.g., stethoscopes, identification badges, pens, etc.).
- Tape the patient's eyes *completely* shut; incomplete closure leads to dehydration of the cornea. P.500
- Consider not placing the syringe on the endotracheal tube (ETT) while intubating; the syringe might be scratching the cornea while you are looking for the vocal cords.
- Place the pulse oximeter on the ring finger instead of the index finger, as patients invariably rub their eyes with the index and middle finger.
- Avoid putting direct pressure on the globe.
- Avoid lubricating ointments like "Lacrilube" that have been sitting in your anesthesia machine for several years-they're great vectors for adenovirus.
- Watch the surgeons carefully; if you suspect that Hibiclens has been administered inadvertently to the patient's eyes, irrigate them immediately with a sterile wash and contact an ophthalmology consultant.
- Check the position of the patient's head and eyes after the patient is placed in the prone position; don't forget to check again after you move the patient into an airplane position.
- Specifically *ask* the patient about ocular symptoms after anesthesia.
- *Never* discount patients' complaints about foreign-body sensations or altered vision.
- When in doubt, seek ophthalmic consultation early.

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Editors: Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.
Title: *Avoiding Common Anesthesia Errors, 1st Edition*

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Be Aware of the Issues and Criteria Pertaining to Discharge of the Postspinal Patient

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It is your first day on rotation in the operating room suite that covers orthopedics, urology, and gynecology cases. At 4 PM, you are paged by the agency nurse who is working in the Post-Anesthesia Care Unit (PACU) that day. She tells you that she collected 400 cc of urine about 2 hours ago via straight catheter from Mr. Johnson (who had a diagnostic knee scope in the middle of the day), but that he still has not been able “to go on his own.” He is getting “antsy” and his family would like to get back to the Eastern Shore before dark. She requests that you come over to sign out the patient. What do you do?

Because regional anesthesia has grown in popularity and allows surgical facilities to discharge patients quickly, more surgical procedures are being performed using peripheral nerve blocks as well as epidural or spinal anesthesia. Spinal anesthesia is less technically difficult and has a faster onset time than epidural techniques; however, a surprising number of complications exist with spinal anesthetics. As these procedures increase in number, the astute clinician must be able to manage complications that arise and be aware of the specific PACU discharge criteria related to spinal anesthesia.

COMPLICATIONS

In general, light sedation is often administered with spinal anesthetics, and such administration decreases the incidence of postoperative respiratory complications as compared to the use of general anesthetics. In one study, nearly two thirds of complications related to general anesthetic use involved postoperative respiratory events. Conversely, the most common complications associated with spinal anesthetics are postdural puncture headache (PDPH) and backache.

POSTDURAL PUNCTURE HEADACHES

PDPH is the most common complication in patients receiving spinal anesthesia, with incidences as high as 25% reported in some studies. Small-bore, 25-gauge, pencil-point needles decrease the incidence of PDPH. The greatest incidence of PDPH occurs when large-bore epidural needles accidentally puncture the dura. PDPH is believed to result from a loss of cerebrospinal fluid, which places traction on the meninges and, thus, on the brain. Patients P.502
with a PDPH typically present with orthostatic, position-related headaches. Valsalva maneuvers, such as coughing and sneezing, exacerbate the head pain. The patients may complain of nausea, vomiting, dizziness, visual and auditory changes, paresthesias, weakness, and cranial-nerve palsies. The pain typically resolves in several days. Conservative measures, such as rest, hydration, and caffeine can alleviate symptoms. More long-standing symptoms can be managed through an autologous epidural blood patch, with success rates approaching 90% for the first blood patch and up to 98% with repeat patches.

BACKACHE

In general, the delivery of anesthesia of any kind is associated with postoperative backache, but the incidence is higher following spinal anesthesia. Epidurals have been associated with a higher incidence and a longer duration of backache than have spinal blocks. The etiology of such backache is unknown, but local anesthetic irritation, direct trauma, and ligamentous strain related to muscle relaxation have been postulated.

SYSTEMIC TOXICITY

Although systemic toxicity is continually a concern with epidural anesthesia, systemic toxicity does not occur with spinal anesthesia, because drug doses are generally considered too low to cause toxic reactions, even if injected intravenously.

TOTAL SPINAL

Total or high spinals occur when excessive amounts of medication are injected intrathecally. The anesthetic spreads to the entire spinal cord and the brain stem, resulting in total sympathetic blockade with resultant bradycardia, hypotension, and respiratory depression. Ponhold and Vicenzi found that the patient's operative position helps determine the incidence of bradycardia and, thus, of sympathetic blockade. Patients in the Trendelenburg position were more likely to develop bradycardia than were those in the supine or hammock position. Respiratory arrest can also occur as the primary and accessory respiratory muscles are paralyzed, and the lower-respiratory brain centers are affected. Adequate supportive measures with antimuscarinics, vasopressors, intravenous fluids, and ventilation can effectively treat high spinal blocks.

NEUROLOGIC INJURY

Neurologic injury is a feared complication of both spinal and epidural anesthesia. The incidence of serious injury to the cauda equina roots and paraplegia is extremely low, but persistent paresthesias and limited motor
weakness are the most common injuries among neurologic complications. Barash and Stoelting state that, “Injury may result from direct needle trauma to P.503
the spinal cord or spinal nerves, from spinal cord ischemia, from accidental injection of neurotoxic drugs or chemicals, from introduction of bacteria into the subarachnoid or epidural space, or very rarely from epidural hematoma.”

Transient neurologic symptoms (TNS) can result from clinical administration of high concentrations of local anesthetics, causing pain to radiate down the back and lower extremities. The pain occurs within 24 hours after spinal anesthesia and generally lasts 48 hours. As the number of procedures done using spinal anesthetics has increased over the past decade, so has awareness and concern about TNS. Hyperbaric lidocaine is a major culprit in causing TNS, and the lithotomy position and knee arthroscopy position increase the rate of TNS. Although the dose of a solution does not appear to affect the incidence of TNS, anesthesiologists believe that hyperbaric solutions surrounding neurologic components pool in dependent locations and fail to mix adequately with cerebrospinal fluid, causing cauda equina syndrome. Anesthesiologists speculate that neural stretching compromises blood flow and

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can exacerbate sciatic-type symptoms. Pollack et al. compared various concentrations of lidocaine and found no difference in their associated incidences of TNS. TNS is, however, a rare complication. In a retrospective study examining the risk of neurologic symptoms in 4,767 subjects, Horlocker et al. showed the safety of spinal anesthesia.

ANTICOAGULATION

Spinal hematoma and other central nervous system (CNS) hematomas are potentially disastrous complications of spinal anesthesia in patients receiving anticoagulation therapy, which is commonly administered to various patients, including those with atrial fibrillation and hypercoagulable disorders; those with orthopedic fractures; and those who are bedridden and debilitated. Antiplatelet-drug use and traumatic or prolonged needle placement also can lead to CNS hematomas. The American Society of Regional Anesthesia and Pain Management has developed practice guidelines for treating neuroaxial anesthesia. The extremely low incidence of neuroaxial hematomas precludes prospective randomized studies. Using these guidelines and understanding the relevant pharmacology permits well-informed clinical decision-making (http://www.asra.com/publications/consensus-statements-2.html).

DISCHARGE CRITERIA

After having general or regional anesthesia, a patient can be evaluated for discharge from the Post-Anesthesia Recovery Unit (PACU), using the Aldrete score, which assigns scores of 0, 1, or 2 in five categories, including extremity movement; the ability to breathe deeply and cough; orientation and consciousness; oxygen saturation; and systemic blood pressure (Table 115.1).

Typically, a score of 9 or greater is required for discharge from the PACU. Peripheral perfusion, including heart rate and blood pressure, should be stable for at least 15 minutes before discharge. Hypothermia can persist, but shivering should resolve. Patients should also understand that their activities, including driving, are restricted postoperatively for at least 24 hours.

TABLE 115.1 ALDRETE SCORE

CATEGORY	PARAMETERS	SCORE
Activity	Moves four extremities on command	2
	Moves two extremities on command	1
	Moves no extremities on command	0
Breathing	Breathes deeply and coughs deeply	2
	Dyspnea	1
	Apnea	0
Circulation	Blood pressure within 20% of preanesthetic level	2
	Blood pressure within 20% to 49% of preanesthetic level	1
	Blood pressure within 50% of preanesthetic level	0
Consciousness	Fully awake	2
	Arousable	1
	Nonresponsive	0
Oxygen saturation	>92% on room air	2
	Supplemental oxygen to maintain saturation >90% on room air	1
	<90% with supplemental oxygen	0

Adapted from Aldrete JA. The post anaesthesia recovery score revisited. *J Clin Anesth* . 1995;7:89-91, with permission.

Regional-anesthetics techniques with decreased parenteral opioid requirements minimize postoperative nausea and vomiting (PONV), pain, and

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somnolence, which are the most common symptoms necessitating PACU admission following general anesthesia. Numerous randomized studies have shown that general anesthesia is associated with a high incidence of PONV, sore throat, longer recovery time and higher opioid requirements; whereas spinal anesthesia is associated with a high incidence of pruritis and urinary retention. Postoperative symptoms, such as pain, nausea, and vomiting, generally resolve within 24 hours, but vomiting should resolve before discharge.

Patients receiving neuroaxial anesthesia, including spinals, must be evaluated for the regression of motor, sympathetic, and sensory blockade before ambulating. The clinician can test the sacral nerves (S4-5) for perineal sensation, first-toe proprioception, and pedal-plantar flexion. Before ambulating, residual motor, sympathetic, and sensory blockade should have resolved. Patients should ambulate without dizziness or difficulty before discharge.

Delayed voiding commonly delays discharge from the PACU. In one study, 18% of patients receiving spinal anesthesia had delayed voiding that delayed their discharge. Motor and sensory functions typically return before

sympathetic nerve function, and residual sympathetic blockade to the bladder and urethra can lead to urinary retention. Once the third sacral dermatome blockade ceases, the micturition reflex generally returns. Epinephrine is not recommended for use when administering spinal anesthetics to ambulatory patients, because, although it can increase the duration of the spinal anesthetic, it further compromises the micturition reflex. Avoidance of long-acting spinal and epidural anesthetics allows bladder function to return before bladder distention begins. *Aconsensus does not exist on whether voiding should be a criterion for PACU discharge*. Recent trials have shown that those at low risk for urinary retention do not need to void before discharge, whereas those at high risk for urinary retention should void before discharge. Marshall et al suggested that even patients with a high risk for urinary retention could be discharged home before voiding with specific follow-up if voiding fails to occur in a given time interval. P.505

Jin et al. showed no correlation between drinking and not drinking and its association to nausea and vomiting. In a review, Marshall et al concluded that withholding fluid intake shortens the postoperative recovery period without evidence of adverse effects. The medical staff should be educated that drinking before discharge may not be needed in ambulatory patients.

Amodification of the Aldrete PACU scoring system, termed the Postanesthesia Discharge Scoring System (PADS), "establishes a pattern of routine, repetitive evaluation of patients home readiness, that is likely to contribute to improved outcome." The PADS scoring system lacks requirements for drinking and voiding. Chung recommended initially using the Aldrete score, followed by home-readiness evaluation with the PADS scoring system, which includes categories for vital signs, activity level, nausea and vomiting, pain, and surgical bleeding.

TAKE HOME POINTS

- Use of spinal anesthetics represents a safe, efficient, and inexpensive technique that offers lower postoperative morbidity when compared with general anesthetics for some of the complications of anesthesia; however, use of spinal anesthetics introduces new postoperative issues that must be confronted.
- Spinal anesthetics are ideally suited for use in ambulatory surgical cases, given their short duration and limited complications; however, they also can serve well in chronically debilitated, hospitalized patients.
- *Although all surgical facilities should have established PACU discharge requirements, no standard exists.*
- At minimum, patients should meet the Aldrete scoring system's requirements.
- An anesthesiologist must ultimately decide a patient's postanesthetic disposition, with the ultimate goal of returning the patient to his preanesthetic state. P.506

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Fasting Guidelines for Children Should Be Simple But Not Too Simple

Justin B. Hauser MD

Among the many routine decisions that must be made concerning the preoperative care of pediatric patients, perhaps none is as consistently complicated as the preoperative fasting guidelines. The anesthesiologist must always balance the risks of a full stomach and pulmonary aspiration against the threat of prolonged fasting, dehydration, and hypoglycemia. While making the safest medical decision, the anesthesiologist must also consider the probability of irritable children, disgruntled parents, and a constantly evolving operating room schedule. Large retrospective studies have shown that, although perioperative pulmonary aspiration is more common in children than in adults, it is still rare. Significant morbidity resulting from aspiration is seen even less frequently.

Practices regarding preoperative fasting guidelines vary significantly, as reported in a recent survey of major pediatric hospitals. Most centers agreed on allowing clear fluids for as many as 2 hours and breast milk for as many as 4 hours before a planned procedure. However, there was less concurrence regarding the appropriate fasting period for infant formula and solids. Some institutions restricted intake of formula in a manner similar to solids. Others recommended a fasting period for formula the duration of which was between the durations recommended for clear liquids and solids and was similar to the duration recommended for breast milk. In addition, certain policies dictated fasting intervals based on the patient's age whereas others placed definitive limits on volumes of ingestible items. These discrepancies, along with frequently unclear policies within individual institutions, have created a great deal of confusion for both parents and health care providers.

In 1999, in an effort to establish clear recommendations regarding nothing by mouth (NPO) status, the American Society of Anesthesiologists (ASA) published Practice Guidelines for Preoperative Fasting and the Use of Pharmacologic Agents to Reduce the Risk of Pulmonary Aspiration. These guidelines provide clinical recommendations based on a review of current literature, a large survey of physicians, and a consensus among anesthesiologists within the Task Force on Preoperative Fasting. As part of their recommendations, the task force emphasizes the importance of a preoperative evaluation of each patient and recognition of comorbidities that may increase the risk of pulmonary aspiration. Gastroesophageal reflux, ileus, and bowel obstruction
are prevalent among the pediatric surgical population and certainly warrant individualized preoperative fasting orders based on the severity of the patient's condition. P.511

The task force's guidelines are intended for healthy patients who are scheduled for elective procedures and provide appropriate fasting intervals for each type of ingested material. Accordingly, clear liquids may be given as many as 2 hours before a planned procedure. Clear liquids frequently given to pediatric patients include water, ginger ale, popsicles without fruit chunks, fruit juices without pulp, and oral rehydration solutions. For breast milk, the recommended fasting interval is 4 hours. Regarding infant formula, the task force recommends a fasting interval of 6 hours. Similar to that for formula, the recommended fast for solids and milk other than breast milk is 6 hours before the procedure.

For patients requiring preoperative or daily medications, decisions must be made on an individual basis. Pediatric patients generally can safely be given their medications on the morning of surgery with a small amount of water. A prospective study in pediatric surgical patients showed no significant change in gastric pH or residual volume in those given oral midazolam mixed with 5 mL of water. Pediatric anesthesiologists generally accept the risk associated with ingestion of premedications, such as midazolam, just before induction of anesthesia due to the other desirable attributes of these drugs. Special consideration must be given to medications that involve the administration of a larger volume of liquid. The Task Force on Preoperative Fasting does not recommend the routine preoperative use of gastrointestinal stimulants, histamine-2 receptor antagonists, antacids, or antiemetics due to insufficient evidence suggesting a role of these agents in reducing the risk of pulmonary aspiration. The guidelines instead emphasize the importance of the fasting intervals for the specific types of ingested liquids and solids.

These NPO recommendations are most easily remembered as the “2, 4, 6” rule (Table 116.1): 2 hours for clear liquids, 4 hours for breast milk, and
6 hours for formula, milk other than breast milk, and solids. Another way to simplify these guidelines is to provide two sets of standardized NPO orders, one for all pediatric patients with procedures scheduled to occur before noon and another for those with procedures scheduled to occur after noon (Table 116.2). For patients having procedures in the morning, no solids, milk other than breast milk, or formula are permitted after midnight. Breast milk is permitted until 3 AM, and clear liquids are permitted until 5 AM. For children scheduled for procedures after noon, no solids, milk other than breast milk, or formula are permitted after midnight. However, breast milk may be given until 8 AM, and these children may have clear liquids until 10 AM. Undoubtedly, conflicts with operating-rooms schedules due to a child's NPO status will forever persist. Nonetheless, these standardized orders are aimed at ensuring that pediatric patients may be safely anesthetized at the time of their procedure while minimizing the number of irritable children and frustrated parents in the surgical waiting room. As with every clinical decision, deciding on the appropriate fasting interval must include consideration of each patient's medical condition and the urgency of the planned procedure. Any significant divergence from these guidelines requires that the anesthesiologist consider the probability of a full stomach and the need for techniques to prevent pulmonary aspiration. P.512

TABLE 116.1 NPO INTERVALS RECOMMENDED BY TASK FORCE ON PREOPERATIVE FASTING

TWO HOURS BEFORE PROCEDURE	FOUR HOURS BEFORE PROCEDURE	EIGHT HOURS BEFORE PROCEDURE
Water, ginger ale	Breast milk	Formula
Fruit juices (no pulp)	-	Milk other than breast milk
Oral rehdyration solutions	-	Solids

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Popsicles	-	-

TABLE 116.2 PROPOSED NPO TIMES FOR PREPRINTED NPO ORDERS

AM PROCEDURE	PM PROCEDURE
Midnight—solids, formula, milk other than breast milk	Midnight—solids, formula, milk other than breast milk
3 AM—breast milk	8 AM—breast milk
5 AM—clear liquids	10 AM—clear liquids

TAKE HOME POINTS

- Implementing reasonable NPO guidelines for children involves considering many factors, including the fact that aspiration of gastric contents is rare but, potentially, very serious.
- Most centers are agreed on guidelines for clear liquids and breast milk.
- The ASA published Practice Guidelines in 1999 to provide clinical guidelines for anesthesia providers. This document stresses awareness of factors that can increase risk of aspiration of gastric contents. These factors, gastroesophageal reflux, ileus, and bowel obstruction, are prevalent among the pediatric surgical population.
- The Task Force on Preoperative Fasting does not recommend the routine preoperative use of gastrointestinal stimulants, histamine-2 receptor antagonists, antacids, or antiemetics.
- NPO guidelines for children are most easily remembered as the “2,4,6” rule.
- One way to simplify the NPO guidelines is to provide two sets of standardized NPO orders, one for all pediatric patients with procedures scheduled to occur before noon and another for those with procedures scheduled to occur after noon.

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Do Not Automatically Cancel the Procedure if the Child has a Runny Nose

Ann G. Bailey MD

One of the most common dilemmas for the pediatric anesthesiologist is deciding whether to anesthetize a child who has just recovered from or currently has an upper-respiratory-tract infection (URI). This situation is challenging even for experienced providers who have lots of judgment and experience in this area—junior anesthesiologists should not be at all hesitant about seeking help and advice.

The first problem is to determine if the child has an URI at all. When is a runny nose secondary to an URI, and when is it due to allergic rhinitis? Remember that the symptoms that the parents are most likely to tell you about ("he's had a runny nose" or "a little bit of a cold") can be very similar to those of other illnesses or allergies. The most reliable way to tell the difference between an URI and allergic rhinitis or other benign entities is to get further history from the parents. If they relate that the child developed symptoms while in daycare or that he has sick siblings, it is probably an URI. Also, you should suspect that the child has an URI if the parents tell you that their child's symptoms are new and not a routine occurrence. There are, however, some children who "keep a cold." In one study, the probability of having an URI in a child younger than 3 years, in daycare, with parents who smoke in crowded house was 0.61 in any given 2-week period! Signs that are associated with an URI include purulent rhinorrhea, fever, sore throat, productive cough, and other lower-tract signs, such as wheezing or rhonchi. Allergic rhinitis is not associated with fever or productive cough, rhinorrhea is usually clear, and it is often associated with a history of atopy.

The second problem is determining whether the URI will affect the child's anesthetic course. Early studies demonstrated that what is thought to be purely an URI might also involve the lower airways and be associated with decreased pulmonary function, and increased reactivity for up to 6 weeks after the infection. In viral airway infections, much of the bronchial hyperreactivity is vagally mediated. Some viruses are thought to produce a viral neuraminidase that decreases the function of M_2 muscarinic receptors. As a result, there is increased release of acetylcholine in virus-infected airways. There may also be a decrease in the activity of airway neutral endopeptidase, an enzyme responsible for inactivating tachykinins. These effects render the lower airways more susceptible to smooth-muscle contraction, thus increasing airway reactivity. P.515

The typical URI also is associated with more airway secretions. The combination of increased secretions with increased reactivity leads to the adverse events often described in clinical studies of anesthetized children with URIs: coughing, breath-holding, laryngospasm, bronchospasm, and episodes of desaturation.

The next question is which procedures should be cancelled to avoid serious complications in the child with a URI? Children who present for an elective procedure with moderate-to-severe signs and symptoms of fever, myalgias, lassitude, wheezing, or rhonchi are easily discerned as being at high risk, and their procedures should be delayed. However, those who present with mild or recent symptoms are also at risk for adverse events. One study evaluated more than 1,000 children scheduled for elective surgery who were either well, had a recent URI (within 4 weeks), or had an active URI. The incidence of adverse respiratory events was greater in the children with recent or active URIs than in the children who were well. Independent risk factors for respiratory complications in children with an URI were copious secretions, presence of an endotracheal tube (ETT) in children less than 5 years of age, history of premature birth, nasal congestion, paternal smoking, history of reactive airway disease, and airway surgery. The more serious complications of laryngospasm and bronchospasm were not different among the groups. A second study compared the cases of 1,280 children with preoperative URIs with those of 20,876 children without preoperative URIs and demonstrated that there was an 11-fold increase in the risk of a respiratory complication if the child with an URI required intubation. The first study also confirmed that an ETT in the presence of an URI contributed to more adverse respiratory events, although these events were primarily breathholding.

Does the increased risk of adverse respiratory events mean that, in every case in which a child has a recent or current URI, the procedure should be delayed for at least 4 weeks (6 weeks for lower-tract symptoms)? Absolutely not, since the vast majority of children with an URI do well upon having an anesthetic. Identifying children who are at higher risk for adverse outcomes and delaying those surgeries is reasonable. The child who has a runny nose for 3 days, who is otherwise well, and who is scheduled for an adenoidectomy will probably do fine. One can improve the likelihood of an uneventful anesthetic by tailoring it to minimize reactivity (See Take Home Points). Sevoflurane, with its mild airway pungency, causes fewer respiratory events when used as a maintenance agent in children with an URI. Additionally, the use of a laryngeal mask airway (LMA) or facemask, when feasible, decreases complications. Administering a dose of atropine (10 mcg/kg intravenously [IV]) or glycopyrrolate (5 to 10 mcg/kg IV), to serve both as an antisialogue and as a vagolytic agent, may decrease risk. Giving a bronchodilator prophylactically to prevent wheezing has not been shown to be effective. In a younger child who will require intubation, one should anticipate a higher incidence of postoperative croup and should consider giving dexamethasone (0.3 to 0.5 mg/kg IV; maximum dose, 10 mg) intraoperatively. P.516

With such a small amount of scientific evidence regarding the risks of anesthesia for the child with an URI, clinical experience and common sense must help to determine our course of action. Charles Cote, MD, summarized it best in an editorial about this dilemma: "We are left to our best clinical judgment about an individual patient undergoing a specific procedure for a specific duration of time by a specific surgeon that requires intubation that may or may not involve admission to the hospital who also has or has had a recent URI."

TAKE HOME POINTS

Strategies for minimizing risks when the patient has an URI include the following:

- Use a LMA or mask instead of ETT, if possible;
- Administer atropine or glycopyrrolate, preoperatively or intraoperatively;
- Use sevoflurane rather than desflurane or isoflurane;
- Use propofol rather than thiopental for an IV induction;
- Have albuterol available, but do not use it prophylactically; and
- Consider dexamethasone in small children who require intubation.

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Put Some Thought Into Which Child, and with Which Drug, You Premedicate

M. Concetta DeCaria MD

The amount of literature on the subject of preoperative medication can be overwhelming. This may be because there is no “ideal” medication available that is free from unwanted side effects. When deciding whether to administer medication to a child before surgery, one should consider, first, whether that child is a candidate for such medication, and, secondly, which medication would be most appropriate for that child. There is no perfect formula for this, but one general rule is to consider both the comfort and the safety of the child when making these choices. This is yet another situation where much can be learned from observing the senior pediatric anesthesia providers. Experienced pediatric anesthesiologists start like the rest of us—with the general rule that both the safety and the comfort of the child must be considered. But they then know how to use the available agents to take an anxious, upset child and transition smoothly to an anesthetized patient.

When choosing whether to give medications preoperatively to a child, it is imperative to consider the consequences of doing so. The advantages of administering medication before surgery are well described and include the dissipation of preoperative anxiety, for both the child and the parent. Preoperative medication can facilitate separation from the parents and can smooth induction in the operating room. Of course, there are also disadvantages to giving medication to children preoperatively, which can include anything from inconvenient side effects, such as prolonged recovery, to dangerous side effects, like oxygen desaturation and loss of airway control.

The perfect preoperative medication would have the following characteristics:

- It would have a relatively rapid and reliable onset;
- It would be free of side effects, such as nausea, the risk for excessive sedation, increased oral secretions, and discomfort from administration;
- It would readily be accepted by the child and would have a route of administration that is not traumatic;
- It would last long enough to accommodate operating-room delays but not prolong recovery or delay discharge; and
- It would not require constant nursing supervision due to side effects.

For outpatient pediatric patients about to have anesthesia, a medication that does not require intravenous (IV) administration would be most useful. One of a child's worst fears associated with coming to the hospital is being stuck by needles. It logically follows that it would be best to avoid P.518 administering drugs via the intramuscular route. Other options then include giving a medication by the oral, rectal, or transmucosal route. The most commonly sited classes of medications used for preoperative purposes are hypnotics, opiods, N-methyl-D-aspartate (NMDA) receptor antagonists, and alpha-2 agonists. As a general rule, most children under approximately 10 months of age do not require sedation preoperatively because they have yet to develop separation anxiety.

HYPNOTICS

The medication used most frequently preoperatively in pediatrics is midazolam, largely due to its safety profile, reliability, anxiolytic effect, ability to be given orally, and its effect on antegrade recall. While it is possible to administer midazolam by virtually any route, in the pediatric population, the oral route is most commonly used.

Acommon and effective dose for oral administration is 0.5 mg/kg. When given intranasally, it has been shown that a dose of 0.2 to 0.3 mg/kg has been helpful in reducing preoperative anxiety. Another efficient method of administration is sublingual (0.2 mg/kg), which works better for older children who can comply with this technique. Rectal midazolam (0.5 mg/kg to 1 mg/kg) is anxiolytic, but approximately 20% of patients who receive midazolam rectally develop hiccups.

Compared to other benzodiazepines, midazolam has a more rapid onset and shorter duration of action. While relatively free of unwanted side effects, oral midazolam does have one problem. It has a very bitter aftertaste, and many children will refuse to finish their doses if they have gotten a little taste of it. Many flavored syrups, the most common of which is cherry, have been used to mask the taste. Encourage your patients to take it all down at once and not to “sip” it. The period between oral administration and onset is 10 to 15 minutes.

For patients who refuse to take the medication orally, it can be given intranasally. Intranasal midazolam causes a burning sensation, and, when oral and intranasal routes of administration were compared in one study, patients preferred oral administration. For short procedures, orally administered midazolam can prolong recovery and delay discharge.

Barbituates have also been historically used for preoperative medication in the pediatric population. The most common of these is the shorter-acting pentobarbital, which can be given safely by both the oral and rectal routes. It can also be given intramuscularly, but this has been associated with prolonged pain at the injection site. The dose of pentobarbital is 2 to 4 mg/kg. It is best given rectally in children younger than 3 years of age. Given orally, it can take from 1 to 1.5 hours to become effective, which can be an obvious drawback to its use. We do not use barbituates as preoperative medication in P.519 our practice.

OPIOIDS

Narcotics, such as sufentanil and fentanyl, have been studied for the purposes of preoperative medication. Problems with the routine usage of opioids include respiratory depression, decreased oxygen saturation, delay in the tolerance of oral intake postoperatively, dysphoria, nausea, and vomiting. Routes of administration are broad and include oral, nasal, rectal, intravenous, and intramuscular administration. In some cases, the transmucosal route (fentanyl oralet) has been used.

When given intranasally, sufentanil can be given in increments of 2 mcg/kg. Compared with the intranasal administration of midazolam, intranasal administration of sufentanil appears to be less unpleasant. However, its administration has been shown to result in an increased difficulty in ventilation in

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approximately one third of patients, due to chest-wall rigidity.

KETAMINE

An NMDA-receptor antagonist, ketamine, can be used for the induction and maintenance of anesthesia. As a preoperative medication, it can be given intramuscularly, orally, rectally, nasally, and intravenously. Advantages of ketamine are good anxiolysis, facilitation of mask induction, and sedation. Potential disadvantages are emergence delirium, increased salivation, and, in some studies, delayed discharge from the postanesthesia care units.

An effective intranasal dose of ketamine for preoperative sedation is 6 mg/kg, with an onset time of approximately 20 to 40 minutes. A rectal dose of ketamine (5 to 10 mg/kg) has been shown to provide adequate sedation within 7 to 30 minutes after administration. Ketamine is given orally in doses from 3 to 6 mg/kg with good effect within 20 to 25 minutes. Notably, there seems to be an absence of emergence delirium associated with the oral administration of ketamine. The ketamine "dart" is still used in some special circumstances. Usually 1 to 2 mg/kg is sufficient, although some use a true induction dose of 4 mg/kg. The addition of glycopyrolate or atropine is recommended.

CLONIDINE

Clonidine, an alpha-2 receptor agonist, has been shown to have many desirable effects, including perioperative sedation, hemodynamic stability, reduced anesthetic requirements, and even postoperative analgesia. The use of clonidine in adult patients is well described. Interest in pediatric use is growing. At doses of 4 mcg/kg, oral clonidine has been shown to provide sedation and analgesia and to facilitate separation from parents. It requires administration 60 minutes before induction when given orally. Determining an optimum dose and providing other safe routes of administration, such as intranasal administration, may increase preoperative use of clonidine.

CHILDREN WITH AN IV LINE

If a child has an IV line, by all means use it for preoperative medication. Remember, with IV administration, doses required will be much smaller and the period between administration and onset will be much shorter.

TAKE HOME POINTS

- Choose which patients need preoperative medication: Determine whether the patient has had surgery before; whether the patient appear to be anxious; and whether the patient will return for repeated procedures.
- Consider which medication would best suit the needs of and be least dangerous to the patient: Determine whether the patient has a history of a difficult airway, breathing problems, or paradoxical reactions to certain sedative medications.
- Consider using methods other than preoperative medication to relieve a child's anxiety. Such methods may include familiarizing the child with the operating room before the date of surgery, when possible, and demonstrating the mask induction on a stuffed animal. Consider allowing a parent to accompany the child to the operating room, realizing that sometimes this creates more anxiety, depending on the child and the parent.
- Keep the safety and personal needs of the child at the forefront of your mind when considering preoperative medication.

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Parents are Present in the Operating Room by Invitation of the Anesthesiologist Only

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Surgery can be a time of *intense* psychological stress, for both the child and the parents, and the stress comes from several sources. First, there is often a lot of anticipatory anxiety leading up to arrival at the operating room (OR). Second, a significant percentage of children may experience anxiety when separated from their parents. Finally, induction can be one of the most challenging times of the perioperative period for the child. The anesthesia provider's job is to minimize all of these stresses for both the parent and the child and still accomplish a safe and expeditious induction.

A number of different interventions, which are discussed elsewhere (see Chapter 118), are useful to reduce preoperative anxiety. These interventions include play therapy with distraction techniques and pharmacologic interventions. For relieving separation anxiety for both the patient and the parent as well as for increasing the patient's compliance in the OR, it has become common practice in many centers to allow one parent to accompany the child to the operating suite. The decision to allow parents into the operating room is still controversial—there are advantages and disadvantages. The obvious benefit of parental presence during induction is that it eliminates the need to separate the child from the parent while the child is still awake. In some cases, parental presence in the OR may eliminate the need for preoperative medication of the child, which can be particularly useful when the risks of using such medication may outweigh the benefits. A parent's presence in the OR decreases the child's fears relating to the unfamiliar OR environment. In both pre-school and school-age children, watching the parent dress up in the gown, mask, and cap can be a humorous distraction. It can be comforting for the child to have his parent with him while being introduced to the unfamiliar OR environment and to sit in the parent's lap until induction is complete.

There are also several potential drawbacks to parental presence at induction of anesthesia. The presence of an anxious parent can actually increase the anxiety of the child, even at very young ages. During the preoperative interview, the anxiety level of the parents must be assessed and taken into account when deciding whether a parent should be invited to accompany
the child. A parent should not be allowed into the operating suite solely to calm his or her own fears. It is better to calm the parent's fears by engaging them P.522
in an open and relaxed discussion about the procedure and the risks and benefits of anesthesia.

The presence of parents in the OR depends, to a certain extent, on the institution's culture. At some hospitals, a parent almost always comes into the OR, with very few exceptions, whereas, at other hospitals, parents quite rarely enter the OR. Regardless of what the local culture is, the final decision of whether to invite the parent into the OR for induction is the anesthesiologist's decision. In making this decision, the anesthesiologist must balance the needs of the parents, patient, OR staff, and anesthesia staff. Most experienced anesthesiologists evaluate each family on a case-by-case basis in the preoperative area, taking into account the child's age, activity level, and overall temperament as well as the baseline anxiety level of the parent. You should consider your options carefully before inviting a parent to enter the OR. Parents that are very upset or hostile are best not brought back to the OR. Non-English speaking parents are generally not good candidates to be present at induction. Generally, only one parent is invited to enter the OR; virtually all experienced anesthesiologists feel that having more than one family member present is a mistake. Finally, in certain emergent or trauma cases, having a parent present might not be appropriate. Once a decision is made, it should be clearly and openly communicated to the parent, with a focus on the child's safety as the ultimate goal.

The anesthesiologist is responsible for informing the parent who is coming into the OR about what to expect. Anesthesia residents and nurse anesthetist students should expect that the majority of the parent-doctor interaction in the OR will probably be done by the senior anesthesia provider. The parent should be told that the mask may upset the child, the child may thrash briefly and appear to be disoriented, the child's eyes may roll back as the child goes to sleep, and the child's breathing may become noisy. There are different styles of doing this—the authors prefer to “brief” the parents as the induction proceeds. The important thing is that the parent needs to be assured that these events are normal. The parent also needs to know that he will be escorted out of the OR as soon as the child is asleep. Occasionally, during a completely routine induction, the child's response to induction may cause a very emotional response in the parent, which can occasionally lead to a vasovagal reaction. For this reason, the parent should *always* be seated during the induction of the child.

There should be a designated OR staff member (not the anesthesiologist!) to escort the parent out of the OR immediately after induction and to be available to comfort and reassure the parent if needed. Parents are sometimes uncertain when to leave the OR, particularly in cases in which the
child's eyes remain open after induction. The parent may wish to confirm that the child is “asleep” or to ask questions. A simple signal that cues both the P.523
parent as well as the designated OR staff is extremely helpful in keeping everyone informed in the steps of the process. An example would be “Okay, Mom, she is asleep now. You can give her a kiss. The nurse will show you back to the waiting area.”

For hospitals that offer parental presence at induction, an educated surgical staff is vital. It is very important that the OR staff be clear that the anesthesiologist makes the decision on whether the parent may enter the OR. Staff should avoid inadvertently misleading parents by suggesting that they are welcome to be present for induction before the anesthesiologist has made that determination. It is important to establish a consistent, hospital-wide policy that is well known by surgeons and their staff and that can be communicated early to parents during their child's preoperative workup in the surgery clinic. In this way, there are no unrealistic expectations on the day of surgery. At some institutions, no parent is allowed to accompany a child younger than 10 to 12 months of age. The rationale is that children younger than 10 to 12 months of age have not developed stranger anxiety and can separate from their parents relatively easily.

There are several nonpharmacologic interventions that can be adjuncts or alternatives to parental presence at induction. The effectiveness of these options is determined primarily by the child's age. For some children, particularly those under 4 years of age, bringing both the child's mother and the child's teddy into the OR may provide extra comfort, so the rule about no siblings is suspended if the additional “family member” is a small stuffed animal (the mother takes the sleep buddy with her when she goes out of the room). For school-age children, allowing the child to choose among various scents for their face mask or printing out an electrocardiogram strip once monitors are placed makes the experience more intriguing. Offering a child the opportunity to be the line leader walking into the operating suite may allow them to display independence and is also a good way to assess the child's ability to separate successfully from parents. Providing adolescents with options regarding the procedure gives them some control and reduces anxiety.

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One example would be to give them the option of an IV or an inhalational induction when either method would be appropriate.

An interesting question is whether the parent should enter the OR when pharmacologic agents have been successfully administered to the child preoperatively. Studies have not shown any clear benefit to the child in combining parental presence in the OR with administration of oral midazolam. Oral midazolam, at a dose of 0.25 to 0.5 mg/kg (maximum dose, 20 mg), results in less patient anxiety than does parental presence in the OR without any preoperative medication.

To summarize, parental presence at induction of anesthesia reduces the child's anxiety level at induction and avoids having to separate the parent and child in the preoperative area. Although being in the OR reduces anxiety and increases satisfaction in parents, these are not the primary reasons to have them in the OR. For a successful experience, the anesthesiologist should make plans clear to the parents as soon as possible and the OR staff should be organized and skilled in having a parent present at a child's induction. Ultimately, the anesthesiologist's goal is to ensure a perioperative experience for the pediatric patient that is safe and not psychologically traumatic. P.524

TAKE HOME POINTS

- The anesthesiologist makes the final decision on whether to have a parent present at induction—don't do it if you are inexperienced or uncomfortable dealing with parents or if the case has high clinical acuity.
- Watch the senior pediatric anesthesiologists—they all have certain tricks and techniques. Borrow freely the techniques you think will work for you.
- The needs of the child are more important than the needs of the parent.
- Be *very* calm when you have a parent in the OR!

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Pay Scrupulous Attention to Eliminating Air Bubbles in Pediatric Intravenous Tubing

Erica P. Lin MD

Fetal intracardiac and extracardiac shunts that persist after birth provide a natural setup for paradoxic emboli. Right-to-left shunting allows blood to bypass the inherent filtering action of the lungs, and emboli arising from the venous system can pass directly into the systemic arterial circulation. *Remember always that even a small air embolus can have devastating results.*

ANATOMY AND PHYSIOLOGY

In utero, blood flows in a parallel circuit with respect to the right and left ventricles. The highest oxygen content is in blood returning from the placenta via the umbilical vein. The ductus venosus is the first shunt encountered and connects the umbilical vein with the inferior vena cava, allowing roughly half of this oxygen-rich blood to be diverted away from the liver. Within the right atrium, a pressure gradient enables a significant portion of this oxygenated blood to be preferentially streamed across the foramen ovale to the left atrium. This allows the blood with the most oxygen content to be efficiently delivered, via the left ventricle and aorta, to the coronary and cerebral circulations. Right-ventricular outflow consists of a mixture of oxygenated blood from umbilical veins and desaturated blood from the superior and inferior vena cavi. Only 10% of right-ventricular blood actually passes into the pulmonary circuit because of high vascular resistance (the combined result of fluid-filled fetal lungs and medial-muscle hypertrophy in the small pulmonary arterioles). The remaining 90% of right-ventricular output is shunted away from the lungs through the ductus arteriosus and into the descending aorta, where it supplies the lower body or returns to the placenta.

The transition between fetal and neonatal circulation occurs primarily in response to changes in resistance throughout the circulatory system. The parallel circulation of the fetus is converted to a series system. Clamping of the umbilical cord eliminates the low-resistance placenta, and systemic vascular resistance increases. Without venous return from the placenta, right-atrial pressure decreases. As breathing is initiated, pulmonary vascular resistance falls dramatically, resulting in an increase in blood flow to the pulmonary circulation. In addition, higher pulmonary arterial oxygen tension (compared to the relatively hypoxemic fetal environment) contributes to lower pulmonary

vascular resistance. Due to remodeling of the pulmonary vasculature, a continued gradual reduction in pulmonary vascular resistance occurs during the first months of life. More blood flow to the lungs translates to greater pulmonary venous return to the left atrium. Elevated left pressure relative to the right atrium facilitates closure of the flap-like foramen ovale. Higher arterial oxygen concentration stimulates closure of the ductus arteriosus. Removal of the placenta also lowers circulating prostaglandin levels, which further facilitates ductal closure. The ductus arteriosus is functionally closed within the first 24 hours after birth; however, permanent closure requires thrombosis and fibrosis, which can take several months. P.526

Anatomic closure of these alternate pathways does not occur immediately after birth, meaning that the shunts reopen in response to physiologic stimulation. For example, premature infants demonstrate less sensitivity to the effects of oxygen on ductus-arteriosus closure. Similarly, the existence and, often, the pharmacologic maintenance of these shunts are necessary for the survival of infants with congenital heart disease until surgical correction is possible. The drawback, however, is that these shunts provide a route for paradoxic emboli. Multiple case reports have described the source of emboli as varying from air bubbles entrained in intravenous lines to thrombus dislodgement from the ductus venosus, umbilical vessels, or renal veins. Such emboli can be catastrophic, resulting in permanent neurologic damage, myocardial infarction, and even death.

PREVENTION

Simple measures can be taken by health care providers to minimize the risk of iatrogenic emboli. While these maneuvers are second nature for practicing pediatric anesthesia providers, the beginning or occasional pediatric provider should allow for extra time for “de-airing the lines” when setting up the room. These preparations should not be rushed.

Intravenous catheters are an especially frequent culprit. Disconnection of the line, fracture of the hub, or a break in the tubing can allow air entry. Medication administration through peripheral veins can also introduce air. Wald et al. demonstrated in their experimental model that an average volume of 0.02 mL of air is delivered per injection, even when careful attention is paid to remove all air before mounting syringes onto connecting lines. Special care should always be taken when preparing infusions. Ideally, fluids should not be prepared in a cold room and then brought into a warm environment. As the temperature is raised, gas bubbles may form because of reduced solubility and the size of existing bubbles will increase. This same principle applies to the infusion of fluids through a warming system. As the solution passes through the warmer, microbubbles can form and tend to coalesce into larger bubbles that can be passed to the patient. Studies have shown that higher

rates of infusion through warmers correspond with decreased amounts of gas escaping from solution. This is presumably due to the shorter amount of time the solution spends in the warming tubing, which, in turn, limits the time for gas to diffuse out of solution. Using distally placed bubble traps or a stopcock for aspiration of any collected air has been suggested. Efforts should also be made to remove air from intravenous fluid bags prior to infusion, as there have been case reports of massive air embolism, especially when such bags are used with pressurized infusion devices. P.527

The risk of paradoxic air embolism is greater in newborns with transitional circulation and patients of any age with cardiac lesions that allow right-to-left shunting to occur. However, as many as 25% of adults have a patent foramen ovale, which can allow paradoxic emboli. For that reason, it is imperative that caretakers always be meticulous in preparing all intravenous infusion systems.

TAKE HOME POINTS

- Newborns are at high risk for right to left shunting.
- Up to 25% of adults have a patent foramen ovale.
- IV setups are a significant source of air.

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In Children, Improper Placement, Use, and Maintenance of Vascular Access Can Result in Serious Morbidity and Even Mortality

Angela Kendrick MD

Anesthesia providers must always be diligent in limiting vascular cannulation errors in kids. Serious morbidity or even mortality can result from the placement, the use, or the improper maintenance of vascular access lines. An important resource for tracking information about anesthesia complications is the American Society of Anesthesiologists (ASA) Closed Claim Project (the editors feel it's in the required reading category). The Closed Claims Project allows a retrospective comparison of past legal claims for different categories of injury. This data base includes data on vascular cannulation complications.

COMPLICATIONS ASSOCIATED WITH PERIPHERAL CATHETERS

The most common intravenous (IV) complications reported in the Closed Claim Project include skin sloughing or necrosis, as well as swelling, inflammation, infection, nerve damage, and fasciotomy scars resulting from the treatment of compartment syndromes. Frequently, in cases of compartment syndromes, an extremity was tucked or covered and the peripheral IV was not visually or tactilely monitored. The infiltration therefore was not noted in a timely manner. Peripheral IVs can also be a source of other complications, such as air embolism, fluid overload, and infection.

PERIPHERAL SITE SELECTION

In addition to the usual extremity sites, scalp veins may be selected for use in infants. Often, a branch of the superficial temporal vein is selected. This vein may have many branches helping to form the superficial venous network seen on the infant scalp. Most centers no longer shave the scalp prior to IV insertion. Stroking the vein with one end of the vein occluded by finger pressure may help you establish the direction of flow in the vein; drainage usually is toward the neck. Smaller tourniquets (even rubber bands) may be appropriately used on infants, and angiocath sizes used in infants are typically 22 or 24-gauge.

The saphenous vein deserves special mention as a site “of choice” for IV placement. The vein has a reliable anatomic location, lying just in front of the medial malleolus, and is often palpable, even in pudgy toddlers. P.529

Before line placement, you should wash your own hands with an alcohol based solution prior to donning your gloves. Nonsterile gloves may be used for peripheral IVs, but maximum sterile barrier precautions (caps, masks, gowns, gloves, and drapes for the field) are required for central venous lines.

MAINTENANCE OF LINE

Proper securing of the line with clear, occlusive dressing, tape, a padded arm (or leg) board; and, then, an over wrap of gauze dressing prevents a carefully won site from being dislodged by vigorous thrashing. Be sure to leave the insertion site exposed so that it can be monitored for signs of leaking or infection. Infectious complications occur in as many as 25% of patients with indwelling intravenous catheters and can result in serious morbidity and mortality. If an infant or child arrives in the operating room with a covered IV site and a flush fluid needle is not easily injected into the infant or child, the IV site should be uncovered and inspected for skin infiltration. Heated compresses should not be used to treat an infiltration; serious burns have occurred with their use.

To prevent excessive fluid administration, microdrip delivery devices should be used. For patients weighing less than 10 kg, it is best to use a device with a calibrated delivery chamber (e.g., Buretrol). These devices should have a valve or seal that prevents air from entering the IV tubing once the chamber is empty. Careful flushing of all lines, including stopcocks, also is important to help to prevent air embolus. For very small patients (e.g., premature infants), placing all fluids on a regulated pump and avoiding gravity drips altogether is best.

CENTRAL LINES

Mechanical Complications.

Arterial puncture, hematoma and pneumothorax are the most common complications of central venous catheters. Serious morbidity, such as perforation of the heart with subsequent pericardial tamponade and catheter or wire embolization, have declined in the most recent closed-claims analysis. Hemothorax, hydrothorax, and injury to the carotid or subclavian artery continue to be significant problems following central line insertion. Ultrasound guidance (see Chapter 21) has been shown to reduce the number of mechanical complications, the number of catheter placement failures, and the time required for placement.

Thrombotic Complications.

Catheter related thrombosis occurs at a relatively high rate (around 15%), depending on insertion site, with femoral venous catheters having the highest rate. The risk of blood clot embolism from catheter-related venous thrombosis in this group is still undefined.

P.530

Infectious Complications.

Maximal sterile-barrier precautions have been shown to reduce the rate of catheter related blood-stream infections. Use of chlorhexidine gluconate (2%) for preparation of the skin prior to insertion of central lines in children older than 2 months of age has been shown to reduce line-related infections. There is a theoretic concern about using this preparatory solution in children younger than 2 months of age because of their higher skin permeability.

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Site Selection.

Data are available to suggest optimal sites for CVC in adults (subclavian). The jugular, subclavian, or femoral veins may all be used for central access in children. Preference is really dependent on what sites are available and who is placing the line. Cuffed and tunneled central-venous catheters or peripherally inserted central catheters (PICCS) are preferred for patients requiring prolonged venous access.

Size and Depth.

Central lines are sized in “French units.” The size in French units is roughly equal to the circumference in millimeters of the external surface of the catheter. A size 1 F catheter is 1 mm in circumference and 0.33 mm in diameter. One should select the smallest diameter catheter that will provide the flow rate needed. Infants and small children often take a 3 F or a 5 F catheter. The depth to which the catheter is to be inserted is determined by calculating the length needed so that the tip of the catheter lies at the superior vena cava (SVC) and the right atrial junction (RA). Formulas exist to calculate this depth based on the infant's height or weight. For example, for either the right internal jugular (IJ) or the subclavian approach, for example, length of insertion in cm = height in cm/10 - 1 for children <100 cm in height.

PICC LINES

You may be asked to insert a PICC line or you may want to use one as access during a procedure. PICC lines are designed for long-term access and may have a single or a double lumen. As with other central lines, maximal sterile-barrier precautions apply for the insertion of these lines. PICCs are usually placed via the arm using the Seldinger approach, feeding a stylet-type catheter a measured distance measured in advance to the SCV-RA junction through an angiocatheter guide. The placement is confirmed by a chest x-ray prior to final securing of the catheter to the skin. Ultrasound guidance may facilitate identification of an appropriate peripheral vein, or it may be used to identify a catheter that has angled up in the neck rather than flowing centrally.

MAINTENANCE OF CENTRAL LINES

Flush solution and dressing change protocols vary from center to center and are device dependent. Some use 10 units of heparin per mL of flush, whereas others up to 1000 units per mL of flush. Percutaneously inserted central line catheters used for dialysis require special caution. Dialysis catheters may

have 1000 to 5000 units of heparin in the line. You must draw off the heparin solution prior to using the line; do not flush it to the patient. The incidence of P.531
central line infections may be reduced by using antimicrobial impregnated catheters, selecting the site properly, taking maximal sterile barrier precautions during insertion, not using topical ointment at the insertion site, disinfecting catheter hubs carefully before use, and removing catheters promptly when they are no longer needed.

Arterial Access.

This type of access is indicated for measuring beat-to-beat blood pressure and sampling arterial blood gas. Mechanical complications include injury to the vessel, thrombosis of the vessel, and embolization. Nerve injury can also result from puncture of a nerve or as a result of hematoma formation near the nerve.

Site Selection.

The radial artery is usually the preferred site in children, as in adults; however, other sites may be used. The radial artery is not an “end artery” to the hand because of the collateral circulation offered by the ulnar artery. It is estimated that 10% of patients do not have good collateral flow to the hand. Use a modified Allen test (occlude both the radial and ulnar arteries, then release the ulnar artery and observe whether the hand's color returns to normal within 6 seconds) to identify those without significant collateral flow to the hand. Other vessels used include the dorsalis pedis, the posterior tibialis, the ulnar, and the femoral. Catheters (22- or 24-gauge) are appropriately sized to reflect the smaller size of the target vessel. Thrombosis is estimated to occur in 1% to 4% of cannulations. Complications increase with the use of larger catheters and prolonged use. Distal perfusion, including capillary refill, skin temperature, and skin color, needs to be checked every 8 hours. If poor perfusion is detected, the catheter should be removed immediately and the extremity should be monitored closely for the return of adequate perfusion.

UMBILICAL CATHETERS

Using the umbilical artery results in mechanical complications similar to those of using other arterial sites; such complications include thrombosis, (which can involve the aorta), infection, and hemorrhage. It also carries a risk of organ infarction. The infectious risks for umbilical artery catheters and umbilical vein catheters (despite a high colonization rate) are about the same, around 5%.

Using umbilical artery catheters placed in a high position (above the diaphragm at a T5 level) results in a lower incidence of vascular sequelae than does using those placed in a low position (below the diaphragm and above the aortic bifurcation).

The single umbilical vein extends from the umbilicus to the ductus venosus and through the liver to the inferior vena cava (IVC). The preferred location of the tip of the umbilical venous catheter is usually in the cephalad

portion of the IVC or at the IVC -RA junction. Air may be introduced into the intrahepatic circulation with insertion of an umbilical venous line; however, this P.532
is usually a transient finding and should not be confused with the portal venous air associated with necrotizing enterocolitis.

In general, umbilical or arterial catheters may be left in place for as long as 5 days. Umbilical venous catheters may remain for as long as 14 days. If there is a suspicion of a catheter-related infection, vascular insufficiency, or thrombosis, the catheter should be removed.

TAKE HOME POINTS

- Use of vascular-access devices poses the same or an even greater risk for children as it poses for adults.
- Remember the saphenous and scalp veins when searching for a peripheral IV site.
- Peripheral IV catheters are not changed routinely in children.
- Children can be burned from hot compresses applied to infiltrated IV sites.
- Central-line placement in children requires maximum sterile barrier techniques and can be facilitated by ultrasound guidance.
- Flushing and dressing-change protocols for pediatric central lines are institution and device specific.
- Perfusion distal to an arterial line must be checked every 8 hours.
- The umbilical artery is a unique cannulation site and involves special and potentially serious risks.

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Always Make Sure You See the Intubation and Airway Equipment You Plan to Use and the Equipment You Think You Might Use

Eugene E. Lee MD

Because pediatric patients come in a variety of sizes (from a 500-gm neonate to a 100-kg teenager), the pediatric anesthesia provider is confronted with a variety of airway issues and must work with a variety of sizes of airway equipment. Preparedness is very important when securing a child's airway; because babies and children desaturate so quickly, there is not much time to look through the anesthesia cart for airway equipment. Having a little foresight and preparing your anesthesia cart with the equipment you *plan* to use and equipment you think you *might* use will increase patient safety and save a lot of stress.

Understanding the anatomic differences between the pediatric and adult airway is the first step in pediatric airway management. Children have a proportionally larger head and tongue, narrow nasal passages, an anterior and cephalad larynx (at a vertebral level of C4, compared with C6 in adults), a long epiglottis, slanted vocal cords, and a cone-shaped larynx, with the narrowest point being at the cricoid cartilage. Remember also that infants are obligate nose breathers and the upper airway is relatively more prone to collapse. The relatively larger head and prominent occiput of children places the head in a naturally flexed position when the patient is placed supine. It is helpful to elevate the shoulders with a roll-this lets the head extend back slightly on the shoulders and optimizes the airway for mask ventilation and laryngoscopy. It also allows easier access to the neck if external laryngeal or cricoid pressure is required.

Remember always that children as well as adults can have difficult airways and that problems can occur both with establishing a mask airway and intubating. Oral and nasal airways are quite useful in children and should always be readily available in a variety of sizes. The appropriately sized oral airway should reach from the angle of the mandible to the corner of the mouth. Nasal airways should not be so long that they enter the esophagus. Laryngeal mask airways (LMAs) have become a mainstay in managing difficult airways in children, much as in adults.

Both curved and straight laryngoscope blades should be available prior to induction—each has its advantages. A curved blade can sweep the tongue out of the way to improve the view when there is an obscured view of the larynx due to prominent adenoidal and tonsillar tissue. Straight blades are useful when the larynx is particularly anterior and cephalad; for example, a small Miller blade can be helpful in infants in picking up the epiglottis if it obscures the view of the larynx. P.534

There are several issues to consider when selecting endotracheal tubes (ETT) for pediatric patients. The ETT must be large enough to permit spontaneous or controlled ventilation but not so large as to damage the trachea. Selecting the largest tube that will enter the patient's trachea will decrease resistance, lessen the likelihood of plugging, allow the passage of suction catheters, and lessen the chance of airway aspiration. However, an ETT that is too large can cause tracheal damage when the pressure of the ETT against the wall of the trachea exceeds the capillary pressure of the mucosa. This pressure is believed to be 25 to 35 mmHg in adults; however, no values are available for children. Since the cricoid cartilage is the narrowest point of the airway in children and the only complete tracheal ring, mucosal trauma from placing too large an ETT usually occurs here. This can result in postoperative mucosal edema and swelling, which presents clinically as stridor, croup, airway obstruction, and increased work of breathing. According to Poiseuillle's law:

$$R = \frac{8lv}{\prod r^4}$$

where R is resistance, l is length (of the airway), v is gas viscosity, and r is the radius (of the airway). Therefore, small changes in airway radius lead to large changes in airway resistance. The appropriate size of ETT can be estimated by a formula based on age: 4 + age/4 = ETT size (internal diameter [ID] in mm). One should always have at least three differentsized endotracheal tubes available: The tube you think is the correct size and a tube 0.5 mm ID larger and one 0.5 mm ID smaller. Correct size is confirmed by easy passage of the ETT and gas leakage of 15 to 25 cm H_2O. No leakage indicates an oversized tube that should be replaced, while excessive leakage may make ventilation difficult, require excessive fresh-gas flows, and pollute the operating room. Traditionally, uncuffed ETTs have been the tubes of choice for children younger than 8 years of age. This was because the funnel-shaped larynx resulted in a natural seal at the level of the cricoid ring and the concern that a cuffed tube would require a small ID ETT, hence more resistance. Practitioners are also concerned that excessive cuff pressures can cause mucosal damage. Cuffed ETTs are available in pediatric sizes and have a number of advantages. They allow for an “adjustable fit,” permitting fewer tube changes to get the right size. A better seal allows higher inspiratory pressure in children with reduced lung compliance. The better seal also results in less leakage, reduced fresh-gas flows, less operating room contamination, and a reduced risk of aspiration. To avoid tracheal damage, a number of precautions should be taken. The initial choice of tube size should be at least one-half size smaller than that for an uncuffed tube. The tube should be placed so that the cuff is distal to the cricoid ring. The cuff pressure should be monitored to make sure it is *less than* 25 to 30 cm water pressure, and the cuff should be inflated no more than is necessary to prevent a leak at the patient's peak inspiratory pressure. P.535

Use the following formula to estimate the correct depth of ETT placement: 12 + age (years)/2 = depth of tube (cm) at the lip. Another easy rule of thumb follows: Depth at the lip is equal to three times the size of the tube. One can also intentionally place the ETT into the right main-stem bronchus and slowly withdraw it until breath sounds are equal. One final note: A stylet is usually not needed for routine intubations in older children but may be useful with a smaller (flimsier) ETT or if there is anterior or other abnormal airway anatomy. If used, stylets should be malleable and soft, with a flexible tip in order to avoid airway trauma.

TABLE 122.1 ESTIMATES FOR USE IN PLACING PEDIATRIC AIRWAY DEVICES

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AIRWAY DEVICE	SIZE AND DEPTH ESTIMATE
Nasal airways	Best estimate is a tube that extends from the ala nasi to the tragus of the ear
Oral airways	Best estimate is a tube that extends from the lips to the angle of the mandible
Endotracheal tube size	Uncuffed: 4 + age (years)/4
	Cuffed: 3.5 + age (years)/4
	Premature infant: 2.5 to 3.0
	Term infant: 3.0 to 3.5
	Depth at lip: 6 plus the weight (kg) for infants weighing from 1 to 4 kg = depth in cm at the lip (e.g., for a 2-kg infant, 2 + 6 = 8 cm at the lip); for older infants and children, roughly three times the size of endotracheal tube placed = the depth in cm at thelip
Laryngoscope blades	Premature infant: “0” Straight Blade (Miller)
	Term infant: “1” Straight Blade
	Toddler: “0” to “1” Robert Shaw or “2” Curved (Macintosh) Blade
	Older children: 2 to 3 curved or straight blade, depending on user preference and patient size
Laryngeal mask airways	Up to 5 kg: 1
	>5 to 10 kg: 1.5
	>10 to 20 kg: 2
	>20 to 30 kg: 2.5

TAKE HOME POINTS

- Understanding the anatomic differences between the pediatric and adult airway is the first step in pediatric airway management.
- Remember always that children as well as adults can have difficult airways and that problems can occur with both establishing a mask airway and intubating.
- Be familiar with the size and depth estimates associated with the use of the most common pediatric airway devices (Table 122.1).
- Be prepared with the appropriate airway devices and always have a backup readily available!

