# Handbook of Pediatric Orthopedics

Paul D. Sponseller

Second Edition



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## Second Edition

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To all whom I have been privileged to teach in the combined mission of caring for growing children: You have been a source of continuous instruction.

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# Foreword

This revised second edition of the *Handbook of Pediatric Orthopedics* is densely packed with vital information about pediatric orthopedics yet is presented in a manner that makes it a useful study guide as well as a valuable reference. Dr. Sponseller has put together a handbook–this word in the title is fitting–that every student and resident rotating on a pediatric orthopedic surgery service should have in their lab coat pocket at all times. In addition it should come out at night to prepare for the following day's clinic and surgical cases. Read it and use it, and you will have a solid foundation in pediatric orthopedics.

This book uniquely houses the essentials of pediatric orthopedics and is primarily targetted at the novice. The text begins with the basic development of the musculoskeletal system and progresses to the common orthopedic pathologies that effect children. The book is relevantly referenced and is filled with the facts, graphs, charts, and tables that are so frequently a part of everyday pediatric orthopedic practice. For example, the evaluation of lower extremity growth is accompanied by the contribution of growth from each of the lower limb growth plates as well as the methods and graphs to calculate growth remaining. These are critical values in planning for corrective procedures, and the several accepted methods for doing so are clearly presented and easy to comprehend. This is but one example of the method of presenting the critical data in an easily usable format. For the more common conditions, the details are present that will allow a reasonable understanding, while for the more obscure pathologies, the prose is appropriately brief or absent. Common things being common, this book contains what everyone who cares for (or plans to care for) children needs to know.

There are several groups of readers who will find the information contained in the *Handbook of Pediatric Orthopedics* particularly helpful. Those who are making their very first entry into the world of pediatric orthopedics often find our specialty initially overwhelming. It is either seen as orthopedics with growth factored into the equation and a myriad of syndromes, or else as pediatrics with too many bones to remember and x-rays that are daunting to interpret. Whether coming from either of these two perspectives, this handbook provides a source of information that is easily digestible and that is certain to quickly provide calm and order. It is just right for the rotating medical student and equally appropriate for residents starting

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their initial pediatric orthopedic experience. It also remains a resource for those beyond the introductory phase to the specialty, potentially for years to come due to the key sources of classification systems, scoring schemes, and graphical data particularly related to skeletal growth. As such, fellows in pediatric orthopedics may also want to refer back to this text at various times in the year (and beyond).

Be ready for rounds, for clinic, for your attending's questions, and most importantly for what you need to know in order to care for your patients on the pediatric orthopedic service. Read this handbook and keep it close by. There is little doubt it will serve you well.

Congratulations and thank you to Dr. Sponseller for bringing us the second edition of this valuable, straightforward, and concise *Handbook of Pediatric Orthopedics*.

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# Preface

The practice of pediatric orthopedics relies heavily on knowledge of principles and on technical skills. Nevertheless, there is also a need for the use of data and standards. In the process of teaching and working with professionals of all levels of training in children's orthopedics, I have tried to compile information that is essential for the practice of our specialty. I gathered these essentials into a practical handbook that is complete enough to be useful. The book is not meant to provide definitive coverage of individual problems or theoretical principles. Rather, it is intended to provide factual information to which we must commonly refer.

The first chapter, Anatomy and Normal Development in Children, gives norms for osseous development, motor development, innervation, and growth patterns and predictions. Chapter 2, Disorders of Skeletal Growth and Development, follows and provides radiographic parameters and clinical standards for treatment. Chapter 3, Skeletal Syndromes and Systemic Disorders in Pediatric Orthopedics, is included because many of these conditions are rare and not immediately familiar to the orthopedist; an overview is given with references at the end. Chapter 4, Neuromuscular Disorders in Pediatric Orthopedics, covers the basics of many of these unusual conditions. After an important chapter on Pediatric Trauma (Chapter 5), Chapter 6, Normal Values and Medications used in pediatric orthopedics, presupposes a knowledge of their pharmacology but gives dosages based on weight as well as their route and frequency. Normal laboratory values for children are listed by age. Last, Chapter 7, Common Procedures, including blocks, traction, special casts, aspiration, and arthrograms, is meant to provide technical information that is not readily available elsewhere.

It is my hope that this handbook will be a practical aid in the day-to-day practice of pediatric orthopedics for those at all levels of experience. I will be pleased to receive any comments or suggestions for the eventual third edition at psponse@jhmi.edu.

## ◆ Acknowledgments

I thank those I have been privileged to help train for making me constantly examine what might otherwise be taken for granted. I also thank my family—Amy, Matt, and Nina—for their inspiration. The careful editing of Sara Cleary, Elaine Henze, Kay D. Conerly, and J. Owen Zurhellen IV and the clear, concise drawings of Hong Cui, MD, have made this book possible.

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# 1 Anatomy and Normal Development in Children

Knowledge of normal growth and development is important for evaluation and diagnosis. This chapter contains relevant anatomy and developmental norms. It also contains a description of normal gait and guidelines for interpreting a gait study.

## ◆ Neurodevelopmental Norms

When evaluating a patient at risk of developmental delay, it is helpful to have norms to determine whether a delay is present. The section below presents the chronological appearance of certain key motor, social, and language skills. The Denver II Developmental Screening Test displays these in graphic form. Although the orthopedic surgeon will not perform the test formally, it provides an excellent summary of developmental milestones. **Table 1.1** provides the norms for milestones.

Information on the Denver II Developmental Screening Test is available from the following:

Denver Developmental Materials Inc.
PO Box 371075
Denver, CO 80237
E-mail: generalinfo@denverii.com

The Denver II Developmental Screening Test can be used with children from birth to age 6 years. It is used to decide which children should be

**Table 1.1** Norms for Motor Milestones

| Skill | Mean Age (mo) | St. Dev. |
|---|---|---|
| Roll from back to stomach | 3.6 | 1.4 |
| Roll from stomach to back | 4.8 | 1.4 |
| Sit tailor-style | 5.3 | 1.0 |
| Sit unsupported | 6.3 | 1.2 |
| Crawl (many never crawl) | 7.8 | 1.7 |
| Pull to stand | 8.1 | 1.6 |
| Cruise | 8.8 | 1.7 |
| Walk | 11.7 | 2 |
| Run | 15 | 3 |

*Source*: Palmer F, Capute A. Keys to developmental assessment. In McMillan J, ed. Oski's Pediatrics, 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2006:789

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referred for diagnostic evaluation. It is not an IQ test. It has four sections: personal–social, fine motor, language, and gross motor. The Denver II scoring chart provides an excellent summary of motor development.

## Psychomotor Skills in Children During Years 1 Through 5

- Neonatal period (first month)
  Supine: Generally flexed and tone a little low
- 2 Months
  Prone: Head sustained in plane of body in ventral suspension
  Social: Smiles on social contact
- 4 Months
  Supine: Reaches and grasps objects and brings them to mouth
  Sitting: No head lag on pull to sitting position
- 4 to 6 Months
  Prone: Rolls over to supine
  Semantics: Turns to his or her own name
- 7 Months
  Sitting: Sits briefly with support of pelvis
  Adaptive: Transfers objects from hand to hand
- 10 Months
  Standing: Pulls to standing position
  Motor: Creeps or crawls
- 12 to 18 Months
  Syntax: Speech generally consists of single-word utterances (comment)
- 12 Months
  Motor: Walks with one hand held, "cruises" or walks holding on to furniture
  Language: Two "words" besides mama and dada
- 15 Months
  Motor: Walks alone, crawls up stairs
- 18 Months
  Motor: Runs stiffly
  Social: Feeds self
- 24 Months
  Motor: Opens doors
  Syntax: Uses two- and three-word combinations (telegraphic speech)
- 30 Months
  Motor: Jumps
- 36 Months
  Motor: Goes up stairs alternating feet, stands momentarily on one foot

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- 48 Months
  Motor: Hops on one foot, throws ball overhand
- 60 Months
  Motor: Skips

## Bibliography

Richter SB, Howard BJ, Sturner R. Normal infant and childhood development. In McMillan J, ed. Oski's Pediatrics. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2006:593–601

### Referral Criteria

Referral should be made if the infant is displaying any of the following:

1. Not rolling by 6 months
2. Not sitting independently by 8 months
3. Handedness develops too early (by 12 months): May indicate abnormality of opposite side
4. Not walking by 18 months
5. No words by 14 months

## ◆ Neurologic Anatomy

### Sensation

Knowledge of dermatomes will help in the evaluation of neurologic conditions. There is, however, some variation and overlap between levels. Injury to a single nerve root may not produce complete loss of sensation within a dermatome. Sensation should be recorded as increased, decreased, absent, or dysesthetic. The sensation of proprioception and vibration is carried in the dorsal column of the spinal cord: light touch in the ventral spinothalamic tract and pain and temperature in the lateral spinothalamic tract. During neurologic root recovery, pain sensation returns before light touch (**Fig. 1.1**).

### Upper Extremity Motor Examination

Injury to the roots or the cord of the cervical spine follows certain patterns. Even though most muscles have innervation from multiple segments, each root has specific muscles and sensory regions for which it is critical. The diagram below is helpful for diagnosing cervical root lesions and spinal cord injury. Motor testing can be performed in one coordinated sequence, from proximal to distal: deltoid (C5), biceps (C5), wrist extension (C6),

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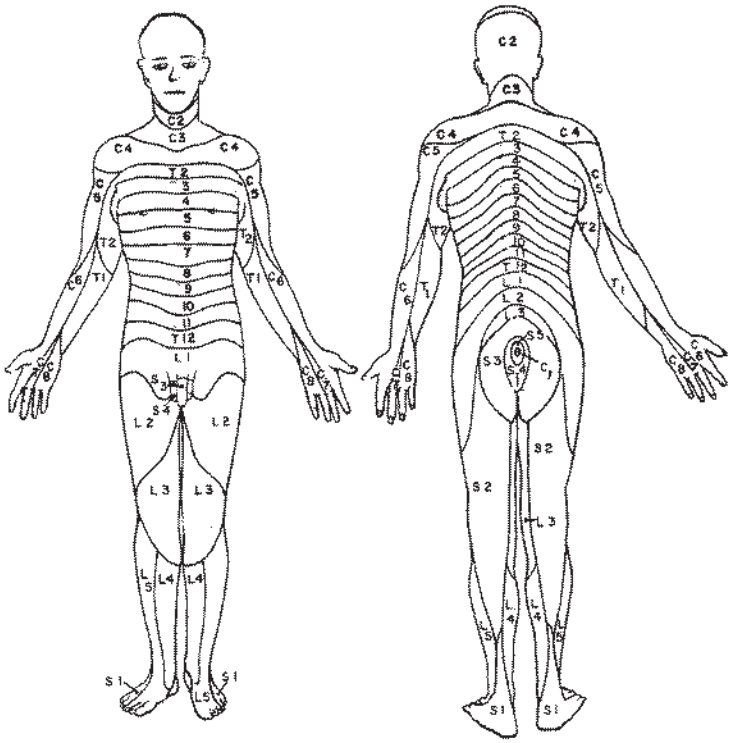


**Fig. 1.1** Dermatomes.

finger extension (C7), finger flexion (C8), finger abduction and adduction (T1) (**Fig. 1.2**).

## Upper Extremity Muscle Innervation

Because most muscles are innervated by multiple segments, it is necessary to know all the roots controlling a given muscle. **Fig. 1.3** indicates the roots contributing to a given muscle in the upper extremity. For purposes of strength grading, the following five-grade scale has been widely used:

- Grade 1: Flicker
- Grade 2: Less than antigravity
- Grade 3: Maintains position against gravity
- Grade 4: Moves against submaximal resistance
- Grade 5: Full strength

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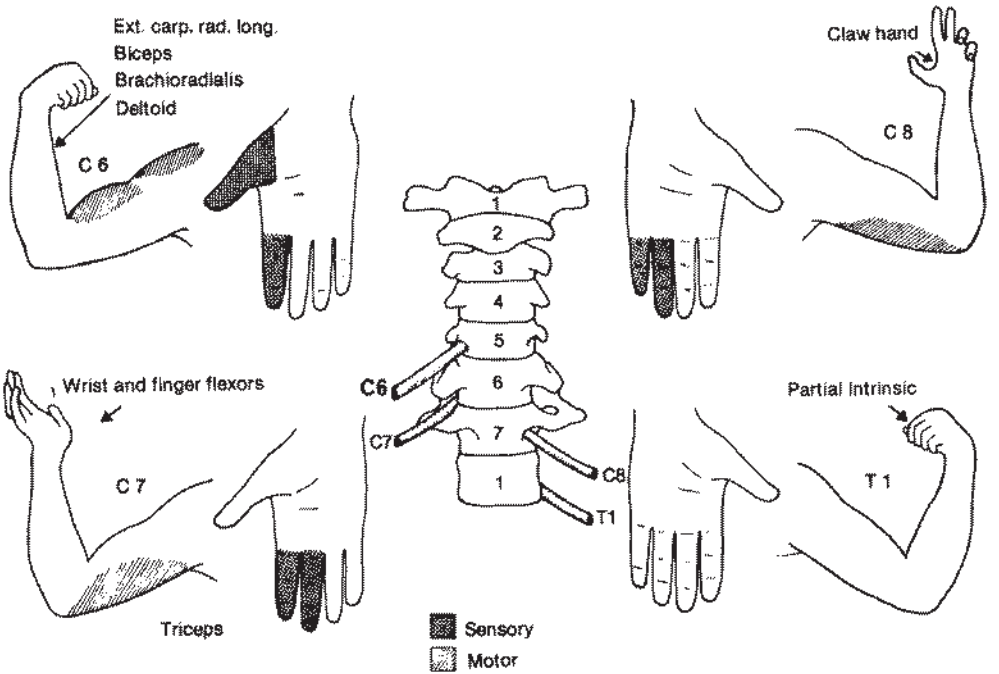



**Fig. 1.2** Sensory and motor innervation C6 to T1. *Note:* Only sensory loss is shown in the shaded hand, and only muscle involvement is shown in the arm. (From McQueen JD, Khan MI. Neurologic evaluation. In Sherk HH, Dunn EJ, Eismont FJ, et al, eds. The Cervical Spine. 2nd ed. Philadelphia: J.B. Lippincott; 1989: 206 (Fig. 4-5). Reprinted with permission.)



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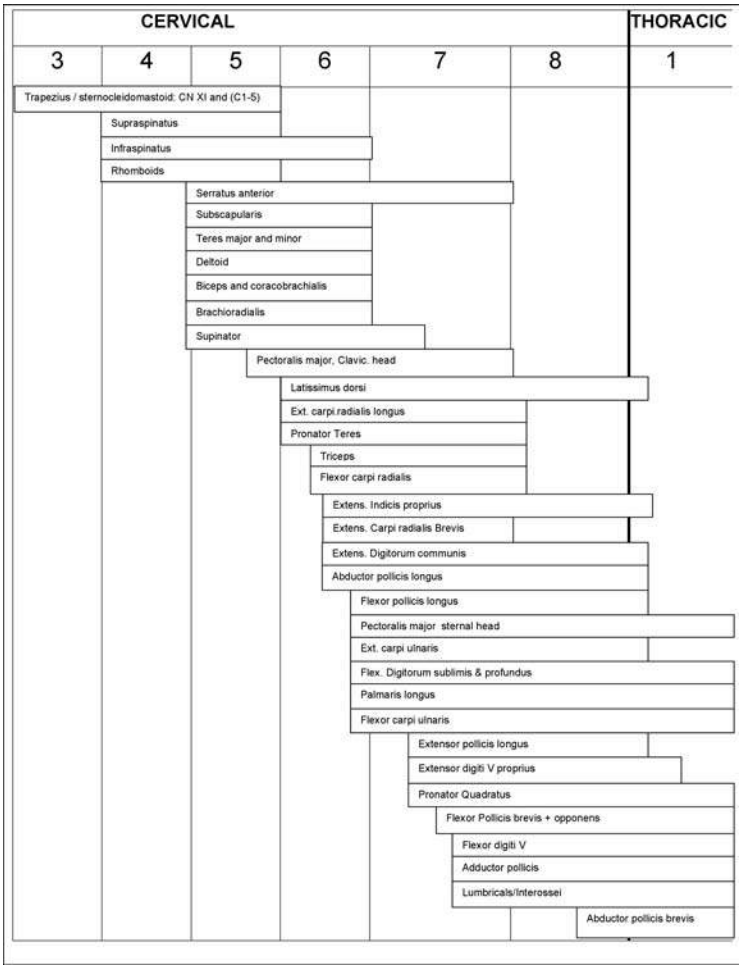



**Fig. 1.3** Innervation of muscles of upper extremity.

## Formation of the Brachial Plexus

Unless one works with the brachial plexus constantly, it is difficult to remember its anatomy accurately. The anatomy is depicted here to understand injuries from birth and later trauma. Most traction injuries involve the upper roots (**Fig. 1.4**).

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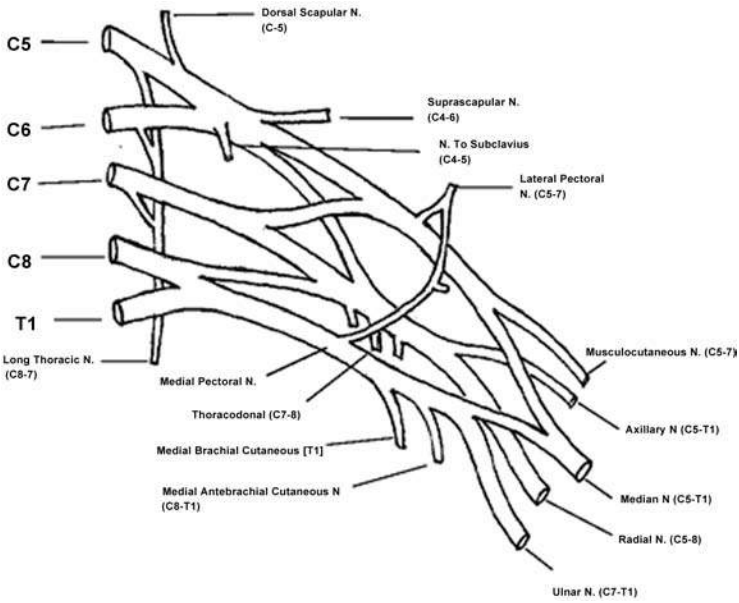



**Fig. 1.4** Formation of the brachial plexus.

## Peripheral Nerve Testing in the Upper Extremity

In evaluating an injury of the upper extremity in children (such as for supracondylar fracture), it is important to have methods to test the peripheral nerves: the median nerve may be tested by grip or finger flexion and the anterior interosseous nerve (a branch of the median that can be selectively injured) by testing distal interphalangeal flexion of the index finger and thumb, forming an "O."

- Radial nerve: By extending the thumb, the wrist or the metacarpophalangeal joints.
- Ulnar nerve: By crossing fingers, abducting fingers, or flexing the distal interphalangeal joint of the fifth finger (**Fig. 1.5**).

## Lower Extremity Motor Innervation

Knowledge of lower extremity motor innervation is important for understanding spina bifida, lumbar disk herniation, spinal cord injury, and other conditions. Innervation of muscles is by descending spinal segments at

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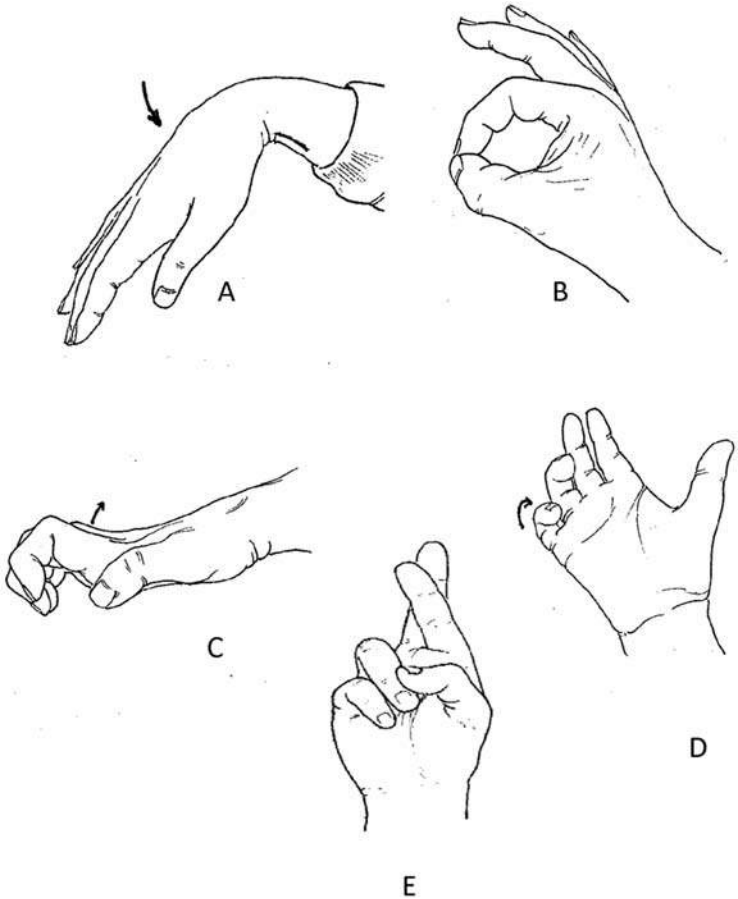



**Fig. 1.5** Documentation of the status of all nerves and circulation before treatment of supracondylar humerus fractures. This involves **(A)** checking active palmarflexion (median nerve); **(B)** flexion of distal interphalangeal joints of the index finger and thumb–anterior interosseous nerve; **(C)** dorsiflexion of the metacarpophalangeal joints–posterior interosseous nerve; **(D)** flexion of the fifth finger distal interphalangeal joint; or **(E)** crossing of index and second fingers–ulnar nerve.

progressively distal levels of the limb, with the notable exception of gluteus maximus, medius, and minimus (L5 through S2).

The most important motors to know are iliopsoas (L1 through L3), adductors (L2 through L4), quadriceps (L2 through L4), hamstrings (L4 through L5), anterior tibialis (L4 through L5), gastrocnemius (S1), and glutei (L5 through S2) (**Fig. 1.6**).

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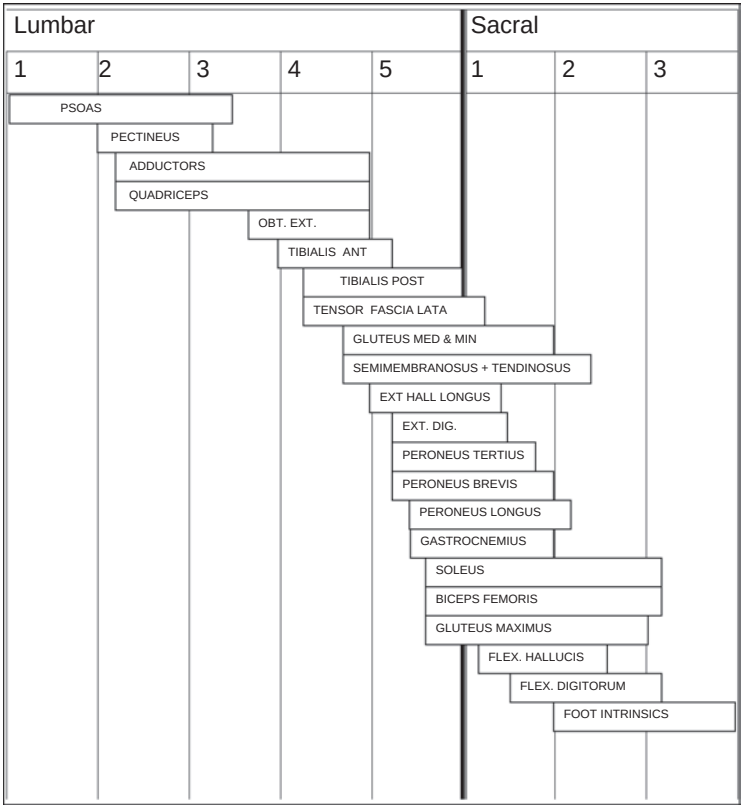



**Fig. 1.6** Segmental innervation of muscles of the lower limb.

## ◆ Skeletal Development

### Appearance of Secondary Ossification Centers and Physeal Closure

In many situations it is important to know whether an epiphysis should be ossified at a given age, such as in evaluating a patient with a hip dislocation, skeletal dysplasia, or elbow fracture. Normal times for appearance and closure of the long bones are given in **Fig. 1.7**; for the hand and foot, see **Fig. 1.8**.

The following are some important milestones:

1. The distal femoral epiphysis is the first to ossify, at ~39 weeks' gestation; the proximal tibia ossifies 1 week later.

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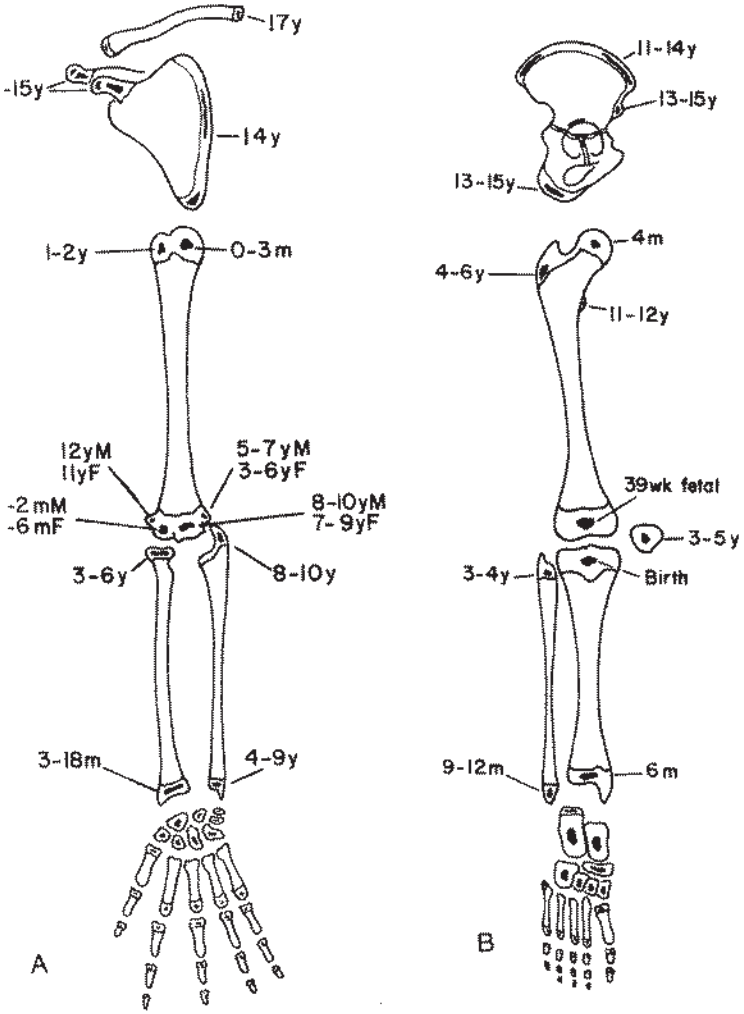



**Fig. 1.7** Age of appearance of secondary ossification centers **(A)** and physeal closure **(B)** in the long bones. (From Ogden JA. Radiologic aspects. In Ogden JA, ed. Skeletal Injury in the Child. 2nd ed. Philadelphia: W.B. Saunders; 1990:84 (Figs. 3-28 and 3-29). Reprinted with permission.)

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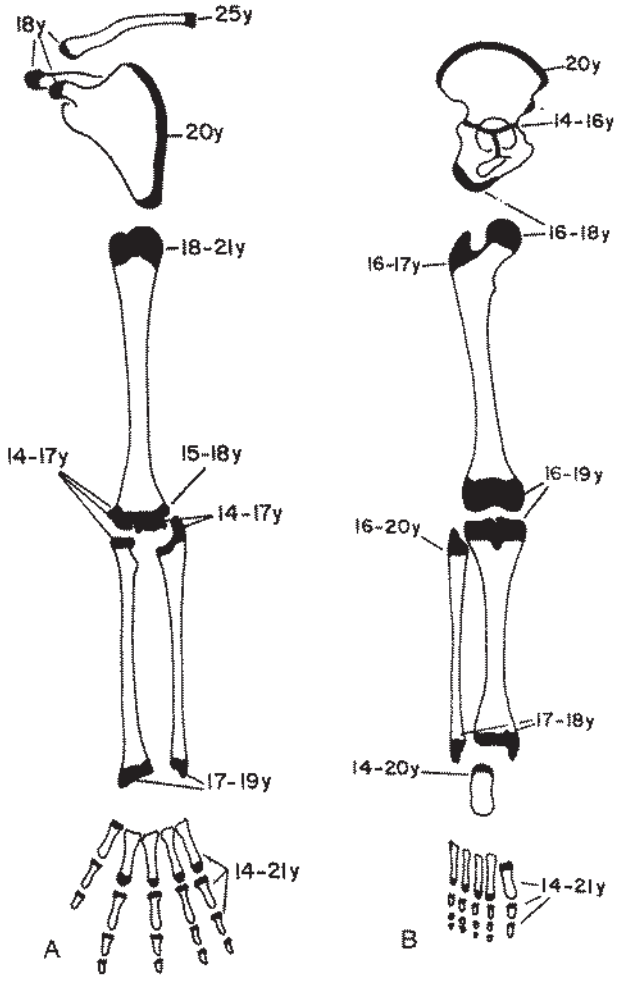



**Fig. 1.7** *(continued)*

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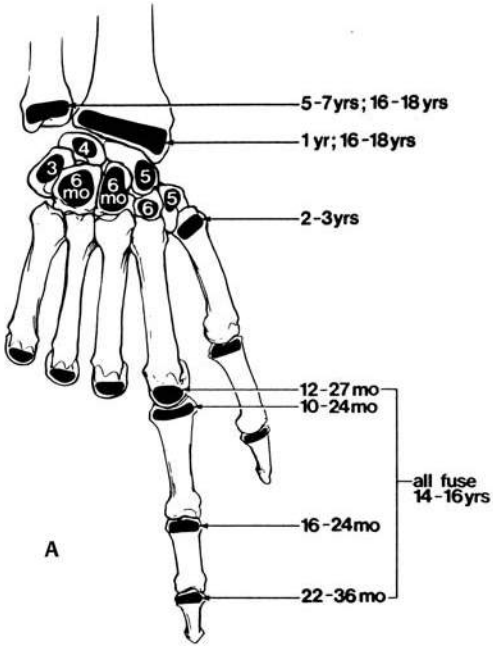



**Fig. 1.8** Age of appearance of secondary ossification centers and physeal closure in the hand **(A)** and foot **(B)**. (**[A]** from O'Brien ET. Fractures of the hand and wrist region. In Rockwood CA Jr, Wilkins KE, King RE, eds. Fractures in Children. 3rd ed. Philadelphia: J.B. Lippincott Co., 1991: 320 (Fig. 4-1). Reprinted with permission. **[B]** from Aitken JT, Causey G, Joseph J, Young JZ. The foot. In Aitken JT, Causey G, Joseph J, Young JZ, eds. A Manual of Human Anatomy. Vol IV, Lower Limb. 2nd ed. Edinburgh: E & S Livingstone; 1966:80 (Fig. 30). Reprinted with permission.)

2. The mean time for ossification of the proximal femoral epiphysis is 4 months, but normal may be up to 11 months. The greater trochanter ossifies at 4 to 6 years.
3. The triradiate cartilage closes before Risser I.
4. The tarsal navicular does not ossify until 3 to 4 years, so its location must be inferred from the position of the first metatarsal.
5. The last physis to close is that of the medial clavicle, at age 20 to 25 years.

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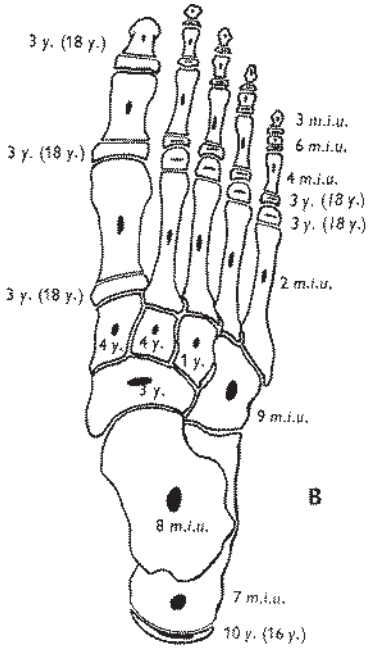



**Fig. 1.8** ***(continued)***

**6.** The sequence of ossification about the elbow can be remembered by the mnemonic CRITOE (**Fig. 1.9**):

- C apitellum (age 2)
- R adial head (age 5)
- I nternal epicondyle (age 7)
- T rochlea (age 9)
- O lecranon (age 10)
- E xternal epicondyle (age 11)

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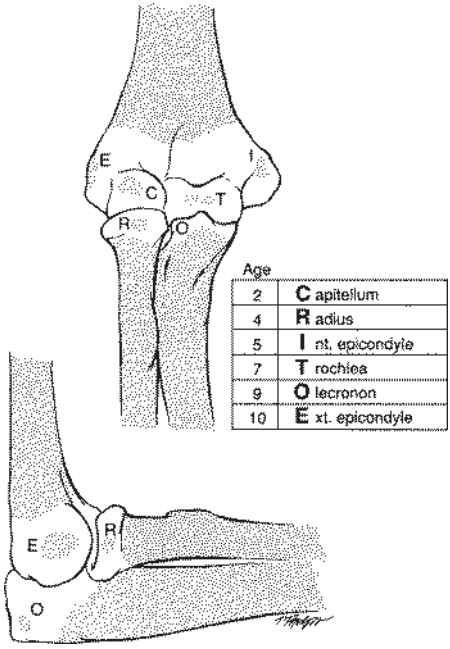



**Fig. 1.9** Appearance of age of ossification centers about the elbow can be summarized by mnemonic CRITOE. (From Sponseller PD. Orthopaedic injuries. In Nichols DG, Yaster M, Lappe DG, Buck JR, eds. Golden Hour: The Handbook of Advanced Pediatric Life Support. St. Louis: Mosby Year Book; 1991:350 (Fig. 18-3). Reprinted with permission.)

7. Angle of distal humeral articular surface: This angle is key to understanding any angular change about the elbow. It is best measured by the Baumann angle, between the humeral shaft and the lateral condylar physis (**Fig. 1.10**). Its normal value is 72 ± 4 degrees. There is no difference between sexes or ages from 2 to 13 years.
8. Determination of skeletal age using the elbow: This can be determined by the olecranon or modified Sauvegrain method (**Fig. 1.11**). The five stages include (1) bipartite olecranon, (2) half-moon–shaped olecranon, (3) rectangular olecranon, (4) partially fused, or (5) fully fused. They occur at 6-month intervals of skeletal age from 11 years in girls and 13 in boys. The fully fused olecranon occurs at skeletal age of 13 in girls and 15 in boys and marks the deceleration of growth velocity.
9. Determination of skeletal age using the Modified Tanner–Whitehouse III Skeletal Maturity Assessment (**Table 1.2** and **Fig. 1.12**)

Patients with curves over 30 degrees at stage 3 to 4 are at high risk of progression to surgery, and patients with curves less than 20 degrees at this stage are at low risk.

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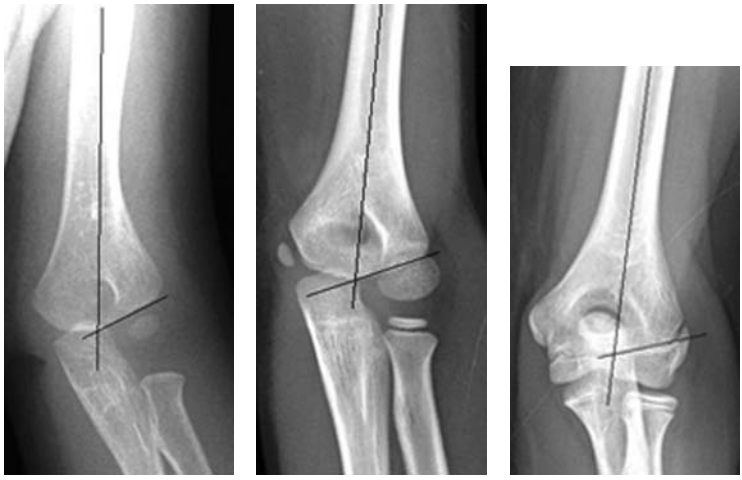


**Fig. 1.10** Differing configuration of the distal humerus and landmarks used for measurement of the Baumann angle.

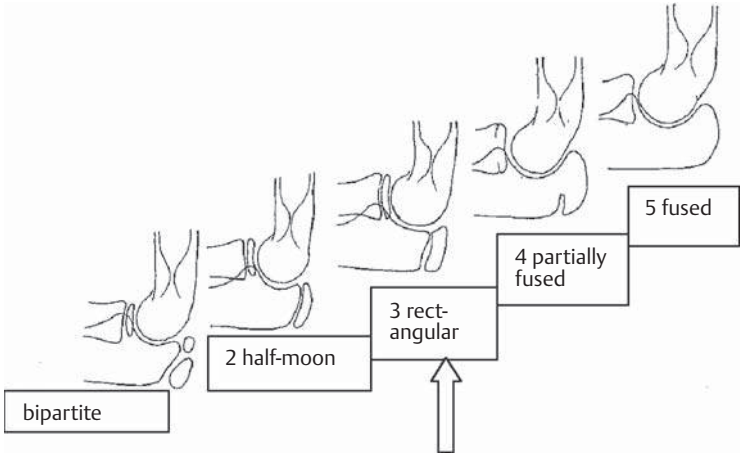



**Fig. 1.11** Simplified Sauvegrain method for skeletal maturity assessment.

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**Table 1.2** Key Findings of the Simplified Tanner–Whitehouse III Skeletal Maturity Assessment

| Stage | Key Features | Tanner-Whitehouse III Stage | Greulich and Pyle Reference | Related Maturity Signs |
|---|---|---|---|---|
| 1. Juvenile slow | Digital epiphyses not covered | Some digits are at stage E or lower | Female 8 year + 10 months, male 12 year + 6 months (note fifth middle phalanx) | Tanner stage 1 |
| 2. Preadolescent slow | All digital epiphyses covered | All digits at stage F | Female 10 year, male 13 year | Tanner stage 2, starting growth spurt |
| 3. Adolescent rapid–early | Preponderance of digits are capped. The 2nd through 5th metacarpal epiphyses wider than their metaphyses | All digits are at stage G | Female 11 and 12 year, male 13 year + 6 months and 14 year | Peak height velocity, Risser stage 0, open pelvic triradiate cartilage |
| 4. Adolescent rapid–late | Any distal phalangeal physes are clearly beginning to close | Any distal phalanges are at stage H | Female 13 year (digits 2, 3, 4), male 15 year (digits 4,5) | Girls typically in Tanner stage 3, Risser stage 0, open triradiate cartilage |
| 5. Adolescent steady–early | All distal phalangeal physes closed; others are open | All distal phalanges and thumb metacarpal are at stage I; others remain at stage G | Female 13 year + 6 months, male 15 year + 6 months | Risser stage 0, triradiate cartilage closed, menarche only; occasionally starts earlier |
| 6. Adolescent steady–late | Middle or proximal phalangeal physes are closing | Middle or proximal phalanges are at stages H and I | Female 14 year, male 16 year (late) | Risser sign positive (stage 1 or more) |
| 7. Early mature | Only distal radial physis is open; metacarpal physeal scars may be present | All digits are at stage I. The distal radial physis is at stage G or H | Female 15 year, male 17 year | Risser stage 4 |
| 8. Mature | Distal radial physis completely closed. | All digits are at stage I | Female 17 year, male 19 year | Risser stage 5 |

*Source*: From Sanders JO, Khoury JG, Kishan S, et al. Predicting scoliosis progression from skeletal maturity: a simplified classification during adolescence. J Bone Joint Surg Am. 2008;90(3):541 (Table 1). Reprinted with permission.

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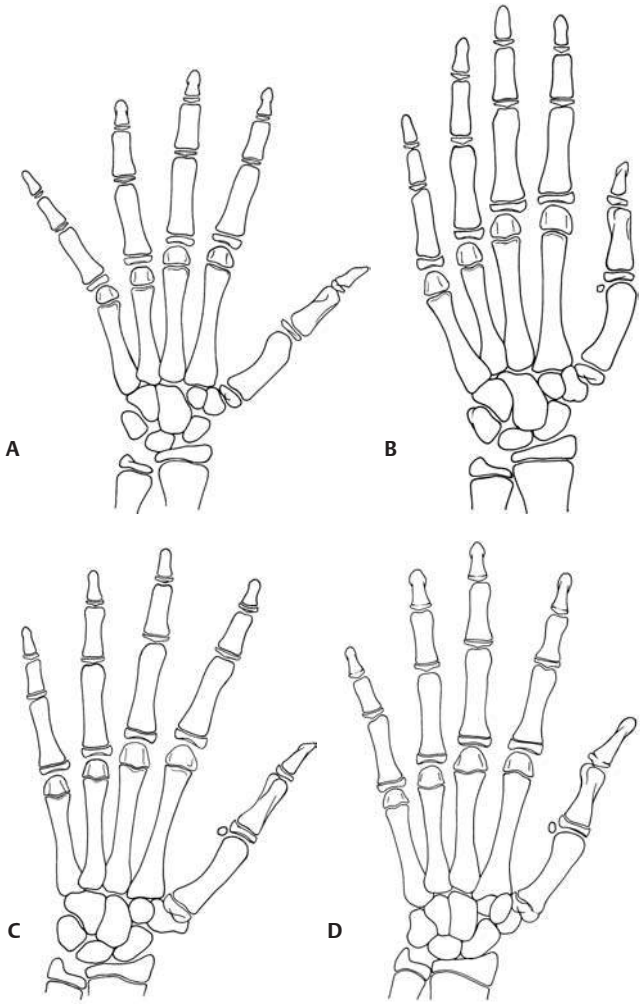



**Fig. 1.12** Simplified Tanner–Whitehouse III method for skeletal maturity assessment. Note that the distal phalangeal epiphyses are the key to the curve acceleration phase (CAP). When they are capped, the phase is beginning, and when they are all closed, the phase has ended. **(A)** Stage 1: All digital epiphyses not covered (epiphyses not as wide as metaphyses). **(B)** Stage 2: All digital epiphyses are covered. **(C)** Stage 3: Most epiphyses cap their metaphyses. Capping is a small bend over the metaphyseal edge. This is also the beginning of the CAP. **(D)** Stage 4: At least one of distal phalanges closed. (From Sanders JO, Khoury JG, Kishan S, et al. Predicting scoliosis progression from skeletal maturity: a simplified classification during adolescence. J Bone Joint Surg Am. 2008;90(3):540-553 (Figs. 1-B, 2-B, 3-B, 4-B). Reprinted with permission.)

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## Cervical Spine Radiographic Normal Values for Children

*Alignment*

1. The cervical spine in children is characterized by increased mobility at C2–3, termed *pseudosubluxation*, which should not exceed 3 mm.
2. The tip of the odontoid should not be more than 1 cm from the basion of the skull (anterior rim of the foramen magnum).
3. The physis of the odontoid normally fuses between 3 and 6 years.
4. The atlas–dens interval should be less than 4 mm.
5. The Power ratio is the ratio of the distance from basion to posterior arch of C1 divided by the distance from the opisthion to the anterior arch of C1. This ratio should be less than 1.
6. The retropharyngeal space should not exceed 8 mm; if greater, it could signify bleeding from a fracture or a dislocation.

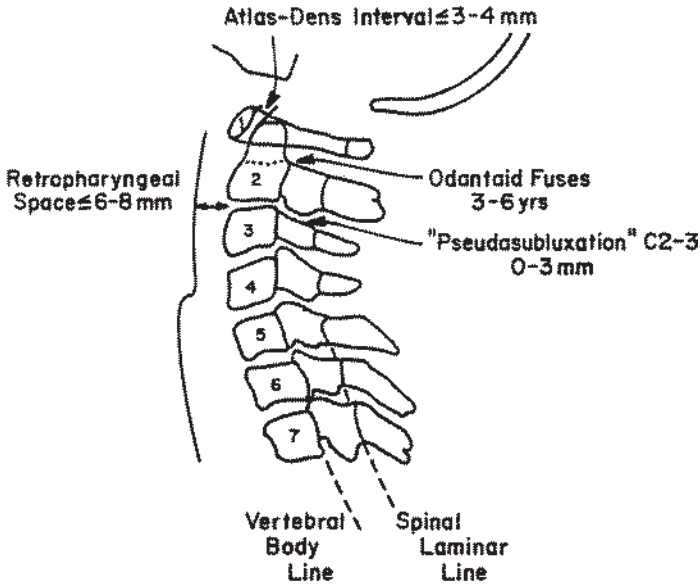



**Fig. 1.13** Normal values of cervical spine alignment for children. (From Sponseller PD. Orthopaedic injuries. In Nichols DG, Yaster M, Lappe DG, Buck JR, eds. Golden Hour: The Handbook of Advanced Pediatric Life Support. St. Louis: Mosby Year Book; 1991:353 (Fig. 18-4). Reprinted with permission.)

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7. The spinal laminae should form a smooth line posteriorly.
8. The vertebral bodies may be wedged anteriorly, especially on their superior surfaces, until age 10 (**Fig. 1.13**).

## Development of the Cervical Spine

*First Cervical Vertebra (Atlas)*

1. Body: Not ossified at birth; the center (occasionally two centers) appears during the first year after birth; the body may fail to develop, and forward extension of neural arches may take its place.
2. Neural arches: Appear bilaterally at approximately the seventh fetal week; most of the anterior portion of superior articulating surface is usually formed by the body.
3. Synchondrosis of spinous processes: Unites by the third year. Union rarely is preceded by the appearance of a secondary center within the synchondrosis.
4. Neurocentral synchondrosis: Fuses at approximately the seventh year (**Fig. 1.14**).

*Second Cervical Vertebra (Axis)*

1. Body: One center (occasionally two) appears by the fifth fetal month.
2. Neural arches: Appear bilaterally by fetal month 7.
3. Neural arches fuse posteriorly by the year 2 or 3.

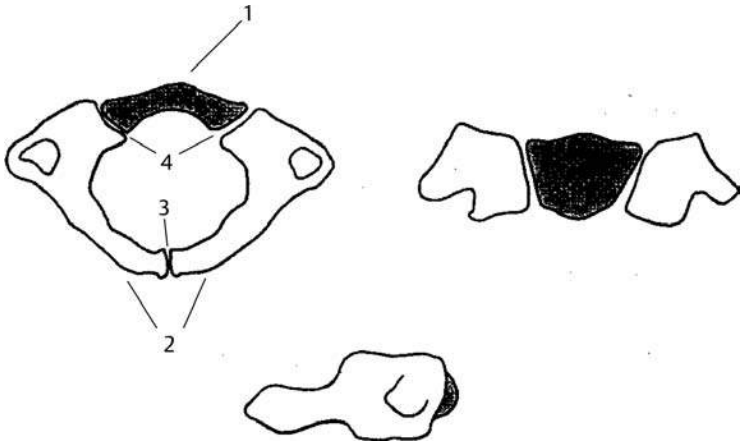



**Fig. 1.14** Axial, coronal, and sagittal views of the developing atlas. The first cervical vertebra is formed by three ossification sites: the anterior arch (gray), or centrum, and the two neural arches (white). (From Oh BC, Wang MY. Cervical anatomy and surgical approaches. In Kim DH, Betz RR, Huhn SL, Newton PO, eds. Surgery of the Pediatric Spine. New York: Thieme; 2008:95 (Fig. 8-1). Reprinted with permission.)

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4. Bifid tip of spinous process: Occasionally a secondary center is present in each tip.
5. Neurocentral synchondrosis: Fuses at 3 to 6 years
6. Inferior epiphyseal ring: Appears at puberty and fuses at around 25 years
7. "Summit" ossification center for odontoid: Appears at 3 to 6 years and fuses with the odontoid by 12 years
8. Odontoid (dens). Two separate centers appear by fetal month 5 and fuse with each other by month 7
9. Synchondrosis between odontoid and neural arch: Fuses at 3 to 6 years
10. Synchondrosis between odontoid and body: Fuses at 3 to 6 years
11. Posterior surface of body and odontoid (**Fig. 1.15**)

## Spinal Growth

The growth of the spine occurs relatively earlier than that of the extremities, but not to the extreme of cranial growth. If arthrodesis is considered in a growing child, the following rules will help predict the consequences for growth.

*Guidelines and Rules of Thumb*

1. T1–S1 growth rates

- 0 to 5 years: 2 cm/year
- 6 to 10 years: 0.9 cm/year
- 10 years: 1.8 cm/year through growth spurt

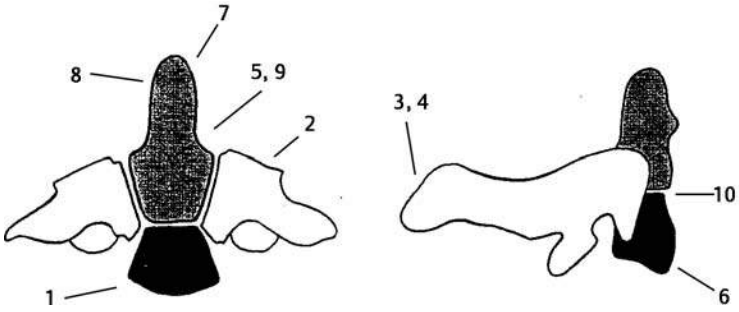



**Fig. 1.15** Coronal and sagittal views of the developing axis. There are four ossification centers present at birth: one center for each neural arch (white), one for the odontoid process (gray), and one for the body (black). (From Oh BC, Wang MY. Cervical anatomy and surgical approaches. In Kim DH, Betz RR, Huhn SL, Newton PO. Surgery of the Pediatric Spine. New York: Thieme; 2008:96 (Fig. 8-2). Reprinted with permission.)

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2. Growth of the T1–S1 segment is two thirds complete by 6 years.
3. Growth remaining from T1–S1 at age 5 is 15 cm.
4. DiMeglio has summarized the growth of the extremities and spine with respect to maturity indicators (elbow growth centers, Risser sign, menarche, triradiate cartilage closure) in **Fig. 1.16**. Triradiate cartilage closure slightly precedes the peak height velocity; olecranon closure occurs at the peak height velocity, and menarche occurs just after peak height velocity; all are at or before Risser sign turns to 1.

Note that posterior arthrodesis alone does not stop growth completely within a fused segment. Further growth can cause relative compression of disk space, exacerbation of preexisting lordosis, or rotation of scoliosis ("crankshaft") (**Fig. 1.17**).

*Spinal Growth Remaining Calculation*

- 0.07 cm × number of spinal segments × growth years left (**Fig. 1.18**)

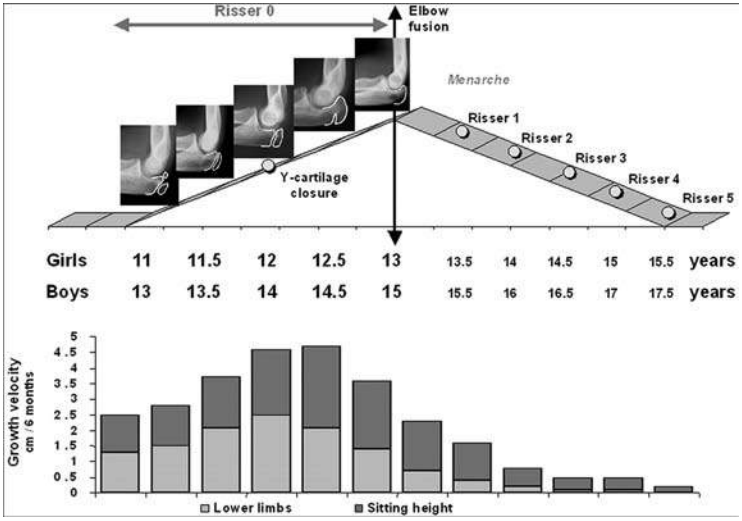



**Fig. 1.16** Simplified skeletal age assessment with the olecranon method during the accelerating pubertal growth phase of peak height velocity and Risser grade 0 from the ages of 11 to 13 years in boys, with a decelerating growth phase after elbow fusion. Y cartilage closure = triradiate cartilage closure. (From Charles YP, Dimeglio A, Canavese F, Daures JP. Skeletal age assessment from the olecranon for idiopathic scoliosis at Risser grade 0. J Bone Joint Surg Am. 2007:89(12):2739 (Fig. 1). Reprinted with permission.)

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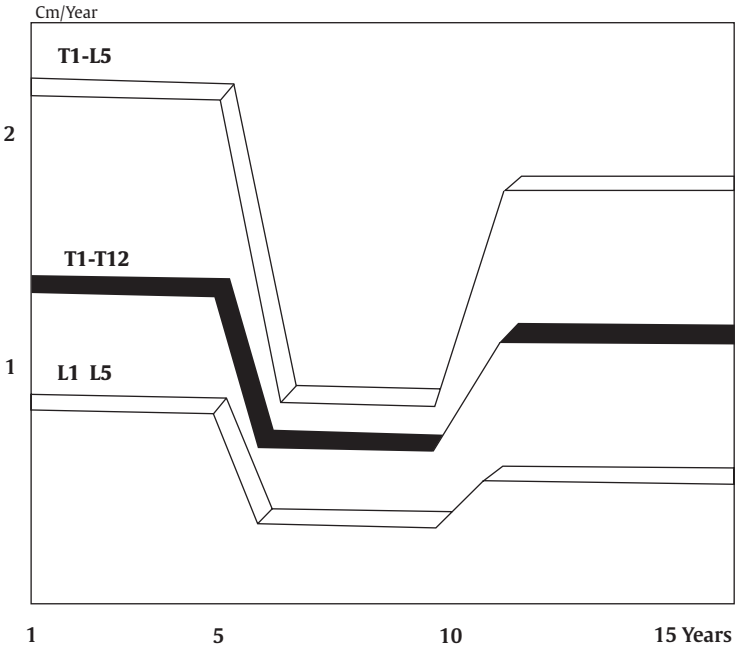



**Fig. 1.17** Approximate growth velocity of the spine by segments: thoracic, lumbar, and combined segments. Note that the greatest velocity is in preschool years, and then a significant drop occurs. (From Dimeglio A. Growth of the spine before age 5 years. J Pediatr Orthop B. 1992;1(2):103 (Fig. 7). Reprinted with permission.)

## Extremity Growth: Relative Contributions to Growth of the Long Bones

The contributions of each physis to longitudinal growth are a reflection of its activity, which in turn influences remodeling potential. A simple rule of thumb is that most growth occurs "away from the elbow and at the knee" in the upper and lower extremities, respectively (**Fig. 1.19**).

