

PHARMACOLOGY MIND MAPS FOR MEDICAL STUDENTS AND ALLIED HEALTH PROFESSIONALS

Prasan Bhandari



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Pharmacology Mind Maps for Medical Students and Allied Health Professionals



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Dr. Prasan Bhandari
Associate Professor, Department of Pharmacology
SDM College of Medical Sciences and Hospital, Sattur, Dharwad, Karnataka, India

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Dedicated to:

ALMIGHTY CREATOR

My parents, Mr. Ramchandra G. Bhandari and Mrs. Asha R. Bhandari

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My sister, Mrs. Veebha (Lochan) V. Prabhu and my brother-in-law, Mr. Vishnu R. Prabhu

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Preface

During my tenure of teaching pharmacology, I noticed that undergraduate students find it difficult to read, remember, revise, and reproduce their subject material from standard textbooks. Empathizing with them, I wanted to write a book to provide them with alternative/supplementary material in a different format.

This book is designed for medical, dental, physiotherapy, and pharmacy students and any other healthcare professionals whose careers involve drug therapy and related aspects.

The book presents condensed and succinct descriptions of relevant and current information pertaining to pharmacology. It is not meant to be a substitute for the comprehensive presentation of information and difficult concepts found in standard textbooks of pharmacology.

Students are expected to master large amounts of information. There are few learning strategies accessible to these students to memorize and recall essential information to succeed in their medical colleges. When medical students receive very large amounts of information, passive learning results. Students remember facts rather than understanding and applying concepts.

As the medical profession continues to change, so do the methods by which medical students are taught. Various authors have accepted the need for alternative teaching and learning approaches that will help medical students to remember huge amounts of information, assimilate critical thinking skills, and explain a range of complex clinical problems.

There is a substantial necessity for faculty to move away from the customary teacher-centered educational method and enhance implementation of an active, student-centered learning environment.

One learning strategy that has been underutilized in medical education is *mind mapping*. Mind maps are multisensory tools that help students to organize, integrate, and retain information. A mind map is a diagram that represents words, concepts, ideas, or other items related to a given topic. Recent work suggests that using mind mapping as a note-taking strategy facilitates critical thinking.

Although the mind map as a learning strategy has not been extensively used in medical education, the latest research recommends using mind mapping in learning, as it increases students' long-term memory.

Mind maps, systematized by Tony Buzan, is a visual technique where information and knowledge are converted to a hierarchical formatted and illustrated diagram, with structural key terms associated with a subject. Mind maps are sprawling network diagrams that radiate out from a central point. The central topic contains a label of a general topic. Lines radiate out from that center to subtopics representing related concepts. More subtopics may radiate from those subtopics.

Mind mapping, a form of visual outlining, may seem superficial, but once mastered it provides a powerful tool for managing information overload and enabling one to quickly capture and organize a massive amount of ideas.

Mind maps are effective and can amplify productivity.

ONE SMALL STEP CAN CHANGE YOUR LIFE.

A mind map is a powerful graphic technique that can be applied to improve learning and clarify thinking. Mind maps can be used as self-learning methods to facilitate understanding of difficult concepts.

Mind mapping uses visual orientation to assimilate information and subsequently help students recall information in an organized manner. It is ideally suited for a last-minute study guide before examinations. This convenient and portable distillation of knowledge aids in memorizing and can save many hours of note taking.

We want to hear what you think. What do you like about the book's format—*the first of its kind in the world for pharmacology*? What do you think could be improved? Please share your feedback by emailing us at prasangeeta2012@gmail.com.

We are grateful to our students and our other colleagues who have taught us most of what we know about teaching. Examinations are stressful, but if you want to succeed, you have to put the work in.

However you choose to study, I hope you find this resource helpful throughout your preparation for your examinations.

Wishing you all the best for your examinations.

God Bless All.

Prasan Bhandari



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Author

Dr. Prasan Bhandari obtained his MBBS degree from one of the oldest institutes in India, Grant Medical College and Sir JJ Group of Hospitals, Mumbai, India. He received his MD in Pharmacology from the academically renowned institute Topiwala National Medical College, BYL Nair Charitable Hospital, Mumbai, India.

Dr. Bhandari has over 23 years of academic, teaching, research, administrative, and industry experience. He has published several articles in both national and international journals and has served as an examiner in several universities for both postgraduate and undergraduate medical, dental, physiotherapy, and other allied paramedical students. He has guided several postgraduate students in their research work and dissertations.

He is currently Associate Professor in the Department of Pharmacology at SDM College of Medical Sciences and Hospital, Dharwad, India.



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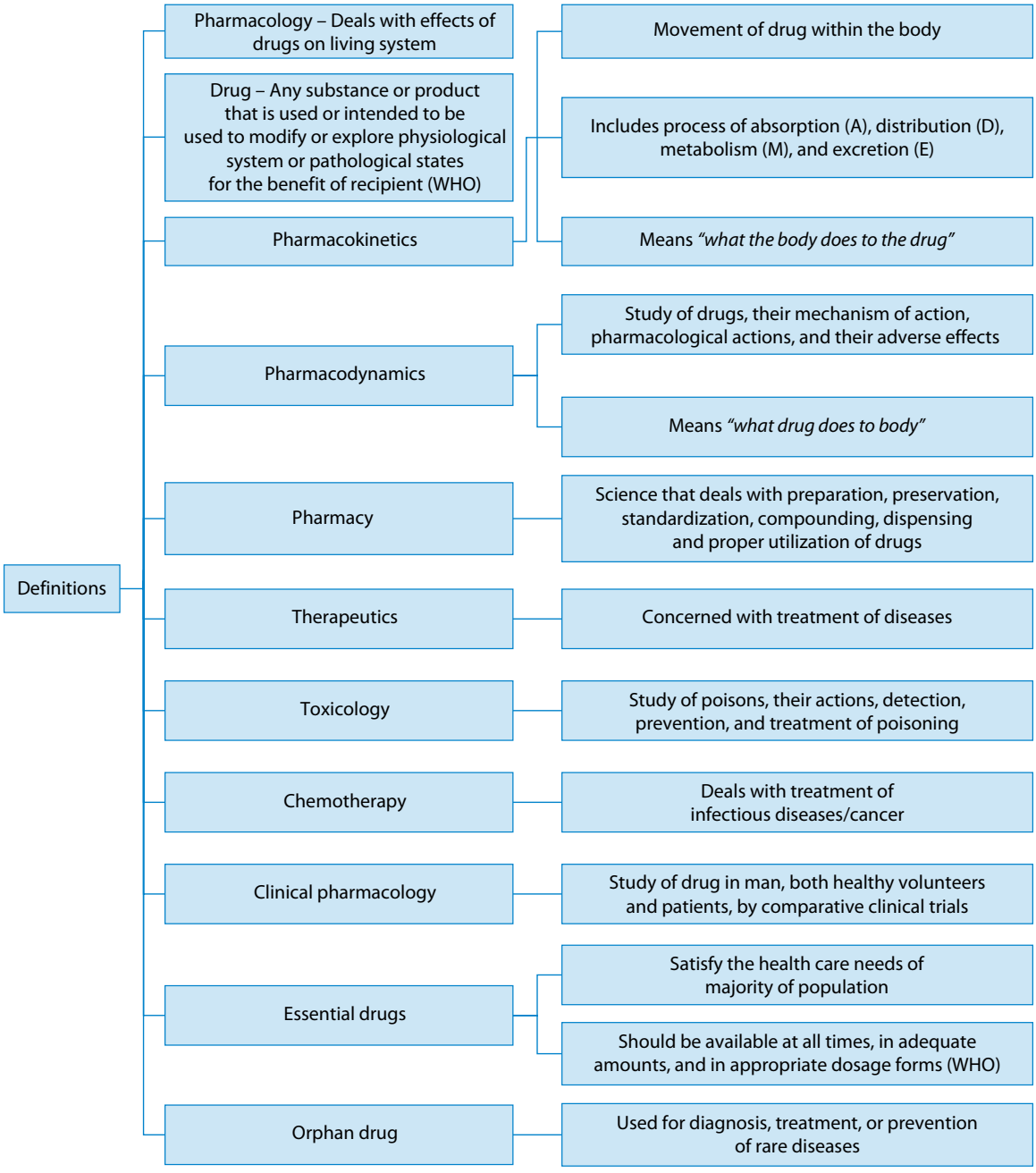
PART I

General pharmacology

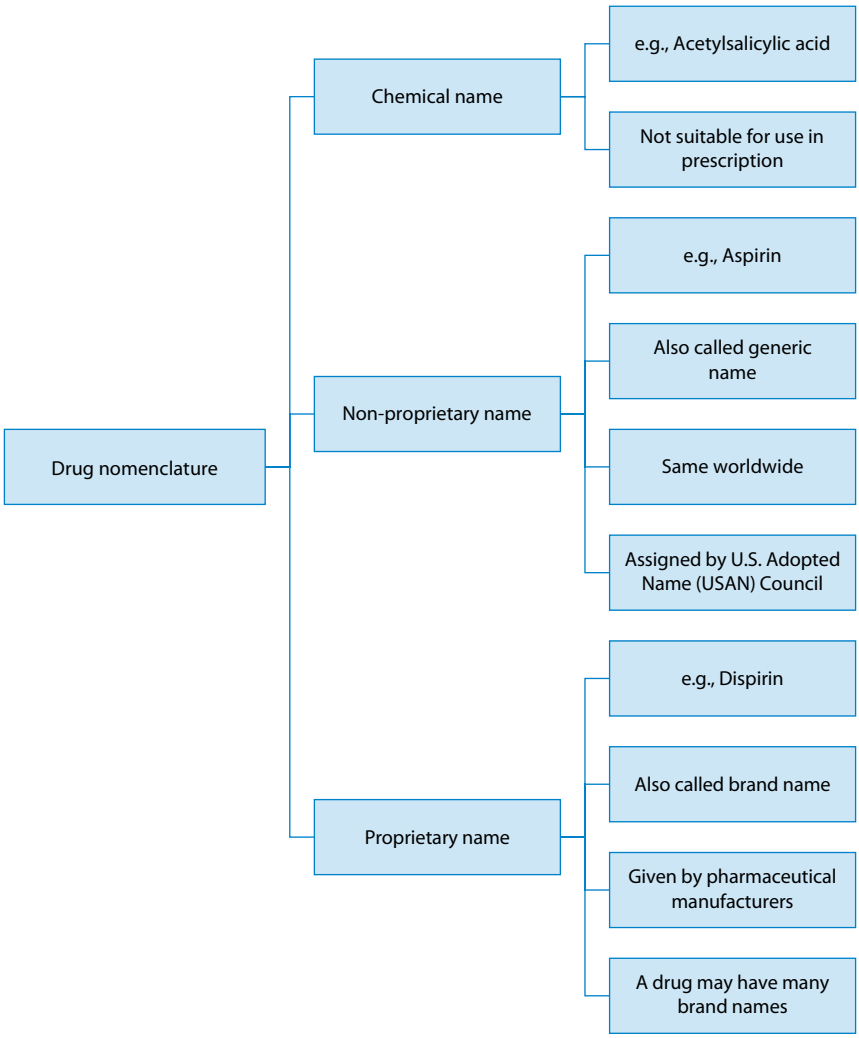
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Definitions, drug nomenclature, and sources of drugs

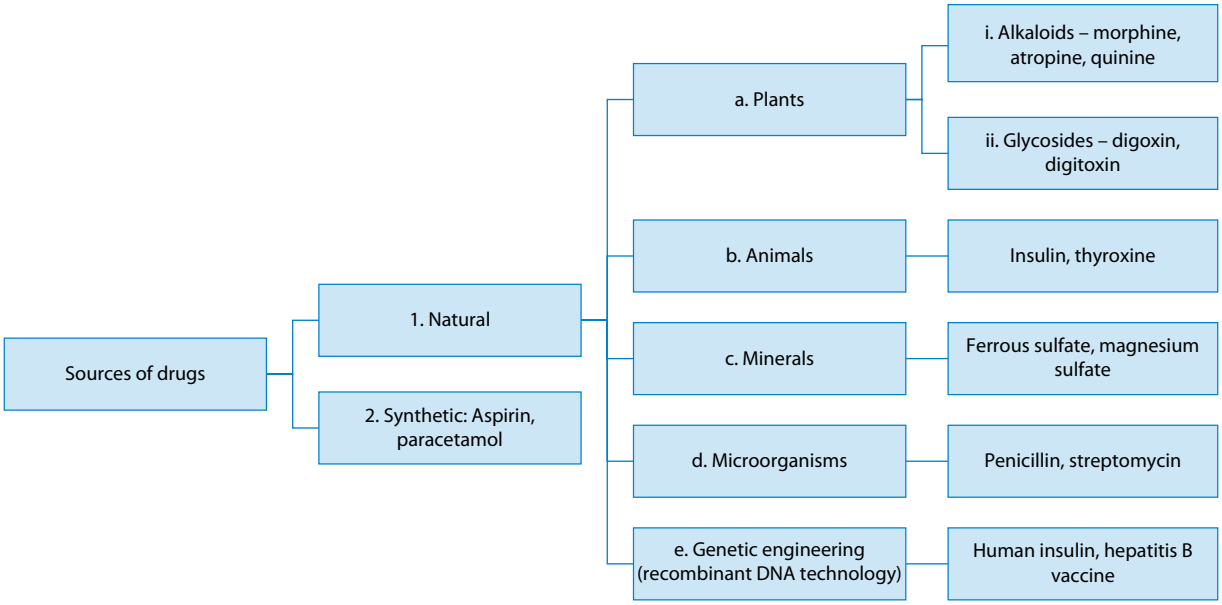
1.1 DEFINITIONS



1.2 DRUG NOMENCLATURE



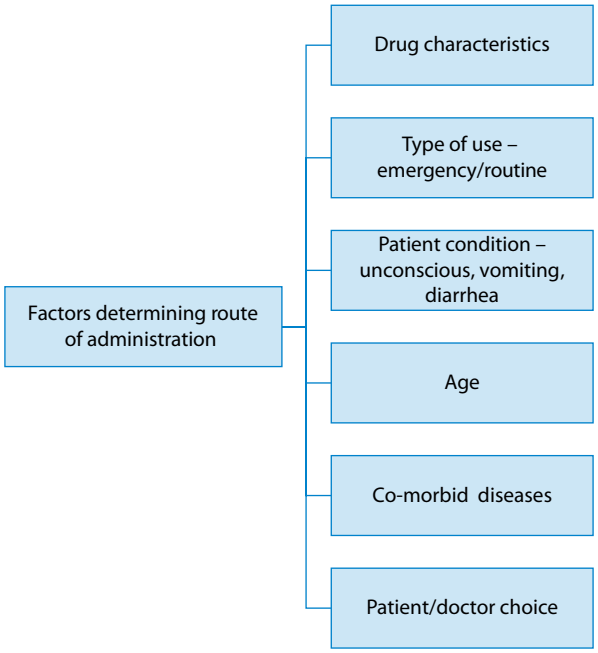
1.3 SOURCES OF DRUGS



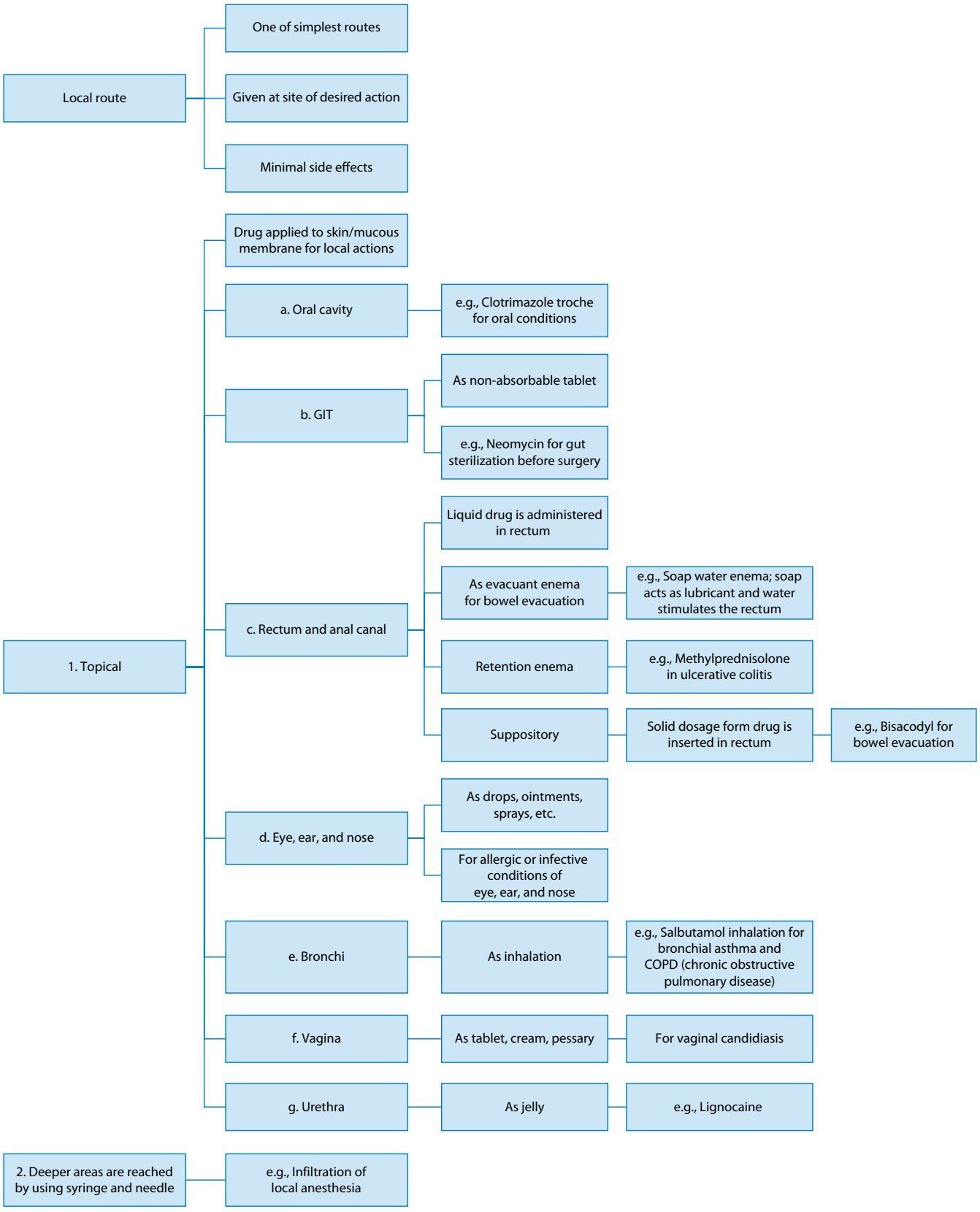
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Routes of drug administration

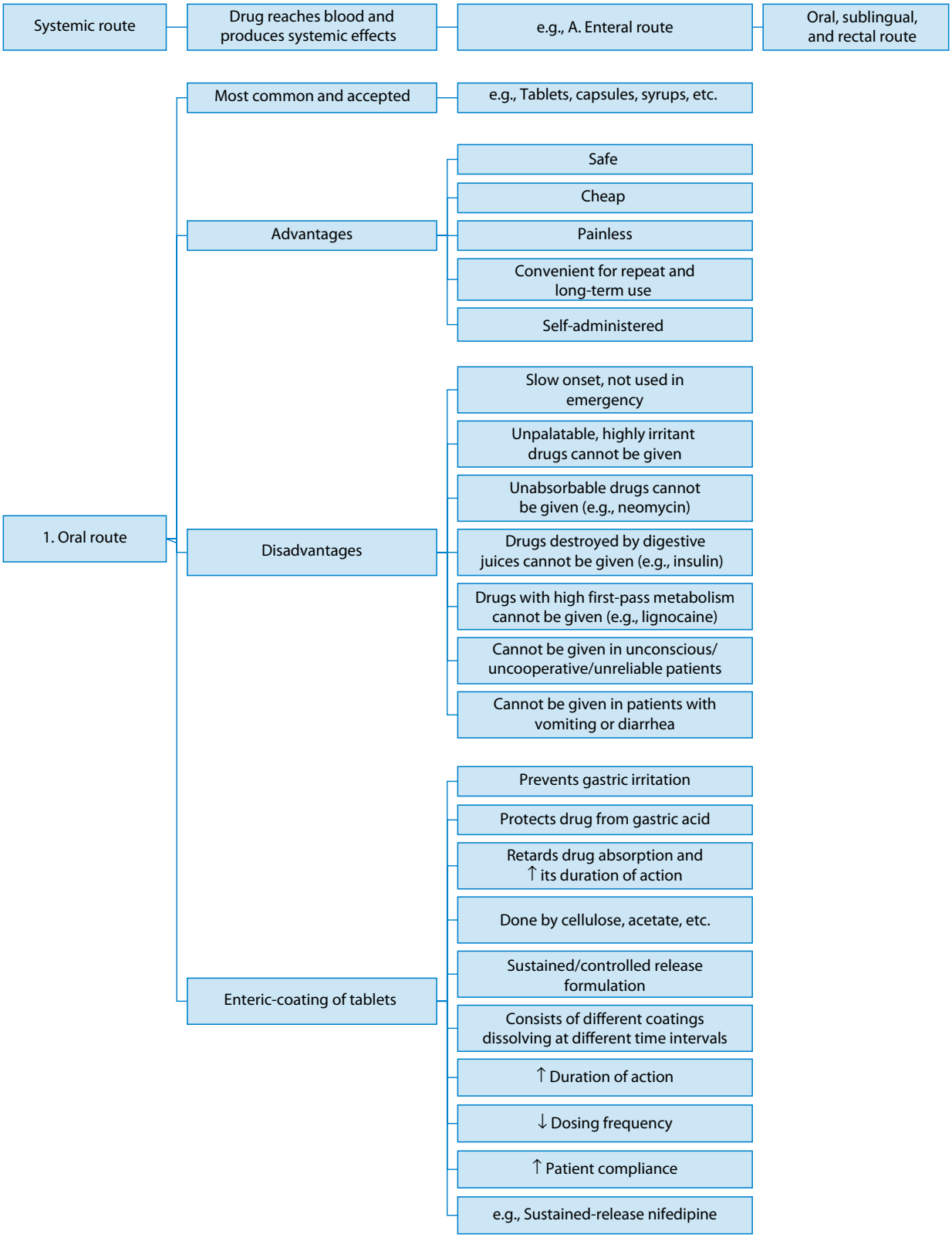
2.1 FACTORS DETERMINING ROUTES OF DRUG ADMINISTRATION



2.2 LOCAL ROUTE

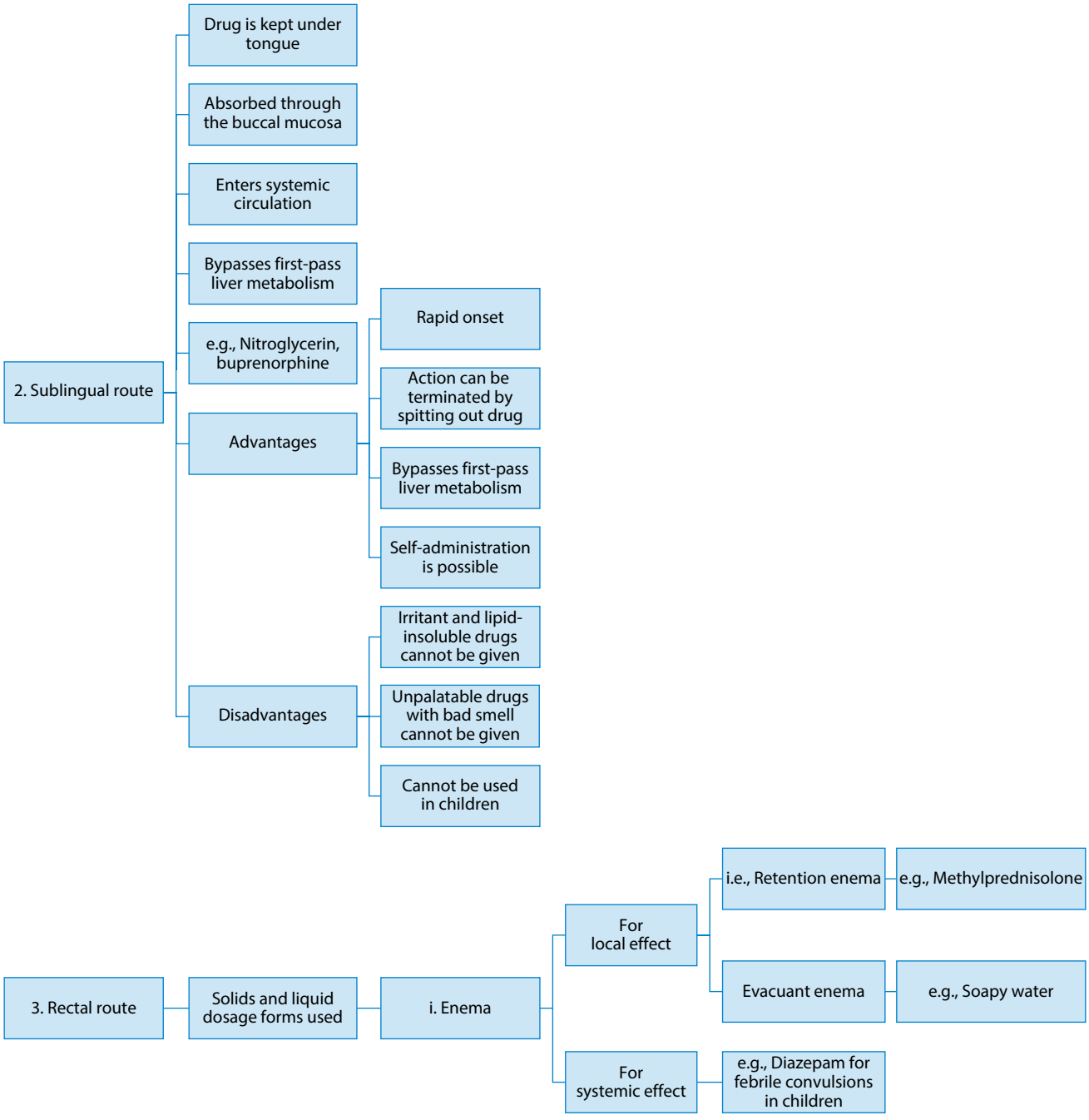


2.3 SYSTEMIC ROUTE



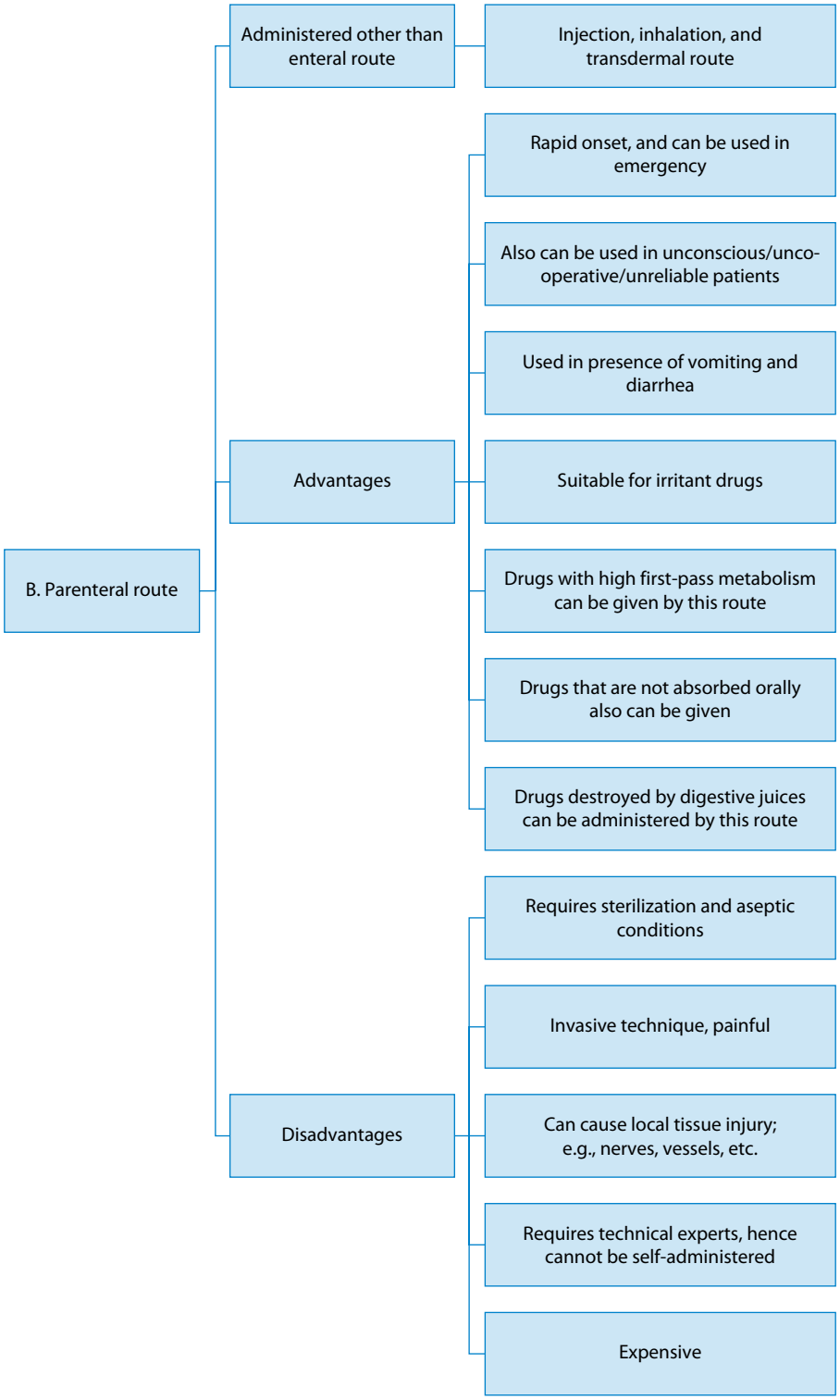
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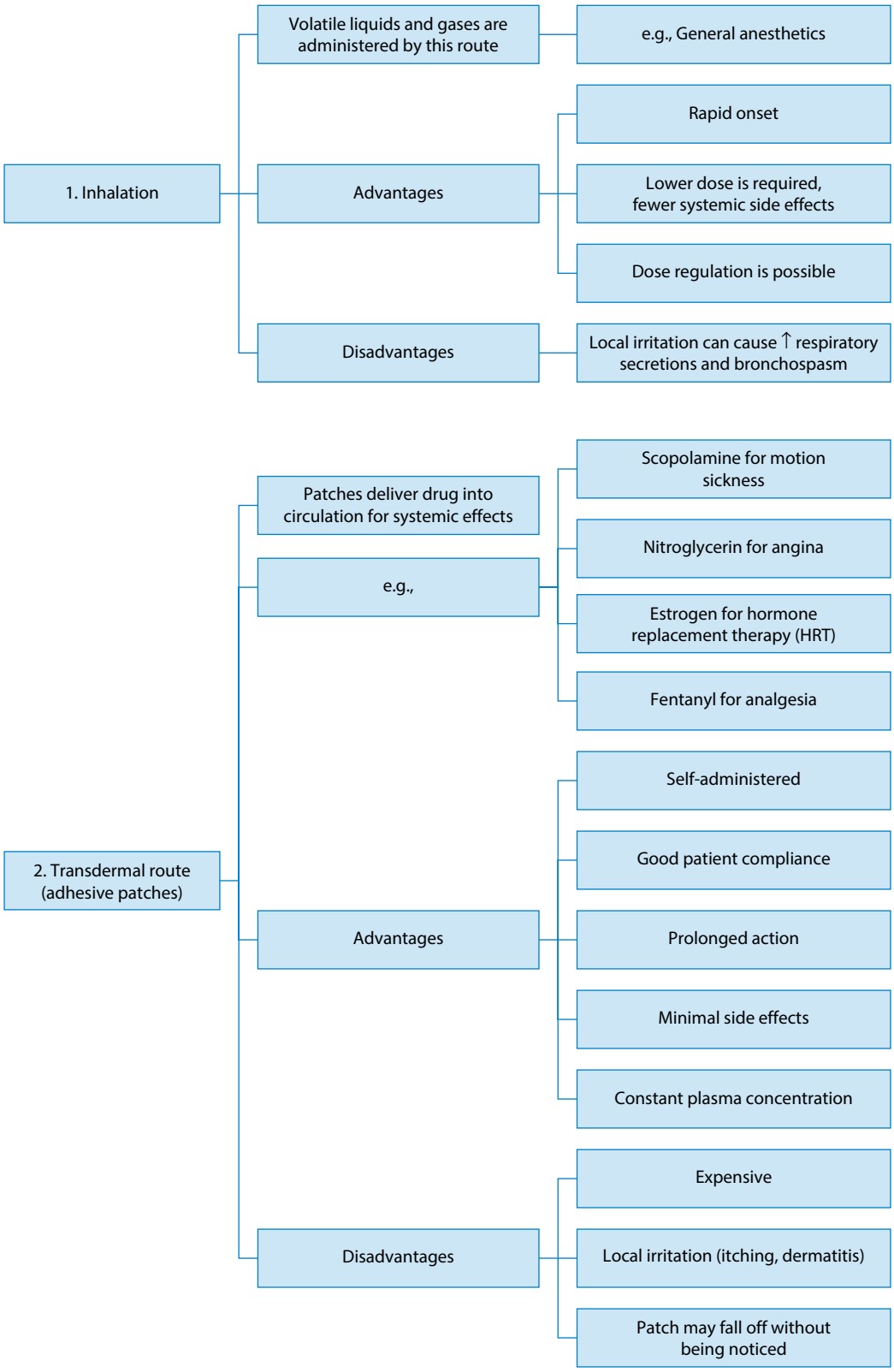
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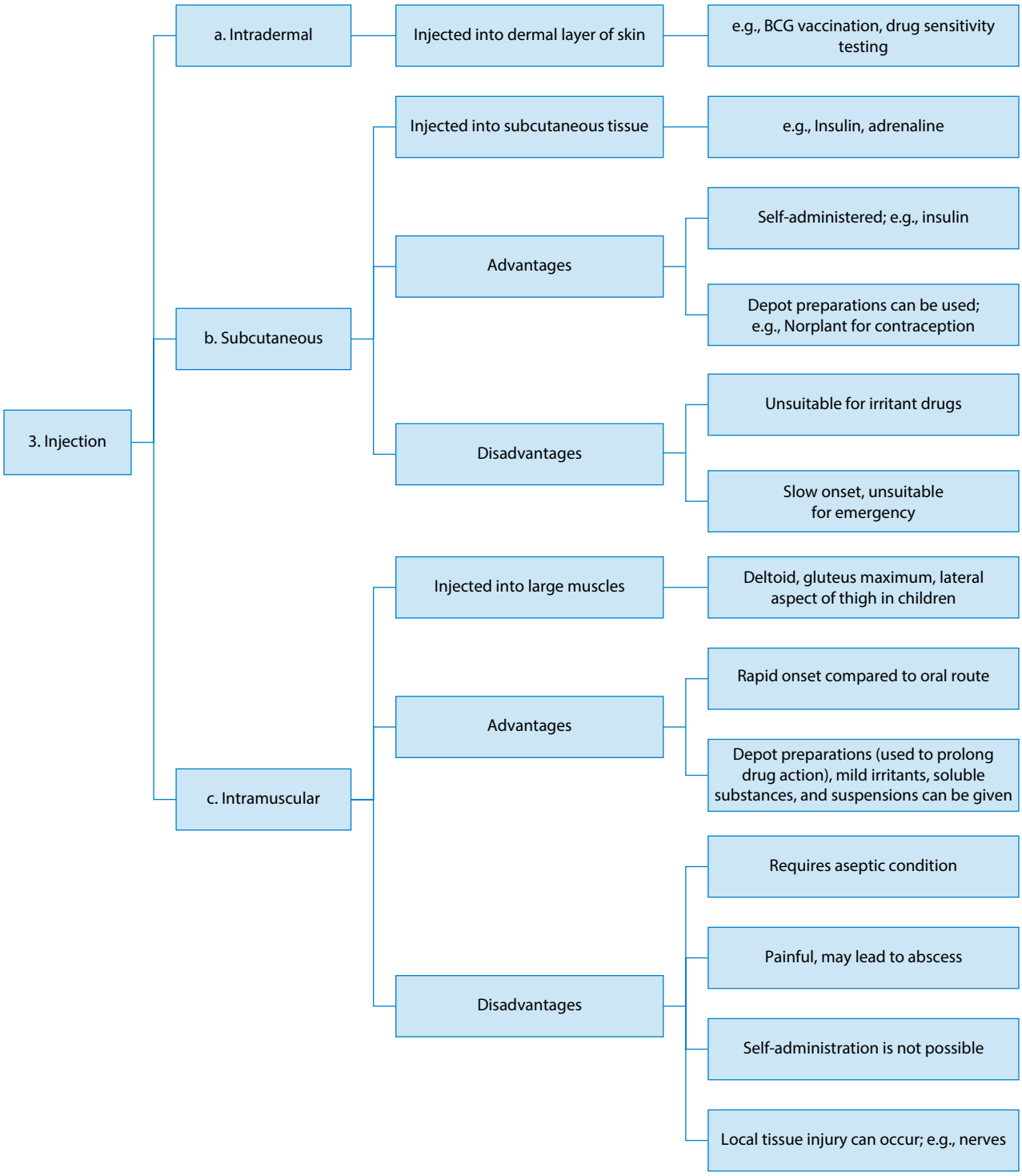
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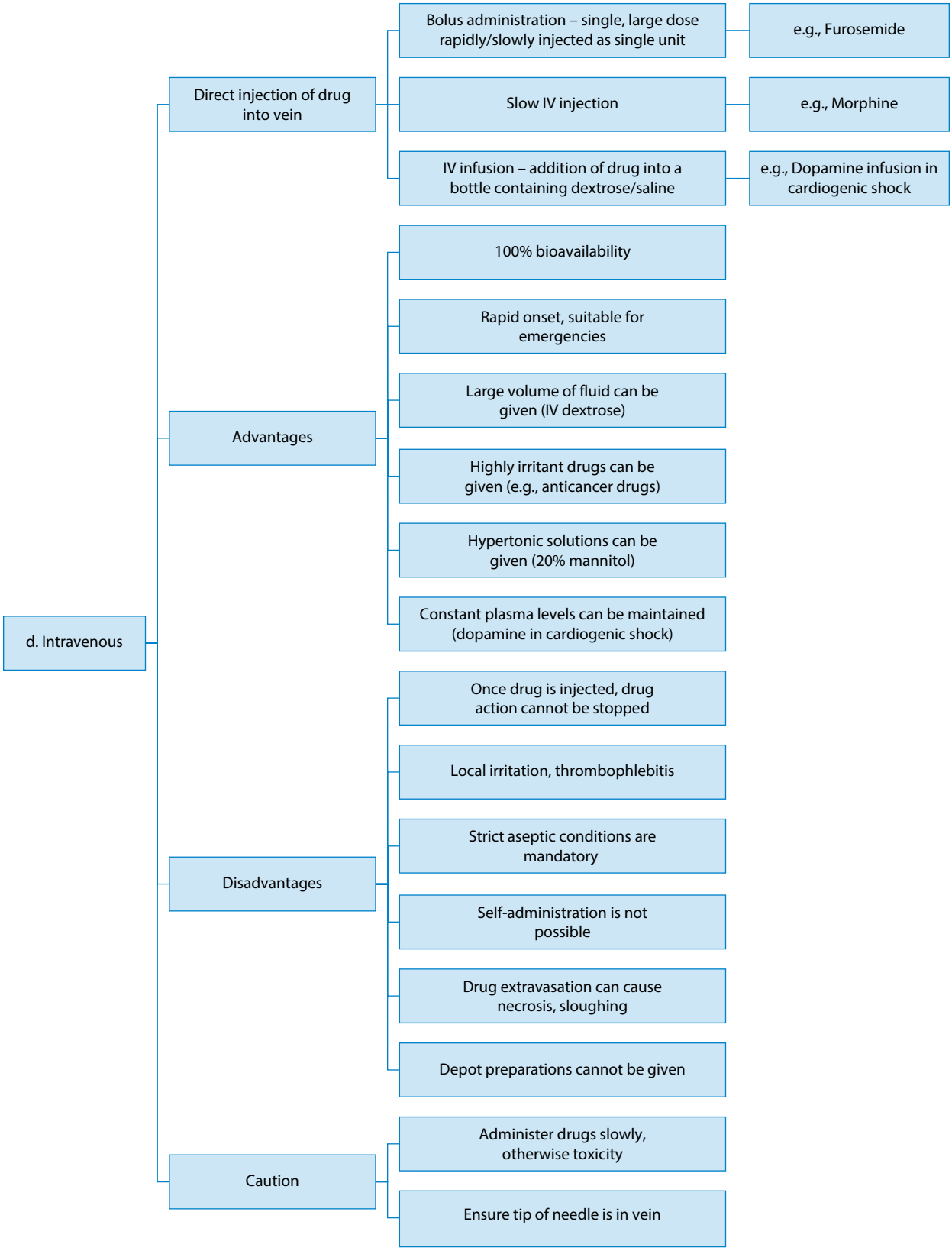
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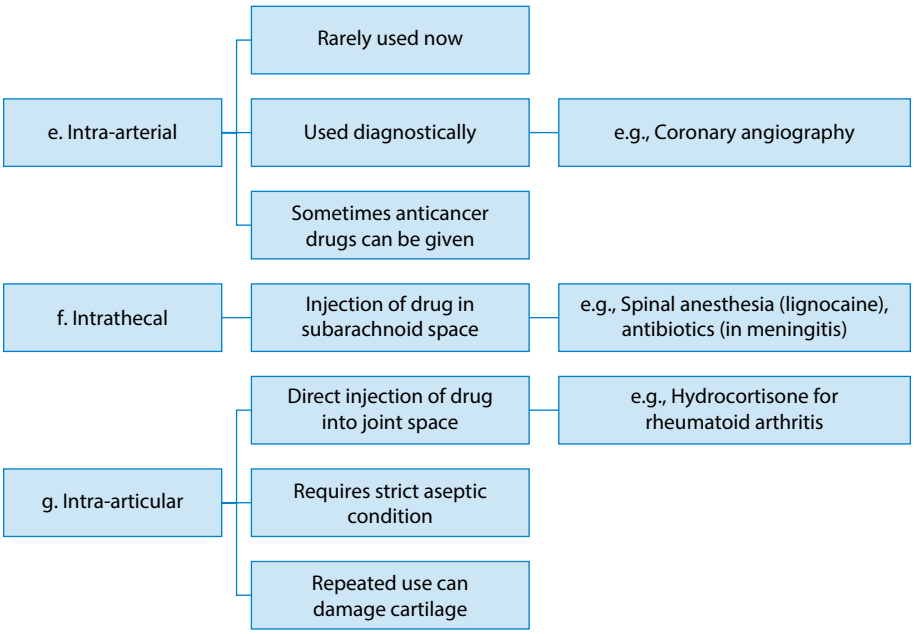
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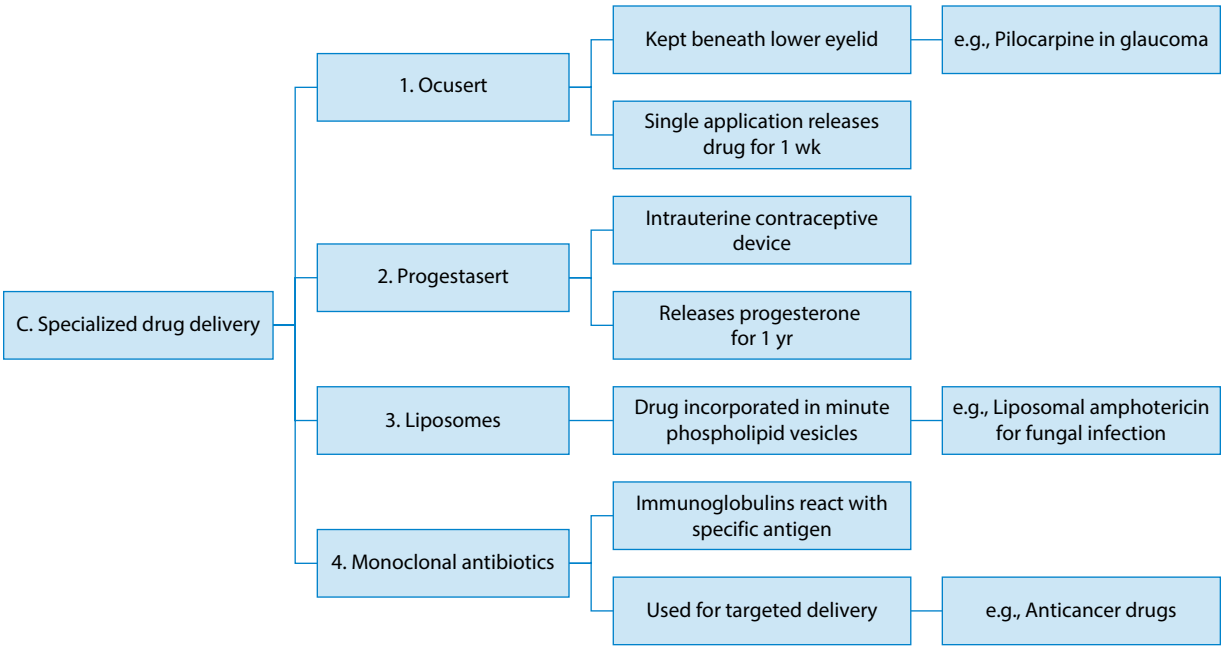


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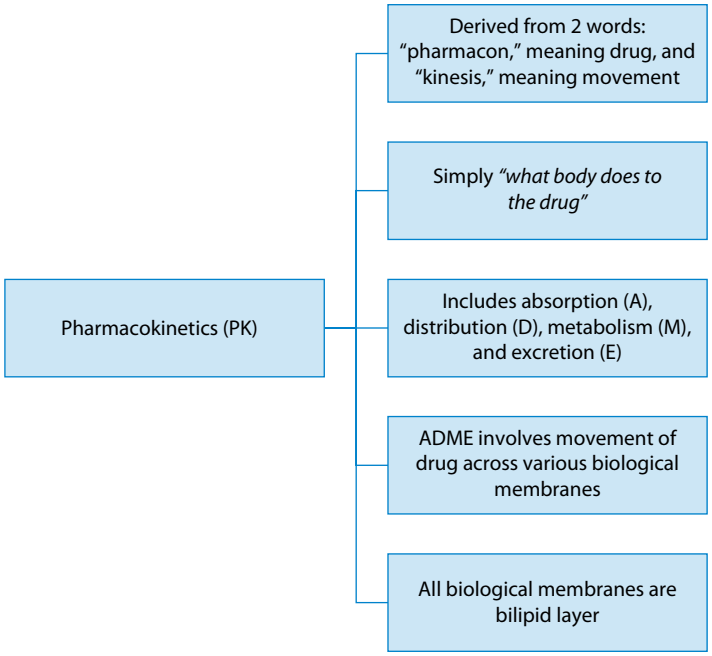
2.4 SPECIALIZED DRUG DELIVERY



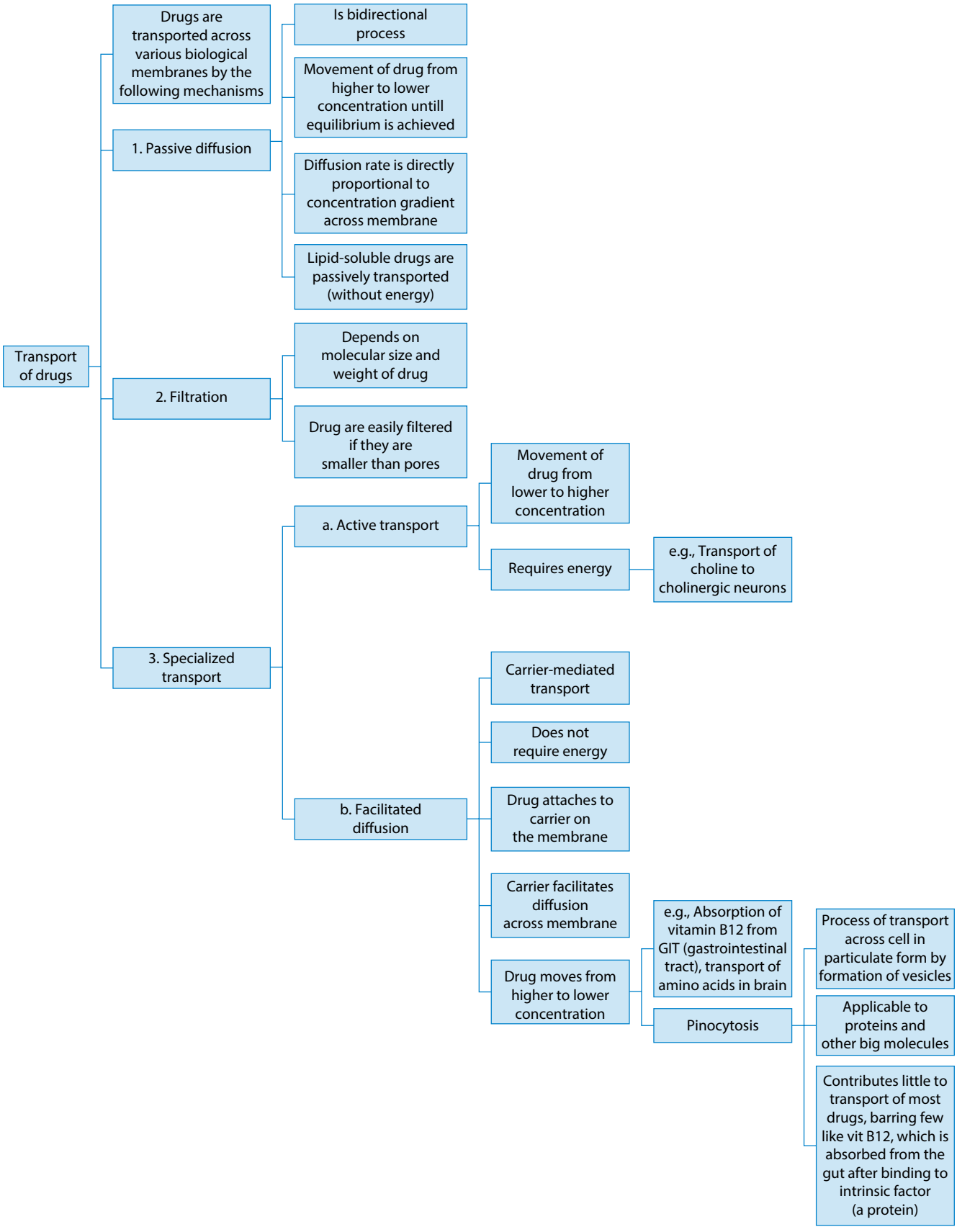
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Pharmacokinetics and applied aspects

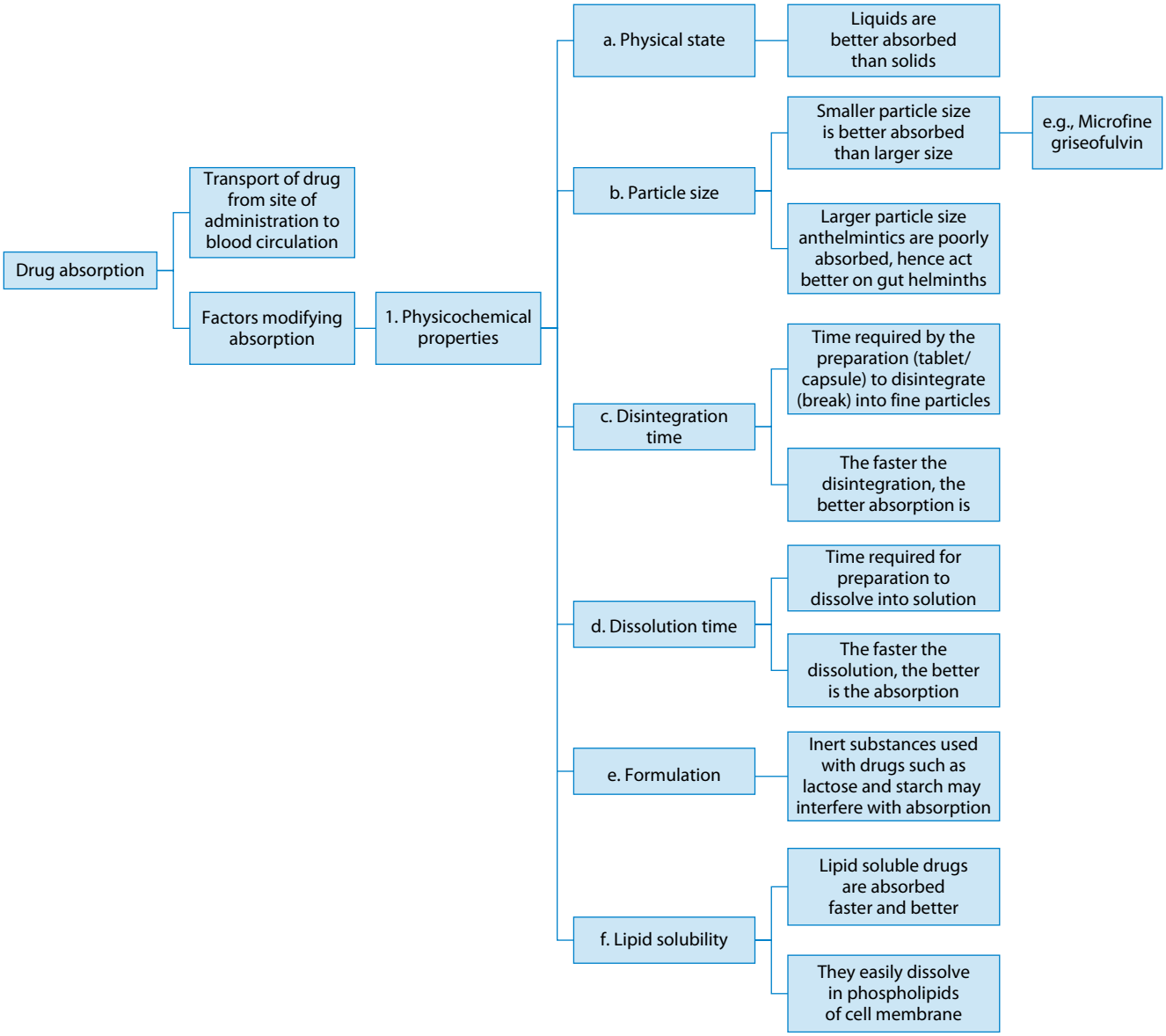
3.1 INTRODUCTION TO PHARMACOKINETICS



3.2 TRANSPORT OF DRUGS

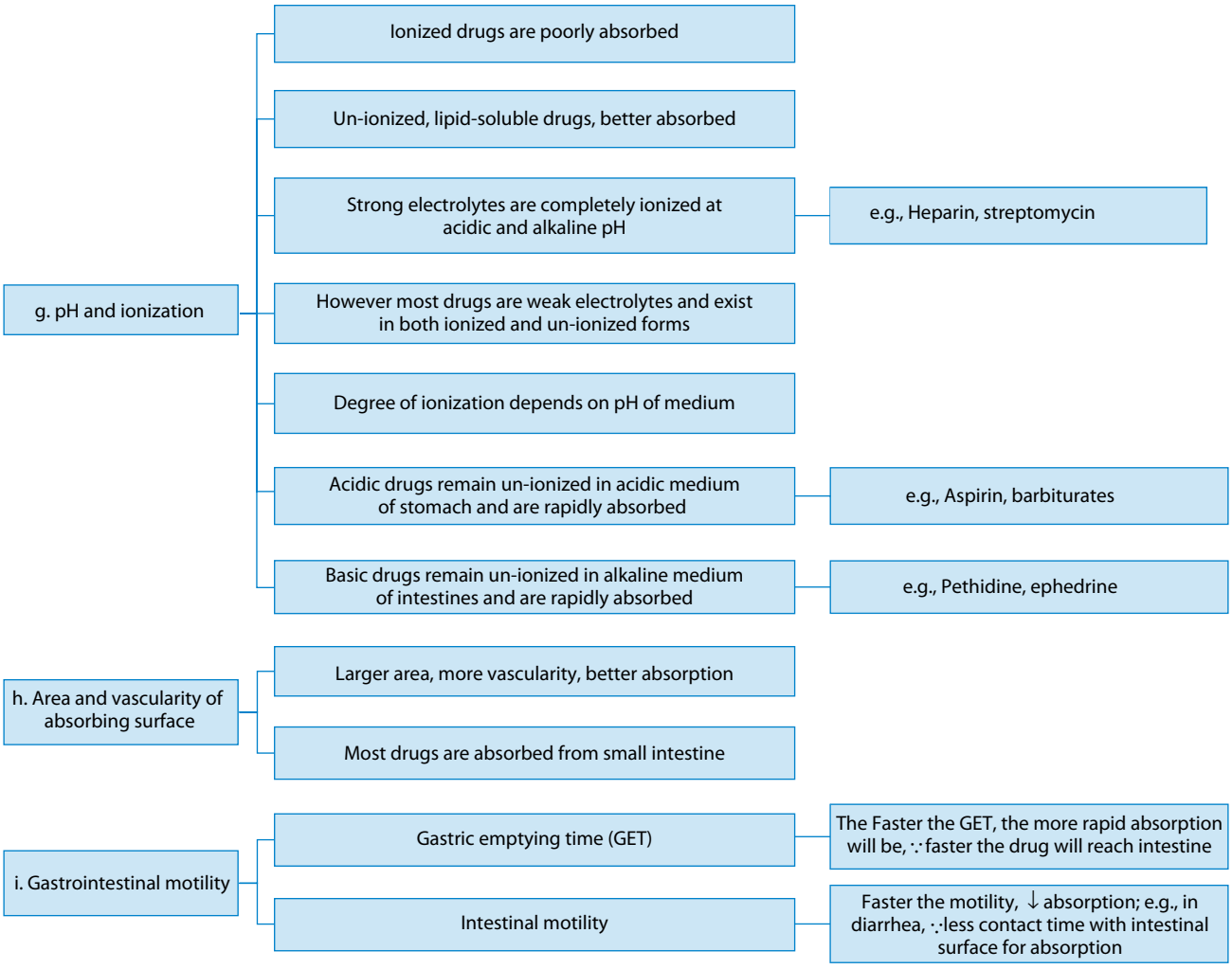


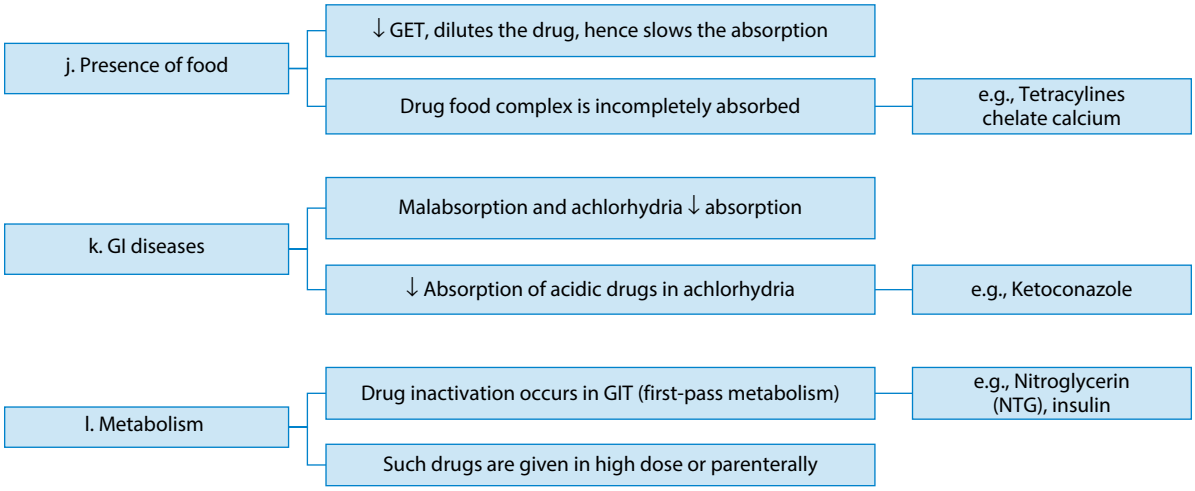
3.3 DRUG ABSORPTION



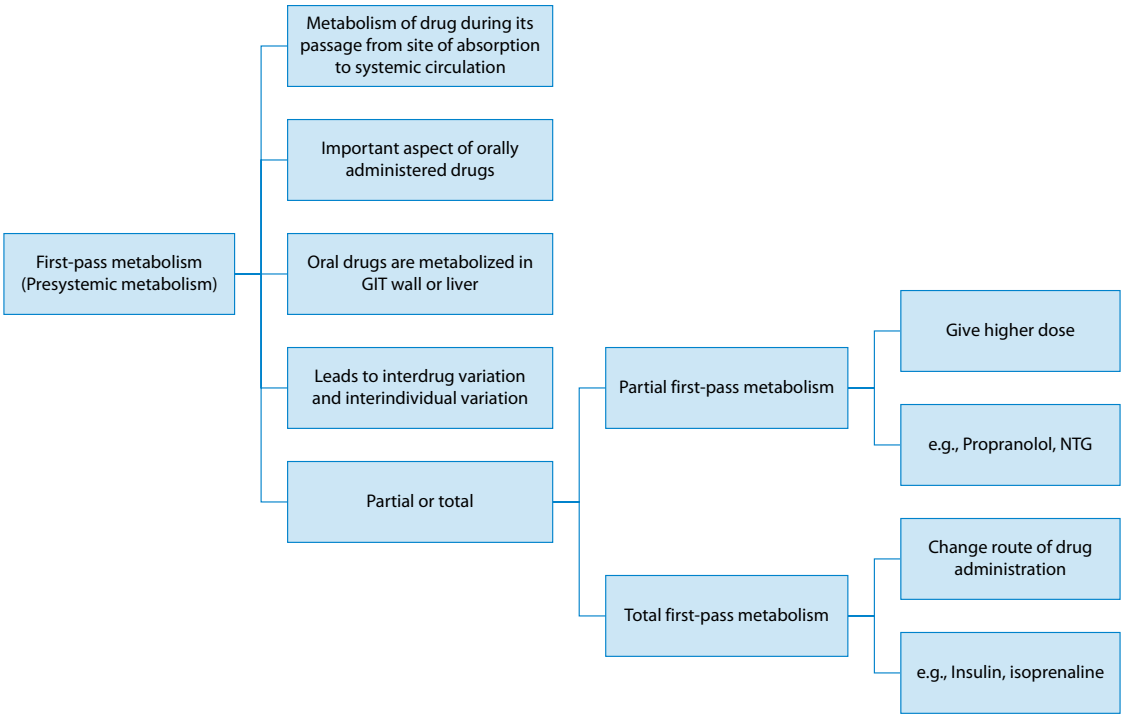
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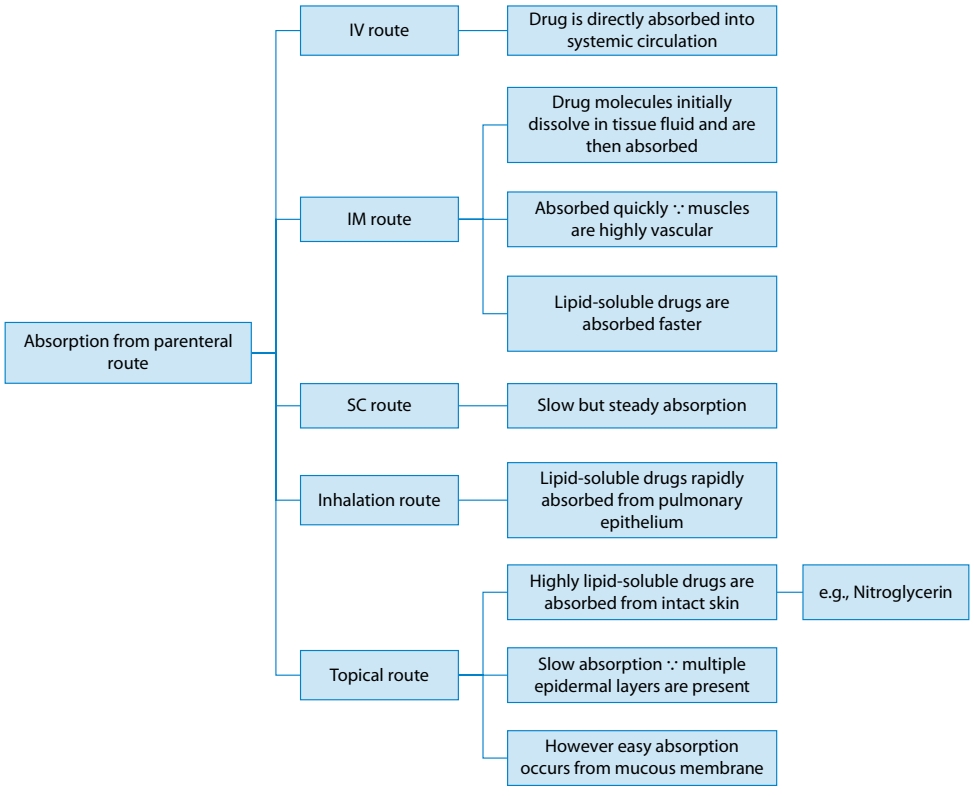




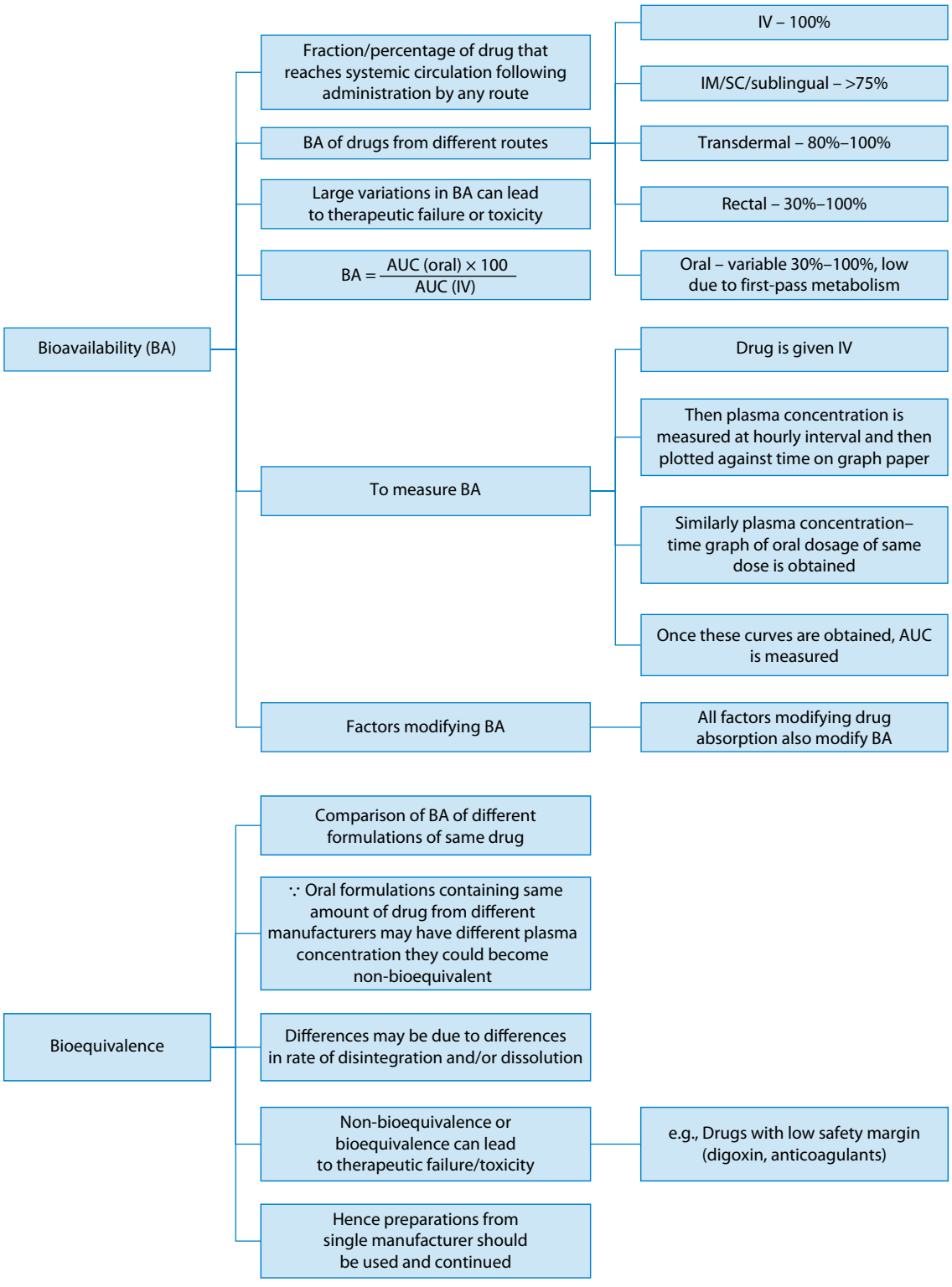
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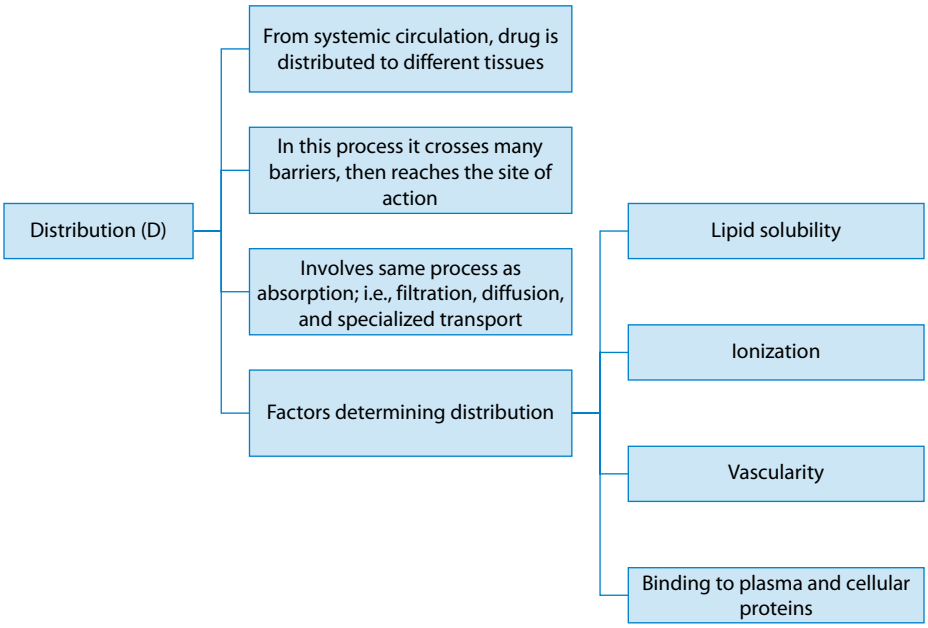
3.5 ABSORPTION FROM PARENTERAL ROUTE



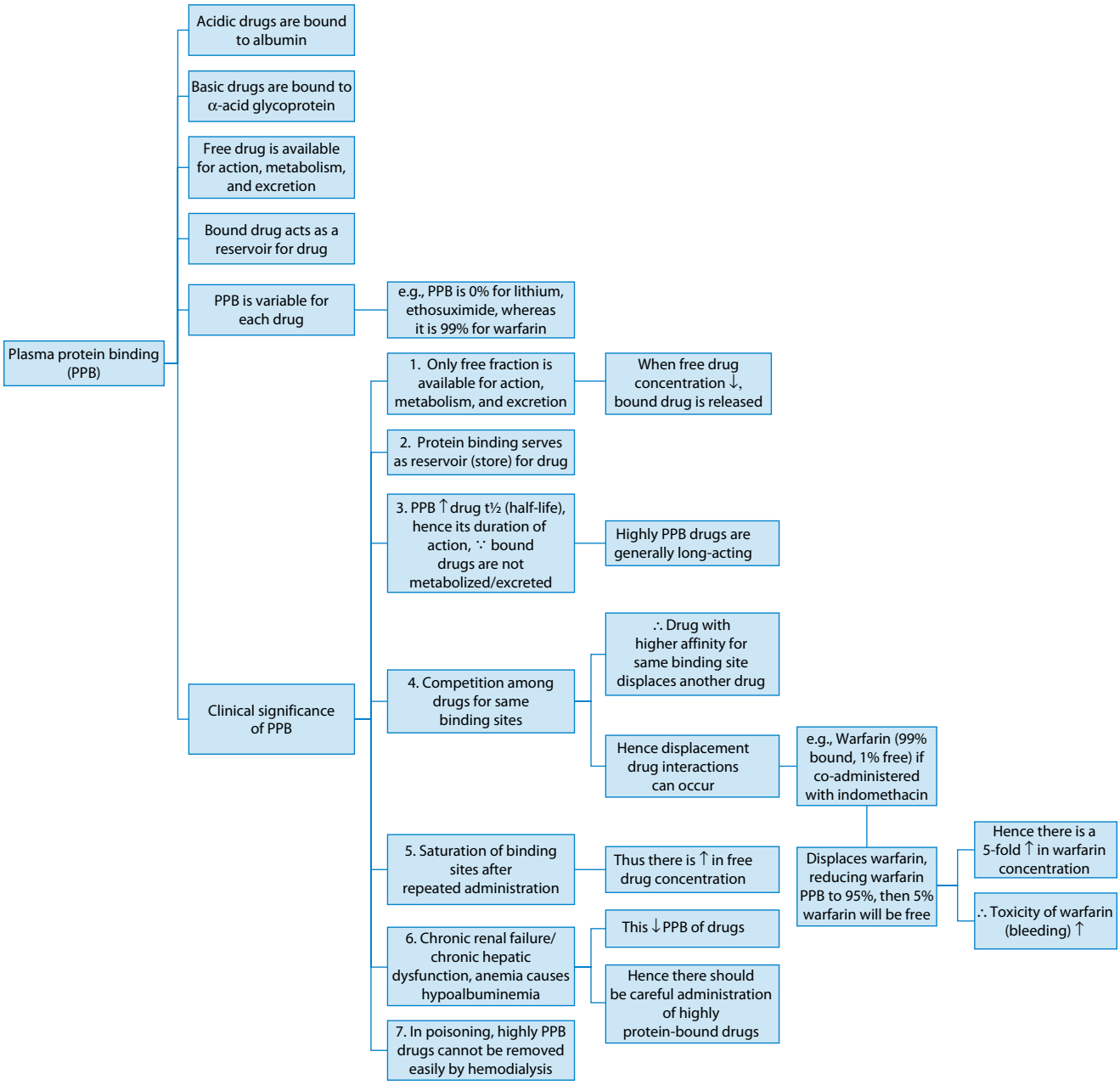
3.6 BIOAVAILABILITY AND BIOEQUIVALENCE



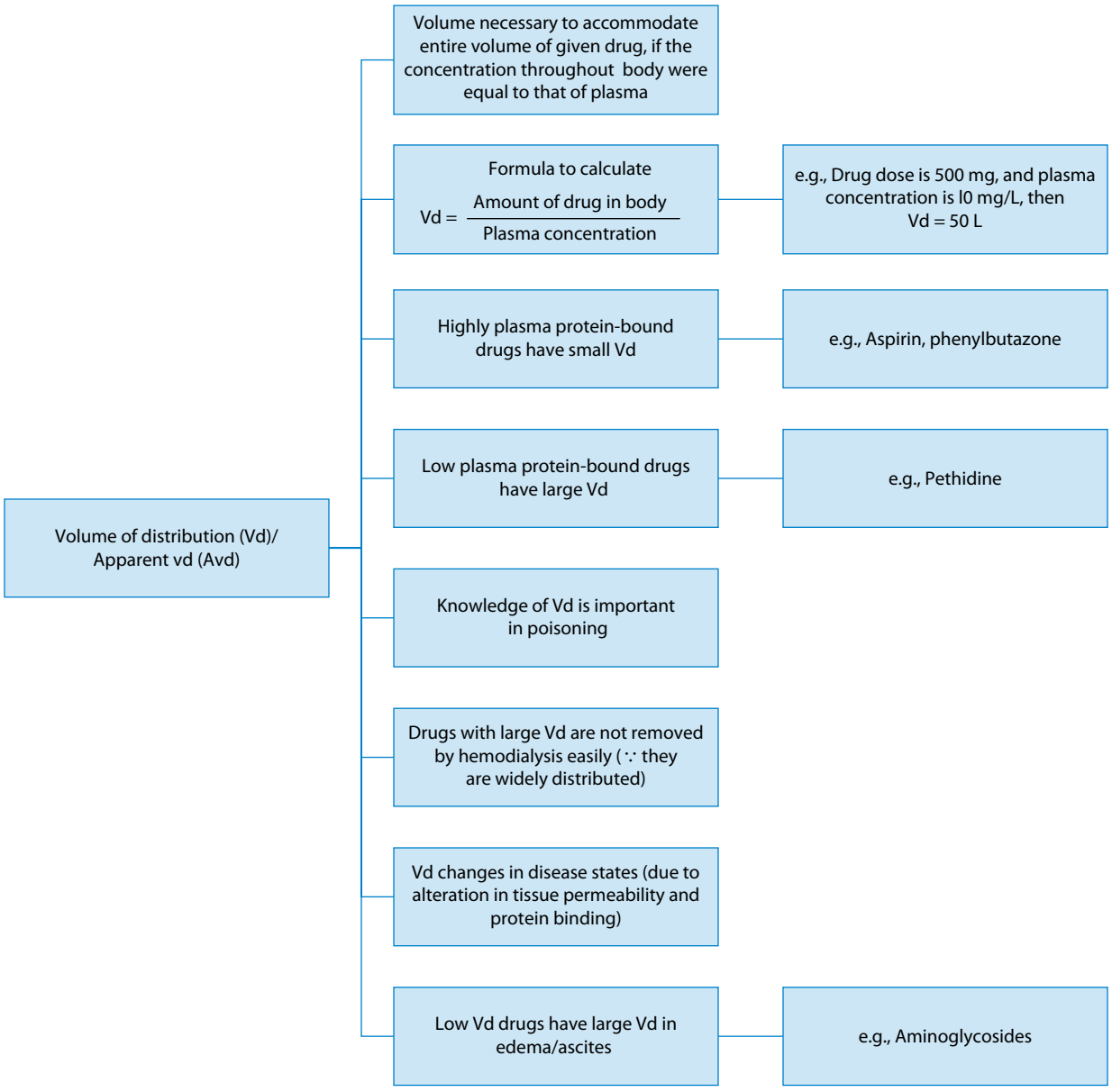
3.7 DRUG DISTRIBUTION



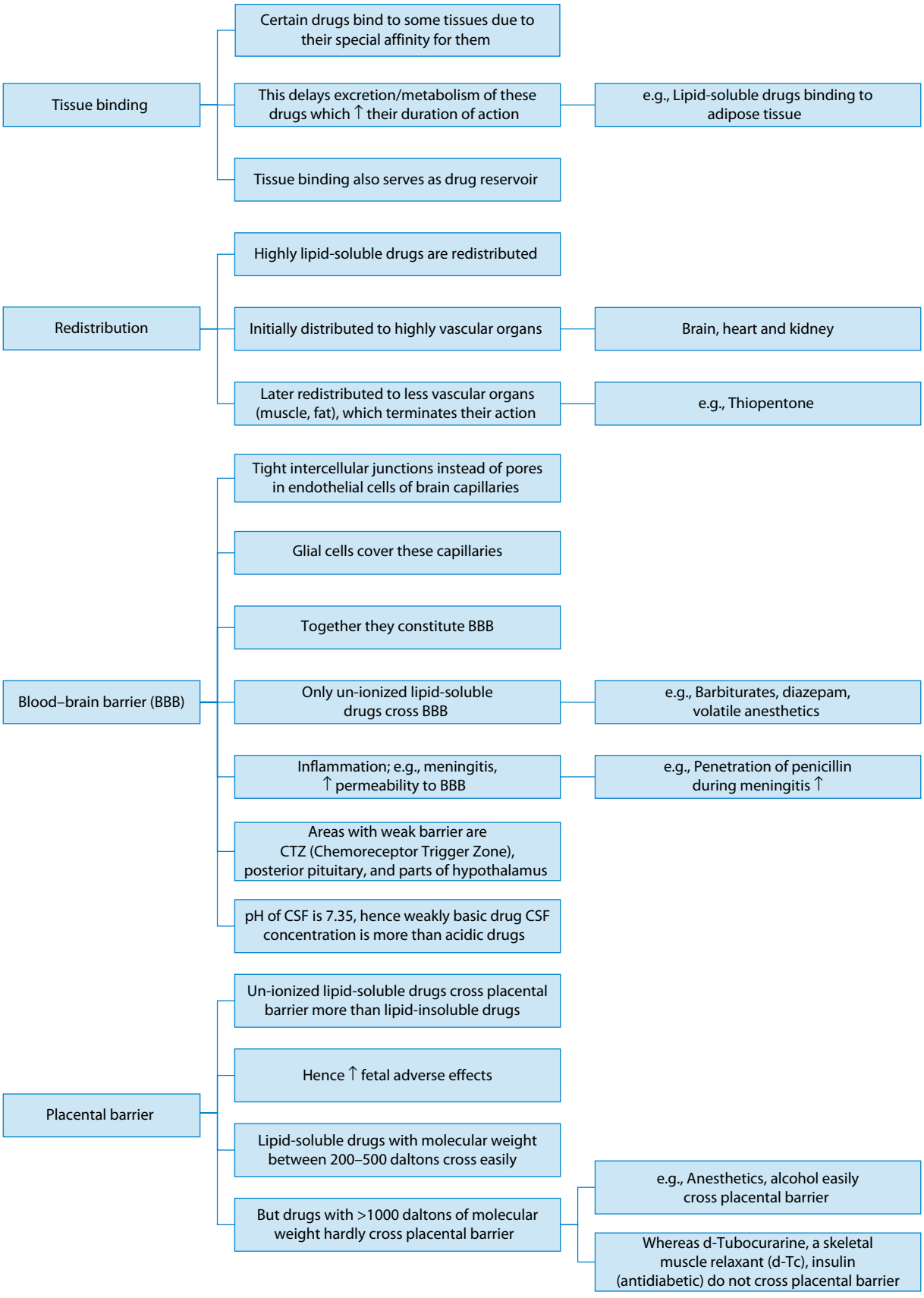
3.8 PLASMA PROTEIN BINDING



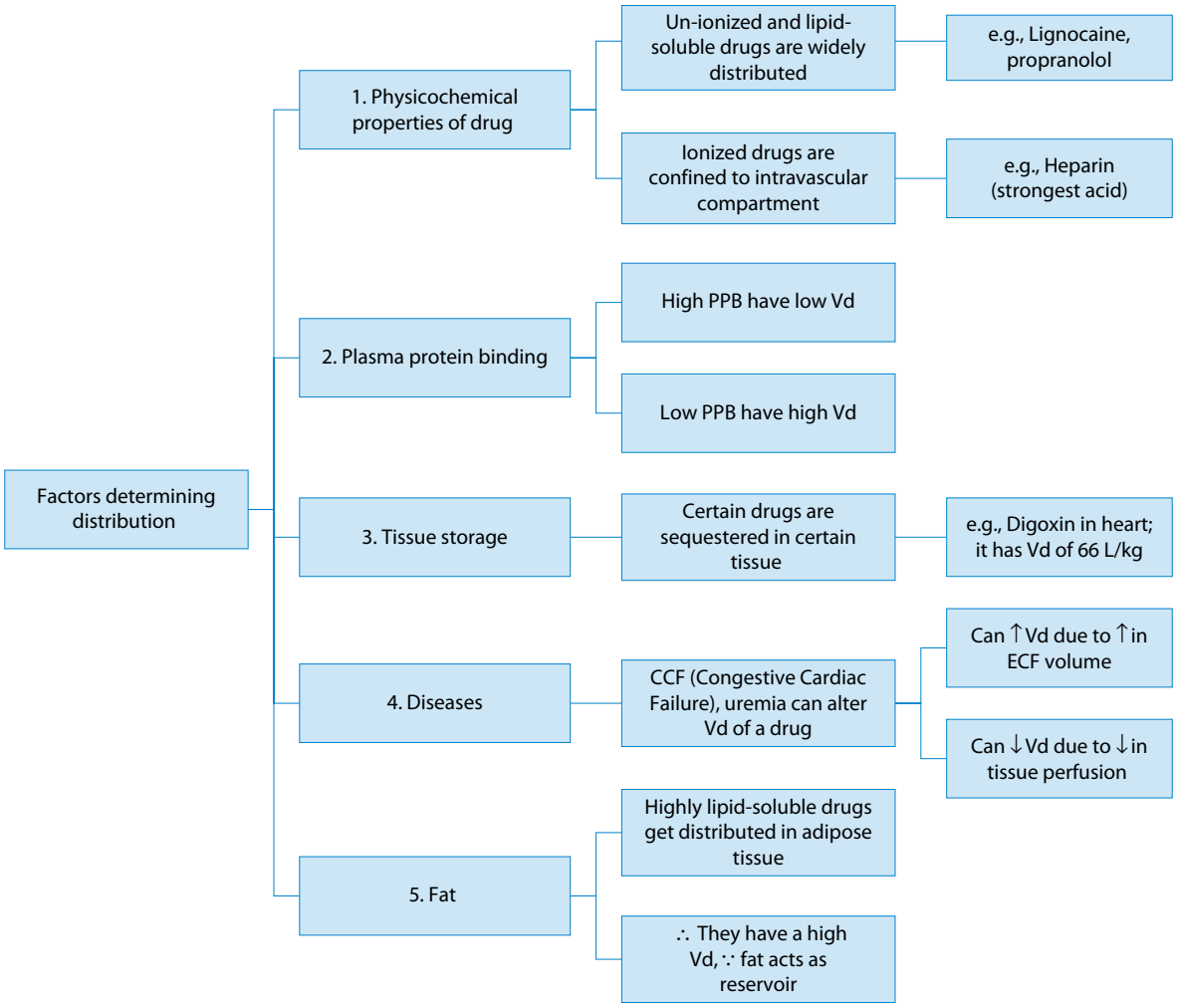
3.9 VOLUME OF DISTRIBUTION



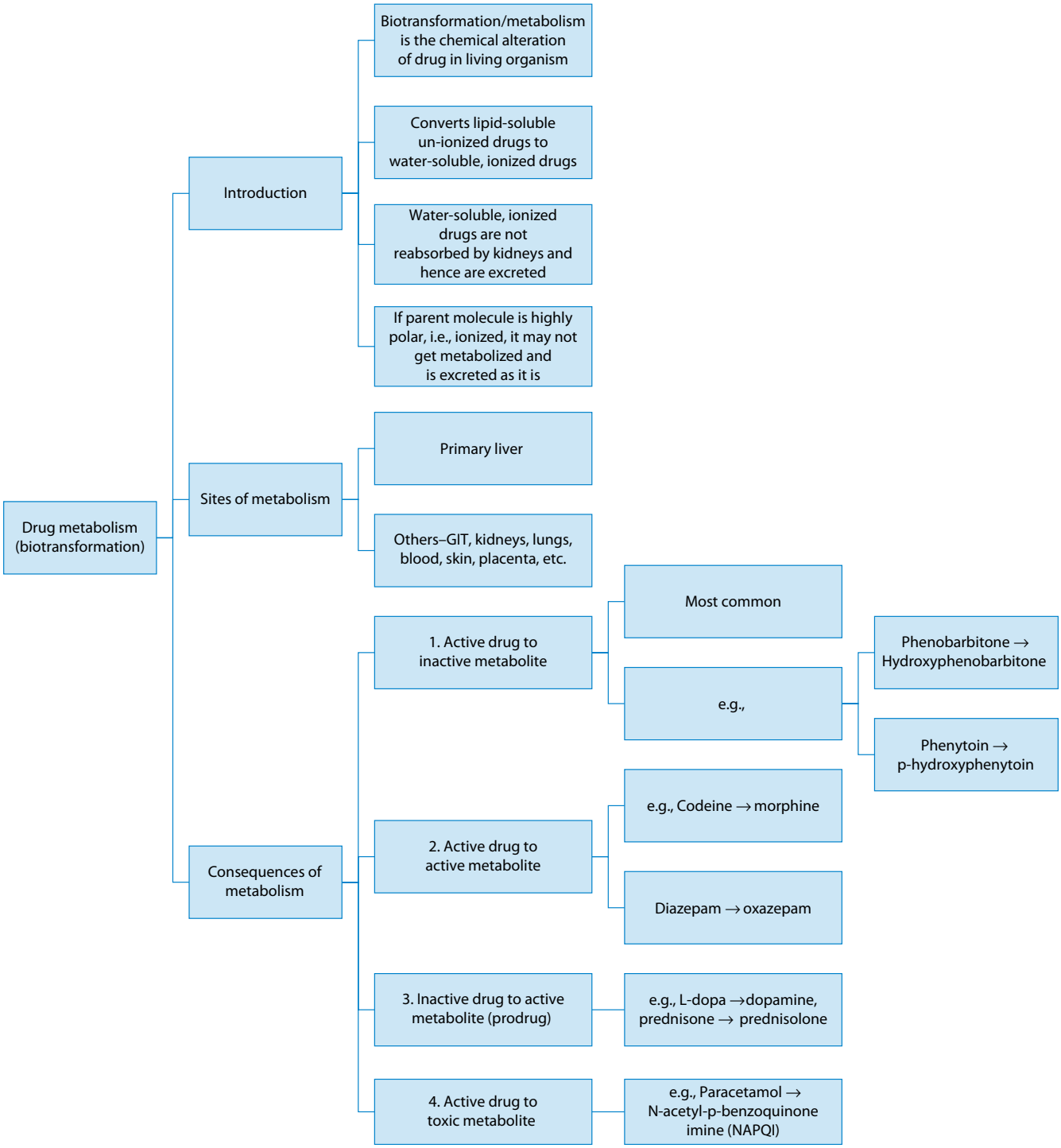
3.10 REDISTRIBUTION, BLOOD–BRAIN BARRIER, TISSUE BINDING, PLACENTAL BARRIER



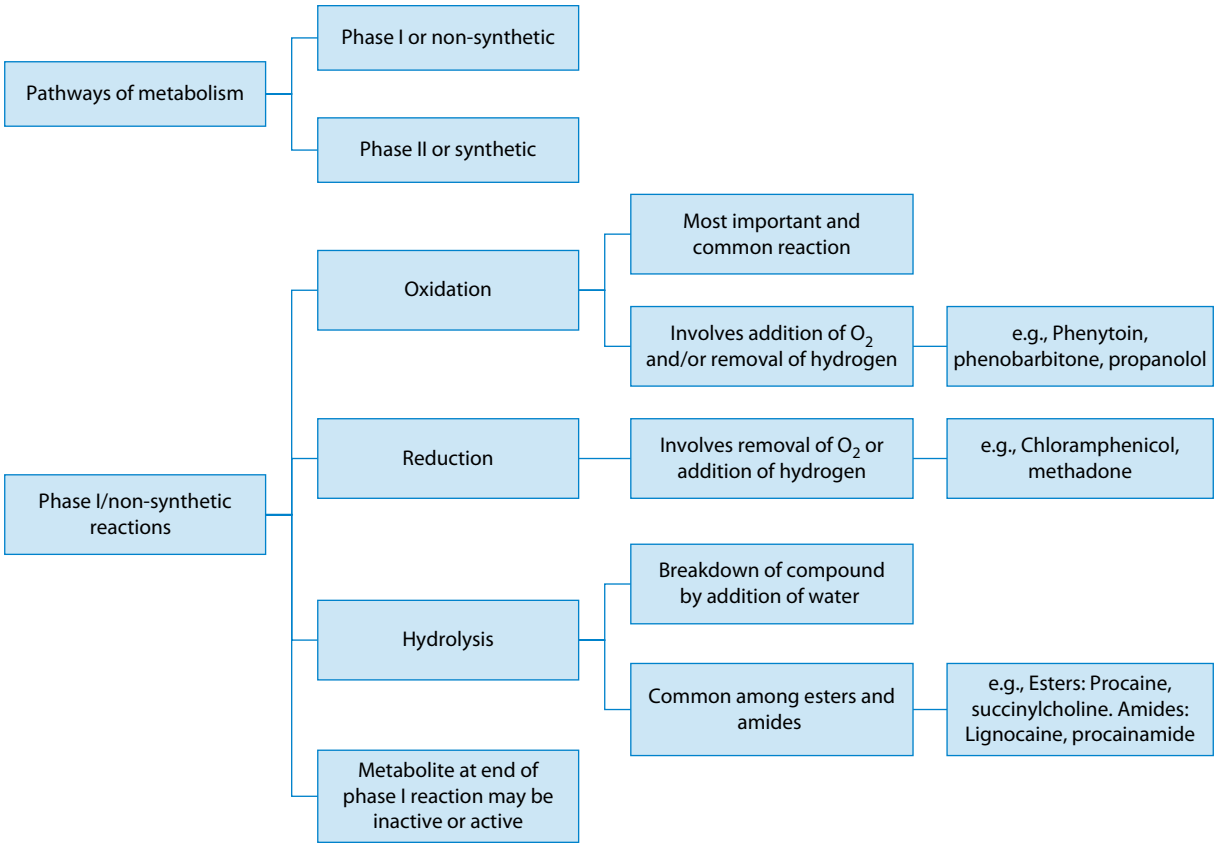
3.11 FACTORS DETERMINING DISTRIBUTION



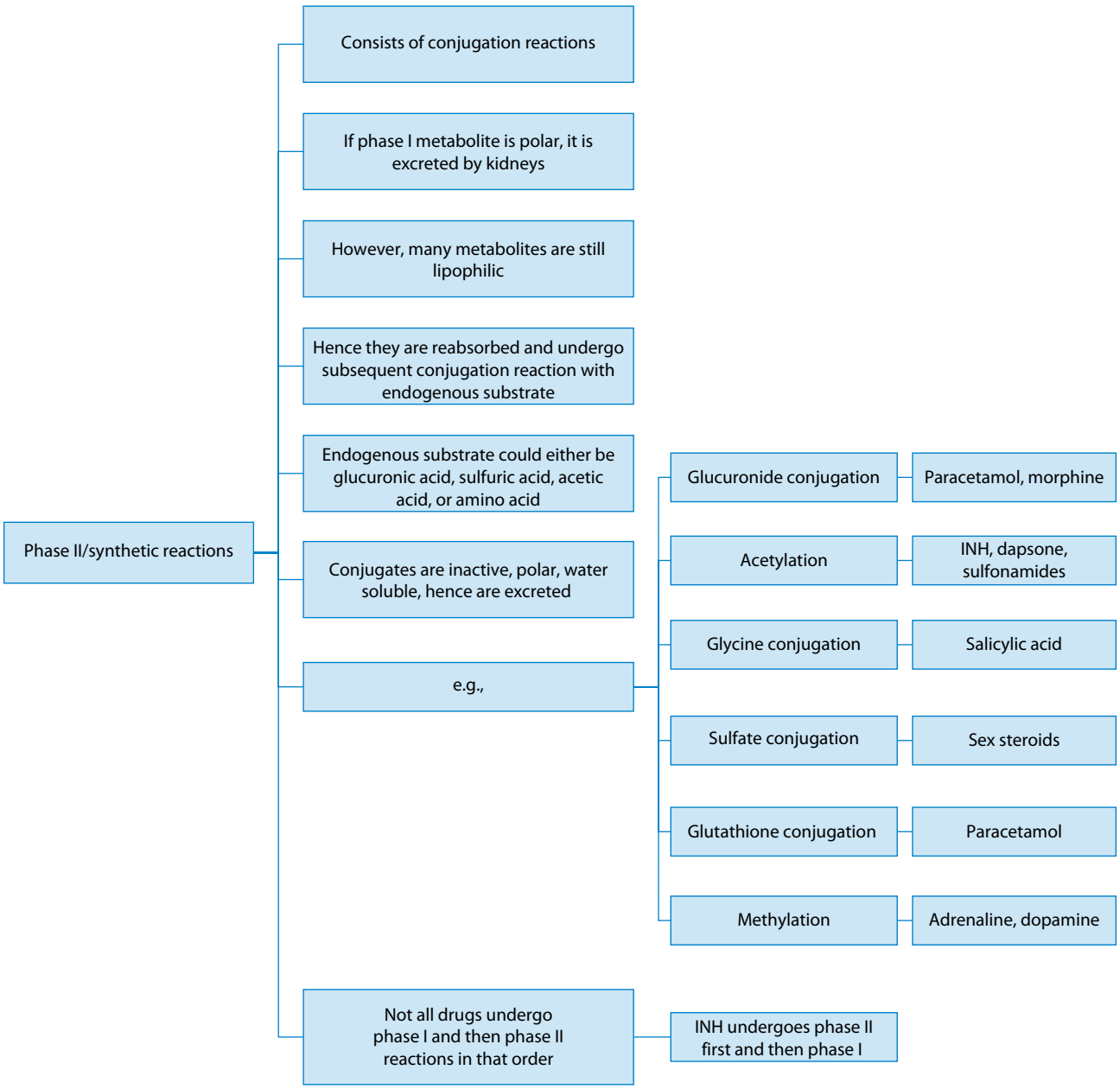
3.12 DRUG METABOLISM (BIOTRANSFORMATION)



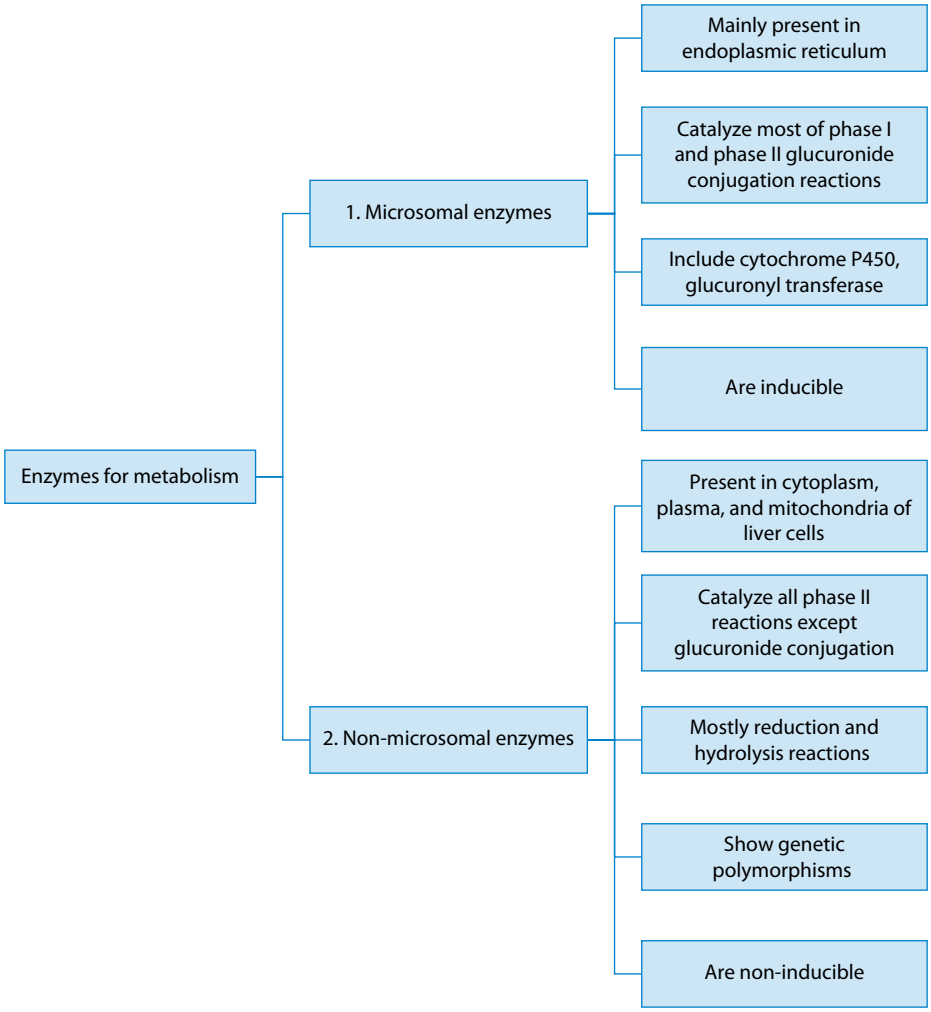
3.13 PATHWAYS OF METABOLISM AND PHASE I REACTIONS