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Always Expect and Be Prepared to Treat Hypoxemia During the Induction and Emergence of a Pediatric Patient

Michael J. Stella MD

The “blue baby” is one of the most urgent and potentially disastrous complications an anesthesiologist may face. The term describes the rapid oxygen desaturation of a child, usually occurring at induction or upon emergence. What is especially difficult about this clinical situation is how *quickly* a pediatric patient can turn blue—it is common to hear the pulse oximeter reading described as “dropping like a stone.” Recognition and preparation can save lives. Never be reluctant to call for help when dealing with a baby who has turned blue—these are challenging situations for even the most experienced pediatric anesthesiologists.

PHYSIOLOGY AND ANATOMY

Toddlers, infants, and neonates are all at significant risk for intraoperative hypoxemic events. Remember that babies are not just small adults; they have important differences in both anatomy and physiology, particularly in the circulatory and respiratory systems. Compared with adults, babies have higher rates of oxygen consumption (6 mL/kg/min in babies, 3 mL/kg/min in adults), as well as lower oxygen reserves or functional residual capacity (FRC). This higher ratio of oxygen consumption to FRC is one of the primary factors affecting the increased rate of desaturation when a baby is apneic or has upper-airway obstruction. Neonates and infants also have fewer alveoli and reduced elastic recoil and compliance of the lung. Coupled with greater chest-wall compliance, these factors create an increased risk of atelectasis and intrapulmonary shunt. A patent foramen ovale (PFO) or a patent ductus arteriosus (PDA) in a newborn creates anatomic shunt, which may cause more rapid desaturation. Also, a newborn can revert to a transitional circulation during the first day or so of life. Therefore, anything that causes an increase in pulmonary vascular resistance (PVR) can result in right-to-left shunting via a PDA or PFO.

Anatomic differences, including a relatively larger tongue, pharyngeal hypotonia, and less rigid supraglottic structures, are present in the airway as well. These contribute to a greater chance of airway obstruction. Negative airway pressure results in pharyngeal obstruction from the tongue sticking to the palate and pharyngeal soft-tissue collapse.

P.538

WHAT CAN GO WRONG

A number of events can lead to hypoxic episodes in babies. For some infants and in some situations, even a slight prolongation in the time to intubate the trachea can result in significant desaturation episodes. This may be especially common with rapid-sequence inductions when mask ventilation is avoided. A relatively common cause of airway obstruction, especially during the cold-and-flu season is laryngospasm. Laryngospasm is frequently the result of extubation during the “excitement” or stage II depth of anesthesia. Pediatric patients also commonly hold their breath during emergence, which can lead to rapid and deep desaturation. This breath holding, or Valsalva-like phenomenon, results in increased intra-abdominal and intrathoracic pressure with glottic closure. Ventilation is difficult, and the resulting increase in PVR causes right-to-left intracardiac shunting via a PFO. Finally, another common cause of airway obstruction during both induction and emergence is relaxation of the genioglossus muscle, which allows the tongue to fall back and occlude the oropharynx.

HOW TO AVOID THESE PROBLEMS

Avoiding these problems is clearly preferable to managing them. Always have appropriately sized oral and nasal airways and a tongue depressor immediately available. Use the proper size and type of laryngoscope blade and proper head and neck positioning to facilitate visualization of the vocal cords. Consider using glycopyrrolate (10 mcg/kg, intravenously [IV]) or atropine (10 mcg/kg IV) as a premedicant to dry oropharyngeal secretions. Administering oxygen in advance is an important adjunct to “buy” time for your intubation attempts. To avoid obstruction due to relaxation of the genioglossus muscle, open the mouth and “unstick” the tongue from the roof of the mouth. Upon emergence, clear the airway and be certain the patient meets extubation criteria and is fully awake. Be certain that the patient remains deeply anesthetized if you are doing a deep extubation. If the patient is emerging from airway surgery, be sure there is adequate hemostasis. Be prepared to provide positive airway pressure if initial respiratory efforts are ineffective due to upper-airway obstruction. Have succinylcholine (1 to 2 mg/kg IV or 3 to 4 mg/kg, intramuscularly [IM]) and atropine (10 mcg/kg IV or 20 mcg/kg IM) readily available.

WHAT TO DO WHEN HYPOXEMIA OCCURS

Even with proper planning, babies may become hypoxemic secondary to difficulty with ventilation. Upper-airway obstruction is treated with an oral or nasal (or both) airway and jaw thrust with continuous positive airway pressure (CPAP) or positive-pressure ventilation. Breath holding is best treated with 100% oxygen and CPAP. This allows the child to receive an assisted breath upon spontaneous inhalation. Laryngospasm is initially treated with positive-pressure ventilation and 100% oxygen, with jaw thrust and temporomandibular joint pressure sometimes being needed. If this fails to break the laryngospasm, succinylcholine and atropine should be readily available and administered. If none of the above maneuvers works, remember the difficult airway algorithm and proceed accordingly. With proper preparation and vigilance, a “blue baby” scenario can be managed safely.

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TAKE HOME POINTS

Always expect and be prepared to treat hypoxemia during the induction and emergence of a pediatric patient:

- Pediatric patients can desaturate *fast* due an increased ratio of oxygen consumption to FRC, which leads to rapid desaturation.
- Never be reluctant to call for help with a hypoxic patient.
- Neonates can be very susceptible to shunting (due to PDA, PFO, and increased PVR).
- Laryngospasm is relatively more common in pediatric than in adult patients, especially during stage II.

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- With genioglossus laxity, a larger tongue can obstruct the oropharynx.
- Be prepared:
  - Have the proper equipment ready.
  - Have the proper medications drawn up beforehand.
  - Administer oxygen in advance appropriately.
- Use CPAP, a jaw thrust, and 100% O_2 for obstructions and laryngospasm.

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The Pediatric Airway is a Scary Repository for all Kinds of Foreign Bodies

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Robert D. Valley MD

So… you are on call with that new ear, nose, and throat surgeon—the one who is new to your hospital and maybe new to the profession (just out of residency!). He wants you in to anesthetize a 10-month-old little girl who choked on “something she picked up off the rug” about 12 hours ago. Her chest X-ray is clear. She has had an intermittent cough and sounds wheezy in Mom's arms. You don't do a lot of little children and wonder if this is the right thing to do. You try talking to the surgeon, but he is distracted as he tries to piece together the seldom-used pediatric rigid bronchoscope. The surgeon assures you he did a “bunch of these” as a resident. Do you proceed or call the hospital administrator on call?

The potential for total airway obstruction, as well as the need to share the airway with the surgeon, makes anesthesia for foreign-body removal challenging. Commonly aspirated foods include peanuts, beans, hotdogs, watermelon seeds, and popcorn. Peanuts are the most common and can be difficult to remove because of the tendency to break into pieces upon attempted retrieval. Aspirated beans are challenging because they often swell once inside the airway and lead to further obstruction and edema. Nonfood items are more commonly aspirated by older children and include small plastic toys, balloon pieces, balls, and marbles.

Foreign-body aspiration should be considered in any child presenting with respiratory distress or with a pulmonary infection that is not responding to standard interventions. A history of a choking episode is found in 80% to 90% of confirmed cases. Chest radiographs are often normal during the first 24 hours. In one large retrospective series of 1,068 cases of foreign-body aspiration in children, radiographs were normal in nearly two thirds of cases. If an inspiratory film is normal, an expiratory film or fluoroscopy may be helpful. These views may demonstrate expiratory mediastinal shift away from the lung field containing the foreign body or postobstructive hyperinflation secondary to air trapping distal to the foreign body.

When foreign-body aspiration is suspected, rigid bronchoscopy is the gold standard. Rigid bronchoscopy permits control of the airway, good visualization, and manipulation of the object with a wide variety of forceps.

Removal of a foreign body from the airway is considered an urgent to emergent procedure, depending on the degree of respiratory distress. A fasting patient is optimal, but the procedure should not be delayed if the airway is compromised or if the foreign body is of organic origin because of its potential to swell and produce asphyxia. The anesthesiologist should decide between using mask induction and intravenous (IV) induction on an individual basis. Most experts recommend mask induction followed by IV placement; however, rapid-sequence induction should be considered if the child has a full stomach. The use of nitrous oxide is a relative contraindication in the presence of air trapping, although most authors conclude that its use during a mask induction poses little risk. Using atropine or glycopyrrolate is recommended to reduce secretions before bronchoscopy, and a dose of a glucocorticoid can be given to reduce inflammation. After a deep plane of anesthesia is obtained, the airway should be examined with a laryngoscope to exclude any posterior pharyngeal foreign bodies and to spray the vocal cords and trachea with lidocaine in order to reduce irritation once the rigid bronchoscope is introduced. P.541

Clinicians are divided over their approach to ventilating these patients. Some prefer spontaneous ventilation, others are equally adamamt that controlled ventilation, with or without muscle relevants, is the safest approach. There is a theoretical risk that a foreign body can be pushed more distally with positive-pressure ventilation. In a prospective randomized clinical trial to compare spontaneous and controlled ventilation during removal of airway foreign bodies in 36 children, it was concluded that routine use of controlled ventilation with muscle relaxants is the better technique. Ventilating a patient through the side port of a rigid bronchoscopy can be difficult, and the amount of inhaled anesthetic administered is unpredictable. Many clinicians would argue that having the patient breath spontaneously is safer. Airway leaks and resistance are highly variable depending on the bronchoscopic instrumentation, patient size, level of the foreign body, depth of anesthesia and relaxation of the patient. Communication between the anesthesiologists and surgeon is essential during the procedure because the distal end of the bronchoscope may need to be occluded. Adequate oxygenation frequently requires the administration of 100% oxygen.

Sixty percent of airway foreign bodies are located in the right lung. Of the remainder, 23% are in the left lung, 3% are in the larynx, 13% are in the trachea or carina, and 2% are in both lungs. During removal of a foreign body, total-airway obstruction may occur. If a single object breaks into multiple pieces, the uninvolved lung may become obstructed. An object, such as a bean, that has swollen can become lodged in the trachea upon attempted removal. The complication of total-airway obstruction is a possibility whenever a foreign body is being removed. Immediate recognition and treatment is essential. If the surgeon can remove the foreign body, it must be done in a timely fashion. If that is not possible, the foreign body can be pushed distally until ventilation is reestablished. If a foreign body is pushed distally to obtain adequate ventilation and can no longer be retrieved bronchoscopically, a thoracotomy may be required. P.542

Maintenance of deep anesthesia, with or without muscle relaxants, is essential for successful removal of an airway foreign body. Complications during bronchoscopy include laryngospasm, bronchospasm, laryngeal edema, arterial desaturations, and upper-airway damage. A retrospective review was done to quantify adverse events in 287 cases in which children had rigid bronchoscopy after mask induction and muscle paralysis for foreign-body aspiration. Anesthesia-related adverse events were seen in 0.7% of cases, whereas complications from rigid bronchoscopy were observed in 7.6% of cases. In this study, the most common anesthesia-related adverse event was laryngospasm upon extubation.

TAKE HOME POINTS

Anesthesia for airway foreign-body removal is challenging, and keys to success include the following:

- Open communication with the surgeon;
- A deep level of anesthesia, with or without paralysis;
- Quick recognition and treatment of total airway obstruction;
- Full emergence in the operating room with the otolaryngologists present; and
- Recognition and treatment of laryngospasm and postextubation stridor.

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Effective Epidural Anesthesia for Children does not Always Require a Catheter

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Pediatric anesthesiologists at major centers often place epidural catheters for complex ureteral implant surgeries and many major lower-abdominal procedures. These catheters are used for both adjunctive anesthesia and postoperative pain control and are best managed by the experienced pediatric anesthesiologist. However, a one-time caudal epidural injection with local anesthetic (the “single-shot caudal”) is a reliable and generally safe technique for patients undergoing smaller abdominal and lower-limb surgeries, such as inguinal hernia repair, hypospadias repair, circumcision, orchiopexy, and club foot repair. The authors feel that the single-shot caudal block is an underused technique, but that it should not be—its technical ease puts it well within the practice parameters of pediatric anesthesiologists who have less experience and anesthesiologists who provide care to pediatric patients only occasionally.

INDICATIONS

The single-shot caudal block is indicated for treating children ranging in age from infancy to approximately 8 years. Single-shot caudal blocks are usually placed at the end of the procedure to maximize the duration of postoperative analgesia; however, practice varies. If this block is placed at the beginning of the procedure, be aware that total anesthetic requirements will be decreased. *Always* discuss the planned block with the pediatric patient's parents. The older child should also be made aware that his or her legs may be difficult to feel after the surgery or that he or she may have trouble walking for a few hours.

CONTRAINDICATIONS

The single-shot caudal block is contraindicated for treating children with infection around the sacral hiatus, coagulopathy, increased intracranial pressure (ICP), uncorrected hypotension, or known anatomic abnormality and children whose parents refuse to consent.

ANATOMY

In patients younger than 1 year of age, the caudal extent of the dura and spinal cord ends at S3-4. The caudal space can be accessed through the sacral hiatus, which is flanked laterally by the prominent sacral cornu and covered by the sacral-coccygeal ligament. P.544

TECHNIQUE

The patient should be placed in lateral decubitus position (left lateral for the right-handed anesthesiologist and right lateral for the left-handed anesthesiologist), with hips and knees flexed to help spread the gluteal muscles away from the sacral hiatus. Using aseptic technique, a 22-gauge needle (or 22-gauge Teflon intravenous catheter) is placed approximately 45 degrees to the skin until a “pop” is felt, which indicates penetration of the sacrococcygeal ligament. The needle should be slightly withdrawn and the angle lowered before advancing a few more millimeters into the canal. Aspiration is necessary to check for blood or cerebrospinal fluid. An initial test dose of 0.1 mL/kg of local anesthetic with 1:200,000 epinephrine can be used to exclude intravascular placement (see below). Resistance to injection should be minimal. Any resistance strongly suggests that the needle tip is not in the epidural space.

DOSING

For single-shot caudal epidural injection, long-acting local anesthetics, such as bupivacaine (0.125% to 0.25%) and ropivacaine (0.2%), are typically used. The volume of local anesthetic depends on the desired blockade level. 0.5 mL/kg can be adequate for sacral blockade, whereas 1.0 mL/kg is needed for upper-abdominal blockade. The maximum volume is 1.25 mL/kg, which will give you a low thoracic level in an infant. The maximum recommended dose for bupivacaine or ropivacaine is 3.0 mg/kg. For older children, the maximum volume of anesthetic injected into the epidural space (regardless of concentration) is usually about 20 mL. To prolong the block's duration, clonidine (1 to 2 mcg/kg) can be added to the local-anesthetic solution.

COMPLICATIONS

Dural Puncture

Unintentional dural puncture can lead to total spinal. Symptoms include apnea, blown pupil, hypotension, and unresponsiveness. Younger children (less than 6 years of age) usually do not exhibit unstable hemodynamics due to an immature sympathetic system and greater centralization of their intravascular space. Treatment is supportive: Airway management, ventilation or oxygenation (or both), and circulatory support (fluids and vasopressors, as indicated).

Intravascular Injection.

Intravascular injection is the most common serious complication of caudal anesthesia. Using a test dose and looking only for heart-rate changes is not a sensitive indicator of intravascular injection in anesthetized children. Changes in electrocardiogram (EKG) morphology, especially peaked T-waves and broadening of the QRS complex, are more sensitive indicators of intravascular (or intraosseous) injection. A caudal block should *never* be placed without continuous EKG monitoring. Even after a negative response to an initial test dose, the anesthesiologist must give the remaining local anesthetic incrementally, looking for EKG changes after administration of each dose. Local-anesthetic toxicity manifests as seizures, hypotension, arrhythmia, and cardiovascular collapse. Support includes airway management, ventilation or oxygenation (or both), circulatory support, and antiseizure medication. The importance of chest compressions cannot be over emphasized, since the primary offset to toxicity is P.545

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redistribution, which cannot occur if circulatory support is not provided. Also, 20% lipid emulsion has recently been identified as a useful treatment for bupivacaine toxicity (see Chapter 128).

Intraosseous Injection.

Intraosseous injection can lead to systemic toxicity similar to that caused by intravascular injections. The thin cortical bone of the sacrum is easily penetrated in pediatric patients. Treatment is the same as for intravascular injection.

TAKE HOME POINTS

Consider incorporating the single-shot caudal injection into your practice even if you are a pediatric anesthesiologist who has less experience or are an anesthesiologist who provides care to pediatric patients only occasionally.

- The typical age range of patients who are good candidates for single-shot caudal blocks is from infancy to approximately 8 years of age.
- Caudal blocks can be performed at the beginning or end of a procedure.
- Always discuss the block with the pediatric patient's parents and tell older children they may have “wobbly legs” when they wake up.
- Dosing varies according to agent, weight, and level of block desired. Maximum doses are given above.
- Maintain high vigilance for dural puncture (total spinal). Children become unresponsive and apneic but may not manifest overt hemodynamic signs.
- Avoidance of intravascular injection depends on close observation of the continuous EKG trace.
- Peaked T-waves and widening of the QRS complex are both sensitive indicators of intravascular or intraosseous injection.

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Keeping Babies Warm in the Perioperative Period is Important, Challenging, and, At Times, Dangerous!

Kathleen A. Smith MD

Virtually all pediatric patients become hypothermic in the perioperative period, unless measures are taken to maintain normothermia. Hypothermia leads to a variety of complications, including delayed emergence, prolonged neuromuscular blockade, platelet dysfunction, increased oxygen consumption upon emergence, and poor wound healing. Thus, the anesthesiologist must continuously monitor core temperature and actively prevent heat loss in the operating room (OR), as well as in the postanesthesia care unit (PACU). Keep in mind that there are actually a number of reasons for heat loss. Also, cooling begins with exposure of the patient upon removal of his clothing; accordingly, do not have the baby undressed until it is necessary.

The most important type of heat loss is via radiation, but convection, conduction, and evaporation also play a role. Most ORs are kept extremely cold, which allows for tremendous radiant heat loss to the OR environment. Next, the surgical site is prepped with cold solution, which partially evaporates from the skin, decreasing the body's temperature further. Other evaporative losses occur from surgical incisions as well as from the airway. Volatile anesthetics cause vasodilation, which brings heat to the cutaneous surface where it can participate in heat exchange with the environment. During the first half hour of anesthesia, it is this redistribution of heat from the core to the periphery that causes the patient's temperature to drop 0.5° to 1.5° C. General anesthesia also impairs normal function of the body's thermoregulatory center, the hypothalamus. Under normal circumstances, the vessels of the body constrict to conserve heat, while metabolic heat production is increased. Peripheral vasodilation is combined with a 20% decease in heat production, thus altering the thermal steady state.

Heat loss is directly proportional to surface area. Because infants and neonates have a very large surface-area-to-volume ratio, they are particularly prone to becoming hypothermic. In addition, metabolic heat production is a function of mass. Therefore, not only do they lose heat faster, but they are less capable of producing heat. Infants and neonates do not shiver. Instead, they produce heat via nonshivering thermogenesis, which takes place primarily in brown fat. This mechanism of heat production, which continues until 2 years of age, is inhibited by anesthesia and sympathetic blockade.

For the many aforementioned reasons, anesthesiologists often battle intraoperative hypothermia on a regular basis. This has led to the development of various mechanisms that can be used to maintain normothermia during anesthesia. Some of the methods are simple and can be of tremendous benefit. Warming the operating room by 1° C will decrease the patient's heat loss by 7%. This is an easy and effective means of preventing heat loss form the start of the procedure. Passive insulators, such as blankets or plastic bags wrapped around the exposed surfaces of the body, may reduce heat losses up to 30%. An infant's head is a major source of heat loss, given its large surface area, and should be wrapped to decrease radiant heat losses. Warming intravenous fluids helps prevent hypothermia during longer procedures with greater fluid requirements. It has been shown that 1 L of room-temperature fluid given to an adult reduces core temperature between 0.25° and 1.0° C. A similar result is seen in children receiving a comparable volume of unwarmed fluid. Circulating water mattresses are used less often to reduce conductive heat losses. Their effectiveness is limited, given that they only reduce the already minimal heat loss from a patient's back. Warming lights, infrared heaters, and warm preparatory and irrigation solutions may also be of benefit. P.547

Perhaps the most popular means of preventing hypothermia intraoperatively is the forced-air warmer. These devices work via convective heat transfer and depend on a device-skin temperature gradient. As helpful as this device can be, improper use may have an effect opposite of that intended and may even be dangerous. The Food and Drug Administration notes several reports of patient injury resulting from the use of warming hoses that are not attached to a warming blanket. This so-called “free-hosing” has led to third-degree burns in several patients. A second pitfall with forced-air warming devices is unintentional patient cooling when either the skin beneath the blanket or the blanket itself becomes wet as a result of messy surgical preparation or leakage of irrigation fluid or body fluids. Little information has been published regarding this phenomenon. To quantify the degree of patient cooling that can result when this occurs, we performed our own miniature experiment. Skin-temperature probes were placed on two warm saline bottles, before covering each of them with a forced-air warming blanket. Both forced-air warming blankets were turned on, set to 43° C, and allowed to equilibrate for 10 minutes. Following this, 125 mL of warm irrigation fluid was poured over the blanket covering bottle A. Bottle B remained dry. The starting temperatures for bottles A and B were 37.8° and 37.3° Celsius, respectively. Ten minutes later, bottle A had cooled from 37.8° to 35.5° C, whereas bottle B remained at 37.3° C. After 15 minutes, bottles A and B were at 34.9° and 37.2° C, respectively.

Clearly, the soiled blanket was not only ineffective at heating but actually might have cooled a patient significantly in a short period of time.

Steps must be taken to prevent forced-air warming blankets from becoming soiled. This starts with awareness of all personnel, especially surgeons. A preparatory job that is sloppy and uses an excessive amount of solution is the anesthesiologist's enemy. In addition, a plastic sheet is included with each forced-air warming blanket. This sheet may be used to make a barrier between the operative field and the blanket itself. This step may reduce the incidence of saturating the blanket, both during the surgical preparation and throughout the surgery. If a warming blanket does become wet, it should be removed and, if possible, replaced with a new blanket. If a warmer must be removed, the OR temperature should be increased. This may be an incentive for all OR personnel to be cautious not to allow the blanket to become wet. P.548

Finally, be aware of iatrogenic hyperthermia. Many practitioners, in an effort to prevent hypothermia, overuse warming techniques, resulting in hyperthermia. This is not uncommon in dental surgery, for example, where virtually the entire body is covered and no body cavities are exposed. This results in very minimal, if any, heat loss. These procedures tend to be long, and the child's basal metabolic rate naturally generates heat. If atropine has been given, sweating is reduced, preventing dissipation of this heat and increasing body temperature. In this situation, a forced-air warming blanket may actually be used to help cool a febrile patient, using the warming blanket's ambient setting.

TAKE HOME POINTS

- We want our pediatric patients to be warm, but not too warm, and we need to keep them warm effectively and safely.
- Don't let the warming blankets get wet!

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Focus on Prevention and Treatment of Emergence Delirium

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Emergence delirium can be a frustrating and stressful situation for everybody involved, including the patients' families and the nurses. A scenario might unfold as follows: A 5-year-old patient with a history of mild attention deficit and hyperactivity disorder has come to your operating room for an emergency esophago gastroduodenoscopy (EGD) to rule out ingestion of a foreign body. The patient is given a midazolam premedication which helps some with the induction. The endoscopist finds nothing in the esophagus or stomach. You wake the child up from the sevoflurane anesthetic and, on the way to the postanesthesia care unit (PACU), he begins crying and thrashing. He catches the PACU nurse on the lip with the back of his head. There is blood. This doesn't look good for you. He is crying and moaning. You can't seem to get his attention even though his eyes are open. Mom comes in and says she has never seen him like this. She starts to cry because her child doesn't recognize her. The nurse looks at you and whispers, “You better do something to make this better!”

Emergence Delirium (ED), also frequently called emergence agitation, is a *diagnosis of exclusion* . A disoriented patient who is incognizant of his or her surroundings and of previously familiar individuals and objects and who is generally inconsolable and uncooperative is diagnosed as having ED, absent another diagnosis. Behavior ranges from incoherence and moaning, to thrashing, agitation, and paranoia or hallucinations. While usually self-limited (<1 hour in duration), ED can lead to many undesirable consequences, such as an inability to monitor the patient, disconnected intravenous (IV) lines, and significant physical trauma to the patient or caregivers (or both).

Hypoxia, inadequate analgesia, hypoglycemia, severe hypercarbia, increased intracranial pressure, nausea, and bladder distention are all factors to consider prior to settling on ED as the diagnosis for a pediatric patient's disorientation and agitation.

The incidence of ED is estimated to range from 5% to 55% and is higher in children who are 2 to 5 years of age than in younger and older children. One of the problems in recognizing and treating the disease has been the lack of a reliable and valid instrument for measuring ED in children. Previously, this precluded comparisons between trials and raised serious questions about the reliability and validity of research results. The development and evaluation of the Pediatric Anesthesia Emergence Delirium (PAED) Scale in 2004 will enable researchers to characterize ED in children accurately and to compare P.550
treatment options in future studies.

Various mechanisms have been postulated to cause ED; a definitive causality has yet to be identified. Volatile anesthetics (halothane, isoflurane, sevoflurane, and desflurane) are associated with an incidence of ED as high as 50%. Sevoflurane and desflurane have been extensively studied. Their low blood-gas-solubility quotient with rapid emergence has been theorized to lead to a higher incidence of ED. Numerous studies have yielded conflicting data but suggest that sevoflurane may be associated with a higher incidence of ED than desflurane. Ketamine, droperidol, atropine, and scopolamine also are associated with ED. Propofol, when used for maintenance of anesthesia, is associated with a very low incidence of ED.

Administering pain medication (fentanyl, 1 to 1.5 mcg/kg IV; or clonidine, 2 mcg/kg IV) may reduce the incidence of ED. Administering midazolam in advance does not consistently reduce the incidence of ED. In pediatric patients, ranging in age from 18 months to 10 years, having outpatient magnetic resonance imaging (a painless procedure) with use of sevoflurane, administering fentanyl (1 mcg/kg IV) 10 minutes before anesthetic discontinuation reduced the incidence of ED by 56%, whereas administering a placebo reduced the incidence of ED by 12%. Similar results were found in a group of 110 developmentally delayed American Society of Anesthesiologists (ASA) Physical Status 1-2 patients having dental restoration (with sevoflurane general anesthesia administered using laryngeal mask array [LMA]). These patients were randomly assigned to receive either fentanyl, 1 to 1.5 mcg/kg, or saline at induction of anesthesia. Notably, administering this dose of narcotic did not increase nausea and vomiting or delay discharge.

Intraoperative use of the alpha-2 adrenergic agonist clonidine, which has sedative and analgesic properties, can also provide ED prophylaxis. A prospective double-blind study examined the cases of 40 2- to 7-year-old boys having circumcision with penile block and sevoflurane general anesthesia. The boys were randomly assigned to receive clonidine, 2 mcg/kg IV, or placebo. Sixteen patients in the placebo group and two in the clonidine group developed agitation. Another prospective study examined the cases of 169 ASA Physical Status 1-2 patients having day surgery (with midazolam, 0.5 mg/kg, administered orally before the procedure and with sevoflurane general anesthesia administered using LMA). These patients were randomly assigned to receive clonidine, 2 mcg/kg IV, or saline prior to induction. The clonidine group had a lower incidence of ED (14.2%) than did the placebo group (33.3%), with no increase in the clonidine group's time to emergence.

The treatment options for ED in the PACU (i.e., rescue therapy) are controversial, even among clinicians with a special interest in this area. There is a lack of published data comparing fentanyl, clonidine, and propofol in formal clinical trials for rescue therapy; however, prospective observational P.551
studies are forthcoming. At present, fentanyl is the drug most frequently reported to be used for rescue therapy. Clonidine, besides being reported to be efficacious for prevention, has also been used anecdotally for treatment. Whatever the true efficacy of clonidine for rescue therapy, its use is not widely reported. Other pediatric anesthesiologists feel they have ample anecdotal evidence that small doses of propofol (1 mg/kg) are successful in treating ED in the PACU and prefer it to clonidine since it is generally a more readily available and familiar drug. Midazolam is thought to simply delay emergence and is not considered very useful for treating ED. Remember that, with time, ED usually resolves; therefore, a commonly employed strategy is gentle restraint of the patient until the ED passes. The decision to wait rather than treat depends on the severity of the ED, the availability of extra PACU staff to restrain the child, and the risk of injury or morbidity secondary to loss of invasive catheters or surgical drains.

TAKE HOME POINTS

- All that is agitation is not pain.
- The price of a rapid emergence may be delirium.
- A little narcotic can make for a smooth landing.

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Title: ***Avoiding Common Anesthesia Errors, 1st Edition***

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Remember that Babies Really Do Feel Pain

Angela Kendrick MD

Pediatric pain management may be as “simple” as providing a single-shot caudal with local anesthetic for postoperative pain relief or as “complex” as providing consultative and management services for children with sickle cell disease, cancer pain, or complex regional pain syndromes.

This chapter is intended to provide you with some strategies for evaluating pediatric pain, to remind you of important pharmacokinetic and anatomic differences between infants and children, on the one hand, and adults, on the other, and to provide you with some tips on avoiding common mishaps associated with the treatment of acute pain in children.

EVALUATION OF PAIN

You must choose the appropriate pain scale. Pain scales are the tools you use to document the presence and severity of pain and the effectiveness of your treatment.

There are a number of behavior-based pain scales. The Children's Hospital of Eastern Ontario Pain Scale (CHEOPS) (Table 128.1) was developed by McGrath et al. to assess pain in the postanesthesia care unit (PACU). Six categories of behavior (crying, facial expression, verbal expression, movement of torso, touching of wound, and movement of legs) are observed and scored, and an appropriate number is charted to indicate the presence or intensity of a response.

The Face, Legs, Activity, Cry and Consolability (FLACC) scale (Table 128.2) is simpler than CHEOPS and is popular for the assessment of both infants and older, nonverbal patients. Parents or chronic caregivers of children with cognitive impairment have been shown to be sensitive to their child's method of expressing pain. This input can be invaluable for interpreting the developmentally disabled child's responses.

Observing physiologic parameters (heart rate, respirations, blood pressure, body temperature) is standard practice in evaluating a patient's overall condition. Physiologic variables are also incorporated into pain assessment, with rapid shallow breathing, elevated heart rate, and increased blood pressure possibly indicating the presence of pain.

Other methods of pain assessment, such as verbal self report, use of numeric scales, and use of the faces scale, are appropriate for use in older children but have their disadvantages. For example, the child may want to avoid a potential painful encounter (fear of needles), so the child denies any pain. A child may fail to understand ordinal numbers and pick a higher “better” number, or the child may pick the smiley face on the faces scale because it is the face he or she likes the best. P.553

TABLE 128.1 CHILDREN'S HOSPITAL EASTERN ONTARIO PAIN SCALE (CHEOPS). RECOMMENDED FOR CHILDREN 1 TO 7 YEARS OF AGE; A SCORE GREATER THAN 4 INDICATES PAIN.

ITEM	BEHAVIORAL	POINTS	DEFINITION	SCORE
Crying	No crying	1	Child is not crying.	
	Moaning	2	Child is moaning or quietly vocalizing; silent cry.	
	Crying	2	Child is crying, but the cry is gentle or whimpering.	
	Scream	3	Child is in a full-lunged cry; sobbing; may be scored with complaint or without complaint.	
Facial	Composed	1	Neutral facial expression.	
	Grimace	2	Score only if definite negative facial expression.	
	Smiling	0	Score only if definite positive facial expression.	
Verbal	None	1	Child not talking.	
	Other complaints	1	Child complains, but not about pain, e.g., “I want to see mommy” or “I am thirsty.”	

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	Pain complaints	2	Child complains about pain.
	Both complaints	2	Child complains about pain and about other things, e.g., “It hurts; I want my mommy.”
	Positive	0	Child makes any positive statement or talks about others things without complaint.
Torso	Neutral	1	Body (not limbs) is at rest; torso is inactive.
	Shifting	2	Body is in motion in a shifting or serpentine fashion.
	Tense	2	Body is arched or rigid.
	Shivering	2	Body is shuddering or shaking involuntarily.
	Upright	2	Child is in a vertical or upright position.
	Restrained	2	Body is restrained.
Touch	Not touching	1	Child is not touching or grabbing at wound.
	Reach	2	Child is reaching for but not touching wound.
	Touch	2	Child is gently touching wound or wound area.
	Grab	2	Child is grabbing vigorously at wound.
	Restrained	2	Child's arms are restrained.
Legs	Neutral	1	Legs may be in any position but are relaxed; includes gentle swimming or serpentine-like movements.
	Squirming or kicking	2	Definitive uneasy or restless movements in the legs or striking out with foot or feet (or both).
	Drawn up or tensed	2	Legs tensed or pulled up tightly to body and kept there.
	Standing	2	Standing, crouching, or kneeling.
	Restrained	2	Child's legs are being held down.

From McGrath PJ. *Medical Algorithms* . www.medal.org, with permission.

TABLE 128.2 FACE, LEGS, ACTIVITY, CRY AND CONSOLABILITY (FLACC) BEHAVIORAL PAIN ASSESSMENT TOOL

CATEGORY	DESCRIPTION	SCORE
Face	0 = No particular expression or smile	0
	1 = Occasional grimace/frown, withdrawn or disinterested	1

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	2 = Frequent/constant quivering chin, clenched jaw	2
Legs	0 = Normal position or relaxed	0
	1 = Uneasy, restless, tense	1
	2 = Kicking or legs drawn up	2
Activity	0 = Lying quietly, normal position, moves easily	0
	1 = Squirming, shifting back and forth, tense	1
	2 = Arched, rigid, or jerking	2
Cry	0 = No cry	0
	1 = Moans or whimpers, occasional complaint	1
	2 = Crying steadily, screams or sobs, frequent complaints	2
Consolability	0 = Content and relaxed	0
	1 = Reassured by occasional touching, hugging or being talked to, distractable	1
	2 = Difficult to console or comfort	2

From Voepel-Lewis T, Merkel S, et al. The reliability and validity of the FLACC Observational Tool as a measurement of pain in children with cognitive impairment. *Anesth Analg* . 2002;95:1224-1229, with permission.

KNOW YOUR HIGH-RISK POPULATIONS

Infants younger than 3 months of age, infants born prematurely that are less than 60 weeks postmenstrual age (formerly called postconceptual age), and any child with a history of apnea or airway compromise, cardiac compromise, or neuromuscular disease are at risk for cardiorespiratory complications following opioid or sedative administration. You need to carefully titrate your dose and monitor these children after administration of opioids or sedatives.

Premature Infants and Neonates Require Special Consideration.

We now know that the preterm infant has the neurophysiologic pathways to experience pain and the inhibitory pathways for modulating the pain response are less developed. Inadequate relief of pain in the neonatal intensive care unit may lead to an accelerated catabolic response, with increased secretion of stress hormones and altered sleep-wake cycles. Inadequate pain relief in the very young may lead to a long-term altered response (increased sensitivity) to pain.

Drug Metabolism in the Neonate.

Newborns have lower levels of plasma proteins, including albumin and alpha (1) acid glycoprotein, than do adults. When protein-bound drugs are administered, newborns have a higher free fraction of drug, which is the pharmacologically active form, than do adults.

Infants may have physiologic processes that impair renal or portal blood flow (necrotizing enterocolitis, abdominal compartment syndrome, reduced systemic cardiac output), so metabolism and elimination of drugs may become unpredictable.

Newborns are slow metabolizers due to liver immaturity. This ability rapidly increases during the first 3 months of life. Hepatic metabolism occurs in two phases:

a. Phase 1 (oxidation, hydroxylation, and hydrolysis or reduction) involves the cytochrome P-450 enzyme system.

b. Phase II enzymes conjugate the metabolites of the phase 1 reactions. Phase II conjugation with glucuronyl transferases may be also be impaired in the newborn.

Since the kidney clears both parent drug and metabolites produced by the liver, renal failure causes accumulation and potential toxicity.

DRUG THERAPY FOR ACUTE PAIN

Opioid Infusions in the Neonate.

Pain from a variety of causes can be managed via a continuous IV infusion with bolus supplementation. The two opioids most frequently used in the neonate are morphine and fentanyl. Neonates who are not intubated need particularly careful monitoring

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a. Morphine does not require Phase I metabolism but undergoes Phase II glucuronidation, producing morphine 3 glucuronide (nonactive) and morphine 3,6 diglucuronide (active). Morphine has a prolonged half-life in neonates (6.8 hours and up to 10 hours in the preterm infant). The loading dose range for IV administration is 50 to 100 mcg/kg. The amount of drug used in constant-infusion doses depends on the patient's rate of metabolism, degree of pain, and other individual characteristics.

b. Fentanyl is highly bound to alpha (1)-acid glycoprotein. Its elimination half-life is prolonged in premature infants and newborns and is even more prolonged after a continuous infusion. The "context-sensitive half time" refers to the time for drug concentration at the sites of action to decrease by half. Newborns receiving fentanyl infusions for longer than 36 hours have a context sensitive half time of greater than 9 hours. Initial IV dosing for acute pain is 0.5 to 1 mcg/kg. Continuous-infusion rates are highly variable and depend on the degree of pain, the duration of administration, and the development of tolerance (see below).

P.556

PCA for Children

a. Use of standard preprinted Patient-Controlled Analgesia (PCA) order sheets, with suggested dose ranges, monitoring standards, and discontinuation of other sedative medications, is important in the care of any infant or child receiving continuous opioid therapy. The orders, the infusions, and their pumps must be reviewed on a daily basis.

b. If true PCA is to be used (rather than a continuous nonadjustable infusion), the child must understand how to push the button .The lockouts must be preset so that only a safe hourly maximum dose is delivered. "PCA by proxy," with the parents or nurse pressing the "pain button," is also used in a number of hospitals. This form of "PCA" is very controversial.

c. Infants and children receiving continuous infusions of opioids develop tolerance and require increasing doses to maintain the same effect. Data from studies done on animals show that young animals develop tolerance at a quicker rate than do adult animals. If continuous opioid infusion therapy has been used for longer than 5 to 7 days, abrupt discontinuation may result in withdrawal symptoms. The duration of drug exposure and the accumulated amount of drug administered are factors to consider when planning your weaning protocol. Weaning protocols usually reduce opioids by 10% to 20% per day, with careful observation for tachycardia, diarrhea, irritability, or other signs of opioid withdrawal. Long-acting opioids, such as methadone and the alpha agonist clonidine, are sometimes useful to assist with the weaning process.

Adjuncts Other than Opioids.

Multimodal therapy is important in treating postoperative pain. Acetaminophen (orally or rectally) given as a scheduled medication (not on an as-needed basis) has a significant opioid-sparing effect. Other nonsteroidal analgesics (Ketorolac) should also be considered for use. Ketamine (small dose, <0.5 mg /kg IV) has also been used in the PACU and in low-dose infusions postoperatively. It is a noncompetitive antagonist of the N-methyl-D-asparate (NMDA) receptor and produces a synergistic effect to opioid analgesia.

REGIONAL ANESTHESIA FOR POSTOPERATIVE PAIN

In contrast to blocks for adults, the majority of blocks for children are placed after the induction of general anesthesia. Detecting an intravascular injection with a test dose of local anesthetic (5 mcg/mL of epinephrine) is less reliable in the presence of inhalational anesthetics. You must watch carefully for an increase in heart rate, a decrease in heart rate, or a change in T-wave morphology (peaked T waves); blood pressure may not increase with intravascular injection. Because the child is anesthetized, central nervous system symptoms may be masked and cardiovascular collapse may seem to occur without warning.

There are important anatomic differences to remember when placing caudal blocks or lumbar epidurals in pediatric patients. The subarachnoid space extends to S3-4 in the newborn before rising to the adult level of S1 at about the age of 1 year. This means the cerebrospinal fluid space is easy to enter when placing a caudal block in an infant. The neonatal spinal chord also extends to a lower lumbar level as low as L3 before receding to the adult level of L1. P.557

Single shot caudal blocks are widely used to provide postoperative pain relief for operations done below the level of the patient's umbilicus. Intravascular injection, intrathecal injection, and intraosseous injection are all complications associated with this block. Careful advancement of the needle, with gentle aspiration after advancement, test dosing, continuous electrocardiogram monitoring, and incremental dosing will help you avoid catastrophic events.

As noted above, local anesthetic toxicity may develop acutely if the local anesthetic is inadvertently given intravenously or intraosseously. Toxicity may also be the result of accumulation of the local anesthetic with continuous, repeated, or inappropriately large dosing.

As with opioids, the pharmacokinetics of local anesthetic metabolism are different in infants. The amide anesthetics are metabolized through the immature cytochrome p 450 system. Infants have a higher steady-state volume of distribution for local anesthetics than do adults, which leads to a prolonged half-life in infants. Local anesthetics are bound by alpha (1) acid glycoproteins; accordingly, a higher fraction of free drug exists in infants.

Infants younger than 4 months of age who receive continuous infusions of local anesthetics (e.g., caudal epidural infusions of bupivacaine) develop increasing plasma levels of bupivacaine. The toxic plasma level of bupivacaine is in the range of 4 mcg/mL. The increasing plasma levels over time, as well as case reports of infants having toxic reactions (seizures, cardiac toxicity) while receiving higher dose infusions, have led to recommendations that infants younger than 4 months of age receive no more than 0.2 to 0.25 mg/kg/hr of bupivacaine. The duration of the infusion should be 48 hours or less. Older infants and children may receive bupivacaine infusions of 0.4 to 0.5 mg/kg/hr.

Ropivacaine has been studied in infants and similar pharmacokinetics apply. An initial epidural dose of 1 to 2 mg/kg, followed by an infusion of 0.2 mg/kg/hr for infants younger than 180 days of age and 0.4 mg/kg for infants older than 180 days of age, is considered safe.

Cardiac toxicity with bupivacaine can be very difficult to treat. Recent reports of adult cases and results from studies done in dogs have suggested that a 20% intralipid infusion may be the first-line therapy. The recommended dose for treatment of local anesthetic toxicity is 1 mL/kg over 1 minute, and it may be repeated twice every 3 to 5 minutes, followed by an infusion at 0.25 mL/kg/min until the patient is hemodynamically stable. P.558

TAKE HOME POINTS

- Select an appropriate pain scale for evaluating the child's pain.
- Family or care giver feedback is necessary in evaluating the child who has cognitive impairment.
- Premature infants, infants younger than 3 months of age, and children with cardiopulmonary compromise, sleep apnea, or neuromuscular disease have a higher risk of adverse events from opioid administration.
- Withdrawal symptoms may occur if administration of opioids is abruptly discontinued after 5 to 7 days of continuous infusion.

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- The potential for local-anesthetic toxicity with use of bupivacaine limits the infusion dose for infants to 0.2 to 0.25 mg/kg/hr.

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Don't Let the Surgeons Discharge Every Pediatric Patient!

Peggy P. Dietrich MD

Robert D. Valley MD

You are at your outpatient surgery center. You run in to see your last patient of the day. It's a 3.5-month-old baby who is scheduled to have an inguinal hernia repair; this case should be a piece of cake! You are confused though when you see the baby. He is awfully little, weighing only 2.1 kg. Speaking with the mother, you find out that she has only had little Tommy at home for the last 2 weeks and that this hernia popped up 2 days ago. Tommy is so tiny because he arrived 14 weeks early. While talking to both the mother and the surgeons, you quickly calculate that he was born at 26 weeks gestation and that he is now just 40 weeks postconceptual age (26 weeks of gestational age plus 14 weeks of chronologic age). Except for the hernia, the mother says all is well. Use of the apnea monitor was discontinued just before Tommy went home. The surgeon says that the hernia needs to be fixed because it is hard to reduce. He asks you, “Can't you just watch him for a couple of hours here then let them go home?” What should you do?

Idiopathic apnea occurs in up to 55% of infants born prior to 37 weeks of gestation and in up to 2% to 3% of full-term infants. All infants, especially those born prematurely, are at risk for postoperative apnea. It is crucial to identify those infants at increased risk for postoperative apnea in order to provide extended monitoring and intervention when necessary.

Apnea is defined as an unexplained pause in breathing lasting 15 to 20 seconds or one lasting less than 15 seconds when associated with bradycardia (heart rate <80), cyanosis, pallor, or marked hypotonia. There are three identifiable types of apnea. Central apnea is characterized by a lack of respiratory effort. Obstructive apnea exists when respiratory effort is present without airflow. Mixed apnea is a combination of both central and obstructive mechanisms.

The most common pattern of apnea in infants has a mixed etiology, withcentral apnea playing the predominant role. Infants, particularly those born prematurely, have an immature central nervous system that manifests as a decreased response to carbon dioxide and a paradoxic response to hypoxia, leading to apnea rather than hyperventilation. Other contributing factors to neonatal apnea include immature intercostal and diaphragmatic musculature, an unstable pliable rib cage, an easily obstructed upper airway, and a lower airway prone to collapse. The long-term consequences of apnea are largely undefined. However, it is reasonable to conclude that hypoxemia associated with repeated apnea increases the likelihood of central nervous system damage. P.560

Anesthesia accentuates a neonate's propensity for apneic events. The first apneic spell can occur up to 12 hours postoperatively. Both inhalational and intravenous anesthetics alter respiratory function. Inhaled anesthetics compromise the infant's immature central nervous system. They have been shown to reduce the central response to respiratory stimulants, including hypercarbia and hypoxia, and to enhance the response to inhibitory afferents. Furthermore, inhaled anesthetics relax pharyngeal musculature, promoting upper-airway obstruction in the neonate who is already prone to obstructive apnea. Intravenous anesthetics, including opioids, also depress the centralnervous-system respiratory centers.

One option for preventing or reducing postoperative apnea in preterm infants is perioperative administration of caffeine citrate (10-20 mg/kg). Caffeine, a methylxanthine, is thought to stimulate breathing by promoting central nervous system excitation, increasing intercostal and diaphragmatic muscle performance, and augmenting chemoreceptor responsiveness to hypercarbia and hypoxemia. Two small studies have demonstrated the usefulness of caffeine to prevent postoperative apnea in infants. Nonetheless, absolute indications for perioperative administration of caffeine remain to be determined by larger studies intended to identify those infants who would most benefit from the drug.

Investigators have attempted to determine whether using regional anesthesia rather than general anesthesia reduces the incidence of postoperative apnea in infants. Arecent meta-analysis included four small studies comparing spinal anesthesia and general anesthesia with respect to the incidence of postoperative apnea following inguinal hernia repair. This meta-analysis failed to demonstrate a statistically significant difference in the percentage of infants with postoperative apnea or postoperative oxygen desaturation based upon anesthetic technique. However, when the patients given preoperative sedatives were excluded, the meta-analysis suggested a lower incidence of postoperative apnea in the spinal anesthetic group. It was concluded that larger studies are needed to clarify the risks and benefits of regional versus general anesthesia in the prevention of postoperative apnea.

Postoperative apnea is reported to have occurred in infants as old as 55 weeks of postconceptual age (postconceptual age = gestational age + age after birth) following extensive surgical procedures. Identifying at-risk infants and determining their need for postoperative monitoring following anesthesia is a great challenge. Many investigators have attempted to identify those infants at greatest risk. In a meta-analysis of eight published reports involving 255 prematurely born infants following inguinal hernia repair, the data revealed that there is a strong inverse relationship between apnea and gestational age and also between apnea and postconceptual age. The authors estimate that the risk of apnea is not less than 1% until postconceptual age is 54 weeks and gestational age is 35 weeks. The meta-analysis found that apnea continuing after hospital discharge and anemia (hematocrit <30), especially for infants older than 43 weeks of postconceptual age, are additional risk factors for postoperative apnea. Finally, the authors determined that the majority of apneic spells resolve without intervention; however, they were unable to identify predictors of the likelihood of self-recovery from an apnea spell. P.561

Based upon reported investigations, general recommendations and guidelines employed at our institution include the following:

1. An infant's need for extended postoperative monitoring is evaluated on a case-by-case basis, considering the patient's gestational and postconceptual age, ongoing apneic events, hematocrit, other comorbidities, and type of surgery.
2. Elective surgery for term infants is delayed until infants are older than 44 weeks of postconceptual age.
3. Premature infants younger than 50 weeks of postconceptual age are admitted and monitored or for at least 18 hours postoperatively. Infants whose postconceptual age is between 50 and 60 weeks are monitored in our postanesthesia care unit for a minimum of 2 hours prior to discharge.

TAKE HOME POINTS

- Postoperative apnea in infants is real.

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- The risk increases with decreasing gestational age at birth and chronologic age at the time of surgery.
- Your facility should have a policy to deal with these high-risk patients in a consistent manner.

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Recognize Predictors and Patterns of Cardiac Arrest in the Anesthetized Child

Kirk Lalwani MD, FRCA

A simple Child, That lightly draws its breath, And feels its life in every limb, What should it know of death? ('We are Seven' - William Wordsworth, 1770-1850)

The death of a child is a tragedy for everyone involved. Unexpected cardiac arrest may occur in anesthetized children despite optimal care. Several factors are known to be associated with an increased risk of anesthesia-related cardiac arrest in children, and the pediatric care provider should be familiar with them in order to minimize the potential risk.

Following the dramatic decline in anesthesia-related mortality rates over the last 50 years, there has recently been a change in the profile of anesthesia-related cardiac arrest in children. Many studies have identified predictors of increased risk for anesthesia-related cardiac arrest in children, but detailed analysis of the root cause of the arrests is based on data collected in the Pediatric Perioperative Cardiac Arrest Registry (POCA). POCA was initiated in 1994 as an offshoot of the American Society of Anesthesiologists (ASA) Closed-Claims Project to identify the most common causes of anesthesia-related cardiac arrest in children and to outline strategies for prevention. The initial POCA findings reported details of 150 cases of anesthesia-related cardiac arrest, which occurred between 1994 and 1997 in 63 North American institutions. The latest report summarizes approximately 300 additional cases submitted from 1998 to 2003.

PREDICTORS OF ANESTHESIA-RELATED CARDIAC ARREST IN CHILDREN

American Society of Anesthesiologists Physical Status.

The strongest predictor of anesthesia-related cardiac arrest in the POCA study was ASA physical status of 3 to 5; such status was associated with a 12-fold increase in the odds of cardiac arrest. ASA physical status as a predictor of increased risk is also supported by data from other studies.

Emergency Surgery.

Emergency surgery was associated with a three-fold increase in risk for cardiac arrest in the POCA study, as well as with an increased risk of other anesthetic complications in a landmark French study. P.563

Age.

The risk of anesthetic complications and cardiac arrest in children varies inversely with age. Neonates and infants are particularly at risk. Approximately 50% of all arrests in the POCA registry occurred in infants (<1 year of age), and 15% occurred in neonates (<1 month of age). Of note, however, is that when underlying disease severity was accounted for (i.e., ASA status), age alone was not a predictor of death in the POCA study.

CHANGING PROFILE OF ANESTHESIA-RELATED CARDIAC ARREST IN CHILDREN

Causes of Cardiac Arrest.

Of the 150 cases of cardiac arrest submitted to POCA, 37% were related to medication. Two thirds were likely due to halothane, alone or in combination with other drugs. Medications were also deemed responsible for 64% of arrests in patients whose ASA physical status was 1 or 2. Based on the analysis of an additional 163 anesthesia-related cases added to the registry, medication-related causes declined from 37% to 20%, primarily due to a decline in cardiac depression induced by volatile agents. Cardiovascular causes of arrest are now the most common, increasing from 32% to 36%. In this category, hypovolemia (frequently due to hemorrhage) and hyperkalemia (secondary to massive transfusion) were the most frequent causes of arrest. Respiratory causes increased from 20% to 27%, with laryngospasm, airway obstruction, inadvertent extubation, difficult intubation, and bronchospasm being the most frequent events. Equipment-related arrests (4%) were commonly due to complications of central-venous-pressure (CVP) catheter placement. Assorted other causes included complex cyanotic congenital heart disease, pulmonary hypertension, myocarditis, prolonged QT syndrome, coronary artery disease, hypertrophic cardiomyopathy, topical vasoconstrictor use, and anaphylactic reactions.

Demographics of Cardiac Arrest.

The percentage of patients whose ASA physical status was 1 or 2 decreased from 33% in 1998 to 27% in 2003, along with the percentage of infants younger than 1 year of age (55% to 36%, $p<0.05$). The POCA investigators attribute the decline in medication-related arrests to these changes; halothane was often responsible for cardiac arrest in infants whose ASA physical status was 1 or 2. The mortality rate did not change significantly from 1998 to 2003 (26% in 1998, 28% in 2003), despite the altered profile of reported cases.

STRATEGIES TO PREVENT CARDIAC ARREST IN ANESTHETIZED CHILDREN

Specific Measures

Sevoflurane.

Halothane has potent negative inotropic and chronotropic effects that can easily produce profound myocardial depression and a precipitous fall in cardiac output in infants and neonates, particularly when the ability of the halothane vaporizer to deliver concentrations in excess of six MAC-multiples is taken into account. The dramatic decline in medication-related deaths in the POCA study has been attributed to the widespread replacement of halothane with sevoflurane for pediatric anesthesia; sevoflurane has minimal effects on heart rate and myocardial contractility. P.564

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Local Anesthetics.

Local anesthetics were used in 3.3% of cases of cardiac arrest reported in the initial POCAseries. Meticulous technique is essential to prevent inadvertent intravascular injection of local anesthetics, which may result in cardiac arrest. Accurately placing the needle, carefully aspirating before injecting, using epinephrine-containing test doses, continuously monitoring electrocardiograms during dosing, and incrementally injecting the local anesthetic solution are some of the precautions used to reduce the incidence of this complication. In the future, widespread adoption of ultrasound-guided regional nerve blocks may significantly decrease the incidence of local anesthetic-related cardiac arrest. Should intravascular injection occur, studies done in animals and anecdotal case reports suggest that recovery from cardiac arrest may be more likely after ropivacaine use than after bupivacaine use. In addition, the use of 20% injectable fat emulsion such as Intralipid® (Fresenius Kahi) reduces the mortality of local anesthetic-induced arrest in animals. Anecdotal reports of human cases suggest Intralipid may be clinically useful and, given its innocuous nature, should be available in locations where regional-anesthetic blocks are placed.

Hypovolemia.

In the POCAstudy, cardiac arrest due to hypovolemia occurred as a result of failure to manage hemorrhage adequately or as a result of hyperkalemia precipitated by massive transfusion. Inadequate intravenous access and failure of the anesthesiologist to keep up with blood loss were implicated in the hypovolemic arrests that occurred secondary to hemorrhage. Hyperkalemia related to massive transfusion occurs as a result of potassium leakage from stored red cells into the plasma. It can be minimized by using packed red blood cells instead of whole blood, requesting the freshest cells available, and avoiding irradiated blood unless indicated. In high-risk situations, such as cases involving infants and children requiring more than one blood volume of replacement and cases in which irradiated blood is necessary (e.g., an immunocompromised child), packed red cells should be washed by the blood bank, and intraoperative serum potassium levels should be carefully monitored.

Central venous pressure Catheter Placement.

Cardiac arrest secondary to CVP catheter placement occurred as a result of cardiac tamponade, pneumothorax, or hemothorax in the POCAstudy. The closed-claims study reported that either ultrasound guidance or pressure waveform analysis would have prevented almost 50% of CVP catheter-placement-related complications. Routine use of an ultrasound device for placement is strongly recommended to increase success rates and decrease complications. P.565

Succinylcholine.

Use of succinylcholine is indicated in pediatric anesthesia cases with difficult or emergency airway management, and rapid-sequence induction is indicated for pediatric patients with a full stomach. However, routine use of succinylcholine has caused several deaths from hyperkalemia in children with undiagnosed muscular dystrophies, such as Duchenne's muscle dystrophy (DMD), prompting the manufacturer to change the recommended indications for succinylcholine use in children. Succinylcholine should not be used in children except as described above. Rocuronium provides adequate intubating conditions for rapid-sequence induction, and can be used in most instances.

Muscular Dystrophy.

Children with progressive muscular dystrophy (Duchenne's and Becker's) are prone to rhabdomyolysis following the use of succinylcholine or volatile anesthetic agents. Arecent editorial has questioned the use of volatile anesthesia (without muscle relaxants) for these patients following the report of yet another death in a child with undiagnosed DMD. Since we are unable to predict which children with DMD will develop clinical rhabdomyolysis following volatile anesthesia, avoiding use of volatile agents in these children and using an intravenous technique instead would be prudent. Since 90% of children with DMD have a family history of DMD, careful questioning about family members and eliciting the child's developmental milestones may trigger suspicion in asymptomatic children that merits preoperative creatine phosphokinase measurement.

General Measures

Perioperative Environment.

The American Academy of Pediatrics has issued guidelines for the creation of a specialized environment for the provision of care for children. These guidelines encompass provider training, experience, and credentialing and the availability of appropriate equipment, drugs, and necessary support services, such as radiology and intensive care.

The Scope of Practice.

Anesthetic outcomes for children are improved when anesthesiologists who are trained or experienced in the care of children are involved. Similarly, for specialized areas, such as congenital heart surgery, high-volume centers have lower mortality rates. Therefore, it is appropriate to transfer such patients to referral centers. The United Kingdom recognized this issue many years ago and has formulated criteria for specialist pediatric centers and providers in the National Health Service.

Continuous Quality Improvement.

Continuous Quality Improvement (CQI) programs include morbidity and mortality conferences and critical incident registries to monitor key quality indicators, identify systematic problems, and implement strategies for the prevention of bad outcomes. Institutional and departmental compliance with professional society guidelines establishes a standard to use in comparing the quality of care among institutions. P.566

CONCLUSION

Rita Mae Brown, the social activist and author, once said that “good judgment comes from experience, and often experience comes from bad judgment.” Pediatric care providers would be wise to study the collective experience of others afforded by elegant database tools such as POCAto heighten their awareness of the risk factors for cardiac arrest, so as to make good judgments without the burden of personal experience.

TAKE HOME POINTS

- Beware of high-risk situations such as cases involving children whose ASA physical status is 3 to 5, cases involving neonates and infants, and cases involving emergency surgery.
- Use sevoflurane instead of halothane.

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- Take care to avoid laryngospasm, and treat it promptly; be vigilant for respiratory obstruction or inadequate ventilation.
- Pay meticulous attention to needle placement and technique for regional anesthesia; use ropivacaine instead of bupivacaine, and become proficient with ultrasound-guided blocks.
- Ensure adequate intravenous access, and assess blood loss relative to blood volume frequently.
- Order fresh or washed packed red cells for infants, and monitor serum potassium levels.
- Place all CVP lines with ultrasound guidance, and don't forget to order a postoperative chest X-ray.
- Use succinylcholine only when absolutely necessary.
- Avoid succinylcholine and volatile agents with suspected or known muscular dystrophy.

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Editors: Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.
Title: *Avoiding Common Anesthesia Errors, 1st Edition*

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The Neurophysiology You Learned in Medical School Really Does Matter During Craniotomies

Jennifer J. Adams MD

Laurel E. Moore MD

You're a new CA-2 carrying the code pager for the *first* time. You are called to the neurosurgical intensive care unit (ICU) for an emergent intubation. On arrival, you find serious badness. Seems a middle-age man scheduled to undergo craniotomy for tumor the next day has become progressively more unresponsive, and in the last 5 minutes, his right pupil has dilated and become unresponsive to light. The ICU team wants to transport him for an emergent head CT but wisely called you first. What is your management plan?