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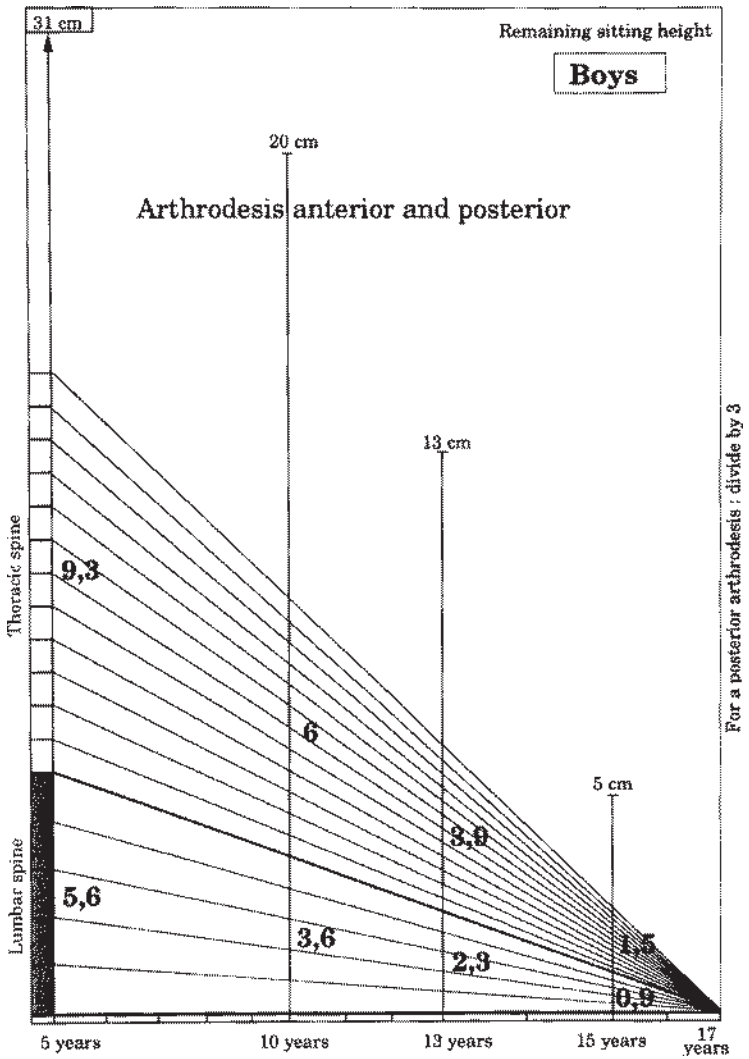



**Fig. 1.18** Growth remaining in the spine by segment for **(A)** boys and ***(continued on page 24)***

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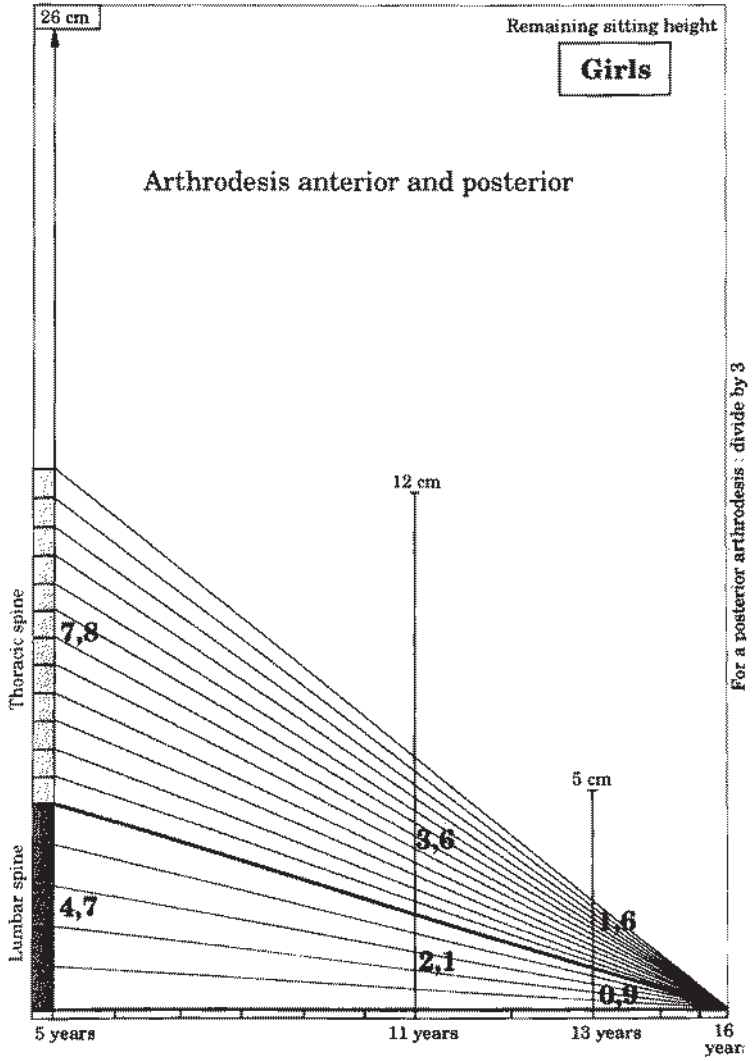



**Fig. 1.18** ***(continued from page 23)*** **(B)** girls. Note that the remaining component of sitting height is due to the head, cervical spine, and cranium. (From Dimeglio A. Growth of the spine before age 5 years. J Pediatr Orthop B. 1992;1(2):104 (Figs. 11 and 12). Reprinted with permission.)

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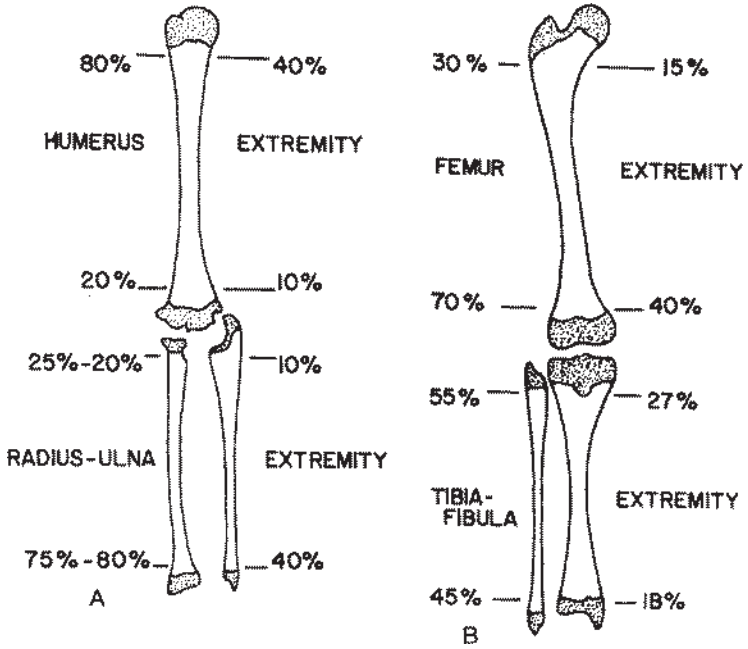



**Fig. 1.19** Relative contributions to growth of the long bones of the upper (**A**) and lower (**B**) extremity. (From Ogden JA. Radiologic aspects. In Ogden JA, ed. Skeletal Injury in the Child. 2nd ed. Philadelphia: W.B. Saunders; 1990:85 (Fig. 3-30A,B). Reprinted with permission.)

Some guidelines for estimating growth:

1. At each physis, this estimation can be done during the preadolescent years using the approximate rates:

   - Proximal femur: ⅛ inch/year
   - Distal femur: ⅜ inch/year
   - Proximal tibia: ¼ inch/year
   - Distal tibia: $^{3}/_{16}$ inch/year

These estimates are true until age 13 for girls or age 15 for boys. For taller-than-average children, these rates are greater. For long growth periods of more than 3 years, it is better to consult growth tables (**Fig. 1.20**).

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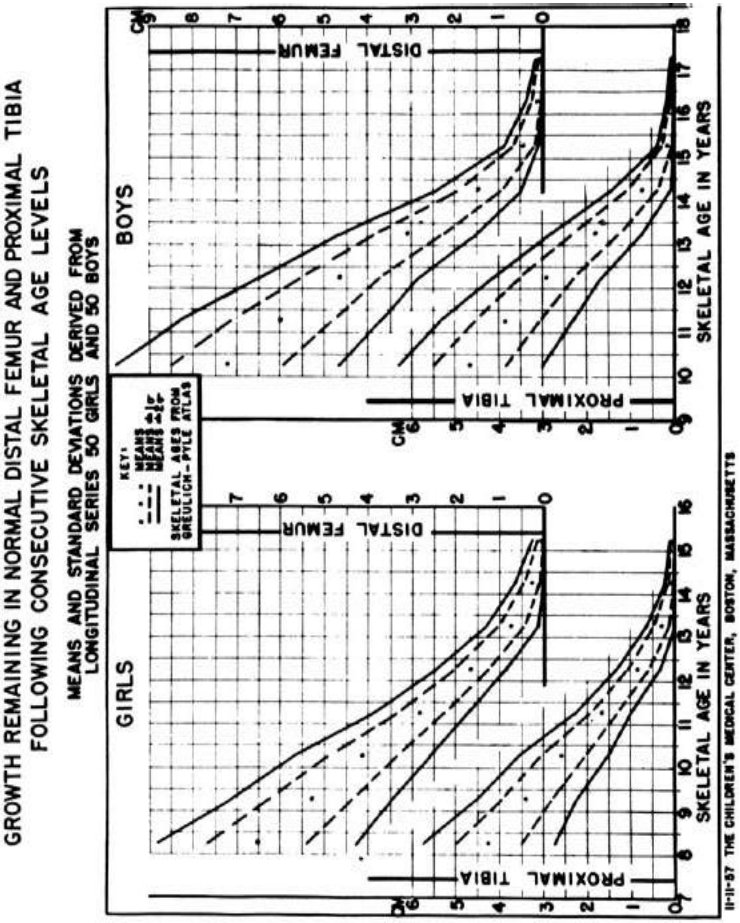



**Fig. 1.20** Growth remaining in normal distal femur and proximal tibia following consecutive skeletal ages. (From Anderson M, Green WT, Messner MB. Growth and predictions of growth in the lower extremities. J Bone Joint Surg Am. 1963;45(1):10 (Chart III). Reprinted with permission.)

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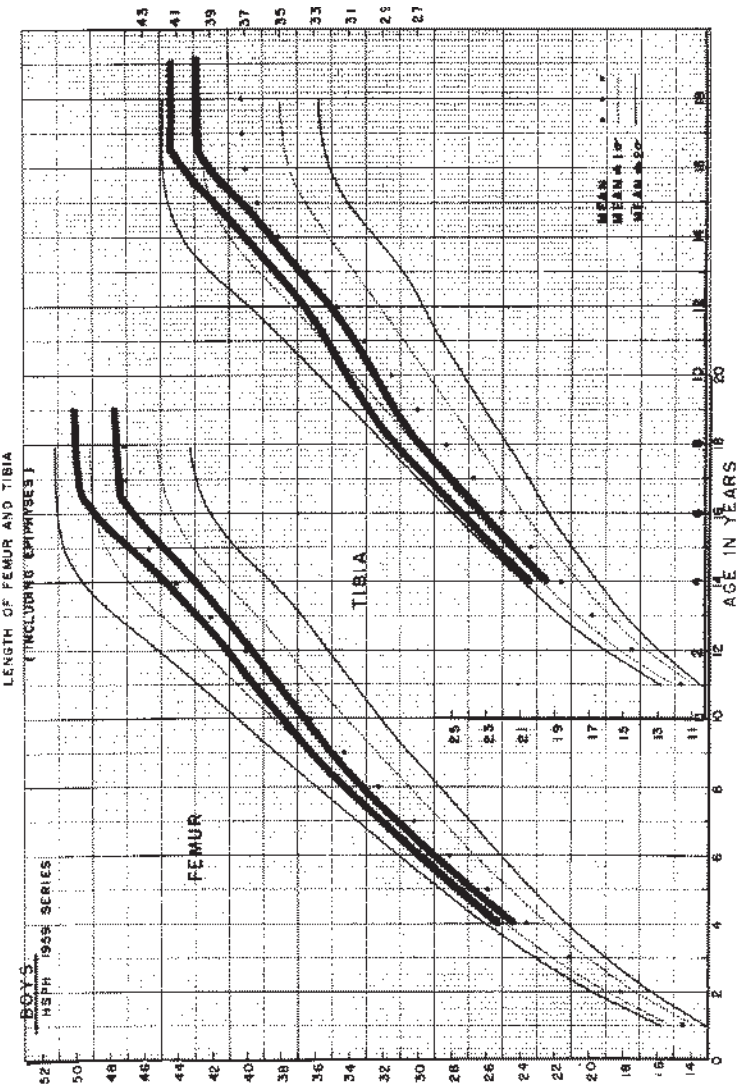



**Fig. 1.21** Length of normal femur and tibia for boys (including epiphyses). (From Anderson M, Messner MB, Green WT. Distribution of lengths of the normal femur and tibia in children from one to eighteen years of age. J Bone Joint Surg Am. 1964;46(6):1201 (Chart II). Reprinted with permission.)

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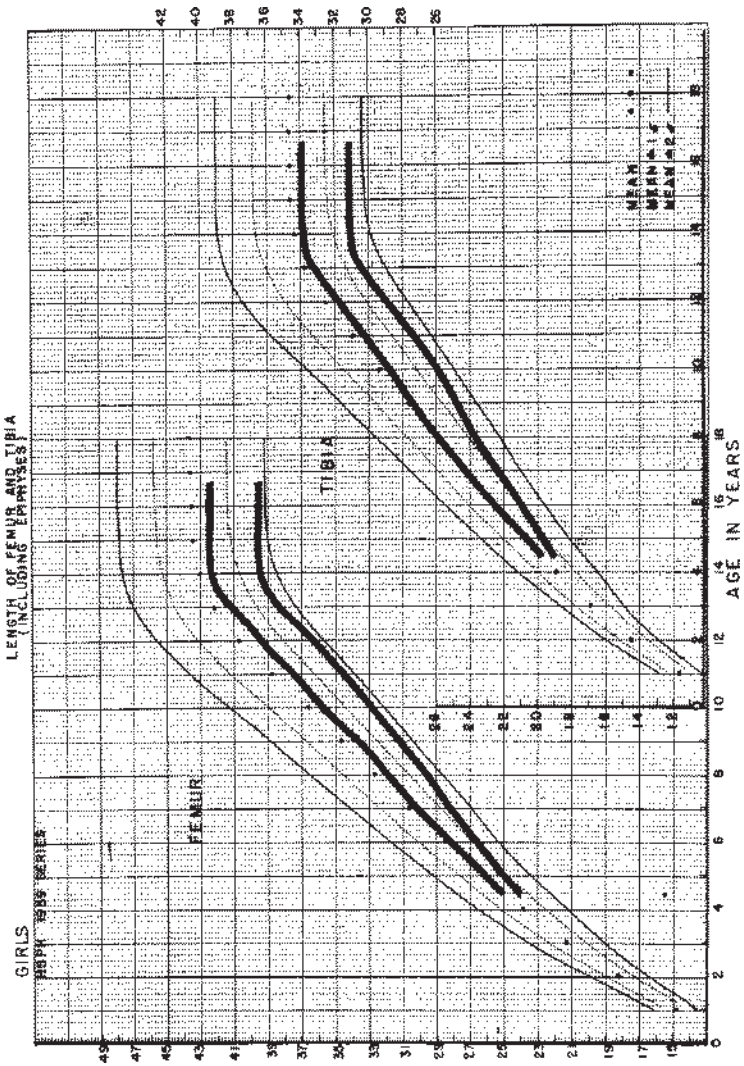



**Fig. 1.22** Length of normal femur and tibia for girls (including epiphyses). (From Anderson M, Messner MB, Green WT. Distribution of lengths of the normal femur and tibia in children from one to eighteen years of age. J Bone Joint Surg Am. 1964;46(6):1200 (Chart I). Reprinted with permission.)

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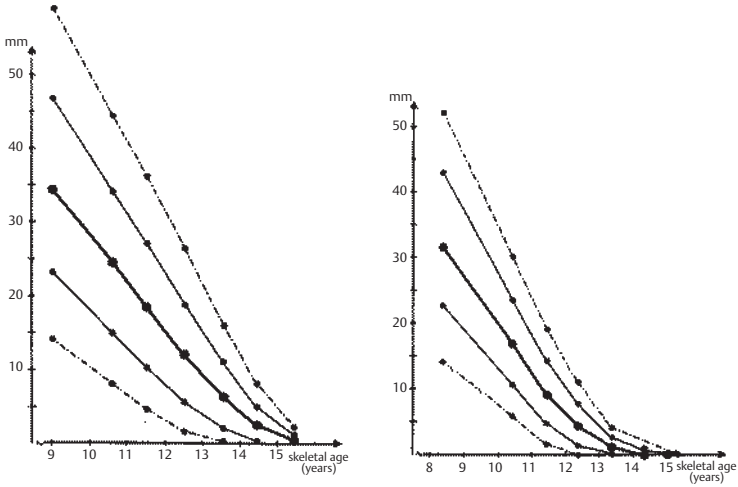
2. Total adult height = 2 × height at age 2.
3. Total adult length of lower extremities = 2 × length at age 4.
4. Growth ceases in girls at 15 to 15.5 years, in boys at 17 to 17.5 years.
5. Another way to estimate adult height:

   - Males = (Father's height + mother's height + 6 cm)/2.
   - Females = (Father's height + mother's height – 6 cm)/2.
   - Two standard deviations = ±5 cm.

6. Another way to estimate adult height is to use the multiplier method (**Table 1.3**, page 36). This takes into account the fact that existing height is a strong predictor of final height.

*Growth Curves for Long Bones*

Growth curves are useful when absolute lengths are needed. **Figs. 1.21 and 1.22** represent the same population data as presented differently in the "growth remaining" curves.




**A** Distal tibia—boys. Calculated remaining growth (—— ±1 SD, — - — ±2 SD).

**B** Distal tibia—girls. Calculated remaining growth (—— ±1 SD, — - — ±2 SD).

**Fig. 1.23** Growth remaining in distal tibia (mean ± 2 SD) for boys **(A)** and girls **(B)**. (From Karrholm J, Hansson LI, Selvik G. Longitudinal growth rate of the distal tibia and fibula in children. Clin Orthop Relat Res. 1984;191:124 (Figs. 2B and 3B). Reprinted with permission.)

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*Growth Remaining Curves*

These are a reformulation of the data shown in **Figs. 1.21** and **1.22**. This format is especially useful for estimating the effects of physeal closure. One must know the patient's skeletal age and percentile rank for height.

*Longitudinal Growth from Distal Tibial and Fibular Physes*

1. Distal tibial and fibular physeal fractures often occur in childhood and adolescence. Growth plate damage may occur. Physeal closure in either bone then must be evaluated for significance of both shortening and angular deformity.
2. The following growth-remaining graphs may be used for prediction of the deformity. The percentile to follow for a given patient may be obtained from the percentile of the patient's rank in **Figs. 1.21, 1.22,** and **1.23**.
3. Length lost from total arrest may be calculated. In general, inequality of less than 1 cm is of no clinical significance.
4. Prediction of angular growth disturbance as a result of peripheral arrest may be calculated from the growth remaining and the width of the physis. Because the average distal tibia is 4 to 5 cm wide, 10 degrees of angulation is likely to occur in boys before the age of 13½ and in girls before 11½.
5. This may also be used to predict the effects of surgical hemiepiphyseodesis.

*Straight-Line Graph for Predictions of Discrepancy*

For growth disturbances that do not change characteristics over time, if skeletal age is known, the eventual inequality of limb length at maturity can be calculated by plotting data points on the straight-line graph developed by Moseley (**Figs. 1.24** and **1.25**). The procedure is explained step by step. Conditions for which the graph may not be appropriate are for those with a phasic nature, for example, a discrepancy that is due to juvenile rheumatoid arthritis or Klippel-Trenaunay syndrome.

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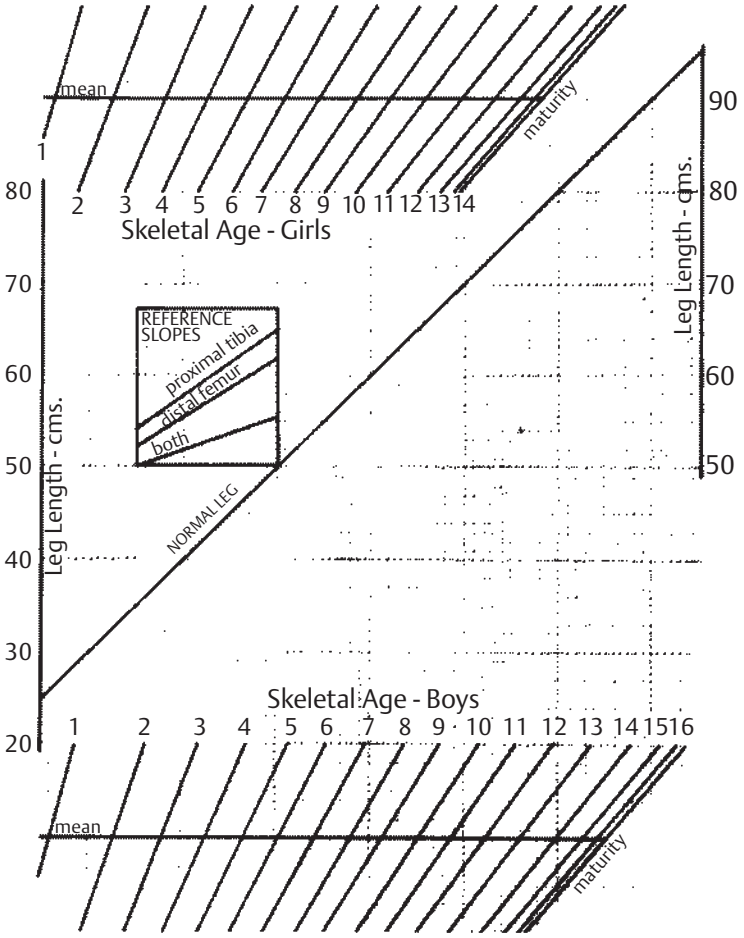



**Fig. 1.24** Moseley straight-line graph for predicting limb-length inequality. (From Moseley CF. A straight-line graph for leg-length discrepancies. J Bone Joint Surg Am. 1977;59(2):176 (Fig. 1). Reprinted with permission.)

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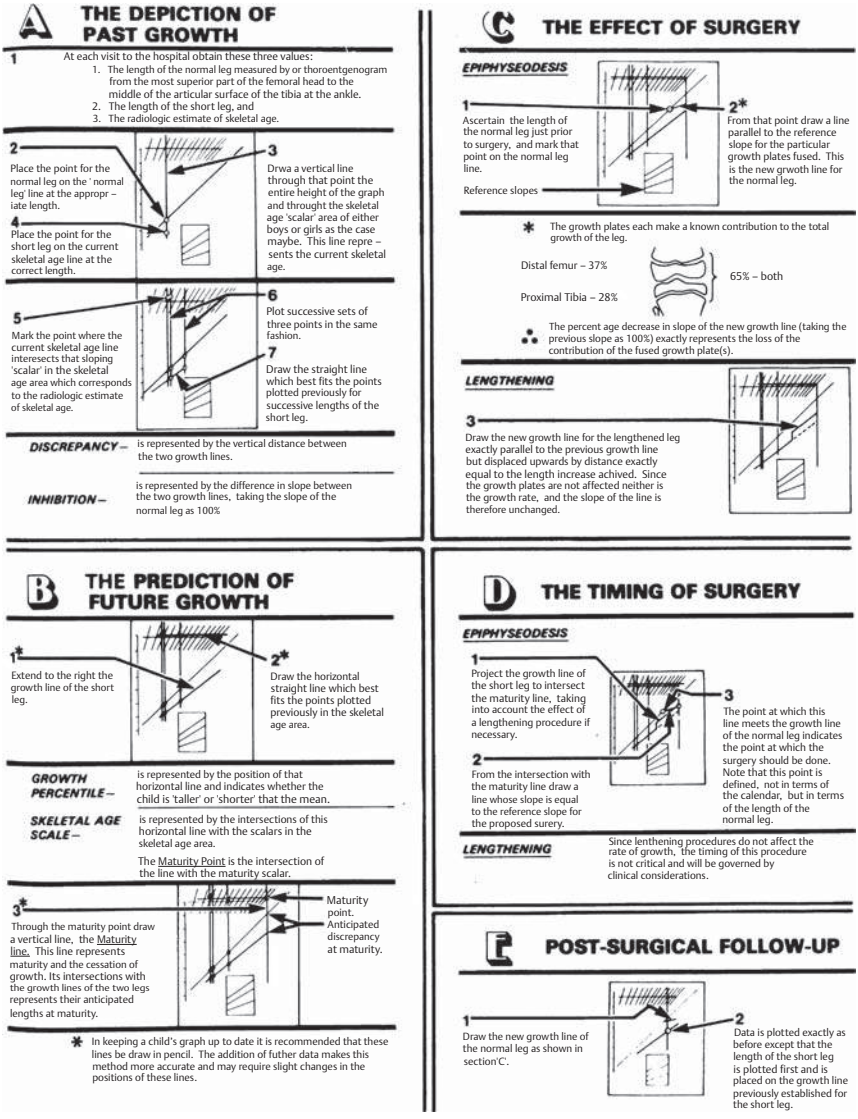



**Fig. 1.25** Instructions for the Moseley graph. (From Moseley CF. A straight-line graph for leg-length discrepancies. J Bone Joint Surg Am. 1977;59(2):177 (Fig. 2). Reprinted with permission.)

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*Upper Extremity Growth*

Growth arrest occurs less commonly in the upper extremity. Occasionally, however, as a result of infection, tumor, or trauma, growth is affected, and it may become necessary to calculate the resulting discrepancy (**Fig. 1.26**).

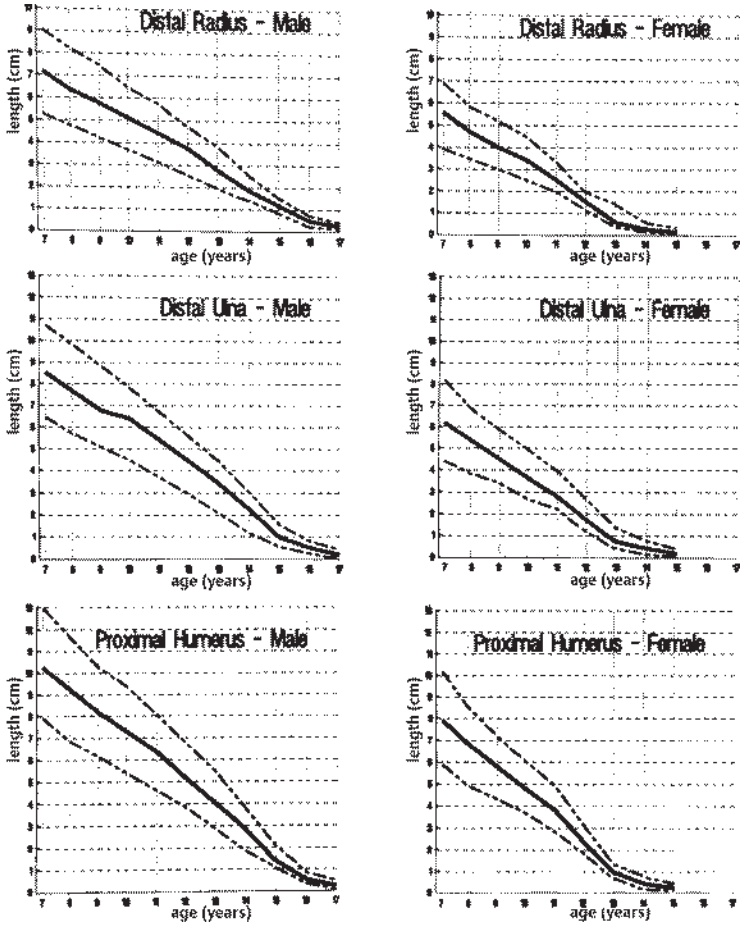



**Fig. 1.26** Growth remaining from major upper extremity physes. These graphs are from the Child Research Counsel, Denver, Colorado. Mean (solid line) and two standard deviations (dotted lines). (From Bortel DT, Pritchett JW. Straight-line graphs for the prediction of growth of the upper extremities. J Bone Joint Surg Am. 1993;75(6):889 (Fig. 2). Reprinted with permission.)

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## *Overall Physical Growth Norms*

Physical growth norms include mean stature for age and their respective percentiles. They are useful in screening for growth disturbances and estimating height at maturity (**Figs. 1.27** and **1.28**).

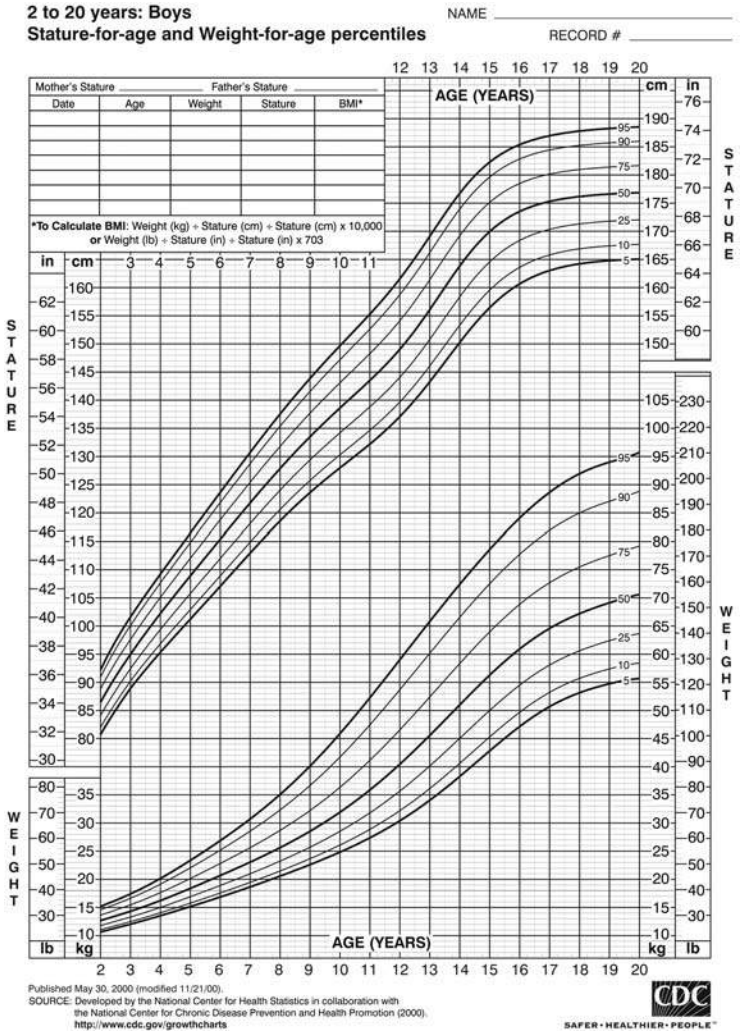



**Fig. 1.27** Age 2 to 20 years: Boys stature-for-age and weight-for-age percentiles.

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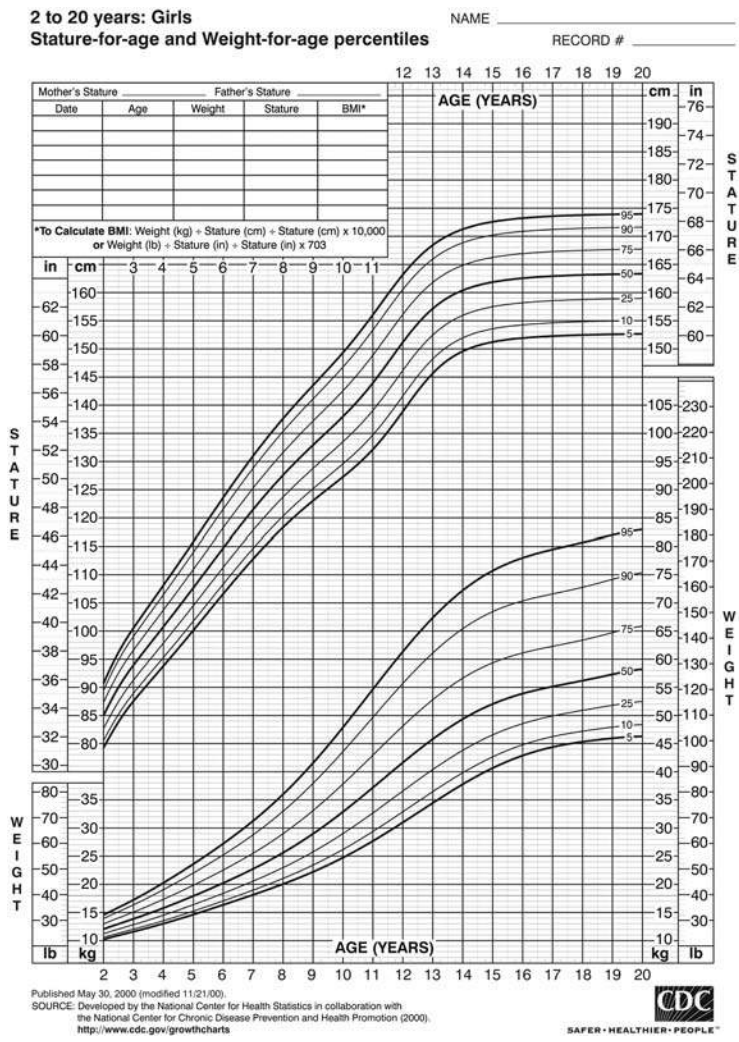



**Fig. 1.28** Age 2 to 20 years: Girls stature-for-age and weight-for-age percentiles.

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**Table 1.3** Multiplier Method for Height (Paley Method)*

| Age (yr) | Height Multiplier | |
|---|---|---|
| | Girls | Boys |
| Birth | 3.30 | 3.53 |
| 0.5 | 2.50 | 2.64 |
| 1.0 | 2.22 | 2.34 |
| 1.5 | 2.04 | 2.16 |
| 2.0 | 1.92 | 2.04 |
| 2.5 | 1.81 | 1.94 |
| 3.0 | 1.73 | 1.86 |
| 3.5 | 1.68 | 1.78 |
| 4.0 | 1.62 | 1.73 |
| 4.5 | 1.57 | 1.67 |
| 5.0 | 1.51 | 1.63 |
| 5.5 | 1.47 | 1.58 |
| 6.0 | 1.42 | 1.53 |
| 6.5 | 1.38 | 1.50 |
| 7.0 | 1.34 | 1.45 |
| 7.5 | 1.31 | 1.42 |
| 8.0 | 1.28 | 1.38 |
| 8.5 | 1.25 | 1.35 |
| 9.0 | 1.23 | 1.32 |
| 9.5 | 1.21 | 1.30 |
| 10.0 | 1.18 | 1.28 |
| 10.5 | 1.16 | 1.26 |
| 11.0 | 1.13 | 1.23 |
| 11.5 | 1.11 | 1.21 |
| 12.0 | 1.08 | 1.19 |
| 12.5 | 1.06 | 1.16 |
| 13.0 | 1.04 | 1.13 |
| 13.5 | 1.03 | 1.11 |
| 14.0 | 1.02 | 1.08 |
| 14.5 | 1.01 | 1.06 |
| 15.0 | 1.01 | 1.04 |
| 15.5 | 1.01 | 1.03 |
| 16.0 | 1.00 | 1.02 |
| 16.5 | 1.00 | 1.01 |
| 17.0 | 1.00 | 1.01 |
| 17.5 | " | 1.01 |
| 18.0 | " | 1.00 |

*The patient's existing height may be multiplied by a number on this table corresponding to age to determine final height.

**Table 1.4** Multiplier Method for Lower Limb (LL) Lengths†

| Age (yr + mo) | LL Length Multiplier | |
|---|---|---|
| | Boys | Girls* |
| Birth | 5.080 | 4.630 |
| 0 + 3 | 4.550 | 4.155 |
| 0 + 6 | 4.050 | 3.725 |
| 0 + 9 | 3.600 | 3.300 |
| 1 + 0 | 3.240 | 2.970 |
| 1 + 3 | 2.975 | 2.750 |
| 1 + 6 | 2.825 | 2.600 |
| 1 + 9 | 2.700 | 2.490 |
| 2 + 0 | 2.590 | 2.390 |
| 2 + 3 | 2.480 | 2.295 |
| 2 + 6 | 2.385 | 2.200 |
| 2 + 9 | 2.300 | 2.125 |
| 3 + 0 | 2.230 | 2.050 |
| 3 + 6 | 2.110 | 1.925 |
| 4 + 0 | 2.000 | 1.830 |
| 4 + 6 | 1.890 | 1.740 |
| 5 + 0 | 1.820 | 1.660 |
| 5 + 6 | 1.740 | 1.580 |
| 6 + 0 | 1.670 | 1.510 |
| 6 + 6 | 1.620 | 1.460 |
| 7 + 0 | 1.570 | 1.430 |
| 7 + 6 | 1.520 | 1.370 |
| 8 + 0 | 1.470 | 1.330 |
| 8 + 6 | 1.420 | 1.290 |
| 9 + 0 | 1.380 | 1.260 |
| 9 + 6 | 1.340 | 1.220 |
| 10 + 0 | 1.310 | 1.190 |
| 10 + 6 | 1.280 | 1.160 |
| 11 + 0 | 1.240 | 1.130 |
| 11 + 6 | 1.220 | 1.100 |
| 12 + 0 | 1.180 | 1.070 |
| 12 + 6 | 1.160 | 1.050 |
| 13 + 0 | 1.130 | 1.030 |
| 13 + 6 | 1.100 | 1.010 |
| 14 + 0 | 1.080 | 1.000 |
| 14 + 6 | 1.060 | NA |
| 15 + 0 | 1.040 | NA |
| 15 + 6 | 1.020 | NA |
| 16 + 0 | 1.010 | NA |
| 16 + 6 | 1.010 | NA |
| 17 + 0 | 1.000 | NA |

*Source*: From Paley D, Bhave A, Herzenberg JE, Bowen JR. Multiplier method for predicting limb-length discrepancy. J Bone Joint Surg Am. 2000;82(10):1440 (Table 5). Reprinted with permission.
*Abbreviations*: NA, not applicable.
†The patient's limb-length inequality may be multiplied by a number from this table corresponding to age to determine the final discrepancy. It may also be used to calculate the amount of growth remaining in the lower limb.

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## The Multiplier Method for Growth Prediction

The multiplier method can be used to predict the growth of long bones or the entire stature as long as the growth is occurring naturally without any intervention. It uses the patient's own initial growth data and multiplies that by a figure known to be the proportion of growth remaining. Therefore, the growth of one limb or one bone can be calculated from just one measurement. If a growing patient has a limb-length discrepancy, that discrepancy is multiplied by the multiplier to calculate the final discrepancy. **Tables 1.3** and **1.4** present the multipliers for different ages.

## Alignment in the Transverse Plane

1. *Femoral anteversion* is the angle between the plane of the femoral head and neck and that of the posterior surface of the femoral condyles (**Fig. 1.29**). Femoral anteversion declines steadily in normal children from a mean of 25 degrees at birth to 15 degrees at adulthood (**Fig. 1.30**). It may be followed clinically by recording internal and external rotation of the hip in extension. It can be estimated in the clinic with the patient prone by internally rotating the hip until the greater trochanter is considered to be directly lateral and measuring the angle formed by the flexed tibia with the vertical (**Fig. 1.31**). Anteversion is most accurately measured by

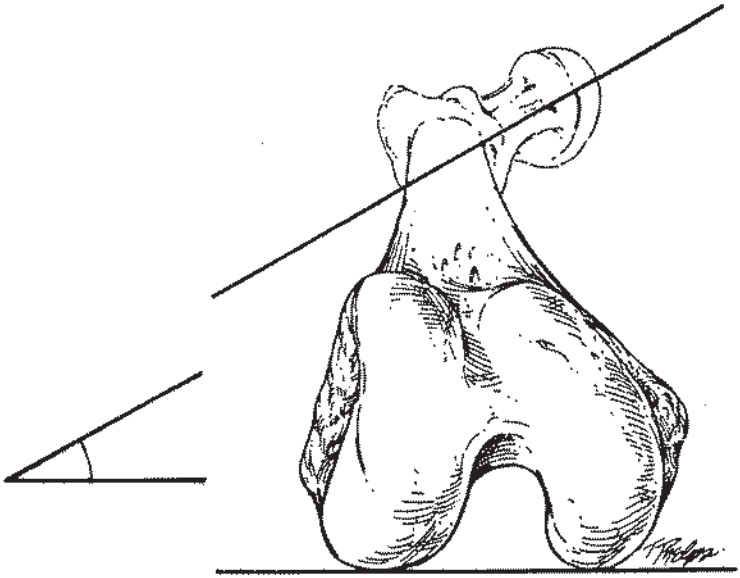


**Fig. 1.29** Femoral anteversion: definition.

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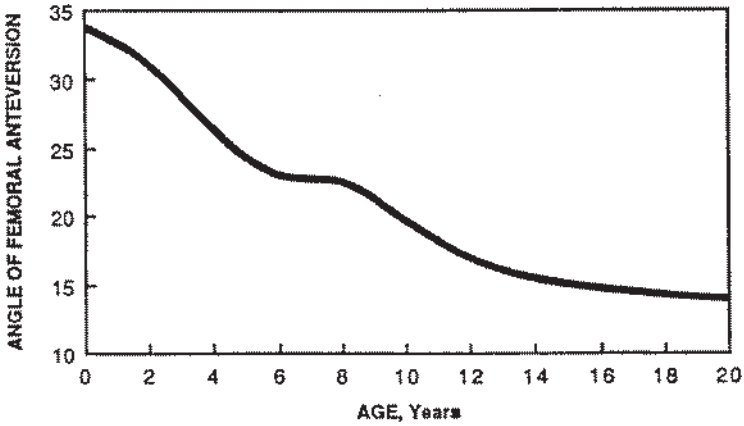



**Fig. 1.30** Normal values for femoral anteversion. (From Shands AR Jr, Steele MK. Torsion of the femur. J Bone Joint Surg Am. 1958;40(4):806 (Graph I). Reprinted with permission.)

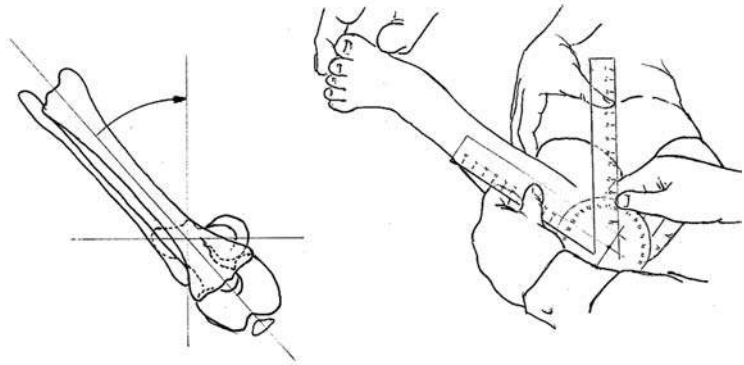


**Fig. 1.31** Clinical measurement of femoral anteversion. This may be estimated with the patient prone by rotating the hip internally until the greater trochanter has reached maximal prominence and then noting the angle formed by the tibia with a vertical line. (Clinical acccuracy described in Ruwe PA, Gage JR, Ozonoff MB, DeLuca PA. Clinical measurement of femoral anteversion. J Bone Joint Surg Am. 1992;74:820–830 [original drawing]).

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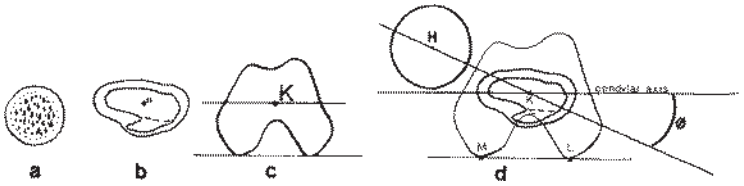



**Fig. 1.32** Measurement of anteversion by computed tomography. **(A)** Center of femoral head. **(B)** Center of base of femoral neck. **(C)** Condylar axis. **(D)** Anteversion is the angle between the two lines so defined. If the femoral head is posterior to the condylar axis, the value of the angle is negative and is termed retroversion. (From Murphy SB, Simon SR, Kijewski PK, Wilkinson RH, Griscom NT. Femoral anteversion. J Bone Joint Surg A.m 1987;69(8):1175 (Fig. 10). Reprinted with permission.)

computed tomography (CT). The method of Murphy is shown here (**Fig. 1.32**). Anteversion can also be measured by standard radiographs using techniques of Ogate or Magilligan.

2. CT measurement of anteversion: On sequential cuts by CT, a line is drawn through the centers of the femoral head and base of the neck. Another line is drawn through the posterior surfaces of the femoral condyles. The angle between these two lines is the femoral anteversion (**Fig. 1.32**).

## Bibliography

Magilligan DJ. Calculation of the angle of anteversion by means of horizontal lateral roentgenography. J Bone Joint Surg Am. 1956;38A(6):1231–1246
Murphy SB, Simon SR, Kijewski PK, Wilkinson RH, Griscom NT. Femoral anteversion. J Bone Joint Surg Am. 1987;69(8):1169–1176

3. Ogate method of determining femoral anteversion using biplane radiographs

   **a.** This method can be used when CT is not readily available. It uses graphs to provide trigonometric calculations of anteversion as well as true neck-shaft angle from two standardized plain radiographs (**Figs. 1.33** and **1.34**). It is built on the fact that the tibia is perpendicular to the condylar axis when the knee is flexed. The method is accurate when positioning is done carefully, to within ± 6 degrees for anteversion and ± 5 degrees for true neck-shaft angle.

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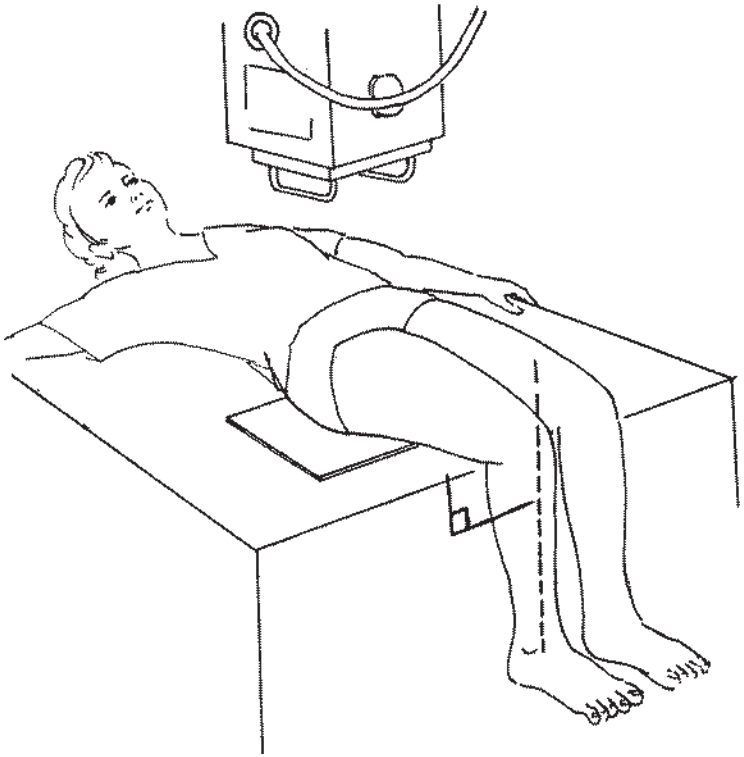


**Fig. 1.33** Positioning for the "projected AP femur X-ray" in the method of Ogate. The projected neck-shaft angle obtained from this film is labeled α and used in the next step in Figs. 1.35 and 1.36 (see).

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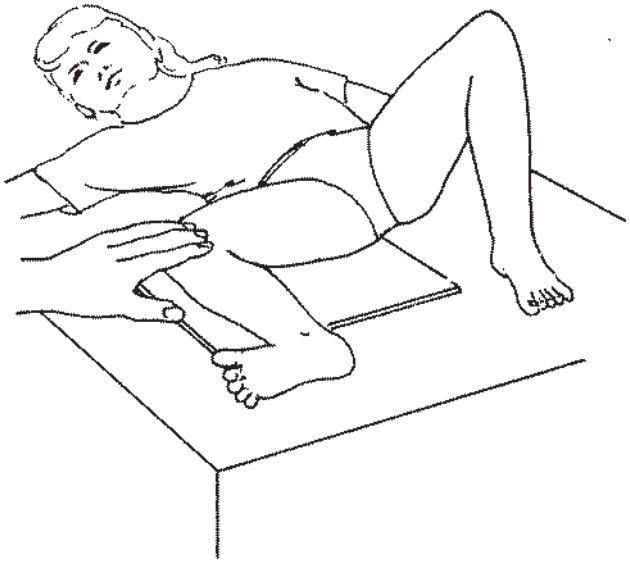


**Fig. 1.34** Positioning for the "projected lateral femur X-ray" in the method of Ogate. The tibia should be freely resting flat on the cassette. The projected neck-shaft angle obtained from this film is labeled β and is used in the next step in Figs. 1.35 and 1.36 (see).

**b.** Technique for obtaining radiographs: The patient is positioned with the knee flexed 90 degrees and perpendicular to the surface of the radiography table. This places the condylar plane of the femur in a true horizontal position. A projected anteroposterior neck-shaft radiograph is taken, and the angle is drawn and labeled α. The patient is then positioned on the side, with the knee flexed and the tibia horizontal. A projected lateral neck-shaft radiograph is taken, and the angle is drawn and labeled β.

**c.** Using the graphs in **Figs. 1.35** and **1.36**, values for true neck-shaft angle and anteversion can be obtained.

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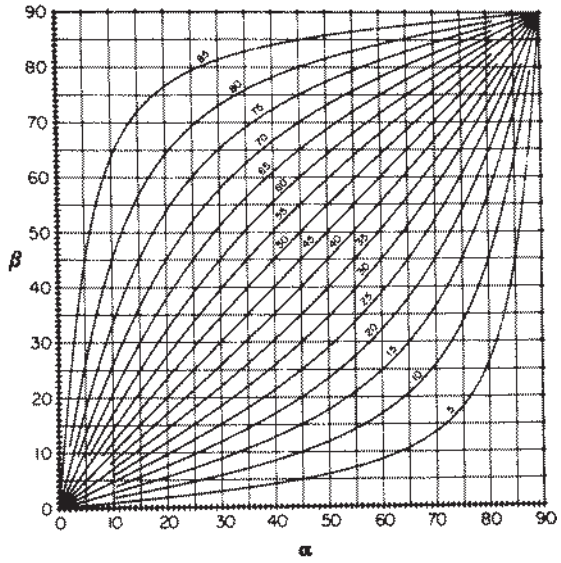



**Fig. 1.35** Determination of true anteversion of the femur using the intersection of projected anteroposterior (α) and lateral (β). (From Ogata K, Goldsand EM. A simple biplanar method of measuring femoral anteversion and neck-shaft angle. J Bone Joint Surg Am. 1979;61(6):849 (Fig. 5A). Reprinted with permission.)

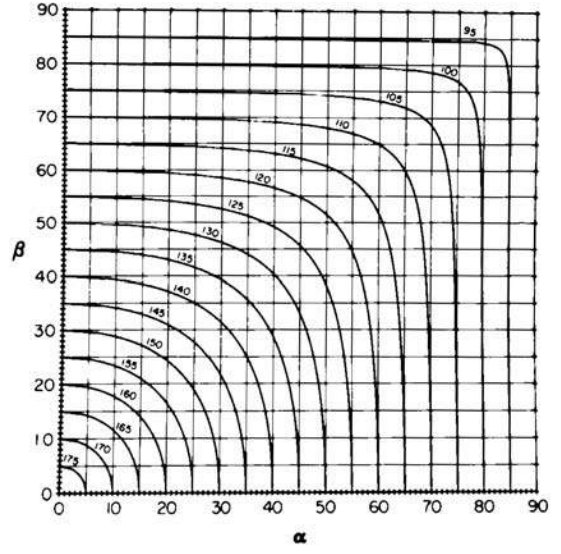



**Fig. 1.36** Determination of true neck-shaft angle. (From Ogata K, Goldsand EM. A simple biplanar method of measuring femoral anteversion and neck-shaft angle. J Bone Joint Surg Am. 1979;61(6):849 (Fig. 5B). Reprinted with permission.)

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## Development of the Tibiofemoral Angle During Growth

The coronal tibiofemoral angle changes dramatically during the first 5 years of life—from varus to excessive valgus to "normal" valgus. This was illustrated by Salenius in the graph redrawn in **Fig. 1.37**. It was made from clinical measurements on 1000 normal children. Radiographs are not generally necessary in the first 12 to 18 months of life. Chapter 2 details the interpretation of abnormal conditions.

*Alignment of the Lower Extremity*

1. Knowledge of normal tibiofemoral alignment is essential for planning osteotomies around the knee. Normal values vary slightly, depending on the width of the pelvis and limb lengths.
2. Definitions

- *Mechanical axis* is the angle of two lines between the centers of the hip, knee, and ankle; normal angle is 0 degrees, inclined 3 degrees from the vertical.
- *Anatomic axis* is the angle between the tibial and femoral diaphyses; normal is 6 degrees.
- The knee joint line should be horizontal.
- Femoral joint angle is 90 - (β + Θ)
- Tibial joint angle is 90 - Θ (**Fig. 1.38**)

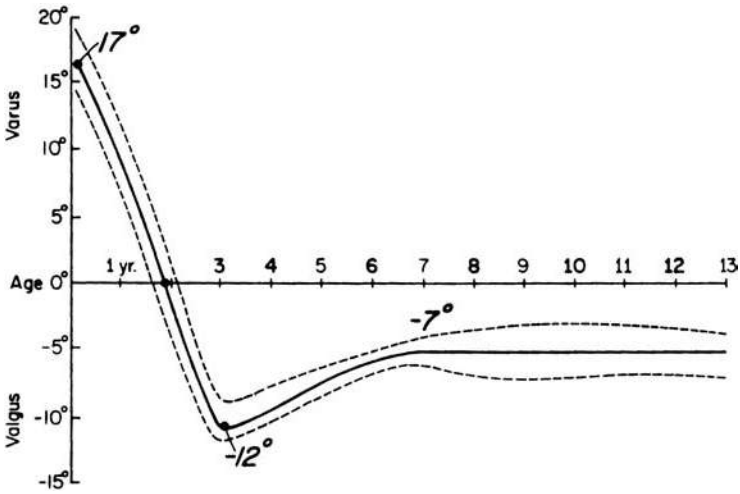



**Fig. 1.37** The tibiofemoral angle during growth. (From Salenius P, Vankka E. The development of the tibiofemoral angle in children. J Bone Joint Surg Am. 1975;57(2):260 (Fig. 1). Reprinted with permission.)

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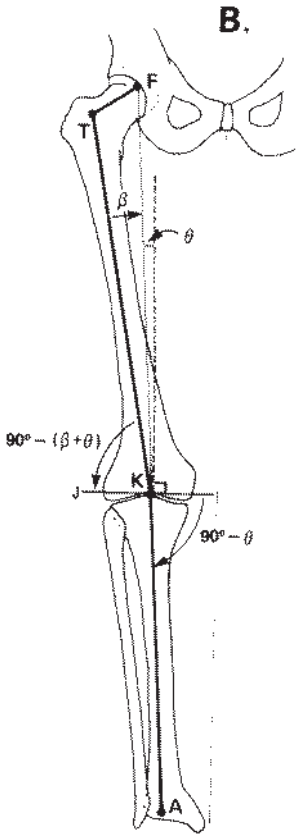



**Fig. 1.38** Alignment of lower extremity. In normal subjects, β = 6 degrees and Θ = 3 degrees; but these may vary depending on the distance between the hip centers, femoral neck-shaft angle, and limb length.

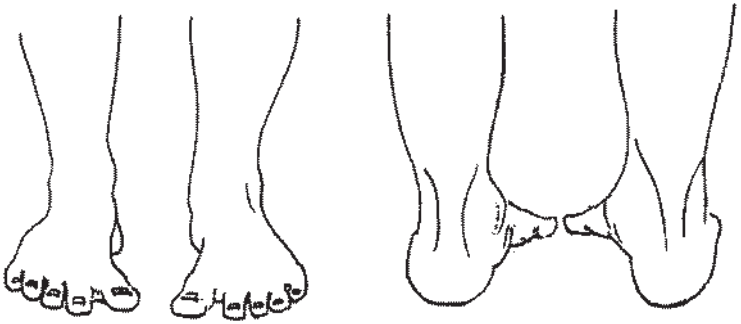


**Fig. 1.39** Appearance of metatarsus adductus.

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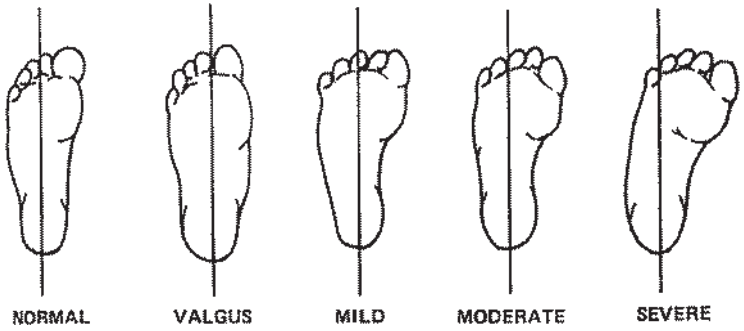



**Fig. 1.40** Quantification of metatarsus adductus. This is done with child prone. A line is drawn through the middle of the heel and examiner notes which toe it intersects. (From Bleck EE. Developmental orthopaedics. III: Toddlers. Dev Med Child Neurol. 1982;24(5):545 (Fig. 20). Reprinted with permission.)