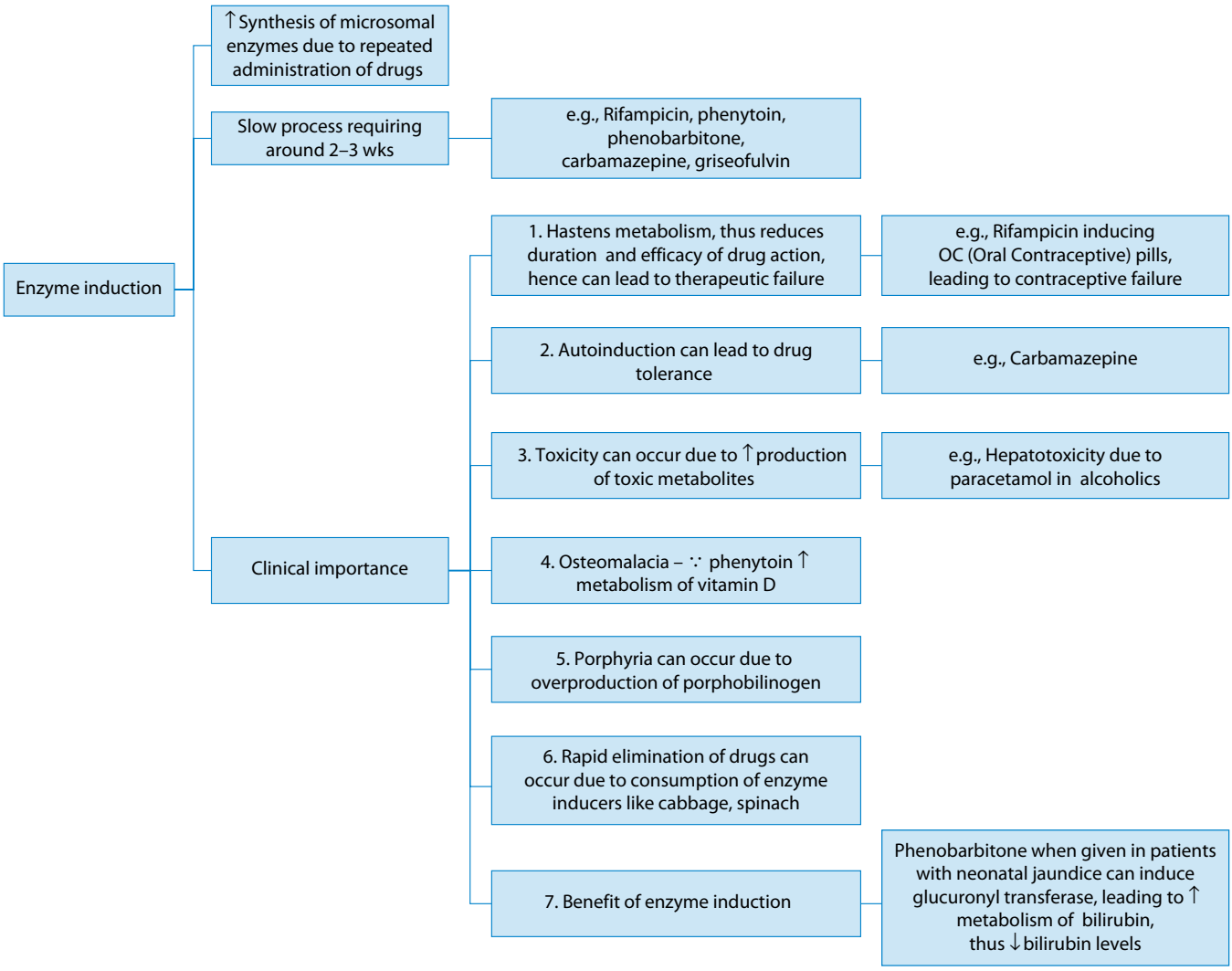
3.14 PHASE II/SYNTHETIC REACTIONS



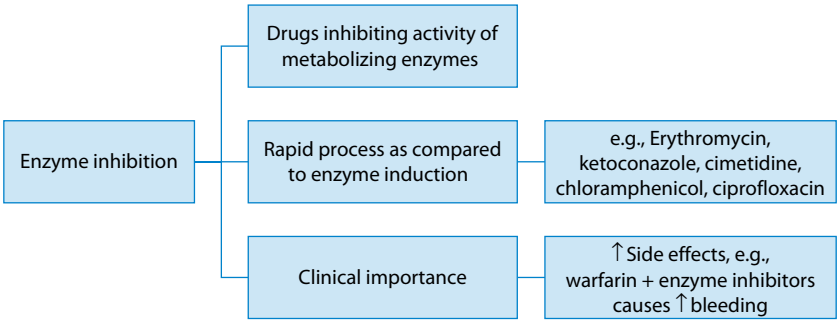
3.15 ENZYMES FOR METABOLISM



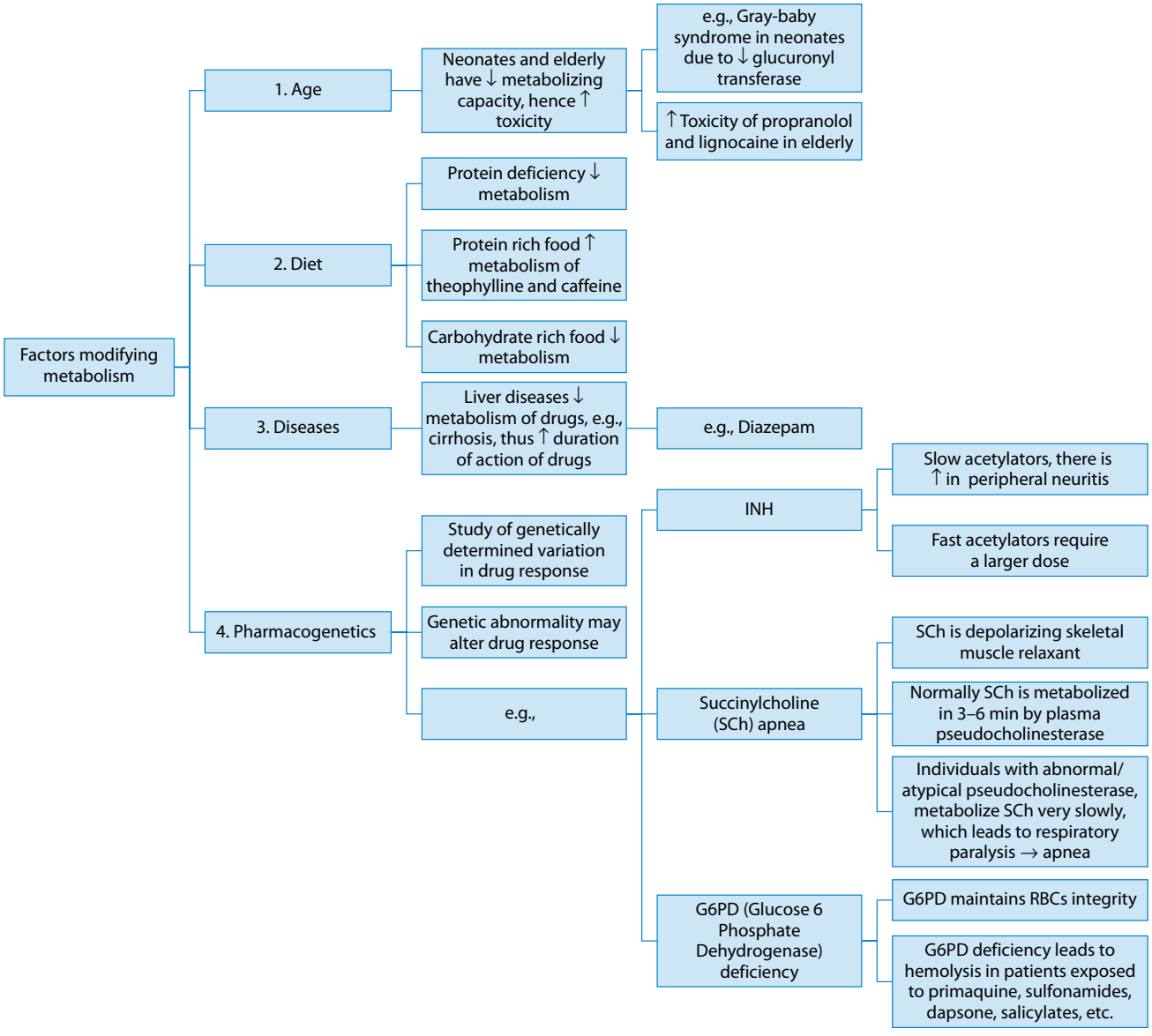
3.16 ENZYME INDUCTION



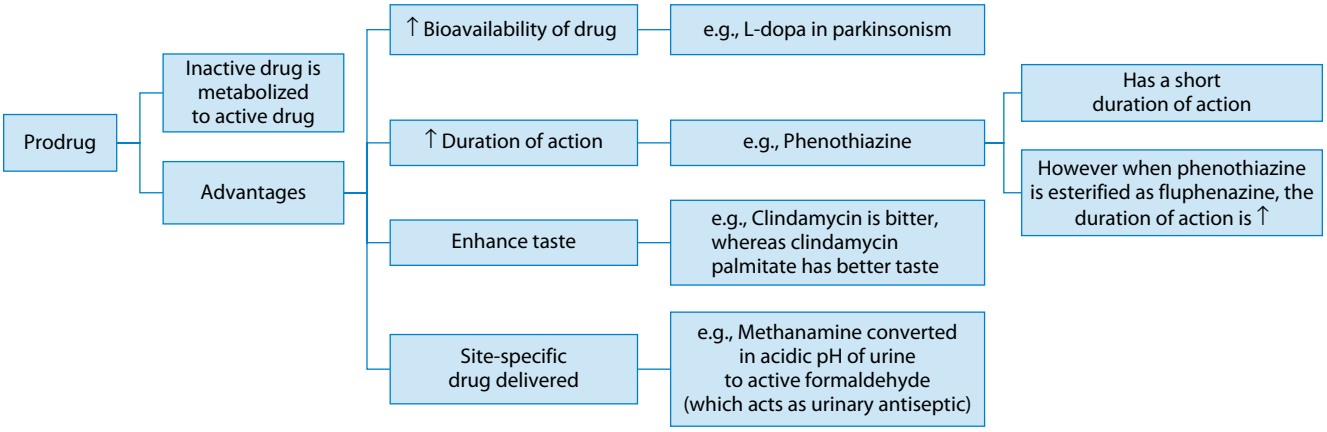
3.17 ENZYME INHIBITION



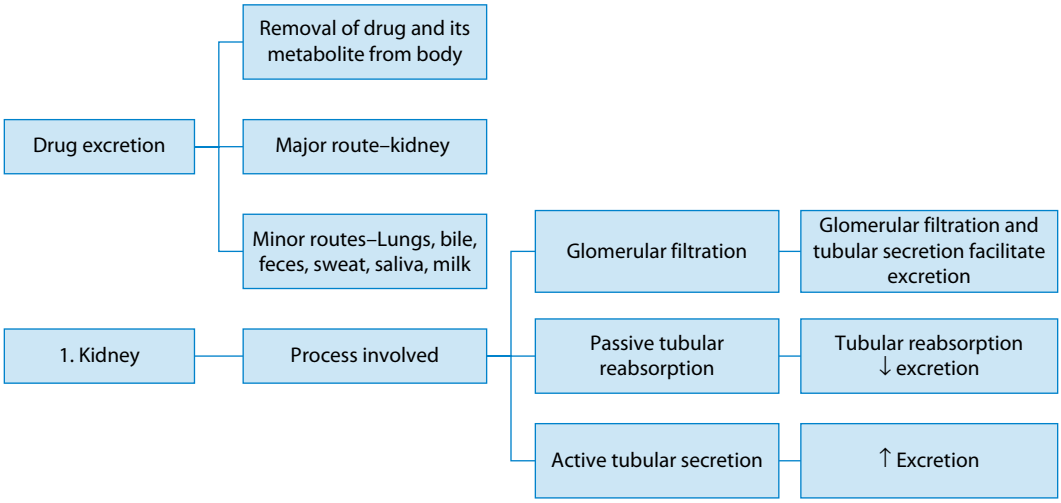
3.18 FACTORS MODIFYING METABOLISM



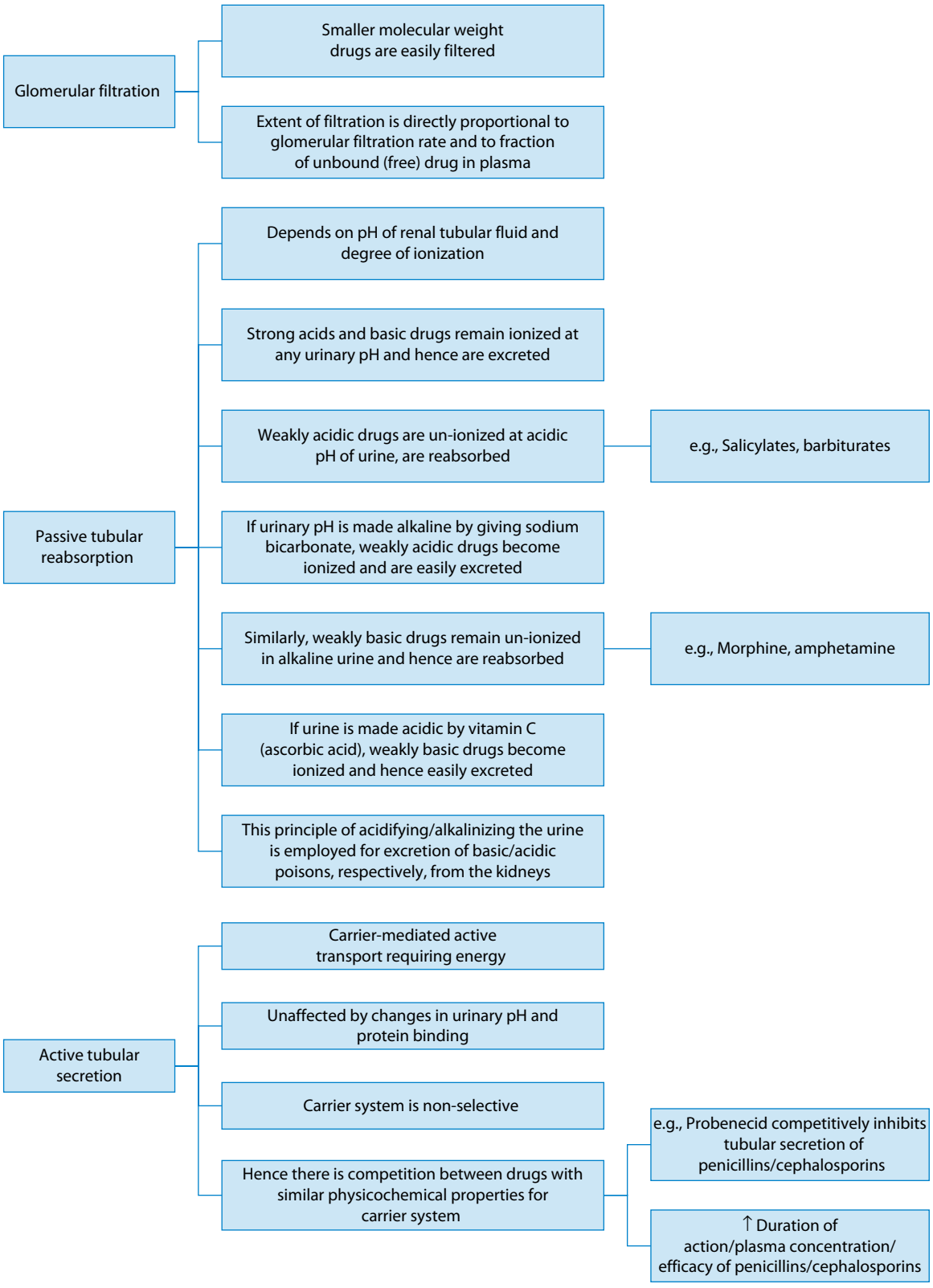
3.19 PRODRUG



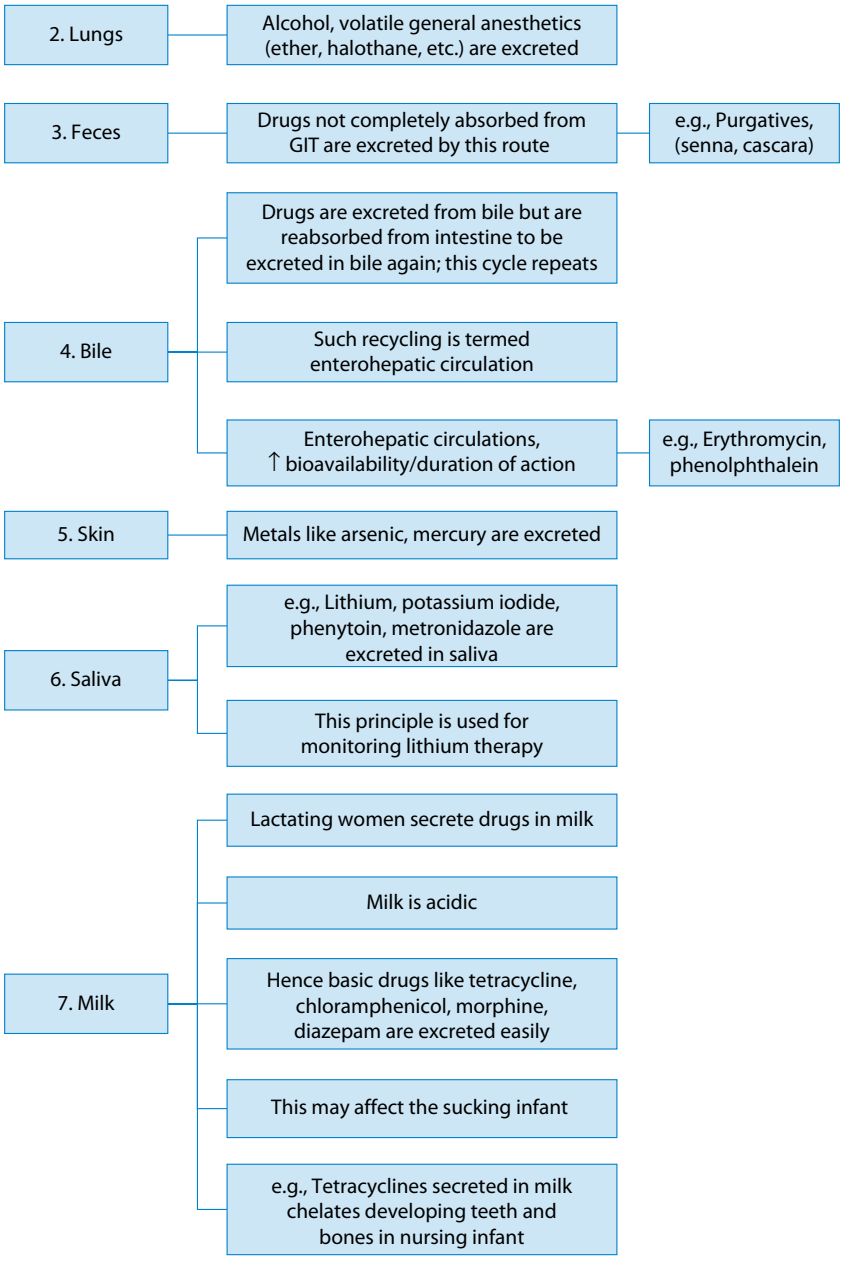
3.20 DRUG EXCRETION



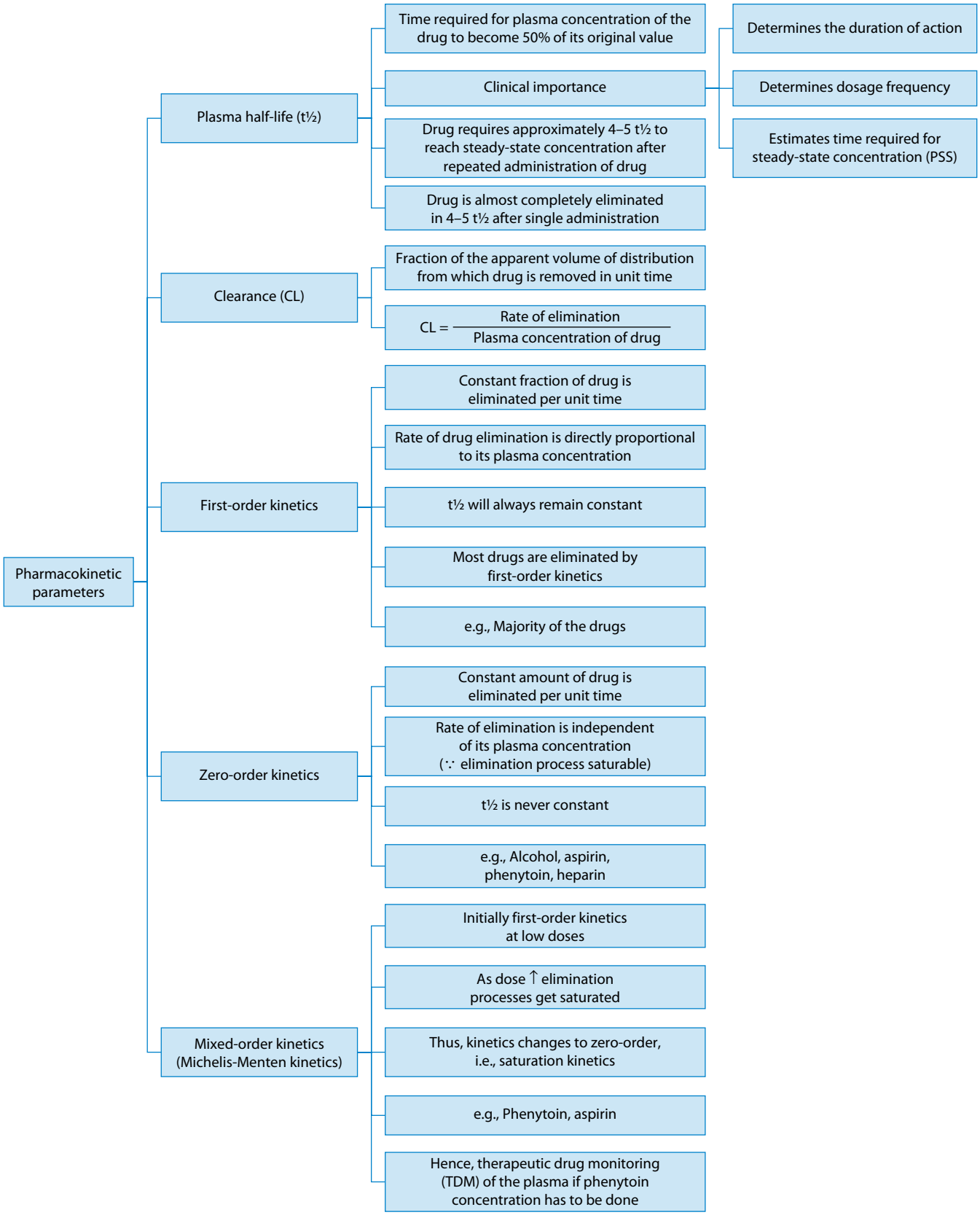
3.21 DRUG EXCRETION BY KIDNEYS



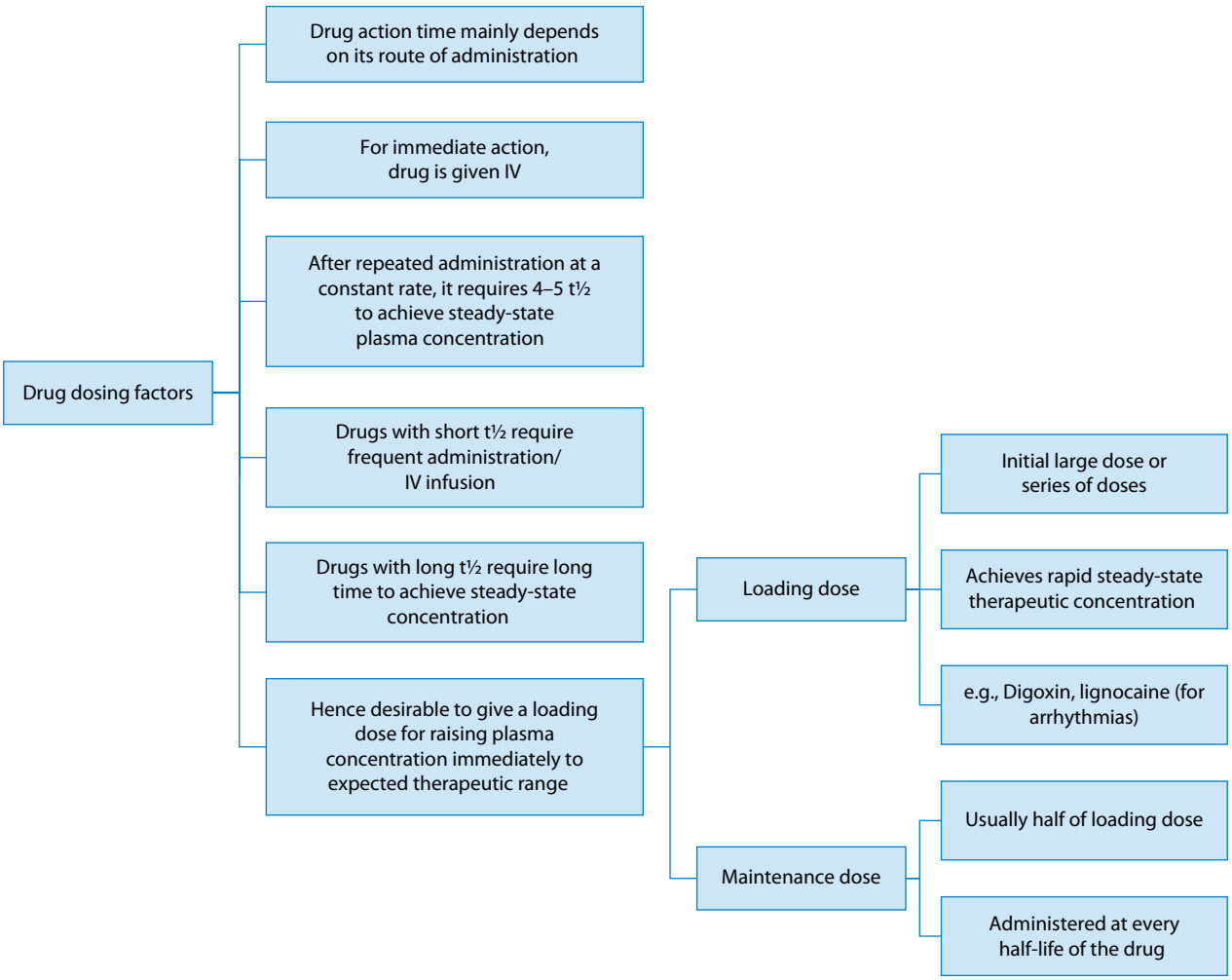
3.22 OTHER ROUTES OF DRUG EXCRETION



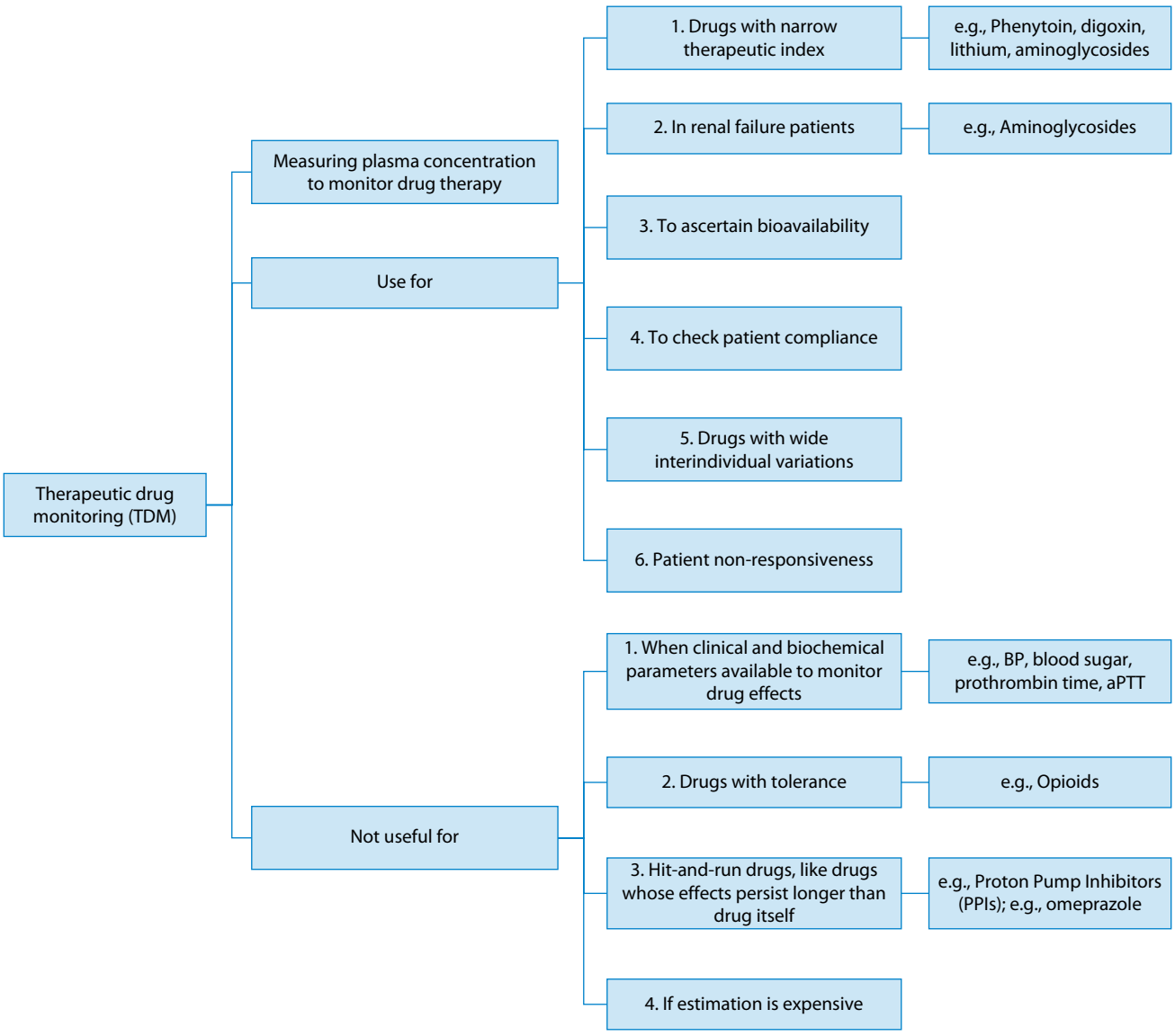
3.23 APPLIED PHARMACOKINETICS



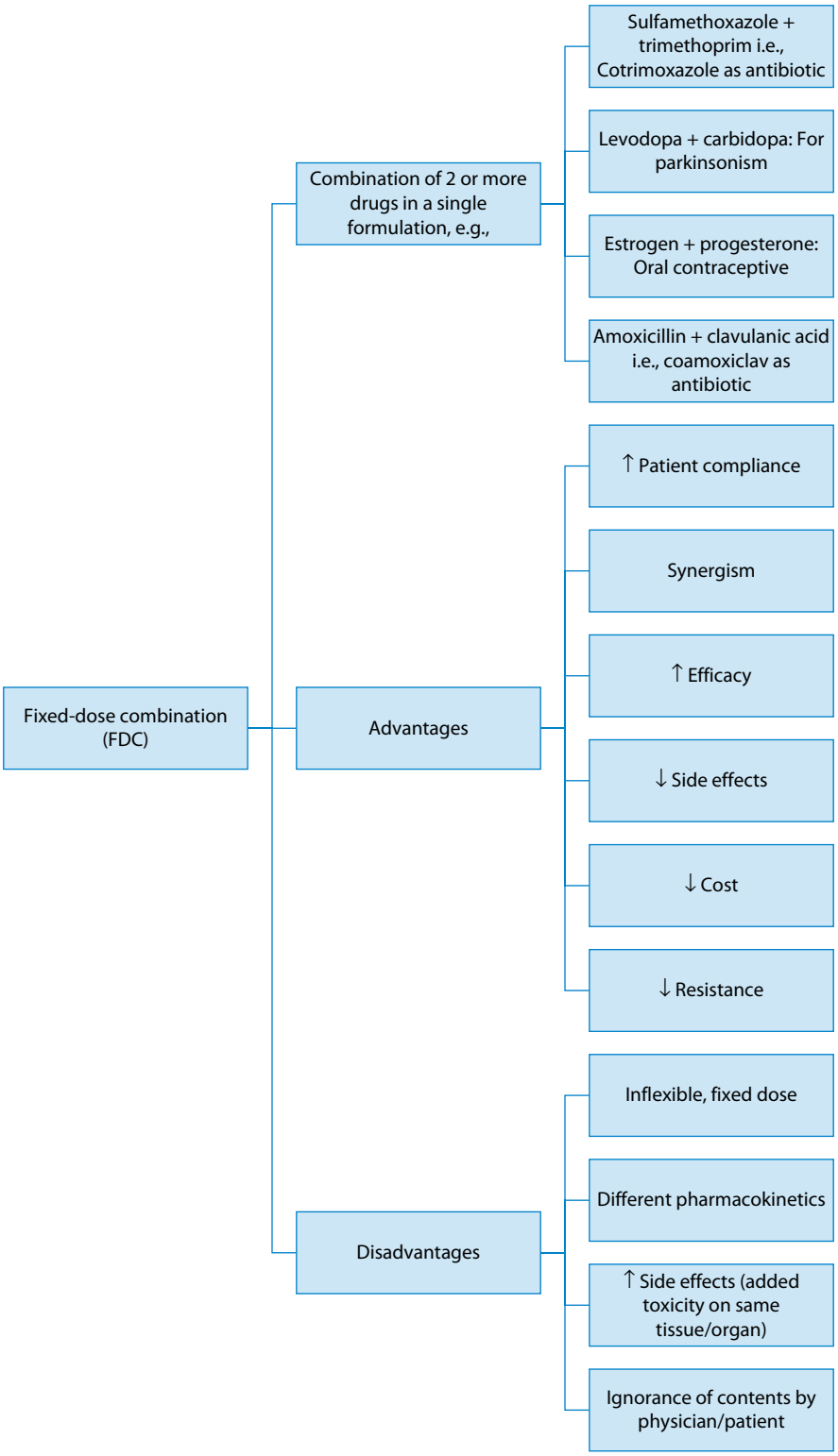
3.24 DRUG DOSING FACTORS



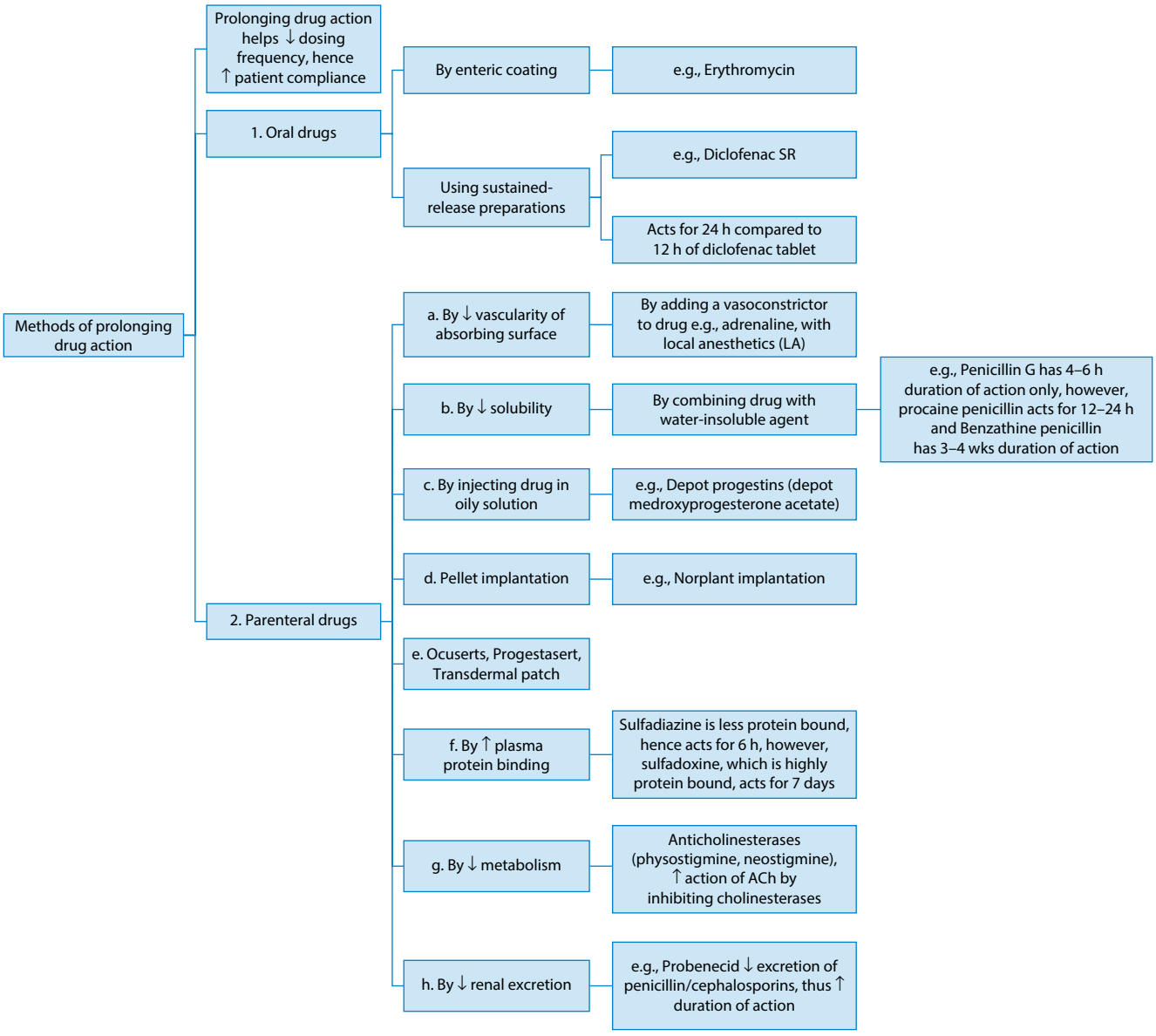
3.25 THERAPEUTIC DRUG MONITORING



3.26 FIXED-DOSE COMBINATION



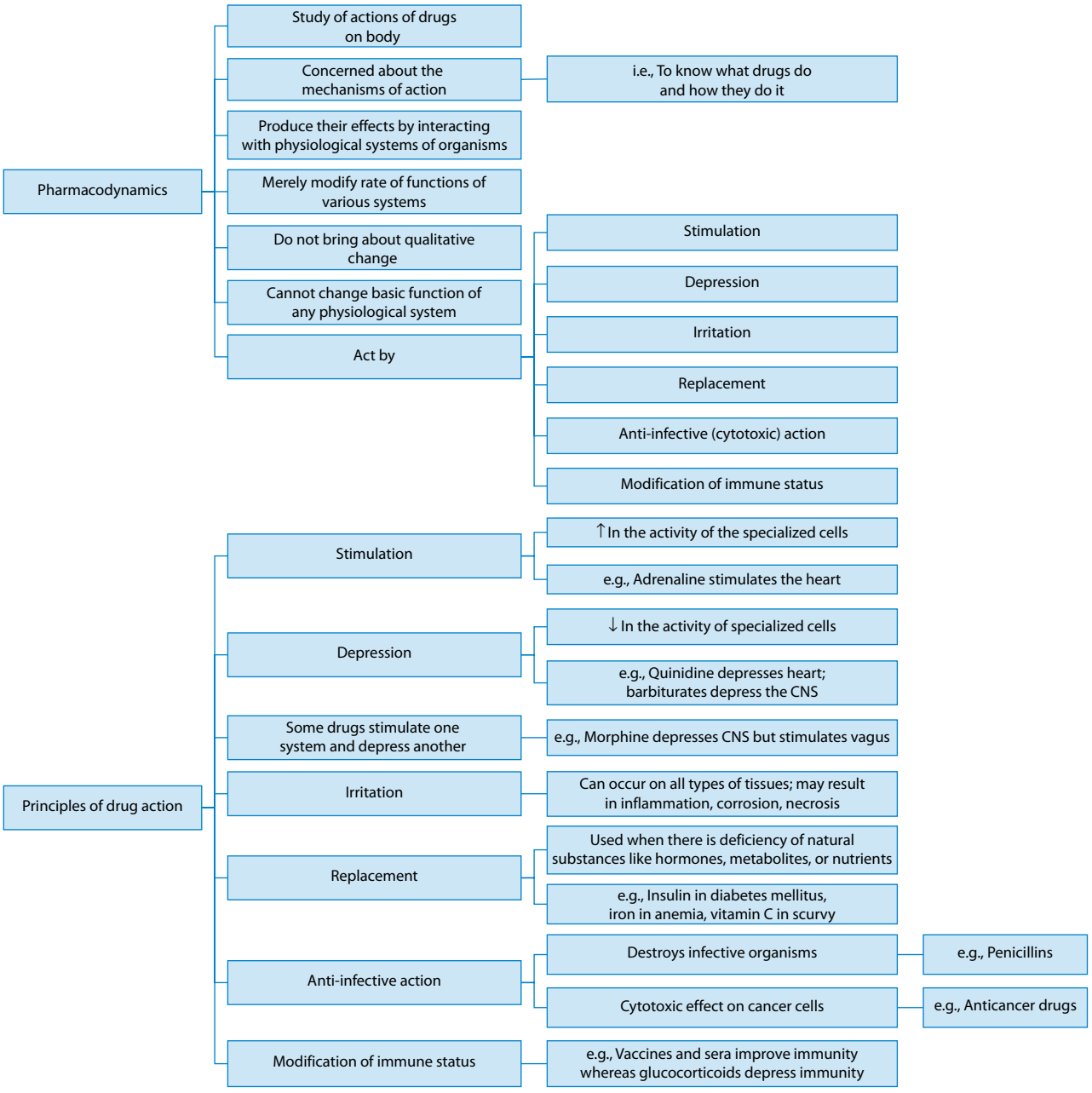
3.27 METHODS OF PROLONGING DRUG ACTION



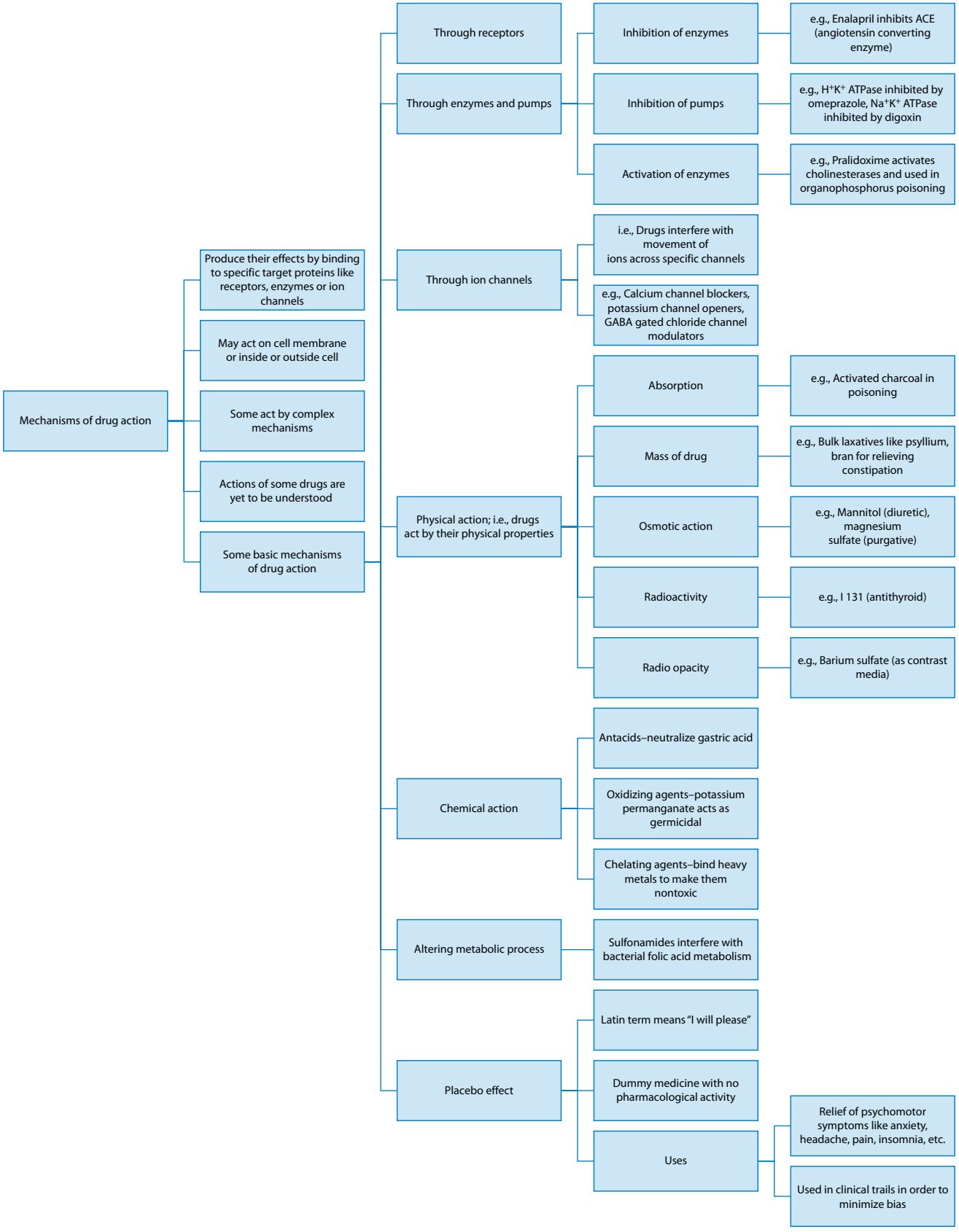
4

Pharmacodynamics

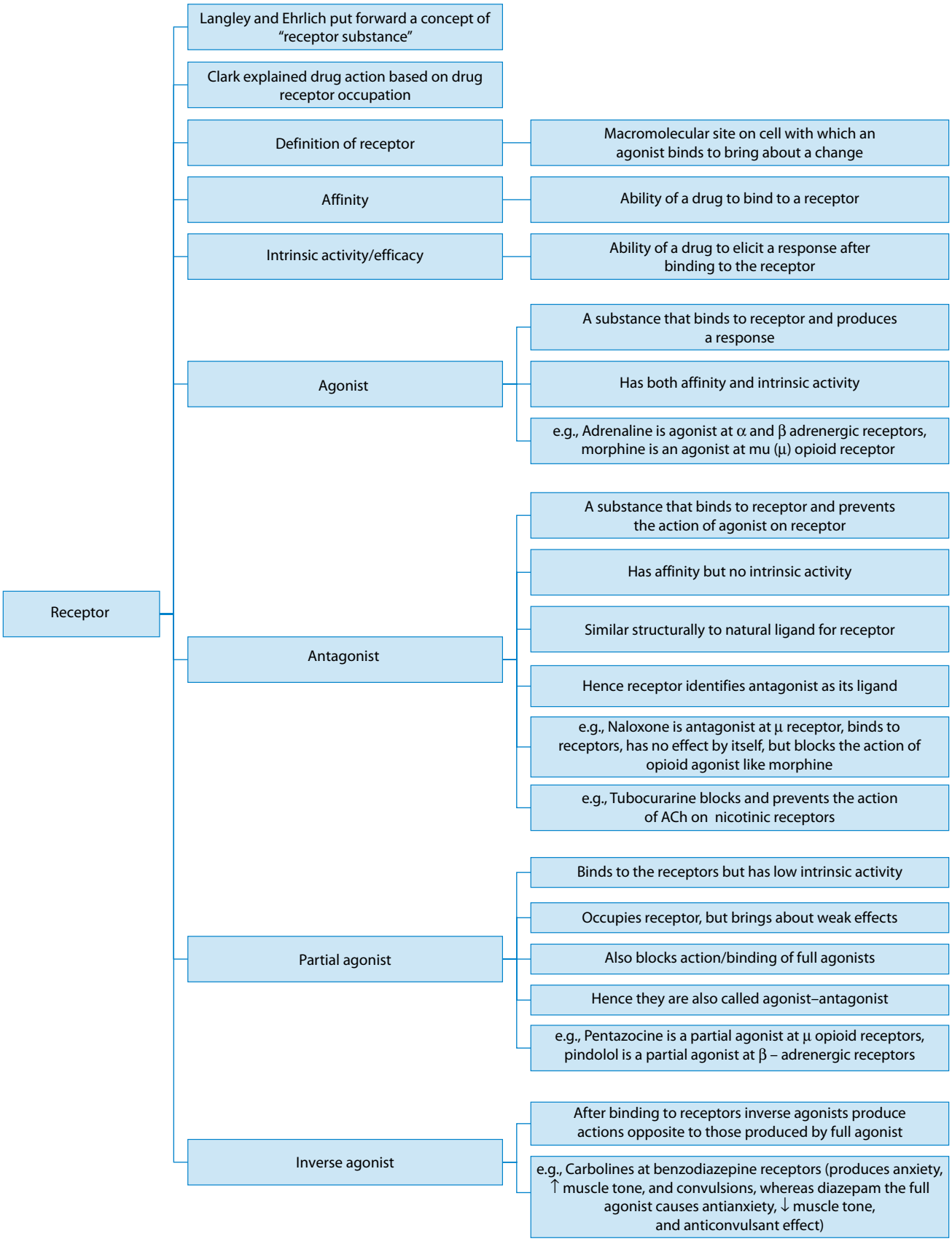
4.1 PHARMACODYNAMICS AND PRINCIPLES OF DRUG ACTION



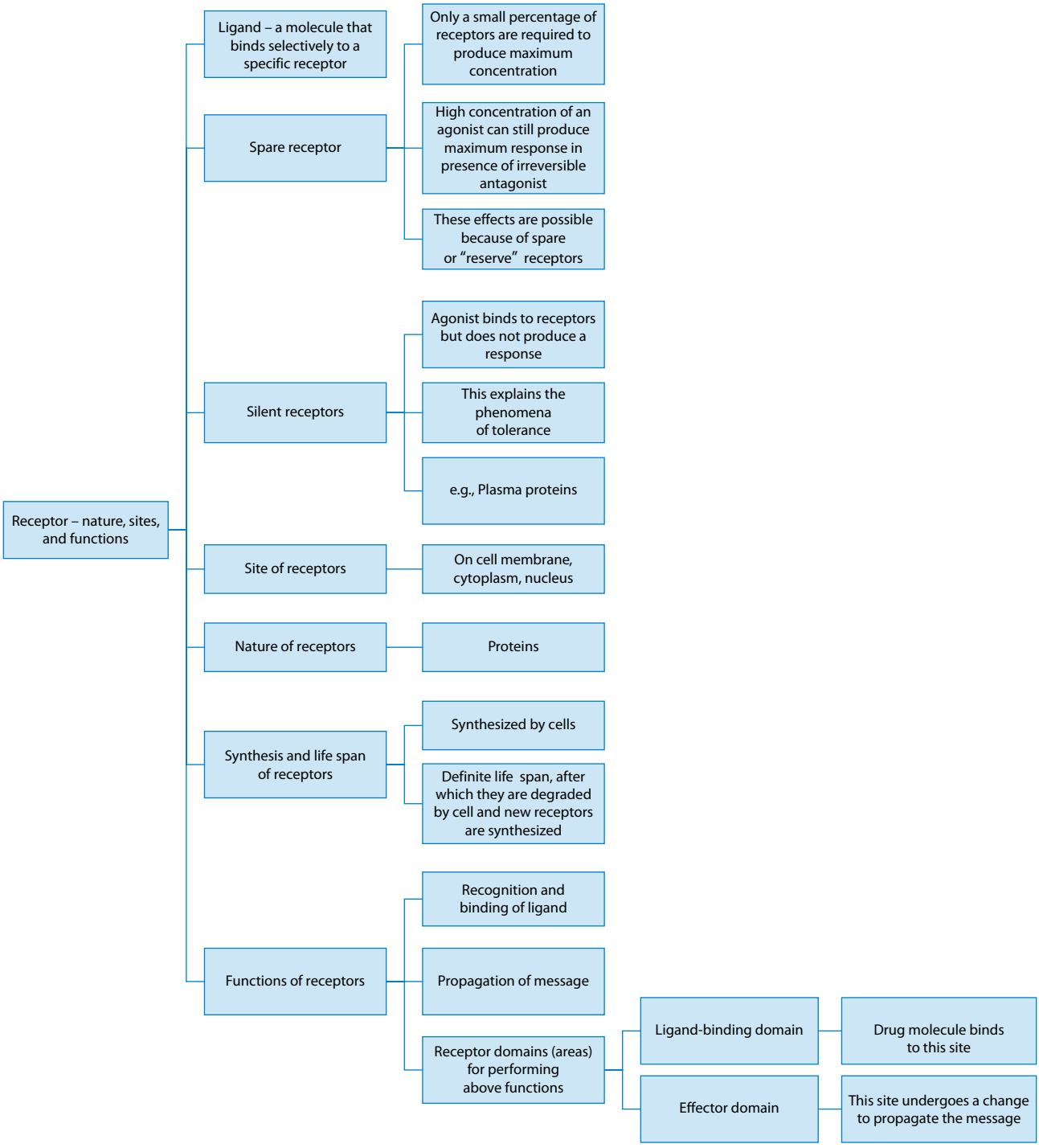
4.2 MECHANISMS OF DRUG ACTION



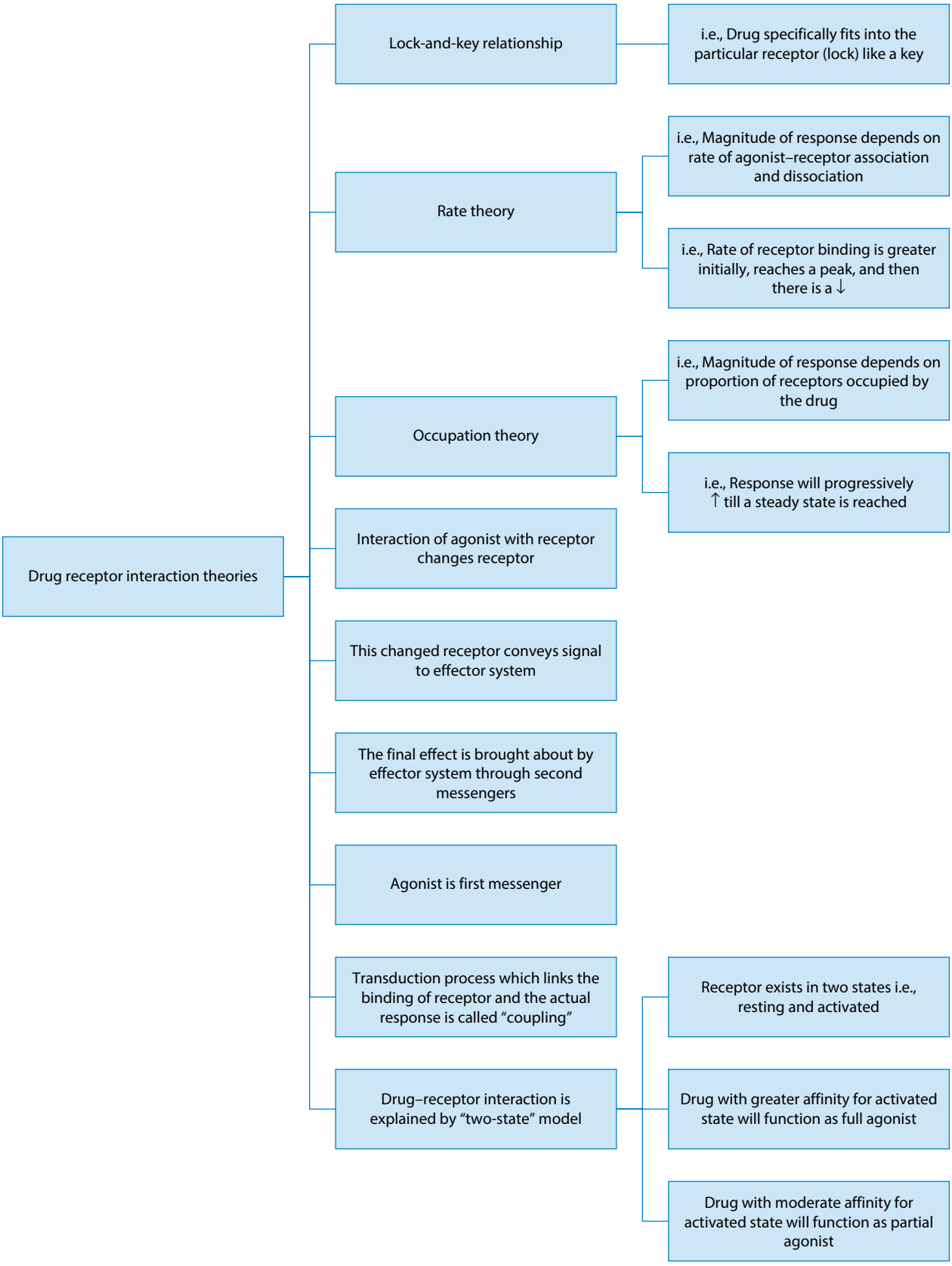
4.3 RECEPTOR


Receptor
Langley and Ehrlich put forward a concept of "receptor substance"
Clark explained drug action based on drug receptor occupation
Definition of receptor
Macromolecular site on cell with which an agonist binds to bring about a change
Affinity
Ability of a drug to bind to a receptor
Intrinsic activity/efficacy
Ability of a drug to elicit a response after binding to the receptor
Agonist
A substance that binds to receptor and produces a response
Has both affinity and intrinsic activity
e.g., Adrenaline is agonist at α and β adrenergic receptors, morphine is an agonist at mu (μ) opioid receptor
Antagonist
A substance that binds to receptor and prevents the action of agonist on receptor
Has affinity but no intrinsic activity
Similar structurally to natural ligand for receptor
Hence receptor identifies antagonist as its ligand
e.g., Naloxone is antagonist at μ receptor, binds to receptors, has no effect by itself, but blocks the action of opioid agonist like morphine
e.g., Tubocurarine blocks and prevents the action of ACh on nicotinic receptors
Partial agonist
Binds to the receptors but has low intrinsic activity
Occupies receptor, but brings about weak effects
Also blocks action/binding of full agonists
Hence they are also called agonist–antagonist
e.g., Pentazocine is a partial agonist at μ opioid receptors, pindolol is a partial agonist at β – adrenergic receptors
Inverse agonist
After binding to receptors inverse agonists produce actions opposite to those produced by full agonist
e.g., Carbolines at benzodiazepine receptors (produces anxiety, ↑ muscle tone, and convulsions, whereas diazepam the full agonist causes antianxiety, ↓ muscle tone, and anticonvulsant effect)

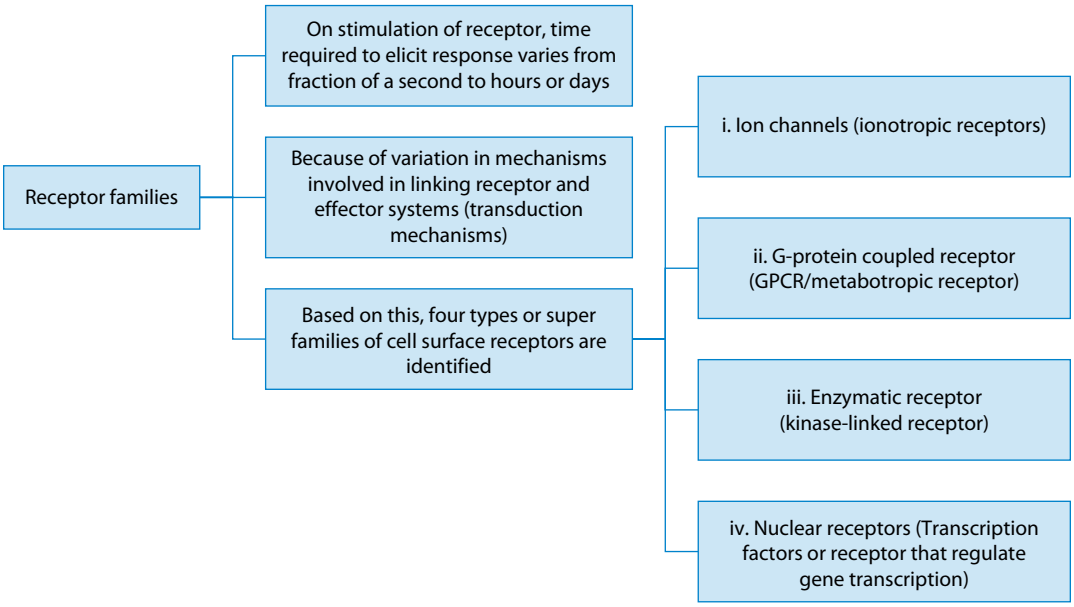
4.4 RECEPTOR – NATURE, SITES, AND FUNCTIONS



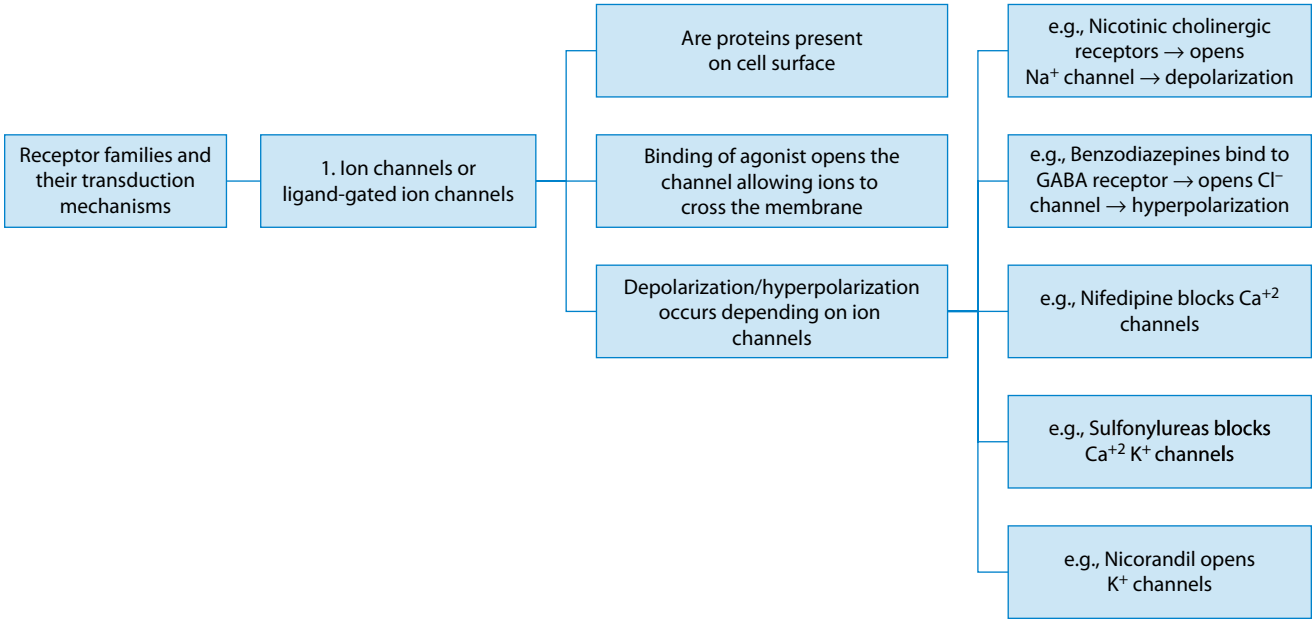
4.5 DRUG RECEPTOR INTERACTION THEORIES



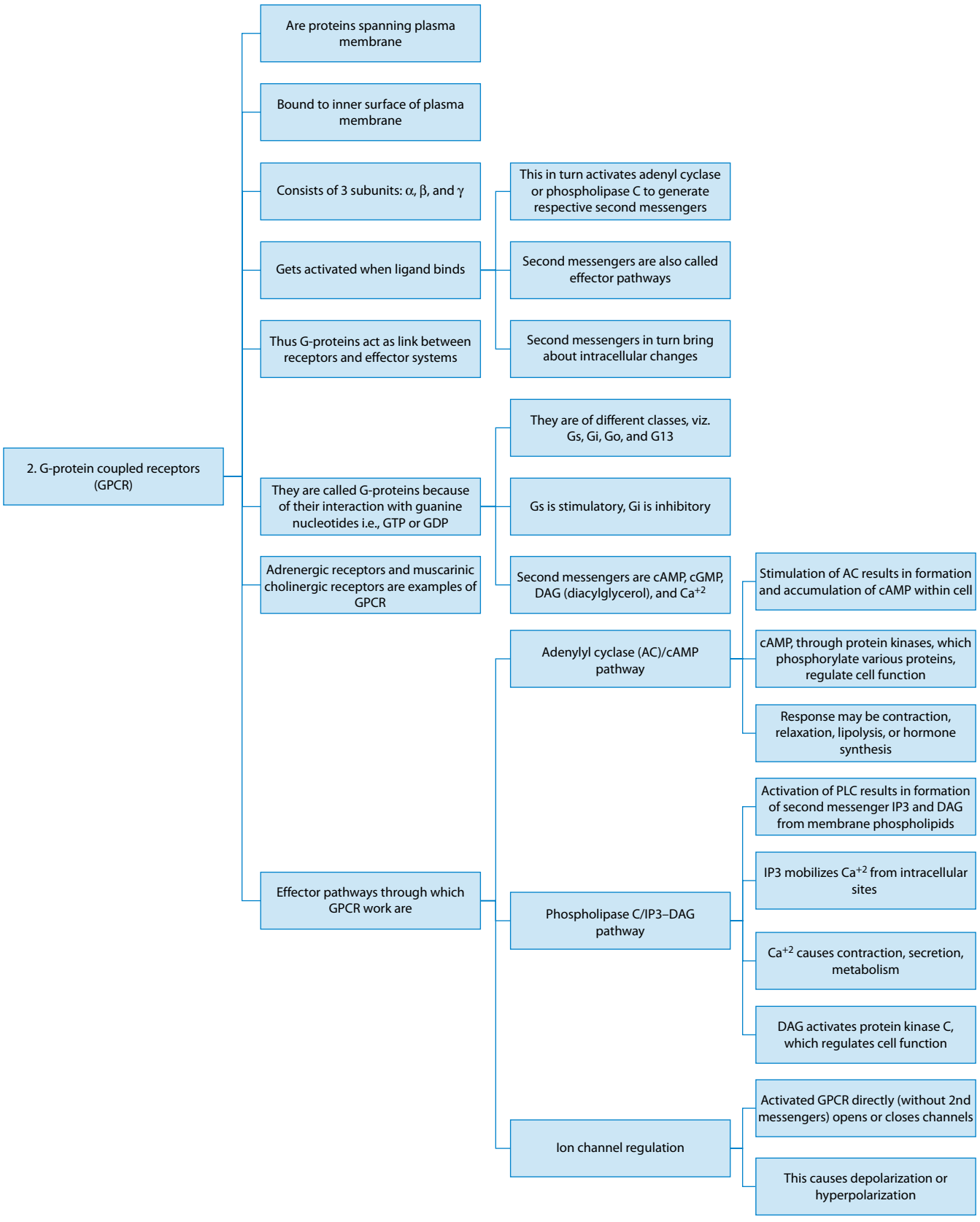
4.6 RECEPTOR FAMILIES



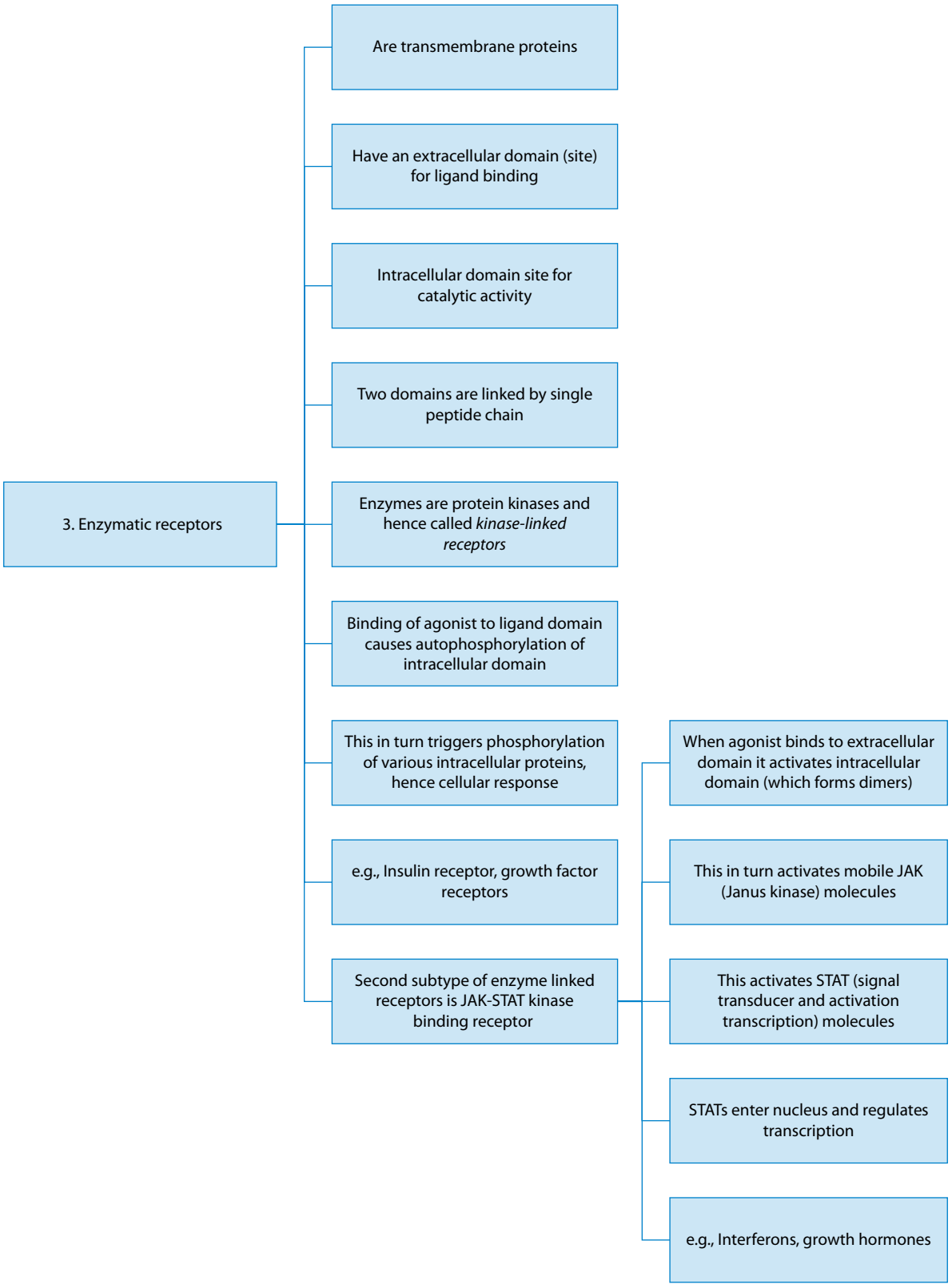
4.7 RECEPTOR FAMILIES AND THEIR TRANSDUCTION MECHANISMS – ION CHANNELS OR LIGAND-GATED ION CHANNELS



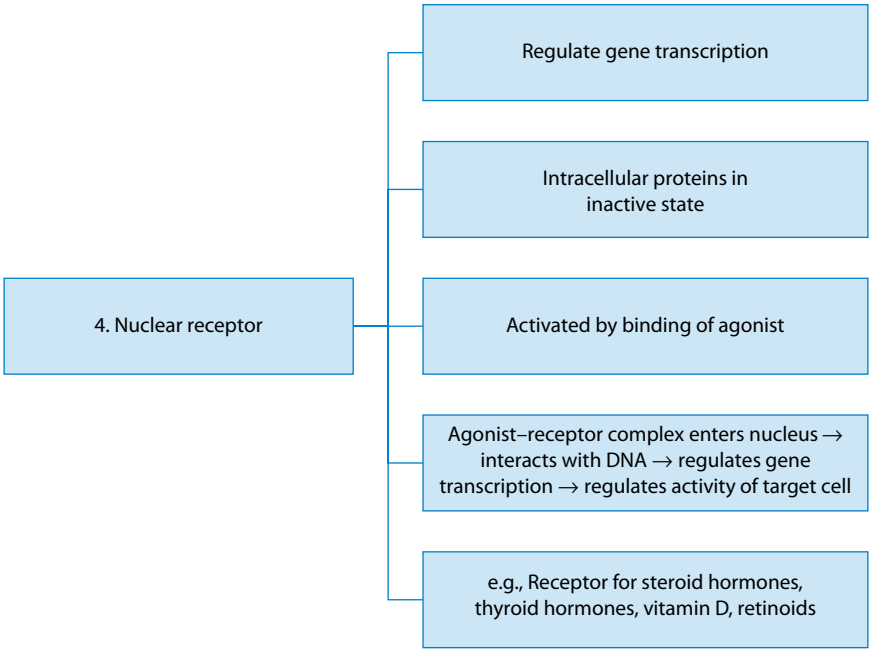
4.8 G-PROTEIN COUPLED RECEPTORS (GPCR)



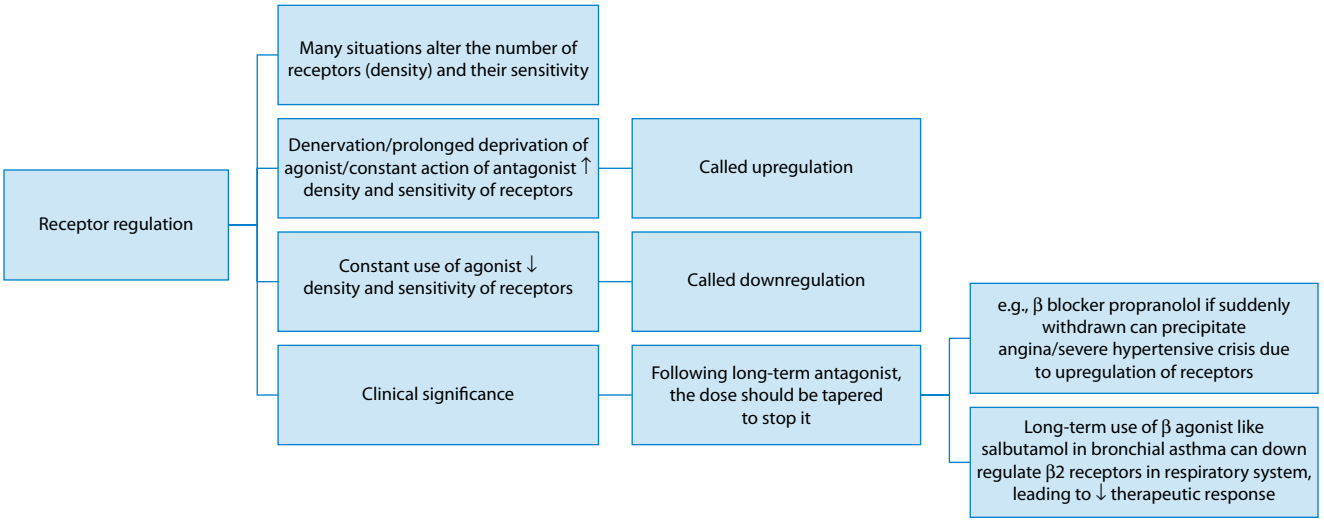
4.9 ENZYMATIC RECEPTORS



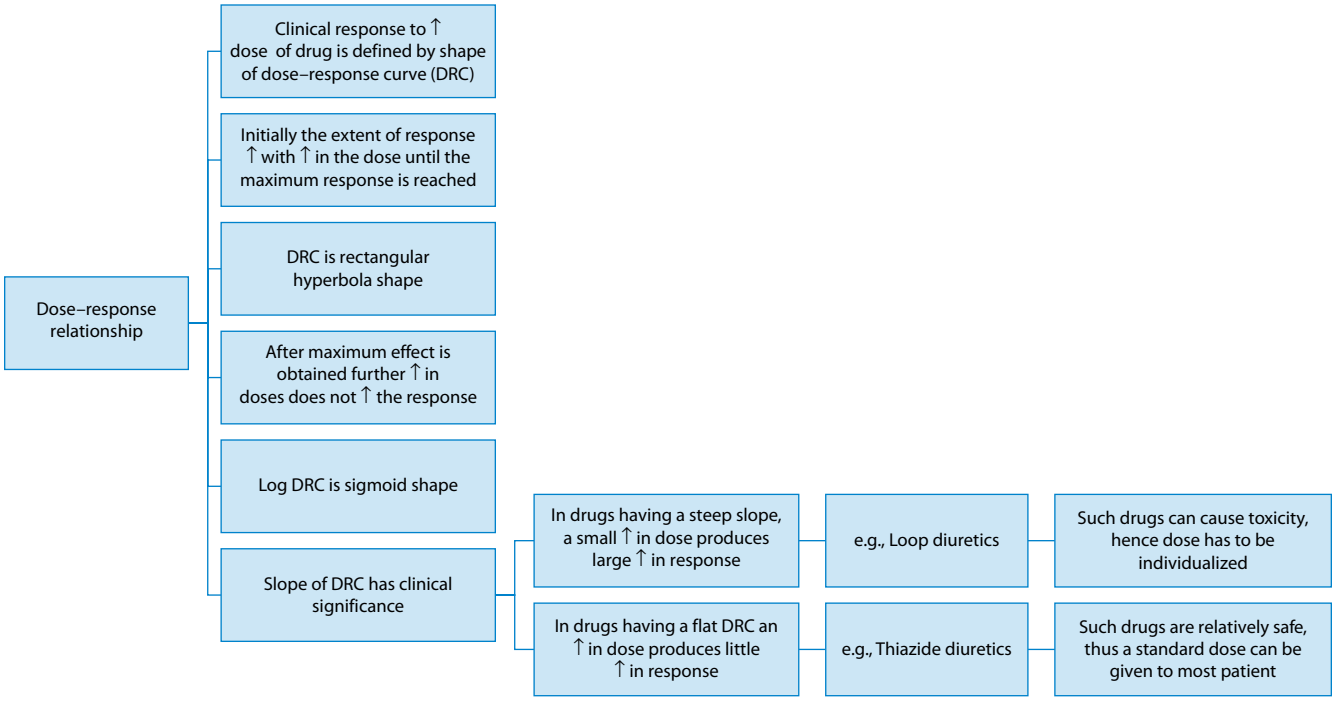
4.10 NUCLEAR RECEPTOR



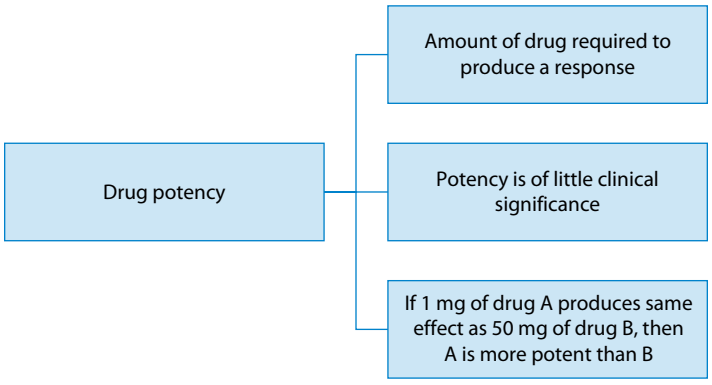
4.11 RECEPTOR REGULATION



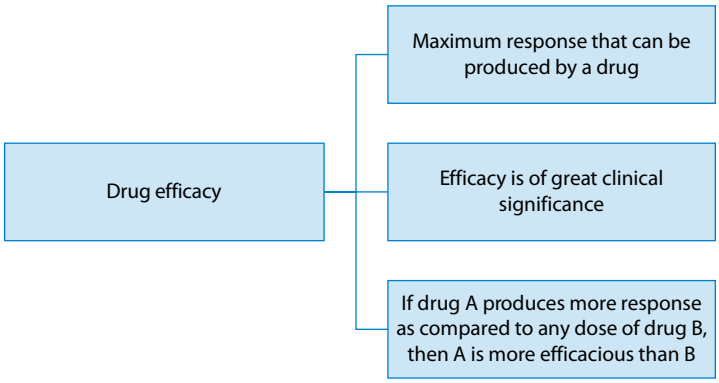
4.12 DOSE–RESPONSE RELATIONSHIP



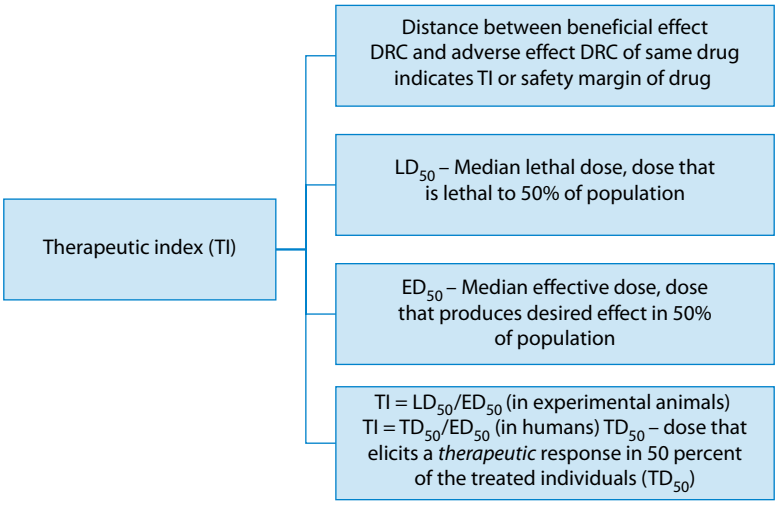
4.13 DRUG POTENCY



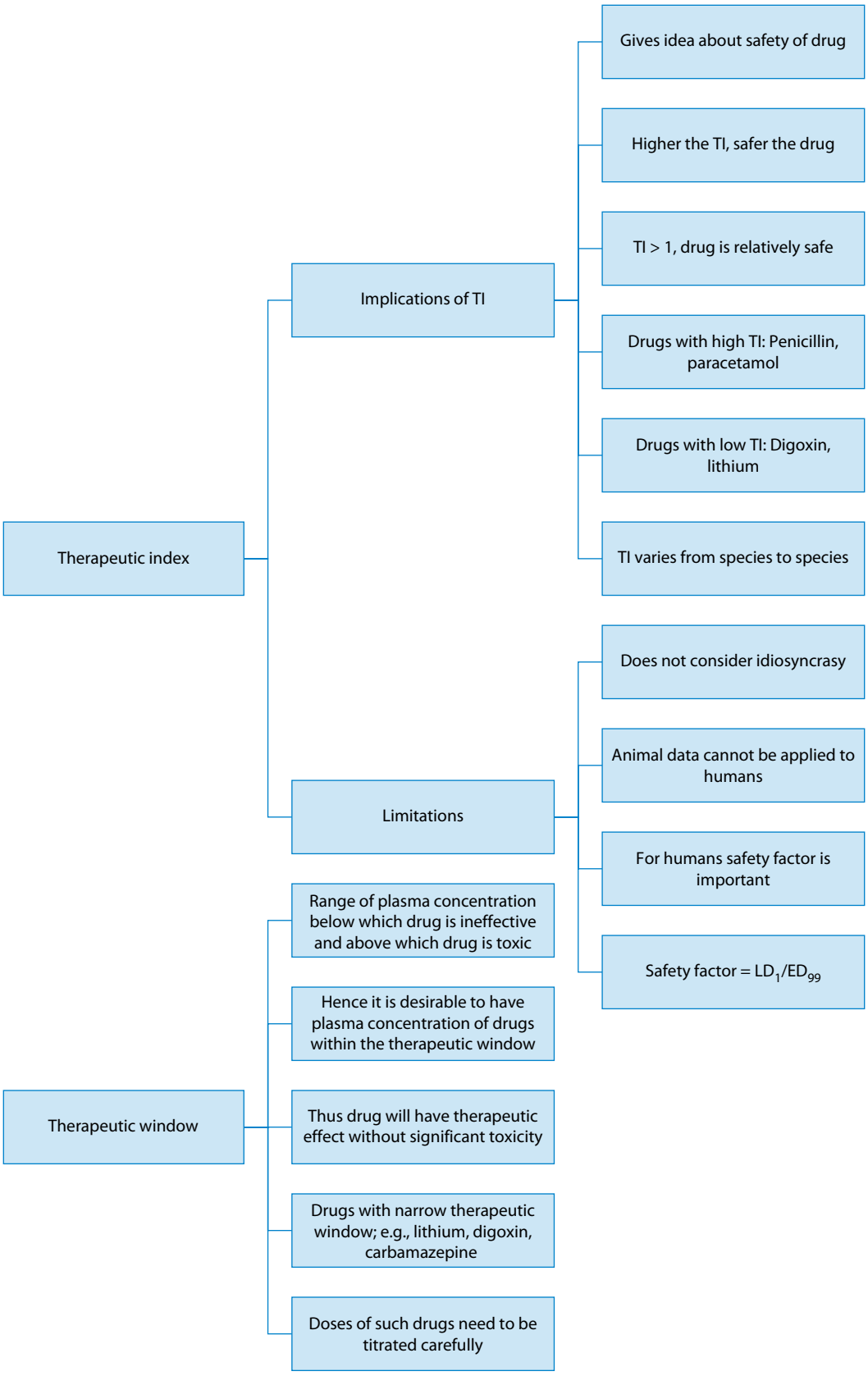
4.14 DRUG EFFICACY



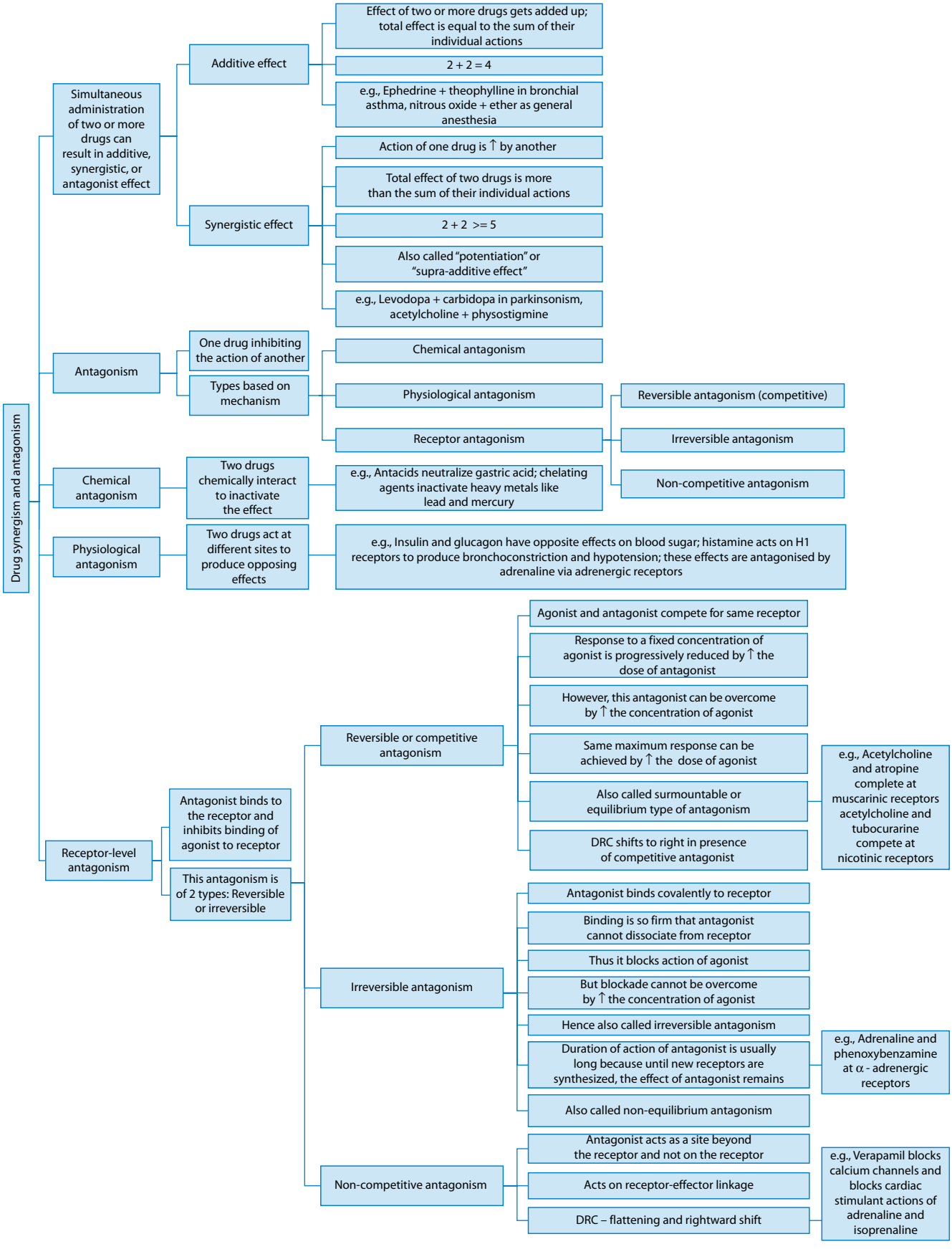
4.15 THERAPEUTIC INDEX (TI)



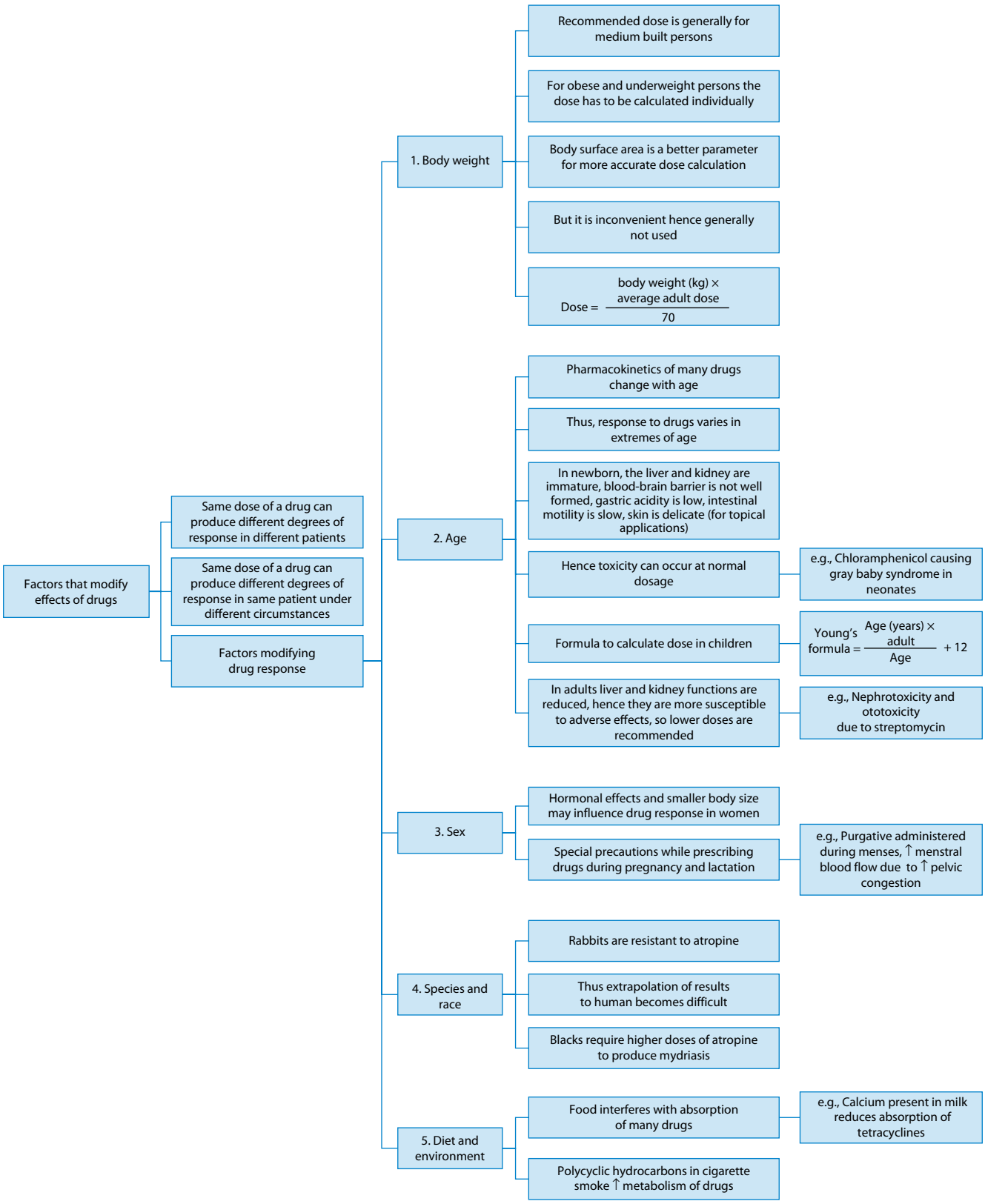
4.16 THERAPEUTIC WINDOW



4.17 DRUG SYNERGISM AND ANTAGONISM

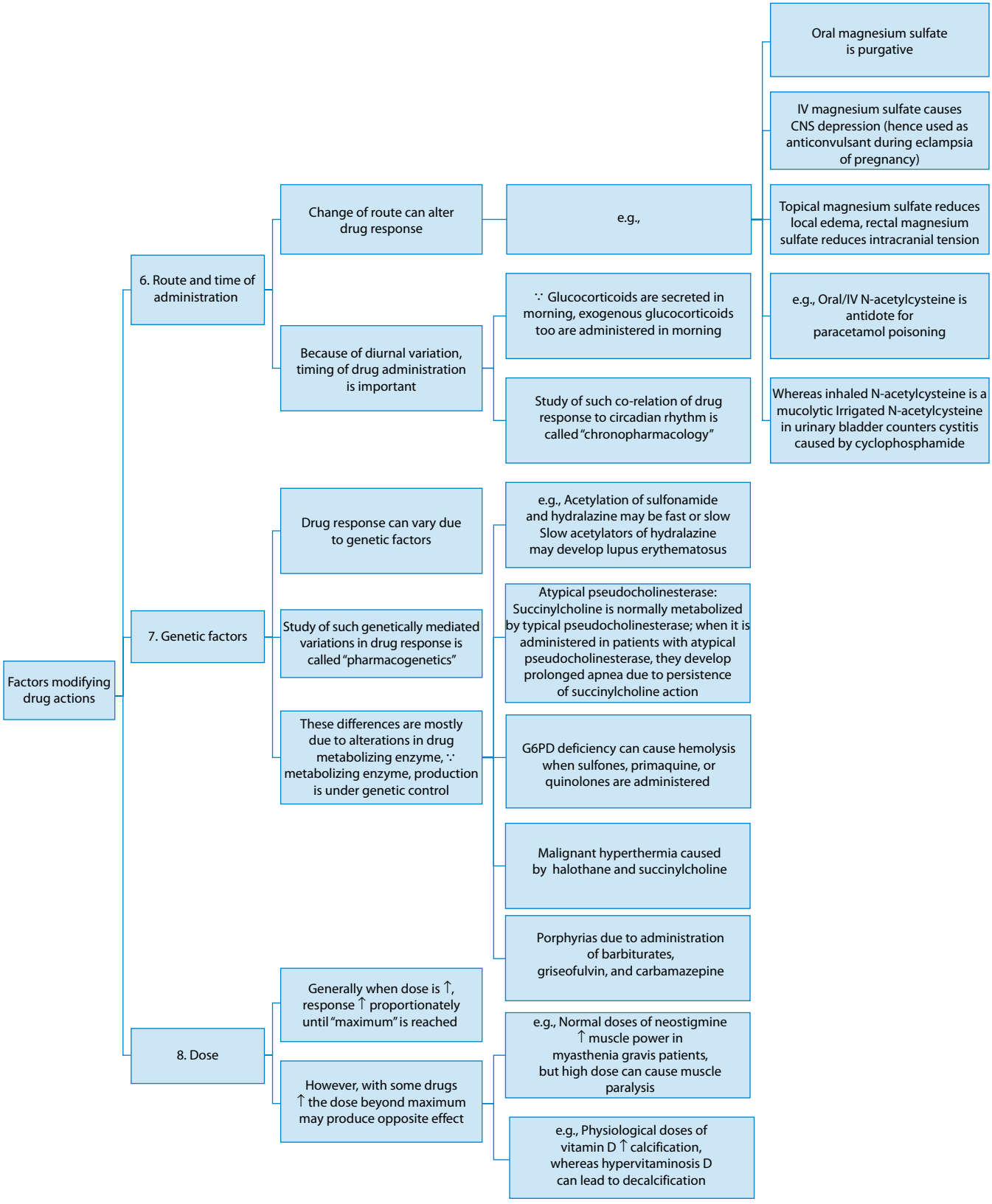


4.18 FACTORS THAT MODIFY EFFECTS OF DRUGS



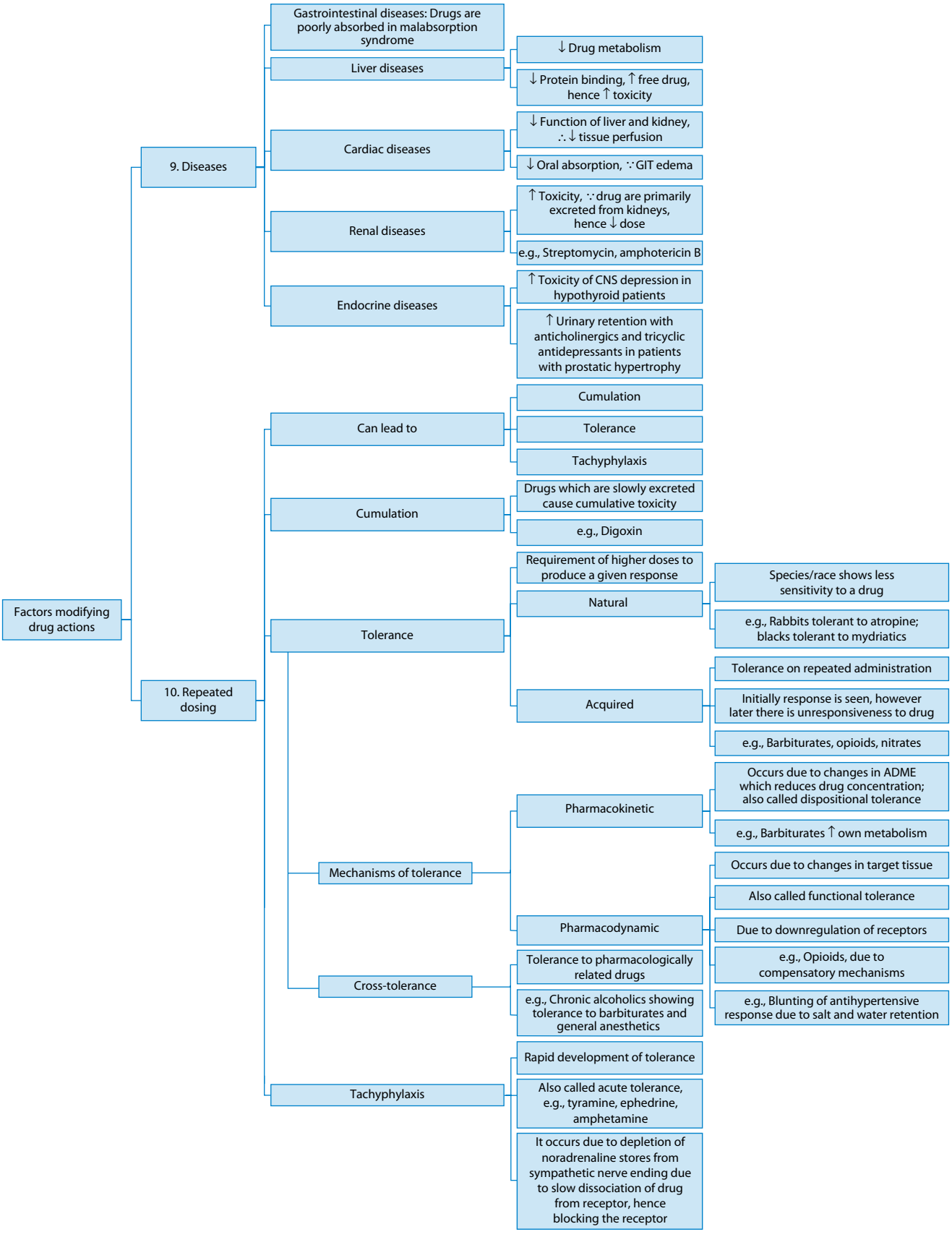
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4.18 FACTORS THAT MODIFY EFFECTS OF DRUGS (Continued)



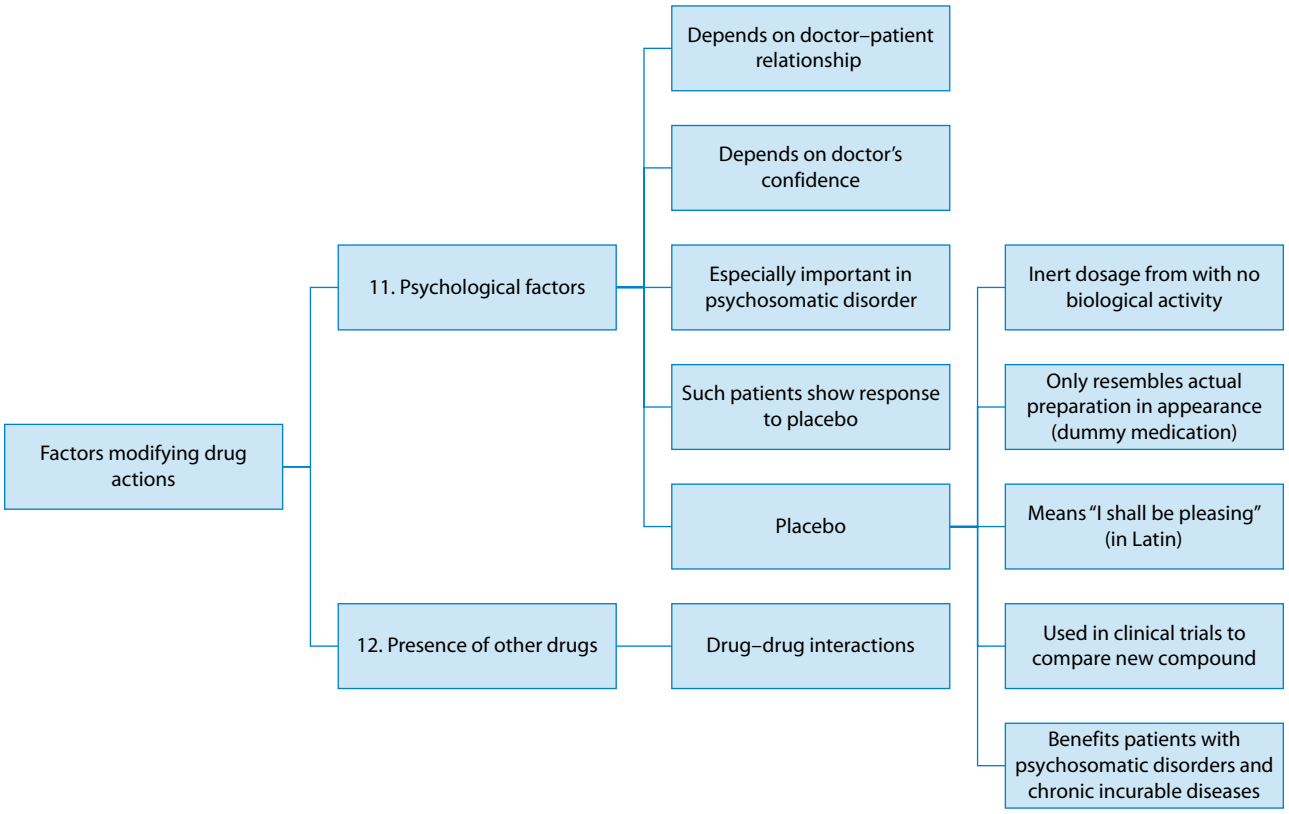
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4.18 FACTORS THAT MODIFY EFFECTS OF DRUGS (Continued)

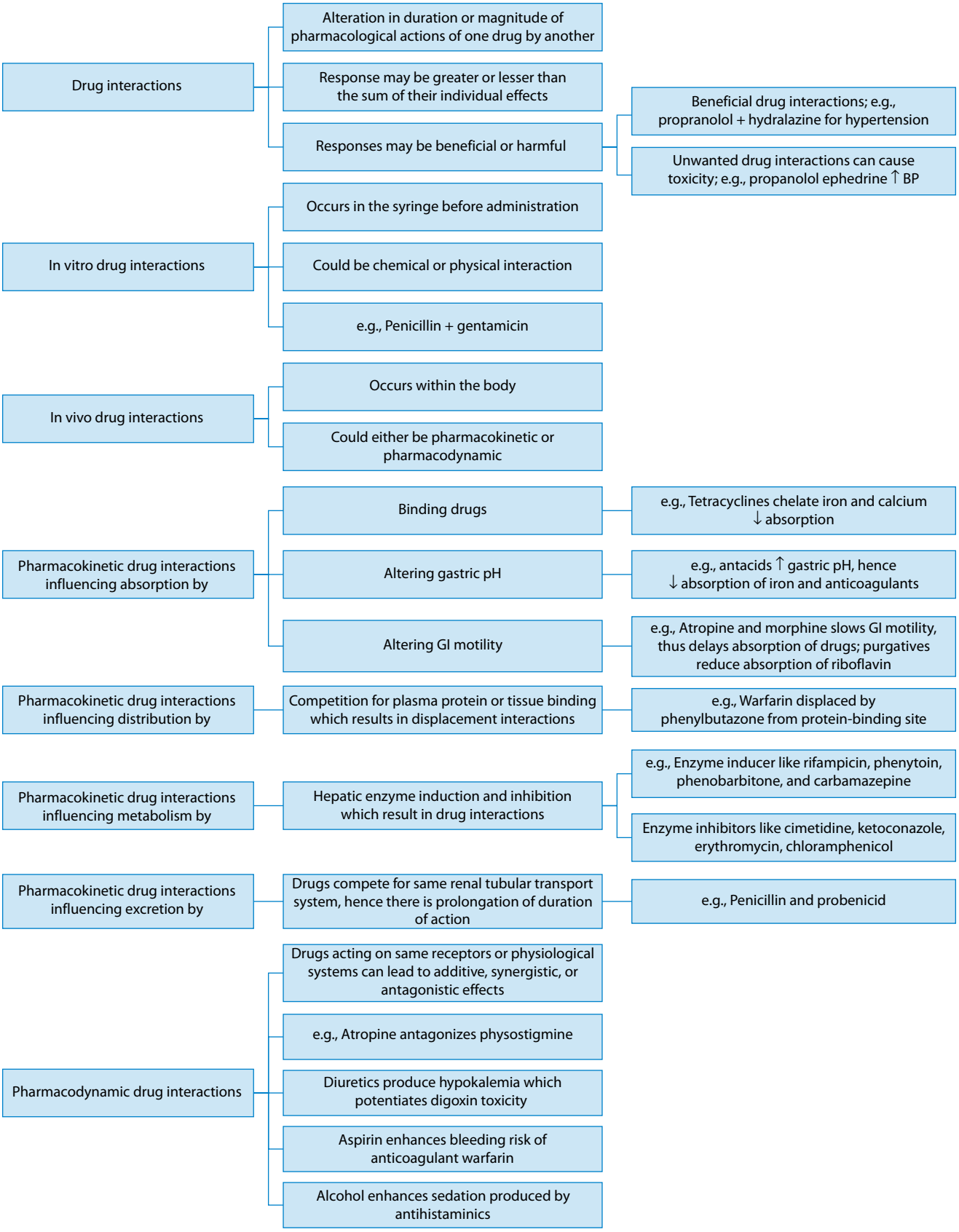


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4.18 FACTORS THAT MODIFY EFFECTS OF DRUGS (Continued)



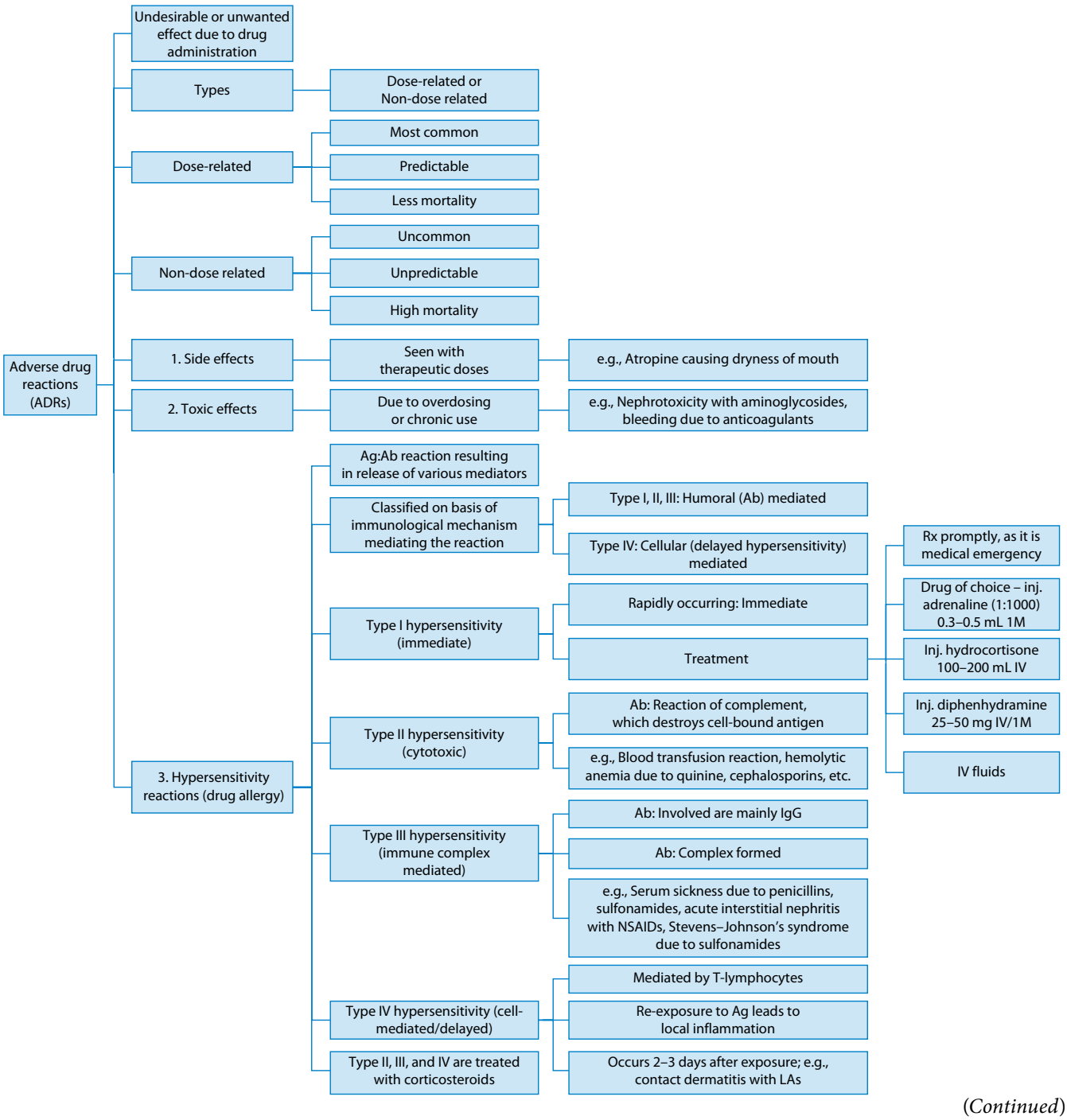
4.19 DRUG INTERACTIONS



5

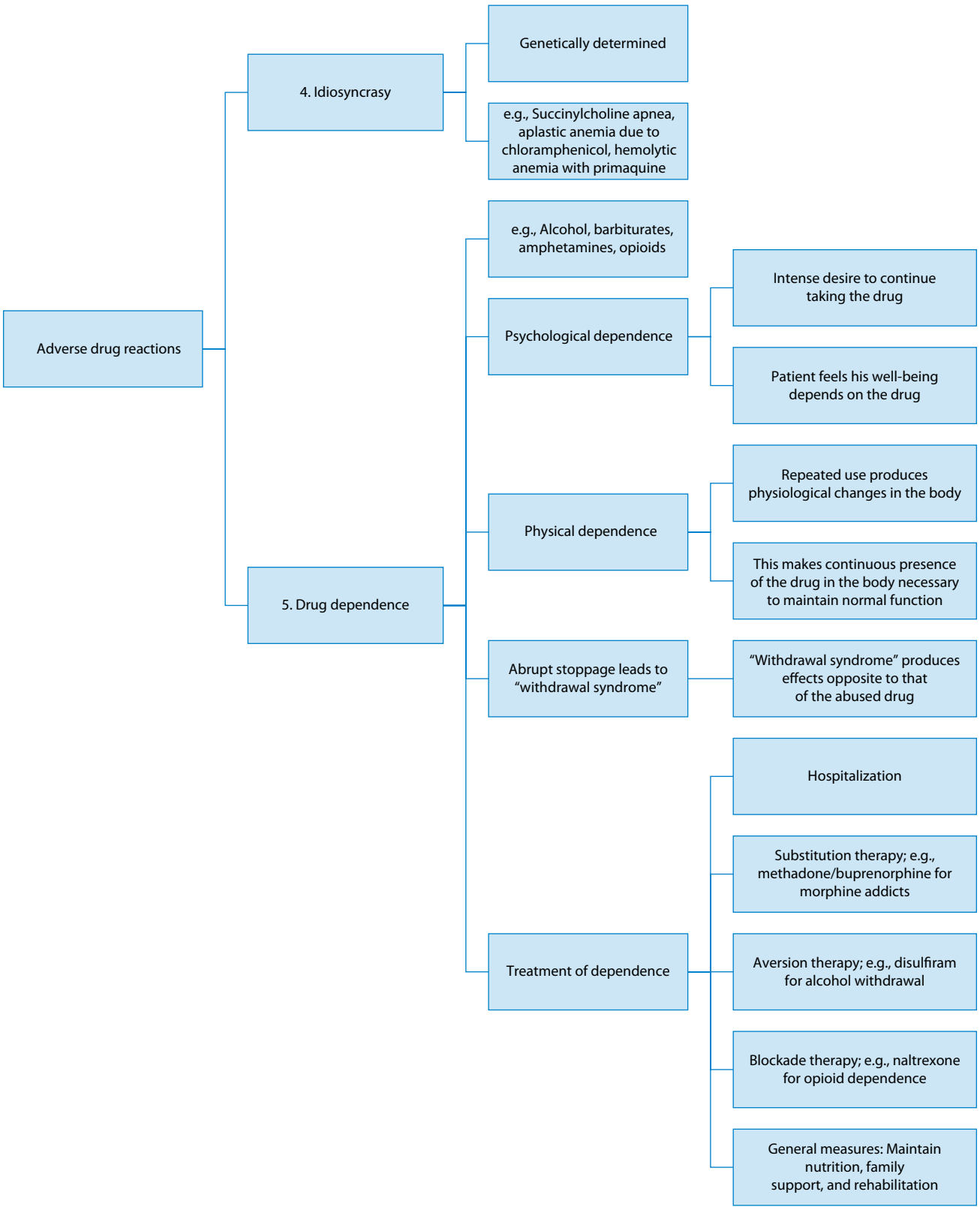
Adverse drug reactions

5.1 TYPES OF ADVERSE DRUG REACTIONS (ADRs)



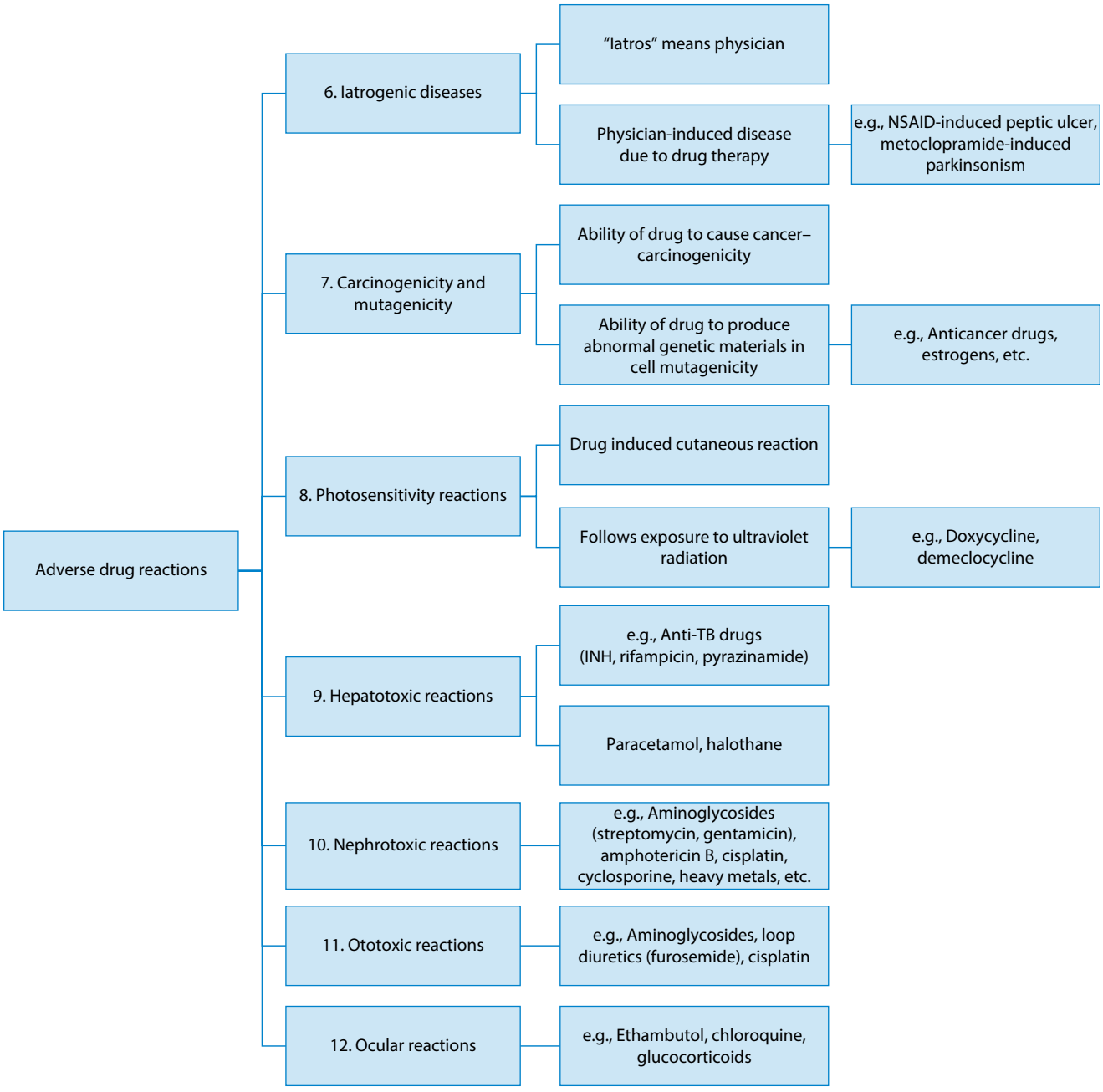
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5.1 TYPES OF ADVERSE DRUG REACTIONS (ADRs) (Continued)



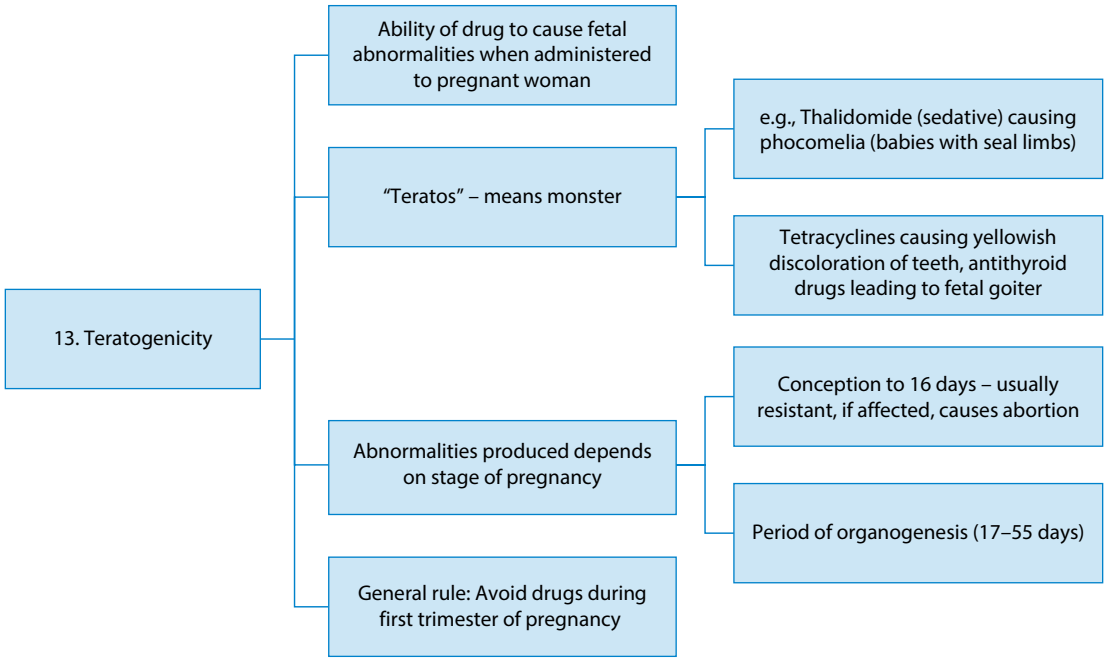
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5.1 TYPES OF ADVERSE DRUG REACTIONS (ADRs) (Continued)

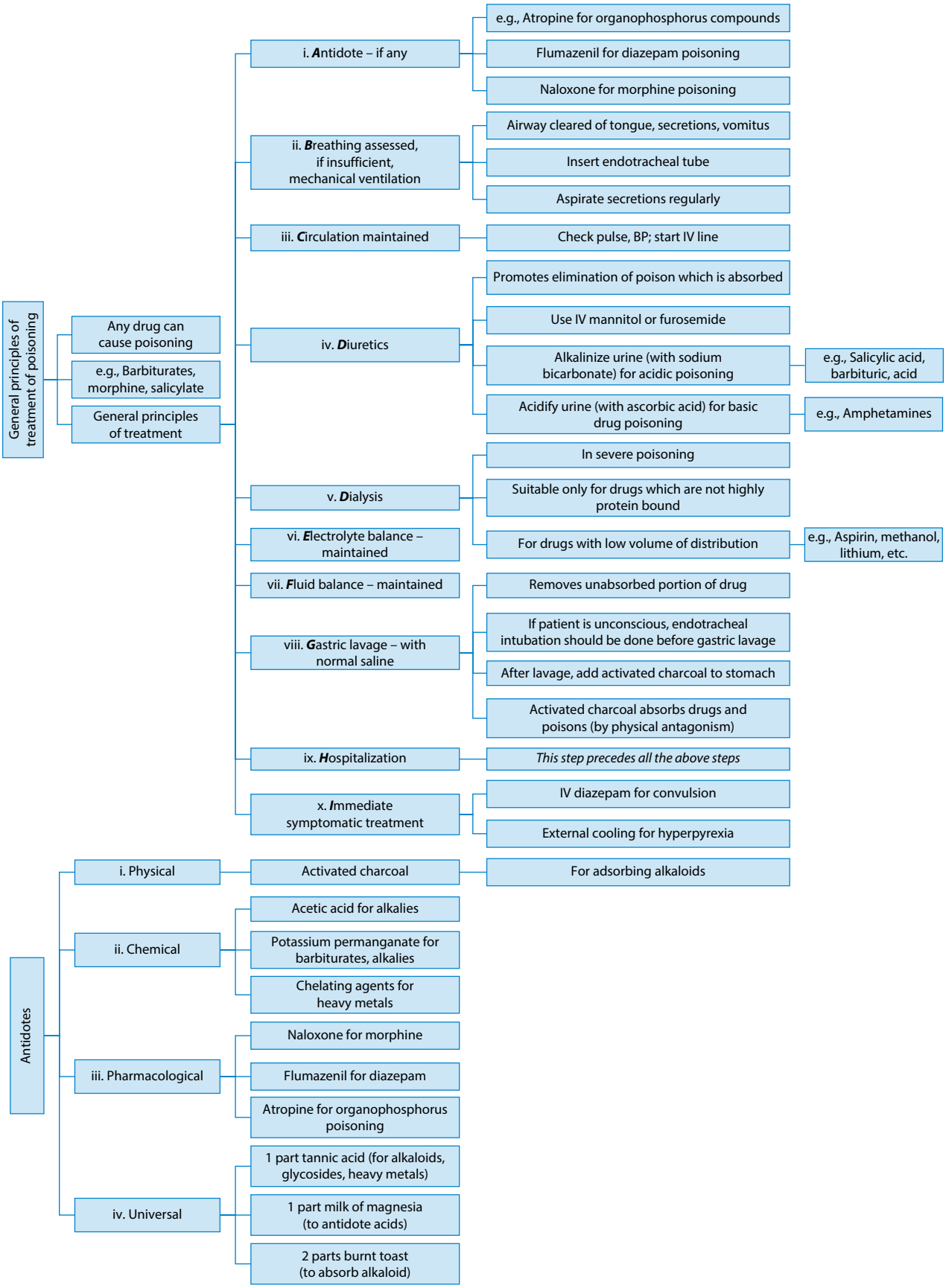


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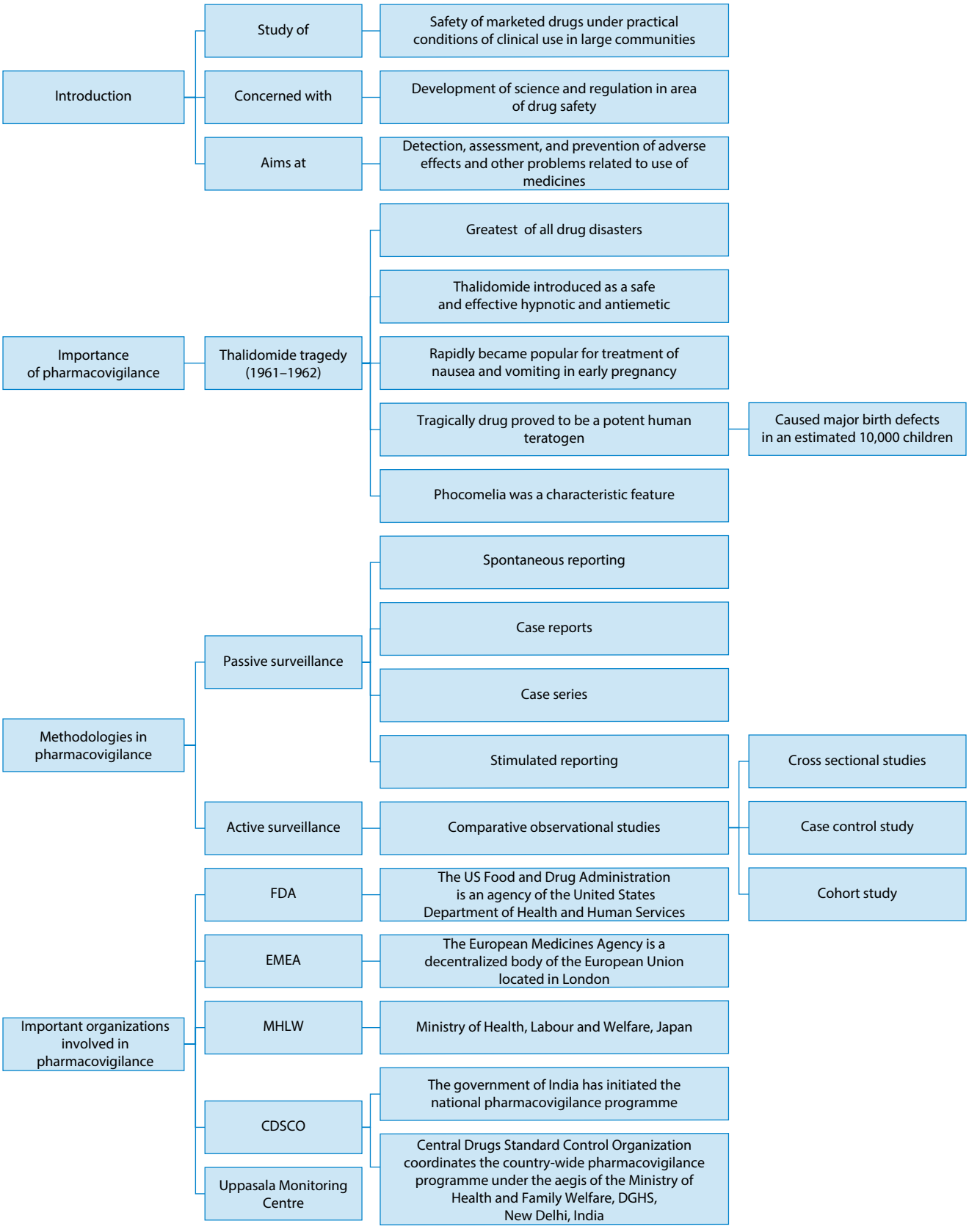
5.1 TYPES OF ADVERSE DRUG REACTIONS (ADRs) (Continued)