In caring for the patient with intracranial hypertension, the role of the anesthesiologist is ultimately to maintain a favorable balance between the delivery of oxygen (and glucose) and the brain's metabolic requirements. Under normal circumstances, flow and metabolism are elegantly controlled to maintain a physiological balance. Under pathological conditions, however, anesthesiologists have a unique role in affecting oxygen delivery and metabolism in order to try to improve oxygen delivery to potentially ischemic brain. For key terms and definitions, see Table 132.1.

When considering oxygen delivery to the brain, it is important to remember that the brain is contained within the indistensible skull. Under normal circumstances, the contents of the skull can be divided into three categories: Brain (80% of intracranial volume), cerebrospinal fluid (CSF) (15%), and blood (5%). Under pathological conditions, tumor or hematoma may gradually or acutely increase intracranial volume. Under chronic conditions, compensatory mechanisms to offset this increasing intracranial volume include increased reabsorption of CSF or diversion of CSF to the spinal canal. Once these compensatory mechanisms are exhausted, however, intracranial pressure (ICP) rapidly increases within the intracranial vault (Fig. 132.1) and cerebral perfusion pressure (CPP) is compromised:

CPP = MAP - ICP

where MAP is mean arterial pressure. Acute increases in intracranial volume, such as seen with head trauma or intracranial hemorrhage, are much more poorly tolerated, and ICP may rise precipitously.

TABLE 132.1 KEY TERMS AND DEFINITIONS

TERM	ABBREVIATION	DEFINITION	UNITS	NORMAL RANGE
Cerebral blood flow	CBF		mL/min/100 g	45-65 mL/min/100 g globally
Mean arterial pressure	MAP	MAP = [SBP + 2(DBP)]/3	mm Hg	Adults: 60-90 mm Hg
Intracranial pressure	ICP		mm Hg	8-12 mm Hg
Cerebral perfusion pressure	CPP	CPP = MAP - ICP	mm Hg	50-70 mm Hg
Cerebral metabolic rate of oxygen	$CMRO_2$	CBF × (CaO_2 - $C_{jv}O_2$)	mL/min/100 g	3.0-3.8 mL/min/100 g
Cerebral vascular resistance	CVR	CPP/CBF	mm Hg/mL/min/100 g	1.5-2 mm Hg/mL/min/100 g

CaO_2, oxygen content arterial blood; $C_{jv}O_2$, oxygen content jugular venous blood.

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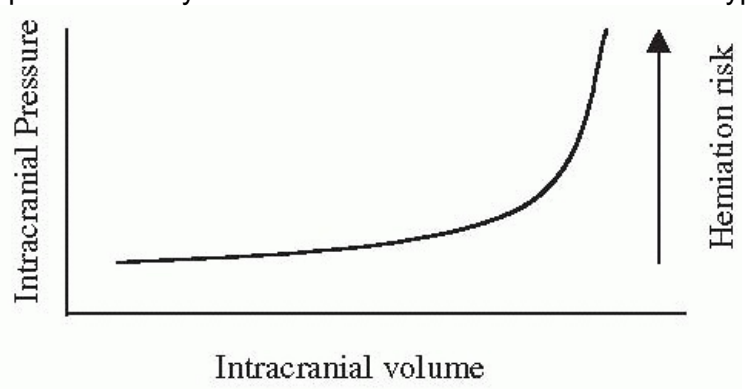


FIGURE 132.1. The intracranial compliance curve demonstrating a stable intracranial pressure (ICP) until some critical intracranial volume is reached, at which time ICP increases exponentially.

Increasing ICP not only places the brain at risk for inadequate oxygen delivery, but there is also the danger of herniation of cerebral tissue, which can rapidly produce death. Cerebral herniation may be associated clinically with Cushing's triad, which includes (a) hypertension, (b) bradycardia, and (c) breathing irregularities.

Under normal circumstances, there are four major determinants of cerebral blood flow (CBF):

1. $PaCO_2$: Carbon dioxide is the major determinant of CBF, and CBF varies directly with $PaCO_2$ between the range of approximately 20 and 80 mm Hg. Below a $PaCO_2$ of 20 mm Hg or above 80 mm Hg, vasoconstriction or vasodilation, respectively, is near maximal, and no further effect of $PaCO_2$ is seen on CBF beyond these limits (Fig. 132.2). It is important to remember that this effect is *temporary*, lasting only 6 to 8 hours. The result of this is that increased hyperventilation can afford additional benefit in patients with intracranial hypertension 6 to 8 hours after initiating hyperventilation. Conversely, rapidly normalizing CO_2 can markedly increase CBF in patients with abnormal intracranial compliance who have been treated with hyperventilation for hours to days.
2. PaO_2: Under normal circumstances, arterial oxygenation has little to no effect on CBF. However, under hypoxic conditions (PaO_2 <50 mm Hg), CBF rapidly increases to maintain oxygen delivery to the brain (Fig. 132.2).
3. $CMRO_2$: As described previously, CBF and $CMRO_2$ are closely aligned in normal brain. Drugs that reduce $CMRO_2$ and thus may benefit patients with intracranial hypertension include barbiturates, etomidate, propofol, benzodiazepines, opioids, and lidocaine. The potent inhaled vapors (excluding nitrous oxide) not only reduce $CMRO_2$ but also act as cerebral vasodilators. Thus, they are not generally considered beneficial in patients with abnormal compliance. Hypothermia is also highly effective at reducing $CMRO_2$. Its clinical relevance, however, is unproven.

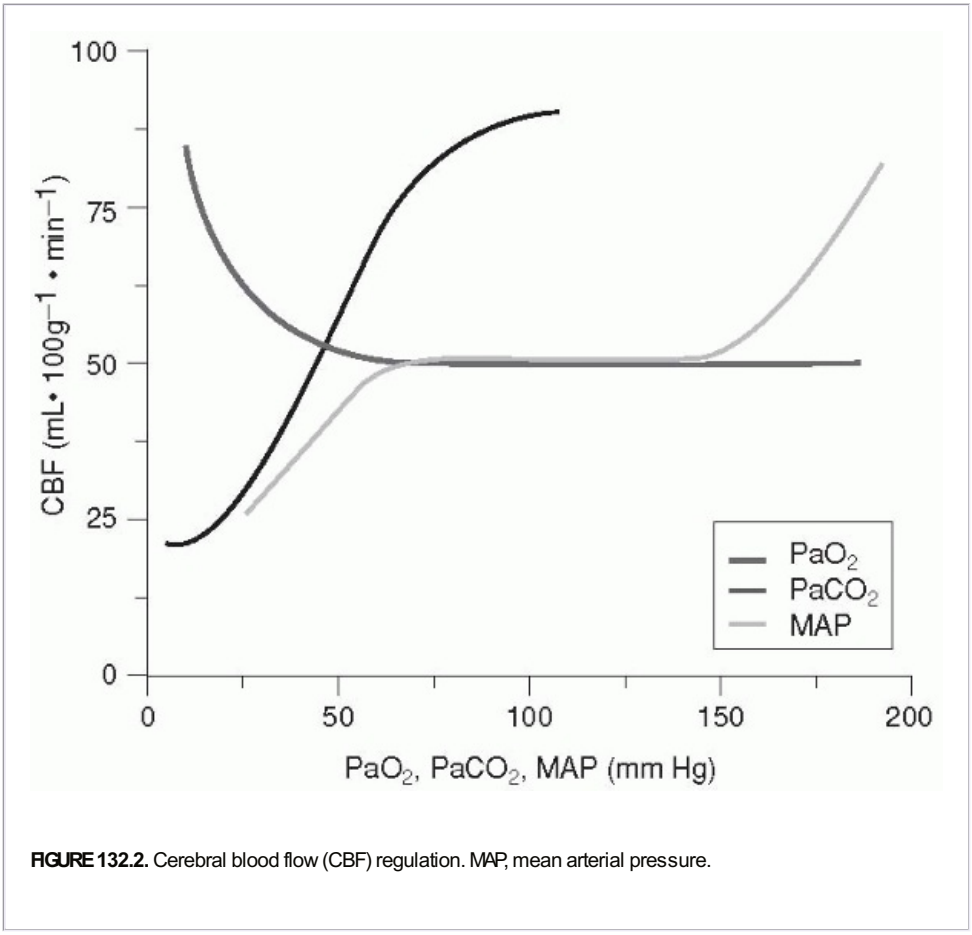


FIGURE 132.2. Cerebral blood flow (CBF) regulation. MAP, mean arterial pressure.

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4. CPP: Normal brain is capable of autoregulation, which means that it maintains a constant CBF over a wide range of CPPs (Fig. 132.3, uninterrupted line). Under pathological conditions, however, autoregulation may be impaired or absent (Fig. 132.3, interrupted line). Some of these conditions include severe brain trauma or subarachnoid hemorrhage. It is critical to maintain a normal or even elevated CPP in these patients so as to maintain adequate oxygen delivery to ischemic regions as perfusion becomes "pressure passive." P.578

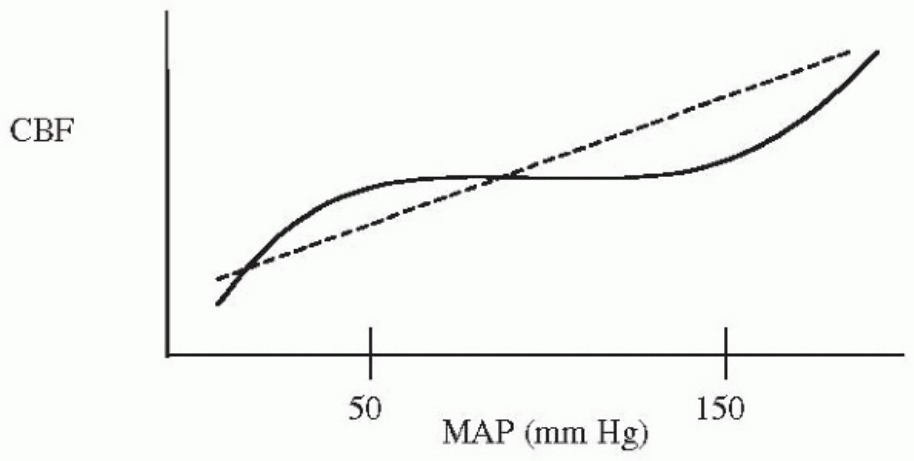


FIGURE 132.3. Diagram depicts cerebral autoregulation (solid line) defined as capacity to maintain a constant cerebral blood flow (CBF) over a wide range of mean arterial blood pressures (MAP). Interrupted line demonstrates pathologic loss of autoregulation in which CBF becomes dependent upon adequate MAP.

These factors, generally controlled by anesthesiologists, have addressed the control of arterial blood flow that, as stated previously, represents a small percentage of total intracranial volume. It is important to remember other modes of reducing ICP, thus optimizing CPP. Reduction of the brain/tissue component contributing to intracranial hypertension falls into the realm of the neurosurgical team. Maneuvers that improve cerebral venous drainage are extremely important to managing ICP and include placing the patient head up and minimizing obstruction to venous drainage (i.e., minimizing airway pressures and intrathoracic pressure). Cerebral interstitial fluid may be rapidly reduced by agents such as mannitol or furosemide. Mannitol, an osmotic diuretic, draws fluid from the interstitial space into the intravascular space. Steroids, when appropriate (e.g., brain tumors), may be administered to minimize tissue inflammation and edema.

TAKE HOME POINTS

- In summary, the anesthesiologist has a unique role to play in the care of patients with intracranial hypertension.
- In the patient example at the beginning of this chapter, it is appropriate to emergently intubate the patient to initiate hyperventilation.
- Drugs to reduce $CMRO_2$ will also be helpful in the acute control of CBF and ICP.
- At this point in time, our understanding of cerebral physiology remains trivial at best. What is occurring in this patient at the level of the microcirculation remains a mystery, essentially a "black box." Until we have ways to evaluate and quantify the balance of oxygen delivery and demand at the cellular level, however, these principles apply to the care of our patients. It is the challenge presented by this "black box" that makes our role as anesthesiologists so exciting and challenging.

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Anesthetic Goals for Cerebral Aneurysm are not the Same as for Routine Craniotomy

James S. DeMeester MD

Cerebral aneurysms typically arise in the circle of Willis at vascular bifurcation points where hemodynamic stress is maximal. Ninety percent of aneurysms occur in the anterior circulation, and only 10% occur in the basilar system. Although cerebral aneurysms, if large enough, can manifest with symptoms of neural compression, the greatest concern is the occurrence of rupture and subarachnoid hemorrhage.

Intracranial aneurysms (ICAs) are responsible for 75% to 80% of episodes of subarachnoid hemorrhage (SAH), which has an incidence of 10-20/100,000, and is associated with high morbidity and mortality. One-third of patients will die from their initial bleed, with another third having severe disability or delayed death. Only the remaining third will have minimal morbidity and an acceptable outcome.

The prevalence of undiagnosed, asymptomatic aneurysms is estimated at 4%; however, surgical clipping only confers significant outcomes benefit when aneurysm size exceeds 10 mm. Timing of surgical intervention becomes more critical after a hemorrhage because the initial 72 hours presents a window for operative management, after which surgery is delayed 10 to 14 days until the risk of vasospasm has decreased. When surgery is indicated, the anesthesiologist must realize that anesthetic considerations for SAH and ICAare unique from those of routine craniotomy.

The primary concern of an anesthesiologist during surgery for aneurysm clipping is the prevention of rupture. ICArupture at the time of induction has a mortality exceeding 75%. The likelihood of rupture is based on aneurysm size, wall strength, history of prior rupture, and transmural pressure. Transmural pressure is

CPP = MAP - ICP

where CPP is cerebral perfusion pressure and MAP is mean arterial pressure. Although mathematically equal to CPP, the concept of transmural pressure and its concern with aneurysmal wall stress is unique to aneurysms. The periods commonly associated with intraoperative rupture are at induction, dura and arachnoid exposure, hematoma evacuation, and during dissection to the aneurysm. Rupture during dissection, or at the time of aneurysm clipping, carries with it much lower morbidity because it is usually rapidly controlled. Anesthetic priorities after a rupture are to maintain cerebral perfusion. P.580
Controlled hypotension in an attempt to reduce bleeding is detrimental *but may be necessary under emergent conditions (e.g., aneurysmal rupture) to enable the neurosurgeon to clip the feeding vessel or aneurysm itself.*

Anesthetic induction should be free from acute increases in blood pressure while preserving CPP. An awake arterial line and an induction with adjunct agents, such as lidocaine, beta blockade, and narcotic, facilitate a smooth induction. Amodification from our management of routine craniotomy is the avoidance of aggressive hyperventilation and hypocapnia at induction. This prevents precipitous decreases in ICP, further increasing the transmural pressure gradient and the risk for rupture. Likewise, aggressive cerebrospinal fluid drainage at this stage can also have similar consequences.

Patients who have experienced a subarachnoid hemorrhage may have regional ischemia and dysfunctional cerebral blood flow (CBF) autoregulation. Accumulation of extravascular blood increases ICP close to MAP, diminishing the perfusion gradient. Extravasated blood also contributes to localized vasospasm and furthers the risk for ischemia. Vasospasm is rare in the first 3 days following a bleed. Risk peaks around day 7 and generally resolves around days 10 to 14. Clues to the presence of vasospasm, especially preoperatively, might include changes in mentation or new neurologic deficits. Newly evolving hemiplegia may indicate involvement of the middle cerebral artery because hemodynamic and respiratory changes may suggest involvement of the posterior circulation. The diagnosis is also made, more accurately, with angiography and transcranial Doppler. Vasospasm has been seen angiographically in 70% to 90% of patients with SAH; however, most symptoms are absent under anesthesia, making its diagnosis challenging.

Preventative and therapeutic interventions for vasospasm include hemodilution, hypertension, and hypervolemia, referred to as “triple H” therapy or HHH. HHH therapy is a strategy to augment CBF past strictures by increasing CPP and intravascular volume. Although acute hypertension should be avoided, maintenance of MAP and volume should be kept high-normal. Pressors are indicated if hypervolemia alone is insufficient. Arule of thumb should be to keep the MAP at normal prior to aneurysm clipping, and high-normal after both temporary and definitive clipping. Remember that HHH therapy is not indicated for elective aneurysm clipping because vasospasm only occurs after the accumulation of *subarachnoid blood*.

Once induction is complete, further positioning, head pinning, and additional neuromonitoring is placed (somatosensory-evoked potential, electroencephalogram). These carry the risk of acute hypertension, and require aggressive blood pressure control and a stable plane of anesthesia. The anesthetic goals during the maintenance period of anesthesia are similar to that of routine craniotomy in that cerebral relaxation is desired. Gentle hyperventilation with the addition of osmotic diuresis facilitates a slack brain, facilitating P.581
surgical dissection. The technique of controlled hypotension during dissection of the aneurysm has been largely supplanted by the use of temporary clips. This lessens the risk of aneurysm rupture or rebleeding during the dissection phase. MAP should be increased after the clip is deployed to enhance collateral perfusion. An isoelectric EEG may be requested during this time, and is achieved by bolus and infusion of propofol. Vasopressors are commonly needed to prevent hypotension.

The goals for anesthesia wake up are to have a comfortable patient free from straining, coughing, and hemodynamic lability. *Patients who are status post subarachnoid hemorrhage and at risk for vasospasm should be intravascularly replete with a normal-to-high normal mean arterial blood pressure.* If surgery was indicated for SAH, then patients who are Hunt-Hess grades I and II should be able to be extubated, facilitating a prompt neurologic examination. Patients who are Hunt-Hess grades III and IV are usually left intubated.

TAKE HOME POINTS

- Anesthetic considerations for surgery for SAH and ICA are unique from those of routine craniotomy.
- The overriding concern is to avoid rupture of the aneurysm and/or subarachnoid hemorrhage.
- ICA rupture at induction carries a mortality rate exceeding 70%!
- Avoid aggressive hyperventilation and hypocapnia at induction.

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- Controlled hypotension during routine dissection of the aneurysm has been largely replaced by the use of temporary clips, but be prepared for controlled hypotension in the case of emergent clipping of an acutely ruptured aneurysm.
- Extubation after aneurysm surgery generally depends on the preoperative Hunt-Hess grade—patients who are grades I or II are generally extubated, whereas patients who are grades III or IV usually have a course of postoperative intubation.

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Evoked Potentials: Don't Approach the Surgeon and Neurophysiologist Until You Know These Principles

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Intraoperative neurophysiological monitoring is used to decrease neurosurgical morbidity and detect neurologic compromise before the damage becomes irreversible. There are numerous modalities for intraoperative monitoring, including somatosensory-evoked potentials (SSEPs), motorevoked potentials (MEPs), brainstem auditory-evoked potentials (BAEPs), and visual-evoked potentials (VEPs). Their selection is based on the area at risk for injury. Monitoring evoked potentials (EPs) can be challenging for the anesthesia provider because many of the anesthetic agents currently used affect signal quality. The anesthesiologist or anesthetist must communicate with the surgeon and neurophysiologist prior to surgery regarding what EPs will be monitored in order to decide on an anesthetic plan that will optimize this monitoring.

EPs are measurements of the electrical potentials produced when the nervous system is stimulated in contrast to the electroencephalogram [EEG], which records spontaneous electrical activity generated by the central nervous system. EPs may be generated by sensory, magnetic, electrical, or cognitive stimulation. They are characterized by both latency and amplitude. The latency of an EP is measured in milliseconds and is the time between a stimulus and the occurrence of the EP. The amplitude is measured in millivolts and is the magnitude of the EP. EPs can be monitored noninvasively from electrodes on the skin or invasively by monitors placed within the surgery field.

SSEPs are in the form of intraoperative neurophysiological monitoring with the widest clinical application. This technique includes stimulating a peripheral sensory nerve repetitively (e.g., median nerve or posterior tibial nerve) and recording the response with electrodes placed over the primary sensory cortex. SSEPs use signal averaging (e.g., repetitive stimulation and response recording) to cancel out background noise (e.g., EEG). In certain surgeries, the recording electrodes can be placed directly in the epidural space immediately proximal to the site of interest. SSEPs monitor the well-being of the dorsal column functions (position, vibratory sense, and light touch) as well as portions of the brainstem and cerebral cortex. Specifically, the pathway includes the peripheral sensory nerves (cell bodies in the dorsal root ganglia) → which ascend via the ipsilateral dorsal column to synapse in the medulla → secondary fibers decussate and ascend to the contralateral thalamus → tertiary fibers ascend from the thalamus to the primary sensory cortex (postcentral gyrus). Note that SSEPs provide little to no information on the anterior columns (read: Motor function), and there are reports of patients with normal intraoperative SSEPs who awaken with new motor deficits. Fortunately, this is rare despite the dorsal columns having a different blood supply (posterior spinal arteries) than the anterior columns (anterior spinal artery). However, it is theoretically possible to have hypoperfusion to the anterior columns (e.g, during a thoracic aortic aneurysm repair) that SSEPs may fail to recognize. SSEPs are monitored during aneurysm surgery (median nerve during middle cerebral artery aneurysms, posterior tibial nerve during anterior circulation aneurysms—remember your homunculus!), brain tumors including the posterior fossa, and some spine surgeries.

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As spine surgery becomes more complex, the need to monitor the anterior columns is becoming increasingly important. Fortunately, our technical ability to monitor MEPs has improved rapidly since the 1990s. MEPs entail applying a current directly or transcranially to the primary motor cortex (precentral gyrus) or spinal cord to initiate an action potential. The action potential then descends from the motor cortex through the pyramidal decussation to the contralateral lateral corticospinal tract. These neurons then synapse on the ventral horn with an alpha motor neuron that travels to the muscle. MEPs can be measured at numerous points along this pathway. Neurogenic MEPs are responses recorded in the periphery following stimulation of the spinal cord. Myogenic MEPs are EPs recorded over the muscle belly as compound muscle action potentials (CMAPs).

BAEPs and VEPs are two additional forms of EPs that may be monitored intraoperatively. BAEPs record subcortical responses from auditory stimuli and monitor the entire auditory pathway, including brainstem nuclei. These prove useful in surgeries near the cerebellopontine angle and particularly help with auditory preservation during resection of acoustic neuromas. VEPs record cortical responses from visual stimuli and monitor the visual pathway. This form of EPs is useful in surgeries near this pathway (i.e., parasellar region). Unlike BAEPs, VEPs are exquisitely sensitive to anesthetic agents.

Because all anesthetic agents have effects on the central nervous system, the anesthetic management of patients undergoing neurophysiological monitoring is vital. Synaptic transmission is more sensitive to anesthetic agents than mere axonal conduction. Therefore, those pathways with multiple synapses (e.g., MEPs, VEPs) are more sensitive to anesthetic agents than those pathways with few (e.g., subcortical SSEPs or BAEPs). The typical effect of most anesthetic agents is to decrease the amplitude and increase the latency of EPs. Anesthetic agents with these effects include the halogenated inhalational agents, propofol, benzodiazepines, and barbiturates. Nitrous oxide has depressant effects on amplitude and minimal effects on latency. Conversely, ketamine and etomidate have minimal effects on EPs and may enhance SSEP amplitude. Opioids also have minimal effects on SSEPs and MEPs, making them a popular choice in EP monitoring.

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Neuromuscular blockade must also be carefully considered when planning anesthetic management. By decreasing neuromuscular transmission, neuromuscular blockers produce a dose-dependent decrease in CMAP amplitude, thus affecting the MEP. Similarly, if electromyography (EMG) is being done (e.g., facial nerve monitoring), profound relaxation will limit signal strength. As one might expect, neuromuscular blockers have no effect on sensation, and therefore, do not interfere with the transmission of an electric potential from the periphery to the central nervous system. Paralysis, in fact, aids in SSEP monitoring because it decreases electromyographic interference. Although neuromuscular blockade decreases the ability to monitor MEPs and EMGs, some degree of relaxation may be needed to help with surgical exposure and to prevent patient movement. Working with the neurophysiologist, a balance can be found to satisfy both the needs of the surgeon and the neurophysiologist. Care should be taken to pad extremities and pressure points, as well as to protect the tongue and oropharynx, to prevent injury during MEP stimulation in patients who are not fully relaxed.

Once the surgical procedure and type of neurologic monitoring is known, anesthetic management can be planned. The anesthetic should provide for hemodynamic stability and patient comfort, while permitting optimal conditions for neuromonitoring. This entails the use of a stable anesthetic and avoidance of medication boluses. If a change is noted in amplitude or latency, it is vital that there be no confusion as to whether the change is due to surgical manipulation or a change in anesthetic management. Furthermore, in the event of a significant change in signals, the possibility of an intraoperative wake-up test should be considered when planning the anesthetic. Medications that are rapidly titrated, such as desflurane, nitrous oxide,

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short-acting opioids, and low-dose propofol, generally provide for good signals and a rapid wake up should this become necessary. For MEPs, particularly in myelopathic patients who have abnormal signals, consideration should be given to using propofol in combination with ketamine, although whether this is better than low-dose volatile agent is unclear. If there is a need to determine whether a loss of signals is real or artifact (e.g., needle displacement), a bolus of etomidate may accentuate poor signals provided that the hardware is in place.

A variety of nonsurgical factors such as temperature, hypotension, anemia, and even brain shrinkage (from cerebral spinal fluid drainage) can affect signals over time. When do these changes become significant? In general, a decrease in amplitude of ≥50%, or a ≥10% increase in latency, deserves investigation. Sudden or unilateral changes certainly suggest surgical trespass. More gradual or bilateral changes are suggestive of anesthetic or physiological change. Regardless of the circumstances, when there is a significant change in signals, the anesthesia team should evaluate (a) whether MAP is adequate and consider increasing MAP 20% above baseline, (b) check hemoglobin and oxygen saturation to ensure adequate oxygen delivery, and (c) ensure that the anesthetic technique has remained stable.

Last, communication is a vital portion of successful EP monitoring. The anesthesiologist must communicate with the neurophysiologist and surgeon about changes in anesthetic management or physiological parameters that may result in changes in signals. Likewise, the neurophysiologist must communicate with the anesthetic and surgical teams of any significant change in signals so they may respond appropriately.

TAKE HOME POINTS

- EP monitoring is actually a "family" of intraoperative monitoring modalities used to decrease neurosurgical morbidity and detect neurologic compromise before the damage becomes irreversible.
- The anesthesia provider should expect to interface with the surgeon, the neurologist, and the EP technician concerning the specific technique to be used for a given case.
- Noninvasive monitoring on the extremities is usually done by placing pad electrodes on the skin, but **beware,** often needle electrodes are used on the scalp.
- SSEPs are the form of intraoperative neurophysiological monitoring with the *widest* clinical application.
- The use of MEPs has increased as spine surgery has become more complex.
- The typical effect of most anesthetic agents is to decrease the amplitude and increase the latency of EPs. Anesthetic agents with the most pronounced effects include the halogenated inhalational agents, propofol, benzodiazepines, and barbiturates. Opioids have minimal effects on SSEPs and MEPs, making them a popular choice in EP monitoring cases.
- Always be ready to re-evaluate your anesthetic if a significant change occurs in the EPs, and always be ready for a wake-up test.

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Don't Treat Hypertension in Neurosurgery Cases Before Considering the Cause(s) and Risks, and Always Recognize that An Abrupt Change in the Patient's Blood Pressure Needs to Be Investigated

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The appropriate management of hypertension during neurosurgical cases requires knowledge of normal and pathological cerebrovascular physiology and consideration of the multiple causes of increased blood pressure. Like all things in anesthesia, simply "treating a number" without considering a cause risks worsening the patient's condition.

Cerebral blood flow (CBF) depends on an adequate cerebral perfusion pressure (CPP) and is well maintained via autoregulation over a wide range of mean arterial pressures (MAPs) in the normally functioning brain. CPP is determined by the difference between MAP and intracranial pressure (ICP):

CPP = MAP - ICP

Simply put, the body's blood pressure, minus the pressure inside a nonexpanding calvaria, gives the perfusion pressure, which drives blood into the cerebral vasculature. Classically, CBF is maintained at a relatively constant level between MAPs of 50 to 150 mm Hg. Because the "average" ICP rests around 10 mm Hg and MAP for healthy adults is generally around 80 mm Hg, a goal CPP of 70 mm Hg should keep normotensive patients safely within the range of autoregulation. Some investigators have therefore adopted this CPP goal; however, there is no "standard" CPP target due to individual variability and disease state.

Chronic hypertension shifts the autoregulatory curve to the right (Fig. 135.1). This means that even "normal" MAPs may be relatively too low to maintain adequate CBF. This stresses the importance of knowing the patient's baseline blood pressure values and taking them into consideration before treating hypertension. Also, autoregulation may be obliterated by certain conditions, such as ischemia (either by stroke or manual surgical retraction), and trauma. Without this ability to constrict blood vessels to maintain adequate CPP, MAPs that are adequate to perfuse a normal brain may be insufficient in the injured one.

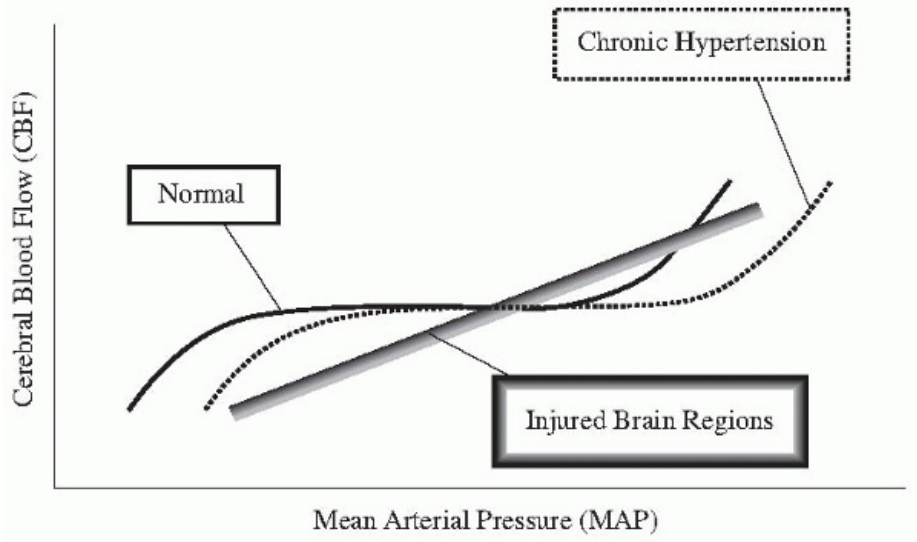


FIGURE 135.1. Autoregulatory curves in three different scenarios: Normal brain function, chronic systemic hypertension resulting in a right-shift of MAPs to maintain cerebral blood flow, and injured brain, resulting in a loss of cerebral vascular autoregulation. (Adapted from Miller RD. *Miller's Anesthesia.* 6th ed. Philadelphia, Pa.: Elsevier/Churchill Livingstone; 2005.)

From the previous formula, one can easily see how intracranial mass, hydrocephalus, or edema, each causing an increase in ICP, will lower CPP, possibly compromising CBF if the MAP is not adequately maintained. The body accommodates for elevated ICP by elevating blood pressure. The Cushing reflex, a triad of hypertension, bradycardia, and respiratory changes, demonstrates the body's attempt to maintain an adequate CPP when ICP increases. Because higher MAPs are necessary to maintain CPP, one can see how treating mild to moderate hypertension in this instance could be detrimental.

For prophylaxis and treatment of subarachnoid hemorrhage (SAH), "triple H" therapy, or hypertension, hypervolemia, and hemodilution (HHH), has traditionally been used to prevent cerebral vasospasm. In theory, increasing CBF by elevating the CPP would improve ischemia caused by cerebral vasospasm, a common complication following SAH. Although no "hard" evidence exists to support routine use of HHH, there is likely benefit in maintaining moderate hypertension in certain patients,and more important, in avoiding hypotension in this patient population.

Of course, like all things in medicine, context is everything. There are certainly times in neurosurgical cases when you want to reach for your labetalol.

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Keeping CPP adequately elevated is a good thing, but there are limits. Although no standard CPP goal has been established, many neuroanesthesiologists try to keep the CPP between 70 and 100 mm Hg, and usually closer to 70 mm Hg in certain brain injuries. Higher CPP (beyond the bounds of autoregulation) leads to an increase in CBF, causing a possible increase in ICP in a poorly compliant cranium.

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So how do you decide which numbers to treat and which to watch? The best answer is to gather all the information you can about a patient. Ideally, one should consult with the surgery team to discuss CPP goals, the potential for increased ICP, and the surgery plan. Also, preoperative blood pressures and nature of the patient's neurologic diseases and comorbidities are necessary to make an informed decision on when to treat.

For certain neurosurgical procedures, hypertension can be almost expected. For example, percutaneous treatments for trigeminal neuralgia have traditionally been performed with an awake or partially awake patient to facilitate placement. This often painful procedure, combined with a patient population that has a higher proportion of chronic hypertensives, leads to increased intraoperative blood pressures. Knowing this, one can see how preoperative antihypertensives combined with vigilant pain control may be more effective than treating hypertension acutely intraoperatively.

TAKE HOME POINTS

- Don't just treat the numbers—think through the physiology and investigate the cause.
- Start your management decisions with firm knowledge of the patient's baseline blood pressure and remember that chronic hypertension shifts the curve to the right.
- There is likely some benefit to at least the hypertension component of “triple H” therapy in subarachnoid hemorrhage, although there are no concrete data.
- Expect a significant degree of acute hypertension with surgery for trigeminal neuralgia. In some institutions, it has been the practice to employ powerful, very short-acting vasodilating or venodilating agents to lower the blood pressure.

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Awake Craniotomy Can Be Done Humanely

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It's 7:00 AM when you learn that your supposedly routine craniotomy for tumor resection is actually an awake craniotomy because of the proximity of the tumor to Broca's area. The patient has been coached by the surgeon, but the family is agitated and fails to understand why anyone would need to have their head opened while awake. What do you need to prepare the room for anesthesia, and what do you say to the family to reassure them?

Awake craniotomies are relatively uncommon procedures that are generally performed in large-volume neurosurgical centers. The usual indication is to facilitate intraoperative electrocorticography to map eloquent brain regions involved in speech, motor, and sensory function in order to minimize neurologic injury to these critical regions during tumor resection. The main objectives of the anesthetic employed for awake craniotomies are to (a) minimize patient discomfort associated with painful portions of the procedure and prolonged restriction of movement, (b) ensure patient responsiveness and compliance during phases of the procedure that require assessment of speech or motor function, and (c) produce minimal inhibition of spontaneous seizure activity. These objectives are achieved by using a variety of techniques that range from minimal sedation to asleep-awake-asleep techniques using a laryngeal mask airway (LMA). There are many important considerations when performing anesthesia for an awake craniotomy. These include pain control, airway management, the possibility of hypoventilation in the setting of increased intracranial pressure, the onset of seizures, the possible complication of venous air embolism (VAE), and patient selection.

Pain control is critical to the care of patients undergoing awake craniotomies. Certain parts of the procedure, such as pin fixation of the head, temporalis muscle dissection, and traction on the dura close to the middle meningeal artery territory can be especially painful. In most institutions, pain control is accomplished with a combination of narcotics and regional anesthesia. Narcotics can be administered as a bolus or as an infusion. The narcotics used should be short acting with minimal effects on electroencephalogram (EEG) monitoring. Fentanyl and remifentanil are appropriate choices. Also, a scalp block is necessary to control pain. Scalp blocks are performed using a 22-gauge intradermal needle with circumferential injection, following a line from the glabella to the occiput and dividing it into four parts

at the intersection of the sagittal and frontal planes (Fig. 136.1). Mayfield pin sites generally require supplementation and should be done in conjunction with the neurosurgeon placing the head holder. The local anesthetic used for injection should have a prompt onset of action, provide analgesia for a significant period of time, and not interfere with electrocorticography. Lidocaine and bupivacaine may be used in combination to achieve rapid onset, long duration and minimal toxicity. Because the scalp block requires a large volume of local anesthetic (as much as 60 cc), it is important for the anesthesiologist to be vigilant in calculating dosages of local anesthetics and monitoring for toxicity. The addition of a third drug such as chloroprocaine or ropivacaine may be necessary to safely achieve the necessary volume of local anesthetic. Avoidance of local anesthetic toxicity is obviously critical in patients immobilized in a Mayfield head holder. It is also important to realize that even with a perfect scalp block and in the absence of somatic pain, many patients will still have a vague (or even severe) headache that will require opioid supplementation.

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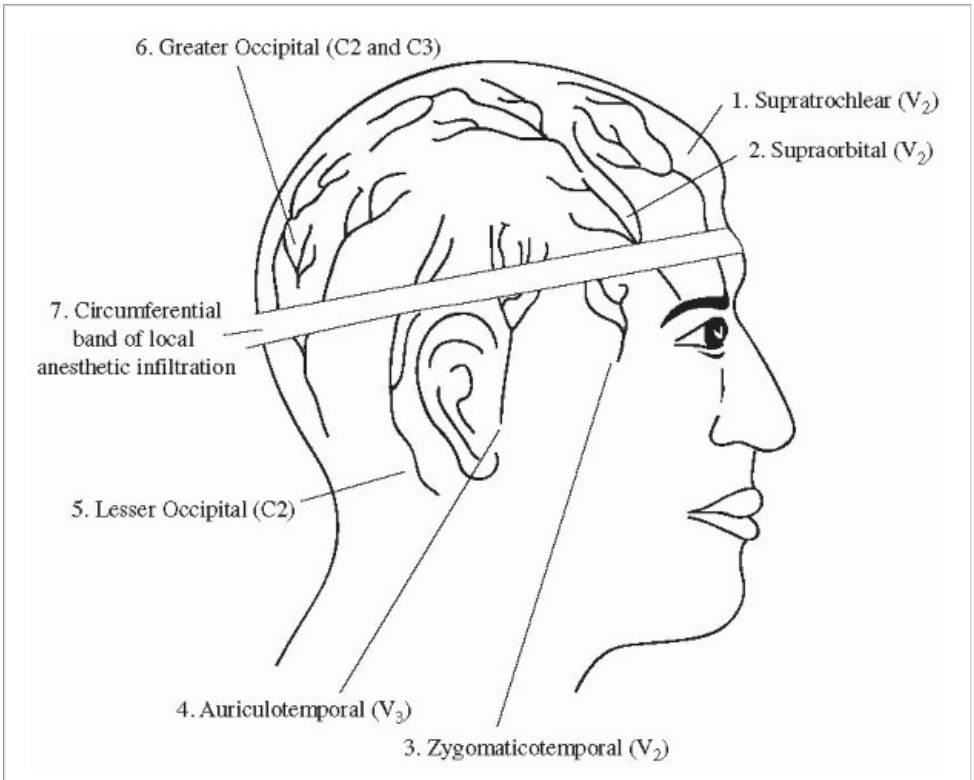


FIGURE 136.1. Schematic anterior and posterior view of the scalp.

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In addition to analgesia, most patients require sedation in order to tolerate the emotional stress of awake craniotomy and simply lying still with an immobilized head for several hours. Many anesthesiologists use an asleep-awake-asleep technique, inserting an LMA for the most stimulating portions of the procedure (line insertion, Mayfield head holder application, and craniotomy) and removing it when patient cooperation becomes necessary. Alternatively, a continuous infusion of propofol or dexmedetomidine with spontaneous ventilation is generally adequate (in conjunction with scalp block and opioids) for even the most stimulating portions of the procedure and does not involve removing the LMA (and thus the potential for coughing) in the immobilized patient. Regardless of whether an LMA is planned, final head position needs to be determined jointly between neurosurgery and anesthesiology so the airway is accessible and so an LMA could be inserted emergently if needed.

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Patients undergoing awake craniotomy are at increased risk of VAE because they are breathing spontaneously. VAE can occur whenever a noncollapsable vein is opened and a pressure gradient exists favoring air entrapment rather than bleeding. This can occur whenever the operative site is above the level of the heart (atmospheric pressure > cerebral venous pressure) or in spontaneously breathing patients where negative intrathoracic pressure is transferred to the cerebral venous system. VAE involves entrainment of air into the venous system, right heart, and pulmonary vasculature. This causes sympathetic reflex vasoconstriction leading to ventilation-perfusion abnormalities. The increased pulmonary vasculature resistance can lead to increased right ventricular afterload, increased pulmonary artery pressure, and increased central venous pressure as the right ventricle begins to fail. Hypotension develops as cardiac output falls. Adult respiratory distress syndrome can be a late sequela. Signs and symptoms in the awake patient include tachypnea, cough, chest pain, hypoxemia, decreased end tidal CO_2, and the classic mill wheel murmur, which can be detected by a precordial stethoscope. Patients may express anxiety or a sense of impending doom. Precordial Doppler is the most commonly used VAE monitoring technique, although it is not quantitative. Once a VAE is suspected, the field should be flooded with saline, the patient placed in the Durant position (Trendelenburg with right side up), nitrous oxide discontinued, the central venous catheter aspirated in an attempt to retrieve entrained air, vasopressors and volume given to treat hypotension, and bilateral jugular vein compression applied to increase cerebral venous pressure and minimize air entry into the heart. Fortunately, this complication remains rare during awake craniotomy.

Another potential consideration during awake craniotomy is the onset of seizures. They can result from cortical stimulation or from local anesthetic toxicity. Treatment includes protection of the airway to maintain respiration and administration of drugs to stop the seizure. Thiopental is an appropriate drug to treat seizures, but it should be held until it is clear that the seizure is not self-limited because the drug may interfere with subsequent EEG localization of the seizure focus. Benzodiazepines may also be used to treat seizures. Generalized tonic-clonic seizures should be managed aggressively in patients immobilized in a Mayfield head holder.

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The final and possibly most important consideration is patient selection. Children and extremely anxious adults are not good candidates for awake craniotomy, although this prohibition is not absolute. It is important that the patient and family understand exactly why the procedure is best performed while the patient is awake. All aspects of the procedure, including the potential for discomfort, must be explained in detail to the patient and his or her family prior to entering the operating room. To maintain credibility, an absolutely honest discussion must be held between the patient and the anesthesiologist, with the assurance that the anesthesiologist will be there to work to keep the patient comfortable throughout. Recognition of the patient's fears and concerns is essential. A comfortable professional relationship with the patient and ongoing conversation and reassurance are the keys to successful completion of the awake craniotomy.

In summary, awake craniotomies are uncommon procedures performed when neurosurgical resection may place eloquent areas of brain at risk for injury. For an awake craniotomy procedure to be a success, the anesthesiologist must take into account many considerations, including pain control, airway management, the onset of seizures, the possible complication of a VAE, and patient selection.

TAKE HOME POINTS

- A scalp block involving circumferential administration of large volumes and adequate concentrations of local anesthesia is required for successful completion of an awake craniotomy. Even when performed by an experienced clinician, patients may experience headache during the awake portions of the procedure and should be prepared in advance for this. Opioid administration is helpful to manage headache.
- Plans for sedation versus general anesthesia by LMA are determined in advance. In both scenarios, the patient is breathing spontaneously with an open calvaria and dura, placing the patient at risk for both hypoventilation and increased intracranial pressure and VAE.
- Head position, determined in conjunction with neurosurgery, should always allow for emergent access to the airway and LMA placement, if necessary.
- An honest and thorough discussion of plans, expectations, and risks needs to be had with the patient and his or her family before entering the operating room. An open relationship between clinician and patient with discussion and reassurance intraoperatively is paramount to patient comfort and safety.

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Don't Be Caught Unprepared for a Wake-Up Test

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You are about to induce anesthesia on a 15-year-old girl for a thoracolumbar fusion when the surgeon casually mentions that, given the degree of kyphoscoliosis, a wake-up test might be required intraoperatively. Yikes! How does this affect your preoperative discussion with the child (and her parents) and your intraoperative management?

Awake-up test is one of several monitoring methods used to evaluate brain and spinal cord function during general anesthesia. The wake-up test allows an intraoperative neurologic examination, which is useful during surgery that may cause reversible central nervous system (CNS) injury. Wake-up tests are traditionally associated with scoliosis procedures during which significant spinal cord distraction may occur. Evoked potential monitoring is commonly performed during these procedures; however, the possibility of false-positive and false-negative results remains. A satisfactory motor response (achieved by asking the patient to "*wiggle your toes* " or "*squeeze my fingers* ") during a wake-up test reassures that both afferent and efferent spinal cord pathways are intact. Furthermore, a wake-up test is indicated in patients with persistent suppression of their evoked potentials despite correction of any identifiable cause and in cases of technical failure. Wake-up tests are rarely done during craniotomy when eloquent regions of brain are at risk (e.g., speech or motor regions). This subtopic is discussed more fully in Chapter 136.

The anesthesia provider must recognize that the potential complications of intraoperative wake-up testing are generally related to overzealous patient movement. These include injury to the patient, injury to operating room personnel, and dislodgement of monitors, intravenous lines, or airway equipment. Patient awareness of the wake-up test postoperatively is *not* necessarily an adverse event unless it is accompanied by awareness of discomfort. It is imperative that the anesthesiologist and patient discuss the wake-up test during the preoperative evaluation, both to acquire informed consent and to decrease potential for undesired patient movement. Patients should be reassured that the wake up will be brief, with maximum attention to their comfort level, and that recall of the wake-up test is possible but not likely.

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In addition to discussing the plan with the patient, the anesthetic regimen should be chosen to facilitate a rapid and smooth emergence for the wake-up test. The earliest anesthetic techniques combined nitrous oxide, opioid, and a volatile anesthetic, allowing for rapid awakening. Since then, there has been some evolution in what is considered the optimum anesthetic technique for major neurosurgical procedures, driven by the use of more sophisticated intraoperative monitoring techniques. For example, motorevoked potentials (MEPs) are rapidly becoming the standard of care for major spine surgery. Unfortunately, transcranial MEPs are exquisitely sensitive to volatile anesthetics; thus, total intravenous anesthesia (TIVA), with some combination of propofol, ketamine, and opioid, is generally believed to provide the best MEP recordings under general anesthesia. Unfortunately, wake up from such an anesthetic may be prolonged. One recent trial compared two anesthetic techniques for wake-up testing and showed volatile anesthetic plus opioid (desflurane and remifentanil) allowed patient reawakening more rapidly than TIVA (propofol infusion with remifentanil or sufentanil).

At the authors' institution, the most common practice during spinal surgery involves volatile anesthetic, low-dose propofol infusion, and remifentanil infusion. The propofol decreases volatile anesthetic requirements, thus improving evoked potential recordings. When it is time for the wake-up test, the propofol and desflurane are discontinued. Remifentanil continues to infuse, providing a baseline level of analgesia throughout the wake-up test. Wake-up tests are done so rarely that surgeons may need to be reminded that it may take up to 30 minutes for a patient to awaken from TIVA.

Neuromuscular blockers must be titrated carefully during cases in which a wake-up test is performed. During the wake-up test, two to three twitches on a train of four nerve stimulators are optimal. This can be accomplished most predictably with a continuous infusion of muscle relaxant throughout surgery. This level of blockade should be sufficient to allow neurologic examination yet produce a weakened patient with less risk of injury or equipment dislodgement. Once the patient's function has been examined, a rapid induction of general anesthesia is desirable. A bolus dose of propofol is our preferred agent, and then the maintenance anesthetic regimen is resumed.

Finally, although the wake-up test is an accepted method to verify motor function in spinal surgery, a normal wake-up test does not guarantee an optimal neurologic outcome. Several cases exist in the literature of patients with normal intraoperative wake-up tests having postoperative complications, including vertebral artery dissection and delayed anterior spinal artery syndrome. Continued vigilance in maintaining stable hemodynamics and neuromonitoring are imperative until the end of the case.

P.598

TAKE HOME POINTS

- It is imperative that the wake-up test be discussed preoperatively with the patient if planned or considered likely during surgery.
- TIVA, a commonly employed anesthetic regiment used to facilitate MEP monitoring, is not ideal for a rapid wake up.
- A basal level of analgesia and incomplete reversal of neuromuscular blockade minimize the risk of vigorous patient movement and subsequent injury during wake-up testing.

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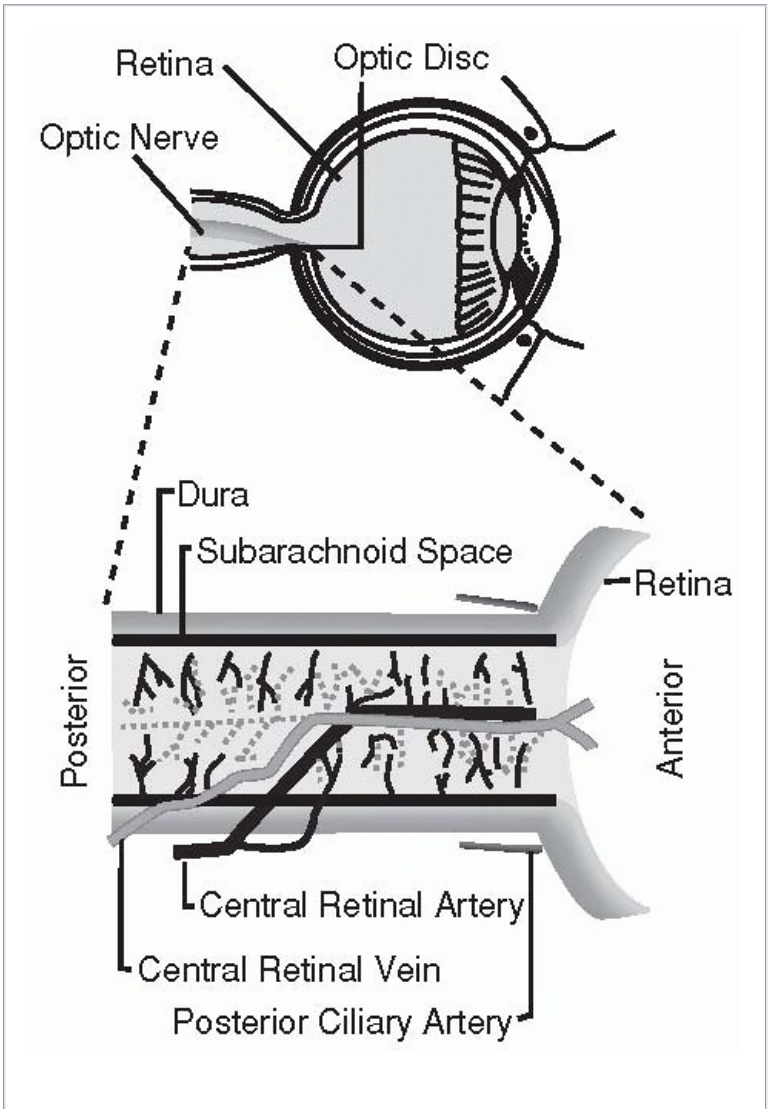
Remember that Loss of Vision is One of the Most Feared and Devastating Complications of Spine Surgery

Laurel E. Moore MD

It is the first day of your first neuroanesthesia rotation and you're all set to start a major spinal reconstruction. You're about to wheel your patient back to the operating room when the patient's spouse (an attorney) tells you that she's recently read about blindness after spine surgery and wants to know what you think and, *specifically*, what you are going to do to prevent blindness?

Postoperative visual loss (POVL) is a devastating injury that fortunately remains rare, with an occurrence rate of between 1/1,000 to 3/10,000 spine surgeries. The time of presentation is variable—patients may present with painless loss of vision immediately post surgery or up to 3 to 4 days postoperatively. The deficit can vary from a mild field cut to complete loss of light perception. Historically, POVL was most closely associated with cardiac surgery and cardiopulmonary bypass. However, with the advent of increasingly complex spine surgeries, the incidence of POVL may also be increasing. This is somewhat surprising because the eye has a dual blood supply: (a) the central retinal artery (CRA), and (b) a series of posterior ciliary arteries. The anterior optic nerve (Fig. 138.1) (anterior to the lamina cribrosa) is supplied by branches of posterior ciliary arteries. These are end arteries and therefore may produce watershed regions, placing the retina at risk for ischemia. The posterior optic nerve is supplied by pial vessels and, occasionally, branches of the CRA. Notably, up to 20% of normal individuals have minimal or absent autoregulation of the optic nerve blood supply.

Unfortunately, the mechanisms of POVL are poorly understood, but they may be categorized into at least two general subgroups. The first of these is central retinal artery occlusion (CRAO). CRAO is the predominant mechanism of POVL following cardiac surgery and is almost certainly embolic following cardiopulmonary bypass. CRAO following spine surgery may not only be embolic, but it may also result from reduced perfusion pressure to the retina by other mechanisms, such as direct pressure on the orbit while in the prone position (“head rest syndrome”). CRAO is generally unilateral and may present with periorbital edema suggesting decreased venous outflow from the orbit, thus reducing retinal perfusion pressure. Funduscopic exam classically reveals a cherry red spot. The prognosis with CRAO is poor, and there is rarely significant improvement in vision. P.600



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FIGURE 138.1. Schematic cross-sectional anatomy of the eye.

More common than CRAO after spine surgery is ischemic optic neuropathy (ION). ION is subdivided into anterior ischemic optic neuropathy (AION) and posterior ischemic neuropathy (PION), depending on whether the ischemic optic nerve is anterior or posterior to the lamina cribrosa (e.g., intraorbital or retro-orbital). These may be distinguished on funduscopic exam. With AION, the funduscopic exam is generally abnormal at presentation, whereas the funduscopic exam with PION is normal at presentation and then becomes abnormal over several weeks as the optic nerve dies. Also, PION is generally more common in spine surgery. The mechanism of ION is poorly understood, but known patient risk factors include (a) history of diabetes and/or atherosclerotic disease, (b) preoperative anemia, (c) *intraoperative hypotension and anemia* , (d) prolonged surgical procedures with large blood loss, and (e) possibly the intraoperative use of vasoconstrictors. ION is more likely to be bilateral than CRAO. In one review of cases, approximately 40% of patients with ION had some return of vision, unlike

CRAO patients. AION has a better prognosis than PION in terms of vision recovery. P.601

Because of the devastating nature of POVL, the American Society of Anesthesiologists recently convened a subcommittee on POVL with the goal of formulating a practice advisory for spine patients. This practice advisory was recently completed and includes the following recommendations[1]:

1. Systemic blood pressure should be continuously monitored in high-risk patients (read: Arterial monitoring). Although all committee members believed that deliberate hypotension was contraindicated in patients with chronic hypertension, there was disagreement as to whether deliberate hypotension was safe in patients without chronic hypertension.
2. A combination of crystalloids and colloids should be used to maintain euvolemia. Central venous pressure monitoring should be considered in high-risk patients.
3. Hemoglobin should be monitored intermittently. The subcommittee could not determine a lower limit below which the risk of POVL could be eliminated. It is the author's belief that hemoglobin should be maintained above 9.0 g/dL at all times for major spine surgery in adults.
4. Although subspecialty neuroanesthesiologists believed that the prolonged use of alpha-agonists could contribute to POVL, the subcommittee believed that there was insufficient evidence to make a statement regarding the use of alpha-agonists.
5. Avoid direct pressure on eye to prevent CRAO. The high-risk patient's head should be maintained in a neutral position with the head higher than the heart whenever possible.
6. For complex spine surgery, consideration should be given to the use of staged procedures in high-risk patients.
7. When POVL is suspected, immediate ophthalmologic consultation should be obtained.

Unfortunately, once POVL is recognized postoperatively, there are few treatment options. For suspected CRAO, some have suggested eye massage or intravenous acetazolamide to reduce intraocular pressure and local thrombolytic therapy, although these maneuvers are of questionable benefit. In general, optimization of hemoglobin and mean arterial pressure are probably of greater benefit. Magnetic resonance imaging (MRI) should be considered to rule out other possible causes of postoperative blindness, including pituitary apoplexy or cortical blindness. Edema of the optic nerve may be visualized on MRI with PION.

TAKE HOME POINTS P.602

- Optimization of mean arterial pressure and hemoglobin with careful presurgical patient positioning are probably the best ways to minimize the risk of POVL.
- Surgeons should consider staged procedures in high-risk patients.
- Because the mechanisms are not fully elucidated, it is probably impossible to completely eliminate the risk of POVL.
- Two clinical practices are recommended: (a) discussion of POVL preoperatively with patients at high risk, and (b) confirmation (and documentation) that the eyes are free of pressure every 15 minutes during prolonged procedures in the prone or lateral position.

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Cannulation for Cardiopulmonary Bypass—Be Careful Where You Point that Thing!

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A64-year-old female insulin-dependent diabetic with severe aortic atheromatous disease and peripheral vascular disease presents for coronary revascularization. Surgery is planned to include the use of cardiopulmonary bypass (CPB). Intraoperative pre-CPB transesophageal echocardiography exam reveals a “mine field” of an ascending aorta with multiple mobile elements and protruding atheromatous plaques. The surgeon requests your help to find an appropriate site for insertion of the arterial perfusion cannula. What is your move in this situation? An epiaortic scan will help better define the extent of atheromatous disease in the ascending aorta. The epiaortic scan did not suggest that it would be safe to place the cannula in the ascending aorta because the stroke risk would be as big as the state of Texas! The surgeon begins to prepare for cannulation of the left femoral artery. Is your left radial arterial line appropriate for systemic pressure monitoring? Yes, but given that the surgical plan involves femoral cannulation in a severely diseased aorta, the patient still has a high risk of stroke from retrograde emboli (e.g., like kicking your foot upstream in a brook where the bottom is covered with moss and loose sand, the upstream current stirs up a load of dirt and debris). The key to placing the cannula in this patient is to recognize that it is going to be difficult to minimize the stroke risk, *and* iatrogenic aortic dissection will also be of major concern.

Cannulation for more complex cardiac surgical operations is not quite as “cookbook” as it may be for coronary surgery. Although the ascending aorta is the most common site of arterial cannulation for CPB, the presence of ascending aortic aneurysms, dissections, severe atherosclerosis, or previous ascending aortic surgery may preclude safe cannulation in this region. *In cannulating an ascending aorta that has severe atheromatous disease, one must ensure that the major force of the arterial cannula flow stream is not directed at a focus of aortic atheromatous disease (i.e., creating “the sand blasting effect”) in order to minimize the risk of stroke.* Optimal selection of the aortic cannulation site is facilitated with epiaortic ultrasound assessment. Alternative sites for arterial cannulation include the femoral artery, axillary artery, innominate artery, common carotid artery, and cardiac apex. The different

cannulation sites will have unique implications for perfusion techniques and hemodynamic monitoring. P.607

FEMORAL ARTERY CANNULATION

The femoral artery is the most common alternative site for CPB cannulation. This vessel is readily accessible and is therefore often used for cannulation to emergently initiate CPB, or when the ascending aorta is not suitable for cannulation such as in an acute ascending aortic dissection. CPB can be initiated with cannulation of the femoral artery and femoral vein or vena cava. On initiation of CPB, the arterial blood flows retrograde through the aorta. *In select emergent cases (e.g., massive pulmonary embolism), femoral cannulation can be accomplished under local anesthesia to avoid the potential hemodynamic collapse that may accompany induction of general anesthesia (related to the blunting of sympathetic outflow) and the initiation of positive-pressure ventilation.*

In the case of aortic dissection or severe aortic atherosclerosis, the descending aorta and femoral artery are often involved. Retrograde perfusion via femoral cannulation may elevate the dissected intima, causing malperfusion with consequent neurologic injury or visceral organ ischemia, or it may cause retrograde embolization from the atherosclerotic aortic wall. *Commonly, the right femoral artery is cannulated in preparation for CPB in patients with an aortic dissection that involves the descending thoracic aorta because it is more common for the dissection to extend into the left femoral artery.*

AXILLARY ARTERY CANNULATION

In comparison to the femoral artery, the axillary artery is usually less affected by atherosclerosis or dissection. Axillary artery cannulation minimizes the need to manipulate an atherosclerotic ascending aorta, which may help reduce the potential for atheroembolic sequelae.