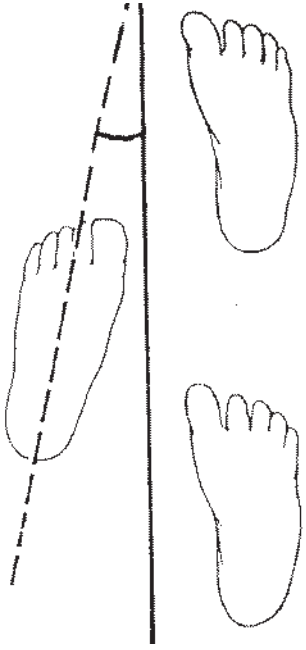


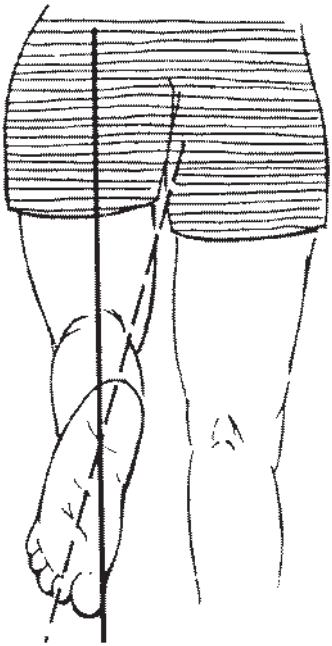
**Fig. 1.41** Measurement of the foot progression angle.

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## Clinical Evaluation of Rotation of the Lower Extremities

Rotational abnormalities can be approached in a systematic fashion. Foot progression angle quantifies the sum of rotations occurring in the femur, tibia, and foot. These components may be assessed by the parameters pictured on the following pages. Normal values throughout growth for all of these parameters are given, as determined by Staheli et al. Treatment for these rotational deformities is rarely indicated, but showing the natural progression to the parents in graphic form can be helpful.

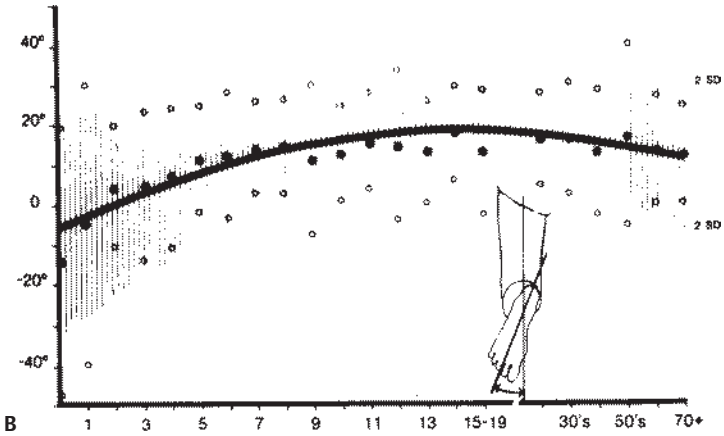
Metatarsal adduction and abduction (**Figs. 1.39** and **1.40**): Forefoot adduction is generally recorded from clinical measurements, but radiographic norms are available. Metatarsal adduction can be quantified by drawing a line through the heel bisector and noting which toe it intersects. Normally



A

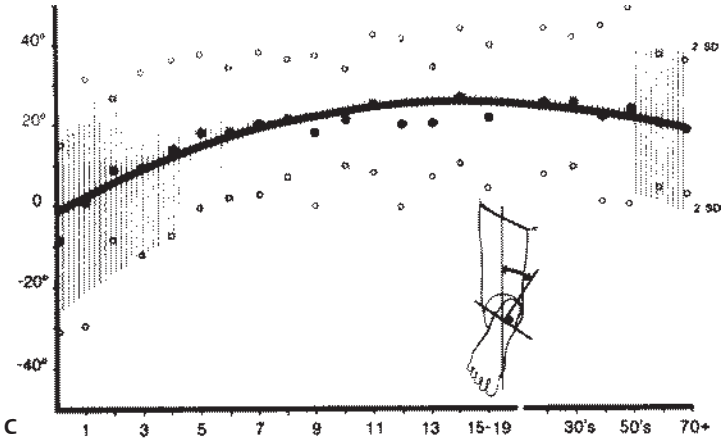
**Fig. 1.42** Measurement of the thigh-foot angle. **(A)** Clinical measurement. **(B)** Normal values with age. **(C)** Transmalleolar axis and normal values for age. ([B,C] from Staheli LT, Corbett M, Wyss C, King H. Lower-extremity rotational problems in children. J Bone Joint Surg Am. 1985;67(1):43 (Figs. 2E and 2F). Reprinted with permission.)

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Thigh-foot angle.

**Fig. 1.42** ***(continued)***




Angle of the transmalleolar axis.

**Fig. 1.42** ***(continued)***

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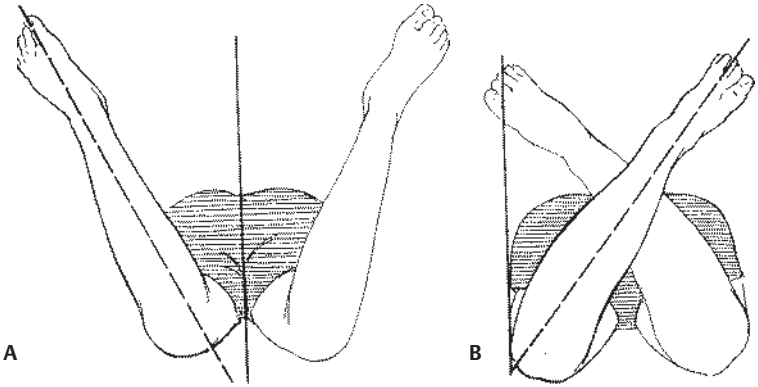



**Fig. 1.43** **(A)** Measurement of internal rotation. **(B)** Measurement of external rotation.

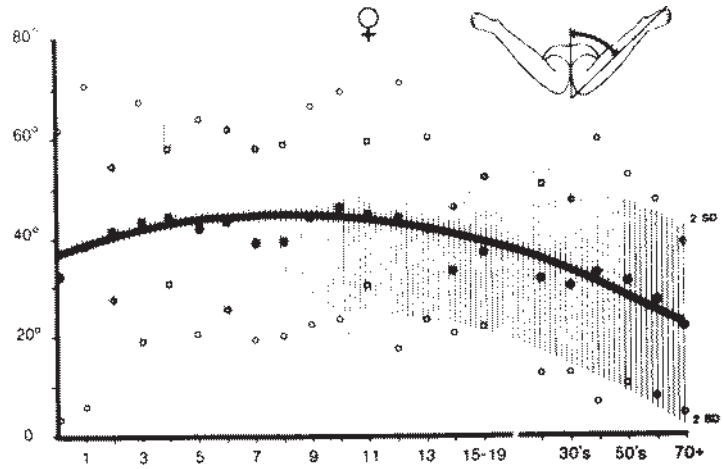
it falls between the second and third toes. Moderate to severe adduction is present when the line falls lateral to the fourth toe.

The foot progression angle is the end product of all rotational components in the lower extremity: hip, femur, knee, tibia, and foot. It is the angle formed by the foot (on the average) with the direction of walking (**Fig. 1.41**).

The *thigh–foot angle* is an approximate clinical measure of tibial torsion. It is assessed with the patient prone and the ankle gently dorsiflexed to a neutral position (**Fig. 1.42A**). Normal values with age are given in **Fig. 1.42B**. When the foot is affected by a rotational problem such as clubfoot, metatarsus adductus, spasticity, or contracture, the tibial torsion can be estimated by the *transmalleolar axis*. This is the angle formed between a line between the two malleoli and the thigh when the patient is prone with the knee bent (**Fig. 1.42C**).

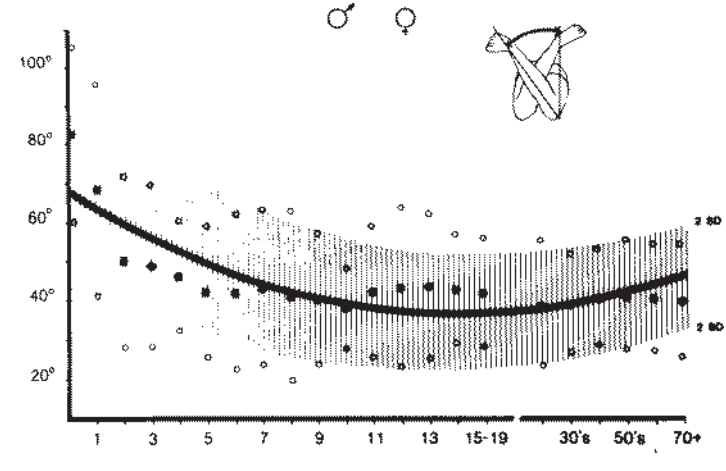
Internal and external rotation of the hip in extension are used to assess the relative contributions of the hip to rotation (**Fig. 1.43A,B**). Anteversion is likely to be present if internal rotation exceeds 70 degrees and external rotation is less than 20 degrees. Graphs of the normal range with age are given in **Fig. 1.43C,D**.

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C

Medial rotation of the hip in female subjects.




D

Lateral rotation of the hip in male and female subjects combined.

**Fig. 1.43** ***(continued)*** Hip rotation is usually measured in extension for assessment of torsion deformities. **(C)** Normal values for internal rotation with age. **(D)** Normal values for external rotation with age. ([C,D] from Staheli LT, Corbett M, Wyss C, King H. Lower-extremity rotational problems in children. J Bone Joint Surg Am. 1985;67(1):43 (Figs. 2C,D). Reprinted with permission.)

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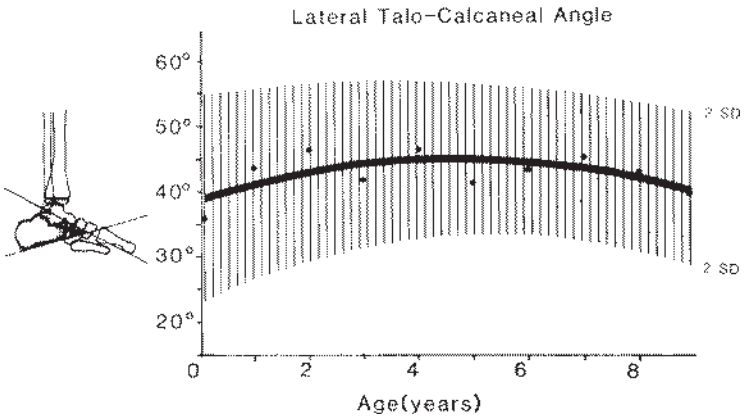



**Fig. 1.44** Lateral talocalcaneal angle in the standing foot. (From Vanderwilde R, Staheli LT, Chew DE, Malagon V. Measurements on radiographs of the foot in normal infants and children. J Bone Joint Surg Am. 1988;70(3):410 (Fig. 2D). Reprinted with permission.)

## Normal Radiographic Measurements of the Pediatric Foot

Radiographs are helpful in interpreting pathology in both the unoperated and the postoperative foot. The films should be taken with the patient standing, if possible. Normal values are given for the lateral talocalcaneal

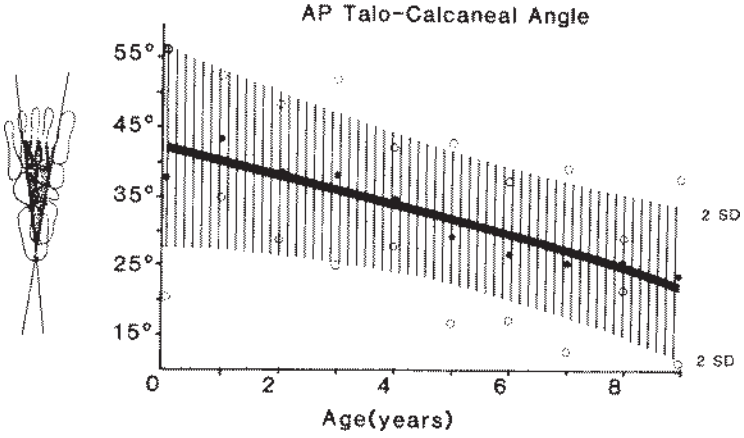



**Fig. 1.45** Anteroposterior talocalcaneal angle in the standing foot. (From Vanderwilde R, Staheli LT, Chew DE, Malagon V. Measurements on radiographs of the foot in normal infants and children. J Bone Joint Surg Am. 1988;70(3):409 (Fig. 2A). Reprinted with permission.)

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angle in **Fig. 1.44** and the anteroposterior talocalcaneal angle in **Fig. 1.45**. Note that a decline in the value of both angles is seen with a varus foot (increasing parallelism), and an increase is seen with a valgus foot (increasing divergence).

## ◆ Normal Gait in Children

Normal gait minimizes energy consumption. Gait deviations occur in response to a patient's neurologic or mechanical abnormalities. To understand gait by visual or laboratory analysis, it must be broken down into component characteristics.

### Definitions

1. *Normal walking gait* is 60% *stance* and 40% *swing*; therefore 20% is spent in *double support* (both limbs on the ground).
2. *Cadence:* Number of steps per unit of time
3. *Stride:* One cycle, including right plus left steps
4. *Kinematics:* Study of motion
5. *Kinetics:* Study of forces that produce movement
6. *Stance phase:* Period when one or both feet are on the ground
7. *First rocker:* First stage of ankle motion in stance, from heel strike to foot flat; decelerates inertia of body; tibialis anterior contracts eccentrically.
8. *Second rocker:* Second stage of ankle motion in stance, from foot flat till heel rise; deceleration of tibia to relax quadriceps; soleus contracts eccentrically.
9. *Third rocker:* Third stage of ankle motion in stance, from heel rise till toe off; accelerates limb; gastrocnemius and soleus contract concentrically.
10. Phases of gait cycle (**Figs. 1.46** and **1.47**).

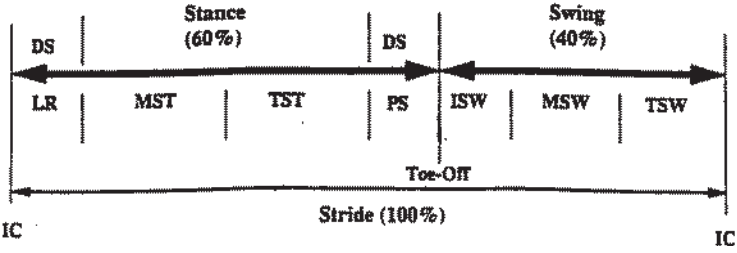



**Fig. 1.46** Phases of gait cycle. LR, loading response; MS, midstance; TST, terminal stance; PS, preswing; ISW, initial swing; MSW, midswing; TSW, terminal swing.

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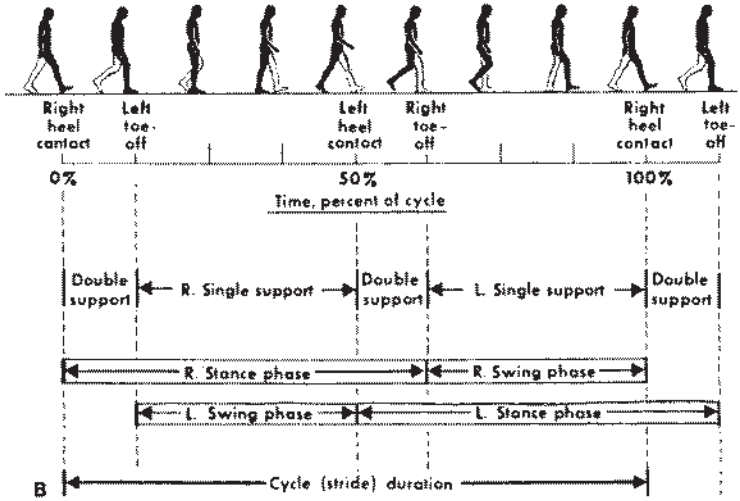



**Fig. 1.47** Phases of gait cycle with figures. (From Inman VT, Ralston HJ, Todd F. Human Walking. Baltimore: Williams & Wilkins; 1981;26 (Fig. 2.6). Reprinted with permission.)

## Normal Parameters of Gait

1. Mature gait: Fully developed by age 7
2. Velocity
   - 2-year-old: 0.78 m/sec ± 0.2
   - 4-year-old: 1.0 m/sec ± 0.2
   - 6-year-old: 1.1 m/sec ± 0.2
   - Adult: 1.5 m/sec ± 0.2
3. Cadence
   - 2-year-old: 180 steps/min
   - 4-year old: 160 steps/min
   - Adult: 116 steps/min
4. Hip flexion: Extension 0 to 40 degrees during normal walking
5. Knee flexion: Extension 5 to 60 degrees
6. Ankle: 5 to 20 degrees plantarflexion (see **Fig. 1.48** for graphs of normal joint motion during gait)

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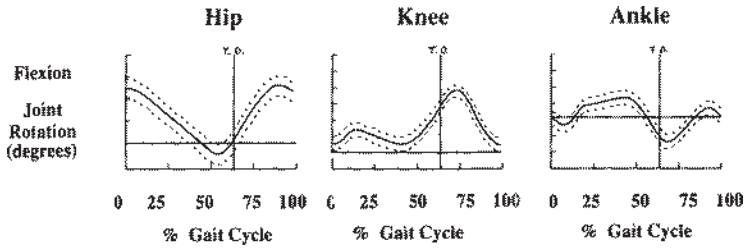



**Fig. 1.48** Normal joint kinematics at the hip, knee, and ankle. (From Gage JR. The clinical use of kinetics for evaluation of pathological gait in cerebral palsy. J Bone Joint Surg Am. 1994;76(4):626 (Fig. 8, part I). Reprinted with permission.)

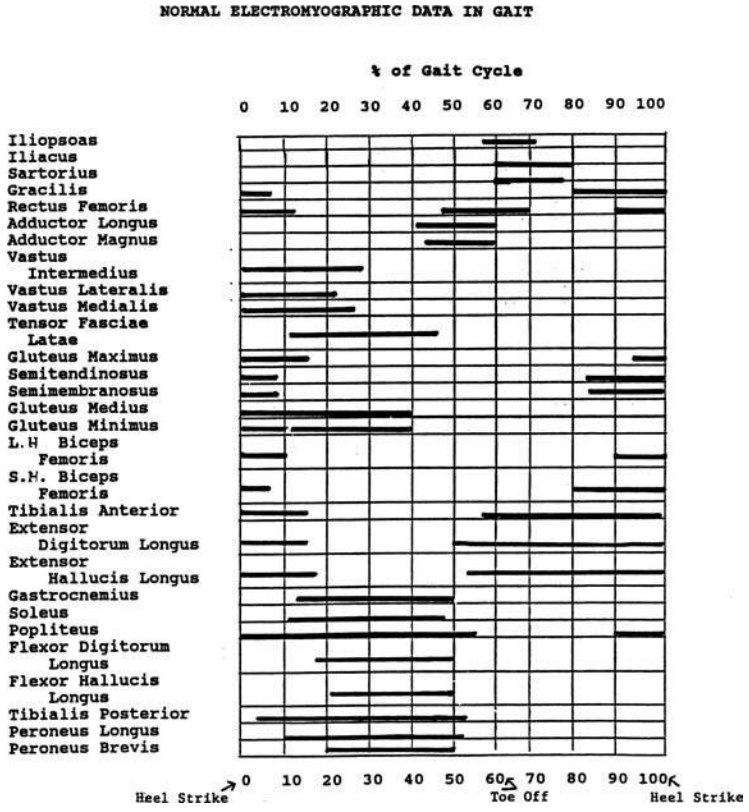



**Fig. 1.49** Muscle activity during gait.

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### Muscle Activity During Gait

1. Muscle activity may be measured by surface electrodes for large muscles or by fine wire electrodes for small muscles. A diagram of normal muscle activity is given in **Fig. 1.49**. Note that electromyelographic data give muscle activity and intensity but not the *force* of contraction.
2. Muscle control by phase:

   **a.** Heel strike: Gluteus maximus, hamstrings, tibialis anterior
   **b.** Loading response: Hamstrings, tibialis anterior, quadriceps, gluteus medius and maximus, adductor magnus
   **c.** Midstance: Soleus, quadriceps, gluteus maximus
   **d.** Terminal stance: Soleus, gastrocnemius, peroneals, toe flexors
   **e.** Preswing: Gastrocnemius, adductor longus, rectus femorus
   **f.** Initial swing: Hip flexors, tibialis anterior, toe extensors
   **g.** Midswing: Tibialis anterior
   **h.** Terminal swing: Hamstrings, quadriceps, tibialis anterior

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# 2 Disorders of Skeletal Growth and Development

Selected pediatric orthopedic conditions are summarized here with emphasis on central concepts and parameters. The goal is to provide a working knowledge for treatment. Principles and specific treatment details are covered more extensively in standard texts. Other major topics are covered in Chapters 3 (genetic syndromes), 4 (neuromuscular disorders), and 5 (trauma).

## ◆ Lower-Limb Length Inequality

### Principles

1. Inequalities of lower-limb length of up to 1 to 1.5 cm are within normal variation of the population.
2. Inequalities of less than 2.5 cm do not cause back pain or noticeable limp.
3. Length inequalities are a much less common cause of limp than are joint disorders (contracture, pain) or muscle weakness.
4. The gait disturbance caused by inequality of limb length is usually subtle and consists of pelvic drop and compensatory flexion of the knee of the long limb and equinus of the ankle of the short limb.
5. Most congenital inequalities of limb length behave in a proportionate fashion with growth; that is, the ratio of the short leg to the long leg is constant throughout growth.

### Congenital Causes

1. Congenital short femur, proximal focal femoral deficiency, tibial or fibular hemimelia, hemiatrophy, hemihypertrophy. Multiplier method predicts final discrepancy (see Chapter 1).
2. Systemic disorders: Ollier disease, fibrous dysplasia, osteochondromatosis, osteogenesis imperfecta, cerebral palsy.

### Acquired Causes

1. Developmental dysplasia of the hip (DDH) (length difference resulting from growth disturbance or osteotomy).
2. Legg–Calve–Perthes disease (may be up to 4 cm if proximal femoral physeal growth slows early.
3. Blount disease
4. Trauma (fracture overlap, growth arrest)
5. Osteomyelitis with physeal arrest
6. Foot deformities

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## Measurement

1. Block method: Palpate height of iliac crests in standing patient. Add height to short limb by measured blocks until equal. This is a good screening method to determine whether further, more precise measurements are needed. Although not as precise as radiographic measurements, it does take into account all factors such as contracture and shortening occurring within the foot or pelvis (**Fig. 2.1**).
2. Tape method: Measure from inferior margin of the anterior superior iliac spine (ASIS) to the medial malleolus. This method can be inaccurate in overweight patients or in those who have had anterior hip surgery and does not include foot discrepancies.
3. Scanogram: Images of both hips, knees, and ankles are taken in one position alongside a radiographic ruler. This method does not account for foot discrepancies or contracture.
4. Computed radiograph: Computes lengths and angles of segments.

## Treatment

1. If discrepancy is less than 2.5 cm in a mature person, no treatment is needed.
2. 2.5 to ~4 cm: Leg lift or epiphyseodesis or shortening of corresponding long segment (see section 1).

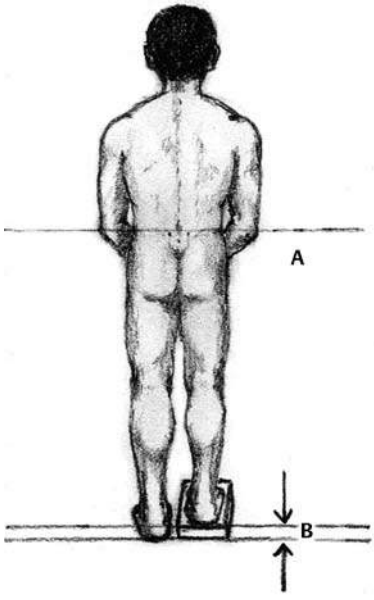



**Fig. 2.1** The standing block test. A block is added under the short limb **(B)** until the pelvis is palpated to be level **(A)**.

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3. >~4 to 5 cm: Limb lengthening if no contraindications or combination of lengthening and shortening and lift.

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## ◆ Developmental Dysplasia of the Hip

### Principles

Developmental dysplasia of the hip (DDH) is caused by forces acting on the hip in utero. The risk is increased by abnormalities of connective tissue. DDH is a spectrum, from hips that are subluxatable to dislocatable (Barlow positive) to dislocated (Ortolani positive). The combined incidence of these groups is 2 to 5 per 1000.

All hips should be screened by a knowledgeable examiner at birth and again within the first few months of life. The infant should be made as quiet and comfortable as possible for the examination, using warmth, contact, low light, feeding, or a pacifier. Abnormal physical signs marked with an asterisk (*) should prompt reexamination or ultrasound, with treatment if these are abnormal.

### Risk Factors in History

The following factors increase the risk of hip dysplasia in descending order and should prompt reexamination or ultrasound:

1. Positive family history of DDH
2. Breech position at the end of gestation (5% are unstable). A breech female should have a screening ultrasound.
3. Large birth weight

### Physical Examination for Developmental Hip Dysplasia in the Newborn

1. Appearance at rest: The affected side is more adducted at rest in unilateral cases and may have a deeper or extra high fold proximally.

*2. Asymmetric passive abduction: Dislocated hip will lack passive abduction compared with the normal side (**Fig. 2.2A**).

*3. The Barlow test will cause pistoning of the proximal femur if dislocatable (**Fig. 2.2B**).

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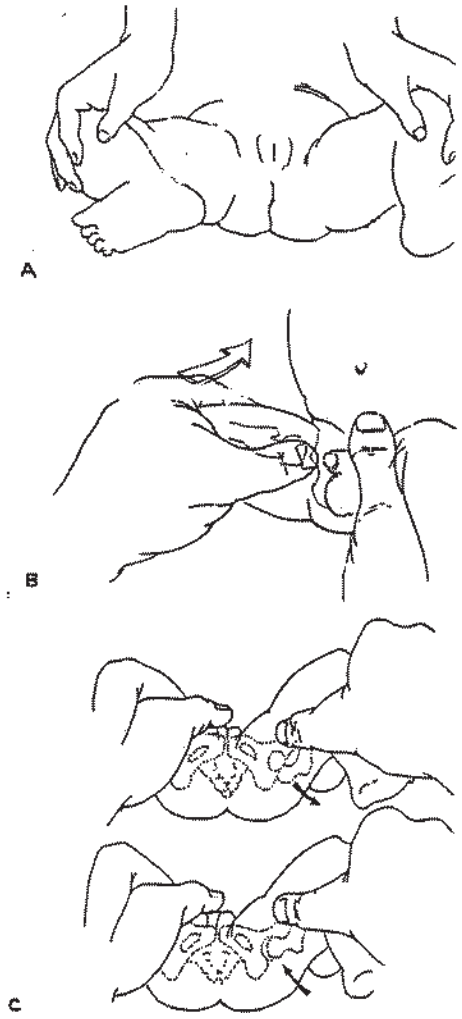



**Fig. 2.2** **(A)** Asymmetric abduction; left side is dysplastic. **(B)** Barlow test (done on one hip at a time). **(C)** Ortolani test.

***4.** The Ortolani test will cause a "clunk" as a dislocated hip is relocated. Examine each hip separately; stabilize the pelvis with the other hand (**Fig. 2.2C**). Note that the Ortolani and Barlow tests are for *translation* of the femur. A "click" *per se* is not a positive test; only 1% of patients with a click have dysplasia. A click may come from the patella or the meniscus of the knee as well as from the fascia lata or a synovial fold in the hip.

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5. Proximal location of greater trochanters is a helpful sign in diagnosing a patient with bilateral irreducible hip dislocations. Also, the femoral head may cause a prominence superiorly.
6. Significant foot deformity or torticollis may increase the risk of hip dysplasia and should prompt a careful examination of the hips.

## Evaluation of the Older Child for Developmental Dysplasia of the Hip

In the older child with hip dysplasia, the signs progressively change. Reducibility of the hip in the awake patient is lost after about 3 months, and one must rely more on indirect signs:

1. Asymmetric passive abduction
2. Galeazzi test will show thigh shortening on the side that is dislocated (**Fig. 2.3A**). The pelvis should be kept level during this test.
3. Leg-length discrepancy
4. Trendelenburg gait
5. Palpable femoral head posterior to the acetabulum
6. Nélaton line (an imaginary line between the ASIS and the ischium) should lie superior to the trochanter (**Fig. 2.3B**).
7. Klisic line between the greater trochanter and the ASIS should project cephalad to the umbilicus (**Fig. 2.3C**).
8. Increased lumbar lordosis (if bilateral) is due to posterior displacement and mechanical disadvantage of hip abductors.

## Ultrasound for Hip Dysplasia

*Principles*

The role of ultrasound in diagnosis of dysplasia varies regionally. Its benefit is its ability to show cartilage and other soft tissues, as well as observe stability in response to stress. Interpretation of an ultrasound considers both static and dynamic findings.

The ultrasound view is named according to the direction of the transducer—transverse or coronal—and the position of the hip—neutral or flexion. The highest frequency possible (3 to 7 MHz) will give the best resolution, but this must usually be reduced with age to obtain adequate penetration.

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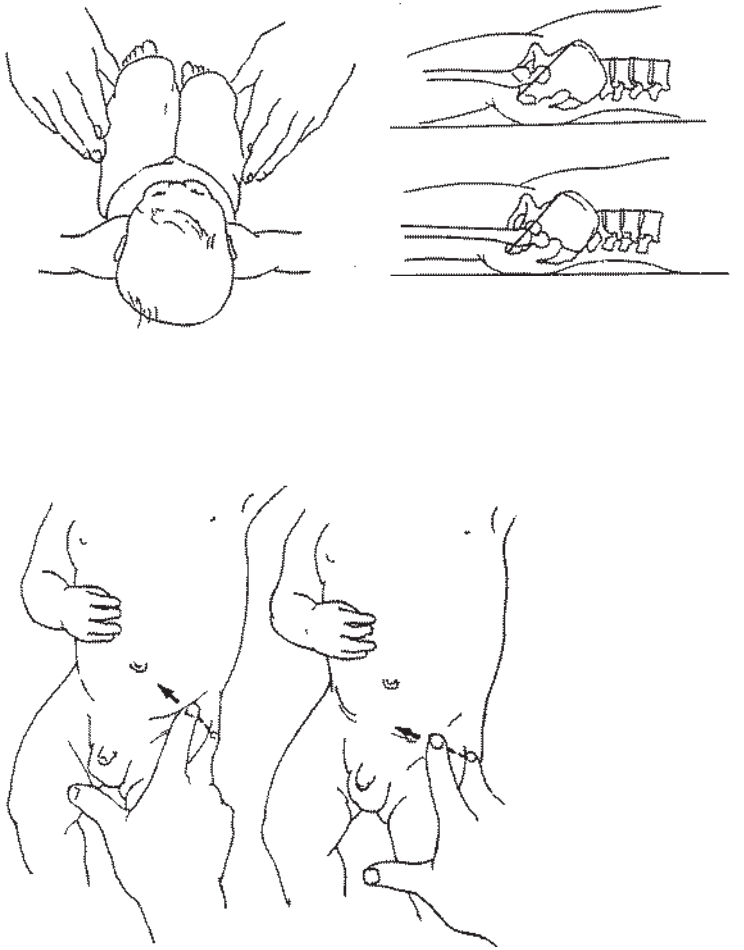


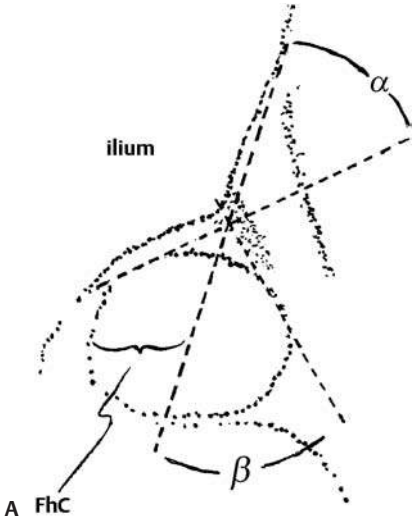
**Fig. 2.3** **(A)** Galeazzi sign shows apparent thigh shortening on dysplastic side (right). **(B)** If dislocated, the greater trochanter will lie proximal to Nélaton line (anterior superior iliac spine to ischium). **(C)** Klisic line in the normal hip falls above the umbilicus.

*Coronal View*

In this view, the landmarks are similar to those seen on a plain radiograph, when the transducer is in the mid acetabular plane.

1. The stability and gross appearance are the most important features.
2. The following other parameters may be checked (**Fig. 2.4**):

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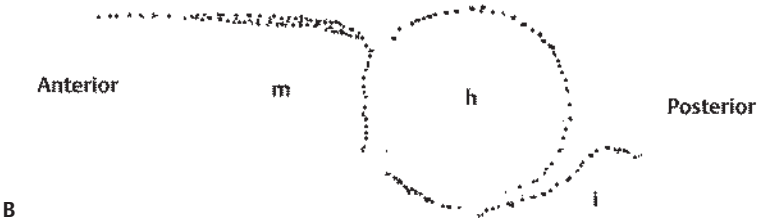




**Fig. 2.4** **(A)** Ultrasound, coronal view. FhC, femoral head coverage. α angle, acetabular roof line. β angle, slope of labrum. **(B)** Transverse view. Note the "U" formed by the metaphysis and the acetabulum.

- **a.** Femoral head coverage: The percent of the femoral head medial to the outer line of the ilium. This should be greater than 50%.
- **b.** Alpha angle, or acetabular roof line, between the lateral ilium and the bony acetabular roof. It is analogous to the acetabular index and should be greater than 60 degrees.
- **c.** Beta angle, or slope of the labrum versus the lateral wall of the ilium. It should be less than 55 degrees, indicating a downward slope of the labrum.

*Transverse View*

In this view the hip is flexed and the transducer is placed posterolaterally in the transverse plane of the body. The combination of echoes from the femoral

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metaphysis and the acetabulum normally form a "U." When dislocated, the femoral head comes to lie lateral and posterior to the acetabulum, and the U is disrupted.

## Radiographic Evaluation of Hip Dysplasia

1. Grades of hip dislocation according to Tonnis indicate the position of the ossific nucleus relative to Perkin vertical line (p) and Hilgenreiner horizontal line (h) (**Fig. 2.5**).

   **a.** Nucleus medial to Perkin line
   **b.** Nucleus lateral to Perkin line
   **c.** Nucleus at Hilgenreiner line
   **d.** Nucleus above Hilgenreiner line

2. The *acetabular index* is the angle formed between the Hilgenreiner line and the inner and outer borders of the acetabular roof (**Fig. 2.6A**, right hip). It is useful in assessing hip development in early years, before the center of the femoral head can be accurately identified. The normal values are shown in **Fig. 2.6B**.
3. Center edge angle of Wiberg (CEA)

   **a.** CEA measures the coverage of the femoral head by the acetabulum (**Fig. 2.6A**, left hip). Long-term follow-up studies by Wiberg have

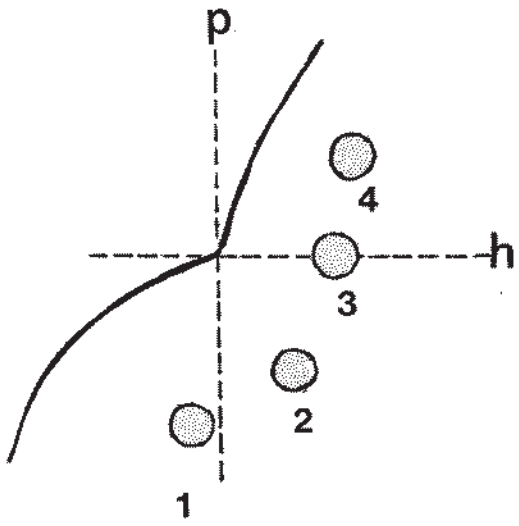



**Fig. 2.5** Tonnis grades of hip dislocation (p = Perkins' line and h = Hilgenreiner's line).

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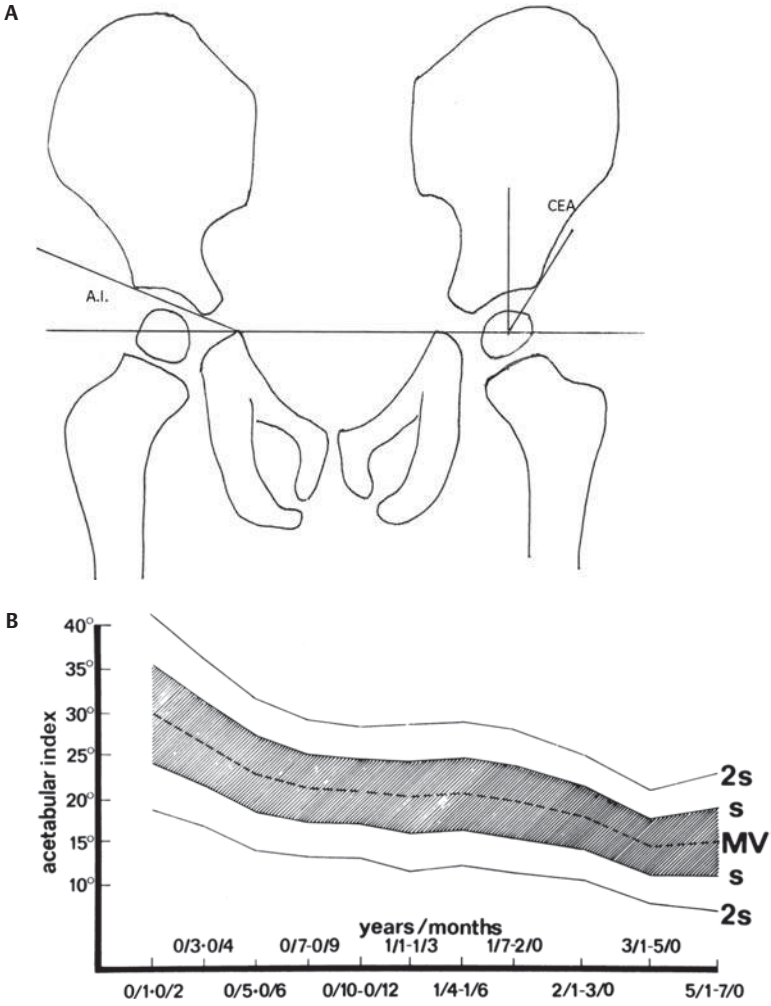



**Fig. 2.6** **(A)** Acetabular index measurement (right hip) and measurement of center edge angle of Wiberg (left hip). **(B)** Acetabular index normal values for age. (Part B from Tonnis D. Normal values of the hip joint for the evaluation of x-rays in children and adults. Clin Orthop Relat Res. 1976;119:41 (Fig. 2). Reprinted with permission.)

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shown a correlation between development of symptoms after maturity and CEA below 20 degrees.

**b.** Normal values

- Lower limit of normal
  - 5 to 8 years: 19 degrees
  - 9 to 12 years: 25 degrees
  - 13: 26 degrees
  - Less precise under 5 years

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### Management of Hip Dysplasia

The following is a general algorithm for management of dysplasia (**Fig. 2.7**). Guidelines given may be modified based upon individual factors.

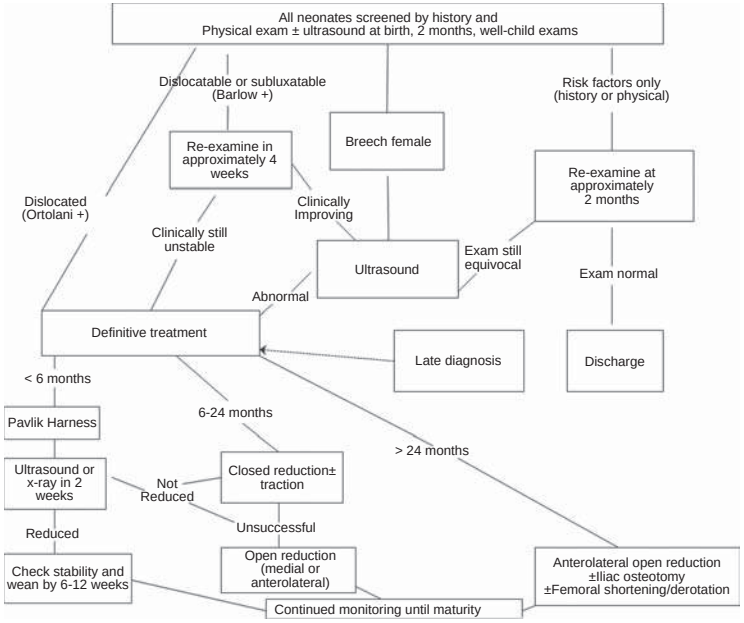



**Fig. 2.7** General algorithm for management of pediatric hip dysplasia.

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## ◆ Legg–Calve–Perthes Disease

Legg–Calve–Perthes disease (idiopathic avascular necrosis [AVN] of the immature femoral head) is most commonly seen in children aged 4 to 10 years. Five percent of patients develop bilateral involvement, but this is almost always at different times (asynchronous). Synchronous involvement should suggest the possibility of skeletal dysplasia, hypothyroidism, or steroid use.

### Symptoms

1. Minimal or no history of trauma history
2. Stiffness
3. Intermittent mild pain or no pain at all

### Signs

1. Mild Trendelenburg gait
2. No pain with gentle motion
3. Limitation of extremes of motion, especially abduction and internal rotation

### Radiographic Findings

1. Chronological sequence
    - **a.** Initially may appear normal
    - **b.** Failure of nucleus to grow versus opposite side
    - **c.** Subchondral "crescent" sign best seen on lateral view, present in one third of cases
    - **d.** Fragmentation of nucleus with resorption
    - **e.** Epiphyseal extrusion outside of acetabulum
    - **f.** Physeal and metaphyseal irregularities
    - **g.** Later, reossification and variable remodeling
    - **h.** Usually loss of epiphyseal and neck height at maturity
2. Staging
    - **a.** Catterall (**Fig. 2.8**)

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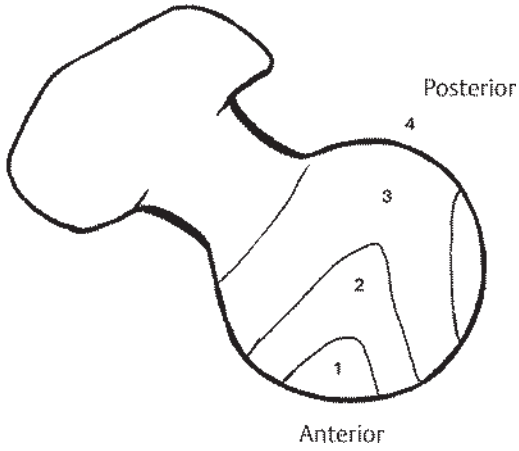



**Fig. 2.8** Catteral classification of Perthes disease as viewed from above.

**1)** Central anterior involvement of head only
**2)** Greater central head involvement but intact medial and lateral column
**3)** Lateral three quarters of femoral head involved with only intact medial column; metaphyseal reaction
**4)** Whole-head involvement, with metaphyseal reaction and remodeling of epiphysis

**b.** Herring Lateral Pillar Classification predicts flattening during healing (**Fig. 2.9**).
**1)** Lateral pillar is intact without radiographic change.
**2)** Lateral pillar is collapsed, but height is still greater than 50%.
**3)** Lateral pillar is collapsed to less than 50% of original height.

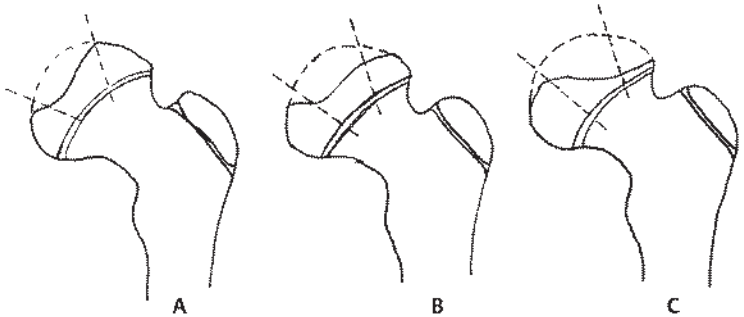



**Fig. 2.9** Herring lateral pillar classification of Perthes.

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3. Prognostic signs
   a. "Head-at-risk signs" of Catterall
      1) Lateral calcification
      2) Lateral subluxation
      3) Gage sign: Lucency proximal and distal to lateral physis
      4) Metaphyseal reaction
      5) Horizontal physis (meaning limb is adducted)
   b. Epiphyseal extrusion (**Fig. 2.10**) greater than 20% carries poor long-term prognosis.
   c. Mose sphericity: Deviation of head periphery from a perfect sphere by more than 3 mm on anteroposterior (AP) and lateral radiograph carries poor long-term prognosis.
   d. Stulberg rating (used after healing) assesses femoral head sphericity and its congruency with the acetabulum. There are five different stages. These have been correlated with long-term outcome (**Fig. 2.11**).
4. Arthrogram, magnetic resonance imaging (MRI): These are not routinely indicated but may be helpful in selected cases.

## Differential Diagnosis

1. Hypothyroidism
2. Multiple epiphyseal dysplasia
3. Spondyloepiphyseal dysplasia

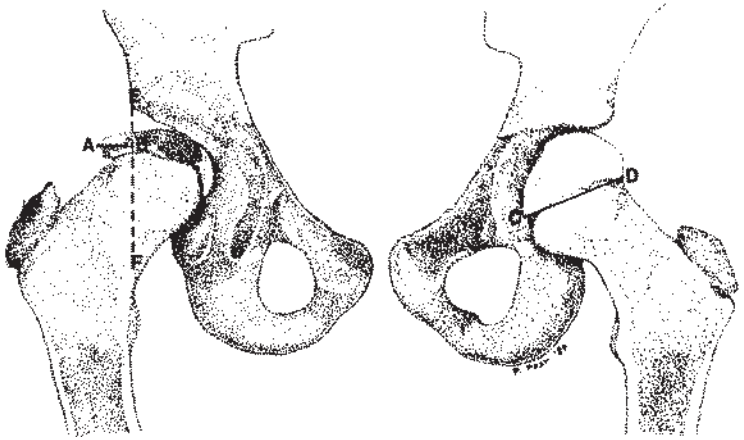



**Fig. 2.10** Measurement of epiphyseal extrusion. (From Green NE, Beauchamp RD, Griffin PP. Epiphyseal extrusion as a prognostic index in Legg-Calve-Perthes disease. J Bone Joint Surg Am. 1981;63(6):902 (Fig. 1). Reprinted with permission.)

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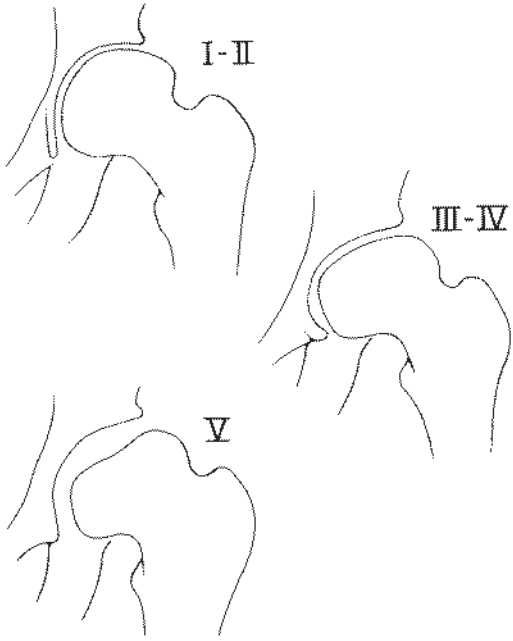



**Fig. 2.11** Stulberg rating of outcome of healed Perthes. Stages I–II are spherical and congruent and have a low likelihood of degenerative joint disease. Stages III and IV are aspherical and congruous and have a risk of degenerative joint disease by middle age. Stage V is aspherical and incongruous, and there is a risk of degenerative joint disease before 50. Source: Stulberg SD, Cooperman DR, Wallensten R. The natural history of Legg-Calve-Perthes disease. J Bone Joint Surg Am. 1981;63(7):1095–1108.

4. Meyer dysplasia (bilateral synchronous early childhood AVN; better prognosis)
5. Storage disorder (Gaucher disease, mucopolysaccharidoses)
6. AVN following trauma, steroids, sickle cell infarct, DDH treatment

### Treatment

1. There is no consensus on a protocol. However, many hips have a poor long-term natural outcome, and some hips appear to be helped by treatment. Containment is indicated if several of the following features are present:

    a. Head involvement greater than 50% (Catterall 3–4, Herring B or B/C border)

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**b.** Age over 8 years
**c.** Collapse or extrusion not established yet

**2.** Containment options

**a.** Abduction brace or Petrie casts
**b.** Femoral varus osteotomy
**c.** Iliac rotational osteotomy or augmentation
**d.** Combinations of b and c

**3.** Late options

**a.** Epiphysiodesis for leg length inequality greater than 2 cm
**b.** Valgus osteotomy for symptomatic hinge abduction
**c.** Trochanteric transfer for persistent abductor weakness
**d.** Epiphyseal osteotomy for femoral incongruity

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## ◆ Transient Synovitis of the Hip

### Overview

**1.** Transient synovitis of the hip is characterized by the acute onset of monarticular hip pain, limp, and restricted hip motion.
**2.** It is the most common cause of hip pain in children.
**3.** Child's age is usually 1 to 4 years, but any age can be affected.
**4.** This condition must be distinguished from septic arthritis.
**5.** Gradual but complete resolution over several days to weeks is the norm.
**6.** Cause is unknown, but it may be immune-mediated.

### Diagnosis

**1.** A diagnosis of exclusion
**2.** Acute onset of unilateral hip pain in an otherwise healthy child
**3.** The patient may be afebrile or have a low-grade fever.

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4. Laboratory values are nonspecific and are often within normal limits.

### Physical Examination

1. Limp and antalgic gait are common.
2. Most patients can bear weight on the involved extremity with assistance.
3. Hip is held in a flexed, externally rotated position. Restricted range of motion, especially abduction and rotation; slow movement is better.
4. Pain is not as great as with septic arthritis.

### Laboratory Tests

1. Laboratory tests are usually nonspecific and within normal limits, but they may help to rule out other diagnoses.
2. Peripheral white blood cell count is normal to slightly elevated.
3. Erythrocyte sedimentation rate averages 20 mm/hour but may be slightly higher.
4. Urinalysis, blood culture, rheumatoid factor, and Lyme titers results are usually within normal limits.
5. Aspiration of joint fluid is not needed if presentation is typical. If done, results are nonspecific.

### Imaging

1. Plain films of the hip: AP and lateral views.
2. In transient synovitis they are normal can but help rule out other diagnoses.
3. Ultrasound may be used to assess for effusion and to guide aspiration if infection cannot be ruled out clinically.
4. MRI is needed only in cases of persistent pain.

### Differential Diagnosis

1. Septic arthritis
2. Osteomyelitis in the femoral neck or pelvis
3. Tuberculous arthritis
4. Psoas abscess
5. Other muscle infection about the hip
6. Juvenile rheumatoid arthritis
7. Idiopathic chondrolysis
8. Acute rheumatic fever
9. Legg-Calvé-Perthes disease
10. Tumor
11. Sacroiliac joint infection

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### Treatment

1. Bed rest at home if diagnosis is clear or in hospital if further workup is needed.
2. Nonsteroidal anti-inflammatory drugs (NSAIDs)
3. Prompt improvement should be seen.
4. Activity as tolerated when clinically improved

### Prognosis

1. Good; no clear evidence of increased risk of AVN

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## ◆ Slipped Capital Femoral Epiphysis

### Background

1. Incidence: 2 to 10 per 100,000
    a. Higher in males than females
    b. Higher in African Americans
    c. 20% have bilateral involvement at presentation
    d. 20% more bilateral later
2. Etiologic factors
    a. Obesity
    b. Trauma: Mild or severe
    c. Endocrine disorders: Hypothyroidism, hypogonadism, rickets, renal failure

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**d.** Down syndrome
**e.** Family history
**f.** Radiation

### Classification

**1.** Loder classification

- Stable: Able to bear weight (even with crutches)
- Unstable: Unable to bear weight

**2.** Chronologic

- Acute: Symptoms of less than 3 weeks' duration
- Chronic: Symptoms for 3 weeks or longer

**3.** Severity

- Grade I: Less than 33% slip of epiphysis on metaphysis
- Grade II: 33 to 50% slip
- Grade III: More than 50% slip
- "Pre-slip": Symptoms are present in patient at risk, but no observable slip is seen; MRI may be positive.

### Clinical Presentation

**1.** Age 9 to 14 years, most common
**2.** Antalgic limp
**3.** Pain in the thigh, knee, or hip
**4.** Leg externally rotated during gait and at rest
**5.** Internal rotation less in flexion than extension
**6.** Seasonal variation: Highest rates in September, lowest in March because of sunlight and vitamin D cycles
**7.** Age–weight test: If age is younger than 10 or older than 16 years or weight is less than 50 percentile, suspect nonidiopathic slip and perform an endocrine workup.
**8.** Height test: If height is below 10th percentile for age, risk of atypical slip is increased increased; perform an endocrine workup.

### Radiographic Features

**1.** Slip is best seen on lateral view.
**2.** AP view

**a.** Physeal widening, irregularity
**b.** Decreased epiphyseal height

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**c.** "Klein's line": Line on lateral femoral neck with slipped capital femoral epiphysis (SCFE) transects less than 20% of epiphysis in child older than 10 years.
**d.** Chondrolysis–joint space narrowing may be seen before treatment (rare).

### Treatment

1. Immediate weight relief (bed rest)
2. Traction for acute slip for comfort or reduction if severe (optional).
3. Fixation in situ

   **a.** Single screw is centrally placed within physis.
   **b.** Second screw may be used if first is not perfect or if slip is severe.

4. Realignment: Main indication is the patient who is dissatisfied with limb deformity resulting from slip; it is not commonly needed.

   **a.** Open realignment and pinning of acute slip
   **b.** Cuneiform osteotomy just below physis
   **c.** Base of neck osteotomy: some series show high AVN rate.
   **d.** Subtrochanteric osteotomy (Southwick)

5. Prophylactic contralateral pinning

   **a.** Cost–benefit studies show prophylactic pinning is justifiable (at the surgeon's discretion).
   **b.** Main indication is a patient in whom diagnosis of late contralateral SCFE may be missed as a result of impaired communication or follow-up.
   **c.** Also valid option in patients with SCFE before growth spurt (in girls younger than 10 years, in boys younger than 12 years)
   **d.** If triradiate cartilage is closed or in a girl older than 13 years or in a boy older than 14 years, there is a low risk of subsequent slip (~7%).

### Complications

1. Chondrolysis: Affects 5% or less and usually improves with time and physical therapy
2. AVN

   **a.** Greater in unstable or acute slips
   **b.** May be focal or complete
   **c.** Some healing is possible in young patients.
   **d.** Some patients can function 10 to 20 years with AVN before salvage is needed.

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## ◆ Developmental Coxa Vara

A varus deformity of femoral neck that is usually progressive. Incidence is 1 in 25,000. Thirty percent of cases are bilateral.

### Radiographic Features

1. Femoral neck shortened and in varus
2. Inverted "Y" appearance of physis as a result of shear plane through metaphysis
3. Triangular fragment on inferior femoral neck
4. Decreased femoral anteversion
5. Mild acetabular dysplasia

### Signs and Symptoms

1. Abnormal gait caused by abductor weakness and leg-length inequality
2. Activity-related hip pain
3. Length inequality if unilateral: Usually less than 2.5 cm

### Differential Diagnosis

1. Cleidocranial dysplasia
2. Metaphyseal dysplasia
3. Morquio syndrome
4. idiopathic

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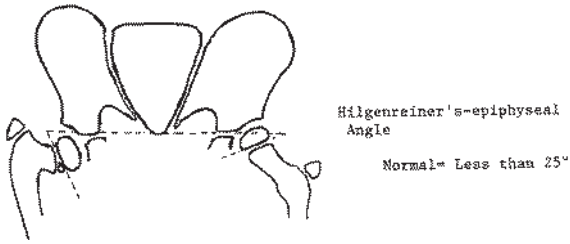



**Fig. 2.12** Hilgenreiner–epiphyseal angle; normal is less than 25 degrees. Note "inverted Y" appearance of physis on involved right hip.