5.2 GENERAL PRINCIPLES OF TREATMENT OF POISONING (MNEMONICS [ABCDEFGHI])



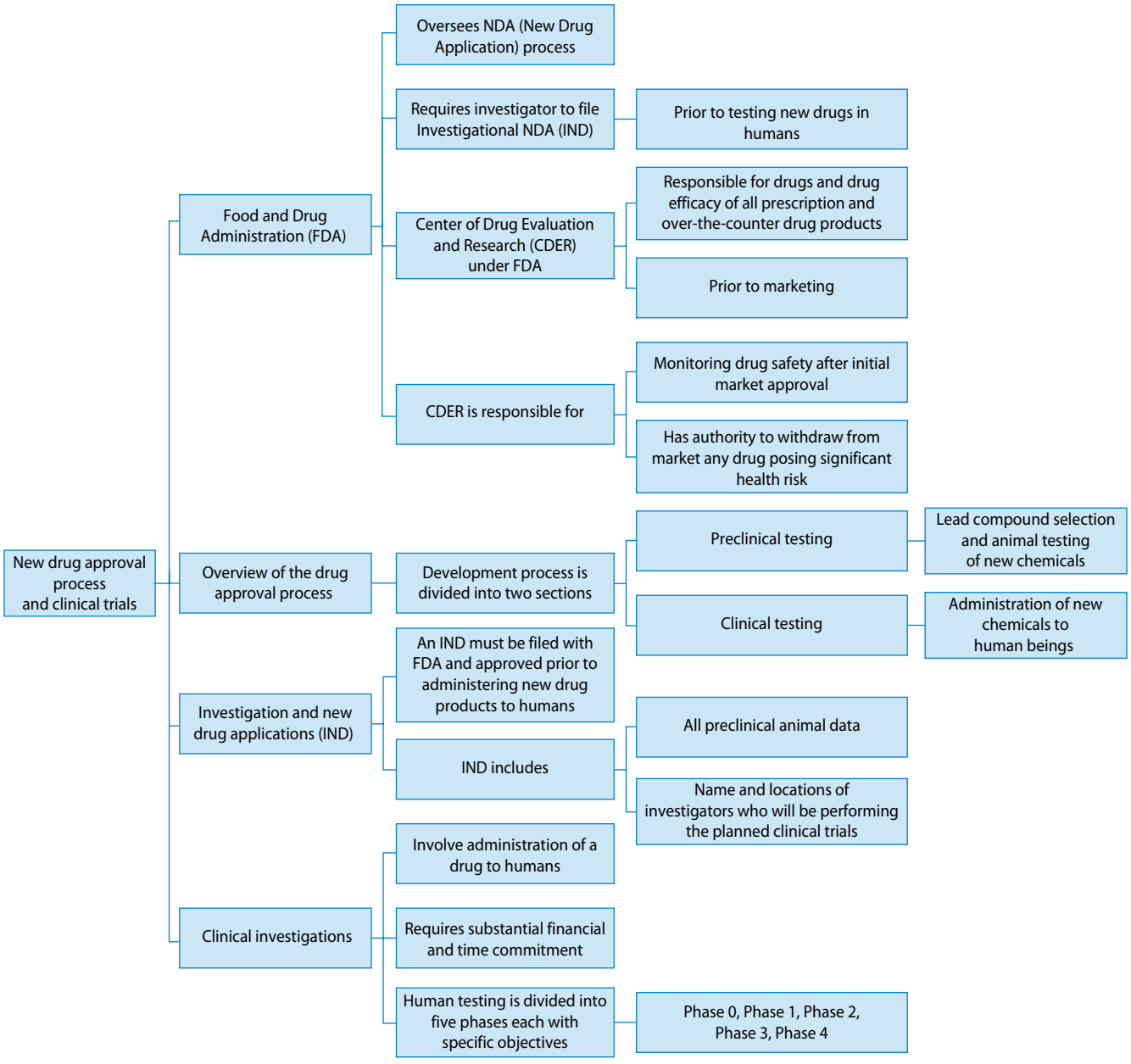
5.3 PHARMACOVIGILANCE



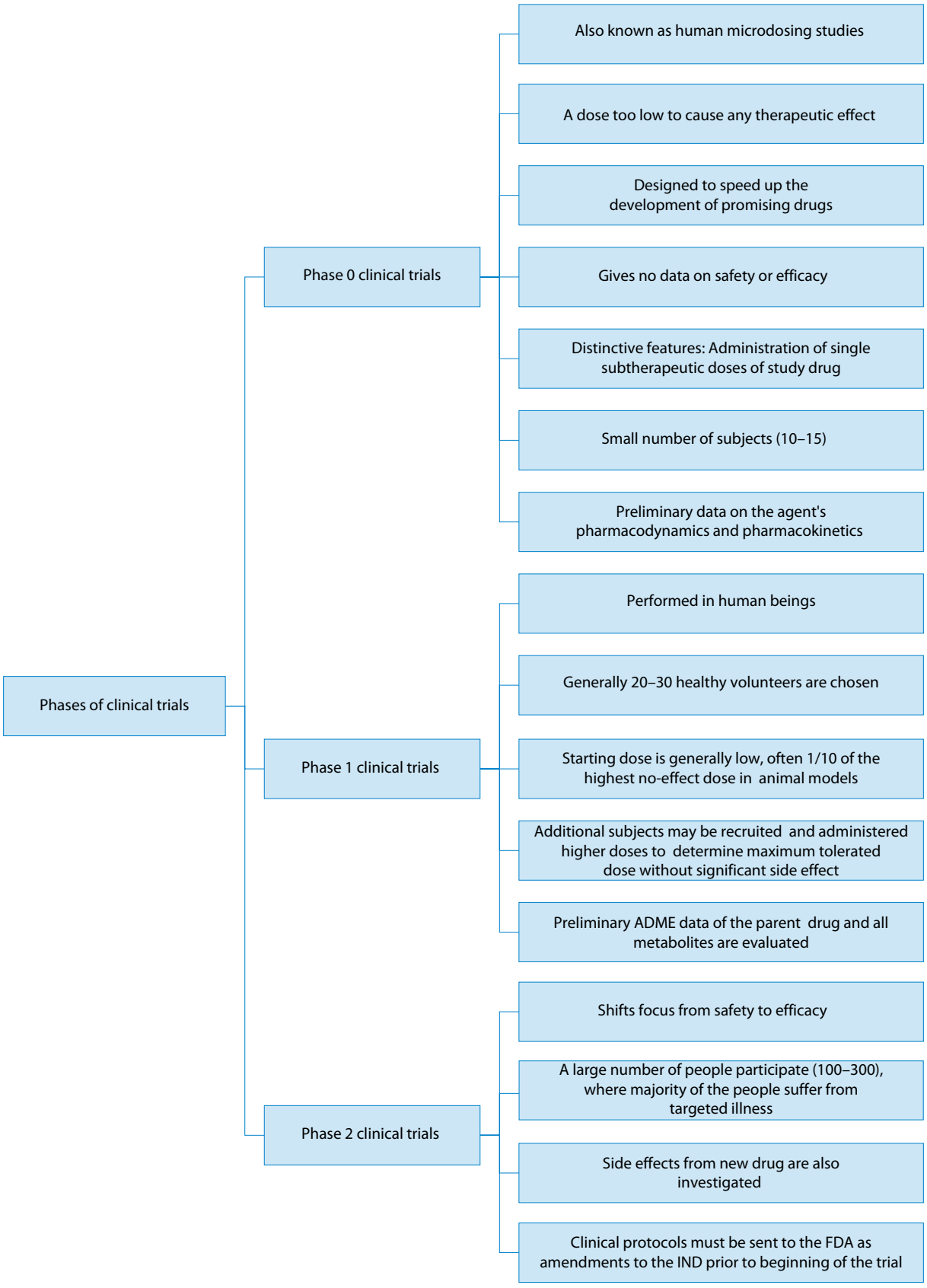
6

New drug approval process and clinical trials

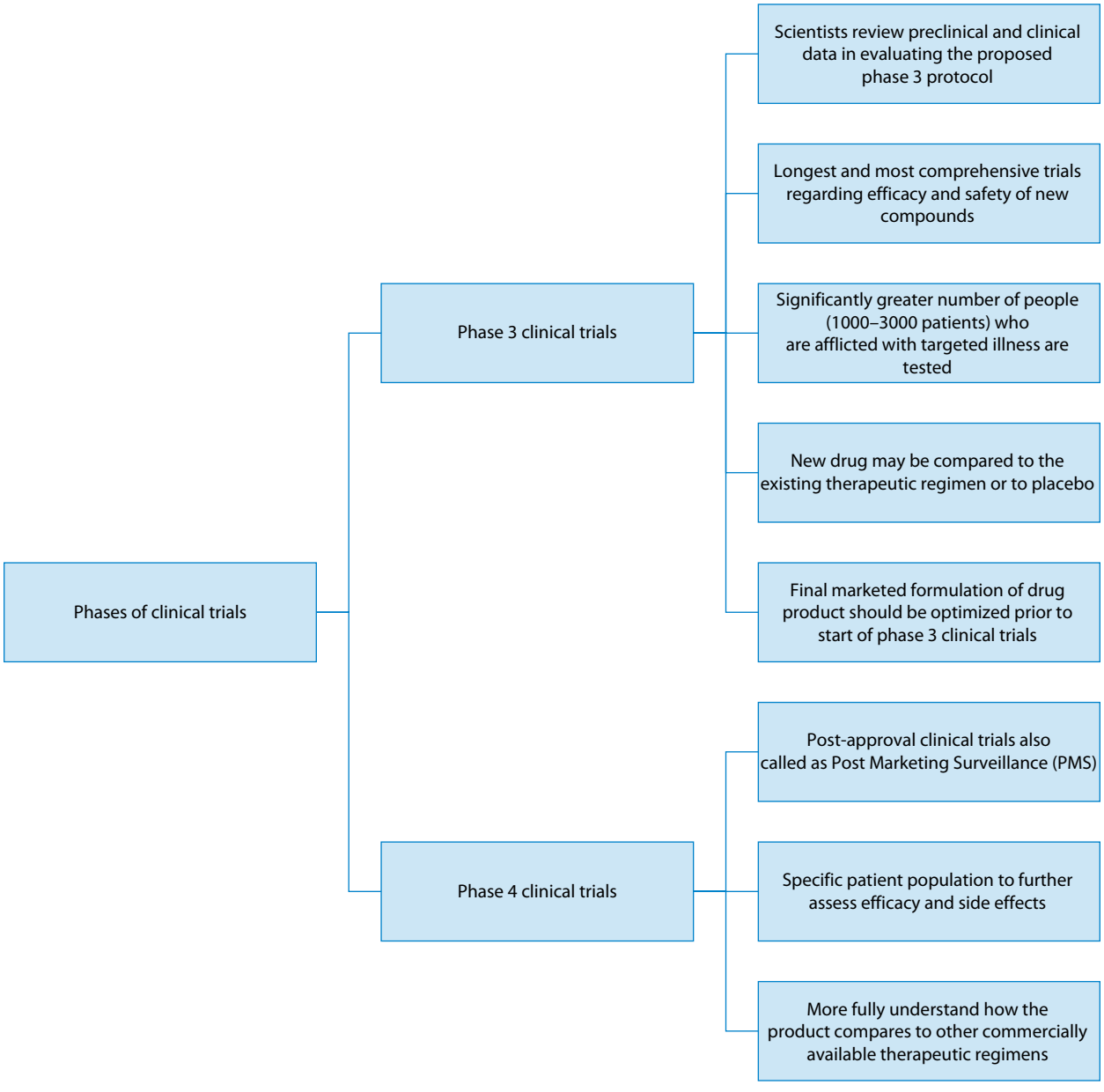
6.1 NEW DRUG APPROVAL PROCESS



6.2 PHASES OF CLINICAL TRIALS (0, 1 AND 2)



6.3 PHASES OF CLINICAL TRIALS (3 AND 4)



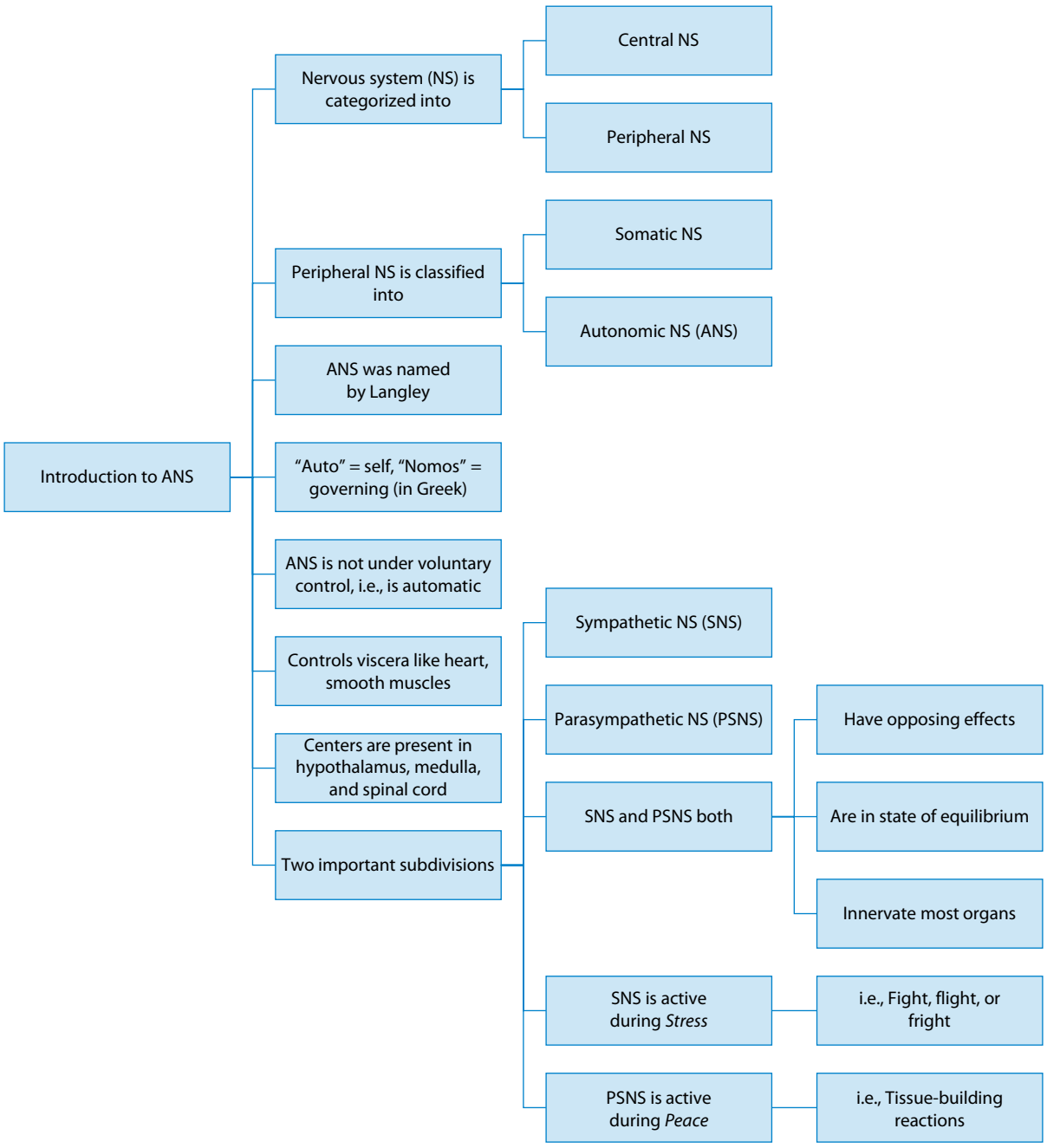
PART II

Autonomic nervous system (ANS) pharmacology

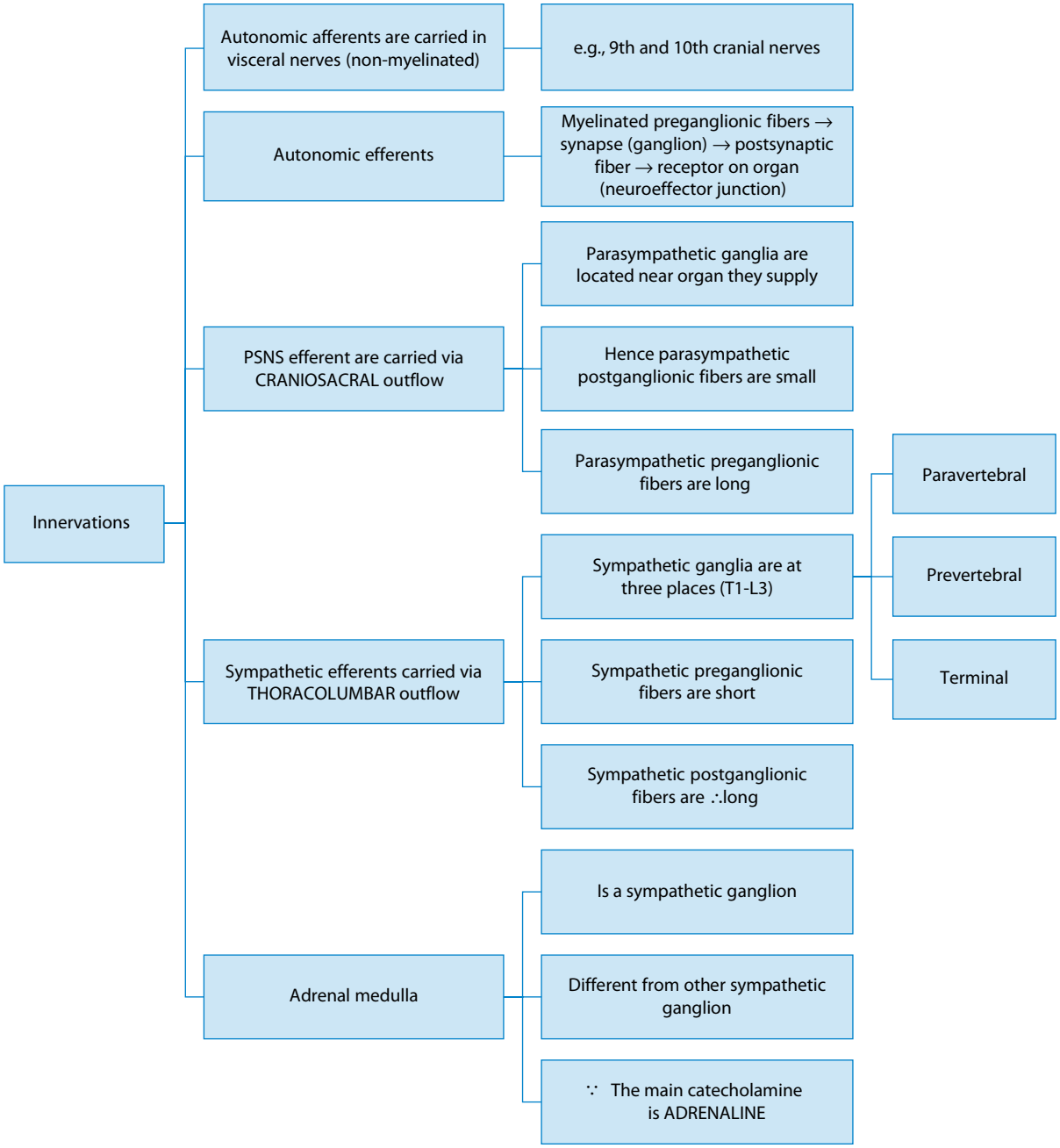
7

Introduction to ANS

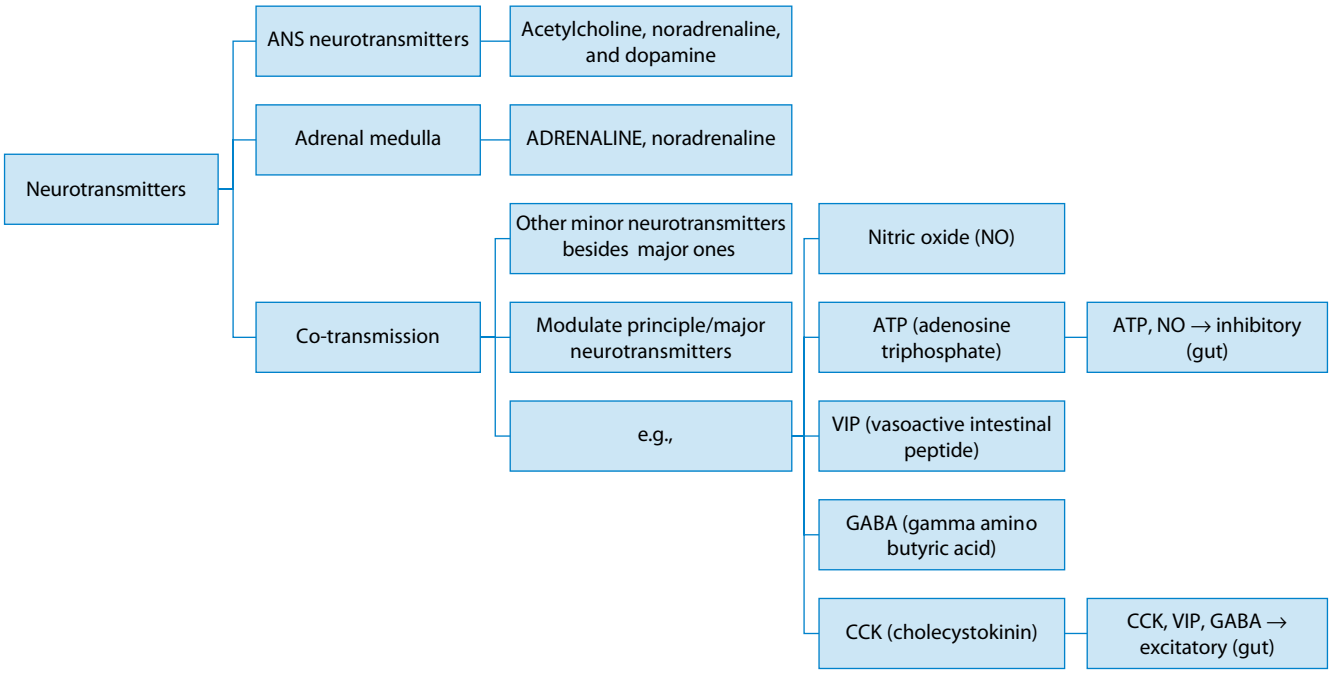
7.1 INTRODUCTION TO ANS



7.2 INNERVATIONS OF ANS



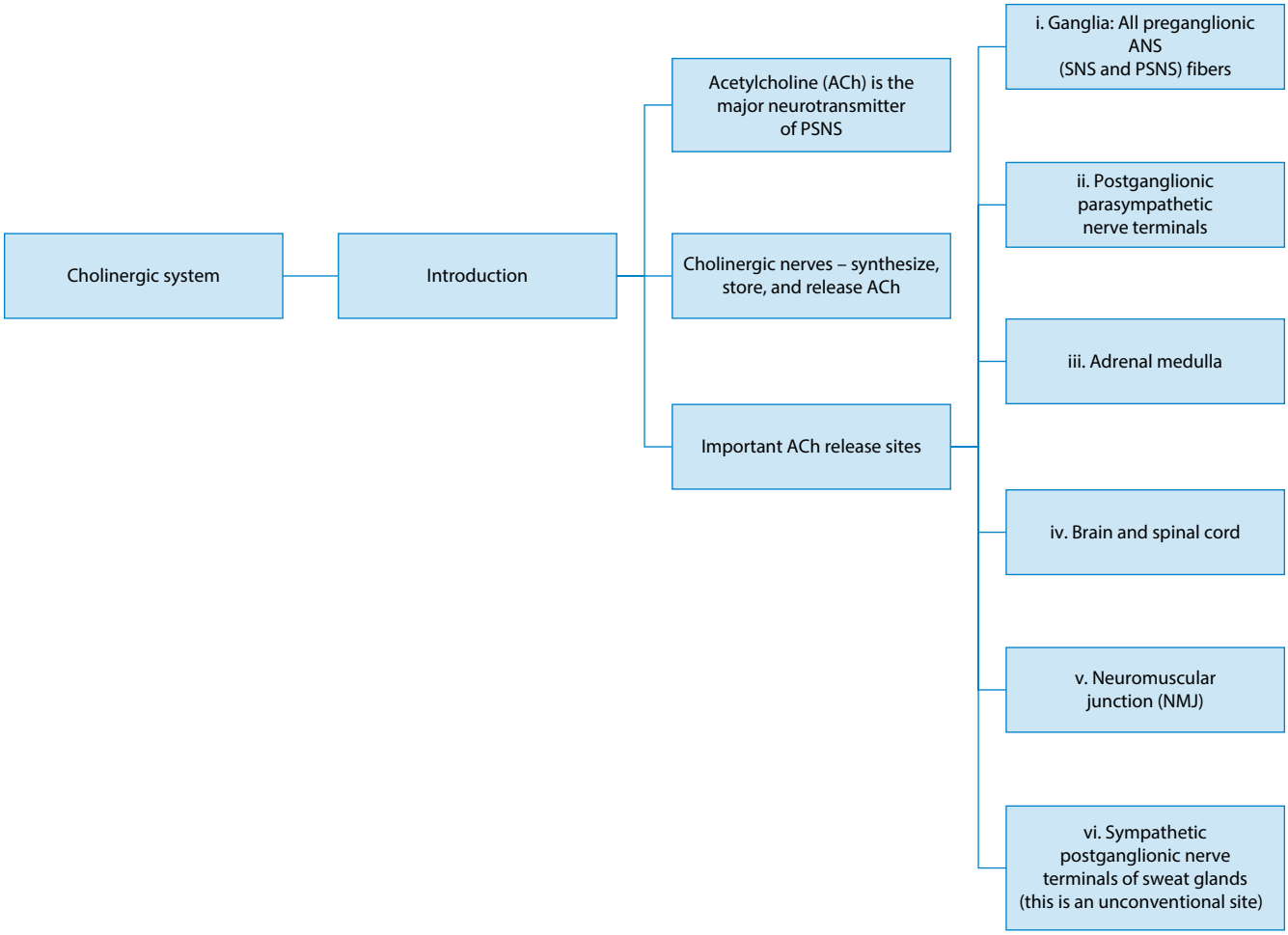
7.3 NEUROTRANSMITTERS



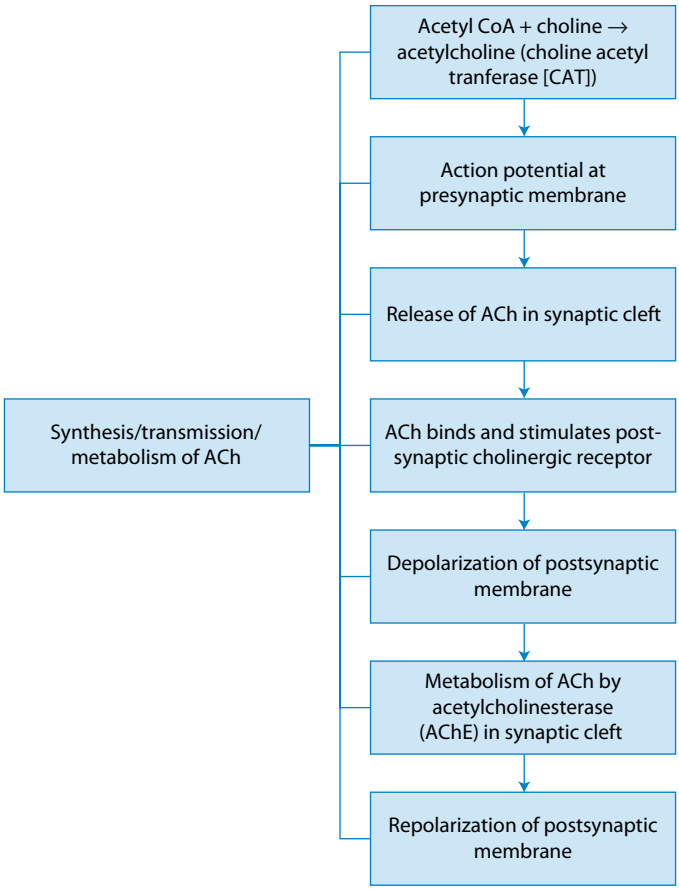
8

Cholinergic system and drugs

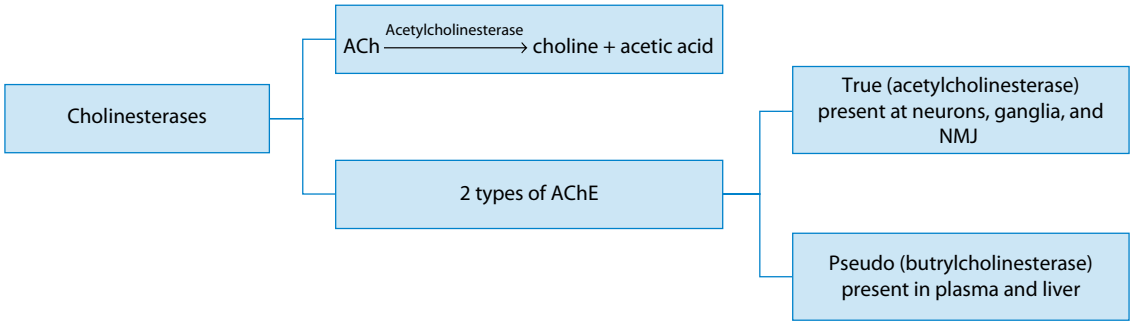
8.1 CHOLINERGIC SYSTEM



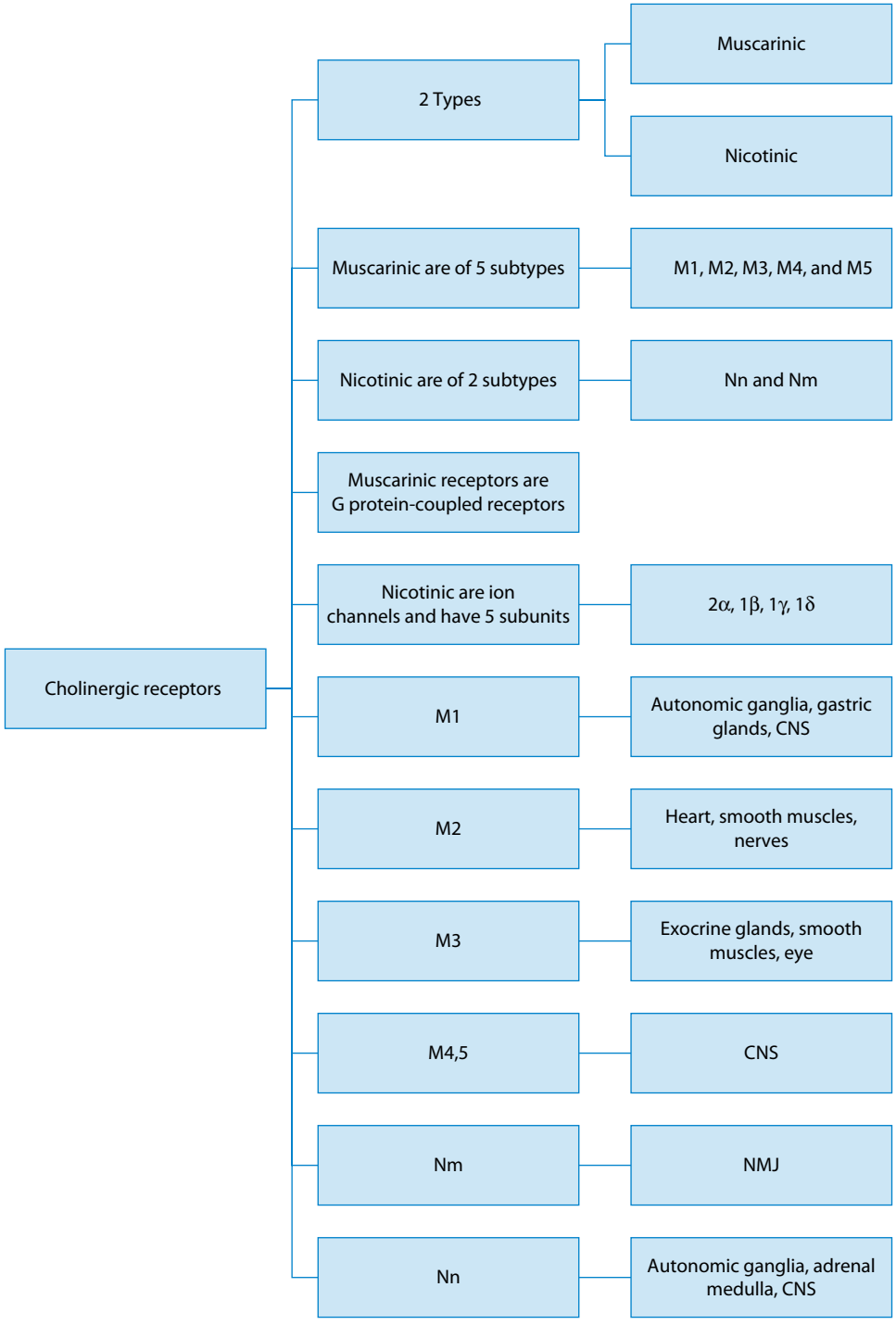
8.2 SYNTHESIS/TRANSMISSION/METABOLISM OF ACh



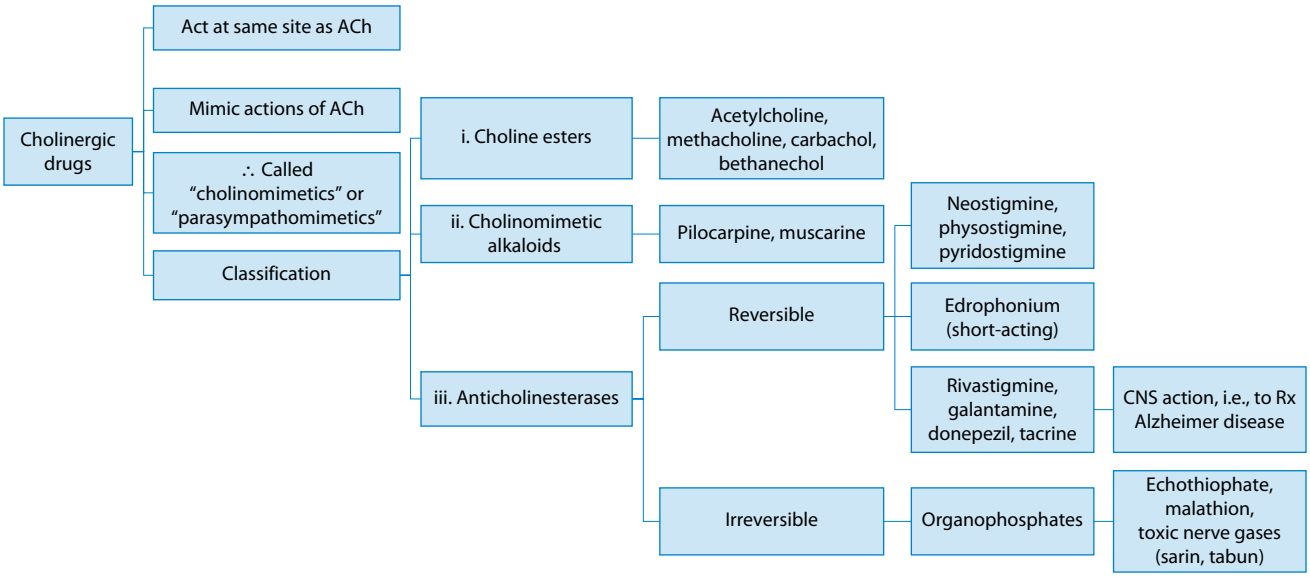
8.3 CHOLINESTERASES



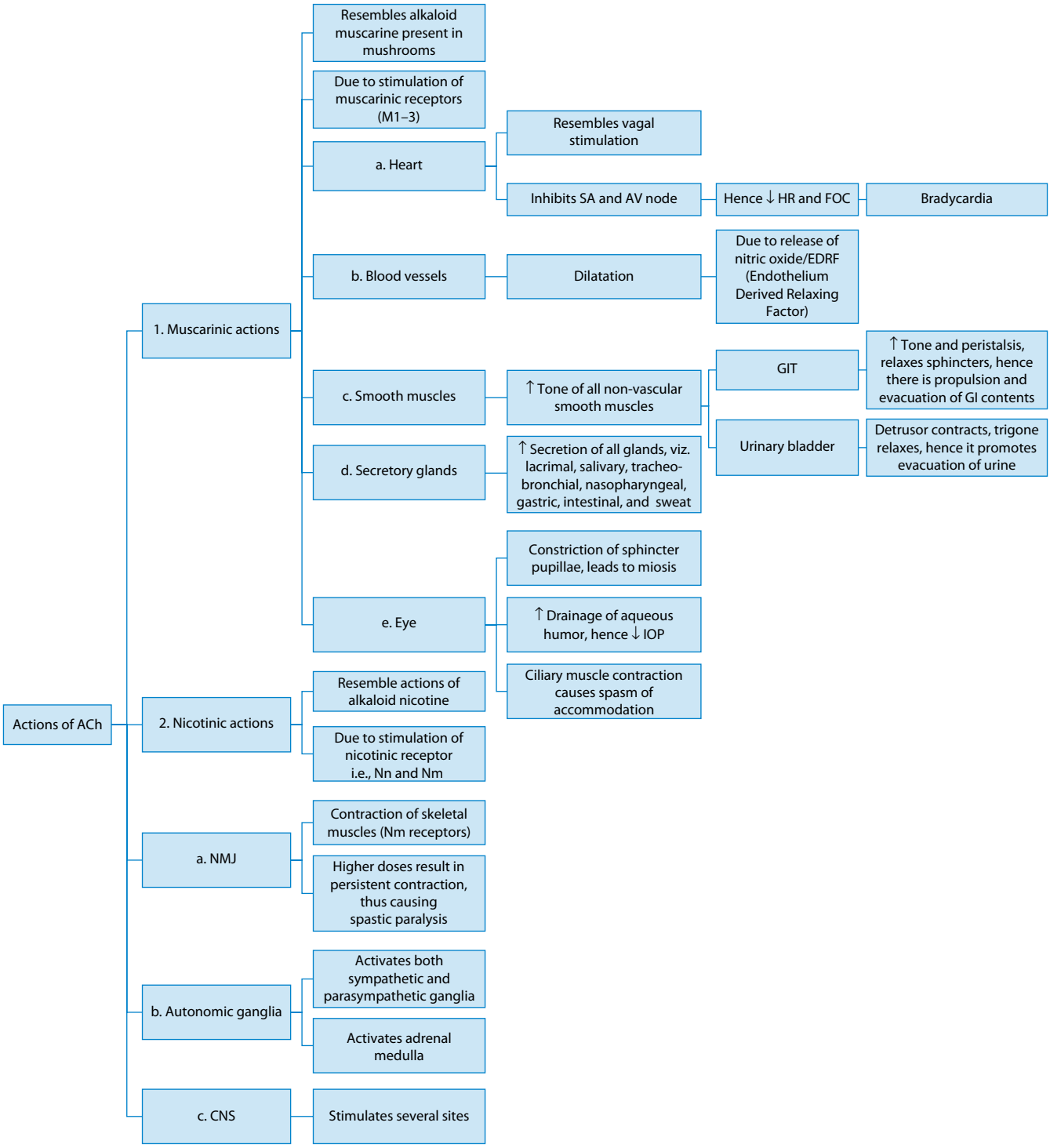
8.4 CHOLINERGIC RECEPTORS



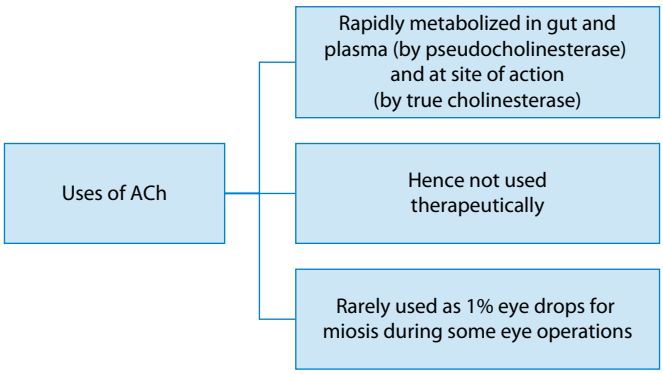
8.5 CHOLINERGIC DRUGS



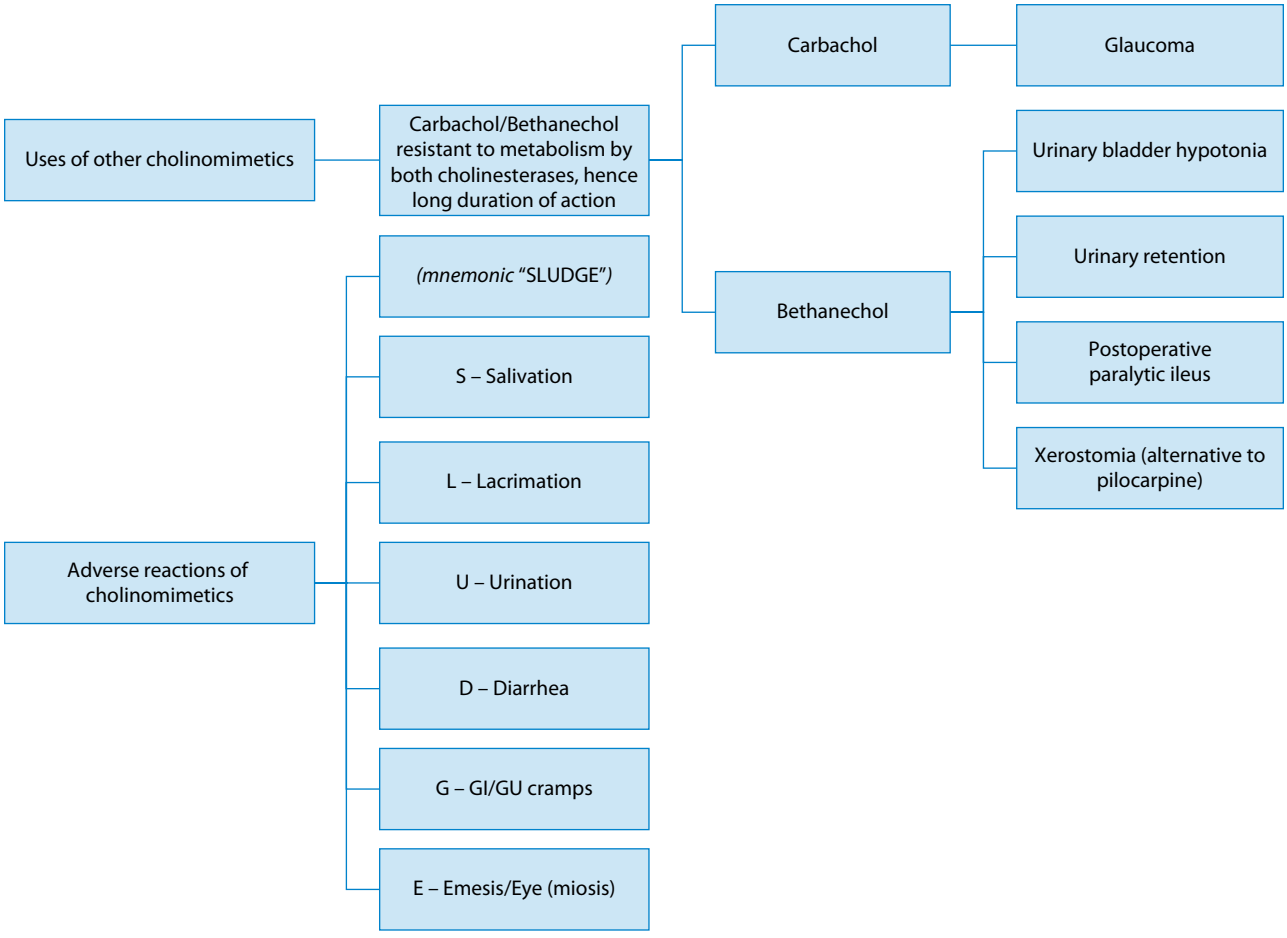
8.6 ACTIONS OF ACh



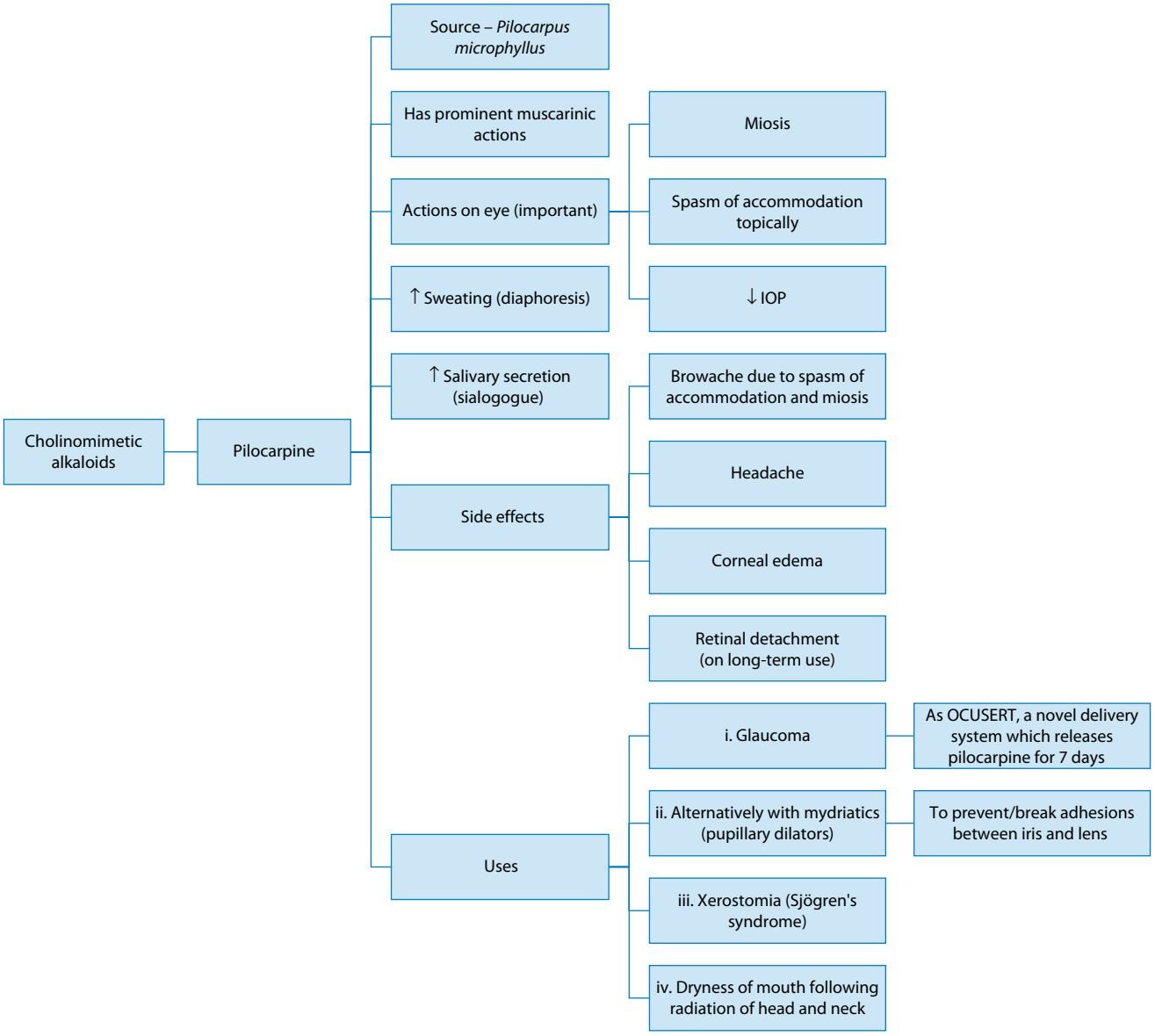
8.7 USES OF ACh AND CHOLINOMIMETICS



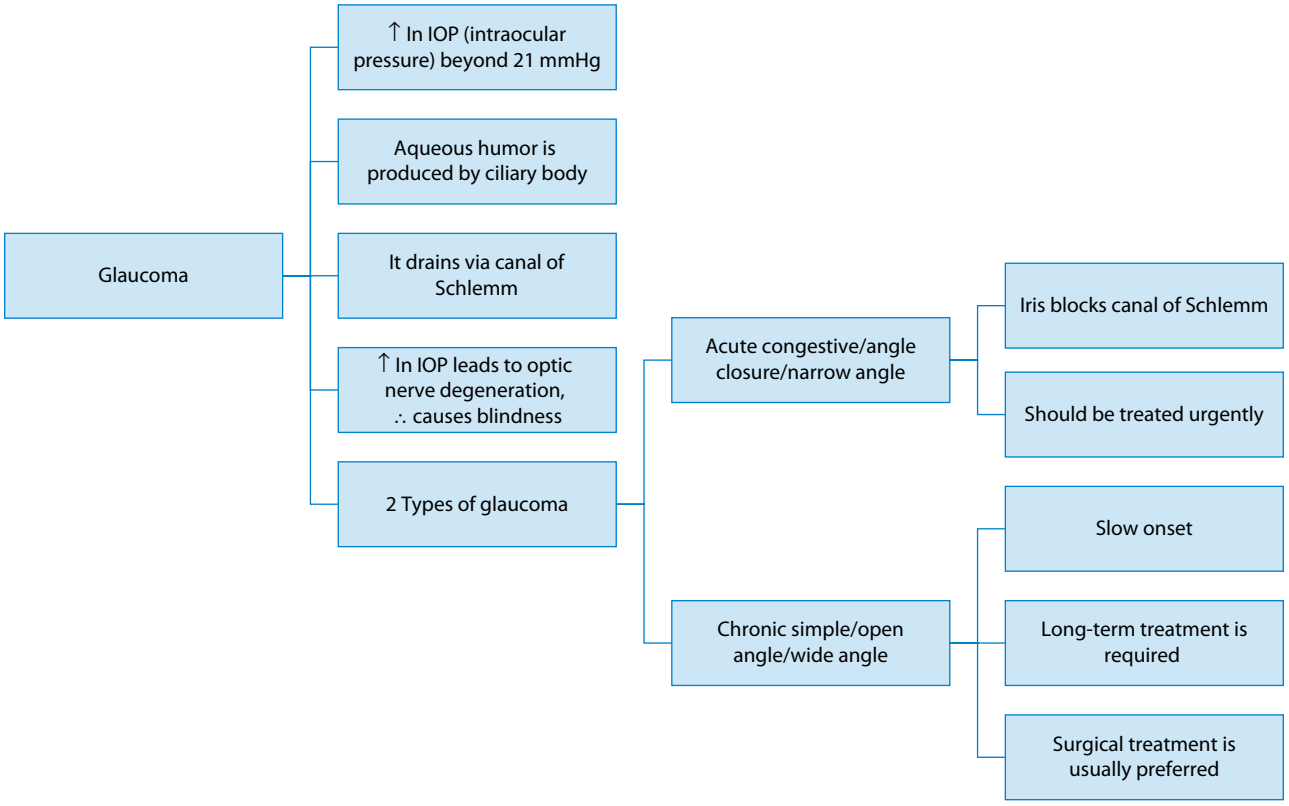
8.8 ADVERSE REACTIONS OF CHOLINOMIMETICS



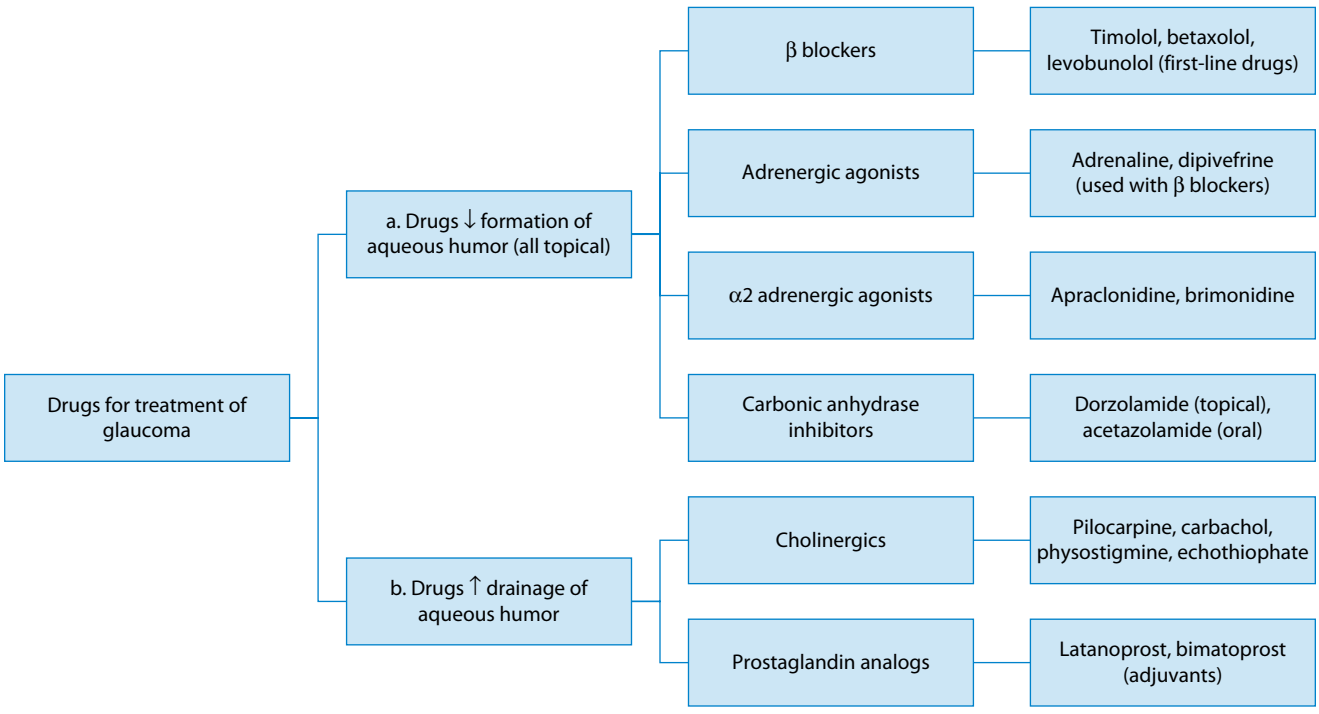
8.9 CHOLINOMIMETIC ALKALOIDS



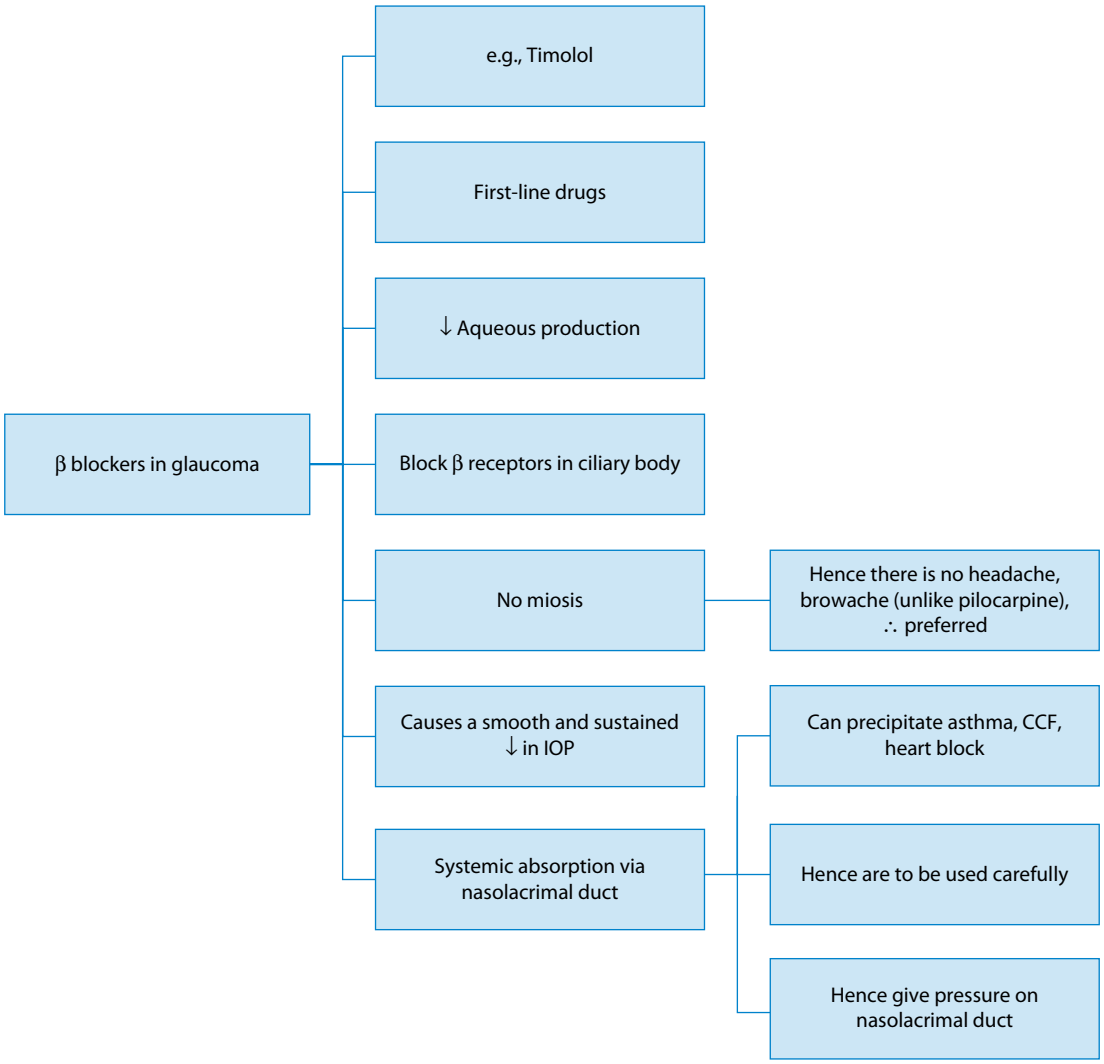
8.10 GLAUCOMA



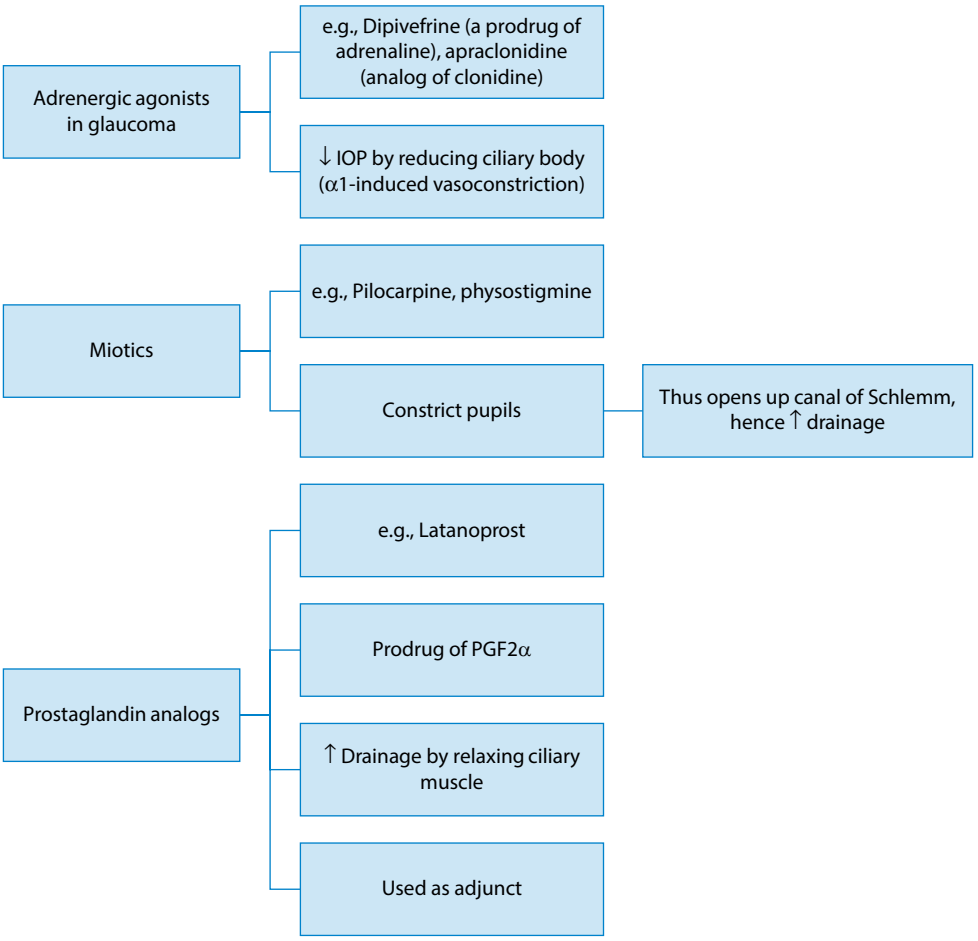
8.11 DRUGS FOR GLAUCOMA



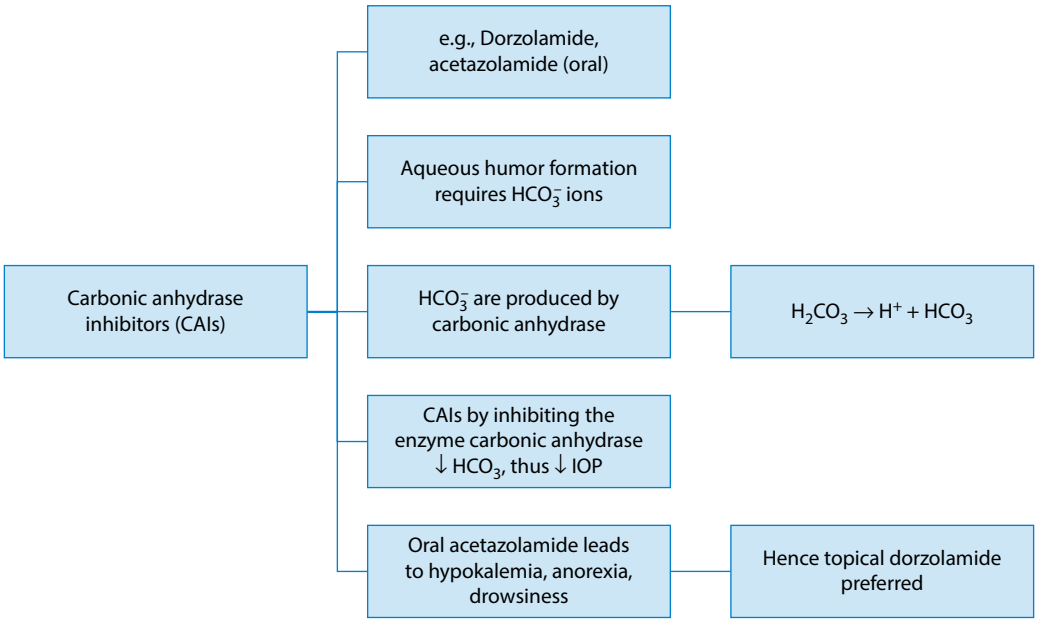
8.12 β BLOCKERS IN GLAUCOMA



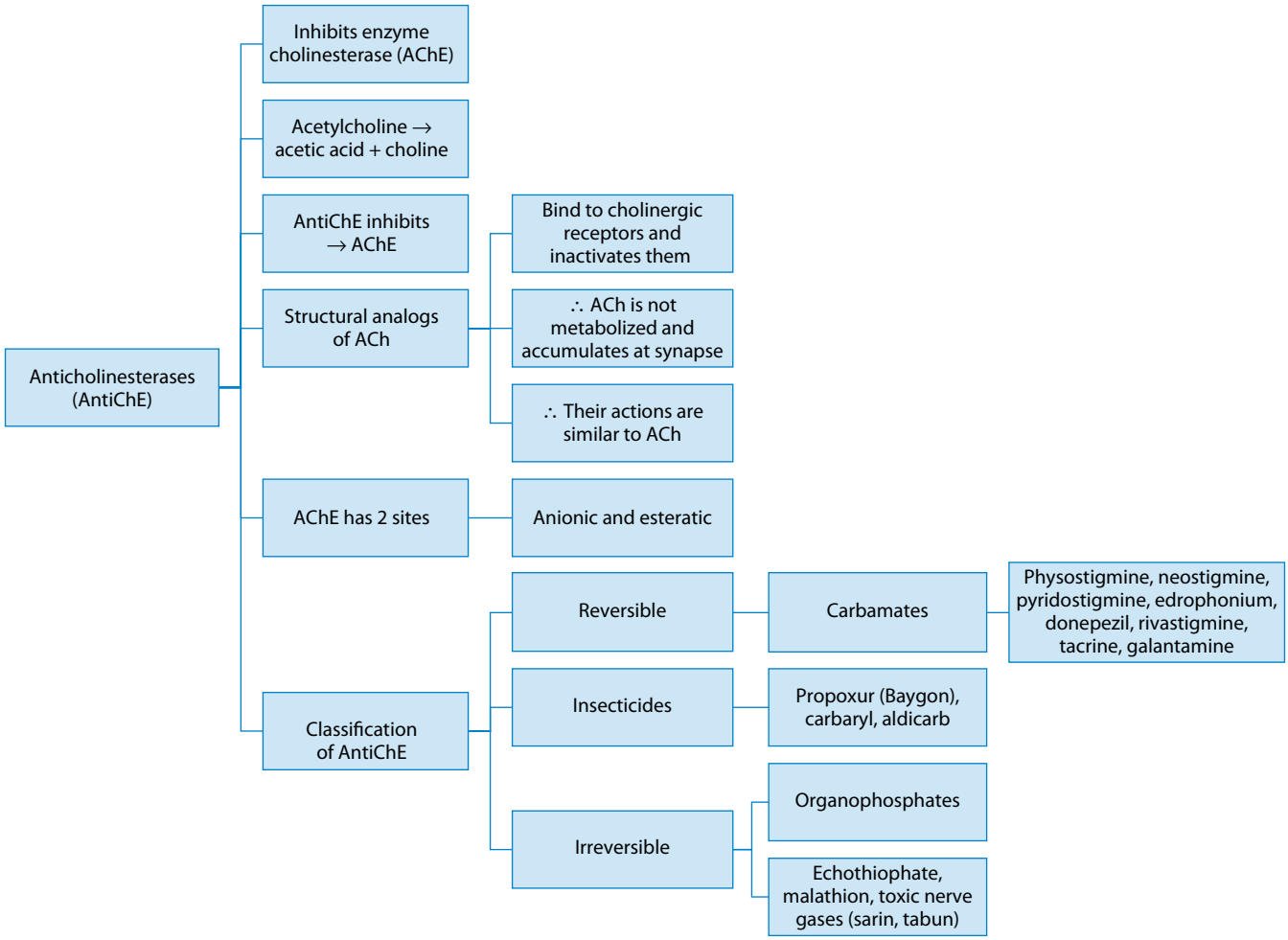
8.13 ADRENERGIC AGONISTS, MIOTICS, AND PROSTAGLANDIN ANALOGS IN GLAUCOMA



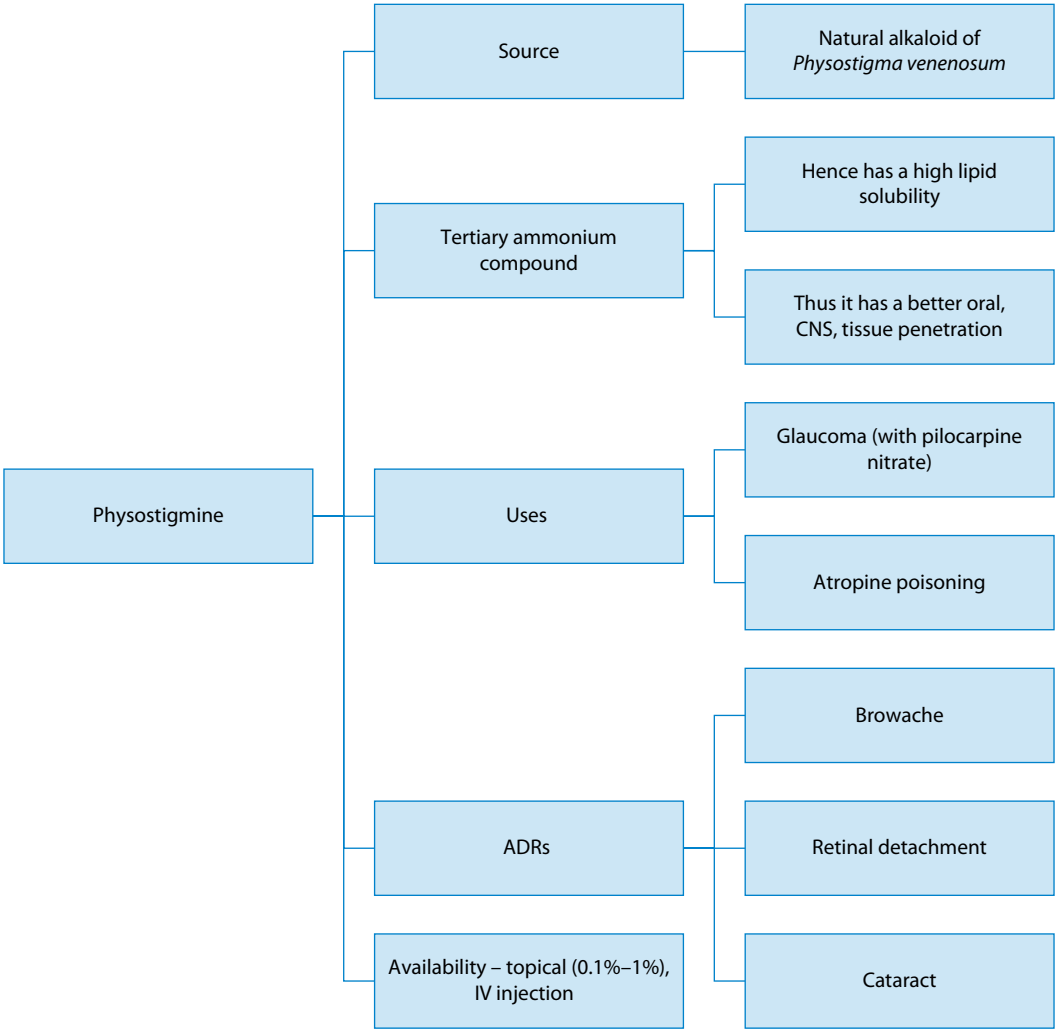
8.14 CARBONIC ANHYDRASE INHIBITORS (CAIs)



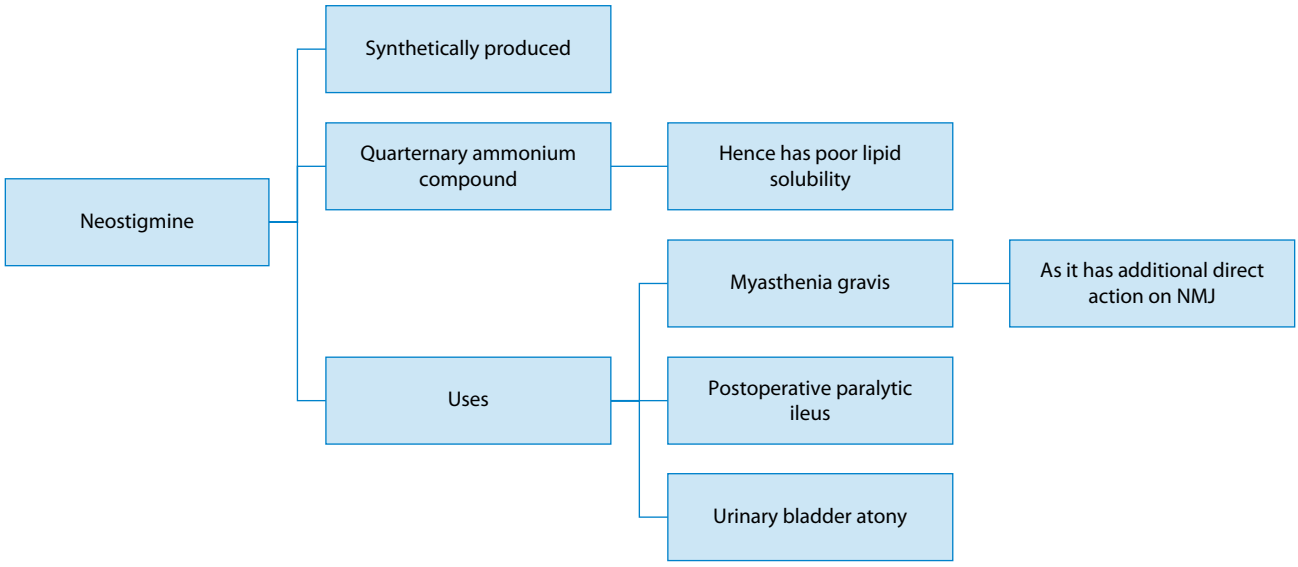
8.15 ANTICHOLINESTERASES (ANTIChE)



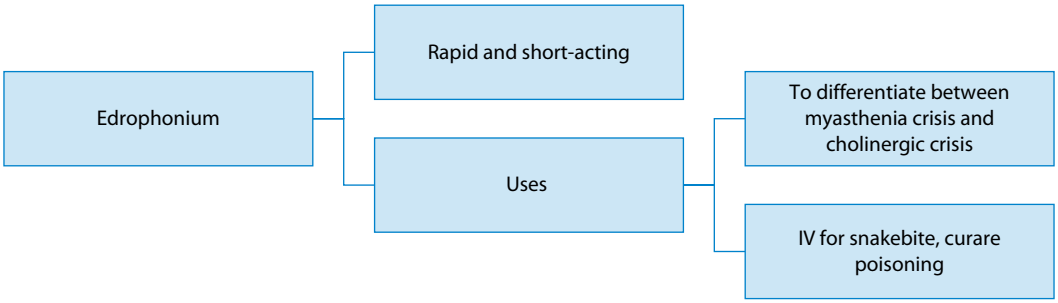
8.16 PHYSOSTIGMINE



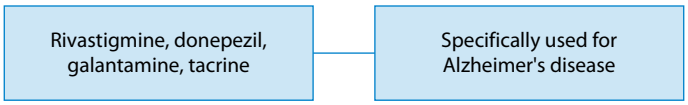
8.17 NEOSTIGMINE



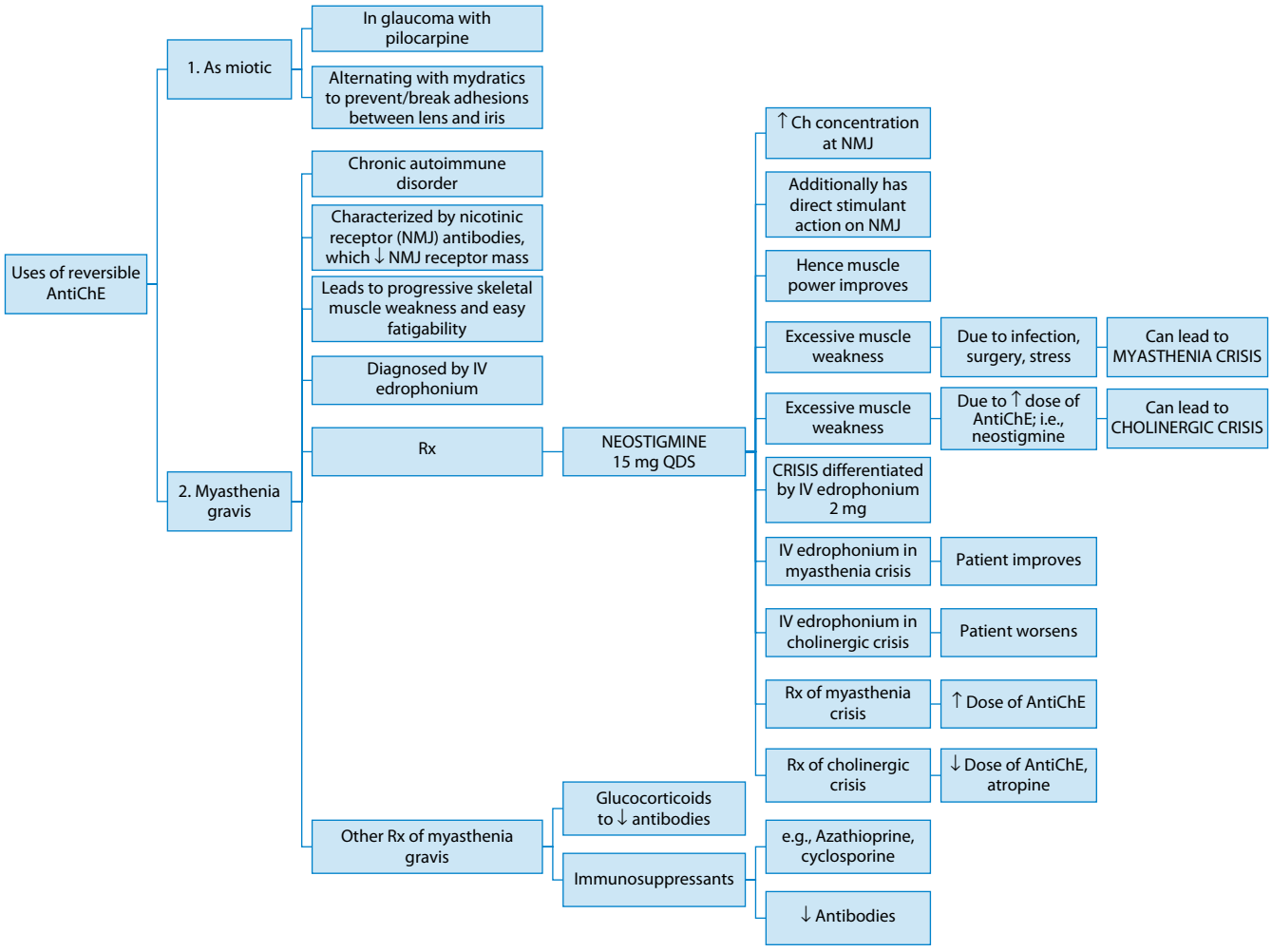
8.18 EDROPHONIUM



8.19 RIVASTIGMINE, DONEPEZIL, GALANTAMINE, TACRINE

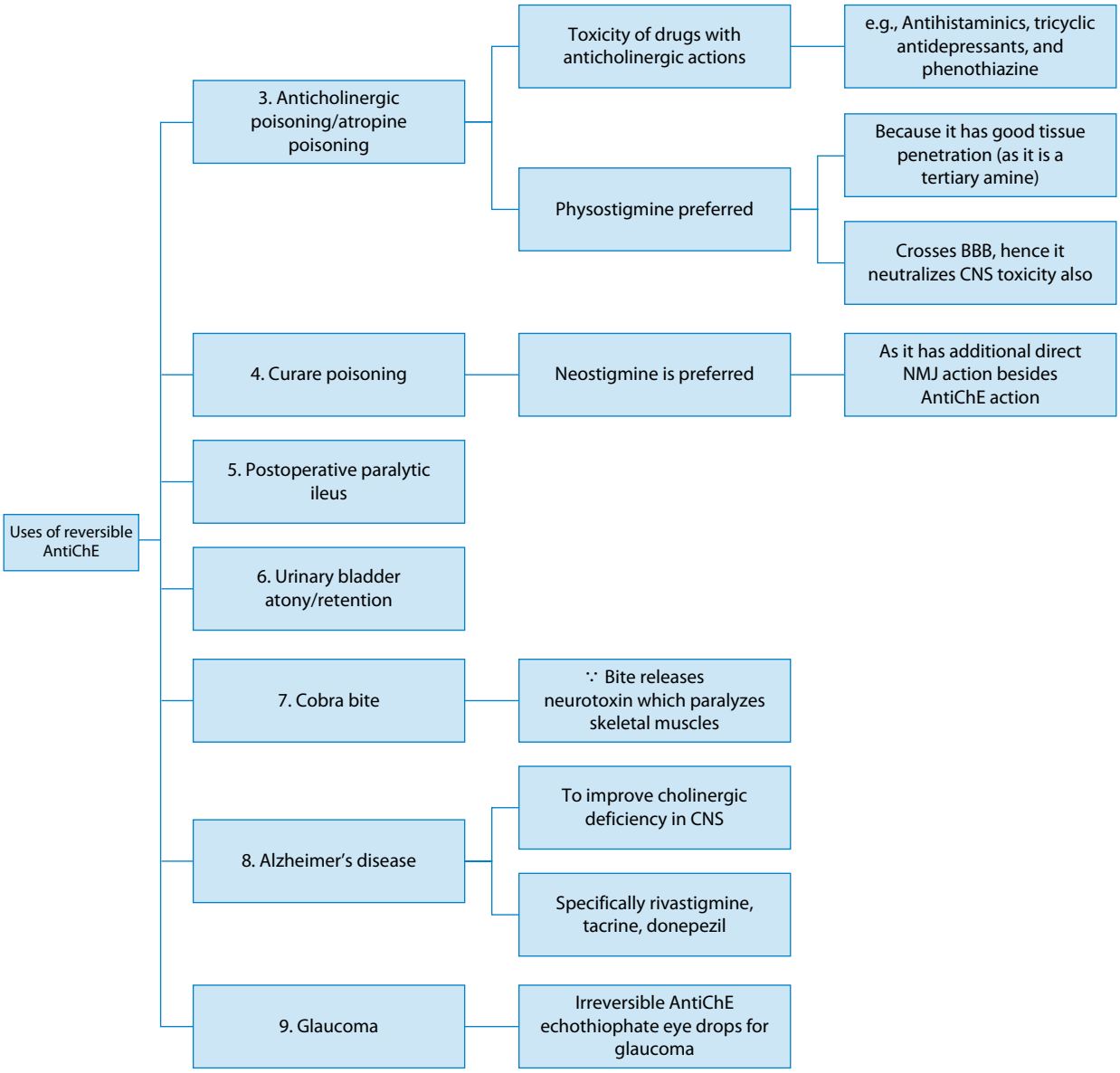


8.20 USES OF REVERSIBLE ANTIChE

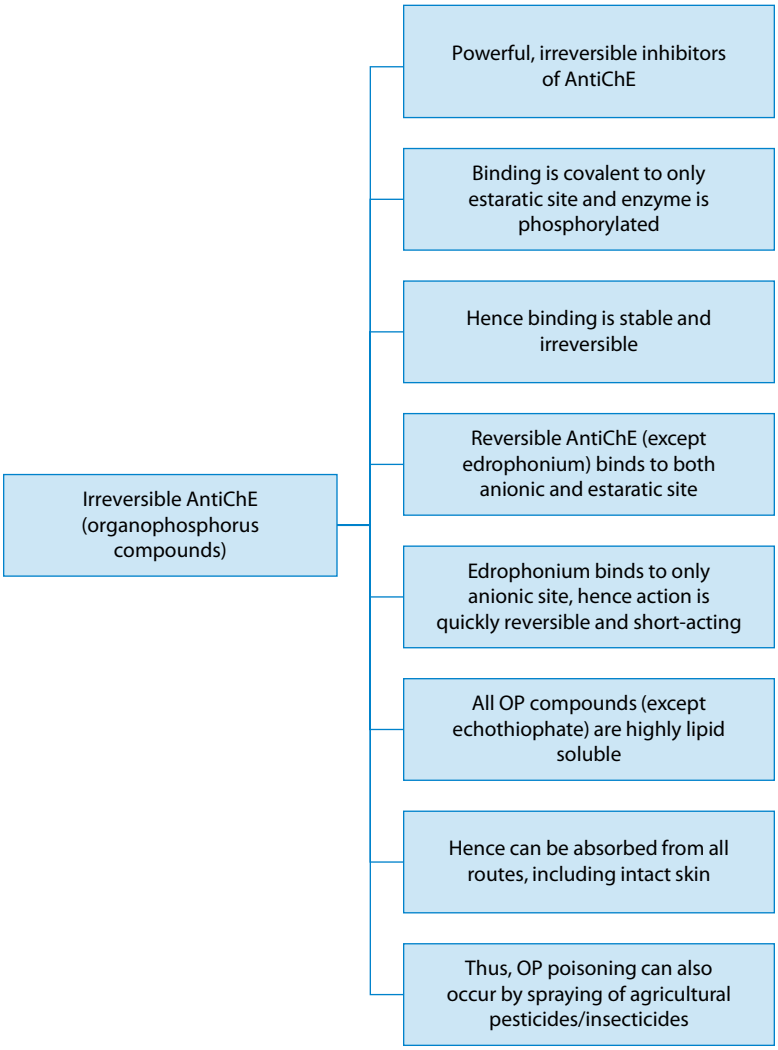


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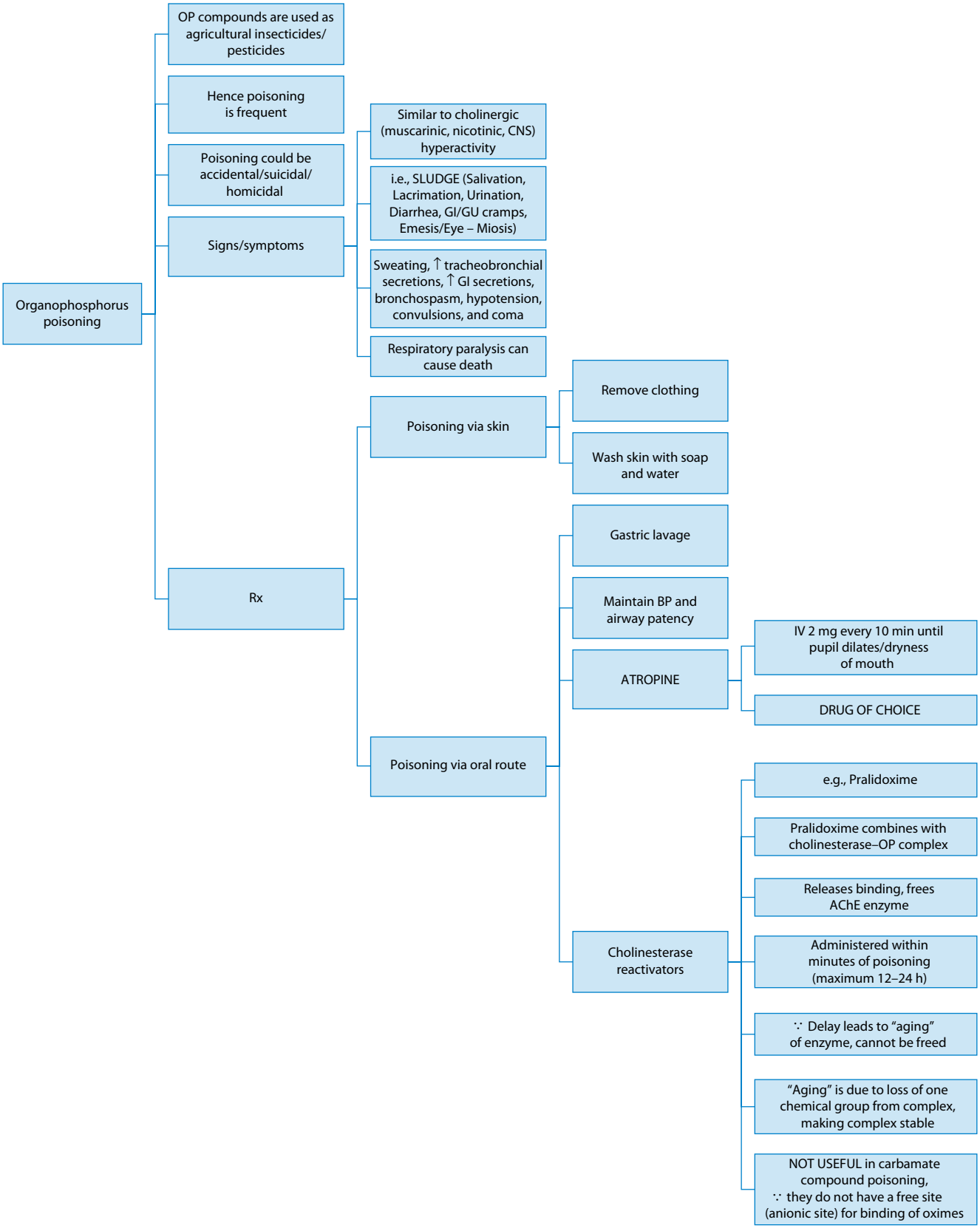
8.20 USES OF REVERSIBLE ANTIChE (Continued)



8.21 IRREVERSIBLE ANTIChE (ORGANOPHOSPHORUS COMPOUNDS)



8.22 ORGANOPHOSPHORUS POISONING



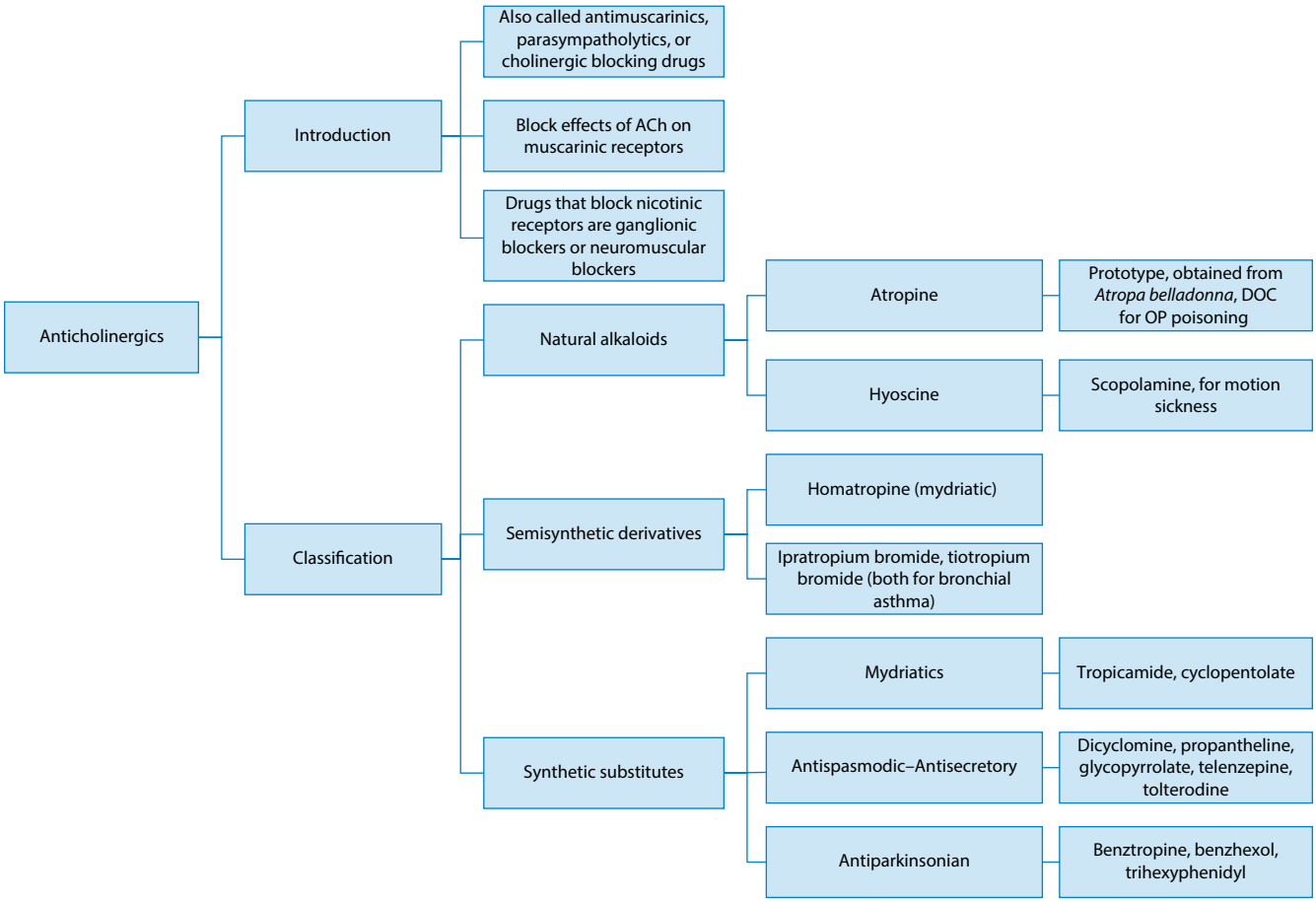
8.23 DIFFERENCES BETWEEN PHYSOSTIGMINE AND NEOSTIGMINE

Physostigmine	**Neostigmine**
1. Natural (*Physostigma venenosum*)	Synthetic
2. Tertiary amine	Quarternary amine
3. Good oral absorption	Poor oral absorption
4. Good tissue penetration	Poor tissue penetration
5. Crosses BBB: CNS effects	Does not cross BBB, no CNS effects
6. Main indication – glaucoma	Myasthenia gravis
7. Used in atropine poisoning	Used in curare poisoning

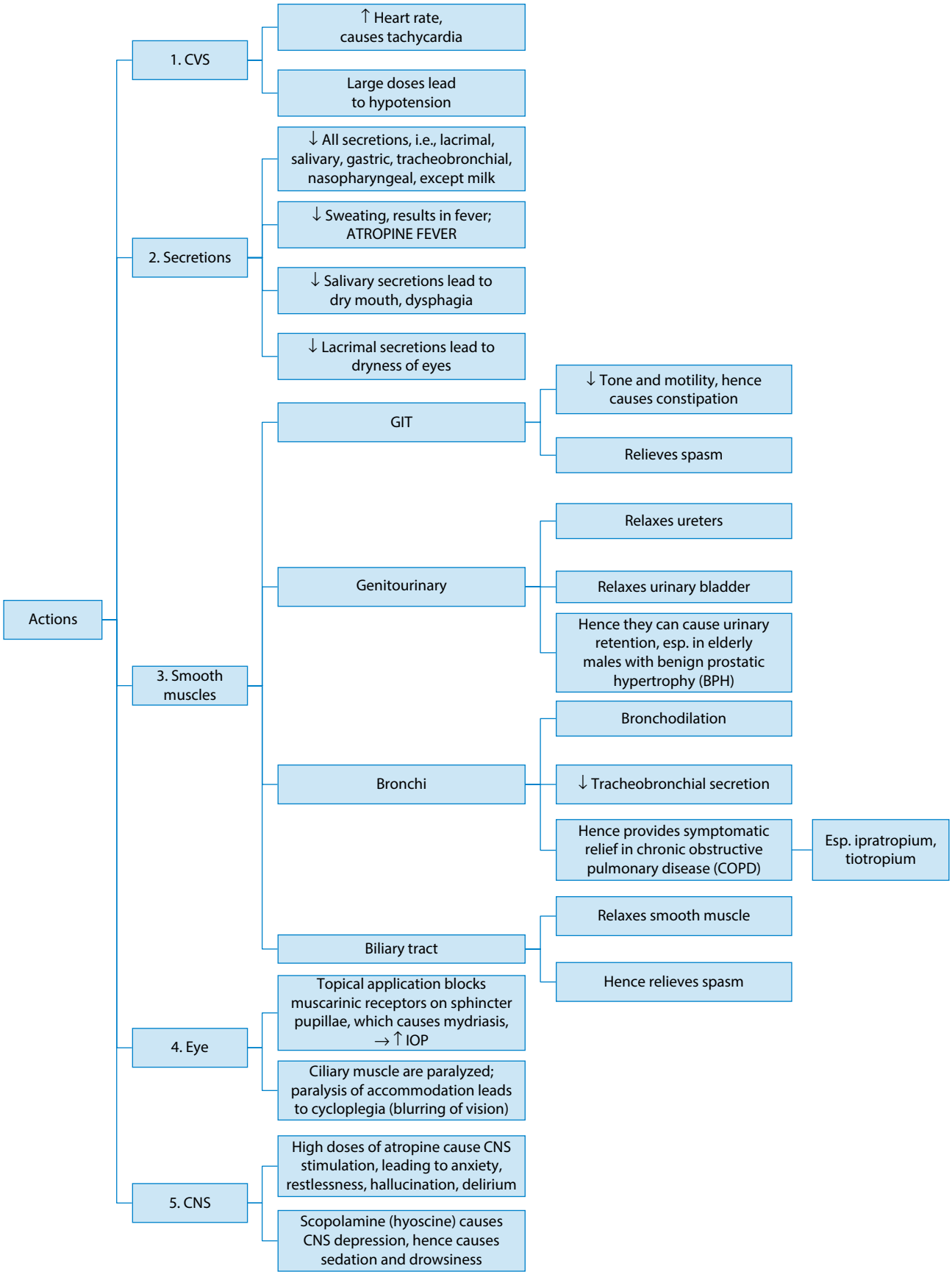
9

Anticholinergics

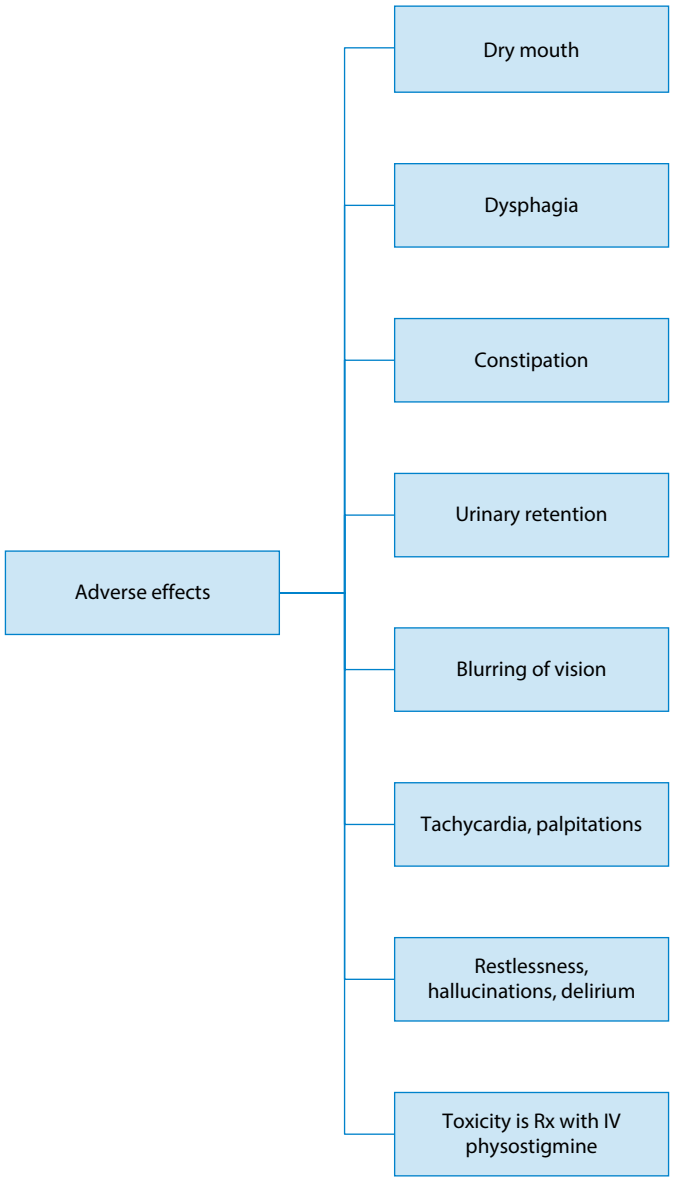
9.1 INTRODUCTION AND CLASSIFICATION



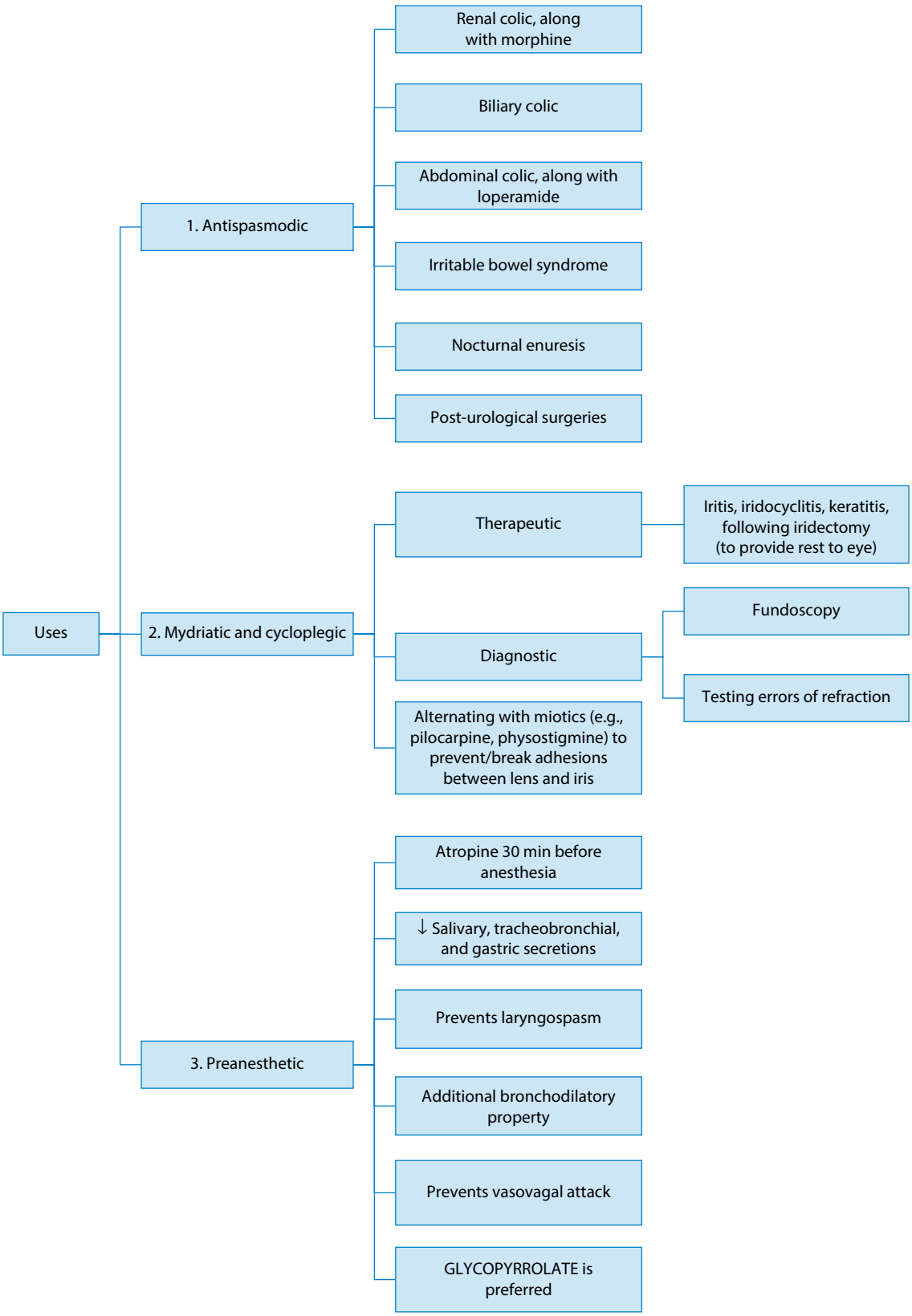
9.2 ACTIONS


Actions
1. CVS
↑ Heart rate, causes tachycardia
Large doses lead to hypotension
2. Secretions
↓ All secretions, i.e., lacrimal, salivary, gastric, tracheobronchial, nasopharyngeal, except milk
↓ Sweating, results in fever; ATROPINE FEVER
↓ Salivary secretions lead to dry mouth, dysphagia
↓ Lacrimal secretions lead to dryness of eyes
3. Smooth muscles
GIT
↓ Tone and motility, hence causes constipation
Relieves spasm
Genitourinary
Relaxes ureters
Relaxes urinary bladder
Hence they can cause urinary retention, esp. in elderly males with benign prostatic hypertrophy (BPH)
Bronchi
Bronchodilation
↓ Tracheobronchial secretion
Hence provides symptomatic relief in chronic obstructive pulmonary disease (COPD)
Esp. ipratropium, tiotropium
Biliary tract
Relaxes smooth muscle
Hence relieves spasm
4. Eye
Topical application blocks muscarinic receptors on sphincter pupillae, which causes mydriasis, → ↑ IOP
Ciliary muscle are paralyzed; paralysis of accommodation leads to cycloplegia (blurring of vision)
5. CNS
High doses of atropine cause CNS stimulation, leading to anxiety, restlessness, hallucination, delirium
Scopolamine (hyoscine) causes CNS depression, hence causes sedation and drowsiness

9.3 ADVERSE EFFECTS

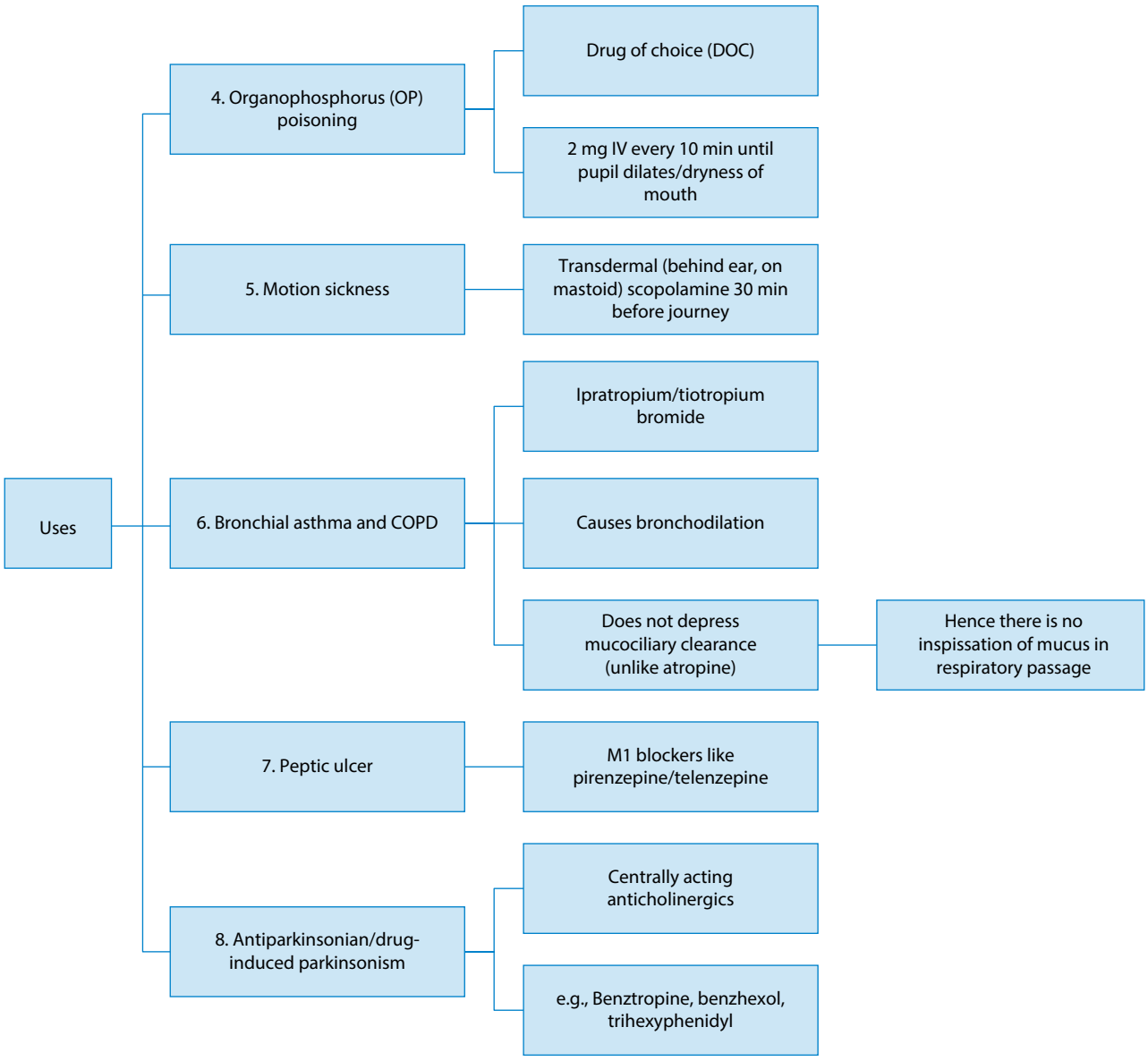


9.4 USES



(*Continued*)

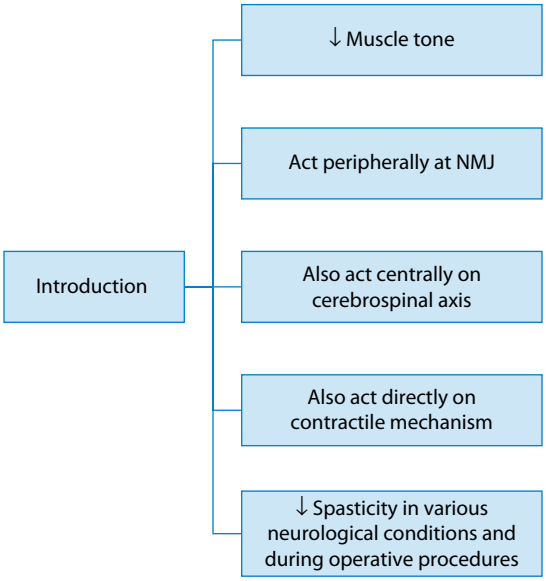
9.4 USES (Continued)