For an operation that requires deep hypothermic circulatory arrest (DHCA), it is also necessary to have a cannulation strategy that promotes cerebral protection. Retrograde cerebral perfusion is accomplished by cannulating the superior vena cava (SVC) and providing retrograde venous blood flow to the brain while concurrently monitoring SVC pressure with an internal jugular central venous line. *Be sure to keep the SVC pressure <25 mm Hg during retrograde cerebral perfusion to protect the cerebral capillary beds.* A designated central venous catheter (e.g., the side arm of the pulmonary artery introducer is often used for this purpose) should be used for monitoring the retrograde cerebral perfusion pressure. The tip of the catheter has to be cephalad to the end of the SVC cannula to guide the retrograde perfusion pressure because inaccurate pressures put the cerebral capillary beds at risk for pressure-related trauma.

However, it may be difficult to control the SVC in surgical procedures that involve a redo sternotomy. Axillary artery cannulation provides

anterograde right carotid artery perfusion in conjunction with innominate artery occlusion during DHCA (Fig. 139.1). Axillary artery cannulation for CPB can be either direct or via an end-to-side interposition graft. P.608

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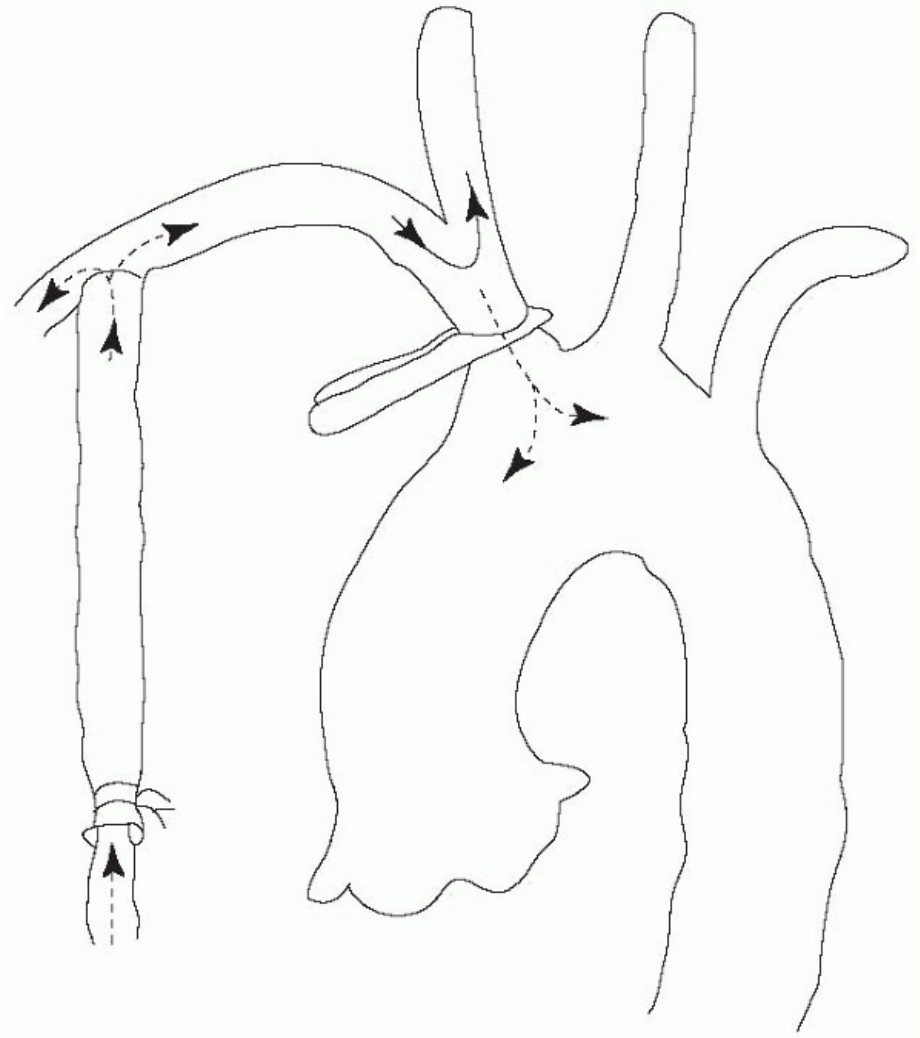


FIGURE 139.1. Right axillary artery cannulation with interpositional graft and antegrade selective cerebral perfusion. Through the right axillary artery, the cardiopulmonary bypass (CPB) pump provides systemic blood flow via an interpositional graft (with clamp off the innominate artery). The arterial pressure recorded from right radial artery will be significantly higher than that of the left radial or femoral arteries. The CPB pump provides antegrade cerebral perfusion via right common carotid artery when the innominate artery is clamped.

INNOMINATE ARTERY CANNULATION

This technique provides the previously mentioned advantages of right axillary artery cannulation, but with a greater simplicity, because there is no need to make an adjunctive incision. It is especially useful in emergency cases (e.g., acute type Adissection).

Line Placement and Pressure Monitoring. The radial artery is the most common peripheral artery used to monitor systemic blood pressure during CPB. In most situations, radial artery cannulation laterality does not produce a clinically significant issue. *However, when axillary or innominate arterial cannulation for CPB is planned, bilateral radial cannulation is recommended so systemic arterial pressure can be more accurately assessed.* Depending on the cannulation technique used when CPB is initiated with right axillary arterial cannulation, the right radial artery may transduce a pressure close to that of the CPB circuit. Therefore, the right radial artery pressure is much higher than the left radial artery or femoral artery pressure, which could lead to hypoperfusion of the brain and other organs. In procedures with right axillary arterial CPB cannulation that plan to employ DHCA, selective anterograde cerebral perfusion via the right common carotid artery can be used if the innominate artery is temporarily clamped. Bilateral radial artery cannulation is recommended in these cases to accurately assess cerebral and systemic blood pressures. In patients who do not need DHCAbut receive axillary arterial CPB cannulation for other reasons, arterial pressure monitoring should be accomplished using the contralateral upper extremity.

Special attention should be paid to central venous pressure (CVP) monitoring while patients are on CPB with SVC cannulation. To accurately monitor the SVC pressure, be sure that the tip of the central venous line or CVP port of the pulmonary artery catheter is cephalad to the end of the SVC cannula.

TAKE HOME POINTS

- In cannulating an ascending aorta that has severe atheromatous disease, one must ensure that the major force of the arterial cannula flow stream is not directed at a focus of aortic atheromatous disease (i.e., creating “the sand blasting effect”) in order to minimize the risk of stroke.
- The femoral artery is the most common alternative site for CPB cannulation. Retrograde perfusion via femoral cannulation may elevate the dissected intima in patients with aortic dissection, causing malperfusion with consequent neurologic injury, visceral organ ischemia, or retrograde embolization from the atherosclerotic aortic wall.
- In select emergent cases (e.g., massive pulmonary embolism), femoral cannulation for CPB can be accomplished under local anesthesia to avoid the potential hemodynamic collapse that may accompany the induction of general anesthesia and the initiation of positive-pressure ventilation.

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- Commonly, the right femoral artery is cannulated in preparation for CPB in patients with an aortic dissection that involves the descending thoracic aorta because it is more common for the dissection to extend into the left femoral artery.
- For an operation that requires DHCA, it is necessary to have a cannulation strategy that promotes cerebral protection.
- When axillary or innominate arterial **CPB** cannulation is planned, bilateral radial cannulation is recommended so systemic arterial pressure can be more accurately assessed. P.610
- The tip of a cannula used to monitor CVP should be optimally positioned relative to the CPB venous cannula to ensure accurate determination of pressure.
- For DHCA, be sure to keep the SVC pressure <25 mm Hg during selective retrograde cerebral perfusion in order to protect the cerebral capillary beds.

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Remember to Ask About A History of Esophageal Disease if Transesophageal Echocardiography is Planned (or Possible)

Thomas R. Elsass MD
Robert W. Kyle DO

Fortunately, there are relatively few complications of transesophageal echocardiography (TEE). When they do occur, however, the complications can be quite serious. Untoward events include severe odynophagia, esophageal rupture, bleeding varices, bleeding ulcers, endotracheal tube dislodgement, and dental and oral trauma. The overall incidence of complications in the immediate postoperative period is ~0.2%. In 7,200 cardiothoracic (CT) surgery patients, severe odynophagia occurred in 0.1% of all patients, and upper gastrointestinal bleed (UGIB) occurred in 0.03%. It has been reported that the incidence of UGIB is even higher. They found that 1.2% of CT surgery patients who underwent TEE had a UGIB within 30 days of the surgery, compared to 0.29% of patients who underwent CT surgery only. It is important to note that bleeding complications often go unrecognized in the immediate perioperative period.

The key, as always, is to avoid complications in the first place. TEE probes should *not* be placed in patients who have an absolute contraindication. These include known or recent (<6 months) esophageal trauma, active variceal bleeding, and tracheoesophageal fistula. Placement of a probe should be considered *carefully* if the patient has any relative contraindications, including esophageal varices, history of UGIB, esophageal strictures, and Barrett esophagus (Table 140.1). To determine whether a patient has a contraindication to TEE**, you must ask the patient about history and look for signs and symptoms** of these disorders. Pertinent signs and symptoms include dysphagia, odynophagia, coffee-ground emesis or hematochezia, liver disease, history of banding, significant longstanding (especially untreated) gastroesophageal reflux disease, and hiatal hernia.

TEE probes are typically placed for certain cardiac surgery procedures (especially valve replacement and repair) but may also be used to evaluate the heart during emergent intraoperative events. The ultimate decision to place a TEE probe depends on the clinical indications and the risk-benefit profile that is determined by the managing anesthesia team.

TABLE 140.1 RELATIVE CONTRAINDICATIONS TO TRANSESOPHAGEAL ECHOCARDIOGRAPHY

History
Dysphagia
Odynophagia
Mediastinal radiation
Recent upper gastrointestinal (GI) surgery
Recent upper GI bleeding
Esophageal pathology
Stricture
Tumor
Diverticulum
Varices
Esophagitis
Recent chest trauma

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From Hensley FA, Martin PF, Gravlee GP. *A Practical Approach to Cardiac Anesthesia.* 3rd ed. Philadelphia, Pa.: Lippincott Williams & Wilkins; 2003.

Here are the three basic guiding principles to follow when placing a TEE probe:

1. Never force the probe. If there is resistance, try something else.
2. Use a lot of lubrication.
3. Use a jaw thrust, and consider asking for help. An extra pair of hands can prove invaluable in difficult situations.

One specific technique for placing a TEE probe is to start by anteflexing the tip to help make the anatomical angle of the pharynx. Simultaneously, perform a jaw thrust and gently twist the probe side to side with careful pressure. Use of a laryngoscope is routine and will assist in displacing the tongue and mandible anteriorly. If you encounter difficulty, you may try retroflexing the probe or approaching from a different angle in the mouth. Again, use lots of lubrication and **never force the probe.** If placing the probe is difficult using standard maneuvers, creating more space for the TEE to pass may be necessary. This can be accomplished with the help of a second operator or different laryngoscope blade to assist in displacing airway structures anteriorly. Also, in smaller patients, it is advisable to empty the stomach and then discontinue the gastric tube. If this is unsuccessful, some institutions will get a STAT intraoperative esophagogastroduodenoscopy (EGD) from the thoracic or gastrointestinal services to evaluate for intrathoracic stomach, tumor, or stricture.

If you are still unable to pass the probe but believe that the surgery necessitates TEE, consider a pediatric probe. Pediatric probes come in biplane and omniplane, are often of sufficient length (especially in women), and are

likelier to cause less injury due to their small diameter. Another alternative to inserting a TEE probe may be to switch strategies and perform epicardial echocardiography (for cardiac cases) or TTE for cases not involving the thorax. P.613

The TEE probe should be considered an invasive monitoring device and care must be taken not to cause additional morbidity once the probe has been placed. One practice is to withdraw the probe to the upper esophagus for cases involving cryoablation (maze) of the posterior left atrium in order to reduce the probability of atrial rupture. Also, during the cardiopulmonary bypass and especially during circulatory arrest, the editor advises that the power to the TEE be discontinued to avoid burn injury to the esophageal tissues (rare) and to avoid unnecessarily warming the heart with the ultrasound signal (especially for cold cardioplegia protection cases).

TAKE HOME POINTS

- TEE is a valuable intraoperative monitor. Complications are rare but serious.
- There are both absolute and relative contraindications to placement of the probe.
- Insertion is facilitated by flexing the probe tip, doing a jaw thrust, removing the gastric tube, performing laryngoscopy, and switching to a smaller probe.
- *Never* force the probe.
- An intraoperative EGD may be warranted to check for significant pathology.
- Know when to withdraw the probe and/or discontinue power.

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Remember that It Is Not Possible to Completely Avoid Myocardial Ischemia Associated with Cardiopulmonary Bypass and the Delivery of Cardioplegia—The Goal Is to Minimize It

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Myocardial protection techniques are critical in obtaining optimal results in cardiac surgery with the aim to decrease myocardial ischemia during cardiopulmonary bypass (CPB). Although some degree of myocardial damage and disturbance of cellular integrity is almost inevitable, in the majority of cases it is reversible when appropriate protection methods are performed. The normal working myocardium consumes 8 mL of O_2/100 g/minute. Oxygen (O_2) consumption decreases to 5.6 mL O_2/100 g/minute in the empty beating heart, to approximately 4 mL/100 g/minute in the fibrillating heart (at a temperature of 32°C), and to 1.1 mL O_2/100 g/minute during CPB with potassium cardioplegia. Cooling the heart to 22°C may further decrease O_2 consumption to 0.3 mL O_2/100 g/minute. Typically, myocardial damage is attributed to a combination of myocardial ischemia due to oxygen supply-demand imbalance and reperfusion injury. Both mechanisms may result in intracellular acidosis, calcium overload, the formation of free radicals, initiation of inflammation, complement activation, and myocardial cellular edema. During CPB, myocardial injury can also occur due to ventricular fibrillation with concurrent ventricular distention, excessive use of inotropes and/or calcium, surgical manipulation, and coronary embolism. Normothermic ventricular fibrillation should be avoided in patients with myocardial hypertrophy because the increased wall tension compromises subendocardial perfusion. Likewise, ventricular distension is a special threat in patients with severe aortic insufficiency. Proper deairing of grafts, cardiac chambers, and venting before and during initial cardiac ejection can prevent formation of air bubbles that may lead to coronary embolism and both ventricular arrhythmias and dysfunction. Not surprisingly, those patients with more severe cardiac risk factors (e.g., severe coronary artery disease (CAD), valvular disease, ventricular hypertrophy, New York Heart Association functional class IV) are at greatest risk of sustaining myocardial injury from inadequate myocardial protection. The area of myocardial protection

strategies is continually growing, and this chapter summarizes the current trends in this evolving field. P.615

CARDIOPLEGIA

Always consider that following the initiation of CPB, it is important to discontinue the administration of all exogenous inotropes so they are not mixed into the cardioplegia because these drugs can potentially increase myocardial energy consumption. Potassium cardioplegia is the most common method of myocardial protection during CPB. Traditionally, cardioplegia is delivered in an anterograde intermittent fashion through the cross-clamped aortic root with a cold, high-potassium, crystalloid solution. The increase in extracellular potassium concentration eventually inactivates the sodium channels, abolishing action potentials and arresting the heart in diastole, thus stopping the energy expenditure associated with electrical and mechanical activity. The composition of cardioplegia varies on an institutional basis; however, all solutions contain 10 to 40 mEq/L. In addition, small amounts of calcium and magnesium and a buffer (usually bicarbonate) are needed in the cardioplegia. Alkaline buffers improve myocardial protection by preventing acid metabolite buildup. With cold cardioplegia, multiple doses are given for the effects of washout, rewarming, and prevention of metabolite buildup. Washout of high-potassium cardioplegia occurs due to the presence of non-coronary collaterals. Standard practice has been to employ systemic and topical cardiac hypothermia to reduce basal metabolic oxygen consumption. O_2 consumption decreases by 50% for every 10°C decrease in myocardial temperature, and myocardial cooling to 25°C allows the interruption of coronary blood supply (aortic cross-clamping) for longer periods of time. However, hypothermia itself may elicit myocardial injury by affecting cellular fluidity, transmembrane gradients, and these changes may result in cellular edema. Recently, it has been suggested that continuous and/or warm cardioplegia provides better protection, although severe, diffuse CAD may preclude even distribution of the cardioplegia and lead to poor protection. So far, studies have not consistently demonstrated the superiority of one method over the other. Although only anterograde cardioplegia was initially used, retrograde perfusion via the coronary sinus while carefully monitoring coronary sinus perfusion pressure (at <40 mm Hg) has become popular, with many centers using a combination of both methods and showing superior protection. Retrograde cardioplegia is particularly advantageous in valve surgery and in patients with critical CAD or total coronary occlusions. This technique is contraindicated in patients with a persistent left superior vena cava draining into the coronary sinus because of the risk of retrograde cardioplegia perfusion to the brain. Most centers currently use blood cardioplegia, a mixture of blood in crystalloids with a final hematocrit of 16% to 20%. In addition to

the O_2-carrying capacity of hemoglobin, blood contains buffers, free radical scavengers, colloids, and other components that protect the myocardium. P.616

MYOCARDIAL PRECONDITIONING WITH ANESTHETIC AGENTS

Ischemic preconditioning describes the heart's ability to be protected during periods of ischemia through an endogenously mediated mechanism. Extensive investigation in this area has demonstrated that volatile anesthetics can confer similar cardioprotective benefits through conformational changes in myocardial protein structure. Clinical research in this area has shown encouraging results for isoflurane, sevoflurane, and desflurane when compared to a primarily propofol-based anesthetic. The majority of these studies demonstrate decreased postoperative troponin elevation, reduced inotropic support, and greater preserved cardiac function. A similar incidence of postoperative complications has been observed between both groups in these trials. Current research in this area is focused on the timing of volatile anesthetic administration during cardiac surgery for maximal preconditioning benefits.

ANTI-INFLAMMATORY THERAPEUTIC STRATEGIES

CPB activates multiple contact, coagulation, fibrinolytic, and cytokine cascades, which result in a systemic inflammatory response. In recent years, this concept of myocardial injury leading to inflammatory mediated responses has prompted research to prevent CPB-induced inflammation. Numerous studies have investigated potential pharmacologic agents, such as corticosteroids, adenosine, phosphodiesterase inhibitors, antioxidants, nitric oxide donors, and complement inhibitors. Mechanical strategies that have been studied include hemofiltration, leukocyte filters, hypothermic CPB, pulsatile CBP

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flow, and uniquely coated circuits (e.g., heparin bonded [Carmeda Bonded Maxima; Medtronic, Inc, Minneapolis, MN] or circuits SMART; Cobe Cardiovascular, Arvarda, CO). The beneficial effects of both pharmaceutical and mechanical strategies have yet to be definitively demonstrated in large clinical studies.

In summary, myocardial protection remains one of the most critical determinants in the success of cardiac surgery. Cold intermittent potassium cardioplegia remains the mainstay of myocardial protection. New techniques include focusing on preventing systemic inflammatory responses and on myocardial preconditioning are not conclusive, and further work remains to be done in these areas. Because cardiac surgery currently carries excellent results in low-risk patients, future innovations in myocardial protection will ultimately benefit those patients with high risk, including those with poor ventricular function, severe valvular disease, and high-grade CAD.

TAKE HOME POINTS

- Myocardial protection remains one of the most critical determinants of the success of cardiac surgery.
- CPB-associated myocardial damage is attributable to O_2 supply-demand imbalance, reperfusion injury, ventricular fibrillation with ventricular distension, use of inotropes and calcium, surgical manipulation, and coronary artery embolism.
- Always consider that following the initiation of CPB, it is important to discontinue the administration of all exogenous inotropes so they are not mixed into the cardioplegia because these drugs can potentially increase myocardial energy consumption.
- Studies to date do not consistently demonstrate that “traditional” cardioplegia with a cold, high-potassium, crystalloid solution is superior to continuous and/or warm cardioplegia.
- Retrograde cardioplegia is advantageous in valve surgery or with critical CAD. Careful attention to avoiding coronary sinus perfusion pressures of >40 mm Hg is of paramount importance.
- An anesthetic based on volatile anesthetics (isoflurane, sevoflurane, and desflurane) can protectively precondition the heart against ischemia damage.
- The relationship between myocardial injury and CPB and a systemic inflammatory response is a subject of ongoing investigation.

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On the Cusp of Disaster: Distinguish between the Anesthetic Management of Stenotic and Regurgitant Cardiac Valves

George A. Mashour MD, PhD

Theodore A. Alston MD, PhD

The initial approach to the patient with cardiac valve disease involves three basic questions:

1. Which valve is affected?
2. Is the lesion stenotic or regurgitant?
3. What is the severity of stenosis or insufficiency?

This chapter reviews disease of the aortic and mitral valve and provides recommendations for anesthetic management during cardiac surgical repair.

AORTIC VALVE DISEASE

Aortic Stenosis. *Stenosis* is defined as a fixed obstruction preventing outflow through a valve. Stenotic lesions of the aortic valve may be either congenital or acquired. Bicuspid valve is associated with a prevalence of approximately 2% and is therefore one of the most common congenital cardiac lesions. Aortic stenosis may also be acquired due to degenerative calcification. The severity of aortic stenosis is characterized by the valve area and mean pressure gradient (Table 142.1).

A low cardiac index can cause underestimation of the severity of aortic stenosis; selected patients benefit from valve replacement for low-output, low-gradient aortic stenosis.

Compensatory changes in response to aortic stenosis include left ventricular hypertrophy. The associated increase in wall thickness and decrease in compliance renders the ventricle preload dependent. Adequate preload is achieved by normovolemia, maintenance of venous return, adequate diastolic filling time, and preservation of sinus rhythm and the associated "atrial kick." Anesthetic goals for the patient with aortic stenosis therefore include the following:

- Avoidance of hypovolemia
- Avoidance of precipitous decrease in systemic vascular resistance, as may happen during induction of anesthesia
- Avoidance of tachycardia, which will decrease diastolic filling time
- Maintenance of sinus rhythm and preservation of atrial kick

TABLE 142.1 SEVERITY OF AORTIC STENOSIS CHARACTERIZED BY AREA AND MEAN PRESSURE GRADIENT

SEVERITY	VALVE AREA (CM^2)	MEAN PRESSURE GRADIENT (MM HG)
Mild	1.5-2.0	<25
Moderate	1.0-1.5	25-50
Severe	0.5-1.0	>50
Critical	<0.5	

The classic technique of high-dose fentanyl induction was initially developed as a management technique for patients with aortic stenosis.

Aortic Regurgitation. Regurgitation or insufficiency is defined as the failure of a valve to prevent backflow of blood. Such lesions of the aorta are due to dilation of the aortic root or leaflet abnormalities and, like stenosis, may be congenital or acquired. Congenital root dilation is associated with Marfan disease and other connective tissue disorders, while acquired insufficiency can result from aortic dissection, chronic hypertension, atherosclerosis, and aortitis. Leaflet abnormalities include bicuspid valves (which can become regurgitant), as well as rheumatic heart disease, endocarditis, and connective tissue disease. The determinants of insufficiency include the valvular orifice area and the diastolic pressure gradient. Like stenosis, adequate preload and sinus rhythm should be maintained. Unlike stenosis, high-normal heart rate and lower systemic vascular resistance improve cardiac performance in the patient with aortic insufficiency. Faster heart rate decreases diastolic time during which insufficiency is manifested, and lower systemic vascular resistance favors forward flow rather than regurgitation.

Intra-aortic balloon counterpulsation is contraindicated in the setting of significant aortic regurgitation. The balloon increases the regurgitation.

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MITRAL VALVE DISEASE

During diastole, the mitral valve allows blood to flow from the left atrium to the left ventricle, and during systole, it protects the left atrium and pulmonary circulation from high pressures. Like the aortic valve, mitral valve lesions can be stenotic or regurgitant.

Mitral Stenosis. The normal area of the mitral valve is 4 to 6 cm^2. Mitral stenosis is defined as an area of <2 cm^2 and is critical at <1 cm^2. Mitral stenosis is typically caused by rheumatic fever and may lead to elevated left atrial pressures, consequent atrial fibrillation, and pulmonary hypertension. Left atrial filling pressures are critical for patients with mitral stenosis. Thus, like aortic stenosis, anesthetic goals include the following:

- Maintenance of preload
- Avoidance of tachycardia
- Maintenance of systemic vascular resistance
- Maintenance of sinus rhythm

P.620

Mitral Regurgitation.

Mitral insufficiency can be either acute or chronic. Acute regurgitation often results from the rupture of chordae tendineae, ischemic papillary muscle, or ruptured papillary rupture. Myxomatous degeneration, rheumatic heart disease, and endocarditis are other causes of mitral regurgitation. As with aortic regurgitation, high-normal heart rate and decreased afterload are beneficial intraoperatively. Although preferable, sinus rhythm is not as crucial in mitral regurgitation as other valvular lesions. In fact, patients with chronic mitral regurgitation often present with atrial fibrillation due to left atrial distension.

Intra-aortic balloon counterpulsation is hemodynamically beneficial in the setting of mitral regurgitation, although atrial fibrillation can decrease the efficiency of the device. The balloon can be lifesaving in cases of acute, severe regurgitation.

INTRAOPERATIVE TRANSESOPHAGEAL ECHOCARDIOGRAPHY

Use of intraoperative echocardiography is essential to cardiac valve surgery.

The initial exam should evaluate the following:

- Valve area
- Peak and mean gradients
- Color Doppler mapping of regurgitant jets
- Valve leaflet structure, movement, coaptation, and apposition
- Annular dilation

Accurate echocardiographic evaluation can help guide surgical planning, especially in the decision between mitral valve replacement and repair. After surgery is performed, echocardiographic evaluation should focus on the following:

- Degree of valvular regurgitation or stenosis
- Paravalvular leak
- Systolic anterior motion of mitral leaflet leading to obstruction of left ventricular outflow tract

TAKE HOME POINTS

- Stenotic lesions of the aortic and mitral valves are best managed by maintaining higher preload, higher systemic vascular resistance, sinus rhythm, and lower heart rate.
- Regurgitant lesions of the aortic and mitral valves are best managed by maintaining normal preload, lower systemic vascular resistance, and higher heart rate.
- Accurate transesophageal echocardiographic evaluation is essential for cardiac valve surgery.

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Antifibrinolytic Agents: Spare the Clot But Spoil the Thrombus

Ala Nozari MD, PhD

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PHYSIOLOGY OF FIBRINOLYSIS

The fibrinolytic system has evolved to dissolve intravascular thrombus and vessel-sealing clot after control of the vascular breach. The system prevents inappropriate accumulation and extension of fibrin. It is important in the repair and remodeling process following injury.

The major fibrinolytic enzyme is plasmin, which circulates as its inert proenzyme plasminogen. The conversion of plasminogen to plasmin is facilitated by the plasminogen activators, thrombin, fibrin, and factor XII (Hageman factor). Tissue-type plasminogen activator (tPA) is produced predominantly in the vascular endothelium in the presence of fibrin, while the urokinaselike plasminogen is found in urine and other secretions. The proteolytic function of tPA is inhibited by plasminogen activator inhibitor (PAI), which is produced by the vascular endothelium (PAI-1) or the placenta (PAI-2).

Plasmin can be inactivated by the serine protease inhibitor alpha 2-antiplasmin and by alpha 2-macroglobulin, a large plasma protein produced by the liver that is found in increased concentration in nephrotic syndrome. Alpha 2-antiplasmin deficiency can lead to a bleeding tendency.

Plasmin functions as a serine protease, cutting specifically C-terminal to the lysine and arginine residues on fibrin. Fibrin monomers, when polymerized, form protofibrils. These protofibrils contain two strands, antiparallel, associated noncovalently. Within a single strand, the fibrin monomers are covalently linked through the actions of coagulation factor XIII. Thus, plasmin action on a clot initially creates nicks in the fibrin, and further digestion leads to solubilization. These soluble parts, called fibrin degradation products, compete with thrombin and slow down the conversion of fibrinogen to fibrin. This effect can be seen in the thrombin clotting time test, which is prolonged in a person who has active fibrinolysis. Soluble fibrin degradation products are cleared by the reticuloendothelial system.

ANTIFIBRINOLYTIC AGENTS

Antifibrinolytic agents (aprotinin, epsilon-aminocaproic acid [EACA], and tranexamic acid) can reduce the amount of blood loss and the need for blood transfusions.

Tranexamic acid (Cyklokapron) and EACA (Amicar) are antifibrinolytic amino acids with the ability to bind to plasminogen by its lysine-binding site, preventing its association with fibrin. They also inhibit the proteolytic activity of plasmin. Tranexamic acid is more potent but less well absorbed orally than EACA. Both drugs are minimally metabolized and are mainly excreted unchanged in the urine, with plasma half-time of 2 to 10 hours. Dosage reduction is indicated in renal impairment. P.623

Besides their role in cardiac surgery, these agents are useful in conditions with bleeding tendency such as patients with von Willebrand disease, patients following prostatic or uterine surgery, and patients with gastrointestinal (GI) bleeding, and as adjunctive therapy in patients with hemophilia.

Side effects are more frequently seen with EACA and include GI symptoms and dizziness. Prolonged use of EACA can rarely cause acute necrotizing polymyositis with myoglobinuria. Antifibrinolytics should not be used in patients with gross hematuria because of the risk for clot formation in the urinary tract with subsequent obstruction and hydronephrosis.

Aprotinin (Trasylol) is a serine protease inhibitor (serpin) isolated from bovine lung tissue. It forms a reversible enzyme inhibition complex with serine proteases by binding to the proteases in a dose-dependent manner. These proteases include plasmin, trypsin, kallikrein, and activated protein C. It inhibits platelet activation by thrombin and is suggested to modulate the systemic inflammation associated with cardiopulmonary bypass (CPB). The antifibrinolytic properties of aprotinin are of particular interest in cardiac surgery because it is suggested to decrease contact activation of the intrinsic coagulation pathway during CPB.

Anaphylactic and anaphylactoid reactions are rare in patients without prior exposure to aprotinin (<0.1%), but patients who are re-exposed have an increased risk of allergic reactions (0.9%-5%). Clinically relevant creatinine elevation (0.7-2 mg/dL over baseline) has been reported in up to 7.7% of cardiac surgery patients treated with aprotinin, but in the majority (80%), renal impairment was associated with perioperative hemodynamic instability and hemorrhage. The proposed mechanism involves the effects of aprotinin on afferent arteriolar tone through inhibition of renin and kallikrein activity. Risk factors for postoperative renal failure include advanced age, duration of CPB >3 hours, preoperative ventricular dysfunction, diabetes mellitus, and pre-existing renal disease. The beneficial effect of aprotinin in achieving hemostasis must also be weighed against risk of pathological thrombosis, even though studies have not confirmed an increased risk of deep venous thrombosis or pulmonary embolism in these patients.

P.624

KEY POINTS

1. Aprotinin significantly reduces transfusion requirements compared to placebo (relative risk, 0.38-0.61) and total blood loss (35%-53%, depending on dose).
2. EACA and tranexamic acid also reduce bleeding. All three drugs significantly reduce the rate of surgical re-exploration. All three drugs refers to Aprotinin, EACA, and tranexamic acid.
3. Antifibrinolytic therapy is not associated with an increased incidence of early SVG occlusion or myocardial infarction.
4. EACA is given as a loading dose of 100 to 150 mg/kg (10 g over 30 minutes) to the patient and into the pump prime solution, followed by a 20 to 30 mg/kg/hour infusion. Aprotinin is administered as a 280-mg bolus (2 million kallikrein inhibitory units) to the patient and into pump prime, followed by 70 mg/hour during surgery.

POTENTIAL MISTAKES IN THE USE OF ANTIFIBRINOLYTICS

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1. Prior to administration of full-dose aprotinin, all patients should undergo an evaluation of risk factors followed by a test dose of aprotinin 5 minutes prior to the loading dose.
2. Consider the risk of anaphylactic reaction, particularly in patients who are being re-exposed to aprotinin. For patients who have received aprotinin within the previous 12 months (the manufacturer changed this recommendation in Dec. 2006 from 6 in 12 months), do not administer the drug until the surgical ability to rapidly achieve CPB is achieved (after aortic cannulation). Do not prime the pump with aprotinin until the risk of anaphylaxis is deemed remote.
3. Postoperative renal failure may be a problem in patients with pre-existing renal disease, advanced age, preoperative ventricular dysfunction, diabetes mellitus, and expected prolonged CPB.
4. Avoid antifibrinolytic treatment in patients with disseminated intravascular coagulation.
5. Remember to correct underlying coagulopathy, factor deficiency, thrombocytopenia, or platelet dysfunction. Note that aprotinin prolongs the celite-activated clotting time and the partial thromboplastin time, and those test results do not indicate anticoagulation. The kaolin-activated clotting time and the prothrombin time are relatively insensitive to the presence of aprotinin in the blood.

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The Downside of Anesthesia for the Descending Thoracic Aorta: Just About All Anesthetic Issues are of Paramount Importance!

Edwin G. Avery IV MD
David A. Shaff MD

Patients undergoing surgery on the descending thoracic aorta (DTA) present a *great* anesthetic challenge. The general categories of aortic pathology affecting the DTA include aneurysm, pseudoaneurysm, coarctation, aortic rupture, dissection, intramural hematoma, infection, and atherosclerotic disease (with or without penetrating ulcers). Constructing a safe anesthetic plan for patients requiring DTA surgery mandates an intimate knowledge of aortic pathology, the planned procedure, and strategies to minimize the deleterious effects of surgical manipulation of the aorta. These patients often present in an urgent or emergent fashion with little time to evaluate serious comor-bidities that frequently accompany aortic disease. *Emergent procedures require recruiting additional anesthesia personnel so preoperative preparation is carried out with optimal efficiency.* The anesthesiologist must review the details of all available imaging studies and create an anesthetic plan that focuses on providing protection and support for all bodily systems affected by the planned procedure.

A successful anesthetic monitoring plan for DTA surgery considers that the nature of these procedures creates hemodynamic instability and hypoperfusion of multiple organ systems. To partially circumvent the effects of interrupting blood flow with aortic cross-clamping, the distal aorta is often perfused using a Gott shunt or partial left-heart bypass (left atrium to distal aorta, iliac or femoral artery bypass with centrifugal pump assist). *Femoral arterial blood pressure monitoring is therefore necessary to assess distal arterial perfusion pressure.* Distal aortic mean arterial pressure (MAP) is commonly maintained between 60 and 70 mm Hg during the partial bypass period, while proximal MAP is maintained at approximately 90 mm Hg. The use of partial left-heart bypass does not routinely incorporate a heater/cooler exchange unit into the extracorporeal circuit and thus puts the patient at risk for developing profound hypothermia as heat is lost through the extracorporeal circuit. *Hypothermia increases the risk of coagulopathic hemorrhage and hyperglycemia (which may exacerbate ischemic injury to the cord and/or heart) in these patients, and thus patient warming systems (e.g., forced convective*

air blankets or hydrogel energy conduction pads) should routinely be used in these anesthetics. P.627

Invasive monitoring is essential for DTA surgery. Caution is warranted in choosing a site for radial arterial cannulation because atherosclerotic disease is highly prevalent in patients requiring DTA surgery. S*evere subclavian or axillary artery plaquing can result in inaccurate hemodynamic data.* Checking bilateral noninvasive cuff pressures can identify significant gradients between the upper extremities. *Central venous access is mandated for these procedures* so cardiac function and filling pressures can be monitored (e.g., by insertion of a pulmonary artery catheter). Intraoperative transesophageal echocardiography (TEE) monitoring can also be useful in this regard. A dedicated central infusion port for the rapid administration of potent inotropes, vasoconstrictors, and vasodilators is also necessary. Aortic cross-clamping places an acutely increased afterload on the left heart that can result in myocardial ischemia and/or acute left ventricular failure in susceptible patients. Careful monitoring for ischemic electrocardiogram changes and/or signs of acute left ventricular failure (e.g., an acute increase in the pulmonary capillary wedge pressure or new wall motion abnormalities observed with TEE) during aortic cross-clamping is mandatory. *Pharmacologic-based afterload reduction (e.g., administration of sodium nitroprusside, nitroglycerin, or fenoldopam) is often indicated during aortic cross-clamping.* Aortic cross-clamp removal can also be accompanied by hemodynamic instability (e.g., hypotension) related to an acute reduction in left ventricular afterload and to the release of lactic acid from ischemic tissue beds distal to the clamp if partial left-heart bypass is not employed. *The transient increase in circulating lactic acid and potassium associated with cross-clamp removal can also instigate ventricular irritability or arrhythmias. Fluid loading and pharmacotherapy (e.g., administration of α_1-agonists and sodium bicarbonate) just prior to and after unclamping can be useful to quell the hemodynamic deterioration.* Large-bore peripheral intravenous access is standard for these procedures to allow for the rapid administration of fluid and/or blood products throughout perioperative period.

The temporary interruption of aortic blood flow associated with DTA surgery to allow for aortic interposition grafting places all distal organs at risk for ischemic injury and infarction. *Potential ischemic injury to the spinal cord is of paramount concern; thus, the anesthetic plan is tailored to both monitor for and prevent such damage.* Protection of the spinal cord during DTA surgery is accomplished with maneuvers that promote blood flow to the cord. Suggested interventions include the preoperative insertion of a lumbar spinal drain, arterial pressure augmentation, neurophysiological monitoring techniques, and pharmacologic support measures. Lumbar cerebrospinal fluid (CSF) drainage is employed to both prevent cord ischemia and treat postoperative paraplegia resulting from DTA surgery. These catheters are managed *without the use of a heparinized or pressurized transducer flush apparatus.* CSF is permitted to drain if the lumbar CSF pressure exceeds 10 mm Hg. Care is taken to ensure a normal coagulation profile prior to catheter insertion and removal. *Arterial pressure augmentation is achieved by maintaining a spinal cord perfusion pressure (MAP - intrathecal pressure) of at least 70 mm Hg, which is accomplished by achieving an MAP of 80 to 100 mm Hg.* Intraoperative neurophysiological monitoring can reveal the acute development of spinal cord ischemia and is accomplished through the use of somatosensory-evoked potentials or motor-evoked potentials. Pharmacologic support measures include the intrathecal administration of preservative-free papaverine (in conjunction with lumbar CSF drainage) and the systemic administration of glucocorticoids, thiopental, mannitol, magnesium, calcium channel blockers, and naloxone. Supportive studies of these drugs in this clinical setting are difficult to interpret, and no widely used standard pharmacologic regimen has been established to date. Intravenous naloxone given at 1 µg/kg/hour has been demonstrated to be associated with improved neurologic outcomes (including reversal of paraplegic symptoms) in this clinical setting. P.628

Left thoracotomy, or a thoracoabdominal incision, is the most common surgical incision used for exposure of the DTA. This surgical approach often necessitates selective lung ventilation, most often of the right lung. Options for selective lung ventilation include lung retraction, insertion of a left bronchial blocker, or the use of a double-lumen endobronchial tube (DLT) (i.e., commonly, a left DLT is employed). The DLT permits pulmonary maneuvers such as the ability to suction the airways and apply continuous positive airway pressure. However, its use may require transition to a single-lumen endotracheal tube at the end of the procedure with the inherent risks after a major surgery.

Consideration of postoperative analgesia is of major concern in this clinical setting because of the painful nature of a thoracotomy. The intra- and postoperative use of thoracic epidural analgesia can provide excellent pain control and permit early extubation to allow for the prompt assessment of

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neurologic function (e.g., rule out paraplegia related to spinal cord ischemia); care must be taken to ensure normal function of the coagulation cascade for both insertion and removal of the catheter to prevent bleeding-related neurologic complications.

TAKE HOME POINTS

- Emergent procedures on the DTA require recruiting additional anesthesia personnel so preoperative preparation is carried out with optimal efficiency.
- Potential ischemic injury to the spinal cord is of paramount concern and thus the anesthetic plan is tailored to both prevent and monitor for such damage.
- When DTA surgery involves the use of a Gott shunt, or partial left-heart bypass, femoral arterial blood pressure monitoring is necessary to assess distal arterial perfusion pressure.
- Hypothermia increases the risk of coagulopathic hemorrhage and hyperglycemia (which may exacerbate ischemic injury to the cord and/or heart) in these patients, and thus patient warming systems (e.g., forced convective air blankets or hydrogel energy conduction pads) are routinely used.
- Invasive monitoring is essential for DTA surgery. Be aware that severe subclavian or axillary artery plaquing can result in inaccurate hemodynamic data. Checking bilateral noninvasive cuff pressures can identify significant gradients in the upper extremities.
- Central venous access is mandated for these procedures so cardiac function and filling pressures can be monitored. Central access also provides a route for administration of important drugs.
- Pharmacologic-based afterload reduction (e.g., administration of sodium nitroprusside, nitroglycerin, or fenoldopam) is often indicated during aortic cross-clamping.
- The transient increase in circulating lactic acid and potassium associated with aortic cross-clamp removal can instigate ventricular irritability or arrhythmias.
- Fluid loading and pharmacotherapy (e.g., administration of α_1-agonists and sodium bicarbonate) just prior to and after aortic unclamping can be useful to quell the associated hemodynamic deterioration.
- Lumbar CSF drainage is employed to prevent and/or treat spinal cord ischemia. These catheters are managed without the use of a heparinized or pressurized transducer flush apparatus.
- Consideration of postoperative analgesia is of major concern in this clinical setting because of the painful nature of a thoracotomy.

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Anesthesia for Ascending Aortic Dissection: It's Like Walking on Eggshells for the Anesthesiologist

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A 74-year-old man arrives at the emergency ward (EW) at 2:31 AM. Gripping his chest, he complains of an intense tearing pain. Medical history is significant for poorly controlled hypertension, hyperlipidemia, and gout. Surgical history is significant for a right hip replacement and cholecystectomy. Vital signs are blood pressure—left: 188/124 mm Hg, right: 125/110 mm Hg; pulse 124/minute; and respirations 24/minute. Physical exam is remarkable for slight confusion, no jugular venous elevation, bilateral basilar rales, holodiastolic diastolic murmur, unremarkable abdomen, and no pedal edema. Pulses are unequal with the right radial pulse obviously weaker than the left radial pulse. Left pedal pulse is weaker than right pedal pulse. The patient has a 14-gauge intravenous catheter in the left forearm. Electrocardiogram shows ST-segment elevation in the inferior leads with a baseline sinus tachycardia. Chest film demonstrates widening of the mediastinum, and a computed tomography (CT) scan confirms a type A dissection extending to the iliac bifurcation. Now, you get paged to the EW to assist with management! Strap in and hold on because you're in for a ride! Forget about getting any sleep for the rest of the night because this patient needs to go to the operating room pronto! Establish arterial monitoring in the left radial artery, reduce the systolic blood pressure to 110 mm Hg, order some blood, and get a quick transthoracic or transesophageal echocardiogram to assess for cardiac tamponade and to determine the mechanism of his aortic insufficiency, which the holodiastolic murmur tipped you off to. Get a lot of help because time is of the essence in these cases, and even when you do your job just right, the patient can still have a bad outcome.

Dissections of the ascending aorta, representing 60% to 70% of all aortic dissections, are true life-threatening emergencies. They involve a tear of the intimal wall of the aorta, creating a false channel. *Due to the high aortic pressures and shear stress created by pulsatile perfusion, the dissection flap can continue to dissect in its proximal or distal extent (often in a spiral manner) and potentially compromise blood flow to multiple vital organs.*

Risk factors for aortic dissection include congenital unicuspid or bicuspid aortic valve, aortic aneurysm, collagen vascular disorders (e.g., Marfan syndrome), cocaine use, chest trauma, and chronic, uncontrolled hypertension. Aortic dissection usually occurs in patients of ages 30 to 80 years, with the peak occurrence in the sixth and seventh decades of life. It affects men more frequently than women. *The Stanford classification of aortic dissection designates type A as involving the ascending aorta, while type B refers to dissections that do not involve the ascending aorta.* Furthermore, using the DeBakey classification of aortic dissection, a type I (involves the entire aorta), type II (involves only the ascending aorta), or type III (involves only the descending aorta) designation can better clarify which aortic segments are involved. P.631

The most common presentation (80% of patients) is chest pain described as “ripping,” “tearing,” “sharp,” “stabbing,” or “pressurelike.” There may be pulse deficits in the extremities, suggesting limb ischemia or systemic hypoperfusion. Aortic dissection resulting in cardiac tamponade (i.e., commonly from aortic root rupture into the transverse pericardial sinus) may produce jugular venous elevation and systemic hypotension; complete cardiovascular collapse is also possible. If the dissection causes severe aortic insufficiency, signs of congestive heart failure (e.g., pulmonary edema) may be present.

Diagnosis of ascending aortic dissection is commonly made by CT, magnetic resonance imaging with angiography (sensitivity 94%-100% and specificity of 95%-100% for all dissections), aortic angiography, or transesophageal echocardiography (TEE) (sensitivity 78%-100% for type A). TEE is a particularly useful diagnostic modality to rule out cardiac tamponade, both for a more detailed analysis of the extent of the dissection and for determining the mechanism/severity of aortic insufficiency (if present). Intraoperatively, TEE can help identify both the true and false aortic lumens, which will facilitate the arterial cannulation required for the initiation of cardiopulmonary bypass (CPB). *Any attempt to perform a preoperative diagnostic TEE must be made in conjunction with an experienced cardiac anesthesiologist because inadequate sedation and blunting of sympathetic autonomic outflow could result in immediate aortic rupture with unrecoverable hemodynamic collapse.*

The mortality rates associated with Stanford type A dissections are approximately 1% to 2% per hour, in the first 48 hours, following initial onset of symptoms. This increases to 60% in the first 6 days and 74% by 2 weeks, and by 6 months, the trend levels off near 91%. The complications associated with type A dissection are listed in Table 145.1. The high degree of morbidity and mortality is usually related to involvement of the coronary ostia, dissection into the aortic root causing severe aortic insufficiency, and proximal dissection causing cardiac tamponade. The dissection can also cause rupture into either atria or the right ventricle.

Acute medical management is directed at control of systemic blood pressure to reduce the shear stress or change in pressure over time (ΔP/Δt) transmitted to the aortic wall. Although there has not been any pressure range proven to be best, a pressure of 100 to 110 mm Hg systolic is the generally accepted goal. This pressure will likely maintain adequate organ perfusion while minimizing the risk of aortic rupture. P.632

TABLE 145.1 COMPLICATIONS OF ACUTE STANFORD TYPE A AORTIC DISSECTION (*N = 513)*

Complication	Percentage of Occurrence
All neurologic defects	18%
Coma/altered consciousness	14%
Myocardial ischemia/infarct	4%

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Acute renal failure	6.2%
Hypotension	26%
Cardiac tamponade	17%
Mortality	30%

Adapted from Bossone E, Rampoldi V, Nienaber CA, et al. Usefulness of pulse deficit to predict in-hospital complications and mortality in patients with acute type Aaortic dissection. *Am J Cardiol* 2002;89:851.

Anesthetic management should be directed at establishing access and includes multiple large-bore intravenous catheters with blood warming devices, as well as a rapid infusion device (if available), central venous access (i.e., to permit monitoring of central filling pressures, assess cardiac output, and deliver both potent α-agonists and inotropes), and at least one arterial line, depending on the surgical plan for the arterial cannulation that is needed for CPB (see Chapter 139 because arterial monitoring can be complicated in these patients, depending on the extent of the dissection). Ultrasonic exam of the common carotid arteries prior to central line placement can reveal both the location of the vessel and whether the dissection has extended into the neck. Such involvement is common with type Adissections, but flow obstruction-related common carotid dissection is not common and portends increased mortality. It may be prudent to avoid cannulating the central venous system on the side of compromised arterial flow, although some experienced anesthesiologists make a sound argument for avoiding central venous cannulation on the side with normal arterial flow.

Have **at least** *4 U of appropriately typed and cross-matched packed red blood cells available in the operating room before the procedure begins.* Necessary monitoring includes standard American Society of Anesthesia monitors with the addition of a Foley catheter (for urine drainage/monitoring), orogastric tube, and temperature monitoring of both the shell and the core. High-dose narcotic induction is a preferred method due to hemodynamic instability frequently observed in these patients. *Induction of general anesthesia for patients with aortic dissection must always proceed with prevention of hypertension as a*

priority. If the airway necessitates a rapid sequence intubation, then esmolol can be used as an adjunct to treat the hypertensive response to laryngoscopy P.633
(these patients are often managed preoperatively with a combination of esmolol and nitroprusside infusions, as indicated in Table 145.2). Intravenous lidocaine (1.5 mg/kg) at the time of induction can also blunt the response to laryngoscopy. The short half-lives of esmolol and nitroprusside make them ideal for blood pressure control in these patients. ATEE probe should be placed after induction, and a complete TEE exam performed with special attention to the extent of the dissection (e.g., involves the aortic arch, coronary ostia, and aortic valve). *Depending on the distal extent of the dissection, circulatory arrest may be required to perform an adequate surgical repair.* Neuroprotection is of paramount concern in cases requiring deep hypothermic circulatory arrest (see Chapter 146).

TABLE 145.2 MEDICATIONS FOR CONTROL OF BLOOD PRESSURE

Medication	Dosage
Esmolol	25-300 mcg/kg/min
Sodium nitroprusside	0.5-2 mcg/kg/min
Nicardipine	1-15 mg/h

If the dissection has caused cardiac tamponade, the transition to positive-pressure ventilation during the induction of general anesthesia can be deleterious for right heart filling and result in cardiovascular collapse. Maintain cardiac filling pressures with intravenous volume administration and a short inspiratoryto-expiratory ratio to maximize right heart filling. If pericardial tamponade is diagnosed or suspected, femoral cannulation for CPB under local anesthesia should be considered, followed by induction and immediate sternotomy. *Decompression of cardiac tamponade prior to bypass may cause rebound hypertension, resulting in aortic rupture and a tremendous increase in mortality risk.* If femoral cannulation for CPB cannot be instituted under local anesthesia prior to inducing general anesthesia, then a careful induction with the gradual transition to assisted and, finally, positive-pressure ventilation should be performed to minimize the risk of cardiovascular collapse. In such cases, prior to induction, a surgeon must be present, the patient fully prepped and draped, and the operating room team ready to establish emergent femoral bypass.

TEE is a useful post-CPB monitor to assess the adequacy of repair. Important post-CPB TEE findings include restoration of flow to the true aortic lumen, assessment of aortic valve repair/replacement, and evaluation of cardiac function. Finally, use of a cerebral oximeter (Somanetics Corporation, Troy, MI) can alert one to the presence of compromised cerebral blood

flow post-CPB. Alternatively, the common carotid arteries can be directly assessed with a surface ultrasound probe via color Doppler interrogation to P.634
confirm that bilateral blood flow is present to the cerebral circulation. *Following confirmation of a successful surgical repair, the anesthesia team should be prepared to both evaluate (e.g., via thromboelastography) and aggressively treat coagulopathic hemorrhage* (see Chapter 149).

TAKE HOME POINTS

- Due to the high aortic pressures and shear stress created by pulsatile perfusion, the dissection flap can continue to dissect in its proximal or

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distal extent (often in a spiral manner) and potentially compromise blood flow to multiple vital organs.

- The Stanford classification of aortic dissection designates type A as involving the ascending aorta, while type B refers to dissections that do not involve the ascending aorta.
- Any attempt to perform a preoperative diagnostic TEE must be made in conjunction with an experienced cardiac anesthesiologist because inadequate sedation and blunting of sympathetic autonomic outflow could result in immediate aortic rupture with unrecoverable hemodynamic collapse.
- Acute medical management is directed at control of systemic blood pressure to reduce the shear stress, or change in pressure over time ($\Delta P/\Delta t$), transmitted to the aortic wall. Although there has not been any pressure range proven to be best, a pressure of 100 to 110 mm Hg systolic is the generally accepted goal.
- Make at least 4 U of appropriately typed and cross-matched packed red blood cells available in the operating room before the procedure begins.
- Induction of general anesthesia for patients with aortic dissection must always proceed with prevention of hypertension as a priority. The short half-lives of esmolol and nitroprusside make them ideal for blood pressure control in these patients.
- Depending on the distal extent of the dissection, circulatory arrest may be required to perform an adequate surgical repair. Neuroprotection is of paramount concern in cases requiring deep hypothermic circulatory arrest (see Chapter 146).
- If the dissection has caused cardiac tamponade, the transition to positive-pressure ventilation during the induction of general anesthesia can be deleterious for right heart filling and result in cardiovascular collapse.
- Decompression of cardiac tamponade prior to the bypass may cause rebound hypertension, resulting in aortic rupture and a tremendous increase in mortality risk.
- Following confirmation of a successful surgical repair, the anesthesia team should be prepared to both evaluate (e.g., via thromboelastography) and aggressively treat coagulopathic hemorrhage.

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Editors: **Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.**
Title: ***Avoiding Common Anesthesia Errors, 1st Edition***

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Freeze—You're Under Deep Hypothermic Circulatory Arrest!

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A 50-year-old male who works as an aeronautical engineer presents sporting a bicuspid aortic valve and a "grande burrito" size (63 mm) ascending aorta. He is scheduled for both aortic valve replacement and ascending aortic replacement requiring deep hypothermic circulatory arrest (DHCA). Following initiation of cardiopulmonary bypass (CPB), the heater/cooler exchange jacket is dropped to 15°C, and after the patient reaches a nasopharyngeal temperature of 18°C, the rest of the operating room team is ready for DHCA. Are you? Hopefully, he'll return to work for another 15 years after this operation, and his space shuttle will have a successful mission. To allow that, you'll have to protect that great brain of his! Cover his head with ice bags, and prepare a brain cocktail with steroids, magnesium, and mannitol. Once circulation is arrested, ensure that the selective anterograde cerebral perfusion circuit flow is appropriate for his body size. Watch the clock; time is the enemy now, and the surgeon needs to be informed of the circulatory arrest time at regular intervals. Following complete rewarming of both core and shell temperatures, make sure that platelets, plasma, and red blood cells are available for transfusion if needed. With these maneuvers and a technically successful surgery, the patient has a great chance of going right back to being a rocket scientist!

Certain adult cardiac surgical procedures, primarily involving the ascending and descending aorta, aortic arch, and pulmonary vasculature require temporary interruption of cerebral blood flow to improve visualization and technical repair. This technique is referred to as DHCA (aka "circ arrest") and is associated with high rates of neurologic compromise. Advanced age (older than 80 years) and DHCA times of >30 minutes are associated with higher rates of neurologic injury. The immediate energy-supply imbalance that occurs with "circ arrest" requires that the anesthesiologist consider the direct application of ice to the head (i.e., inducing an ice cream headache) and administration of a fancy drug cocktail in an attempt to preserve neurologic function. In addition, selective cerebral perfusion during DHCA has been observed to be of clinical benefit.

MECHANISM OF BENEFITS

The brain is exquisitely vulnerable to ischemic episodes due to its high cerebral metabolic rate of oxygen consumption ($CMRO_2$) and yet relatively limited reserve of high-energy phosphates. Hypothermia decreases the metabolic rate of neural tissue and increases the safe time that the brain can survive without oxygen and metabolic substrate. Silence of the electroencephalogram is only achieved in 50% of patients at 18°C but up to 99% at 12.5°C. Other suggested mechanisms of protection include inhibition of capsases, maintenance of microvascular integrity, prevention of glutamate-associated transient spikes in activity, suppression of inflammation, and decrease in edema formation. Most clinicians will cool systemically to between 15°C and 20°C and then maintain circulation at this temperature for approximately 5 minutes before bypass flow is ceased.

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The acceptable duration of circulatory arrest depends on the degree of hypothermia. According to the Kirkland normogram, at 15°C, 30 minutes is possible, and at 10°C, up to 40 minutes is possible. There is no consensus opinion about the acceptable duration, and the limit likely varies between individuals.

Known risks of hypothermia include injury to blood elements associated with cooling and warming, platelet dysfunction, and coagulopathy.

DIRECT APPLICATION OF ICE—THE ICE CREAM HEADACHE

Direct application of ice-filled bags to the head has been advocated to facilitate cooling and to reduce passive rewarming during the period of arrest. Placement of ice-filled bags around the entire head with careful attention to protect the eyes, nose, and ears from direct contact should be done as soon as cooling is initiated. Ice should also be applied to the occiput. The ice bags should be removed on rewarming if you want to keep the respect of your surgical colleagues!

BLOOD GAS MANAGEMENT

Pediatric literature and animal experimentation supports **"pH stat"** blood gas management (temperature corrected values), in which CO2 is added to the blood to facilitate cerebral vasodilation and more uniform distribution of cooling. The results in adult patients are not as clear. Blood gas management using **"alpha stat"** techniques (temperature uncorrected values) without additional CO_2 may be beneficial during rewarming to avoid excessive cerebral perfusion and embolic injury commonly seen due to the blood vessel-rich nature of the brain.

BRAIN COCKTAIL ANYONE? (PHARMACOLOGIC ADJUNCTS)

There is no universally accepted medical protocol for DHCA that has been validated in randomized controlled clinical trials. **Barbiturates** (Phenobarbital) may be administered in an attempt to decrease $CMRO_2$ by burst suppression.

Large doses are required and result in hemodynamic compromise and longer periods of intubation. **Volatile anesthetic agents** may provide protection by ischemic preconditioning. **Glucocorticoids** are often administered due to several potentially beneficial mechanisms, including decreased lipid, eicosanoid, free radical, and edema formation. In addition, normal cerebral blood flow may return earlier after DHCA. *The obvious risks of uncontrolled hyperglycemia must be recognized and treated.* **Magnesium** has several potential beneficial mechanisms, including inhibition of NMDA receptors, which limits the excitotoxic cascade that commonly leads to central nervous system damage. **Ketamine** also inhibits NMDA receptors, and several in vitro studies show benefit. The benefits of **mannitol** may be due to limitation of free radical formation, improved blood viscosity, and a decrease in edema formation. Volatile anesthetic agents may confer cerebral protection through inhibition of excitotoxicity, improvement in brain oxygenation, and decreasing the metabolic rate.

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SELECTIVE CEREBRAL PERFUSION

Selective cerebral perfusion is proposed as a mechanism to provide the brain with substrate while the rest of the body is subject to circulatory

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arrest.

Retrograde cerebral perfusion is the practice of providing oxygenated blood flow and nutrients from the bypass circuit into the superior vena cava. Other benefits may include a decreased risk of air or atheromatous emboli and maintenance of cerebral hypothermia. *Care is taken to maintain a pressure <25 mm Hg (measured via an internal jugular catheter). Generally, flows of 200 to 600 mL/minute are achieved.* Risks include ineffective perfusion due to valves, edema formation, injury to the venous system, and cerebral hemorrhage.