### Treatment

1. Observe if Hilgenreiner-epiphyseal angle is less than 45 degrees (**Fig. 2.12**)
2. Valgus-derotation osteotomy if greater than 45 degrees and progressive and symptomatic or if greater than 60 degrees at diagnosis

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## ◆ Proximal Focal Femoral Deficiency

A spectrum of congenital femoral anomalies including a temporary or permanent discontinuity in the proximal femur

### Classification (Aitken) (Fig. 2.13)

1. A: Femur is in continuity but appears discontinuous early.
2. B: Varus/shortening is more extreme, but most ossify later.

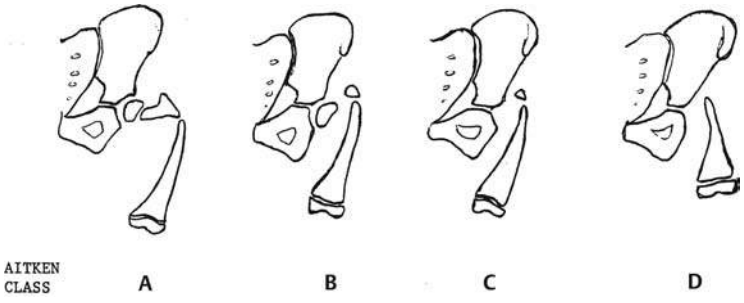



**Fig. 2.13** Proximal focal femoral deficiency: Aitkin classification.

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3. C: Small acetabulum but no femoral head
4. D: No femoral head or acetabulum

Other classification systems exist but are less widely used.

### Characteristics

1. 15% are bilateral (most type D).
2. More than 50% have other lower extremity anomalies, most commonly fibular hemimelia.
3. Affected limb is a constant proportion to the length of the normal limb.

### Problems

1. Limb-length inequality (if unilateral)
2. Pelvic–femoral instability
3. Malrotation of lower extremity (flexion–abduction–external rotation)
4. Proximal muscle weakness

### Treatment

1. Bilateral
    a. Patients walk without prostheses if feet are all right.
    b. Nonfunctional feet may need surgery.
    c. Extension prostheses may be used when desired to increase height.
2. Unilateral
    a. Hip abnormalities: Valgus osteotomy and lengthening for types A and B; consider femoropelvic arthrodesis for type D versus containment of thigh segment in a prosthesis.
    b. Knee: Offer rotationplasty if foot and ankle are strong and positioned distal to the contralateral knee. Syme disarticulation is another option. Both are combined with fusion of anatomic knee to increase lever arm.
    c. Foot: Prosthesis equalizes length and provides plantigrade foot (**Fig. 2.13**).

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## ◆ Bladder Exstrophy

A spectrum of anomalies that may involve the bladder, pelvis, intestinal tract, and external genitalia.

1. The *most common* form is "classic" exstrophy, which involves a widened pelvis with an anterior diastasis, an open bladder, and a complete epispadias.
2. The *mildest* form is epispadias, which may have a closed bladder but widened pelvic symphysis.
3. The *most pronounced* expression of this spectrum is cloacal exstrophy, which usually involves all of the above as well as omphalocele and a lumbosacral neural tube defect. It often includes anomalies of the spine and extremities.
4. The incidence of bladder exstrophy is around 1:25,000 live births. Males are more commonly affected.

### Clinical Features

1. Defect in lower abdominal wall
2. Open bladder and urethra
3. In cloacal exstrophy, abdominal wall defect is larger and lower intestinal tract is exposed.
4. A spinal and a neurological examination should be performed. Often in cloacal patients there is lipomeningocele or myelomeningocele. Hip dislocation, foot deformity, or partial sacral agenesis may occur.

### Radiographic Features

Separation of pubic bones, typically about 4 to 5 cm at birth, and increases steadily with age (**Fig. 2.14**). The iliac wings are externally rotated and "flattened." The ischiopubic bones are slightly underdeveloped. The hips themselves rarely show dysplasia.

### Treatment

1. The urologist usually performs the reconstruction in several stages, including closure of the bladder and lower abdominal wall soon after birth, followed by epispadias closure at the same time or at a later date. Surgery to achieve continence is commonly performed after the age at which children are normally continent and may consist of bladder neck suspension.
2. Orthopedic surgery of the pelvic deformity is indicated only if it is needed to achieve urologic goals.

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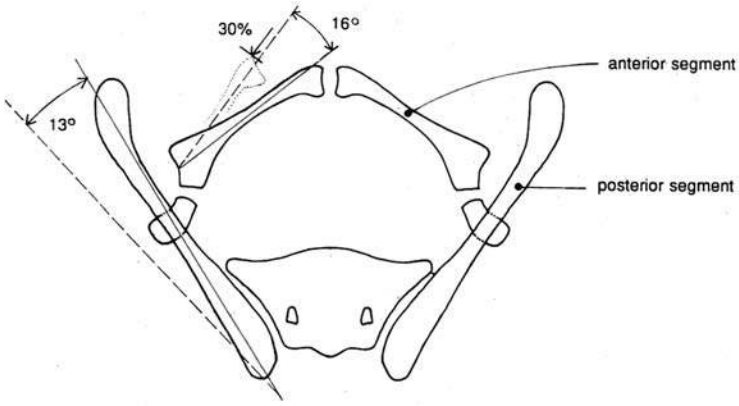



**Fig. 2.14** Schematic representation of pelvic differences in classic exstrophy versus normals in the transverse plane.

3. In the neonatal period, pubis may be approximated manually and temporarily held with suturing.
4. In an older child, iliac osteotomy is indicated either anteriorly or posteriorly.

### Prognosis

The hip function in the untreated patient with classic bladder exstrophy is good. Children walk at a normal age, although they have an increased external foot-progression angle. This becomes less pronounced over time. Adults with exstrophy have an increased incidence of pain in the region of the sacroiliac joints. One natural history study suggests an increased incidence of degenerative disease of the hip in patients with uncorrected exstrophy. Patients with exstrophy are usually fertile.

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**Table 2.1** Comparison of Types of Tibia Vara (by Age)

| Parameter | Infantile (0–3 yr) | Juvenile (3–10 yr) | Adolescent (11 yr and older) |
|---|---|---|---|
| Pain | No | No | Usually |
| Site of varus | Proximal tibia only; medial plateau may be tilted or depressed. Femur often in valgus | Tibia | Distal femur and proximal tibia in varus |
| Risk of bar | Yes | No | No |
| Treatment | Observe or brace; hemiepiphyseodesis if mild; osteotomy of femur and tibia as indicated; may need lengthening later | | • Hemiepiphyseodesis if mild, growing<br>• Osteotomy of femur and tibia |

## ◆ Tibia Vara

A focal varus deformity of the proximal tibia resulting from overload causing disordered medial growth. Tibia vara may have onset in the infantile, juvenile, or adolescent period (**Table 2.1**).

### Radiographic Features

1. Normal alignment of the lower extremity is shown in Chapter 1's Fig. 1.38.
2. In infantile tibia vara, changes involve physeal depression with lucency and beaking of the corresponding metaphysis and epiphysis. These changes have been staged 1 through 7 by Langenskjold (**Fig. 2.15**).

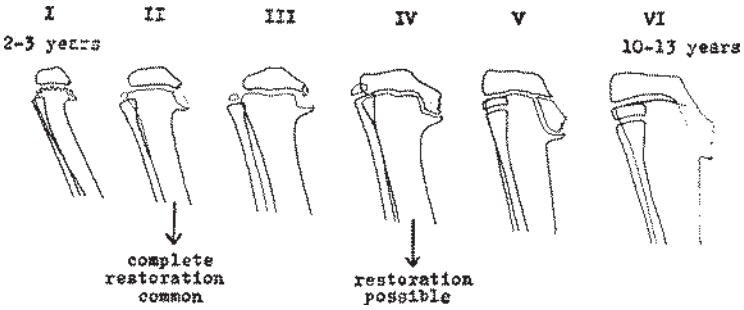



**Fig. 2.15** Langenskjold stages of infantile tibia vara. (From Langenskiold A. Tibia vara. (Osteochondrosis deformans tibiae.) A survey of 23 cases. Acta Chir Scand. 1952;103(1):1–22 (Fig. 5). Reprinted with permission.)

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3. Infants under age 2 years have inadequate ossification to assign Langenskjold stages. As an alternate means of early diagnosis, the metaphyseal–diaphyseal angle may be drawn (**Fig. 2.16**). An angle of greater than 16 degrees is diagnostic of infantile Blount disease. An angle of less than 11 degrees is rarely seen in patients with Blount disease and rules this diagnosis out.
4. Medial physeal slope greater than 60 degrees may predict recurrent bowing after osteotomy (**Fig. 2.17**).
5. Medial plateau depression greater than 30 degrees may be seen in neglected infantile tibia vara (**Fig. 2.18**).



**Fig. 2.16** Metaphyseal-diaphyseal angle used to detect tibia vara in very young children. High diagnostic likelihood if angle is greater than 11 to 16 degrees.

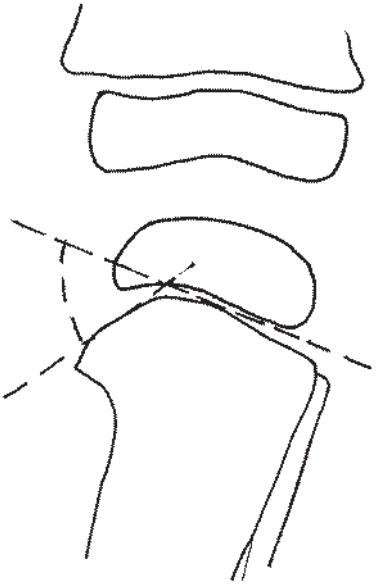


**Fig. 2.17** Metaphyseal slope is used to predict the risk of recurrent deformity. Values over 60 degrees are at increased risk.

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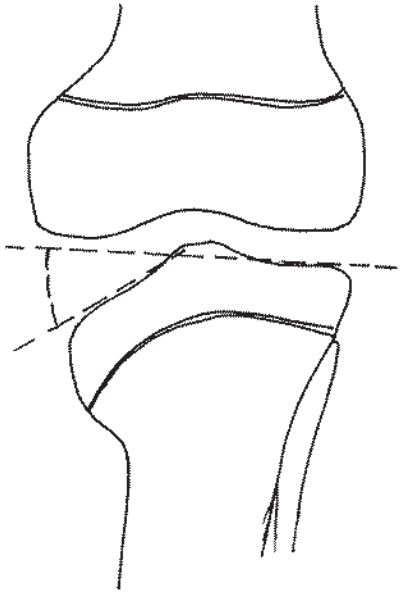


**Fig. 2.18** Medial plateau depression is unique to infantile tibia vara. If greater than 25 degrees, the medial side may need to be selectively elevated.

6. Mechanical axis deviation quantifies the degree of varus. The axis line is drawn from the center of the hip to the center of the ankle. The degree of varus (or valgus) is quantified as follows (**Fig. 2.19**).

### Differential Diagnosis

1. Rickets: Many types
2. Achondroplasia
3. Metaphyseal chondrodysplasia (Schmidt)
4. Trauma or infection of medial physis
5. Physiologic bowing
6. Focal fibrocartilaginous dysplasia

### Treatment

1. Infantile
   a. Observe or brace (day versus night)
   b. Valgus derotation osteotomy if not better by age 4 or Langenskjold stage 4. Hemi-epiphyseodesis is another option.

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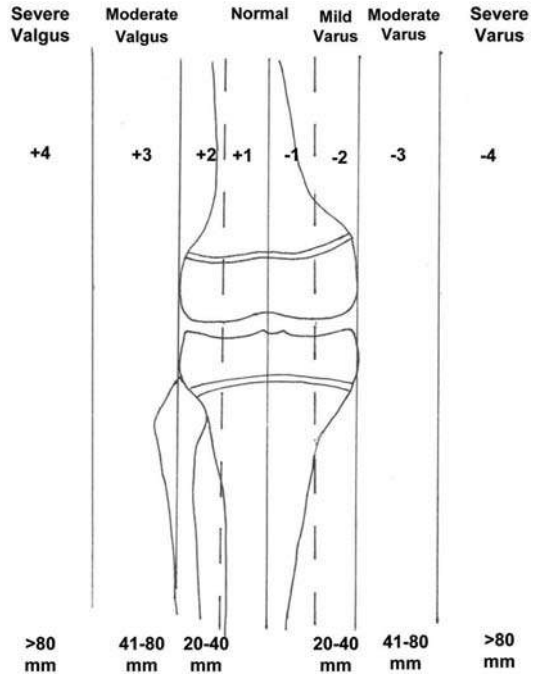



**Fig. 2.19** Mechanical axis deviation. The location of a straight line between the hip and the ankle is graded in zones as shown.

- **c.** Consider MRI for bar if physis is narrow or medial physeal slope is greater than 60 degrees. If bar is found, resect it or close lateral side of physis.
- **e.** Consider tibial plateau elevation if medial depression exceeds 25 degrees.
- **f.** Correct femoral valgus deformity if greater than 10 degrees.
- **g.** Equalize leg lengths as needed.

**2.** Adolescent tibia vara

- **a.** Correct if deformity exceeds 10 degrees or pain is persistent.
- **b.** Lateral hemiepiphysiodesis is an option if deformity is not severe (Mechanical axis deviation 1 to 2) and patient has two or more years of growth remaining.
- **c.** Osteotomy of proximal tibia and distal femur as appropriate.
- **d.** Equalize leg lengths if discrepancy is greater than 2.5 cm.

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## ◆ Other Angular Deformities at the Knee

Angular deformities may occur as a result of trauma or metabolic disorders, or they may be idiopathic or physiologic. If deviation from normal alignment is greater than 10 degrees, correction may be indicated in some cases. Treatment may be done by osteotomy and internal or external fixation or by hemiepiphysiodesis. The goal is to restore a horizontal joint line, physiologic angulation (see Chapter 1's Fig. 1.38) and near-equal limb lengths.

Hemiepiphysiodesis may be the simplest method for growing patients. The theory is shown in **Fig. 2.20**. Prerequisites include (1) predictable growth pattern, (2) sufficient growth remaining to correct defect, and (3) limb lengths close to equal.

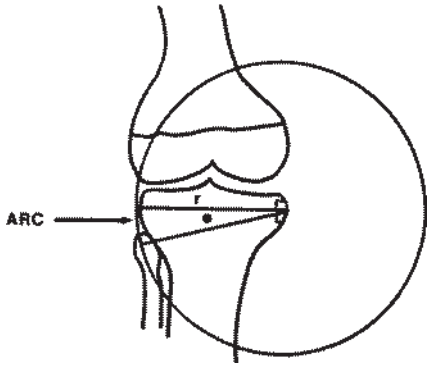
### Planning

1. Locate the site of deformity—distal femur, proximal tibia, or both—and measure its degree.
2. Using the width of the physis and the desired degree of correction, draw a horizontal line on the figure.
3. Draw a dot at the intersection of this horizontal line with the patient's tibial or femoral percentile.
4. A vertical line from this point downward indicates the age at which hemiepiphysiodesis should be performed. If done before puberty, it will indicate the length of time before correction is complete. Allow slight overcorrection before removing plates.

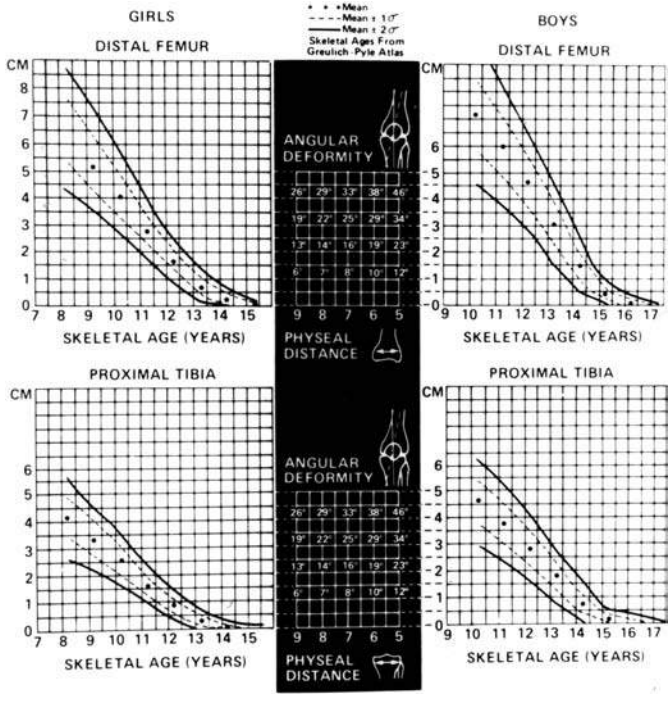
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A




B

**Fig. 2.20** **(A)** Theory of angular correction by asymmetrical tethering. **(B)** Angular correction is calculated from metaphyseal width and growth remaining. (From Bowen JR, Leahey JL, Zhang ZH, MacEwen GD. Partial epiphysiodesis at the knee to correct angular deformity. Clin Orthop Relat Res. 1985;198 (Figs. 2 and 3). Reprinted with permission.)

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## ◆ Patellofemoral Disorders

Patellofemoral disorders are common, especially in adolescence. History should include duration of symptoms, recent changes in activity, and inciting factors.

### Physical Examination

1. Patient to indicate location of pain
2. Check pain on compression of patella.
3. Apprehension test (guarding with lateral pressure)
4. Assess effusion
5. Note retinacular tightness (reverse tilt)
6. Measure tibial and femoral rotational alignment
7. Assess ligament and meniscal integrity
8. Measure valgus
9. Examine active tracking
10. Observe gait

### Differential Diagnosis

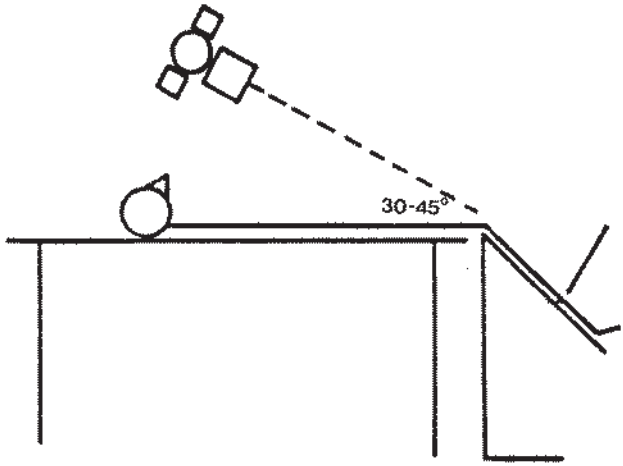
1. Plica
2. Saphenous nerve entrapment
3. Fat pad impingement
4. Patellar osteochondritis
5. Iliotibial band syndrome
6. Patellar malalignment
7. Patellar subluxation
8. Arthrosis

### Radiographic Evaluation

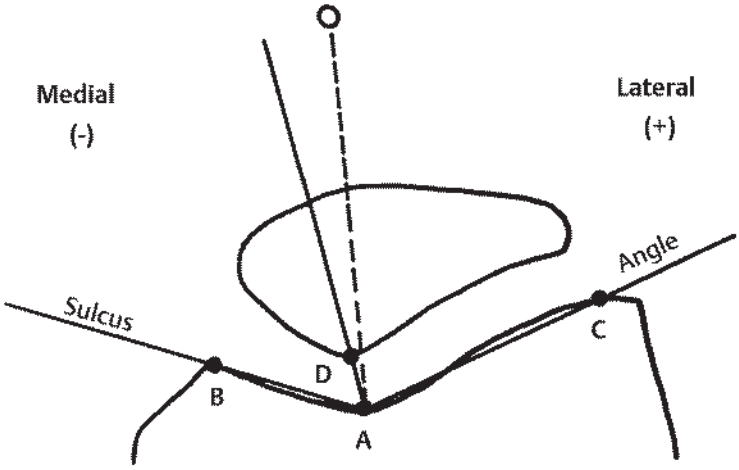
Radiographs are not needed on initial evaluation of all cases. However, if the problem is recalcitrant, the following studies may help:

1. Merchant view: A "sunrise" view taken at 30 to 45 degrees of knee flexion (**Fig. 2.21A**). From this, the sulcus angle can be measured, which should be greater than 17 degrees (**Fig. 2.21B**).

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A Merchant View




B The Congruence Angle

**Fig. 2.21** **(A)** Merchant view: a sunrise view with knee flexed 30 to 45 degrees. **(B)** Interpretation. Sulcus angle should be at least -17 degrees. (From Merchant AC, Mercer RL, Jacobsen RH, Cool CR. Roentgenographic analysis of patellofemoral congruence. J Bone Joint Surg Am. 1974;56(7) (Figs. 2 and 5). Reprinted with permission.)

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2. The lateral view will show the Insall ratio, which is the length of tendon divided by the length of patella (**Fig. 2.22**). The normal value is 1.0, and the upper limit of normal is 1.2.
3. Computed tomography may be helpful to assess patellar tilt. It should be done in 20 degrees of flexion. Normal tilt is less than +8 degrees.
4. MRI is an option to assess for osteochondral lesions.

### Treatment

Treatment of patellofemoral malalignment should start with conservative measures. These may include the following:

1. Stretching of retinaculum, iliotibial band, quadriceps, and hamstrings
2. Strengthening of quadriceps: Low resistance, high repetition, knee not flexed over 45 degrees
3. NSAIDs
4. Orthotics to decrease foot pronation, if present
5. Elastic brace or patellar taping
6. In the resistant case, surgical correction may be offered and may include the following:

    a. Lateral release (for tilt)

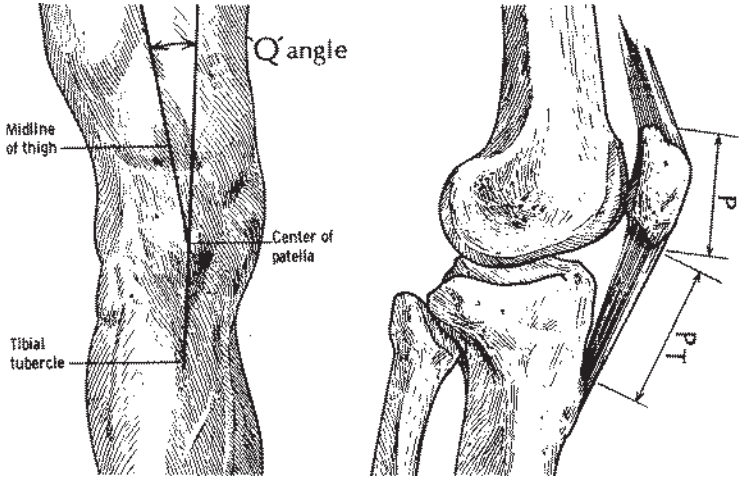



**Fig. 2.22** Insall the ratio to evaluate the patella alta: Length of the tendon is divided by the length of the patella. The patella alto is defined as a ratio of greater than 1.2. (From Insall J, Falvo KA, Wise DW. Chondromalacia patellae: a prospective study. J Bone Joint Surg Am. 1976;58(1):2 (Fig. 2). Reprinted with permission.)

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**b.** Tibial tubercle transfer + vastus medialis oblique advancement (for subluxation)
**c.** Limited debridement (for severe cartilage surface degeneration)
**d.** Optional correction of severe anteversion or torsion

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## ◆ Discoid Lateral Meniscus

### Background and Classification

1. Incidence: About 1%; usually lateral
2. Classification: Based on the degree of attachment to the tibial plateau

   **a.** Wrisberg ligament type: Absent lateral meniscotibial ligaments. Only ligament of Wrisberg present, attaching meniscus to the posterior cruciate ligament. This type is the most anomalous and unstable type.
   **b.** Complete type: Meniscus covers the entire tibial plateau, but ligaments are intact.
   **c.** Incomplete type: Stable incomplete discoid meniscus with intact ligaments.

### Clinical Signs and Symptoms

1. "Snapping knee," typically before the age of 8 years
2. A palpable snap and shift of the tibial plateau during movement
3. Joint pain and swelling occur as the meniscus develops a tear.
4. Feelings of "catching" with knee motion

### Imaging

1. Plain radiographs are indicated initially to rule out any bony anomalies. These may show widening of lateral joint space.
2. MRI shows discoid meniscus and any tears.
3. A true discoid meniscus will appear as a continuous bow-tie appearance on three or more consecutive sagittal plane images. In the coronal plane, the discoid meniscus occupies more than one third of the joint diameter at the midpoint. False negatives can occur with the unstable (Wrisberg) type of discoid meniscus, which may maintain a relatively semilunar shape.

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### Treatment

1. If the patient is asymptomatic and the discoid meniscus is an incidental finding, excision is not recommended.
2. Substantial tears of a stable discoid meniscus may require a resection in which the peripheral rim is left intact (meniscoplasty).
3. In cases in which the meniscus is unstable, repair and contouring may be attempted, but a complete meniscectomy is often required.

### Prognosis

There is a paucity of long-term follow-up data for both untreated discoid menisci and for partial discoid meniscectomy.

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## ◆ Popliteal (Baker) Cyst

### Background

1. Popliteal cysts are the most common masses found in the popliteal fossa.
2. These synovial-lined ganglion cysts arise from synovial fluid through communication between the knee joint and the bursa between the semimembranosus and medial gastrocnemius muscles.
3. Located along the medial side of the popliteal fossa, posterior to the medial femoral condyle, between the two tendons.
4. Rarely associated with conditions such as meniscal tears, arthritides, and pigmented villonodular synovitis.
5. Prevalence of popliteal cysts in asymptomatic children is around 2%.

### Signs and Symptoms

1. Rarely causes symptoms and is usually an incidental discovery
2. May wax and wane in size but should not cause limp or disability
3. However, patients may present with complaints of pain if the cyst ruptures.

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### Physical Features

Superficial, firm or rubbery, smooth, nontender, slightly mobile and nonpulsatile mass. Most cysts will transilluminate with a handheld light (**Fig. 2.23**). If so, imaging is unnecessary.

### Imaging

1. Differential diagnosis: Lipomas, xanthomas, vascular tumors, or fibrosarcomas
2. History and transillumination are the best ways to differentiate.
3. If in doubt, ultrasound can be used to distinguish between fluid-filled cyst and solid tumors. MRI is another option for those rare cases when clinical confirmation is in doubt.

### Treatment

Most cysts (85%) disappear without intervention or cause few symptoms (15%). Surgical excision is rarely indicated. Parents should be informed that the cyst may vary in size for a while, but it should eventually regress over the years. Only if the diagnosis is in doubt or if clinical symptoms are persistent should surgery be undertaken.

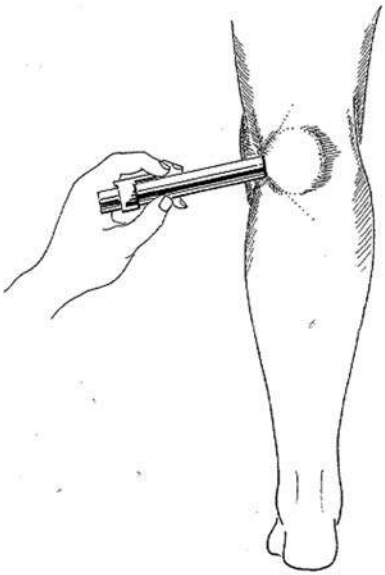


**Fig. 2.23** Transillumination of the popliteal cyst. The cyst picks up light more than surrounding tissues.

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## ◆ Congenital Dysplasia (Anterolateral Bow/Pseudarthrosis) of the Tibia

### Background and Natural History

1. Congenital dysplasia may be sporadic or associated with neruofibromatosis-1.
2. Etiology is unknown.
3. Anterolateral bowing of distal tibia resembles genu varum, but the apex is more distal and has an anterior component; it is unilateral only.
4. Bowing often progresses to pseudarthrosis, which is usually very difficult to heal with cast, plate, or electrical stimulation. The distal apex contributes to difficulty with fixation.
5. 1–4 cm shortening of the affected side
6. Clinically no pain until fracture (which produces little pain)

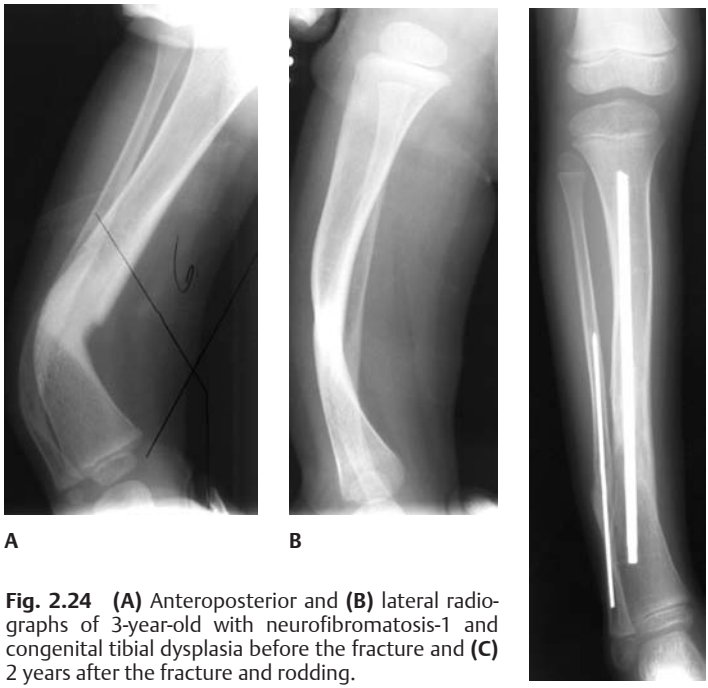
### Radiographic Appearance (Fig. 2.24)

1. Narrowing of tibia at bow
2. Narrowing or scalloping of medullary canal
3. Tapering of bone ends
4. May be associated with fibular bowing or pseudarthrosis

### Treatment

1. Protect with brace before fracture.
2. Fibular strut if fracture is imminent.
3. If fractured, options include long intramedullary (Williams) rod ± rhBMP-2, Ilizarov treatment, free vascularized fibula, or combinations.
4. Separate treatment for limb-length inequality and valgus ankle may be required.

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**A** **B** **C**

**Fig. 2.24** **(A)** Anteroposterior and **(B)** lateral radiographs of 3-year-old with neurofibromatosis-1 and congenital tibial dysplasia before the fracture and **(C)** 2 years after the fracture and rodding.

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Richards BS, Oetgen ME, Johnston CE. The use of rhBMP-2 for the treatment of congenital pseudarthrosis of the tibia: a case series. J Bone Joint Surg Am. 2010;92(1):177–185

## ◆ Posteromedial Bow of Tibia

### Background and Natural History

1. Present at birth. Etiology unknown. Not a prior fracture
2. Bowing resolves with time and growth over 5 to 10 years.
3. No increased risk of fracture

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4. Shortening proportionate throughout growth; mean 2 to 5 cm at maturity

### Clinical and Radiographic Appearance (Fig. 2.25)

1. Foot appears dorsiflexed and in valgus; limited plantar flexion
2. Leg slightly short at birth.
3. Tibia and fibula have posteromedial bow of distal third with sclerotic cortices.

### Treatment

1. Observation with or without stretching and orthosis for foot support
2. Osteotomy rarely necessary for bowing
3. Leg-length equalization as indicated, with lift and surgery near maturity

## Bibliography

Pappas AM. Congenital posteromedial bowing of the tibia and fibula. J Pediatr Orthop. 1984;4(5):525–531

Shah HH, Doddabasappa SN, Joseph B. Congenital posteromedial bowing of the tibia: a retrospective analysis of growth abnormalities in the leg. J Pediatr Orthop B. 2009;18(3):120–128

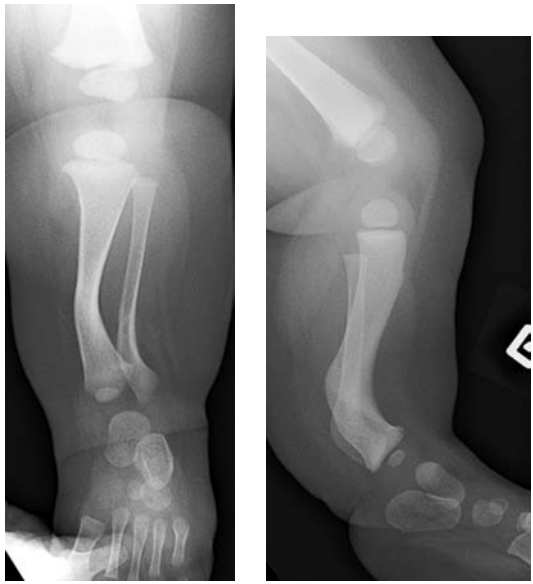


**Fig. 2.25** Posteromedial bowing of the tibia and fibula in a 1-year-old.

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## ◆ Clubfoot

Talipes equinovarus may be unilateral or bilateral with equal frequency. Although most cases are idiopathic, other causes or associations should be considered:

1. Neurogenic: Spinal dysraphism, tethered cord, arthrogryposis
2. Connective tissue disorders: Loeys-Dietz syndrome, Larsen syndrome, diastrophic dwarfism, spondyloepiphyseal dysplasia
3. Mechanical: Oligohydramnios, congenital constriction bands
4. Syndromes: Freeman–Sheldon, Pierre-Robin, tibial hemimelia, Mobius

### Physical Findings

A variable number of these findings may be present:

1. Medial or posterior crease
2. Curved lateral border
3. Calf atrophy
4. Cavus
5. Equinus
6. Forefoot adduction and supination

The foot may be given a score from 1 to 20 if each positive finding is awarded 1 point (**Table 2.2**).

**Table 2.2** DiMeglio Clubfoot Scoring System

| Parameter | Points | | | | |
|---|---|---|---|---|---|
| | 4 (degrees) | 3 (degrees) | 2 (degrees) | 1 (degrees) | 0 (degrees) |
| Equinus | 45–90 | 20–45 | 20–0 | 0–20 DF | > + 20 DF |
| Varus | 45–90 | 20–45 | 20–0 | 0–20 DF | >20 val |
| Supination | 45–90 | 20–45 | 20–0 | 0–20 pro | >20 pro |
| Adductus | 45–90 | 20–45 | 20–0 | 0 >–<20 abd | >20 abd |
| Posterior crease | | | | Yes | No |
| Medial crease | | | | Yes | No |
| Cavus | | | | Yes | No |
| Abnl muscle fcn | | | | Yes | No |

*Abbreviations*: Abd, abduction; Abnl, abnormal; DF, dosiflexion; fcn, function; pro, pronation; val, valgus.
*Scoring*: very severe = 16–20, severe = 11–16, moderate = 6–10, postural = 1–5.

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### Radiographs

Radiographs are useful mainly if surgery is indicated or in postsurgical follow-up. AP and lateral films should be taken with the foot as plantigrade as possible. Normal values for AP and lateral talocalcaneal angles are given in Chapter 1's Figs. 1.44 and 1.45. Equinovarus correlates with increasing parallelism of the talus and calcaneus. Cuboid can be neutral or subluxated medially.

### Treatment

1. Serial manipulation and long-leg cast treatment (Ponseti method): This treatment produces the most normal foot growth and mobility; it is described in greater detail in Chapter 7. Sequence of correction:
   - **a.** Dorsiflexion of first ray
   - **b.** Counterpressure on dorsolateral aspect of talus
   - **c.** Progressive external rotation and dorsiflexion of hindfoot
   - **d.** Achilles' tenotomy if correction of hindfoot equinus is not complete after correction of other components
   - **e.** Maintaining correction after cast removal with foot abduction orthosis. This is a straight or reverse last shoes attached to bar with feet externally rotated 60 degrees.
   - **f.** Foot abduction orthosis is worn full-time for 2 months, then nightly for 3 to 5 years.
   - **g.** Relapses are salvaged by repeat cast program.
   - **h.** Feet with late recurrence are treated by Achilles' tendon lengthening and transfer of anterior tibialis to third cuneiform.
2. Operative correction, for severe relapses and syndromes, may include the following:
   - **a.** Lengthening of heel cord and posterior tibialis
   - **b.** Posteromedial capsulotomies
   - **c.** Complete subtalar release
   - **d.** Calcaneocuboid release
   - **e.** Lateral column shortening

## Bibliography

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Ponseti IV. The Ponseti technique for correction of congenital clubfoot. J Bone Joint Surg Am. 2002;84-A(10):1889–1890, author reply 1890–1891

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## ◆ Tarsal Coalition

Tarsal coalition is a fibrous, cartilaginous, or bony connection of two or more tarsal bones. It is present in about 3% of the population and is bilateral in half of these. Calcaneonavicular (CN) and talocalcaneal (TC) bars are equally common.

### Signs and Symptoms

1. Foot or ankle pain and stiffness occurs at about age 8 to 12 years for CN bar, 12 to 16 for talocalcaneal bar.
2. Limitation of subtalar movement is greater with TC coalition.
3. Pes planus: Variable
4. Peroneal guarding or "spasm"

### Radiographs

1. Oblique view of midfoot is diagnostic for CN bar with narrowing, irregularity, or fusion of the space between the two bones (**Fig. 2.26A**).
2. Lateral view shows pointed projection of calcaneus or "anteater nose" in CN coalition.
3. Harris view shows obliquity and sclerosis of sustentaculum in TC coalition and may show fusion across middle facet.
4. Computed tomography (CT) is the definitive study for a TC coalition. The plane of the tomograms should be the coronal plane of foot with knees flexed and sole flat on gantry. Reconstructions in all planes should be viewed (**Fig. 2.26B**).
5. CN and TC coalitions may coexist.

### Treatment

1. If discovered incidentally and asymptomatic, observe.
2. Arch support
3. Walking cast for 3 to 6 weeks
4. Bar resection if conservative treatment fails for TC bar less than 50% and no degenerative changes or CN bar of any size
5. Arthrodesis

## Bibliography

Lemley F, Berlet G, Hill K, Philbin T, Isaac B, Lee T. Current concepts review: Tarsal coalition. Foot Ankle Int. 2006;27(12):1163–1169

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**Fig. 2.26** **(A)** Oblique view of the midfoot is diagnostic for the calcaneonavicular bar with narrowing, irregularity, or fusion of the space between the two bones. **(B)** Computed tomography is the definitive study for a talocalcaneal coalition. The plane of the tomograms should be the coronal plane of foot with knees flexed and sole flat on gantry. Reconstructions in all planes should be viewed.

## ◆ Flatfoot

### Background and Definition

1. Etiology is a combination of factors including connective tissue properties, muscle tone, and genetics.
2. Achilles contracture increases eversion.

### Signs and Symptoms

1. Usually asymptomatic
2. Severe pressure on plantar surface may cause pain.

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3. The natural history is generally benign in all but the most pronounced cases.

### Physical Features and Examination

1. Examine elbows, wrists, and knees for evidence of ligamentous laxity.
2. Determine range of hindfoot and forefoot motion to classify as flexible or rigid.
3. Measure ankle dorsiflexion.
4. Observe patient standing on toes, checking for reappearance of the arch as an indicator of structural integrity of the foot during push-off.
5. Inspect skin of sole for signs of pressure concentration over navicular and talar head.

### Differential Diagnosis

1. Connective tissue disorders: Marfan, Ehlers Danlos, osteogenesis imperfecta
2. Neurologic disorders: Spinal dysraphism, diplegia
3. Tarsal coalition: Fibrous or bony
4. Congenital malformation: Fibular hemimelia, vertical talus
5. Accessory navicular

### Treatment

1. Observation if asymptomatic
2. Soft arch support if symptoms are severe
3. Surgical reconstruction if unresponsive, with structural weakness and pressure concentration

## Bibliography

Kasser JR. The pediatric foot. In Morrissy RT, Weinstein SL eds. Lovell and Winter's Pediatric Orthopaedics, 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2008:1257–1329

## ◆ Calcaneovalgus Foot

Positional deformity in newborns. The foot is dorsiflexed at the ankle and in valgus.

### Physical Features

Passively, the foot may be plantarflexed to neutral or below, but it does not have a full range of normal plantarflexion. There is no deformity of the tibia.

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### Radiographs

Not indicated in the typical case because the physical examination is diagnostic. If radiographs are obtained, they show no bony abnormalities other than the dorsiflexed position. This differentiates it from posteromedial bow of the tibia.

### Treatment

No treatment is necessary. Deformity improves with time. No casting, bracing, or splinting is necessary.

### Prognosis

Spontaneous resolution is the rule.

## Bibliography

Kasser JR. The pediatric foot. In Morrissy RT, Weinstein SL eds. Lovell and Winter's Pediatric Orthopaedics, 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2008: 1257–1329

## ◆ Congenital Vertical Talus

Essential feature is a dorsolateral dislocation of the talonavicular joint with associated contractures.

### Etiology

1. Idiopathic
2. Myelomeningocele
3. Arthrogryposis
4. Larsen syndrome

### Physical Findings

1. Reversal of arch with plantar convexity,"rocker bottom"
2. Forefoot is dorsiflexed and everted; hindfoot is plantar-flexed.
3. Normal arch is not fully reproducible.
4. Crease in sinus tarsi

### Radiographic Findings

1. Talus is almost "vertical" and is in line with tibia
2. Talar axis does not line up with first metatarsal.
3. Diagnostic test: Talus and first metatarsal still do not line up with stress plantarflexion lateral film of foot (**Fig. 2.27**).

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**Fig. 2.27** Diagnostic test for congenital vertical talus. The talus and first metatarsal still do not line up with stress plantarflexion lateral film of foot.

### Treatment

1. Plantarflexion casting (into clubfoot position)
2. Percutaneous pinning of talonavicular joint
3. Open reduction via dorsal or plantar approach, with tendon lengthening
4. Subtalar fusion if valgus persists

## Bibliography

Dobbs MB, Purcell DB, Nunley R, Morcuende JA. Early results of a new method of treatment for idiopathic congenital vertical talus. J Bone Joint Surg Am. 2006;88(6):1192–1200

Seimon LP. Surgical correction of congenital vertical talus under the age of 2 years. J Pediatr Orthop 1987;7(4):405–411

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## ◆ Hallux Valgus

### Background and Definitions

1. First metatarsophalangeal (MTP) angle is greater than 15 degrees.
2. Onset before age 10 is termed *juvenile hallux valgus*.
3. Onset between age 10 and 18 is termed *adolescent hallux valgus*.
4. Positive family history and ligamentous laxity are common.

### Clinical Features

1. Asymptomatic or pain over medial prominence
2. Tightness of Achilles' tendon may be present.
3. Pes planus is usually present.
4. Range of toe movement should be measured.

### Radiographic Features

1. Standing AP and lateral radiographs are needed.
2. Metatarsophalangeal (MTP) angle greater than 15 degrees is hallux valgus.
3. An intermetatarsal angle (between the 1st and 2nd metatarsals) greater than 10 degrees indicates metatarsus primus varus.
4. The distal metatarsal articular angle (DMAA) is formed between the shaft of the first metatarsal and the line perpendicular to the articular surface of the MTP joint. The DMAA is expected to increase in juvenile or adolescent hallux vagus.
5. MTP joint congruity, relative lengths of the metatarsals, metatarsocuneiform orientation, proximal phalangeal articular angle, and lesser metatarsal orientation should also be assessed.

### Treatment

1. Conservative: Shoes should have soft material and a low heel. Arch supports may help with pronation. Night splints have not proven effective.
2. Surgery is indicated only if there is significant pain despite conservative treatment.
3. Metatarsus primus varus (MPV) in adolescent: Lateral hemiepiphyseodesis of first metatarsal base.
4. Deformity of interphalangeal joint: Osteotomy of proximal phalanx.
5. MTP less than 25 degrees: Soft tissue reconstruction.
6. MTP greater than 25 degrees: Correct MPV and reorient joint.
7. Address significant hindfoot valgus with osteotomies.
8. Arthrodesis of the MTP joint is the most reliable way of hallux valgus correction in cerebral palsy.

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### Complications

Stiffness of the MTP joint, persistent pain, AVN of the metatarsal head, and a high rate of recurrence

## Bibliography

Aronson J, Nguyen LL, Aronson EA. Early results of the modified Peterson bunion procedure for adolescent hallux valgus. J Pediatr Orthop. 2001;21(1):65–69
Groiso JA. Juvenile hallux valgus: a conservative approach to treatment. J Bone Joint Surg Am 1992;74(9):1367–1374

## ◆ Toe Walking

### Background

1. Common in toddlers.
2. Commonly caused by shortened Achilles' tendon.
3. Some eventually adopt normal walking with growth.
4. Persistent toe walking beyond 3 years of age should prompt examination for underlying neuromuscular problems.
5. However, most children have what is termed, by exclusion, *idiopathic toe walking*.
6. Many patients have a positive family history.
7. May be associated with autism, language disorders.

### Signs and Symptoms

1. Painless
2. Occasionally frequent falling

### Physical Examination

1. Examine with the child in shorts.
2. Assess for ataxia, muscle weakness, Gower sign.
3. Range of ankle dorsiflexion should be noted, with the knee both flexed and extended.
4. Look for calf pseudohypertrophy.
5. Hamstrings and adductors should be checked for tightness.

### Differential Diagnosis

1. Cerebral palsy
2. Muscular dystrophy
3. Tethered cord syndrome
4. Charcot–Marie–Tooth disease

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5. Arthrogryposis
6. Freidreich ataxia

### Treatment

1. Physical therapy
2. Casting: Increased ankle dorsiflexion can be achieved by stretching and serial casting. The cast should be changed weekly until the desired ankle range of motion is obtained. After casts are off, continue stretching.
3. Achilles' tendon lengthening, percutaneous or open.

## Bibliography

Kalen V, Adler N, Bleck EE. Electromyography of idiopathic toe walking. J Pediatr Orthop. 1986;6(1):31–33

## ◆ Macrodactyly

### Background

1. Macrodactyly is overgrowth of one or several adjacent digits or rays of a hand or foot.
2. It is present at birth, although some cases worsen disproportionately.
3. Growth of the enlarged digits ceases when the patient reaches skeletal maturity.

### Classification

1. Incidence is less than 1:10,000.
2. Most cases are idiopathic; others are associated with Klippel–Trenaunay–Weber, neurofibromatosis-1 (NF-1), or Proteus syndrome.
3. Usually unilateral
4. Etiology unknown
5. No genetic risk if not NF-1

### Signs and Symptoms

1. Painless in child; premature arthritis in adult
2. Difficulty with shoe wear
3. Impaired push-off (foot) or clumsiness (hand)

### Physical Features (Fig. 2.28)

1. Enlargement is greater distally than proximally. The nail is especially enlarged. All tissues are affected.

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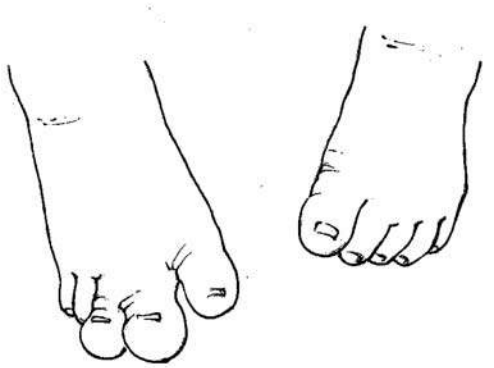


**Fig. 2.28** Physical features of macrodactyly.

2. Tissues on the plantar or palmar surface of the digit are more enlarged than those on the dorsal side, causing the digit to become hyperextended (dorsiflexed).
3. Central digits are more commonly involved than border digits.
4. Syndactly may co-exist.
5. Metatarsals and carpals are rarely significantly enlarged.
6. If two digits are involved, they grow away from each other.
7. Rarely enlargement is disproportionate.
8. Skin may display hemangioma.
9. Range of interphalangeal motion is decreased.

### Tests

Genetic testing is available for Proteus syndrome and NF if these are suspected.

### Imaging

1. Plain films should be made to help assess and document the extent of overgrowth and the segments involved.
2. Skeletal maturity is often advanced in the enlarged rays.
3. MRI is rarely necessary.

### Differential Diagnosis

1. Hemihypertrophy, in which all segments of a limb are uniformly overgrown
2. Acrodactyly, in which overgrowth of all digits is greatest distally
3. Growth hormone excess: Acromegaly

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### Treatment

1. Shoe modification if needed
2. Surgery
    a. Resection of the most enlarged ray, if the width of the hand or foot is greatly increased.
    b. Phalangectomy may make the length more even if the width is not a problem (if nail loss is acceptable).
    c. Epiphysiodesis (closure of the growth plates); the correction occurs more gradually with time.
3. Debulking of fat may improve the appearance, especially of the plantar fat hypertrophy.
4. Discretion advised in staging multiple procedures to avoid tissue ischemia.

## Bibliography

Akinci M, Ay S, Erçetin O. Surgical treatment of macrodactyly in older children and adults. J Hand Surg Am. 2004;29(6):1010–1019
Akinci M, Ay S, Erçetin O, Glutting J. Surgical treatment of macrodactyly in older children and adults. J Hand Surg Am. 2004;29(6):1010–1019
Barsky AJ. Macrodactyly. J Bone Joint Surg Am. 1967;49(7):1255–1266
Dennyson WG, Bear JN, Bhoola KD. Macrodactyly in the foot. J Bone Joint Surg Br. 1977;59:355–359

## ◆ Torticollis

Malposition of the head and neck with lateral flexion to one side and rotation to the contralateral side. The child holds the ear closer to one shoulder and the chin closer to the other.

### Differential Diagnosis

1. Muscular torticollis resulting from contracture of sternocleidomastoid
2. Atlantoaxial rotatory instability
3. Congenital malformation of upper cervical spine
4. Reflux response (Sandifer syndrome)
5. Ocular or auditory abnormality
6. Brainstem abnormality or tumor

### Physical Examination and Findings

1. Measure range of motion to each side
2. Thorough neurologic examination

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3. Palpate for sternocleidomastoid contracture (typically tight on the side of lower ear).
4. Examine eye movements and hearing.
5. Plagiocephaly is a sign of early onset, longer duration.

### Imaging

1. Plain films centered on upper cervical spine
   a. Usually difficult to interpret because of rotation
   b. Include open mouth AP if possible
2. CT with reconstructions usually necessary
3. MRI if suspect neurologic abnormality or if surgery is planned

### Treatment

1. Muscular torticollis: Stretching, possible lengthening
2. Atlantoaxial rotatory instability: Stretching if duration less than 1 week; traction 1 to 3 weeks; fusion if over 1 month

## ◆ Idiopathic Scoliosis

Idiopathic scoliosis is the most common pediatric spinal deformity. It is transmitted as an autosomal dominant condition with incomplete penetrance. The initial evaluation should rule out other causes and determine maturity, curve size, type, and appropriate treatment.

1. Infantile scoliosis (onset 0–3 years): obtain MRI
2. Juvenile scoliosis (onset 4–9 years): obtain MRI
3. Adolescent scoliosis (onset >9 years)

### History

1. How curve was discovered
2. Presence or absence of significant pain
3. Family history
4. Menarchal status
5. Medical and surgical history

### Physical Findings and Examination

1. Record height and weight.
2. Assess trunk and extremities for cutaneous lesions, congenital malformation, connective tissue disorder, or atrophy.
3. Measure pelvic height for inequality of lower-limb length.

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4. Neurologic examination
    a. Strength, reflexes all extremities
    b. Abdominal reflex testing
5. Estimate physical maturity (breast appearance, facial or axillary hair)
6. Assess curve
    a. Shoulder elevation
    b. Trunk balance C1 through S1, coronal and sagittal
    c. Curve level
    d. Intrinsic pelvic deformity
    e. Kyphosis and lordosis

### Differential Diagnosis (in Absence of Overt Vertebral Malformations)

1. Genetic or connective tissue
    a. Marfan syndrome, Loeys–Dietz
    b. Ehlers–Danlos
    c. NF
    d. Prader–Willi
    e. Stickler syndrome
    f. Many others
2. Neurologic
    a. Syringomyelia
    b. Brainstem or cord tumor
    c. Friedreich ataxia
    d. Charcot–Marie–Tooth
    e. Polio
    f. Thoracic level paralysis or dyscoordination of any cause
3. Neoplastic and other
    a. Tethered cord/occult dysraphism
    b. Osteoid osteoma
    c. Osteoblastoma
    d. Postradiation
    e. Spinal cord tumor

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## Scoliosis Screening: Forward-Bend Test (Fig. 2.29)

1. Patient stands with feet together, knees straight, palms together. Check shoulders and pelvis for obliquity, and equalize leg lengths with blocks if necessary. Observe sagittal profile for focal kyphosis.
2. Have patient bend slowly all the way over.
3. Check thoracic spine.
4. Check lumbar spine.
5. Scoliometer measurement of trunk asymmetry (**Fig. 2.30**). Measures the angle of trunk rotation. This is roughly coordinated with Cobb angle. A scoliometer reading of 5 or less is 99% sensitive and 97% specific for curves under 20-degree Cobb angle. The mean Cobb measurement for

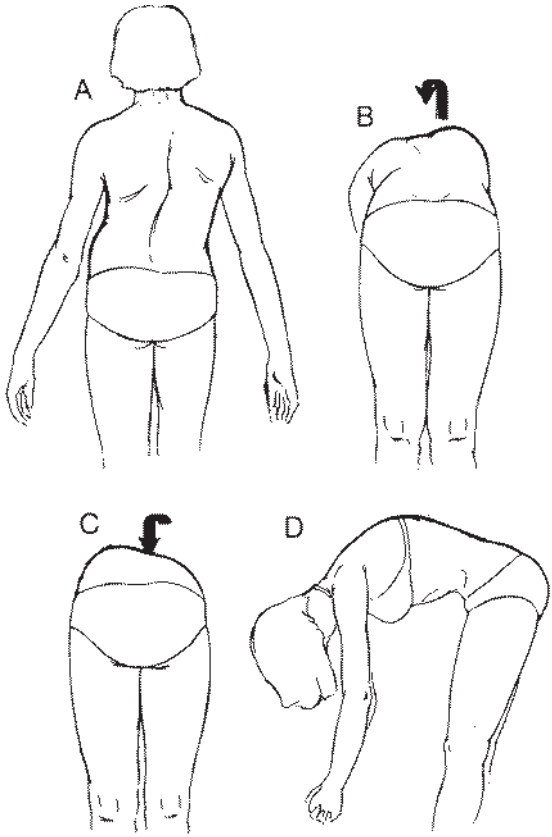



**Fig. 2.29** Forward bending test for spinal deformity.

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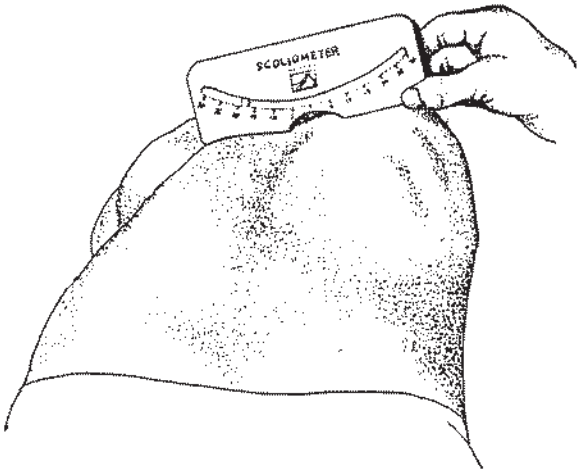



**Fig. 2.30** Technique of scoliometer measurement. (From Bunnell WP. An objective criterion for scoliosis screening. J Bone Joint Surg Am. 1984;66(9):1383 (Fig. 3). Reprinted with permission.)

curves 5 degrees by scoliometer is 11 degrees. A scoliometer reading of 7 or less is 88% sensitive and 86% specific for curves under 25 degrees with a mean Cobb of 20 degrees. The scoliometer is adequately sensitive for scoliosis screening: 95% of curves greater than 20 degrees measure 7 degrees or more on the scoliometer.