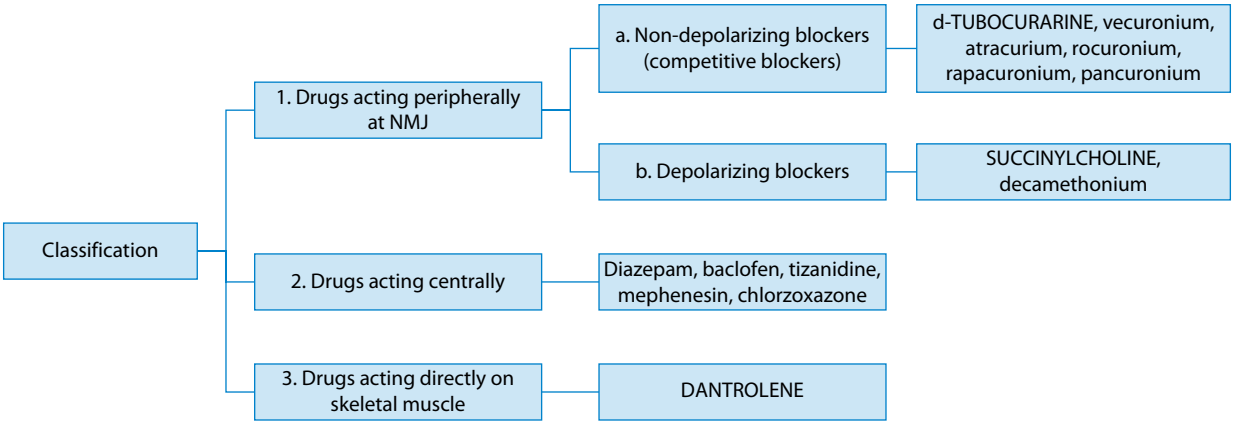
10

Skeletal muscle relaxants

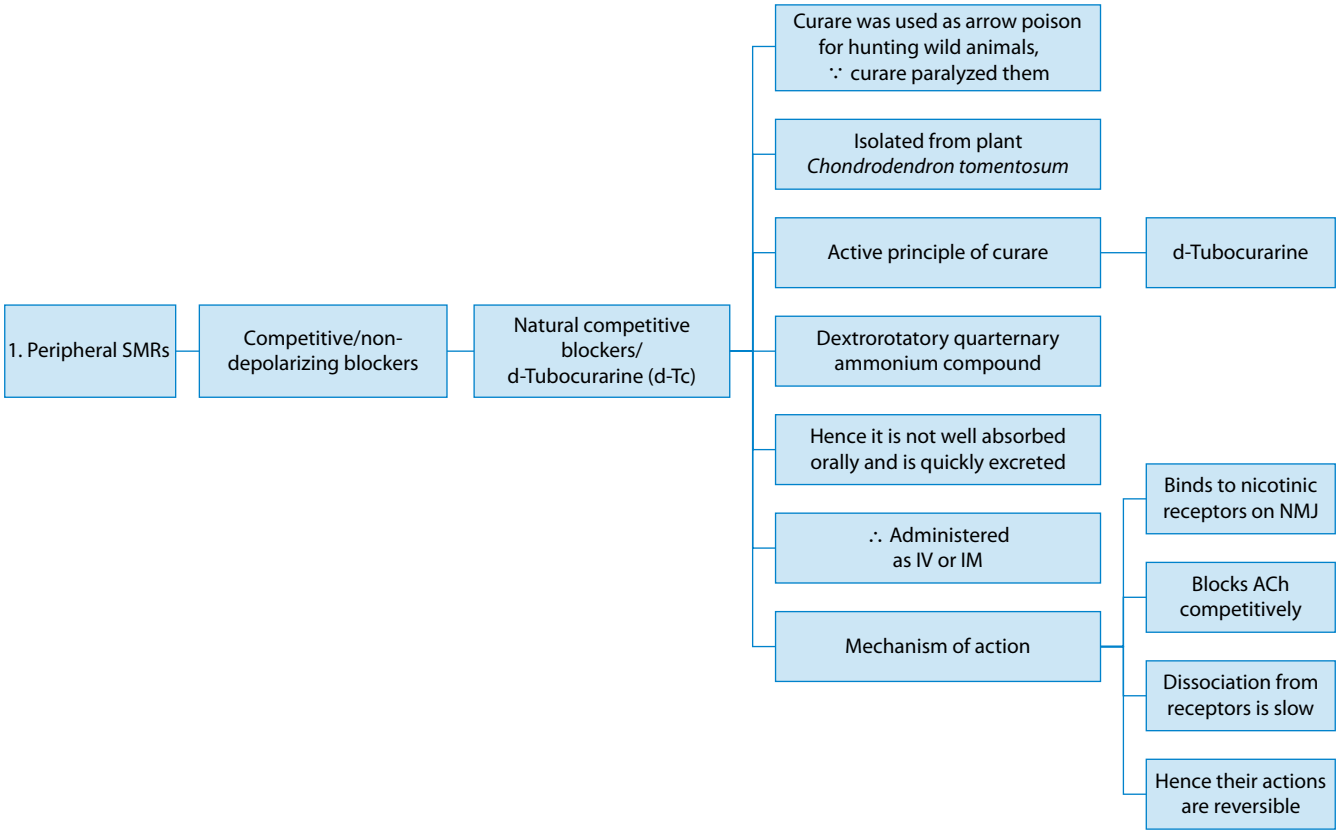
10.1 INTRODUCTION



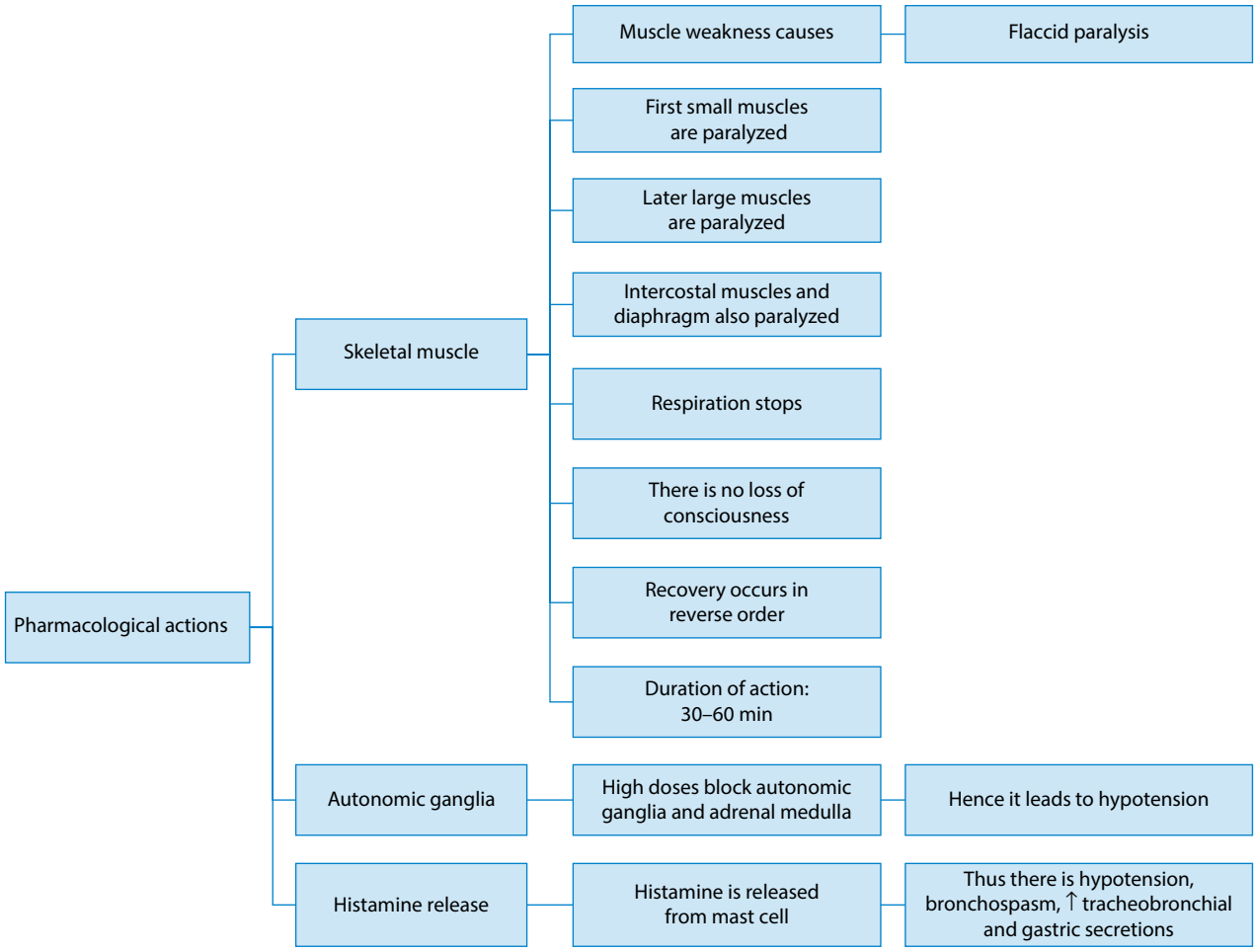
10.2 CLASSIFICATION



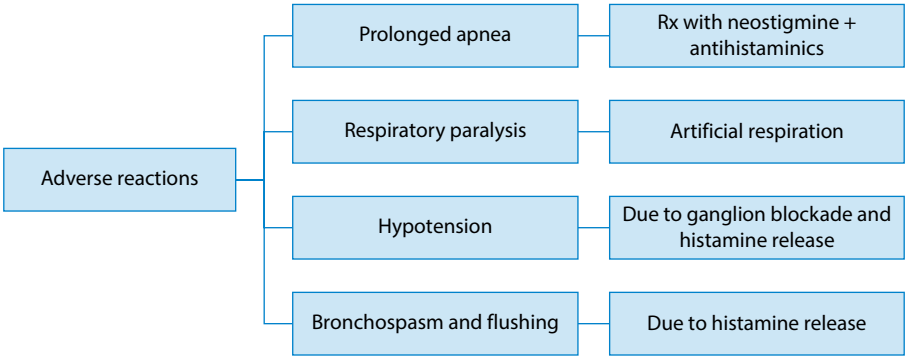
10.3 PERIPHERAL SMRs



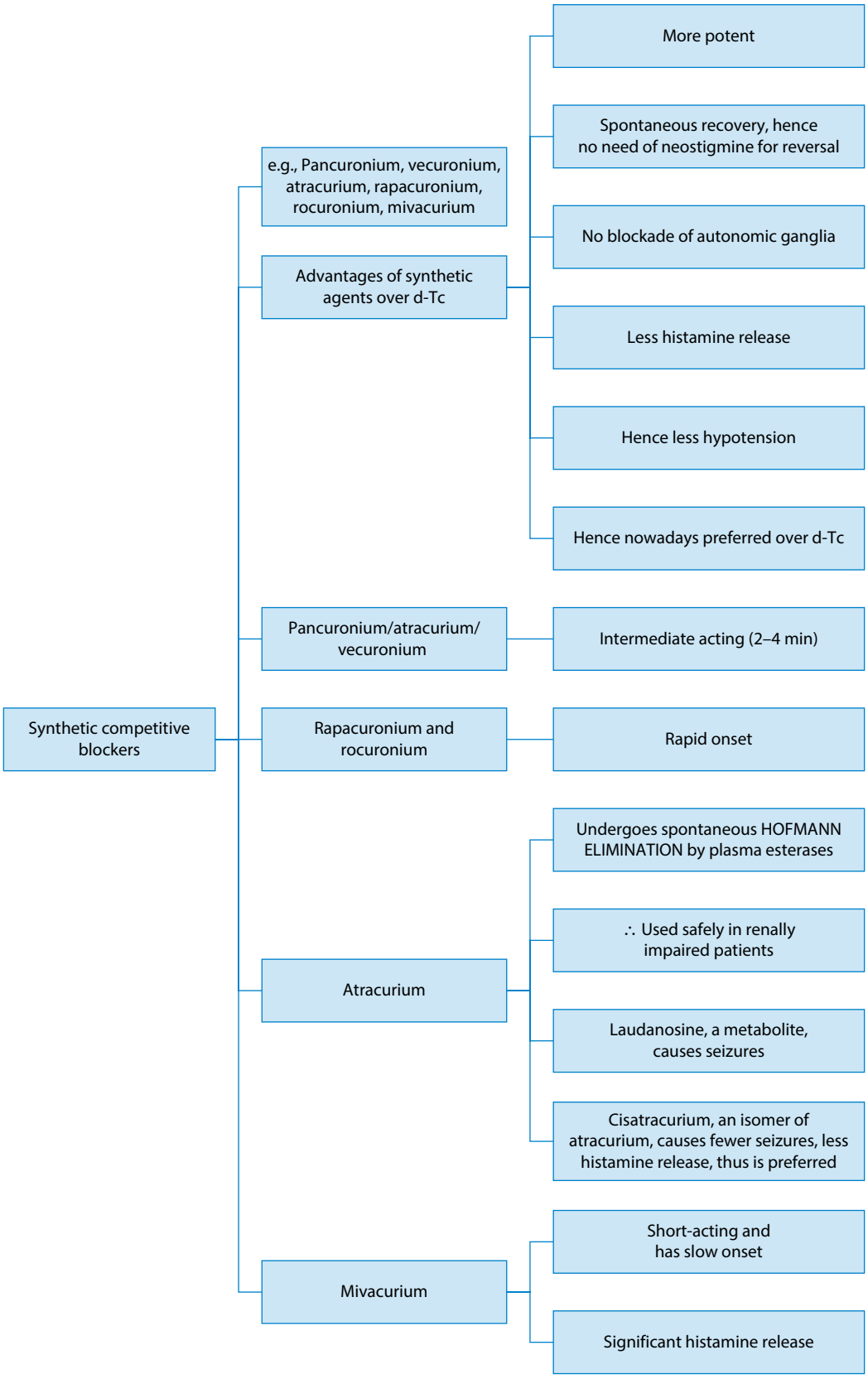
10.4 PHARMACOLOGICAL ACTIONS



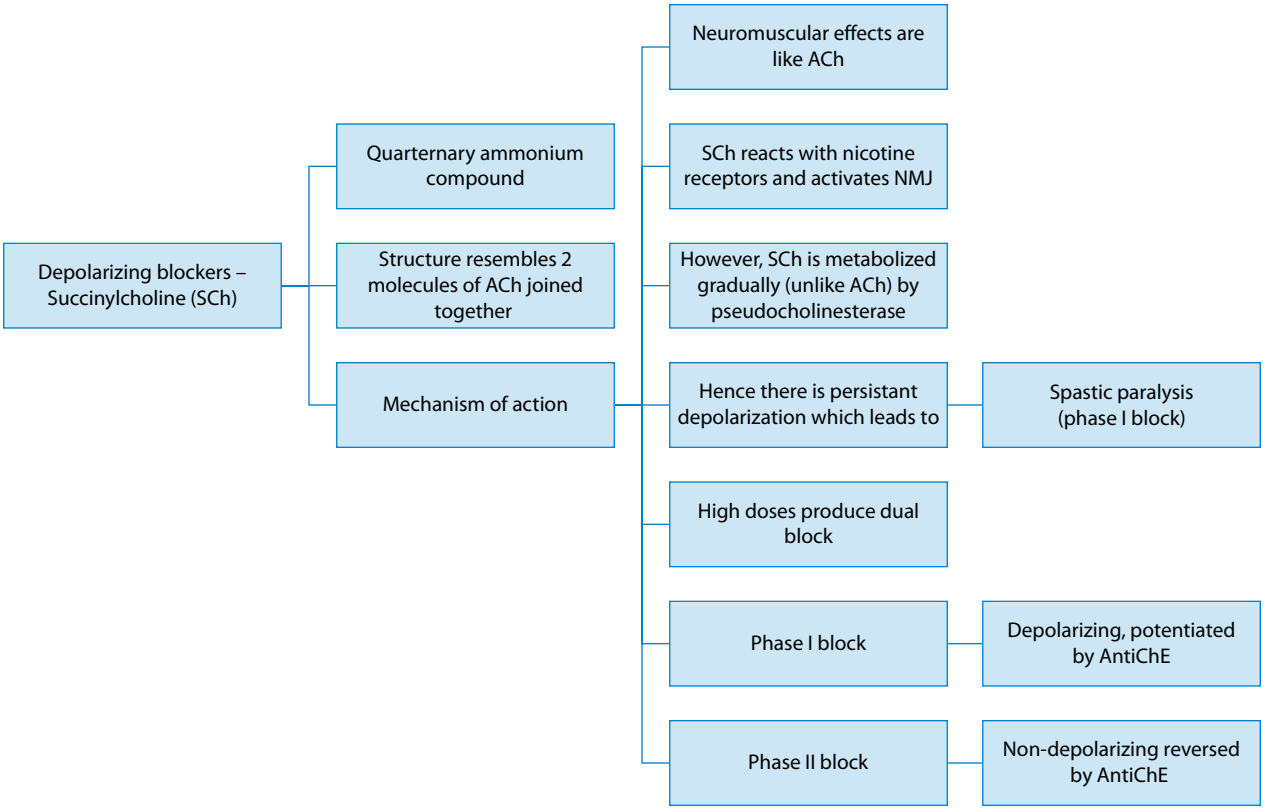
10.5 ADVERSE REACTIONS



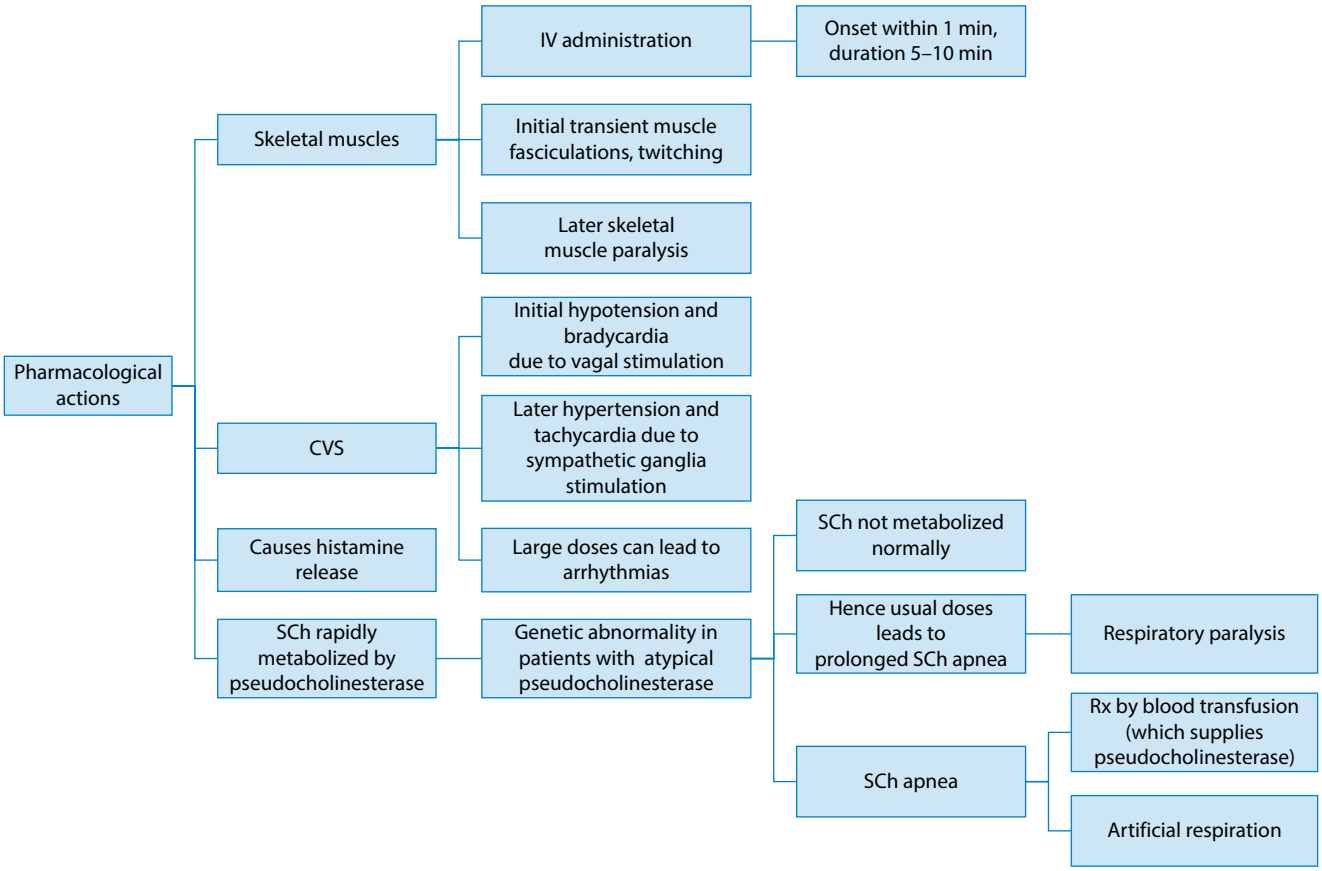
10.6 SYNTHETIC COMPETITIVE BLOCKERS



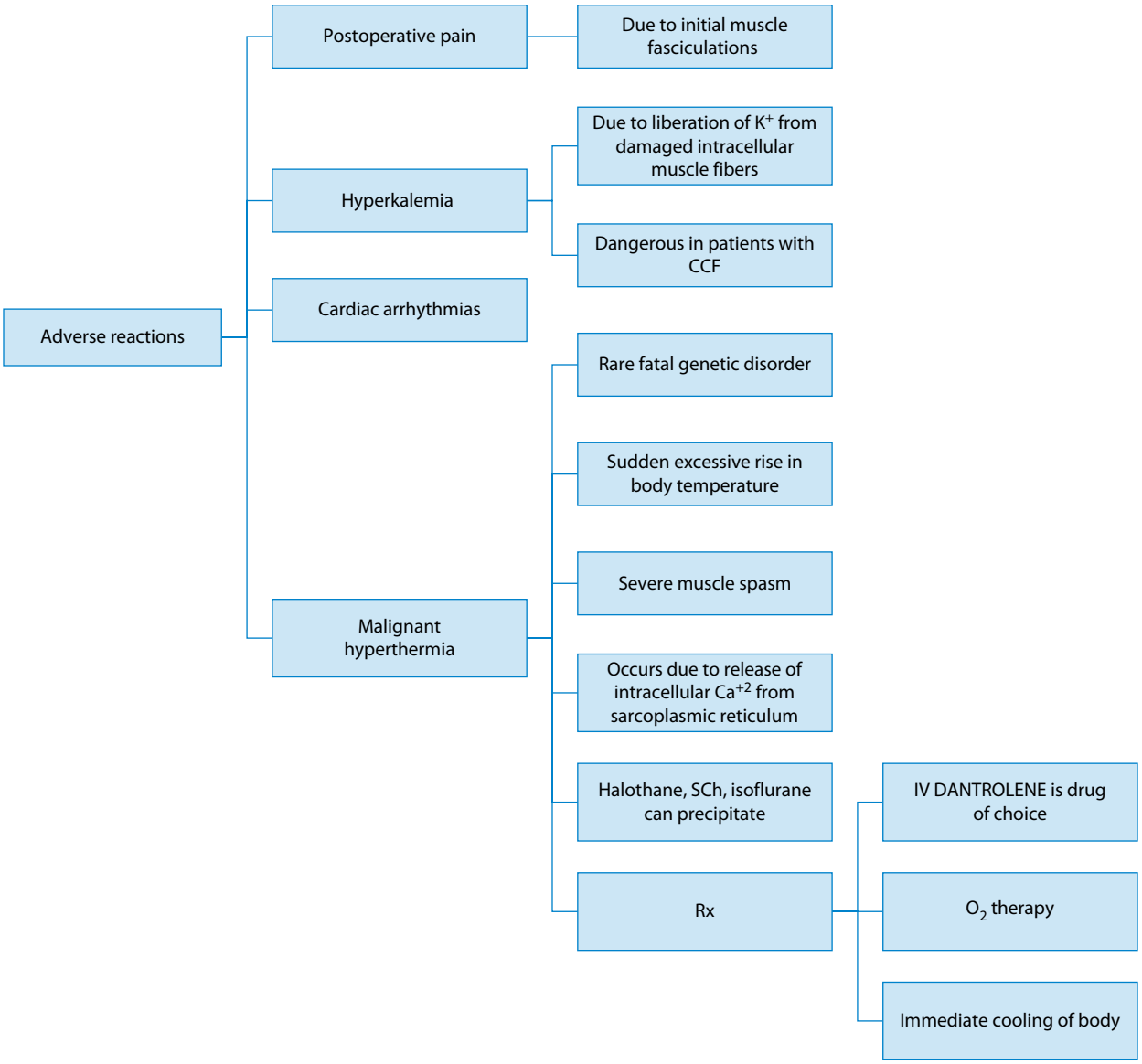
10.7 DEPOLARIZING BLOCKERS – SUCCINYLCHOLINE (SCh)



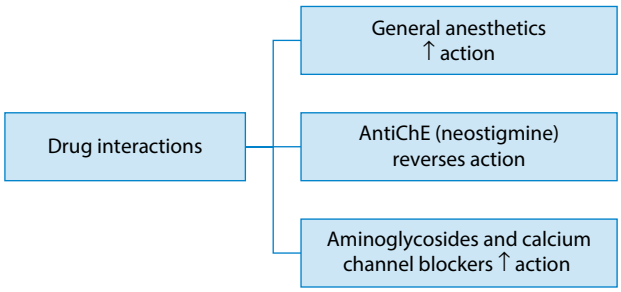
10.8 PHARMACOLOGICAL ACTIONS



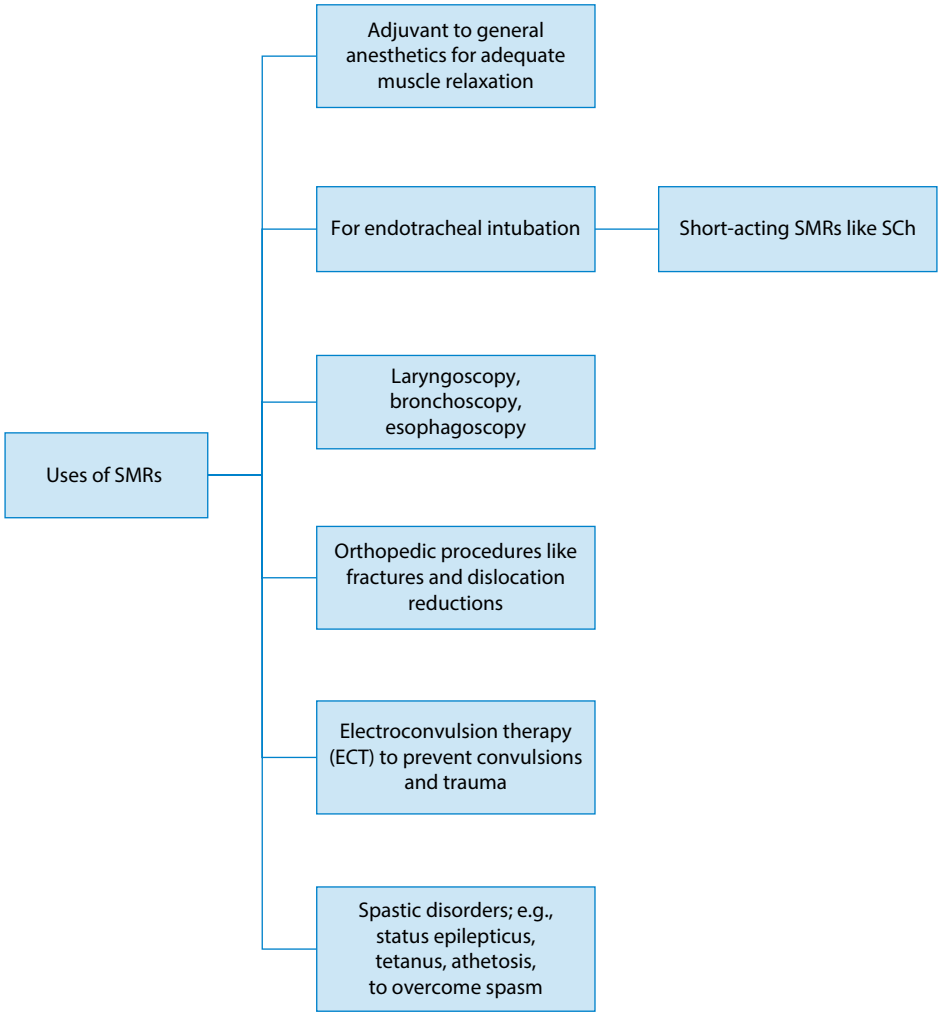
10.9 ADVERSE REACTIONS



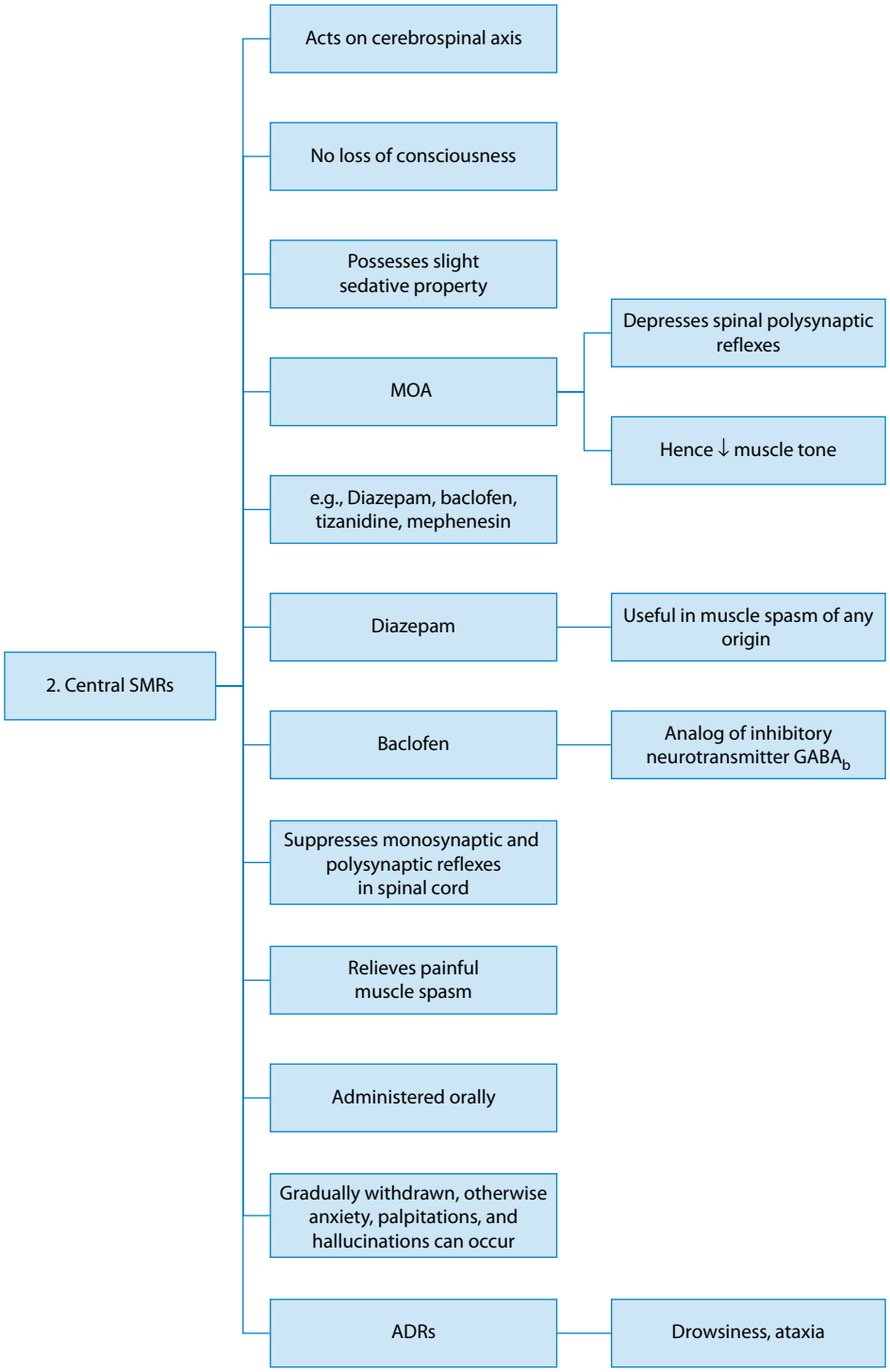
10.10 DRUG INTERACTIONS



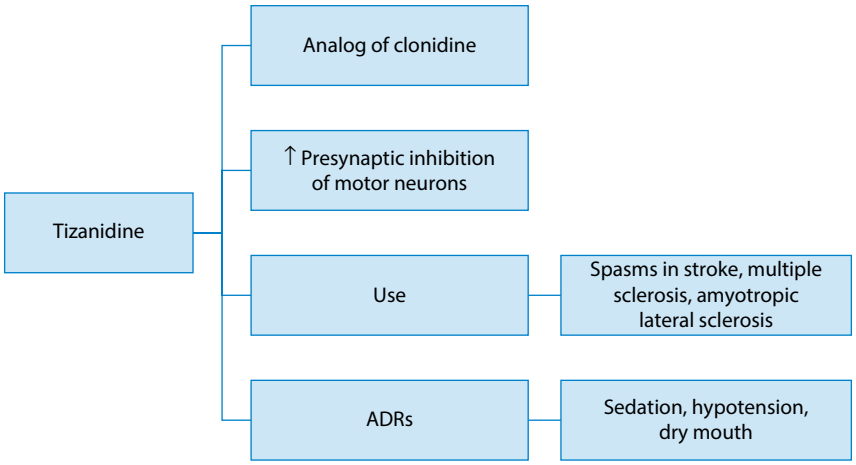
10.11 USES OF SMRs



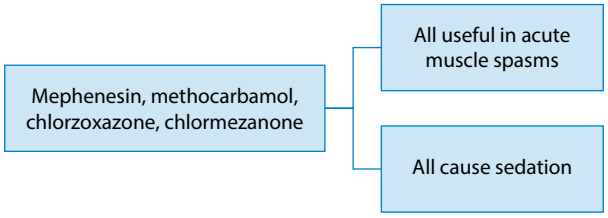
10.12 CENTRAL SMRs



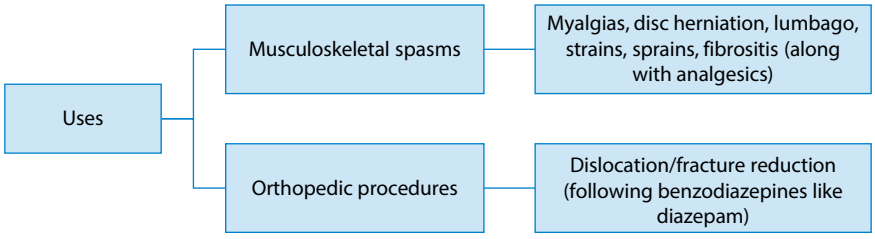
10.13 TIZANIDINE



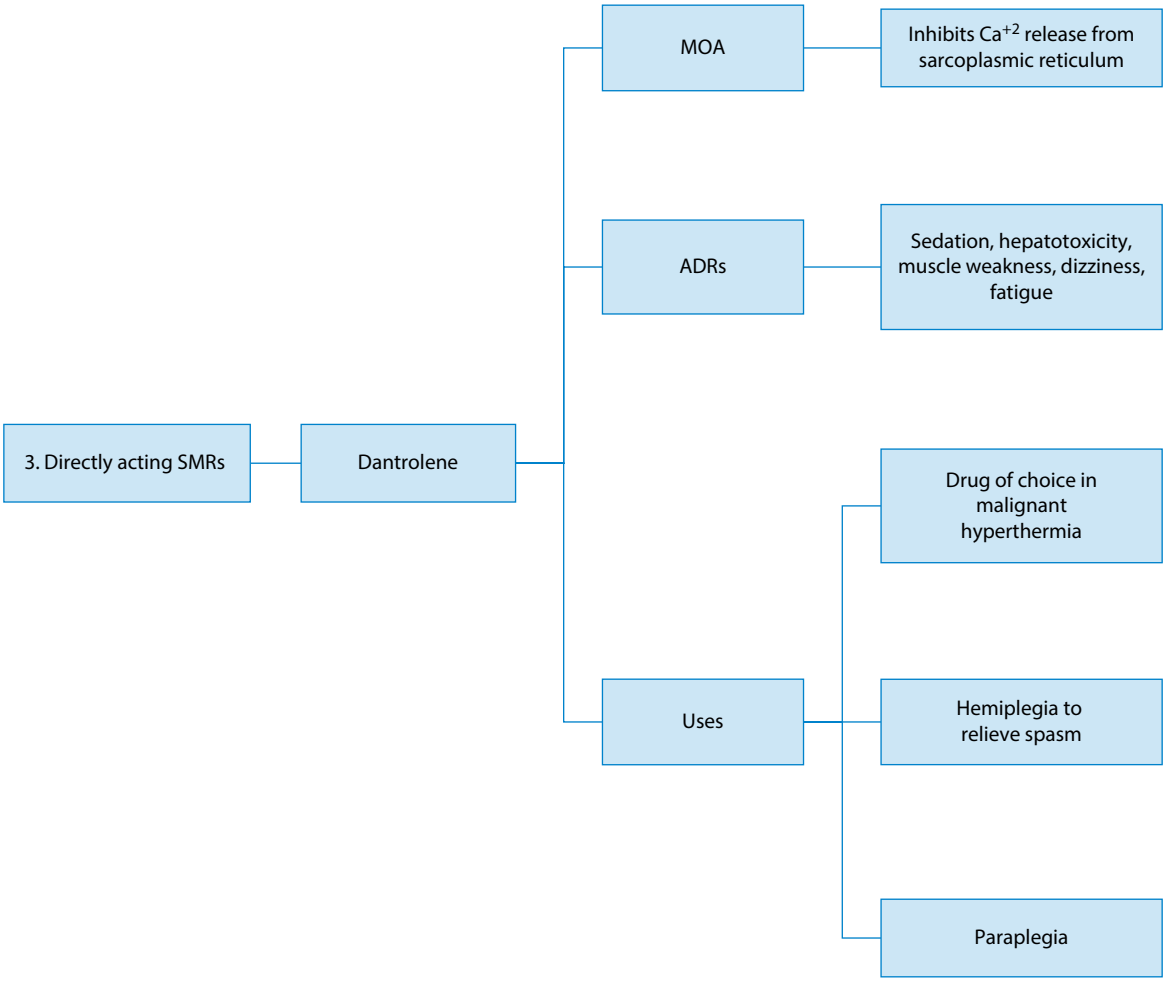
10.14 MEPHENESIN, METHOCARBAMOL, CHLORZOXAZONE, CHLORMEZANONE



10.15 USES



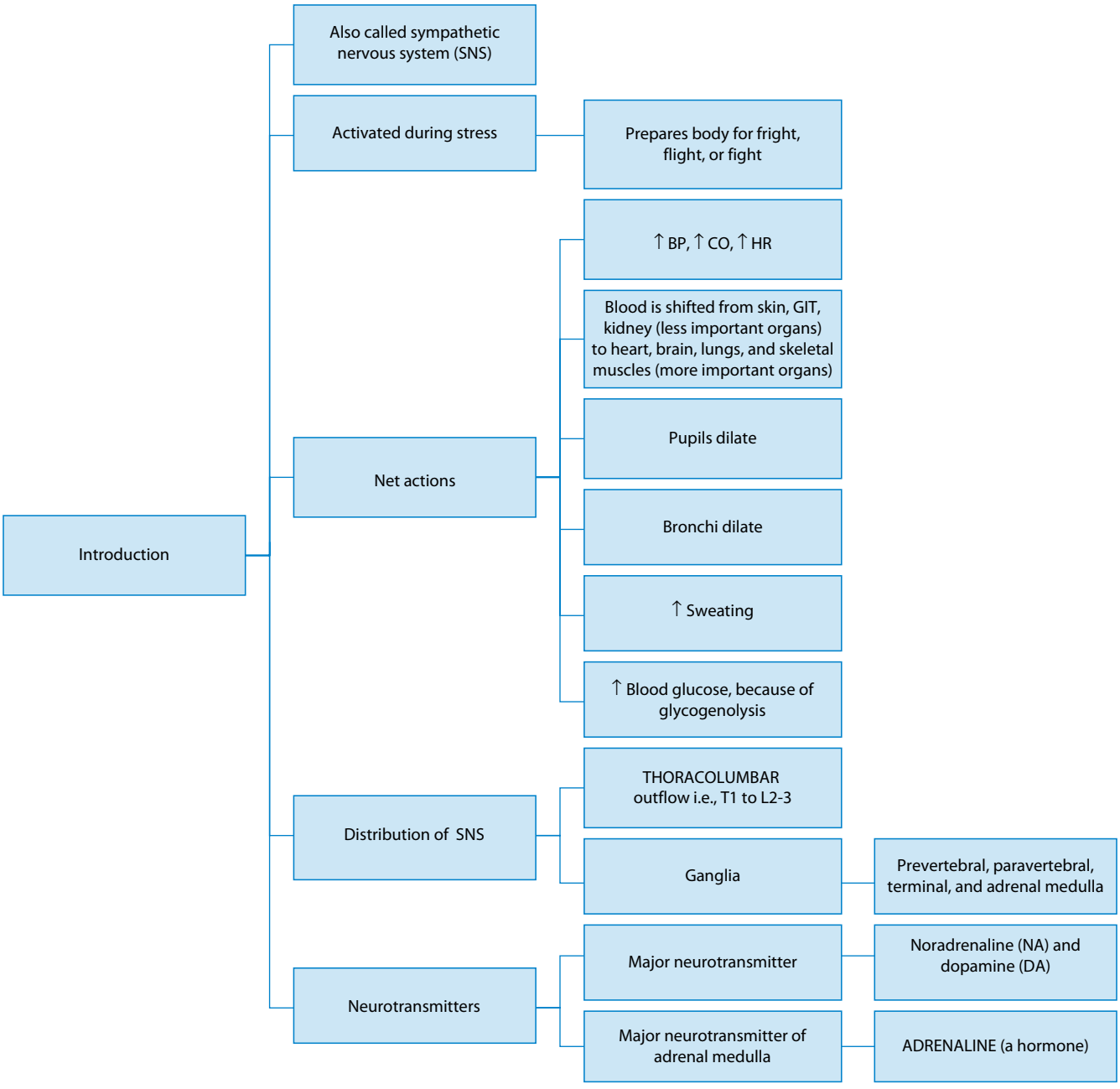
10.16 DIRECTLY ACTING SMRs



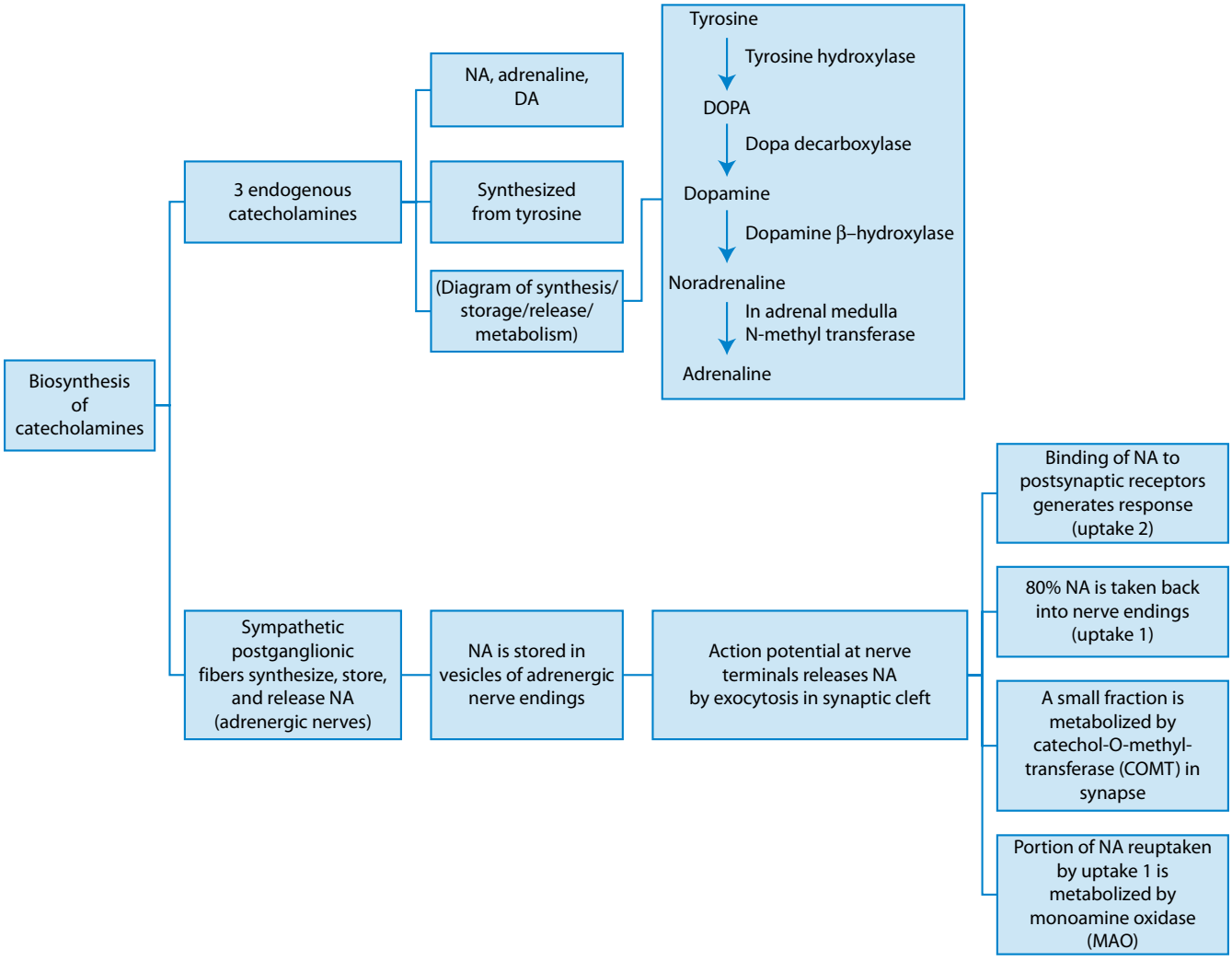
11

Adrenergic system and drugs

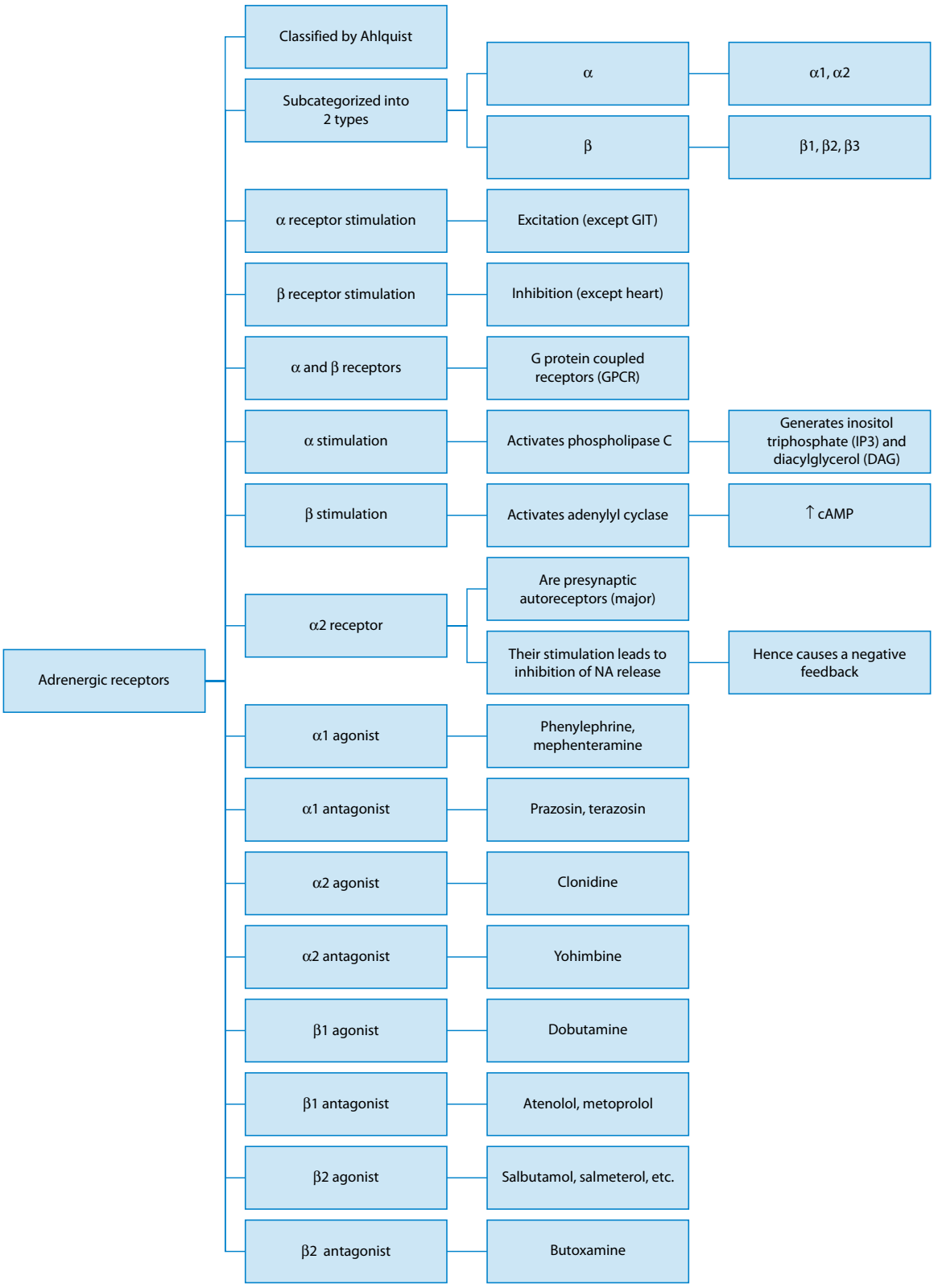
11.1 INTRODUCTION, DISTRIBUTION OF SNS, NEUROTRANSMITTERS



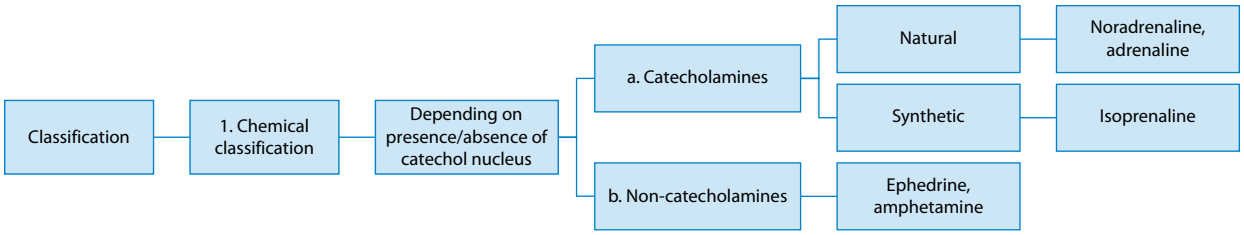
11.2 BIOSYNTHESIS OF CATECHOLAMINES

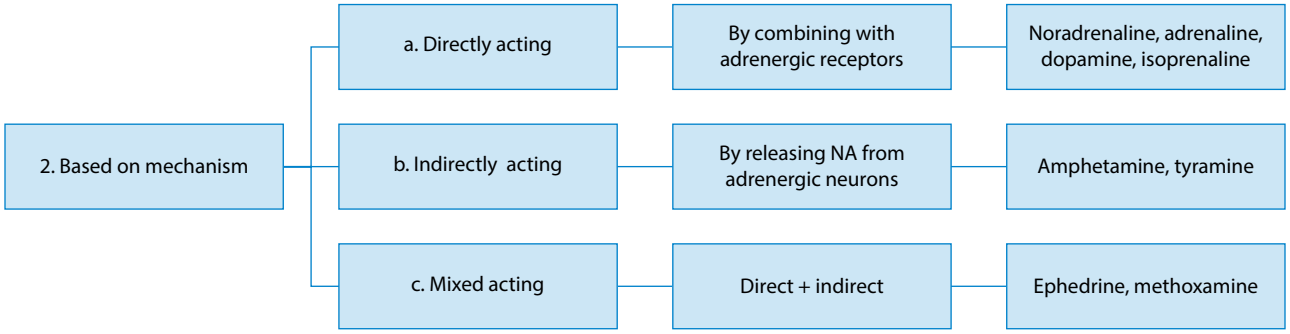


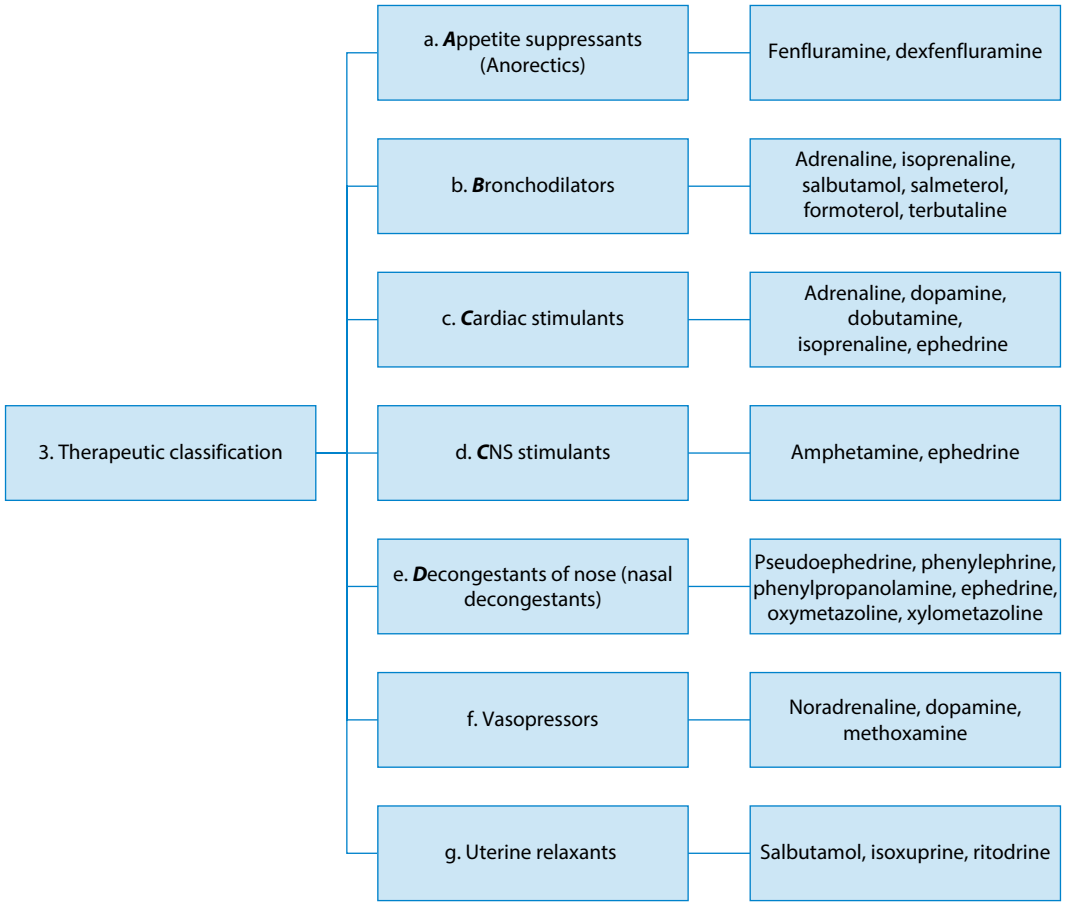
11.3 ADRENERGIC RECEPTORS



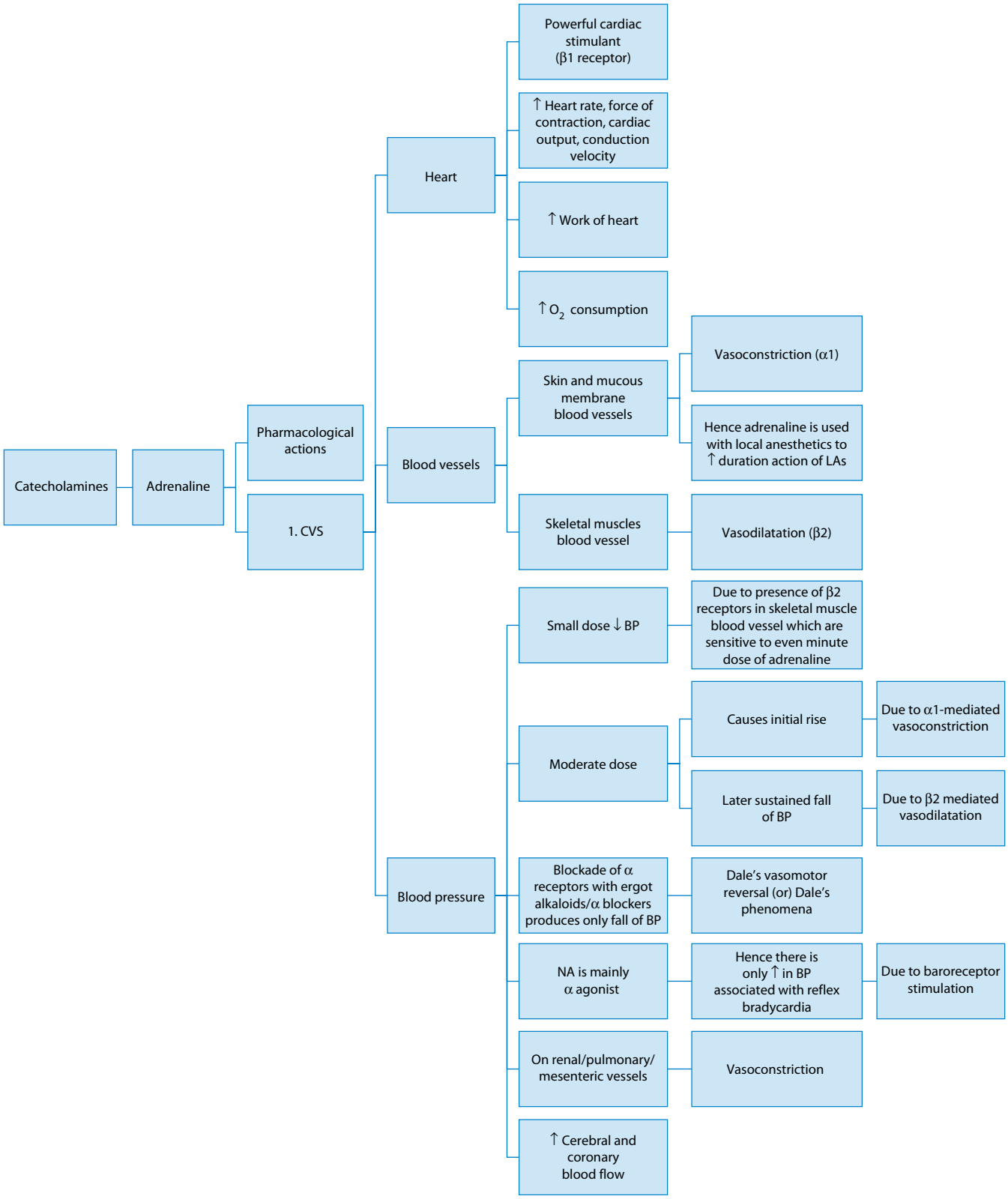
11.4 ADRENERGIC DRUGS (SYMPATHOMIMETICS) – CLASSIFICATION





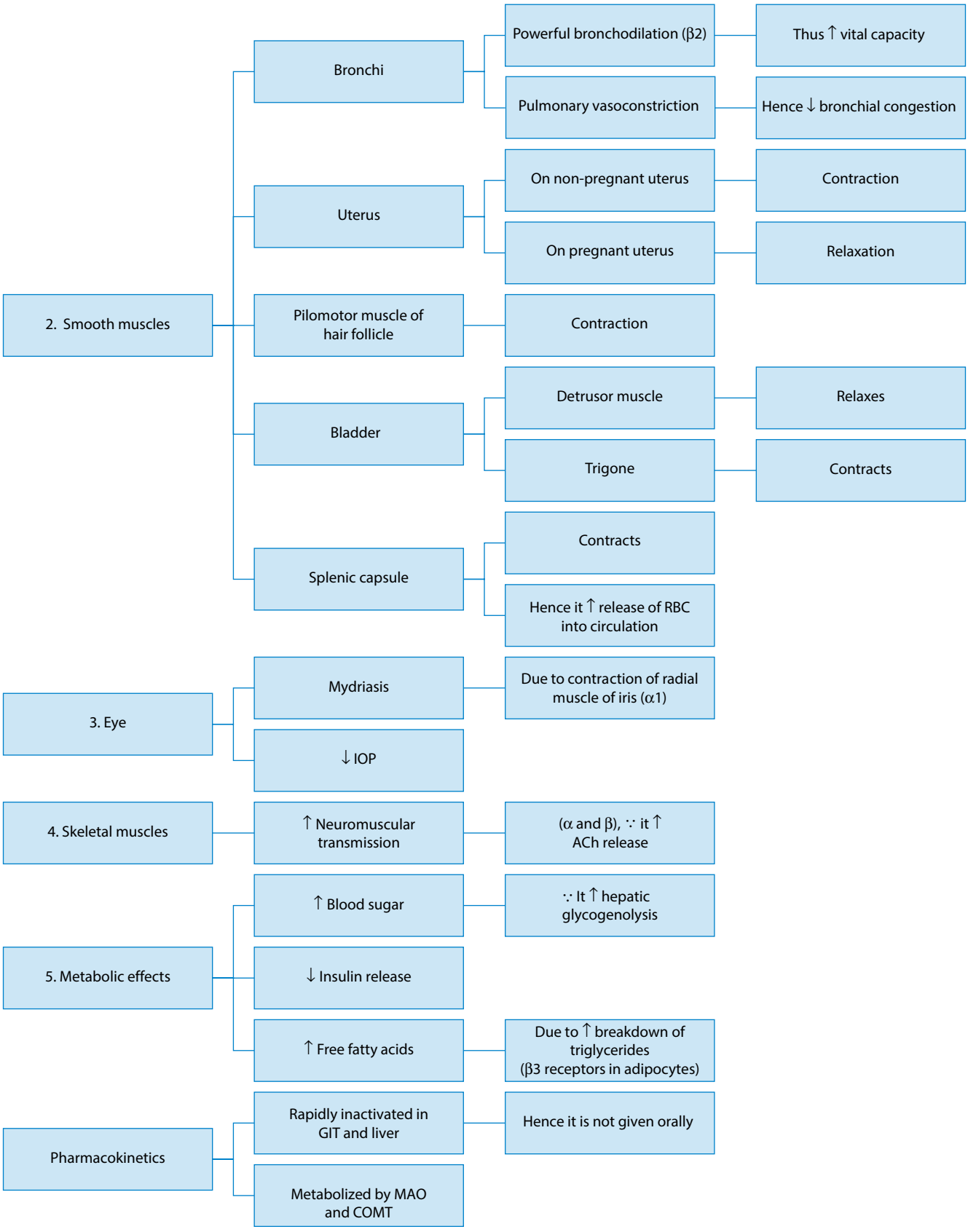


11.5 CATECHOLAMINES – ADRENALINE – PHARMACOLOGICAL ACTIONS

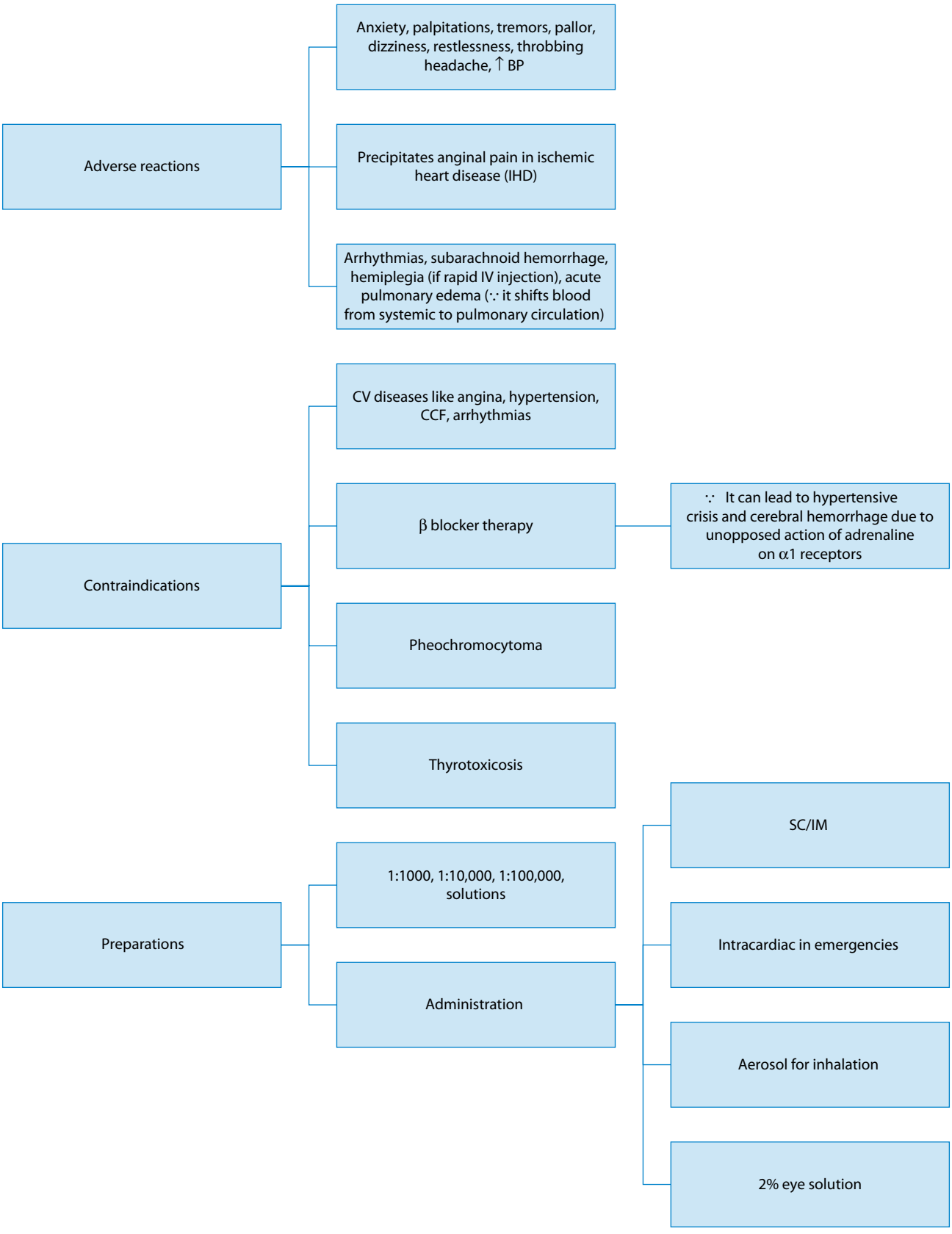


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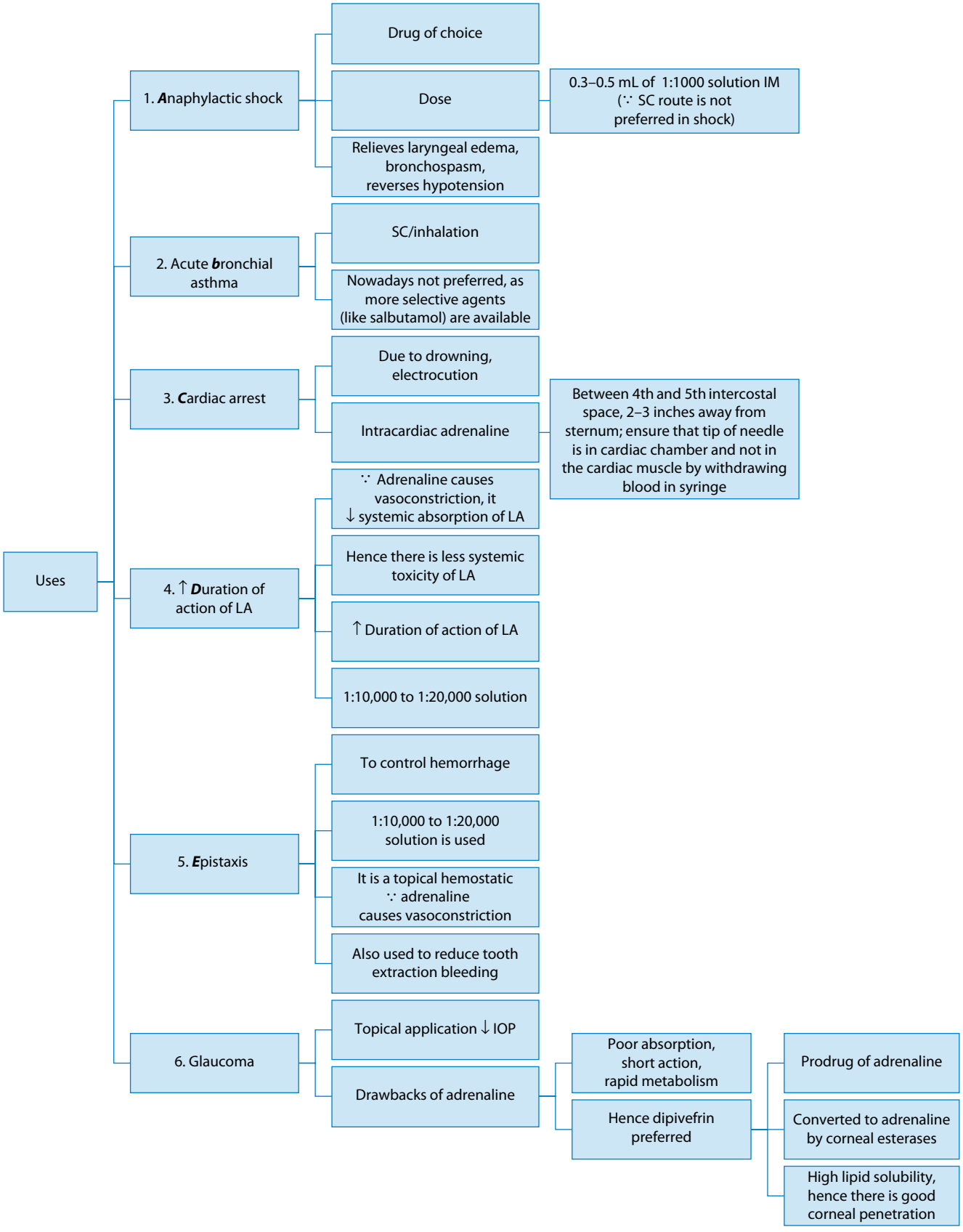
11.5 CATECHOLAMINES – ADRENALINE – PHARMACOLOGICAL ACTIONS (Continued)



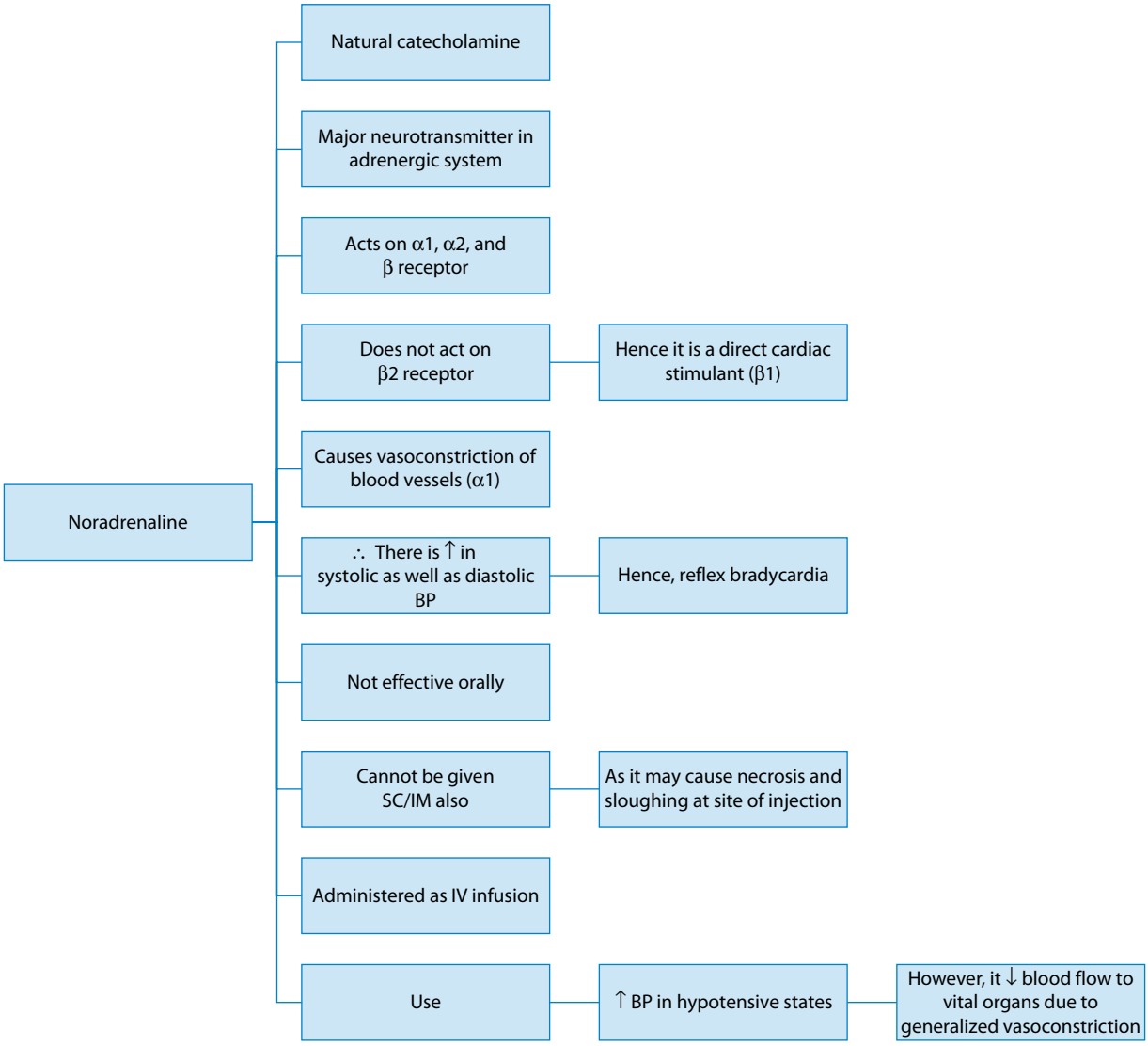
11.6 ADVERSE REACTIONS, CONTRAINDICATIONS, PREPARATIONS



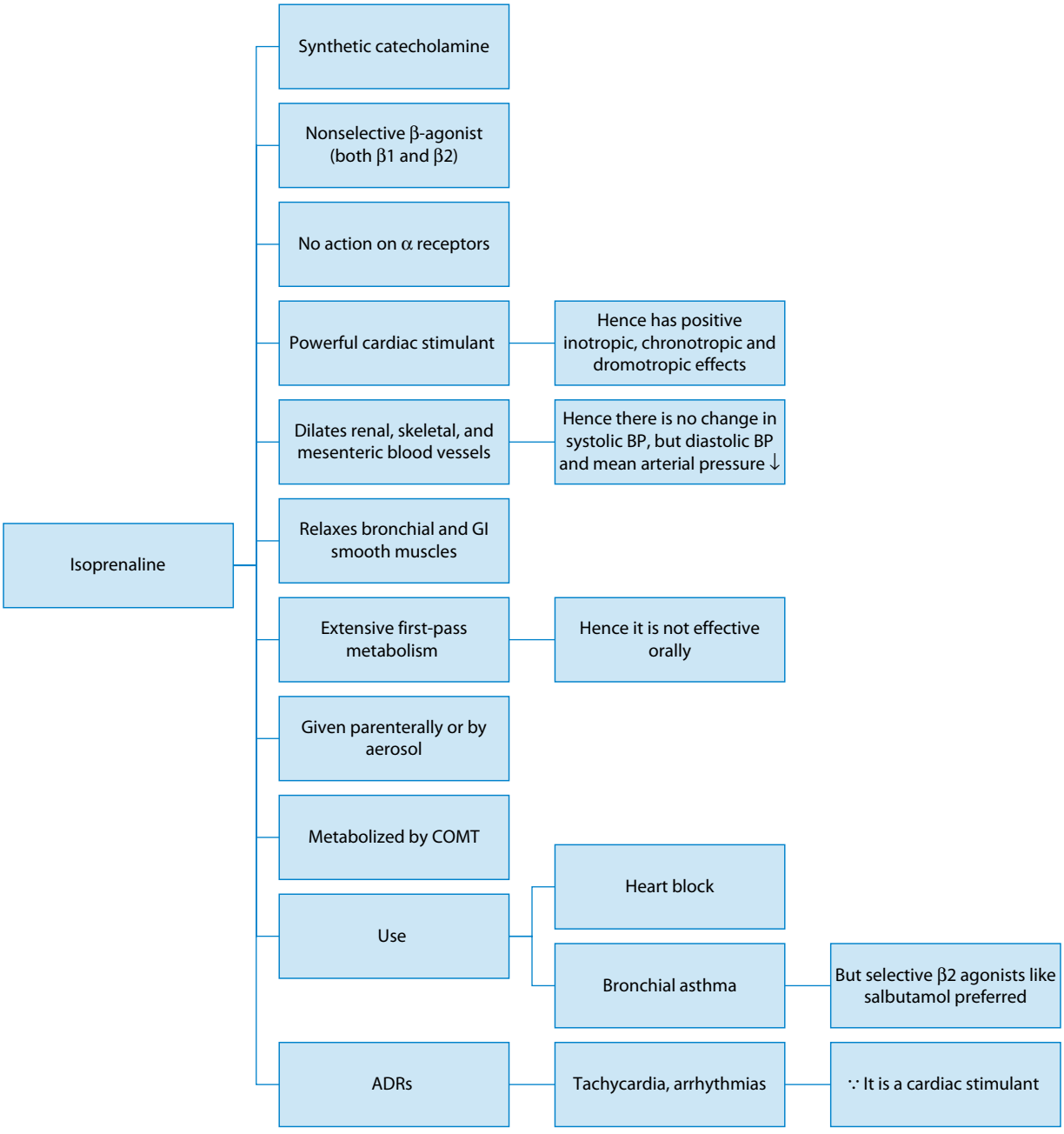
11.7 USES OF ADRENALINE



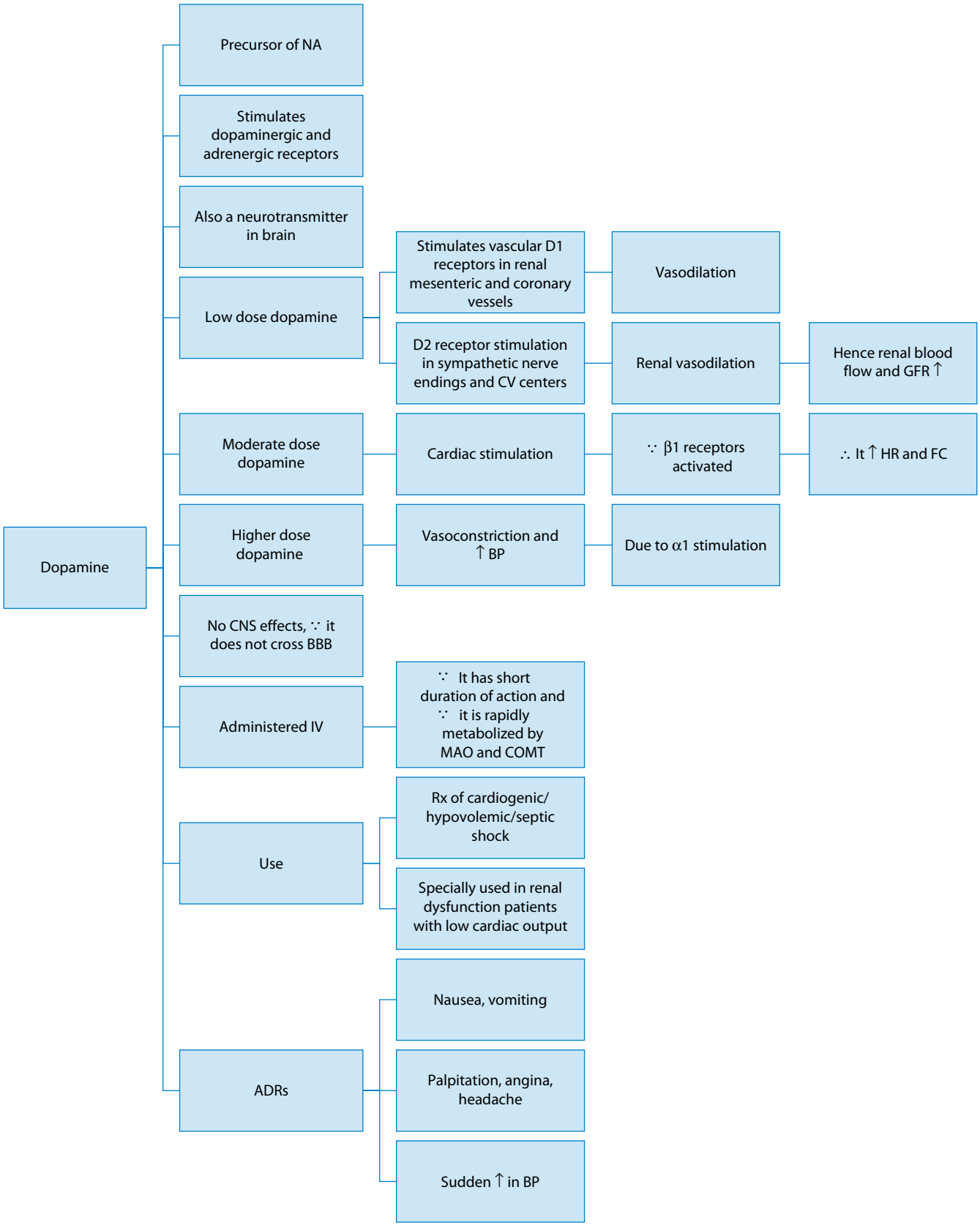
11.8 NORADRENALINE



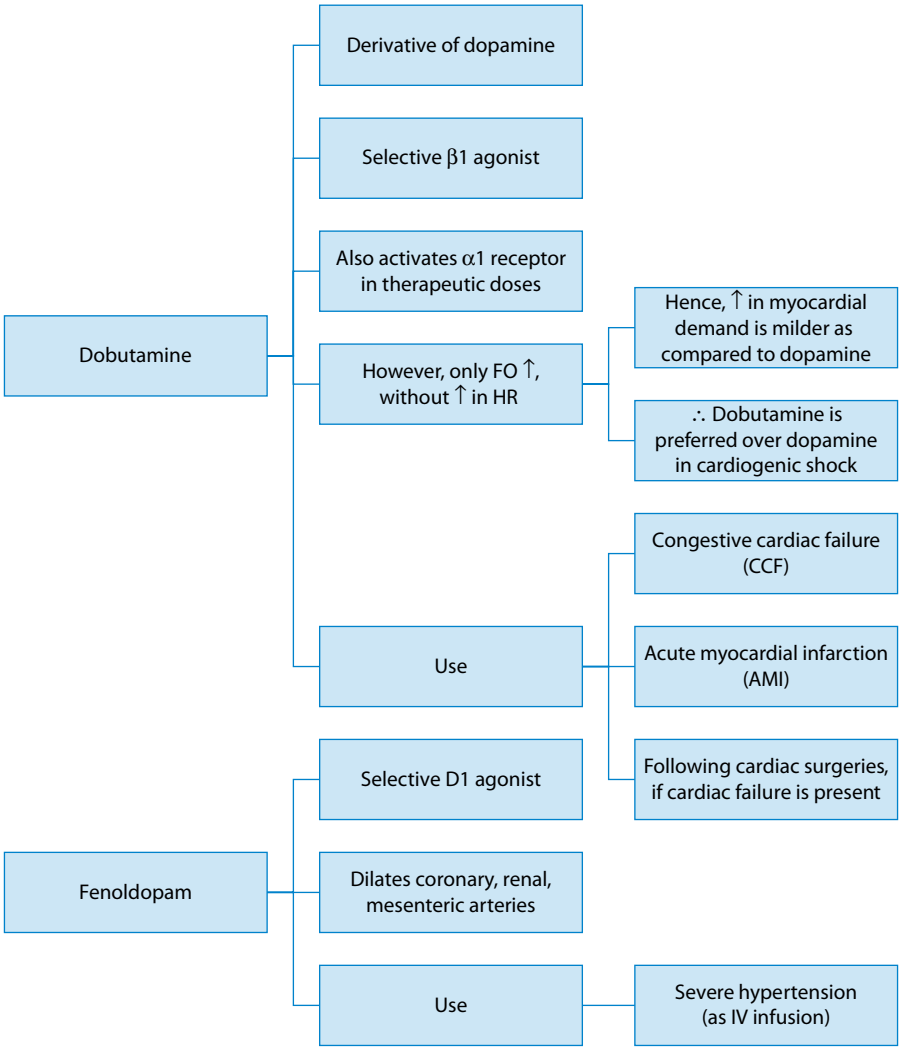
11.9 ISOPRENALINE



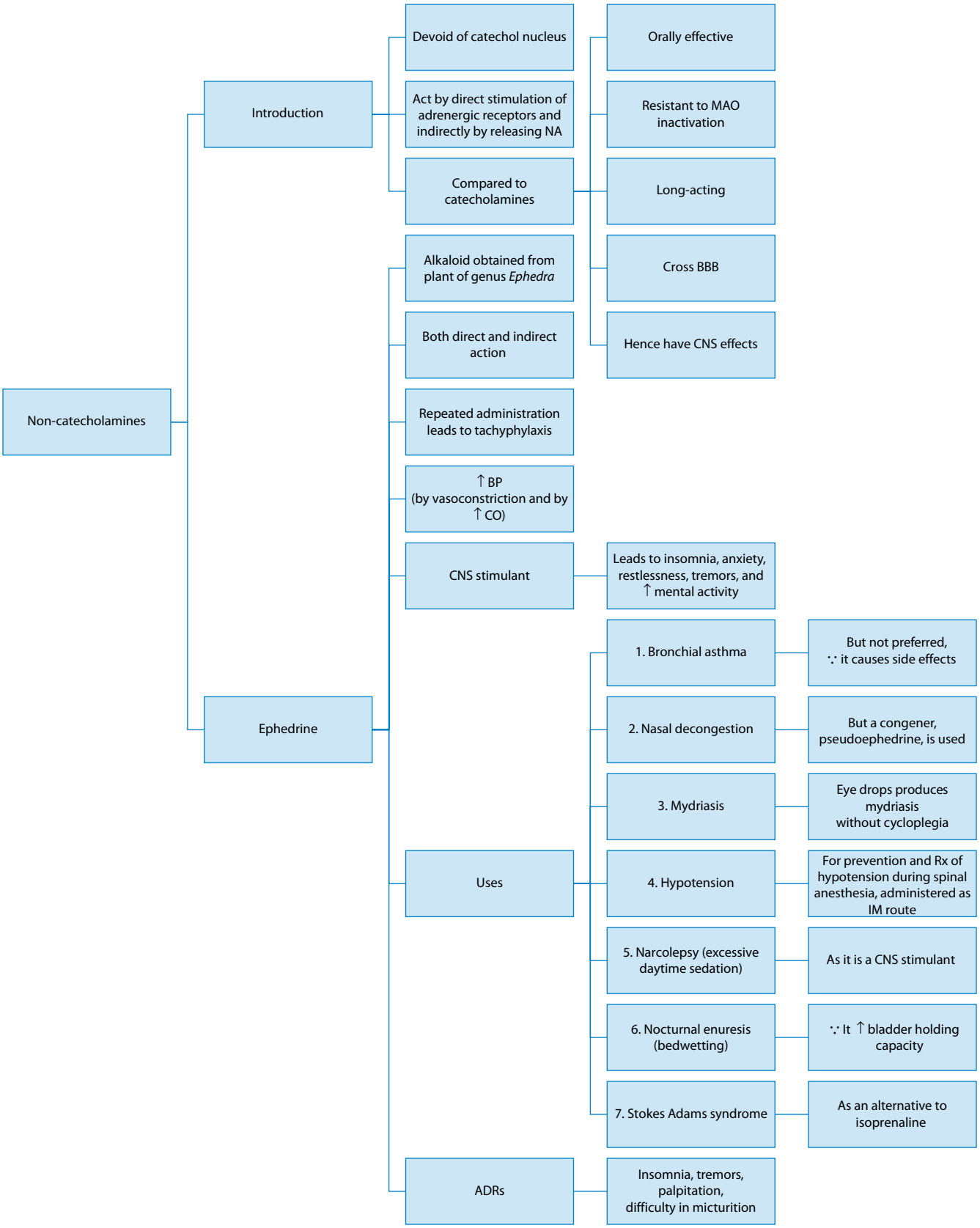
11.10 DOPAMINE



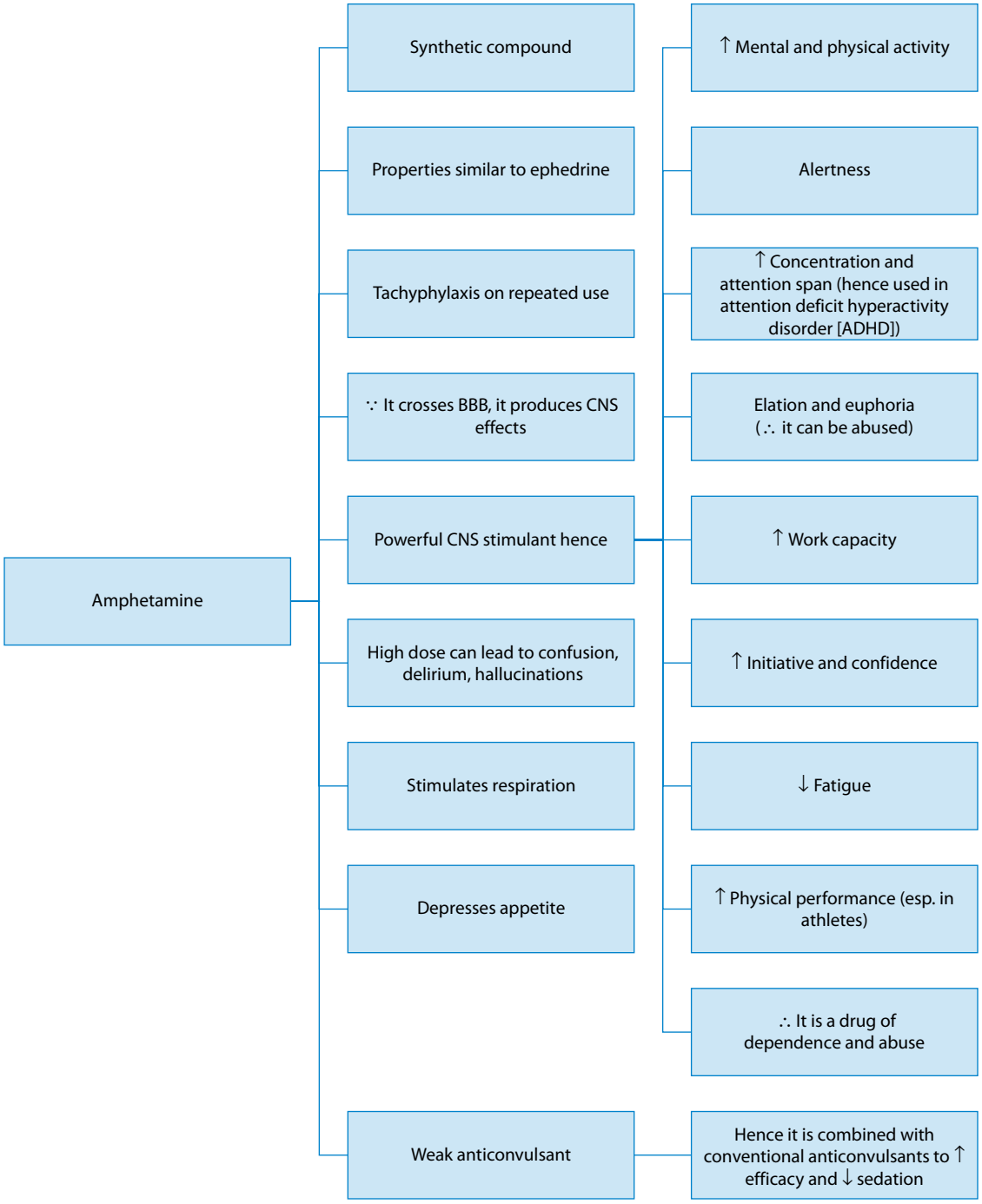
11.11 DOBUTAMINE, FENOLDOPAM



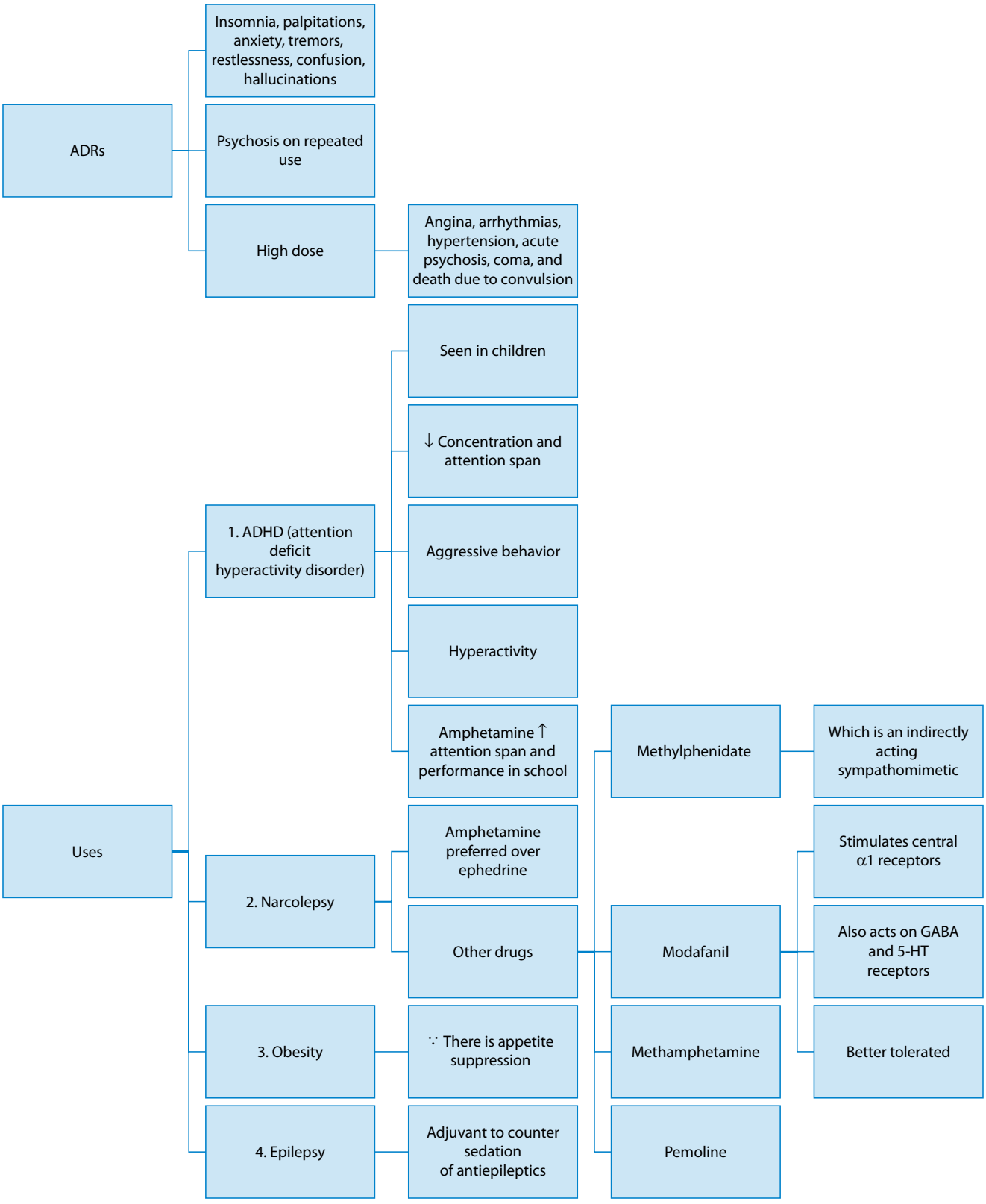
11.12 NONCATECHOLAMINES – INTRODUCTION AND EPHEDRINE



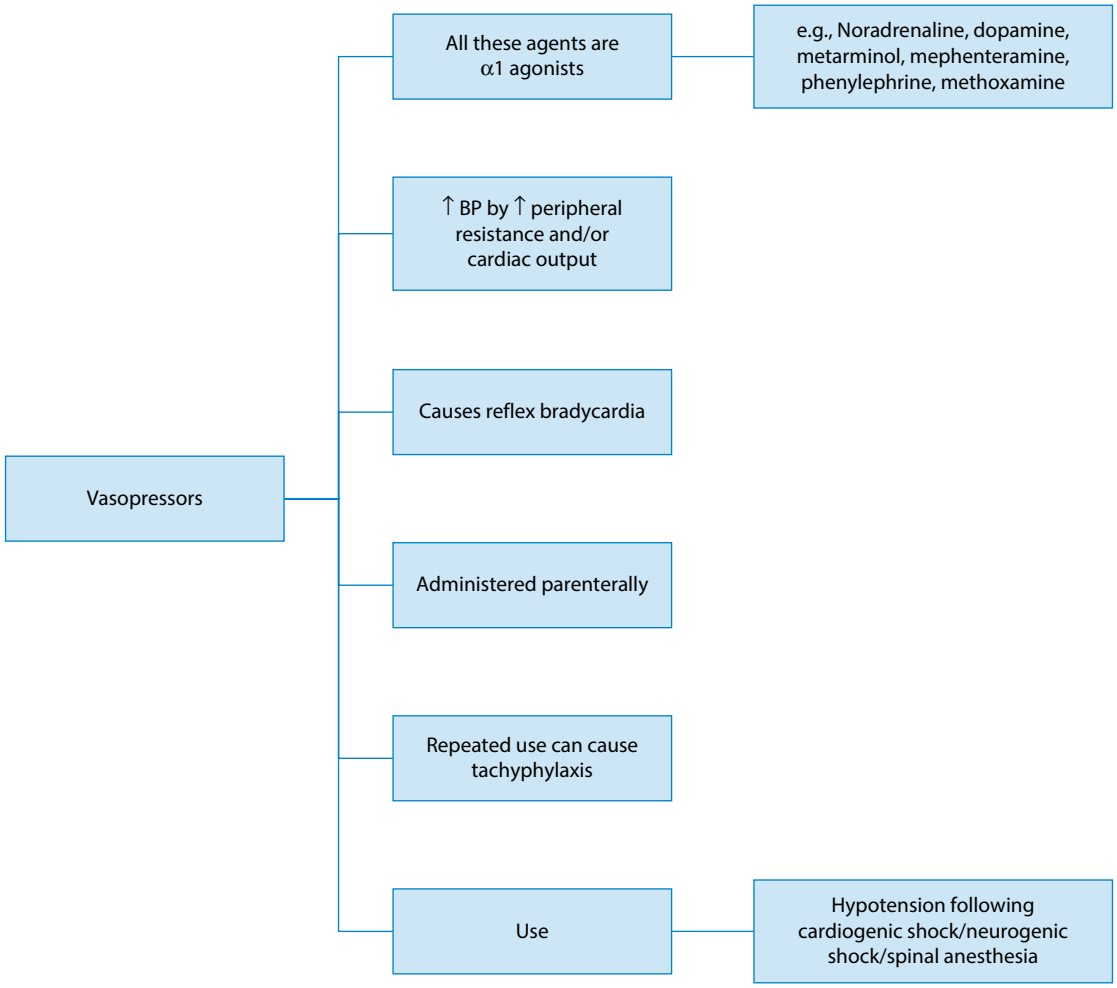
11.13 AMPHETAMINE



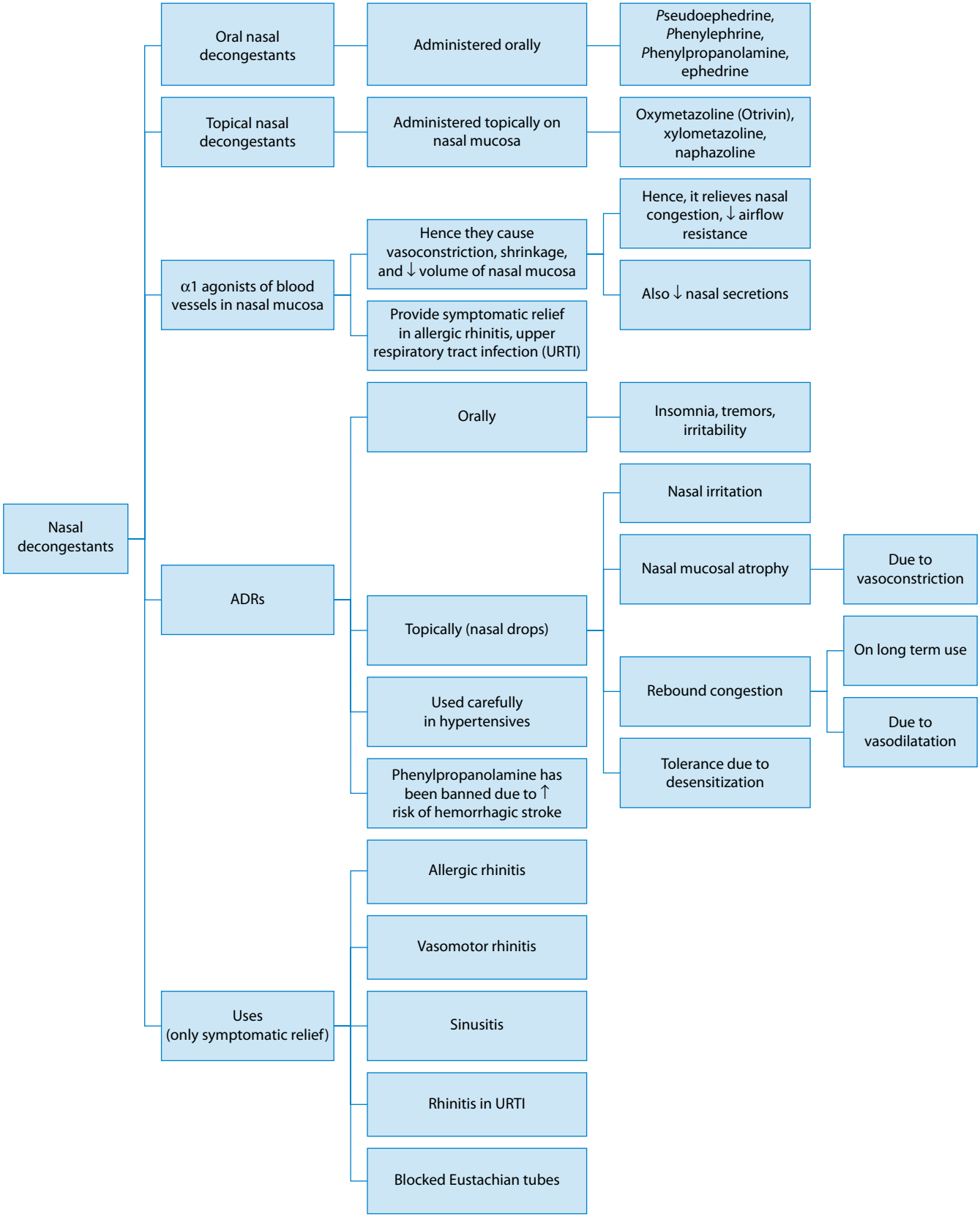
11.14 ADRs, USES



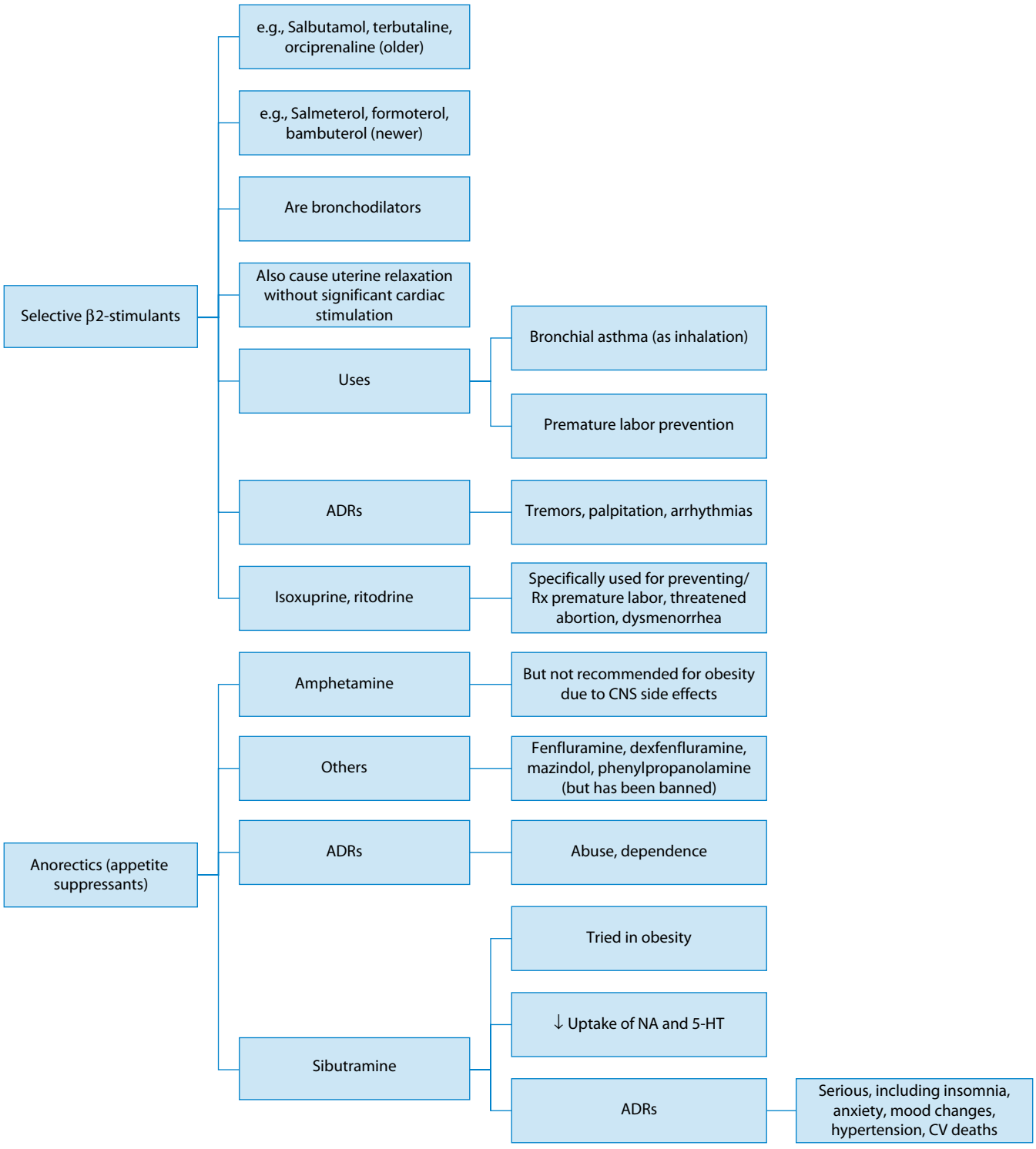
11.15 VASOPRESSORS



11.16 NASAL DECONGESTANTS



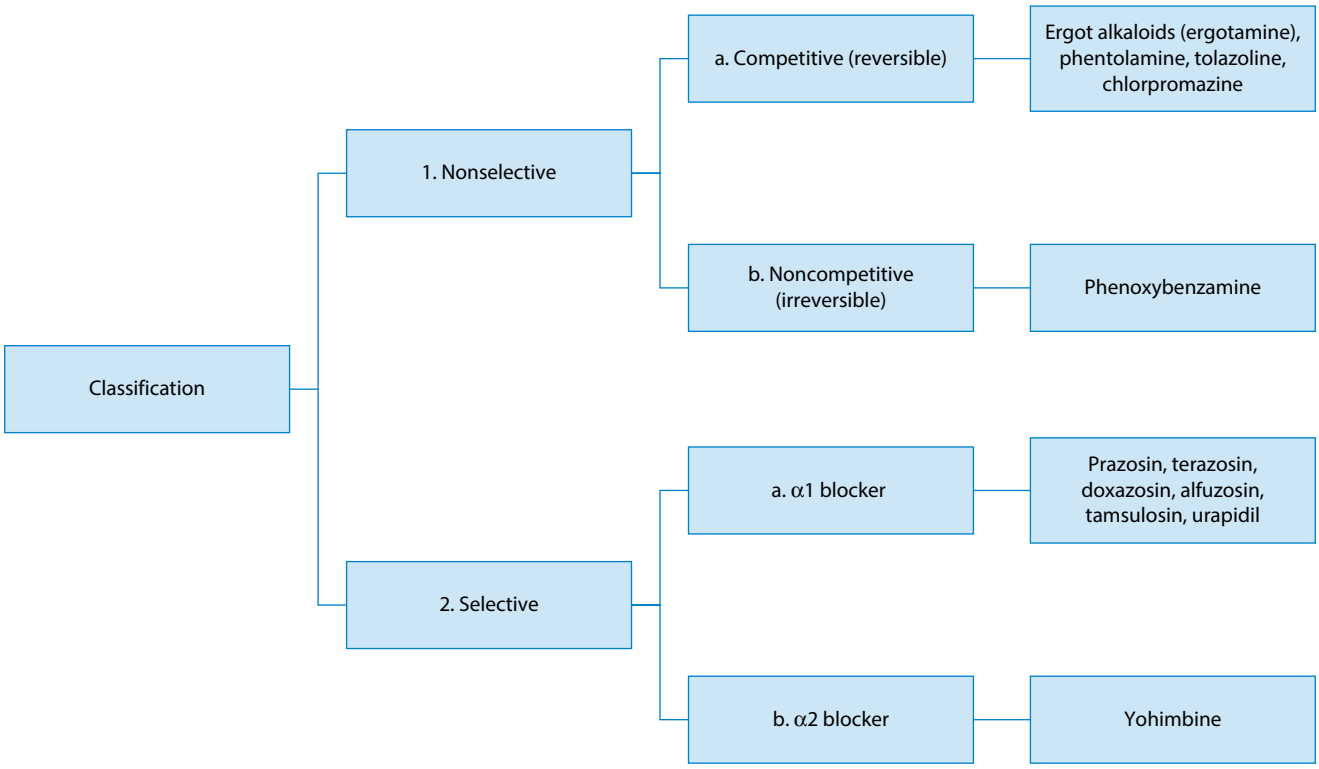
11.17 SELECTIVE β2-STIMULANTS, ANORECTICS (APPETITE SUPPRESSANTS)



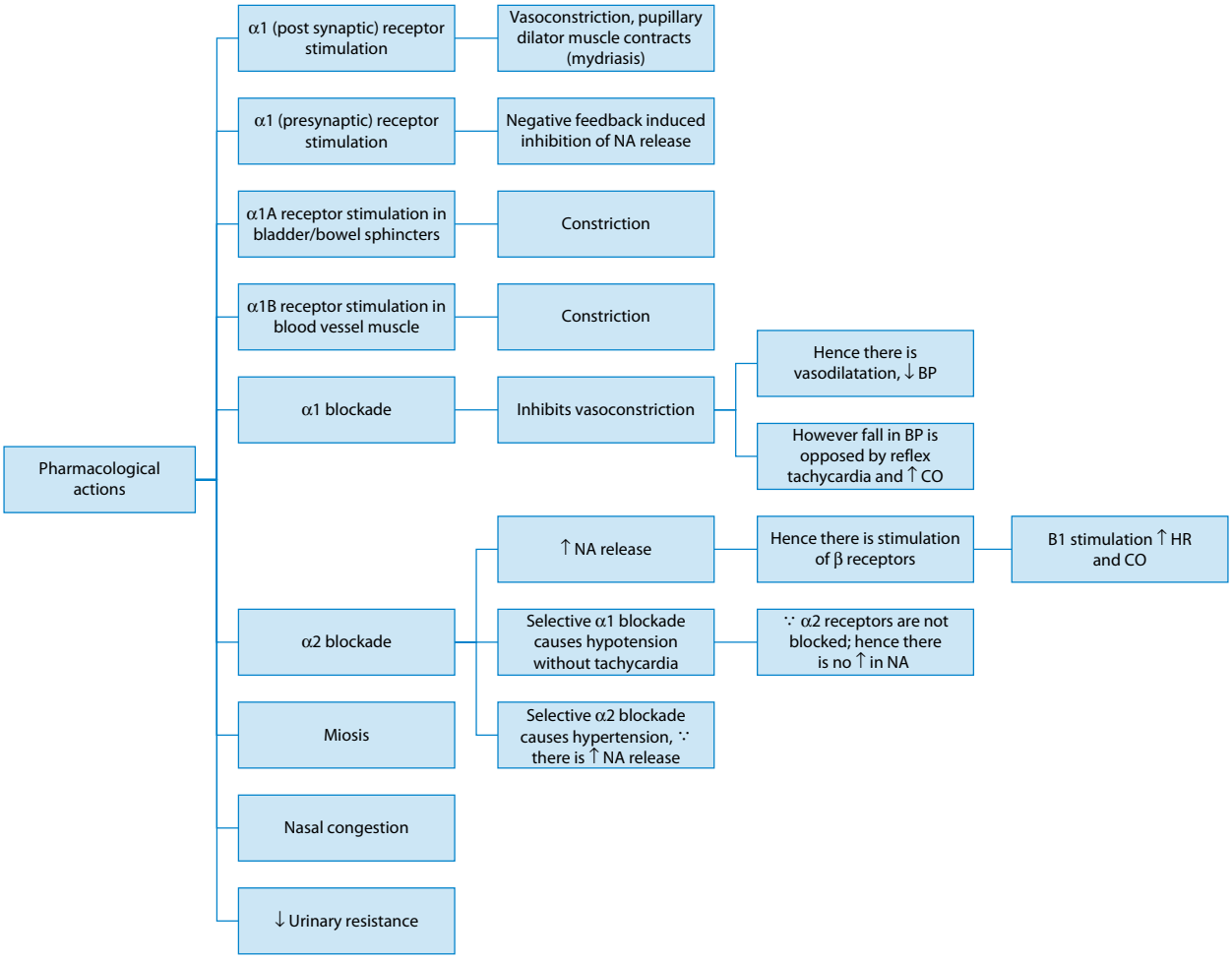
12

Alpha-adrenergic blocking agents (α blockers)

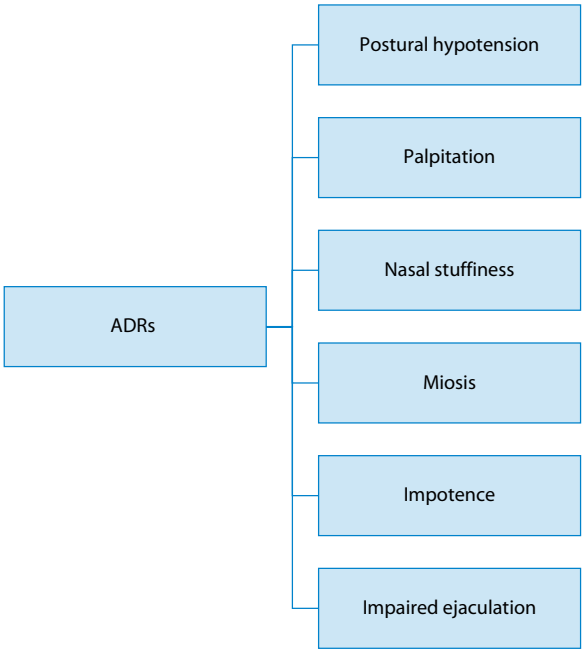
12.1 CLASSIFICATION



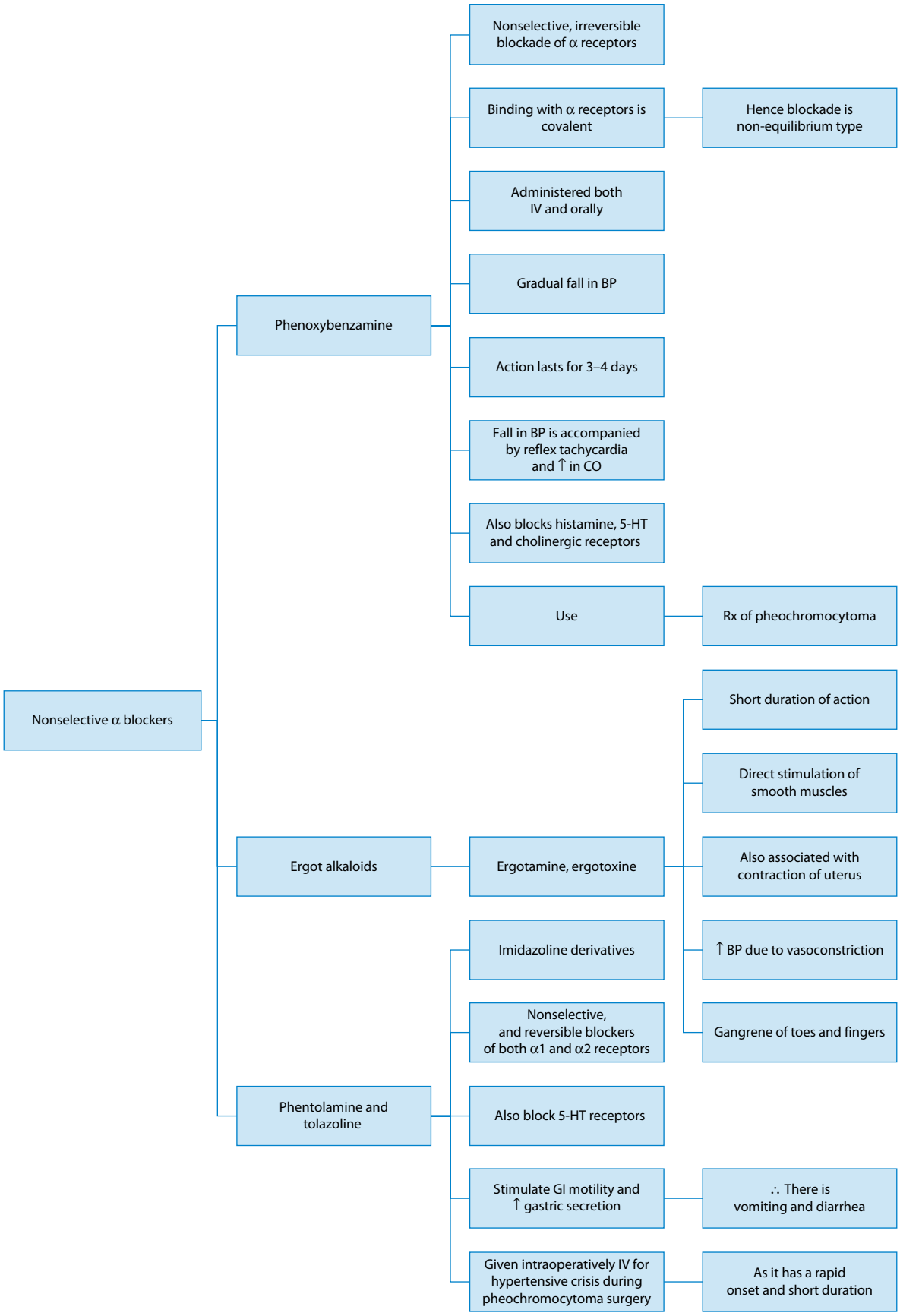
12.2 PHARMACOLOGICAL ACTIONS



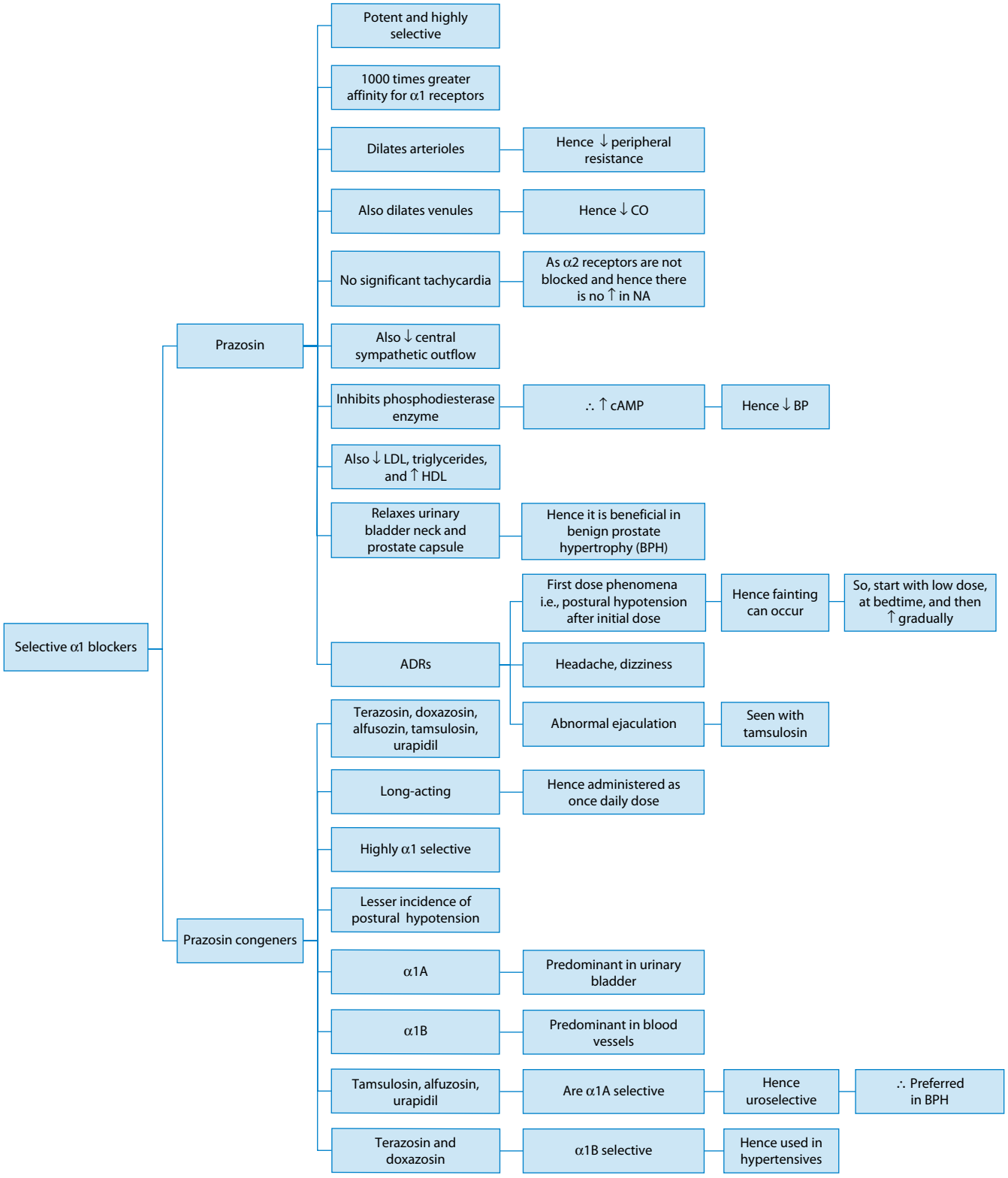
12.3 ADRs



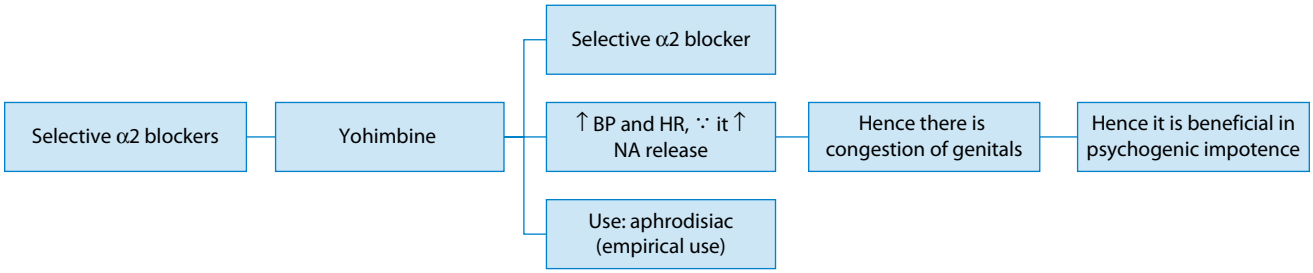
12.4 NONSELECTIVE α BLOCKERS



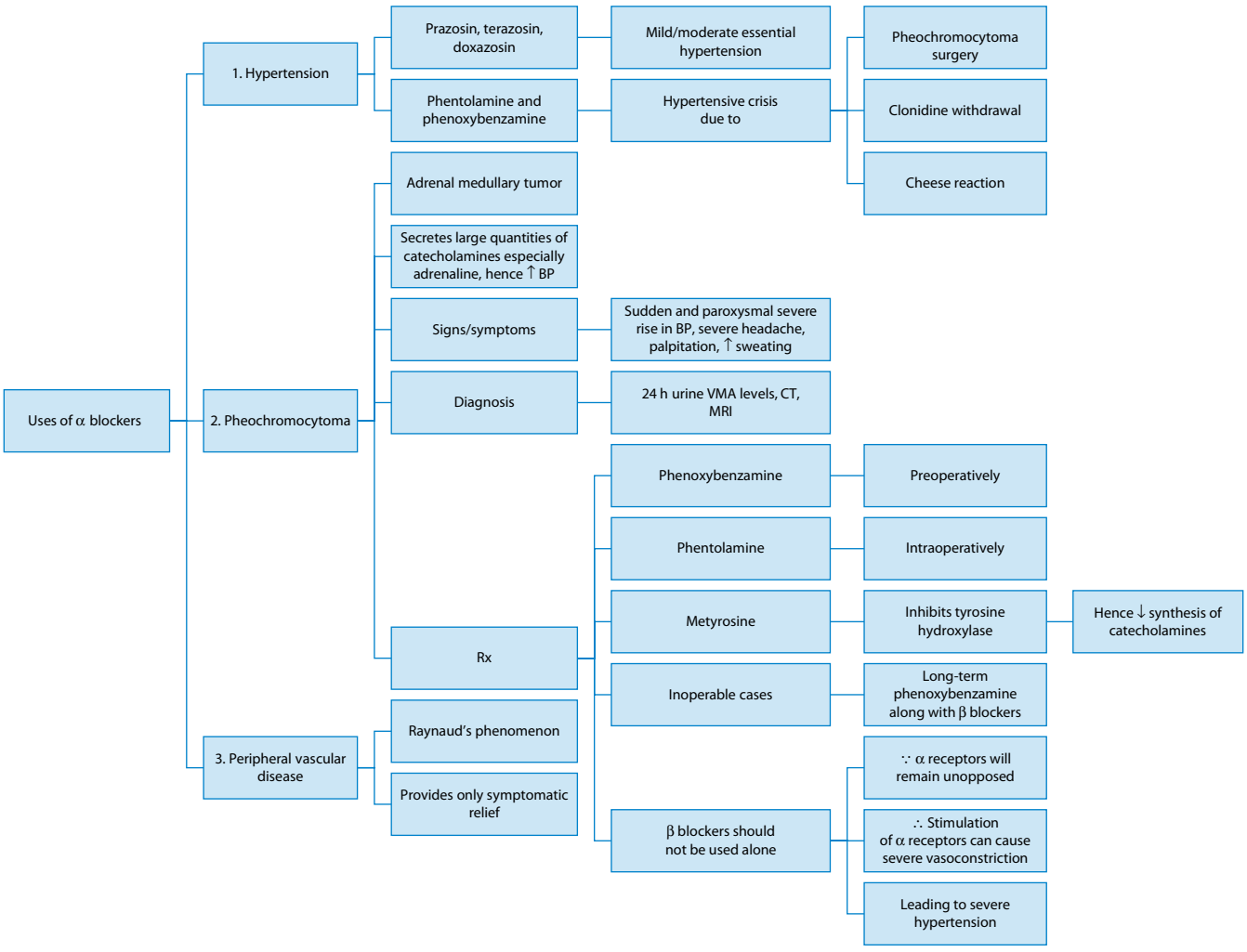
12.5 SELECTIVE α1 BLOCKERS



12.6 SELECTIVE α2 BLOCKERS

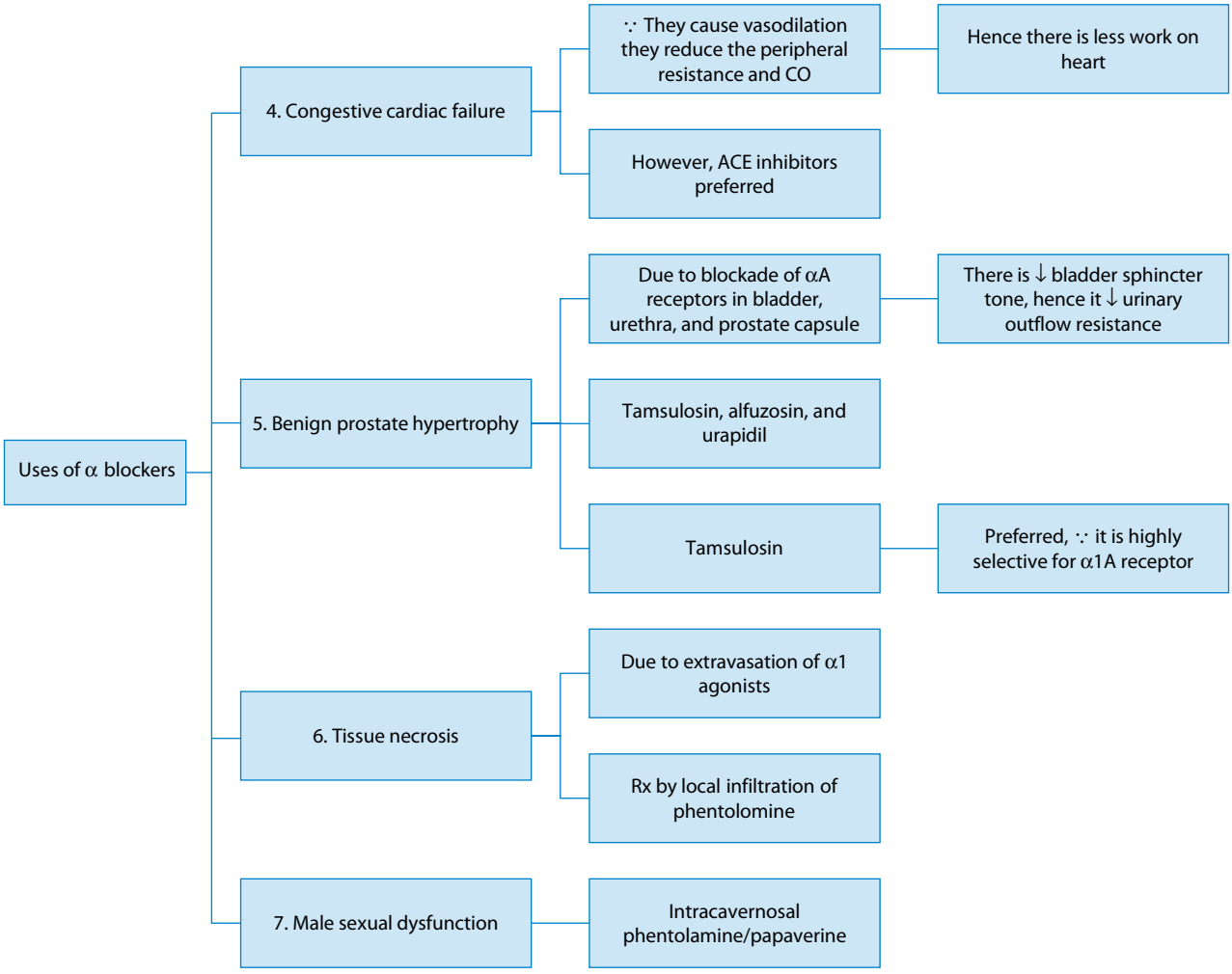


12.7 USES OF α BLOCKERS



(*Continued*)