Selective anterograde cerebral perfusion is the practice of intermittent or continuous perfusion of the brain via cannulation of the brachiocephalic vessels, axillary artery, or aortic arch vessels. This is from last page usually initiated once full cooling has occurred and the arch vessels are exposed. Flow rate is approximately 15 cc/kg/minute when perfusion is via the axillary artery. Cerebral perfusion depends on an intact circle of Willis and may prolong the period of arrest. *Potential risks include crowding of the surgical field, injury to arch vessels, embolization, and malperfusion.*

REWARMING

Rewarming starts as soon as surgical conditions warrant. Usually, the body is warmed 1°C every 3 minutes of bypass time. The bypass temperature should never be more that 10°C higher than body temperature. Warming should be terminated when the body is between 35°C and 36°C.

TAKE HOME POINTS

- Directly apply **ice** bags to head, including occiput, with protection of eyes, ears, and nose. Ice should be placed as soon as cooling is initiated
- Consider use of **corticosteroids** (methylprednisolone 30 mg/kg, generally 1,000 mg) administered at start of the case
- Provide maintenance anesthetic maintained **a volatile anesthetic agent** at 1 MAC prior to DHCA
- Empty **"pH stat"** blood gas management during cooling
- Administer intravenous **mannitol** (0.25 g/kg, usually 25 g) 5 minutes before initiation of circulatory arrest
- Administer **magnesium sulfate** (100 mg/kg) just prior to DHCA
- Carefully manage **hyperglycemia** before and after DHCA
- Consider the use of either selective anterograde or retrograde cerebral perfusion during the period of DHCA with the knowledge that the weight of existing evidence is supportive of the use of the selective anterograde technique
- Employ **"alpha stat"** management during rewarming
- Provide controlled **rewarming** no faster than 1°C every 3 minutes, with a gradient no more than 10°C between body and perfusate

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Editors: **Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.**
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Remember that Anesthesia for Left Ventricular Assist Device Surgery is Especially Challenging: Do Not Neglect These Essential Principles

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Giora Landesberg MD, DSc, MBA

The chronic shortage of donor hearts for transplantation has created the need for ventricular assist devices (VADs) in patients with end-stage heart failure, for either temporary (bridge to transplantation) or permanent implantation (destination therapy). Left ventricular assist devices (LVADs) support the left ventricle (LV) by diverting either the entire or partial LV blood flow via a cannula in the apex of the LV to the aorta using an external mechanical force. Anesthesia for these cases can be challenging. Consider the options for a patient on a transplant list with end-stage heart failure (ejection fraction [EF] 21%), worsening symptoms, and new signs of renal failure. An intra-aortic balloon pump is placed. What is involved in the perioperative assessment and management of this patient?

CLASSIFICATION OF LEFT VENTRICULAR ASSIST DEVICES

There are two main types of VADs: Pulsatile and nonpulsatile. The pulsatile or displacement VADs use valves and a pusher plate, displacing blood drained from the LV through a reservoir into the aorta. Pulsatile devices are further divided into extracorporeal or paracorporeal (Abiomed BVS 5000, Abiomed, Inc., Danvers, MA and the Thoratec, Thoratec Corp., Pleasanton, CA), in which the pump is external to the body. These LVADs are in use only for temporary assist (up to 30 days). The other type is intracorporeal (HeartMate I Thoratec IVAD, Thoratec Corp., Pleasanton, CA and Novacor World Heart Inc., Oakland, CA), in which the pump is implanted inside the peritoneum or the thorax, and only the power supply is external. The nonpulsatile or rotary VADs are also divided into two types: The centrifugal (either extracorporeal or intracorporeal), which are rarely used anymore, and the more modern axial flow pumps (Jarvik 2000, Jarvik Heart, Inc., New York, NY and the HeartMate II, Thoratec Corp., Pleasanton, CA), in which a small electromagnetically actuated impeller drive shaft rotates and propels blood at a flow that can be controlled according to the needs of the individual patient. The axial flow pumps are smaller, less than one-fifth the size of the
pulsatile pumps, allowing their implantation in children and small adults. Axial rotary pumps can also be easily adjusted to operate in parallel to the pulsating LV, thus allowing the heart to gradually recover and wean from the LVAD. P.641

Indications. Candidates for heart transplant are those with a cardiac index <2 L/minute/m^2 and a pulmonary capillary wedge pressure (PCWP) >20 mm Hg.

Contraindications. Contradictions include infection (blood cultures are negative, especially for fungus, at least 1 week before device placement), renal failure (creatinine >5.0 mg/dL), severe liver impairment, stroke, severe pulmonary dysfunction, and severe pulmonary dysfunction. ***Anesthetic Considerations.*** These patients are critically ill, are often dependent on inotropes and/or intra-aortic balloon pump counterpulsation therapy, and have often had previous cardiac operations and other end-organ dysfunctions. Their fixed low stroke volume strongly depends on preload and heart rate to maintain marginal performance, which is already on the flat or even descending limb of the Starling curve. Excessive increase in afterload may decrease stroke volume and output dramatically. They are often maintained on angiotensin-converting enzyme (ACE) inhibitors and β-blockers to decrease afterload and diuretics to minimize volume overload. Amiodarone is also commonly used; this blocks the sympathetic system by noncompetitive α- and β-blockade. Its combination with ACE inhibitors that block the renin-angiotensin system and decrease cardiovascular responsiveness to catecholamines may lead to increased vasoconstrictor requirements during cardiopulmonary bypass (CPB). Circulating endogenous catecholamines are high in patients with heart failure, which makes them prone to cardiovascular decompensation during induction or maintenance of anesthesia. Changes in volume of distribution and the decreased clearance of drugs are likely due to altered metabolism by the liver and elimination by the kidney.

Monitoring.

Monitoring should include standard cardiac hemodynamic monitoring, arterial line, pulmonary artery catheter (inserted either before or after induction) and transesophageal echocardiography (TEE). Large-bore intravenous lines are prudent for rapid administration of fluid and blood products. Pulmonary vascular resistance (PVR) provides an indication of the need for pulmonary vasodilators on weaning from bypass:

PVR = 80(MPAP - PCWP)/CO

where MPAP is mean pulmonary artery pressure and CO is cardiac output. Continuous CO catheters may be useful after LVAD placement for continuous assessment of right-sided CO, while the LVAD monitor continuously
displays left-sided flow. Discrepant values may indicate aortic regurgitation or intracardiac shunting. P.642

TEE has a major role in managing LVAD surgery. Before bypass, TEE helps optimize left ventricular filling; **exclude patent foramen ovale** (PFO), artial septal defects (ASD), and aortic insufficiency; evaluate right ventricular function; assess degree of tricuspid regurgitation; assess the aorta for calcification, plaque, or dilation; and examine all chambers for thrombus, particularly at the apex of the LV, where the LVAD inflow cannula is inserted. Exclusion of even the smallest PFO, ASD, or ventricular septal defect (VSD) is of paramount importance due to the risk of hypoxia secondary to the rightto-left shunt after bypass and LV decompression if the PFO/ASD/VSD is not surgically sutured. Efforts to detect a PFO should include intravenous bubble contrast injection during and following the release of a Valsalva maneuver and with increased preload (Trendelenburg position). *Caution should be exercised when executing a Valsalva maneuver in these patients because it may precipitate a malignant arrhythmia and subsequent cardiovascular collapse.* During bypass, TEE is used to assess appropriate placement of the inlet cannula and its orientation (toward the mitral valve), **device** activation, and deairing. After bypass, TEE is used to monitor right ventricular function and tricuspid regurgitation, decompression of the LV and left atrium, air entrainment if the LV collapses, and **Doppler assessment of LVAD flows** to rule out partial cannula obstruction.

Anesthetic Induction.

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Maximal preoxygenation is extremely important and may take time due to the low functional residual capacity. Hemodynamic stability is usually achieved with low-dose fentanyl (5-10 mcg/kg), midazolam, and etomidate. The stress of intubation, hypoxia, or hypercarbia should be avoided because they may lead to rapid decompensation, and low-dose norepinephrine or phenylephrine is often administered to augment blood pressure.

Maintenance of Anesthesia.

Low-dose isoflurane (0.4%-1.0%) is often used to avoid awareness to the extent that the hemodynamic state allows. Mean blood pressure of 70 mm Hg is acceptable and is often supported by low-dose norepinephrine. Aprotinin or ε-aminocaproic acid is administered to reduce bleeding and blood product consumption.

Weaning from Bypass.

The major concerns on weaning from CPB are right ventricular failure (in 25% of the patients), control of pulmonary and peripheral resistances, and bleeding. Of note, axial flow devices function best in the presence of lower systemic and PVR; therefore, drugs such as milrinone, dobutamine, or isoproterenol are preferentially used to lower resistance and support the RV. Inhaled nitric oxide or PGE1 have been used successfully to reduce pulmonary hypertension. Conversely, some patients may require an increase in systemic vascular resistance with the use of norepinephrine (1-20 µg/minute) or vasopressin (0.03-0.07 U/minute). RV failure may become evident only after LVAD operation due to the improved cardiac output and venous return, combined with the loss of developed LV systolic pressure related to ventricular unloading by the LVAD. Excessive decompression of the LV may cause LV collapse and LVAD inlet occlusion, which may lead to air entry through suture lines or interstices of the graft, with potential catastrophic air embolism, particularly in pulsatile LVADs. TEE is used to measure LVAD flow by continuous-wave Doppler through the inflow cannula and comparing that to the flow set on the device.

TAKE HOME POINTS

- LVAD patients are extremely sick and are often dependent on both intrinsic and extrinsic adrenergic support. Anesthesia and hemodynamic support should be carefully planned to avoid catastrophic hemodynamic collapse during induction and before going on bypass.
- TEE plays a major role during all stages of LVAD surgery and anesthesia.
- After bypass, the major issues are tuning RV function and pulmonary resistance as well as assuring proper LVAD function and hemostasis.

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Off-Pump Cardiac Surgery: What Do You Mean No Pump Break for the Anesthesiologist?

Biswajit Ghosh MD

Vipin Mehta MBBS, MD, DARCS, FFARCSI

A68-year-old male is admitted for elective coronary revascularization. Coronary angiography reveals severe three-vessel coronary artery disease. Left ventricular ejection fraction (LVEF) is 45% by echocardiogram. Preoperative chest film reveals a heavily calcified ascending aorta. Past medical history is notable for hypertension (HTN), diabetes mellitus type 2 (DM-2), stroke 3 years prior (no residuum), and a 45 pack-year smoking history prior to quitting 3 years earlier. In addition, he underwent right carotid endarterectomy (CEA) 2 years ago, and his most recent blood tests reveal chronic, moderate renal insufficiency. During your preoperative anesthesia evaluation, he informs you that the surgeon has told him that he plans an off-pump coronary artery bypass (OPCAB) surgery for him. The patient recently read an article about "beating heart surgery" and is terrified about the prospect of undergoing this procedure! He wants to know what benefits he can expect from OPCAB versus standard coronary artery bypass grafting (CABG). He asks, "Doc, am I going to have another stroke while I am asleep?" What do you tell him?

OPCAB is a good option for this patient, especially because his surgeon performs this procedure frequently. The patient's age, history of a stroke, prior CEA, DM-2, calcified ascending aorta, and history of both HTN and tobacco abuse suggest he is at great risk for perioperative stroke, especially if the diseased aorta is heavily manipulated. Avoiding aortic manipulation and cardiopulmonary bypass (CPB) may minimize the propensity for stroke in this case. The patient should be informed that OPCAB surgery could be associated with less neurologic complications than standard CABG in his case.

OPCAB surgery does not offer any additional protection to the kidneys over conventional bypass. The possibility of blood product transfusion may be less in OPCAB surgery. Time to extubation, intensive care unit (ICU) length of stay, and hospital length of stay may be reduced in OPCAB surgery.

BACKGROUND

The first CABG performed without CPB was reported by Kolessov in 1967.The advancement and standardization of CPB and myocardial perfusion techniques resulted in a waning interest for this technique during the 1970s. Since the 1990s, there has been a notable resurgence of interest in the OPCAB technique at select centers. Of note, approximately 25% of all CABG in the United States in 2003 was done without CPB. P.645

WHO QUALIFIES FOR OPCAB?

Selection of OPCAB patients depends on multiple factors: Institutional practice, expertise of the surgeon, number of vessels to be revascularized, site and degree of stenosis, and location of the vessel (i.e., *intramyocardial vessels are technically more difficult to graft during OPCAB surgery*). Patients who may not be good candidates for OPCAB include those with a cardiothoracic ratio >0.7, vessel diameter <1.5 mm, left main disease, LVEF <35%, acute myocardial infarction with hemodynamic instability, morbid obesity, and severe pulmonary disease. Although these are all relative contraindications, experienced OPCAB surgeons are often not deterred by such risk factor profiles from attempting OPCAB.

SURGICAL TECHNIQUE

The position of the patient varies from supine (standard midline sternotomy and reverse J inferior sternotomy) to supine with right lateral tilt for the left anterior minithoracotomy in the fourth or fifth intercostal space.

The sequence of graft placement is determined in order of increased cardiac displacement, and accordingly, the anterior wall vessels are grafted initially, followed by inferior wall vessels and then lateral wall vessels. The concept behind this is that the increasingly revascularized heart tolerates a greater degree of displacement and the hemodynamic insults associated with this repositioning. Exceptions to this approach exist according to individual anatomy.

Adequate exposure and stabilization of the operative vessel is critical for the success of anastomosis completion. This goal is achieved using retractors, stabilizing devices, stabilizing sutures, and possibly intracoronary shunts. OPCAB equipment varies and may function by compression and/or suction to achieve stabilization. Development of these devices has played a significant role in the progress and safety of OPCAB surgery.

Proximal and distal occlusion of the coronary artery is accomplished by encircling suture or soft, flexible, Silastic string around the vessel. Distal occlusion is not always necessary. Intracoronary shunts are often used to reduce ischemia during this time, and along with a carbon dioxide-containing saline mister-blower, provide better visualization of the anastomosis.

ANESTHETIC MANAGEMENT

Patients should be evaluated thoroughly regarding cardiac status (e.g., number of vessels to be revascularized, site and type of lesion, LVEF, presence of any significant valvular abnormality). A careful history, physical examination, and review of the diagnostic studies, focusing on the pulmonary, renal, hepatic, and neurologic status, are of paramount importance. P.646

Premedication should be customized according to the patient's condition. Scopolamine should be avoided to prevent delayed anesthesia recovery.

Anesthetic management goals of OPCAB include maintenance of hemodynamic stability (especially during frequent repositioning of the heart and intermittent coronary occlusion), *maintenance of normothermia, early detection and prompt management of myocardial ischemia, provision of adequate postoperative analgesia, rapid emergence, early extubation, and early patient mobilization.*

Patientwarming systems (e.g., forced convective air warming blanket [Bair Hugger, Arizant, Inc., Eden Prairie, MN] or hydrogel energy conduction pads [Kimberly-Clark Patient Warming System, Kimberly-Clark Health Care, Roswell, GA]) *and warmed intravenous (IV) fluids should be routinely used.* Room temperature may need to be adjusted to prevent passive loss of heat to the environment and the potential development of a hypothermiarelated

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coagulopathy.

Arterial catheterization, large-bore peripheral IV access (14- or 16-gauge), and central venous access (e.g., a triple-lumen catheter or pulmonary artery catheter) are standard for OPCAB. Pacing and defibrillator capabilities should be immediately available if they are required; the pacing pulmonary artery catheter can be a useful adjunct in this regard.

Monitoring of electrocardiogram (ECG) leads II and V5 is important for detection of ischemia. Transesophageal echocardiography (TEE) is also helpful for early detection of ischemia (e.g., segmental wall motion abnormality or development of ischemic mitral regurgitation) and the development of ventricular systolic or diastolic dysfunction, and for monitoring the volume status. The cardiac displacement required for this procedure can make obtaining diagnostic TEE images challenging. The ECG diagnosis of myocardial ischemia is also challenging as a result of the frequent axis shifts of the heart that accompany repositioning.

Any of the available IV induction agents can be titrated, depending on the patient's hemodynamic status. Fentanyl 7.5 to 10 mcg/kg or sufentanil 0.5 to 1 mcg/kg is usually adequate to blunt the hemodynamic response to airway manipulation. *Always remember that high-dose, narcotic-based anesthetics are not advisable when early extubation is the goal.* Intermediate-acting muscle relaxants (e.g., vecuronium, rocuronium, cisatracurium) are preferable over the longer-acting agents.

Any of the inhalational agents (isoflurane, sevoflurane, or desflurane) can be used for maintenance. Nitrous oxide can also be used but should be turned off promptly if conversion to CPB is anticipated.

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Hypotension associated with retraction and compression of the heart is primarily managed by preloading with crystalloid and/or colloid solution. In general, IV fluid is used more liberally as compared to standard CABG. Overall, fluid management should be guided by the cardiac filling pressures and echocardiographic appearance of the volume status of the heart. Hypotension can also be managed by the Trendelenburg position, lightening of anesthesia, and use of vasopressors such as Neo-Synephrine, norepinephrine, and ephedrine. Underlying arrhythmias should be corrected pharmacologically or electrically (e.g., with cardioversion or pacing). Inotropes such as milrinone, dobutamine, or epinephrine may be used in patients with a low cardiac output state. *Always consider that the hemodynamic instability associated with OPCAB positioning may be related to compromised right heart filling; therefore, it is frequently helpful for the surgeon to open the right pleural space to allow for more efficient right heart filling during maximal cardiac displacement.*

Management of intraoperative ischemia includes the IV administration of nitroglycerin and the use of beta-blockers (to reduce myocardial oxygen consumption) and alpha-adrenergic agonists to maintain coronary perfusion pressure. Endocardial or epicardial pacing may be required in the event of severe bradycardia or heart block. Coronary perfusion and cardiac output should be optimized instead of merely treating the systemic blood pressure. Use of intracoronary shunting during the distal anastomosis can be helpful. Initiation of intra-aortic balloon pump counter pulsation therapy may be needed in select cases. The surgeon can also release the coronary occlusion or pericardial compression temporarily, if feasible. Ischemic preconditioning (transient occlusion of the coronary artery) before initiating completion of the distal anastomosis has been observed to reduce myocardial damage in some studies. *In resistant cases, CPB may have to be initiated following appropriate systemic heparinization.*

Anticoagulation varies among different institutions from a full dose of heparin (3-4 mg/kg or 300-400 IU/kg) in multivessel anastomoses to often much lower doses when one or two vessel anastomoses are planned. *Heparin reversal strategies are also variable in that it is not routinely or completely reversed.* Some literature suggests that OPCAB patients are hypercoagulable, presumably due to the lack of hemodilution and platelet dysfunction associated with CPB.

Extubation in the operating suite can be contemplated with hemodynamically stable patients that demonstrate acceptable postoperative thermal and metabolic profiles. The majority of patients are extubated in the ICU within 1 to 2 hours, although this varies on case-by-case and institutional basis.

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TAKE HOME POINTS

- In OPCAB, the anesthesiologist plays a more active role as compared to on-pump CABG, where the perfusionist maintains the patient's circulation during revascularization. Continuously communicating with the surgeon; observing the operative field to follow/anticipate the steps of surgery; frequently changing the position of the operating table/heart; and closely monitoring the patient's ventilation, hemodynamics, and hematologic parameters, coupled with delivery of prompt and appropriate corrective measures, requires vigilance from the anesthesia provider.
- Hemodynamic instability associated with OPCAB is common. Allowing time for the heart to adjust to the position while providing appropriate supportive measures is often all that is needed to achieve acceptable hemodynamics.
- Always consider that the hemodynamic instability associated with OPCAB positioning may be related to compromised right heart filling; therefore, it is frequently helpful for the surgeon to open the right pleural space to allow for more efficient right heart filling during maximal cardiac displacement.
- The principal advantages of OPCAB as compared to on-pump CABG are directly associated with avoiding CPB and the complications related to it, which include platelet dysfunction, coagulation abnormalities, increased blood loss, increased transfusion of blood products, metabolic/stress response related to extracorporeal circulation, and CPB-associated systemic inflammatory response.
- Avoiding aortic manipulation minimizes the complications related to it (e.g., aortic dissection, dislodgement of the atheromatous plaques and embolization).
- OPCAB may facilitate early extubation, early ICU discharge, and shorter hospital lengths of stay. OPCAB may reduce the overall cost associated with coronary surgery by as much as 15% as compared to standard CABG.
- The incidence of type I neurologic complications such as stroke, transient ischemic attack (as diagnosed on anesthesia emergence), or cerebrovascular accident occurring later in the postoperative course is significantly lower in some OPCAB cohorts as compared to standard CABG patients.
- The incidence of postoperative atrial fibrillation is lower in OPCAB patients as compared to standard CABG for patients older than 70 years. The evidence is less clear in younger patients.
- Studies have failed to show any evidence that OPCAB provides protection against postoperative pulmonary dysfunction as evidenced by observed alveolar-arterial oxygen gradients.

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- The incidence of postoperative renal dysfunction is not reduced in OPCAB patients.
- Early patency of the grafts is comparable in OPCAB and standard CABG. The success of long-term OPCAB graft patency is not clear at present.

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Editors: **Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.**
Title: ***Avoiding Common Anesthesia Errors, 1st Edition***

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Coagulopathy in the Cardiac Surgical Patient—It's Not Just about the Numbers

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You are the anesthesia provider for a 71-year-old man with multivessel coronary artery disease and normal preoperative labs who is undergoing a whopper of a coronary artery bypass graft (six vessels!) on cardiopulmonary bypass (CPB). Anticoagulation was managed with appropriate heparinization and protamine reversal as dictated by the HEPCON Heparin Management System. Amicar (Medtronic, Inc., Minneapolis, MN) was given pre-CPB in anticipation of a long CPB run (187 minutes). Following heparin neutralization, the floodgates appear to burst open and profuse bleeding from the surgical field is apparent; the hemoglobin measured just before CPB separation is 10 mg/dL. The surgeon notes that there is no apparent "surgical bleeding," only diffuse oozing from all tissues and anastomoses. He requests immediate administration of fresh frozen plasma (FFP), cryoprecipitate, banked red blood cells (RBCs), and "rescue aprotinin therapy." Central laboratory coagulation studies are sent off STAT. FFP, platelets, and banked RBCs are immediately available in the operating room. What is the best course of treatment for the patient at this time?

Start a thromboelastograph (TEG) right away if available; it will help diagnose the nature of the coagulopathy within 15 minutes. Do not administer "rescue aprotinin" to patients already treated with an antifibrinolytic. In the absence of preoperative antiplatelet or antithrombotic therapy, the bleeding is most likely due to the platelets getting knocked around in the CPB circuit (mechanical shear stress activation) during the prolonged pump run. Administer 6 U of platelets pronto! Platelets should not be given too quickly to avoid hypotension associated with bradykinin release. Give crystalloid to maintain intravascular volume. Do not inject concentrated calcium solutions in the same intravenous line as the platelets because it will activate them in the tubing! Following administration of the platelets, the field becomes considerably less oozy, and the central laboratory reports a platelet count (drawn pretransfusion) of 140 th/mm3; the activated partial thromboplastin time (aPTT) is normal, and the prothrombin time (PT) is prolonged. No additional blood products are given. The platelet count was not helpful in diagnosing the nature of this coagulopathy because many of the counted platelets were dysfunctional or "spent platelets." The PT was
also misleading. Pretransfusion TEG confirms poor platelet function and normal coagulation protein function. P.651

If anyone is going to experience a clinically significant perioperative coagulopathy, it will probably be a cardiac surgical patient. The complexity of cardiac surgical procedures distinguishes the coagulopathy seen in these patients. Remember that cardiac surgery patients are routinely exposed to variable time periods of CPB. Whether the Silastic CPB circuit is uncoated, bonded with heparin, or bonded with a uniquely charged coating (Smart tubing, SMART; Cobe Cardiovascular, Arvarda, CO), the coagulopathy observed has been shown to correlate with the length of time spent on CPB (i.e., there is an increasing incidence of coagulopathy as CPB time increases). *In general, CPB times exceeding 3 hours should alert the clinician to the potential for development of a clinically significant coagulopathy.* Blood-synthetic tubing/filter interfaces and blood-air interfaces within the CPB circuitry initiate activation of systemic inflammatory pathways. Systemic inflammation is tied to the coagulation system and can provoke imbalance toward hypocoagulability. CPB-associated mechanical trauma to the blood elements (e.g., CPB induces shear stress injury, which activates platelets) causes anemia, thrombocytopenia, and a functional platelet defect that contributes to the development of coagulopathy. In addition, the same processes associated with the conduct of cardiac surgery reproducibly manifest an antifibrinolytic state that is only partially overcome through the pre-CPB initiation of antifibrinolytic agents. Furthermore, because many cardiac surgery patients have undergone previous percutaneous coronary interventions (e.g., coronary stenting and/or angioplasty) or present with a low cardiac output syndrome, they are routinely treated with antiplatelet and/or antithrombotic agents. The cardiologists love to give these drugs that cause cardiac surgery patients to bleed! These treatments are associated with increased postoperative hemorrhage and the increased need for transfusion of allogeneic blood products. Several antithrombotic (e.g., low-molecular-weight heparin) and antiplatelet (e.g., clopidogrel) agents cannot be neutralized pharmacologically and will require the administration of allogeneic blood products to overcome their effects.

The potentially costly and morbid consequences of mistreating the coagulopathy observed in cardiac surgery patients calls for the use of point-of-care testing as well as standard central laboratory coagulation tests. The PT measures the integrity of the extrinsic and common coagulation pathways. It can be prolonged in the setting of factor VII deficiency, Coumadin therapy, or vitamin K deficiency. Unfractionated heparin can also prolong the PT due to inactivation of factor II. *The PT is a poor predictor of postoperative hemorrhage in cardiac surgery patients and is therefore not appropriate to use as an independent test of coagulation.* The aPTT tests the integrity of the
intrinsic and final coagulation pathways. The aPTT is prolonged in patients being treated with unfractionated heparin as well as in patients with deficiencies of factors XII, XI, IX, and VIII; high-molecular-weight kininogen; and kallikrein. The activated clotting time (ACT) is the point-of-care test that is used in cardiac surgery to monitor the effect of blood heparin levels, and it correlates with the bleeding related to dysfunction within the intrinsic and final coagulation pathways. Of note, the ACT does not correlate well with plasma heparin levels in hypothermic, hemodiluted patients on CPB and thus may potentially mislead clinicians to underdose heparin. Patients with marked thrombocytopenia (<50,000/μL) and those treated with platelet inhibitors may exhibit an abnormally prolonged ACT. Platelet lysis, anesthesia, and surgery can decrease the measured ACT value. *The platelet count will not always predict whether a bleeding cardiac surgery patient requires a platelet transfusion because CPB frequently induces a functional platelet defect.* P.652

Increasingly, TEG is being employed as a point-of-care test to rapidly evaluate the integrity of the coagulation cascade, platelet function, platelet-fibrin interactions, and fibrinolysis in cardiac surgery patients. TEG involves testing the viscoelasticity of a clot and the temporal nature of clot formation. TEG values include the reaction time (R), the coagulation time (K), or angle (α), the maximal amplitude (MA), and the percent lysis at 30 minutes (LY30). R represents the time for initial fibrin formation, which is a measure of the intrinsic, extrinsic, and common pathways. K or α are measures of the speed of clot formation; they reveal the level of functional fibrinogen. MA is an index of the tensile clot strength and is predominantly determined by platelet function and platelet-fibrin interaction. LY30 indicates the percentage of clot lysis that has occurred after 30 minutes, and it can reveal abnormal fibrinolytic states. Particular coagulation defects have characteristic TEG tracings that the clinician is aided in interpreting by the software that drives the TEG device. A number of trials have confirmed that the use of TEG in cardiac surgery is more predictive of bleeding than routine coagulation studies. The concurrent use of TEG with a transfusion algorithm has been demonstrated to result in significant reductions of re-exploration for hemorrhage and in the need for transfusion of blood products.

TAKE HOME POINTS

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- The complexity of the cardiac surgery-associated coagulopathy calls on the clinician to employ the results of both central laboratory coagulation tests (e.g., activated partial thromboplastin time [aPTT], prothrombin time [PT], and platelet count) and point-of-care tests (e.g., activated clotting time [ACT], thromboelastography [TEG]) to guide the administration of allogeneic blood products and ultimately restore normal coagulation function. P.653
- The ACT does not correlate well with plasma heparin levels in hypothermic, hemodiluted patients on cardiopulmonary bypass (CPB) and is generally a poor test for use in revealing trace amounts of unfractionated heparin when compared to the aPTT.
- The platelet count will not always predict whether a bleeding cardiac surgery patient requires a platelet transfusion because CPB frequently induces a functional platelet defect.
- The post-CPB initiation of antifibrinolytic therapy (e.g., ε-aminocaproic acid, tranexamic acid, or aprotinin) has not been prospectively evaluated in cardiac surgery patients and therefore is not recommended as a primary treatment for this coagulopathy. The safety of this practice has not been determined, and administration of these agents should begin prior to CPB.
- The combined use of aprotinin with either ε-aminocaproic acid or tranexamic acid has not been prospectively studied as a treatment option for this coagulopathy and therefore should not be considered.
- Cardiac surgery-associated coagulopathy should be anticipated in patients that have experienced CPB times in excess of 3 hours.
- Several antithrombotic and antiplatelet agents cannot be neutralized pharmacologically and require the administration of allogeneic blood products to overcome their effects. Select patients treated with these agents will benefit from the delay of elective cardiac surgery.
- Rebound heparinization should be considered as a contributing cause of cardiac surgery-associated coagulopathy, and can be effectively diagnosed (e.g., through the use of the heparinase TEG or the HEPCON Heparin Management System) and treated (e.g., administration of additional protamine sulfate).

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Remember There Are Specific Do's and Don'ts of Anesthesia Care for Hypertrophic Obstructive Cardiomyopathy

Cosmin Gauran MD

Edward George MD, PhD

A 27-year-old professional snowboarder experienced a syncopal episode while training on a closed ski slope at Snowbird. The "Dude" (as he likes to call himself) was found at the scene in ventricular tachycardia and was successfully cardioverted by the emergency medical response team. On questioning, he admitted to experiencing some vague chest discomfort and shortness of breath over the past several months that he attributed to the altitude (>8,000 ft) and some "vigorous aprés ski celebrating." Following some strong encouragement from his adoring entourage, he agreed to forego the rest of the day's snowboarding to visit the local hospital. Medical evaluation demonstrated the presence of hypertrophic obstructive cardiomyopathy (HCM).

Now, adorned with his orange lens shades, he presents to your preoperative clinic for anesthesia consult prior to surgical septal myomectomy. Review of his past medical and surgical history is unremarkable. Family history is significant for a paternal uncle and grandfather with sudden death in their twenties. He takes atenolol 100 mg each morning and has no allergies. Vital signs are blood pressure 110/62 mm Hg, heart rate regular at 64/minute, and 100% oxygen saturation on room air. Physical exam revealed a noticeable double apical impulse and III/VI systolic ejection murmur at the left sternal border with radiation to the sternal notch, but not to the common carotid arteries. The murmur intensifies with upright posture, Valsalva maneuver, and squatting. The electrocardiogram showed normal sinus rhythm with electrical criteria for ventricular hypertrophy and diffuse depression of the ST segments. Chest film revealed a minimally increased cardiac silhouette. Echocardiography revealed ventricular hypertrophy with a marked asymmetrically enlarged upper interventricular septum. The mean gradient across the left ventricular outflow tract (LVOT) was calculated to be 55 mm Hg. The mitral valve showed signs of systolic anterior motion (SAM) and mild to moderate mitral insufficiency. Cardiac catheterization revealed normal coronary anatomy and confirmed the LVOT gradient. He wants to know how soon he could be back on the slopes.

HCM (formerly known as HOCM or idiopathic hypertrophic subaortic stenosis [IHSS]) is increasingly recognized as a relatively common (i.e., estimated incidence of approximately 1/500) and underdiagnosed congenital cardiac abnormality. Although the disease is known to be a frequent cause of sudden cardiac death in young people, the majority of affected patients is minimally symptomatic and has a normal lifespan. P.655

HCM is an autosomal disease with variable penetrance associated with mutations in the sarcomeric proteins. The main pathological mechanisms include hypertrophy, obstruction, diastolic dysfunction, and arrhythmias. Asymmetric hypertrophy is the trademark of the disease, with the interventricular septum being the most common area of involvement. Upper septal hypertrophy creates an area of narrowing and increases the velocity of the blood in the LVOT, which in turn leads to a Venturi effect and SAM of the mitral valve. The end result of this physiological pattern is a characteristic dynamic obstruction in mid to late systole. SAM resulting in significant mitral regurgitation will decrease forward cardiac flow. Increased inotropism, chronotropism, and hypovolemia can accentuate the dynamic obstruction in HCM patients. The diastolic dysfunction associated with HCM translates into decreased ventricular compliance and leads to requirements for higher cardiac filling pressures and a dependence on the atrial contribution for adequate cardiac output. Any type of arrhythmia can be associated with HCM, particularly atrial fibrillation (i.e., there is an incidence of up to 25%) and malignant ventricular arrhythmias; thus, the risk of sudden cardiac death becomes apparent.

Medical management of HCM involves aggressive treatment with beta-blockers to reduce the inotropic state of the heart, to decrease heart rate to allow adequate filling time, and to promote maintenance of normal sinus rhythm. Therapeutic approaches available for symptomatic patients not responding to medical management are septal ablation with alcohol injection using percutaneous techniques (increasingly popular, although not an option for all HCM patients) and the Moro surgical septal myomectomy. The Moro procedure involves a transaortic approach to the LVOT and uses cardiopulmonary bypass with cardiac arrest. Complete heart block and creation of a ventricular septal defect (VSD) are associated complications of this procedure. HCM patients with a history of a malignant ventricular arrhythmia should have an implantable cardioverter-defibrillator (ICD) placed.

TAKE HOME POINTS

- In providing anesthesia care for an HCM patient, DO be certain that the patient has undergone a recent echocardiographic assessment of their cardiac anatomy to gauge progression of their risk for dynamic obstruction. A complete history (i.e., take care to inquire about shortness of breath, syncope, anginal symptoms, palpitations, and history of arrhythmias) and physical exam may provide clues to pathology progression but should not replace echocardiographic examination in elective surgeries. P.656
- In providing anesthesia care for an HCM patient, DO be certain that if the patient has a strong family history of sudden death, a personal history of syncope, or a malignant arrhythmia that they have a functional ICD, or are cared for perioperatively in a monitored bed with the means to immediately provide electrical therapy for a perioperative malignant arrhythmia. A negative electrophysiology study is not a sensitive predictor as to whether an HCM patient will suffer a malignant arrhythmia.
- Although the preferred anesthetic approach for HCM patients requiring both cardiac and noncardiac surgery is the use of general anesthesia, DO consider that regional anesthesia can be safely used (for noncardiac surgical procedures only) as long as the patient is given adequate anxiolytics and volume loaded prior to central neuraxial blockade. Vigilant maintenance of systemic vascular resistance with alpha-agonists (e.g., phenylephrine infusion) is required with both general and regional anesthetic techniques. Arterial catheterization and monitoring promotes the success of these techniques.
- In providing anesthesia care for any procedure (cardiac surgery or noncardiac surgery) for an HCM patient, DO consider the use of central pressure monitoring (i.e., following hemodynamic trends can be useful in these patients that may have elevated cardiac filling pressures related to chamber noncompliance) or preferably transesophageal echocardiography (TEE) to assess the adequacy of ventricular filling and the degree of dynamic LVOT obstruction.
- In providing anesthesia care for an HCM patient undergoing septal myomectomy, DO consider that complete heart block and creation of a VSD are complications of this procedure. Preoperative placement of an AV Paceport pulmonary artery catheter will provide the ability to

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deliver cardiac pacing in the event of perioperative complete heart block. Performing a complete post cardiopulmonary bypass TEE exam is essential and will rule out the presence of an iatrogenic VSD. In addition, it is important to document the resolution of the LVOT gradient following septal myomectomy.

- In providing anesthesia care for a patient without a history of HCM who has undergone a mitral valve repair (MVR), DO consider that SAM is a potential complication in this circumstance, although the genetic sarcomeric abnormality does not exist in these patients. Medical treatment may be all that is needed, and the return to cardiopulmonary bypass for MVR revision following MVR is not always necessary. Experienced anesthesia and surgical clinicians should be involved in such a decision and base their therapy on the postcardiopulmonary bypass TEE exam results.
- In treating hypotension in HCM patients, DO NOT employ agents (e.g., dopamine, milrinone, norepinephrine, ephedrine) with mixed inotropic, chronotropic, and peripheral vascular effects. Consider that the increase in heart rate and the inotropic state of the heart can acutely worsen hypotension related to dynamic LVOT obstruction. Phenylephrine infusion, beta-blockers, and maintenance of adequate intravascular volume are the treatments of choice in HCM patients. P.657
- In planning the anesthesia induction of an HCM patient, DO NOT rely heavily on agents (e.g., propofol or thiopental) that acutely sympathectomize the patient and abruptly reduce both venous and arterial tone. Narcotic and benzodiazepine combinations can have more subtle sympathetic depressive effects when used judiciously. Most contemporary volatile anesthetics (e.g., isoflurane, sevoflurane, desflurane) act as systemic vasodilators and myocardial depressants, and thus they should be used cautiously if at all.

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Managing Guests on the Labor Deck Requires Clear Communication Consistently, Firmness When Necessary, and Calmness Always

Christopher E. Swide MD

The labor and delivery suite is one of the few places in the hospital where family and friends are almost universally present during procedures that are fraught with risk (the other being the induction of pediatric patients with the parent present). The obstetric anesthesiologist must have not only the clinical skills to deal with the medical management of parturients but also the social and communication skills to manage guests. In the obstetrics environment, the patient's family and friends are often in the immediate vicinity. They can be distracting to care providers and may become stressed themselves by witnessing emergency care procedures. Unlike other chapters in this book that address anesthetic emergencies, this chapter focuses on methods for handling the potential repercussions of managing anesthetic emergencies in the presence of guests.

Every anesthesia provider must develop a practice model concerning guests. It is important that this model meet the needs of all "clients" (mothers-to-be, family and guests, the baby, the obstetrician, and the labor and delivery nurses) as well as his or her own needs. Fortunately, there is help available in handling these situations:

1. **Follow institution policies.** Hospital policies govern who can be in the delivery room or operating room (OR), and include processes of limiting access for medical cause as determined by the obstetric and anesthesia providers.
2. **Communicate with the labor nurses.** The nurses taking care of the patient often have a good understanding of the relationship dynamics of the patient's family and friends. Thus, the nurses can provide excellent advice on how to best broach the subject of presence during procedures. It is also important that the nurses, physicians, and other providers on the unit make a unified decision.
3. **Communicate with the obstetric provider.** The obstetric provider can be invaluable in helping the patient and her guests understand that the people she selected to be present at her delivery may be asked to leave the room if the care management plan necessitates it.
4. **Communicate with the patient.** Discuss the potential of asking guests to leave the room if necessary during a procedure as part of the basic patient interview. It is the author's practice to *always* discuss this and to make sure the guests know that it is in the best interest of the patient. P.661
5. **Always remain calm and professional.** Even in the midst of a real emergency or with an angry patient or guest, this is an important rule. If you must ask a guest to leave, try to frame it in terms of being able to best help the mom and the baby, rather than making it a contest of wills or control struggle.
6. **Use the institution's patient advocate.** Patient advocates are trained professionals who are invaluable in dealing with stressed and angry patients, family members, and health care providers. These advocates can be used if the previous strategies are ineffective. This step almost always works to avoid utilization of the last step, which is activation of the security team.

If all else fails, and if the anesthesia provider believes that guest presence puts the patient (or the provider) at risk, the last step is calling in the institution's security team to escort the guests from the scene. Proper preparation and utilization of the other techniques almost always solves the problem before the need for security.

HELPFUL HINTS FOR MANAGING GUESTS

These tips are considered reasonable by most obstetric anesthesia providers.

Before the start of care:

- You MUST check privately with the mother-to-be regarding her desire for guests in general as well as individual guests in particular.
- Try to ascertain how much medical knowledge the guest has. Sometimes guests with a degree of medical knowledge may have control issues.
- In discussion with guests, always mention that the pace can quicken even in routine deliveries and find out how people will feel about being asked to step out.
- There are warning signs that may predict for a difficult or disruptive guest: confrontational and angry interactions with staff, poor interactions with the patient, and poor communication skills. Do not allow these people in the room.
- Exercise caution in allowing "guests to support guests." This situation sometimes arises with when the couple having the baby are themselves very young—for example, an older female family member might request to be in the room to support a teenaged father. This is felt by obstetric anesthesia providers to be a somewhat gray area. Allow it only if you feel the extra person(s) will help the care team better care for the patient.

For the labor and delivery rooms: P.662

- There is no real limit on the number of support people except for space. The nurses will often help provide crowd control in the labor rooms so things do not get out of hand. Usually everyone except one person is asked to leave during procedures; however, anesthesia providers may reserve the right to have all guests leave the room if the epidural is anticipated to be difficult.

For the OR:

- Usually only one person is allowed in the OR for C sections, sometimes more if the anesthesia provider has met them and agreed beforehand to have them as guests.

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- Guests may come and go from the OR only with an escort. Have a prearranged method to get a guest who gets woozy out of the delivery room.
- After the baby is delivered, guests may approach the baby warmer only if the okay is given by the delivery team (including the pediatric team, if they are there).
- Usually no guests are allowed in the OR until the block is in and working. However, a support person can occasionally be allowed into the room for the block, if the anesthesia providers believe that it may **help them** do their job more efficiently.

In general:

- Guests must be a minimum of 18 years of age to be in the room for C sections (unless the guest is the father of the baby). There is no age minimum for labor and delivery rooms.
- Expect as you gain experience that your practices will both loosen and tighten. This author has loosened up on the number of people in the OR, provided they do not interfere with the tasks of the anesthesia team, but tightened up about moving guests out if needed and *very* much tightened up about only one support person for labor epidurals and no videotaping of procedures.
- The doula is a member of the patient's care team and does not count as a guest.
- No cell phones are allowed in clinical areas.

A final note from F. Jacob Seagull, PhD: Successful obstetrical anesthesia providers instinctively use a human factors approach for the management of guests in the obstetrical suite. In other words, you will do well to create an environment in which expectations are clear, and plans are discussed in advance. Doing this means that when the time comes, you will be activating an existing plan, not explaining a new plan, managing expectations, explaining your actions or trying to calm worried guests. By discussing the actions beforehand, you can address any questions while everyone is calm. You can effectively "offload" the bulk of the tasks involved in getting people out of the labor and delivery rooms to your low-workload times. This offloading is what experienced anesthesia providers do in planning any difficult case, by preparing in advance for contingencies.

Tell the guests that they may be asked to leave for a number of reasons pertaining to the ongoing management of the patient, (noise level too high, arrival of extra care team providers, need to treat the patient for nonlifethreatening events such as vomiting, a new peripheral IV, etc.) but that are not indicative of a true crisis. Guests will have a cognitive framework of the range of possible circumstances in which they might be asked to leave, and therefore may not assume the worst. They will be happy to "implement a plan" when the time comes; you will be happy that you don't have to explain the situation. The goal is to try to arrange a win-win situation.

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"I Want My Epidural Now!" the Effects of Epidural Analgesia on Progress of Labor and Delivery

Patrick G. Bakke MD

Jennifer Cozzens MD, MPH

It is common on the labor and delivery ward to hear the nurses and doctors tell patients that they cannot have an epidural until cervical dilation reaches 4 cm. The reasons given for this include that labor will be prolonged, cesarean section rates increased, or that pain relief is just not needed before that point. In stark contrast is the American College of Obstetrician and Gynecologists (ACOG) recommendations that timing of epidural analgesia for labor and delivery is when the patient asks. That said, what are the impacts of epidural analgesia on labor and delivery?

The stages of labor are typically divided into the first, second, and third stages of labor. The first stage of labor can be separated into the early and active phases. Traditionally, early labor involves the onset of contractions and cervical dilation until 4 cm. The next portion of the first stage is known as the active phase and involves the cervix dilating from 4 to 10 cm. The second stage of labor is from complete cervical dilation until birth. The third stage of labor is from the time of birth until delivery of the placenta. In 1993, Thorpe published a paper stating that there was an increased risk of dystocia in nulliparous women who had epidural analgesia for labor prior to cervical dilation of 4 cm. This is where the "no epidural until after 4 cm" idea originated. Subsequently, in several well-designed randomized clinical trials, Chestnut showed this not to be the case. Since the 1990s, clinical studies have not shown that epidurals increase the cesarean section rate, so we can reassure our patients that the available evidence does not support delaying pain relief due to fear of increasing a woman's risk of cesarean section.

However, epidurals may affect the duration of labor. That is, the placement and use of an epidural for labor and delivery may prolong the first and second stages of labor. Typically, the first stage may be prolonged up to 40 minutes. Failure to progress should not be defined until the patient has achieved the active stage of labor. During the active stage, a number of indices are used to define failure to progress; however, epidural analgesia has not been associated with failure to progress to the second stage of labor. The second stage of labor has been reported to be 15 to 36 minutes longer with the use of epidural analgesia. The ACOG suggests that a prolonged second stage of labor should be considered when the second stage exceeds 3 hours if regional anesthesia is administered or 2 hours in the absence of regional anesthesia in nulliparous women. In multiparous women, such a diagnosis can be made if the second stage of labor exceeds 2 hours with regional anesthesia or 1 hour without. There is no significant prolongation of the third stage of labor with epidural analgesia.

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Because use of an epidural in labor can prolong the first and second stages of labor, does this have an impact on instrument delivery or cesarean section? There may be an increased incidence of forceps- or suction-assisted delivery in patients who use epidural analgesia during labor. This is believed to be due to decreased maternal efforts at expulsion during delivery and not dystocia. For cesarean sections, populationwide studies have shown that the use of epidural analgesia for labor and delivery does *not* increase the rate of cesarean deliveries.

Most important, patients have lower pain scores and higher patient satisfaction with an epidural for labor and delivery compared with other forms of analgesia. When you couple these facts with the findings of neonatal Apgar scores, which are higher in infants born to mothers who used an epidural versus intravenous analgesia during labor, it is hard to argue against the use of epidurals for pain control during labor and delivery.

TAKE HOME POINTS

- The proper timing of an epidural is when the patients request the epidural and the obstetric providers are committed to delivery.
- The first stage of labor can be prolonged by as much as 40 minutes.
- The second stage of labor can be prolonged by 15 to 36 minutes.
- Neither failure to progress nor second stage arrest is increased by the use of an epidural.
- Instrumented delivery for dystocia is not increased.
- Cesarean section rates are not increased.
- Patients have lower pain scores and higher satisfaction.

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If Carefully Selected, Patients in Labor Can Ambulate Safely—The Key is the Use of Low-Dose Epidural Analgesia

Christopher M. Colville MD

Christopher E. Swide MD

Effective analgesia for women in labor has become a reality since the 1950s. Labor suites have progressed from the time of sedated mothers and babies to modern central neuraxial techniques managed by professional anesthesia providers. Women can now enjoy a comfortable labor with safe, effective pain control. Many women and their obstetric providers believe that walking during labor can speed progress and provide comfort to the parturient. We can now provide effective pain control while preserving motor function, so the question becomes "does ambulation play a role for those patients with labor epidurals?"

What does ambulation do for mothers? The medical literature is unclear as to whether ambulation truly helps with the outcome of labor despite the common wisdom that this is true. Ambulation for the laboring patient theoretically can result in fairly substantive benefits. Faster labors, fewer instrumental and operative deliveries, and better maternal and infant outcomes are some of the alleged advantages to having the parturient walking early and often. Decreases of up to 2 hours in the first stage of labor, decreased requests for analgesia, lower rates for instrumental deliveries, and improved Apgar scores have all been reported in patients who walked after placement of the labor epidural. However, other studies have not found statistically significant changes in length of labor (from epidural insertion), Apgar scores, or cesarean delivery rate for these walking patients. The current literature has not revealed evidence of harm to the mother or baby if ambulation is allowed in the properly selected laboring patient. Patient satisfaction seems markedly improved because the majority of women who did ambulate in labor with analgesia reported that they would do so again for future pregnancies. Implicit in this satisfaction may be the decreased motor block that allows ambulation in the first place. With increased lower extremity mobility, parturients seem to feel greater independence from the labor bed. The low-dose epidural dosing studied (and used in many centers) also allows patients to void with greater autonomy. Patients have reported a somewhat greater satisfaction of the entire laboring experience when being allowed to walk, even if that ambulation is of short duration (e.g., walking across the room to the toilet). What is clear is that women like to walk in labor, and they particularly like to walk with adequate pain control, so patient satisfaction can be improved even if we cannot prove any medical benefits of ambulation.

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Is it safe for laboring patients with epidural analgesia to ambulate? Safety concerns can be separated into two main classes: maternal safety and fetal safety. Maternal safety depends on having a protocol in place that addresses the main safety concerns in ambulatory epidurals, which are patient falls and hemodynamic stability. Patients who are allowed to ambulate with neuraxial labor analgesia need to be carefully selected and approved by the attending obstetrician or midwife and anesthesia provider after careful review of the patient's medical and obstetric history. In addition, this "freedom from the bed" should only be permitted with close ambulation supervision and with appropriate equipment (e.g., portable pumps, easily mobile intravenous [IV] stands). If medically dictated, at the discretion of the attending obstetrician or midwife, fetal heart rate monitoring will need to be portable, with mechanisms in place for remote monitoring units. Also, having a "rest" area at the nurses' station where the mom can have her blood pressure, heart rate, and fetal heart rate measured every 15 minutes facilitates nursing efficiency. A standard protocol is waiting 30 to 45 minutes after initial dosing, and then having the anesthesia provider examine the patient for signs of motor weakness and hemodynamic instability. A simple test is the modified Bromage score. If the patient can flex the leg at the hip and resist the examiners hand, then she is given a score of 5, and if she can stand from the bed with minimal assistance (deep knee bend), then she is given a score of 6 and is cleared to ambulate. To test hemodynamics, the patient is asked to stand, and both systolic blood pressure and fetal heart rate are measured to look for significant changes. Ambulation is then allowed using the patient's support person or designated helper to walk with her for assistance. The use of appropriate IV poles can help facilitate walking. Fetal safety depends on maintaining adequate perfusion of the placenta by ensuring maternal safety. For properly selected patients, there is no evidence in the literature of fetal/maternal harm from walking in labor.

How do we provide labor analgesia and ambulation? The key is in the use of epidural solutions using low concentrations of local anesthetic with narcotics. Examples of effective solutions are 1/16% to 1/20% bupivacaine or ropivacaine, with 0.5 to 1 mcg/cc sufentanil, or 1 to 2 mcg/cc fentanyl, which can provide effective analgesia without motor blockade. Techniques using low-dose combined spinal-epidural analgesia (2.5 mg plain bupivacaine, 5 µg fentanyl for the spinal with epidural dosing 40 minutes later with 0.1% or 0.05% bupivacaine and 2 µg/mL fentanyl) have been shown to allow for effective ambulation. In addition, earlier ambulation of the laboring patient can be obtained by omitting the lidocaine-epinephrine test dose and replacing it with an epidural bolus of 12 mL of 0.125% bupivacaine or using one of the dilute local anesthetic/narcotic solutions. The attending anesthesiologist needs to weigh the benefits of a test dose against the benefits of ambulation in this patient population.

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Is ambulation with analgesia (primarily epidural based) the best solution for every laboring patient? As with everything in medicine, there are no absolutes. However, it is clear that there seems to be little harm in allowing selected patients to ambulate if the appropriate analgesic technique is chosen. Finally, the patient and her family, as well as the entire labor and delivery staff (obstetricians, family medicine physicians, midwives, nurses, certified registered nurse anesthetists, and anesthesiologists), need to be educated on the benefit of ambulation with analgesia.

TAKE HOME POINTS

- Patients can walk in labor with analgesia.
- Patients generally like this option to be available to them (even if they choose not to ambulate).
- Patient selection is a key to success. The obstetric provider needs to evaluate the patient and approve ambulation with analgesia.
- Epidural and Combined Spinal Epidural (CSE) solutions must be chosen to allow minimal motor block (all use combinations of dilute local anesthetics with narcotics).
- Proper monitoring must be available.
- Patients, nursing staff, and obstetric providers must be educated on the benefits of ambulation, and a proper protocol must be in place for all

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Nothing By Mouth Guidelines During Labor—"Can I Please Just Have Some Water?"

Patrick G. Bakke MD

There has been increasing pressure to liberalize oral intake during the active stage of labor. This has potentially disastrous consequences for patients with comorbid disease or those needing an emergent general anesthetic. Nothing by mouth (NPO) policies were first introduced on labor and delivery wards in the late 1940s and early 1950s. The hope was that by making patients NPO, the devastating consequences of pulmonary aspiration of gastric contents could be avoided if general anesthesia becomes necessary. Curtis Mendelson, MD (who was actually an obstetrician, not an anesthesiologist), first illustrated the dire consequences of aspiration in 1946. In his study of more than 44,000 pregnancies, 66 women experienced pulmonary aspiration of gastric contents. Of these 66, only 2 died, and the suggested cause of death was asphyxiation by solid food. Those who developed aspiration pneumonitis survived. This may be a good point to emphasize—volume, pH, and particulate matter are the main determinates of pulmonary injury and outcome following aspiration.

Factors that increase the risk of aspiration for the laboring woman can be broken down into two broad categories. First are the physiological changes due to pregnancy. These changes start in the second trimester. Increasing intragastric pressure is due to the expanding uterus. At the same time, the lower esophageal sphincter is pushed up and to the left, a similar situation to a hiatal hernia. Sphincter tone is decreased due to circulating hormones, mainly progesterone. This leads to an incredibly high incidence of gastric reflux in pregnant women, even if asymptomatic. Gastric pH becomes more acidic as the placenta begins to produce gastrin. The active phase of labor slows gastric emptying. It should be noted, however, that pregnancy, by itself, appears to have no effect on gastric emptying.

Second are the difficulties that laboring women present in airway management. It is well known that the morbidity and mortality associated with anesthesia in pregnant women are primarily related to difficulties in controlling the airway and intubation. The airway is more edematous with increased vascularity and friability. Young women tend to have intact dentition. Increase in breast size during pregnancy and the gravid abdomen make positioning of the laryngoscope difficult at best.

So, we have a situation in which there is an increased gastric volume, decreased pH, and delayed gastric emptying during the active phase of labor. The good news is that there have been advances in anesthetic techniques since 1946. The advent of rapid sequence intubation with cricoid pressure in the 1970s and the more heavy reliance on regional techniques may have played a role in the decline seen in maternal deaths since Mendelson's study. In addition, treatment with H_2 blockers and nonparticulate antacids may have helped. There has been increased training of anesthesiologists since that time as well. If we couple these with NPO guidelines, it would seem that we are following the Hippocratic Oath to do no harm. Or are we? P.670

In the 1960s, a body of literature began to emerge regarding maternal ketosis in the third trimester of labor. The increased metabolic demands of labor only serve to increase the production of ketones. These concerns led to the use of dextrose-containing intravenous solutions. However, the downsides of this practice quickly led to its discontinuation. One of the best studies of eating during labor came from Scrutton et al. in 1999. This prospective randomized study compared water, light diet, or low-fat diet on labor outcome; aspiration risk; and metabolic profile. Although the numbers were small, they found no difference on length or outcome of labor or fetal outcome. The water-only diet had higher ketone levels, while the fed groups had higher glucose levels. Ultrasound was used to evaluate gastric volumes. The fed groups were found to have significantly higher residual gastric volumes. It is interesting to note that many studies, including the previously mentioned one, found that women were less interested in eating as labor progressed.

Current recommendations from the American College of Obstetricians and Gynecologists are for a laboring patient to be on clear liquids during the active phase of labor or NPO if a surgical delivery is anticipated or likely. Current midwifery guidelines stress that the risk of aspiration is almost exclusively anesthesia related. As such, self-determination is advocated regarding eating during labor with consideration of limiting solid or liquid oral intake with increasing risk of operative delivery. Recommendations are as follows:

- Clear liquids during the active phase of labor
- NPO for patients with additional reasons for slowed gastric emptying
- NPO for patients with an increased risk for operative delivery
- Nonparticulate antacids with H2 blockers prior to all operative deliveries
- NPO for 8 hours prior to elective operative delivery, clear liquids for 2 hours

TAKE HOME POINTS

- Aspiration of gastric contents has long been recognized as a rare but potentially devastating complication in the obstetrics suite.
- Aspiration risks occur both as the result of the physiological changes of pregnancy and as the need to manage the more difficult airway of the pregnant patient. P.671
- There has been and continues to be increased pressure on anesthesiologists to allow relaxed NPO standards.
- It has been documented that ketosis increases in the fasting parturient.
- Practical guidelines are suggested that allow a moderate amount of clear liquids in the "uncomplicated" laboring patient but maintain stricter limits for complicated, high-risk, or possible surgical patients.
- The editor recommends that anesthesia providers read Mendelson's classic 1946 study. It is one of the most influential papers in the field of anesthesia. A synopsis can be found in the September 1999 ASA newsletter at www.asahq.org that includes the following passage:

  Dr. Mendelson argued quite aggressively for better-trained personnel in the administration of anesthesia to his patients. He clearly was not happy regarding the poor, inexperienced anesthesia support his specialty was receiving at this time and suggested methods by which

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obstetricians could overcome this problem. In the discussion section of his 1946 article, Mendelson stated, “The anesthetic deserves special consideration.” He further goes on to address several important issues in the anesthetic care of the obstetrical patient, suggesting that local anesthesia would eliminate the dangers of “incompetently administered general anesthesia.” Dr. Mendelson also listed several important skills in airway management in which he believed that individuals administering an anesthetic should become proficient (e.g., skill in laryngoscopy).

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Be Prepared for the Presence of a Doula in Both the Labor and Delivery Room

Christopher E. Swide MD

Doulas are becoming increasingly popular in the United States and are active on many labor suites across the country. DONAInternational is the oldest and largest of the doula organizations in the United States. It has more than 5,800 members, including 2,300 certified birth doulas in all 50 states. Many hospitals and health systems hire doulas directly, but it is also common for doulas to contract separately with the patient. The term *doula* is derived from Greek and refers to the most important female servant in the ancient Greek household. Today's doulas are caregivers whose primary role is the emotional support of the mother during labor and delivery. Anesthesiologists and nurse anesthetists working on labor units will encounter doulas but are often confused about their role in the birth experience. It is important for them to have a general understanding of this caregiver's role to facilitate optimal care for obstetric patients.

Doulas are not licensed providers; therefore, there are no state requirements for doula education. However, DONAInternational offers a certification program that consists of completing a reading list; completing a workshop of a minimum of 16 hours; and documentation of previous experience in childbirth care by training in childbirth education or midwifery, work experience as a labor nurse, or observation of a childbirth preparation series. After completion of this program, the new doula must provide service to at least three clients and document the births with a 500- to 700-word firsthand account of each experience. In addition, the new doula must provide evaluations from three clients, three primary care providers, and three nurses or midwives.

Doulas do not provide clinical care to the mother. They do not make clinical decisions or offer opinions that might influence their clients' decisions. A doula that is also a midwife, nurse, or other care provider cannot function in those roles while serving as a client's doula. Their primary role is emotional support of the mother, and facilitating positive communication between the mother, her nurses, midwives, and physicians. They maintain continuous presence during labor and help the mother's partner be part of the experience. Although many doula clients do not want regional anesthesia for labor, a significant number of patients still expect or decide to receive a

labor epidural for pain relief. It is important for the anesthesia provider taking care of these patients to interact with the patient and the doula. Patients with doulas must consent to having the doula present for confidential medical discussions. The doula will expect to be in continuous attendance with the mother, and this should be respected unless there are medical or hospital policy rules that do not allow it. Doulas can be helpful to the anesthesiologists and nurse anesthetists, often comforting the mother during the placement of the regional block and maintaining communication patterns between the patient, the nurses, physicians, other caregivers, and the partner during labor. If a patient needs cesarean section, the doula should be included in the discussion and planning made to accommodate the doula in the operating room if feasible. In the case of urgent cesarean section requiring a general anesthetic that precludes doula presence, the situation should be discussed with patient and doula, and all efforts made to preserve the team approach for delivery of the baby and to maintain respectful interactions.