## Bibliography

Bunnell WP. Outcome of spinal screening. Spine. (Phila Pa 1976) 1993;18(12):1572–1580

### Radiographic Assessment

1. A radiograph should be made if the physical examination indicates that a curve may require treatment. Posteroanterior (PA) technique minimizes dose to gonads and breast but gives slightly less detail. A lateral film should be ordered only if needed for evaluation of pain or sagittal deformity.
2. Look for other anomalies such as congenital malformation, vertebral erosion, or pedicle widening or thinning. The curve magnitude may be described by the Cobb measurement (**Fig. 2.31**) and the direction of convexity and levels involved. The interobserver measurement error (Cobb) is around 5 degrees for idiopathic scoliosis and greater than 10 degrees

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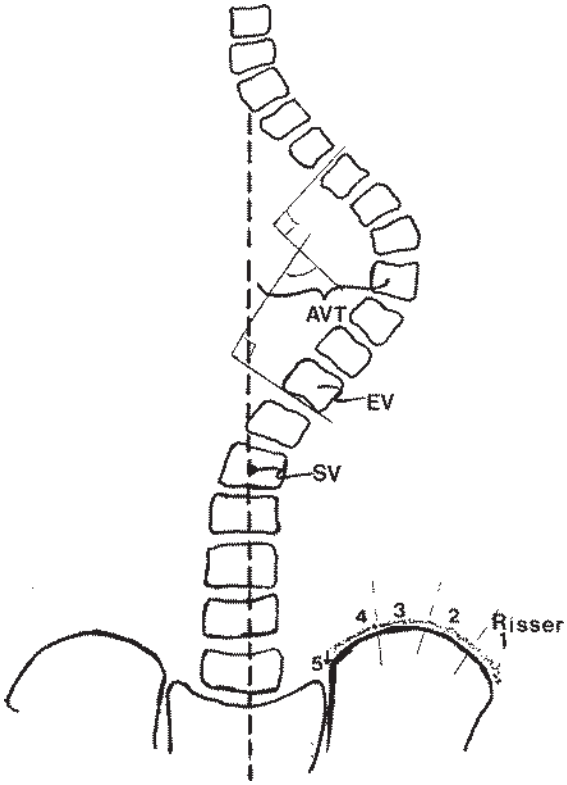



**Fig. 2.31** Cobb method of measurement. The angle between the upper and lower end vertebrae (EV) in the curve. Risser sign reflects maturation of the ilium. Grade 5 is fused epiphyses. It has a rough correlation with skeletal age and helps to predict the end of skeletal growth. It should not be used in isolation. The stable vertebra (SV) is the lowest vertebra bisected by a vertical line from the center of the sacrum. The apical vertebral (AVT) is measured with respect to the center sacral line.

for congenital scoliosis. Skeletal maturity may be roughly estimated by the Risser sign but should be correlated with physical examination (Tanner) and bone age if needed.

3. Rotation in the adolescent may be estimated by the method of Nash and Moe (**Fig. 2.32**) or by CT or the Perdriolle method.
4. Rotation may be estimated in the infantile or juvenile patient using the rib–vertebral angle difference (RVAD) of Mehta (**Fig. 2.33**).

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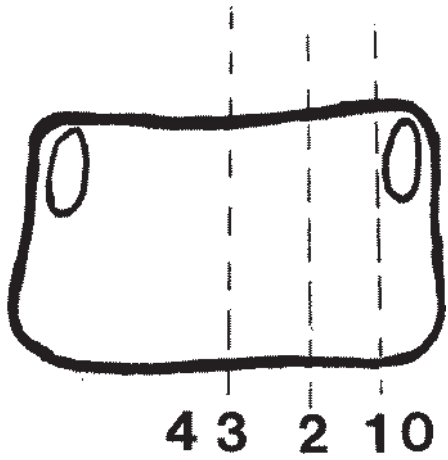



**Fig. 2.32** Rotation may be estimated by the method of Nash and Moe according to the location of the pedicle on the convex side.

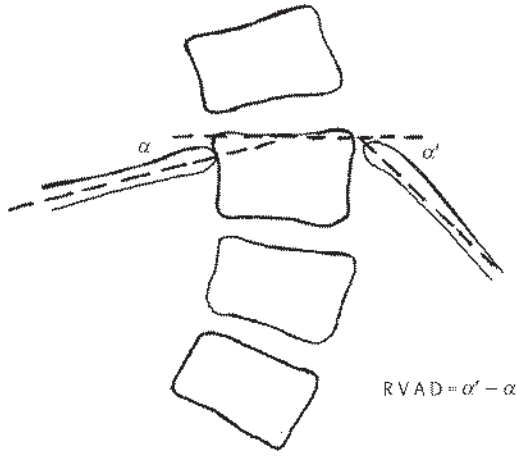



**Fig. 2.33** Rotation in infantile curves may be estimated by the rib vertebral angle difference of Mehta. The difference is between the angles formed by the two ribs versus the endplate of the apical vertebra.

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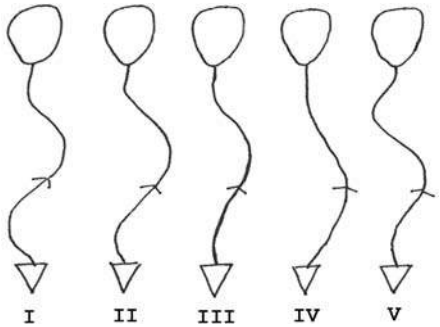



**Fig. 2.34** King classification of thoracic curve types.

**5.** Curve types are named for level of the apex:

- **a.** Thoracolumbar curves have an apex at or between T12 and L1.
  - **1)** Thoracic curves have an apex above this.
  - **2)** Lumbar curves have an apex below this.
- **b.** Thoracic curves have been classified by King into five types (**Fig. 2.34**).
  - **1)** True double major curves: Both are of equal magnitude and flexibility.
  - **2)** False double major curves: Lumbar curve is smaller and more flexible with less prominence.
  - **3)** Thoracic curve only; lumbar curve does not cross middle
  - **4)** Long thoracic curve; returns to midline at L4
  - **5)** Double thoracic curve
- **c.** Lenke classification of curve types is more comprehensive and incorporates sagittal characteristics (**Fig. 2.35**).
  - **1)** Main thoracic
  - **2)** Double thoracic
  - **3)** Double major
  - **4)** Triple major
  - **5)** Thoracolumbar/lumbar
  - **6)** Thoracolumbar/lumbar: Main thoracic

### Treatment Guidelines

These represent the mainstream of thought, but each case must be managed individually (**Fig. 2.36**).

**1.** Infantile scoliosis

- **a.** If patient is under age 1 or the curve less than about 25 degrees, observe with follow-up in about 4 months.

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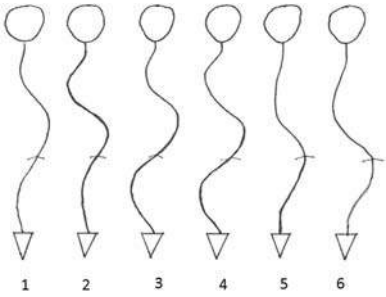


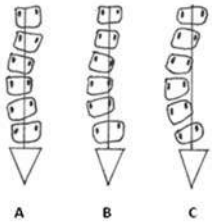

**Fig. 2.35** Lenke classification of curve types.

Curve Type

| Type | Proximal Thoracic | Main Thoracic | Thoracolumbar/ Lumbar | Curve Type |
|---|---|---|---|---|
| 1 | Non-Structural | Structural (Major*) | Non-Structural | Main Thoracic (MT) |
| 2 | Structural | Structural (Major*) | Non-Structural | Double Thoracic (DT) |
| 3 | Non-Structural | Structural (Major*) | Structural | Double Major *(DM) |
| 4 | Structural | Structural (Major*) | Structural | Triple Major* (TM) |
| 5 | Non-Structural | Non-Structural | Structural (Major*) | Thoracolumbar/Lumbar (TL/L) |
| 6 | Non-Structural | Structural | Structural (Major*) | Thoracolumbar/Lumbar (TL/L Main Thoracic (TL/L-MT) |

*Major = Largest curve identified by Cobb Measurement always structural
Structural curve = does not bend to 25 degrees or includes T2-5 or T10-L2 hyphosis 20 degrees

Modifiers




| Lumbar Spine Modifier | CSVL to Lumbar Apex |
|---|---|
| A | CSVL Between Pedicles |
| B | CSVL Touches Apical Body (ies) |
| C | CSVL Completely Medial |

| Thoracic Sagittal Profile T5-T12 |
|---|
| - (Hypo) < 10° |
| N (Normal ) 10° - 40° |
| + (Hyper) > 40° |

Lumbar modifiers: A-center sacral vertical line (CSVL) falls between pedicles of apical vertebra; B: CSVL touches pedicles of apical vertebra; C: CSVL does not touch apical vertebra

Thoracic Sagittal modifier for T5-12: + (> 40°), N (10-40°), - (<10°)

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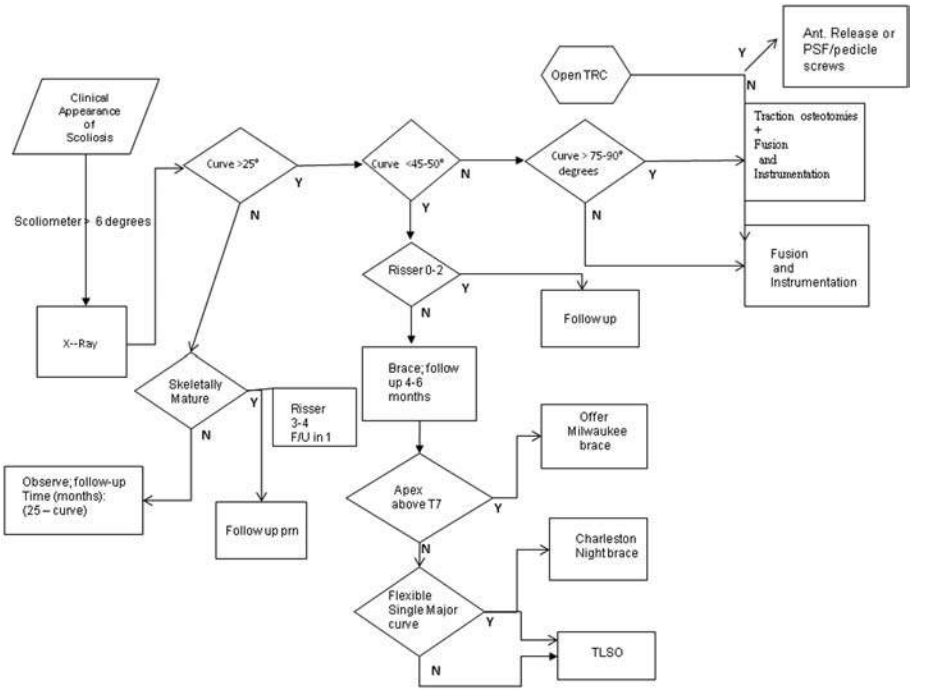



**Fig. 2.36** Idiopathic scoliosis treatment algorithm.

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**b.** Obtain MRI if curve is greater than about 25 degrees.
**c.** If RVAD is greater than 20 degrees or curve is greater than 35 degrees, consider Mehta casting followed by bracing in younger patients.
**d.** If curve progresses to greater than about 60 degrees, consider growing rods.

**2.** Juvenile scoliosis

**a.** If curve is greater than 25 degrees, order MRI.
**b.** If curve is greater than 25 degrees, consider full-time brace.
**c.** If curve progresses beyond 50 to 60 degrees, consider growing rods (or stapling) if the patient is younger than 9 years or definitive fusion if over the patient is 10 years or older.

**3.** Adolescent scoliosis

**a.** Curve over 25 degrees, Risser 0 to 2: Brace full-time or part-time (part-time braces less effective for double curves or those over 35 degrees)
**b.** Risser 3+: Observe or night-brace for relevant patterns
**c.** Discontinue brace when Risser 5 and height gain is less than 1 cm in 6 months
**d.** Curve greater than 50 to 60 degrees: Discuss surgery

## ◆ Back Pain in Children

Although disabling back pain in children is rare, back pain of some degree is experienced by over 30% of children. History should include the following:

**1.** Mechanism of onset
**2.** Duration
**3.** Severity (1 to 10)
**4.** Interference with school, play, or sports

### Physical Examination

**1.** Neurologic examination
**2.** Spinal range of motion
**3.** Location of pain
**4.** Straight leg raising

### Differential Diagnosis

**1.** Developmental

**a.** Spondylolysis (most common)

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- **b.** Scheuermann kyphosis (second most common)
- **c.** Tethered cord

**2.** Traumatic

- **a.** Herniated nucleus pulposus or endplate
- **b.** Musculoligamentous strain
- **c.** Fracture

**3.** Infectious

- **a.** Discitis
- **b.** Osteomyelitis
- **c.** Tuberculosis
- **d.** Sacroiliac joint infection

**4.** Tumor

- **a.** Benign bony neoplasm
  - **1)** Osteoid osteoma
  - **2)** Osteoblastoma
  - **3)** Aneurysmal bone cyst
  - **4)** Eosinophilic granuloma
- **b.** Malignant bony neoplasm
  - **1)** Ewing sarcoma
  - **2)** Osteogenic sarcoma
  - **3)** Leukemia
- **c.** Neoplasm: Neural
  - **1)** Glioma
  - **2)** Epidermoid
  - **3)** Neuroblastoma

**5.** Inflammatory

- **a.** Ankylosing spondylitis
- **b.** Enteropathic arthritis

**6.** Extraspinal

- **a.** Neural
- **b.** Intestinal
- **c.** Vascular
- **d.** Psychologic

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### Warning Signs of a Serious Underlying Disorder

1. Neurologic abnormality
2. Repeated interference with function (school, play, sports)
3. Prolonged stiffness
4. Fever

### Imaging and Treatment

If warning signs are present, aggressive workup including plain radiographs, bone scan, or MRI is indicated. If not, close follow-up with appropriate activity modification followed by an exercise program for abdominal and extensor muscles may be used at first. Further tests and treatment should then be undertaken if needed.

## Bibliography

Curtis C, d'Hemecourt P. Diagnosis and management of back pain in adolescents. Adolesc Med State Art Rev. 2007;18(1):140–164

## ◆ Congenital Scoliosis

### Introduction

1. Definition: Scoliosis that is due to primary vertebral malformation
2. Types
    a. Failure of segmentation (Bar): Most deforming (progression 5 degrees or more/year)
    b. Failure of formation (hemivertebra)
        1) Segmented (normal growth plates on either side): Progress up to 2 degrees per year.
        2) Semisegmented
        3) Unsegmented (nonprogressive)
3. Progression is greatest in first 2 years of life or in adolescent growth spurt.

### Associated Findings

1. Spinal dysraphism
2. VATER (vertebral defects [imperforate] anus, tracheoesophageal [fistula], radial and renal [dysplasia]) or VACTERLS association (vertebral anal, cardiac, tracheal, esophageal, renal, limb) malformations
3. Goldenhar syndrome (oculoauriculovertebral dysplasia)

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4. Arthrogryposis
5. Multiple pterygium syndrome
6. Skeletal dysplasia
7. Klippel–Feil syndrome
8. Sprengel anomaly (congenital scapular elevation)

### Imaging

1. Compare films from infancy
2. Coned AP and oblique films
3. Cardiac auscultation
4. Renal ultrasound or intravenous pyelogram
5. MRI if there is neurologic asymmetry or if surgery is planned
6. *Note:* Measurement error is greater than in idiopathic scoliosis—10 to 19 degrees.

### Treatment

1. Bracing has little or no value.
2. Document progression with films taken in same position every 6 to 12 months.
3. If progressive:
   a. Acceptable deformity: Fuse in situ (anterior and posterior if well-formed vertebral body)
   b. Unacceptable deformity
      1) Anterior and posterior convex growth arrest if under age 5
      2) Osteotomy or excision
      3) Careful instrumentation of flexible segment of curve is an option.

## ◆ Scheuermann Kyphosis

Developmental wedging of three or more adjacent vertebrae over 5 degrees. Incidence is about 5% of general population.

### Clinical Features

1. Thoracic or thoracolumbar kyphosis
2. Pain within curve, usually during growth spurt
3. Mild scoliosis may coexist.
4. Tight hamstrings

### Radiographic Findings

1. Wedging of three or more vertebrae

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2. Irregularity of end plate
3. Disk space narrowing
4. Schmorl nodes

### Treatment

1. Exercise program: Strengthen abdominal and hip extensor muscles and stretch hamstrings.
2. NSAIDs
3. Bracing of curve if 50 to 70 degrees and skeletal maturity less than Risser 3.
4. Operative correction: Optional for curves greater than 75 degrees if pain persists or deformity is objectionable to patient.
    a. Posterior column shortening and fusion alone if flexible.
    b. Anterior release and posterior fusion if rigid.

## Bibliography

Lowe TG. Scheuermann disease. J Bone Joint Surg Am. 1990;72:940–945

## ◆ Spondylolysis and Spondylolisthesis

Spondylolisthesis is the most common identifiable cause of back pain in children. The prevalence begins to plateau at around 6% by age 6 years. Etiology is a stress fracture in a susceptible pars interarticularis. The fifth lumbar vertebra is the most commonly involved. Twenty percent of pars defects are unilateral.

### Classification

1. Dysplastic
2. Isthmic (most common)
3. Degenerative
4. Traumatic
5. Pathologic

### Risk Factors

1. Risk factors for isthmic spondylolysis
    a. Positive family history
    b. Spina bifida occulta of L5
    c. Excessive stress
        1) Scheuermann kyphosis

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**2)** Gymnastics
**3)** Athetosis
**4)** Football lineman

**2.** Risk factors for progressive slip

**a.** Preadolescent age
**b.** Female
**c.** Dysplastic slip
**d.** High-grade slip (III or IV)
**e.** High slip angle

### Signs and Symptoms

Signs and symptoms most commonly develop in adolescence.

**1.** Symptoms

**a.** Low back pain: Activity related; worse with extension
**b.** Pain in buttocks or proximal thighs

**2.** Signs

**a.** Stiff-legged gait
**b.** Limited forward flexion
**c.** Prominent ilia




**Fig. 2.37** Measurement of percent slip is performed with respect to a line drawn from the posterior cortex of the sacrum. Note that the reference line from the superior vertebra is parallel to the sacral line, not the L5 cortex. (From Bradford DS. Spondylolysis and spondylolisthesis. In Lonstein JE, Bradford DS, Winter RB, Ogilvie JW, eds. Moe's Textbook of Scoliosis and Other Spinal Deformities. 3rd ed. Philadelphia: W.B. Saunders; 1995;406 (Fig. 19-5A). Reprinted with permission.)

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**d.** Palpable step-off of spinous process if greater than 25%
**e.** Weakness of ankle or bladder dysfunction (rare)

### Plain Radiographic Findings

1. Pars defect on lateral film
2. Pars defect on oblique film (Scotty dog's neck)
3. Elongation of pars
4. Vertebral body slippage (see **Fig. 2.37** for measurement technique)
5. Relative lumbosacral kyphosis (**Fig. 2.38**)

### Additional Radiographic Studies

1. Bone scan of spine with single-photon emission computed tomography if plain films are negative or need to determine acute versus chronic.
2. CT to visualize or confirm subtle spondylolysis
3. MRI if significant neurologic physical finding exists

### Treatment

1. Activity restriction as indicated
2. Consider brace treatment for healing of acute slip.
3. Strengthen abdominal and extensor muscles.
4. Fusion if severe pain or symptoms persist or if slip is greater than 50%
5. Repair of defect if slip is less than 5 mm and symptomatic and patient is under age 25

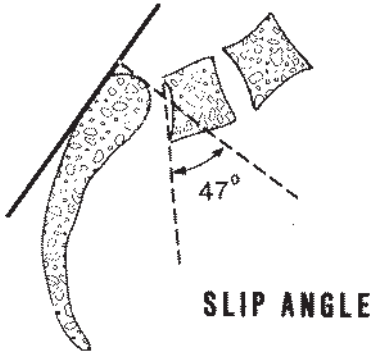



**Fig. 2.38** Measurement of slip angle (lumbosacral kyphosis). Note that the sacral reference line is drawn perpendicular to the posterior cortex because the sacral end plate may be rounded. (From Bradford DS. Spondylolysis and spondylolisthesis. In Lonstein JE, Bradford DS, Winter RB, Ogilvie JW, eds. Moe's Textbook of Scoliosis and Other Spinal Deformities. 3rd ed. Philadelphia: W.B. Saunders, 1995;406 (Fig. 19-5B). Reprinted with permission.)

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6. Reduction is controversial and usually considered only if slip is high grade and deformity is the primary complaint.
7. Skeletally immature patients should be followed up during growth to watch for progression.

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Hu SS, Tribus CB, Diab M, Ghanayem AJ. Spondylolisthesis and spondylolysis. Instr Course Lect. 2008;57:431–445

## ◆ Osteochondroses

Disordered behavior of growing cartilage under load. This may include compressive or tensile loads. Most common age to present is 7 to 12 years.

### Classification

1. Spine: Scheuermann kyphosis
2. Upper extremity
   - **a.** Panner (capitellum)
   - **b.** Madelung (distal radial physis)
3. Lower extremity
   - **a.** Perthes
   - **b.** Osgood–Schlatter (tibial tubercle)
   - **c.** Blount (medial tibial physis)
   - **d.** Kohler (tarsal navicular)
   - **e.** Freiberg (2nd metatarsal head)
   - **f.** Sever disease (calcaneal apophysis)

### Treatment

Cartilage heals and remodels with time. Minimize load. Bracing may help (Blount, Scheuermann). Reconstruct if deformity develops (Perthes, Scheuermann, Blount, Madelung).

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McKenzie W. Localized disorders of bone. In: Morrissy RT, Weinstein SL, eds. Lovell and Winter's Pediatric Orthopaedics, 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2005:346–348

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## ◆ Musculoskeletal Tumors

Most pediatric skeletal tumors are benign. The most common primary malignancies include osteosarcoma, Ewing sarcoma, and rhabdomyosarcoma. Secondary skeletal involvement may occur with leukemia and neuroblastoma and peripheral neuroectodermal tumor.

### Symptoms

Symptoms that can indicate malignancy include night pain that is not activity related, rapid increase in pain, increasing fatigue, or bruising.

### Laboratory Studies

1. Erythrocyte sedimentation rate:
   a. Mildly elevated for most malignant tumors
2. Complete blood cell count: Abnormal in leukemia and lymphoma

### Radiologic Studies

1. Plain films: Location, morphology, and host reaction are most diagnostic features. Locations of most common benign and malignant tumors are shown in **Fig. 2.39**.
2. Radionuclide scans: Very sensitive for malignant bone and soft tissue tumors; may be negative in eosinophilic granuloma
3. CT: Best when bony changes need to be better defined
4. MRI: Does not show bony detail well but does show soft tissue and intramedullary detail well

### Tumor Types

1. Benign
   a. Eosinophilic granuloma: Reticuloendothelial lesion usually centrally located in one or several bones; poorly or well circumscribed. Usually waxes and wanes spontaneously.
   b. Osteoid osteoma: Painful nidus surrounded by sclerosis; usually found in patients aged 6 to 25 years.
   c. Osteochondroma (osteocartilaginous exostosis), metaphyseal, solitary, or multiple; growth ceases at maturity.
   d. Chondromyxoid fibroma: Eccentric local lesion, usually in lower extremity progressively enlarging. Patients aged 10 to 25 years.
   e. Chondroblastoma epiphyseal tumor of adolescence; lucent with foci of internal calcification.

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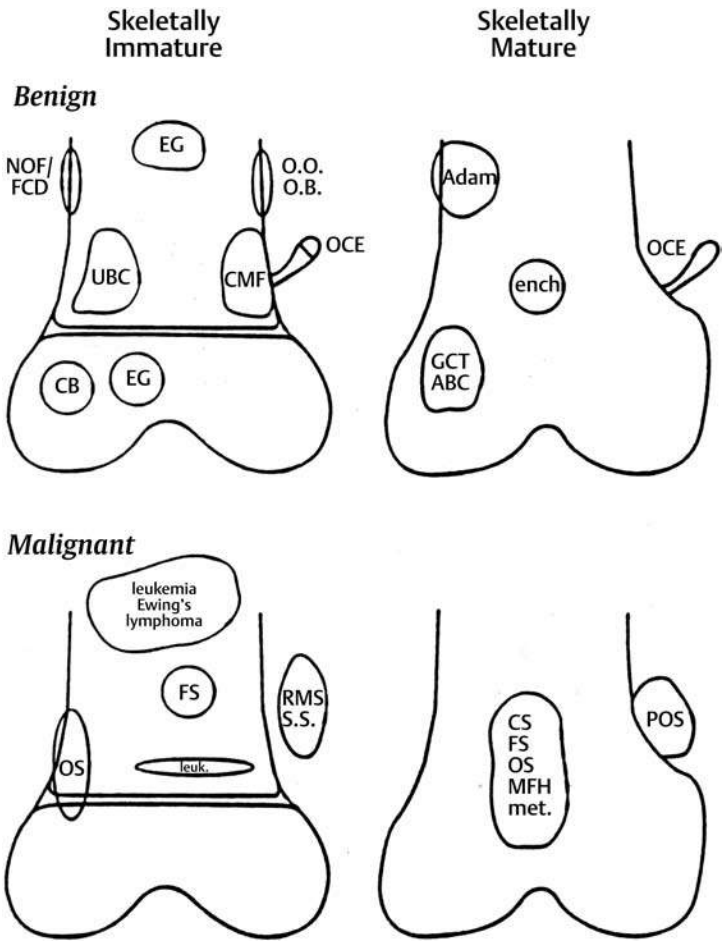



**Fig. 2.39** Sites of musculoskeletal tumors in skeletally immature and skeletally mature children. See text for explanation of abbreviations.

- **f.** Unicameral bone cyst: Central lucent metaphyseal lesion usually of proximal humerus or femur; expands and thins cortex; resolves at maturity.
- **g.** Nonossifying fibroma: Eccentric intracortical deficit in metaphysic; resolves by maturity.
- **h.** Adamantinoma (adam): Sclerotic anterior cortical deficit, usually of tibia.
- **i.** Enchondroma (ench) central lucent defect with internal calcification.

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**j.** Giant cell tumor: Lucent epimetaphyseal tumor just after maturity.
**k.** Aneurysmal bone cyst (ABC): expansile metaphyseal lesion of late adolescence that destroys cortex but leaves thin shell.

**2.** Malignant

**a.** Fibrosarcoma
**b.** Osteosarcoma: Most common primarily malignant bone tumor, metaphyseal; located in fastest growing regions
**c.** Chondrosarcoma: Central or peripheral, expansile tumor of young adults
**d.** Ewing sarcoma: Small cell tumor of diaphysis; patients aged 5 to 15; usually lasts with extensive periosteal reaction

### Tumors Common to Specific Locations in Children

**1.** Long bones (**Fig. 2.39**)
**2.** Spine

**a.** Posterior elements
  **1.** Aneurysmal bone cyst
  **2.** Osteoid osteoma
  **3.** Osteoblastoma
**b.** Vertebral body
  **1.** Histiocytosis
  **2.** Hemangioma
  **3.** Osteosarcoma
  **4.** Ewing sarcoma
  **5.** Chordoma

**3.** Ribs

**a.** Fibrous dysplasia
**b.** Ewing sarcoma
**c.** Chondrosarcoma
**d.** Metastasis

**4.** Pelvis

**a.** Ewing sarcoma
**b.** Fibrous dysplasia
**c.** Aneurysmal bone cyst
**d.** Osteoblastoma
**e.** Eosinophilic granuloma

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**f.** Leukemia
**g.** Osteosarcoma

**5.** Scapula

**a.** Ewing sarcoma
**b.** Osteoblastoma
**c.** Aneurysmal bone cyst

### Staging

**1.** Malignant tumors (**Table 2.3**)
**2.** Benign tumors

**a.** Latent
**b.** Active
**c.** Aggressive; May expand into soft tissues or metastasize (**Table 2.4**)

## ◆ Musculoskeletal Problems in Hemophilia

Hemophilia A (factor VIII) and B (factor IX deficiency) are the two most common bleeding disorders, followed by von Willebrand disease. Factor levels below 5% of normal level indicates risk of serious bleeding. These conditions should be jointly managed by hematology and orthopedic specialists (**Table 2.5**).

### Treatment of Acute Hemarthropathy

**1.** Factor replacement 50% every 48 hours × 6 days
**2.** Aspiration
**3.** Immobilization
**4.** Rehabilitation

**Table 2.3** Malignant Tumors

| Surgical Stage | Surgical Grade (G) | Site (T) | Metastases (M) |
|---|---|---|---|
| IA | Low ($G_1$) | Intracompartmental ($T_1$) | $M_0$ B |
| | Low ($G_1$) | Extracompartmental ($T_2$) | $M_0$ |
| IIA | High ($G_2$) | Intracompartmental ($T_1$) | $M_0$ |
| | High ($G_2$) | Extracompartmental ($T_2$) | $M_0$ |
| III | Any | Any T | |

*Source*: Enneking WJ. Musculoskeletal Tumor Surgery. New York: Churchill Livingstone, 1983.

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**Table 2.4** Common Bone Tumors

| Type of Tumor | Abbreviation | Name |
|---|---|---|
| Benign | | |
| | EG | Eosinophilic granuloma |
| | OO/OB | Osteoid osteoma/osteoblastoma |
| | OCE | Osteochondroma |
| | CMF | Chondromyxofibroma |
| | CB | Chondroblastoma |
| | UBC | Unicameral bone cyst |
| | NOF/FCD | Nonossifying fibroma/fibrous cortical defect |
| | Adam | Adamantinoma |
| | Ench | Enchondroma |
| | GCT | Giant cell tumor |
| | ABC | Aneurysmal bone cyst |
| Malignant | | |
| | FS | Fibrosarcoma |
| | OS | Osteosarcoma |
| | CS | Chondrosarcoma |
| | MFH | Malignant fibrous histiocytoma |
| | POS | Parosteal osteosarcoma |
| | Met | Metastasis |
| | SS | Synovial sarcoma |
| | RMS | Rhabdomyosarcoma |

### Treatment of Subacute Hemarthropathy

1. Factor replacement 30% X 2–6 weeks.
2. Strengthen and increase ROM.
3. Consider synovectomy if not resolved

**Table 2.5** Factor Doses and Kinetics

| Factor | % Rise After l Unit/kg Dose | Approximate Half-life (h) |
|---|---|---|
| VIII | 2 | 12 |
| IX | 1 | 24 |

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### Control of Bleeding after Fracture

1. Factor replacement 50% X 1–2 days.
2. Factor replacement 30% X 1 week.

### Radiographic Evaluation

*Arnold Classification*

1. Soft tissue swelling
2. Osteopenia, epiphyseal overgrowth
3. Subchondral cysts; wide or square contours
4. Irregular joint space
5. Absent joint space

## Bibliography

Heyworth BE, Su EP, Figgie MP, Acharya SS, Sculco TP. Orthopedic management of hemophilia. Am J Orthop. 2005;34(10):479–486

## ◆ Musculoskeletal Infections

### Evaluation/Workup

Obtain all necessary cultures before starting antibiotics.

1. Blood cultures in all, if possible
2. Spinal tap if indicated
3. Aspiration of bone, joint, or abscess
4. MRI if multiple tissues involved
5. Bone scan if location difficult on physical examination

### Differential Diagnosis

1. Transient synovitis
2. Postinfectious arthritis
3. Juvenile rheumatoid arthritis

**Table 2.6** Empiric Antibiotic Selection for Skeletal Infection

| Age (mo) | Antibiotic |
|---|---|
| <1 | Ampicillin/sublactam + vancomycin |
| 1–4 | Vancomycin + ceftriaxone |
| >4 | Clindamycin; or vancomycin +Rifampin for severe infections |

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**Table 2.7** Organisms Causing Osteomyelitis

| Organism | % |
|---|---|
| *Staphylococcus aureus*, MS | 22 |
| *Staphylococcus aureus*, MR | 22 |
| *Staphylococcus epidermidis* | 5 |
| Grade A β-hemolytic streptococcus | 4 |
| *Pseudomonas aeruginosa* | 4 |
| *Enterobacter cloacae* | 1 |
| *Kingella kingae* | 1 |
| *Streptococcus pneumoniae*, *Escherichia coli*, enterococcus, *Candida* spp. | each <1 |
| No growth/unknown | 38 |

4. Ewing sarcoma
5. Rheumatic fever

### Treatment

Empiric antibiotics recommendations by age (**Table 2.6**):

### Organism

Varies with age; staphylococcal most common at all ages (**Tables 2.7** and **2.8**)

**Table 2.8** Organisms Causing Septic Arthritis

| Organism | % |
|---|---|
| *Staphylococcus aureus*, MS | 14 |
| *Staphylococcus aureus*, MR | 5 |
| Group A β-hemolytic streptococcus | 6 |
| Streptococci | 4 |
| Staphylococcus epidermidis | 3 |
| Neisseria gonorrhea | 2 |
| *Candida* spp. | 2 |
| Haemophilus influenza | 1 |
| Group B streptococcus | 1 |
| Escherichia coli | 1 |
| Enterobacter cloacae | 1 |
| No growth/unknown | 63 |

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Copley LA. Pediatric musculoskeletal infection: trends and antibiotic recommendations. J Am Acad Orthop Surg. 2009;17(10):618–626
Gafur OA, Copley LA, Hollmig ST, Browne RH, Thornton LA, Crawford SE. The impact of the current epidemiology of pediatric musculoskeletal infection on evaluation and treatment guidelines. J Pediatr Orthop. 2008;28(7):777–785

## ◆ Lyme Disease in Orthopedics

### Background

1. Definition: An immune-mediated disorder in reaction to infection by the spirochete *Borrelia burgdorferi*. The disease was first characterized after an epidemic of involvement in Old Lyme, Connecticut, in the mid-1970s. Since then, other endemic areas have been identified. It may include rash, arthritis, synovitis, carditis, or neurologic manifestations.
2. It may have acute and chronic stages. Acute stage consists of rash and early arthritis. The chronic stage involves arthritis, carditis, and neuritis.
3. There are three major endemic areas in the United States:
    a. Upper mid-Atlantic area from Massachusetts to Maryland
    b. Upper Midwest (especially Wisconsin and Minnesota)
    c. Western states of Oregon, Utah, Nevada, and California
4. The human leukocyte antigen (HLA)-DR4 haplotype predicts increased risk of disease.

### Signs and Symptoms

1. Acute: Spreading rash known as erythema chronicum migrans, beginning 3 to 30 days after a tick bite
2. Fever
3. Headache
4. Malaise
4. Migratory arthralgias and myalgias
5. Chronic swelling of large joints, most commonly the knee
6. Involvement of one or more joints
7. Pain, which may be minimal, as in juvenile rheumatoid arthritis, or acute, resembling bacterial arthritis
8. Cardiac involvement, possibly including atrioventricular block or myocarditis
9. Neurologic involvement, possibly including seventh cranial nerve (facial) palsy, meningoencephalitis, or peripheral neuropathy

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### Physical Findings

1. Erythema chronicum migrans: spreading, oval rash
2. Examine for cranial nerve or peripheral nerve palsy
3. Examine all joints for effusion, even if painless
4. Auscultate patient's heart

### Laboratory Tests

1. Erythrocyte sedimentation rate is usually elevated.
2. Tests for Lyme disease include two methods of antibody detection.
   a. Enzyme-linked immunosorbent assay for spirochete is sensitive but not specific. A titer of more than 1:80 is considered positive. If positive, this test should be followed by Western blot test, a gel electrophoresis technique, which is more specific.
   b. Arthrocentesis is not a specific test for Lyme disease, but it is often performed to rule out other disorders. The white blood cell count is 25,000 to 90,000 and may include up to 95% polymorphonuclear leukocytes. The spirochete is not recoverable from joint fluid.
3. Electrocardiography may be indicated to demonstrate atrioventricular block.

### Imaging

1. Plain radiographs of the affected area are indicated.
2. Joint changes may include soft tissue swelling in early stages, osteopenia if the inflammation has been present for several weeks, and joint space narrowing if it has been chronic.

### Differential Diagnosis

1. Juvenile rheumatoid arthritis
2. Bacterial arthritis
3. Rheumatic fever

### Treatment

1. Consult other specialists, such as those in infectious disease, neurology, rheumatology, or cardiology, as appropriate.
2. Start treatment with oral (early stages of disease) penicillin or amoxicillin empirically.
3. Tetracycline is also an option for children who are older than 8 years. It should not be used in younger children because of potential discoloration of teeth.

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### Prognosis

Prognosis is usually good unless late joint changes or neurologic complications have occurred.

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Rose CD, Fawcett PT, Eppes SC, Klein JD, Gibney K, Doughty RA. Pediatric Lyme arthritis: clinical spectrum and outcome. J Pediatr Orthop. 1994;14(2):238–241
Steere AC. Lyme disease. N Engl J Med. 1989;321(9):586–596

## ◆ Congenital Constriction Band Syndrome

### Background

1. Synonyms: Amniotic band syndrome or Streeter dysplasia. The details of etiology are unknown but relate to in utero bands or strands of tissue that encircle and damage the fetus. There are four basic features of the syndrome (some or all of these may be present):

   **a.** Bands encircling the skin of extremities
   **b.** In utero amputation of digits or portions of extremity
   **c.** Acrosyndactyly (terminal syndactyly with slit-like separations proximally)
   **d.** Secondary effects of constriction: nerve palsy, vascular or lymphatic occlusion, clubfoot, or other contractures.

2. Incidence: Around 1 per 5000 live births. Upper extremities are more commonly involved than lower extremities. The incidence of clubfoot is as high as 30%. If clubfoot occurs, it may be more resistant to treatment.

### Physical Features and Examination

All extremities should be carefully examined, especially the tips of the digits, which may have only subtle involvement. Rarely there is significant neurovascular impairment distally. In some cases, this may become worse after birth and may require urgent release of the bands. Syndactyly and hypoplastic nails are often present. The clubfoot may be paralytic if ipsilateral to a band, or it may resemble idiopathic clubfoot when contralateral.

### Differential Diagnosis

1. Hair thread constriction may occur in infants, and the threads may be difficult to see after swelling occurs.
2. "Michelin tire baby syndrome," which is extensive, and symmetrical rolls of skin

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### Treatment

1. The constriction bands may need urgent release if there is circulatory constriction in a newborn, but this is rare.
2. Usually the bands can be excised and reconstructed when the child is 6 to 12 months of age. This can be done circumferentially at one sitting. The scarred groove should be removed until the tissues have a normal depth on either side. Dissect band off of neurovascular structures and muscle so that there is no residual constriction. The remaining edges can then be reapproximated using Z-plasty techniques so that the scar does not form a recurrent constriction.
3. Some bands are shallow enough that no surgery is needed.
4. Partial amputations may not have been completed by birth. If the tissue bridge is minimal, it can be tied off. Otherwise the amputation will need to be completed in the operating room. Amputations through bone in growing children often present later with spike formation, which requires revision.
5. Clubfoot associated with constriction band syndrome should be treated initially with the Ponseti method. The recurrence rate is significant. If multiple recurrences develop, limited open release or tendon transfer may be indicated.

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# 3 Skeletal Syndromes and Systemic Disorders in Pediatric Orthopedics

This chapter summarizes developmental syndromes that involve skeletal abnormalities. A diagnosis or discussion of prognosis may be required. Because knowledge of a large number of facts rather than basic principles is required, this chapter was created to help. It is organized according to presenting features (see below). More detailed discussion of these conditions is available in the cited references.

## ◆ Skeletal Dysplasias

Skeletal dysplasis involve basic and systemic abnormalities of bone and cartilage growth and development. Usually short stature occurs.

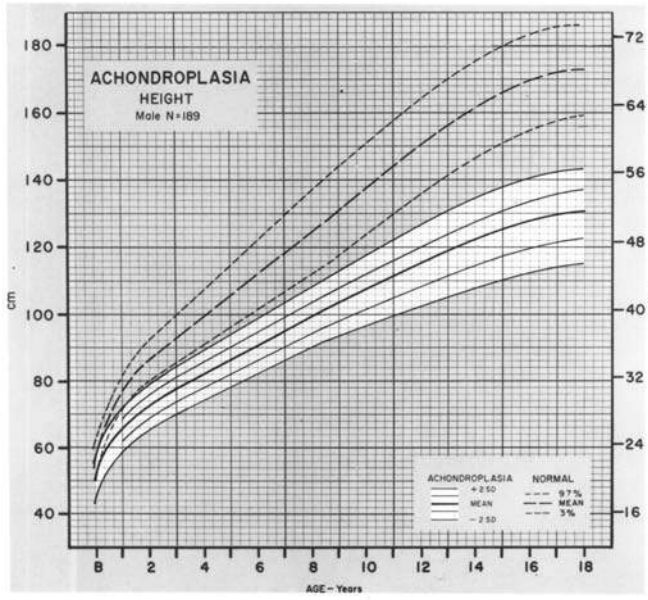
**1.** Achondroplasia

- **a.** Autosomal dominant with frequent new mutations. Most common skeletal dysplasia.
- **b.** Genetic defect: Fibroblast growth factor receptor protein 3 (a gain-of-function mutation).
- **c.** Major features
  - **1)** Midface hypoplasia
  - **2)** Rhizomelic dwarfism
  - **3)** Genu varum (variable)
  - **4)** A 3- to 6-month delay in motor milestones
  - **5)** Thoracolumbar kyphosis, often resolving with growth
  - **6)** Spinal stenosis is greatest in the lumbar spine, greatest distally, but may affect entire spine, including foramen magnum, and cause severe developmental delay.
- **d.** Height graph (**Fig. 3.1**)
- **e.** Treatment: Monitor for spinal stenosis and persistent kyphosis. Correction of knee and ankle deformities at patient's discretion. Limb lengthening is an option and is usually successful but time consuming.

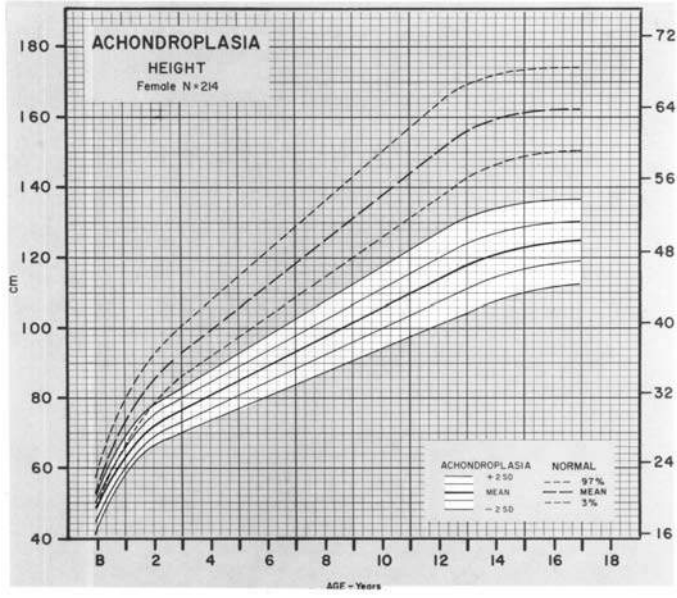
**2.** Pseudoachondroplasia

- **a.** Although these patients are also rhizomelic, the epiphyseal involvement in this syndrome causes major differences from achondroplasia.
- **b.** Genetic defect: COMP (cartilage oligomeric matrix protein); found in extraterritorial matrix).

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A




B

**Fig. 3.1** Comparison of growth patterns of normal-stature and achondroplastic persons. **(A)** Males. **(B)** Females. (From Horton WA, Rotter JI, Rimoin DL, Scott CI, Hall JG. Standard growth curves for achondroplasia. J Pediatr. 1978;93(3):436 (Figs. 1 and 2). Reprinted with permission.)

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**c.** Major features
  **1)** Rhizomelic shortening of extremities
  **2)** Variable knee deformities (often varus on one side, valgus on the other)
  **3)** Mild platyspondyly; minimal scoliosis; no stenosis
  **4)** Odontoid hypoplasia, possible C1-C2 instability
  **5)** Epiphyseal deformation; eventual degeneration
  **6)** Ligamentous laxity
**d.** Treatment: Screen cervical spine; correct major limb malalignment. Many patients require joint arthroplasty as adults.

**3.** Diastrophic dysplasia

**a.** Autosomal recessive
**b.** Genetic defect: Diastrophic sulfate transporter (DTST)
**c.** Major features
  **1)** "Cauliflower ear" developing at around 6 months of age
  **2)** Rhizomelic shortening of extremities
  **3)** Contractures of major joints with later degenerative joint disease (DJD)
  **4)** Hands: Hitchhiker thumb, symphalangism
  **5)** Dislocated hips: Occasionally
  **6)** Equinovarus or other foot deformities
  **7)** Cervical spina bifida with severe kyphosis: Sometimes resolves
  **8)** Scoliosis of thoracic and lumbar spine
**d.** Treatment: Screen and monitor the cervical spine. Correct foot deformities, scoliosis, and limb contractures. Arthroplasty as indicated.

**4.** Spondyloepiphyseal dysplasia congenita

**a.** Autosomal dominant with frequent new mutations
**b.** Genetic defect: Collagen 2A1
**c.** Major features
  **1)** Extreme short stature
  **2)** Odontoid hypoplasia/os odontoideum: May have instability
  **3)** Platyspondyly, scoliosis
  **4)** Coxa vara, epiphyseal irregularity, DJD
**d.** Treatment: Screen or stabilize the cervical spine. Correct scoliosis as indicated. Treatment for hip dysplasia is of uncertain benefit. Joint arthroplasty is often indicated in adulthood.

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**5.** Spondyloepiphyseal dysplasia (SED) tarda

- **a.** Variable transmission; diagnosis late in childhood
- **b.** Genetic defect: SEDL (a tracking protein) or others
- **c.** Major features
  - **1)** Irregular ossification, DJD
  - **2)** Hips may resemble Perthes, but in SED bilaterally synchronous
  - **3)** Osteoarthritis of other joints
  - **4)** Scoliosis

**6.** Multiple epiphyseal dysplasia

- **a.** Autosomal dominant
- **b.** Gene defect: COMP (found in matrix) or collagen 9 or DTST
- **c.** Major features
  - **1)** Variable; usually mild short stature because of short limbs
  - **2)** Irregular epiphyseal ossification with deformity, pain, DJD
  - **3)** Hips, knees, ankles are most involved. Patella may show "double layer."
  - **4)** Usually presents in late childhood to adulthood

**7.** Metatropic dysplasia

- **a.** Major features
  - **1)** Epiphyseal or metaphyseal enlargement: "Knobby" joints with contractures
  - **2)** Cervical stenosis, instability
  - **3)** Scoliosis, kyphosis, later onset
  - **4)** Coccygeal tail
  - **5)** Thoracic hypoplasia; may cause respiratory compromise
  - **6)** Initially short-limb dwarfism; becomes short-trunk type with onset of scoliosis

**8.** Chondrodysplasia punctata (Conradi–Hunerman)

- **a.** Autosomal dominant, recessive, and X-linked
- **b.** Major features
  - **1)** Multiple asymmetric epiphyseal calcifications
  - **2)** Rhizomelic form may have cervical stenosis or kyphosis as well as thoracolumbar scoliosis.
  - **3)** Good prognosis for dominant form. Decreased life expectancy for other forms

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**9.** Multiple hereditary exostoses (MHE)

- **a.** Inheritance: Autosomal dominant
- **b.** Genetic defect: At least three have been described; EXT-1 and -2 on different chromosomes. EXT-1 produces more serious form.
- **c.** Clinical appearance: Mild short stature
- **d.** Categories of problems
  - **1)** Local impingement on tendons, nerves, spinal canal, ribs
  - **2)** Asymmetrical growth in two-bone segments (forearms and legs) leading to valgus at knees, ankles, elbow, and wrists and possibly radial head dislocation.
  - **3)** Leg-length inequality (usually less than 4 cm)
  - **4)** Risk of malignant degeneration (in about 1% of patients)
  - **5)** Osteochondromas may grow silently in spinal canal; monitor neurologic examination.
  - **6)** Patients with MHE often heal incisions with wide scars or keloids.
- **e.** Radiographic features
  - **1)** Osteochondromas in metaphysis, pointing away from joint
  - **2)** Cortex of osteochondroma is confluent with that of host bone.
  - **3)** May be sessile or pedunculated
- **f.** Treatment
  - **1)** Resect lesions only when symptomatic. Increased rotation not predictable in forearm.
  - **2)** Correct knee and ankle valgus when greater than 10 degrees.
  - **3)** Monitor in adulthood every 2 years, possibly with bone scan.
  - **4)** Obtain spine magnetic resonance imaging (MRI) if there is any question of involvement.

**10.** Dysplasia epiphysialis hemimelica (Trevor disease)

- **a.** Definition: Epiphyseal osteochondroma; no genetic pattern
- **b.** Clinical features: Presents in first decade of life; restricted joint motion, enlarged joint, or locking. Knee, foot, and ankle are most commonly involved.
- **c.** Radiographs: Multiple opacities in exostotic cartilage; these eventually coalesce.
- **d.** Treatment: Resection, attempting to preserve normal cartilage

## Bibliography

Kettelkamp DB, Campbell CJ, Bonfiglio M. Dysplasia epiphysealis hemimelica: a report of fifteen cases and a review of the literature. J Bone Joint Surg Am. 1966;48(4):746–765, discussion 765–766

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**11.** Multiple enchondromas (Ollier disease)

- **a.** Genetic defect: *PTH*/*PTHRP*
- **b.** Clinical presentation
  - **1)** Angular deformity
  - **2)** Bony irregularity
  - **3)** Limb-length inequality
- **c.** Radiographic features
  - **1)** Diffuse enchondromas in metaphysis; occasionally epiphyses. Usually asymmetrical
- **d.** Treatment
  - **1)** Angular or length correction of limb
  - **2)** Monitor for malignancy, especially in Maffucci syndrome

**12.** Cleidocranial dysplasia

- **a.** Autosomal dominant
- **b.** Genetic defect: *CBFA1*, a transcription factor
- **c.** Clinical features
  - **1)** Persistently open skull sutures with bulging calvarium
  - **2)** Hypoplasia or aplasia of clavicles
  - **3)** Wide symphysis pubis
  - **4)** Hip abnormalities (coxa vara)
  - **5)** Short middle phalanx of fifth finger
  - **6)** Scoliosis with or without syringomyelia
  - **7)** Multiple dental abnormalities

## Bibliography

Jensen BL. Somatic development in cleidocranial dysplasia. Am J Med Genet. 1990; 35(1):69–74

**13.** Dyschondrosteosis (Leri–Weill disorder)

- **a.** Genetic defect: *SHOX* pseudoautosomal genes
- **b.** Major features
  - **1)** Mild short stature (<25 percentile)
  - **2)** Madelung deformity (dorsoulnar deficiency of distal radial growth)
  - **3)** Relative shortening of forearm and leg; varus or valgus deformity
  - **4)** Females predominant

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## ◆ Other Syndromes Involving Short Stature

**1.** Cornelia de Lange (Brachmann de Lange type)

- **a.** Genetic defect: *NIPBL*; or microdeletion on chromosome 3
- **b.** Major features
  - **1)** Synophrys (single eyebrow)
  - **2)** Down-turned mouth
  - **3)** Mandibular spur in infancy
  - **4)** Hirsutism
  - **5)** Gastroesophageal reflux
  - **6)** Small for gestational age, with continued growth retardation
  - **7)** Motor, speech, and intellectual delay
  - **8)** Cardiac abnormalities
- **c.** Orthopedic involvement
  - **1)** Upper extremity anomalies (~100%)
    - **a)** Micromelia, phocomelia
    - **b)** Decreased number of fingers
    - **c)** Lobster-claw hand
    - **d)** Proximally placed thumb
    - **e)** Elbow anomalies
  - **2)** Lower extremities
    - **a)** Miscellaneous foot deformities and contracture
    - **b)** Avascular necrosis (AVN) of femoral head in 10%
- **d.** Treatment: Correct lower extremity abnormalities if limiting ambulation; upper extremities: individualized treatment

**2.** Riley–Day familial dysautonomia

- **a.** Ashkenazi Jews only: Autosomal recessive
- **b.** Sympathetic overactivity is key feature
- **c.** Major features
  - **1)** Deficient sensation of pain and proprioception
  - **2)** Gastroesophageal reflux, pneumonia
  - **3)** Variable life expectancy
- **d.** Orthopedic abnormalities and implications
  - **1)** Scoliosis/kyphosis before age 8; poor brace tolerance; fuse early
  - **2)** Fractures from osteopenia or dyscoordination
  - **3)** AVN of femoral head, distal femur, talus
  - **4)** Hip dysplasia

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**3.** Nail–patella syndrome

- **a.** Autosomal dominant, normal life expectancy
- **b.** Orthopedic features
  - **1)** Nails grooved, small, or absent, especially on thumb
  - **2)** Multiple knee anomalies: patella tripartite, small or absent, lateral femoral condyle hypoplastic, (valgus) osteochondritis dissecans of lateral femur and talus
  - **3)** Elbow: Capitellar hypoplasia, cubitus valgus, flexion contracture
  - **4)** Iliac horns
  - **5)** X-linked hypophosphatemic rickets

## ◆ Sclerosing Bone Disorders

**1.** Fibrodysplasia ossificans progressiva

- **a.** Progressive, disabling heterotopic ossification or ankylosis. Incidence 1:1,000,000
- **b.** Etiology: Enhanced signaling of *BMP4*
- **c.** Characteristic shortening/valgus of great toe
- **d.** Ossification starts as tender, hard nodule; progresses proximal to distal, posterior to anterior
- **e.** Do not biopsy; may accelerate the process
- **f.** Genetics: Usually a spontaneous mutation but may be transmitted as autosomal dominant

**2.** Progressive diaphyseal dysplasia (Camurati–Engelman disease):

- **a.** Clinical features: Pain, fatigue, muscle atrophy
- **b.** Etiology: activating mutation in transforming growth factor beta (TGF-β) genes
- **c.** Radiographs: Symmetrically widened, sclerotic diaphyses; epiphyses spared tibia, femur most commonly involved
- **c.** Treatment: Osteotomies only if marked deformity. Possible role for bisphosphonates

**3.** Melorheostosis

- **a.** Syndrome involving asymmetrical extraosseous longitudinal hyperostotic streaks resembling molten wax; limb pain and soft tissue contracture
- **b.** Treatment: Analgesics, bracing contracture releases, and bone shortening

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4. Osteopathia striata

   a. Linear intraosseous metaphyseal striations
   b. Autosomal dominant
   c. Asymptomatic
   d. No treatment required

5. Osteopoikilosis

   a. Multiple symmetrical intraosseous epiphyseal–metaphyseal "spots"
   b. Autosomal dominant
   c. Asymptomatic

## ◆ Fibrous Dysplasia

1. Background: Inheritance—somatic mutation that produces mosaic distribution of lesions in one (monostotic) or many (polyostotic) bones

   a. The molecular basis is a postzygotic activating mutation in the *GNAS1* gene, which encodes for the cyclic adenosine monophosphatase (cAMP)-regulating α subunit of the Gs protein complex.
   b. Histologically the fibrous tissue undergoes ossification to small, irregular trabeculae ("alphabet soup"). The disease process is most active during growth and causes weakening of the bone and pathologic fracture.

2. Clinical manifestations: Monostotic accounts for 80% of cases. Polyostotic is found in several bones on one side of the skeleton or scattered throughout the skeleton. In the axial skeleton, the craniofacial bones and the ribs are the most common sites; in the appendicular skeleton, the tibia and proximal femur are the most common sites.

   a. Monostotic fibrous dysplasia usually presents without symptoms, and the lesion is found when a radiograph is taken for unrelated reasons.
   b. Polyostotic fibrous dysplasia often results in distortion of the skeletal and facial configuration. The peak incidence of fractures is during the first decade of life, followed by a decrease thereafter. Lesions of the femoral neck may cause progressive coxa vara, leading to the shepherd's crook deformity; this is the most common angular deformity in polyostotic fibrous dysplasia. Spinal involvement and scoliosis may also occur.
   c. Skin lesions include café-au-lait spots with an irregular border ("coast of Maine").

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**d.** Fibrous dysplasia may be associated with significant endocrine disturbances: hyperthyroidism, phosphaturia, precocious puberty, and diabetes mellitus. The McCune–Albright syndrome includes the triad of polyostotic fibrous dysplasia, café-au-lait spots, and precocious puberty.

**3.** Imaging findings: Elongated lesion with symmetric cortical thinning and outward expansion, the characteristic diaphyseal "long lesion in a long bone." Lesion shows few trabecular markings and has a ground-glass appearance. Some may be entirely radiolytic or radiodense. There may be an associated angular deformity. Fibrous dysplasia shows excessive uptake on bone scan.

**4.** Treatment: Unnecessary for asymptomatic lesions of fibrous dysplasia. Large or symptomatic lesions may be treated by curettage and allografting. Lesions of the femoral neck should be treated with metallic support with or without cortical bone grafting because of the risk of fatigue fracture. If symptomatic varus deformity is present, treatment should include valgus osteotomy with cortical bone grafting and rigid internal fixation. Because of poor bone quality, intramedullary fixation is preferable to plates and screws alone for lesions of the femoral shaft. Deformity may occur at stress risers, similar to osteogenesis imperfecta. Medical management includes the use of bisphosphonates. Early studies suggest that bisphosphonates decrease pain, improve the radiologic appearance of the lesions, and decrease the fracture rate.