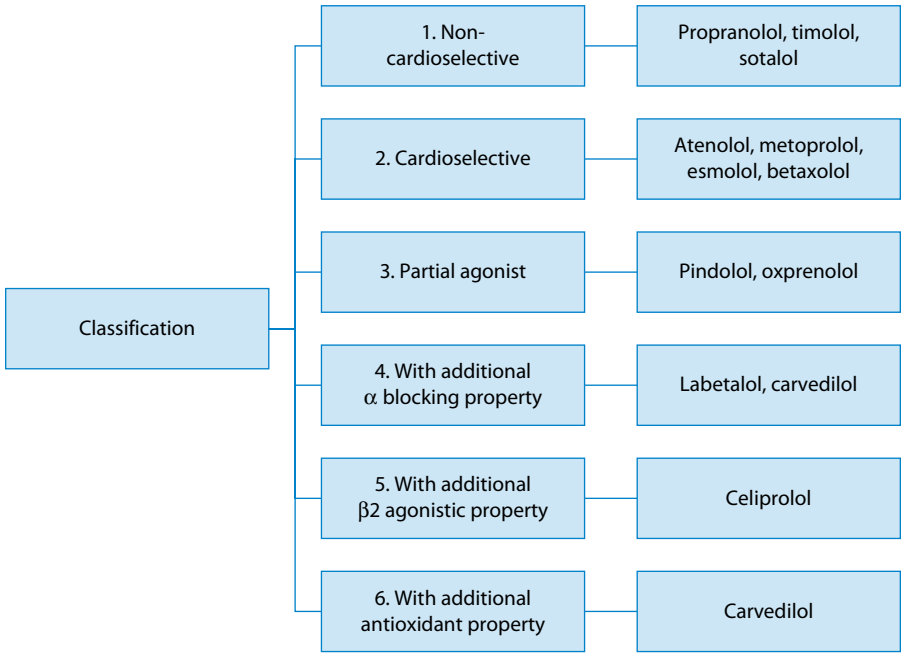
12.7 USES OF α BLOCKERS (Continued)



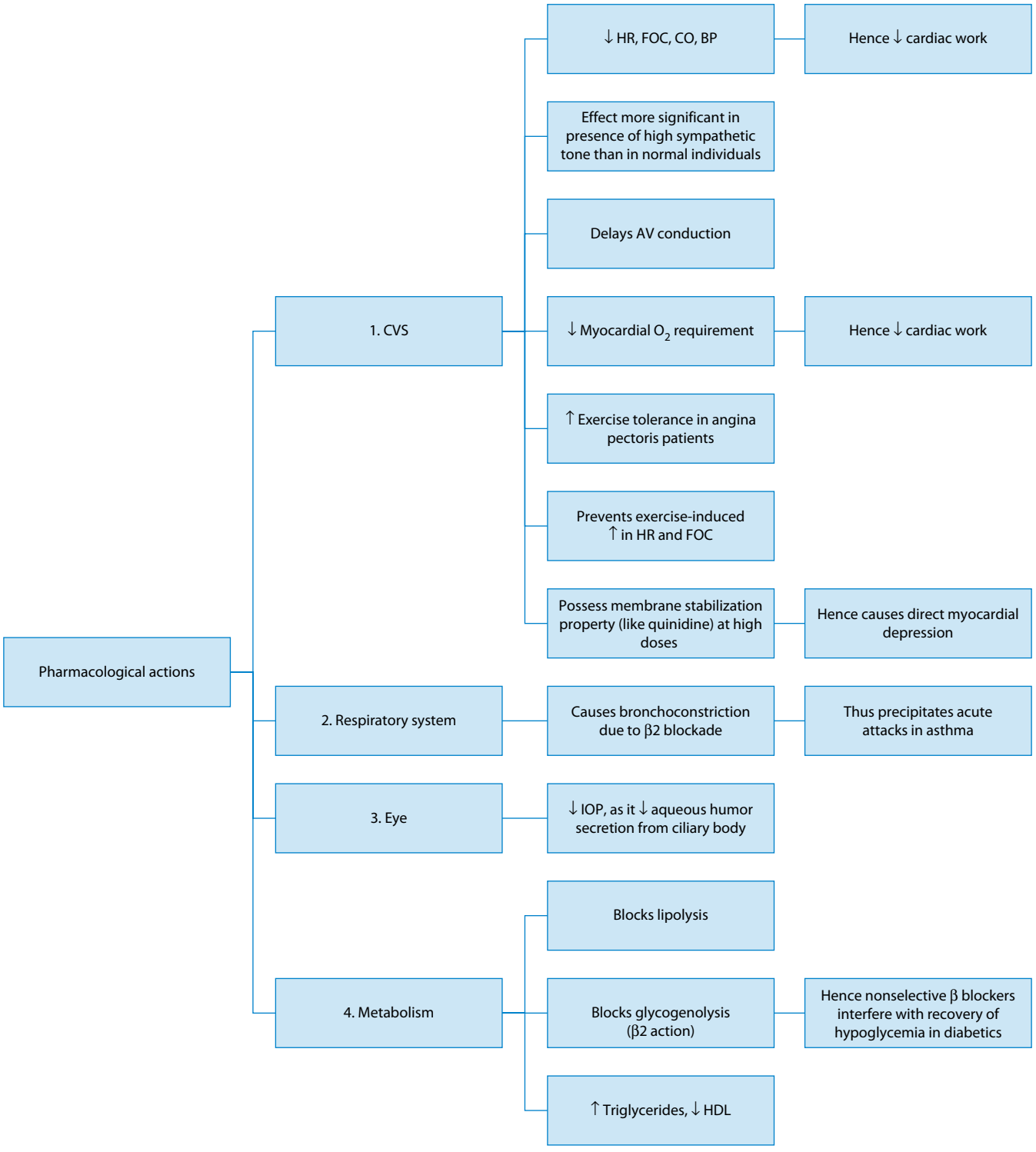
13

Beta-adrenergic blockers (β blockers)

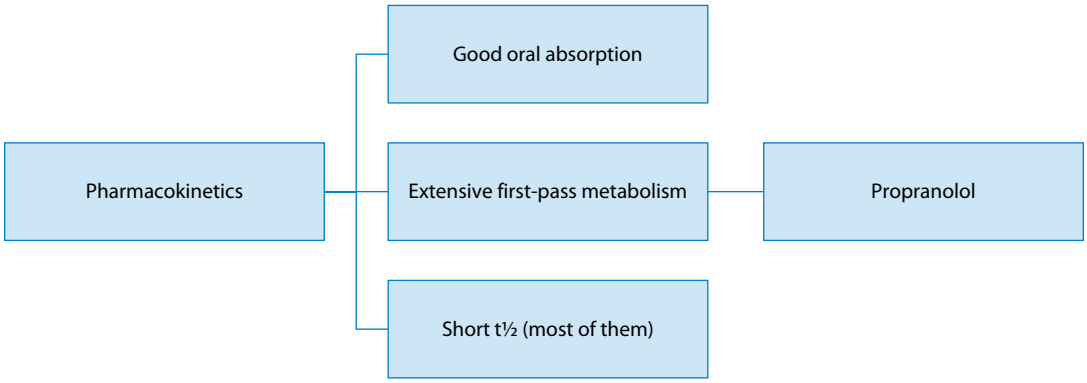
13.1 CLASSIFICATION



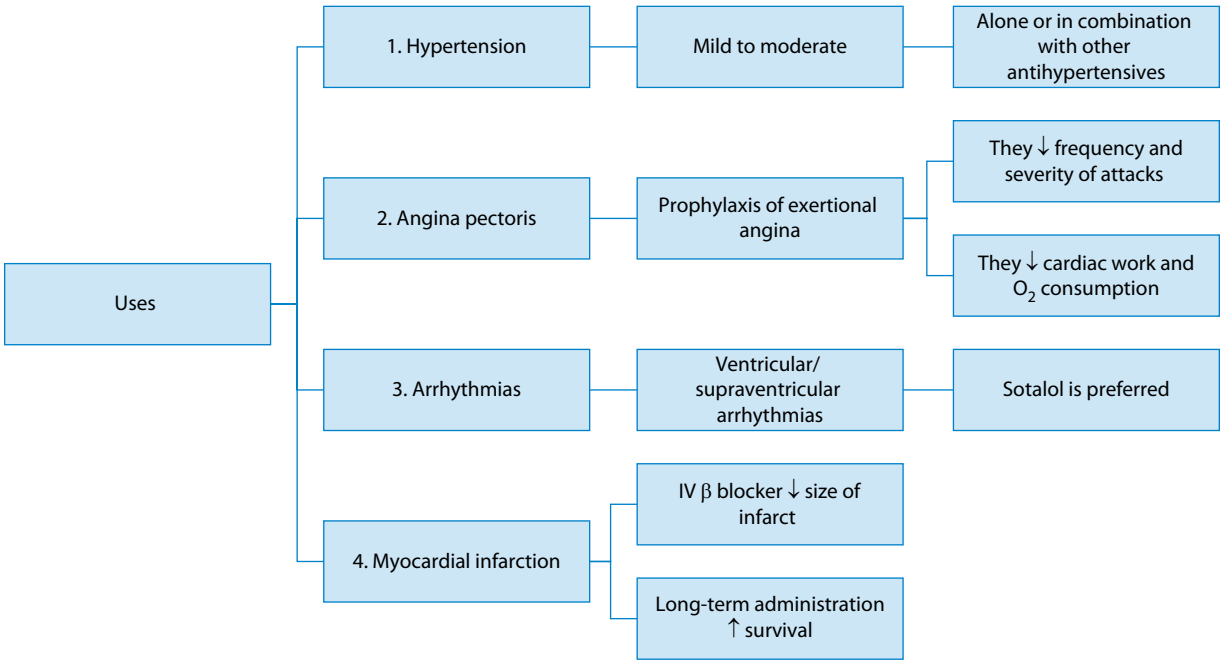
13.2 PHARMACOLOGICAL ACTIONS



13.3 PHARMACOKINETICS

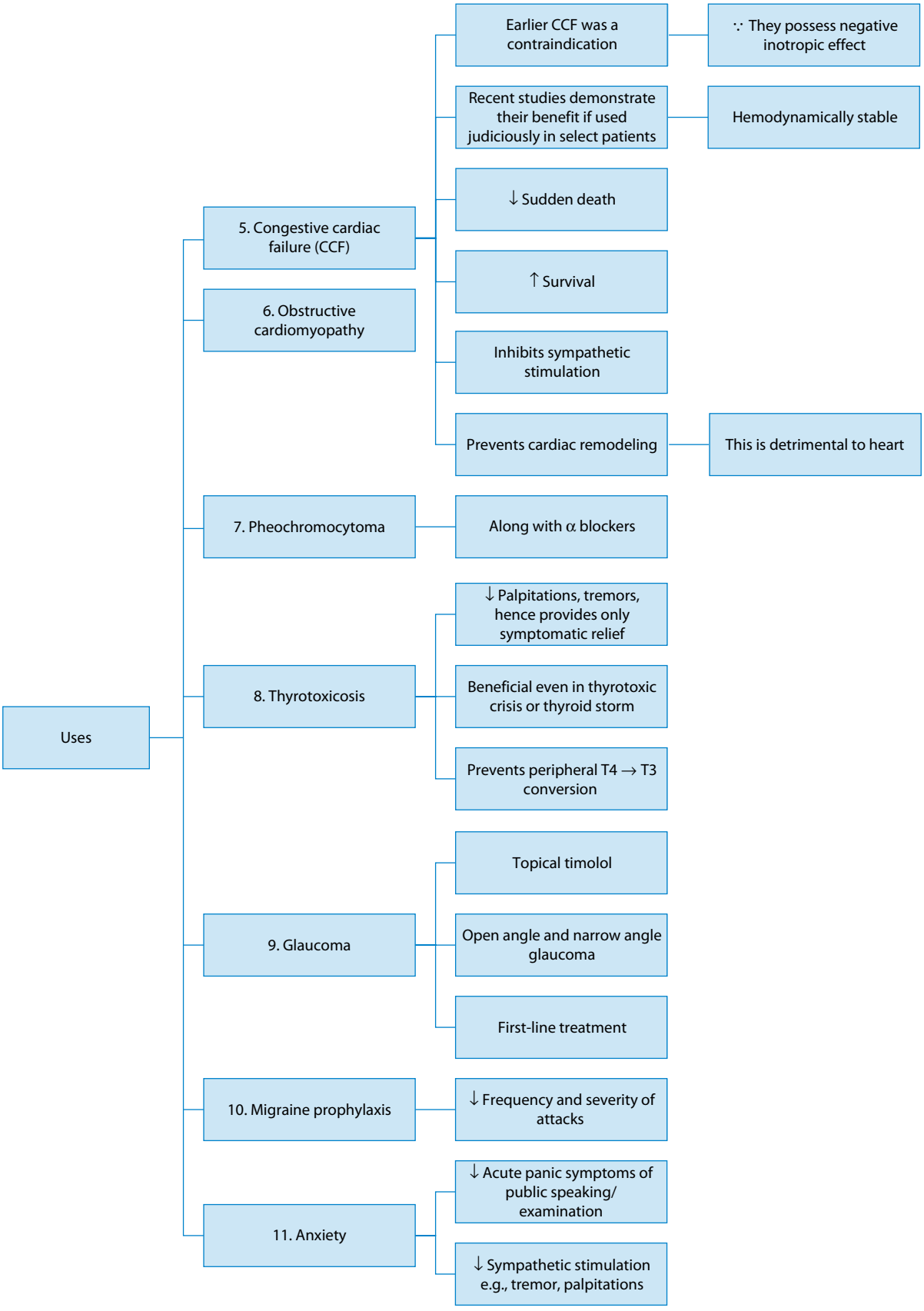


13.4 USES

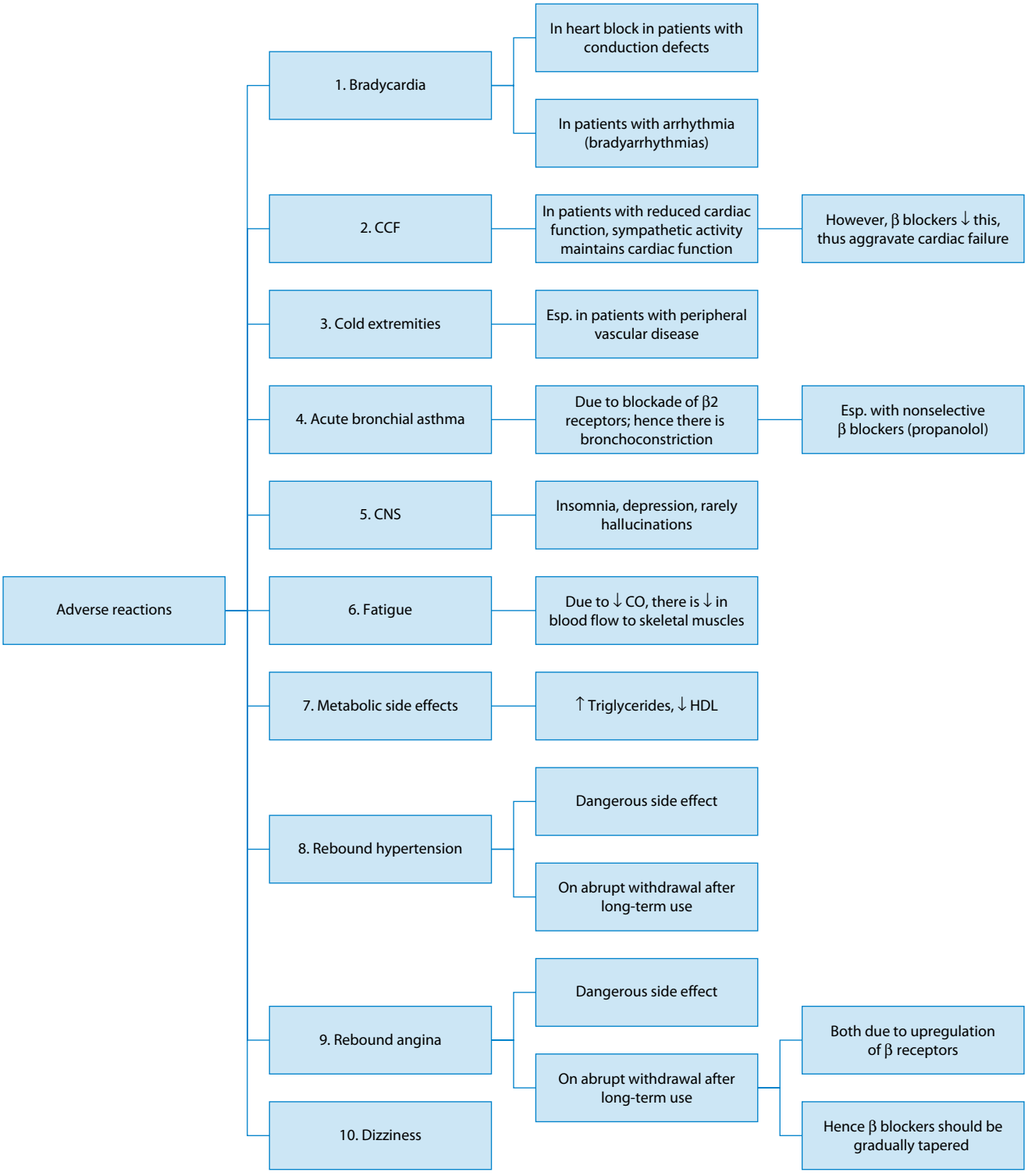


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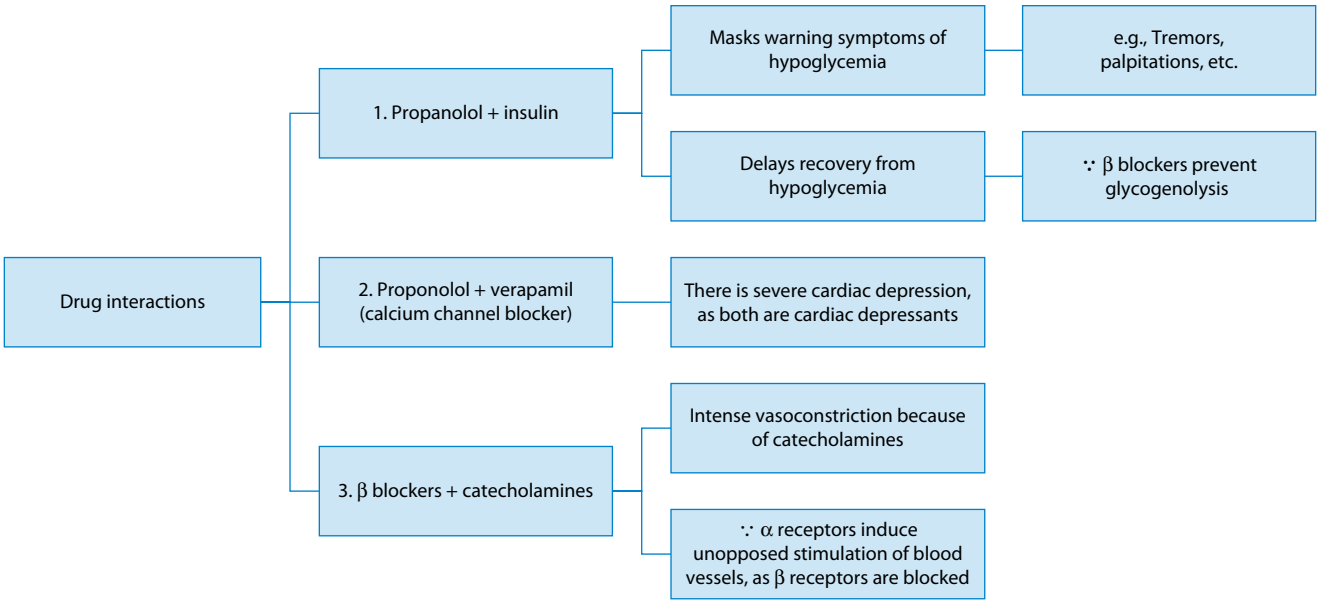
13.4 USES (Continued)



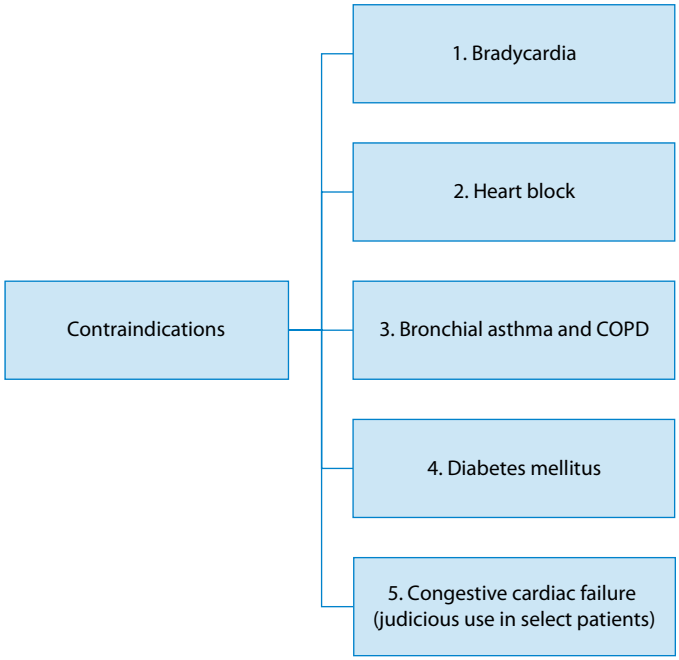
13.5 ADVERSE REACTIONS



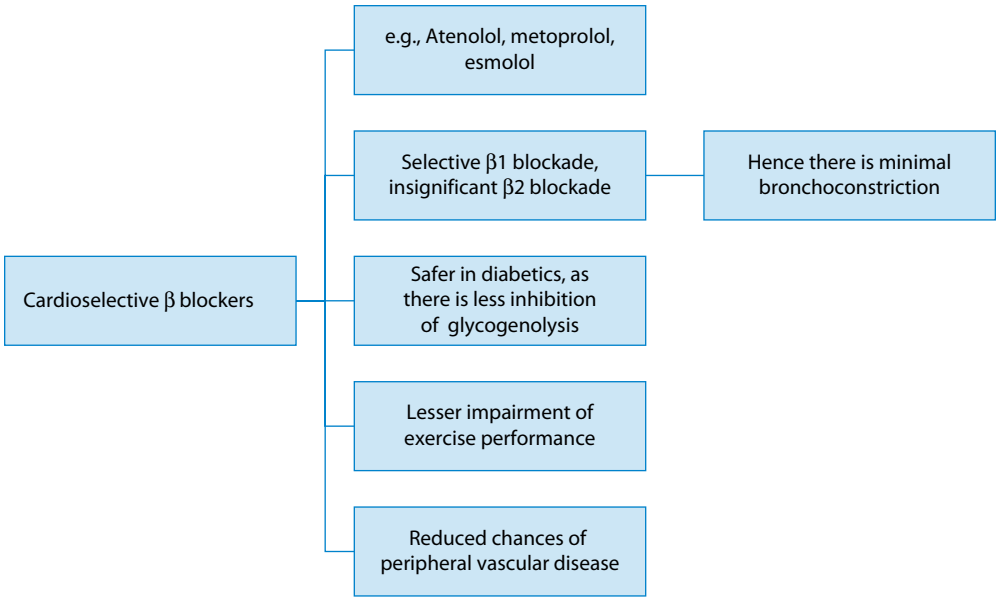
13.6 DRUG INTERACTIONS



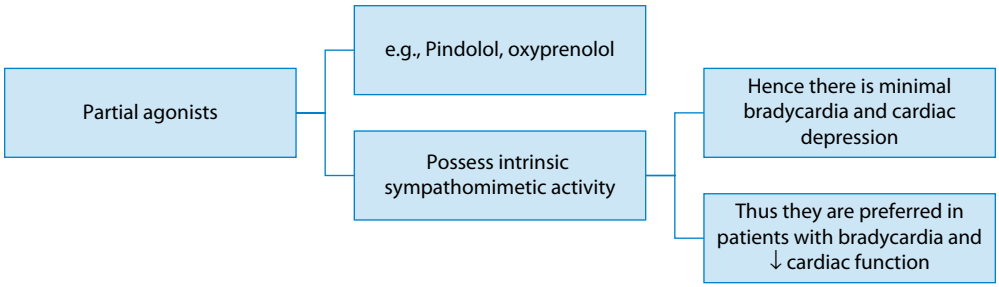
13.7 CONTRAINDICATIONS



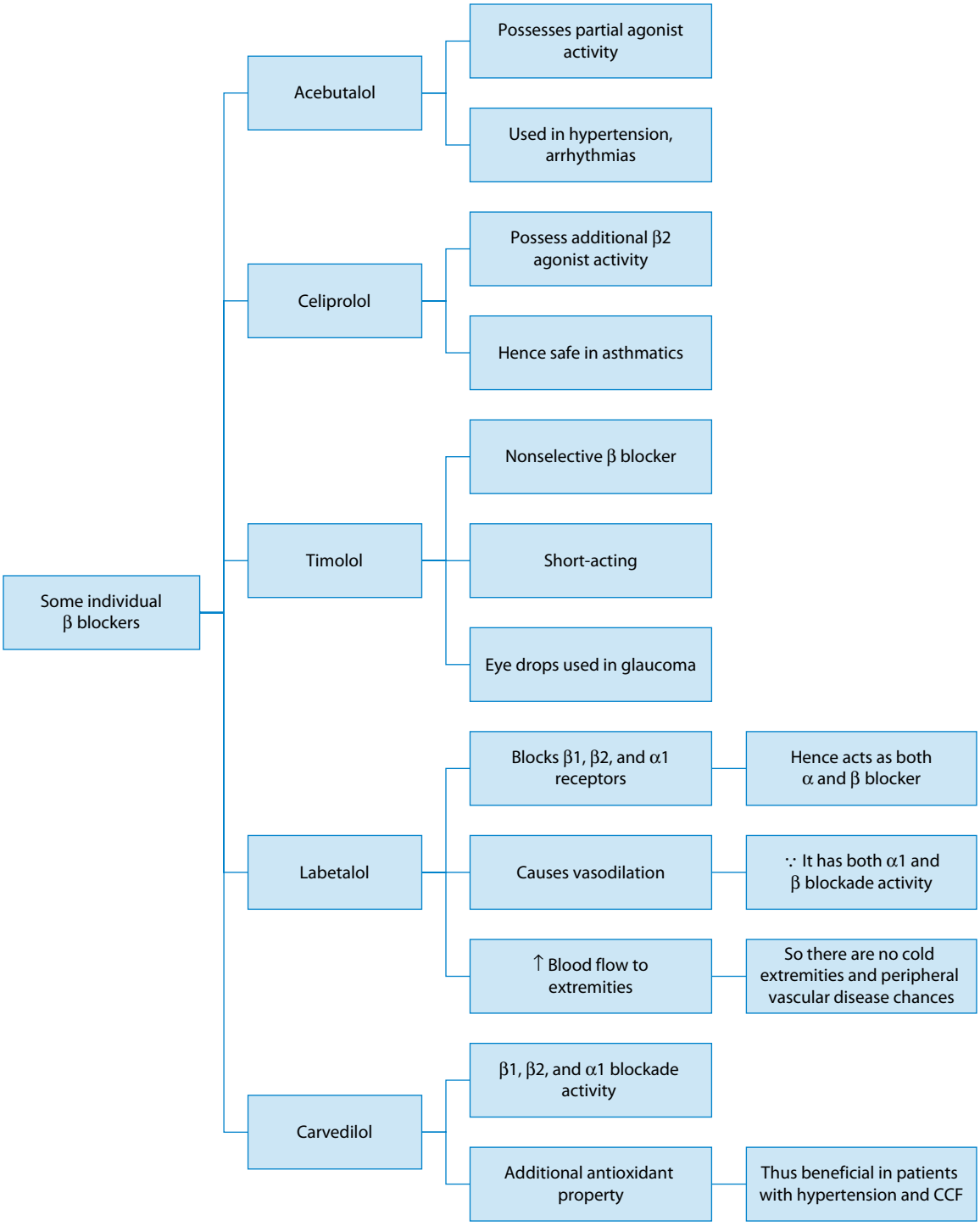
13.8 CARDIOSELECTIVE β BLOCKERS



13.9 PARTIAL AGONISTS



13.10 SOME INDIVIDUAL β BLOCKERS





Taylor & Francis
Taylor & Francis Group
http://taylorandfrancis.com

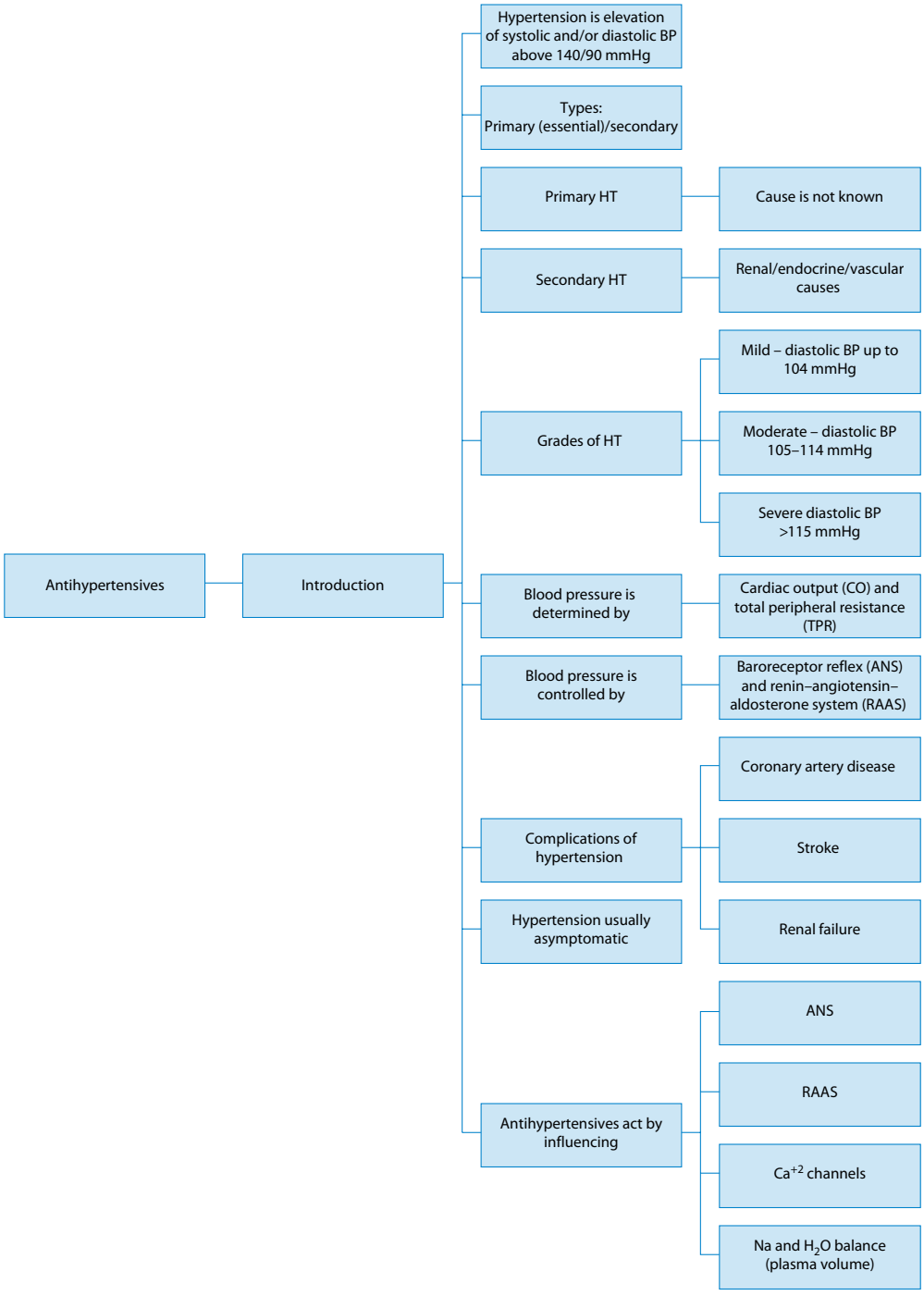
PART III

Cardiovascular pharmacology

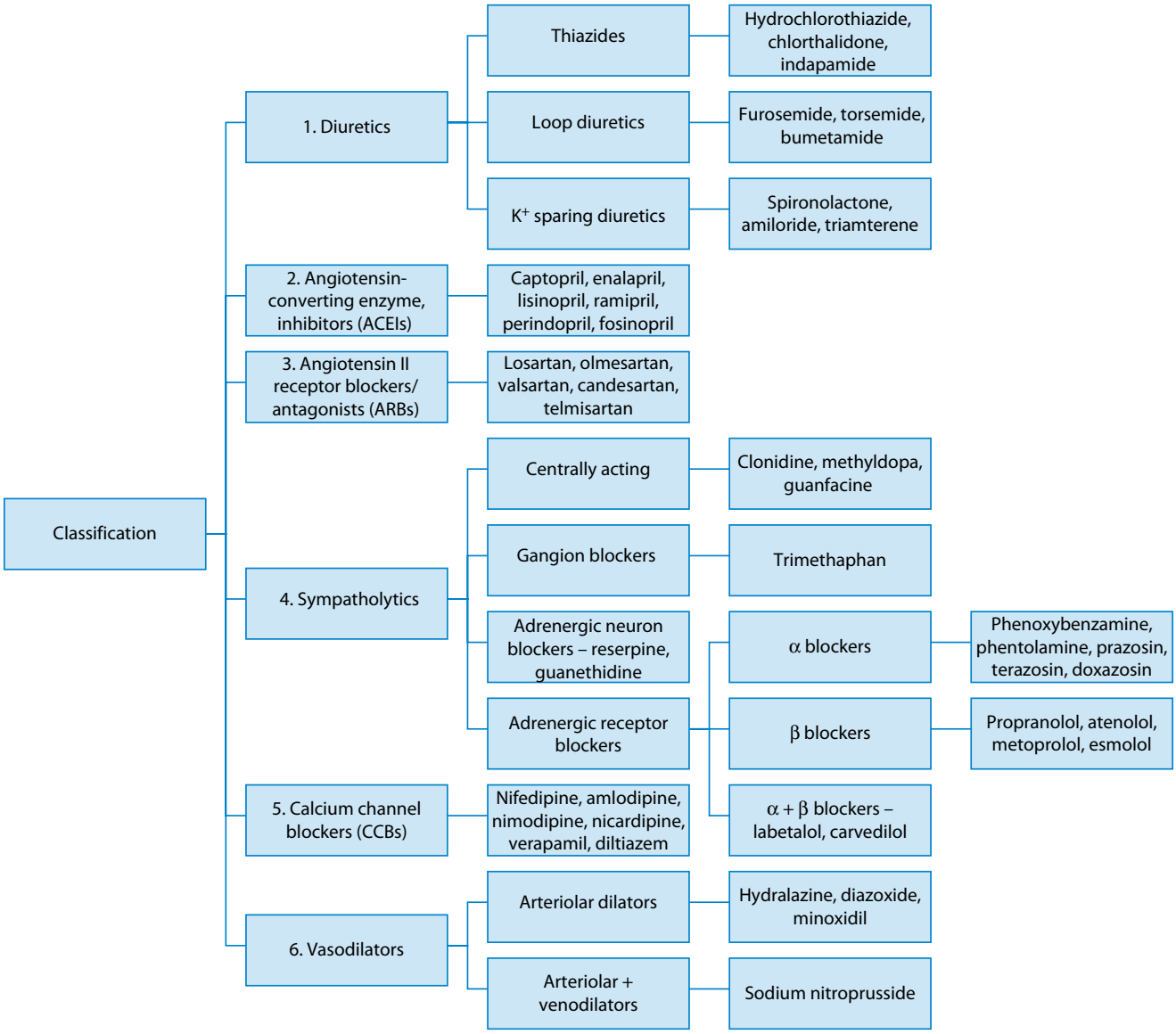
14

Antihypertensives

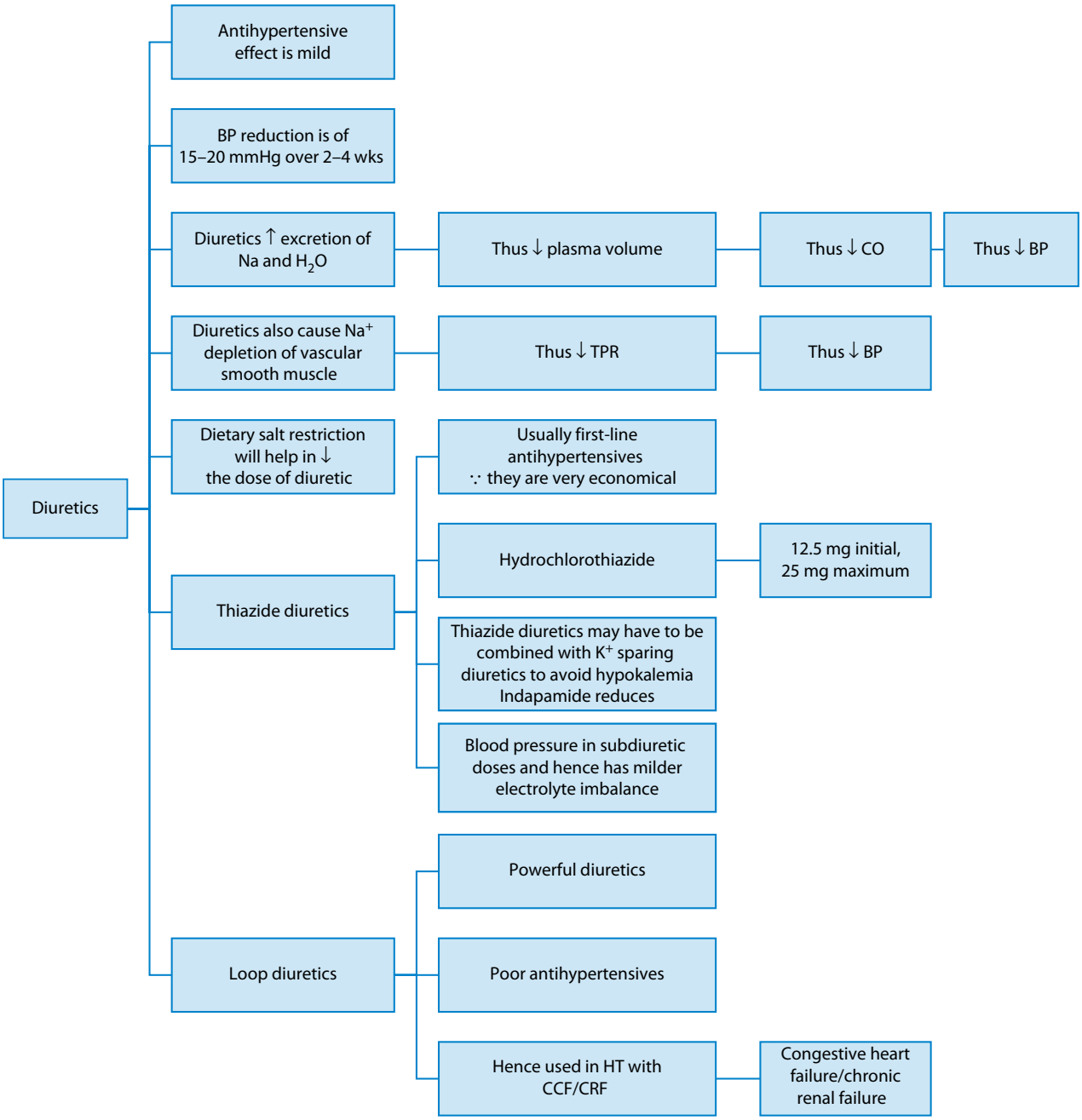
14.1 INTRODUCTION



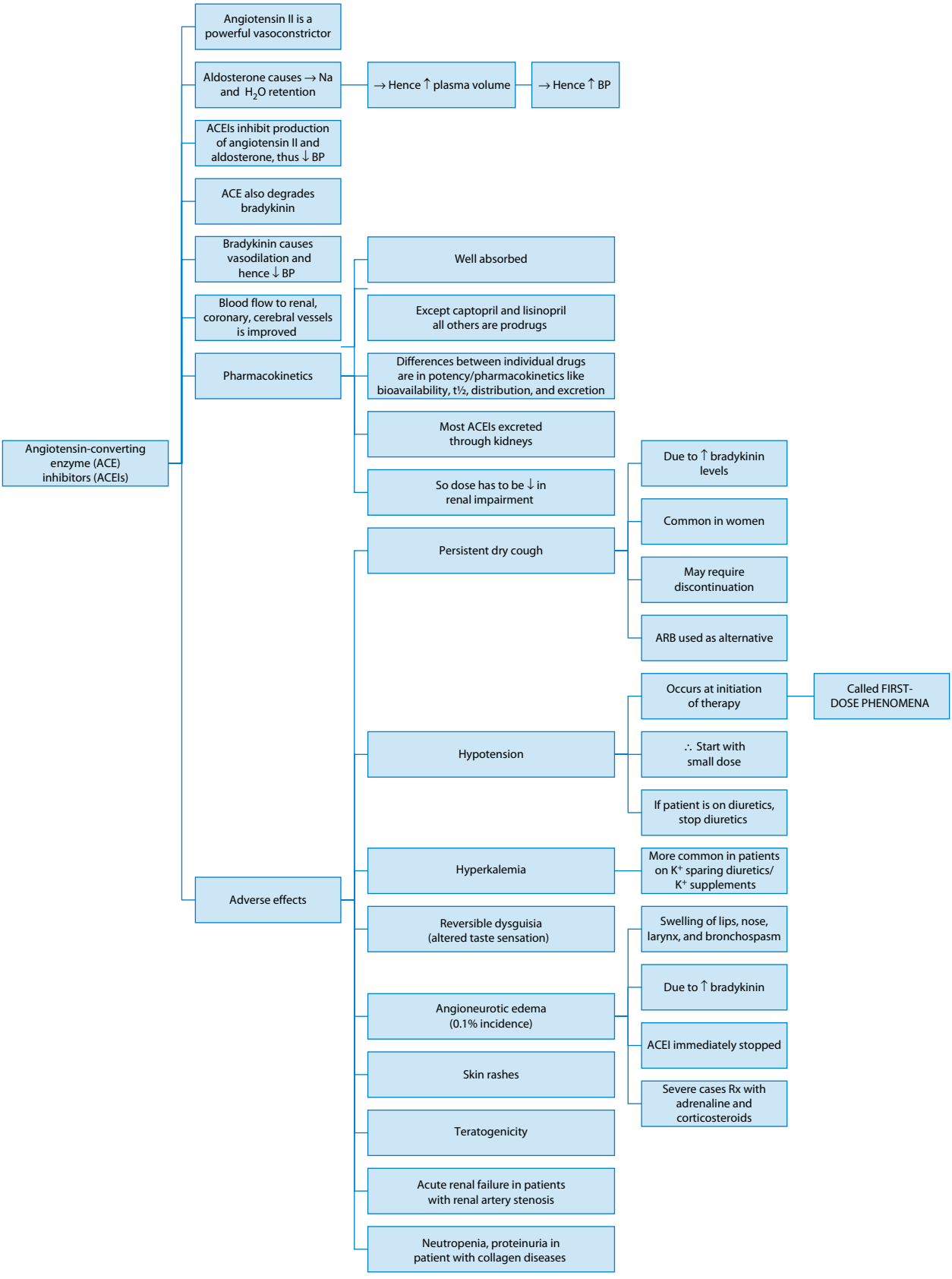
14.2 CLASSIFICATION



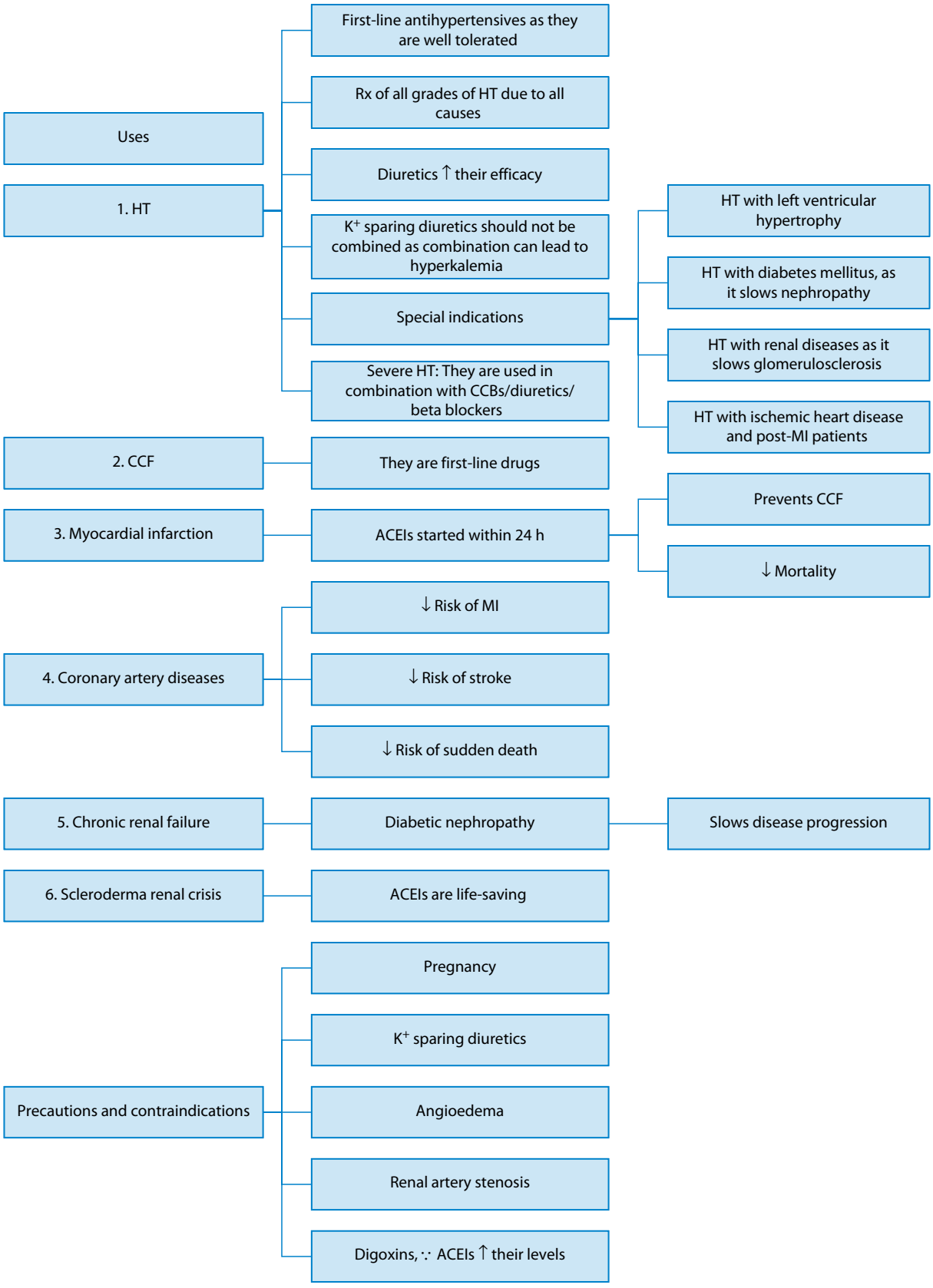
14.3 DIURETICS



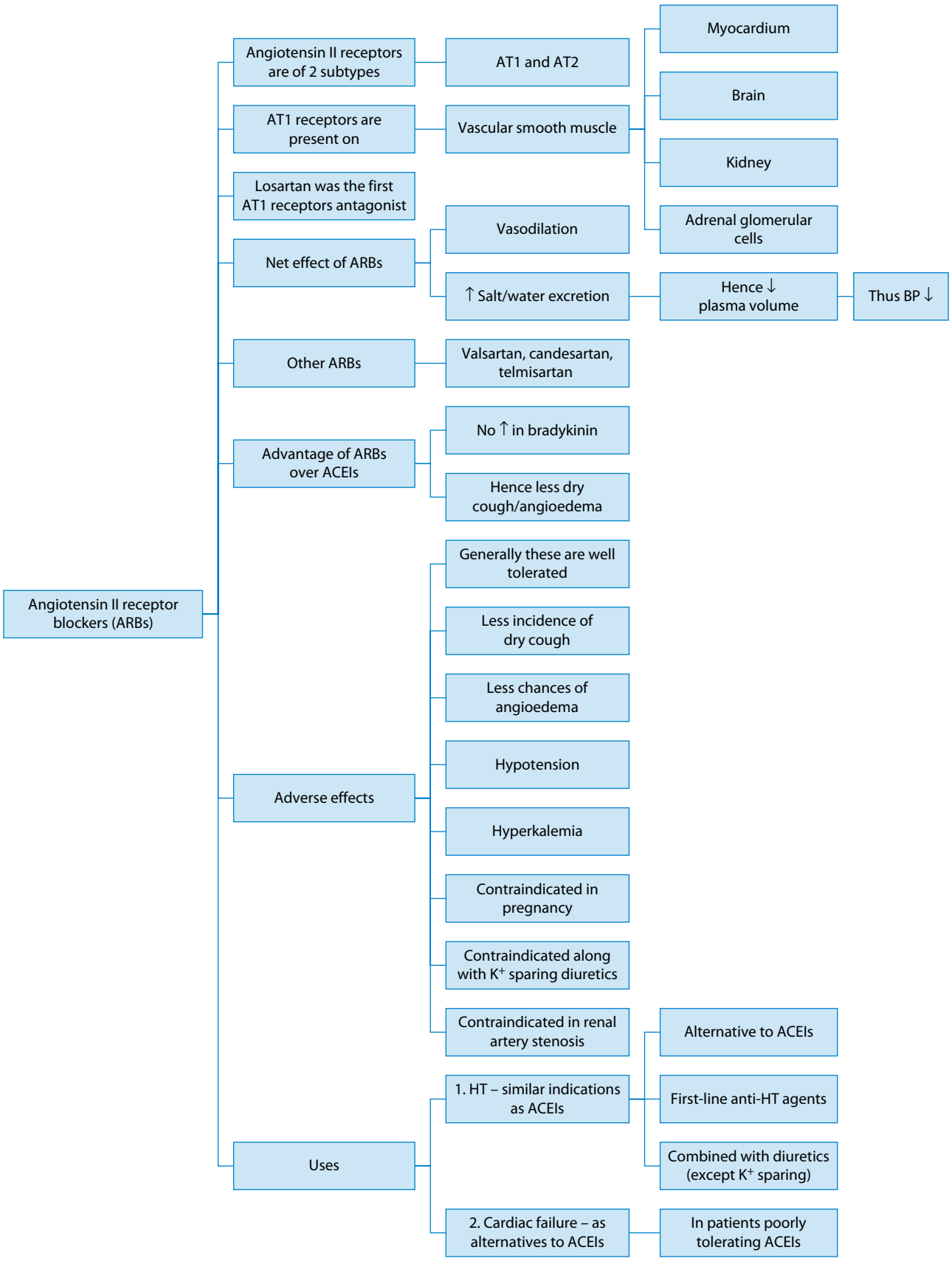
14.4 ANGIOTENSIN-CONVERTING ENZYME (ACE) INHIBITORS (ACEIs) AND ADRs



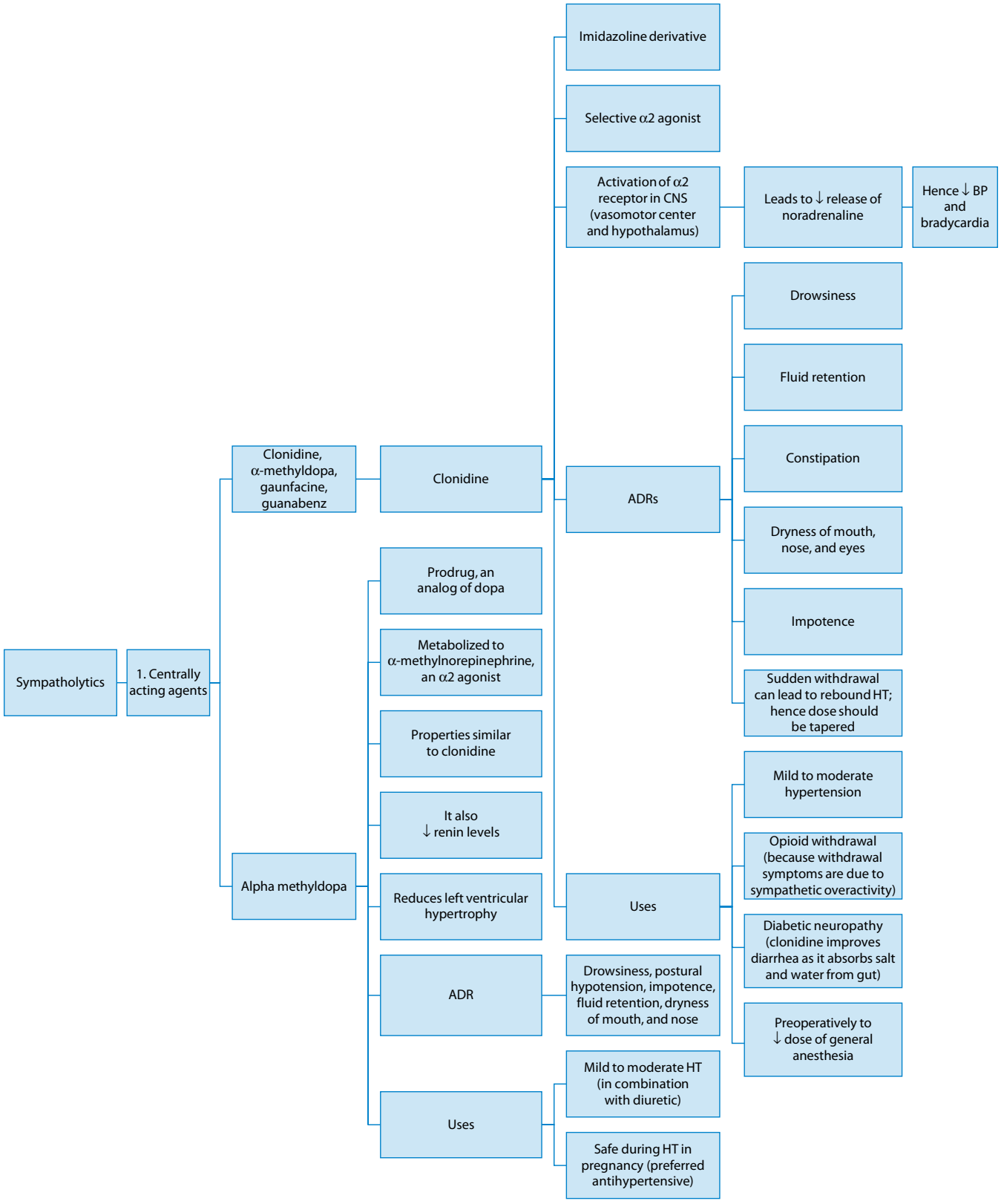
14.5 ANGIOTENSIN-CONVERTING ENZYME (ACE) INHIBITORS (ACEIs) – USES, PRECAUTIONS, AND CONTRAINDICATIONS



14.6 ANGIOTENSIN II RECEPTOR BLOCKERS (ARBs)

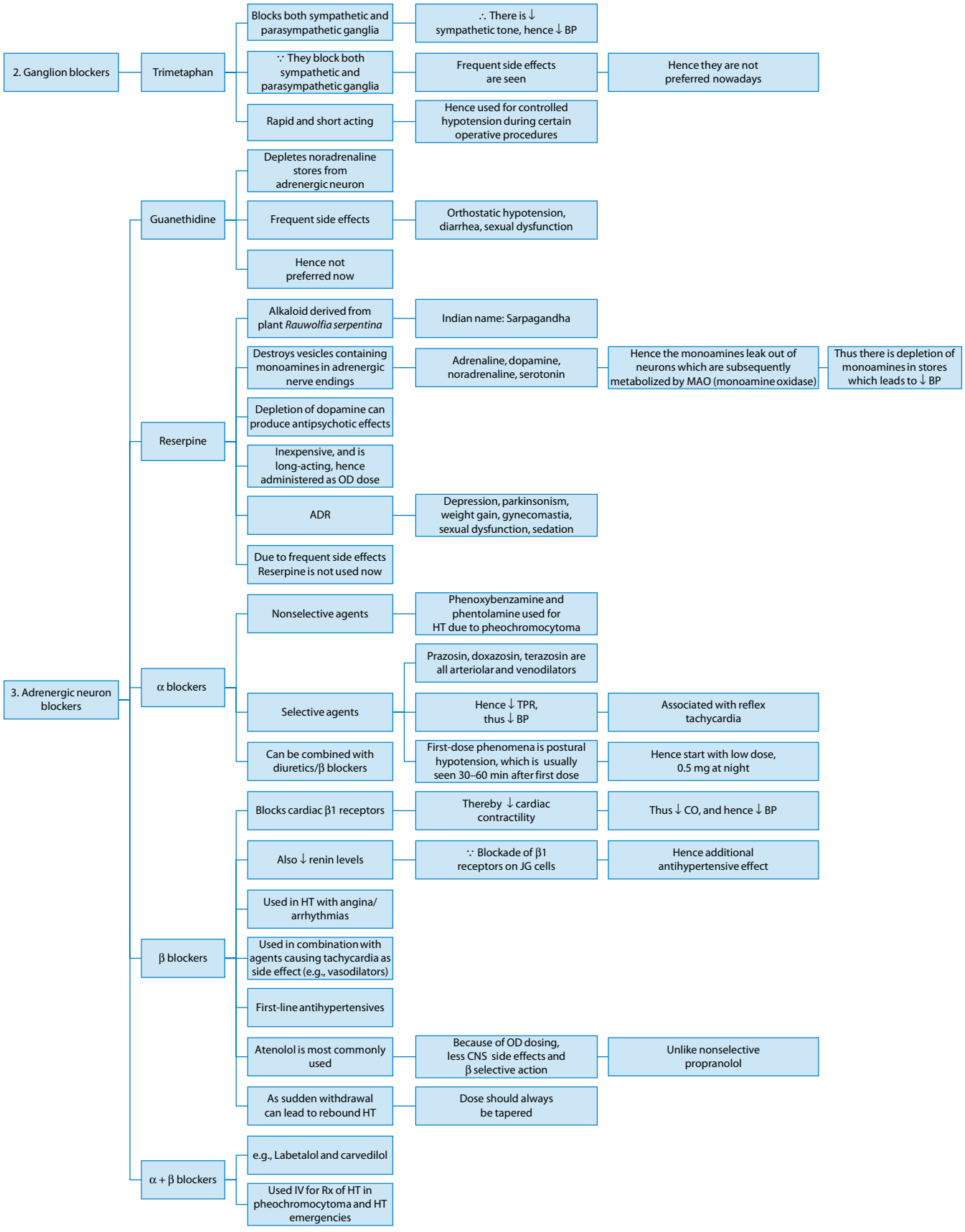


14.7 SYMPATHOLYTICS

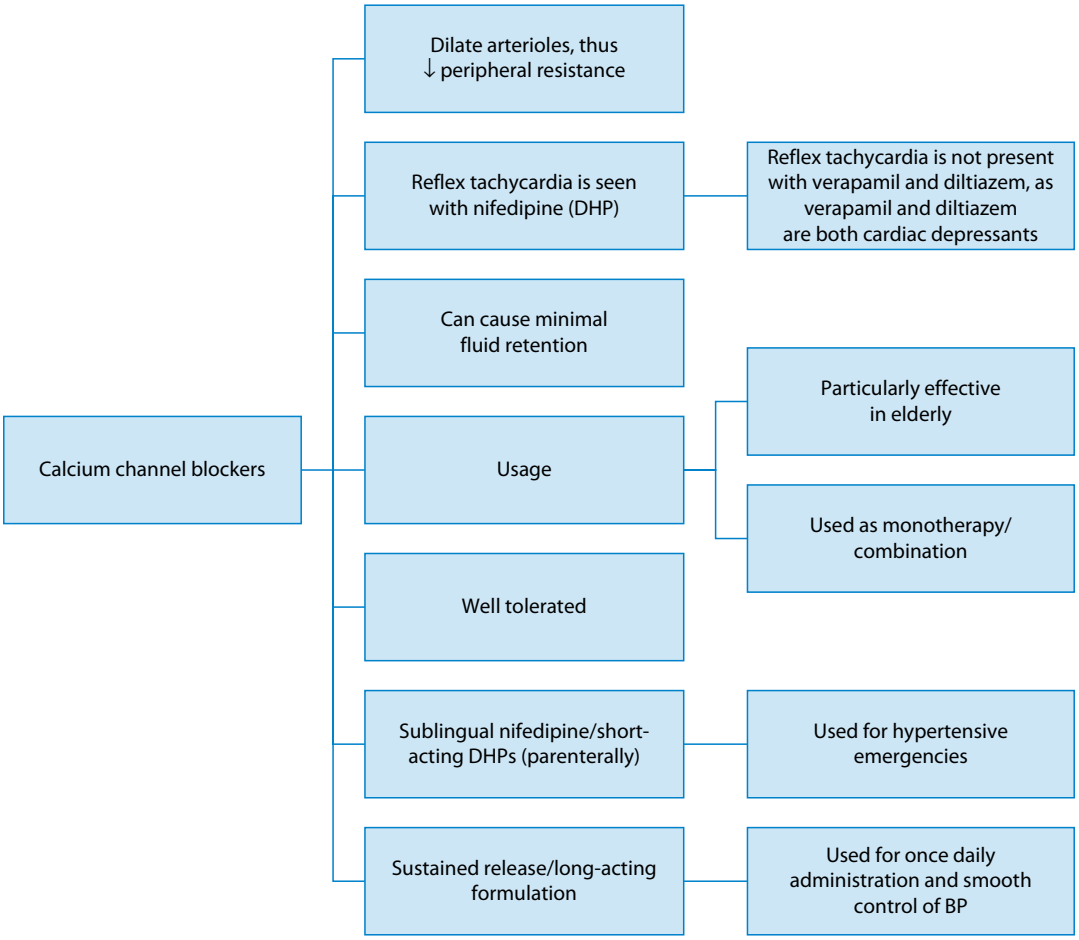


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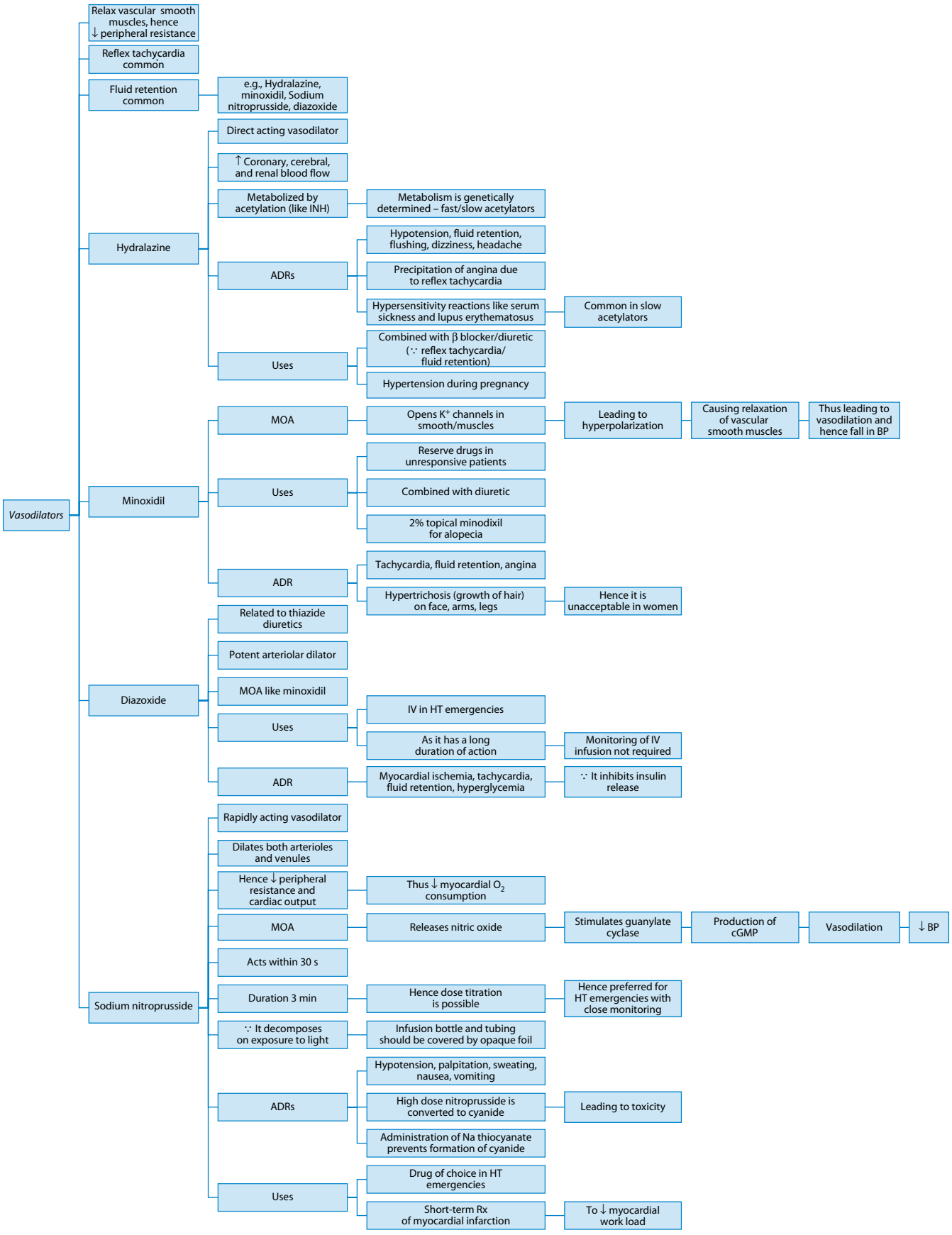
14.7 SYMPATHOLYTICS (Continued)



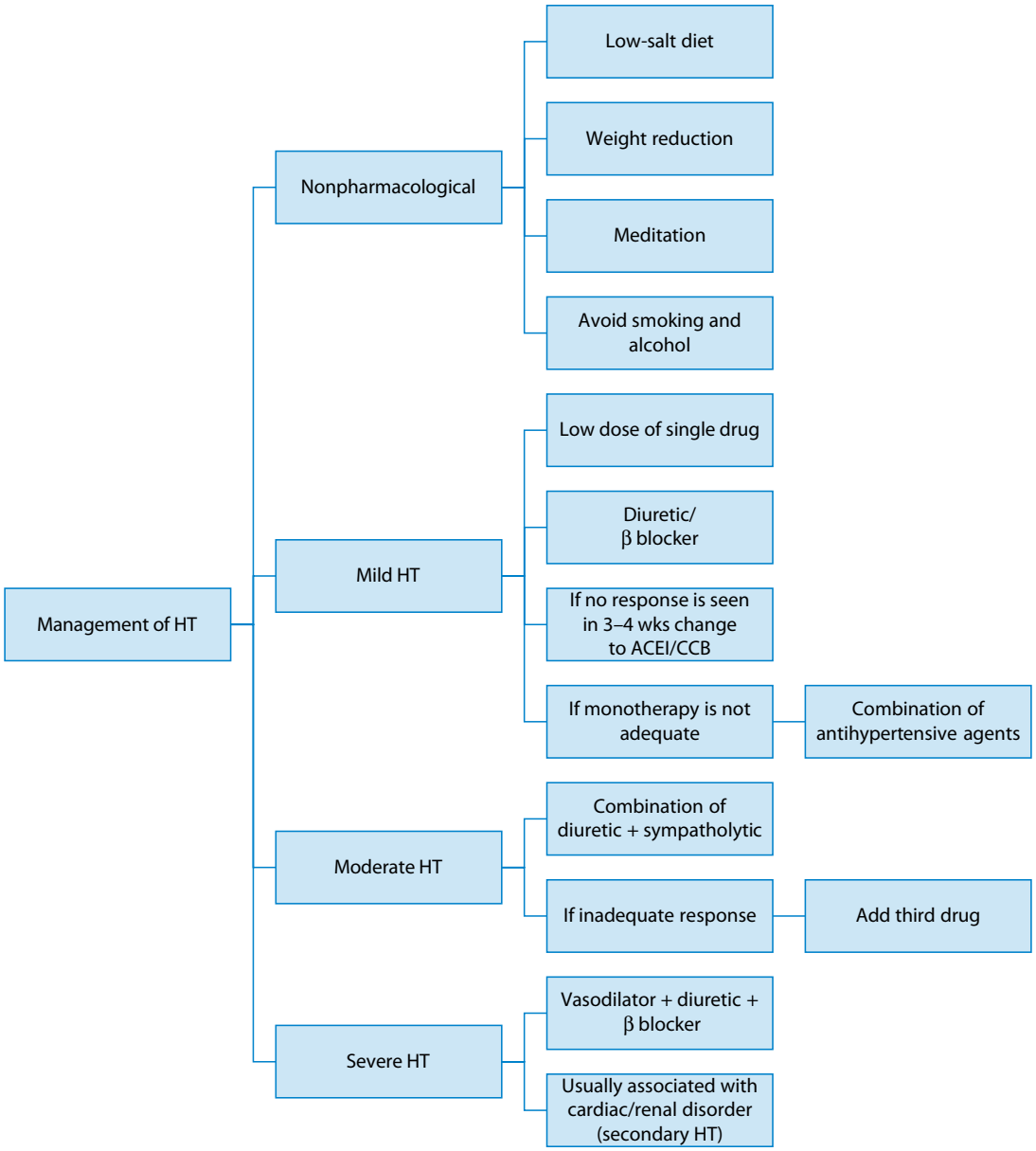
14.8 CALCIUM CHANNEL BLOCKERS



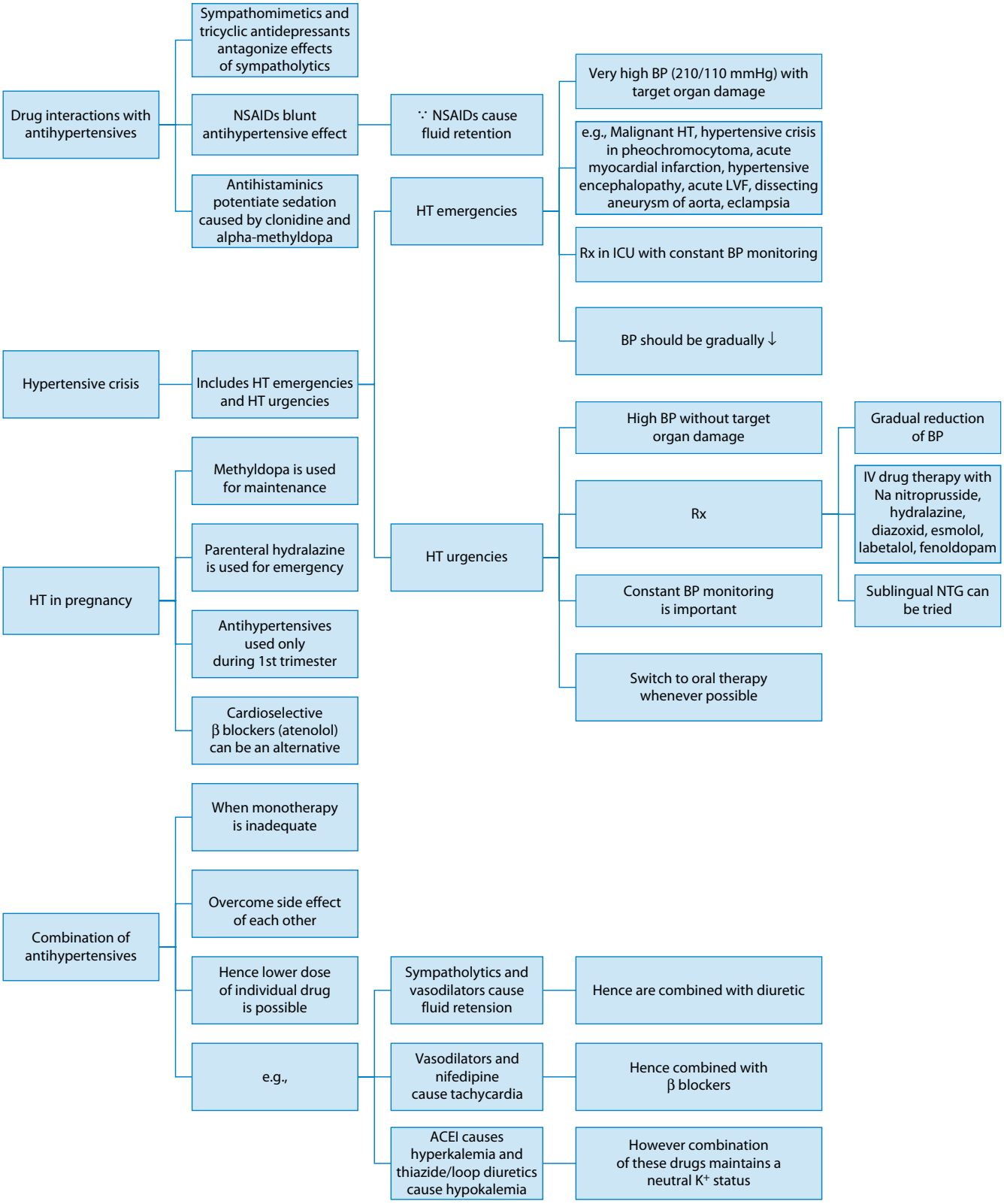
14.9 VASODILATORS



14.10 MANAGEMENT OF HT



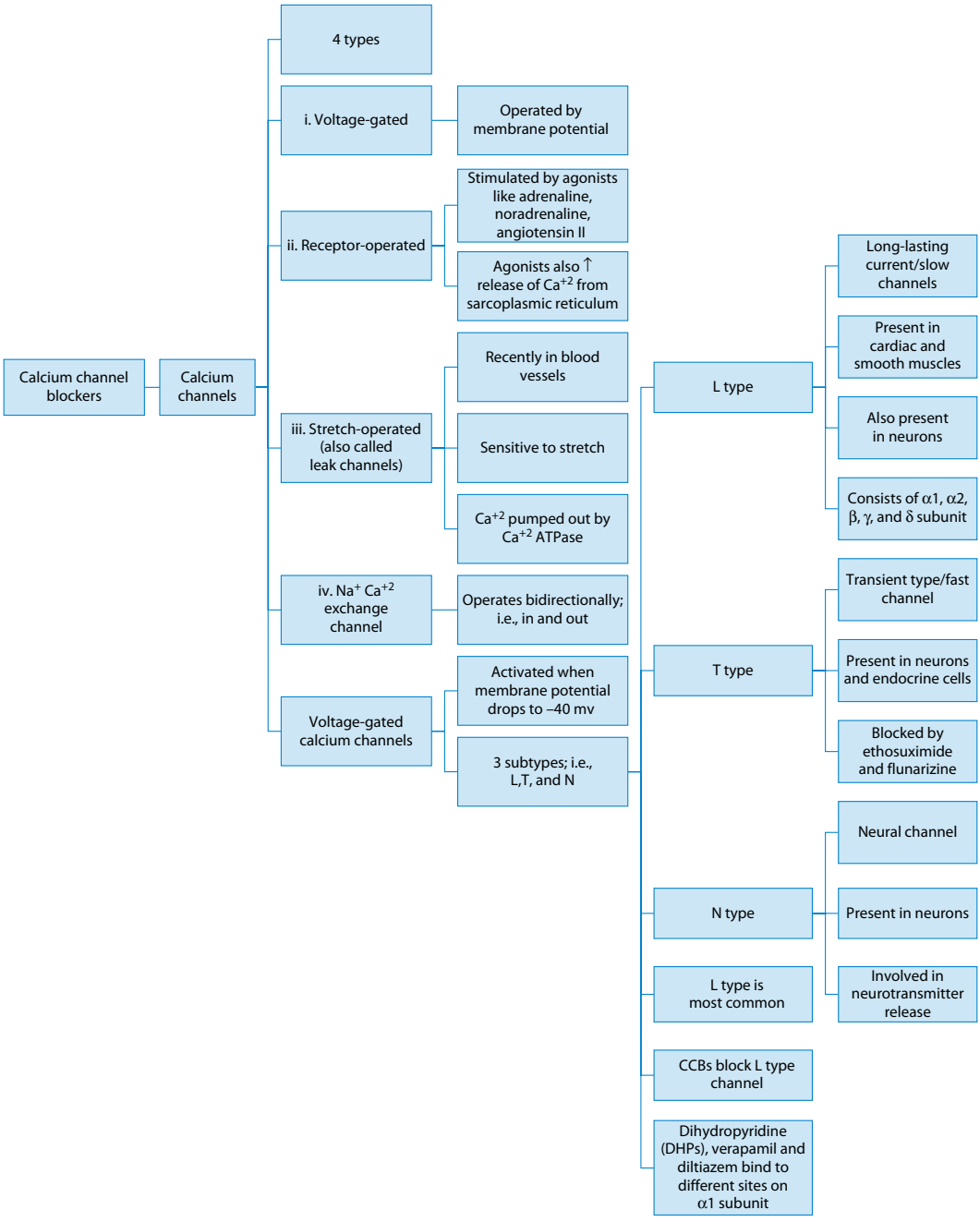
14.11 DRUG INTERACTIONS WITH ANTIHYPERTENSIVES, HYPERTENSIVE CRISIS, HT IN PREGNANCY, COMBINATION OF ANTIHYPERTENSIVES



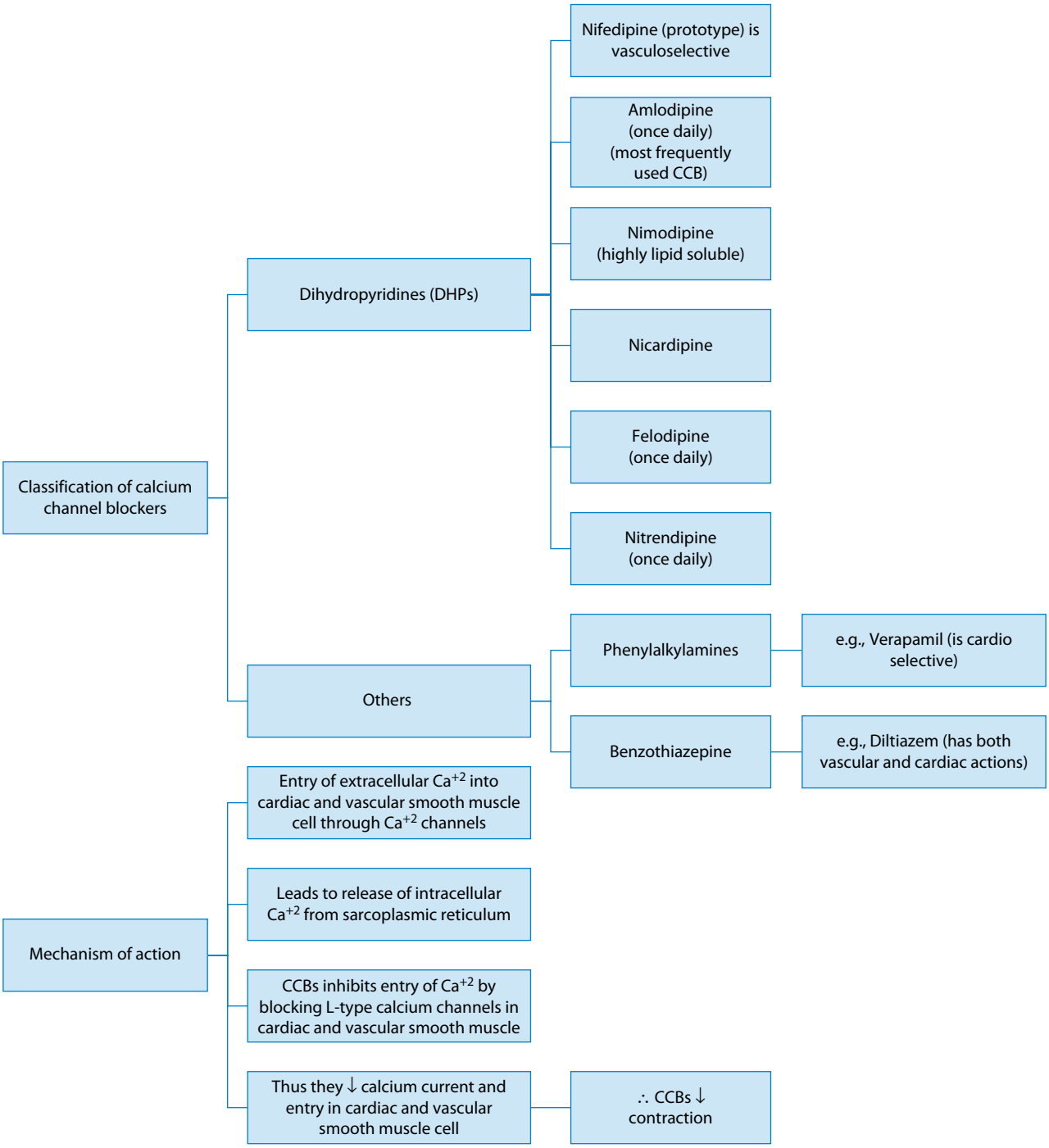
15

Calcium channel blockers, drug treatment of angina pectoris, and myocardial infarction

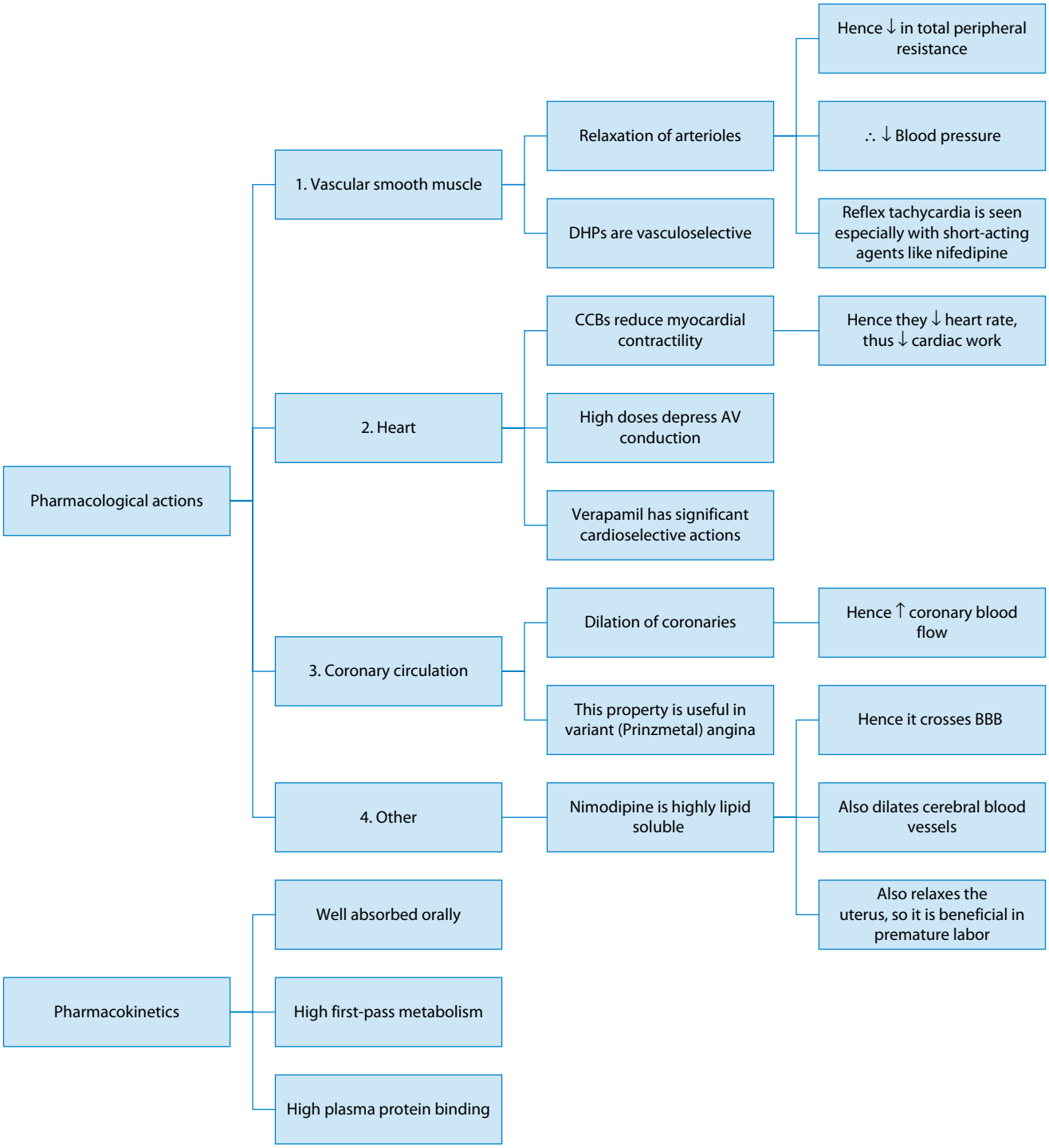
15.1 CALCIUM CHANNELS



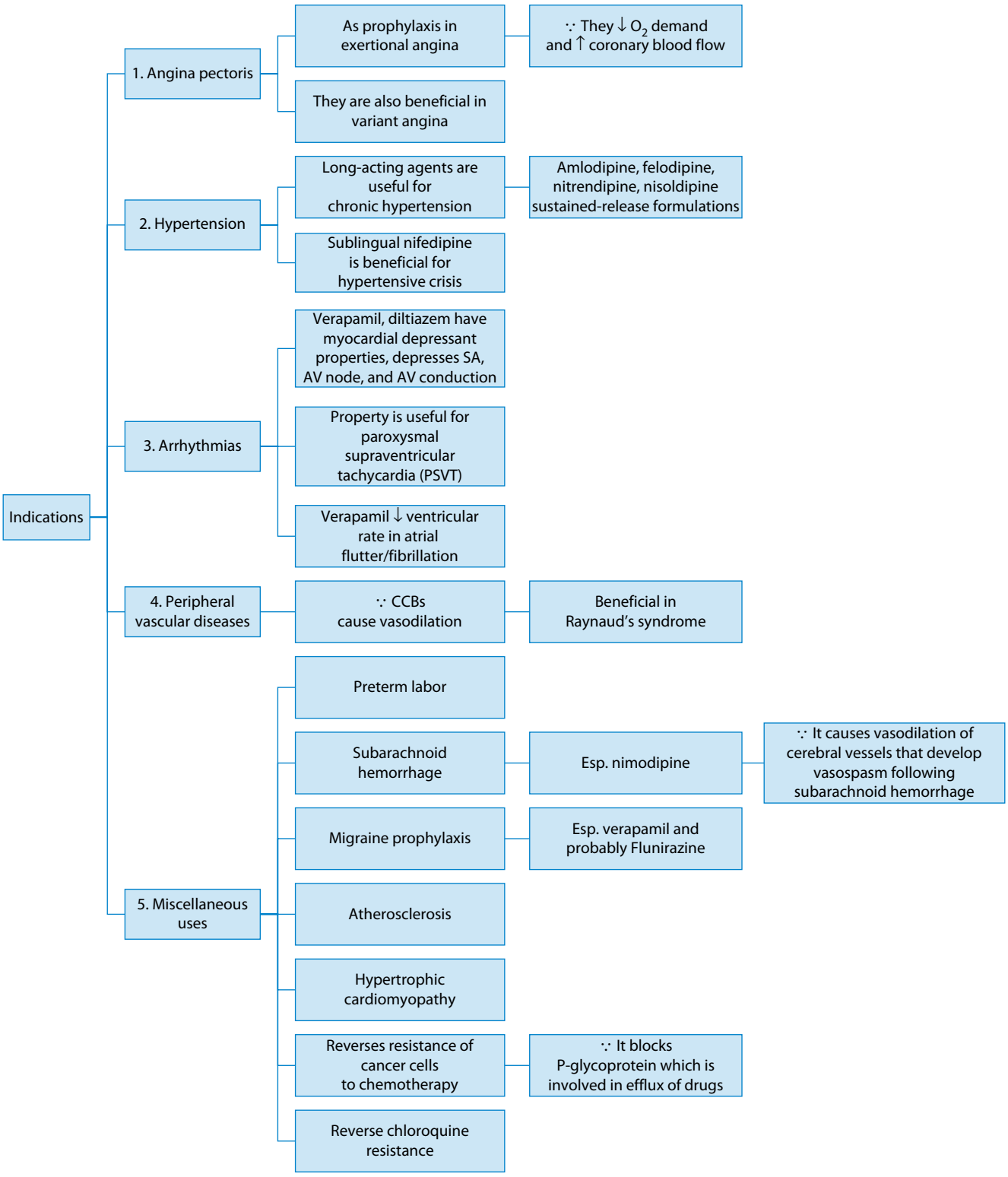
15.2 CLASSIFICATION OF CALCIUM CHANNEL BLOCKERS AND MECHANISM OF ACTION



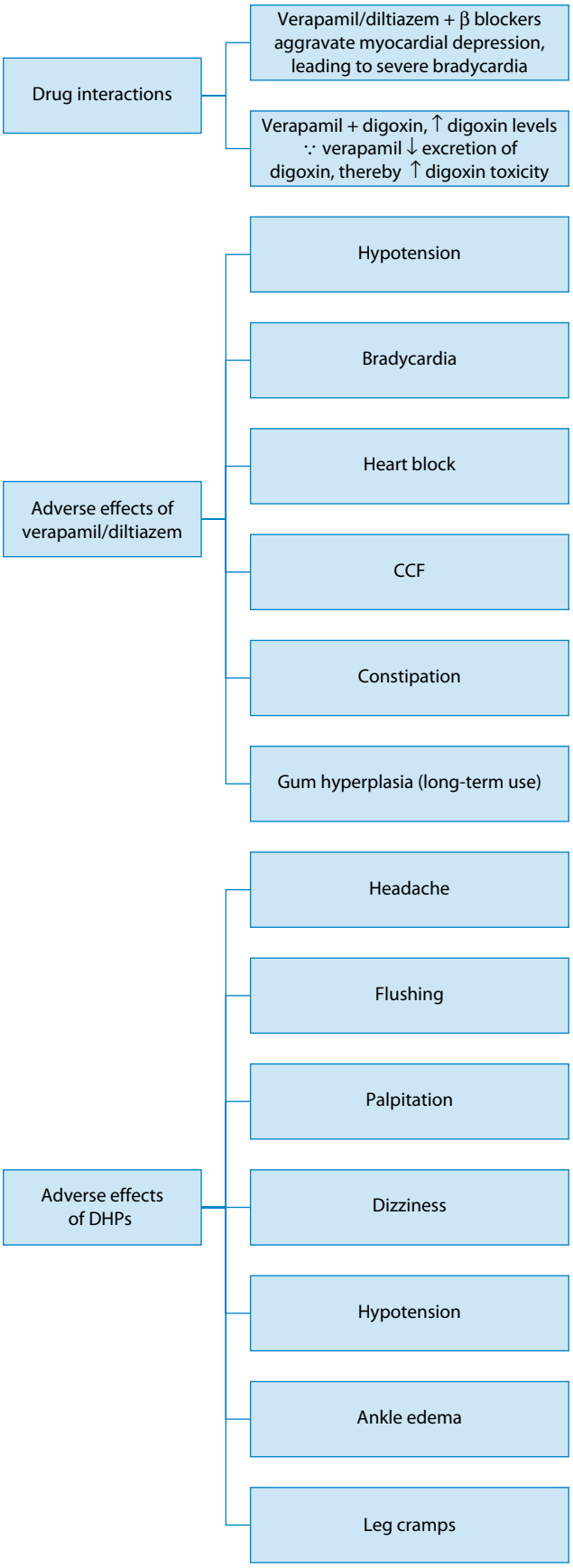
15.3 PHARMACOLOGICAL ACTIONS AND PHARMACOKINETICS



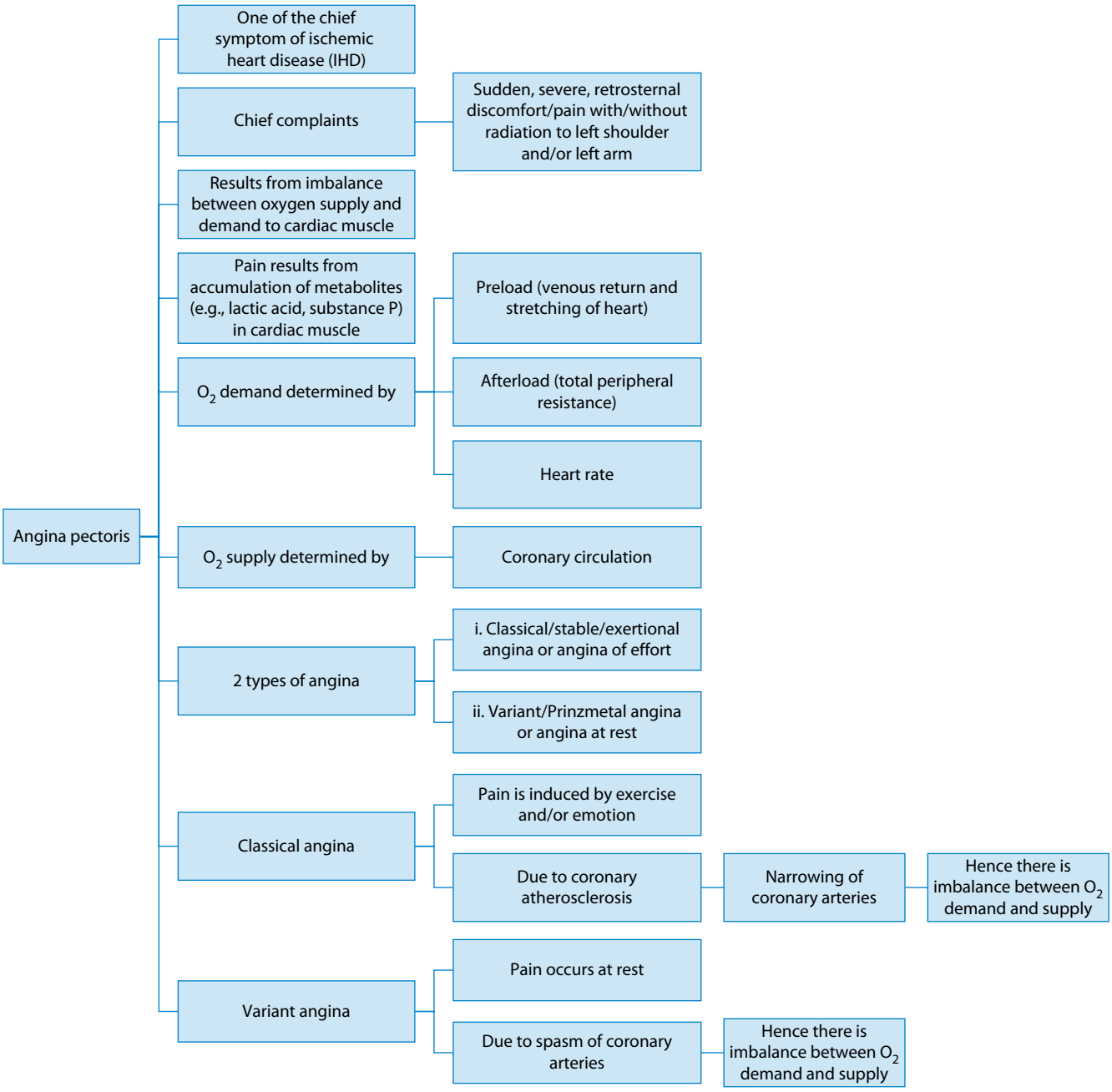
15.4 INDICATIONS



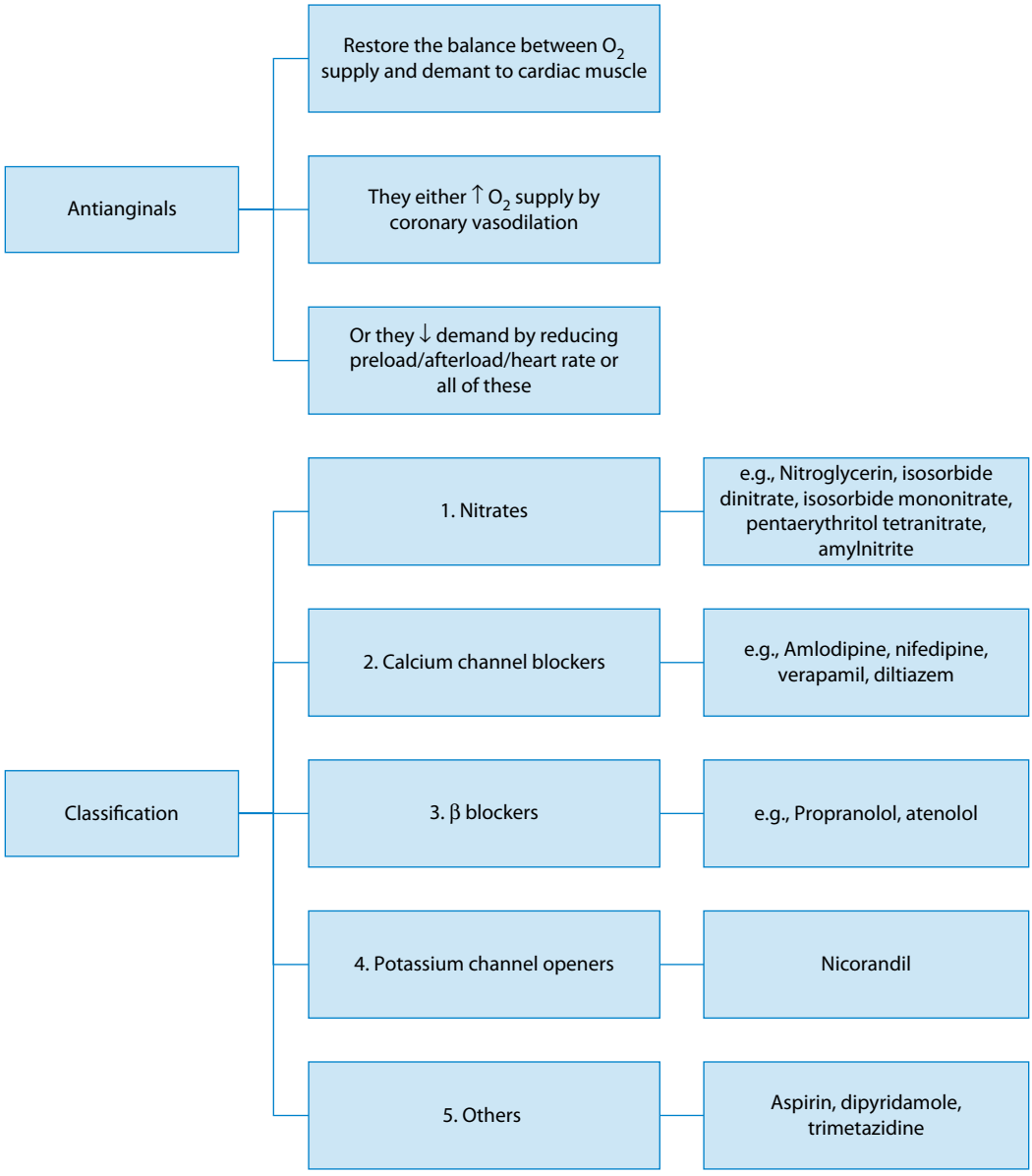
15.5 DRUG INTERACTIONS AND ADRs



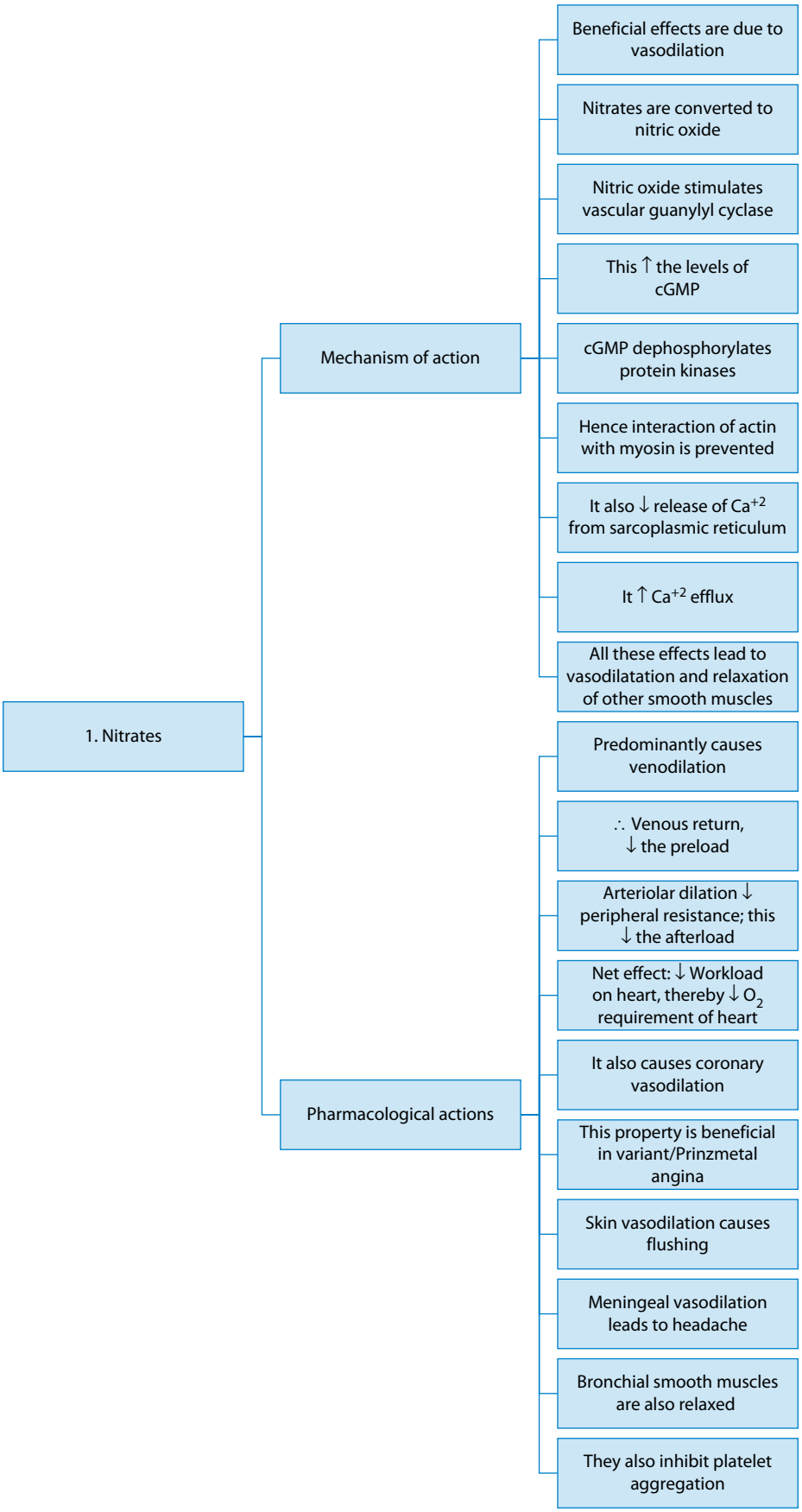
15.6 ANGINA PECTORIS



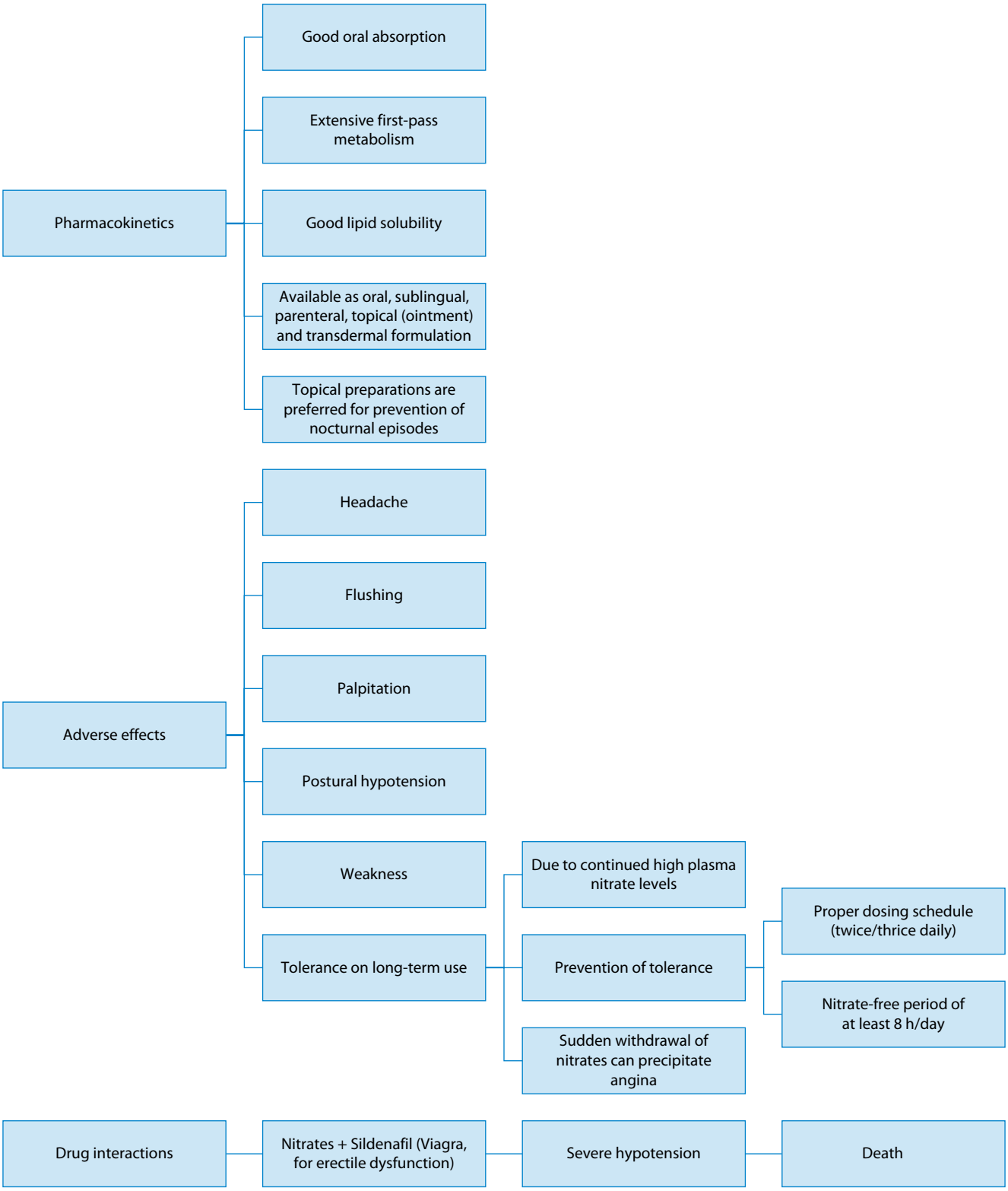
15.7 ANTIANGINALS – CLASSIFICATION



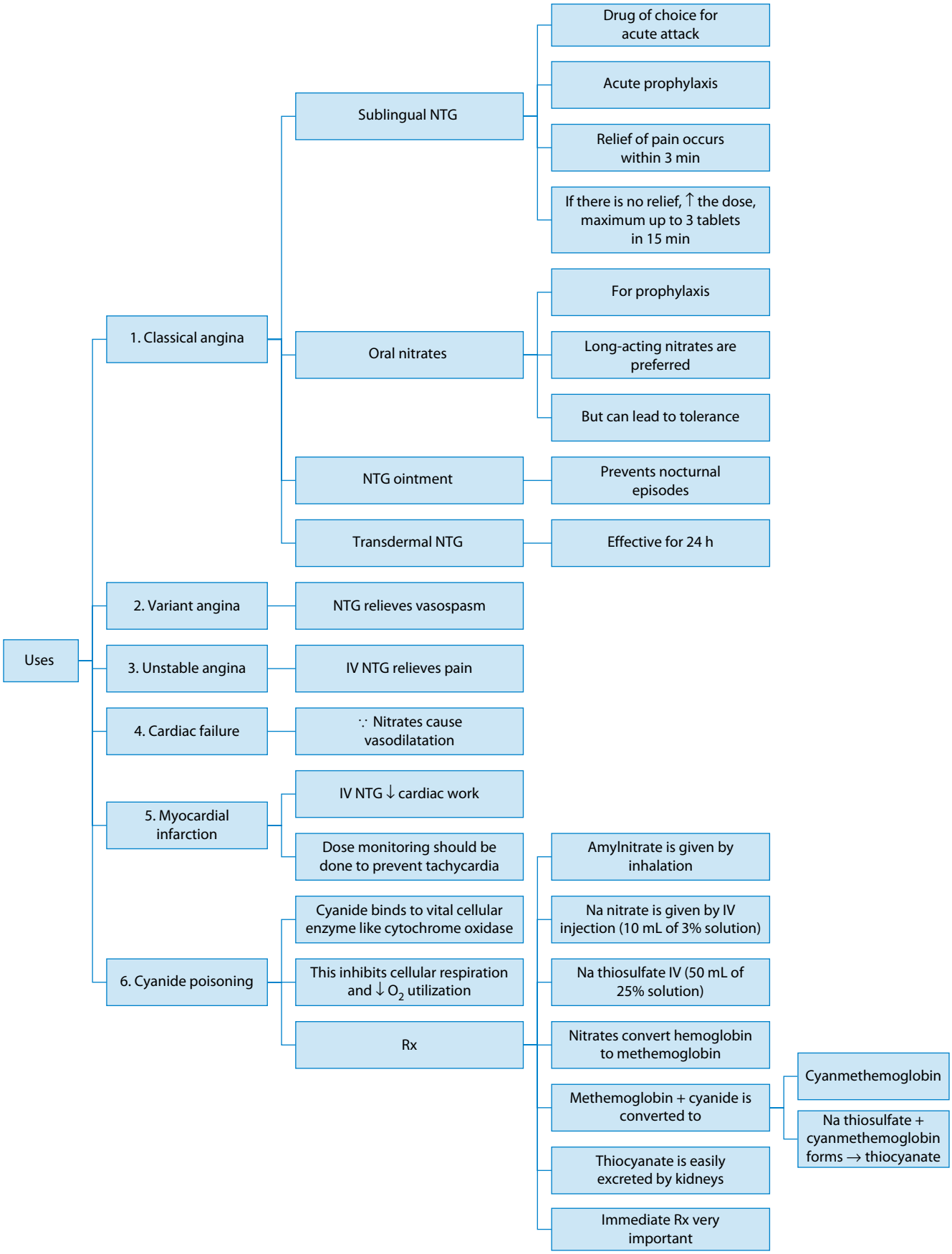
15.8 NITRATES – PHARMACOLOGICAL ACTIONS



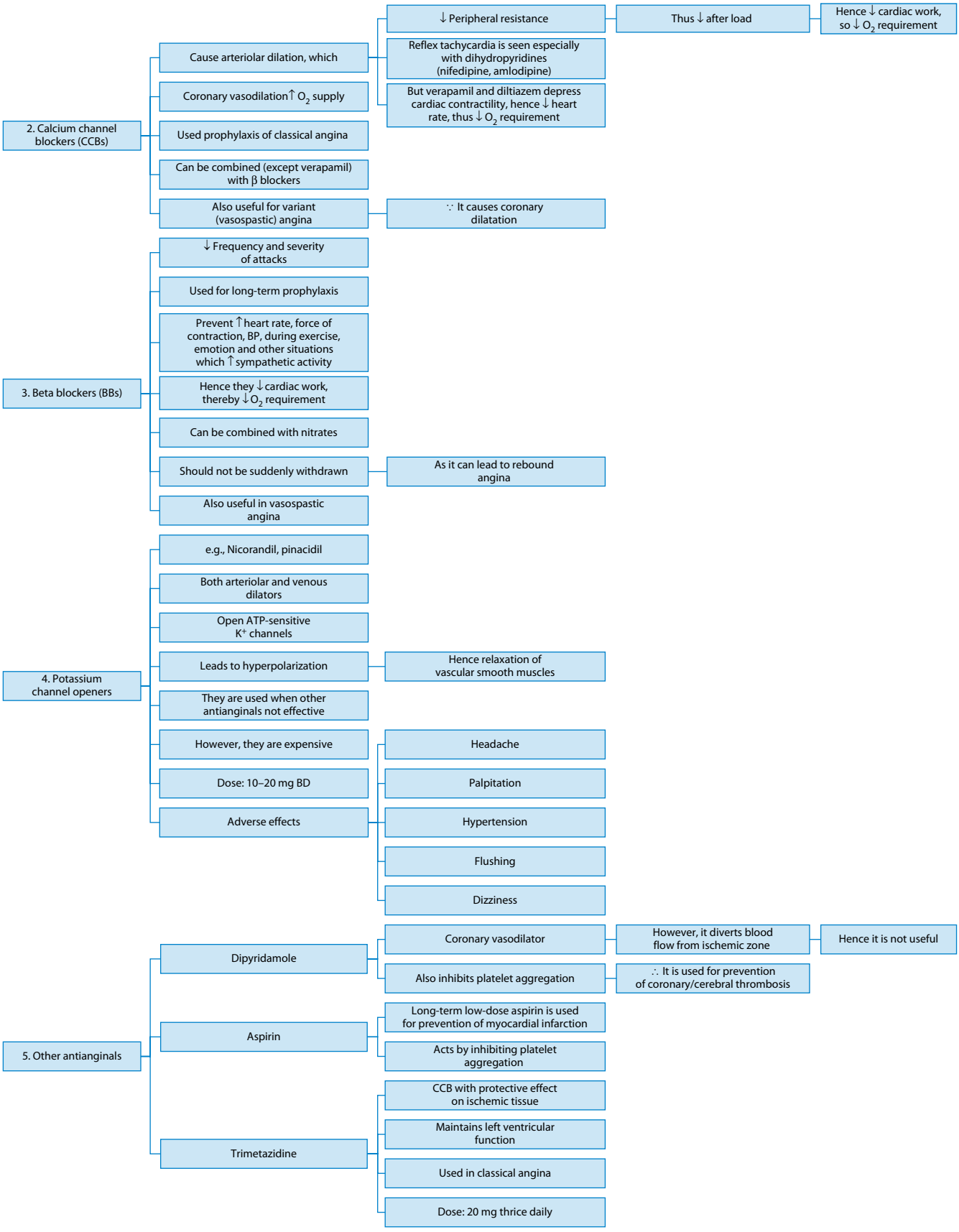
15.9 PHARMACOKINETICS, ADRs, AND DRUG INTERACTIONS OF NITRATES



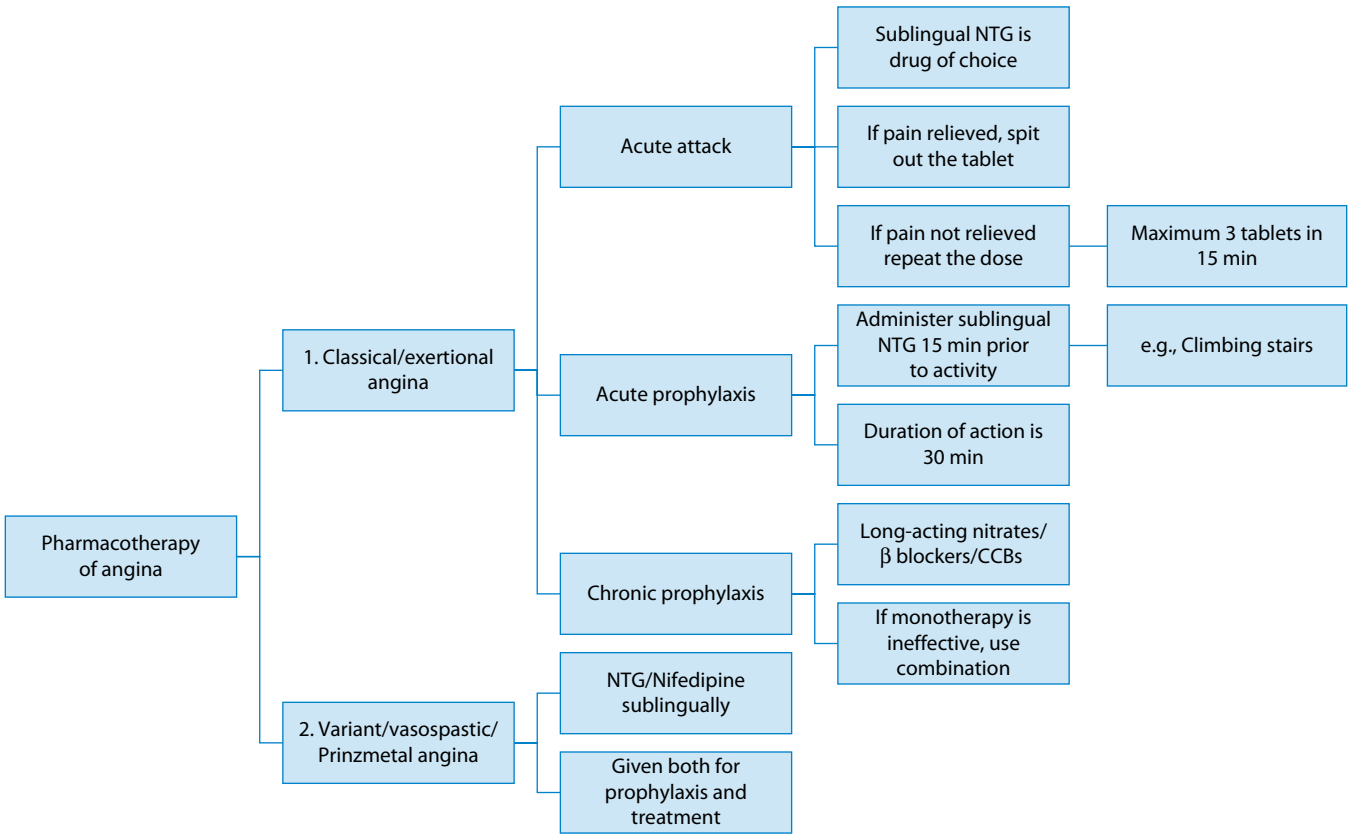
15.10 USES OF NITRATES



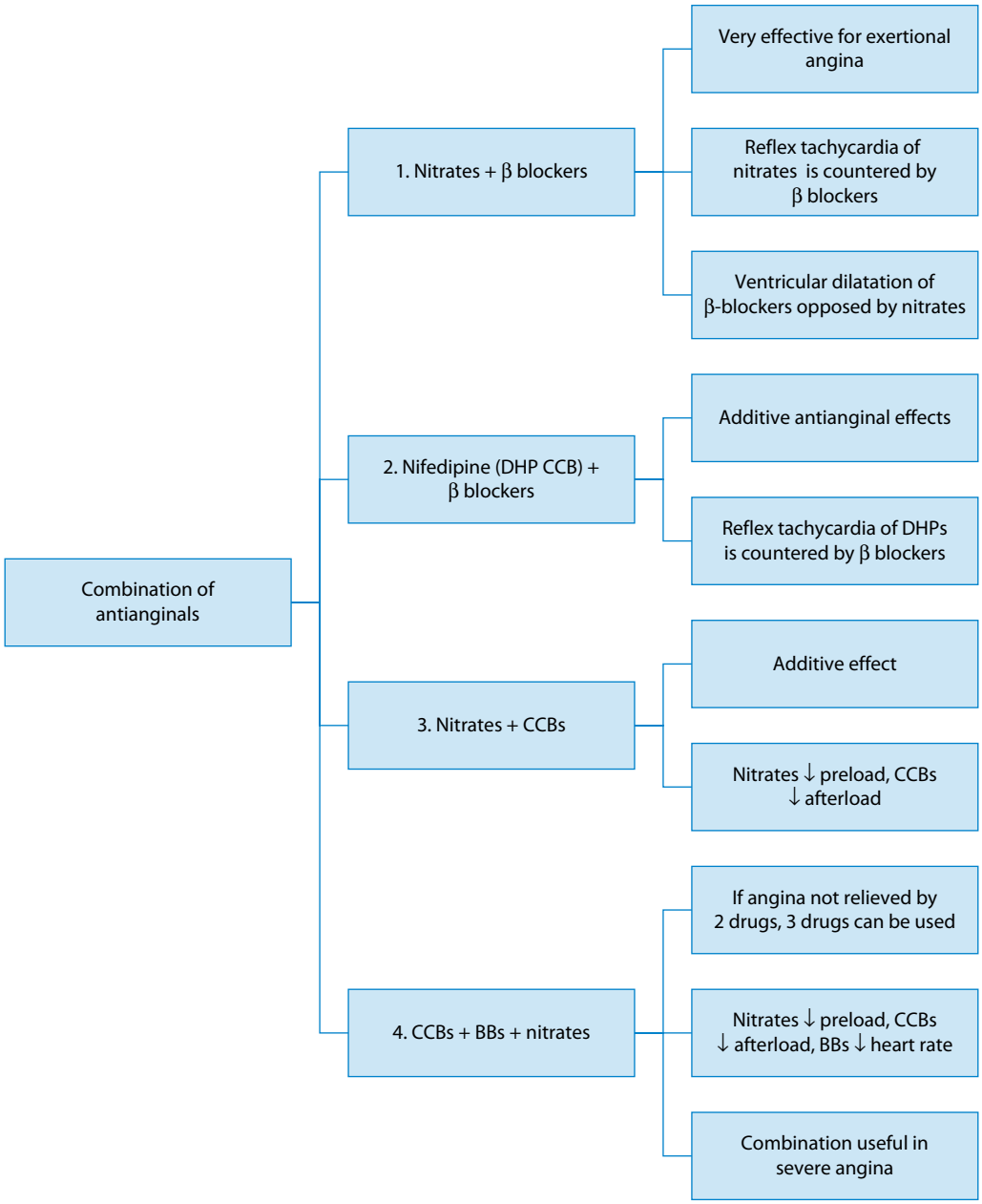
15.11 CALCIUM CHANNEL BLOCKERS (CCBs), BETA BLOCKERS (BBs), POTASSIUM CHANNEL OPENERS, AND OTHERS AS ANTIANGINALS