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In summary, doulas can provide obstetric patients with excellent emotional support and increase the mother's satisfaction with her birth experience. It is important for anesthesia providers to understand the doula's role and incorporate the doula into the overall care plan so patients who choose doulas can have an optimal birth experience.

TAKE HOME POINTS

- Take the time to understand the doula's role in childbirth.
- Respect the mother's choice in choosing a doula to participate.
- Introduce yourself to both the patient and the doula.
- Maintain respectful and professional interactions throughout the care of the patient.
- Learn the policy of your labor unit concerning doulas.
- Make patient care decisions and recommendations as you would with any patient in labor. Your standard of care does not change with patients using doulas.

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Urgent Cesarean Section: What's Good for Baby Is Good for Mommy?

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Karen Hand MD

Nearly one fourth of all births in the United States today are via cesarean section, a dramatic increase from the 5.5% of births in 1970. A considerable number of these fall into the classification of urgent, nonelective cesarean. In some cases (i.e., massive maternal hemorrhage, dystocia), both the mother and the baby are in jeopardy. Quite commonly, however, there is nonreassuring fetal status without concomitant maternal distress. The parturient presenting for urgent cesarean section brings with her unique anesthetic challenges, and anesthesia-related complications in this setting can be associated with considerable maternal morbidity and mortality. Indeed, anesthesia is the seventh leading cause of maternal mortality in the United States.

PHYSIOLOGICAL CHANGES ASSOCIATED WITH PREGNANCY

There are numerous physiological changes that occur by the third trimester of pregnancy, many of which potentially make anesthesia more hazardous. The anesthesiologist must be familiar with the major physiological changes that occur with pregnancy and the potential anesthetic implications (Table 156.1).

INDICATIONS FOR URGENT CESAREAN SECTION

The anesthesiologist must be in close communication with the obstetrician in determining the indication and true urgency for cesarean section. A distinction must be made between *urgent* and *emergent or stat* section, as those in the former category may allow time for regional anesthesia, while those in the latter nearly always require general anesthesia in the absence of a functioning epidural catheter. Although each individual case must be considered in consultation with the obstetrician, those falling in the **urgent but not emergent** category generally include (a) variable decelerations with prompt recovery of fetal heart rate (FHR), (b) dystocia, (c) previous classic cesarean and active labor, (d) active genital herpes and ruptured membranes, (e) ruptured membranes and abnormal fetal presentation (i.e., transverse, breech, multiple gestation), and (f) rapidly deteriorating maternal illness (pre-eclampsia, cardiac, pulmonary). Conversely, those indications generally falling into the **emergent or stat** category include (a) prolonged fetal bradycardia or persistent late decelerations (rule of 60s = FHR <60/minute **or** deceleration lasting longer than 60 seconds), (b) massive maternal hemorrhage (placenta previa, placental abruption), (c) prolapsed umbilical cord, and (d) uterine rupture.

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TABLE 156.1 PHYSIOLOGICAL CHANGES ASSOCIATED WITH PREGNANCY

SYSTEM	PHYSIOLOGICAL CHANGES AT TERM GESTATION (COMPARED WITH PREPREGNANT VALUES)	ANESTHETIC IMPLICATIONS
Respiratory	30%-40% increase in oxygen consumption 20% decrease in FRC 45% increase in minute ventilation Increased airway edema and mucosal friability	Rapid development of hypoxemia during apnea, potentially difficult intubation
Cardiovascular	50% increase in cardiac output 20% decrease in SVR Possible aortocaval compression by gravid uterus 55% increase in plasma volume 45% increase in blood volume	Aortocaval compression→ decreased preload→ hypotension in supine parturient→ uteroplacental insufficiency
Gastrointestinal	Anatomic displacement of stomach cephalad and left Decreased lower esophageal sphincter tone Delayed gastric emptying? Increased gastric acid secretion?	Increased risk for aspiration (Mendelson syndrome)

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Nervous	Increased sensitivity to IV anesthetics	Adjust doses accordingly
	MAC of volatile agents reduced 15%-40%	
Miscellaneous	Engorged breasts Increased sensitivity to nondepolarizing MR	Difficult laryngoscope insertion, reduce dosages of NDMR

FRC, functional residual capacity; SVR, systemic vascular resistance; IV, intravenous; MAC, minimum alveolar concentration; MR, muscle relaxant; NDMR, non depolarizing muscle relaxant.

APPROACH TO THE URGENT CESAREAN SECTION

Ideally, the obstetric anesthesiologist should be familiar, at least superficially, with all patients present in the labor and delivery suite. Practically speaking, however, this is difficult on a busy service. Therefore, it is imperative that an attempt be made to identify those patients at increased risk for cesarean section (see Indications for Urgent Cesarean Section section). **Once again, a good working relationship with the obstetrician will facilitate this process. At the first sign of trouble, the obstetrician should contact the anesthesiologist to allow the maximum amount of time possible for evaluation and formulation of an anesthetic plan.** Once identified, a thorough pre-operative evaluation should be performed on each patient deemed to be at risk.

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An aggressive approach should be taken in managing potentially confounding factors in each patient's anesthetic management. In those patients where difficult airway management is anticipated (i.e., excessive pharyngeal edema, obesity, short thyromental distance, poor mouth opening, poor neck extension), early placement and testing of an epidural catheter should be strongly considered. This will allow prompt initiation of regional anesthesia in the event that cesarean becomes necessary. Similarly, those patients presenting with hypertensive disorders of pregnancy (pre-eclampsia, HELLP [**H**emolysis, **E**levated **L**iver **E**nzymes, and **L**ow **P**latelets] syndrome) in whom coagulopathy is developing (increasing international normalized ratio, falling platelet count) are candidates for prompt placement and testing of an epidural catheter before coagulation parameters deteriorate to a level deemed unsafe for regional anesthesia.

All patients presenting for cesarean section should have at least one well-functioning 18-gauge or larger intravenous (IV) catheter in place. Each patient should also have a current type and screen. If atypical antibodies or other factors are present that will make cross-matching difficult, 2 to 4 U of cross-matched blood should be made available in the operating room prior to proceeding. In those patients deemed to be at increased risk for peripartum hemorrhage, it is recommended that at least one additional large-bore IV catheter be placed and that blood be available for rapid administration.

Standard American Society of Anesthesiologists (ASA) monitors are required for each cesarean section. Additional monitoring may be necessary and should be considered on a case-by-case basis. Supplemental oxygen should be administered to all patients. Left uterine displacement should be performed in all parturients to decrease the likelihood of aortocaval compression and the accompanying hemodynamic compromise to mother and fetus.

Anonparticulate antacid should be administered to **all** patients shortly prior to or on arrival to the operating room. We commonly administer an H_2-blocker and metoclopramide at the same time. Although current data are conflicting, conventional teaching states that a gastric pH of <2.5 and gastric volume of >25 mL places patients at increased risk for aspiration pneumonitis.

Frequent reassurance and comfort given to the expectant mother is paramount prior to/during the urgent cesarean section. The anesthesiologist is frequently in the best position to do this and should become adept at doing so. Otherwise, unoccupied personnel should also tend to the expectant father and other family members because this can be a time of great confusion and emotional distress for all involved.

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REGIONAL ANESTHESIA

Consideration should be given to the use of regional anesthesia, even in the face of urgent cesarean. Clearly, if a functional epidural catheter is in place, this will often provide the quickest and safest route to surgical anesthesia. For the emergent cesarean section in our practice, we dose the epidural catheter initially with 15 to 20 mL of 3% 2-chloroprocaine. Because of the low potential for systemic toxicity due 2-chloroprocaine's rapid clearance by plasma esterases, this large dose can be administered safely as a bolus or incrementally with very short dosing intervals. Adequate anesthesia generally develops within 3 to 5 minutes. It is important to remember that due to 2-chloroprocaine's short duration of action, the catheter must be redosed in approximately 25 to 35 minutes to prevent regression of the block to an unacceptable level. This is most often done with a longer-acting agent, such as 2% lidocaine with 1:200,000 epinephrine, 0.5% ropivacaine, or 0.5% bupivacaine.

Many **experienced** practitioners believe that they can successfully place a spinal anesthetic in the same amount of time it takes to induce general anesthesia. Communication with the obstetric team regarding this plan can allow the spinal to be placed while the team prepares for surgery. A sufficient block will generally have time to develop while the abdomen is prepped and draped. If complicating factors are present that may make placement of a spinal anesthetic difficult (i.e., obesity, scoliosis, previous lumbar spinal surgery), then it is best to not waste time trying to place a regional block and proceed directly to a general anesthetic. Regional anesthesia is best avoided in the face of significant bleeding/hypovolemia because the accompanying sympathectomy can cause catastrophic hemodynamic collapse.

Regardless of the type of anesthetic planned, each patient's position on the operating table should be optimized for airway management, and the proper equipment and drugs necessary for general anesthesia should be prepared in advance.

GENERAL ANESTHESIA

As mentioned previously, prior to induction of general anesthesia, a nonparticulate antacid (i.e., Bicitra) should be administered, a well-functioning IV catheter and appropriate monitors should be in place, and the patient's position should be optimized for airway management. Depending on the patient's body habitus, this may involve placing a “ramp” of towels or sheets or placement of a commercially available intubating wedge under the patient's shoulders to facilitate intubation. Several small endotracheal tubes (6.0-6.5 mm cuffed) and a variety of laryngoscope blades should be available. It is often helpful to have a short-handled laryngoscope because engorged breasts can interfere with insertion of the blade into the patient's mouth. An

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intubating stylet may also be helpful and should be at hand. Finally, backup airway equipment, including laryngeal mask airways (LMAs) of different sizes, intubating LMAs, Combitube or other airway devices, and the ability to provide jet ventilation should be immediately available. P.678

Preoxygenation is mandatory prior to induction of anesthesia. Full denitrogenation takes approximately 3 to 5 minutes of normal tidal volume breathing with 100% oxygen. However, some studies have demonstrated four to six vital capacity breaths to be nearly as effective as the traditional method. The urgency of the situation and clinical judgment should dictate which method is undertaken. **It should be emphasized that the anesthesiologist must take control of the situation and should not proceed until the previous criteria are met. With proper training and direction of operating room personnel, adequate preparation can be carried out in a very short period of time. The mother's life should not be placed in jeopardy if the anesthesiologist deems the situation unacceptably risky.** As the situation and urgency dictate, possible alternatives include awake intubation or, on rare occasions, local infiltration by the obstetrician, although the block achieved in this instance is generally not ideal.

Prior to induction, the abdomen should be prepped and draped, and the surgical team should be ready to proceed. Rapid sequence induction with properly administered cricoid pressure by an assistant is the usual method indicated for cesarean general anesthesia (GA). Depending on the situation, any of the available induction agents, including propofol (2 to 3 mg/kg), sodium thiopental (3 to 4 mg/kg), etomidate (0.2 to 0.3 mg/ kg), or ketamine (1 to 2 mg/kg) may be indicated. Succinylcholine (1 mg/kg) is administered immediately after the induction agent. If succinylcholine is contraindicated, rocuronium is an acceptable alternative (rapid sequence dosage should be reduced 30% to 50% from nonpregnant values). As soon as anesthesia is induced, the surgical team is allowed to proceed. Anesthesia is generally maintained with a low concentration (i.e., 0.5 MAC) of volatile agent in 50% to 60% nitrous oxide. Opioids are generally minimized until after delivery of the fetus to minimize respiratory depression in the neonate. If poor uterine tone is of concern after delivery of the fetus, the concentration of volatile agent may be further reduced. Extubation is carried out awake, assuring that airway reflexes are intact.

TAKE HOME POINTS

- Physiological changes of pregnancy potentially make anesthesia for urgent cesarean section hazardous.
- Communication with the obstetric team, early identification and evaluation of potential candidates, and proper anesthetic planning can greatly reduce anxiety surrounding an urgent cesarean section and may be lifesaving. P.679
- Early placement and testing of an epidural catheter should be considered in at-risk parturients.
- Preparation for general endotracheal anesthesia for cesarean section includes, at a minimum, administration of a nonparticulate antacid, placement of a well-functioning 18-gauge or larger IV catheter, application of standard ASA monitors, optimizing position for intubation, and adequate preoxygenation prior to induction.

The key to managing these patients is to remember that you have an obligation to both mom and baby, and a “nonreassuring” tracing for the baby should not trigger an unsafe situation for mom. With proper planning, communication, and teamwork, these difficult situations can be managed to provide the best possible outcomes for both the mother and her child.

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Pain Control After Cesarean Section—"How Can I Take Care of My Baby if I Just Had Surgery?"

Patrick G. Bakke MD

Pain control after cesarean section (C section) is crucial for the comfort of the new mother. In addition, poor pain control on postoperative day 1 when the mother is mobilizing can hinder her ability to care for her child. The discomfort results from two distinct pathways of nociception: (i) the somatic component derived from the surgical wound, and (ii) a visceral component derived from the ongoing contractions of the uterus. There are also concerns about the development of residual pain after C section. The most commonly used routes for delivering analgesics for postoperative pain control following C section are systemic and neuraxial. Commonly employed medications are opiates, nonsteroidal anti-inflammatory drugs (NSAIDs), and local anesthetics.

Systemic delivery of analgesics traditionally involved intravenous (IV) or intermuscular injection of opiates. It is now clear that the use of the IV route results in more patient satisfaction, overall lowered pain scores, and decreased nursing workload. IV patient-controlled analgesia (IVPCA) results in even higher patient satisfaction scores due to the availability of a more consistent level of analgesia and a greater measure of patient control. Opiate side effects of pruritus, nausea and vomiting, urinary retention, constipation, sedation, and respiratory depression must be monitored and treated accordingly. Although all opiates are present to some degree in breast milk, they have not been shown to be a problem with neonatal sedation. Morphine has the least presence in breast milk. Meperidine, however, is best avoided due to accumulation of its active metabolite in breast milk. It is interesting to note that patients with epidural opiate/local anesthetic infusions had lower pain scores than those with an IVPCA; however, patient satisfaction is still reported to be greater in the IVPCA group.

Neuraxial delivery of opiate analgesics has become more common with the use of spinal or epidural anesthesia for C section. Using neuraxial techniques for the primary anesthetic may help prevent central sensitization with resulting hyperalgesia and allodynia. In fact, residual pain is more prevalent in patients after C section who have had a general anesthetic than those receiving a neuraxial technique. Certain patient factors such as pre-eclampsia, low platelet count, or anticoagulation must be kept in mind when planning a neuraxial technique. In addition, there is a subset of patients who will refuse neuraxial techniques, and their wishes need to be respected. P.681

When given intrathecally, preservative-free morphine can give excellent pain control up to 24 hours. There seems to be little effect on pain scores with doses above 0.1 to 0.2 mg. However, with escalating doses, the side effects of pruritus, nausea and vomiting, urinary retention, and respiratory depression increase. Of these side effects, the most feared is delayed respiratory depression, which can occur 12 to 14 hours after administration. Abouleish et al. studied 856 patients who had spinals for elective C section—0.2 mg of preservative-free morphine was included, and <1% (all of whom were morbidly obese) met the criteria for respiratory depression. Clearly, this route and dose can be used with safety. (*Note:* Most obstetrics units will discharge a **low-dose** "Duramorph mom" to the floor with the caveat that no IV narcotics can be prescribed in the first 12 hours without assessment from the anesthesia service—generally, this is not an issue because most moms can be transitioned directly from Duramorph to oral pain medication.) Fentanyl or sufentanil can also be used for analgesia in the intrathecal space. These drugs have fewer propensities for nausea, vomiting, or itching. They are faster in onset and have a shorter duration of action, likely due to their greater lipid solubility. Respiratory depression, if it is to occur, usually does so within 30 minutes.

Epidural solutions containing both local anesthetics and opiates provide excellent analgesia. The presence of local anesthetics reduces the opiate requirements. However, the solution must be dilute enough not to cause significant motor or sensory weakness to the lower extremities. A commonly employed strategy is to remove the epidural catheter on postoperative day 1 when the patient is expected to ambulate. Plus, bowel function has usually returned by then, allowing oral administration of medications for pain relief.

Although opiates are excellent at treating the somatic portion of pain, they are somewhat inadequate with visceral pain. NSAIDs play their biggest role here. NSAIDs have a well-documented opiate-sparing effect. What is commonly overlooked, however, is the role NSAIDs play centrally by inhibiting production of prostaglandins in the spinal cord. Prostaglandins are pronociceptive in central sensitization. Diclofenac and ketorolac have both been shown to be particularly effective. It must be noted that even though the American Academy of Pediatricians regards ketorolac as safe for use in breastfeeding women, the manufacturer states that its use is contraindicated during labor and delivery or by nursing mothers, and the U.S. Food and Drug Administration has a black box warning because of the possible effects on fetal circulation and uterine contractions.

Last, we should not overlook the role of the surgeon. Local infiltration of the wound has marked immediate postoperative effects on pain reduction. Some advocate the silo elastic "pain balls" hooked to a subdermal catheter for postoperative pain control. P.682

In summary, there are a number of modalities for pain relief following C section. Opiates may be given either systemically or by intrathecal route. Local anesthetics mixed with opiates in the epidural space are very effective. NSAIDs have opiate-sparing effects and help prevent central sensitization that can lead to residual pain. Our surgical colleagues can help by using local injections or infusions in the surgical wound.

TAKE HOME POINTS

- IVPCA results in the highest patient satisfaction scores after C section.
- Epidural solutions with mixed local anesthetics and opiates result in the lowest pain scores after C section.
- Neuraxial techniques for the primary anesthetic may lead to less residual pain after C section.
- Preservativefree morphine given via the intrathecal route is safe up to 0.2 mg and is not more effective for pain relief beyond that dose.
- NSAIDs have an opiate-sparing effect.
- NSAIDs also help prevent central sensitization.
- Surgeons should use local anesthesia in the wound.

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Be Aware of the Consequences of Aortocaval Compression and the Supine Hypotensive Syndrome When Caring for a Pregnant Patient

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Ryan C. Romeo MD

Positioning in the pregnant patient is very important. About 10% of women at term experience the supine hypotensive syndrome caused by complete or near-complete occlusion of the inferior vena cava (IVC) by the gravid uterus. A resultant drop in venous return decreases cardiac output (CO), leading to systemic hypotension for which the cardiovascular system cannot compensate. The nature and severity of symptoms range from unspecific symptoms such as pallor, sweating, nausea, and vomiting, to severe maternal hypotension, loss of consciousness, cardiovascular collapse, and consecutive fetal depression. Inferior vena caval compression can begin to occur in pregnant women as early as 13 weeks' gestation and may be exaggerated by multiple gestations (twins) and obesity.

Complete or near-complete obstruction of the IVC can occur when the parturient is supine. Collateral circulation can allow for some venous return, but the result is a decrease in right atrial filling pressure. This drop in preload leads to a decrease in CO. The lateral decubitus position causes partial caval obstruction, but the right ventricular filling pressure is unaltered due to collateral circulation, and there is little effect on the CO.

Aortic compression also occurs. There is little aortic compression at term in the lateral position. In the lateral tilt position, 40% of patients will have a fall in femoral artery pressure, indicating a compression of the aorta. In the supine position, the fall is even greater and inversely proportional to arterial pressure. Thus, there is enhanced compression of the aorta when the patient is hypotensive.

Objective parameters of fetal well-being deteriorate in the supine position compared to the lateral or tilted position. Neonatal outcome, as accessed by umbilical cord pH and Apgar scores, is also compromised in the supine position. These effects are explained by aortocaval compression and the resulting impairment in uteroplacental blood flow.

CARDIOVASCULAR SYSTEM ADAPTATION TO PREGNANCY

Table 158.1 summarizes the body's cardiovascular adaptation to pregnancy. Labor augments CO. Uterine contractions add 300 mL to the central circulating volume and relieve the degree of vena caval compression. CO, heart rate, and stroke volume all increase substantially shortly after delivery. The P.684
maximum rise in CO is coincident with the autotransfusion of blood from the evacuated uterus. There is a decline in aortocaval compression, and the loss of the placental shunt. The cardiovascular changes and parameters return to prelabor values within an hour after delivery. They gradually return to prepregnant values within 2 to 4 weeks postdelivery.

TABLE 158.1 SUMMARY OF THE BODY'S CARDIOVASCULAR ADAPTATION TO PREGNANCY

Maternal oxygen consumption	Increased
Heart rate	Increased
Cardiac output	Increased
Stroke volume	Increased
Peripheral vascular resistance	Decreased
Mean arterial pressure	Decreased
Central venous pressure	Unchanged
Blood volume	Increased
Plasma volume	Increased
Erythrocyte volume	Increased

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Hemoglobin	Decreased

POSITIONING THE PREGNANT PATIENT

Aortocaval compression and the supine hypotensive syndrome can be minimized by having the parturient lie in the lateral position. For cesarean section or when the lateral position is not practical, left uterine displacement using the lateral tilt position should be employed. Place a wedge under the patient's right hip to elevate the hip 10 to 15 cm and displace the uterus to the left, away from the IVC. This should be done from 20 weeks' gestation to term.

When placing an epidural catheter or performing a spinal anesthetic, the sitting position may be preferred because it has been shown to be associated with less decrease in CO than the maximal lateral flexed position when placing an epidural in laboring women.

Cardiopulmonary resuscitation in the parturient is complicated by vena caval compression. The patient should be placed in the lateral tilt position and an assistant should move the uterus further off the IVC by lifting it with two hands to the left and toward the patient's head. Cesarean section plays an important role in the resuscitation of the mother in cardiopulmonary arrest and is advocated if the resuscitation is not successful within 5 minutes. It is believed that occlusion of the IVC is relieved completely by emptying the uterus, whereas it is only partially relieved by manual uterine displacement or an inclined position.

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DETERMINANTS OF UTERINE BLOOD FLOW

Uterine blood flow (UBF) represents about 10% of the CO at term, which is about 500 to 700 mL/minute. In comparison, the nonpregnant uterus sees about 50 mL/minute. UBF is directly related to perfusion pressure and inversely related to vascular resistance.

$$\text{UBF} = \frac{\text{Uterine arterial} - \text{Uterine venous pressure}}{\text{Uterine vascular resistance}}$$

Thus, UBF decreases when perfusion pressure decreases or when vascular resistance increases.

UBF may decline when the perfusion pressure decreases because of decreased uterine arterial pressure, which occurs during systemic hypotension. Not only will UBF decrease because of the decline in perfusion pressure, but uterine vascular resistance also increases because of increased concentrations of vasopressin and angiotensin II, which are secreted in an attempt to maintain systemic blood pressure.

UBF may decline when perfusion pressure decreases because of increased uterine venous pressure. This typically occurs with vena caval compression, during uterine contraction, and during the Valsalva maneuver that accompanies pushing during the second stage of labor.

UBF may decline because of increased uterine vascular resistance. This may occur secondary to endogenous catecholamines (stress) or exogenous vasoconstrictors such as phenylephrine administration. However during pregnancy, anatomical adaptations result in near maximal vasodilation of the uterine arteries, resulting in a low-resistance pathway for delivery of blood to the placenta. Therefore, adequate uteroplacental blood flow depends on the maintenance of a normal maternal perfusion pressure.

There is little evidence available for supporting autoregulation of blood flow in the placenta, and the circuit mainly depends on maternal blood pressure.

EFFECTS OF ANESTHESIA ON UTERINE BLOOD FLOW

Regional anesthesia has the following effects on UBF. It can increase UBF by causing pain relief, decreased sympathetic activity and decreased maternal hyperventilation. It can decrease UBF by causing hypotension, unintentional intravenous injection of local anesthetic and/or epinephrine, and absorbed local anesthetic (little effect). Adjuvant drugs used in regional anesthesia can have varying effects on UBF, and these effects can be found in more comprehensive texts.

General anesthesia has minimal effects on UBF. The drugs used to induce general anesthesia such as barbiturates and propofol may decrease UBF by causing a degree of hypotension. Usual clinical doses of inhalation agents have little or no effect on UBF. Noxious stimuli secondary to light anesthesia may cause the release of catecholamines and thereby decrease UBF.

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TAKE HOME POINTS

- The supine hypotension syndrome can be life threatening to both the mother and the fetus.
- Aortic compression in the supine position is actually a separate but related phenomenon.
- The detrimental effects to the fetus when the mother is allowed to assume the supine position are well documented.
- Left uterine displacement should be used once the patient is past 20 weeks' gestation, regardless of whether she experiences symptoms in the prone position.
- There is little if any autoregulation of UBF, the main determinant is maternal blood pressure.
- If the mother is undergoing cardiopulmonary resuscitation, she should be placed in the left uterine displacement position and the uterus should be manually lifted off the vena cava.

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Headaches and Hypertension: Management of Pre-Eclampsia in the Obese Patient

Patrick G. Bakke MD

The incidence of hypertensive disorders of pregnancy is 2% to 8%. These disorders can be thought of as a continuum from gestational hypertension to eclampsia. Pre-eclampsia is defined as systolic blood pressure (SBP) ≥140 and diastolic blood pressure (DBP) ≥90, diagnosed after 20 weeks' gestation and accompanied by proteinuria >300 mg/24 hours. Severe pre-eclampsia is defined as SBP ≥160, DBP ≥110, proteinuria >5 g/24 hours. In addition, signs of end-organ dysfunction (oliguria, elevated serum creatinine, pulmonary edema, visual disturbances, headaches, epigastric pain), fetal growth restriction, oligohydramnios, or HELLP syndrome (**H**emolysis, **E**levated **L**iver **E**nzymes, and **L**ow **P**latelets) make the diagnosis of severe pre-eclampsia. Eclampsia is diagnosed by the presence of seizures.

The etiology of pre-eclampsia is poorly understood. Currently, it is believed that initially there is abnormal trophoblastic invasion during placental implantation. Trophoblastic invasion of the decidual and myometrial portions of the spiral arteries results in an increase in diameter of these arteries. In pre-eclampsia, only the decidual portions change; the myometrial segments remain small and exceptionally responsive to vasomotor stimuli. This abnormality explains the drop in placental perfusion and the predisposition to uterine growth retardation. In turn, this leads to the changes that characterize the systemic maternal disease. There is endothelial dysfunction that can cause platelet aggregation, downregulation of circulating anticoagulants, and capillary leaks. In addition, an imbalance between decreased prostacyclin I_2 and increased thromboxane A_2 exists. Prostacyclin I_2 is a potent vasodilator and activator of platelets. Thromboxane A_2 is a potent vasoconstrictor and promoter of platelet aggregation. Endothelial dysfunction may impair nitric oxide and increase endothelin-1. Endothelin-1 is also a potent vasoconstrictor and platelet aggregator. These changes result in generalized vasospasm, reduced intravascular volume, decreased glomerular filtration rate, generalized edema, and a hypercoagulable state.

Obesity, among others, is an independent risk factor for developing pre-eclampsia. When the two entities are combined, anesthetic management can be quite challenging. Current obstetric management involves treating elevated blood pressure with labetalol first, then using oral nifedipine. These patients will likely be receiving magnesium sulfate for seizure prophylaxis. It is crucial to have open and complete communication with the obstetricians regarding these patients. The only definitive treatment is delivery of the fetus. P.688

The anesthetic management of labor should involve the early placement of a continuous lumber epidural catheter. Given the obese body habitus and generalized edema, intravenous line placement may be difficult at best. Next, it is important to check a platelet count and prothrombin time/international normalized ratio prior to proceeding. Early involvement of anesthesia can allow placement before platelet counts fall to an unacceptable level. Most obstetric anesthesiologists are comfortable placing an epidural with platelet counts >80,000. Obesity increases the technical difficulty of placing an epidural catheter. Help should be recruited to ensure optimal positioning. Early placement of an epidural has benefits besides patient comfort. Controlling the pre-eclamptic patient's pain can blunt the hypertensive response to pain, decrease circulating catechols, and improve uteroplacental blood flow. In addition, early epidural catheter placement may avoid a general anesthetic in case of a C section. Fluid bolus must be done with some caution because too much fluid may result in pulmonary edema. If there is presence or suspicion of pulmonary issues, boluses of 250 mL at a time may be wise. Vasoactive medications such as ephedrine or phenylephrine may have a much greater effect than anticipated on the already exceptionally responsive vascular system.

In the event of an operative delivery, the combination of pregnancy, pre-eclampsia, and obesity all conspire against us. The method of choice is still an epidural for C section. If the catheter has been placed early, you are in luck. If not, refer to the previous paragraphs for consideration of placing the epidural. In the past, spinal anesthesia was a relative contraindication in the pre-eclamptic. It was believed that the drop in blood pressure sometimes seen with a spinal would be extreme given the vasoconstricted and volume depleted state of the pre-eclamptic. A body of evidence now exists that shows that a spinal can be used safely. Combined spinal epidural may be a wise choice given the increased operative difficulties obesity may present. Fluid boluses and support with vasopressors should be done cautiously as previously mentioned. SBP should be maintained at no less than 20% of baseline values in order to maintain placental perfusion.

If general anesthesia is required, there are a few extra considerations for the obese pre-eclamptic above and beyond the healthy obstetric patient. Again, communication with the nurses, obstetricians, and patient must be clear. For example, a patient with a terrible airway and extremely low platelets may benefit from a controlled C section instead of an emergent procedure. Next, careful consideration must be given to the overall medical condition of the patient. During the history and physical, extra attention should be paid to the airway, cardiac, pulmonary, abdominal, and neurologic systems. The airway may be even more edematous. Signs of heart failure should be explored. If the patient requires oxygen to maintain oxygen saturation or if there are rales on auscultation, pulmonary edema is likely. Complaints of right upper quadrant pain or a tender enlarged liver on abdominal examination may herald impending Glisson capsule rupture. Headache or visual changes are especially worrisome on neurologic exam. Labs should be recent with complete blood count, metabolic panel, liver function tests, and a coagulation panel at the least. Two large-bore intravenous (IV) catheters should be in place. The medications that the patient is receiving for blood pressure control and seizure prophylaxis must be noted. The anesthesiologist must also check the fetus by way of prenatal records and fetal heart tracing. This is also a time to decide on monitors. A mild pre-eclamptic will likely need no more than American Society for Anesthesiologists' standard monitoring. However, in the case of the severe pre-eclamptic, presence of pulmonary edema, renal insufficiency, or inability to obtain adequate venous access, invasive monitoring in the form of an arterial line, central line, or even pulmonary artery catheter may be necessary. A difficult airway cart should be present given the likelihood of airway difficulties. On induction, the response to direct laryngoscopy needs to be sufficiently blunted to prevent intracranial bleed. Besides the usual induction agent, lidocaine and opiates, it may be necessary to use esmolol, nitroglycerine, or even nitroprusside to control the hypertensive response. Magnesium sulfate should be continued in the perioperative period. When it is time to extubate, a leak test should be performed to ensure that airway edema has not increased with intraoperative fluid administration to a point that compromises airway patency. The obese patient should be awake, following commands, fully reversed, and seated upright prior to extubation. P.689

Postoperatively or postpartum, laboratory values should be checked prior to removing the epidural catheter. The nadir of the platelet count can be 2 to 3 days postpartum, and it may take 5 to 6 days to reach an acceptable level for epidural removal. Pulmonary edema risk is greatest 2 to 3 days postoperatively when fluid is mobilized. Magnesium should be continued for 24 hours because the risk of seizure is greatest in the postpartum period.

TAKE HOME POINTS

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- Clear and early communication among the obstetrician, nursing staff, and anesthesiologist is crucial.
- The airway may be even more challenging in the obese pre-eclamptic.
- Be involved with the IV access—most experienced obstetric anesthesia providers will try for at least one decently running 18g peripheral line with these patients. P.690
- Pay close attention to the platelet count and laboratory values before epidural placement and before epidural catheter removal.
- Place the epidural catheter early.
- A spinal is acceptable for a C section.
- Do not hesitate to place invasive lines.
- Continue the magnesium; seizure risk is greatest postpartum. Despite therapy, patients may become eclamptic. Therapy to treat the acute onset of seizures and airway support should be immediately available. Historically, thiopental was used to good effect—50 mg (or 2 cc) will stop the seizures. Because thiopental is now not always immediately available, propofol 20 to 120 mg can be used for seizure control, with succinylcholine 80 mg used for emergency airway control and intubation.
- Be careful with fluid administration; pulmonary edema is most likely 2 to 3 days postoperatively.
- Patients will likely be sensitive to vasopressors.
- Prevent hyperdynamic responses during direct laryngoscopy.
- Early regional anesthesia for labor can prevent the need for general anesthesia and the potential airway issues.

One final note (and a common folklore saying in the labor and delivery suite): Do NOT let a pre-eclamptic patient “get away from you,” or you will be the one with the headaches and hypertension!

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Acupuncture Has Significant Efficacy for the Treatment of the Pregnant or Laboring Patient

Leena Mathew MD

The perception of acupuncture as a complementary medical modality (as opposed to an *alternative* therapy) has grown a lot in the last ten years, as the integration of complementary and conventional medicine continues to take root in the Western world. This is reflected by the surge of scientific studies and increasing evidence that establishes acupuncture as an accepted modality in Western medicine. Approximately 1 million U.S. consumers use acupuncture annually, primarily seeking relief of pain, and acupuncture is now available as a treatment option in most chronic pain clinics.

At present, the research data on acupuncture are mixed. Most trials on the efficacy of acupuncture are equivocal or contradictory. The randomized controlled trials using acupuncture to treat addictions and tinnitus have been predominantly negative. Multiple reviews of identical conditions such as asthma, depression, and bronchitis use overlapping studies and have conflicting interpretations.

However, notable exceptions are randomized controlled trials of acupuncture for emesis, labor pain, and dental pain, which have been overwhelmingly positive. The World Health Organization mentions acupuncture as a nonpharmacologic method to be used in the obstetric population, especially during labor, and emphasizes the necessity of clinical studies to further validate the role of acupuncture. Theoretically, acupuncture is an ideal adjunct for the obstetrics population because it poses a low risk to the mother and fetus and is relatively free of side effects.

The term *acupuncture* comes from the Latin words *acus* meaning needle and *punctura* meaning puncture. It is an ancient science dating back almost 5,000 years. It relies on the stimulation of well-demarcated points situated along set meridians. It is a concept of medicine, which in simple terms suggests that internal homeostasis depends on the unobstructed flow of *Qi* (chee) through these meridians.

Athorough understanding of surface anatomy is essential to the practice of acupuncture, which involves the placement of fine disposable stainless steel needles at select acupuncture points. There are 365 acupuncture points that lie along 14 meridians, which correspond to organ systems. Acupuncture needles are inserted at these points and stimulated by various techniques (similarly, acupressure involves holding pressure over the acupoint). These needles range from 1/4 in. to several inches in length and a few thousandths to several thousandths of an inch in diameter. One inch to 1.5 in. is the most commonly used length of needle. Longer needles are used in certain specialized therapies. After needle placement, manual twirling of the needle is performed to elicit a characteristic sensation called *De Qi* . Other methods used include electrical stimulation, laser, and moxibustion.

P.692

The mechanism of action of acupuncture is not completely understood, but it is hypothesized that it involves the rhythmic discharge of nerve fibers that can cause the release of endorphins and oxytocin. One popular theory explaining how acupuncture modulates pain is the neurohumoral hypothesis of acupuncture analgesia. The neurohumoral hypothesis, based on more than 100 scientific papers, states that the pain-relieving properties of acupuncture are partly mediated by a cascade of endorphins and monoamines that are activated by stimulating De Qi, a sensation of numbness and fullness. De Qi is associated with the stimulation of A-delta afferents, which set the cascade in motion. It is interesting that different manners of needle manipulation are known to produce different therapeutic effects.

Although the exact scientific mechanisms remain largely unknown, the analgesia produced by electroacupuncture of different frequencies may be mediated by different endogenous opioids. It is suggested that at lowfrequency (2-Hz) electroacupuncture analgesia is mediated by enkephalin, whereas dynorphins play a role at high-frequency (100-Hz) electroacupuncture analgesia. Among the endorphins, enkephalin and beta-endorphin act at mu and delta receptors. The endomorphins act at the mu receptors alone. Dynorphins are purely kappa receptor agonists. Some studies have suggested that analgesia induced by 2-Hz electroacupuncture is mediated by the m receptor and that of 100-Hz electroacupuncture by k opioid receptors, and that this analgesia is reversible by naloxone. With 2-Hz stimulation electroacupuncture, the release of beta-endorphins into the peripheral blood improves pain, modulates stress, and coordinates contraction of the uterus caused by oxytocin. In animal studies, beta-endorphin levels have been observed to rise in the brain tissue of animals after both acupuncture and exercise. Acupuncture may also decrease sympathetic discharge by the inhibitory effects that the hypothalamic beta-endorphinergic system has on the vasomotor center.

Acupuncture-mediated analgesia is further modulated through neurohumoral pathways. The stimulation of the acupuncture point by needling selectively activates myelinated nerve fibers, and there is neuronal activation at the level of dorsal horn, nucleus raphe magnus in the brainstem, hypothalamus, and thalamus. Subsequently, there is inhibition of the small unmyelinated pain-carrying fibers. The endorphins also allow for a sense of well-being and relaxation.

P.693

In the obstetric population, acupuncture may be used effectively in the treatment of first-trimester nausea and vomiting or hyperemesis, carpal tunnel syndrome, headaches, migraine, backache, sciatica, and other chronic pain syndromes. Acupuncture has also been used to aid in the correction of malpresentation, induction of labor, and pain relief in labor. In the postpartum period, acupuncture has been used to treat postpartum depression and pain from breast engorgement. To date, the evidence about acupuncture for labor analgesia is quite encouraging, and the data available from scientific studies suggests that the analgesic effect is superior to placebo. However, the data are limited, and elegant studies are few.

BRIEF OVERVIEW OF THE COMMON ACUPUNCTURE POINTS USED IN OBSTETRICS

Nausea and Vomiting.

Hyperemesis occurs in 1% to 2% of pregnancies. Acupressure or acupuncture done at P6 point has been shown to be efficacious in the treatment of nausea and vomiting. The P6 point/Neiguan point lies on the pericardial (P) meridian. It is located between the tendons of the flexor carpi radialis and the palmaris longus 2 cun (pronounced as “soon”) proximal to the distal wrist crease. One cun is the interphalangeal distance on the third finger (Fig. 113.2).

Acupressure may be self-administered by using a finger to hold pressure over this point for 15 to 20 minutes. Acupressure is not as efficacious as acupuncture. With placement of the acupuncture needle at this point, the patient may report a sense of heaviness or warmth in the hand, which is called De Qi. If the patient reports continuous sharp pain, the position of the needle must be readjusted. Arandomized blind crossover comparison of P6

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acupuncture versus placebo showed a significant decrease in the resolution of nausea and decreased episodes of emesis.

Labor Analgesia.

Although acupuncture may not be a complete analgesic, it may act to provide some analgesia and relaxation. One study comparing acupuncture with conventional care showed that women receiving acupuncture requested significantly fewer pain-relieving interventions than the women not receiving acupuncture. Among the women who received acupuncture, 86% said they would use it again for childbirth, although the results concerning the analgesic effects of acupuncture were not always successful. There were no differences in labor outcomes between the two groups, including the duration of labor, cesarean section rates, and measures of infant wellness.

Acupuncture during labor provides relaxation. It has been suggested that if the parturient is more relaxed, she is likely to have more control and consequently cope better with the pain. Some studies suggest decreased requirement for epidural analgesia in patients treated with acupuncture; however, other studies suggest that acupuncture did not decrease the need for additional analgesics during labor. Although many points are used, there are two classic points that may be beneficial in labor: P.694

- **LI 4 (Hegu):** This point is located at the base of the anatomical snuff box—on the dorsum of the hand, between the first and second metacarpals, approximately in the middle of the second metacarpal bone on the radial side (Fig. 160.1). The needle is inserted to a depth of 0.5 cm.
- **SP 6:** This point is located on the spleen meridian and is an empirical point located 3 cun above the medial malleolus (Fig. 160.2). It is especially useful in difficult labor. It may also aid in cervical dilatation. It is needled with a perpendicular or oblique proximal insertion to a depth of 1 cm.

Another point that is quite useful is the DU 20 (Baihui). It is located at the top of the scalp equidistant from both pinnae (Fig. 160.3). It is needled with perpendicular insertion 0.5 to 1 cm using a 0.25-mm diameter needle. It allows for intense relaxation and mild sedation. It is never subjected to electroacupuncture stimulation.

For labor analgesia, LI 4 and SP 6 are stimulated bilaterally. Ideally, the treatment should be started at the beginning of the active phase in the first stage of labor. Fine 0.25-mm diameter stainless steel filiform needles are inserted into the acupoints to a depth of 10 mm at LI 4 and 15 mm at SP 6. Once the patient reports a De Qi sensation, which is basically a sore and distending sensation, the needles are retained for 2 minutes. At this time, the free end of the needles can be connected to the electroacupuncture apparatus and stimulation set at a frequency of 2 to 100 Hz and electric current of 14 to 30 mA. Gradually, the stimulation strength can be increased. The needles can be removed after 15 to 30 minutes. This can be repeated as needed during the course of labor. It is recommended that when 7 to 8 cm of cervical dilatation is achieved, the procedure can be repeated. P.695

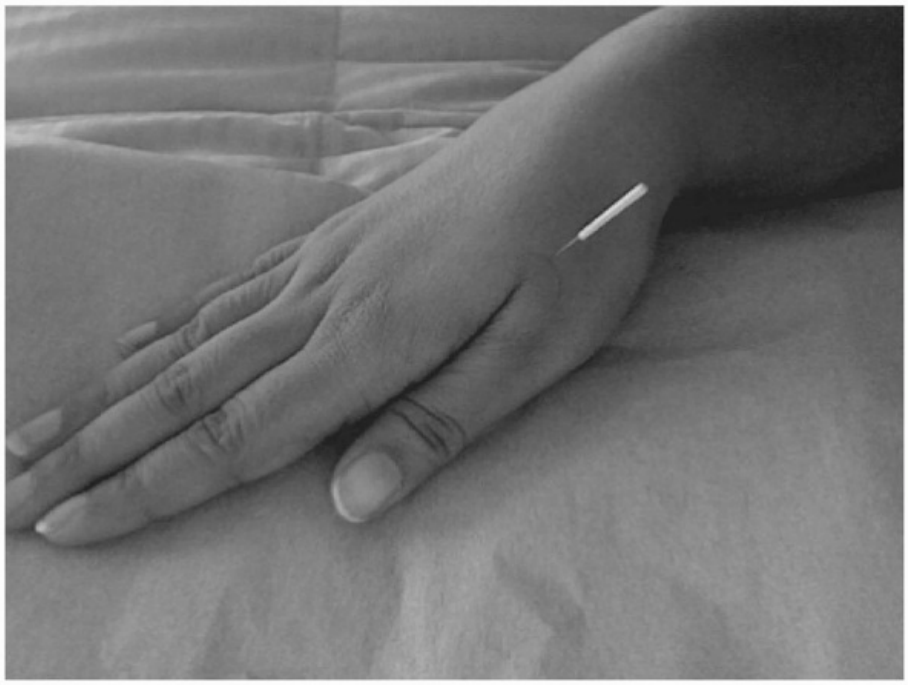


FIGURE 160.1. Acupuncture at the LI 4 point.

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FIGURE 160.2. Marking of the SP 6 point.



FIGURE 160.3. Acupuncture at the DU 20 point.

Chronic Pain During Pregnancy.

Acupuncture can also be used to relieve chronic pain syndromes such as low back and neck pain, carpal tunnel syndrome, headaches, migraines, and sciatica. Acupuncture decreases pain and has been shown to diminish disability in low back pain better than physiotherapy during pregnancy. The insertion of needles at points LI 4 and DU 20, along with other points as prescribed as per the patient's needs, is of benefit. For carpal tunnel syndrome, bilateral needles are placed in the SP 6 and LI 4 points, and either left in place or stimulated with low-frequency stimulation for 30 minutes.

Headaches and migraines can also be treated similarly with acupuncture treatment. Adequate acupuncture can avoid the risk of side effects, but more important reduced the intensity and frequency of muscle pain, the degree of headaches, and decreased tenderness over trigger points.

POSTPARTUM DEPRESSION

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Comparison of the current randomized controlled trials suggests some evidence for the utility of acupuncture in depression. Acupuncture modalities were as effective as antidepressants employed for treatment of depression in the limited studies available for comparison. However, placebo acupuncture treatment was often no different from intended verum acupuncture. Efforts are now being made to standardize complementary approaches to treat depression, and further research is warranted.

TAKE HOME POINTS

- Acupuncture is an increasingly common therapeutic modality in Western medicine.
- The efficacy of acupuncture for the treatment of some disorders has not been clearly established; however, the results of randomized controlled trials for the treatment of obstetric patients have been overwhelmingly positive.
- Acupuncture is most likely mediated by a combination of endogenous opioids and neurohumoral pathways.
- In the obstetric population, acupuncture may be used effectively in the treatment of first-trimester nausea and vomiting or hyperemesis, carpal tunnel syndrome, headaches, migraine, backache, sciatica, and other chronic pain syndromes.
- Studies (to date) have not shown improvement in several key obstetric outcomes, but parturients overwhelmingly report that they would request it for a subsequent labor.

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Always Be Prepared for Emergent Delivery as a Consequence of External Cephalic Version

Angela M. Pennell MD

Breech presentation affects 3% to 4% of term pregnancies. Because a fetus is more likely to be safely delivered vaginally from the vertex position, external cephalic version is a technique used by obstetricians to change the presentation of the fetus from breech to vertex. The obstetrician is balancing the risks of the version (umbilical cord compression, and fetal and/or maternal hemorrhage leading to urgent or emergent delivery) against the risks of a potentially difficult breech vaginal delivery (leading to urgent or emergent delivery). The procedure is typically scheduled after 38 weeks' gestational to decrease the possibility of the fetus reverting back to a breech position and is sometimes performed with the adjunctive use of tocolytics.

Overall, external cephalic version is successful in 35% to 86% of nonlaboring, term parturients. Maternal and fetal factors that increase the chance of successful external cephalic version include that the patient is not obese, the cervix is not fully dilated, amniotic membranes are intact, the fetus is in a frank breech or transverse lie, there is normal amniotic fluid volume, the presenting part of the fetus has not entered the pelvis, and there is posterior positioning of the fetal back. Both obstetric and anesthesia staff also generally acknowledge that the success of external cephalic version may depend to a considerable degree on the skill and experience level of the obstetrician combined with the level of pain tolerated by the patient.

The question of whether the use of neuraxial anesthesia (spinal or epidural) facilitates successful external version and improves outcomes is an interesting and unanswered one. There is also the dilemma of what drug to use and how much to give. Most anesthesiologists tend to use “labor doses” for version, and because labor epidurals are quite routine in current anesthetic practice, the matter seems a simple one. When giving anesthesia for external version, however, for every pro there is a con. Patients are sometimes quite anxious about the procedure and fearful of the pain. Assuring the patient that she will have little, if any, pain, and then providing her with a painfree experience, can help the obstetrician perform the version with fewer maneuvers by providing a relaxed and reassured patient. However, removing pain as a limiting factor in the procedure may also *increase* the potential for a greater number of maneuvers and more aggressive attempts.

This may in turn increase the potential for significant complications, such as placental abruption. This is equally true if the patient is unable to feel pain from a resulting abruption or uterine rupture *after* the trial of external version is complete. Also, the risks of neuraxial anesthesia, including hypotension, infection, paresthesias, bleeding/epidural hematoma, subdural puncture headache, and, most important, fetal distress, are incurred by the patient in addition to the risks of the version. If the attempted version does result in a call for urgent or emergent delivery, it is advantageous for the patient, anesthesiologist, and obstetrician to have the block already in place and working, unless, of course, it is determined that the block contributed significantly to the inability to monitor the maternal vital signs or to the fetal difficulties. It is incumbent on the anesthesia provider to document **before the version is attempted** that the block is stable and the mother and fetus are at baseline.

P.699

Currently, there is not a plethora of data available on the safety of neuraxial anesthesia for external version. There have been several trials and retrospective studies on the success rate of external cephalic version with adjuvant neuraxial techniques. According to a search of the Cochrane Pregnancy and Childbirth Group trials register, three randomized trials were analyzed with patients receiving spinal or epidural anesthesia versus no analgesia. In summary, two of the trials revealed a decrease in external cephalic version failure, noncephalic births, and cesarean section, with one trial showing no benefit. However, the differences among the conflicting trials were not statistically significant. The retrospective reviews of neuraxial anesthesia for external cephalic version have shown overall success but suggest further studies to evaluate maternal and fetal safety related to its use. Finally, the American College of Obstetricians and Gynecologists indicates that there is not enough consistent evidence to recommend either spinal or epidural anesthesia for external cephalic version.

Of course, any anesthesia professional providing neuraxial anesthesia for external cephalic version must be prepared for emergent cesarean section (it is wise to be prepared for a call for emergent delivery even if the anesthesia team has *not* provided a block for the procedure). Prior preparations in the operating room should include a recent anesthesia machine check; intravenous fluids; emergency syringes of succinylcholine, atropine, lidocaine, epinephrine, ephedrine, and/or phenylephrine; uterotonics, including oxytocin and methergine; spinal anesthesia kits; and intubating equipment. For a patient who has an existing epidural, use a bolus of 15 to 20 mL of 3% chloroprocaine or 15 to 20 mL of 2% lidocaine with 1:200,000 epinephrine plus or minus fentanyl. An urgent cesarean section or a poor candidate for general anesthesia may permit time for the placement of spinal anesthesia using 12 mg of 0.75% bupivacaine. An emergent surgical intervention or

failure of neuraxial analgesia will require general anesthesia with a rapid sequence induction using thiopental 3 to 4 mg/kg or ketamine 0.5 to 1 mg/kg plus succinylcholine 1 to 1.5 mg/kg to facilitate intubation. Maintenance of anesthesia can be accomplished with 50% nitrous oxide plus 0.75 MAC volatile anesthetic prior to umbilical cord clamping and 70% nitrous oxide plus <0.50 MAC volatile anesthetic post cord clamping with the addition of morphine or midazolam as indicated.

P.700

TAKE HOME POINTS

- External cephalic version is typically performed in near term patients and has a highly variable success rate.
- Be aware when a version is imminent or underway on the labor unit—this is not a good time to go down to the lobby to pick up the food.
- Many factors influence the success of the attempted version, including skill of the obstetrician.
- There are significant pros and cons to providing analgesia for the version—anxiety and pain may hinder the version but also act as a harbinger of impending trouble.
- Anesthesia providers will typically place peripheral access, even if no analgesia is requested.

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Obstetric Anesthesia/analgesia *can* Work in a Small Hospital: the Key Principles are Commitment, Flexibility, and Planning

James S. Hicks MD, MM

The ability to offer obstetric (OB) conduction anesthesia to laboring patients in a small hospital where 24-hour in-house anesthesia coverage is not available or feasible is both a challenge and an opportunity to provide excellent service both to patients and obstetric colleagues. To be successful, interposing obstetric conduction anesthesia services within a surgical schedule and managing the same service after usual operating room hours require careful planning, interdisciplinary cooperation, and ongoing continuous quality improvement.

Among the challenges that must be addressed to provide satisfactory small hospital OB services are the following:

- Recognize and minimize the potential delay in the elective surgical schedule required to perform epidurals
- Recognize and minimize the delay in responding to requests for epidural anesthesia due to the unavailability or on-call status of the anesthesia provider
- Manage the potential inability to provide timely emergency cesarean section anesthesia
- Recognize the need for obstetric nurses to assist with management of epidural infusions and provide them with appropriate education and experience
- Meet regulatory requirements

Surmounting these obstacles requires a commitment on the part of the anesthesiology service that obstetric anesthesia is no less of a priority than surgical anesthesia. This does not imply that obstetric anesthesia will take priority on the surgical schedule, but simply that the efforts to meet the needs of obstetric patients are genuine, compassionate, and equivalent to our efforts to provide excellent surgical anesthesia.

Successful rural OB service is achievable. The American Society of Anesthesiologists' (ASA) Consultation Program has provided hospitals with the expertise of board-certified anesthesiologists who are specially trained in assessing anesthesia practices and in providing recommendations for improvement. The program also provides ASA with a multiple-exposure snapshot of many anesthesia practices, many of which involve rural anesthesia coverage. The following recommendations are the result of experiences drawn from observations gained from this program throughout the United States: P.702

1. Convene a “council of stakeholders” composed of anesthesia providers, obstetricians (and, if applicable, family practitioner and certified nursemidwife obstetric providers), surgeons, emergency physicians, obstetric nurses, pharmacy, and hospital administration. Obtain consensus from this group that labor epidural analgesia for all patients desiring such is a worthwhile and desirable goal. To gain this consensus, council members must acknowledge that the needs of the patients may require customization of policies that would, if otherwise applied, preclude the ability to have labor analgesia coverage. For example, pharmacists need to acknowledge the need to maintain adequate supplies of premixed epidural solutions, even if this results in occasional outdates of solutions. Obstetric nurses must be willing to titrate epidural anesthesia infusions (given clear parameters and orders) in the face of a national organization's recommendations to the contrary. Surgeons must acknowledge the occasional delay between cases for the anesthesia provider to place an epidural catheter. None of these roadblocks has proven to be an obstruction to the small hospital and anesthesia service that are determined to offer comprehensive OB anesthesia, however.
2. Meet as an anesthesia service and adopt standardized processes for epidural and cesarean section techniques and procedures so anesthesia providers can easily manage blocks, surgical anesthetics, and postoperative patients of colleagues. Ideally, this should include standardization of
    - Labor epidural agent mixture
    - Order sheet, anesthesia record (if different from the surgical record), billing forms and practices
    - Cesarean section techniques and preferences (opioid mixtures and local anesthetic mixtures for both elective cesarean section spinals and labor epidurals used for cesarean section)
3. Perform a task analysis of all tasks necessary to accomplish a labor epidural (e.g., examining the chart; taking history and performing physical examination; obtaining pump, infusion bag, gloves, epidural tray and anesthesia cart, placing, test dosing, and securing catheter and beginning infusion).
4. Prepare an educational course for obstetric nursing staff that can be recorded for future additions to the staff. This should include anatomy, physiology, pharmacology, and practical work with the technique of epidural placement, dosage management, and management of known complications. Request that nursing staff complete all possible steps in the task analysis (step 3) that can be safely accomplished by them prior to your arrival. This step is vital to your ability to place an epidural quickly during a break in a busy surgical schedule. P.703
5. Certify the competence of nurses successfully completing the labor analgesic course after observing them manage a given number of epidurals. (This will give them increased assurance in managing epidural infusions in the face of contradictory information from national organization recommendations.)
6. Inform surgical colleagues of your intent to provide labor epidural services with the minimal interruption of the surgical schedule and request their cooperation.
7. Plan in advance for labor epidurals whenever possible. This means contacting the labor floor early in the morning for patients who will likely need an epidural during the morning, as well as inserting and testing a catheter before the beginning of the surgical schedule. As soon as the patient then

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requires pain relief, the infusion may begin without a bolus dose at the usual rate. This results in an effective dose being achieved in approximately the same time frame as would have occurred had the usual sequence of notify-attend-insert-test-bolus taken place.

8. An anesthesia provider's rest *is* important when he or she is on call for the operating room and/or is expected to provide anesthesia the following day, as is often the case at smaller hospitals. By maintaining frequent communication with obstetric nursing staff, checking the labor floor before departing for the day or after an on-call surgical case, and encouraging nurses to call sooner rather than later for patients who require epidurals, return trips to the hospital or loss of sleep can be minimized.
9. Obtain assurance from emergency department physicians that they will respond to the extremely rare instance of adverse reaction to an established epidural analgesic, and educate them on the possible complications. The use of a test dose coupled with an extremely low local anesthetic concentration (the author's department uses 0.055% bupivacaine with 1 mcg of sufentanil and 1.7 mcg of epinephrine per mL) make the possibility of such complications almost nonexistent.

One final note: The question is often asked, “Do I have to remain in the hospital during the entire course of a labor epidural?” According to ASA, the answer appears in the introduction to the *Guidelines for Regional Anesthesia in Obstetrics* : “Because the availability of anesthesia resources may vary, members are responsible for interpreting and establishing the guidelines for their own institutions and practices….” Thus, ASA acknowledges that local convention based on capability and personnel are determining factors in obstetric anesthesia services and allows hospitals to meet their standards in ways that are best for patients.

TAKE HOME POINTS

- The first step in providing OB services in a small or rural hospital is recognizing the challenges.
- There must be commitment to the idea that obstetric analgesia/anesthesia is no less important than surgical anesthesia.
- The consensus must be across a wide range of medical and administrative staff.
- Standardization of anesthetic techniques for labor epidurals and cesarean sections is of paramount importance.
- Labor nurses must willingly accept training in certain aspects of the setup and management of labor epidurals.
- Complications due to labor epidurals can be minimized by standardized, careful test dosing of catheters and very low-dose epidural infusions.

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Editors: Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.
Title: *Avoiding Common Anesthesia Errors, 1st Edition*

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Remember that Anesthesia for the Pregnant Patient Having Nonobstetric Surgery Is Not Limited to any Particular Agents or Techniques

L. Michele Noles MD

The primary goals in the anesthetic management of patients undergoing nonobstetric surgery during pregnancy (NOSP) are to ensure maternal safety, avoid intrauterine fetal hypoxia and acidosis, know the implications of fetal exposure to anesthetic agents, and avoid preterm labor (PTL). Also, it is fairly common for these patients to experience severe anxiety over possible injurious effects to their pregnancies from both anesthesia and surgery—the anesthesia provider must maintain a working knowledge of the pertinent issues and be able to maintain a calm and reassuring demeanor.

NOSP is fairly common, involving approximately 75,000 patients per year or 1% to 2% of pregnant women, and has specific implications for anesthetic management based on the physiologic changes in the pregnant woman and the presence of a live fetus. The most common nonobstetric surgeries performed on pregnant women include appendectomy, cholecystectomy or biliary tract procedures, breast surgeries, ovarian surgeries, procedures related to carrying a pregnancy to term (i.e., cervical cerclage), and the range of trauma-related procedures. Emergent surgical procedures should proceed as the mother's medical condition warrants. Elective surgery should be postponed until 6 weeks postpartum when the physiological changes of pregnancy have resolved and fetal exposure can be avoided. It is advantageous to proceed with necessary surgery during the second trimester if possible, when organogenesis has been completed and the higher myometrial irritability of the third trimester has not yet begun. The American College of Obstetricians and Gynecologists (ACOG) recommends an obstetrics consultation prior to surgery on any pregnant patient undergoing nonobstetric surgery.