## ◆ Marfan and Related Disorders

**1.** Marfan syndrome (MFS)

**a.** This is a disorder of fibrillin-1, which also affects TGF-β distribution and has multiple effects on the skeleton and connective tissue. Because some features may be seen in the general population, the following diagnostic criteria have been developed.

**b.** Diagnostic (Ghent) criteria: Two major criteria (*below) and involvement of another system; system involvement (*indicates major manifestation)

**1)** Genetic: MFS diagnosis in first-degree relative or fibrillin gene mutation known to cause MFS*

**2)** Skeletal: (*major involvement if four features are found)

**a)** Pectus excavatum or carinatum

**b)** Dolichostenomelia (long, narrow limbs; arm span–height greater than 1.05)

**c)** Arachnodactyly (long, narrow digits with positive thumb and wrist sign). "Thumb" or Steinberg sign is when the entire distal phalanx of the thumb protrudes beyond the ulnar border of the clenched fist. "Wrist" or Walker–Murdoch sign is posi-

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tive when the thumb can overlap the nail of the fifth finger when clasping the opposite wrist) (**Fig. 3.2**).

- **d)** Vertebral column deformity (increased kyphosis, scoliosis greater than 20 degrees)
- **e)** Significant hindfoot valgus
- **f)** Facial features including a high narrow cranium, down-slanting eyes, narrowly arched palate
- **g)** Elbow flexion contracture
- **h)** Protrusio acetabulae

**3)** Ocular

- **a)** *Ectopia lentis (superolateral dislocation)
- **b)** Flat cornea
- **c)** Retinal detachment
- **d)** Myopia

**4)** Cardiovascular

- **a)** *Dilation of aortic root with ascending arch aneurysm
- **b)** *Aortic dissection: Usually ascending segment
- **c)** Aortic valve regurgitation

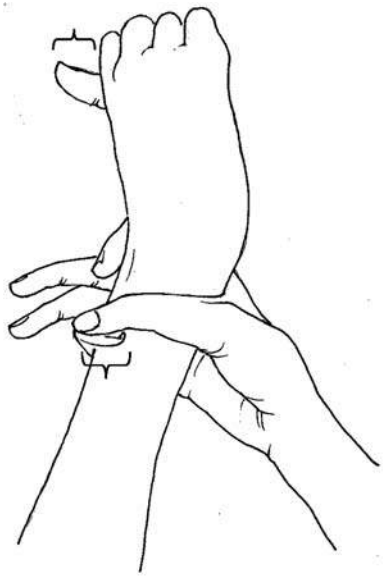


**Fig. 3.2** Illustration of the thumb and wrist signs. Thumb sign: The entire distal phalanx of the thumb protrudes beyond the ulnar border of the clenched fist. Wrist sign: Thumb covers the entire fifth fingernail when wrapped around the opposite wrist.

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**d)** Mitral valve regurgitation
**e)** Abdominal aortic aneurysm

**5)** Pulmonary
**a)** Spontaneous pneumothorax
**b)** Apical bleb

**6)** Skin
**a)** Striae atrophicae ("stretch marks")
**b)** Hernia

**7)** Central nervous system
**a)** Dural ectasia
**b)** Learning disability
**c)** Hyperactivity

**c.** Implications
**1)** Monitor aortic and cardiac status
**2)** β-blocker or losartan for aortic dilation
**3)** Restrict from vigorous exertion
**4)** Counsel regarding genetics
**5)** Treat skeletal deformity if symptomatic. Bracing does not usually control spinal deformity.

**2.** Homocystinuria: This disorder may be mistaken for MFS but is most readily distinguished by mental retardation. It has the following major features:

**a.** Mental retardation
**b.** Dislocated lens (inferomedial)
**c.** Arachnodactyly
**d.** Joint stiffness
**e.** Cavus feet
**f.** Scoliosis or kyphosis
**g.** Diagnosis: Urine amino acid screen
**h.** Treatment: Vitamin B6 administration, methionine restriction

**3.** Congenital contractural arachnodactyly (Beals syndrome)

**a.** This is also a MFS look-alike, so much so that the original MFS patient had this syndrome.
**b.** Genetic defect: Fibrillin-2
**c.** Clinical features
**1)** Face: Oval with recessed jaw, flattened ears
**2)** Eyes: Occasional intraocular coloboma
**3)** Heart: Congenital septal and valve defects

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4) Skeleton: Flexion contractures, which partially improve with time
5) Hands: Contracture of proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints
6) Scoliosis: Appears by mid-childhood

**4.** Achard syndrome

**a.** Clinical features
1) Arachnodactyly
2) Generalized ligamentous laxity
3) Mandibular hypoplasia

**5.** Stickler syndrome (hereditary arthro-ophthalmopathy)

**a.** Genetic etiology: A mutation in type II collagen
**b.** Clinical features
1) Progressive myopia beginning in first decade
2) Retinal detachment
3) Abnormal epiphyseal development, eventual DJD. May resemble Perthes
4) With or without mild joint hypermobility
5) With or without marfanoid habitus
6) Autosomal dominant

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Beals RK. Hereditary arthro-ophthalmopathy (the Stickler syndrome): report of a kindred with protrusio acetabuli. Clin Orthop Relat Res. 1977;125(125):32–35

**6.** MASS (mitral valve prolapse, aortic anomalies, skin and skeleton changes) phenotype

**a.** An overlap connective tissue disorder
**b.** Genetic etiology: Unknown/variable
**c.** Clinical characteristics: Mitral valve, aortic root, skin and skeleton

**7.** Shprintzen–Goldberg syndrome

**a.** Clinical syndrome of connective tissue
**b.** Genetic defect: Fibrillin 1
**c.** Clinical features: Craniosynostosis, exophthalmos, low-set ears, arachnodactyly, camptodactyly, developmental delay

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**8.** Loeys–Dietz syndrome

- **a.** Connective tissue disorder characterized by triad of hypertelorism, arterial tortuosity and aneurysms, and bifid uvula
- **b.** Genetic defect: TGF-β receptor protein 1 or 2
- **c.** Other clinical features: Clubfeet or skewfeet, knee and elbow hyperextensibility, bifid anterior or posterior arch of C1, cervical instability, scoliosis, dural ectasia
- **d.** Treatment: Screen for aneurysms; rule out cervical instability; consider angiotensin-converting enzyme (ACE) inhibitor. Conservative treatment preferred for feet. Spine treatment as for MFS

## ◆ Arthrogryposis/Contracture

**1.** Arthrogryposis multiplex congenita

- **a.** This is the classic contractural syndrome seen by orthopaedists
- **b.** Etiology is unknown; likely multifactorial
- **c.** Major features are skeletal and may affect all four limbs, just uppers, or just lowers. Involvement is usually greatest distally within each limb.
  - **1)** Hips: Frequently dislocated. Contractures are usually into abduction and external rotation
  - **2)** Knees: Flexion more common than extension
  - **3)** Clubfoot, vertical talus: Common, often resistant to cast treatment
  - **4)** Upper extremities: Often extended at elbows: adducted, stiff fingers
  - **5)** Spine: Paralytic scoliosis, torticollis
- **d.** Treatment
  - **1)** Joint range of motion can be shifted but not increased. Stretching, splinting are indicated
  - **2)** Guided growth procedures early; osteotomies most useful near end of growth
  - **3)** Fractures common after manipulation or cast

**2.** Larsen syndrome

- **a.** Genetic defect: Filamin gene (*FLNB*)
- **b.** Orthopedic features
  - **1)** Dislocated hips, hyperextended or dislocated knees
  - **2)** Clubfeet, vertical tali, or other foot deformities
  - **3)** Normal muscle mass
  - **4)** Elbow dislocations
  - **5)** Cervical kyphosis and instability
  - **6)** Thoracic and lumbar scoliosis

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**c.** Nonorthopedic features
  **1)** Flattened face; depressed, widened nasal bridge
  **2)** Cleft palate

**d.** Treatment: Surgically correct and stabilize cervical kyphosis if present. Correct hips, knees, and feet using standard principles to maximize function

**3.** Freeman–Sheldon (whistling face) syndrome

**a.** Genetics: Recessive or dominant; mechanisms unknown

**b.** Clinical findings
  **1)** Small mouth and chin (may cause difficulty with intubation)
  **2)** Fingers arthrogrypotic, flexed and ulnar deviated
  **3)** Clubfeet or vertical talus
  **4)** Scoliosis/kyphosis

**4.** Mobius syndrome

**a.** Congenital facial diplegia

**b.** Variable absence of shoulder girdle muscles

**c.** Clubfeet and hand contractures or anomalies

**5.** Pterygium syndromes

**a.** Multiple pterygium (Escobar) syndrome
  **1)** Genetic defect: Acetylcholine receptor
  **2)** Static disorder with flexion contractures and webs at all flexion creases
  **3)** Short stature
  **4)** Congenital spinal anomalies
  **5)** Treatment: Judicious guided growth and osteotomies

**b.** Popliteal pterygium syndrome
  **1)** Webs only across perineum and knees
  **2)** Facial malformations
  **3)** Contractures are due to a single cord from ischium to calcaneus, with nerve bow-strung across the joint

## ◆ Vascular Abnormalities

**1.** Klippel–Trenaunay–Weber

**a.** Clinical features
  **1)** Cutaneous hemangioma (port-wine stain)
  **2)** Varicose veins
  **3)** Limb hypertrophy: Width or length

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- **b.** Genetic basis: None known
- **c.** Treatment
  - **1)** Initially treated with compressive therapy
  - **2)** Focal limb deformity treated as needed

**2.** Maffucci syndrome

- **a.** Defect in parathyroid receptor protein
- **b.** Major features
  - **1)** Multiple enchondromata
  - **2)** Limb shortening and deformity
  - **3)** Cavernous hemangiomas
  - **4)** Risk of sarcomatous transformation

**3.** Sturge–Weber syndrome

- **a.** Major features
  - **1)** Port-wine hemangioma in trigeminal distribution
  - **2)** Neurologic sequelae resulting from meningeal hemangioma

**4.** Blue–Rubber bleb nevus syndrome

- **a.** Autosomal dominant
- **b.** Clinical features
  - **1)** Bluish cavernous hemangiomata on trunk, upper arms
    - **a)** May bleed from gastrointestinal tract locations
    - **b)** May cause pain
  - **2)** Regional hyperhidrosis

**5.** Kasabach–Merritt syndrome

- **a.** Clinical features
  - **1)** Solitary or multiple cavernous hemangiomas on the trunk or extremities
  - **2)** Consumptive coagulopathy secondary to above

**6.** Ataxia-Telangiectasia (Louis–Bar)

- **a.** Clinical features
  - **1)** Telangiectasia on conjunctivae, face, neck, and arms
  - **2)** Progressive ataxia and dysarthria
  - **3)** Skeletal problems: Equinovarus or valgus feet, scoliosis, limited ambulation
  - **4)** Immunodeficiency, infection, and cancer risk
  - **5)** Increased sensitivity to ionizing radiation

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**6)** Decreased life expectancy

**7)** Genetics: Autosomal recessive. Defect in *AT1* gene; related to immunoglobulin superfamily

**7.** Hemophilia A (see also Chapter 2)

**a.** Deficiency of factor VIII. X-linked inheritance. Males are affected, and females are carriers.

**b.** Clinical features: Bleeding into muscles, joints, and central nervous system. Often first noted at circumcision.

**1)** Factor VIII level less than 1% = severe; 2 to 5%, moderate; and greater than 5% = mild.

**2)** After several bleeds, joints develop synovial hypertrophy and are prone to rebleeds (target joints).

**3)** Most common target joints: Knees, ankles, elbows, shoulders

**c.** Treatment: Prevent target joint by early aspiration, irrigation, and rest. Hemophilic arthropathy: Joint effusion present for over 6 weeks. Consider synovectomy. Severe arthritis, consider joint replacement.

**8.** Sickle cell anemia

**a.** Disorder of β chains of hemoglobin. Hemoglobin S is a point mutation replacing a glutamic acid to valine. Hemoglobin C replaces this with lysine at the same position. S mutation is present in 1:500 African Americans. Hemoglobin SS produces severe sickling.

**1)** Hemoglobin SC produces moderate sickling.

**2)** Sickle–thalassemia produces mild sickling.

**3)** Sickle cell trait is heterozygous; one normal and one S globin inherited; no clinical sickling

**b.** Clinical features

**1)** Dactylitis: Swelling of digits in infants

**2)** Sickle crisis: Infarcts of muscle, bone, other tissue

**3)** Growth, maturation diminished or delayed

**4)** Infections with *Salmonella* or *Staphylococcus* spp.

**c.** Treatment

**1)** Rehydration, analgesics

**2)** Transfusion

**3)** Core decompression for early AVN

## ◆ Overgrowth Syndromes

**1.** Generalized bodily overgrowth

**a.** Prader–Willi

**1)** Partial deletion of paternal chromosome 15

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**2)** Major findings
- **a)** Infantile hypotonia
- **b)** Obesity beginning after age one
- **c)** Mental retardation
- **d)** Cryptorchidism
- **e)** Short stature
- **f)** Eyes slant upward and lateral

**3)** Orthopedic findings
- **a)** Developmental dysplasia of the hip: 10%
- **b)** Scoliosis: 50%
- **c)** Small hands and feet

**4)** Treatment: Growth hormone improves growth and muscle mass

**b.** Bardet–Biedl

**1)** Multiple genetic mutations may be causative

**2)** Major findings
- **a)** Truncal obesity
- **b)** Mental retardation
- **c)** Hypogonadism
- **d)** Retinitis pigmentosa
- **e)** Renal abnormalities

**3)** Orthopedic abnormalities
- **a)** Postaxial polydactyly (feet more than hands)

**c.** Beckwith–Wiedemann

**1)** Autosomal dominant; partial deletion chromosome 15

**2)** Major findings
- **a)** Large stature
- **b)** Omphalocele
- **c)** Macroglossia (may partially regress)

  Hypoglycemia

  Multiple organ enlargement; risk of Wilms tumor. Ultrasound every 3 months until age 7

**3)** Orthopedic abnormalities
- **a)** Leg-length inequality
- **b)** Neurologic damage if hypoglycemia not controlled
- **c)** Polydactyly, idiopathic scoliosis, radial head dislocation variable

**2.** Asymmetric overgrowth

**a.** Idiopathic hemihypertrophy

**1)** Skeletal findings
- **a)** One side of the body larger in all dimensions

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**b)** Growth proportionate over time
**c)** Lower extremities most often affected; trunk and upper extremities may or may not be affected

**2)** Genitourinary system
**a)** Wilms tumor (nephroblastoma): Around 5%
**b)** Medullary sponge kidney
**c)** Renal malposition

**3)** Vascular system: Aortic, cerebral vascular or congenital heart abnormalities

**b.** Russell–Silver syndrome
**1)** Genetically heterogeneous
**2)** Short stature
**3)** Small, triangular face; may be asymmetrical
**4)** Genitourinary and genital malformations
**5)** Orthopedic features
**a)** Hemihypertrophy
**b)** Other miscellaneous skeletal findings (pseudoepiphysis of second metacarpal, clinodactyly)

**c.** Goldenhar syndrome (hemifacial microsomia, oculoauriculovertebral dysplasia)
**1)** Usually not inherited
**2)** Major features
**a)** Epibulbar dermoids
**b)** Preauricular skin tags; facial asymmetry

**3)** Orthopedic abnormalities
**a)** Congenital vertebral anomalies
**b)** Other aspects of VATER (vertebral defects, imperforate anus, tracheoesophageal fistula, radial and renal dysplasia) association

**4)** Implications: Monitor for scoliosis; be aware of difficult intubation

**d.** Klippel–Feil syndrome
**1)** Key feature
**a)** Cervical spine fusions; may have stenosis; instability

**2)** Genetics: Usually sporadic; rarely inherited
**3)** Possible skeletal associations
**a)** Congenital or "idiopathic" scoliosis of lower spine: 60% of patients
**b)** Sprengel deformity (30%)
**c)** Upper extremity and hand anomalies

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4) Nonskeletal associations
   a) Genitourinary and renal malformations
   b) Hearing impairment
   c) Cardiac anomalies
   d) Facial asymmetry
5) Implications
   a) Screen early for hearing impairment, genitourinary malformations (ultrasound)
   b) Look for other skeletal malformations
   c) Counsel regarding activity and anesthesia if neck is unstable

**e.** Proteus syndrome
1) A hemartomatous disorder affecting all three germ layers. Most likely a postzygotic mutation
2) Characterized by its variability and progressive nature, named for Greek god able to change shape at will
3) Macrodactyly, asymmetrical tissue overgrowth, and nevi are key features.
4) Definitive diagnosis can be established by scoring as in **Table 3.1**
5) Treatment: Restoration of alignment by epiphyseodesis, osteotomy

## ◆ Neurofibromatosis Type 1

Neurofibromatosis type 1 (NF-1) is the most prevalent skeletal disorder caused by a single gene defect. This gene, *NF1*, codes for neurofibromin. It is related to the GTPase Activating Protein pathway and has tumor-suppressive function.

### Diagnosis

At least two of the following National Institutes of Health criteria:

1. Cafe-au-lait spots (six or more spots ≥1.5 cm after puberty or ≥0.5 cm before puberty)

**Table 3.1** Proteus Syndrome

| Diagnostic Criteria | Points |
|---|---|
| Macrodactyly and/or hemihypertrophy | 5 |
| Thickening of skin | 4 |
| Lipomas and subcutaneous tumors | 4 |
| Verrocous epidermal nevus | 3 |
| Macrocephaly | 2.5 |
| Other minor abnormalities | 1 |

Definitive diagnosis is made if score is ≥13 points; questionable diagnosis, 10–13 points; diagnosis excluded < 10 points.

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2. Subcutaneous NF
3. Positive biopsy
4. Positive family history (first-degree relative affected)
5. Skeletal manifestation
    a. Long-bone pseudarthrosis
    b. Dystrophic curve of spine
    c. Elephantiasis neuromatosa
6. Optic glioma
7. Two or more Lisch nodules (hemartomas in iris)
8. Axillary or inguinal freckling

### Features of Dystrophic Spinal Curve

1. Severe apical rotation or wedging
2. Paravertebral mass
3. "Spindling" of transverse process
4. Thinning of rib
5. Foramenal enlargement
6. Vertebral scalloping

### Orthopedic Implications

1. Bracing is not effective for dystrophic curves.
2. Dystrophic curves require more aggressive treatment (fuse if 50 degrees; anterior and posterior if >70 degrees or kyphosis >50 degrees)
3. Preoperative MRI or computed tomography/myelogram on all dystrophic curves
4. Rule out sarcoma if unexplained pain or localized growth occurs

## ◆ Ehlers–Danlos Syndromes

This group of connective tissue abnormalities has at least 11 different subtypes. Beighton later suggested six descriptive types (classic, hypermobility, vascular, scoliotic, arthrochalasis, dermatosparaxis) and five unspecified types. Many other patients do not fit precisely into one of the groups. Most are disorders of collagen types 1, 5, or its processing enzymes. They are listed in **Table 3.2**.

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**Table 3.2** Types of Ehlers-Danlos Syndrome

| Names | Type | Genetics | Skeletal Manifestations | | | Other Problems |
|---|---|---|---|---|---|---|
| | | | Dislocations | Joint Laxity | Scoliosis | |
| Classic: gravis | 1 | AD | + | + | + | Aneurysms, viscus rupture, hernias |
| Classic: mitis | 2 | AD | - | +/− | - | —– |
| Benign hypermobile | 3 | AD | + | + | - | Mitral valve prolapse |
| Vascular | 4 | AD/AR | + | Fingers | - | Aneurysms, spontaneous rupture |
| Unspecified X-linked | 5 | X | - | - | - | Intramuscular hemorrhage, “floppy baby” |
| Ocular–scoliotic | 6 | AR | + | + | ++ | Ocular complications |
| Arthrochalasis multiplex | 7 | AR | + | + | + | Short stature |
| Unspecified: periodontosis | 8 | AD | - | +/− | - | Necrobiosis of skin; periodontosis |
| Unspecified: occipital horn | 9 | X | + | + | - | Occipital horns, skeletal dysplasia |
| Unspecified: platelet dysfunction | 10 | AR | - | Hands | - | Platelet defect |
| Unspecified: familial laxity | 11 | AD | Petallae, hips | | + | |

*Abbreviations:* AD, autosomal dominant; AR, autosomal recessive.

## ◆ Osteogenesis Imperfecta

Osteogenesis imperfecta (OI) comprises a group of disorders of type I collagen causing bone fragility and in some cases blue sclerae, hearing loss, and abnormal dentin. The osseous fragility tends to improve after puberty.

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## Sillence Types

- Type I
  - **a.** Variable osseous fragility (minimal through moderately severe)
  - **b.** Blue sclerae (at all ages)
  - **c.** Early hearing loss
  - **d.** Autosomal dominant
- Type II (lethal perinatal OI)
  - **a.** Extremely severe osseous fragility, with stillbirth or neonatal death
  - **b.** Subgroup A—radiographs show broad, crumpled long bones and broad ribs with continuous beading. Autosomal dominant or new mutation
  - **c.** Subgroup B—radiographs show broad crumpled long bones, ribs show discontinuous beading or are not beaded. Autosomal recessive
  - **d.** Subgroup C—radiographs show thin, fractured long bones and thin, beaded ribs. Autosomal recessive (?)
- Type III
  - **a.** Autosomal recessive
  - **b.** Fractures at birth, then progressive deformity
  - **c.** Normal sclerae and hearing
- Type IV
  - **a.** Moderate osseous fragility
  - **b.** Normal sclerae (blue in infancy)
  - **c.** Variable deformity of long bones and spine
  - **d.** Autosomal dominant
- *Note:* The value of opalescent dentin for subcategorization of OI is uncertain.

## Additional Types (After Sillence)

- Type V
  - **a.** Hyperplastic callus
  - **b.** Radial head dislocation
  - **c.** Moderate fracture rate
  - **d.** Normal type I collagen

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- Type VI
  - **a.** Moderate osseous fragility
  - **b.** "Fish-scale" pattern of lamellae on histology
  - **c.** Excessive osteoid deposition
  - **d.** Normal type I collagen
- Type VII
  - **a.** Rhizomelic short stature
  - **b.** Normal type I collagen
  - **c.** CRTAP (cartilage-associated protein) defect
  - **d.** Reported only in Canadian First Nations Community
- Type VII
  - **a.** Short stature and normal sclerae
  - **b.** Bulbous metaphyses
  - **c.** Extreme osteopenia
  - **d.** Normal type I collagen; attributable to defect in leprecan, which hydroxylates proline #986 of COL 1A1

### Bruck Syndrome

1. OI with multiple congenital contractures
2. Genetic defect in bone-specific telopeptide lysl hydroxylase
3. Contractures of knees, feet and ankles, elbows

## ◆ Mucopolysaccharidoses

Mucopolysaccharidoses are recessive disorders of glycosaminoglycan (mucopolysaccharide) storage, all autosomal recessive. They have delayed appearance of signs and symptoms corresponding to the accumulation of storage products. Most are progressive. **Table 3.3** describes their features.

## ◆ Malformations of the Hand and Foot

### Syndactyly

1. Terminology
   - **a.** Extent: Partial or complete
   - **b.** Simple: Skin only
   - **c.** Complex: Synostosis

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**Table 3.3** The Mucopolysaccharidoses

| Number | Name | Genetics | Enzyme Defect | Clinical Features |
|---|---|---|---|---|
| I-H | Hurler | AR | µ-L-iduronidase | Diagnosis at 1–3 yr; corneal clouding, MR, kyphoscoliosis. Some amelioration with enzyme replacement, BMT, gene therapy |
| I-S | Scheie | AR | µ-L-iduronidase | Corneal clouding, aortic abnormality, normal intelligence, longer survival |
| II | Hunter | XR | Iduronate sulfate sulfatase | Clear cornea, mild MR, ± kyphosis |
| III | SanFillipo A,B,C,D | AR | | Dementia, seizures |
| IV-A | Morquio-A | AR | B galactosidase - 6-sulfate sulfatase (increased urinary keratan sulfate) | Short trunk, odontoid hypoplasia with cervical instability, flame-shaped vertebrae, kyphosis (TL) |
| IV-B | Morquio-B | AR | B galactosidase | Milder form |
| VI | Maroteaux–Lamy | AR | Arylsulfatase B | Corneal clouding, normal intelligence, ± cervical stenosis, ± TL kyphosis |
| VII | Sly | AR | B-glucuronidase | May have epiphyseal dysplasia |

*Abbreviations:* AR, autosomal recessive; BMT, bone marrow transplant; MR, mental retardation; TL, thoracolumbar; XR, X-linked recessive.

**d.** Polysyndactyly: Hidden duplicated skeletal structures
**e.** May be associated with Apert syndrome, Saethre–Chotzen, Poland, or other syndromes

**2.** Isolated syndactyly (five types)

**a.** Long-ring syndactyly is most common.
**b.** Look for duplicated phalanges, abnormalities of metacarpals and tarsals.
**c.** Autosomal dominant
**d.** Minimal risk of associated anomalies

**3.** Poland syndrome

**a.** Simple syndactyly of variable number of fingers
**b.** Short fingers (absent or hypoplastic middle phalanges)
**c.** Absent sternocostal head of pectoralis major

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4. Acrocephalosyndactylies
   a. Apert syndrome
      1) Complete complex syndactyly D2–4 with common nail, progressive interphalangeal synostosis of hands and feet. Medial deviation of great toe and tarsal synostosis
      2) Craniosynostosis
      3) Occasional cervical fusions, usually without deformity
   b. Crouzon syndrome
      1) Craniosynostosis
      2) Calcaneocuboid coalition, C-spine fusion
   c. Many others: Saethre-Chotzen, Carpenter, etc.

5. Congenital constriction bands (Streeter's bands)
   a. Distal (acral) syndactyly
   b. Thumb rarely involved
   c. Cutaneous rings or amputations
   d. May have distal paresis or deformity (clubfoot)
   e. No known mendelian basis but genetic contribution possible

## Polydactyly

1. Ulnar (postaxial): Frequently isolated, especially in African Americans
2. Radial (preaxial): More frequently associated with syndromes, especially radial ray defects
3. Radial clubhand: This is a spectrum that includes hypoplasia to complete absence of preaxial parts. It may be isolated or associated with the following:
   a. Blood dyscrasias
      1) Fanconi: Anemia to progressive pancytopenia, not present at birth; about a third with renal anomalies; often fatal
      2) TAR (thrombocytopenia, absent radii) syndrome; neonatal thrombocytopenia, usually improves with time; frequent knee anomalies
   b. Congenital heart defects
      1) Holt–Oram syndrome: Variable cardiac and preaxial deficiency; most commonly atrial septal defect and hypoplastic thumb
   c. Craniofacial anomalies (Nager syndrome)
   d. Congenital scoliosis
      1) VATER, Goldenhar syndrome (oculo-auriculo-vertebral dysplasia)
      2) Klippel–Feil syndrome

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4. Implications

   a. Examine previous chest and abdominal films for vertebral anomalies, or take new ones
   b. Evaluate face, jaw, palate
   c. Do complete blood and platelet counts
   d. Ask about feeding (esophageal abnormalities)
   e. Listen to heart, possibly echo
   f. Evaluate genitourinary system: Urinalysis, possibly echo
   g. Chromosome analysis if multiple anomalies found outside of the particular syndrome

### Ulnar Clubhand

1. Usually a mild dysgenesis; few frequent associations
2. May be seen with Cornelia de Lange syndrome

### Amputated Limbs

1. Single: Usually an isolated anomaly but may be associated with idiopathic scoliosis
2. Congenital ring constriction syndrome

   a. Nongenetic, variable, rings with grooves in skin, occasionally with lymphatic or vascular impairment
   b. Transverse amputation with proximal limb normal
   c. Syndactyly (distal with proximal fenestrations)
   d. Clubfeet
   e. Craniofacial defects

## ◆ Syndromes with Predominant Spinal Deformity

### VA(C)TER(LS)

1. A syndrome or association of unknown etiology, characterized by

   - Vertebral anomalies
   - Anorectal atresia
   - Cardiac anomalies
   - TE (tracheosesophageal) fistula
   - Renal and radial anomalies (renal atresia, duplication; radial clubhand or preaxial upper-limb hypoplasia)
   - Lower-limb abnormalities (duplicated hallux or other anomalies)
   - Single umbilical artery

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2. Clinical implications: In patient seen for vertebral anomaly, search for other abnormalities. Obtain renal ultrasound or MRI.

### 22Q Deletion Syndrome

1. Common chromosomal deletion syndrome encompassing DiGeorge syndrome, velocardiofacial syndrome, conotruncofacial syndrome, and others
2. Key anomalies include skeletal, palatal, cardiac, and immunologic.
3. Spinal features: Platybasia, occipitalization of the atlas, atlas arch defects, block vertebrae, stenosis, up-turned (swoosh) C2 lamina, thoracolumbar anomalies
4. Other skeletal anomalies: Equinovarus feet, polydactyly, rib anomalies

### Wildervanck Syndrome

Deafness, Klippel-Feil anomaly, abducens palsy, Chiari malformation

### Goldenhar Syndrome

See preceding sections.

## ◆ Syndromes Caused by Teratogens

### Fetal Alcohol Syndrome

1. Growth disturbance (of both length and weight) through childhood
2. Central nervous system dysfunction and decreased head size; learning deficit/attention deficit disorder/mental retardation
3. Dysmorphic face (mild): Small eyes, flat philtrum, thin upper lip
4. Orthopedic features
   a. Contractures (elbows, metaphalangeal and interphalangeal joints)
   b. Miscellaneous synostoses
   c. Hip dislocations, clubfeet
   d. Congenital cervical fusion (C2-C3 = most)

### Fetal Hydantoin Syndrome (from Maternal Use of Phenytoin)

1. Growth retardation: Mild
2. Mental retardation: Mild
3. Face: Hypertelorism, cleft lip
4. Hands: Hypoplasia, absence of phalanges, mostly distal

### Warfarin

Fetal bleeding and teratogenesis

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## ◆ Chromosome Abnormalities

### Down Syndrome

1. Trisomy, mosaicism, or translocation of chromosome 21
2. Major findings
    - **a.** Mental retardation, variable
    - **b.** Congenital heart defects: arteriovenous communis, ventriculoseptal defect
    - **c.** Gastrointestinal anomalies
    - **d.** Short stature
    - **e.** Leukemia (1%), seizures, diabetes, hypothyroidism: Less frequent
    - **f.** Orthopedic
        - **1)** Delayed walking (1½ to 5 years of age)
        - **2)** Ligamentous laxity
        - **3)** C1-C2 or occiput: C1 laxity: Radiographs at about age 4 and yearly if atlanto-dens interval is greater than 5 mm. Fuse if signs of myelopathy exist.
        - **4)** Scoliosis, idiopathic-like
        - **5)** Hip dislocations: Acute, subacute, or habitual
        - **6)** Slipped capital femoral epiphysis
        - **7)** Perthes disease
        - **8)** Patellar subluxation or dislocation
        - **9)** Metatarsus adductus or hallux valgus

### Turner Syndrome

1. Monosomy X
2. Major findings
    - **a.** Low birth weight and persistent growth retardation
    - **b.** Normal intelligence
    - **c.** Low hairline and webbed neck
    - **d.** Renal and cardiac anomalies, coarctation
    - **e.** Absent or hypoplastic gonads
    - **f.** Orthopedic
        - **1)** Genu and cubitus valgus
        - **2)** Scoliosis, idiopathic

### Noonan Syndrome

1. Turner-like phenotype but normal chromosomes. Defect in *PTPN11* in some cases

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2. Major findings

   a. Mental retardation
   b. Hypertelorism, ptosis, downward-slanting eyes
   c. Increased severity of scoliosis, end-plate changes
   d. Pulmonary stenosis common
   e. Thrombocytopenia in some cases

### Klinefelter Syndrome

1. Genetics: 1. 47 XXY
2. Clinical features

   a. Asthenic habitus, long legs
   b. Failure of secondary sexual development
   c. Scoliosis
   d. Proximal radioulnar synostosis

### "Cri-du-Chat" Syndrome

1. Genetics: 5P
2. Clinical findings

   a. Profound mental retardation
   b. Multiple hand and foot anomalies
   c. Scoliosis, congenital

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# 4 Neuromuscular Disorders in Pediatric Orthopedics

This chapter summarizes neuromuscular diseases that affect the skeleton as a result of pathologic conditions of the spinal cord or peripheral nervous system. These diseases may present a fully developed picture or show early subtle findings, such as a mild deviation of gait. The tables in this chapter organize the neuromuscular diseases according to the location of pathology reference.

## ◆ Evaluation

The elements of evaluation of neuromuscular disease include the following:

1. History: Prenatal, birth (gestation, weight, apgar scores), developmental (milestones), family
2. Physical examination
    - **a.** Motor strength, tone
    - **b.** Deep tendon reflexes
    - **c.** Cranial nerves
    - **d.** Cerebellar signs
3. Serum creatine phosphokinase (CPK)
    - **a.** Elevation directly related to amount of muscle necrosis or membrane disorder. Abnormal in Duchenne and Becker muscular dystrophies (>20 times normal in childhood and early teens), myopathies, or myositis
    - **b.** Minimal to mild elevation in other dystrophies
4. Immunocytochemistry: Directly differentiates Duchenne from Becker dystrophies and other conditions. Requires small amount of muscle tissue
5. DNA mutation analysis: Requires small amount of blood or amniotic fluid, allows prenatal diagnosis
6. Electromyelography (EMG): Distinguishes myophatic from neuropathic weakness
    - **a.** Myopathic process: Polyphasic low-voltage signal; fibrillations and sharpwaves
    - **b.** Neuropathic process: Initially, brief biphasic low-voltage fibrillation
    - **c.** Chronic process: Prolonged polyphasic fibrillation, increased amplitude

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**7.** Nerve conduction studies

- **a.** Abnormally slowed conduction velocities in conditions involving peripheral nerves only
- **b.** Normal conduction velocities in spinal muscular atrophy
- **c.** Normal conduction velocity (>49 m/s in arms, >39 m/s in legs) for patients over 5 years of age. Younger children have slower conduction velocity.

**8.** Muscle biopsy: Biopsy minimally involved muscle in chronic conditions and severely involved muscle in acute conditions. Vastus lateralis used for proximal myopathy, should be done only in centers capable of doing special histochemistry

- **a.** Type I fibers: Oxidative metabolism, slow twitch
- **b.** Type II fibers: Anaerobic metabolism, fast twitch
- **c.** Myopathic process
  Necrosis, phagocytosis, and inflammation
  Irregularly sized fibers
  Type I predominance less than 50%
- **d.** Neuropathic process
  Small group atrophy
  Fiber-type grouping, angular fibers
  Type II predominance greater than 80%
- **e.** Electron microscopy (glutaraldehyde) is used to differentiate architectual changes within the congenital myopathies

**9.** Nerve biopsy

- **a.** Sural nerve most commonly biopsied
- **b.** Guillain Barré syndrome: Mononuclear infiltrates and focal acute demyelination
- **c.** Hypertrophic neuropathies: Nerve fiber loss interstitial fibrosis and "onion bulb" formation

**10.** Electrocardiogram

- **a.** Abnormal in Duchenne muscular dystrophy (sinus tachycardia and right ventricular hypertrophy), Friedreich ataxia, and myotonic dystrophy

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## ◆ Cerebral Palsy

### Definition

Static brain injury or lesion in prenatal, perinatal, or postnatal period (up to 2 years)

### Causes

Prematurity, infection, hypoxia, ischemia, embolus, trauma, brain malformation

### Physiologic Types

Pyramidal (spastic), extrapyramidal (dystonic or athetoid), mixed

### Anatomic Types

1. Diplegic: Usually premature. Pyramidal tracts involved; magnetic resonance imaging (MRI) shows periventricular leukomalacia; cognition preserved; legs affected, most distally; equinovalgus feet most common; hips rarely subluxate; scoliosis is rare.
2. Hemiplegic: Focal cortical lesion; risk of seizure; cognition preserved; ipsilateral arm and leg involved. Equinovarus foot is most common; hip and spine involvement is rare. Limb shortening is usually minor.
3. Totally involved (quadriplegic): Usually diffuse cortical problem. Cognition may be affected; problems with spine and trunk balance; respiration and swallowing function affected. Risk of seizure. Hip subluxation, scoliosis, and limb deformities are common.
4. Other types: Monoplegia, asymmetric diplegia

### Function

*Gross Motor Functional Classification System (***Fig. 4.1***)*

1. I: Walks without restrictions, but speed and coordination are impaired.
2. II: Walks in and outdoors and climbs stairs using railing; difficulty with uneven surfaces
3. III: Uses crutches to walk. Propels own manual wheelchair
4. IV: Walks short distance with walker; uses wheelchair more
5. V: Trunk and all extremities are involved. No independent mobility

### Hip Subluxation Rating

Reimer migration index (MI) is the percentage of femoral head lateral to Perkin line (A/B below); quantitates MI (**Fig. 4.2**).

### Treatment

1. Physical therapy
2. Mobility aids (orthoses, crutches, walker, wheelchair)

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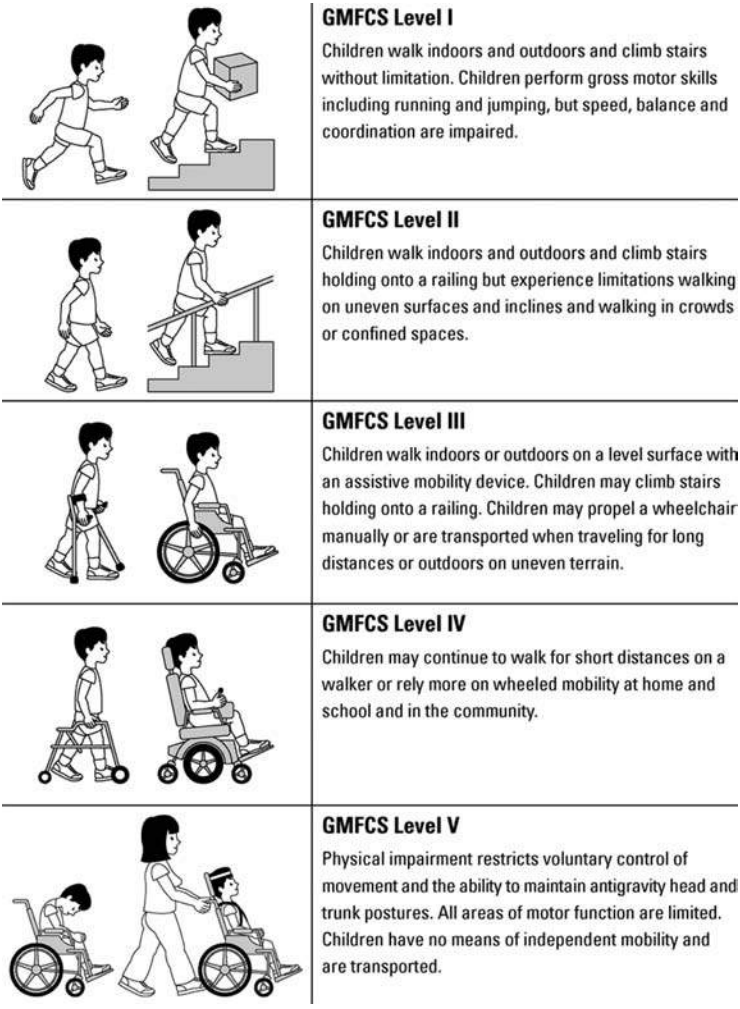



**Fig. 4.1** Gross motor functional classification system. (From Graham HK. Classifying cerebral palsy. J Pediatr Orthop. 2005;25(1):128 (Fig. 1). Reprinted with permission.)

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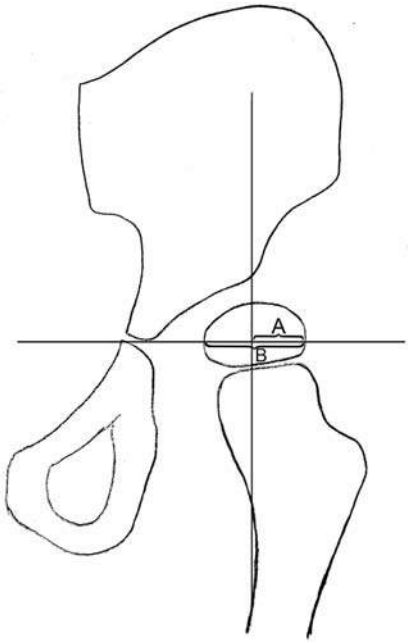



**Fig. 4.2** The percentage of femoral head lateral to Perkin line (A/B) quantitates the Reimer Migration Index.

3. Oral medications for spasticity, dystonia
4. Botulinum toxin for temporary tone management
5. Selective dorsal rhizotomy for lower extremity spasticity
6. Intrathecal pump for generalized spasticity
7. Orthopedic muscle lengthening and transfer
8. Osteotomies for bony deformity
9. Arthrodesis of spine or feet if collapse impairs function

## ◆ Disorders of Spinal Cord Peripheral Nerves and Muscles

### Anterior Horn Cell Diseases

(**Table 4.1**)

### Neuropathies

(**Table 4.2**)

### Muscular Dystrophies

(**Table 4.3**)

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**Table 4.1** Anterior Horn Cell Disorders

| Disease | Age at Diagnosis | Inheritance Pattern | Life Expectancy | Signs | Orthopedic Manifestations | Laboratories |
|---|---|---|---|---|---|---|
| Polio | Variable | None (infectious) | Related to level of involvement | Asymmetric flaccid paralysis, asymmetric or absent DTR | Contractures, scoliosis | Depends on phase of illness |
| Spinal muscular atrophy | | | | | | |
| Type I or Werdnig–Hoffman | Birth to 6 mo | Autosomal recessive gene defect on 5 q<br>SMN gene | Most die in infancy | Marked general weakness, no head control, absent DTR normal sensation | Fractures | |
| Type II | 6–12 mo | Autosomal recessive gene defect on 5 q | Early to mid-adulthood | Normal until ~6 mo, independent head control in sitting position, never ambulate | Hip subluxation, scoliosis, contructure | EMG: high-amplitude polyphasic<br>NCS: widespread fascicular pattern of denervation |
| Type III Kugelberg–Welander | 1–2 yr | Autosomal recessive gene defect on 5 q | Over 45 yr | Normal until ~1 yr, ambulate until 2nd decade, then wheelchair, never can run or climb stairs | Kyphosis, scoliosis | DNA test for SMN, same as type II |

*Abbreviations:* DTR, deep tendon reflex; EMG, electromyelography; NCS, nerve conduction study.





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**Table 4.2** Neuropathies

| Disease | Age of Diagnosis | Inheritance Pattern | Life Expectancy | Signs | Orthopedic Manifestations | Laboratories |
|---|---|---|---|---|---|---|
| Guillain-Barré syndrome | Variable | None; postviral infection | ~5% mortality | Ascending pain, paresthesia, and weakness | Contractures | ↑ CSF protein |
| Friedreich ataxia (spinocerebellar degeneration) | Before 10 yr | Autosomal dominant chromosome 9 defect | <40 yr | Wide-based gait weakness, tremor, nystagmus, uses wheelchair by age 30; areflexia cardiomyopathy | Pas cavus, scoliosis, ataxia, speech disorder | ↓↓ Sensory and ↓ motor conduction velocity |
| Hereditary motor and sensory neuropathies | | | | | | |
| Type I: Demyelinating form of Charcot-Marie-Tooth | Second decade of life | Autosomal dominant | Normal | Foot deformity, intrinsic wasting, absent reflexes, length-dependent weakness, sensory ↓ | Equinocavovarus clawing, gait abnormality, hip dysplasia, scoliosis (10%) | ↓ Motor conduction velocity |
| Type II or Charcot-Marie-Tooth disease (axonal form) | Wide variation | Autosomal dominant | Normal | "Stork legs," foot deformity, distal weakness | Cavus foot deformities, occasional UE intrinsic weakness, gait abnormality | Normal or sl ↓ motor conduction velocity |
| Type III or Dejerine-Sottas disease | Infancy to early childhood | Autosomal recessive | Wide variation | Same as CMT but more severe | Cavus foot, scoliosis | ↓↓ Motor conduction velocity, ↑ CSF protein |

*Abbreviations:* CSF, cerebrospinal fluid.

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**Table 4.3** Muscular Dystrophies

| Disease | Age at Diagnosis | Inheritance pattern | Abnormal gene | Life Expectancy | Signs | Orthopedic Manifestations | Laboratories |
|---|---|---|---|---|---|---|---|
| Duchenne muscular dystrophy (DMD) | 2–6 yr | X-linked recessive (⅓ have new mutation) | Xp21 | ~20–30 yr | Late walker, calf pseudohypertrophy, Meryon and Gower signs, mild mental retardation | Contractures: equinovarus, knee flexion, hip flexion–abduction, wheelchair dependence, scoliosis | ↑↑ CK, ↓↓ dystrophin |
| Becker muscular dystrophy | Childhood | X-linked recessive | Xp21 | Adulthood | Weakness, pseudohypertrophy | Contractures, wheelchair dependence late | ↑ CK, ↓ dystrophin |
| Limb-Girdle dystrophy | 2nd to 3rd decade of life | Autosomal recessive and dominant forms | Many | Wide range | Weakness of pelvic or shoulder-girdle muscles | Similar to DMD, milder | ↑ CK, normal dystrophin |
| Fascioscapulohumeral dystrophy | 2nd decade of life | Autosomal dominant | 4q | Normal | Facial and shoulder-girdle weakness, expressionless face | Scapular winging contractures | Mild ↑ CPK, normal dystrophin |
| Emery-Dreifuss dystrophy | 2–4 yr | X-linked recessive | Xq28 | Mid-adult | Muscle weakness, AV block | Toe-walking, elbow flexion contractures neck extension | CK min ↑ normal dystrophin |
| Congenital myopathies | | | | | | | |
| Nemaline-body myopathy | Variable | Sarcomere proteins | | Variable | | Scoliosis proximal weakness | |
| Central core myopathy | Infancy | | | | Nonprogressive | Kyphoscoliosis equinovarus feet | |
| Centronuclear myopathy | | X-linked recessive | Xq28 | | | DDH | |

*Abbreviations:* AV, arteriovenous; CK, creatine kinase; CPK, creatine phospokinase; DDH, developmental dysplasia of the hip.



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**Table 4.4** Myotonic Disorders

| Disease | Age of Diagnosis | Inheritance pattern | Abnormal gene | Life expectancy | Signs |
|---|---|---|---|---|---|
| Myotonia congenita | 0–10 yr | Autosomal dominant | | Normal | Myotonia with initial movement. General muscular hypertrophy. Dilantin and procainamide can decrease myotonia |
| Paramyotonia congenita | Childhood | Autosomal dominant | | Normal | Cold-induced episodes of paradoxical myotonia and flaccid paresis associated with $K^+$ abnormalities treated with quinine or ion exchange resins |
| Myotonic dystrophy | 2nd to 3rd decade of life | Autosomal dominant | CTG expansion | Decreased | Progressive weakness begins distally. Gonadal atrophy, cataracts, heart disease, and mental defects associated. Develop C spine subluxation and wheelchair dependency |
| Congenital myotonic dystrophy | Infancy | Autosomal dominant | Large CTG expansion | Normal | Hypotonia, difficulty swallowing, and expressionless faces. Develop clubfeet, contractures and dislocated hips, mental defects, and lenticular opacities associated.<br>EMG: Pathognomonic "dive bomber" pattern disease becomes more severe with second-generation equinovarus feet |

*Abbreviations:* EMG, electromyelography; CTG, cytosine, thymine, guanine

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### Myotonic Disorders

(**Table 4.4**)

### Spina Bifida

1. Classification: Myelomeningocele or lipomeningocele
2. Associated abnormalities: Congenital spine deformity, Sprengel deformity, Chiari malformation, tethered cord, neurogenic bladder. High risk of latex allergy
3. Motor classification and implications
    - **a.** Thoracic/high lumbar: Minimal short-term ambulation; highest risk of associated deformities
    - **b.** Midlumbar: May ambulate but stop at maturity; highest risk of hip dislocation
    - **c.** Low lumbar (quadriceps and hamstrings function): Moderate walking with limp
    - **d.** Sacral: Near-normal walking
4. Treatment
    - **a.** Physical therapy to optimize strength and motion; match equipment to functional level
    - **b.** Monitor for tethered cord (increasing weakness, back pain, unexpected scoliosis)
    - **c.** Brace feet, ankles, and knees if able to improve function
    - **d.** Treat hip dislocations only if L5 level or below
    - **e.** Release contractures only if functionally significant
    - **f.** Treat clubfeet aggressively with casts, braces, and surgery as needed
    - **g.** Monitor for potential skin pressure areas and prevent with bracing or surgery
    - **h.** Avoid latex
    - **i.** Optimize vitamin D intake
    - **j.** Recognize fractures presenting as warm, swollen limb in the absence of trauma

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# 5 Pediatric Trauma

## ◆ Pediatric Trauma

This chapter provides basic principles and algorithms for trauma management in children. Common diaphyseal fractures are not covered if there are no unique pediatric features. References are provided at the end of each section for further information.

## ◆ Basic Principles

### Physeal Fractures

**1.** Classification

- **a.** There are several classification systems, including those of Aitken, Salter and Harris, and Peterson. Their goals are to (1) facilitate communication, (2) predict the risk of growth disturbance, and (3) determine treatment.
- **b.** The classifications provide information on
  - **1)** Physeal alignment
  - **2)** Articular alignment
  - **3)** Stability and risk of displacement
- **c.** Physeal damage can occur from
  - **1)** Step off at the level of the physis, with bar formation
  - **2)** Damage to and death of physeal cells
  - **3)** Ischemia of the physis if severe soft tissue damage occurs
- **d.** The classifications are usually predictive of the risk of growth disturbance. Salter 1 and 2 are typically at low risk of growth disturbance because the growth plate is not traversed by the fracture. However, there are some notable exceptions because damage may occur to the physis which is not visible on plain films. This is most common in the distal femur and the distal tibia, where there is a high risk of growth disturbance even in the "benign" fracture types such as Salter I and II. This may be due to complex physeal anatomy as well as compressive forces involved. By contrast, there is a low risk of growth plate damage in Salter 1 and 2 fractures of the distal radius and ulna and the proximal humerus.
- **e.** Salter–Harris classification: The most widely used (**Fig. 5.1**). Note that type V fractures are rarely, if ever, seen.
- **f.** Peterson classification: Recognizes a broader spectrum of injuries (**Fig. 5.2**)

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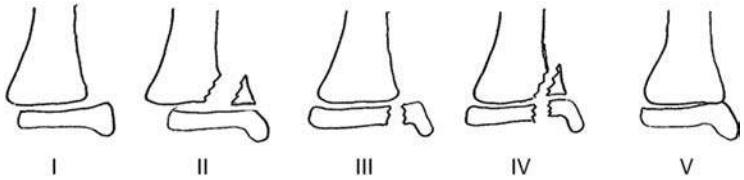



**Fig. 5.1** Salter–Harris classification of physeal injuries.

## Management of the Polytrauma Patient

1. Definition: A patient with more than one organ system injured or more than one component within one organ system.
   - **a.** Laboratory studies: Complete blood cell count, type and cross, urinalysis, blood urea nitrogen, creatinine, amylase, electrolytes
   - **b.** Indications for radiographic studies
     - **1)** Cervical, thoracic, lumbar spine
       - **a)** Tender
       - **b)** Unconscious or heavily sedated
       - **c)** Neurologically abnormal
     - **2)** Pelvis
       - **a)** Tender
       - **b)** Unconscious
       - **c)** Hematuria present
     - **3)** Skull
       - **a)** Head trauma and loss of consciousness for longer than 5 minutes, hematoma
       - **b)** Skull depression
       - **c)** Focal neurologic signs
       - **d)** Cerebrospinal fluid from nose or middle ear
       - **e)** Blood in middle ear
     - **4)** Computed tomography (CT) of the head
       - **a)** Glasgow Coma Scale less than 8
       - **b)** Focal neurologic signs
       - **c)** CT of the abdomen
       - **d)** Shock
       - **e)** Severe head injury
       - **f)** Abnormal abdominal examination

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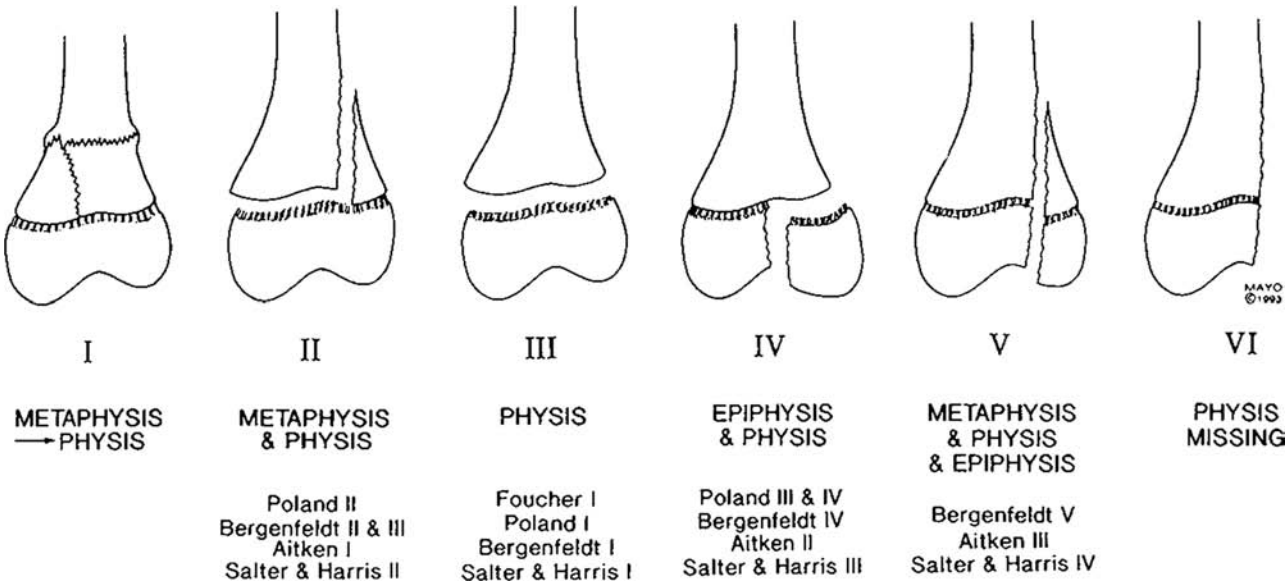



**Fig. 5.2** Peterson classification of physeal injuries. (From Peterson HA. Physeal fractures: Part 3. Classification. J Pediatr Orthop. 1994;14(4):443 (Fig. 8). Reprinted with permission.)

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**2.** Initial evaluation

- **a.** Physical examination
  - **1)** Primary survey: To detect most urgent priorities (ABCDE)
    - A Airway
    - B Breathing (ventilation)
    - C Circulation (hemorrhage)
    - D Disability (neurologic status)
    - E Exposure (temperature)
  - **2)** Secondary survey
    - **a)** Complete physical examination
    - **b)** History of event
    - **c)** Medical history
    - **d)** Laboratory and radiographic results
    - **e)** Reevaluation and stabilization
  - **3)** Normal vital signs for children (**Table 5.1**)
  - **4)** Glasgow Coma Scale (**Table 5.2**)

**3.** Adjuncts in management

- **a.** Intracranial pressure measurement indications
  - **1)** Glasgow Coma Scale less than 5 or less than 8 if shock is present
  - **2)** CT scan showing mass or shift
  - **3)** Progressive neurologic deterioration
- **b.** Parenteral nutrition
  - **1)** Indicated in polytrauma patient if enteral feeding not expected within 24 hours
- **c.** Repeat physical examination

**Table 5.1** Normal Vital Signs by Age

| Age | Pulse (beats/min) | Respirations (per min) | Blood Pressure (mm Hg, S/D) |
|---|---|---|---|
| 1–6 mo | 130 ± 45 | 30–40 | 80/46 |
| 6–12 mo | 114 ± 40 | 24–30 | 95/65 |
| 1–2 yr | 110 ± 40 | 20–30 | 99/65 |
| 2–6 yr | 105 ± 35 | 20–25 | 100/60 |
| 6–12 yr | 95 ± 30 | 16–20 | 110/60 |
| 12 yr | 80 ± 25 | 12–16 | 120/60 |

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**Table 5.2** Glasgow Coma Scale

| Response | Score |
|---|---|
| Eye opening | |
| None | 1 |
| To pain | 2 |
| To voice | 3 |
| Spontaneous | 4 |
| Verbal | |
| none | 1 |
| incomprehensible | 2 |
| inappropriate | 3 |
| disoriented | 4 |
| oriented | 5 |
| Motor | |
| none | 1 |
| decerebrate | 2 |
| decorticate | 3 |
| withdraws from pain | 4 |
| localizes to pain | 5 |
| obeys commands | 6 |
| **Total** | **Up to 15 points** |

**1)** Should be performed at 24 and 48 hours because of the incidence of missed injuries
**2)** Bone scan is an alternative

**d.** Indications for deep venous thrombosis prophylaxis in polytrauma
**1)** Oral contraceptive use
**2)** Vascular injury
**3)** Sickle cell anemia
**4)** Prolonged immobility in older adolescent

## ◆ Pediatric Shoulder Injuries

### Principles

**1.** The proximal humeral physis is one of the most active in the skeleton, contributing 80% of the length of humerus; therefore it has tremendous remodeling potential.