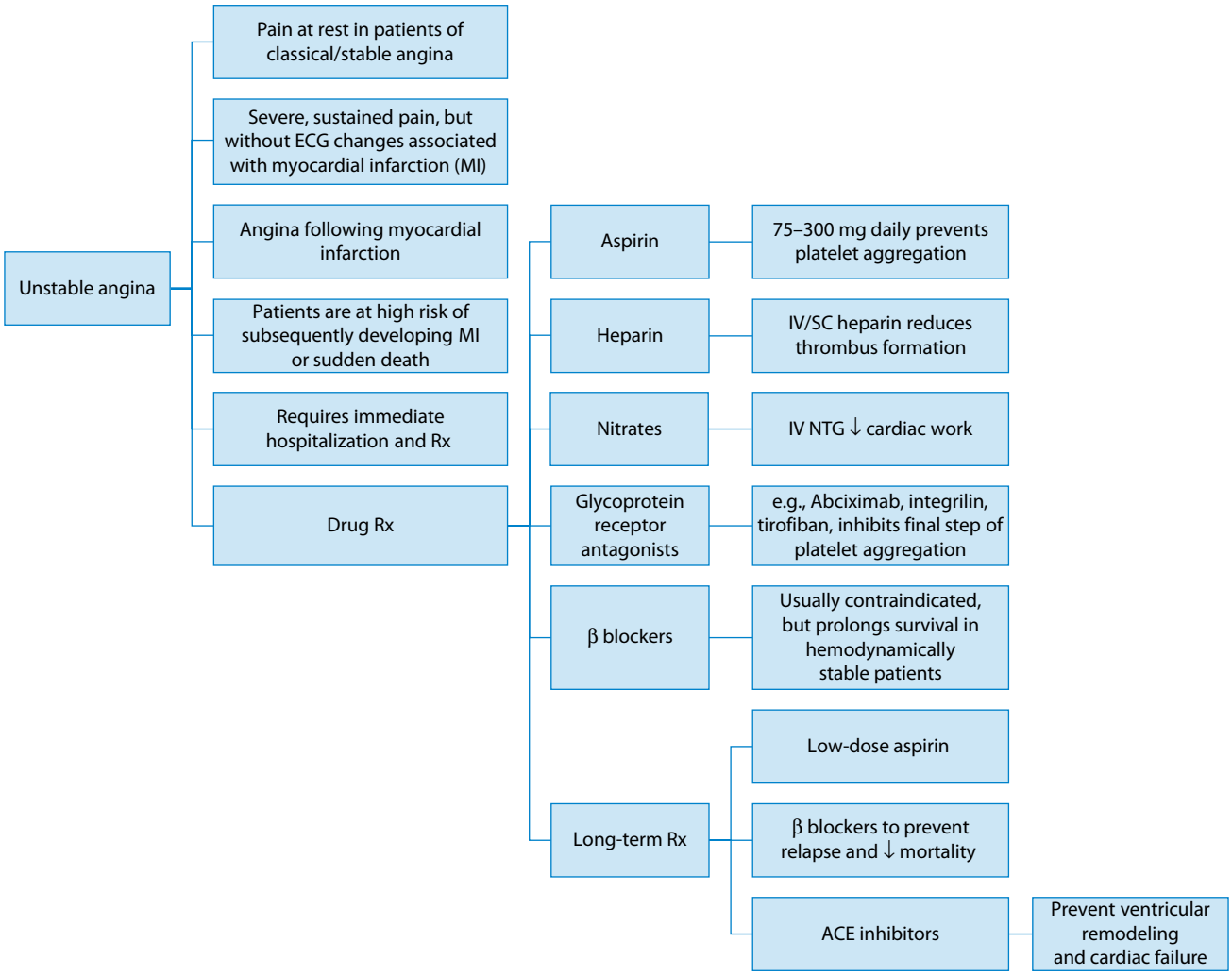
15.12 PHARMACOTHERAPY OF ANGINA



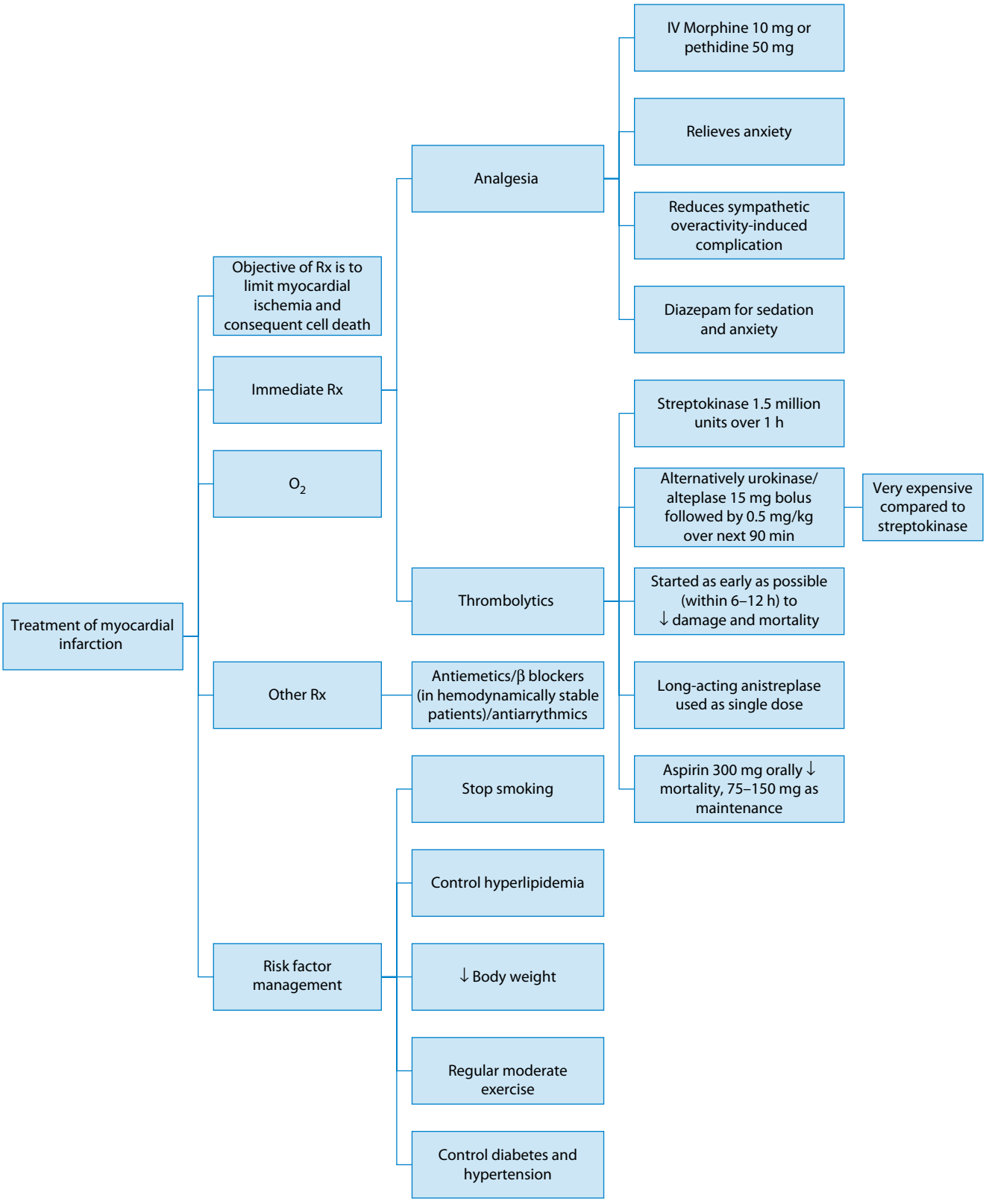
15.13 COMBINATION OF ANTIANGINALS



15.14 UNSTABLE ANGINA AND TREATMENT



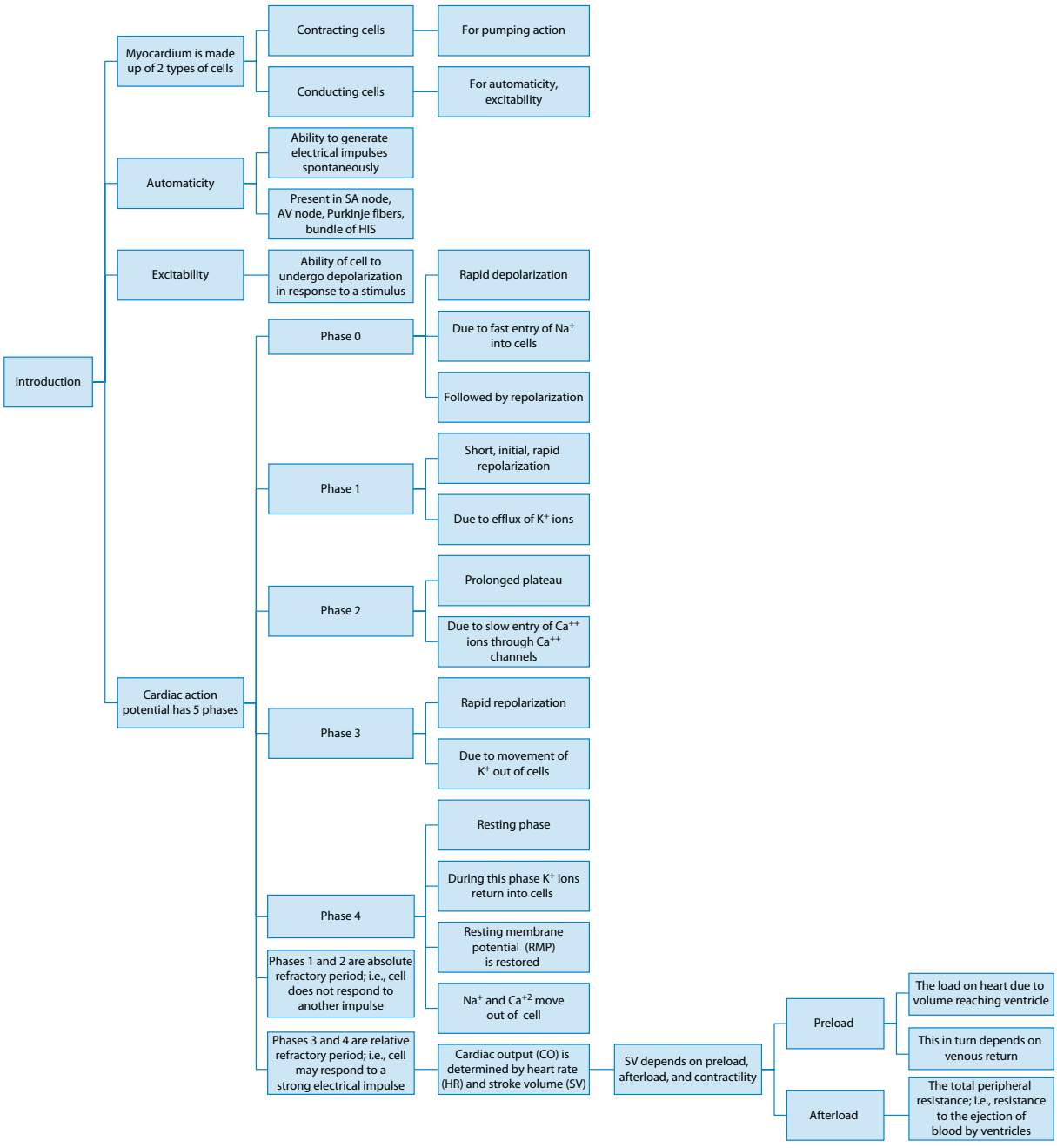
15.15 TREATMENT OF MYOCARDIAL INFARCTION



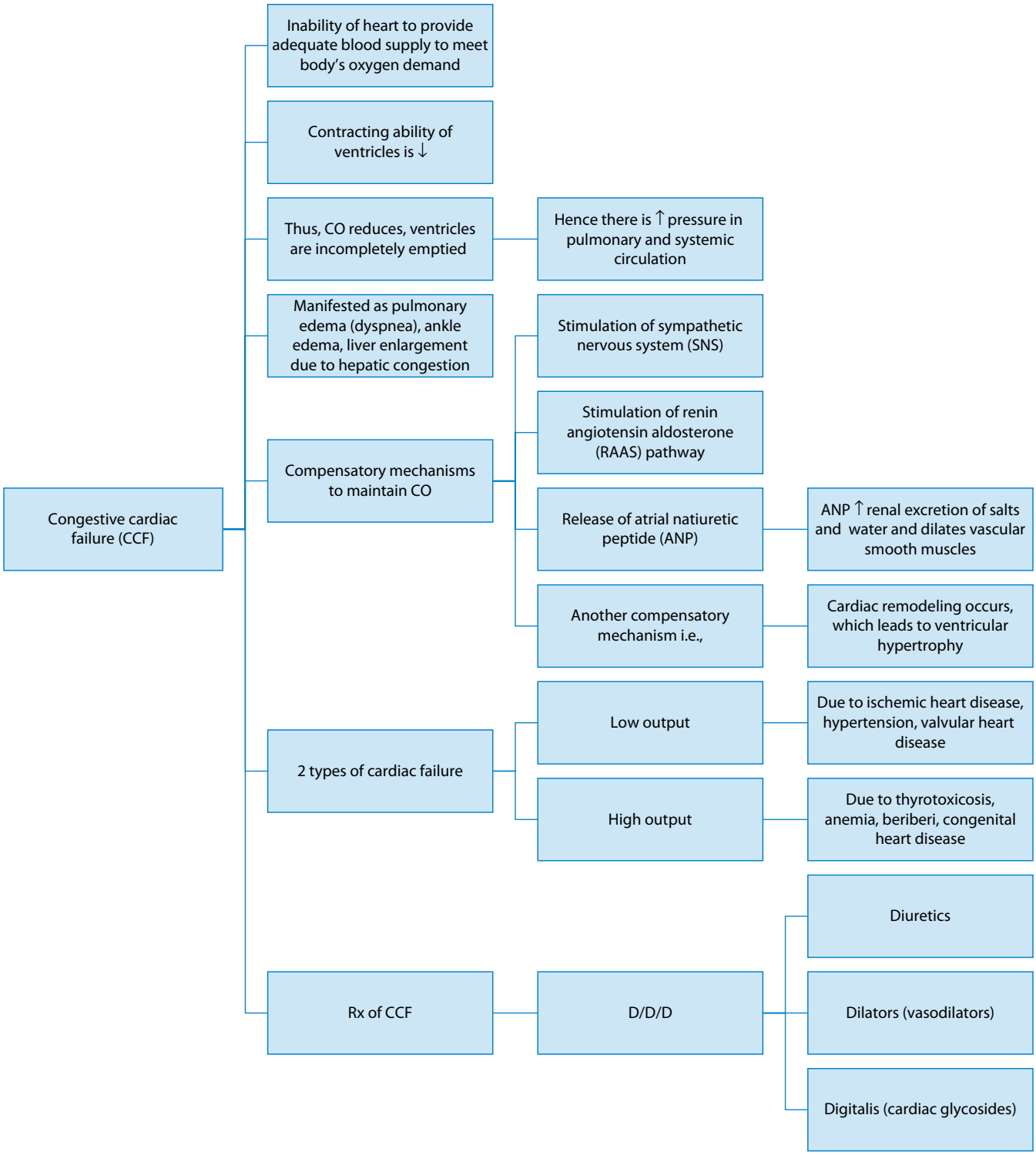
16

Cardiac glycosides and treatment of cardiac failure

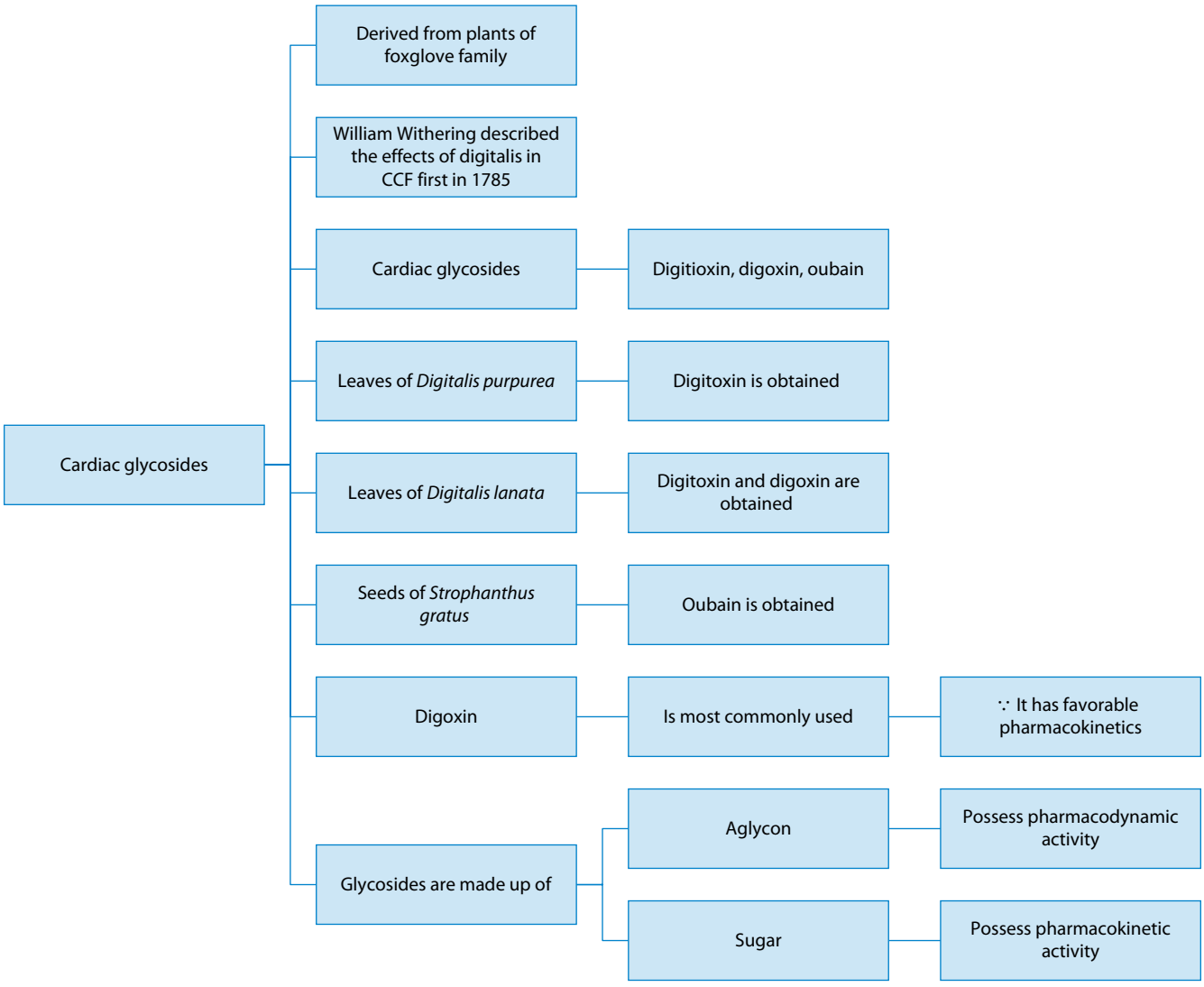
16.1 INTRODUCTION



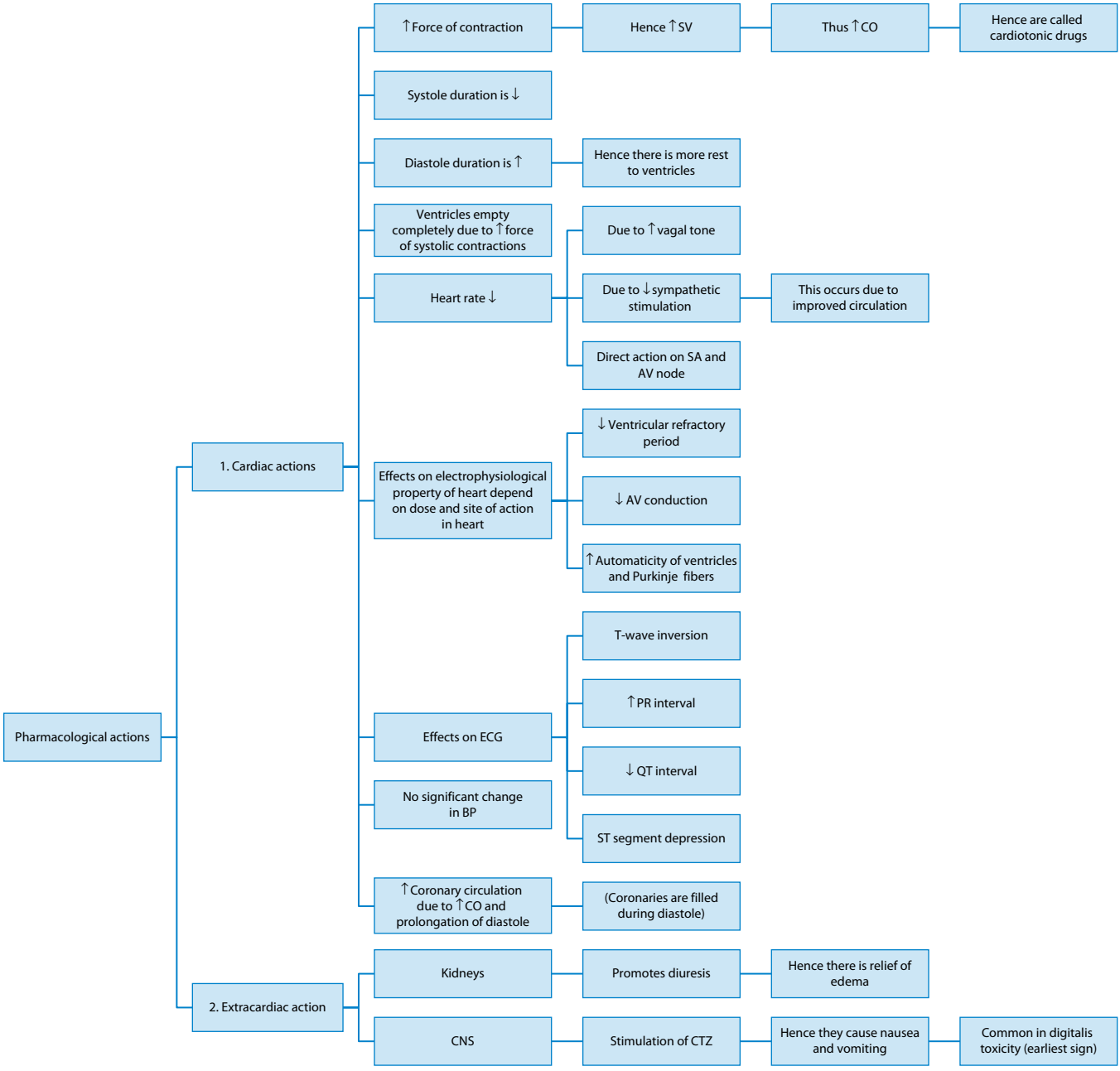
16.2 CONGESTIVE CARDIAC FAILURE (CCF)



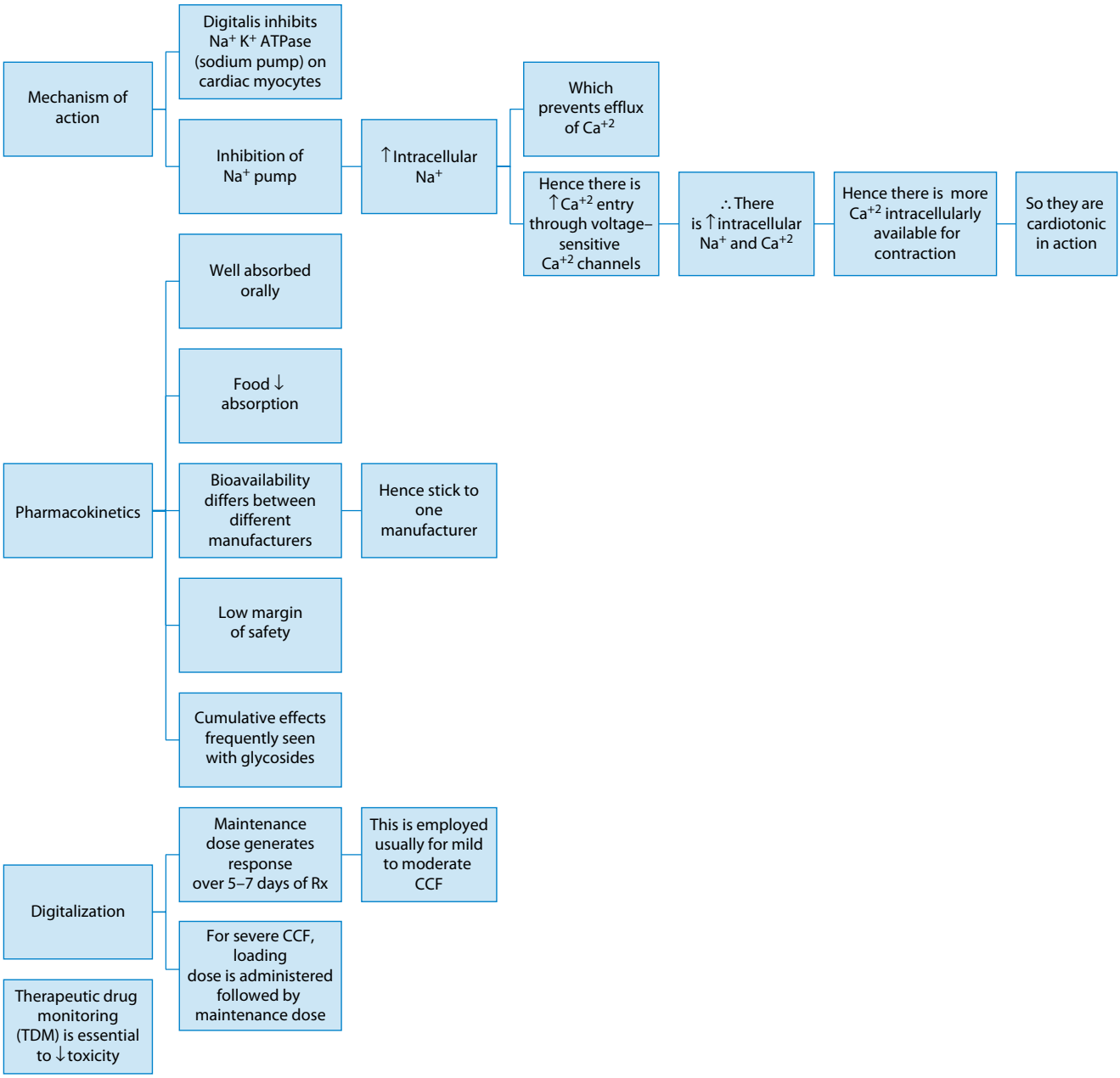
16.3 CARDIAC GLYCOSIDES



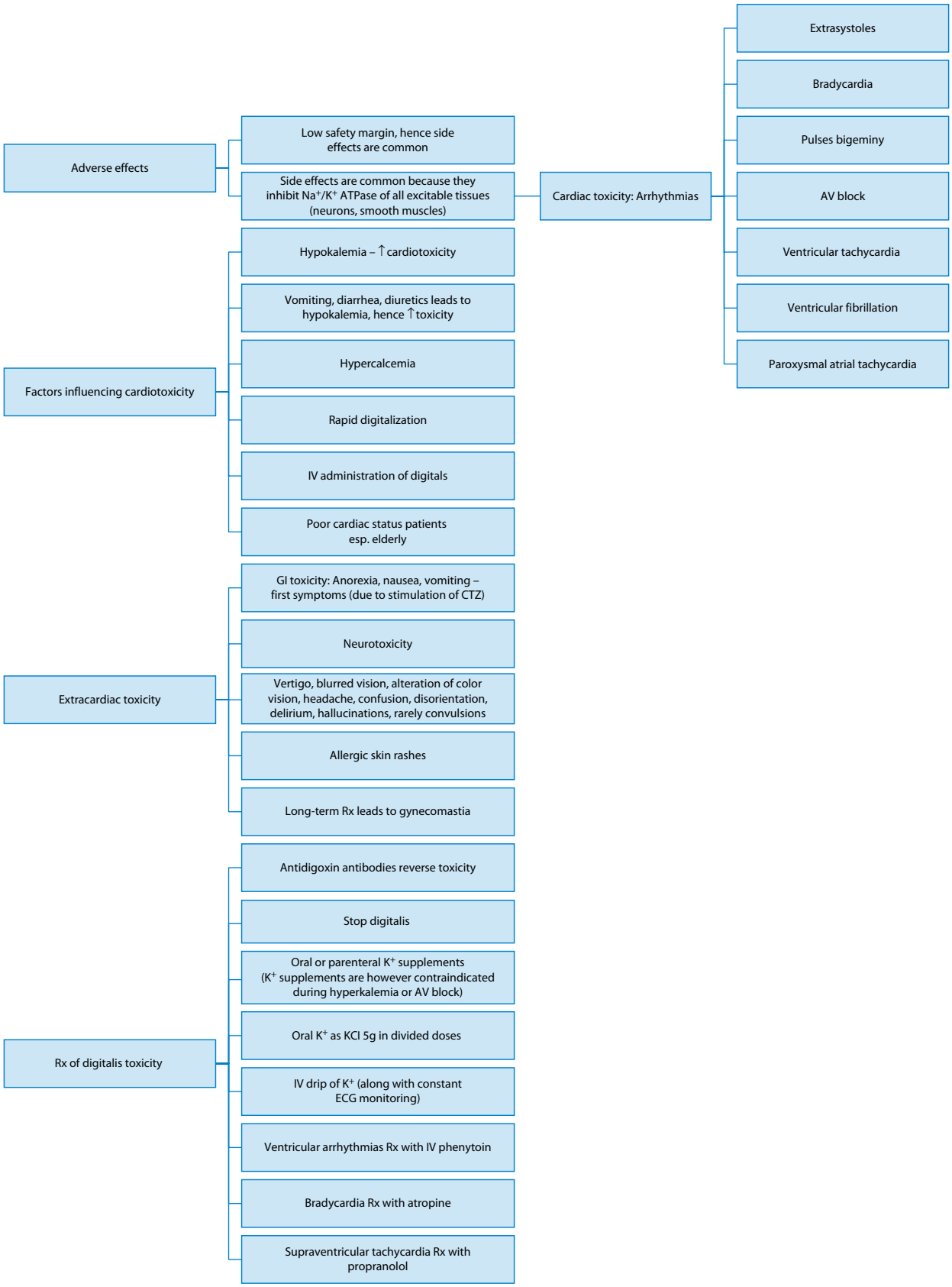
16.4 PHARMACOLOGICAL ACTIONS



16.5 MECHANISM OF ACTION, PHARMACOKINETICS, DIGITALIZATION



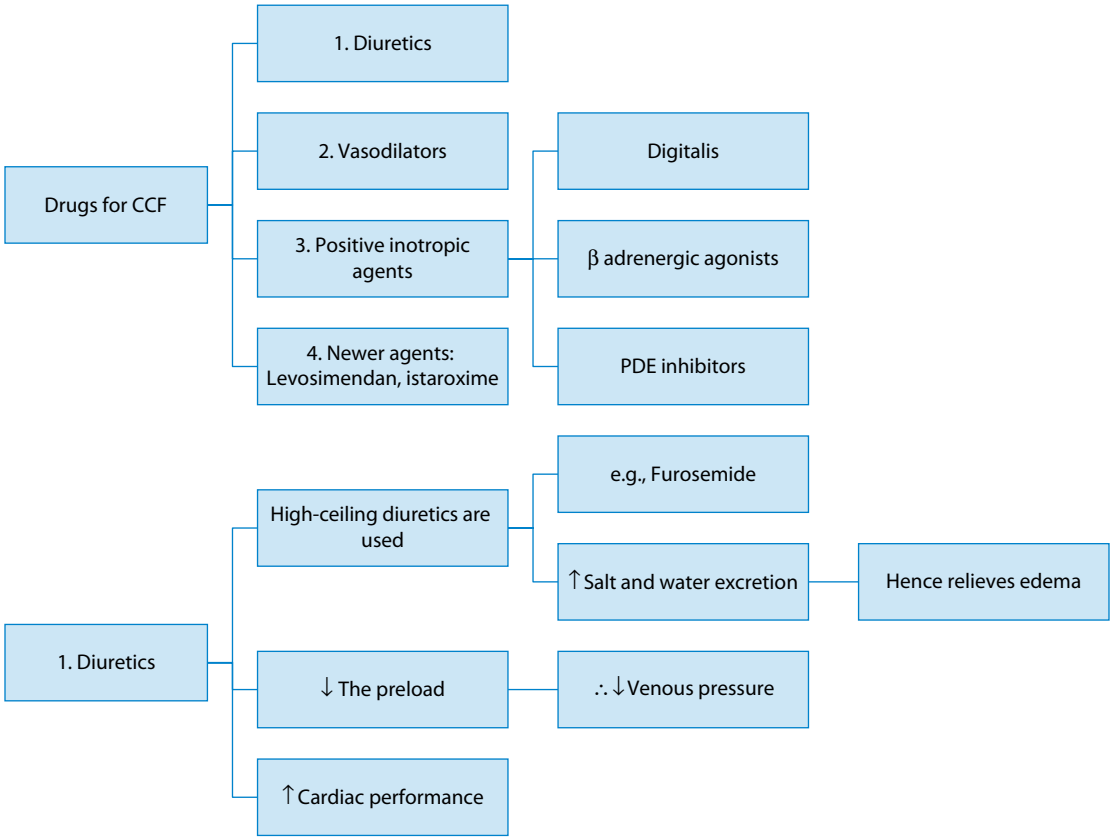
16.6 ADVERSE EFFECTS



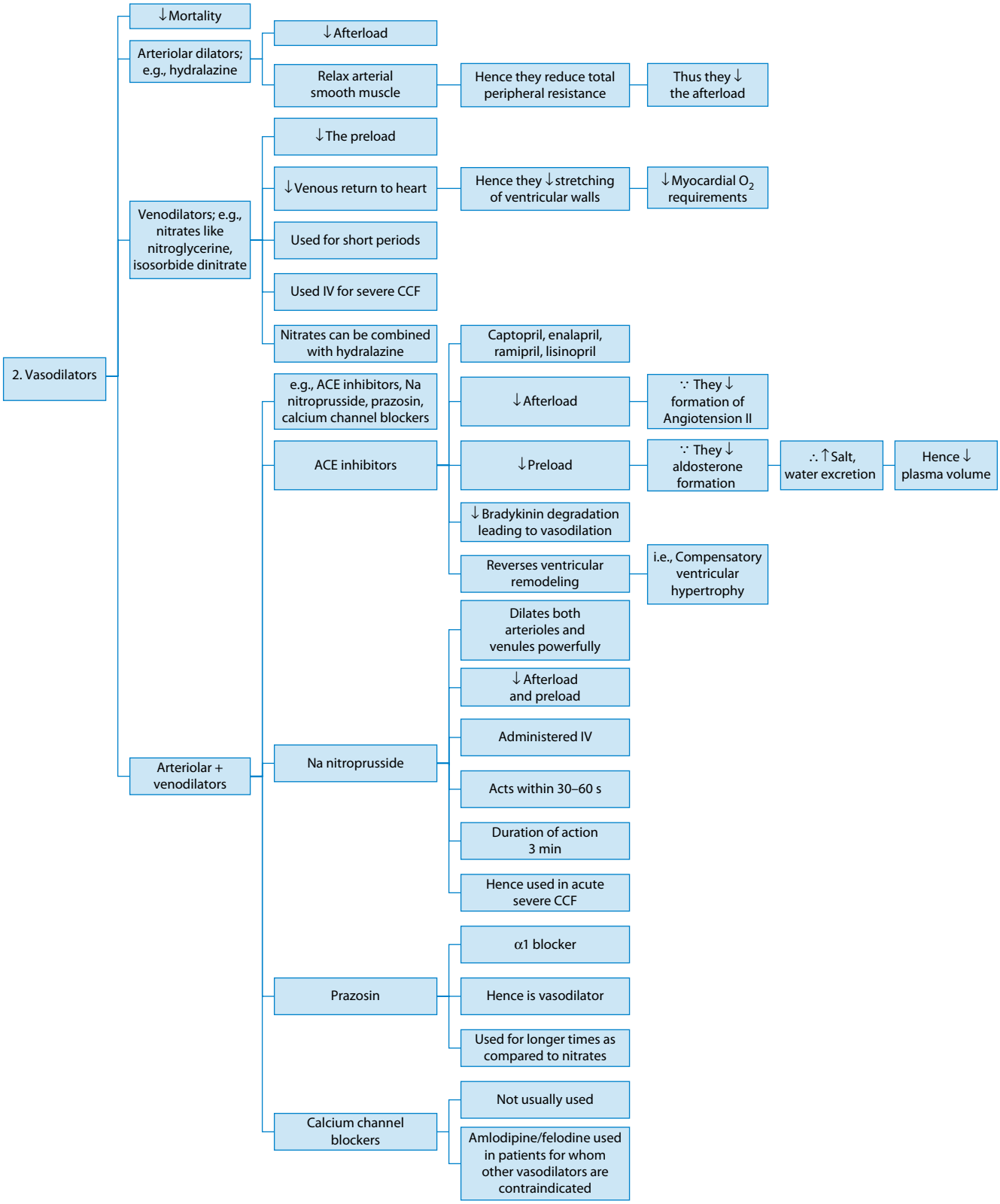
16.7 DRUG INTERACTIONS, USES, PRECAUTIONS, AND CONTRAINDICATIONS



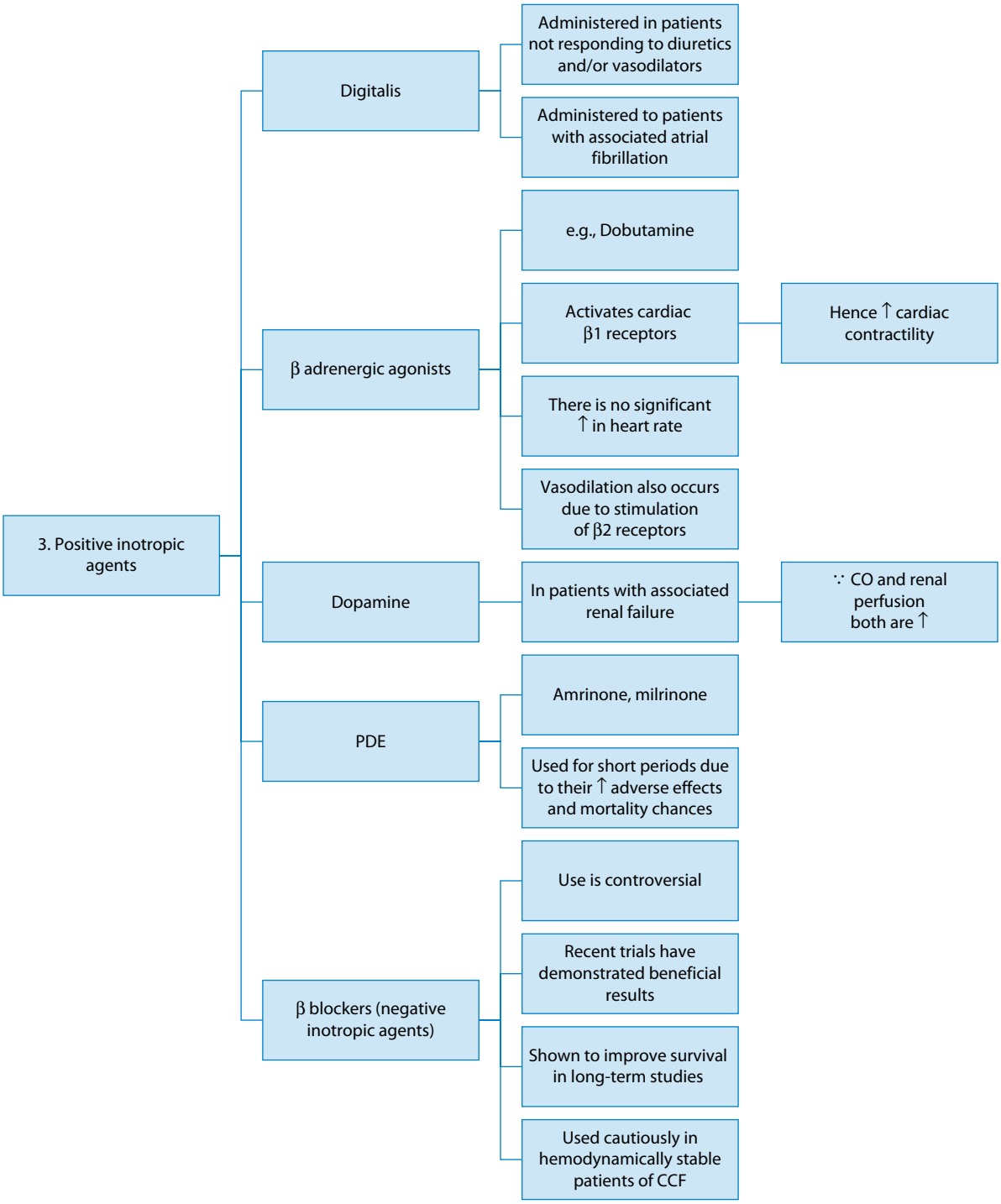
16.8 DRUGS FOR CCF, DIURETICS



16.9 VASODILATORS



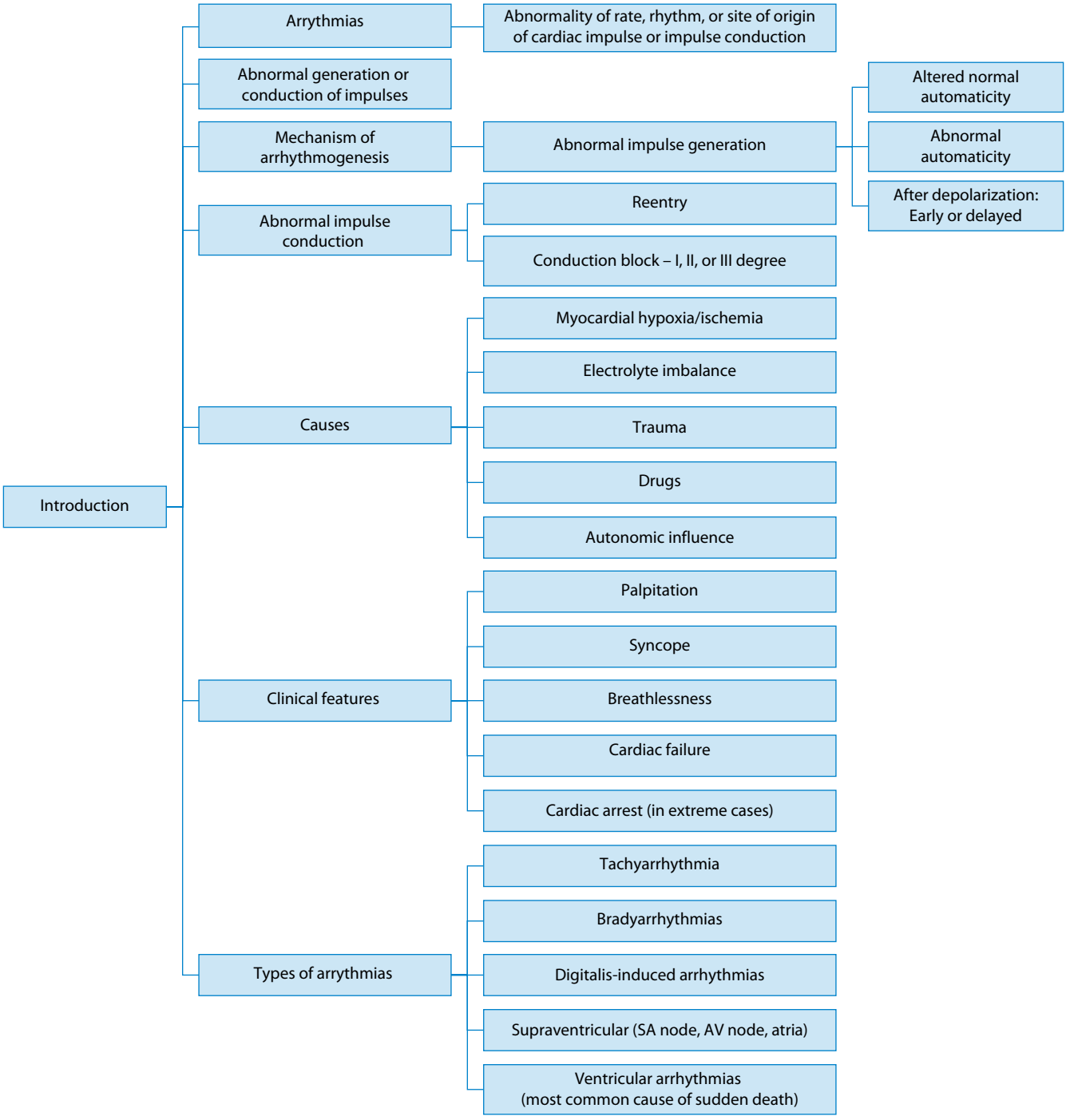
16.10 POSITIVE INOTROPIC AGENTS



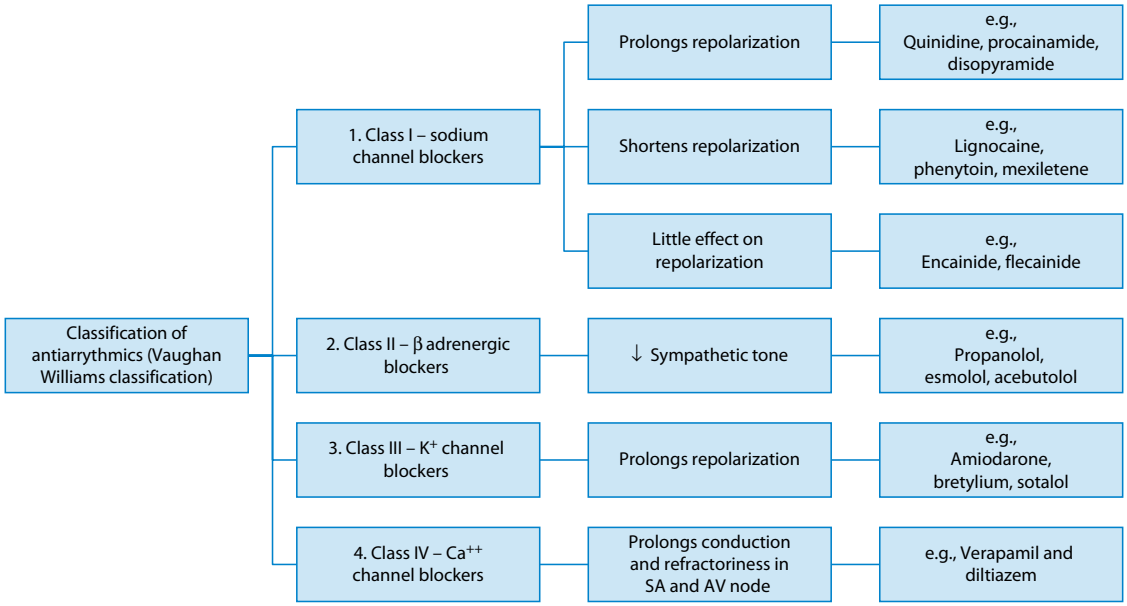
17

Antiarrhythmics

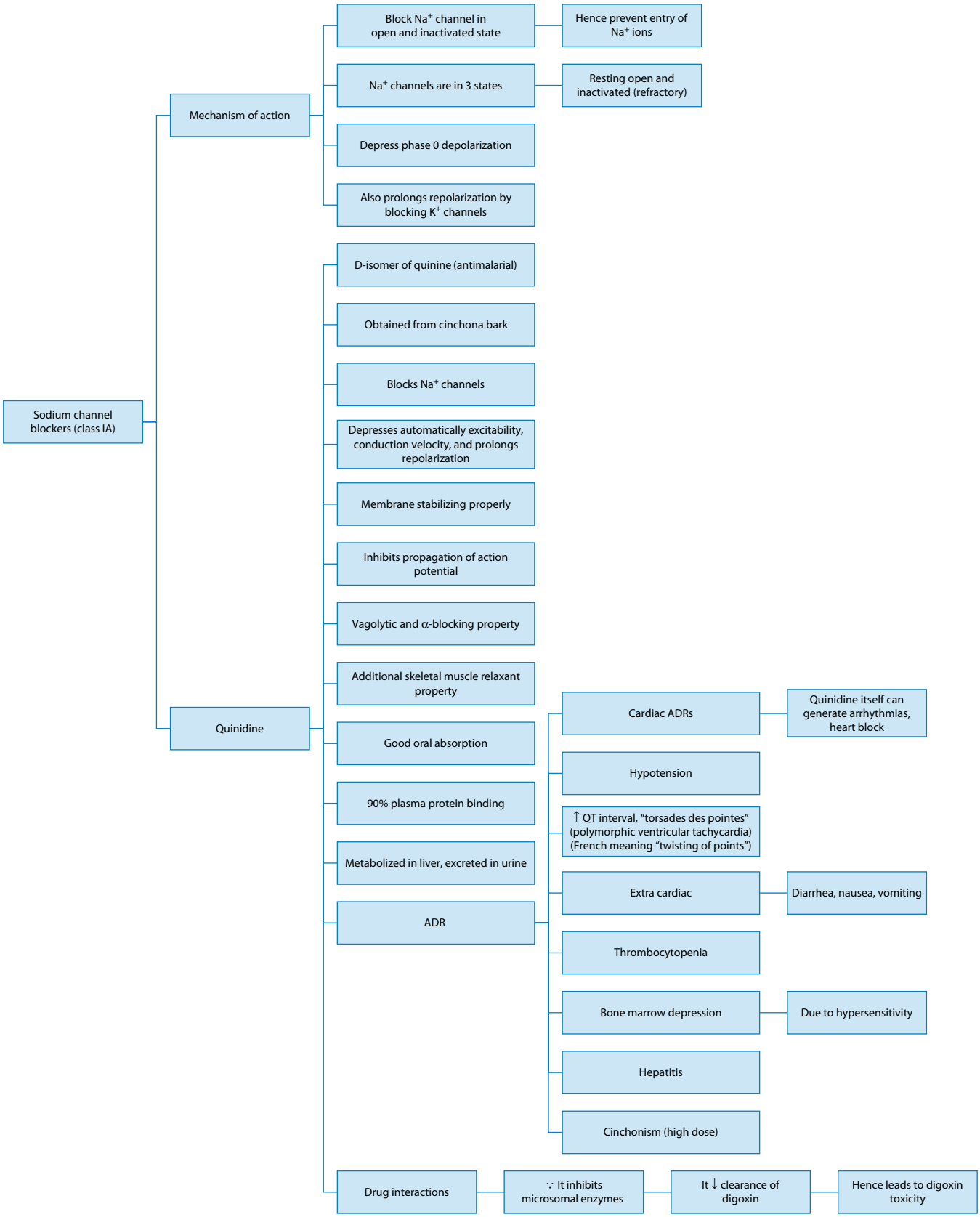
17.1 ARRYTHMIAS



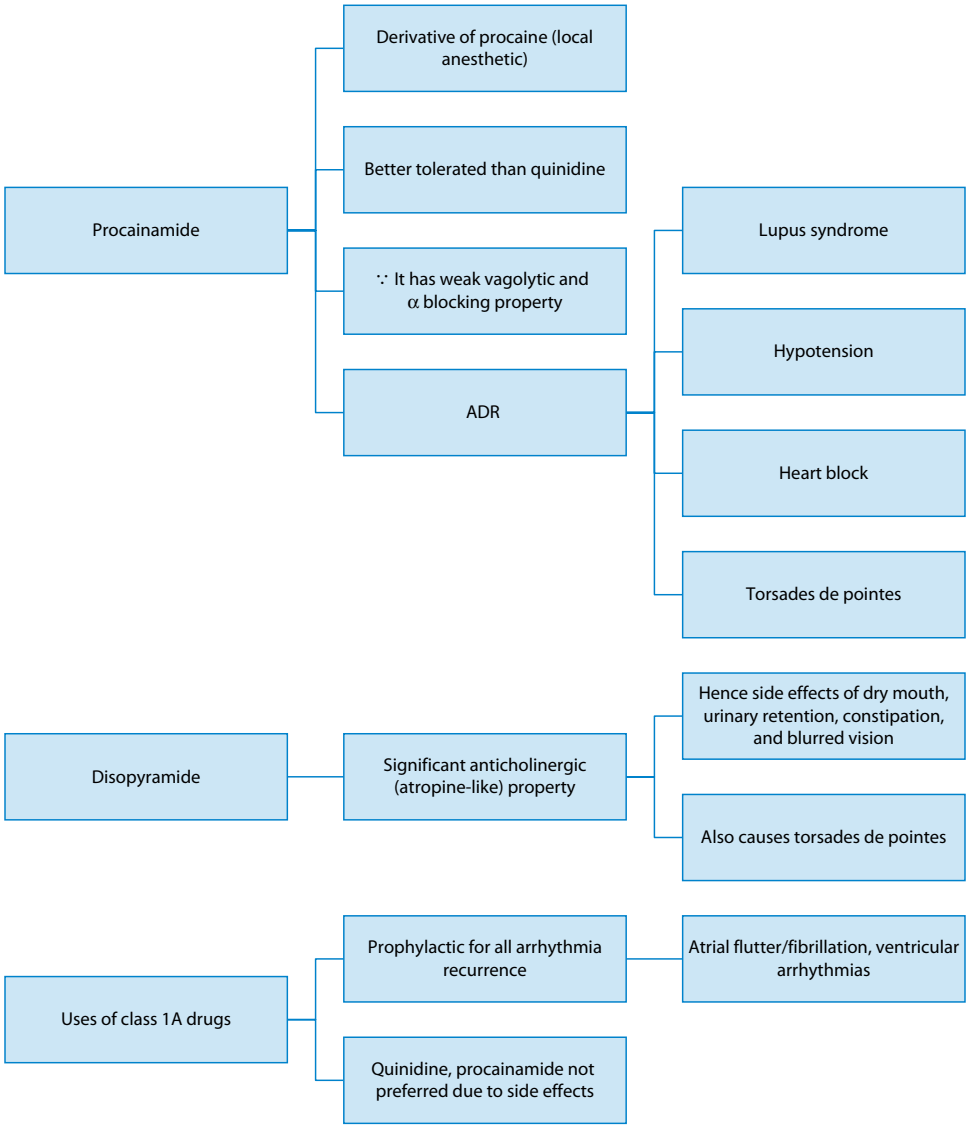
17.2 CLASSIFICATION OF ANTIARRYTHMICS



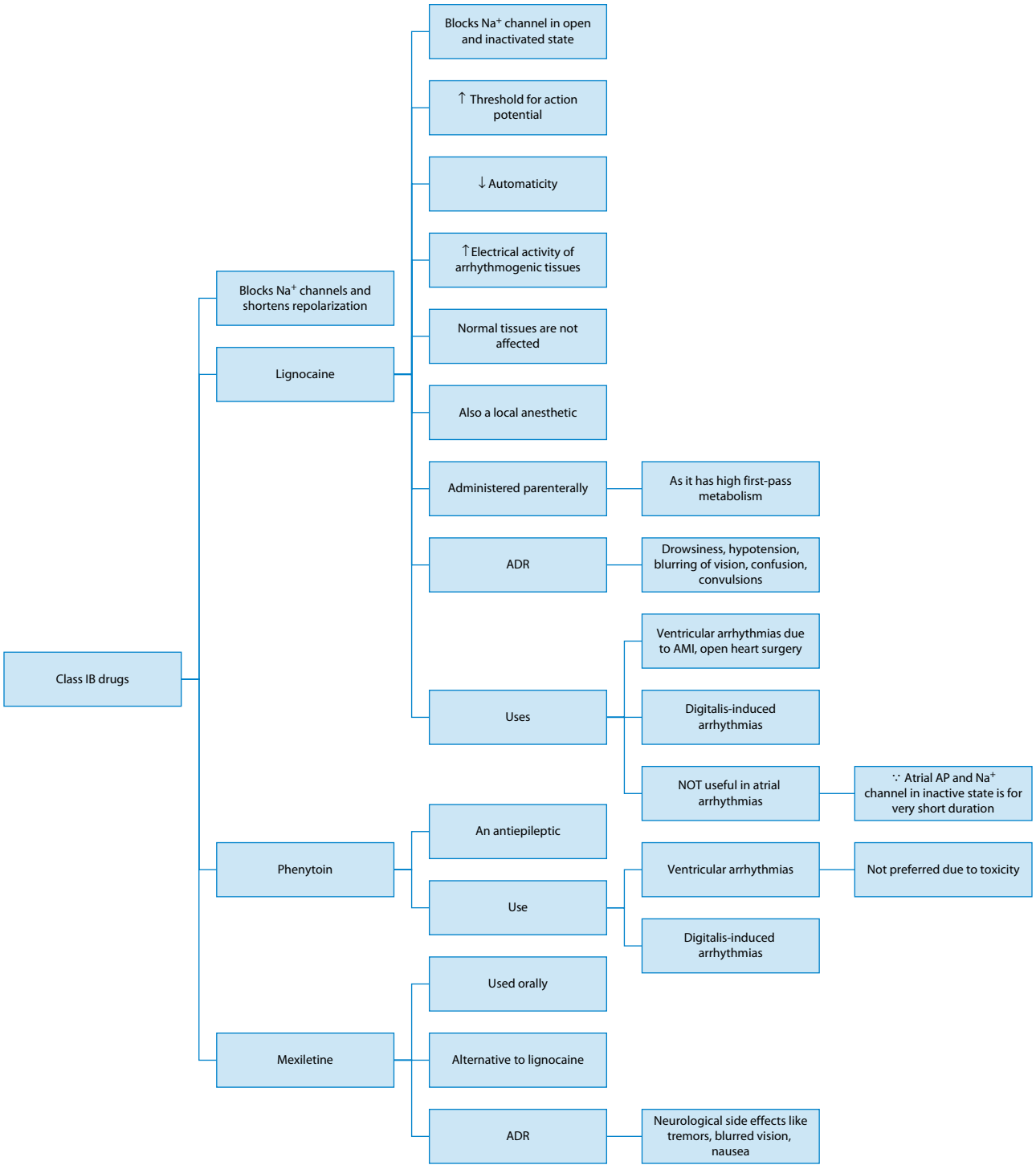
17.3 SODIUM CHANNEL BLOCKERS (CLASS IA) AND QUINIDINE



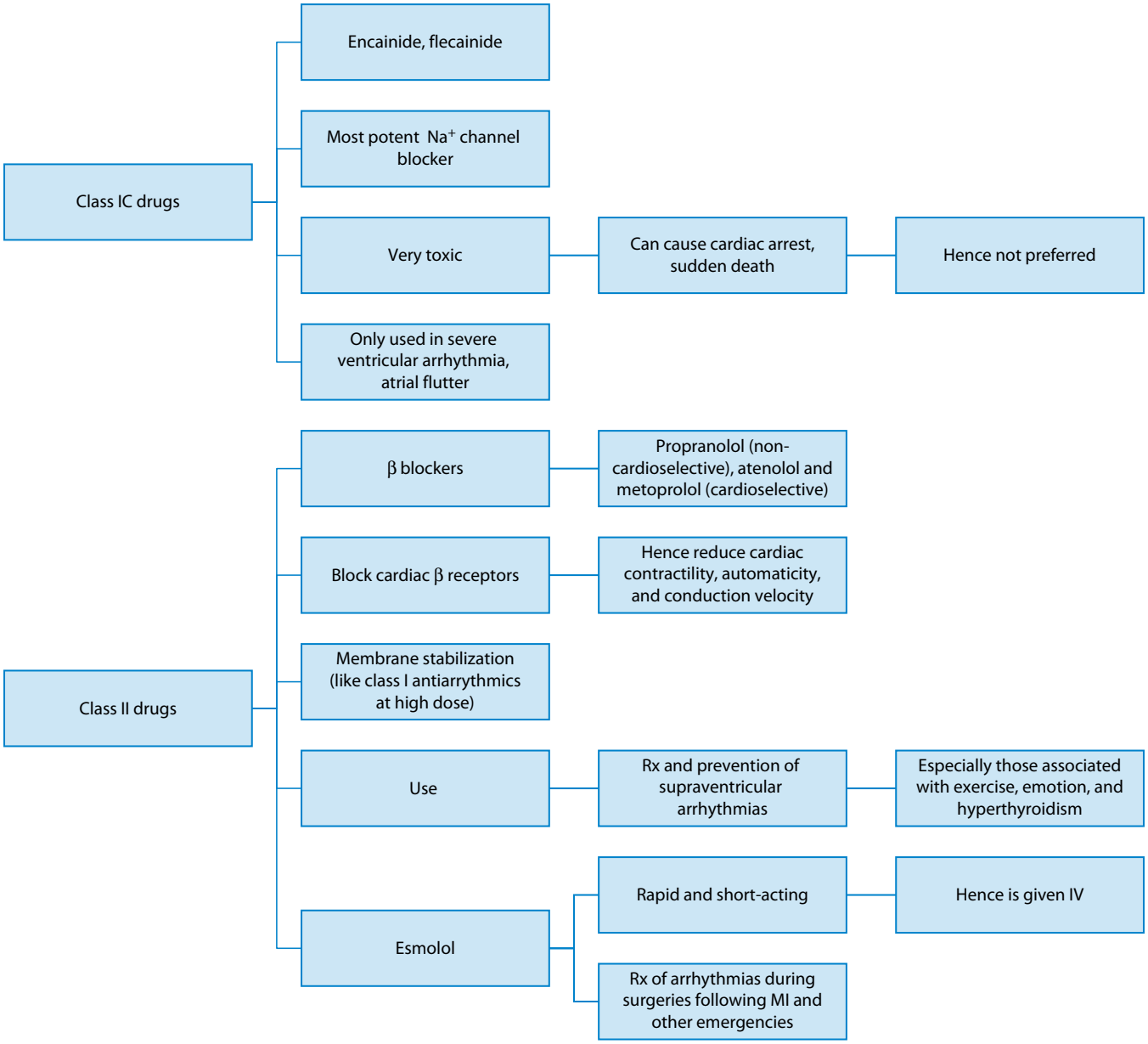
17.4 SODIUM CHANNEL BLOCKERS (CLASS IA) AND PROCAINAMIDE, DISOPYRAMIDE, AND USES OF CLASS 1A DRUGS



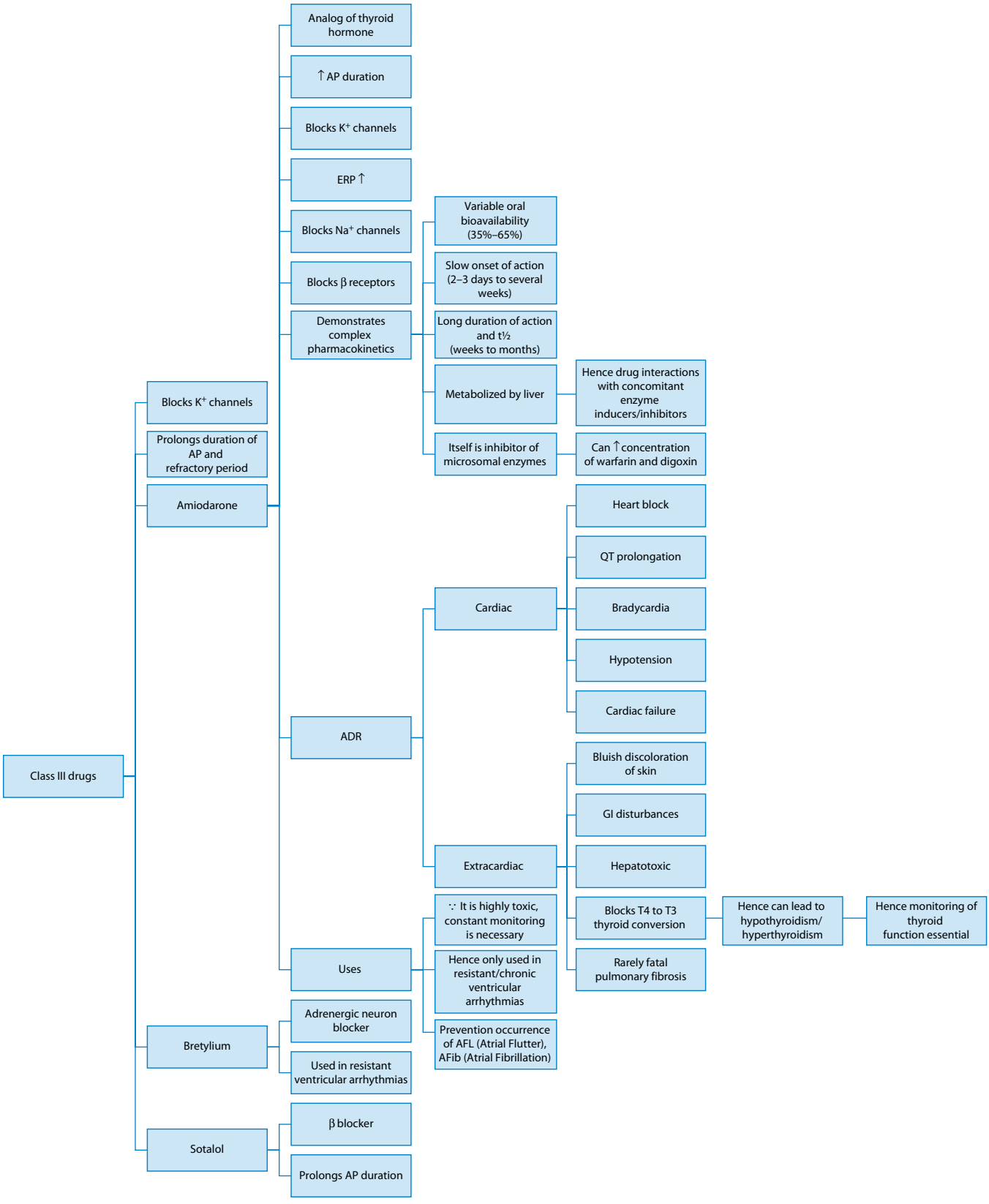
17.5 CLASS IB DRUGS – LIGNOCAINE, PHENYTOIN, AND MEXILETINE



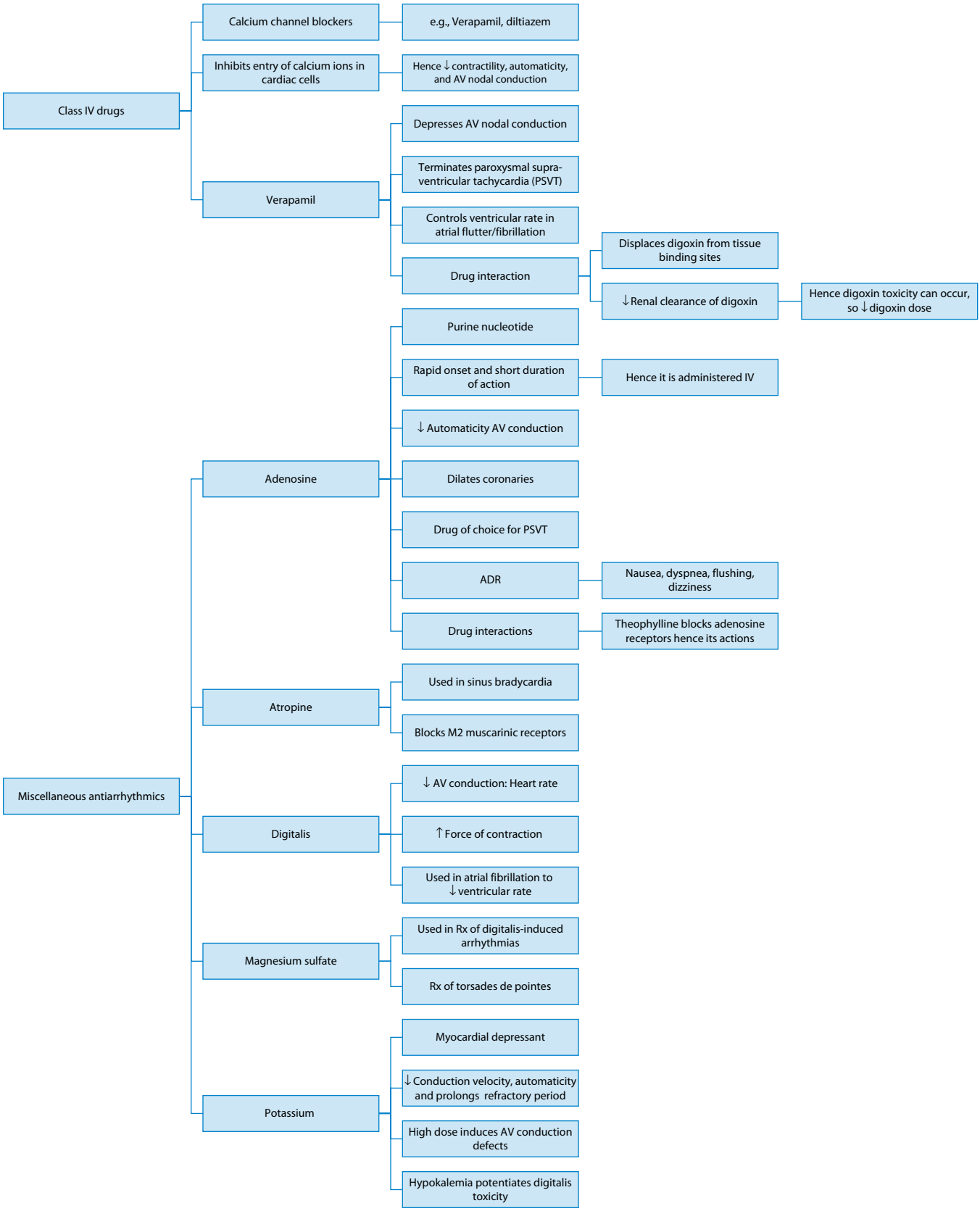
17.6 CLASS IC DRUGS AND CLASS II DRUGS



17.7 CLASS III DRUGS AND AMIODARONE



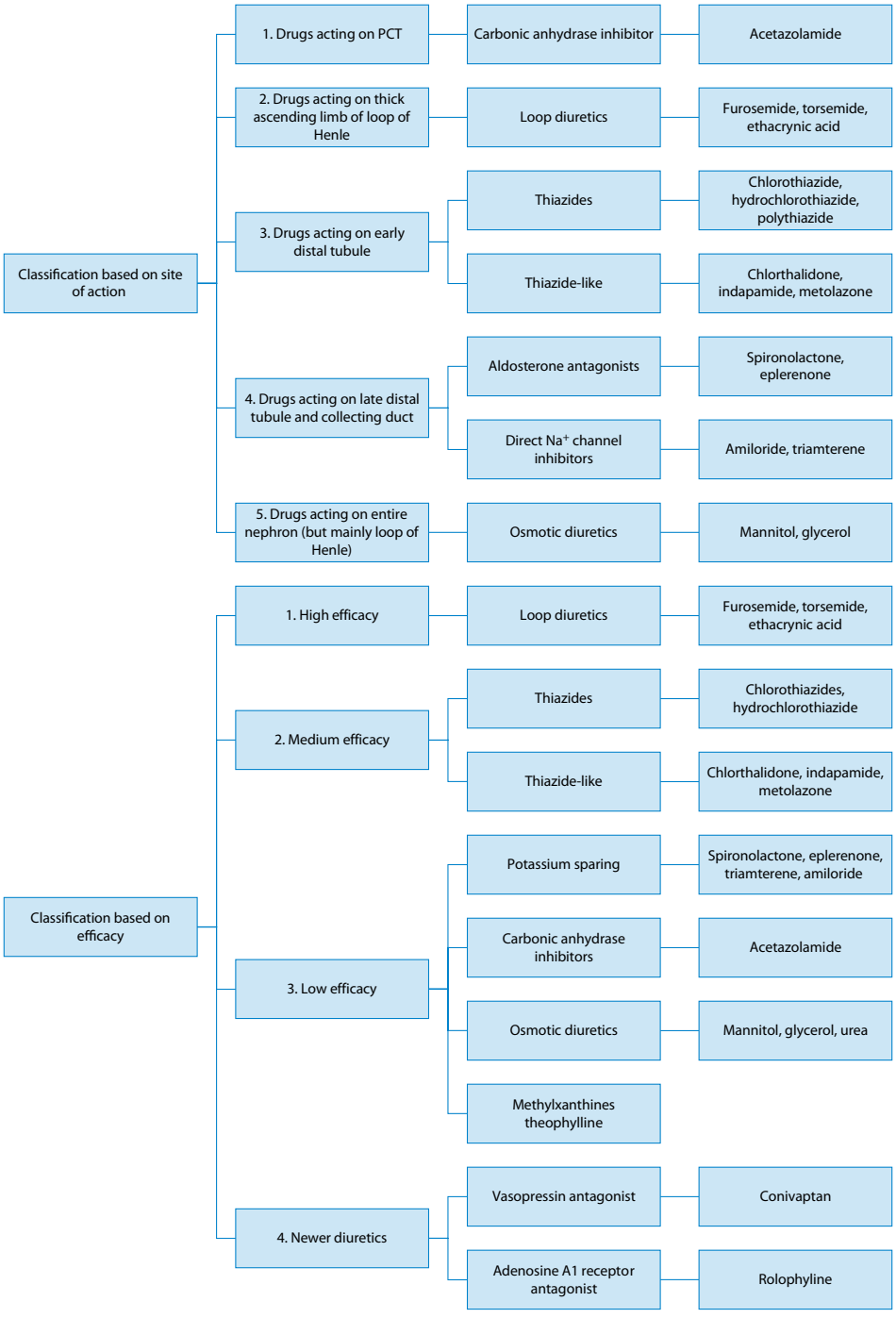
17.8 CLASS IV DRUGS AND MISCELLANEOUS AGENTS



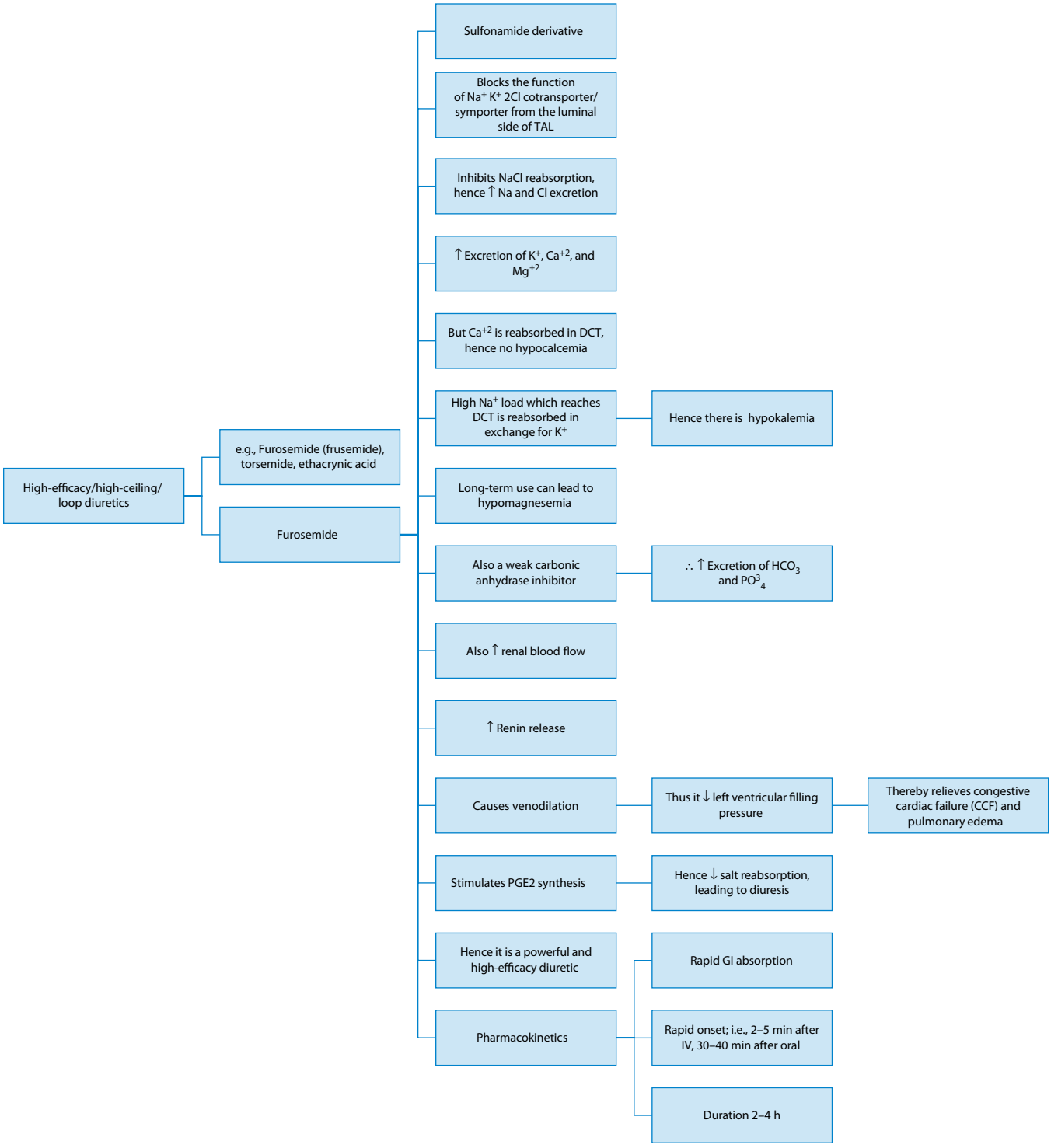
18

Diuretics and antidiuretics

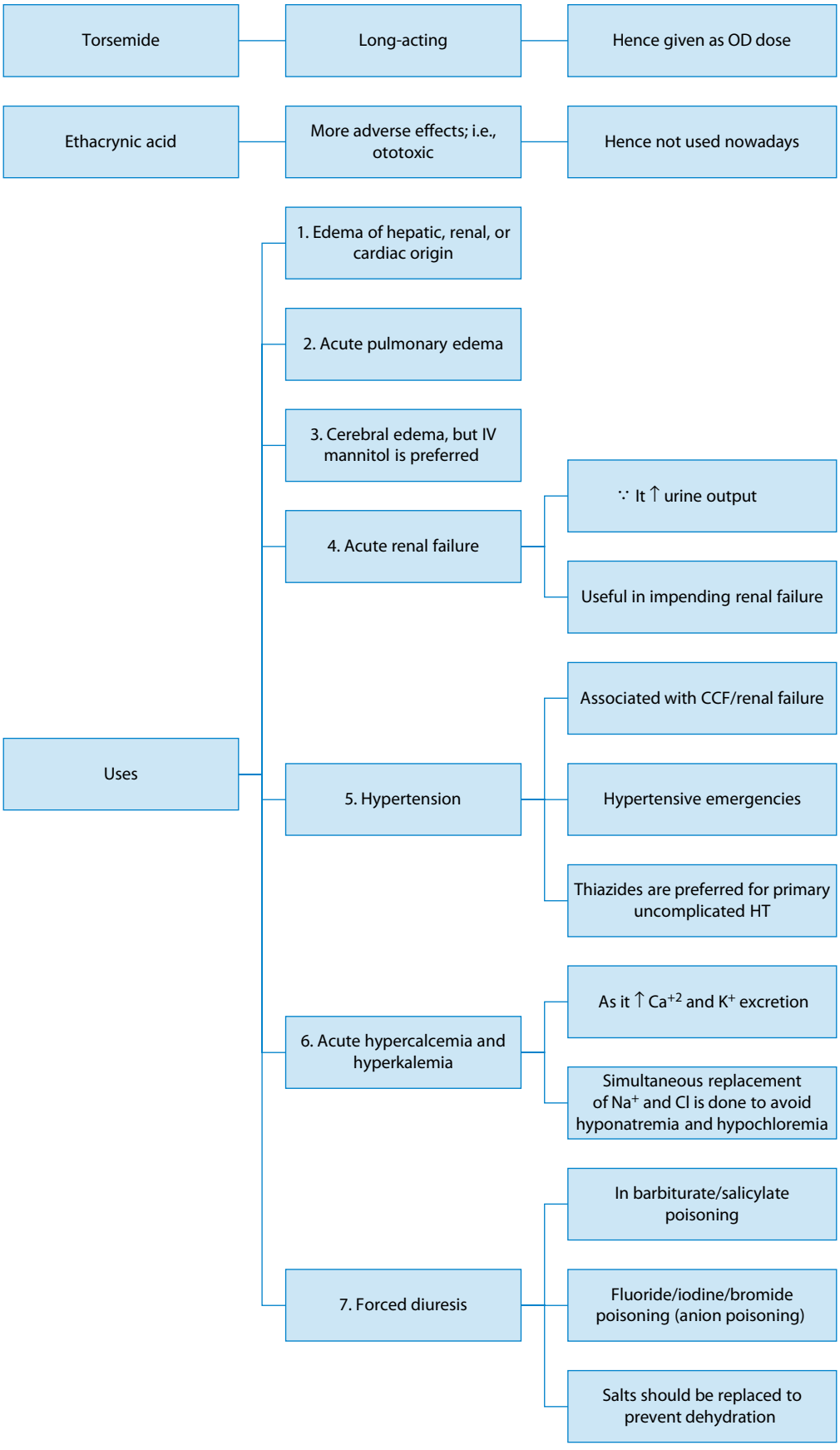
18.1 CLASSIFICATION



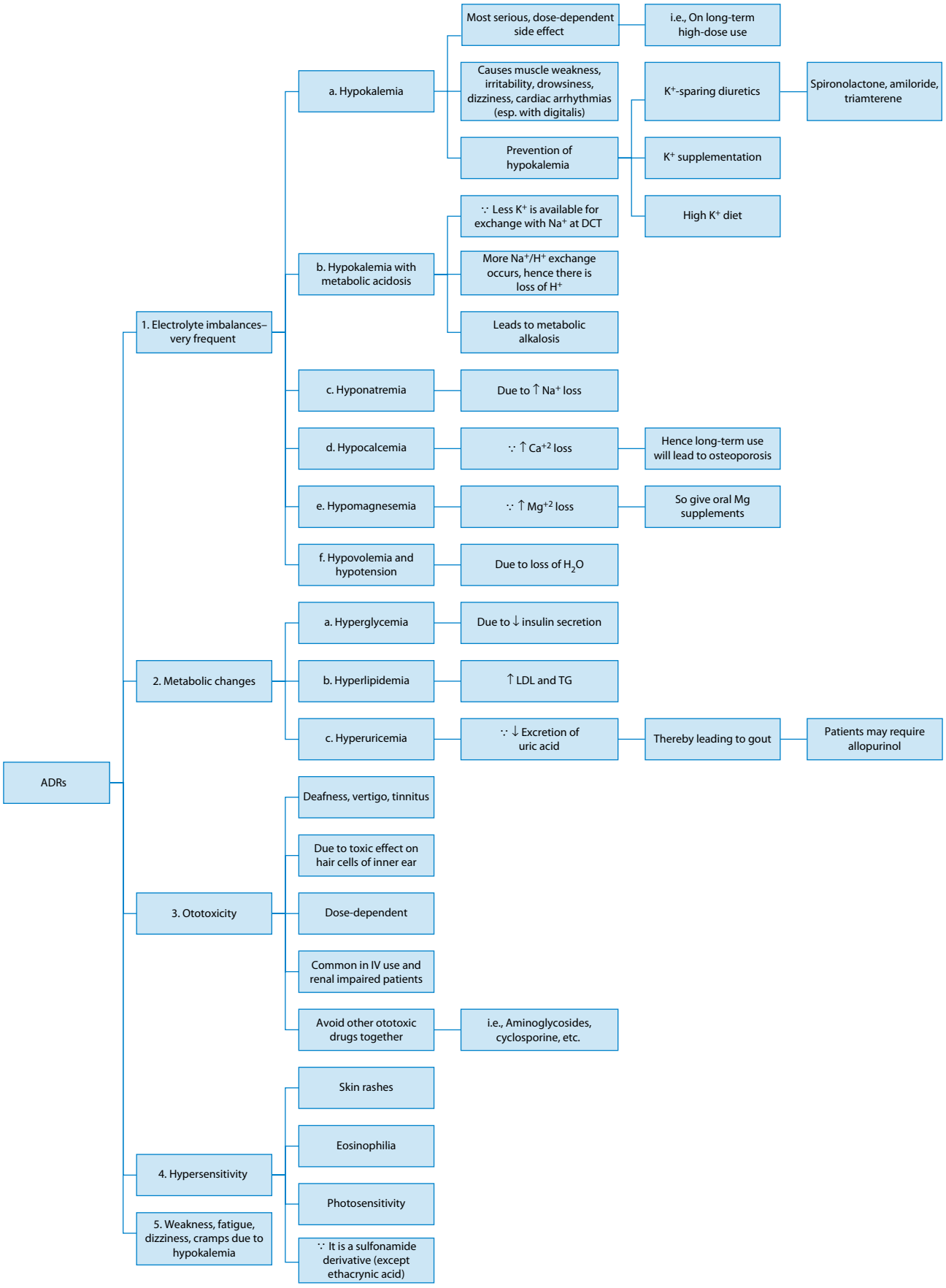
18.2 HIGH-EFFICACY/HIGH-CEILING/LOOP DIURETICS



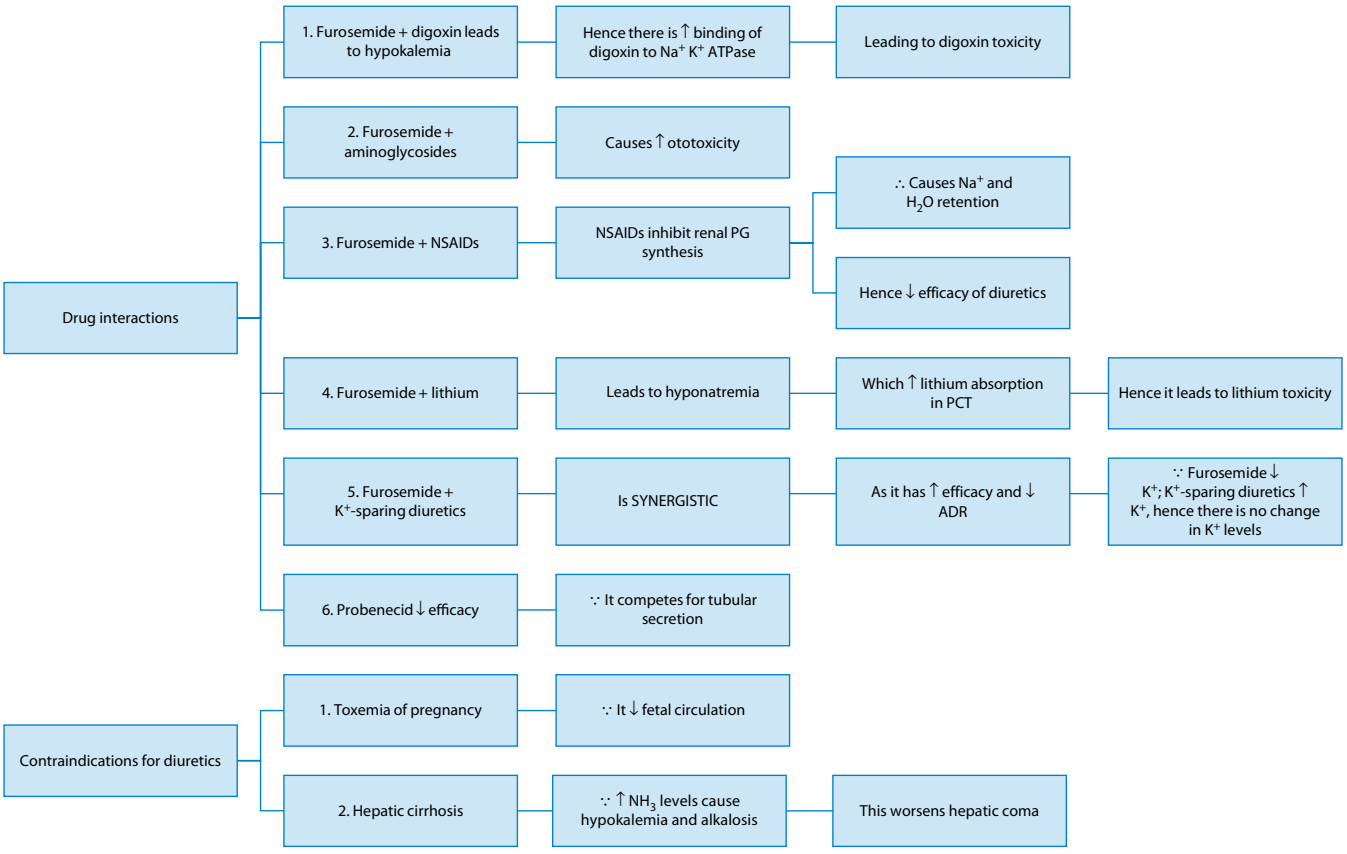
18.3 OTHER LOOP DIURETICS AND USES



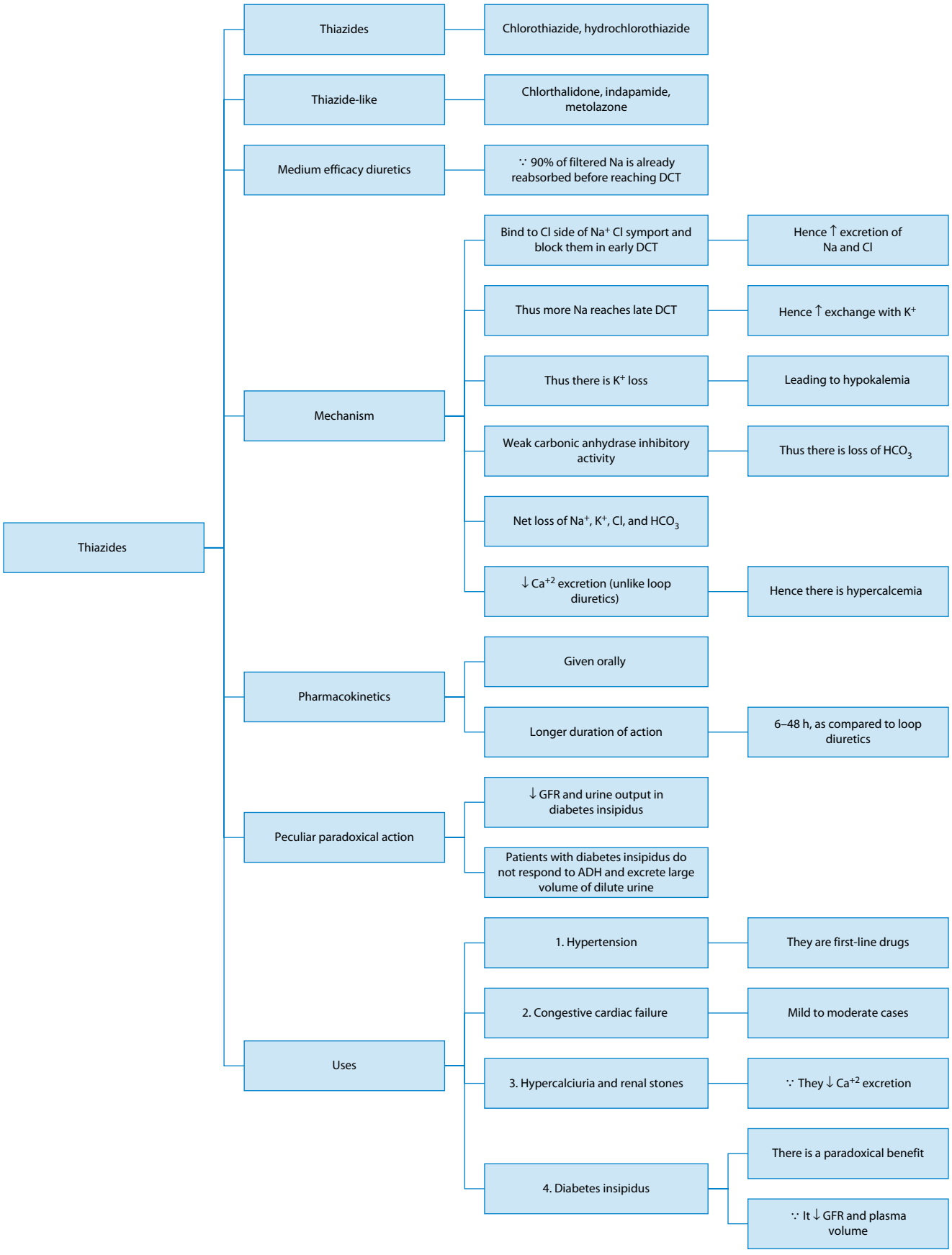
18.4 ADRs


ADRs
1. Electrolyte imbalances–very frequent
a. Hypokalemia
Most serious, dose-dependent side effect
i.e., On long-term high-dose use
Causes muscle weakness, irritability, drowsiness, dizziness, cardiac arrhythmias (esp. with digitalis)
Prevention of hypokalemia
K+-sparing diuretics
Spironolactone, amiloride, triamterene
K+ supplementation
High K+ diet
b. Hypokalemia with metabolic acidosis
∵ Less K+ is available for exchange with Na+ at DCT
More Na+/H+ exchange occurs, hence there is loss of H+
Leads to metabolic alkalosis
c. Hyponatremia
Due to ↑ Na+ loss
d. Hypocalcemia
∵ ↑ Ca+2 loss
Hence long-term use will lead to osteoporosis
e. Hypomagnesemia
∵ ↑ Mg+2 loss
So give oral Mg supplements
f. Hypovolemia and hypotension
Due to loss of H2O
2. Metabolic changes
a. Hyperglycemia
Due to ↓ insulin secretion
b. Hyperlipidemia
↑ LDL and TG
c. Hyperuricemia
∵ ↓ Excretion of uric acid
Thereby leading to gout
Patients may require allopurinol
3. Ototoxicity
Deafness, vertigo, tinnitus
Due to toxic effect on hair cells of inner ear
Dose-dependent
Common in IV use and renal impaired patients
Avoid other ototoxic drugs together
i.e., Aminoglycosides, cyclosporine, etc.
4. Hypersensitivity
Skin rashes
Eosinophilia
Photosensitivity
∵ It is a sulfonamide derivative (except ethacrynic acid)
5. Weakness, fatigue, dizziness, cramps due to hypokalemia

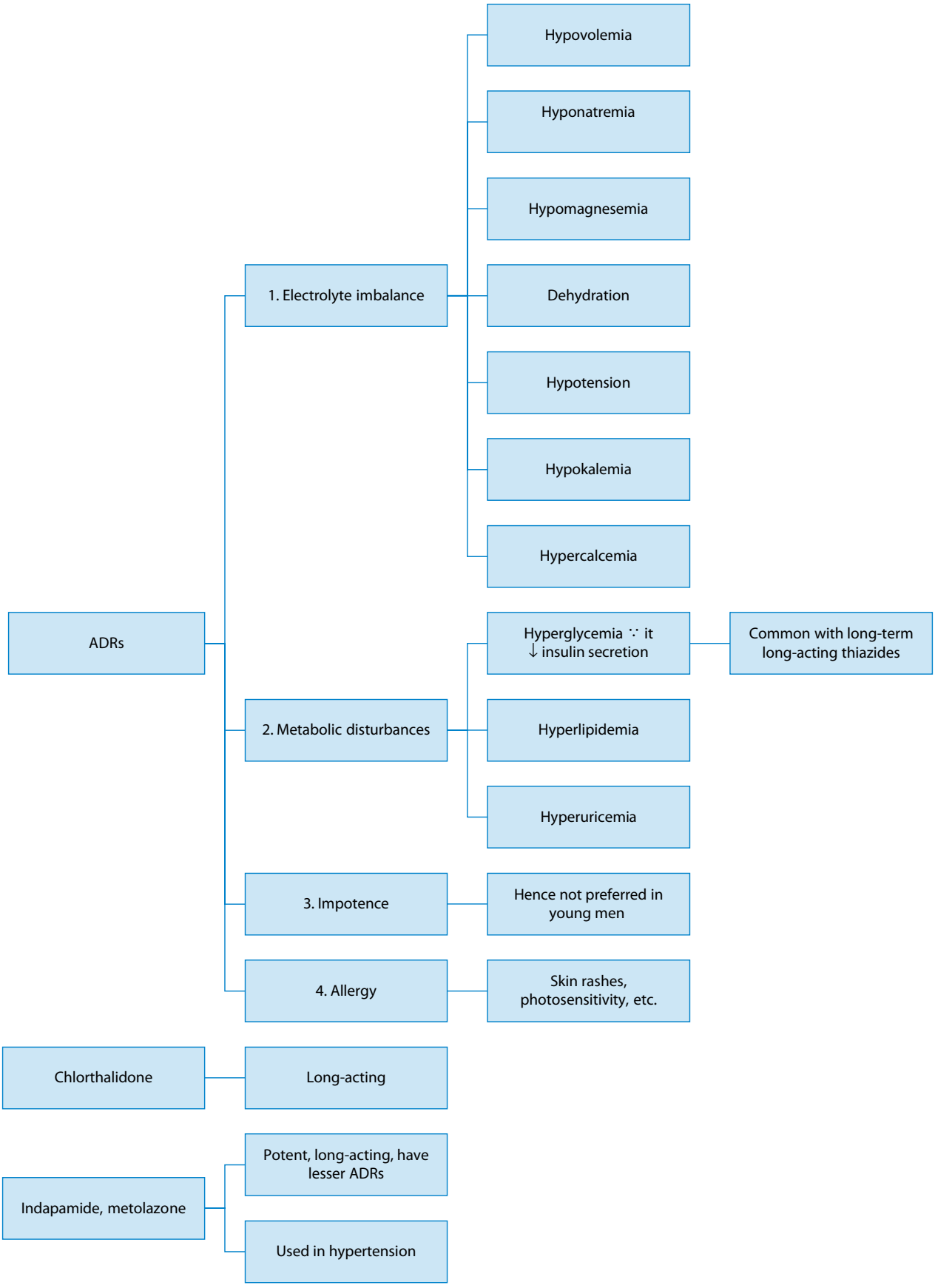
18.5 DRUG INTERACTIONS AND CONTRAINDICATIONS



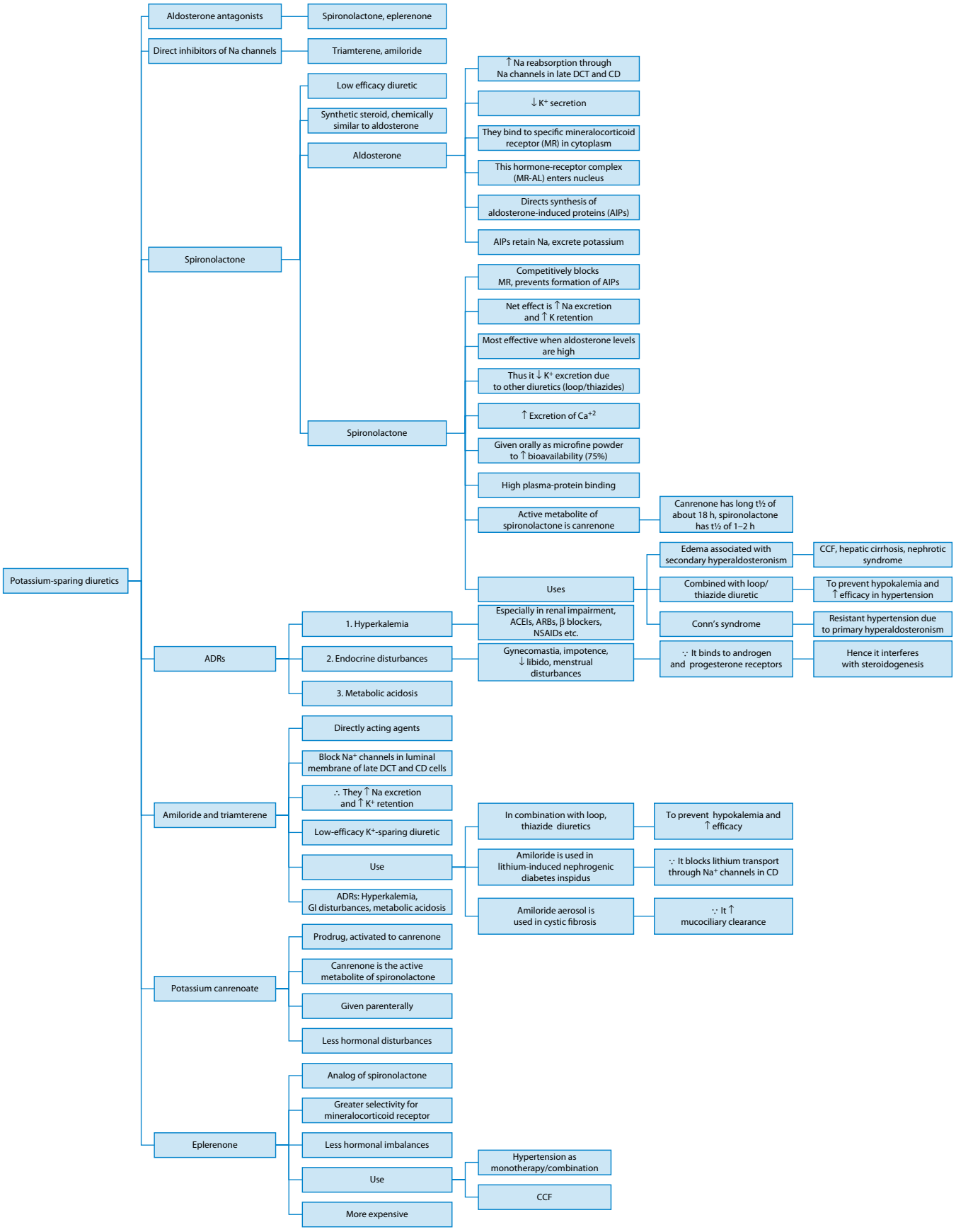
18.6 THIAZIDES AND THIAZIDE-LIKE DIURETICS