ANESTHETIC IMPLICATIONS OF MATERNAL PHYSIOLOGICAL CHANGES DURING PREGNANCY

Pregnant women have a decreased respiratory reserve due to their increased oxygen consumption (15% to 20%) coupled with decreased functional residual capacity (15% to 20%). Thus, they develop hypoxemia and hypercapnia more rapidly than a nonpregnant patient during hypoventilation or apnea. Due to laryngeal edema, engorged and friable oronasopharyngeal

mucosa, weight gain affecting the tissues of the neck, and increased breast size, they also have an increased likelihood of difficult airway management and intubation. P.706

Pregnant women are at increased risk of regurgitation of gastric contents and aspiration pneumonitis due to impaired lower esophageal sphincter tone. Both hormonal and mechanical factors contribute as the pregnancy progresses. There are conflicting data regarding whether pregnant women have slower gastric emptying, increased gastric volume, and/or decreased pH of gastric contents. However, a pregnant patient should be considered at significant risk for aspiration after 18 weeks' gestation or earlier if symptoms of gastroesophageal reflux exist.

An airway plan for a pregnant patient should include aspiration prophylaxis premedication with at least a nonparticulate antacid (Bicitra); addition of an H2 blocker and/or metoclopramide is at the anesthesiologist's discretion. Use supplemental oxygen during sedation, and for general anesthetics, plan a full 5-minute preoxygenation, a classic rapid sequence induction, and oral intubation with an endotracheal tube that is one size smaller than usual. If difficult intubation is anticipated, opt for awake oral fiber-optic intubation.

During pregnancy, cardiovascular changes include an increased cardiac output, dilutional anemia, decreased vascular responsiveness, an increased risk of thromboembolism, and, after 20 weeks' gestation, a risk of aortocaval compression by the gravid uterus leading to supine hypotension syndrome. Even normal physiological compensation for aortocaval compression can be blunted by general and regional anesthetics that interfere with sympathetic nervous system responses to hypotension. The result is a profound decrease in venous return and cardiac output, leading to impressive maternal hypotension and significantly decreased placental perfusion. Uterine blood vessels are maximally dilated at term, leaving no reserve for vascular autoregulation with perfusion pressure changes. Positioning with left uterine displacement by placing a wedge under the right hip, adequate hydration, and judicious use of pressors can help maintain maternal normotension. Ephedrine has long been the initial pharmacologic agent of choice in hypotensive pregnant women. To minimize risk of thromboembolism, thromboprophylaxis including preinduction placement and activation of pneumatic compression stockings is recommended.

The central nervous system changes in the pregnant patient include a 30% decrease in MAC of volatile anesthetics even in early pregnancy. An engorged epidural venous plexus leads to a one-third decrease in the local anesthetic volume needed for neuraxial anesthetics and an increased risk of inadvertent intravascular injection.

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OPTIMIZATION OF THE FETAL ENVIRONMENT

Optimization of the fetal environment follows from preserving uterine blood flow and fetal oxygen delivery, which are directly dependent on maintaining maternal oxygenation, normocapnia, and normotension. Maternal hyperventilation and overly aggressive positive-pressure ventilation lead to uterine artery constriction and decreased venous return, respectively, thereby causing decreased uterine blood flow. Faced with hypoxia, the fetus is unable to significantly increase its oxygen extraction because it is already near maximum.

The extent of fetal and uterine activity monitoring should be decided on with a consultant obstetrician. Personnel trained in the interpretation of fetal heart rate (FHR), and uterine activity tracings should be involved perioperatively. In general, fetal monitoring in pregnancies less than 24 weeks should include, at minimum, documentation of presence of FHR pre- and postoperatively with or without uterine activity monitoring. After 24 weeks, continuous perioperative FHR and uterine activity monitoring is ideal. Loss of beat-to-beat variability under general anesthesia is typical, but persistent fetal bradycardia is almost always an indication of fetal distress. The anesthesiologist may benefit from this indirect measure of fetal well-being to respond with changes in maternal oxygenation, blood pressure, fluid load, positioning, and/or recommendations for altering the placement of surgical retraction instruments.

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TERATOGENICITY AND IMPLICATIONS OF FETAL EXPOSURE TO ANESTHETIC AGENTS

Although maternal hypoxia, hypotension and acidosis pose a greater risk to the fetus, considerable concern exists regarding the potential harmful effects of anesthetic agents on the fetus. There is wide consensus that no anesthetic agent in common use, including induction agents, volatile agents, anxiolytics, muscle relaxants, or opioids, appears to be teratogenic or safer than other agents. The known association between NOSP and an increased risk of fetal loss, growth restriction, and an increased frequency of low-birth-weight neonates is generally attributable to the patient's underlying medical condition, site of surgery, or surgical procedure. No drug or anesthetic technique has been shown to impact the onset of preterm labor (PTL). Some anesthesiologists prefer to use “tried and true” drugs such as morphine because patients may be somewhat reassured by the fact that a drug has been used with hundreds of thousands of pregnant women over a period of decades without incremental ill effect.

Numerous studies have failed to show the association once believed to exist between benzodiazepines, specifically diazepam, and cleft lip and palate abnormalities. Nitrous oxide (N_2O) is known to interfere with methionine synthase activity and DNA synthesis. These known cellular effects have not been shown to lead to adverse effects in human pregnancy. However, a cautious approach advocated by some experts in the field includes limiting N_2O to 50% or less and limiting its use in lengthy operations. Volatile agents may provide a margin of safety from the point of view of protection from PTL due to their depressant effect on myometrial contractility. P.708

Neuromuscular blocking agents do not cross the placenta in clinically significant amounts and thus fetal exposure is minimal. Maternal plasma cholinesterase concentration is decreased; however, the impact on succinylcholine use is usually clinically negligible.

REGIONAL ANESTHETIC TECHNIQUES

Many anesthesiologists prefer to use regional techniques whenever possible during NOSP. Although evidence is lacking to support that this approach is safer for the mother and baby, there certainly is strong theoretical reasoning to support this, particularly for extremity surgeries for which regional techniques such as subarachnoid blocks, epidural blocks, and peripheral nerve blocks can provide complete surgical anesthesia. It is important to remember that pregnant patients are more sensitive to local anesthetics, so techniques should be employed that reduce the total volume of local anesthetic solution required for anesthesia. In our practice, we use spinal and epidural anesthesia in lower extremity surgery, and brachial plexus blocks in upper extremity surgery, in pregnant patients agreeable to regional anesthesia. We also consider the use of the Bier block when appropriate. Although there are no published studies on peripheral nerve blocks in pregnant patients, we find that reducing the local anesthetic concentration by 20% to 30% while maintaining the same volume as a nonpregnant patient works to give effective anesthesia and reduces local anesthetic toxicity. Similarly, we reduce the amount of local anesthetic used for spinal anesthesia—7.5 mg of hyperbaric bupivacaine generally provides an adequate level for lower extremity surgery.

LAPAROSCOPIC SURGERY

Laparoscopic surgery has become an acceptable safe alternative to open procedures in the pregnant patient. The physiological changes of pneumoperitoneum are magnified in the pregnant patient, thus pneumoperitoneum pressures should be minimized (8-12 mm Hg) not to exceed 15 mm Hg. Fetal $paCO_2$ correlates directly with maternal $paCO_2$ such that maternal hypercapnia results in fetal acidosis and potential myocardial depression and hypotension. The resultant need for hyperventilation and increased ventilatory pressures can lead to decreased venous return, cardiac output, and ureteroplacental perfusion.

TAKE HOME POINTS P.709

- Nonobstetric surgery for the pregnant patient is actually fairly common.
- Obtain an obstetrics consult before initiating the anesthetic if clinical urgency permits; if not, obtain it as soon as the mother's condition permits.
- Remember that the status of the fetus is optimized by optimizing the status of the mother—this may involve a light sedative to control hyperventilation in an anxious patient having elective surgery all the way to an arterial blood gas to direct resuscitation if the mother has sustained trauma.
- Be especially careful and conservative with airway management!
- Remember that fetal outcome depends much more on the condition of the mother and the nature of the surgical case than the type of anesthesia or the specific anesthetic agents.

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Editors: Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.
Title: *Avoiding Common Anesthesia Errors, 1st Edition*

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Do Not Guess at the Equivalent Dose When Determining An Opioid Conversion

Amit Sharma MD

Opioids are robust analgesic medications that have played a core role in the management of acute and chronic pain for decades. The term *opioid* is used for drugs that are derived from the opium plant *Papaver somniferum* . The term *narcotic* is derived from the Greek word “narcosis” (meaning stupor), which, in legal context, includes opium derivatives, their semisynthetic substitutes, and certain stimulants such as cocaine. Thus, all opioid medications are narcotics, but all narcotics are not opioids. Anesthesiologists constantly deal with opioid management for their patients in the perioperative setting. This frequently involves changing one opioid to another or changing the delivery route of a given opioid. The complex pharmacology of these drugs, coupled with significant interpersonal variation seen clinically with these medications, often makes this job cumbersome. Mastering the art of these challenging conversions requires understanding some of the basic pharmacology of these drugs.

Opioid medications are delivered by all possible routes: topical, transdermal, transmucosal, oral, rectal, and parenteral (subcutaneous, intramuscular, or intravenous [IV]). They bind to *Mu, Delta,* and *Kappa* types of opioid receptors in central and peripheral nervous system to cause analgesia or certain untoward side effects. Based on their affinity to *Mu*-receptors, there is a considerable divergence among these medications. Fentanyl, for instance, is almost 15 times as potent as hydrocodone, which in turn is 4 to 5 times stronger than morphine for its analgesic properties. Moreover, there is a significant degree of interpersonal variability for any given opioid, depending on the type of pain, psychological factors, therapeutic drug interactions with other medications, age (elderly are considered to be more sensitive), certain pathophysiological states (increased sensitivity with central nervous system comorbidity), hepatic and renal dysfunction, and even genetic variations. Thus, 2 to 4 mg of IV morphine may not provide adequate pain control in a young patient after shoulder arthroscopy but may produce sedation and respiratory depression in an elderly man with arthritis pain. A brief outline of pharmacokinetic properties of commonly used opioids is given in Table 164.1.

To resolve some of these complex issues, Houde et al. and Bruera et al. performed single-dose relative potency studies on opioid medications. Based on these relevant studies, an equianalgesic dose table (Table 164.2) has subsequently been formulated that describes relative potencies between these diverse opioids for both oral and parenteral routes of administration. The term *equianalgesic* is used for two doses with comparable pharmacologic analgesic effects, either of different drugs or of the same drug but with different routes of delivery. Thus, the first two columns of Table 164.2 list the oral and parenteral doses of opioids that are equivalent to 10 mg of parenteral morphine. These values essentially account for pharmacodynamic and pharmacokinetic differences among these drugs. Table 164.2 is a stepping stone in solving complicated issues related to opioid management.

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TABLE 164.1 OPIOID ANALGESIC GUIDE

	ORAL (MG)	PARENTERAL (MG)	DURATION (H)	PEAK EFFECT (H)	$T_{½}$ (H)
MSO4	30	10	3-6 (O)	1-2 (O)	1.5-2
			3-4 (P)	0.5-1 (P)	
Hydromorphone	7.5	1.5	3-6 (O)	1-2 (O)	2-3
			3-4 (P)	0.5-1 (P)	
Sustained release oxycodone	20	—	8-12	3-4	4-6
Oxycodone	30	—	3-6	1-2	2-3
Hydrocodone	30	—	4-8	1-2	3.5-4.5
Methadone	10[a]	5[a]	4-6	1-2	15-30
	2-4[b]	2-4[b]			
Levorphanol	4[a]	2[a]	6-8	1-2 (O)	12-16
	1[b]	1[b]		0.5-1 (P)	

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Fentanyl	—	0.1	1-2	<10 min	1.5-6
Oxymorphone	15	1	4-6 (O)	1.5-3 (O)	NA
			3-4 (P)	0.5-1 (P)	
Codeine	200	130	4-6		3
Meperidine	300	75	2-4		3-4

$T_{½}$, elimination half-life; O, oral; P, parenteral; NA, not applicable.

[a] For acute pain management purposes.

[b] For chronic pain management purposes.

OPIOID CONVERSIONS IN PERIOPERATIVE SETTING

Opioids are the analgesic agents of choice in intra- and postoperative setting. Patients are often given these drugs via IV route using a microprocessor-controlled infusion pump (patient-controlled analgesia or PCA). The patient can activate a button (demand) to deliver a specified amount of drug (demand dose) into their IV line. Once the microprocessor is activated, it is programmed to go into dormant (or sleep) mode for a specified time during which no further demand doses are delivered (lockout interval). Physicians can also program a maximum 1-hour cumulative dose that serves as an additional line of safety. Standard PCA guidelines for opioid-naive patients are described in Table 164.3. Furthermore, a continuous infusion mode can be added to this program to deliver a specified dose on a baseline (basal infusion). Although basal infusion is avoided in opioid-naive patients, they are often a helpful tool in managing acute-on-chronic or cancer-related pain issues.

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TABLE 164.2 PHARMACOKINETICS OF COMMONLY USED OPIOIDS

MEDICATION	ABSORPTION	DISTRIBUTION	METABOLISM	ELIMINATION
Morphine	40% GI tract[a] T_{max} = 30 min	25%-30% reversibly PPB	Liver: <5% demethylation >95% glucuronidation to M3G 50% (inactive) and M6G 15% (active but does not cross BBB)	Renal—Mainly as M3G/M6G and 10% unchanged Feces—7%-10% $T_{½}$ = varies
Hydromorphone	24% GI tract[a] T_{max} = 30-60 min	8%-19% PPB	Liver: Glucuronidation >95% to H3G <5% to 6-hydroxy reduced metabolites	Renal—Mostly as inactive metabolites $T_{½}$ = 2.6 h
Oxycodone	60%-87% GI tract[a] T_{max} = 1.6-3.2 h[b]	45% PPB	Liver: Noroxycodone (weak analgesic) Oxymorphone (analgesic) via CYP450 2D6	Renal: 50% Conjugated oxycodone 19% Free oxycodone 14% Conjugated oxymorphone $T_{½}$ = 3.2-4.5 h[b]
Fentanyl transmucosal	25% buccal mucosa (rapid) 25% GI tract[a] T_{max} = 20-40 min	Highly lipophilic PPB = 80%-85% α-1 AG>Alb>Lipo	Liver and intestinal mucosa by CYP450 3A4 isoform to norfentanyl (inactive)	Renal (>90% inactive metabolites, 7% unchanged) Feces (1% unchanged) $T_{½}$ = 40-50 min
Fentanyl transdermal	92% skin 13%-21% unbound fraction in plasma are finally achieved T_{max} = 12-24 h	Highly lipophilic PPB = 80%-85% α-1 AG>Alb>Lipo	Liver CYP450 3A4 isoform to norfentanyl (inactive) by oxidative N-dealkylation	Renal (75% inactive metabolites, <10% unchanged) Feces (9% metabolites) $T_{½}$ = 17 h (13-22)

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Methadone	Excellent GI absorption T_{max} = 2-3 h	PPB = major site: alpha 1-acid glycoprotein	Liver CYP3A4	Renal and feces pH-dependent urinary excretion $T_{½}$ = 8-59 h

T_{max}, peak plasma concentration time; PPB, plasma protein bound; M3G, morphine-3-glucuronide; M6G, morphine-6-glucuronide; BBB, blood-brain barrier;

$T_{½}$, plasma half-life; H3G, hydromorphone-3-glucuronide; α-1 AG, α-1 acid glycoprotein; Alb, albumin; Lipo, lipoprotein.

[a] Drug that escapes hepatic and first-pass elimination.

[b] Depending on type of preparation, such as immediate release, or sustained or controlled release.

Modified from Sharma A, Raja SN. Assessment and management of chronic pain and palliative care. In: Sieber FE, ed. *Geriatric Anesthesia* . The McGraw-Hill Companies, Inc. 2007;319-336.

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TABLE 164.3 INTRAVENOUS OPIOID PATIENT-CONTROLLED ANALGESIA GUIDELINES FOR OPIOID-NAIVE ADULTS WITH ACUTE PAIN

DRUG (USUAL STANDARD CONCENTRATIONS)	USUAL STARTING DEMAND DOSE	USUAL DOSE RANGE	USUAL STARTING LOCKOUT (MIN)	USUAL LOCKOUT RANGE (MIN)
Morphine (1.0 mg/mL)	1.0 mg	0.5-2.5 mg	10	6-10
Hydromorphone (0.2 mg/dL)	0.2 mg	0.05-0.4 mg	10	6-10
Fentanyl (10 mcg/mL)	10 μg	10-50 μg	6	6-8

From American Pain Society. *Principles of Analgesic Use in the Treatment of Acute Pain and Cancer Pain* . 5th ed. Glenview, Ill.: American Pain Society; 2003;20.

Opioid conversions in perioperative setting are required in many different scenarios. Some of the patients undergoing surgical procedures are already on opioid medications for their pain management. These medications need to be taken into consideration when planning for postoperative PCA pain regimen. Similarly, PCA can be used in any setting where oral pain management becomes impossible. Prior to calculating the new dose of any alternate opioid, it is recommended to note the average 12- to 24-hour usage of current opioid medications. Subsequently, the equianalgesic conversion can be done by using the following formula:

$$\text{Dose of alternate opioid drug or route in a given period} = \frac{\text{Dose of current opioid drug (s)}}{\text{EAN of current opioid drug (s)}} \times \text{EAN of alternate opioid drug or route}$$

where EAN is the equianalgesic number of an opioid given by a specific route (Table 164.2). For instance, a patient who takes 30 mg of long-acting morphine preparation twice a day along with three to four tablets of short-acting morphine for his or her chronic back pain takes approximately 180 mg of oral morphine. For any PCA orders to be written in this situation requires correction for this additional morphine. Calculation of equianalgesic dose (EAD) for IV morphine can be done as

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$$\text{24-hour dose of IV morphine} = \frac{180}{30} \times 10 = 60 \text{ mg or } 2.5 \text{ mg of morphine per hour } (60/24)$$

Thus, 2.5 mg of IV morphine per hour is added to the PCA orders. An increase in the total dose by 25% to 50% is recommended for treating moderate to severe pain in patients who are on opioid therapy for longer than 5 days. Subsequent changes in PCA orders should only be made after five half-life ($T_{½}$)

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periods. Thus, treatment of postoperative pain following a spine surgery in the previously mentioned patient would require approximately 3 to 3.75 mg of IV morphine per hour. Half of this dose can be safely administered as basal infusion, with the remaining provided as demand doses with suitable lockout interval. Only 10% to 20% increment is recommended for an opioid-naive patient (therapy less than 5 days) under analogous circumstances.

Following repeated usage, the rotation of one *Mu*-opioid receptor agonist to another often results in evidence of pharmacologic supersensitivity toward the new agent, a concept known as *incomplete cross-tolerance*. Thus, conversion of one opioid to another requires a reduction in the total dose of new opiate by 25% to 50% to compensate for incomplete cross-tolerance and to avoid overdose. For example, if the aforementioned patient was on 20 mg of sustained-released oxycodone and five to six doses of 10 mg of short-acting oxycodone for breakthrough pain, the EAD calculations would suggest changing it to roughly 40 mg of IV morphine. In practice, though, this dose of morphine is likely to cause overdose or side effects in this particular patient. If pain had been adequately controlled on previous regimen, then this dose should be reduced by 25% to 50% (0.8- to 1.2 mg of IV morphine per hour). Again, half of this dose can be given as basal infusion and the remainder as demand doses. It is important to understand that due to significant interpersonal variations, a “one size fits all” technique would lead to both physician and patient dissatisfaction. After a therapy is initiated, regular pain assessments should be made, and treatment should be titrated accordingly.

$$\text{24-hour dose of IV morphine} = \left\{\frac{40}{20} + \frac{60}{30}\right\} \times 10$$

$$= 40\ \text{mg/24 hours or } 1.67\ \text{mg of morphine per hour}$$

Conversion of transdermal fentanyl to other opioid drugs is slightly intricate. Rather than using the EAD formula, it is probably safer and easier to use a separate transdermal fentanyl table as recommended by the manufacturer (Table 164.4). For example, a patient receiving 50 μg/hour of transdermal fentanyl corresponds to 135 to 224 mg of oral morphine (mean = 180 mg). This range can now be used to convert to any other opioid drug or route using the standard method, as previously described. A conservative estimate would mean that it is equivalent to the lower end of this range and vice versa. In our practice, we often accept the mean of this range (180 mg in this case) and adjust the dose further based on the patient's response. In short, to accommodate 50 μg/hour of transdermal fentanyl into IV morphine PCA, we need to add 60 mg morphine/24 hours.

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TABLE 164.4 RECOMMENDED IINITIAL TRANSDERMAL FENTANYL DOSE BASED ON DAILY ORAL MORPHINE DOSE

ORAL 24-H MORPHINE DOSE (MG/D)	TRANSDERMAL FENTANYL DOSE (μG/H)
60-134	25
135-224	50
225-314	75
315-404	100
405-494	125
495-584	150
585-674	175
675-764	200
765-854	225
855-944	250
945-1034	275
1035-1124	300

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$$\text{24-hour dose of IV morphine} = \frac{180}{30} \times 10$$
$$= 60 \text{ mg or } 2.5 \text{ mg of morphine per hour } (60/24)$$

OPIOID CONVERSIONS FROM INTRAVENOUS TO ORAL FORM

Conversion of parenteral opioids to oral formulation is based on similar principles but may pose certain unique problems. Often, these calculations are requested by the surgical team at the time of the patient's discharge. Any overestimation with these calculations may impose unwanted side effects on the patient. It is essential to teach the surgical teams in your hospital that these steps should be taken at least 24 to 48 hours prior to the patient's discharge, which is usually a sufficient time to assess the patient's response to the new medical regimen. A basic guide for these conversions is shown in Fig. 164.1. For acute pain management, a smaller proportion of total drug should be prescribed as a long-acting formulation. As the pain-inducing injury (or postsurgical inflammation) heals, lesser amounts of short-acting medications will be used by the patient. Based on this response, the long-acting preparations can be reduced afterward and eventually discontinued. On the contrary, more aggressive conversions should be reserved for cancer pain, where as much as half to two-thirds of total dose can be given as long-acting preparation, and dose can be escalated further based on breakthrough drug usage (Fig. 164.1).

P.718

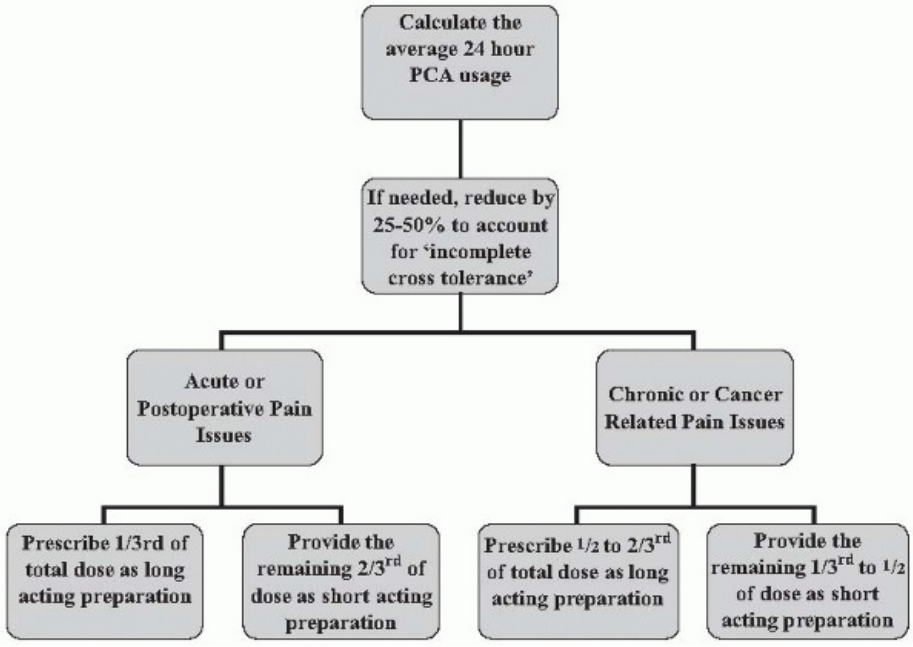


FIGURE 164.1. Changing the parenteral opioid dose to oral form.

TAKE HOME POINTS

- The use of opioid medications in the perioperative setting is increasing.
- Equianalgesic opioid drug conversions are easy to perform and give a reasonable estimation of the initial dose.
- Common errors in these conversions can be avoided by following the described method.
- The safety of these calculations can be enhanced by initially accepting the conservative side of dose range and later adjusting the dose according to the patient's response.
- When in doubt, always err on using smaller doses of basal infusion or long-acting preparations.
- Physicians should also be prepared to recognize and manage any possible side effects or add adjuvant medications to the regimen when needed. Compassion and diligence eventually lead to smooth transitions, which is often a gratifying experience for both the physician and their patients.

P.719

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Consider Discussing the Use of Ketorolac (Toradol) with Your Surgical Team Before the Need Arises

Amit Sharma MD

"In a fight between an anesthesiologist and a surgeon, the patient is always defeated."

It was another fine morning. We were finishing our first scheduled laparoscopic urologic surgery case when I received a Stat page to the operating room (OR). As I was reaching the OR, I could hear our famous urologist roaring at my resident. Apparently, my patient-compassionate resident had used ketorolac (Toradol; 30 mg IV) for postoperative pain control, and our surgical friend firmly believed that it would lead to renal failure or post-surgical bleed. I tried dodging his logic of so-called experience with my shield of scientific data leading to more noise pollution in the OR. The end result of our *battle for Middle Earth* was the formulation of the Eleventh Commandment by our respected colleague: "NO TORADOL for any of my patients EVER." Currently, ketorolac is perhaps one of the leading causes of tense anesthesiologist-surgeon relationships, and it is briefly discussed in this chapter.

Ketorolac tromethamine (commercially available as Toradol) is a member of the pyrrole-pyrrole group of nonsteroidal anti-inflammatory drugs (NSAIDs). It is available for intravenous (IV) or intramuscular (IM) administration as 15 mg in 1 mL and 30 mg in 1 mL (60 mg in 2 mL) solution. It is also available in tablets of 10 mg for oral use. It is a peripherally acting analgesic agent that inhibits prostaglandin synthesis. The drug possesses no sedative properties, making it a good agent in the perioperative setting. The bioavailability with oral, IM, or IV use is 100%, with peak analgesic activity time of 2 to 3 hours via oral route and 1 to 2 hours when used parenterally. Ketorolac is highly protein bound; metabolized in the liver via hydroxylation and conjugation, and eliminated by the kidneys (60% metabolites, 40% parent drug). It has a half-life of about 5 to 6 hours. The drug half-life is prolonged and unpredictable in the elderly and in the presence of renal impairment, having poor correlation with creatinine clearance values. Its pharmacokinetics are relatively unchanged in the presence of mild to moderate hepatic dysfunction. Thirty milligrams of IV ketorolac provide similar analgesic results as 4 mg of IV morphine, but a similar dose via IM route is equivalent to 6 to 8 mg of IM morphine. When used with opioids for postoperative pain management, ketorolac reduces opioid requirements, lowering their potential side effects. P.721

Currently, ketorolac is indicated for short-term (≤5 days) management of moderately severe pain in the postoperative setting. It may be given as a single or multiple dose on a regular or as needed schedule up to a maximum of 5 days. Its frequently used dose profile is depicted in Table 165.1. IV injection is recommended to be given over at least 15 seconds, while the IM dose must be given slowly and deeply into the muscle. The time of onset of ketorolac is roughly 30 minutes. Peak effects are seen in 1 to 2 hours via either route, and clinical effects last for 4 to 6 hours. Mixing parenteral formulations with morphine, meperidine, promethazine, or hydroxyzine in the same syringe leads to precipitation and should be avoided.

Despite these desirable attributes, use of ketorolac comes with its own set of problems. Its relevant side effects and absolute contraindications are described in Table 165.2. Prostaglandins play a positive role in maintenance of platelet function and renal perfusion. The potent inhibitory effects of ketorolac on prostaglandin synthesis might jeopardize either of these vital phenomenons. Ketorolac use should be avoided in patients on therapeutic doses of anticoagulants, including prophylactic low-dose heparin therapy. It should also be avoided preoperatively, in any major surgical case, or when hemostasis is critical. It should be kept in mind that ketorolac-induced increased risk of bleeding cannot be detected on routine testing, via platelet count, prothrombin time, or activated partial thromboplastin time. It should also be used with extreme caution, if at all, in patients with impaired renal function. Ketorolac may cause dose-dependent reduction in renal prostaglandin formation and may precipitate acute renal failure. This is more likely to occur in patients with impaired hepatic or renal functions, dehydration, heart failure, concomitant use of diuretics, and advanced age. It is, therefore, not surprising that our surgical colleagues get really nervous with ketorolac use.

Regardless of these intimidating concerns, ketorolac use has been shown to be relatively safe in innumerable studies. A recent meta-analysis showed that NSAIDs caused a clinically unimportant transient reduction in renal function in the early postoperative period in patients with normal preoperative renal function. The authors concluded that NSAIDs should not be withheld from adults with normal preoperative renal function because of concerns about postoperative renal impairment. Ketorolac has been shown to effectively control postoperative pain after major abdominal, orthopedic, or gynecologic surgery, or after ambulatory laparoscopic procedures. Simultaneous use of ketorolac with opioids results in a 25% to 50% reduction in opioid requirement, resulting in reduction of side effects, more rapid return to normal gastrointestinal (GI) function, and a shorter in-hospital stay. Although many published case reports and case series have shown an increase in intra- and postoperative bleeding following tonsillectomy in children after ketorolac use, such findings have not been reproduced in adults. Ketorolac does increase bleeding time to some extent, but its actual contribution to surgical site bleeding is perhaps inconsequential. Besides, these risks can be reduced to minimal by adhering to its strict treatment guidelines. P.722

TABLE 165.1 RECOMMENDED DOSE OF KETOROLAC IN PERIOPERATIVE SETTING

	INTRAMUSCULAR (IM)		INTRAVENOUS (IV)		
	SINGLE DOSE SCHEDULE	MULTIPLE DOSE SCHEDULE	SINGLE DOSE SCHEDULE	MULTIPLE DOSE SCHEDULE	ORAL (PO)†
Age 2-16 years	1 mg/kg Max = 30 mg	NR	0.5 mg/kg Max = 15 mg	NR	NR
Age > 16 but <65 and BW ≥110 lbs	60 mg	30 mg Q 6° Max = 120 mg	30 mg	30 mg Q 6° Max = 120 mg	1st Dose = 2 tabs followed by 1 tab Q 4-6° Max = 40 mg/d

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Age ≥65 Or BW <110 lbs Or Renal Impairment	30 mg	15mg/kg Q 6° Max = 60 mg	15 mg	15 mg Q 6° Max = 60 mg	1st Dose = 1 tab followed by 1 tab Q 4-6° Max = 40 mg/d

NR = not recommended; BW = body weight; Q = every; ° = hours; lbs = pounds; mg = miligrams.

† = Recommended ONLY as a continuation therapy of IM or IV ketorolac therapy.

P.723

TABLE 165.2 COMMON CONCERNS WITH KETOROLAC USE

RELEVANT SIDE EFFECTS	CONTRAINDICATIONS
▪ Headache (17%) ▪ Nausea (12%) ▪ Drowsiness or dizziness (6%-7% each) ▪ Dyspepsia (12%) ▪ GI pain (13%) ▪ Peptic ulceration ▪ GI bleeding and/or perforation ▪ Platelet function inhibition ▪ Hypersensitivity reactions: range from bronchospasm to anaphylactic shock ▪ Acute renal failure ▪ Interstitial nephritis ▪ Nephrotic syndrome ▪ Fluid retention and edema (4%) ▪ Oliguria, elevated BUN and creatinine ▪ Elevated liver enzymes	▪ Active peptic ulcer disease ▪ Recent GI bleeding or perforation ▪ H/O peptic ulcer disease or GI bleeding ▪ Advanced renal impairment ▪ Renal failure risk due to volume depletion ▪ Suspected or confirmed cerebrovascular bleeding ▪ Hemorrhagic diathesis ▪ Incomplete hemostasis ▪ High risk of bleeding ▪ Prophylactic use before any *major* surgery ▪ Intraoperative use when hemostasis is critical ▪ H/O hypersensitivity reaction to ketorolac ▪ H/O allergic reaction to aspirin or other NSAIDs ▪ Concurrent use with aspirin or other NSAIDs ▪ Intrathecal or epidural use ▪ In labor and delivery

GI, gastrointestinal; BUN, blood urea nitrogen; H/O, history of; NSAIDs, nonsteroidal anti-inflammatory drugs.

It is imperative for anesthesiologists to be aware of issues related to ketorolac prior to its use. Thorough knowledge would help us enhance the pleasant nature of the "Toradol conversation" with our surgical associates. No textbook can teach us communication skills, which would certainly come in handy during these intricate dialogues. Surgeons do have the right to know the anesthetic management and our plans for postoperative pain control. A formal discussion would help them understand the benefits of ketorolac and would also help their patients in the long run.

P.724

TAKE HOME POINTS

- Ketorolac tromethamine is a nonselective NSAID available in both parenteral and oral formulations for the management of moderately severe pain in the postoperative setting for ≤5 days.
- Administering 30 mg of IV ketorolac has the same equivalent analgesic effect as 4 mg of IV morphine, with much lower incidence of nausea.
- Ketorolac is contraindicated in patients with a history of peptic ulcer disease, bleeding problems, and advanced renal impairment, or in patients at risk for renal failure due to volume depletion, prior to major surgery, in the presence of uncertain hemostasis.
- Ketorolac has been shown to be a safe and effective analgesic agent in the postoperative setting.
- A mutual agreement between the anesthesiologist and the surgeon prior to the use of ketorolac would result in a more cordial atmosphere and better patient outcomes.

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The Basal Infusion Mode in Patient-controlled Analgesia Is Both Friend and Foe

Amit Sharma MD

Patient-controlled analgesia (PCA) is the core therapeutic drug delivery system in acute and acute-on-chronic pain settings. Historically, in 1971, Sechzer published the first report showing the safety and efficacy of delivering small incremental doses of opioids using a machine. Since his initial description, this novel technique of analgesic drug delivery has been enormously refined and widely accepted. It emanated from the basic pharmacologic principle that a given analgesic medication needs to attain a specific serum concentration to produce analgesia. The lowest drug levels at which this is accomplished is the minimum effective analgesic concentration (MEAC) of that particular drug. Maintaining a constant serum drug concentration above MEAC would thus result in sustained analgesia. It is almost impossible to achieve stable serum drug levels with intermittent oral and intramuscular drug delivery methods. They often result in *peaks* of high drug concentrations that cause side effects and *troughs* of lower drug levels leading to inadequate analgesia (Fig. 166.1). These variations in drug levels can be avoided with the use of intermittent small intravenous (IV) doses or a continuous infusion.

Using these principles, opioids are now often delivered intravenously using highly sophisticated microprocessor-controlled units (e.g., PCAmachine) in numerous acute and certain selected chronic pain settings. These machines allow several modes of drug administration, some of which are as follows:

1. **Demand-Only Mode (DOM):** This is the most commonly used method. The patient can self-administer a fixed dose (demand dose) into their IV.
2. **Continuous Plus Demand Mode (CDM):** A fixed baseline (basal or background) dose is infused intravenously every hour in addition to demand dosing.
3. **Variable-Rate Infusion Plus Demand Mode (VID):** In addition to patient dosing, continuous basal rate is preprogrammed on an internal clock to vary, turn on, or turn off during specified hours of the day.
4. **Variable-Rate Feedback Infusion Plus Demand Mode (VFID):** Basal infusion rate is varied by the PCAmachine's microprocessor and is based on patient's usage. P.726

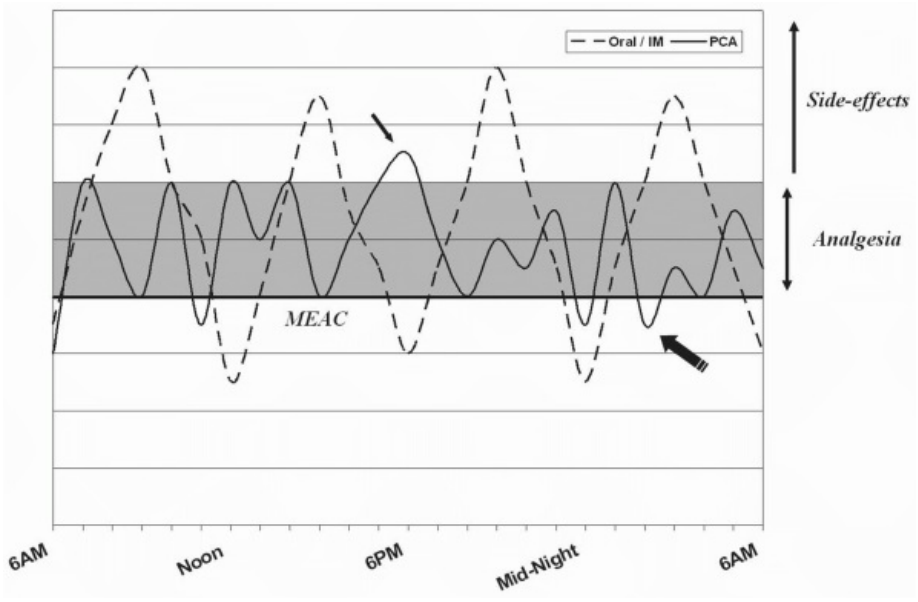


FIGURE 166.1. Comparison of systemic drug concentrations (*y* axis) during conventional oral or intramuscular (IM) opioid drug delivery system and during patient-controlled analgesia (PCA) use. The dark line demonstrates the minimally effective analgesic concentration (MEAC), and the shaded area represents the analgesic window. Serum drug concentrations above the analgesic window (*thin arrow*) result in clinically relevant side effects, while those below (*bold arrow*) cause poor pain control.

Given the widespread use of PCAin postoperative settings, it is imperative for all anesthesiologists to have a precise understanding of their functioning. Although standard PCAguidelines exist for postoperative analgesia (Table 164.3), confusion often prevails with regard to basal infusion. Proponents of basal infusion claim that it allows better patient satisfaction because the demand frequency with CDMis lower than in DOM. It seems that the constant IV infusion of opioid would maintain a certain level of serum drug levels and would then be easier to reach the MEAC using fewer demand doses (Fig. 166.2). In reality, this perception is oversimplistic and does not account for multiple variables on which drug pharmacokinetics actually depends. For instance, the serum drug concentration required to reach MEAC varies from one individual to the next. This situation is further complicated by the fact that significant interpersonal variability exists for the safe *analgesic window*, which is defined as the serum drug concentrations that are associated with clinically relevant analgesia without any side effects (shaded area in Fig. 166.1). Certain individuals, such as elderly people, have relatively narrow analgesic windows, and use of basal infusion would pose undue risks for side effects in these patients. P.727

The futility and risks of basal infusion, especially in opioid-naïve patients, has been shown in multiple studies (Table 166.1). Guler et al. showed

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significant improvement in pain scores with the use of CDM technique in the postoperative pain setting, but most other studies have failed to replicate those results. Even with similar analgesic response, opioid consumption has been shown to be higher with CDM than with DOM in numerous studies. Moreover, the low-dose continuous nighttime-only infusion (VID mode) failed to show any improvement in patients' sleep or comfort in most studies (Table 166.1). CDM technique in these studies failed to show any major increase in the incidence of severe side effects. Other studies and case series, in contrast, have frequently linked the additional background opioid infusion to myriad side effects. Some of the feared complications include respiratory depression and higher chances of programming errors.

Despite additional risks, use of continuous basal infusion is recommended for opioid-tolerant patients by some authors and is frequently used in cancer patients in clinical practice. It can also be a handy tool in the presence of certain personality traits and some neurologic disorders. One of the important prerequisites of PCA usage is the patient's willingness and capacity to self-administer demand doses at appropriate times. Patients with external locus of control, severe depression, and learned helplessness are often unable to use the demand doses adequately. Also, patients with neurologic problems such as Parkinson disease or motor neuron diseases might find the use of the demand button challenging. A low-dose basal infusion can be slowly titrated in these subgroups with improved results. Caution is certainly advised in all patients whenever basal infusion is being considered. Risk factors for respiratory depression with IV PCA usage include age older than 70 years; presence of renal, hepatic, pulmonary, or cardiac impairment; obesity and sleep apnea (suspected or history); use of concurrent central nervous system depressants; and upper abdominal or thoracic surgery. Basal infusion should be avoided in these patients, whenever possible.

P.728

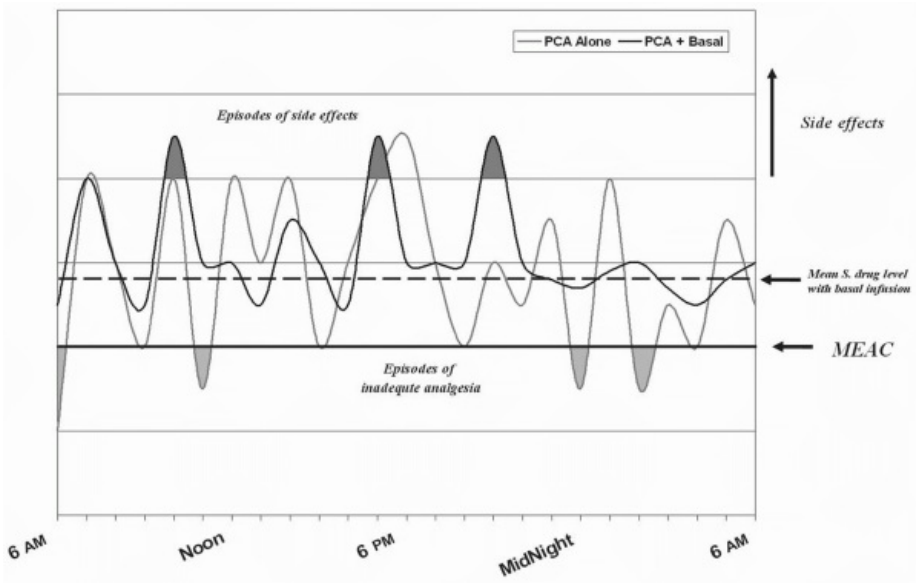


FIGURE 166.2. Optimum use of basal infusion during opioid drug delivery using a patient-controlled analgesia (PCA) system. Under ideal situations, continuous basal infusion raises serum drug concentrations closer to or above minimally effective analgesic concentration (MEAC) (*dotted line*). This may reduce the number of demand doses required to stay in the analgesic window and may reduce the episodes of inadequate analgesia. As evident, it may also increase the chances to develop clinically relevant side effects.

P.729

TABLE 166.1 RELEVANT TRIALS COMPARING IV PCA DEMAND-ONLY AND CONTINUOUS PLUS DEMAND MODE

STUDY, YEAR	TYPE OF STUDY METHOD	RESULTS
Guler 2004	Randomized controlled trial 60 adults undergoing elective CABG surgery IV morphine PCA: DOM vs. CDM Follow up: 24 h postoperative	Similar sedation scores in both groups CDM group had [check mark] Significantly lower VRS scores after first hour [check mark] Greater cumulative morphine consumption [check mark] No episodes of hypoxemia or hypertension
Dal 2003	Randomized double-blind trial 35 adults patients undergoing elective open heart surgery IV morphine PCA: DOM vs. CDM Follow up: 44 h postoperatively	[check mark] Less morphine consumption at 44 h in DOM (P = 0.0006) [check mark] No significant difference between two groups in VAS scores, blood levels of morphine, and adverse effects
Russell 1993	Controlled trial 62 patients undergoing gynecologic surgery IV morphine PCA: DOM vs. CDM (1 mg/h) Follow-up: VAS scores and SpO_2 for 24 h postoperatively	[check mark] Significant increase in total morphine consumption in CDM [check mark] Similar pain scores in both groups [check mark] No difference in severity of postoperative desaturation between two groups

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Parker 1992	Randomized controlled trial 156 adult women undergoing elective abdominal hysterectomy IV morphine PCA: DOM vs. CDM (POD 0—continuous infusion of 1 mg/h, POD 1-2—nighttime infusion, 10 PM-8 AM)	[check mark] No improvement in patient's ability to sleep or to rest comfortably at night [check mark] Similar numbers of patient demands and supplemental bolus doses, opioid usage, and recovery parameters in the two treatment groups [check mark] CDM group had six programming errors; in addition, therapy was discontinued in three patients due to hemoglobin oxygen desaturation

CABG, coronary artery bypass grafting; IV, intravenous; PCA, patient-controlled analgesia; DOM, demand-only mode; CDM, continuous plus demand mode; VRS, verbal rating scale; VAS, visual analog scale; SpO_2, oxyhemoglobin saturation.

The concept of PCA involves the administration of drug based on the patient's demand. It also applies to alternative routes of drug delivery, such as transdermal, subcutaneous, oral transmucosal, epidural (patient-controlled epidural analgesia [PCEA]), intrathecal, and regional (patient-controlled regional analgesia [PCRA]). Although this chapter mainly focuses on IV PCA basal infusion, similar notions can be extrapolated to these alternative modes of drug delivery, with few exceptions. In general, for postoperative pain control, demand dose systems using local anesthetic and an opioid combination (PCEA and PCRA alone) provide similar analgesic response with superior patient satisfaction rates when compared to continuous infusion techniques. They also require fewer interventions by nursing staff and acute pain team members. However, addition of a small-dose continuous infusion along with demand boluses has been shown to reduce total anesthetic consumption with similar analgesic results. It is thus recommended that PCEA and PCRA management should be optimized with the addition of a low-dose basal infusion on a routine basis. P.730

TAKE HOME POINTS

- PCA is being universally employed to enhance analgesia in acute and certain chronic pain conditions.
- Overall, PCA with DOM provides better pain control with no worsening of side effects when compared to intermittent intramuscular or oral regimen.
- In opioid-naive patients, use of background or basal infusion leads to increase in opioid consumption with no significant improvement in pain control.
- Basal infusion can be effectively used in the presence of opioid tolerance, certain complex personality traits, and neuromuscular diseases, as well as in cancer-related pain states.

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Management of Perioperative Pain in Opioid Dependent Patient—Out of the Frying Pan, Into the Fire

Amit Sharma MD

"To be conscious that you are ignorant is a great step to knowledge."

---Benjamin Disraeli

Medical science has witnessed a dramatic shift in the awareness and treatment of chronic pain over the past few decades. Numerous organizational and regulatory bodies have declared pain as the fifth vital sign to encourage physicians to actively pursue and treat pain problems. Moreover, expansion in life expectancy has caused a relative increase in the elderly population, which carries the brunt of chronic pain problems. Given this scenario, it is not uncommon for anesthesiologists to encounter patients who are taking opioids. Some of them are on certain colossal doses that widen our eyes. Lack of knowledge on our behalf, bundled with uncertainty about opioid dose requirements and expectations of a smooth perioperative course by family and the surgical team, certainly make these cases challenging. These difficult situations can be handled with poise by knowing some fundamental principles related to the perioperative care of chronic opioid-dependent patients.

Anesthetic care of these intricate cases begins with early recognition of opioid-tolerance issues (Table 167.1) and formulation of a clear plan. A preoperative visit allows the anesthesiologist to perform a thorough medical evaluation, outline a postoperative pain management plan, and advise on therapeutic options. Information about the patient's opioid and adjuvant medication doses should be gathered. An electrocardiogram should be obtained to look for any QT interval prolongation, which is sometimes seen with the use of high doses of methadone. Prolongation of the QT interval is also seen with tricyclic antidepressant medications, which are frequently used as adjuvant drugs in chronic pain patients. A prolonged QT interval can predispose a patient to ventricular tachycardia, ventricular fibrillation, or torsades de pointes, and must be identified. A detailed discussion should be had with the patient to address his or her anxieties and fears. Patients are often worried about excessive pain following the surgery, which is frequently a reflection of their previous experiences. Anesthetic management options should be discussed. Whenever possible, a regional anesthetic technique should be chosen and explained to the patient.

TABLE 167.1 PERIOPERATIVE ISSUES IN OPIOID-DEPENDENT PATIENTS

- Tolerance or increased analgesic requirements
- Poor pain control
- Opioid-induced hyperalgesia
- Opioid withdrawal
- Excessive anxiety
- Gastric aspiration
- Cardiac arrhythmias
- Physician's biases

There have been numerous studies indicating delayed gastric emptying in opioid-tolerant patients. Delayed gastric emptying is presumably related to an increase in pyloric sphincter tone associated with prolonged opioid use. Thus, opioid-dependent patients should be encouraged to stay "nothing by mouth" for longer durations to reduce the aspiration risk. They should also be advised to continue their analgesic regimen until the day of surgery to prevent any withdrawal or falling behind on opioid requirements. The only analgesic medication that should be reduced during the perioperative period is intrathecal baclofen, which has been suggested to enhance the effects of neuromuscular-blocking drugs and increase the incidence of hypotension and excessive sedation. Any such decision must be made after talking to the patient's pain physician.

Patients should be advised to arrive early on the day of surgery. Appropriate pre-emptive analgesia should be given in the preoperative period. Oral acetaminophen (1 g orally 1-2 hours preop) and certain nonsteroidal anti-inflammatory drugs (NSAIDs) (piroxicam 20 mg oral or Celebrex 200 mg oral—2 hours preop), with or without antiepileptic drugs (gabapentin 600 mg oral—2 hours preop), should be given. The anesthetic plan should again be discussed in detail with the patient, and regional anesthesia should be considered, whenever feasible. Recent reports have indicated that erroneous

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doses of fentanyl can be delivered from the transdermal system (fentanyl patch) when a warming blanket is placed on or near the patient. Transdermal fentanyl patches should be removed prior to taking the patient to the operating room. Furthermore, every effort should be made to avoid placing warming devices at the site from where fentanyl patches have been removed because similar effects can be seen with an increased uptake from the dermal fentanyl "depot."

If opioid medications have been discontinued preoperatively for any reason, a long-acting opioid should be titrated intravenously at the time of induction to achieve a spontaneous respiratory rate of 14 to 16/minute. An intravenous (IV) bolus of ketamine (0.5 mg/kg) should also be administered intraoperatively and then followed with an infusion of 4 µg/kg/minute. Ketamine is an NMDA receptor antagonist drug that has been shown to reduce the postoperative opioid usage in opioid-dependent patients. Clonidine and dexmedetomidine (α 2-adrenergic agonists) have also been shown to have similar effects. If pre-emptive analgesia was not begun preoperatively, then 1 g of acetaminophen (per rectal) and IV ketorolac (30 mg) or flurbiprofen should be considered at the time of (or following) induction. Last, but not least, the surgical team should be encouraged to infiltrate a long-acting local anesthetic solution at the site of incision. Intraoperative pain management often requires as much as 50% to 300% higher doses of opioids in an opioid-dependent patient, than in an opioid-naive patient for a similar surgical procedure. If general anesthesia is required, these patients should be reversed early and allowed to return to spontaneous ventilation. IV opioids should then be titrated to achieve a respiratory rate of 12 to 14/minute prior to extubation.

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As expected, postoperative pain management is equally difficult in these patients. Unfortunately, intraoperative opioid usage does not help in estimating postoperative opioid requirements. Therefore, continuous regional techniques certainly come in handy under these circumstances. If epidural technique is used, a local anesthetic, in combination with a potent lipophilic opioid drug (e.g., fentanyl or sufentanil), should be used. Even when regional techniques are used, a patient-controlled analgesia (PCA) with a moderate demand-only dose should be used for supplemental analgesia. When regional techniques are not an option, PCA (with basal infusion and a moderately higher demand dose) should be considered as a second choice. Patient's preoperative total opioid usage should be taken into account while prescribing PCA basal infusion. These oral-to-IV opioid conversions are discussed in Chapter 164. A two- to four-fold increment in opioid requirement should be expected in the postoperative period than those used by an opioid-naive patient. The patient should be monitored in a postanesthetic care unit for oversedation or respiratory depression, and frequent evaluations should be done to ensure timely adjustments in PCA settings. If ketamine was begun during the intraoperative course, it should be at least resumed for 24 to 48 hours postoperatively. Acetaminophen and NSAIDs should also be continued during the postoperative period, with hepatic and renal function monitoring. The patient should be subsequently referred to the chronic pain management service of the hospital for a close follow-up.

TAKE HOME POINTS

- Perioperative pain management in opioid-dependent patients is demanding.
- A preoperative visit certainly helps address many issues, including patient's concerns.
- Pre-emptive analgesia is the most important step leading to reduction in postoperative opioid requirements.
- Whenever possible, regional techniques should be used for intraoperative and postoperative management.
- Adjuvant medications such as acetaminophen, NSAIDs, ketamine, and α 2-adrenergic agonists also help reduce opioid requirement in this difficult population.
- A close follow-up by acute and chronic pain management teams is warranted to ensure best postoperative care in opioid-dependent patients.

P.735

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Do Patients with Congenital Insensitivity to Pain Need Anesthetics and Postoperative Opioids?

Katarina Bojanic MD
Toby N. Weingarten MD
Juraj Sprung MD, PhD

HEREDITARY SENSORY AND AUTONOMIC NEUROPATHY

Congenital hyposensitivity to pain, hereditary sensory and autonomic neuropathy (HSAN), is a group of rare genetic disorders characterized by varying degrees of sensory loss, including nociceptive hyposensitivity, loss of other types of sensation, and various degrees of autonomic dysfunction. Sensory loss, especially the loss of pain sensation, is associated with selfmutilation that may require frequent surgery (Fig. 168.1). Little is known regarding whether or how much these patients require anesthesia for surgery or opioids postoperatively. Furthermore, because of various degrees of autonomic dysfunction, these patients may be at increased risk of perioperative anesthetic complications.

CLASSIFICATION OF CONGENITAL HYPOSENSITIVITY TO PAIN

Congenital hyposensitivity to pain disorders were categorized by Dyck et al. into five different types of HSANs (I-V). Different expressions of sensory loss are possible within each HSAN category, and some patients may have variable pain sensation. The types of HSAN are distinguished by the mode of inheritance, clinical features, degree of autonomic nervous system abnormalities, loss or degeneration of sensory fibers, and increasingly specific molecular genetic abnormalities:

- **HSAN I** is the only autosomal dominant disorder. It is characterized by onset later in life and a sensory deficit that is more pronounced in the legs than in the hands. In HSAN I, sensory deficits overshadow autonomic dysfunction.
- **HSAN II** patients tend to have pain hyposensitivity of the upper and lower limbs. Many have defective tactile sensation, whereas a minority may have areas of normal trunk sensation. Relevant autonomic dysfunction may include episodic hyperthermia and swallowing deficiencies.
- **HSAN III**, also known as Riley-Day syndrome or familial dysautonomia, has a higher prevalence in Ashkenazi Jews. These patients typically present in infancy with a profound dysautonomia (poor feeding with repeated vomiting, failure to thrive, and temperature and vasomotor dysregulation associated with hypertension or hypotension), recurrent pulmonary infections, diminished peripheral pain and temperature sensation, and absence of vibratory perception. P.737
- **HSAN IV**, also known as congenital insensitivity to pain *with* anhidrosis, is characterized by hyposensitivity to superficial and deep visceral pain, mild to moderate mental retardation, and recurrent episodes of hyperpyrexia due to absence of sweating (no innervation of sweat glands).
- **HSAN V**, also known as congenital insensitivity to pain *without* anhidrosis, resembles HSAN IV but is associated with a selective absence of small sensory myelinated fibers (Aδ fibers), which are important for sensing the sharp, well-localized, and prickling sensations of pain. These patients typically respond to tactile, vibratory, and thermal stimuli.

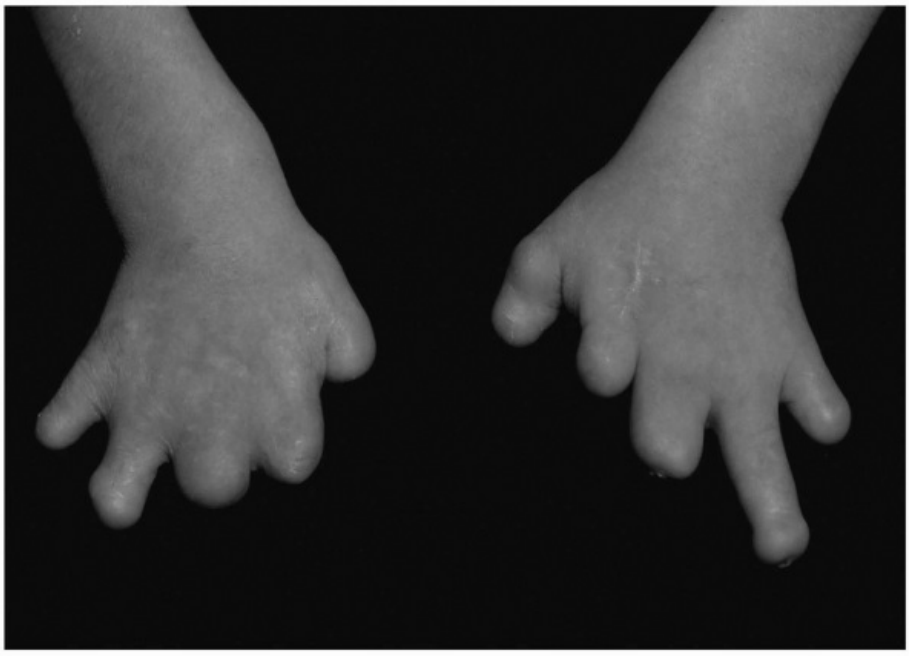


FIGURE 168.1. The hands of a 12-year-old boy with hereditary sensory and autonomic neuropathy (HSAN) II showing acromutilation. This type of

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self-mutilation may be found in all types of recessively inherited HSANs and is attributed to loss of pain sensation, neglect of injury, excessive surgery, and indifferent personality. (From Weingarten TN, Sprung J, Ackerman JD, et al. Anesthesia and patients with congenital hyposensitivity to pain. *Anesthesiology.* 2006;105:338-345. Used with permission.)

ANESTHESIA AND HEREDITARY SENSORY AND AUTONOMIC NEUROPATHY DISORDERS

No consensus exists regarding the intraoperative analgesic needs of HSAN patients. Some patients with HSAN have tactile hyperesthesia, and some may have partially preserved pain sensation; therefore, most reports have described the use of volatile anesthetics in standard concentrations. In addition, P.738
anesthetics are necessary to ensure cooperation and immobility during surgery for pediatric patients, especially for those with mental retardation. The optimal dose of anesthetics for patients with HSAN has yet to be determined. At present, inhalational anesthetics should be titrated in accordance with the patient's hemodynamic response. A case reported by Layman showed that, in a 30-year-old man with congenital insensitivity to pain, bilateral lower extremity amputation could be performed by using only heavy sedation, without opioids or general anesthesia.

Most reports have documented minimal or no use of opioids postoperatively in patients with HSAN IV. When opioids were used perioperatively, no reasoning for their use was offered. The use of opioids in these patients may have followed the anesthesiologists' daily practice of administering opioids with every anesthetic. This “automatism” may occur because HSAN is rarely encountered by anesthesiologists, and because scarce information and no recommendations exist regarding opioid requirements for these patients. Furthermore, some patients with HSAN may have partially preserved nociception, and others may have preserved mechanoreceptor, cooling and warming sensations; therefore, they may sense some aspects of intense surgical stimulation.

Despite decreased pain perception in patients with HSANs, anesthetic administration for these patients has generally been similar to that for patients with normal pain perception. Postoperatively, these patients have minimal or no pain, and most did not require opioid medications.