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2. Ossification

   a. Begins at 6 months in proximal humeral epiphysis, and growth ceases at 15 years in girls and 18 years in boys
   b. Ossific center for greater tuberosity appears at age 1; medial clavicle physis closes at around 23 years

### Birth Fracture

1. Risk factors

   a. Difficult delivery
   b. Large size
   c. Breech presentation

2. Presentation

   a. "Pseudoparalysis" –limb moves little
   b. Rule out sepsis, brachial plexus injury

3. Diagnosis

   a. Plain radiographs or ultrasound

4. Treatment: Ace wrap or arm to chest for 2 weeks

### Proximal Humeral Fractures

1. Background

   a. Age-based patterns: Preadolescent usually has fracture of metaphyseal region; adolescent, physeal fracture; Salter II or I
   b. Mechanism: Axial load or abduction; external rotation
   c. Muscle insertions with respect to physis: Internal and external rotators all on proximal fragment; deltoid and pectoralis, distal fragment displaces anteriorly and medially

2. Criteria for acceptable alignment

   a. Child under age 12 years: Virtually any alignment is acceptable.
   b. Child over age 12 year: Shortening or overlap less than 3 cm
   c. Angulation less than 45 degrees

3. Classification of displacement (pediatric)

   a. Neer and Horowitz
      - I: Less than 5 mm translation

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- II: 5 mm to 33%
- III: 33 to 66%
- IV: Greater than 66% (most are grade IV)
- Translation itself is not a problem. *Note:* Appearance on emergency department film is not the same as the appearance later. Angulation usually improves.

**4.** Treatment methods

**a.** Sling
**b.** Traction
**c.** Shoulder spica
**d.** Abduction brace
**e.** Internal fixation

**5.** General recommendations

**a.** Sling and swathe as long as any growth remains
**b.** Closed or open reduction and internal reduction and internal fixation if
**1)** Unacceptable angulation or shortening; older than 12 years
**2)** Severe head injury with spasticity
**3)** Polytrauma: To facilitate management
**4)** Vascular injury
**5)** Tenting skin: Risk of breakdown

**6.** Fracture through unicameral bone cyst (UBC)

**a.** Common cause of proximal humerus fracture in child
**b.** Differential diagnosis
**1)** Eosinophilic granuloma
**2)** ABC (aneurysmal bone cyst)
**3)** Fibrous dysplasia
**4)** Fibrous cortical defect
**c.** Treatment of UBC
**1)** Sling to heal fracture: 4 to 6 weeks
**2)** Cyst regresses about 20% of time
**3)** Assess and discuss the risk of refracture with family
**a)** Depends mainly on cortical thickness
**4)** Inject or bone graft if:
**a)** Persistently thin cortex
**b)** High desired activity level
**c)** Family prefers active treatment

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5) May need several injections

### Sternoclavicular Injuries

1. Medial clavicle is last epiphysis to appear and to close (≤ 23 years old).
2. It provides 80% of clavicle growth.
3. Injury may be a dislocation or a fracture.
4. CT is most accurate if diagnosis is unclear.
5. Signs of *significant* displacement:
   a. Venous congestion
   b. Decreased pulse
   c. Difficulty breathing, swallowing
   d. Sensation of choking
6. Treatment
   a. Anterior displacement: usually no treatment
   b. Posterior displacement: treat if significant symptoms
   c. Closed reduction; may use towel clips
   d. Internal fixation: use sutures if unstable

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## ◆ Pediatric Elbow Injuries

### Lateral Condyle Fracture

1. Background
   a. Vascular supply to the capitellum and lateral trochlea enters posteriorly
   b. Fracture may occur with varus or valgus force
   c. Distinguish this injury from a transphyseal separation by lack of swelling and tenderness medially and by alignment of the humerus with the forearm (**Fig. 5.3**); ultrasound may help.
   d. Internal oblique radiograph shows the fracture best.

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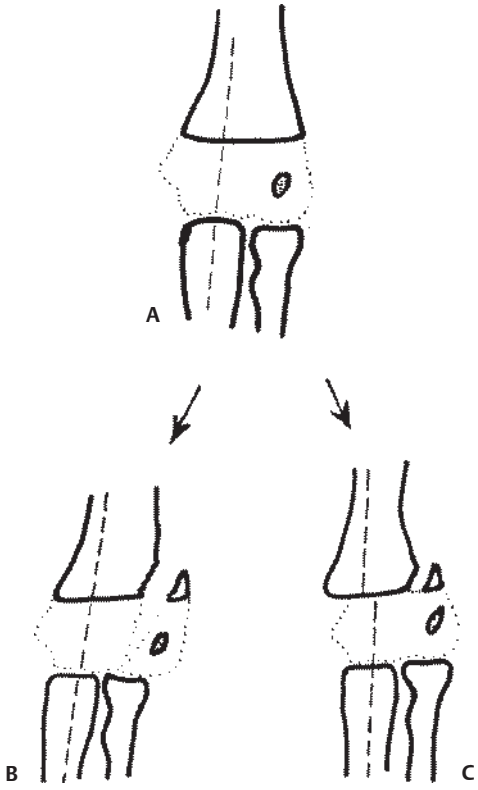



**Fig. 5.3** Differentiation of lateral condyle from distal humeral physeal fracture. In the latter, displacement of the entire forearm follows the metaphyseal fragment. In minimally displaced fractures, physical examination, ultrasound, arthrogram, or magnetic resonance imaging may be helpful. **(A)** Normal alignment of ulna and humerus. **(B)** Alignment maintained in lateral condyle fracture. **(C)** Alignment is lost in distal humeral Salter II physeal fractures.

**2.** Principles and significance

- **a.** Lateral condyle fracture is one of the few pediatric fractures in which nonunion is not rare.
- **b.** Cast is rarely able to maintain reduction of a displaced lateral condyle.

**3.** Treatment recommendations

- **a.** Displacement less than 2 mm→ splint in 90 to 100 degrees of flexion; pronation: recheck at 5 and 10 days (open reduction, internal fixation [ORIF] if further displacement)

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**b.** Displacement greater than 2 mm or follow-up is unreliable:
attempt closed reduction (optional)

reduced / not reduced

internally fix / ORIF

**1)** Avoid posterior dissection; visualize reduction anteriorly
**2)** Internal fixation: Two divergent pins preferred
**3)** May cross physis if needed
**4)** May use screw; remove later
**5)** Usually remove pins and splint at 6 to 8 weeks

**c.** Fiberglass allows best visualization of fracture
**d.** Late presentation (displaced and ununited longer than 6 weeks)
**1)** Approach anteriorly with graft and rigid fixation if in good position (early). Operate only for lateral instability symptoms if after 3 weeks. Avoid excessive dissection.
**2)** Valgus osteotomy if significant deformity exists
**3)** Ulnar nerve anterior transposition if deformity is increasing

## Medial Epicondyle Fractures

**1.** Background

**a.** Medial epicondyle begins to ossify at 4 to 6 years; fuses at around 15 years of age
**b.** Fracture most common in ages 9 to 12 years
**c.** Significance of this fracture
**1)** Medial collateral ligament of ulna attaches to the base of the epicondyle.
**2)** Entrapped medial epicondyle may be missed.
**3)** Look for medial condylar extension.

**2.** Treatment

**a.** Principle: Displacement is well tolerated unless forceful loading is anticipated.
**b.** Indications for ORIF
**1)** Epicondyle in joint despite attempt at manipulation
**2)** High valgus stresses anticipated (dominant arm of throwing athlete, etc.) in patient with displaced epicondyle. “Stress test” in 15 degrees of flexion may be of interest, but most are unstable acutely; no specific guidelines for interpretation of this test exist.

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**c.** Technique of ORIF
  **1)** Exposure is easier in prone position. May use screw or percutaneous pin. Ulnar nerve transposition is not recommended in most cases.

**d.** Indications for closed reduction: All cases not meeting the preceding criteria for ORIF, including the following special cases:
  **1)** Acute ulnar neuropathy: Likely to resolve with time; not a specific indication for ORIF or ulnar nerve transposition
  **2)** Displacement greater than 5 mm but not intra-articular
  **3)** Epicondyle fracture with elbow dislocation
  **4)** Technique of closed reduction: Extend elbow, wrist, and fingers simultaneously with slight valgus

## Elbow Dislocations

**1.** Background

**a.** More common in older children
**b.** Before skeletal maturity, about 60% have associated fracture:
  **1)** Medial epicondyle
  **2)** Proximal radius
  **3)** Coronoid
  **4)** Olecranon
**c.** *Open* dislocations have a high incidence of arterial injury.

**2.** Treatment

**a.** Carefully examine for associated fractures, especially of radial head or lateral condyle.
**b.** Closed reduction with sedation or anesthesia is successful in most cases.
**c.** Admit or instruct parent in vascular examination
**d.** Splint for 2 to 3 weeks
**e.** Follow-up to rule out posterolateral instability
**f.** If dislocation is missed for longer than 1 week, open reduction is needed.

## Nursemaid's Elbow (Annular Ligament Entrapment)

**1.** Caused by longitudinal traction in child 1 to 4 years old
**2.** Elbow held slightly flexed, pronated, guarded
**3.** Radiographs are normal.
**4.** Treatment: Flex and supinate (**Fig. 5.4**); immobilization usually is not needed.

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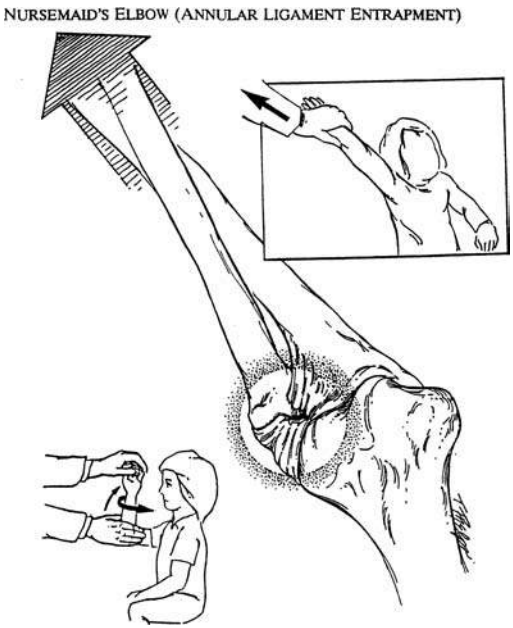



**Fig. 5.4** Nursemaid's elbow. Mechanism is traction and pronation. Reduction by is flexion and supination. (From Sponseller PD. Bone, joint, and muscle. In Oski FA, ed. Principles and Practice of Pediatrics. 2nd ed. Philadelphia: J.B. Lippincott; 1994:1037 (Fig. 38–25). Reprinted with permission.)

## Radial Head and Neck Fractures

**1.** Background

- **a.** Ossification develops in about 4 to 5 years
- **b.** Much of radial neck is intracapsular
- **c.** Wilkins classification
  - **1)** Primary displacement of *radial head*
    - **a)** Valgus fractures
    - **b)** Salter l and II
    - **c)** Salter IV
    - **d)** Metaphyseal
    - **e)** Fractures with elbow dislocation
    - **f)** Reduction injuries
    - **g)** Dislocation injuries
  - **2)** Primary dislocation of *radial neck* (i.e., Monteggia variant)

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**2.** Treatment guidelines

- **a.** More than 30 degrees of angulation: Accept, begin range of motion in 1 to 2 weeks
- **b.** 30 to 45 degrees of angulation: Manipulate but accept closed result
- **c.** More than 45 degrees (approximately) or translocated: Manipulate but require ORIF if unsuccessful

**3.** Manipulation techniques (with patient under anesthesia)

- **a.** Traction in extension
  - **1)** Use fluoro to profile fracture.
  - **2)** Press on displaced fragment.
  - **3)** Apply varus stress to forearm.
- **b.** Flexion–pronation technique
  - **1)** Flex elbow 90 degrees.
  - **2)** Rotate forearm from full.
  - **3)** Supination to pronation while applying pressure anteriorly to radial head
- **c.** Percutaneous K wire leverage:
  - **1)** Elbow in extension
  - **2)** Use fluoroscopy.
  - **3)** Pin starts proximally in the "corner"of radial head, behind the posterior interosseous nerve.
- **d.** Metaizeau technique: 2 to 2.5 mm intramedullary rod from the distal radial metaphysis advanced retrograde into the epiphysis to obtain or maintain reduction

**4.** Maintaining reduction

- **a.** Flexion to 90 degrees if stable
- **b.** For unstable fractures, prefer oblique K wire from proximal to distal fragment. Protect with posterior splint.

### Monteggia Fracture–Dislocation

**1.** Principle: Radial head dislocates in the direction in which a line through the distal ulnar fragment is pointing. Even a slight ulnar bend may be a sign of radial head subluxation (**Fig. 5.5**).

**2.** Bado classification (based on the direction of radial head dislocation)

- **a.** Anterior
- **b.** Posterior
- **c.** Lateral
- **d.** Anterior with fracture of proximal third of the radius

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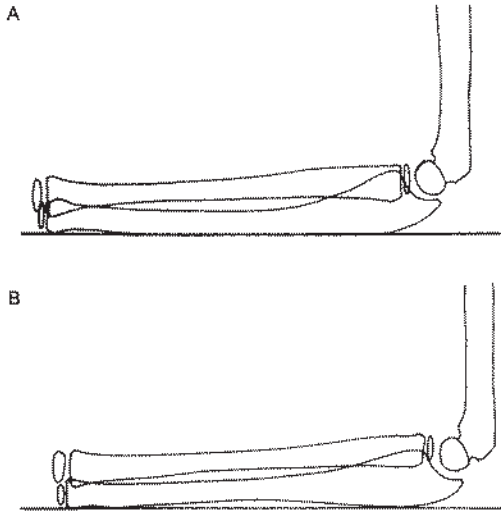



**Fig. 5.5** Ulnar bow sign. **(A)** Maximum deviation of the elbow cortex from a straight line should be 1 mm. **(B)** If greater, it should suggest plastic deformation of the ulna, and radial head subluxation is possible. (From Lincoln TL, Mubarak SJ. "Isolated" traumatic radial-head dislocation. J Pediatr Orthop. 1994;14(4):455 (Fig. 1AB). Reprinted with permission.)

**3.** Treatment

- **a.** Reduction mechanism used to reduce the ulnar fracture, with supination added for anterior and lateral types of dislocation
- **b.** Failure to achieve or maintain radial head reduction: Intramedullary pinning or plating of ulna
- **c.** If this does not reduce the radial head, openly reduce it.

**4.** Late-presenting Monteggia fracture–dislocation can be operatively reduced up to several years after injury. Correct the ulnar bow and reconstruct the annular ligament (Bell Tawse procedure).

## Supracondylar Humerus Fracture

**1.** Classification

- **a.** Direction of displacement
  - **1)** Most are due to hyperextension; periosteal hinge is posterior.
  - **2)** Few are due to flexion: Periosteal hinge is anterior.
- **b.** Degree of displacement
  - **1)** Type I: Undisplaced
  - **2)** Type II: Hinged/greenstick intact; posterior cortex
  - **3)** Type III: Completely displaced

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2. Principle: Type III fractures have an appreciable incidence of nerve and artery damage. They have little intrinsic stability. Best results in type III occur with pin fixation. Carefully document the status of all nerves and circulation before treatment. This involves (1) checking active palmar flexion (median nerve); (2) flexion of distal interphalangeal joints of index finger and thumb, anterior interosseous nerve; (3) dorsiflexion of the metacarpophalangeal joints, posterior interosseous nerve; (4) flexion of the fifth finger distal interphalangeal joint; or (5) crossing of the index and second fingers, Ulnar nerve (**Fig. 5.6**).
3. Treatment
    a. Type II: Correct hyperextension and any angulation. Flex > 90 degrees. Consider pin fixation.
    b. Type III: Percutaneous pin fixation (**Fig. 5.7**)—one medial and lateral or two lateral pins. Both pins should start distal to the fracture site.

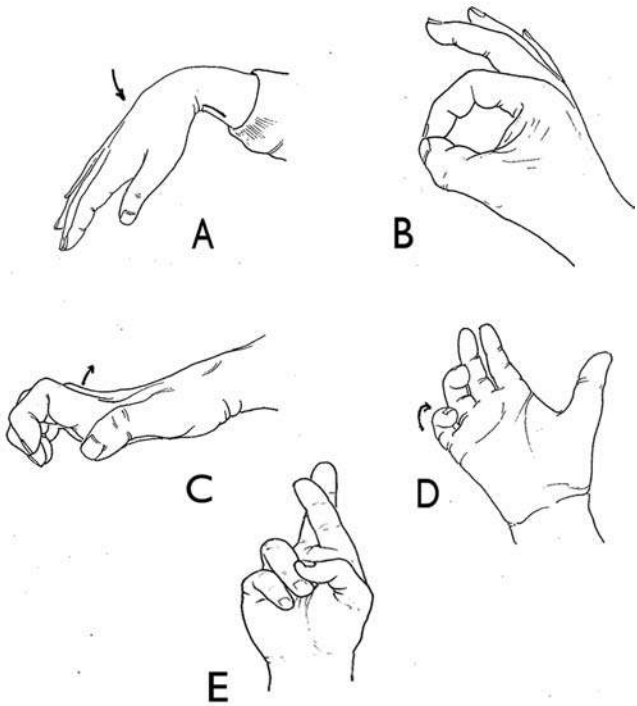



**Fig. 5.6** Documentation of the status of all nerves and circulation before treatment of supracondylar humerus fractures. This involves **(A)** checking active palmarflexion (median nerve); **(B)** flexion of distal interphalangeal joints of the index finger and thumb–anterior interosseous nerve; **(C)** dorsiflexion of the metacarpophalangeal joints–posterior interosseous nerve; **(D)** flexion of the fifth finger distal interphalangeal joint; or **(E)** crossing of index and second fingers–ulnar nerve.

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**Fig. 5.7** One technique of closed reduction and percutaneous pinning. Longitudinal traction is applied in slight flexion to correct angulation and yet allow visualization. Fluoroscopy receiver serves as platform.



**Fig. 5.8** Desired pin placement for medial and lateral pin technique.

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The lateral pin should engage a portion of the capitellum, and the medial pin should be slightly medial and anterior on the epicondyle to avoid the ulnar nerve (**Fig. 5.8**). Make a small incision to clear a tract. If two lateral pins are used, one should cross the lateral third of the fracture, and one should cross the central third of the fracture (**Fig. 5.9**).

**c.** If anatomic closed reduction is not possible, perform open reduction. Check alignment of the fracture using Baumann angle (**Fig. 5.10A**); normal is 72 ± 4 degrees. Also check the anterior humeral line (**Fig. 5.10B**), which should intersect the anterior one third to one half of the capitellum.

**d.** Aftercare: May begin protected range of motion at around 3 weeks with temporary splint removal. Remove pins at 6 weeks.

**4.** Nerve injury

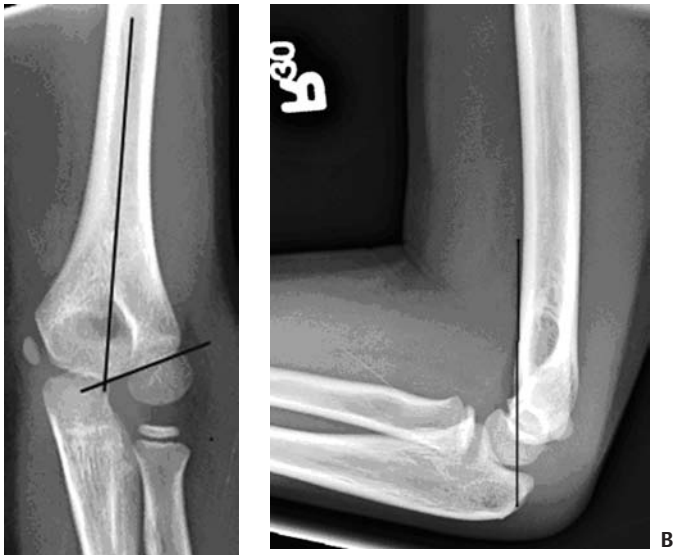
**a.** Frequency: Radial > median > anterior interosseous > ulnar

**b.** Treatment: If deficit is present before reduction, it is probably a neuropraxia resulting from the injury; proceed with closed reduction. If no return by 5 months after injury, then obtain electromyelogram; explore and perform neurolysis if no recovery.



**Fig. 5.9** Lateral pin fixation for type III supracondylar humerus fractures.

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A B

**Fig. 5.10** **(A)** Baumann angle (normal, 72 degrees); **(B)** anterior humeral line should intersect anterior or middle third of capitellum.

**5.** Arterial insufficiency

- **a.** Reduce fracture; do not hyperflex.
  - **1)** If perfusion returns, pin fracture.
  - **2)** If perfusion does not return, perform an open exploration through an anterior Henry approach.
  - **3)** If artery is entrapped, release and watch.
  - **4)** If in spasm, use lidocaine.
  - **5)** If an intimal tear occurs, repair.
  - **6)** If transected, vein graft.
- **b.** Measure compartment pressures after reperfusion and perform fasciotomy if needed.

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## ◆ Pediatric Hand and Wrist Fractures

### Hand Fractures

1. Distal phalanx fractures
   - **a.** Pediatric mallet finger: Often an open injury
   - **b.** Clean thoroughly
   - **c.** Replace nail under fold
   - **d.** Closed reduction or ORIF of the fracture as indicated
   - **e.** Follow-up to rule out infection
2. Phalangeal neck (subcondylar) fractures
   - **a.** Principles
     - **1)** Most occur in proximal phalanx.
     - **2)** Volar angulation is the most common.
     - **3)** Minimal remodeling occurs in this region.
   - **b.** Treatment of displaced fracture
     - **1)** Closed reduction
     - **2)** Transarticular percutaneous pinning for 3 weeks
     - **3)** Buddy-tape for 1 week.
     - **4)** Late presentation: May openly reconstruct up to 4 weeks post fracture
   - **c.** Malunion: Loss of flexion may occur if malposition with bony impingement is allowed to persist.
   - **d.** Treatment: Volar approach and removal of bony block to flexion
3. Phalangeal shaft fractures
   - **a.** Less common in children than adults
   - **b.** May accept 10 degrees dorsal/palmar angulation
   - **c.** Immobilization: short arm cast/splint
4. Metacarpophalangeal joint injuries
   - **a.** Collateral ligament does not protect physis of proximal phalanx or metacarpal head.
   - **b.** "Extra octave" fracture of small finger
     - **1)** Reduce using pencil in web space as a fulcrum.
     - **2)** Reduction should be maintained when pressure is released.
     - **3)** Hold with rolled cotton gauze between digits.

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- **c.** Intra-articular fractures
  - **1)** If fragment is greater than 25% of the joint surface or involves any tendon insertion, it needs to be reduced to within 2 mm of anatomic alignment.
  - **2)** Growth arrest is seen in less than 1% of patients.
- **d.** Dislocation
  - **1)** Often complex dorsal dislocation
    - **a)** Volar plate entrapped
    - **b)** Joint space wide, diaphyses parallel
    - **c)** Sesamoids interposed
    - **d)** Skin dimpled on volar surface
  - **2)** Treatment
    - **a)** One or two attempts at closed reduction
    - **b)** Open reduction: Volar or dorsal approach
    - **c)** Incise superficial transverse ligament lateral to volar plate

**5.** Pediatric thumb metacarpal fractures

- **a.** Metaphyseal or Salter I and II fractures: Attempt closed reduction and cast; pin if unacceptable
- **b.** Pediatric Bennett fractures: Closed reduction or ORIF if more than 1 mm displacement
- **c.** Gamekeeper's fracture
  - **1)** Stener lesion, usually a Salter III type
  - **2)** ORIF if displaced more than 1 mm or normal radiographs
  - **3)** With positive stress test at 45 degrees of flexion

**6.** Scaphoid fractures

- **a.** Much less frequent in children than adults
- **b.** May occur with distal radius fracture
- **c.** If diagnosis is made within 0 to 3 months, it will usually heal with a short-arm thumb spica cast.
- **d.** Delayed union of more than 3 months → bone graft

## Distal Radial Physeal Fractures

**1.** Salter I and II are most common.
**2.** One or two gentle reductions + with adequate analgesia or anesthesia

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3. Immobilize in neutral position or pronation.
4. Growth arrest is rare.
5. Complications
    a. Compartment syndrome
    b. Median nerve injury

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## ◆ Spine Fractures

### Cervical Spine Fractures

1. General principles
    a. Child should be transported on a special backboard to accommodate large head: Recess under head or lift under shoulders
    b. Obtain radiographs if
        1) Unconscious patient
        2) Neck pain
        3) Head or facial bruising in motor vehicle accident
    c. Recommended films
        1) Lateral, anteroposterior, open mouth
        2) Obliques only if dislocation or subluxation is suspected
    d. Normal values
        1) See Chapter 1's Fig. 1.13. Also refer to this section for normal ossification patterns
    e. Algorithms are shown for "clearing" (ruling out injury) in the trauma patient with normal radiographs but with tenderness or altered consciousness (**Figs. 5.11, 5.12, 5.13,** and **5.14**).

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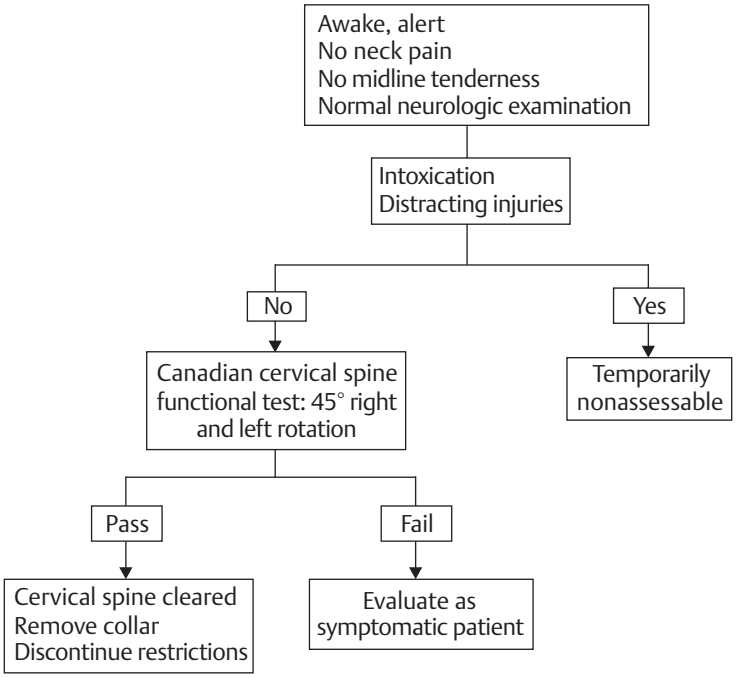



**Fig. 5.11** Evaluation of the asymptomatic blunt trauma patient. (From Anderson PA, Gugala Z, Lindsey RW, Schoenfeld AJ, Harris MB. Clearing the cervical spine in the blunt trauma patient. J Am Acad Orthop Surg. 2010;18(3):149–159 (Fig. 1). Reprinted with permission.)

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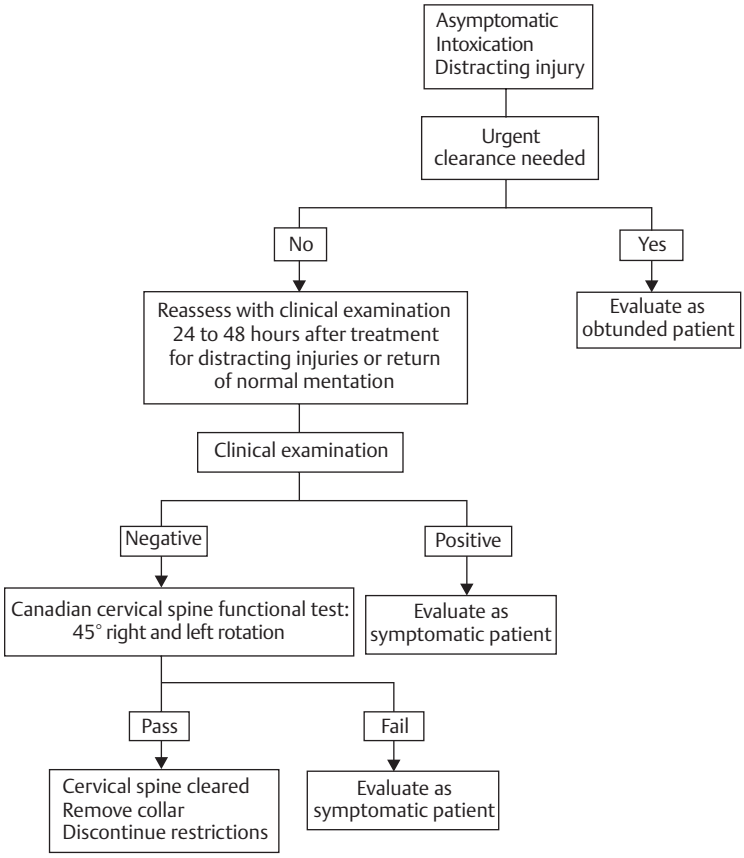



**Fig. 5.12** Evaluation of the temporarily unassessable trauma patient. (From Anderson PA, Gugala Z, Lindsey RW, Schoenfeld AJ, Harris MB. Clearing the cervical spine in the blunt trauma patient. J Am Acad Orthop Surg. 2010;18(3):149–159 (Fig. 2). Reprinted with permission.)

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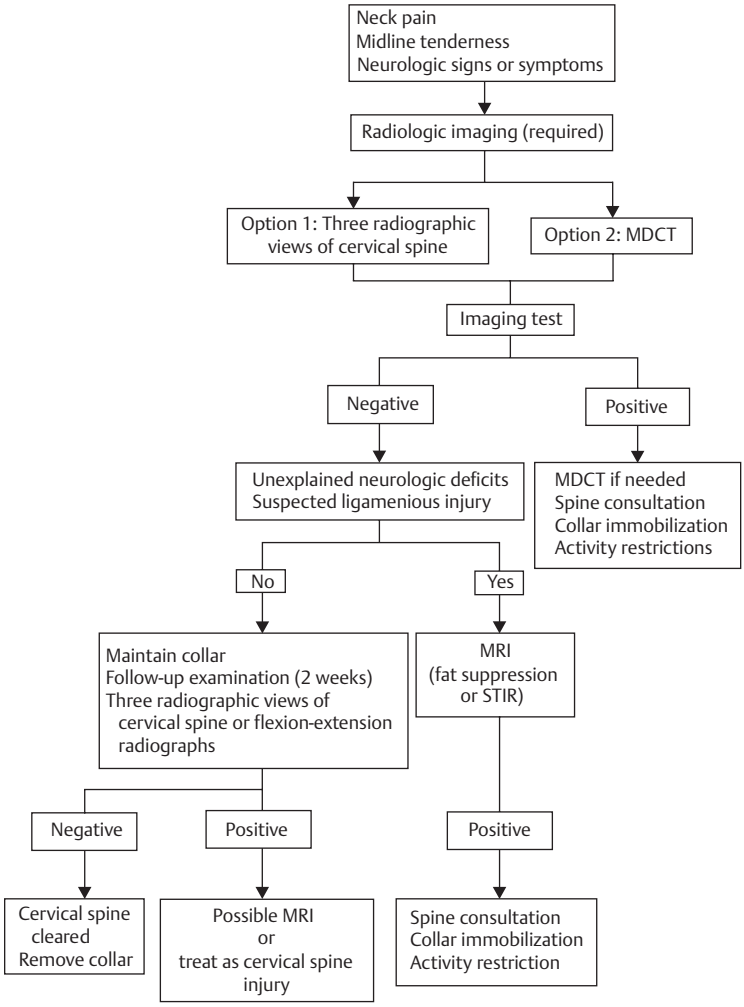



**Fig. 5.13** Evaluation of the symptomatic trauma patient. (From Anderson PA, Gugala Z, Lindsey RW, Schoenfeld AJ, Harris MB. Clearing the cervical spine in the blunt trauma patient. J Am Acad Orthop Surg. 2010;18(3):149–159 (Fig. 3). Reprinted with permission.)

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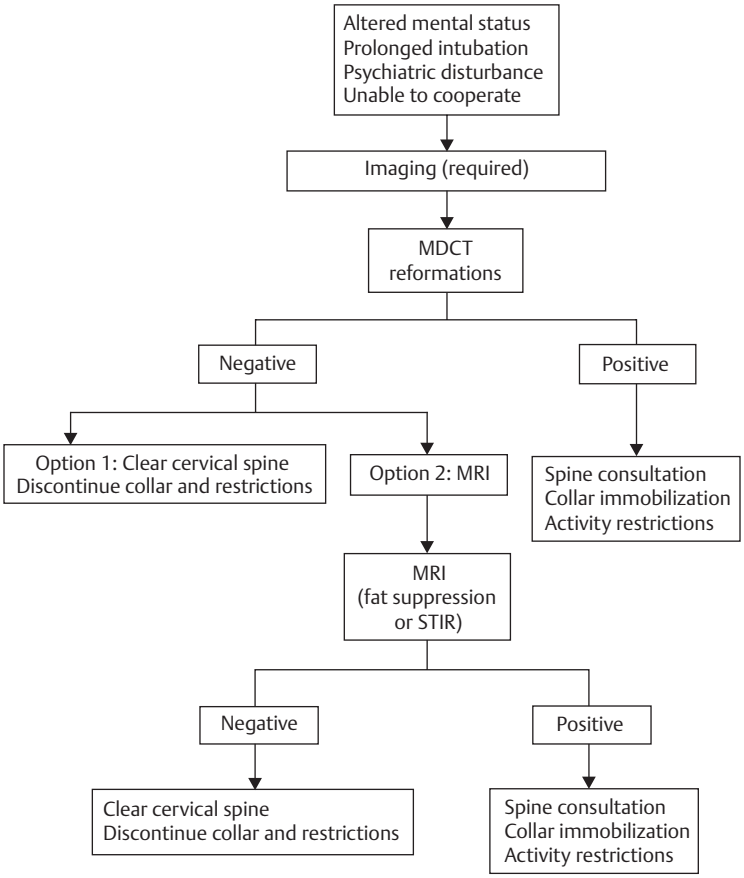



**Fig. 5.14** Evaluation of the obtunded trauma patient. (From Anderson PA, Gugala Z, Lindsey RW, Schoenfeld AJ, Harris MB. Clearing the cervical spine in the blunt trauma patient. J Am Acad Orthop Surg. 2010;18(3):149–159 (Fig. 4). Reprinted with permission.)

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**2.** Atlanto-occipital displacement (**Fig. 5.15**)

- **a.** Usually skull is distracted and displaced forward. Suspect if dens–basion distance is greater than 12 mm or occipital condyles not resting in the superior facets of the atlas, or the Power ratio is greater than 1. Confirm with CT or magnetic resonance imaging. Document neurologic status.
- **b.** Immobilize with recessed backboard and minimal or no traction.
- **c.** Fusion of occiput to C1 or C2 is the most commonly accepted treatment.

**3.** Odontoid fracture

- **a.** Reduce and hold in halo or Minerva for 8 weeks; then a Philadelphia collar for 4 weeks

**4.** Atlas (C1–Jefferson) fracture

- **a.** Minimum (<7 mm) spread of lateral masses → Philadelphia collar
- **b.** If significant spread of lateral masses (>7 mm) is seen, then traction for 4 weeks followed by collar

**5.** C1-C2 rotatory subluxation (**Fig. 5.16**)

- **a.** CT scan is best study to confirm diagnosis
- **b.** Symptom duration less than 1 week → collar, analgesics, bed rest, exercises to reduce
- **c.** Symptom duration longer than 1 week → halter traction

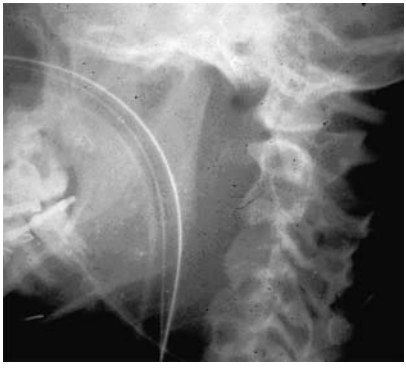





**Fig. 5.15** Atlanto-occipital displacement.

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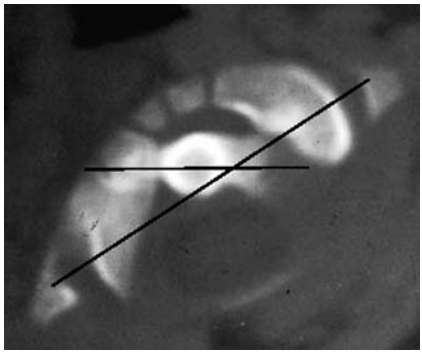


**Fig. 5.16** C1-C2 rotatory subluxation.

**d.** Symptom duration longer than 1 month → halo traction, attempt reduction. Fuse in situ if not reducible

**6.** Transverse ligament insufficiency or os odontoideum (**Fig. 5.17**)

**a.** Assess with flexion–extension views

**1)** 3 to 4 mm: Normal



**Fig. 5.17** Transverse ligament insufficiency.

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**2)** 4 to 8 mm: Collar, restrict activities
**3)** More than 8 mm or any neurologic abnormalities → posterior fusion C1-C2

**7.** C2 pedicle fracture (Hangman's)

**a.** If C2-C3 disk is intact, immobilize in collar or halo.
**b.** If C2-C3 disk disrupted, consider anterior spine fusion.

### Thoracic and Lumbar Spine Fractures

**1.** Compression fracture

**a.** If less than 20%, mobilize as tolerated.
**b.** If greater than 20%, thoracolumbar spinal orthosis for comfort; mobilize as tolerated.

**2.** Burst fractures

**a.** If neurologically normal, cast 6 to 8 weeks and then mobilize as tolerated.
**b.** If neurologic deficit is present, decompress anteriorly or posteriorly and fuse.

**3.** Flexion–distraction (chance) (seatbelt) injuries (**Fig. 5.18**)
**a.** Posterior elements distracted through facets, lamina, or pedicles. Minimal to no compression anteriorly
**b.** Treatment: Attempt reduction in extension and immobilize for 6 to 8 weeks.
**c.** If reduction is not obtained or is still unstable or if significant abdominal injury exists, then fuse.

## ◆ Femoral Shaft Fractures

### Background Principles

**1.** Mechanism: Pedestrian struck by car; fall or sports; passenger in motor vehicle accident
**2.** Acceptable reduction: Varus/valgus of up to 10 degrees, anterior/posterior bow of 20 degrees
**3.** Overgrowth of about 1 cm occurs between ages 2 and 10 years.
**4.** Family factors are important in choosing treatment.
**5.** Consider child abuse if patient is younger than 2 years.
**6.** Hip spica cast is not a good way to maintain length.

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**Fig. 5.18** Flexion-distraction (Chance) (seatbelt) injury.

## Treatment

1. Age younger than 6 years: Resting overlap less than 2 cm or telescope test shows less than 3 cm of shortening:
   Yes → spica cast
   No → traction (in hospital or at home) or external fixator
2. Age 6 to 10 years
   a. If resting overlap is less than 2 cm or telescope test shows less than 3 cm of shortening, a spica cast is an option if the parents choose it.
   b. Preferred options:
      1) Flexible IM rods/plate/fixator
      2) Traction: in hospital or at home
3. Age older than 10 years
   a. IM rod with careful preparation of entry hole to avoid disrupting vessels at femoral neck (trochanteric entry)
   b. External fixator
   c. Plate

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### Time to Union (Mean)

1. Infant: Less than 4 weeks.
2. Age 2 to 4 years: 4 to 6 weeks.
3. Age 4 to 6 years: 6 weeks.
4. Age 6 to 8 years: 6 to 8 weeks.
5. Times are longer for open or high-energy injuries.

### Subtrochanteric Fractures

1. Overgrowth also occurs here: 1 cm on the average.
2. One can accept angulation of 25 degrees in any plane.
3. Fracture tends to develop anterior bow.
4. Fracture is harder to image in cast.
5. Treatment

    a. ORIF, plate
    b. External fixator
    c. Traction for 3 weeks in 90-degree flexion, then spica in 90–90 flexion or

## ◆ Physeal Injuries About the Knee

### Normal Anatomy and Growth

1. Length of lower limbs doubles between 4 years and maturity.
2. Distal femoral physis

    a. Quadripodal shape
    b. Ligaments concentrate stress on this physis.
    c. Growth is 1 cm/year till age 13½ (girls), 15½ (boys).
    d. Blood supply to physis comes from epiphyseal vessels primarily, with some contribution from periosteal vessels.

3. Proximal tibial physis

    a. An anterior extension continues down to the tubercle.
    b. Ligaments, fibula, and semimembranosus insertion protect physis.
    c. Growth is 8 mm/year.

### Distal Femoral Physeal Fracture

1. Most common physeal injury about the knee
2. Mechanism

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**a.** Hyperextension, or
**b.** Valgus

**3.** Findings

**a.** Hemarthrosis, especially in Salter III, IV
**b.** May be missed in polytrauma patient.
**c.** Ligamentous injury may coexist.

**4.** Radiographs

**a.** Obliques and tunnel views if needed
**b.** Stress view if occult fracture suspected
**c.** Plain tomograms or CT for complex Salter III and IV if fracture pattern or displacement is in question
**d.** When to obtain arteriogram
**1)** If vascular examination is abnormal
**2)** Proximal tibia physeal fracture
**3)** Incidence of vascular injury ≤ 1%
**4)** In most cases, arteriogram is not necessary; especially in varus–valgus injuries. Instead, check circulation before and after reduction and instruct caregivers in monitoring it.

**5.** Treatment

**a.** Gentle closed reduction
**b.** One may accept 5 degrees varus/valgus in Salter types I and II
**c.** Open reduction if irreducible closed
**d.** Pin if unstable
**e.** ORIF all displaced type IV fractures
**f.** Ensure physeal alignment by direct inspection as well as fluoro (at fracture and periphery).
**g.** Immobilization
**1)** Long leg cast if limb is slender and fracture is stable
**2)** Spica cast otherwise
**h.** Begin range of motion by 6 weeks
**1)** Follow-up at least 1 year to rule out growth plate injury

**6.** Results

**a.** 25 to 50% have length discrepancy greater than 1 cm.
**b.** 25% have angular deformity >5 degrees.

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**7.** Treatment of physeal bridge

- **a.** Imaging
  - **1)** Growth lines visible on plain film should be present and parallel to physis if growth is normal.
  - **2)** Tomograms (plain, not CT) if bridge is suspected
  - **3)** MRI: Discuss with radiologist before study
- **b.** Indications for resection:
  - **1)** Area of bar is less than 50% of physeal area.
  - **2)** Growth remaining more than 2 years

### Proximal Tibial Physis Injury

**1.** One quarter as common as distal femur physeal injury; 5% have popliteal, peroneal injuries.

**2.** Mechanism

- **a.** Hyperextension
- **b.** Valgus
- **c.** 50% occur in sports.

**3.** Treatment

- **a.** Closed versus open reduction; based on standard criteria
- **b.** Close vascular monitoring

### Tibial Tubercle Fractures

**1.** Background

- **a.** Many have history of Osgood–Schlatter lesion.
- **b.** Frequency in males is greater than in females.
- **c.** Usual age range is 14 to 16 years.
- **d.** Almost always seen in jumping sports

**2.** Treatment

- **a.** Closed if minimally displaced and patient can activity actively extend knee
- **b.** ORIF
  - **1)** Clear bed of interposed tissue.
  - **2)** Use screw if fragment is large and the patient is near maturity.
  - **3)** Otherwise, suture tendon and periosteum.

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3. Complications: Rarely seen

   a. Recurvatum: Only if patient is very young (i.e, younger than 11 years)
   b. Lack of flexion

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## ◆ Physeal Injuries About the Ankle

### Background

1. Normal growth

   a. Distal tibial and fibular epiphyses appear at around the age of 2 years and close at age 15 to 16 years.
   b. Anterolateral portion closes last.
   c. Distal fibular physis is normally at the level of tibial plafond.

### Fracture Classification (Dias)

1. SER (supination–external rotation)
2. PER (pronation–external rotation)
3. SPF (supination–plantar flexion)
4. SI (supination–inversion)
5. Axial loading
6. Tillaux
7. Triplane

### Nonarticular Physeal Fractures

1. Closed versus open reduction: Standard criteria (5 degrees varus/valgus is acceptable)

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2. Check rotation radiographically and clinically by thigh–foot angle.
3. Use long leg cast in most cases if displaced.

### Tillaux Fractures

1. Anterior inferior tibiofibular ligament avulses unfused epiphyseal fragment.
2. Mechanism = SER
3. Treatment: Reduce with internal rotation gap greater than 2 mm → ORIF

### Triplane Fracture

1. Mechanism usually SER
2. May be 2, 3, or 4-part
3. Concern is mainly articular congruity rather than growth remaining (most patients with this fracture are nearing maturity).
4. Treatment
    a. Attempt closed reduction.
    b. If it looks all right, get CT afterward to confirm.
    c. ORIF if more than 2-mm spread or any vertical displacement
    d. Anterolateral incision; Reduce posteromedial fragment first, then medial incision if needed.

### Salter IV

1. Reduce and fix if there is any longitudinal displacement or more than 2-mm spread.

### Physeal Arrest

1. Distal tibia is third most common site of growth arrest after fracture.
    a. Represents 25% of all physeal bars
    b. May occur with Salter II as well as III, IV, and V
2. Implications
    a. Counsel family of this risk at time of fracture
    b. Follow up for a year or longer after injury
3. Partial arrest
    a. Calculate potential angulation based on growth remaining
    b. Some guidelines

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1) 8 mm = 10-degree deformity will occur if bar forms before age 13½ (boys) or 11½ (girls)
2) 1-cm growth remains after age 13 (boys), age 11 (girls)
3) Refer to growth remaining chart (Fig. 1.23).

4. Bar resection versus epiphysiodesis

   a. Latter simpler, more predictable
   b. Patient's choice
   c. There is less need to do resection than in other areas of skeleton because length considerations are lessened.

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# 6 Normal Values and Medications

This chapter includes laboratory values and medications the pediatric orthopedic surgeon needs to know. Pediatric dosages for medication must be calculated based on body weight. Normal laboratory values vary according to age. When giving agents that affect respiration, a plan should also be in place for monitoring and treatment of any adverse responses.

## ◆ Normal Laboratory Values

(**Tables 6.1** and **6.2**)

## ◆ Antibiotics

(**Table 6.3**)

## ◆ Other Medications Frequently Used in Pediatric Orthopedics

(**Table 6.4**)

**Table 6.1** Normal CBC by Age

| Age | HCT (%) mean – 2 SD | WBC/mm X 100 Mean ± 2 SD (Normal Ranges) |
|---|---|---|
| Newborn | 51 (42) | 18.1 (9–30) |
| 6 mo | 36 (31) | 11.9 (6–17.5) |
| 6–24 mo | 36 (33) | 10.6 (6–17) |
| 2–6 yr | 37 (34) | 8.5 (5–15.5) |
| >6 yr | 40 (35) | 8.1 (4.5–13.5) |

*Abbreviations:* HCT, hematocrit; SD, standard deviation; WBC, white blood cell count.

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**Table 6.2** Normal Lab Values by Age

| Parameter | Normal values |
|---|---|
| Sodium | 135–145 mg/dL |
| Potassium | 3.5–5.0 mg/dL |
| Phosphorus | 8–10.5 mg/dL |
| Alkaline phosphatase | |
| Infant | 150–400 U/L |
| 2–10 yr | 100–300 U/L |
| 11–18 yr (male) | 50–375 U/L |
| 11–18 yr (female) | 30–300 U/L |
| Erythrocyte sedimentation rate | 1–5 mm/h |
| Creatinine | |
| Infant | 0.2–0.4 mg/dL |
| Child | 0.3–0.7 mg/dL |
| Adult | 0.5–1.0 mg/dL |
| Glucose | |
| 1 wk–16 yr | 60–105 mg/dL |
| >16 yr | 70–115 mg/dL |
| Albumin | |
| 3–4 mo | 2.8–5.0 mg/dL |
| 1 yr | 3.5–5.0 mg/dL |
| 2 yr–adult | 3.3–5.8 mg/dL |
| ALT (SGPT) | |
| <1 yr | <54 U/L |
| >1 yr | 1–30 U/L |
| AST (SGOT) | |
| <1 yr | 25–75 U/L |
| >1 yr | 0–40 U/L |

Source: *The Harriet Lane Handbook*, 1994.
*Abbreviations:* ALT, alanine aminotransferase; AST, aspartate aminotransferase; SGOT, serum glutamic-oxaloacetic transaminase; SGPT, serum glutamic–pyruvic transaminase.

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**Table 6.3** Antibiotic Guidelines

| Name | Dose | Interval | Route | How Supplied | Comments |
|---|---|---|---|---|---|
| Amikacin | 15–22 mg/kg/day | q8–12h | i.v., i.m. | Inj. 50,150 mg/mL | Peak 20–30 mg/kg, through 5–10 mg/kg |
| Amoxicillin | Child: 20–50 mg/kg/day | q8h | p.o. | Drops: 50 mg/mL; suspension: 125, 250 mg/mL | Less GI irritation than ampicillin |
| | Adult: 250–500 mg/dose | q8h | p.o. | Caps: 250, 500 mg; chewable: 125, 250 | |
| Amoxicillin & clavulanic acid (Augmentin) | Child: 20–40 mg/kg/day (approx.) | q8h | p.o. | Suspension: 125 & 250 mg/mL; tabs: 250–500 mg | Used with influenza, *Staphylococcus aureus*, β lactamase producers |
| Ampicillin | 100–400 mg/kg/day | q6h | p.o./i,m./i.v. | Drops: 100 mg/mL; suspension: 125, 250, 500 | Maximum oral dose 4 g daily; may cause nephritis |
| Carbenicillin | 400–600 mg/kg/day | q6h | p.o./i.m.<br>i.v. (cut dose to ½) | | |
| Cefaclor (Ceclor) | 40 mg/kg/day child | q8h | p.o. | | Use with caution in renal impaired or pcn allergy |
| Cefadroxil (Duricef, Ultracef) | Child: 30 mg/kg/day | q12h | p.o. | | |
| | Adult: 500–1000 mg/day | | | | |
| Cefamandole | Child: 50–150 mg/kg/day | q4–6h | i.m., i.v. | | |
| | Adult: 4–12 g/day | | | | |

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|---|---|---|---|---|---|
| Cefazolin (Ancef, Kefzol) | Child: 50–100 mg/kg/day | q6–8h | i.m., i.v. | | Use with caution in renal failure or pcn allergy |
| Cefaperazone (Cefobid) | Child: 100–200 mg/kg/day | q12h | i.m., i.v. | | |
| | Adult: 2–4 g/day | | | | |
| Cefotaxime (Claforan) | Child: 100–200 mg/kg/day | q6–8h | i.m., i.v. | | |
| | Adult: 2–12 g/day | | | | |
| Cefoxatin (Mefoxin) | Child: 80–160 mg/kg/day | q4–6h | i.m., i.v. | | |
| | Adult: 4–12 g/day | | | | |
| Ceftazidime (Fortaz/Ceptaz) | Child: 90–150 mg/kg/day | q8–12h | i.m., i.v. | | |
| | Adult: 2–6 g/day | | | | |
| Ceftizoxime | Child: 150–200 mg/kg/day | q8h | i.m., i.v. | | |
| | Adult: 2–12 g/day | | | | |
| Ceftriaxone (Rocephin) | Child: 50–75 mg/kg/day | q12–24h | i.m., i.v. | | |
| | Adult: 1–4 g/day | | | | |
| Cefuroxine (Zinacef) | Child: 75–150 mg/kg/day | q8h | i.m., i.v. | | |
| | Adult: 2–4.5 g/day | | | | |
| Cephalexin (Keflex) | Child: 25–50 mg/kg/day | q6h | p.o. | Drops: 100 mg/mL; suspension: 125, 250 mg/5 mL; tabs: 250, 500, 1000 mg; caps: 250, 500 mg | |
| | Adult: 1–4 g/day | | | | |

*(continued on p. 214)*



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**Table 6.3** *(continued from p. 213)*

| Name | Dose | Interval | Route | How Supplied | Comments |
|---|---|---|---|---|---|
| Cephalothin (Keflen) | Child: 80–160 mg/kg/day | q4–6h | i.m., i.v. | | |
| | Adult: 2–12 g/day | | | | |
| Chloramphenicol | 50–100 mg/kg/day | q6h | p.o., i.v. | | Monitor levels in infants |
| Ciprofloxacin (Cipro) | 500–750 mg | b.i.d. | p.o. | | Not recommended under 16 yr |
| | 200–400 mg | q2h | i.v. | | |
| Clindamycin | Child: 20–30 mg/kg/day | q.i.d | p.o. | Caps: 75, 150, 300 mg | May cause pseudo-membranous colitis |
| | 25–40 mg/kg/day | q8–8h | i.m., i.v. | Suspenion: 75 mg/5 mL | |
| | Adult: 600–1800 mg/day | q6–8h | p.o. | | |
| | 600–3600 mg/day | q6–12h | i.m., i.v. | | |
| Cloxacillin (Tegopen) | Child: 50–100 mg/day | q.i.d. | p.o. | Caps: 250, 500 mg; solution: 125 mg/5 mL | |
| | Adult: 1–4 g/day | | | | |
| Dicloxacillin | Child: 50–100 mg/kg/day | q.i.d. | p.o. | | |
| | Adult: 500–2500 mg/day | q.i.d. | p.o. | | |
| Doxycycline | 5 mg/kg/day | q12h | p.o., i.v. | Caps/tabs: 50,100; suspension: 25 mg/5 mL | Do not use in children under 8 yr |
| Erythromycin | Child: 30–50 mg/kg/day | q6–8h | p.o., i.v. | | Multiple GI discomfort; give after meals; caution with liver disease |

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|---|---|---|---|---|
| | Adult: 1–4 g/day | q6h | p.o., i.v. | |
| Ethambutol | 15–25 mg/kg/day | q.d. | p.o. | Do not use in children under 12 yr; |
| Gentamicin | Child: 6–7.5 mg/kg/day | q8h | i.v. | monitor levels: peak 6–10 mg/L; trough 2 mg/L |
| | Adult: 3–5 mg/kg/day | | | |
| Methacillin | Child: 100–400 mg/kg/day | q4–6h | i.m., i.v. | |
| | Adult: 4–12 g/day | | | |
| Metronidazole (Flagyl) | Load with 15 mg/kg, then 7.5 mg/kg/dose | q6h | i.v.<br>i.v., p.o. | |
| Oxacillin | Child: 50–100 mg/kg/day | q6h | p.o. | |
| | Adult: 500–1000 mg/dose | | | |
| Penicillin G | Child: 100,000–4000,000 uits/kg/day | q4–6h | i.v. | Probenecid may prolong |
| | 25–50 mg/kg/day | q6–8h | p.o. | |
| | Adult: 2–24 million units/day<br>125–500 mg/dose | q4–6h<br>q6h | i.v.<br>p.o. | |
| Penicillin G (potassium) | Child: 25–05 mg/kg/24 h | q6h | p.o. | Must be taken 1 h before or 2 h after meals |

*(continued on p. 216)*



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**Table 6.3** *(continued from p. 215)*

| Name | Dose | Interval | Route | How Supplied | Comments |
|---|---|---|---|---|---|
| | Adult: 250–500 mg/dose | q6h | p.o. | | |
| Rifampin | 0–20 mg/kg/dose | q12–24h | i.v., p.o. | | Colors secretions red |
| Tetracycline HCL | Older child: 25–50 mg/kg/day | q6h | p.o. | | Do not use under age 8 yr |
| | Adult: 1–2 g/day | | | | |
| Ticarcillin | 200–300 mg/kg/day | q4–6h | i.m., i.v | | Contains Na |
| Tobramycin | Child: 6–7.5 mg/kg/day | q8h | i.v. | | Check levels; peak 6–10 mg/L, trough <2 mg/L |
| | Adult: 3–5 mg/kg/day | | | | |
| Vancomycin | Child: 10 mg/kg/dose | q8h | i.v. | | Benadryl can reverse red man syndrome |
| | Adult: 2 g/day | q8–12h | | | |

*Abbreviations:* i.m., intramuscular; i.v., intravenous; pcn, penicillin; p.o., orally; q.d., every day; q.i.d., four times daily.