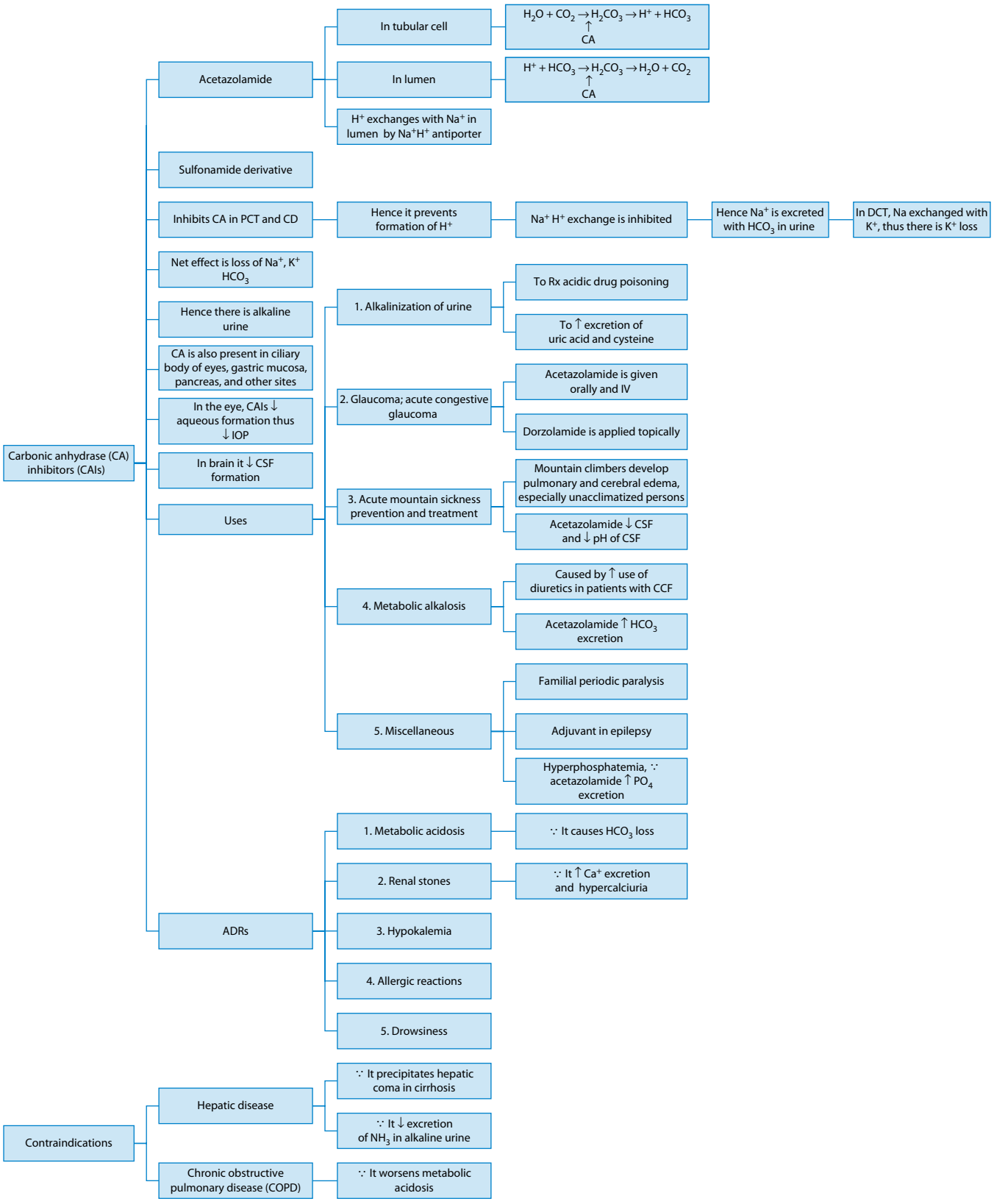
18.7 OTHER THIAZIDE DIURETICS AND ADRs



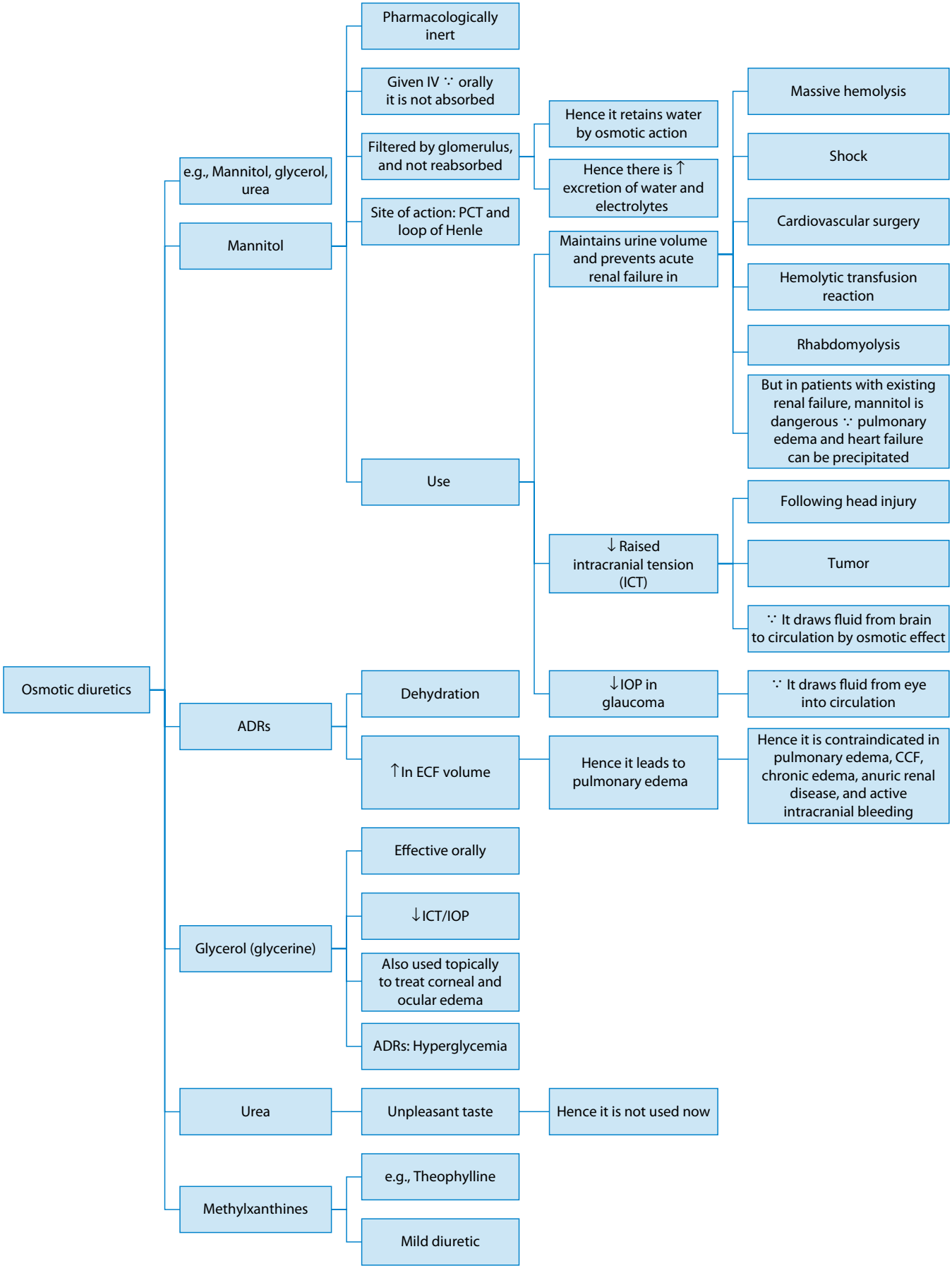
18.8 POTASSIUM-SPARING DIURETICS



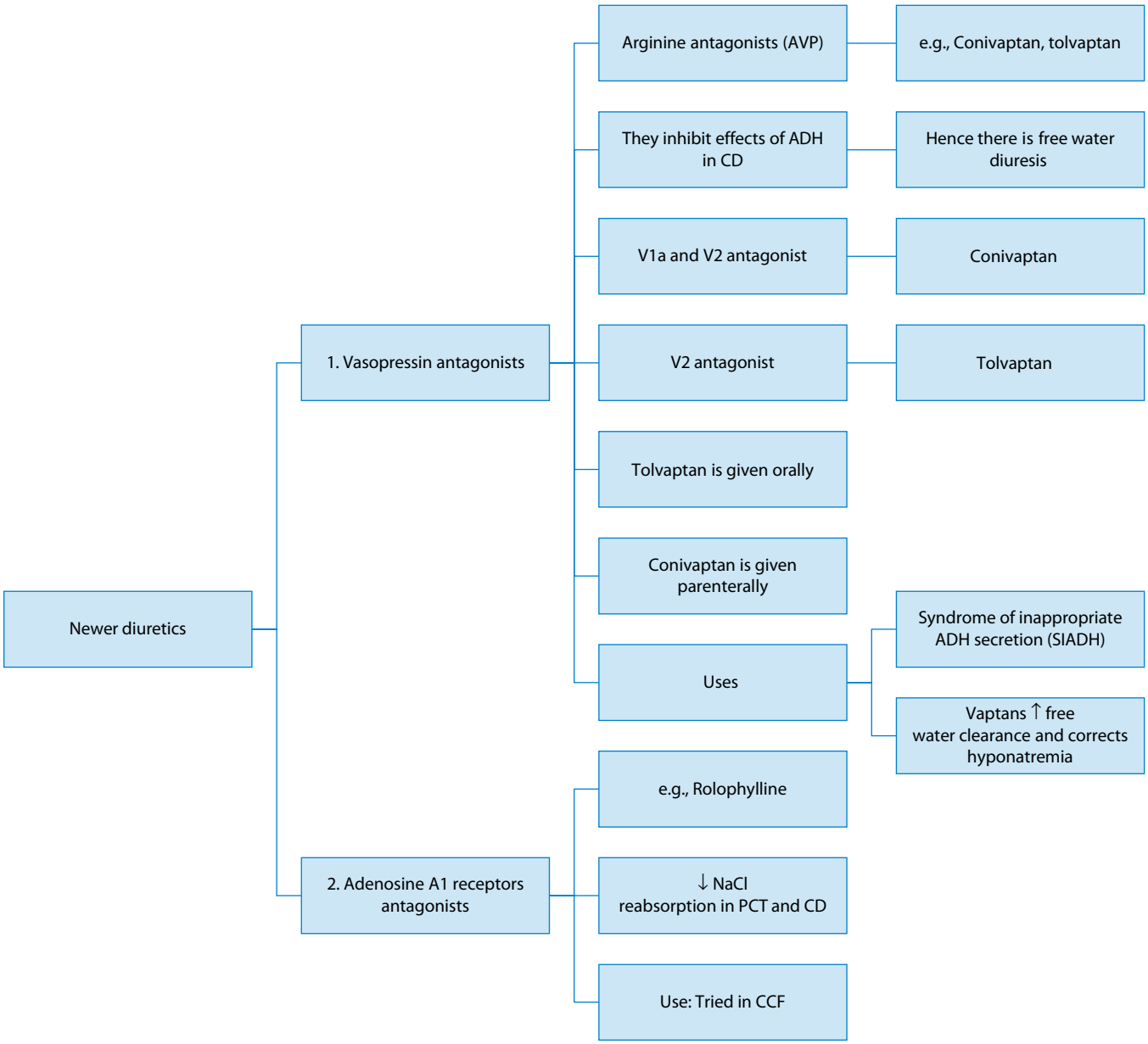
18.9 CARBONIC ANHYDRASE (CA) INHIBITORS (CAIs)



18.10 OSMOTIC DIURETICS



18.11 NEWER DIURETICS

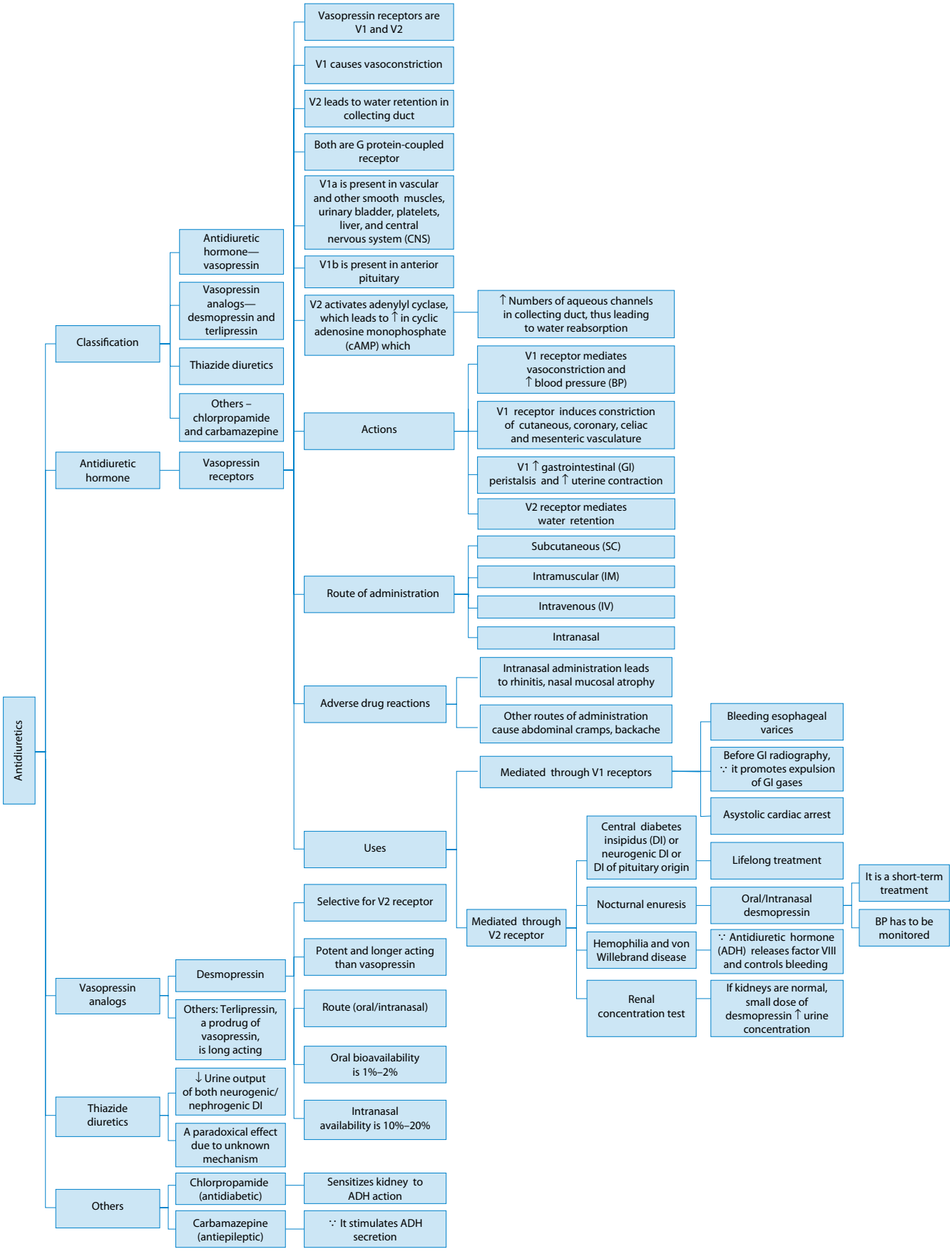


18.12 TABLE ON DIFFERENCES BETWEEN DIURETICS

Thiazide	**Furosemide**
Medium efficacy	High
Acts on early DCT	TALH
Inhibits Na^+ Cl symport	Na^+ K^+ 2Cl cotransport
Onset 1 h	20–40 min
Duration of action long: 8–12 h	8–6 h
No response on ↑ dose	Dose dependent ↑
Causes hyperuricemia	No change
↑ Blood sugar	No change
No ototoxicity	Ototoxic
Use: Hypertension	Edema

Furosemide	**Spironolactone**
Sulfonamide	Steroid
Acts on TALH	DCT and CD
Na^+ K^+ 2Cl cotransport blocked	Aldosterone blocker
High efficacy	Low efficacy
Quick onset (minutes)	Slow onset (days)
Hypokalemia	Hyperkalemia
Causes ototoxicity	Causes gynecomastia, hirsutism
Use – edema	Hyperaldosteronism, as adjuvant to diuretics
Caution: Allergy to sulfonamides	Caution: Peptic ulcer

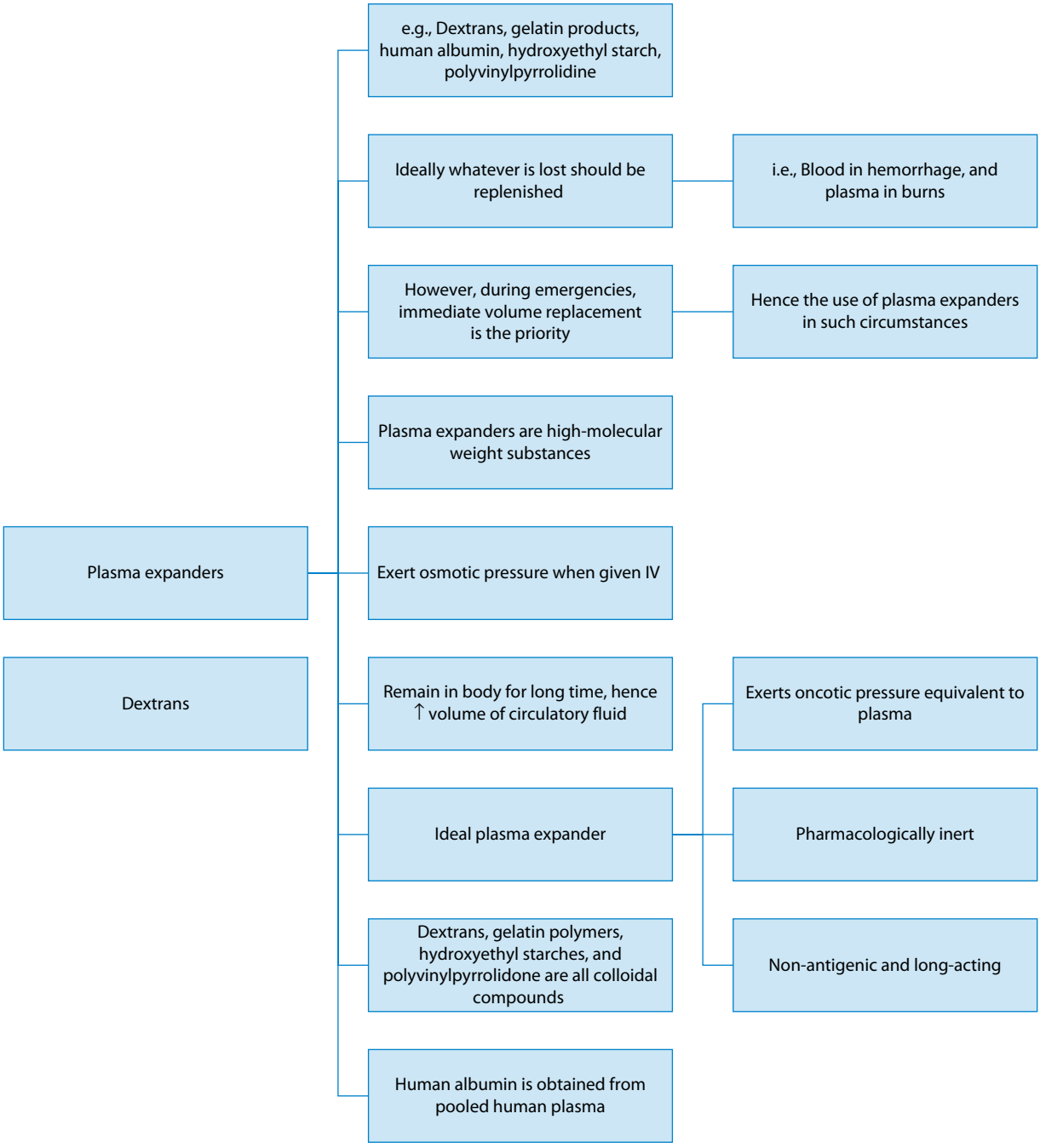
18.13 ANTIDIURETICS



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Pharmacotherapy of shock

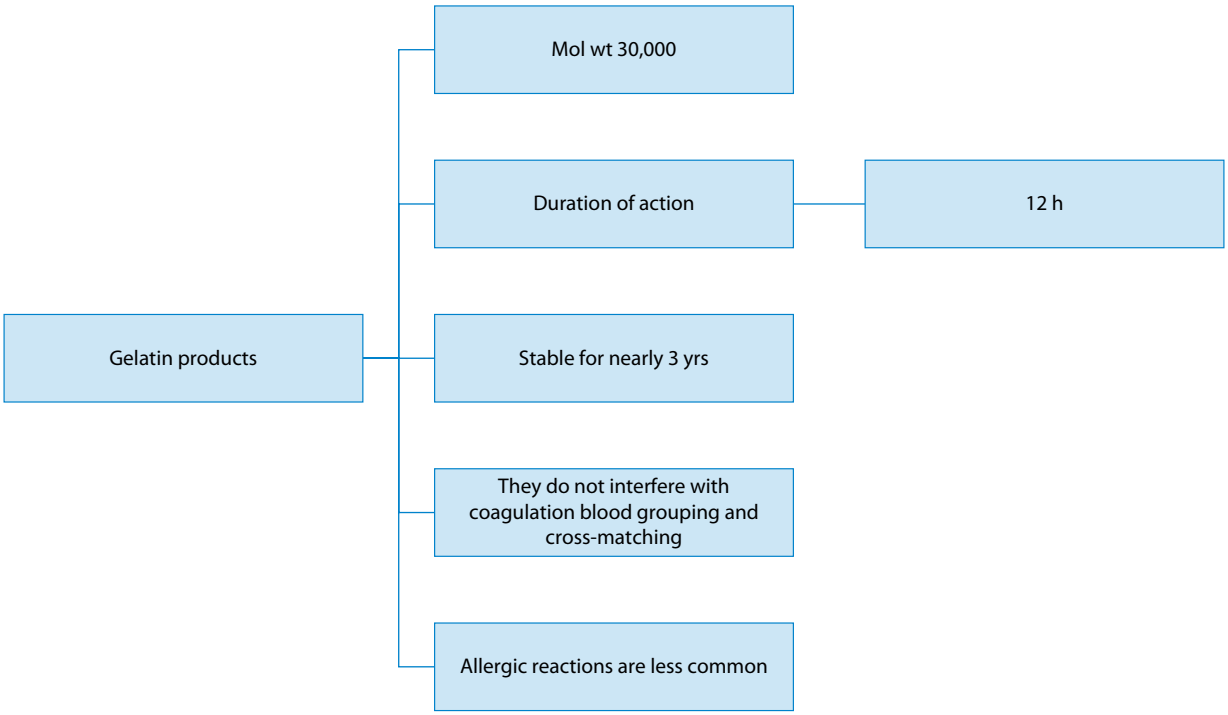
19.1 PLASMA EXPANDERS



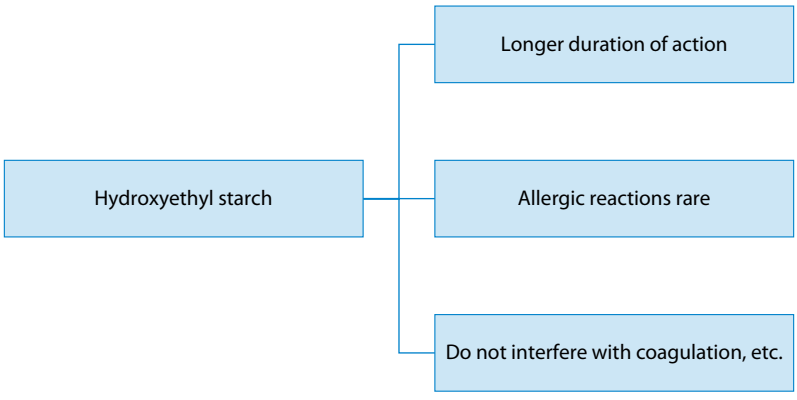
19.2 DEXTRANS



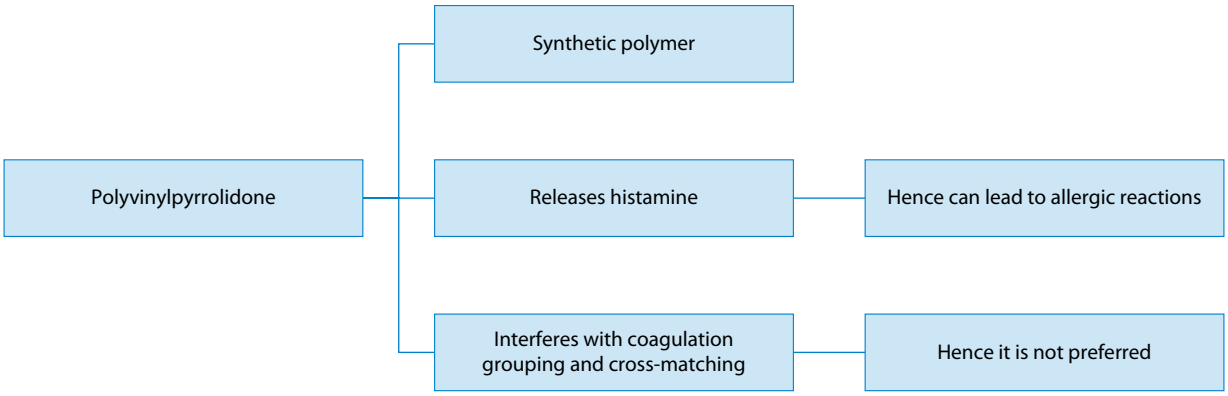
19.3 GELATIN PRODUCTS



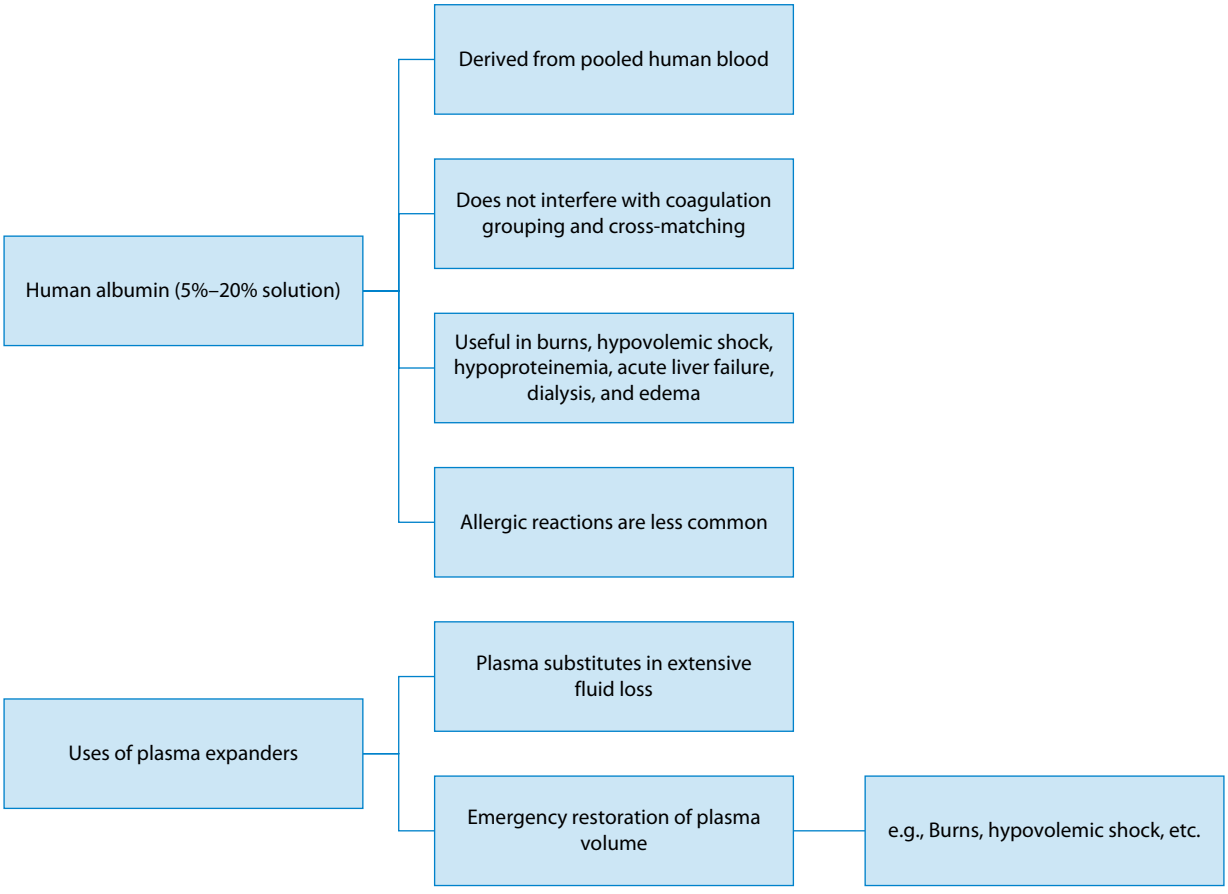
19.4 HYDROXYETHYL STARCH



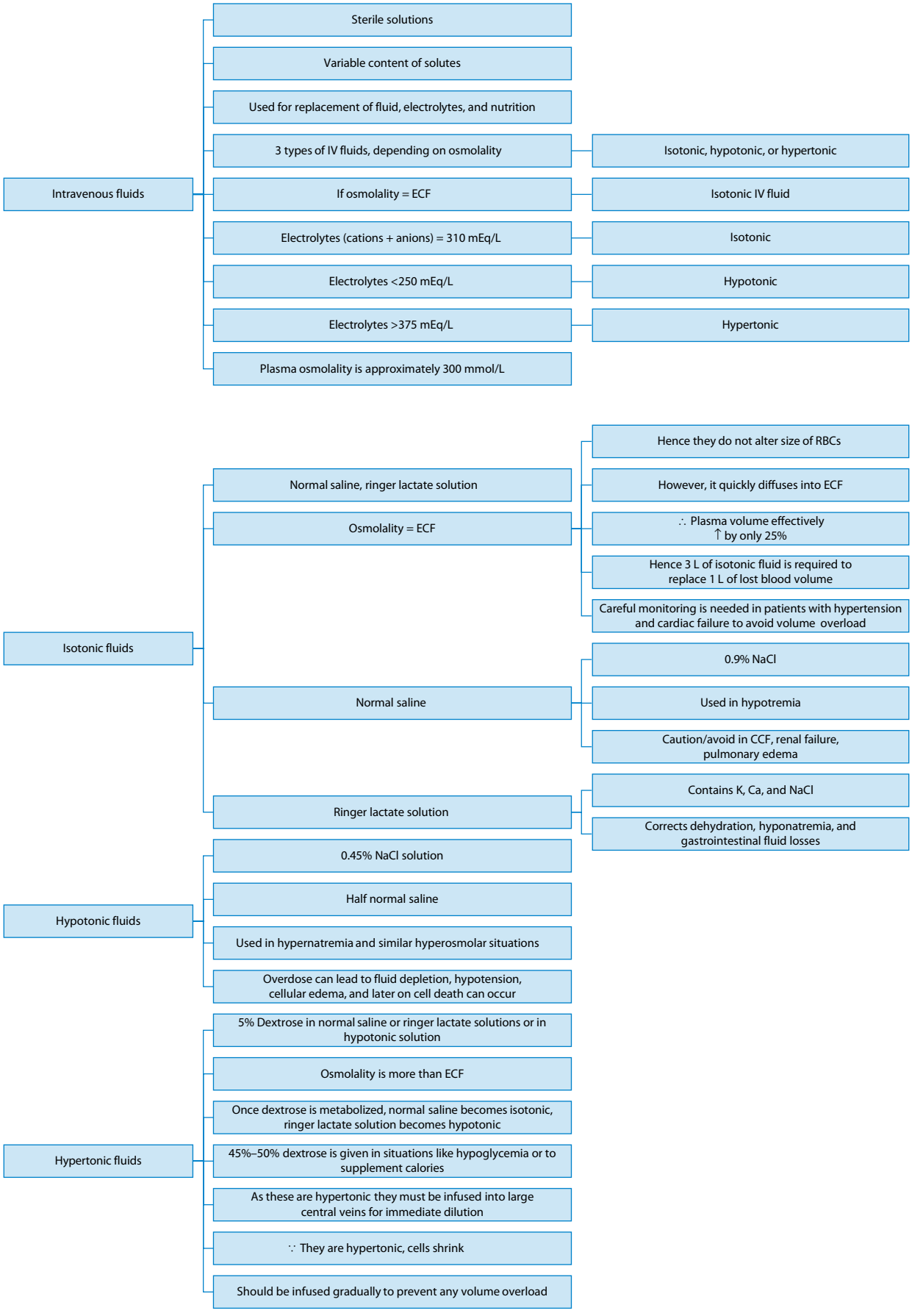
19.5 POLYVINYLPYRROLIDONE



19.6 HUMAN ALBUMIN



19.7 INTRAVENOUS FLUIDS



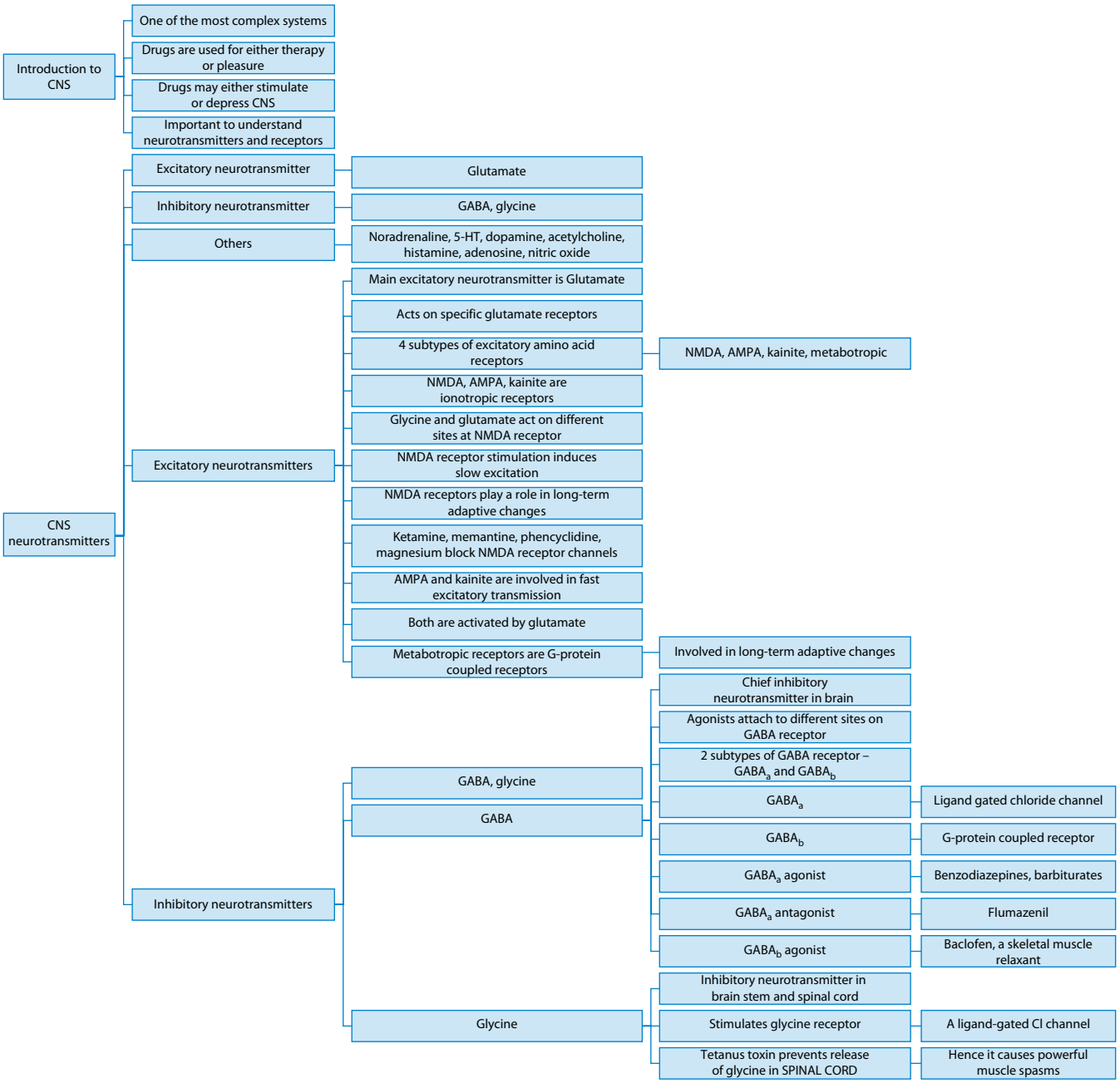
PART IV

Central nervous system (CNS) pharmacology

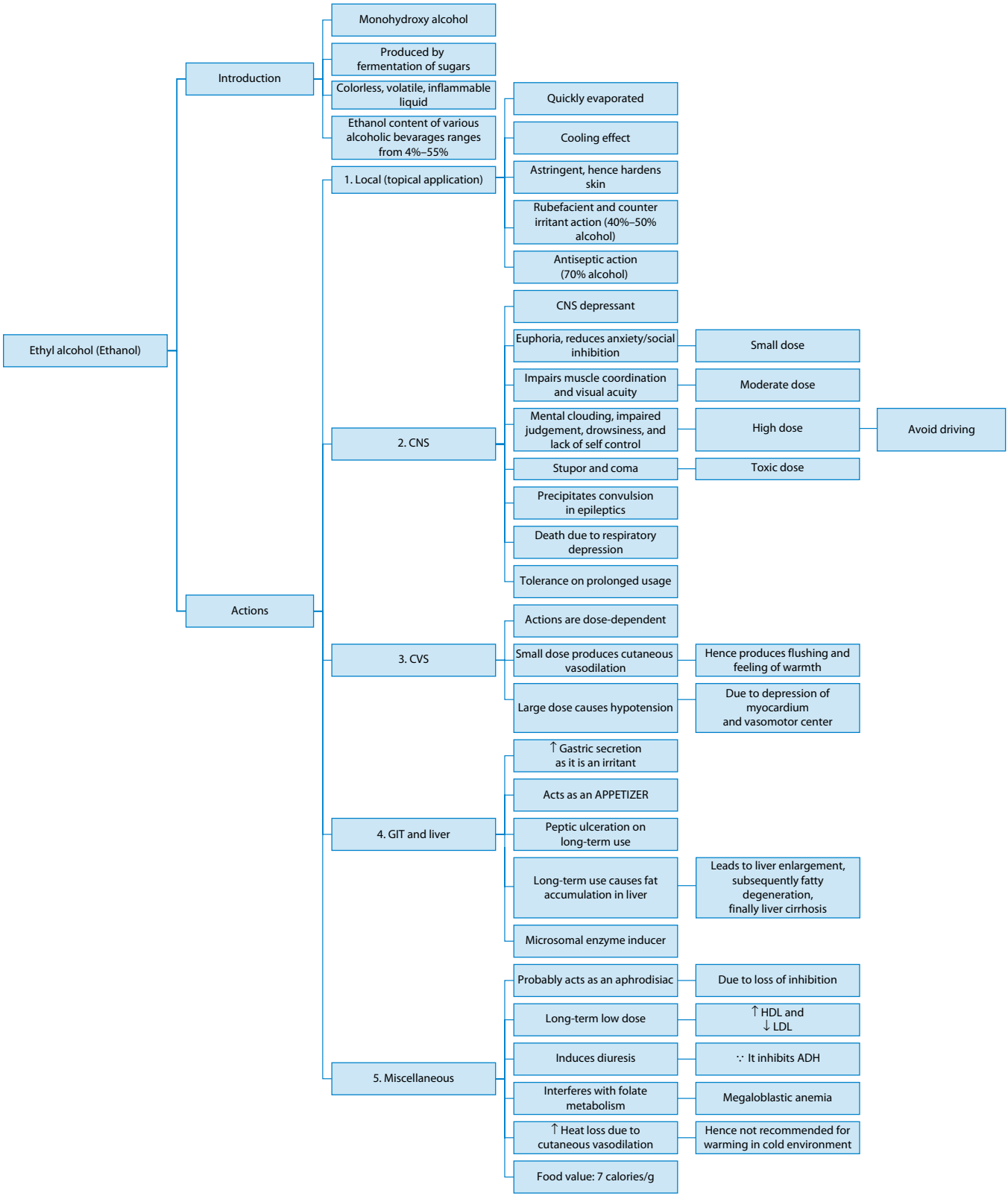
20

Introduction to CNS and alcohol

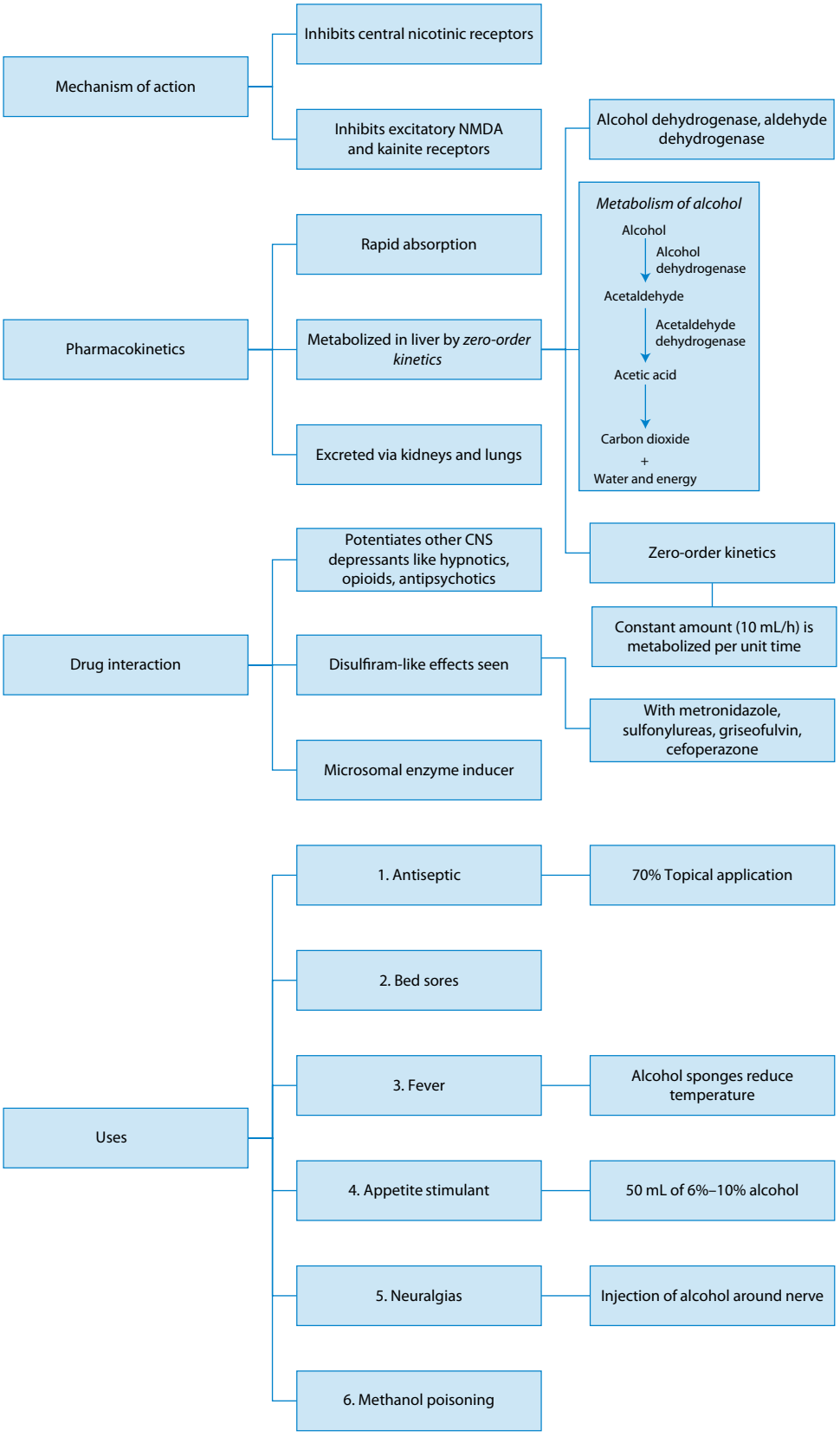
20.1 INTRODUCTION TO CNS, CNS NEUROTRANSMITTERS, EXCITATORY NEUROTRANSMITTERS, INHIBITORY NEUROTRANSMITTERS



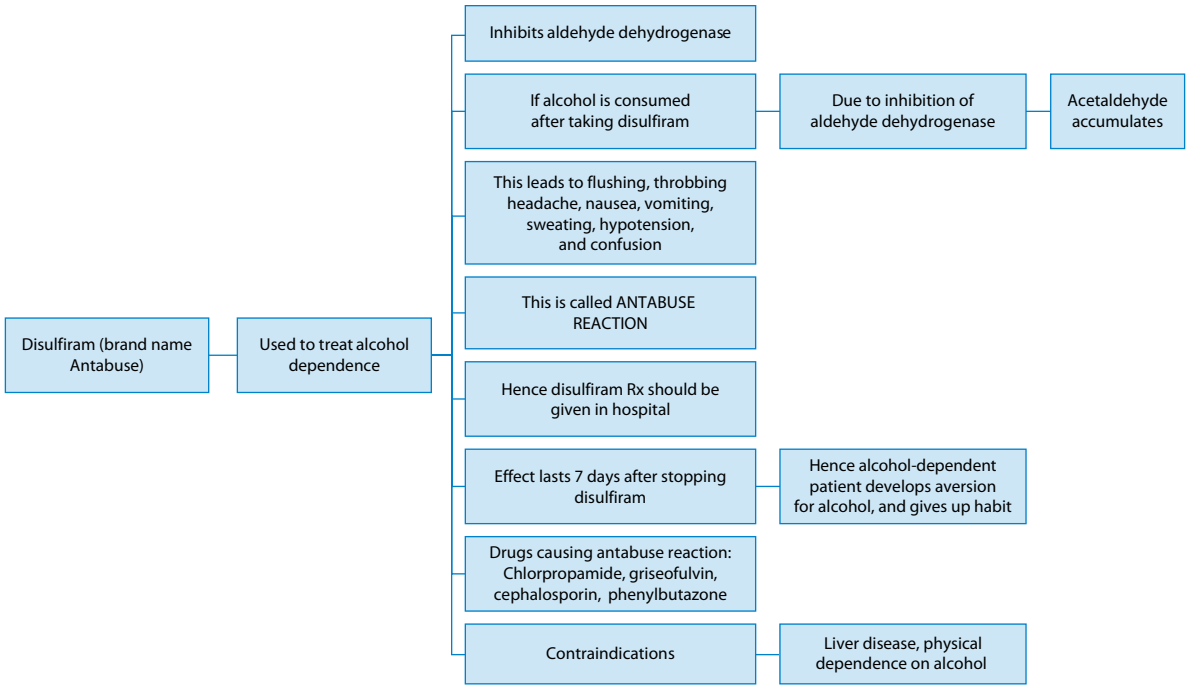
20.2 ALCOHOLS, ETHYL ALCOHOL – INTRODUCTION AND ACTIONS



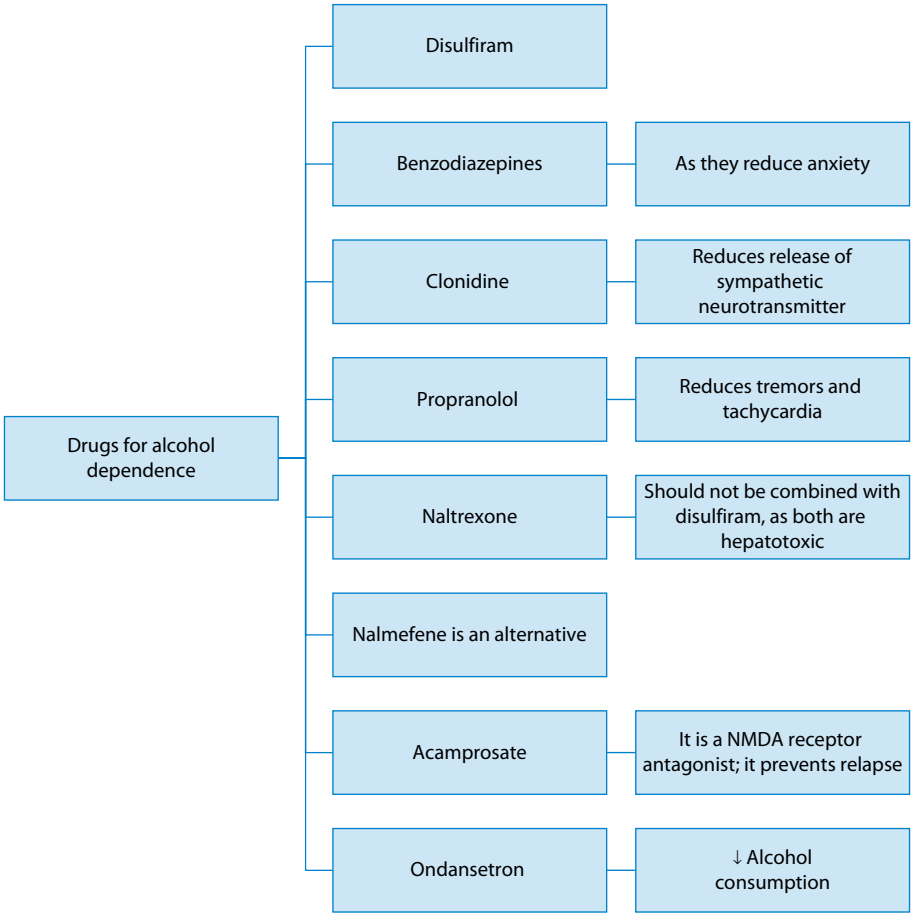
20.3 MECHANISM OF ACTION, PHARMACOKINETICS, DRUG INTERACTION, AND USES



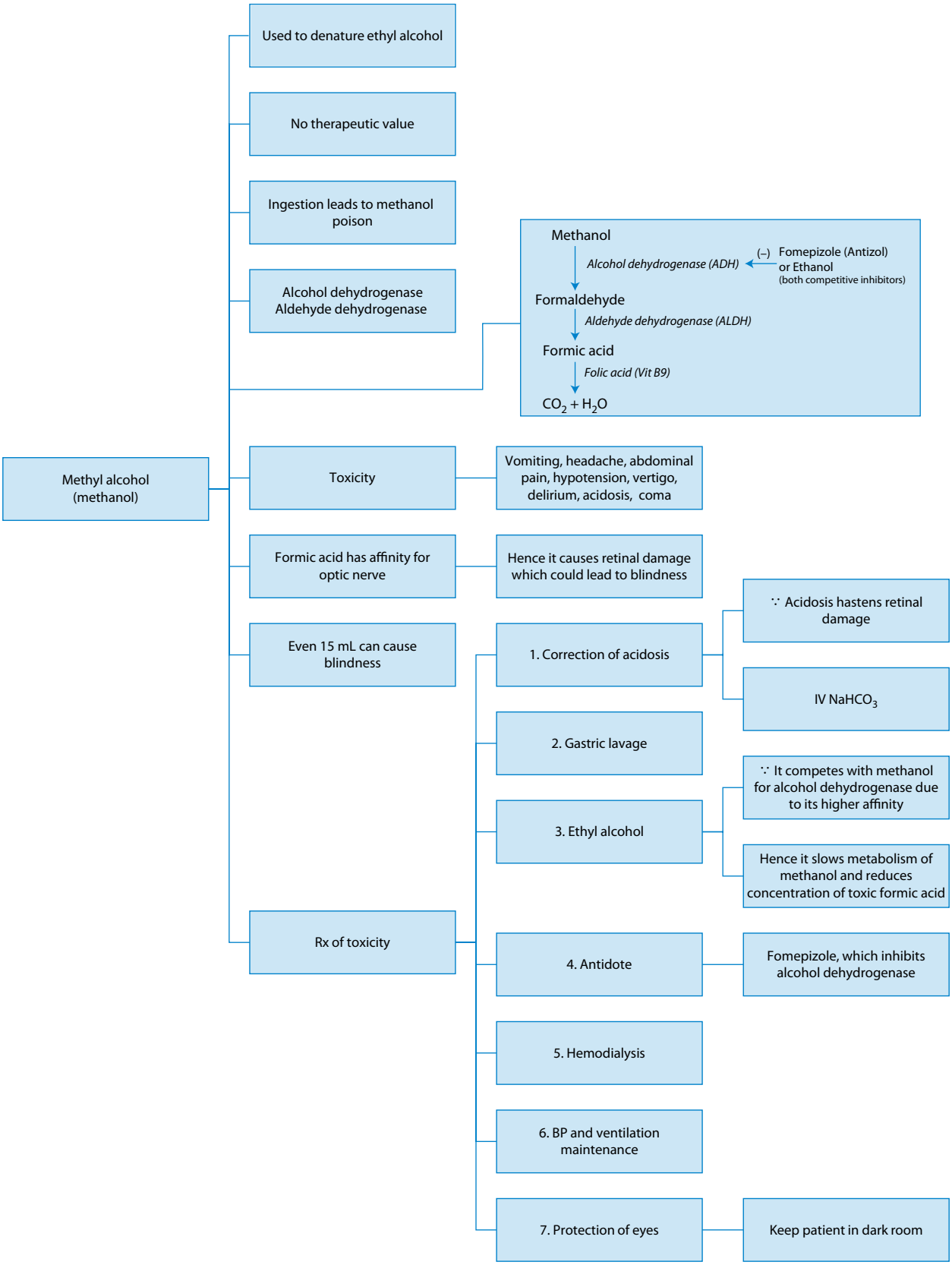
20.4 DISULFIRAM



20.5 DRUGS TO TREAT ALCOHOL DEPENDENCE



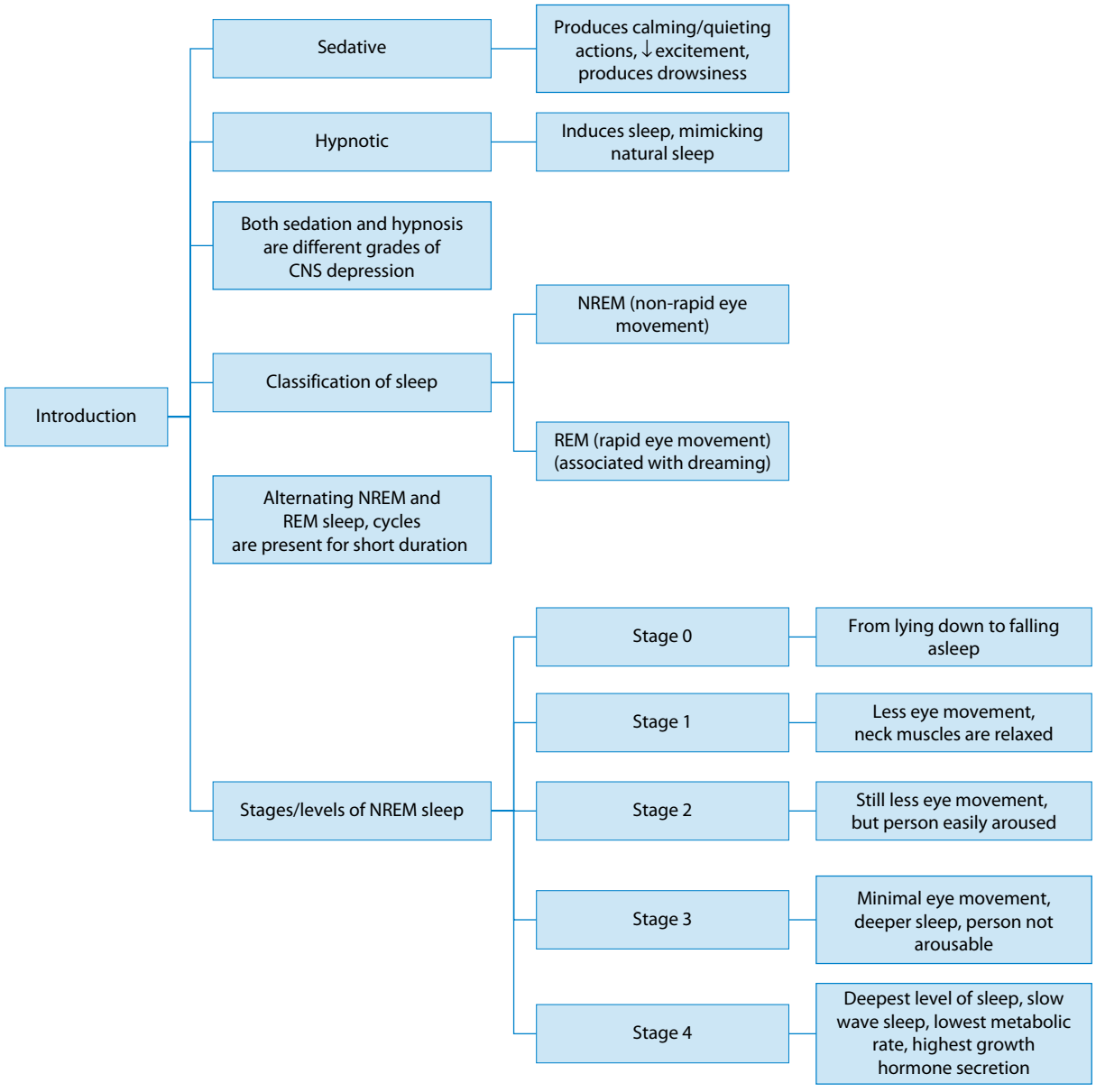
20.6 METHYL ALCOHOL (METHANOL)



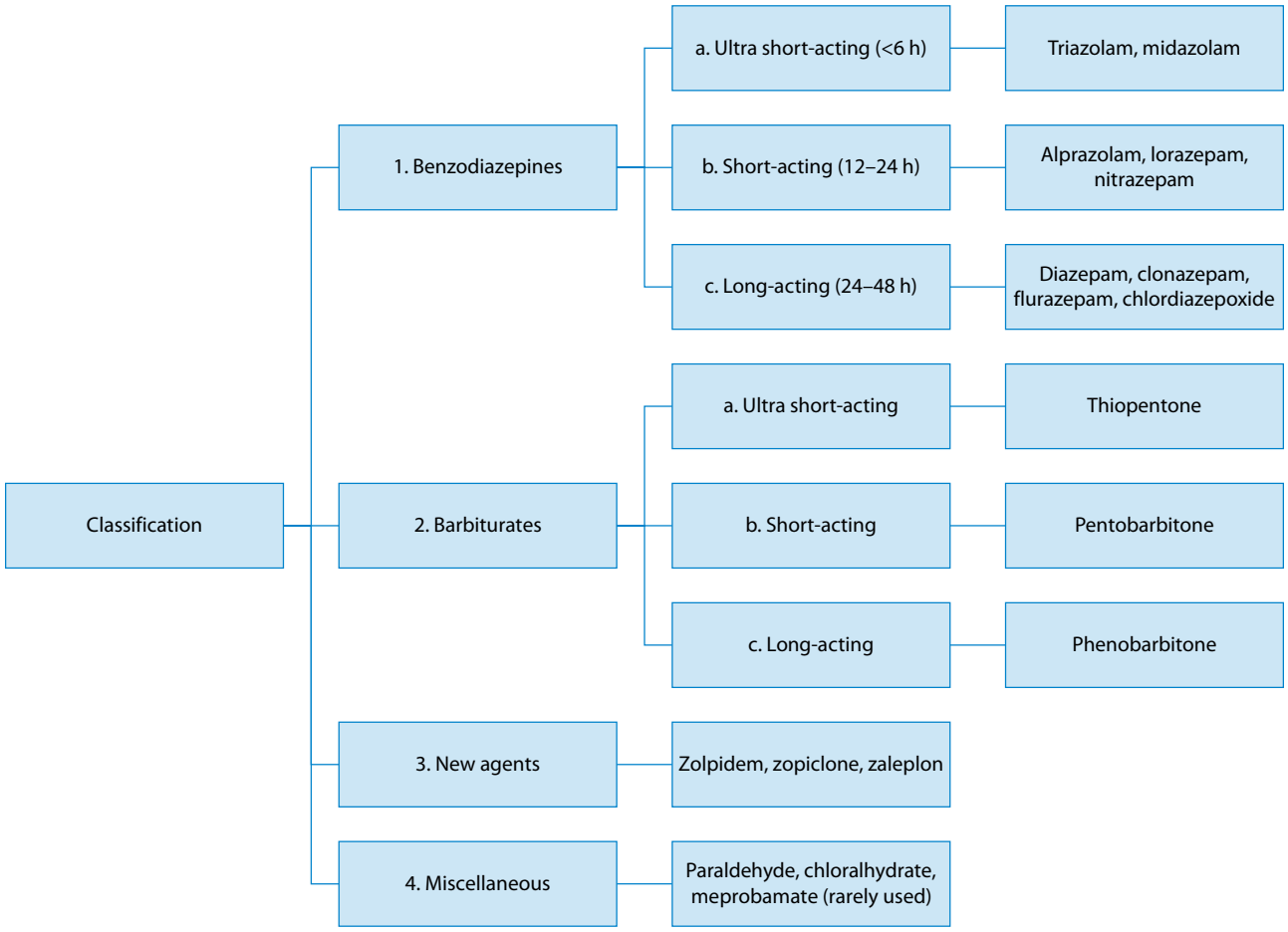
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Sedative hypnotics

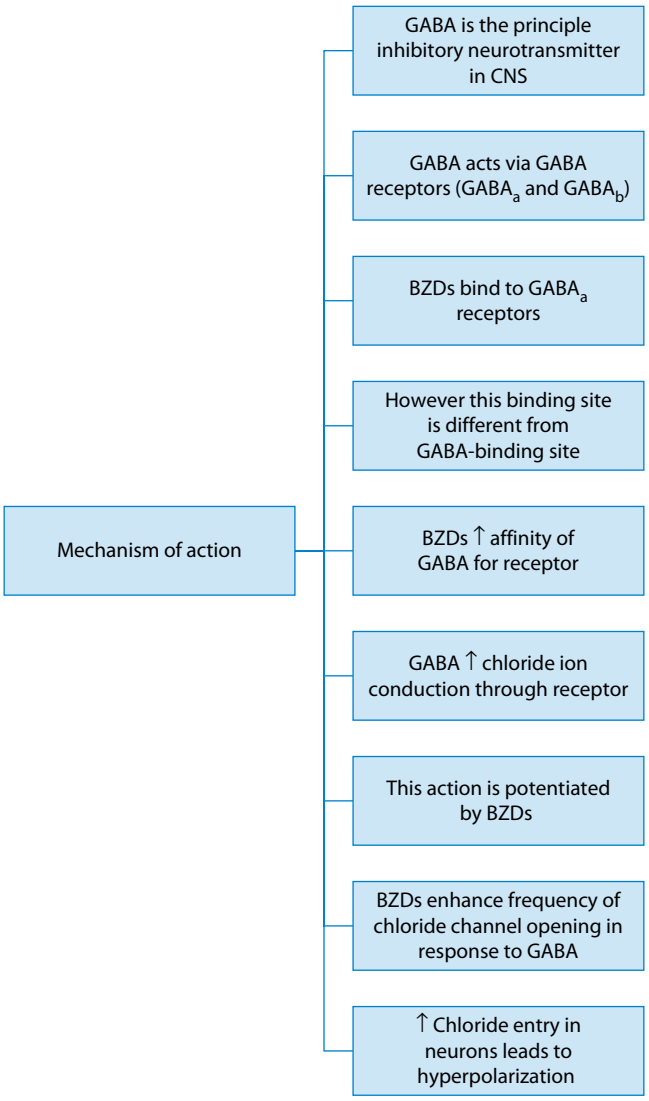
21.1 INTRODUCTION TO SEDATIVE HYPNOTICS



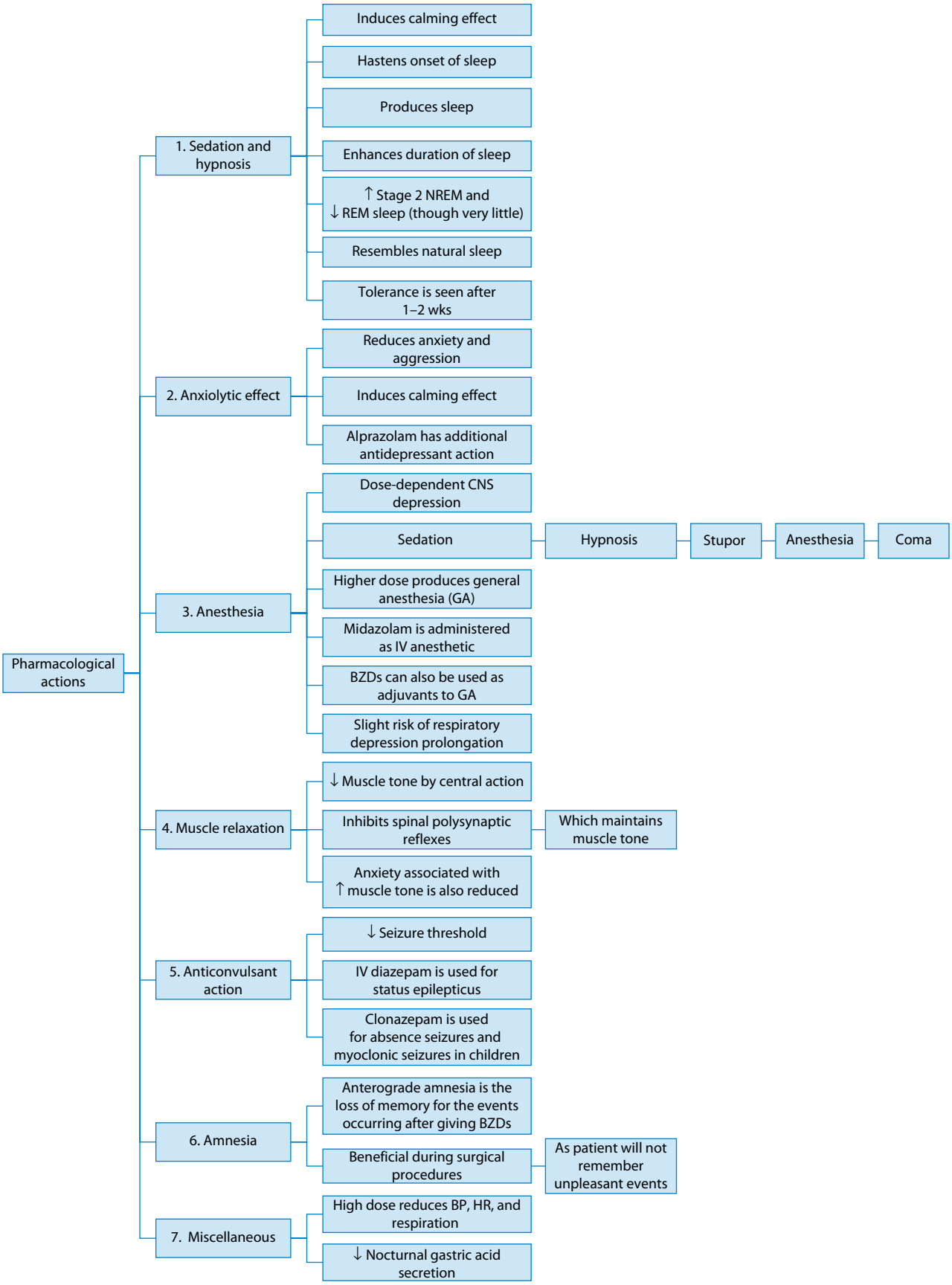
21.2 CLASSIFICATION



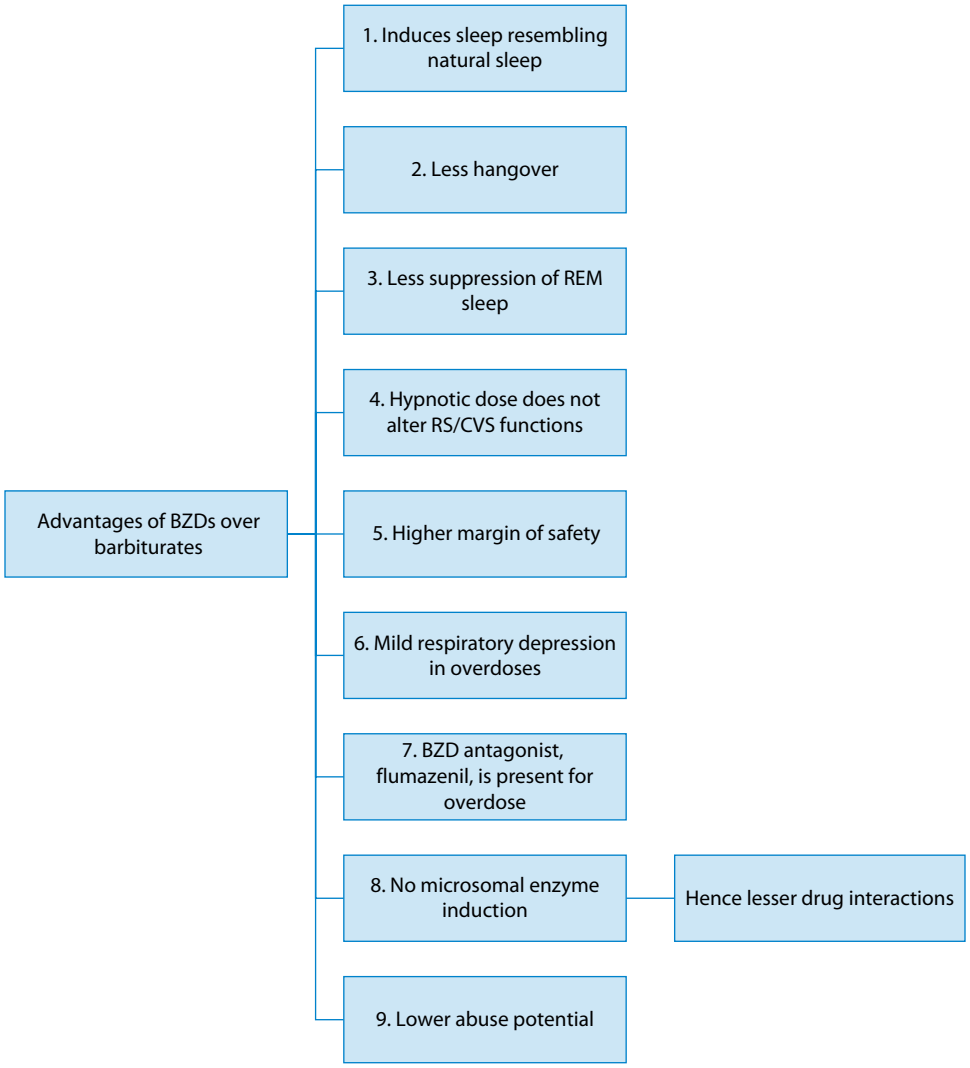
21.3 MECHANISM OF ACTION



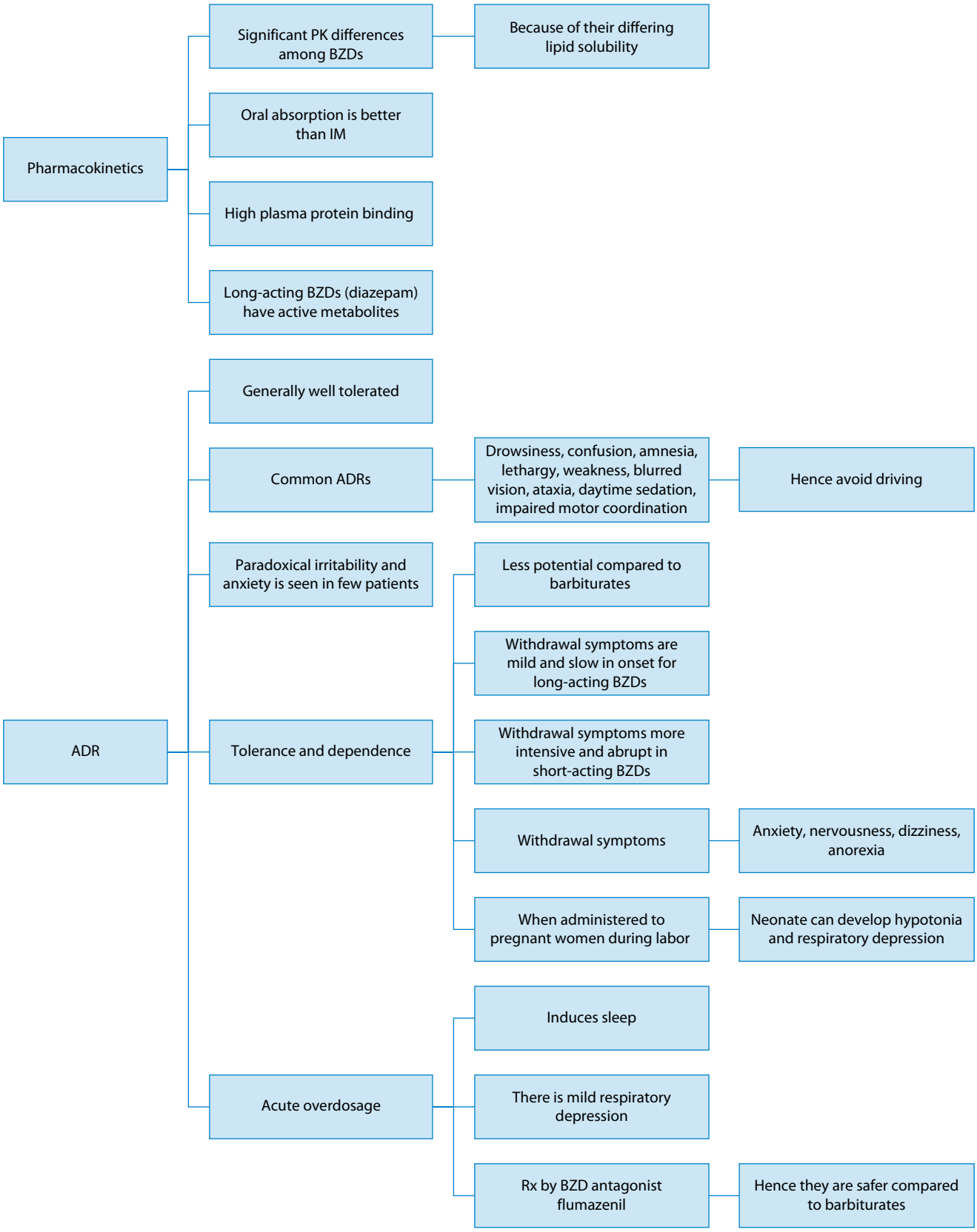
21.4 PHARMACOLOGICAL ACTIONS



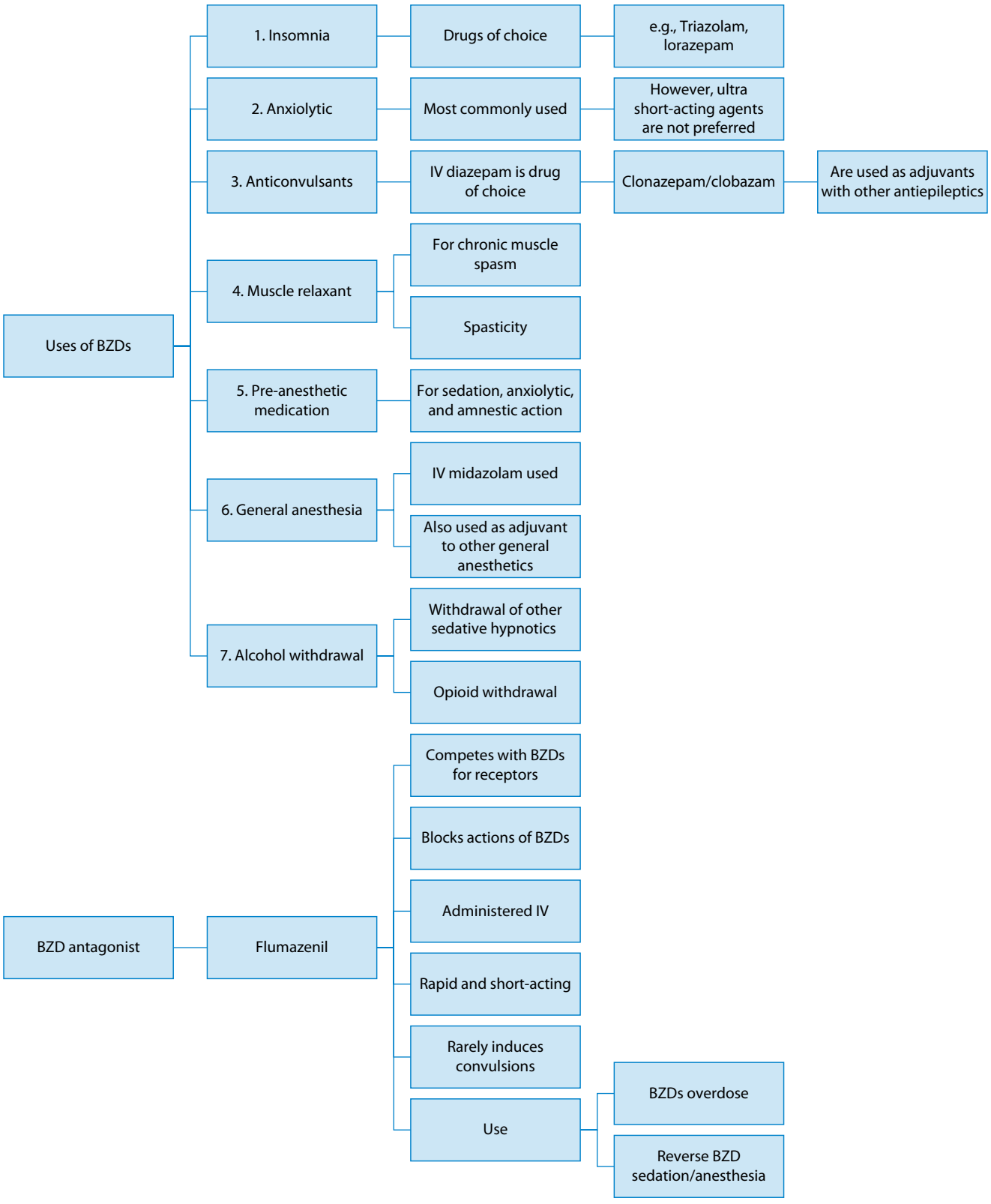
21.5 ADVANTAGES OF BZDs OVER BARBITURATES



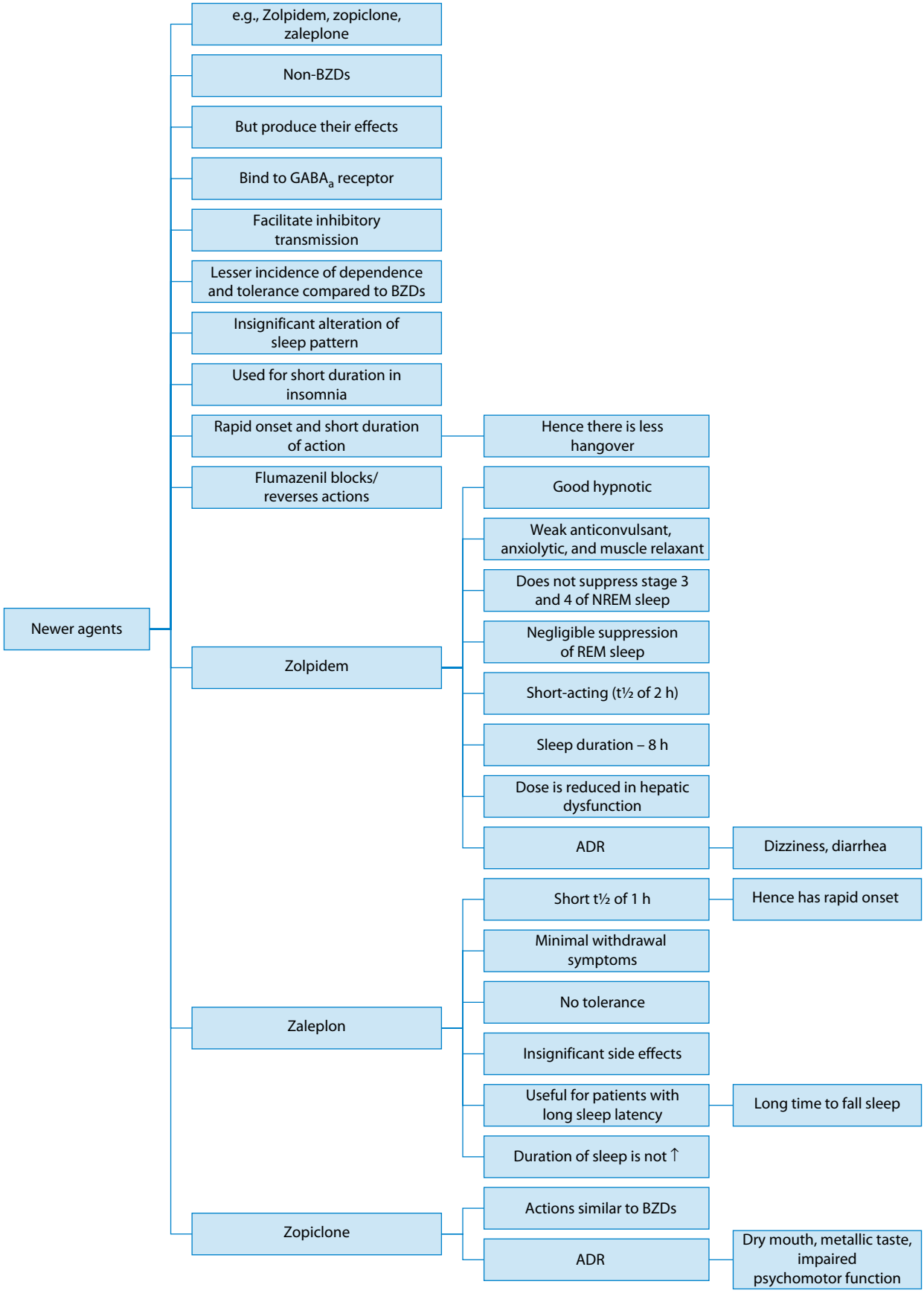
21.6 PHARMACOKINETICS AND ADRs



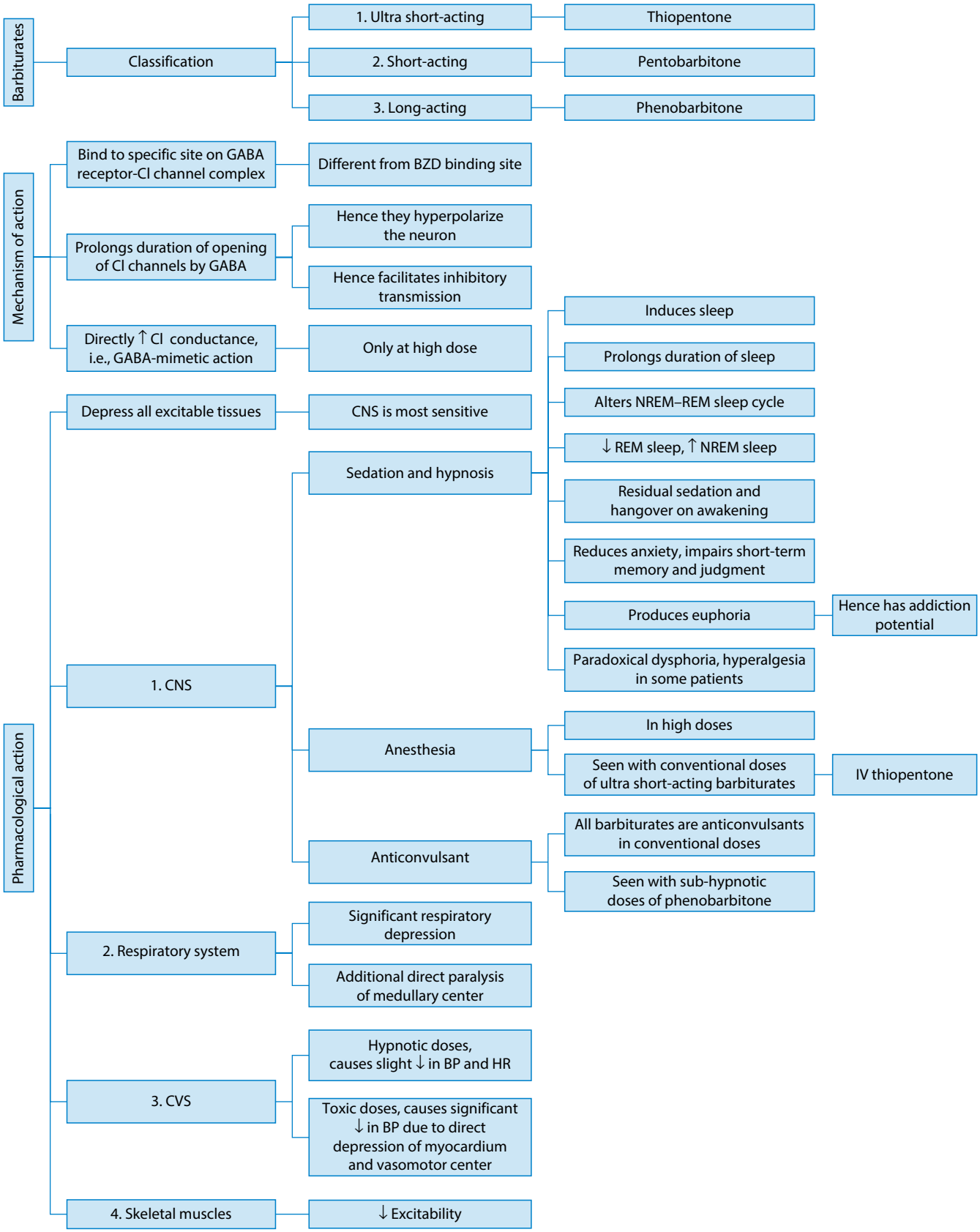
21.7 USES OF BZDs AND BZD ANTAGONIST



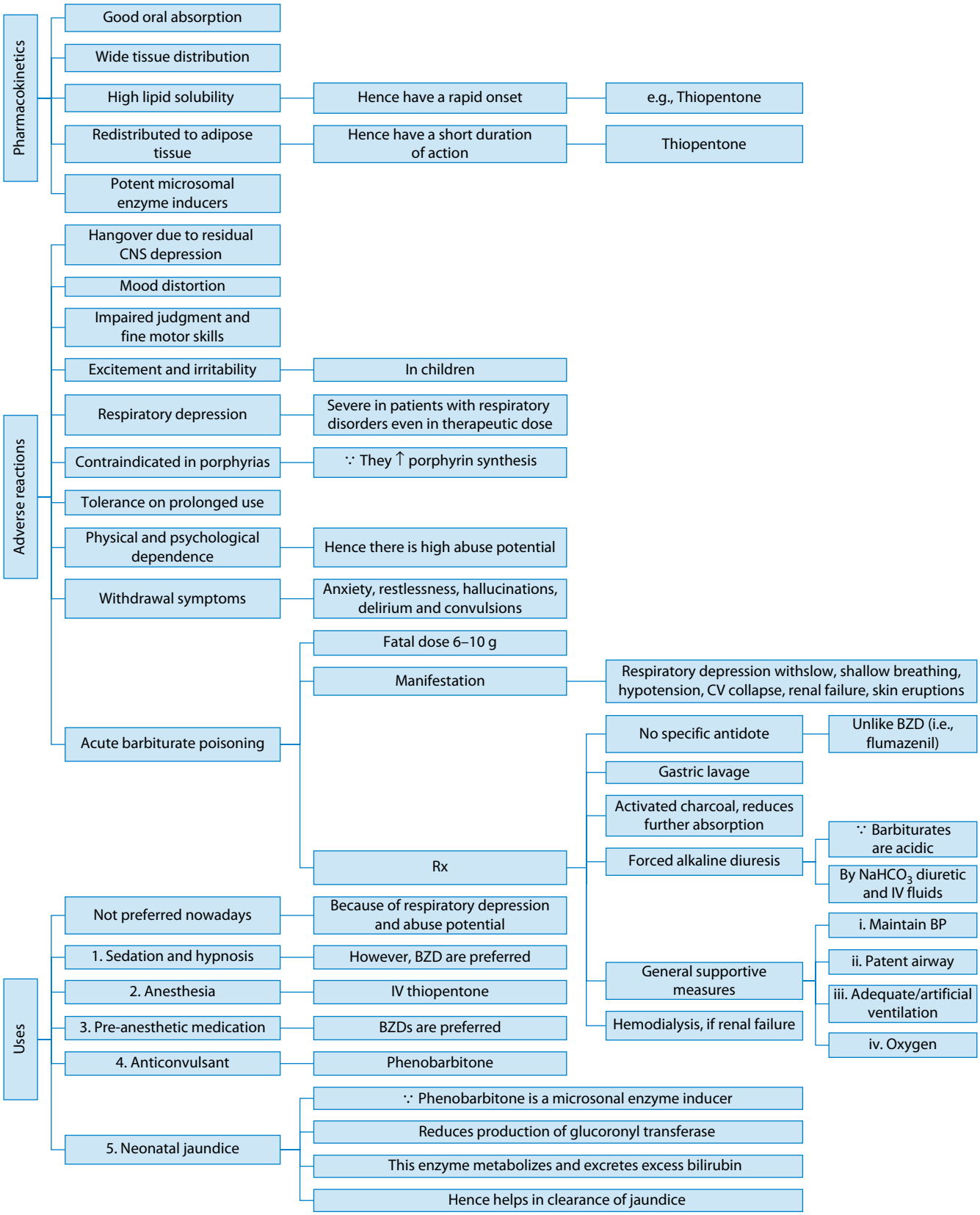
21.8 NEWER AGENTS



21.9 BARBITURATES CLASSIFICATION, MECHANISM OF ACTION, AND PHARMACOLOGICAL ACTION



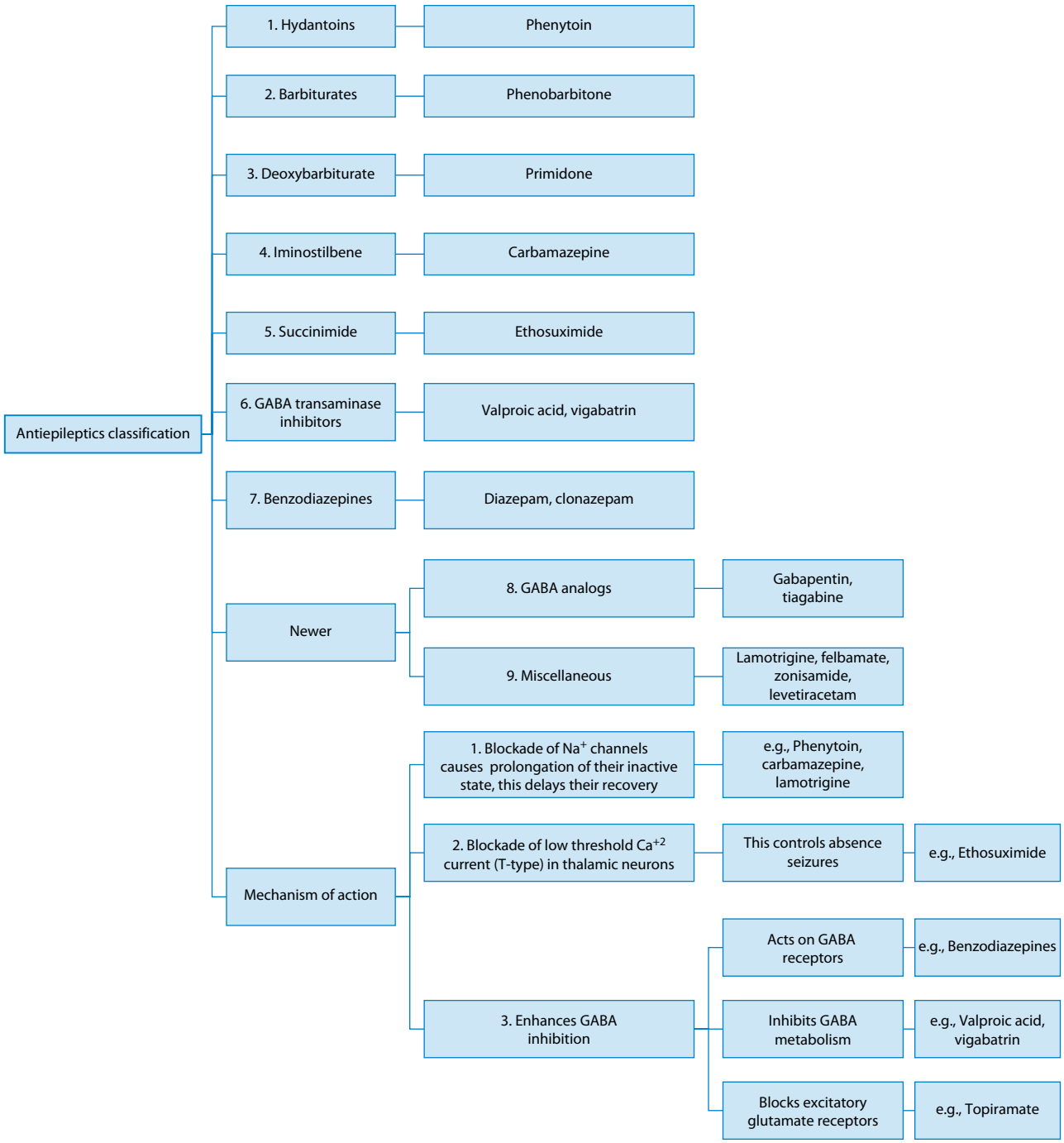
21.10 PHARMACOKINETICS, ADVERSE REACTIONS, AND USES



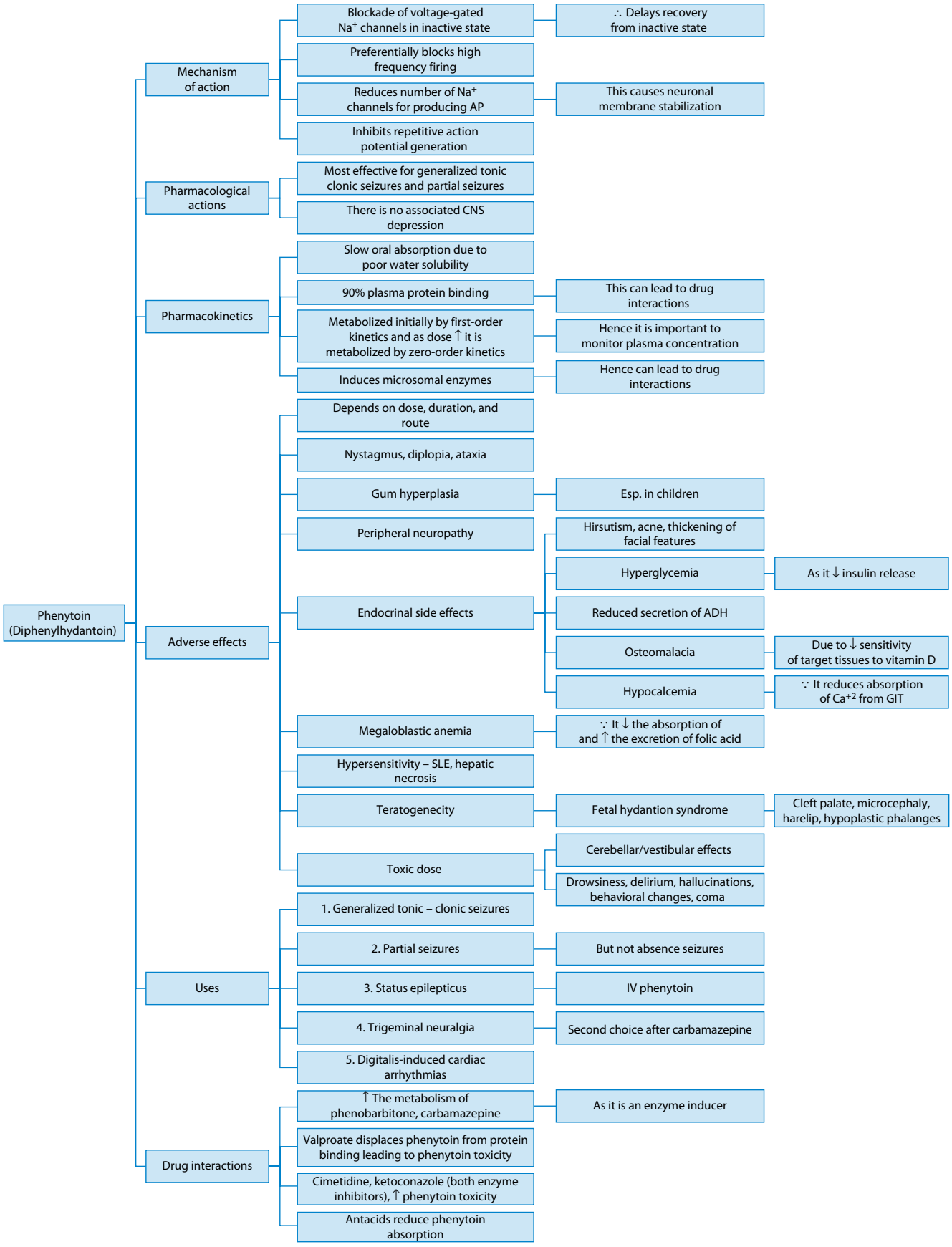
22

Antiepileptics

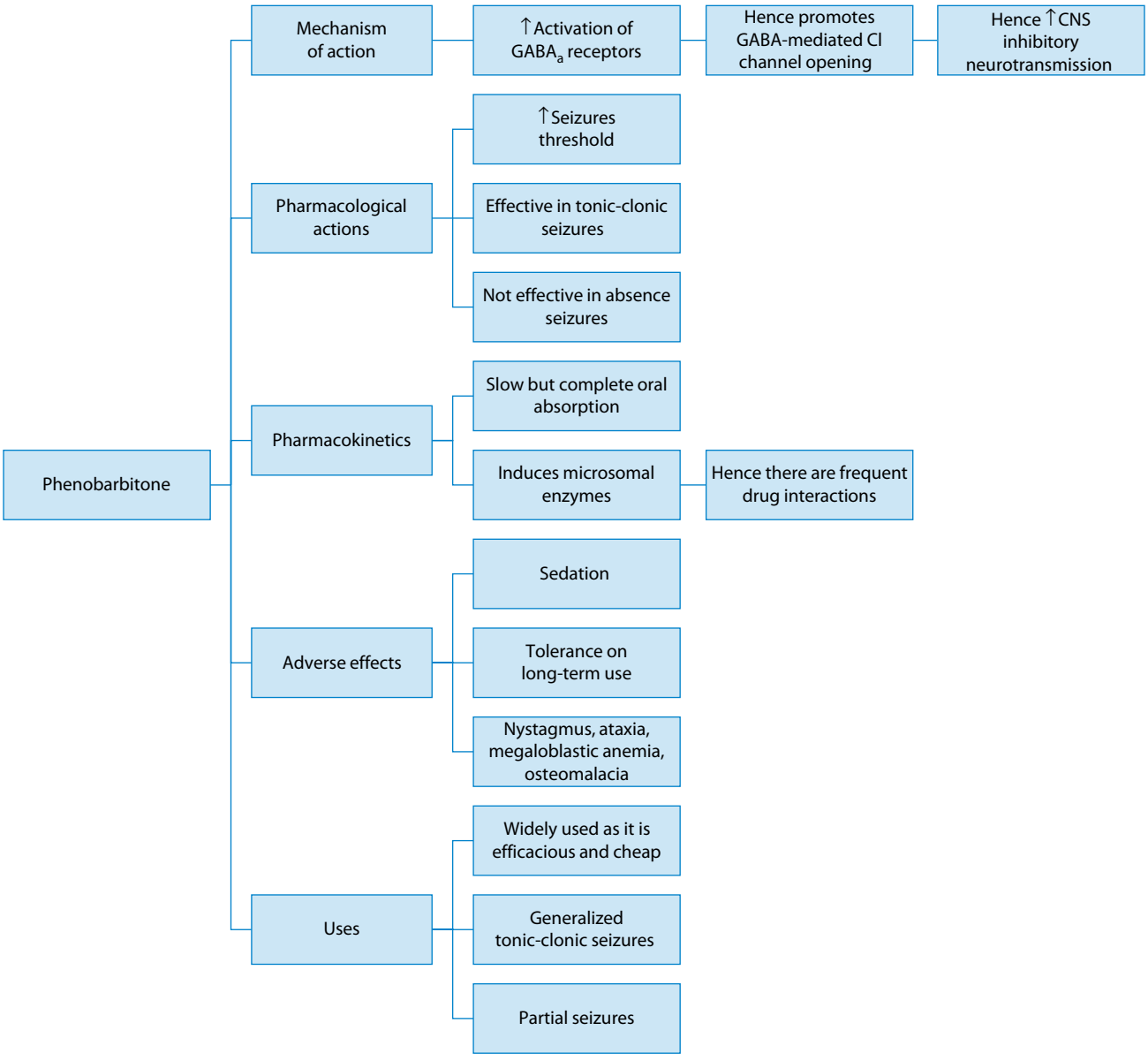
22.1 ANTIEPILEPTICS CLASSIFICATION AND MECHANISM OF ACTION



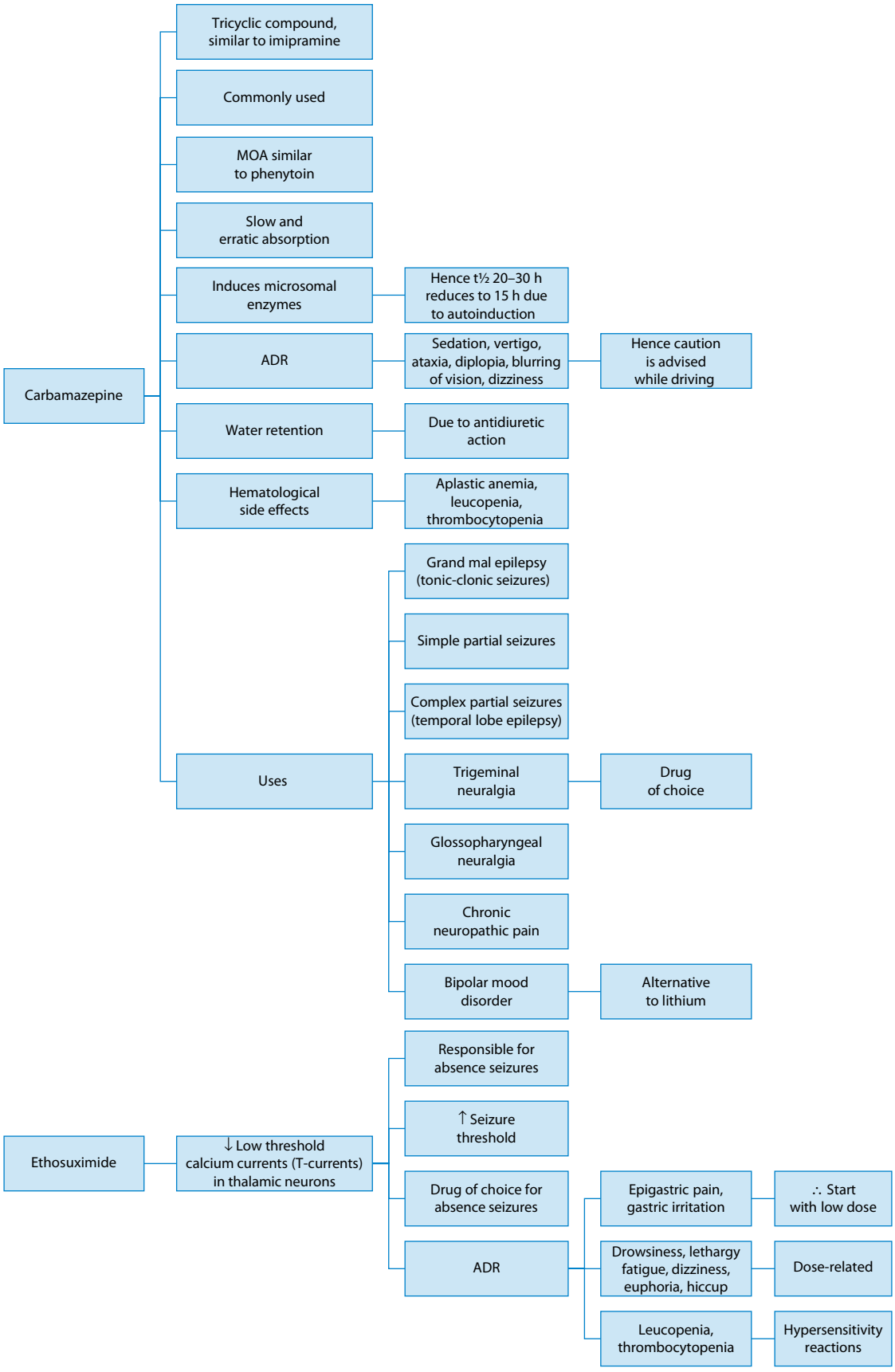
22.2 PHENYTOIN



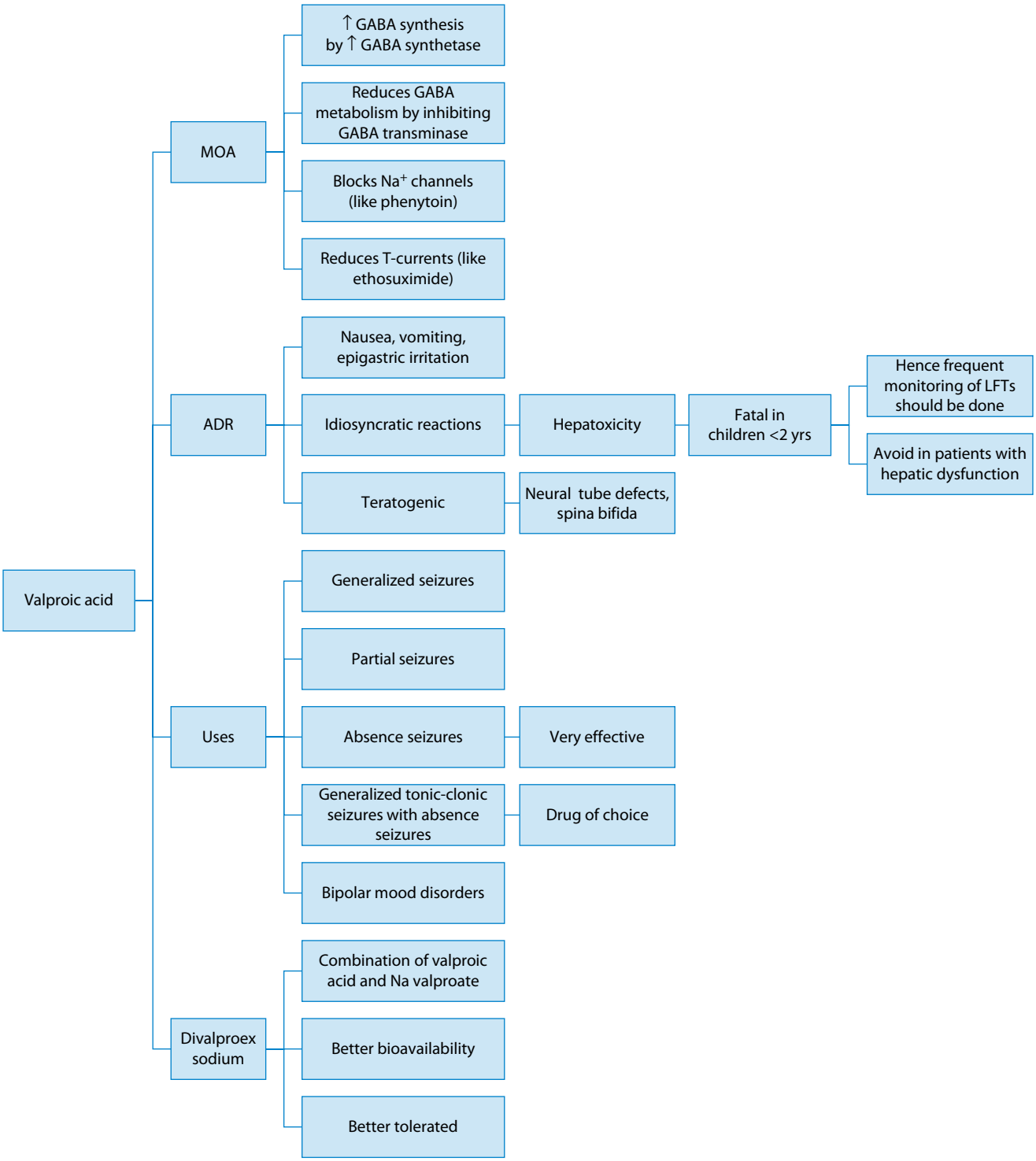
22.3 PHENOBARBITONE



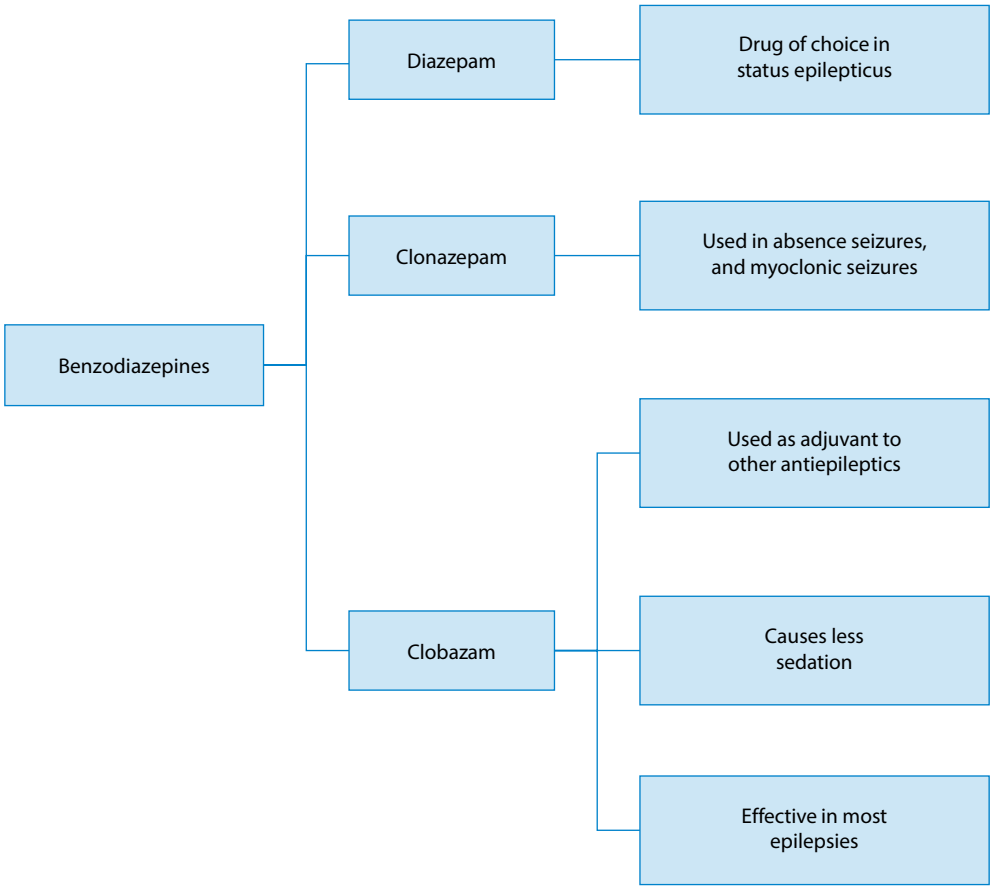
22.4 CARBAMAZEPINE AND ETHOSUXIMIDE



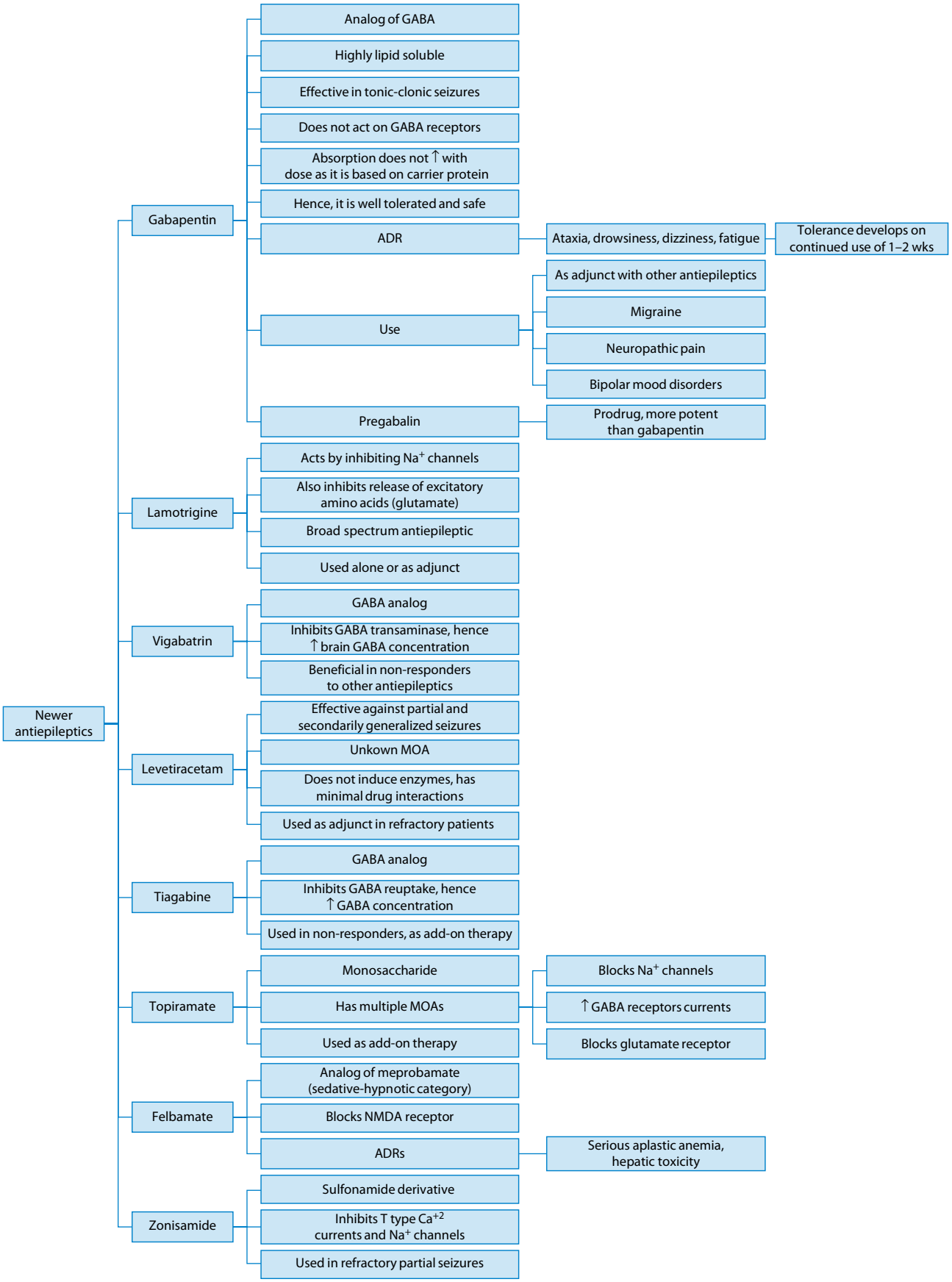
22.5 VALPROIC ACID



22.6 BENZODIAZEPINES



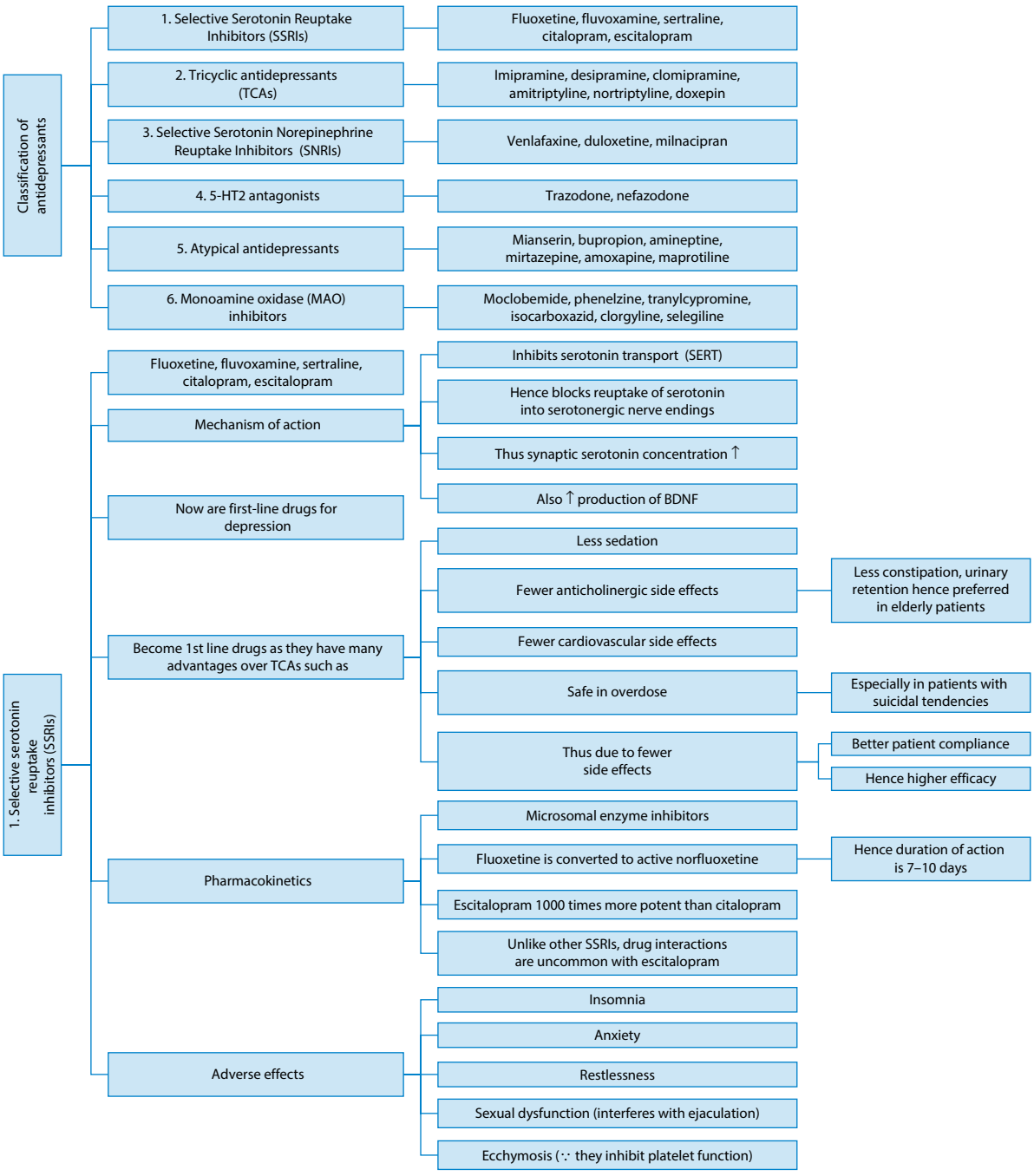
22.7 NEWER ANTIEPILEPTICS



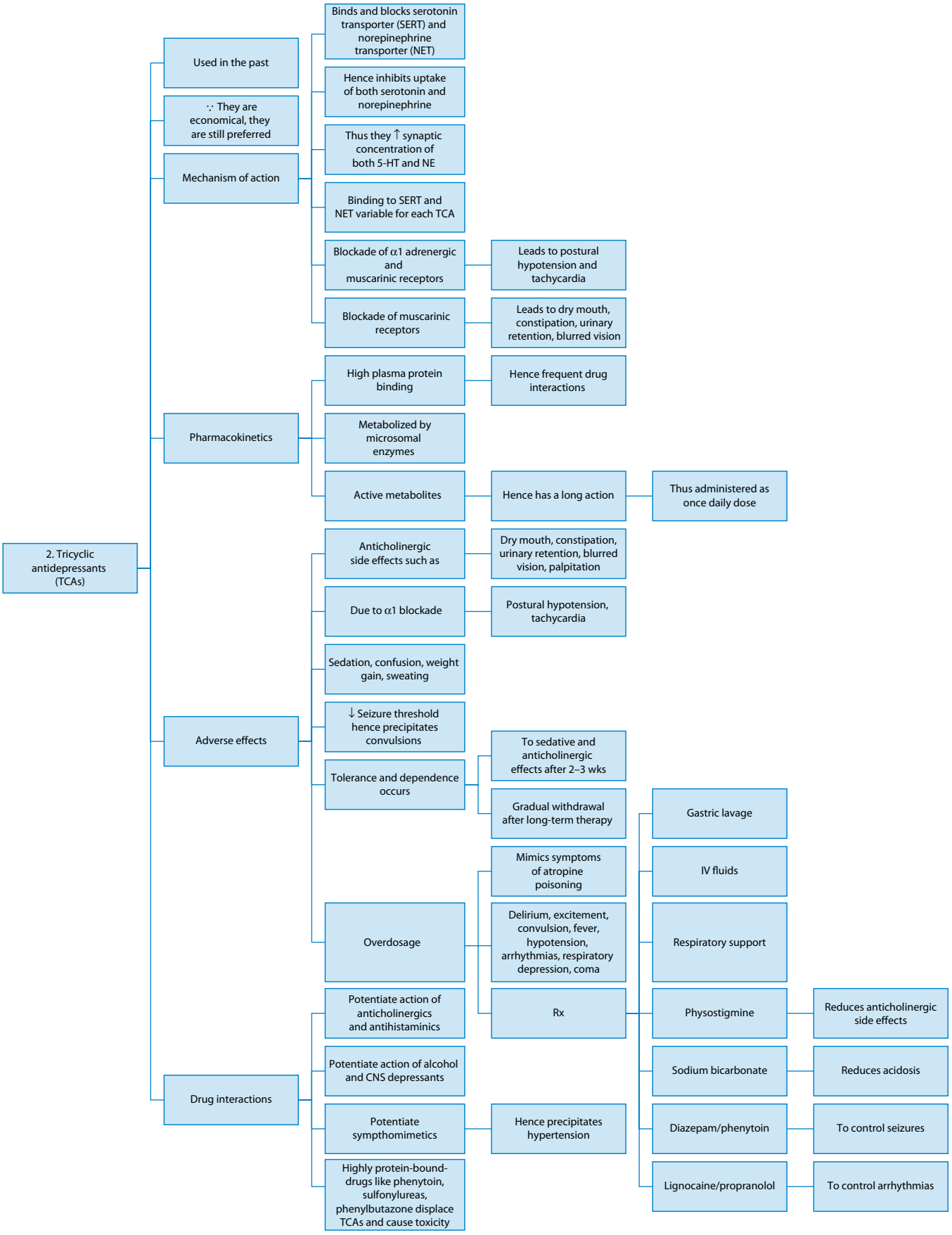
23

Antidepressants

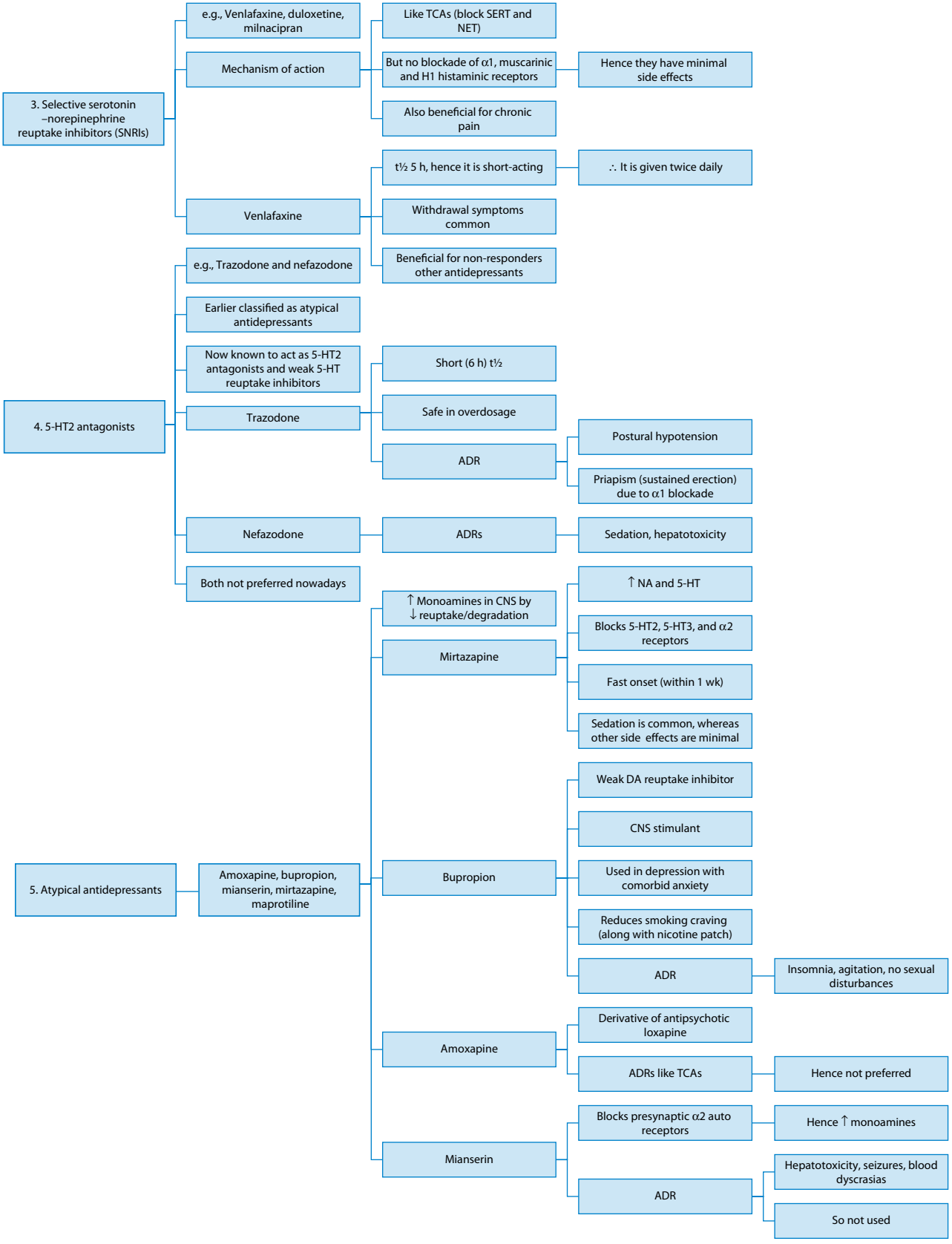
23.1 CLASSIFICATION OF ANTIDEPRESSANTS AND SELECTIVE SEROTONIN REUPTAKE INHIBITORS (SSRIs)