PERIOPERATIVE MANAGEMENT OF BODY TEMPERATURE

Besides hyposensitivity to pain, patients with HSAN have various degrees of autonomic dysfunction that may affect the course of anesthesia. An important element of anesthesia is prevention of autonomic reflexes. Because patients with HSAN IV have anhidrosis, management of temperature homeostasis in daily life may be difficult. One study indicated that almost 20% of these patients died of hyperpyrexia during the first 3 years of life. Thus, perioperative thermoregulation is a concern, and continuous temperature monitoring is of great importance in these patients. Only one report has documented intraoperative hyperthermia in a patient with hyposensitivity to pain; in general, normothermia can be easily maintained with alterations of environmental temperature. Atropine inhibits the activity of the sweat glands and, in healthy children, may cause hyperthermia. However, because children with HSAN IV lack innervation of sweat glands, the use of atropine should not be
associated with hyperthermia. Of note, malignant hyperthermia is not associated with HSAN IV; the genetic mechanisms for precipitating hyperpyrexia in P.739
malignant hyperthermia and HSAN IV are fundamentally different. All triggering agents associated with malignant hyperthermia (succinylcholine, halogenated agents, and others) have been used without any complications in HSAN patients.

TAKE HOME POINTS

- Knowledge regarding the safety of anesthesia in patients with HSAN II, IV, and V is scarce. To date, anesthesia has not been associated with any major adverse events in patients with HSAN disorders.
- Although patients with profound congenital insensitivity to pain may undergo major surgery without general anesthesia, most reports indicate that these patients received standard doses of anesthesia for operations.
- Factors other than analgesia, such as immobilization, prevention of autonomic reflexes, anxiolysis, and sedation, are equally important aspects of these patients' anesthetic management.
- Generally, patients with HSAN II, IV, and V do not need opioids postoperatively, even after major operations.

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Rule Out Facet Arthropathy Before Initiating Expensive and Invasive Maneuvers for Back and Spine Pain

Amit Sharma MD

Spinal pain is one of most common and most intricate problems faced by physicians today. Pain of just the lumbar back alone is believed to be the most common etiology of chronic pain syndrome. In addition to the significant personal morbidity, spine-related pain has an enormous economic impact on society—the total cost of low back pain exceeds $100 billion per year in the United States alone. About one third of the overall costs are direct health care expenditures due to overuse of unnecessary diagnostic modalities and therapeutic maneuvers with unproven benefits. The remaining two thirds of the cost are indirect, due to lost wages and reduced productivity. Given this scenario, it is important to formulate precise evidence-based diagnostic and therapeutic plans for patients with spinal pain.

Facet arthropathy has been implicated as the etiology in 67% of patients with chronic neck pain, 48% of patients with chronic thoracic pain, and as many as 40% of patients with chronic lumbar pain. Thus, the high prevalence of facet arthropathy makes it a leading differential cause of almost all spinal pain issues. The facet (or zygapophyseal) joints are true synovial joints connecting the adjacent vertebra posteriorly. They are formed by the inferior articular process of superior vertebra and the superior articular process of inferior vertebra. Two adjacent vertebral bodies thus have a pair of facet joints preventing axial movements of one vertebra over another. The articular surfaces of each facet joint are covered with cartilage, and its edges are covered with synovial membrane and fibrous capsule. Primary dorsal rami of spinal nerves exit from the neural foramen just above and medial to the facet joints and send off medial and lateral branches. This medial branch, in turn, supplies corresponding facet joint and paraspinous muscles. Each facet joint receives innervation from the medial branch of exiting nerve root of corresponding vertebral level and from the level above. Facet joints can develop degenerative changes under certain circumstances (trauma, osteoarthritis, inflammation of synovial capsule, or joint subluxation) and can become quite painful. This painful degeneration of facet joints is known as *facet syndrome* or *arthropathy*.

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PRESENTATION

Cervical facet arthropathy pain presents as pain involving the head, neck, shoulder, suprascapular and scapular area, and upper arm. Distribution of pain varies, depending on the specific facet joint involved. For instance, involvement of the C2-3 joint leads to pain in the occipital area, C3-4 in the neck, and C4-5 along the lateral aspect of the nape of the neck and shoulder area. The associated physical findings may include decreased range of movements at the neck, exacerbation of pain with neck extension, and some degree of alleviation with flexion. Palpation over the specific facet joint may also aggravate pain. Thoracic facet arthropathy presents mainly as thoracic pain along with paraspinous tenderness. The presentation of lumbar facet arthropathy is often as axial lumbar back pain with stiffness, especially in the morning. Pain also involves the hips, buttocks, or thighs and is often exacerbated by prolonged sitting or standing. Similar to cervical facet syndrome, paraspinous tenderness over the affected joint may be elicited. Movements such as hyperextension and lateral rotation over the spine (facet loading maneuver) that stress the facet joints will also aggravate the pain.

DIAGNOSIS

The differential diagnosis for pain that mimics facet arthropathy includes migraine and tension headaches, degenerative disc disease (discogenic pain), spondylosis, spondylolysis, spondylolisthesis, myofascial pain, and sacroiliitis. Unfortunately, the clinical picture is similar in most of these conditions, and it is difficult to distinguish between them by symptomatology or physical signs alone. Laboratory data and imaging studies are not helpful in diagnosing facet arthropathy because degenerative changes of the facet joints are seen on the plain films and on the computed tomography and magnetic resonance imaging scans in asymptomatic subjects. The only reliable way to identify the presence of facet arthropathy is if amelioration of symptoms occurs after local anesthetic blockade of the facet joint's nerve supply. Historically, this was done by injecting the local anesthetic solution inside the facet joint itself under fluoroscopic guidance. In current practice, the same effect is achieved by injecting the anesthetic solution adjacent to the nerve branches (medial branches) supplying these joints.

This catch-22 situation involving a high prevalence of facet arthropathy and only a single invasive mode of diagnosis would seem to indicate that all patients with spinal pain should be subjected to the intervention of facet block. In fact, this scenario is probably too aggressive and would cause discomfort in many patients with other etiologies of spine pain. Hence, identification of the predictive factors for facet arthropathy became imperative to avoid imposing this rather uncomfortable testing on every patient. In 1998,

Helbig and Lee formulated a scoring system to predict a positive response to facet joint injection. Based on a limited study on 12 subjects, 30 points were allocated to back pain associated with groin or thigh pain, and to the reproduction of pain with extension-rotation at spine (positive facet loading). Similarly, 20 points were given to the presence of well-localized paraspinal tenderness and to corresponding positive radiographic changes, while a negative score of 10 was assigned to the existence of pain below the knee. Using this system, a score of 60 points or more was shown to indicate a very high probability of satisfactory response to facet injections. This scoring system can be used as a clinical guide to decide whether diagnostic facet injections are indicated for any given patient.

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Diagnostic nerve blocks have limitations. Single diagnostic facet injections or medial branch blocks have been shown to have a high sensitivity but a low specificity. False-positive results have been shown in 63% of single local anesthetic blocks at cervical level, and these numbers are as high as 62% and 30% at thoracic and lumbar levels, respectively. Presence of placebo response and spread of local anesthetic solution in the adjacent epidural space have been reasoned as the probable causes of these high numbers. Many authors thus recommend double diagnostic blocks using different local anesthetic solutions such as lidocaine and bupivacaine. A differential duration response (lidocaine block being short lasting and vice versa) is taken as dual confirmation before aggressive therapeutic options are exercised.

The blocks are always done under fluoroscopic guidance. Because every facet joint has a dual innervation, it is crucial to block both nerves. At cervical level, C2-3 facet joint is supplied predominantly by the medial branch of C3 dorsal ramus (also called the third occipital nerve). The remaining joints are supplied by medial branches of the dorsal rami exiting at that vertebral level and from the level above. Thus, C4-5 facet joint is supplied by medial branches of C4 and C5 dorsal rami (C4 being exiting from C3-4 neural foramen). Likewise, at lumbar levels, each facet receives nerve supply from the

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dorsal rami of the same level and from the level above. Because L4 dorsal ramus exit from L4-5 neural foramen, medial branches of L3 and L4 dorsal rami need to be blocked to achieve L4-5 facet block. The interventional pain physician should also be aware of the correct positions of these medical branches to optimally perform these blocks. At cervical level, the target area is the central part or "waist" of the articular pillar on the A-P view (Fig. 169.1) and the midpoint of articular pillar projection on the lateral view (Fig. 169.2). At the lumbar levels, the target area is the junction of superior articular process and superior border of transverse process of the level below (Fig. 169.3) for L1-4 medial branches. The L5 medial branch lies at the junction of the sacral ala and superior articular process of S1.

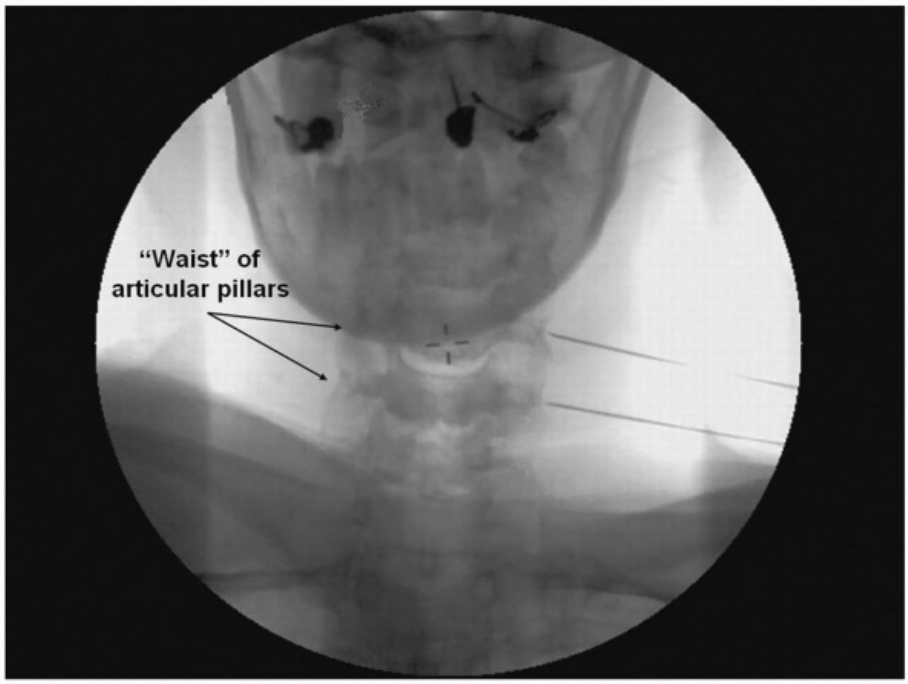


FIGURE 169.1. A-P view of needle placement for cervical medial branch blocks. The needle tip is positioned at the midpoint of articular pillar of corresponding cervical vertebral body (*arrows*).

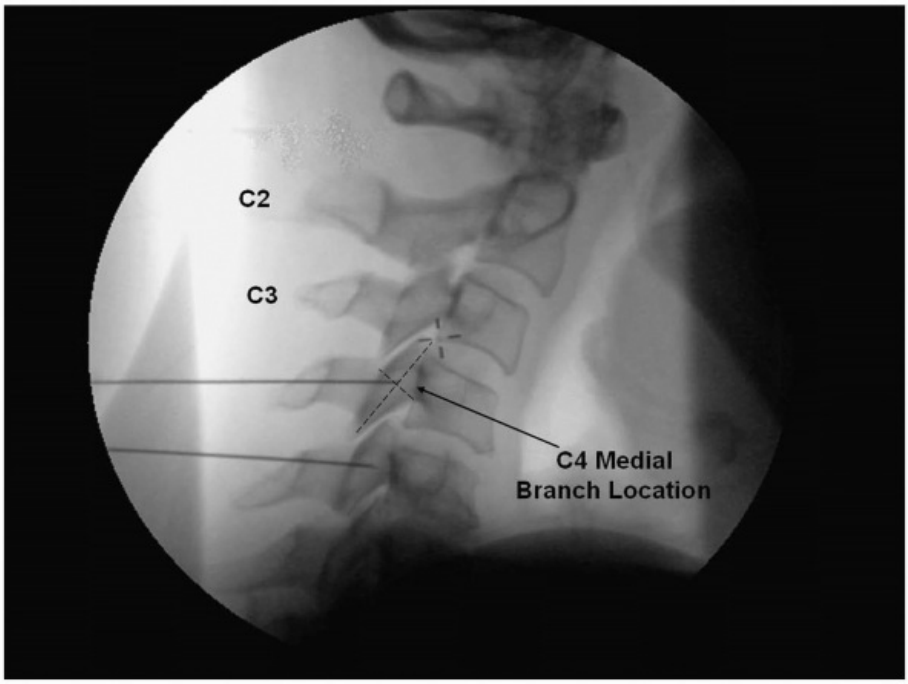


FIGURE 169.2. Lateral view of needle placement for cervical medial branch blocks. The needle tip is positioned at the midpoint of articular pillar of corresponding cervical vertebral body.

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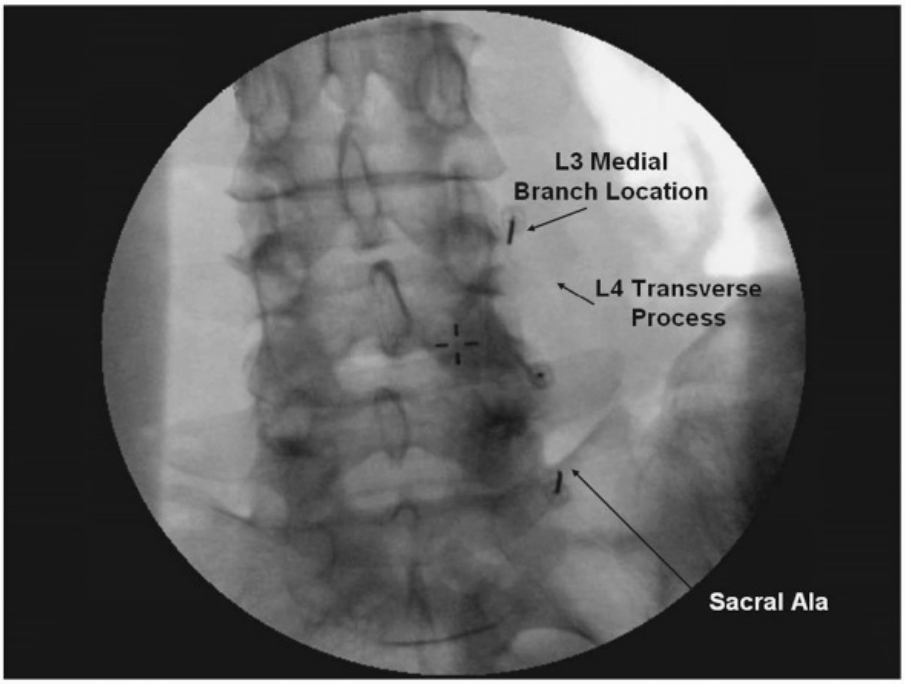


FIGURE 169.3. Placement of needle under fluoroscopic guidance for diagnostic (or therapeutic) medial branch blocks. Note that the L3 nerve root exits below the L3 transverse process and sends a medial branch (L3 MB) that runs along the junction of the superior articular process of the L4 vertebral body and the L4 transverse process.

MANAGEMENT

After the diagnosis of facet arthropathy has been made, patients are often offered therapeutic interventions with relatively long-term pain relief. Previously, this was achieved by intra-articular injection of steroids. With the advancement of radiofrequency technique, better results can be achieved by radiofrequency neurotomy (rhizotomy) or denervation (ablation) of corresponding medial branches. A detailed description of the mechanism of action of radiofrequency or pulsed-radiofrequency is beyond the scope of this chapter. The technique is similar to the diagnostic blocks, wherein radiofrequency probes are positioned at the specified sites of medial branches using 22-gauge spinal needles as introducers. After positive sensory testing (50 Hz) at <0.5 V and negative motor testing (contraction of ipsilateral paraspinal muscles but not lower extremity musculature) at 2 V, ablation is carried out for 90 to 120 seconds. Outcome results of radiofrequency techniques vary, depending on the inclusion criteria and level of blockade (Table 169.1). In general, radiofrequency procedure is relatively safe, gives a prolonged pain relief, and can be safely repeated with similar results.

TABLE 169.1 SELECTED OUTCOME STUDIES FOR RADIOFREQUENCY ABLATION OF MEDIAL BRANCHES FOR FACET ARTHROPATHY RELATED PAIN

AUTHOR (YEAR)	LEVEL TYPE	N[a]	DIAGNOSTIC BLOCK(S) THERAPY FOLLOW-UP	RESPONSE
Lord (1996)	Cervical Randomized, double-blind, placebo-controlled trial (rhizotomy vs. sham procedure)	24	Dual double-blind, placebo-controlled local anesthetic block *90 s RF lesion at 80°C* 12 mo	Median time before return of preprocedure pain = 263 d in study group (8 d in sham group).
Stolker (1993)	Thoracic Prospective study	36	Single local anesthetic block *RF lesion at thoracic MBs* 18-54 mo	After a median follow-up of 31 mo, 16 patients (44%) were pain free and 14 patients (39%) had more than 50% pain relief.
Van Kleef (1999)	Lumbar Randomized, double-blind, placebo-controlled trial (rhizotomy vs. sham procedure)	31	Single local anesthetic block *60 s RF lesion at 80° C* 12 mo	Statistically significant differences in effect on the VAS scores, global perceived effect, and the Oswestry disability scale. 3, 6, and 12 mo after treatment, there were significantly more success patients in the RF group compared with the sham group.
Dreyfuss (2000)	Lumbar Prospective audit	15	Double local anesthetic block *2 lesions, 90 s RF*	60% of the patients obtained at least 90% relief of pain at 12 mo while 87% obtained at least 60% relief.

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			lesion at 85°C 12 mo	

RF, radiofrequency; MB, medial branch; VAS, visual analog scale.

[a] Number of patients included in study.

TAKE HOME POINTS

- Facet arthropathy is a leading cause of spinal pain.
- Clinical and radiographic analysis helps little in its diagnosis.
- Diagnostic local anesthetic blocks are the only method to identify this disease and should be considered early, if the clinical picture (based on a rigorous epidemiologic evaluation) is suggestive.
- Therapeutic radiofrequency ablation of medical branches innervating the specific facet joint gives relatively long-term pain relief.

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When Evaluating Back Pain, Always Examine for Trigger Points Before Ordering Expensive Imaging Studies

Leena Mathew MD

A45-year-old patient presents to the pain clinic complaining of lower back pain and insisting that he needs to be “run through the CT scanner.” He was in his usual state of health until experiencing back pain 6 weeks ago when he spent a weekend planting trees at his mother-in-law's house (in other words, seriously overdoing it). Although the initial pain subsided after 2 weeks, he had recurrence of the same pain 2 weeks ago when he slipped and fell on the sidewalk. Since then, the pain has steadily escalated in intensity to its current level. It is a constant nonradicular pain that he describes as tightness and an aching sensation mostly localized in the left low back. There is no sensory or motor deficit associated with this. He denies any bowel or bladder dysfunction. He also denies any constitutional symptoms such as fever, night sweats, chills, and loss of weight or appetite. There is no increase of pain with Valsalva maneuver. Pain worsens with increased levels of activity, especially with lateral flexion, extension, and rotation at the lumbar spine. His physical exam was within normal limits, except for left paraspinal muscle spasm associated with a trigger point.

When a patient presents with low back pain, there is often immediate focus on obtaining imaging studies to establish a diagnosis. However, indiscriminate expensive imaging studies can add greatly to total health care expenses both in terms of the acute episode of back pain and in the long-term management of a chronic condition. A careful history and physical exam will usually exclude diagnoses that may require immediate operative treatment from those that can be safely and efficiently managed with other modalities. If imaging is sought, many practitioners advocate just a basic x-ray (if low back, flexion and extension are also recommended).

It is crucial to remember that myofascial pain is one of the most common reasons for low back pain. There is a primary or idiopathic etiology, as well as a secondary etiology, stemming from radiculopathy, disc or facet syndrome, dystonia, or postural imbalance such as a leg length discrepancy. The failure to recognize this can result in apparent treatment failures.

The presence of trigger points is the classic hallmark of myofascial pain. Because trigger points are not visualized on any imaging studies or laboratory tests, they are identified only by careful palpation. Trigger points may be appreciated as bands or knots within involved muscles. Palpation of trigger points P.749
produces pain concordant with the patients' usual pain. The pain from the trigger point is centrifugally referred in the affected muscle along the myofascial plane. The most common muscles affected are those in the neck, shoulder girdle, and low back.

Muscles are sprained when placed under constant stress. A sprain usually affects a few fibers in the body of the muscle. The initial inflammation settles within 1 to 2 weeks. If sensitization of the dorsal horn occurs at the corresponding level in the spinal cord, there is an increase in tone within the whole muscle. Chronically, the taut band remains as a painless “latent” trigger point. This makes the muscle vulnerable to further injury because the latent trigger point makes the muscle less pliable. In some patients, the sensitization leads to a self-perpetuating loop that keeps the trigger points active for many months after the original injury.

Treatment of myofascial pain syndrome varies. It includes trigger point injections, spray and stretch, transcutaneous electrical nerve stimulation, ultrasound therapy, dry needling, massage therapy, and elimination of causative and perpetuating factors. The major goal of trigger point therapy is to relieve both pain and the tightness of the involved muscles. An interesting feature of trigger point injections is that they are useful as both a diagnostic *and* a therapeutic tool.

Trigger point injections should always be done in conjunction with a physical therapy program that focuses on stretching, strengthening, and improving the range of motion. The mechanism of trigger point inactivation by injection is unknown. Simons and Travell suggested several possible mechanisms: (i) mechanical disruption of the trigger point, (ii) depolarization block of the nerve fibers by the released intracellular potassium, (iii) washout of the neurosensitizing inflammatory mediators by injected fluid or local hemorrhage, (iv) interruption of the central feedback mechanism, and (v) focal necrosis of the trigger point by the injected drug.

Trigger point injection is contraindicated for patients with a fear of needles, as well as for patients that are pregnant, exhibit signs of infection, have bleeding disorders, or have allergies to anesthetic agents. Although some practitioners like to document that aspirinlike pharmaceuticals have been avoided 1 week before trigger point injection, American Society of Regional Anesthesia currently does not require that the patient be off of aspirin for 1 week prior (in fact, it is likely that most of these patients will actually be on some anti-inflammatories for musculoskeletal pain).

Injecting the trigger points may provide the patient with symptom relief while the primary disease pathology is being addressed. It is a simple, low-risk procedure, which if done correctly decreases the need for aggressive
and expensive interventions. Myofascial trigger points are eliminated by infiltration with a local anesthetic, which in turn blocks the reflex mechanisms P.750
perpetuating the syndrome and allowing the taut muscle fibers to relax. This action provides relief of the referred pain syndrome.

Trigger point therapy begins with accurate identification of the point. After palpation, these points are marked. Using sterile preparation and technique throughout, the needle is introduced at a 30- to 60-degree angle, just beyond the dermis into the myofascial plane. When the myofascial plane is entered, a gritty sand paper sensation or crackling sound is appreciated. The length of the needle depends on the muscle being accessed. For superficial muscles, a 25- to 27-gauge 1-in. needle is used. For deeper muscles such as the gluteus maximus or quadratus lumborum, a 25-gauge 5-cm needle may be needed. The point is then injected with a local anesthetic with or without steroid. The local anesthetic injected may be 1% to 2% lidocaine or 0.125% to 0.5% bupivacaine. Although 0.5% bupivacaine is used by some practitioners, caution must be exercised when injecting over the site of major nerves. Remember that longer-acting local anesthetics are more mycotoxic. One milliliter of local anesthetic is sufficient at each trigger point because larger volumes decrease the specificity of the block and may be ultimately mycotoxic. Although multiple trigger points may be injected simultaneously, it must be kept in mind that the more points that are injected, the less specific the trigger point injection would be for diagnostic purposes. The most symptomatic points are targeted first. One to 10 trigger points can be injected at a visit. When multiple points are injected, care should be exercised not to exceed toxic doses of the local anesthetic. Although it is believed that the addition of steroids to the local anesthetic may increase the duration of analgesia and also act as a local anti-inflammatory agent, there is little evidence to support any beneficial effect of adding corticosteroid to the injection. If the practitioner so chooses, a total of 10 to 40 mg of triamcinolone or methylprednisolone or 2 to 8 mg of dexamethasone may be used in divided doses.

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Repeated trigger point injections with corticosteroids may cause local myofibrillar scarring and suppression of the pituitary adrenal axis, resulting in a poor long-term functional outcome. Epinephrine should never be used in the local anesthetic injectate mixture. The injection may be repeated in 1 to 2 weeks along with a program of physical therapy focusing on stretching, strengthening, and improving range of motion.

Some practitioners will place the needle in the marked point and "dry needle" it by moving the needle back and forth horizontally in the plane of the trigger point. Saline has also been used as an injectate. Most of the studies seem to suggest that the injection of saline or local anesthetic is not as important as the act of needling itself. There is a paucity of good studies stating superiority of one technique over the other. The efficacy of needling techniques compared to placebo is not supported or refuted by the evidence suggested from clinical trials.

Botox A injections may be considered in resistant cases after successful treatment with local anesthetic with or without steroid injection has been documented. In this event, trigger points are deactivated by injecting with botulinum toxin A (Botox A). Botox blocks motor nerve impulses from reaching the injected muscle fiber, producing pain relief because of intense relaxation. Botox binds irreversibly to specific receptors, producing muscle relaxation, which lasts up to 3 months. Muscle tone slowly returns as new receptors are produced.

Long-term outcomes of Botox use in myofascial pain syndrome have not been published. In fact, no study of myofascial, lumbar, or generalized neck pain have described more than two injections, with the second injection usually 3 months post the first injection. Long-term adverse effects, such as development of antibodies to the toxin or muscle weakness/atrophy, have not been described but remain a theoretical concern.

TAKE HOME POINTS

- The diagnosis of myofascial pain depends on careful history and physical exam to identify trigger points.
- Again, it is important to make sure that there are no radicular symptoms or red flags/constitutional symptoms prior to forgoing the "expensive imaging/workup." Many practitioners are advocating a basic x-ray (if low back, flexion and extension are also recommended). The thinking behind this is that a bone-involving process (i.e., a vertebral compression fracture) will have point tenderness, and this can often mimic a trigger point in presentation.
- Trigger points may represent a feedback loop involving dorsal horn cells in the spinal cord.
- Trigger point injections have *both* a diagnostic and a therapeutic value.
- The mechanism of trigger point deactivation by injection is not known, but because it is a simple, low-risk procedure, it should be considered when appropriate.
- Lidocaine or bupivacaine *without* epinephrine is typically used. Multiple trigger points can be injected per treatment, but exercise caution not to exceed the toxic dose.
- Addressing trigger points early may obviate the need for expensive imaging studies and restore early functionality.
- Botox A injections have been used in resistant cases. However, there are no long-term studies to date that examine outcomes pertaining to this practice.

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Title: ***Avoiding Common Anesthesia Errors, 1st Edition***

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Know the Complications of Epidural Corticosteroid Injections

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Epidural corticosteroid injections for back and radicular pain have been performed since the 1950s. Although these injections are generally considered safe, several important complications can occur.

Corticosteroids may be injected epidurally via the interlaminar, transforaminal, and caudal approaches. Common corticosteroids used include betamethasone, methylprednisolone, triamcinolone, and dexamethasone. These vary in potency, duration of action, preparation (solution or suspension), and additives (benzyl alcohol or polyethylene glycol). These factors may have a role in potential complications.

Most complications are relatively mild and include headache, vasovagal reactions, transient increase in pain, dural puncture, and systemic effects such as hyperglycemia. Serious complications are rare. Arachnoiditis is inflammation of the arachnoid mater, which leads to painful paresthesias and weakness. Infectious complications, such as meningitis or epidural abscess, can be devastating or fatal. Paralysis can also occur. Careful and thoughtful performance of epidural corticosteroid injections can reduce the rate of these complications.

ARACHNOIDITIS

Adhesive arachnoiditis can occur after intrathecal administration of corticosteroids. The cause of arachnoiditis is unknown, but particulate corticosteroids in suspension and additives used as preservatives have been implicated. Arachnoiditis can occur if a portion of an epidural corticosteroid injection is delivered to the intrathecal space. Negative aspiration, however, does not guarantee that inadvertent partial injection into the intrathecal space will be prevented.

Administration of a contrast agent under fluoroscopy before injection of a corticosteroid can localize the needle in the epidural space. Use of a microbore attachment ("pigtail") limits movement of the needle during syringe changes, thereby limiting the chance of needle migration into the intrathecal space. Attention must be paid to the contents of the epidural kit and injectant to ensure that they do not contain neurotoxic agents or preservatives. Antimicrobial skin preparations should be allowed to dry before needle placement because these agents may be neurotoxic.

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PARALYSIS

Paralysis after epidural corticosteroid injection can have immediate or late onset. In cases of immediate-onset paralysis, a vascular source is usually suspected, such as vascular trauma or embolism from intravascular injection. The vessels of particular concern are radicular arteries feeding the spinal cord and, at the cervical level, the ascending cervical, deep cervical, and vertebral arteries. The anterior two-thirds of the cord is supplied by the anterior spinal artery. At the cervical level, the radicular and segmental medullary arteries are fed by the ascending cervical and deep cervical arteries. A recent cadaveric study demonstrated that 22% of the posterior portion of the cervical vertebral foramina, once considered the "safe" location for needle placement, contained radicular cervical or segmental medullary arteries.

In the thoracic and lumbar spine, the anterior spinal artery is fed by radicular arteries from the lumbar and intercostal arteries. Of particular concern is the artery of Adamkiewicz, typically located between T5 and L2 on the left. Its origin has considerable anatomic variability. Radicular arteries enter the spinal column through the foramen, putting them at risk during transforaminal injections. Previous spine surgery appears to be a risk factor for anterior spinal cord infarctions associated with epidural injections.

To limit the possibility of intravascular injection, needle placement should be confirmed by administering contrast agent under fluoroscopic imaging. Digital subtraction angiography provides increased sensitivity in detecting intravascular injections. A test dose of local anesthetic can confirm intravascular needle placement by resulting in temporary paralysis, or, in cases of cervical injections, seizures. Use of a microbore attachment limits needle movement while syringes are changed between injections. A pencil-point spinal needle may decrease the risk of vascular cannulation. Corticosteroid solutions rather than particulate formulations may decrease the possibility of embolism during inadvertent intravascular injection. Cases of cervical anterior cord syndrome and cerebellar injury have occurred after cervical transforaminal epidural injections, despite needle placement using fluoroscopy and real-time computed tomography (CT). We recommend that such injections be performed only with extreme caution and after careful consideration of the risks and benefits.

Late onset paralysis may be due to several complications, including compression of the spinal cord from hematoma or epidural abscess; thrombosis due to vascular trauma may also be a source. A preprocedural history should be taken to exclude the use of anticoagulant or antiplatelet medication or an underlying bleeding disorder. Screening laboratory tests on all patients are not indicated. Minimizing needle manipulation in the epidural space further decreases the risk of vascular trauma. Because spinal cord compression can be such a devastating complication, it is important to keep a high index of suspicion for epidural hematoma and abscess. Magnetic resonance imaging (MRI) is becoming the gold standard for diagnosis of these lesions, although CT with or without myelography is also useful.

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INFECTIONS

Infections due to epidural injections are rare but include meningitis, epidural abscess, and soft tissue or skin infections. Risk factors appear to include remote extraspinal infections and an immunocompromised state, including that resulting from diabetes mellitus, chronic corticosteroid use, and cancer. Fig. 171.1 shows MRI findings for a patient in whom discitis developed after an intralaminar epidural corticosteroid injection.

Several precautions should minimize the risk of infection. Strict adherence to sterile technique is an absolute. Skin preparation with 10% povidone iodine was found to leave 35% of epidural needles contaminated with *Staphylococcus epidermidis* or *Staphylococcus aureus*; therefore, use of chlorhexidine should be strongly considered. Administration of antibiotics active against *S. aureus* and *S. epidermidis* to patients with risk factors has also been advocated. Needle passage should avoid superficial skin infections. The classic triad of fever, back pain or tenderness to palpation, and focal

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neurologic signs does not appear in all patients with epidural abscess. Most patients present with worsening back pain only, making a delay in diagnosis common. The clinician must maintain a high degree of suspicion in any patient complaining of back pain after an injection. Measurement of nonspecific markers of inflammation, such as sedimentation rate and C-reactive protein, are highly sensitive in identifying spinal infections. If epidural abscess is suspected, it is necessary to immediately perform MRI, CT, or CT myelography. These patients frequently need surgical intervention.



FIGURE 171.1. AT2 sagittal magnetic resonance imaging scan of the lumbar spine showing evidence of discitis after an intralaminar epidural corticosteroid injection at the L5/S1 interspace. The patient had a history of prior empyema and recurrent pulmonary infections

SYSTEMIC EFFECTS

Exposure to corticosteroids has both transient and long-term effects. Facial flushing and hyperglycemia have been reported after epidural corticosteroid injections. Long-term exposure to corticosteroids can result in more serious complications such as depression of the immune system, osteoporosis, and hypertension. Using the lowest dose that is likely to be effective should minimize systemic adverse effects. Corticosteroid injections should be limited to a reasonable number per patient per year.

TAKE HOME POINTS

- Epidural steroid injection is an established technique for the treatment of back and radicular pain. A variety of steroid agents and anatomic approaches are used.
- Mild complications include headache, vasovagal reactions, transient increase in pain, dural puncture, and systemic effects such as hyperglycemia.
- More serious complications include arachnoiditis, paralysis, infection, and the long-term systemic sequelae of steroid use.

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Chemical Neurolysis Has Good Efficacy in the Treatment of Intractable and Terminal Cancer Pain, But Precise Administration of the Appropriate Anesthetic and Lytic Agents is Imperative

Leena Mathew MD

Chemical neurolysis involves the administration of a chemical agent that destroys nociceptive neural structures to create long-lasting analgesia. Neurolytic blocks are most often done in the setting of intractable pain due to cancer and inoperable chronic illnesses with a short life expectancy.

These agents may be used at peripheral nerves, paravertebral somatic nerves, along the sympathetic chain, or at various epidural or intrathecal levels. The rationale for choosing an agent is based on patient positioning factors. Adiagnostic block using local anesthetic with amelioration of symptoms is essential prior to proceeding with neurolytic blocks. The success and duration of the block can vary anywhere from partial to excellent pain relief lasting from weeks to months, depending on the type of block, agent used, and the skill of the physician. The most common cause of an unsuccessful block is incorrect placement of the neurolytic agent.

Alcohol and phenol are the most commonly used neurolytic agents. Other agents, such as hypertonic saline, have also been tried with varying degrees of success.

PHENOL

Phenol is also known as carbolic acid. It is an aromatic compound with the chemical formula C6H5OH. It is comprised of a hydroxyl group bonded to a phenyl ring. It is prepared as a sterile, commercial-grade phenol in a maximum concentration of 6.7% solution in water. Although it has poor solubility in water, it is highly soluble in organic solvents such as alcohol and glycerol. The addition of small amounts of glycerol may increase its concentration to 15%. The shelf-life of phenol exceeds 1 year when the solution is refrigerated and not exposed to light. Phenol turns red on exposure to sunlight and air because of oxidation.

Phenol can be diluted with saline and is compatible when mixed with radiocontrast dye to allow fluoroscopic guidance during injection of the agent and to monitor spread of the solution. When mixed with glycerol, it slowly diffuses from the solution. With glycerol, phenol forms an extremely viscous hyperbaric solution from 4% to 10%. This is an advantage when injected intrathecally because it allows for limited spread localized in a small target area. For the injection of intrathecal phenol, the patient is placed in lateral decubitus position with the painful side down. When mixed with water, concentrations of 3% to 10% are possible. However, in this formulation, it is a more potent neurolytic with a wider spread.

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The choice of diluents depends on the extent of neurolysis desired. At low concentrations, phenol has local anesthetic properties (the use of higher concentrations of phenol also predisposes to a higher incidence of vascular injury). With increasing concentrations, phenol causes increasing neural damage. At concentrations >5%, there is protein coagulation with segmental demyelination and Wallerian degeneration. At 6%, nociceptive fibers are lysed. With higher concentration, axonal abnormalities and nerve root damage are seen. Concentrations <5% may only provide a sensory block. Some studies have shown that 5% phenol is equipotent to 40% alcohol. Degeneration takes 14 days, and regeneration is complete in 14 weeks. Effects of the block cannot be evaluated until after 24 to 48 hours, to allow time for the local anesthetic effect of phenol to dissipate. The neurolytic effect may be clinically evident only after 3 to 7 days. If inadequate pain relief is obtained after 2 weeks, this may indicate incomplete neurolysis and require repetition of the procedure.

The subsequent fibrosis that occurs following phenol injection makes nerve regeneration more difficult, but not impossible. Nerve regeneration can occur as long as the nerve cell body is intact, at a rate of 1 to 3 mm/day. Nerve arborization and neuroma formation can occur at the site of nerve disruption and can be a focus of a neuropathic type of pain.

Awedge pillow is used to help roll the patient 45 degrees posteriorly so the dorsal column of the effected side is most inferior, or the painful side down. The bevel should face down and the injection must be done slowly because of the high viscosity of the drug. Warming the phenol can make it easier to inject. Phenol has systemic side effects, including central nervous system (CNS) stimulation, cardiovascular depression, nausea, and vomiting. Systemic doses of >600 mg can cause convulsive seizures and CNS depression. Doses < 100 mg are less likely to cause serious side effects. Accidental intravascular injection causes tinnitus and flushing. Chronic toxicity can cause hepatic and renal damage. Phenol is metabolized in the liver by conjugation to glucuronides and oxidation to equinol compounds or to carbon dioxide and water. Excretion of metabolites is via the kidneys.

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ALCOHOL

Ethyl alcohol has a destructive effect similar to phenol. In 1933, Labat and Greene noted that 33.3% alcohol injection produced satisfactory analgesia in the treatment of painful disorders. Alcohol is primarily used for intrathecal neurolysis, sympathetic blockade, celiac plexus block, and chemical hypophysectomy for patients with diffuse pain secondary to metastatic disease.

Its mechanism of nerve destruction is similar to phenol. Alcohol causes dehydration; extraction of phospholipids, cholesterol, and cerebrosides; and precipitation ofmucoprotein and lipoprotein. This causes sclerosis, demyelination, and Wallerian degeneration of nociceptive structures.

Alcohol is available commercially as a 95% solution. Although 50% to 100% alcohol is used as a neurolytic agent, the minimum concentration required for neurolysis has not been established. Alocal anesthetic is commonly used as a diluent. Following its injection, the patient complains of severe burning pain along the nerve's distribution, which may last for a minute and is subsequently replaced by a warm, numb sensation.

It is hypobaric relative to cerebrospinal fluid. The patient is kept in the lateral decubitus position with the painful side up, and the patient is rolled forward by 45 degrees so the dorsal horn of the effected side is the most superior part of the spinal column. Atuberculin syringe is then used to inject alcohol in 0.1-mL increments. Each segment needs at least 0.3 mL per segment, is readily soluble in body tissues, and produces severe burning pain on injection. A

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maximum volume of 0.7 mL can be used at one session for intrathecal neurolysis. For the lysis of the sympathetic chain, more dilute concentration may be used, but larger volumes may be needed and up to 20 cc may be used.

It spreads quite rapidly from the injection site and requires 12 to 24 hours before the effects of the injection can be assessed. Similar to phenol, if inadequate pain relief is achieved after 2 weeks, the block should be repeated.

Aconcentration of 95% will reliably lyse sympathetic, sensory, and motor components of the nerve. Ninety to 98% of the injected alcohol is rapidly metabolized by oxidation in the liver principally by the enzyme alcohol dehydrogenase.

HYPERTONIC SALINE

The use of hypertonic saline by intrathecal injection to treat intractable pain was first reported by Hitchcock in 1967. The most commonly used solution is the 10% aqueous solution, and this is available as a pharmaceutical preparation. The mechanism of neurolysis is not well elaborated. It causes severe pain on injection, and local anesthetic is first injected before the saline solution. When administered intrathecally, hypertonic saline can cause an increase in intracranial pressure, increase in blood pressure, heart rate, and respiratory rate.

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MISCELLANEOUS AGENTS

In the past, other agents have been used to promote neurolysis, including ammonium salt solutions, chlorocresol, and distilled water. However, their neurolytic effect is unpredictable, and their use is now relegated to history. Butyl aminobenzoate (Butamben) is a substance that has been reported to cause neurolysis and is under investigation.

INDICATIONS FOR CHRONIC NEUROLYSIS

- Management of chronic, intractable, nonterminal pain that is not responsive to other modalities
- Treatment of pain in patients who have short life expectancy (<1 year)
- Alternative management to treat spasticity in order to improve balance, gait, self-care, and global rehabilitation

SELECTION CRITERIA FOR INTRATHECAL NEUROLYSIS

- Well-established diagnosis
- Life expectancy: <12 months
- No response to conservative therapies
- Pain localized to two to three dermatomes
- Somatic pain
- Unilateral symptoms—if bilateral symptoms exist, then the intrathecal block should be staggered

Psychological evaluation is also important in completely evaluating the patient before neurolysis is considered. Extensive communication with the patient is necessary, and the risks and benefits of the procedure should be explained before proceeding. Understanding and acceptance of complications by the patient and the family is essential.

An important difference between neurolytic blocks for pain relief versus spasticity is that motor or mixed nerves are targeted preferentially in the management of spastic disorders.

COMPLICATIONS OF CHEMICAL NEUROLYSIS

Skin and Other Nontarget Tissue Necrosis and Sloughing.

This is due to damage of the vascular supply to the skin, causing ischemia, and chronic trauma to denervated tissue. Necrosis of muscles, blood vessels, and other soft tissues has also been reported. Flush the needle with 0.2 mL of air prior to removal.

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Neuritis.

Partial destruction of the somatic nerve followed by its regeneration is responsible for neuritis. This complication has been reported as frequently as up to 10%. Neuritis is less likely to occur with a subarachnoid or ganglion neurolytic block. The symptoms of neuritis present as hyperesthesia and dysesthesia. Some patients may perceive the postneurolytic neuritis to be worse than the original pain problem. It is one of the limiting factors in the use of chemical neurolysis.

Anesthesia Dolorosa.

Pain may persist in an area of anesthesia. The symptom is a poorly understood but well-established complication that patients must be warned of prior to proceeding with neurolytic blocks. It is probably a result of an imbalance in afferent input and the resultant central nervous system changes. Alocal anesthetic block done a few hours prior to the performance of the neurolytic block seems to prevent the development of this complication. The pain from this condition may be treated by the adjuvant analgesics such as tricyclic antidepressants, membrane stabilizing agents, and anticonvulsants.

Paralysis.

This is the most feared major complication. It occurs infrequently and is usually temporary.

Perineal and Sexual Dysfunction.

About 1.4% and 0.2% of patients will have bowel or bladder dysfunction at 1 week and 1 month, respectively. Usually, there is return of function.

Systemic Complications.

Hypotension occurs secondary to vasodilatation after a sympathetic block. Thrombosis of vascular structures can occur if injected into or around major

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vessels. Hypertonic saline when administered intrathecally, causes an increase in the intracranial pressure, hypertension, tachycardia, and tachypnea.

TAKE HOME POINTS

- Chemical neurolysis is considered in patients with intractable pain refractory to other therapies.
- It is a useful analgesic modality in terminally ill patients with a life expectancy of < 1 year.
- Alcohol and phenol are the most commonly used agents for neurolysis. The choice of agent depends largely on patient positioning factors.
- Prior to performing a neurolytic block, a diagnostic block should be done to demonstrate efficacy and symptomatic improvement.
- Phenol is hyperbaric, and alcohol is hypobaric.
- Phenol has intrinsic local anesthetic properties that render it painless during injection, unlike alcohol, which burns during injection.
- The side effects can be varied and serious, ranging from skin necrosis and sloughing, vascular thrombosis, hypotension, neuritis to paraplegia, and loss of bowel and bladder function.

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Understanding the Human Factor

F. Jacob Seagull PhD

Have you ever accidentally poured orange juice in your cereal instead of milk? Have you ever tried to drive from work to the store and ended up driving home by accident? Most likely you have. Have you ever accidentally put sugar into your shoes instead of talcum powder? Probably not. Why not? Because “mistakes” do not happen randomly when humans are involved—they occur in systematic patterns. Some are likely, some are not.

Understanding how people think allows us to understand the types of mistakes that they might make and the types that they are unlikely to make. The study of how people act in the context of work—including the mistakes that they make—is known as “human factors.”

Applying the principles of “human factors psychology” to the domain of anesthesia can create an environment in which a serious mistake is much less likely to occur. Giving the wrong drug should be like putting sugar in your shoes, not like putting juice in your cornflakes. Anesthesia has been at the forefront of human factors in the medical domain for a number of years, adopting safety initiatives, making continuous process improvements, and attaining advances that significantly reduced the likelihood of adverse events. Anesthesia has been singled out by the Institute of Medicine as the medical discipline that can serve as a model of “safety culture.”

The concept of human factors has its roots in aviation. It was first used by the U.S. Air Force to help push human performance to the limit in order to beat the enemy. The fighter pilots in World War II were surrounded by advanced cockpit technology in life or death situations—not entirely unlike an anesthesiologist in an operating room. By designing the pilots' environment to support the task at hand, human factors helped give them the edge they needed.

The field of human factors is multidisciplinary, encompassing domains from psychology to engineering to anthropology to computer science and more. Many different aspects of human activity relevant to anesthesiology fall under the scope of human factors. A few of the more relevant aspects are described briefly in this chapter.

DECISION MAKING

People make decisions in a very different manner than computers. Instead of using precise calculations, people often use heuristics, or rules of thumb, to decide what to do. Heuristics can be a powerful tool that lets a person make quick decisions when relevant information is incomplete or unavailable. P.767
Unfortunately, there are times when using heuristics may lead to systematic biases in decision making, or put more simply, can make a mistake more likely. Human factors psychology has examined a wide array of decision-making aspects, including heuristics and biases. Through the application of human factors principles, one can remediate many common biases, leading to better, safer decisions in critical anesthesia situations.

TECHNOLOGY DESIGN AND COGNITIVE ERGONOMICS

In a fighter jet, well-designed avionics displays can mean the difference between a kill and a crash. In anesthesia, clear monitoring displays, easy-to-program infusion pumps, and clearly identifiable syringes and drugs can have a similar effect. Technology designed with human factors' “cognitive ergonomics” considerations can reduce the likelihood of making an error and improve the odds of recovering from adverse events.

COMMUNICATION

Human perceptual abilities have their limits and weaknesses. Whether interacting with a piece of technology or with another human, people must take in and make sense of information in the environment and convert the perceptual signals into concepts and knowledge about the world. Application of the principles of human factors can cater to human perception's strengths and compensate for perceptual weaknesses, facilitating clear, concise, and error free communication.

ENVIRONMENTAL DESIGN

It is easier to place a central line in a quiet, calm, well-lit room than in the shadow of a noisy code team during an emergency resuscitation. Environmental factors such as noise, lighting, and work space layout can influence the margin of safety in which care is provided.

COMPLEX SYSTEMS

Each individual artifact with which we interact undoubtedly influences the ultimate success or failure of our endeavors. However, beyond these individual influences, there is an overarching interaction among the multitude of systems, artifacts, and people that can have an influence greater than the sum of the parts. Ultimately, it is most often a system failure, and not a failure in the function or design of an individual artifact, that leads to catastrophic failure and adverse events in anesthesia. Human factors addresses issues arising from the complexity of highly coupled technical systems, and can help offer effective system design, or even impart effective strategies for interacting with flawed systems.

Understanding some key aspects of human factors can provide you with points of leverage in critical situations, and perhaps more importantly, can help you prepare yourself properly to avoid critical situations before they arise. The chapters in this unit touch on a small selection of relevant topic areas where consideration of human factors can contribute to safe and effective practice of anesthesia care. P.768

TAKE HOME POINTS

- Mistakes do not happen randomly but rather in systematic patterns.
- The Institute of Medicine paper that singled out anesthesia as the medical discipline that can serve as a model of “safety culture” is an important document that is now driving much of the pay-for-performance/quality movement.
- Anesthesia providers at all levels will increasingly be called on to understand, master, and integrate the basic principles of human factors

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psychology into the delivery of anesthetic care.

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Editors: Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.
Title: *Avoiding Common Anesthesia Errors, 1st Edition*

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Learn From the Pilots to Minimize Errors in Anesthesia Management: Recognition and Prevention

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Crew resource management (CRM), also known as crisis resource management, emphasizes a team approach to managing a situation in which the role of human factors is considered. Included in the CRM model are the effects of fatigue, predictable perceptual errors (e.g., misreading monitors or mishearing instructions), and the effects of various management and organizational styles in high-stress, high-risk environments. Originally developed in the aviation industry, CRM training fosters a unified environment with effective communication in which junior personnel are unconstrained about alerting others when something is amiss. If an error occurs, the goal of the team is to capture the error before it progresses and causes an adverse event.[1]

APPLICATION OF AVIATION-ADOPTED CREW RESOURCE MANAGEMENT TO ANESTHESIA PRACTICE

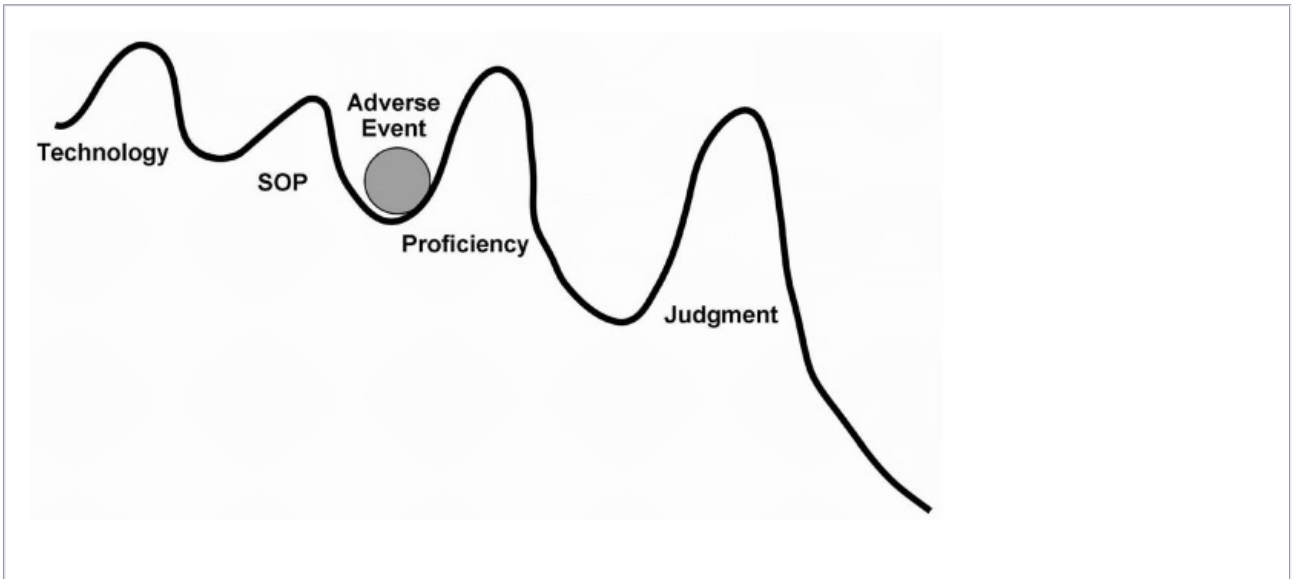
The practice of anesthesiology and the operation of commercial aircraft share some key characteristics: interaction with team members, continuous monitoring, and standard operating procedures (SOPs). The aviation industry has taken great strides in enhancing safety by adopting and implementing CRM in the 1980s. Likewise, these safety procedures captured interest in the anesthesia community as a method for decreasing risk to patients.

In 1978, a commercial airliner experienced a minor landing gear indicator malfunction, which required the crew to troubleshoot the problem before landing. The aircraft was low on fuel, and even with the installation of new digital fuel gauges and warnings, the three-person crew did not realize the low fuel situation until the engines began to flame out. The flight circled the airport until the plane ran out of fuel and crashed. The experienced crew was focused on resolving the landing gear problem, did not coordinate their actions, and did not identify another developing adverse event, even in the presence of modern technology.

The widespread adoption of CRM principles can decrease adverse events in aviation and in the perioperative setting. Adverse events are rarely caused by a single factor and typically occur when a number of conditions are met. According to Rampersad and Rampersad, adverse events often occur along the following trajectory of adversity: P.770

1. A catalyst event (e.g., landing gear malfunction vs. patient oxyhemoglobin desaturation).
2. A system fault (e.g., failure of the new digital fuel gauges to alert the crew to a low fuel situation vs. failure of the pulse oximeter alarm to alert the anesthesiologist of the desaturation).
3. Loss of situational awareness (e.g., cockpit crew was focused on the landing gear problem vs. anesthesiologist preoccupied with charting and does not recognize the desaturation).
4. Human error (e.g., lack of recognition of the low fuel situation vs. lack of continuous monitoring of the vital signs by the anesthesiologist).

Barriers to adverse events that exist in the operating room include technology, SOPs, proficiency, and judgment. An adverse event can be thought of as a ball rolling down a hill. Technology, SOP, proficiency, and judgment represent barriers to stop the ball (Fig. 174.1). At any time, the ball can be stopped by recognition and resolution of the event by one of the four barriers. If the ball continues to roll past the barriers, an adverse event occurs. The angle of the hill (steepness) can also be modified. Anesthesiologist fatigue increases the slope of the hill, allowing the ball to more easily pass through the four barriers. Higher patient acuity, such as elevated American Society of Anesthesiologists physical status, similarly increases the slope because the higher acuity of disease makes these patients less able to tolerate errors. P.771



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FIGURE 174.1. Dynamic error model. The ball represents an adverse event. Any one of the four barriers (peaks) can prevent the ball from rolling down the hill. If the ball rolls over all four hills, an adverse event occurs. (From Rampersad C, Rampersad SE. Crew resource management for people who don't fly the airplane. Presented at the Society for Pediatric Anesthesia winter meeting; Fort Myers, Fla.; February 17, 2006. Available at: http://www.pedsanesthesia.org/meetings/2006winter/pdfs/Friday_Rampersad_Crew%20Resources.pdf. Accessed March 30, 2007. Used with permission.)

Certain behaviors in the operating room increase risk to patients. According to Helmreich, failures of CRM can be divided into four categories: communication, leadership, conflict, and vigilance.

Communication

- Failure to inform the team of the patient's problem (e.g., surgeon fails to inform anesthesiologist of surgical bleeding before a decrease in blood pressure is observed).
- Failure to discuss alternative procedures (e.g., patient is high risk for the selected procedure, and an alternative, less-invasive approach could be performed).

Leadership

- Failure to establish leadership in the operating room team (e.g., anesthesiologist does not direct the actions of other team members during an intraoperative cardiac arrest).

Conflict

- Overt hostility and frustration (e.g., patient deteriorates while surgeon and anesthesiologist argue over who is responsible for newly developed tension pneumothorax).

Vigilance

- Failure to monitor situation and other team member activities (e.g., distracted anesthesiologist fails to detect decrease in blood pressure after a monitor power failure; anesthesiologist treats hypertension while the transducer is “sitting” on the floor).
- Failure to plan for lapsed vigilance (e.g., anesthesiologist does not have appropriate resuscitation drugs ready).

CRM applied to the anesthesia and operating room team encompasses the effective integration and practice of these components. The practice of anesthesiology is amenable to improvement, and many elements of safety enhancements in the aviation industry are applicable. Integration of CRM practices in every operating room is vital to ensure prevention and effective management of adverse events.

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Editors: **Marcucci, Catherine; Cohen, Norman A.; Metro, David G.; Kirsch, Jeffrey R.**
Title: ***Avoiding Common Anesthesia Errors, 1st Edition***

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In a Noisy Operating Room, Use a "Spelling Alphabet" to Communicate the Patient's Name or Check in Blood Products

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You are about to deliver anesthesia in a chaotic emergency situation. The operating room (OR) desk paged you in the cafeteria to tell you that a patient is coming over from the floor with a carotid blowout. The surgical intern is riding on the stretcher to apply pressure to the bleeder. You are told that there are blood products in the blood bank but that you have to call down there stat and let them know what to pack in the cooler. The problem is, you don't know the name of the patient. You ask the person who is on the OR desk to identify the patient, but due to background noise in both the OR and the cafeteria, you cannot catch the name she says. It might be Atkins, it might be Adkins, or it might be Adams. You just can't hear. You ask several times, each time a little louder. She replies, each time a little louder. You still can't hear the name, and you feel yourself getting tachycardic. Suddenly, on the telephone, you hear the calm voice of your CRNA colleague (who served as a U.S. Air Force medic in Vietnam), saying slowly, "The patient is Alpha-Delta-Kilo-India-November-Sierra." Armed with the correct information, you place the appropriate call to the blood bank. Mr. Adkins does fine.

Spelling alphabets are also commonly called radio alphabets, telegraph alphabets, or police letters. There are a number of different versions, but each uses a single phonetically distinct word to replace the name of the letter with which it starts. Spelling alphabets have their origins deep in military and communications history, and have been used at least since the beginning of the 20th century. They arose because early aviation and military radios were plagued by static. Because so many letters in the English language are phonetically similar (e.g., *B, C, E, T, D, P*), a burst of static would have made it impossible to accurately transmit letter-based information. Thus, in World War II, the sequence *B-E-D* would have been "called" as Baker-Echo-Dog. Spelling alphabets are used in at least 30 languages (from Finnish to Urdu), and there is a numeric code as well. Spelling alphabets are often called phonetic alphabets, but this is actually a misnomer. Phonetic alphabets are written representations of spoken sound as opposed to spoken representations of written symbols.

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ORs are noisy places. Decibel levels have been measured under a variety of situations and for different types of procedures. The "average" noise in a routine case will approach 70 to 80 db. In certain orthopedic procedures, such as total joint replacement, noise levels are routinely 110 db, which is the same level of noise in some boiler rooms, and will intermittently approach 140 db, which is high enough to put the surgeons (and other OR staff) at risk for occupationally associated hearing loss.

Operating noise is known to have detrimental effects on anesthesia providers. In a study by Murthy et al., when anesthesia residents were exposed to a 90-minute cassette of OR noise at 77 db, they had lower scores on tests for mental efficiency and short-term memory. More important, the same noise level significantly affected both speech reception threshold and speech discrimination, which led researchers to conclude that there is a marked decrease in the ability to discriminate spoken words at ambient OR noise levels. The use of a spelling alphabet may, therefore, have the same efficacy for anesthesia providers as for the military and pilots.

TABLE 175.1 NATO SPELLING ALPHABET

A	ALPHA
A	Alpha
B	Bravo
C	Charlie
D	Delta
E	Echo
F	Foxtrot
G	Golf
H	Hotel
I	India

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J	Juliet
K	Kilo
L	Lima
M	Mike
N	November
O	Oscar
P	Papa
Q	Quebec
R	Romeo
S	Sierra
T	Tango
U	Uniform
V	Victor
W	Whiskey
X	X-ray
Y	Yankee
Z	Zulu

The spelling alphabet used in World War II was modernized in the 1950s, as military pilots returned to their civilian careers. The most commonly used alphabet today is known as the international radiotelephony or NATO spelling alphabet (Table 175.1). It is simple to memorize and easy to use. P.775

TAKE HOME POINTS

- The noisy OR introduces the possibility of verbal miscommunication and is a cause of errors and breaches in patient safety.
- The spelling alphabet is a time-tested method of more accurately transmitting letter-based information.
- If you ever check in blood with a veteran (especially one who has been in a combat area), he or she will use it automatically.

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