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**Table 6.4** Other Medications Frequently Used in Pediatric Orthopedics

| Medication | Dose | Interval | Route | Comments/Side Effects |
|---|---|---|---|---|
| Acetaminophen (Tylenol) | 65 mg/kg/24 h | q4–6h | p.o./p.r. | Hepatotoxicity; exacerbates g6PD |
| Albuterol (Proventil, Ventolin) | 2–5 yr: 3 mg/kg/day | q8h | p.o., inhalant nebulizer | Tachycardia |
| | 6–11 yr: 2 mg/dose | | | |
| | >12 yr: 2–4 mg/dose | | | |
| Ascorbic acid (vitamin C) | 35–50 mg/kg | q.d. | p.o. | |
| Aspirin | 65 mg/kg 24 h; Max 3–6 g/24 h | q4–6h | p.o./p.r. | GI upset, bleeding. Do not use for chicken pox or flu-like symptoms therapeutic levels 150–300 mg/L |
| Baclofen | Initial dose 5 mg | t.i.d. | p.o. | Avoid abrupt withdrawal |
| | Increase 5 mg t.i.d. q3 days until maximum of 10–15 mg t.i.d. for child 2–7 | | | |
| | 20 mg t.i.d. for child 7–8 yr | | | |
| | 20 mg q.i.d. for adult | | | |
| Beclomethasone | 1 to 2 inhalations | q6h | | |
| Bisacodyl (Dulcolax) | 2–11 yr, 5–10 mg | p.r.n. | p.r. | Effect takes 30 min |
| | >11 yr, 10 mg | | | |
| Calcium carbonate (Os-Cal, Tums) | | q.i.d. | p.o. | May cause constipation |
| Chloral hydrate - sedative/hypnotic | 50–100 mg/kg/dose | | | |

*(continued on p. 218)*



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**Table 6.4** *(continued from p. 217)*

| Medication | Dose | Interval | Route | Comments/Side Effects |
|---|---|---|---|---|
| Cimetidine | Children: 20–40 mg/kg/day | q8–12h | | Contraindicated in renal, cardiac disease |
| | Adults: 1.2 g/day | | | |
| Codeine | 1 mg/kg/dose | q4h | p.o./i.m. | |
| Dantrolene for malignant hyperthermia crisis | 1 mg/kg; repeat until signs & symptoms normalize, up to 10 mg/kg | | | |
| Diazepam (Valium) | Sedative: 0.1–0.3 mg/kg/day | q2–4h | i.m./p.o. | |
| | Anticonvulsant: 0.1–0.3 mg/kg/dose | | i.v. bolus | |
| | Rate should not exceed 5 mg/min Maximum dose: infants and toddlers, 5 mg. Older children, 15 mg. May repeat q15 min, x2 | | | |
| Diphenhydramine (Benadryl) | Child: 5 mg/kg/day | q6h | p.o., i.v. | |
| | Adult: 100–200 mg/day | q6h | p.o., i.v. | |
| Dimetapp (decongestant/antihistamine) | 1 mo–2 yr: 1.25 mL | q6–8h | p.o. | |
| | 2–4 yr: 3.75 mL | q6–8h | p.o. | |
| | 4–12 yr: 5 mL | q6–8h | p.o. | |
| | >12 yr: 5–10 mL | q6–8h | p.o. | |
| | 1 tab | q12h | p.o. | |
| Ducosate sodium - laxative (Colace) | < 3 yr: 10–40 mg/24 h | q6–12h | p.o. | |
| | 3–6 yr: 20–60 mg/24 h | q6–12h | p.o. | |

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|---|---|---|---|---|
| | 6–12 yr: 40–120 mg/24 h | q6–12h | p.o. | |
| | >12 yr: 50–200 mg/24 h | q6–12h | p.o. | |
| Ducosate and casanthranol (laxative) (Pericolace) | 5–10 mL | q.h.s. | p.o. | |
| Fentanyl | 0.5–3.0 µg/kg/dose | q1h | i.v. | Give over 3 min |
| Ferrous sulfate | Drops (15 mg Fe/0.6 mL) | q8h | p.o. | |
| | Syrup (18 mg Fe/5 mL) | | | |
| | Elixir (44 mg Fe/5 mL) | | | |
| | Tablet (60 mg Fe/tab) | | | |
| | Dose 3 mg Fe/kg/24 h | | | |
| Folic acid (vitamin) | Dose 1 mg/d | | p.o. | |
| Furosemide (Lasix) | Child: 1 mg/kg/dose (may increase by 1 mg/kg/dose) | q6–12h | i.v. | |
| | Adult: 20–80 mg/dose | | | |
| Haloperidol (sedative) | 0.01–0.1 mg/kg | q2h | p.o. | |
| Hydromorphone (Dilaudid) | 1–4 mg | q4–6h | p.o., i.v., i.m. | Fewer side effects than morphine sulfate |
| Ibuprofen (Motrin, Advil) | 20–40 mg/kg/day (suspension = 100 mg/tsp) (tablets = 200, 400, 600 mg) | q6–8h | p.o. | |
| Ketamine (hypnotic) | 4–8 mg/kg | | i.m. | May cause laryngospasm, respiratory depression |
| | 0.5–2 mg/kg | | i.v. | |

*(continued on p. 220)*



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**Table 6.4** *(continued from p. 219)*

| Medication | Dose | Interval | Route | Comments/Side Effects |
|---|---|---|---|---|
| Ketorolac (Toradol) | Child: 1 mg/kg load, 0.5 mg/kg dose | q6h | i.v. | Do not use parenterally > 5 days |
| | Adult: 10 mg | q6h | p.o. | |
| Lidocaine - local anesthetic | Up to 1 mg/kg for regional block | | | |
| Meperidine HCL (Demerol) | Child: 1–1.5 mg/kg/dose | q6h | p.o./i.m./i.v. | |
| | Adult: 50–150 mg/dose | q6h | p.o./i.m./i.v. | |
| Methylprednisolone (steroid dose for spinal cord injury) | 30 mg/kg bolus, then 5.4 mg/kg/h × 23 h | | I.v. | |
| Midazolam (sedative/amnestic; Versed) | 0.05–0.15 mg/kg/dose | q4h | i.m., s.c. | |
| | 1.0 mg/kg/dose | | p.r. | |
| Morphine sulfate | 0.1–0.3 mg/kg/dose | q4h | i.m., s.c. | |
| | 0.1 mg/kg/dose | q2h | i.v. | |
| Naloxone (Narcan) | Continuous: 0.025–2 mg/kg/h | | | Short-acting, may need redose |
| | 0.01–0.1 mg/kg/dose | | | |
| | Up to maximum 2 mg/dose. Repeat q 3–5 min | | | |
| Naproxen | 10 mg/kg/day (suspension = 125 mg/tsp) (tablets - 250 & 375 mg) | q12h | p.o. | |
| Nystatin (antifungal, topical) | Infants: 1 mL | q6h | p.o. | |

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|---|---|---|---|---|
| | Children: 2–3 mL | q6h | p.o. | |
| Ondansetron (Zofran) | .15 mg/kg/dose | q4h x3 | i.v. | |
| Oxycodone | Child: 0.05–0.15 mg/kg/dose | q6h | p.o. | Abuse potential, urinary retention |
| | Adult: 5 mg | | | |
| Paraldehyde (sedative/hypnotic) | 0.3 ml/kg/dose | | i.m./p.o./p.r. | |
| Paragoric (analgesic) | 0.25–0.5 mL/kg/dose | q6h | p.o. | |
| Prochlorperazine (antiemetic; Compazine) | (>2 yr only) 0.4 mg/kg/day | q6–8h | p.o./p.r. | |
| | 0.13 mg/kg (single dose) | | i.m. | |
| Promethazine (Phenergan) | Child: 0.25–0.5 mg/kg/dose | q4h | p.o./i.m./i.v. | |
| Trimethobenzamide (antiemetic; Tigan) | 20–45 kg 100 mg | q6–8h | p.o. | |
| | <20 kg 100 mg | q6–8h | p.r. | |
| Rantidine HCL (Zantac) | Child: 2–4 mg/kg/day | q12h | p.o. | |
| | 1–2 mg/kg/day | q12h | i.v. | |

*Abbreviations:* b.i.d., twice daily; GI, gastrointestinal; G6PD, glucose-6-phosphate dehydrogenase; i.m., intramuscular; i.v., intravenous; pcn, penicillin; p.o., orally; p.r., per rectum; q.h.s., at bedtime; q.i.d., four times daily; s.c., subcutaneous.



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**Table 6.5** Medical Items Containing Latex

| Latex-Containing Items | Latex-Free Items |
|---|---|
| Gloves | Safe gloves |
| Eudermic | Tactaylon |
| Ultraderm | Neolon |
| Micro-Touch | Dermaprene |
| Bio Gel D | Elastyren |
| Neutralon | Vinyl examination gloves |
| Perry Derma-Guard | 3M tapes (Microfoam/Micropore) |
| Safeskin | Silktape |
| Pristine | Dermaclear/dermacil tape |
| Sensi-Derm | (Johnson and Johnson) |
| Neutraderm | Neclor oximeter probes |
| Brown Milled | Catheters: Ureteral |
| Baxter exam gloves | Suction, Silastic Foley's (argyle, Bard, Surgicath, Mentor) |
| Cloth tape | |
| Red rubber urinary catheters | Cautery cords (Olsen, Valley Laboratory) |
| Red rubber endotracheal tubes | Cautery pads (3M) |
| Red rubber nasopharyngeal airways (Rusch) | Ear tubes (Richard, Xomed) |
| Penrose drains | Jackson-Pratt drains |
| Band-Aids | Hemovac drains (latex inside) |
| Coban dressings | Steri-drapes (3M) |
| Condom catheters (Texas) | Steri-strips (3M) |
| Dental dams | Tegaderm (3M) |
| BP cuff and attached tubing | Ureteral catheter (Surgicath) |
| Tourniquets | Vessiloops (Devon Industries) |
| Intravenous tubing injection ports | Xeroform (Sherwood Medical) |
| Buretrol latex diaphragm | Salem sump tubes |
| Nondisposable temperature probes | Silicone Foley catheters (sherwood) |
| Black anesthesia masks | Standard ETTs (Portex, Mallinkrodt) |
| Ventilator bellows * | Plastic oral airways |
| Fresh gas flow anesthesia machine tubing | Portex nasal airways |
| Nuk nipples and some other feeding nipples and pacifiers | |
| Medication vial stoppers* | |

*Not considered to be a risk with usual use.
*Abbreviations:* BP, blood pressure; ETT, endotracheal tube.

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## ◆ Latex Allergy Prevention

### Etiology

1. Multiple exposures
2. Genetic predisposition?

### Patients at Risk

1. Myelodysplasia
2. Exstrophy
3. Cerebral palsy with shunt
4. Other congenital urologic anomalies

### Management

1. Avoid exposure to latex (**Table 6.5**).
2. Treat acute episodes with epinephrine, bronchodilators, steroids, volume.

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# 7 Common Procedures

This chapter covers procedures that are commonly needed in the daily care of children but are not well described in surgical atlases, including techniques for the application of traction in children, administration of regional blocks, and aspiration of major joints.

## ◆ Skin and Skeletal Traction

### Indications for Skin Traction

Skin traction is used for conditions that require moderate traction for reduction or comfort. It is limited by the shear resistance of skin, which is about 2 to 4 pounds per limb in younger children and 5 to 7 pounds per limb in older children. If these parameters are exceeded, blisters may form. Skin is also more sensitive when it has been traumatized or when there is swelling from an adjacent fracture. Skeletal traction is an option if skin traction is not suitable. Some indications for skin traction include the following:

1. Treatment of developmental dysplasia of the hip up to about age 2 to 3 years
2. Restoring abduction in stiff, adducted hips with Perthes disease
3. Femur fractures under age 6
    a. As definitive treatment if a cast is contraindicated and there is no spasticity
    b. As temporary treatment until definitive stabilization is performed
4. To promote resolution of transient synovitis

### Skin Traction Application

1. Apply tincture of benzoin (if desired) followed by a single layer of soft roll. Do not apply the traction strips directly to skin. Application of more than a single layer of soft roll would allow traction to slip off when weight is applied.
2. Place an adhesive strip in a sugar-tong or "U" shape on the limb as shown in **Fig. 7.1**. Be sure to pad the malleoli well. If the traction weight is to approach the limit stated above, distribute the shear over the maximum area by bringing the wrap up to the thigh.
3. Roll Ace wrap around leg to hold traction strip in place. Do not roll too tightly.
4. Place spreader in loop of adhesive strip. Make sure the malleoli are free.

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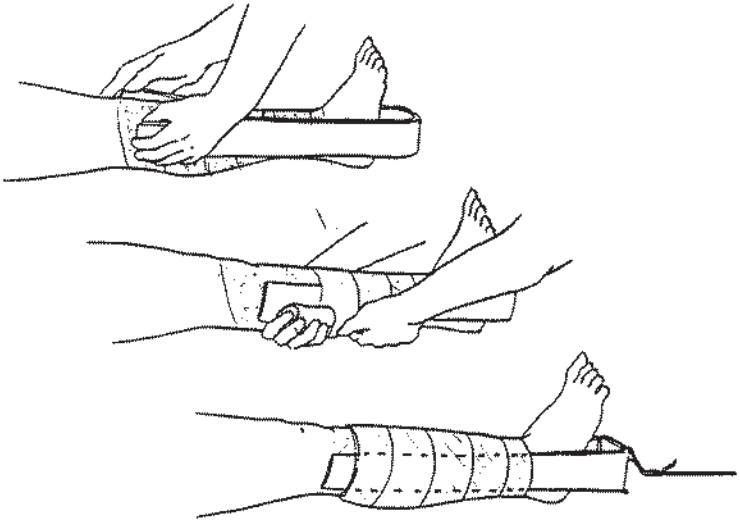


**Fig. 7.1** Application of skin traction to lower limb.

## Skeletal Traction Pin Placement

1. Collect equipment including Steinmann pin set, hand drill, sterile gloves and towels, Lidocaine, and syringe with needle and scalpel.
2. Inject local anesthetic into the entrance and exit areas, ensuring that the periosteum is thoroughly numb. Femoral nerve block is also an option (see below).
3. Make a small skin incision at the entry site. Use a hemostat to spread the soft tissue down to bone. *Note:* For a femoral pin, enter medially 1 cm above the physis, which is at the adductor tubercle (**Fig. 7.2A**). For a tibial pin, enter laterally 1 cm distal to and posterior to the base of the tibial tubercle (**Fig. 7.2B**).
4. Have an assistant stabilize the limb with longitudinal traction and counter pressure. Select a pin and insert it into the entry hole. Feel the anterior and posterior edges of the bone by gently "walking" the pin along the cortex. Determine the midpoint, and press the tip of the pin in. Adjust the angle of the pin to ensure its exit at the proper position. Maintain pressure.
5. Drill through the bone until the pin tents the skin on the far side.
6. Make a small incision to allow the pin to exit the skin. Advance the pin and place traction bow. Cut any sharp ends off the pin and protect.

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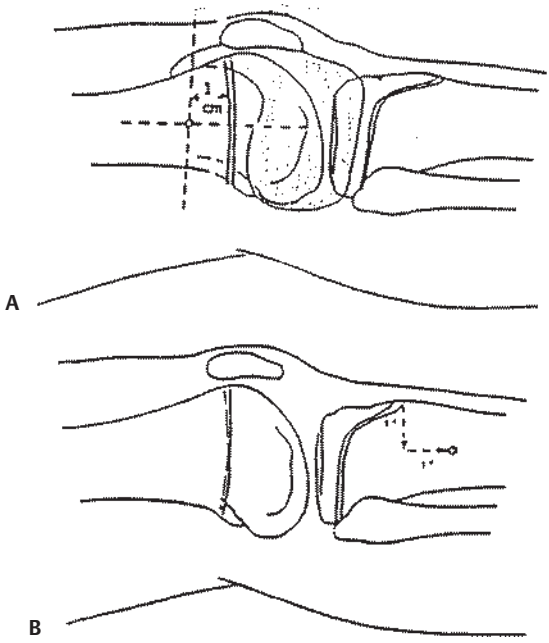



**Fig. 7.2** Insertion sites on children for **(A)** femoral pin, **(B)** tibial pin.

## Traction Assembly

1. Modified Bryant's

    a. Use for developmental dysplasia of the hip and, rarely, hip fractures for infants (**Fig. 7.3**). If a higher angle of flexion is desired, a horizontal bar may be used over the top of the crib.

2. Split Russell's

    a. Use in children with hip synovitis or awaiting treatment of fracture of midshaft of femur (**Fig. 7.4**). This may be applied as skin or skeletal traction. The hip and knee are flexed 20 to 30 degrees. The term *split* is used because in the original Russell's traction, the same weight was used to provide the vertical as well as the longitudinal traction.

3. Ninety–ninety traction (skeletal traction): Use for treatment of femur fractures in children (**Fig. 7.5**). It is appropriate for fractures of the proximal, midshaft, or distal femur. Greater supervision is required to obtain an accurate radiograph, however.

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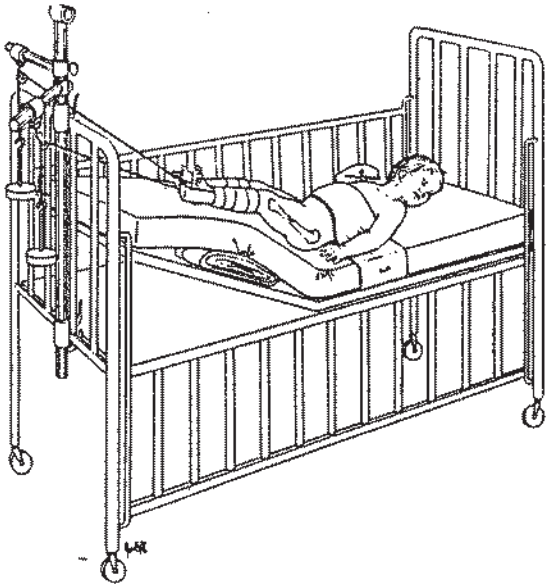


**Fig. 7.3** Modified Bryant's traction.

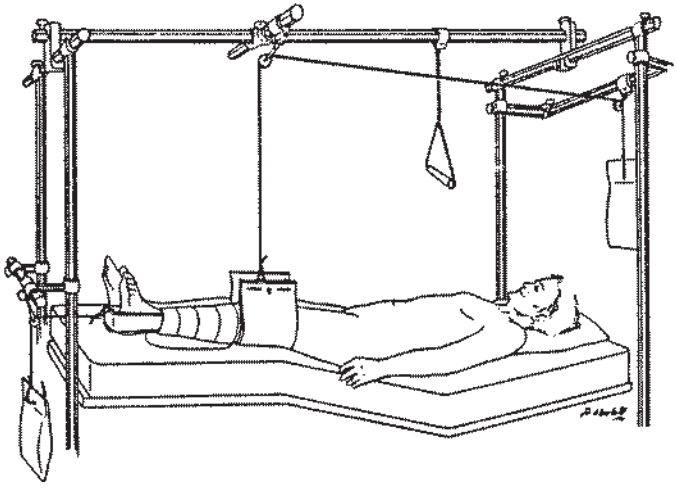


**Fig. 7.4** Split Russell's traction.

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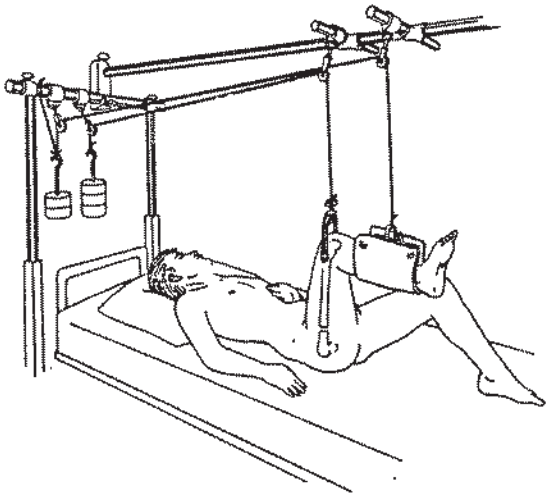


**Fig. 7.5** "Ninety-ninety" traction.

## Pediatric Halo and Halo–Vest Application

1. Indications: Use for reduction and immobilization of upper cervical spine fractures and dislocations, such as odontoid fractures, rotatory subluxation, and facet subluxations. Also used for treatment of thoracolumbar spine deformities in preparation for surgery.
2. Technique: Choose halo size so that there is at least 1.5-cm space between the ring and the cranium circumferentially. Titanium is preferred for magnetic resonance imaging (MRI) compatibility. Stabilize the head while obtaining circumferential access by moving to the end of the backboard. Local anesthetic can be injected into desired areas. Systemic analgesia may be given as needed. Anterior pin sites should be 1 cm above the eyebrow and lateral to its midportion to avoid supraorbital and supratrochlear nerves. Posterior pin sites should be diametrically opposite to these. In children under age 12, six to eight pins should be used for increased stability. Torque should be increased incrementally from 2 in-pound at age 2 to 8 in-pound at maturity.
3. If used for traction, weight is increased gradually from around 5 pounds initially. Monitor neurologic status at least once per shift, more frequently if weight is increased. This should include check of cranial nerves and upper and lower extremities.
4. Gardner-Wells tongs: These tongs can be used for temporary traction. This assembly consists of two pins, one above each external auditory meatus, inserted until the indicator tab pops out.

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## ◆ Regional Blocks

### Intravenous Regional Anesthesia (Bier Block)

1. Indications: Upper extremity fractures requiring closed reduction but not amenable to single-nerve block
2. Contraindications
   - **a.** Fracture above distal humerus
   - **b.** Vascular injury, compartment syndrome
   - **c.** Allergy to local anesthetics
3. Premedication
   - **a.** Chloral hydrate, 50 to 100 mg/kg either orally or rectally or
   - **b.** Other sedative of choice
   - **c.** Have available Valium for administration 1 to 2 mg intravenously every 2 minutes as needed for convulsions (incidence less than 0.5%)
4. Technique
   - **a.** Intravenous access in each upper extremity
   - **b.** Single- or double-cuff tourniquet on upper arm of injured side
   - **c.** Exsanguinate by gravity for 1 to 2 minutes, and then inflate cuff; if double cuff, use most proximal first.
   - **d.** Inflation pressure: 200 to 250 mm Hg
   - **e.** Inject lidocaine, 0.5%: 0.6 to 1.0 ml/kg (3–5 mg/kg)
   - **f.** Reduce fracture, apply splint or cast, and confirm with radiograph.
   - **g.** If tourniquet pain occurs, change from proximal to distal cuff.
   - **h.** Release: Tourniquet must be up for at least 30 minutes to allow tissue binding of lidocaine.
   - **i.** Deflate tourniquet for few seconds, and then reinflate and repeat over 2 minutes to allow gradual release of anesthetic.

## Bibliography

Barnes CL, Blasier RD, Dodge BM. Intravenous regional anesthesia: a safe and cost-effective outpatient anesthetic for upper extremity fracture treatment in children. J Pediatr Orthop. 1991;11(6):717–720

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## Axillary Block

**1.** Indications

- **a.** To provide anesthesia for reduction of fractures of the distal humerus or below the hand, wrist, forearm, elbow, distal arm
- **b.** For wound debridement, closure, or incision

**2.** Contraindications

- **a.** Allergy to local anesthetics
- **b.** Inability to abduct shoulder
- **c.** Bleeding diathesis
- **d.** Vascular injury or compartment syndrome

**3.** Premedication to decrease anxiety

- **a.** Intravenous line in case it is needed
- **b.** Children
  - **1)** Chloral hydrate 50 to 100 mg/kg administered orally or rectally or
  - **2)** Intravenous titration with benzodiazepine/narcotic

**4.** Technique of block administration (**Fig. 7.6**):

- **a.** Draw up anesthetic agent: Lidocaine or Mepivacaine 5 mg/kg in children, 7 mg/kg in adults (usual adult dose is 40 cc of 1% solution)
- **b.** Intravenous line and sedation as desired
- **c.** Shoulder abduction about 90 degrees
- **d.** Palpate axillary artery, pectoralis and latissimus muscles, and humeral head
- **e.** Prep and drape axilla
- **f.** Puncture skin, attempt to puncture axillary artery
- **g.** Advance the needle just through the artery with frequent aspiration checks. Inject one third of the anesthetic agent.
- **h.** Slowly withdraw the needle to just superficial to the artery, and inject the remainder of the agent, 2 to 3 mL at a time.
- **i.** Apply direct pressure on the injection site, and fold the extremity across the chest to avoid distal runoff of the solution.
- **j.** Ultrasound guidance is an option for localization of artery and plexus.

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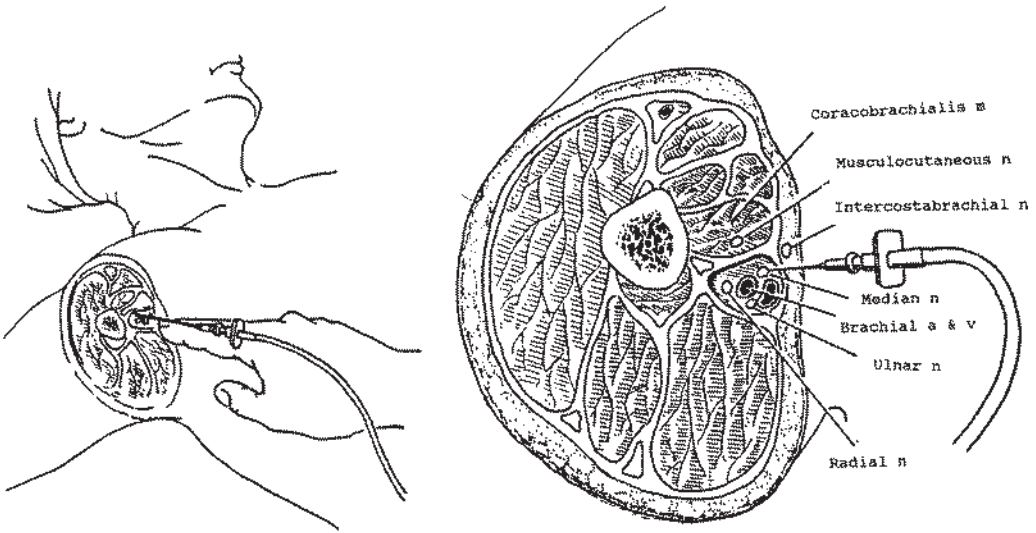



**Fig. 7.6** Landmarks for axillary block.



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## Femoral Nerve Block

**1.** Indications

- **a.** Anesthesia of anteromedial thigh or medial aspect of leg or proximal foot (medial border)
- **b.** Surgery of anterior thigh: Minor surgery
- **c.** Part of multiple lower extremity block for knee and ankle surgery
- **d.** Relief of postoperative pain in the knee
- **e.** Placement and removal of skeletal traction pins
- **f.** Manipulation and reduction of fracture of femur

**2.** Contraindications

- **a.** Ulceration or infection in the groin

**3.** Technique

- **a.** Draw up anesthetic agent: Lidocaine up to 4 mg/kg or Marcaine (bupivacaine) up to 0.5 mg/kg. Usual adult dose is 20 mL of 1% lidocaine or 10 to 15 mL of 0.5% bupivacaine.
- **b.** Patient lies supine with thigh on a flat surface.
- **c.** Landmarks (**Fig. 7.7**)
  - **1)** The femoral nerve is the most lateral structure in the femoral triangle.
  - **2)** Point of entry: One fingerbreadth lateral to the femoral artery below the inguinal ligament
- **d.** Insertion
  - **1)** Locally infiltrate the point of entry.
  - **2)** Place the middle finger of the nondominant hand on the femoral artery.
  - **3)** Insert the needle one fingerbreadth lateral to the artery cephalad at an angle of 30 degrees.
  - **4)** Once the position of the needle is confirmed, aspirate for blood.
  - **5)** Inject the appropriate volume of local anesthetic.

## Popliteal Block

**1.** Indications: For analgesia during or after procedures on the mid to lower leg or foot. May need to be combined with block of saphenous nerve if complete anesthesia is desired.

**2.** Technique: Prepare the popliteal region. Flex the knee to determine the outline of medial and lateral hamstring. Ideal location of injection is midway between these two structures, 2 to 3 cm proximal to the knee flexion crease. Use 22- to 25-gauge needle. Aspirate while injecting.

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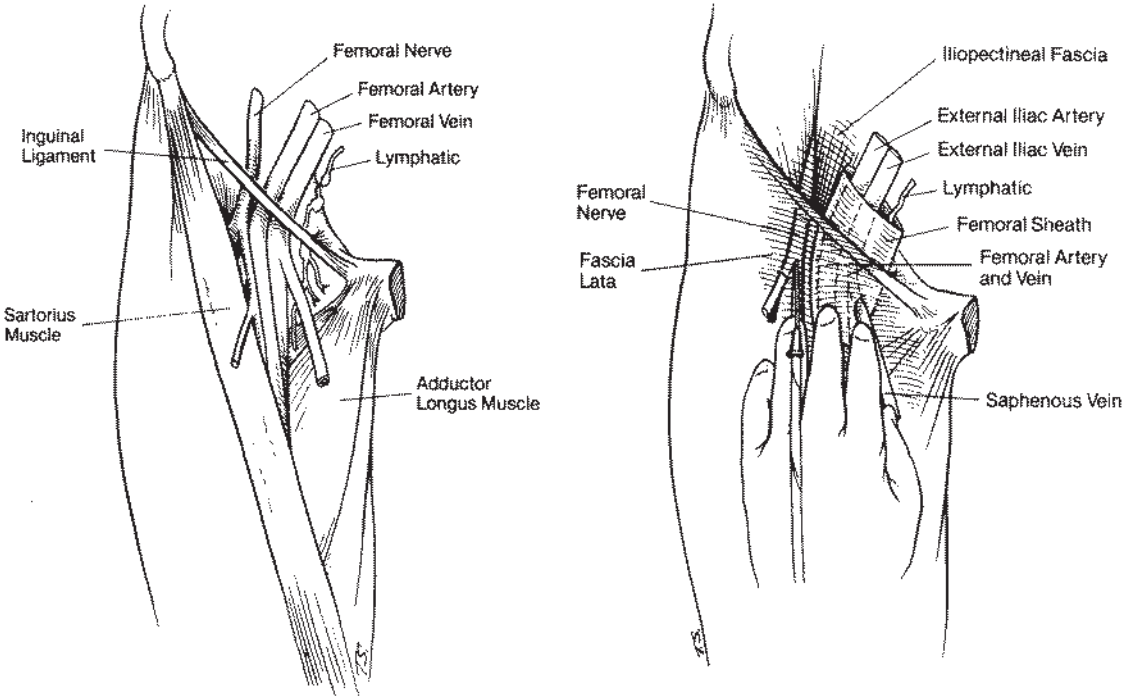



**Fig. 7.7** Landmarks for femoral block. Needle insertion is one fingerbreadth lateral to femoral artery. (From Pai U, Raj PP. Peripheral nerve blocks. In Raj PP, ed. Handbook of Regional Anesthesia. New York: Churchill Livingstone; 1985: 204. Reprinted with permission.)



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Neurovascular bundle is over two thirds of the distance between the skin and bone. If blood is obtained, withdraw the needle slightly. Nerve stimulator or ultrasound is a useful adjunct. Inject desired volume (up to 1 mg/kg of bupivacaine if no allergy). Disperse it medially and laterally. Allow at least 20 to 30 minutes for effect. Epinephrine may be used for longer effect if there is no vascular compromise.

## ◆ Injection and Aspiration

### Technique of Hip Arthrogram/Aspiration

**1.** Indications

**a.** To assess reduction and morphology of the cartilaginous epiphysis
**b.** To rule out infection

**2.** Hip arthrogram with or without aspiration may be performed via anterior, medial, or lateral approach (**Figs. 7.8** and **7.9**). An arthrogram is usually performed with any aspiration to verify that the needle is within the joint rather than in other tissue spaces. Ultrasound may also be used. Confirmation of joint entry is important before injecting arthrogram dye to avoid extra-articular contrast material obscuring the field. Confirmation is accomplished by three methods:

**a.** The sensation of needle popping through the capsule and abutting cartilage (with paradoxical motion on hip rotation)
**b.** Radiographic viewing of needle tip (fluoroscopy or ultrasound)
**c.** The saline acceptance test

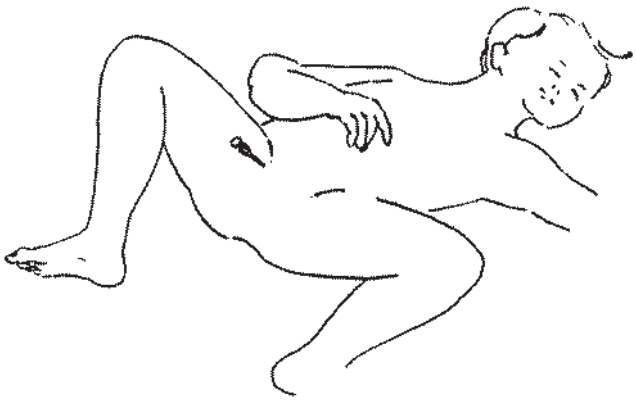


**Fig. 7.8** Medial approach for hip aspiration. Needle is inserted just posterior to adductor longus and directed toward anterior superior iliac spine.

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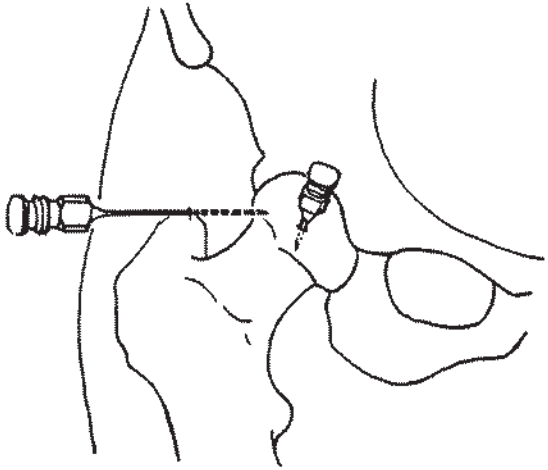


**Fig. 7.9** Anterior approach for hip aspiration is two fingerbreadths lateral to the femoral artery, below the inguinal ligament. The lateral approach is just over tip of the trochanter.

**3.** Equipment

- **a.** Choice of sedation or anesthesia
- **b.** Fluoroscope, fluoroscopy table, shields for both doctor and patient, or ultrasound
- **c.** Sterile preparation, drape, and gloves
- **d.** Needle, 18 to 20 gauge, 1½ to 3 inches long
- **e.** Sterile saline (nonbacteriostatic)
- **f.** Contrast media
- **g.** 10- to 20-mL syringes (2)
- **h.** Intravenous extension tubing (2)
- **i.** Cell count and culture tubes if needed

**4.** Procedure

- **a.** Prepare and drape the area in a fashion that allows the examiner to move the hip during study.
- **b.** Attach syringes to the tubing; fill one with saline, the other with Renografin and label.
- **c.** Localize the skin entry site and needle direction with fluoroscopy. The entry can be anterior, medial, or lateral.

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**d.** Advance the needle until it is felt to pop through capsule and abut cartilage. There should be paradoxical motion with hip rotation; the needle head should move in the opposite direction of the hip.

**e.** Confirm location with radiographs.

**f.** Check for the formation of mucin string on any fluid obtained.

**g.** Inject 1 to 3 mL of saline. If it flows easily and much of the fluid can be reaspirated, the needle is in the proper location. If much resistance occurs even though needle position is good, it is probably in the femoral head cartilage. If fluid injects easily but does not withdraw, it is probably outside the joint.

**h.** If joint fluid is to be analyzed and cultured, do it now (before injecting contrast).

**i.** Detach the saline-filled syringe and tubing; attach a contrast line and use fluoroscopy during initial injection to detect spill early. Inject just enough dye to outline the joint. If closed reduction is to be performed, minimize capsule distention.

**j.** Obtain motion studies and plain copy radiographs if needed.

**k.** If the study is being done to rule out infection, it is best done in the fluoroscopy suite with sedation rather than in the operating room. This allows adequate time to analyze joint fluid before deciding on treatment.

### Sacroiliac Joint Aspiration or Injection

**1.** Indications: To rule out infection or to assess the sacroiliac joint as site of pain

**2.** Equipment

**a.** Fluoroscope, table, gowns

**b.** Needle (18-gauge, 3-inch)

**c.** Prep and drape

**d.** Lidocaine

**e.** Renografin-60

**3.** Technique

**a.** The technique described here takes advantage of the relatively simple plane of the distal one third of the joint.

**b.** Turn the patient prone with the unaffected side tilted 10 to 30 degrees up (**Fig. 7.10**).

**c.** Adjust the tilt until the caudal one third of the affected SI joint is parallel to beam

**d.** Select the needle entry point using fluoroscopy.

**e.** Infiltrate skin as necessary.

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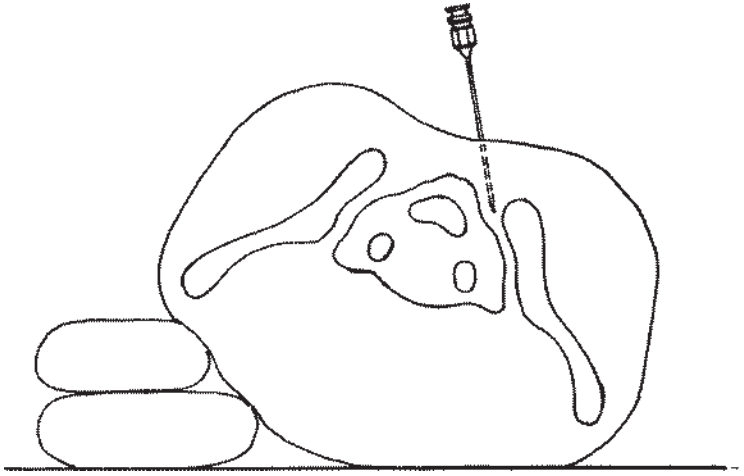


**Fig. 7.10** Positioning for aspiration of the sacroiliac joint.

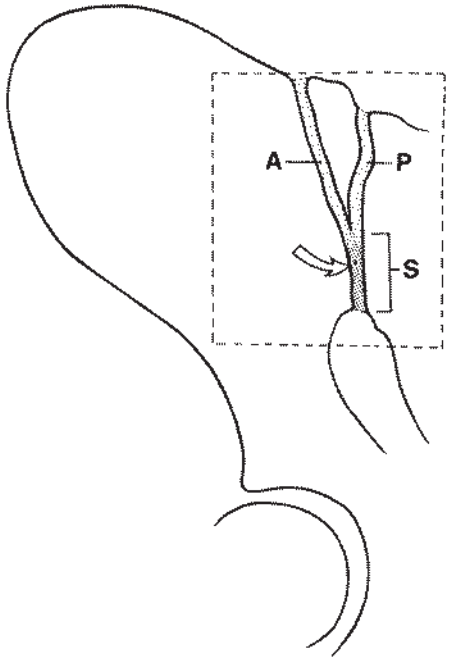



**Fig. 7.11** Desired needle placement as seen radiographically. (From Hendrix RW, Lin PLP, Kane WJ. Simplified aspiration or injection technique for the sacro-iliac joint. J Bone Joint Surg Am 1982;64(8): 1250 (Fig. 1-B). Reprinted with permission.)

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**f.** Insert needle (3 to 5 cm for average grown patient), and check for depth with lateral view as needed (**Fig. 7.11**).

**g.** Aspirate joint; wash with saline as necessary.

**h.** Confirm joint entry with contrast.

## ◆ Hip Spica Cast Application

### Indications

To immobilize a femur fracture or hip dislocation; to immobilize patient after hip surgery

### Preparation

Pain and motion should usually be controlled by general anesthesia or sedation. Local block (femoral block, above) can be added. Adequate help is needed to control the head and legs; ideally one or two knowledgeable or interested assistants are needed.

Goals are to create proper position of immobilization, adequate room for respiration, wide perineal window, and position of patient for best ease of sitting and transport. Discuss steps and define the position of the cast for which the team should aim.

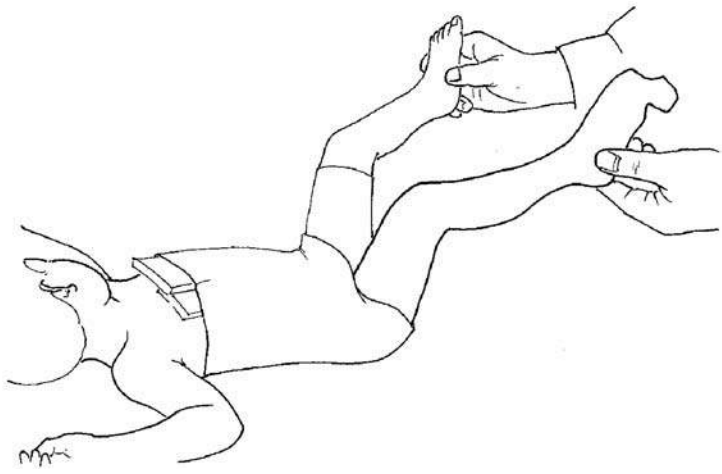
### Equipment

Equipment needed includes a spica table, one or two layers of cast liner or stocking, abundant cast padding, foam or felt for bony prominences, towels to create space for the abdomen, cast material, and cast tape. For infants, waterproof cast material may be used to allow immersion for bathing. Extra cast "splints" are used to reinforce the groin area. Bar between the legs is preferred by some orthopedists.

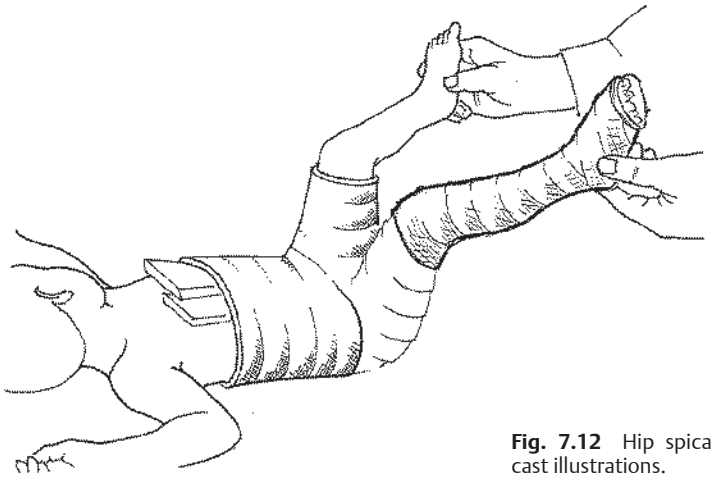
### Procedure

1. If analgesic block is to be used, do it now.
2. Cast liner or stockinette is applied over the trunk and leg(s). For femur fracture the foot is most commonly included; for developmental dysplasia of the hips (DDH) and postoperative cast, the cast may be stopped above the ankle.
3. Foam or felt padding may be applied over SI joints or other prominences (**Fig. 7.12A**).
4. Patient is positioned on spica table with extra room allowed for the torso portion. Towels may be placed on the abdomen as temporary

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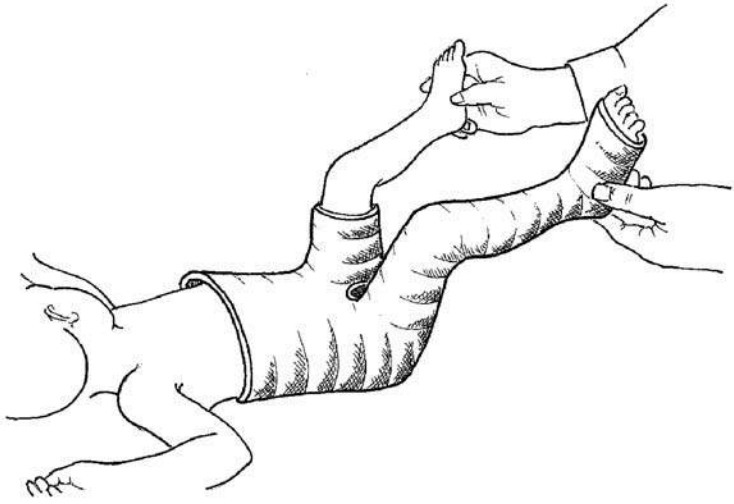
A



B

**Fig. 7.12** Hip spica cast illustrations.

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C



D

**Fig. 7.12** (continued)

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spacers to allow room for respiration. Legs are held in position of flexion and abduction.

5. For femur fracture, apply the cast in sections. An above-the-knee cast (angle less than 90 degrees) is applied to the affected leg with good padding in the popliteal fossa. Cast is applied around the torso as well (**Fig. 7.12B**). Both are allowed to set.
6. Then the reduction of the femur can be performed, maintaining gentle traction and allowing for slight extra valgus. Create narrow medial-to-lateral mold. Add splints across the groin before the final layer because the hip joint of the cast is the weakest area (**Fig. 7.12C**).
7. For closed reduction of DDH, pay attention to the angle of hip flexion (>90 degrees) and abduction (for stability and safe zone >20 degrees). Use padding and molding over affected trochanter(s) to assist reduction.
8. Turn the edges of the cast down. Use tape to hold perineal edges of the liner and create a wide perineal window. Remove towels if used (**Fig. 7.12D**).
9. Window over abdomen or G-tube if needed
10. Radiographs or other imaging of hip or femur if indicated
11. Perform neurologic and vascular checks.
12. Arrange wheelchair and car seat.

## ◆ Ponseti Technique for Clubfoot Cast

### Indications

The Ponseti technique is indicated for treatment of clubfoot in children under age 12 to 18 months who have not had surgery. It relies on guided correction of the three-dimensional malrotation of the foot and uses prolonged bracing to maintain the correction during growth. The goal is to avoid operative dissection of the bones of the foot and ankle, which could disturb growth and create incongruity. Invasive procedures are minimized and are directed at the tendons as indicated.

### Procedures

1. The infant is kept as relaxed as possible, using judicious feeding and minimizing bright light, noise, or other stimuli. Manipulation assesses the components of cavus, adduction, hindfoot varus, and equinus. In bilateral cases, usually one foot is tighter than the other, and this should be noted.
2. Padding is applied, and the foot is held by one worker who is positioned on the lateral side of the foot being treated. The foot is held in the corrected position. Another worker applies the cast of plaster or soft fiberglass. The long-leg cast is applied from the tips of the toes to midthigh

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with the leg flexed 70 to 90 degrees. Avoid changing positions during this stage.

3. Final molding is performed before and during setting of the cast. This involves the following forces:

   **a.** Dorsiflexion of the first ray, with a finger under the metatarsal head and thumb over talar neck to correct the cavus (**Fig. 7.13A**)

   **b.** Abduction of the first ray to correct the adduction

   **c.** External rotation of the foot to correct the varus

   **d.** Gentle dorsiflexion of the ankle.

   **e.** Mold the above-knee portion of the cast to prevent slipping off.

   **f.** The cast is changed weekly until the malrotation and adduction are slightly overcorrected (**Fig. 7.13B**). If the hindfoot equinus does not correct beyond neutral into dorsiflexion, an Achilles' tenotomy is indicated. This is needed in most cases, usually after three to five cast changes. Stress dorsiflexion lateral radiograph may be obtained if there is any question.

   **g.** Achilles' tenotomy is performed under local anesthesia in the office (my preference) or in the operating room with sedation. The Achilles' tendon is marked, 1 cm above the calcaneal insertion. It is infiltrated with local anesthetic. After 5 to 10 minutes, it is reprepped and incised with a #11 scalpel or Beaver blade. A distinct "give" should be felt. If not, redirect the blade slightly to ensure a complete tenotomy. Bleeding should be controlled with pressure over a sterile dressing. Then a new corrective cast is applied for 2 weeks.

   **i.** After the last cast, the feet are held in corrected position in a foot abduction orthosis. This is a bar with the feet held in 65 degrees of external rotation and 10 degrees of dorsiflexion on the involved side(s). It is worn full time for 2 months. After that, it is worn at night and nap time for 3 to 5 years, depending on the correction. Compliance with this phase is critical to success of the treatment.

   **j.** During the follow-up phase, a family member should work on stretching the foot into external rotation and eversion and dorsiflexion. As the child matures, instruct on active exercises in these directions.

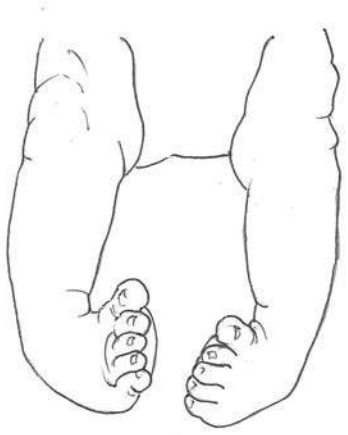
   **k.** If recurrence is noted, repeat the casting protocol and resume bracing.

   **l.** If residual deformity is noted at around 4 to 5 years, this can usually be corrected by minimally invasive surgery. Equinus can be corrected by an open Achilles' tendon lengthening. Internal rotation and adduction can be corrected by transfer of the anterior tibialis tendon to the third cuneiform or base of the third metatarsal. Any fixed deformity should be corrected first.

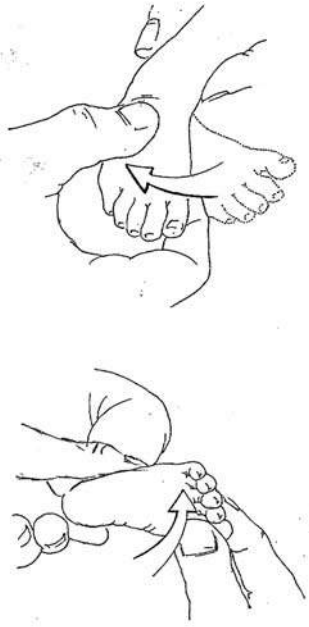
   **m.** Rarely, resistant cases may require open releases.

   **n.** Tips and tricks

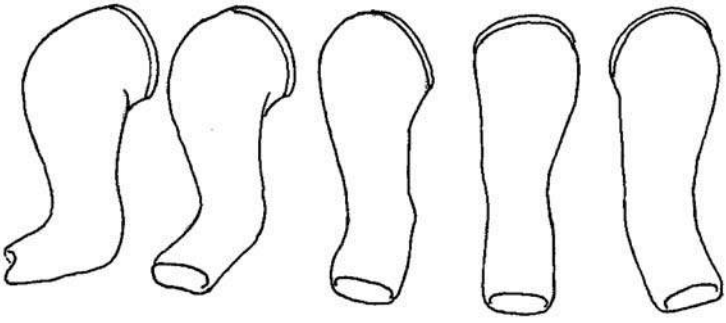
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A



B



C

**Fig. 7.13** Ponseti method for correction of clubfoot.

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1. The fulcrum for the correction involves counter pressure over the talar head, not the calcaneocuboid joint. The goal is to rotate the foot around the talar head and then to dorsiflex it at the tibiotalar joint.
2. If the cast is repeatedly kicked off by the infant, use tincture of benzoin on the skin, increase the degree of knee flexion slightly, and increase the supracondylar molding.
3. If the subcutaneous padding of the foot is thin over the talar head, adhesive padding over this area should be used.
4. If the family is not able to ensure orthosis wear, meet with them more frequently. Use interpreters if needed. Involve other family members if available.
5. One reason for poor tolerance of abduction orthosis is residual deformity. Assess and consider further casting into an overcorrected position.
6. Several varieties of foot abduction orthoses are available. There are foot pieces of soft leather (available through www.mdorthopaedics.com). One design has hinges to allow some kicking movement.
7. If the infant will not tolerate the foot abduction orthosis, a single-leg knee-ankle-foot orthosis may be ordered that will hold the foot in external rotation and dorsiflexion.

## ◆ Minerva Cast Application

### Indications

Maintenance of torticollis correction; immobilization of upper cervical spine injuries such as reduced or nondisplaced odontoid fracture

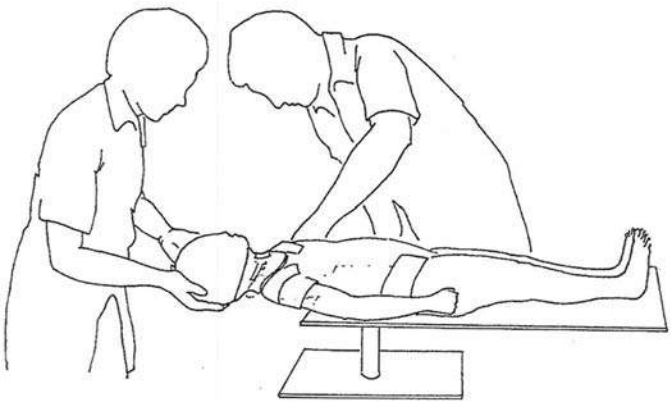
### Equipment

Adequate sedation or analgesia. Cast liner for head. Philadelphia collar to create padded contouring around chin (**Fig. 7.14A**). Padding for base of neck. Head position can be temporarily maintained using manual support (**Fig. 7.14B**) or halter (**Fig. 7.14C**). Hair may need to be trimmed. Thin cast material around the head and neck; wider for torso.

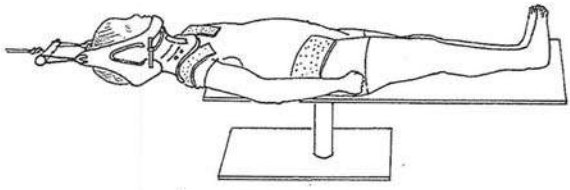
### Procedure

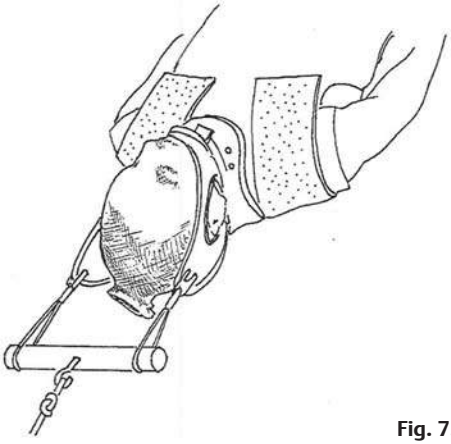
Maintain head position using halter or hands. Apply Philadelphia collar around neck. Add cast liner around torso. Apply cast and reinforce around neck. Trim and pad (**Fig. 7.14D**).

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A

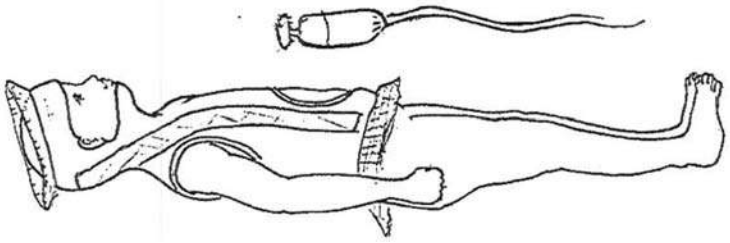




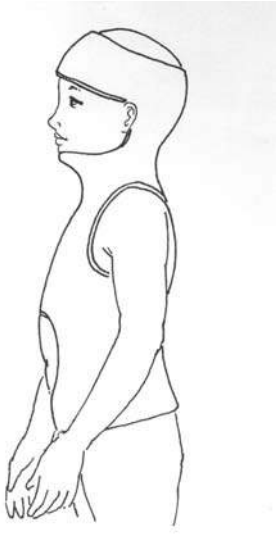
B

**Fig. 7.14** Minerva cast illustrations.

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C



D

**Fig. 7.14** (continued)

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