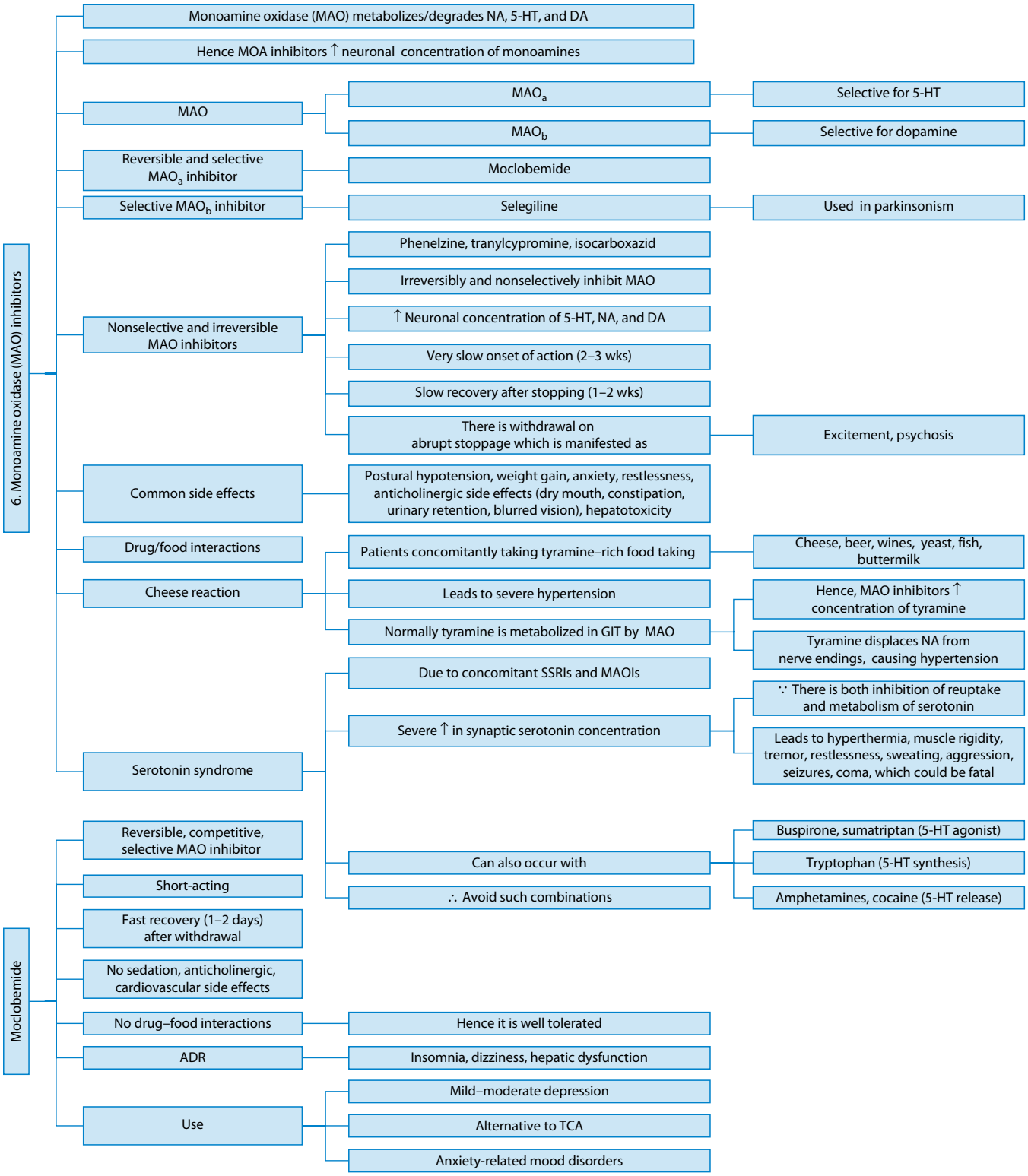
23.2 TRICYCLIC ANTIDEPRESSANTS (TCAs)



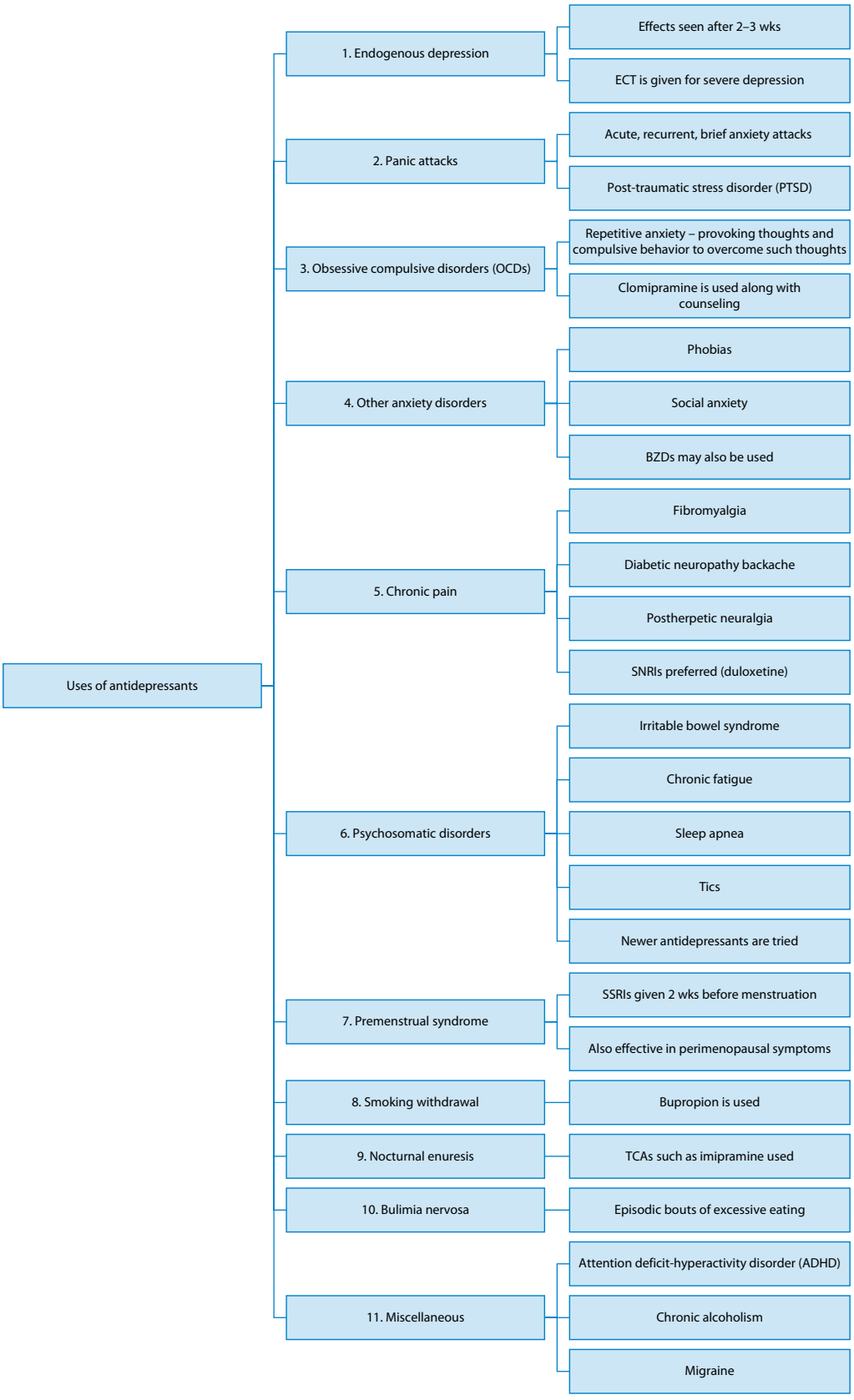
23.3 SELECTIVE SEROTONIN – NOREPINEPHRINE REUPTAKE INHIBITORS (SNRIs), 5-HT2 ANTAGONISTS, AND ATYPICAL ANTIDEPRESSANTS



23.4 MONOAMINE OXIDASE (MAO) INHIBITORS



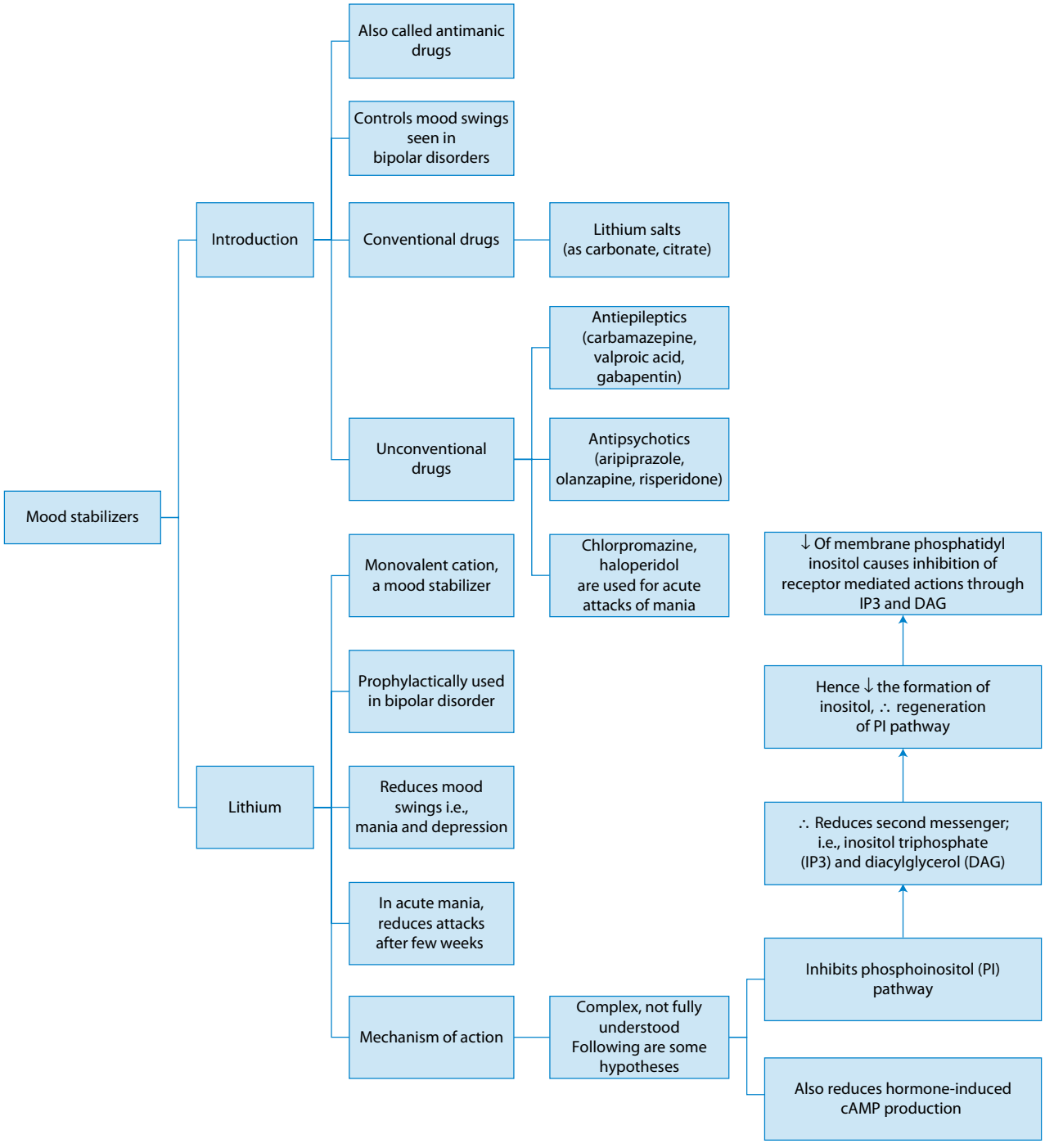
23.5 USES OF ANTIDEPRESSANTS



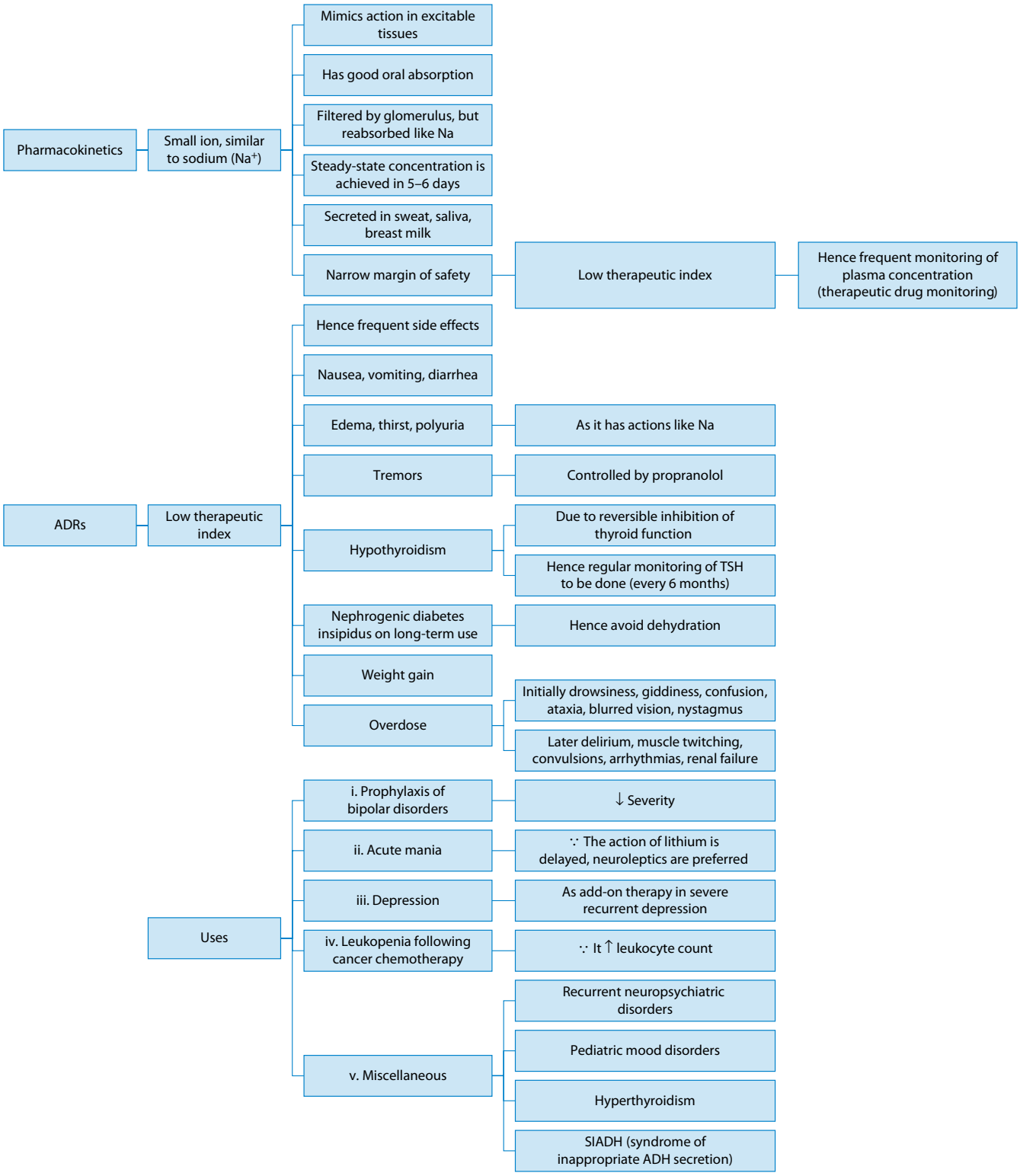
24

Mood stabilizers and lithium

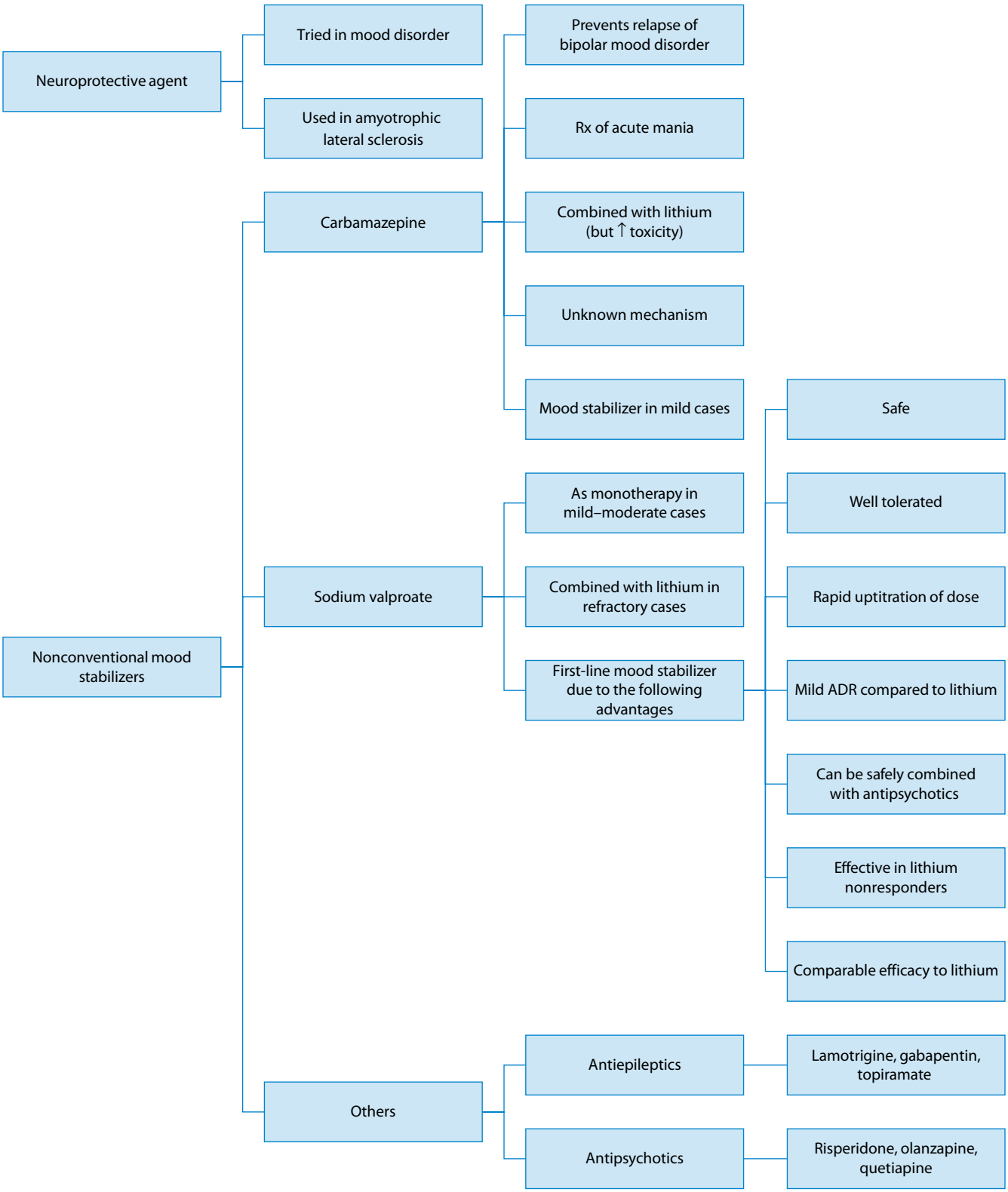
24.1 MOOD STABILIZERS – INTRODUCTION AND LITHIUM



24.2 PHARMACOKINETICS, ADRs, AND USES



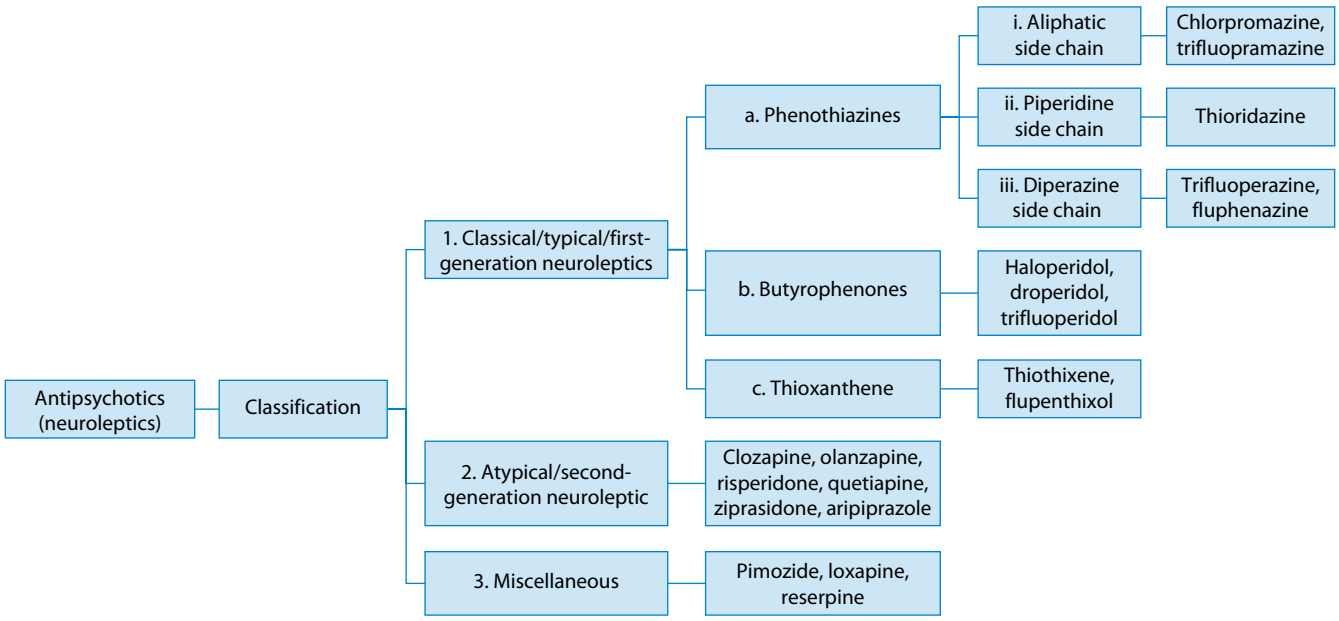
24.3 RILUZOLE AND NONCONVENTIONAL MOOD STABILIZERS



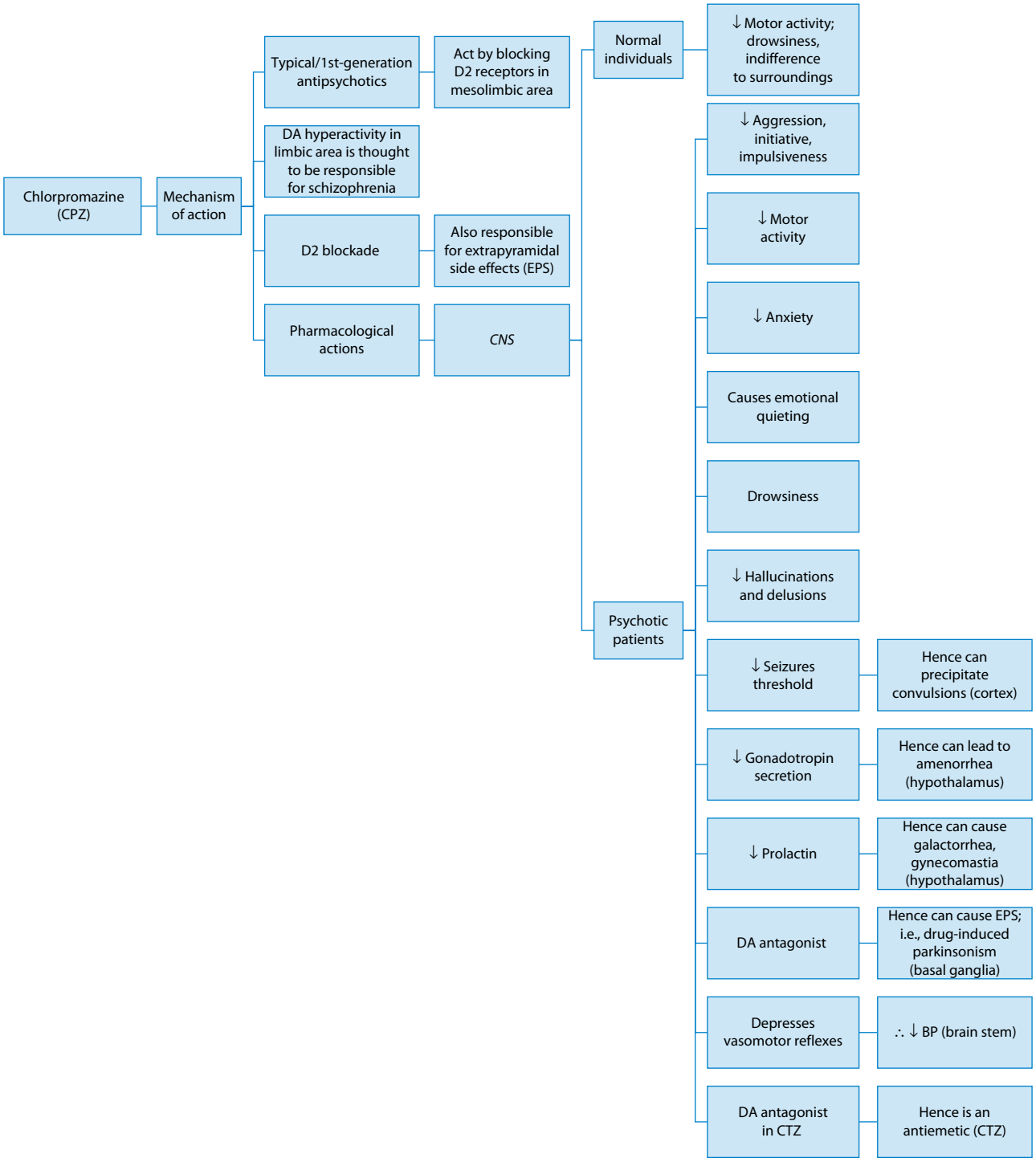
25

Antipsychotics

25.1 ANTIPSYCHOTICS CLASSIFICATION

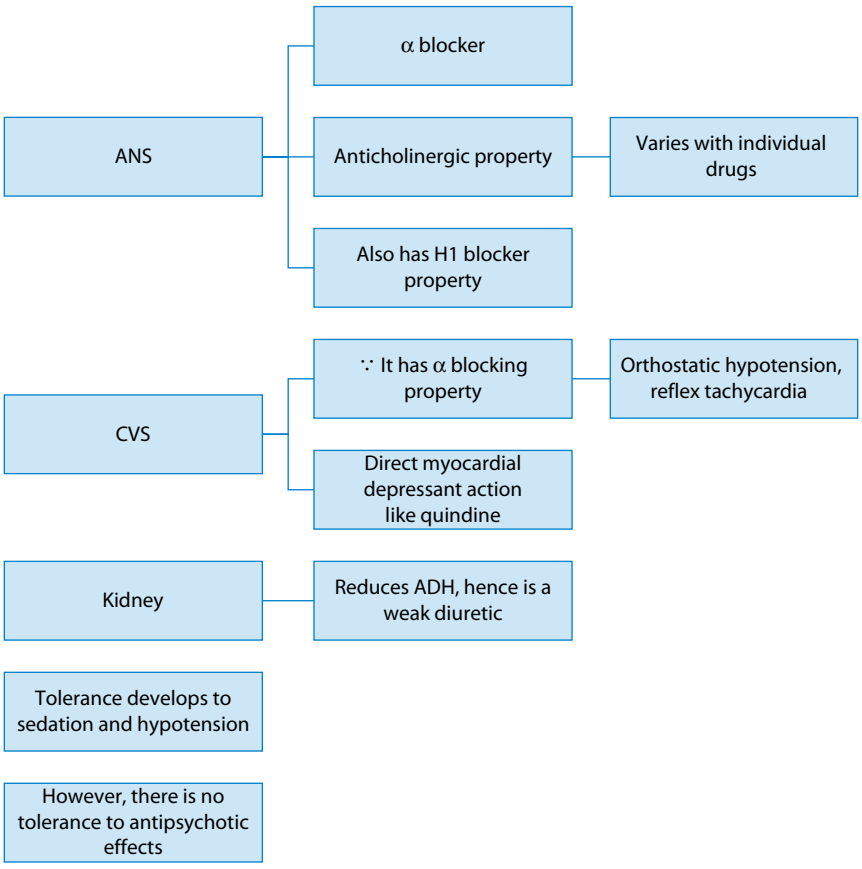


25.2 CHLORPROMAZINE (CPZ) – MECHANISM OF ACTION

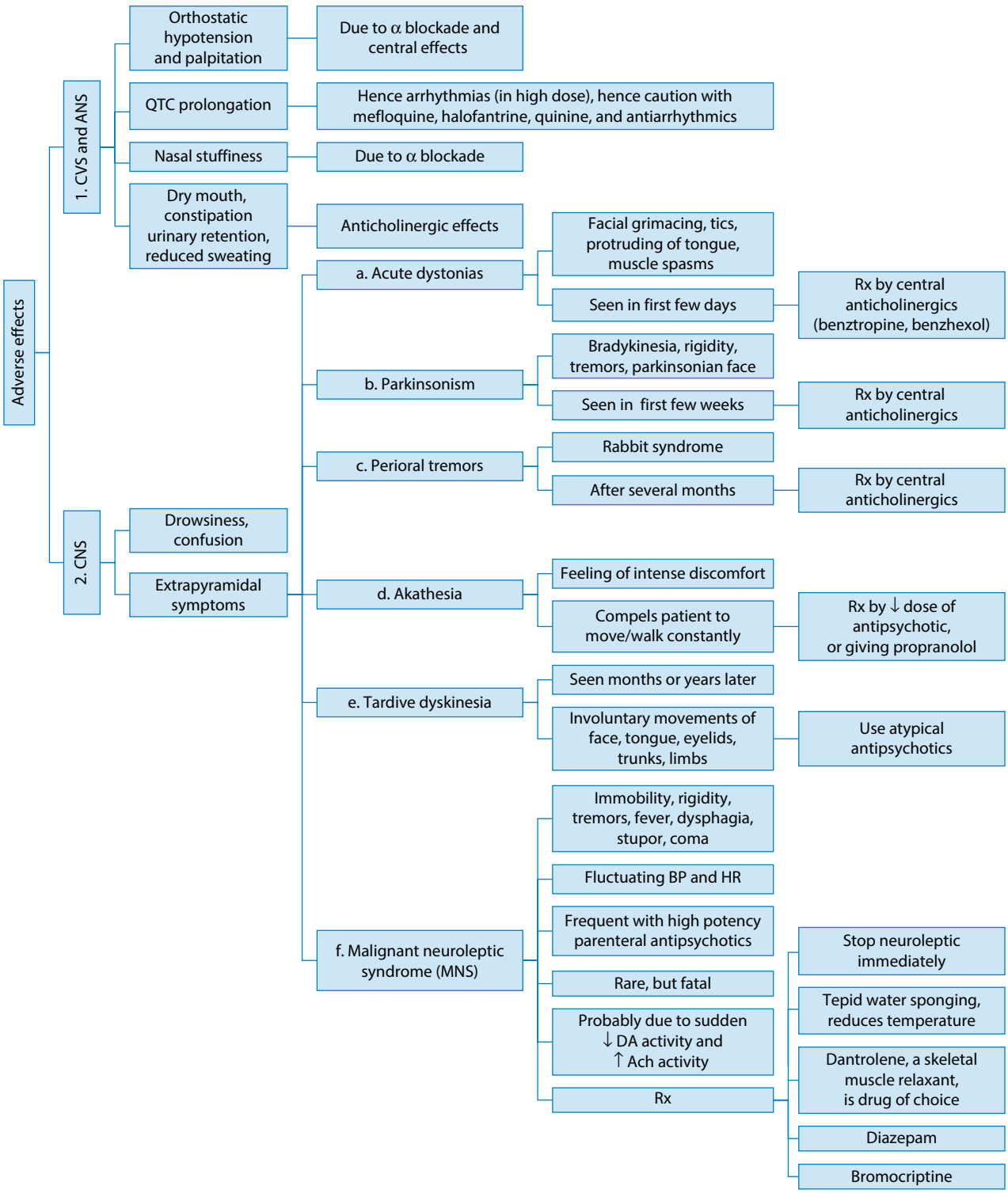


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25.2 CHLORPROMAZINE (CPZ) – PHARMACOLOGICAL ACTIONS (Continued)

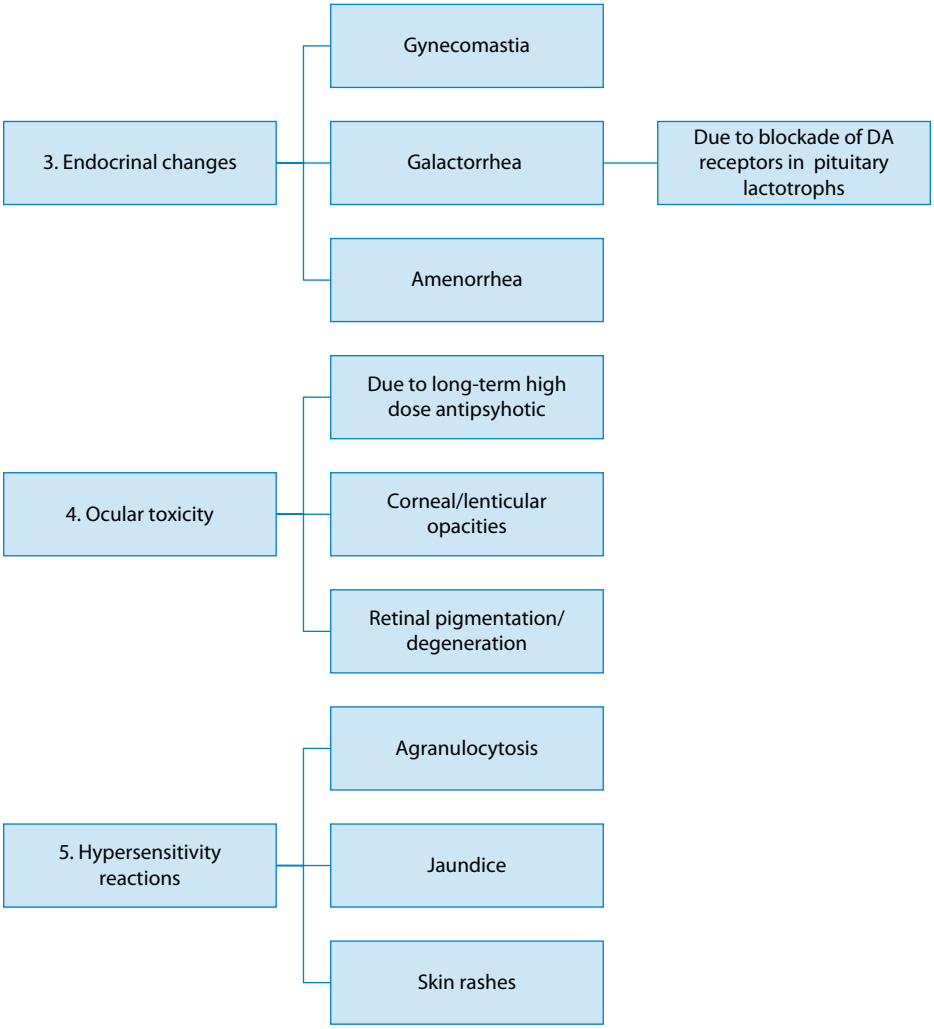


25.3 ADVERSE EFFECTS

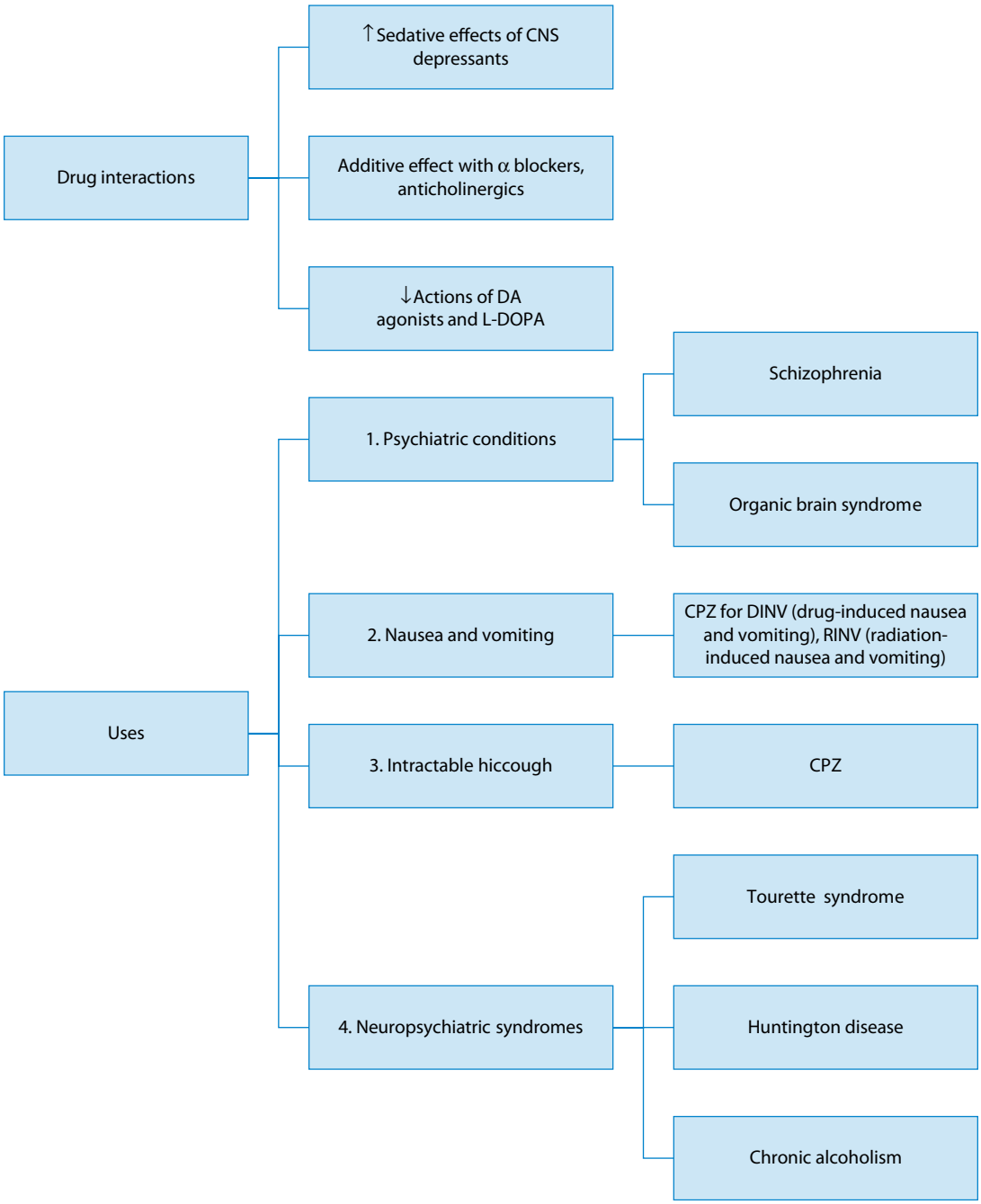


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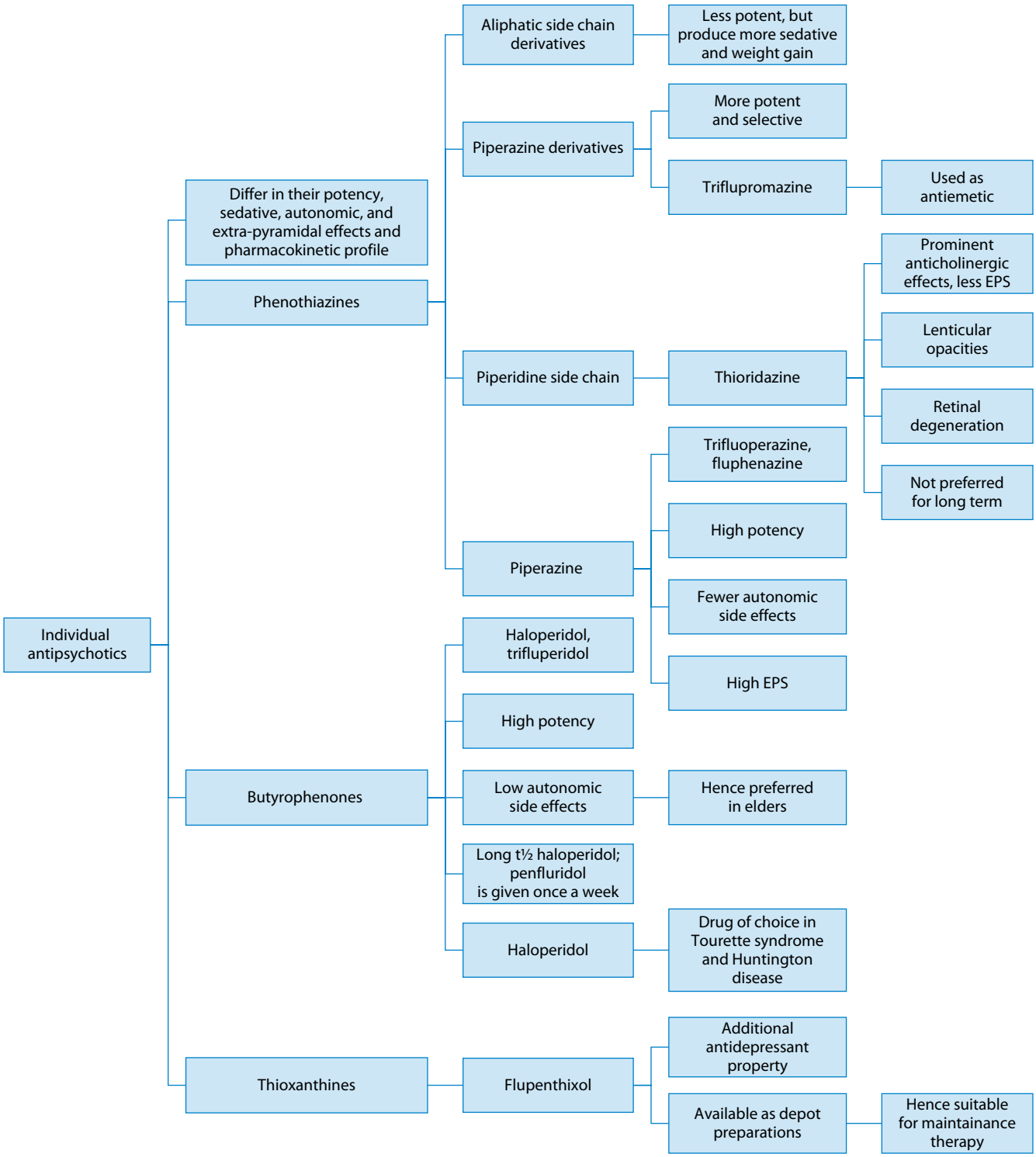
25.3 ADVERSE EFFECTS (Continued)



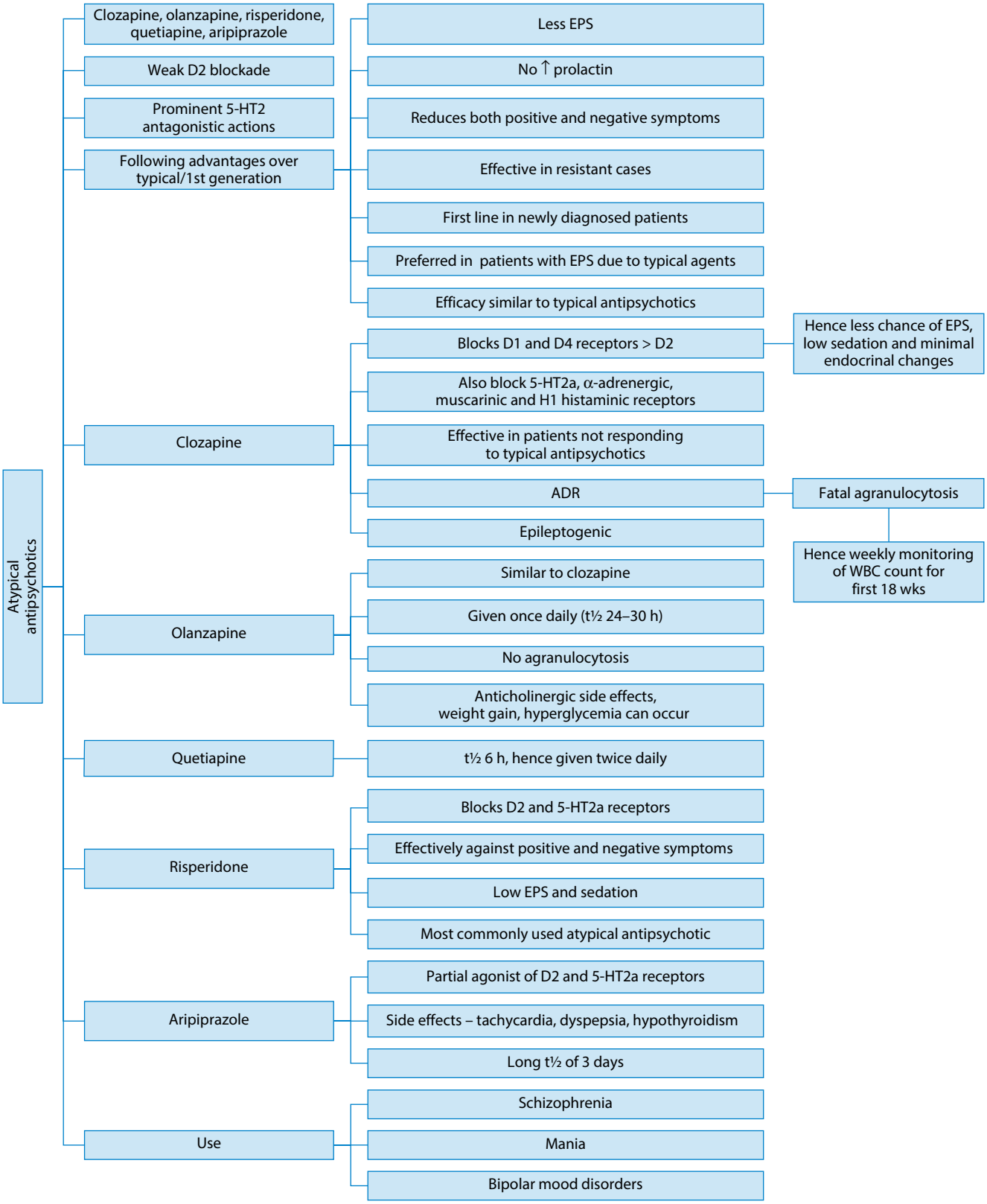
25.4 DRUG INTERACTIONS AND USES



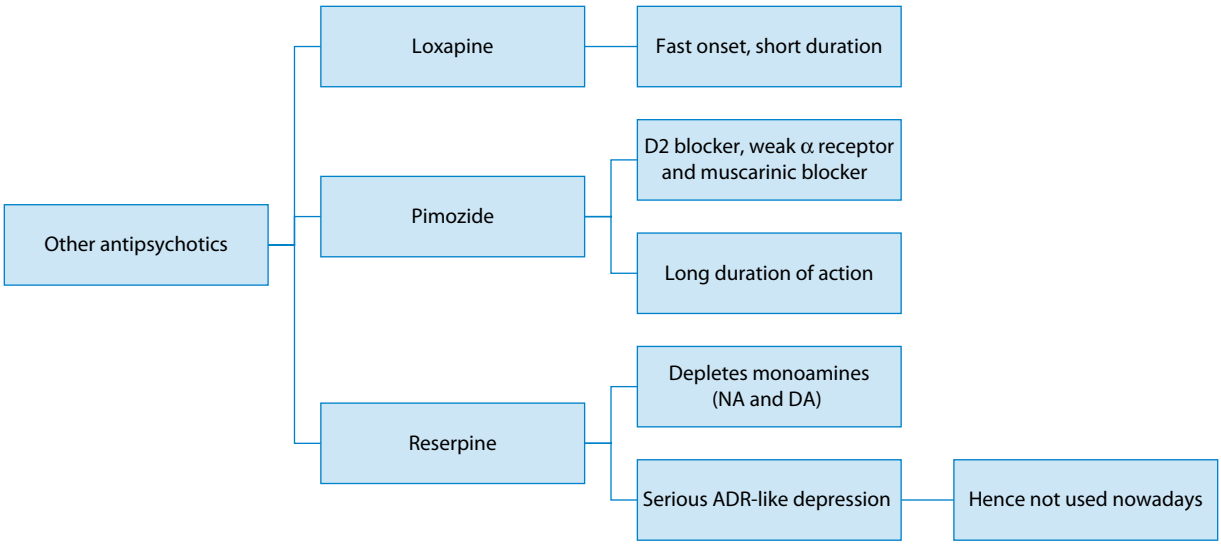
25.5 INDIVIDUAL ANTIPSYCHOTICS



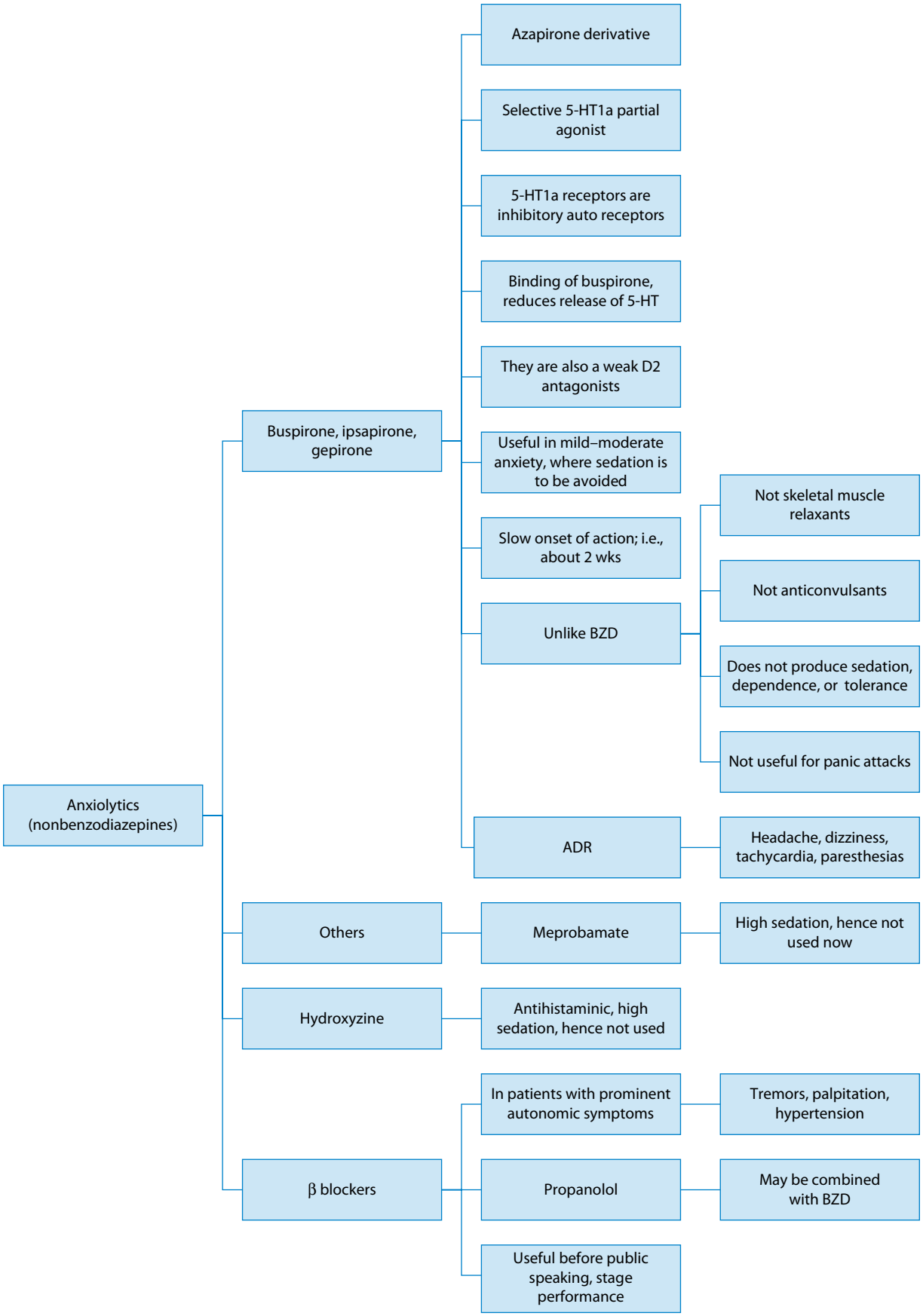
25.6 ATYPICAL ANTIPSYCHOTICS



25.7 OTHER ANTIPSYCHOTICS



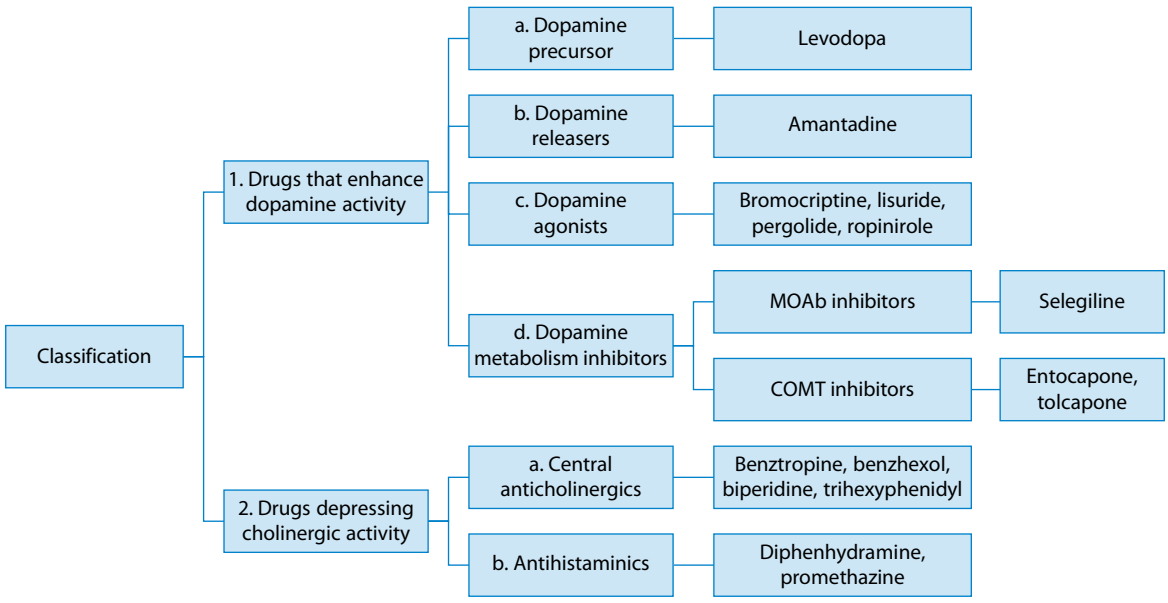
25.8 ANXIOLYTICS (NONBENZODIAZEPINES)



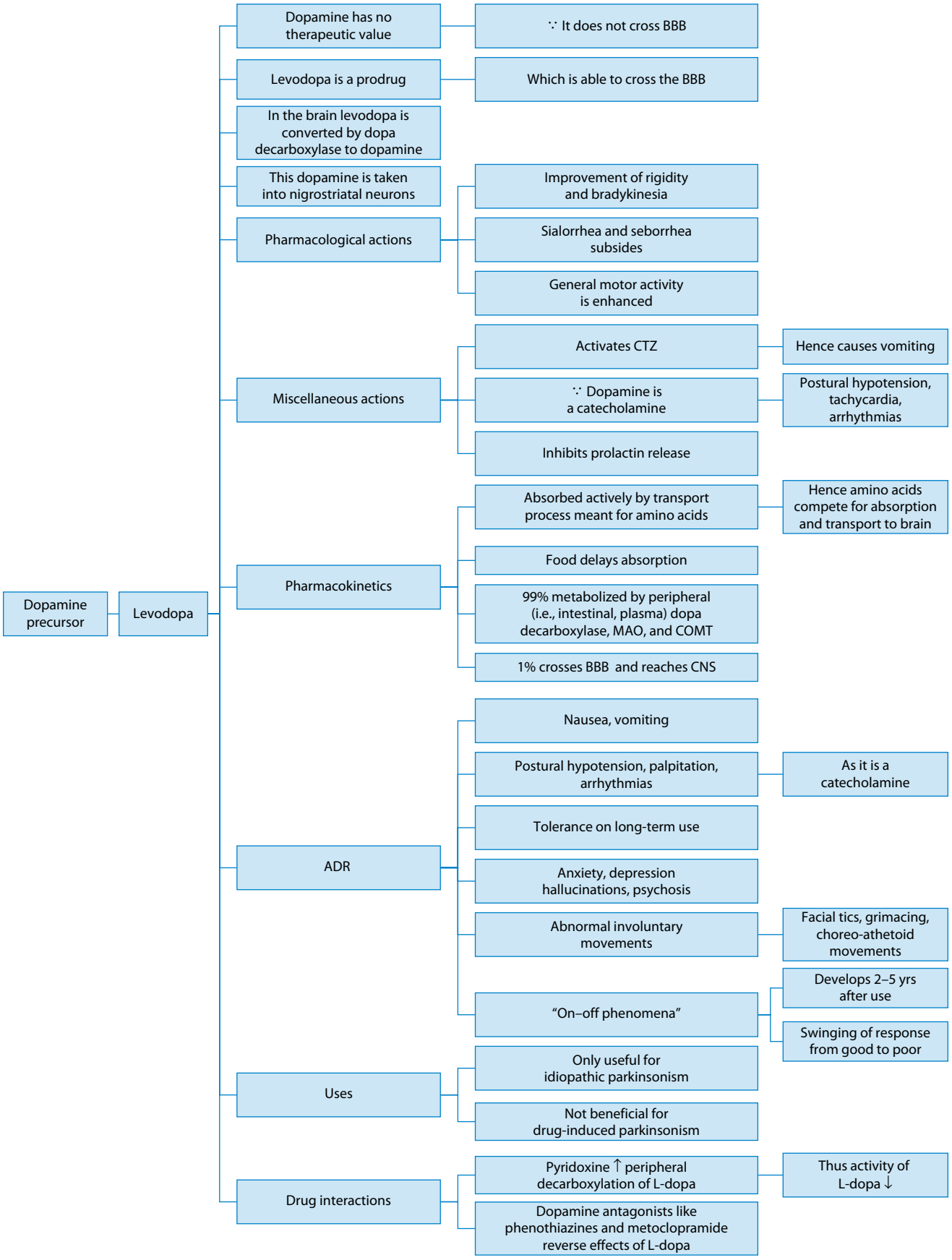
26

Drug treatment of Parkinsonism and Alzheimer's disease

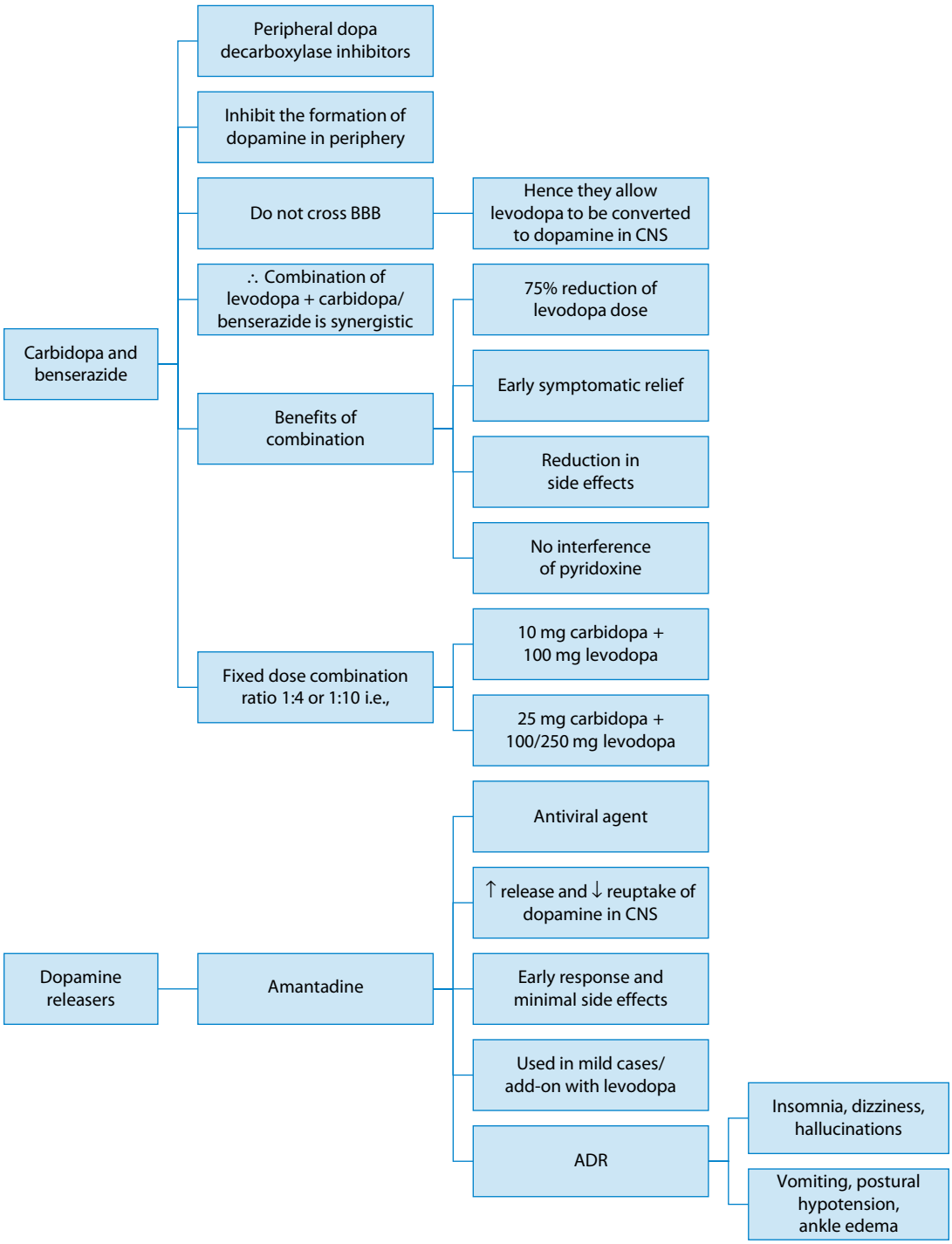
26.1 CLASSIFICATION OF ANTIPARKINSONIAN DRUGS



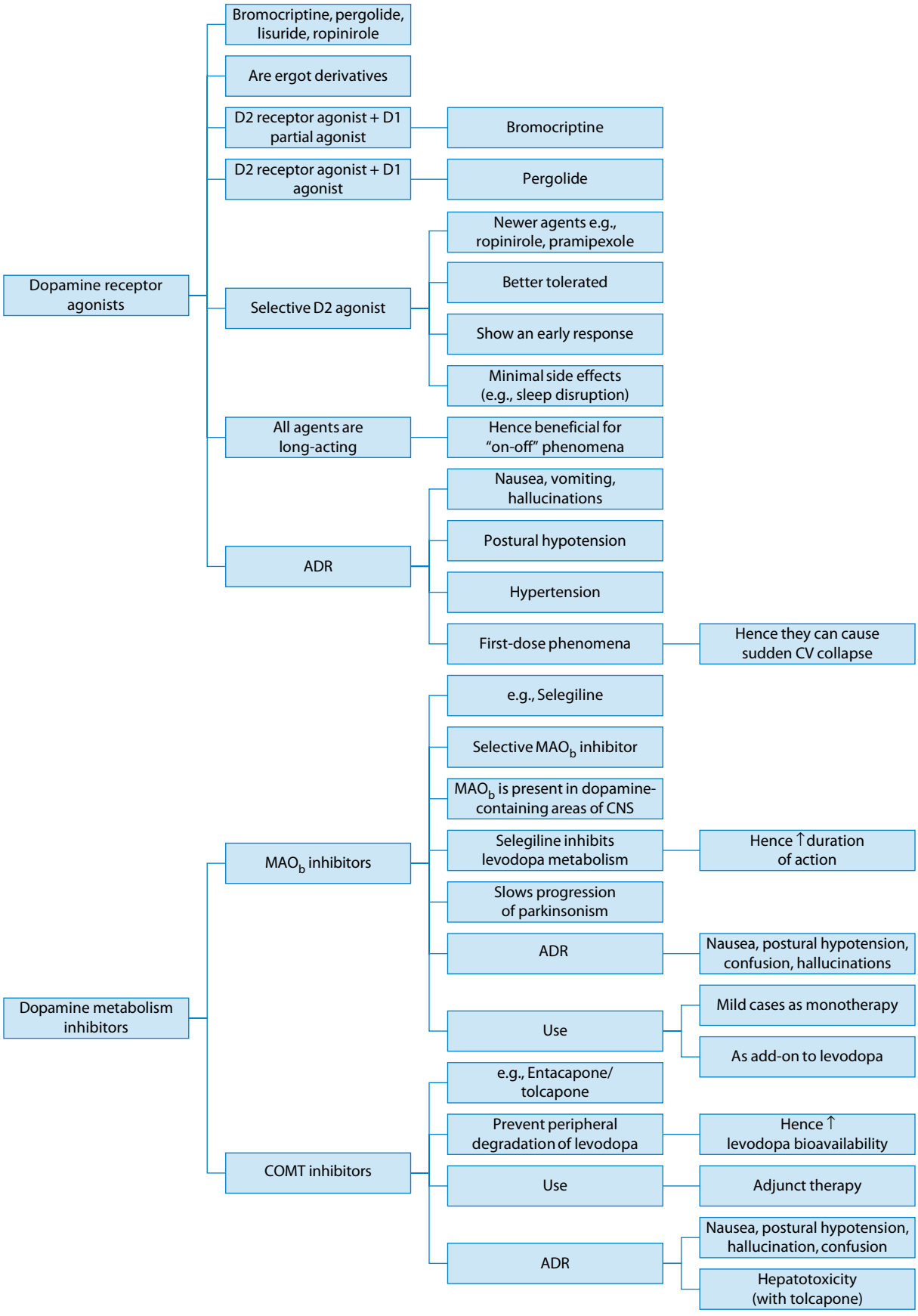
26.2 DOPAMINE PRECURSOR – LEVODOPA



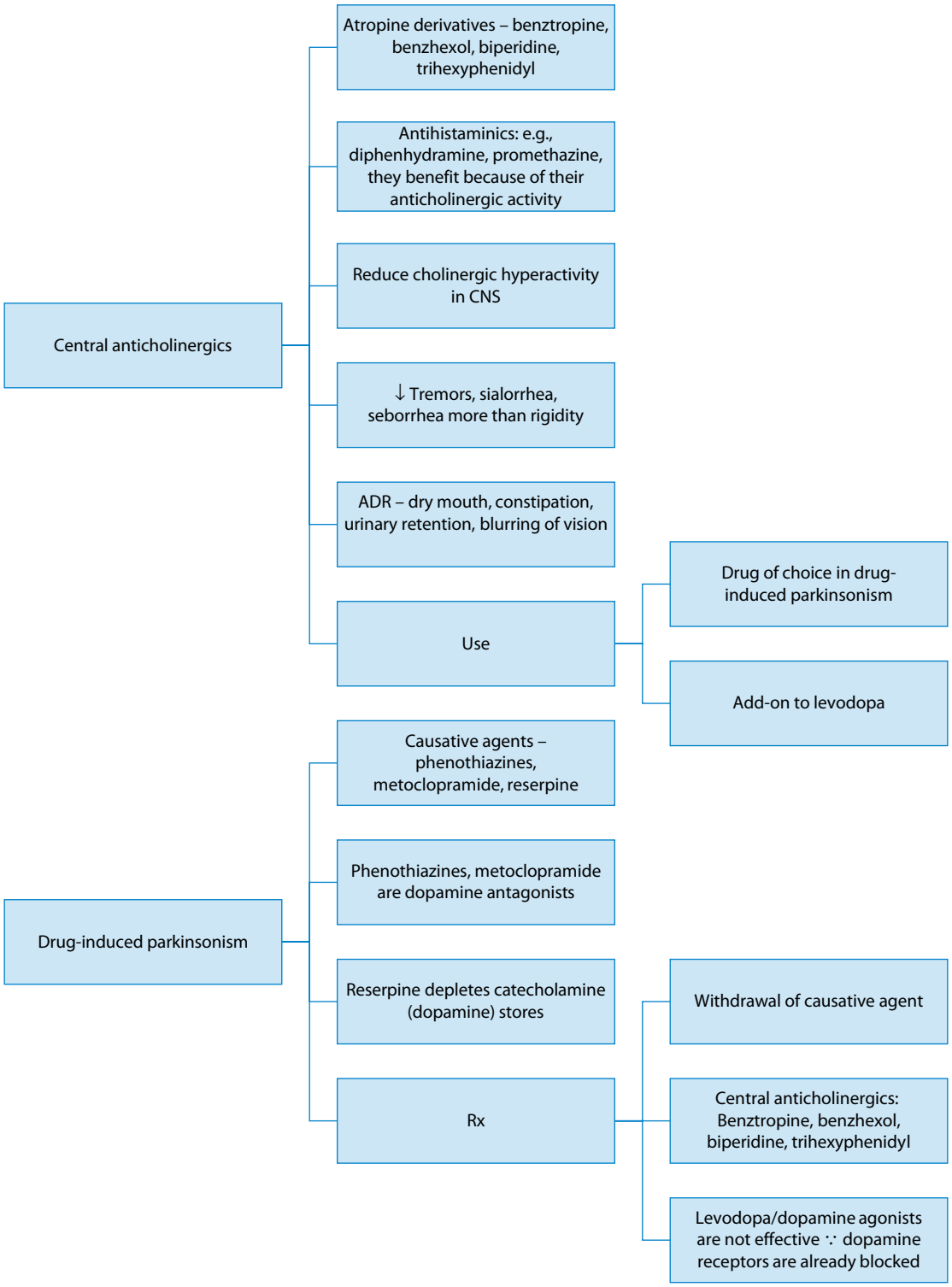
26.3 CARBIDOPA AND BENSERAZIDE, DOPAMINE RELEASERS



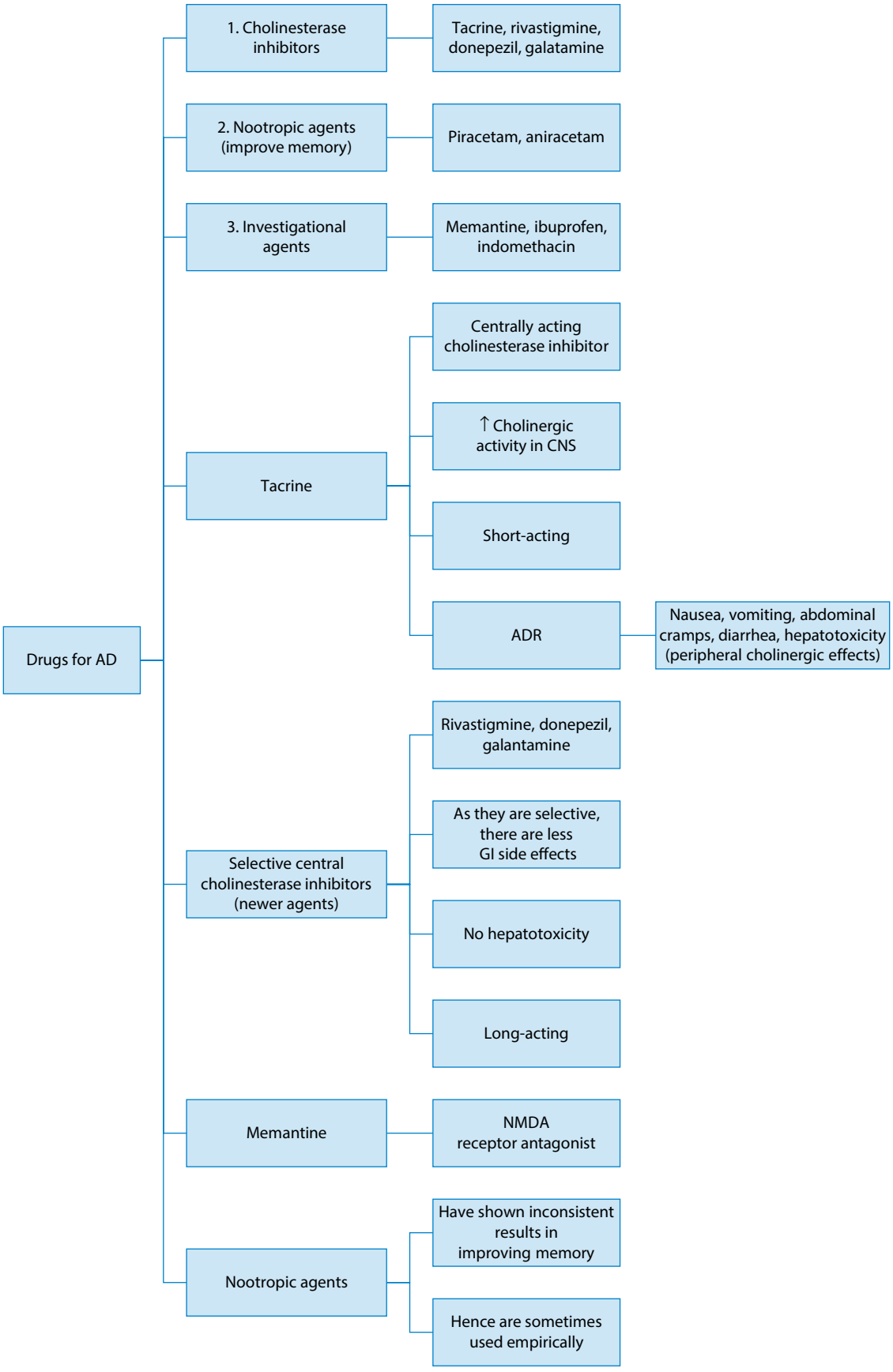
26.4 DOPAMINE RECEPTOR AGONISTS AND DOPAMINE METABOLISM INHIBITORS



26.5 CENTRAL ANTICHOLINERGICS AND DRUG-INDUCED PARKINSONISM



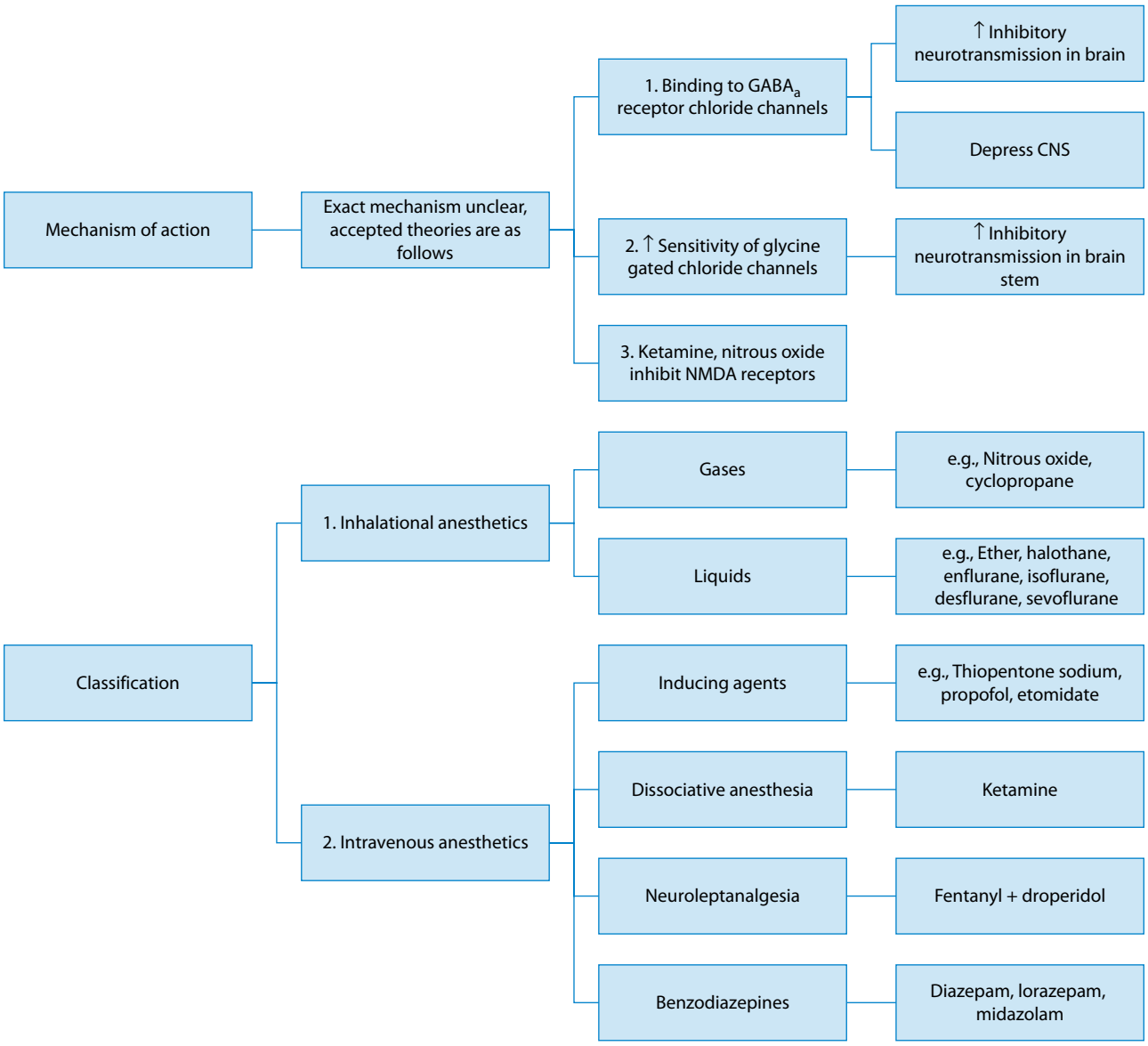
26.6 DRUGS FOR ALZHEIMER'S DISEASE (AD)



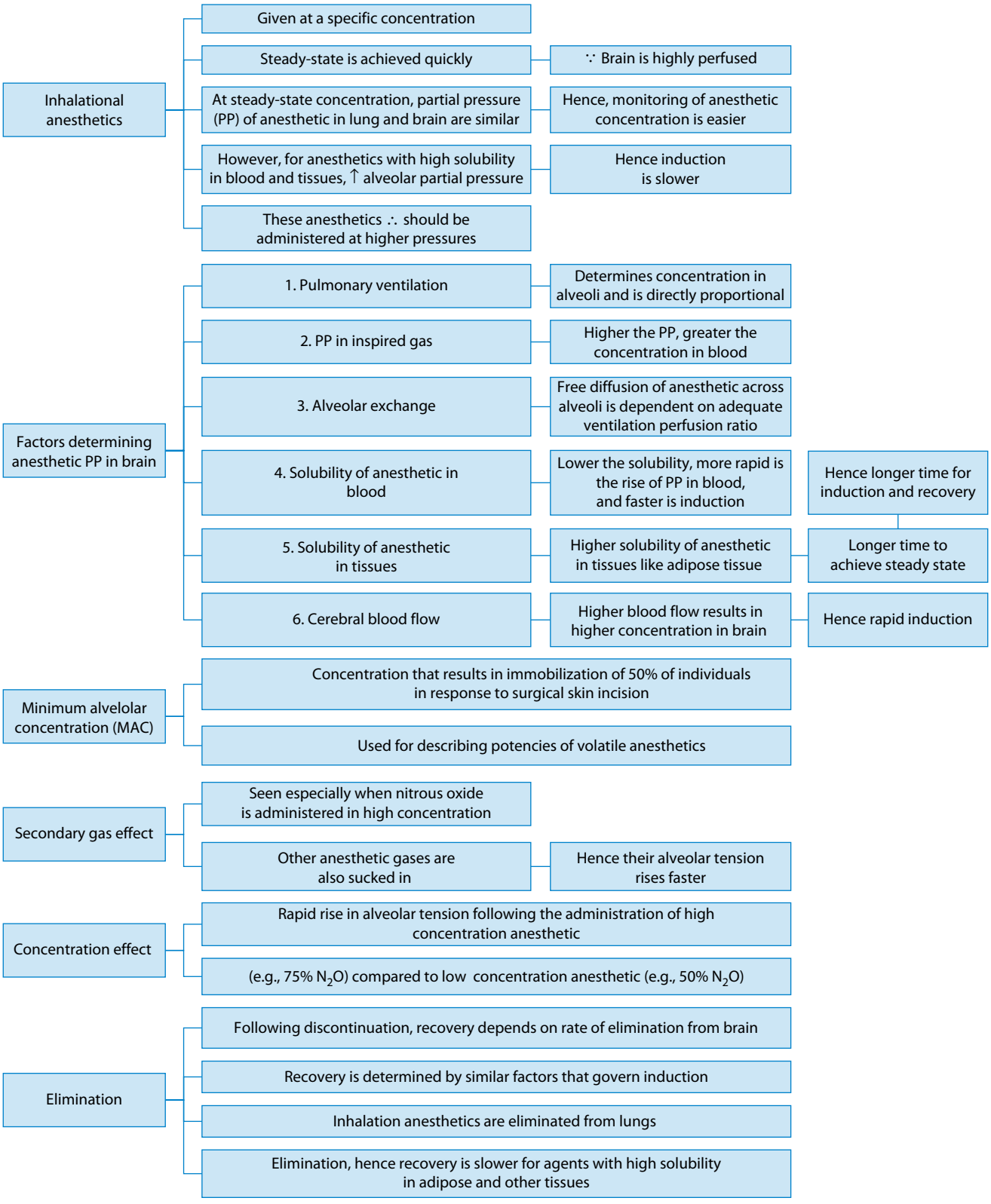
27

General anesthetics (GA)

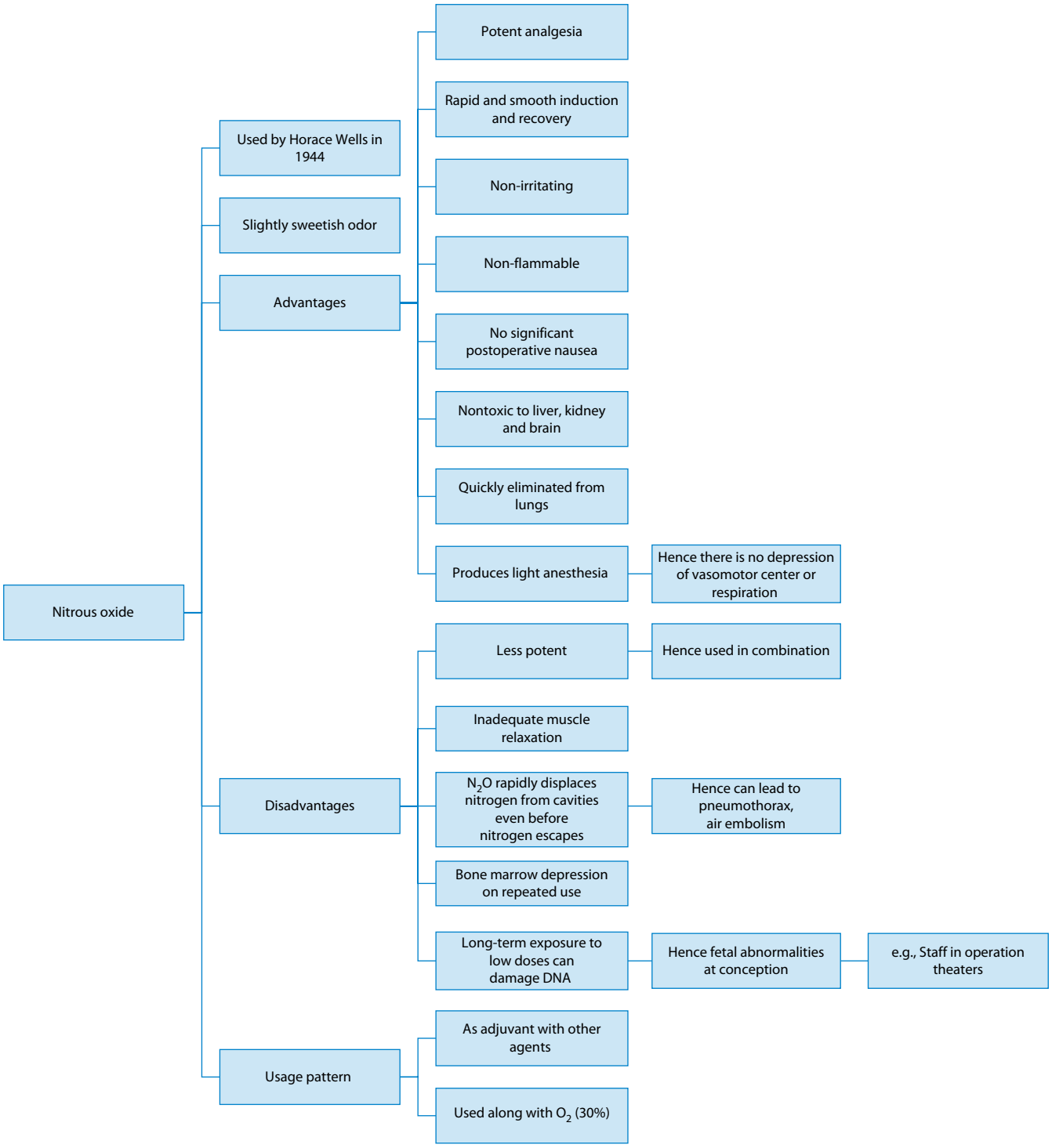
27.1 MECHANISM OF ACTION AND CLASSIFICATION



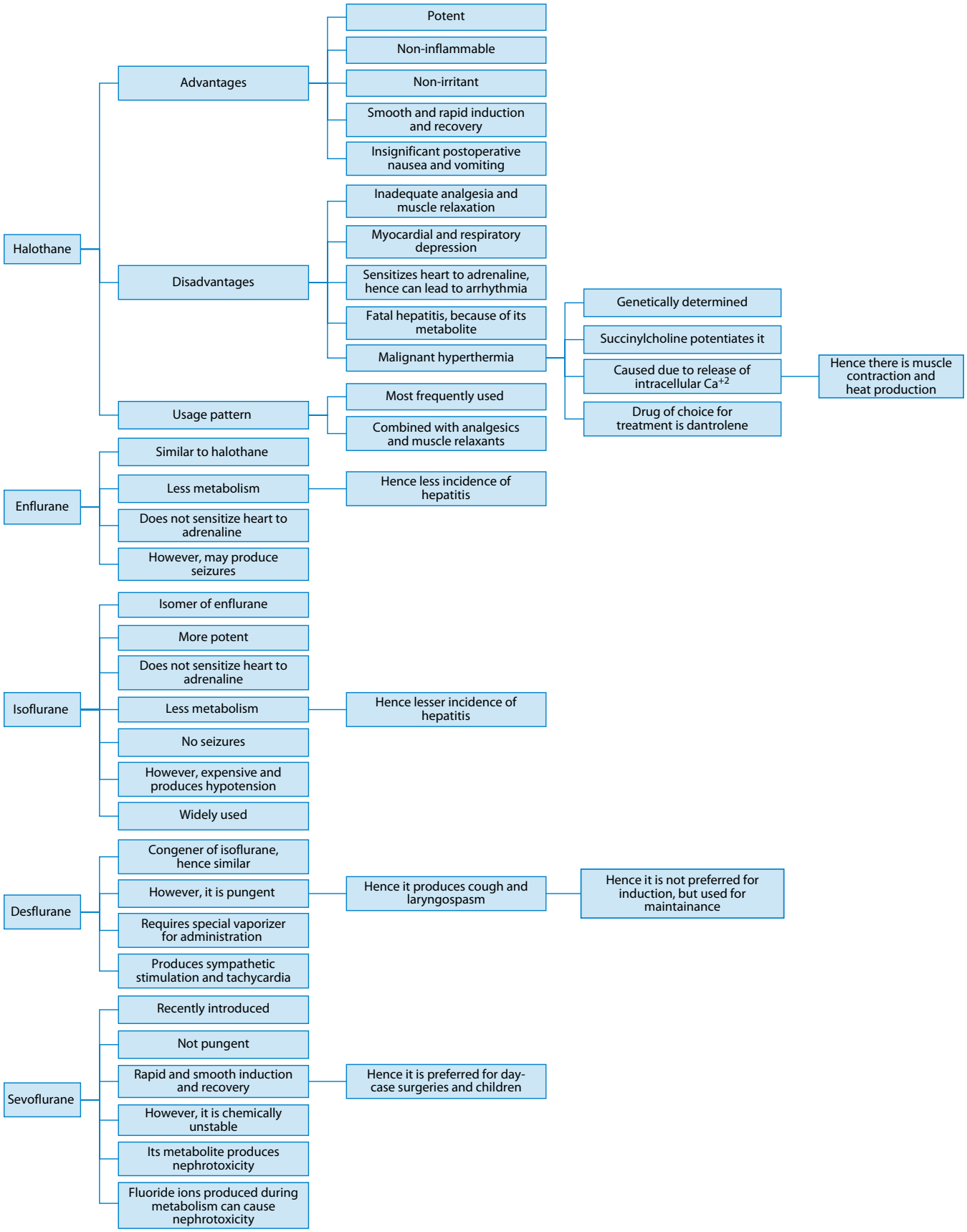
27.2 INHALATIONAL ANESTHETICS AND FACTORS DETERMINING ANESTHETIC PP IN BRAIN



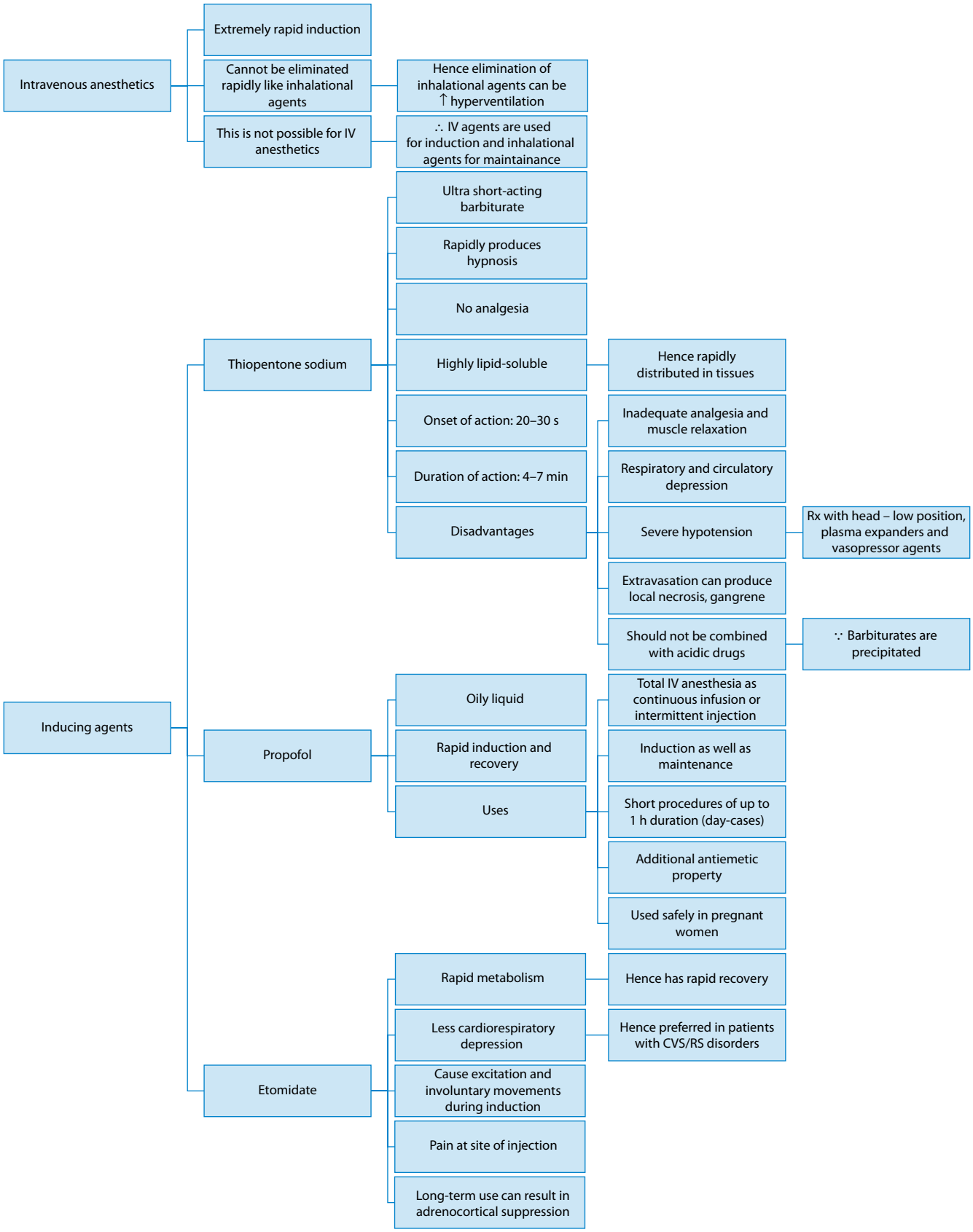
27.3 NITROUS OXIDE



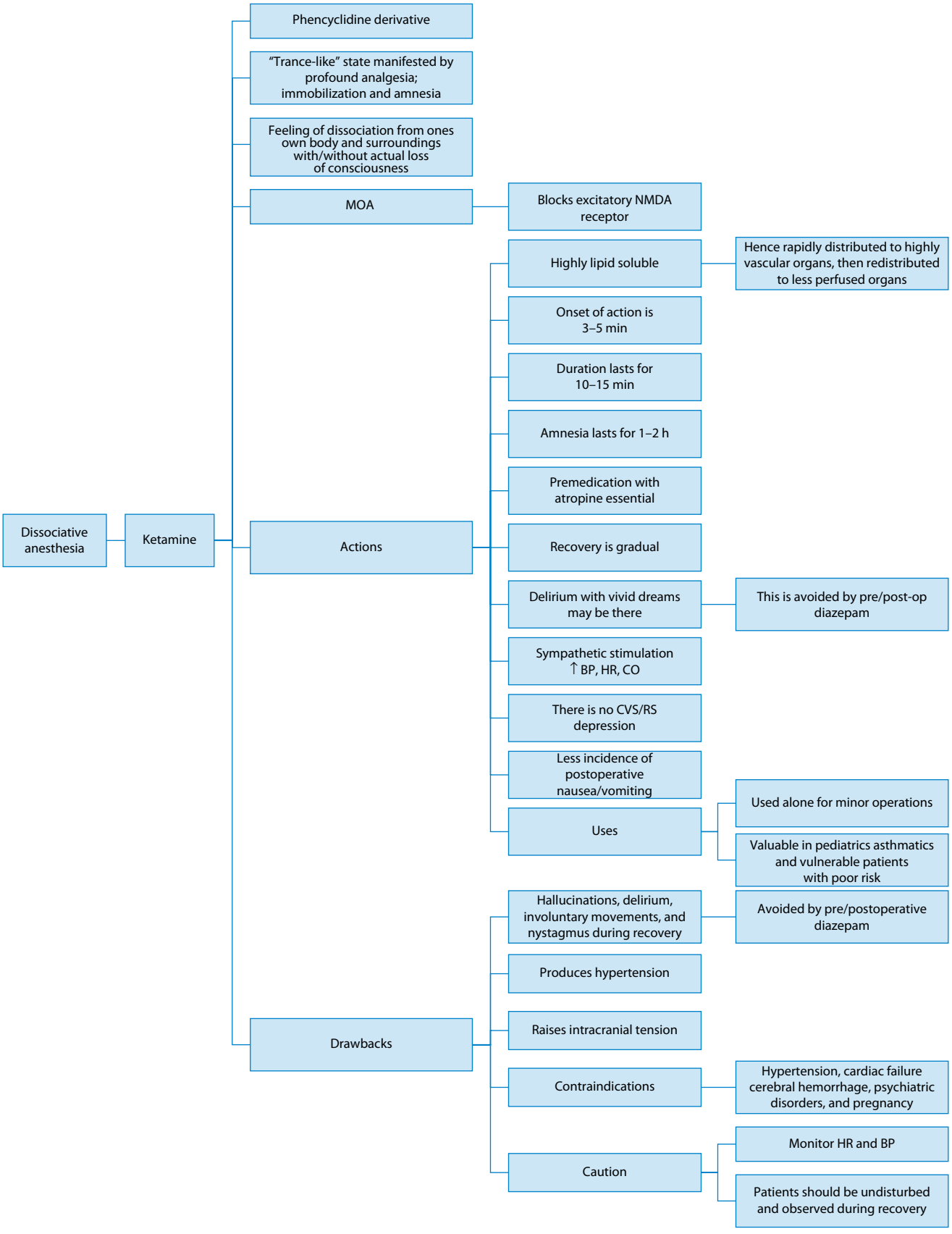
27.4 HALOTHANE AND CONGENERS



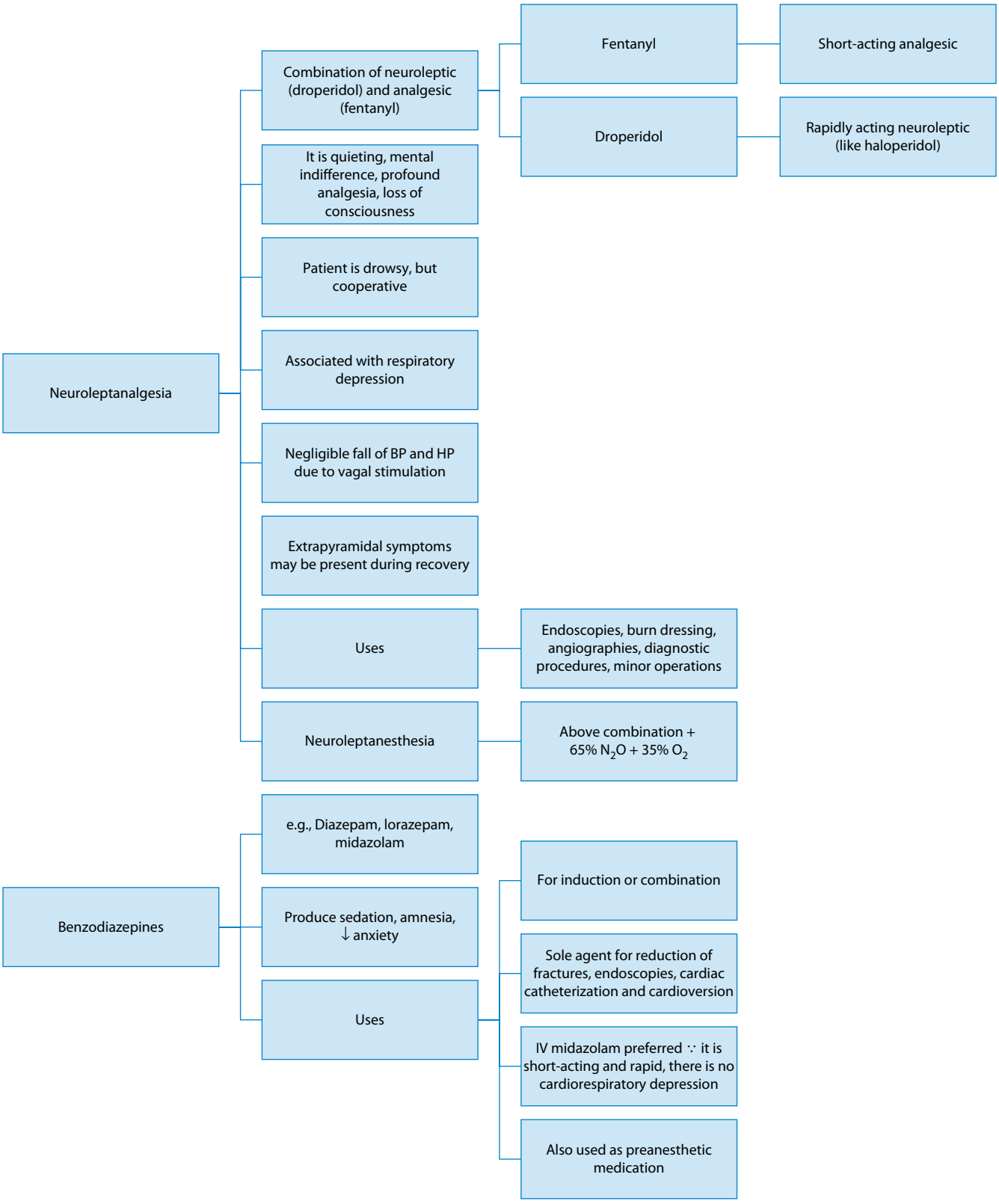
27.5 INTRAVENOUS ANESTHETICS AND INDUCING AGENTS



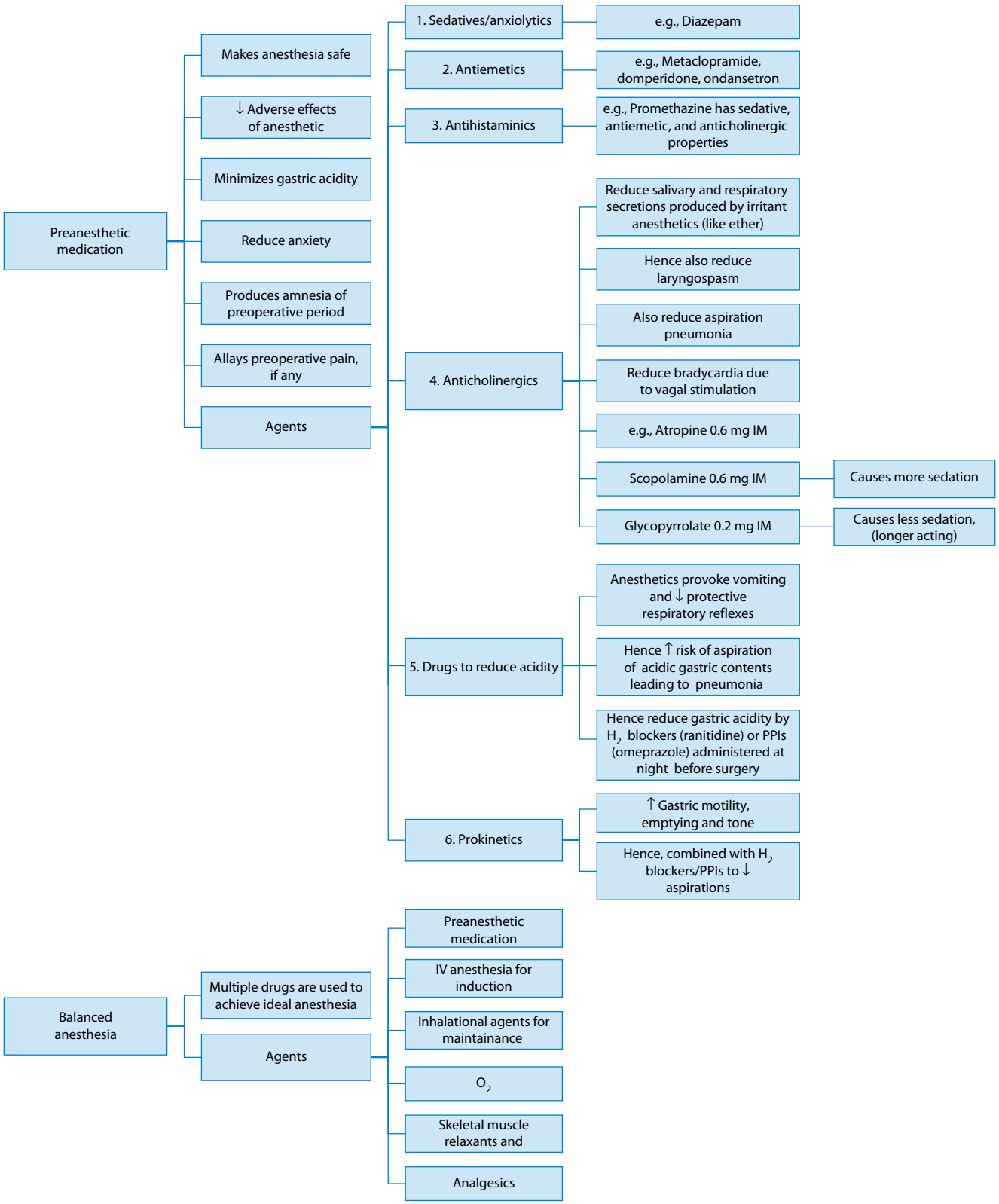
27.6 DISSOCIATIVE ANESTHESIA (KETAMINE)



27.7 NEUROLEPTANALGESIA AND BENZODIAZEPINES



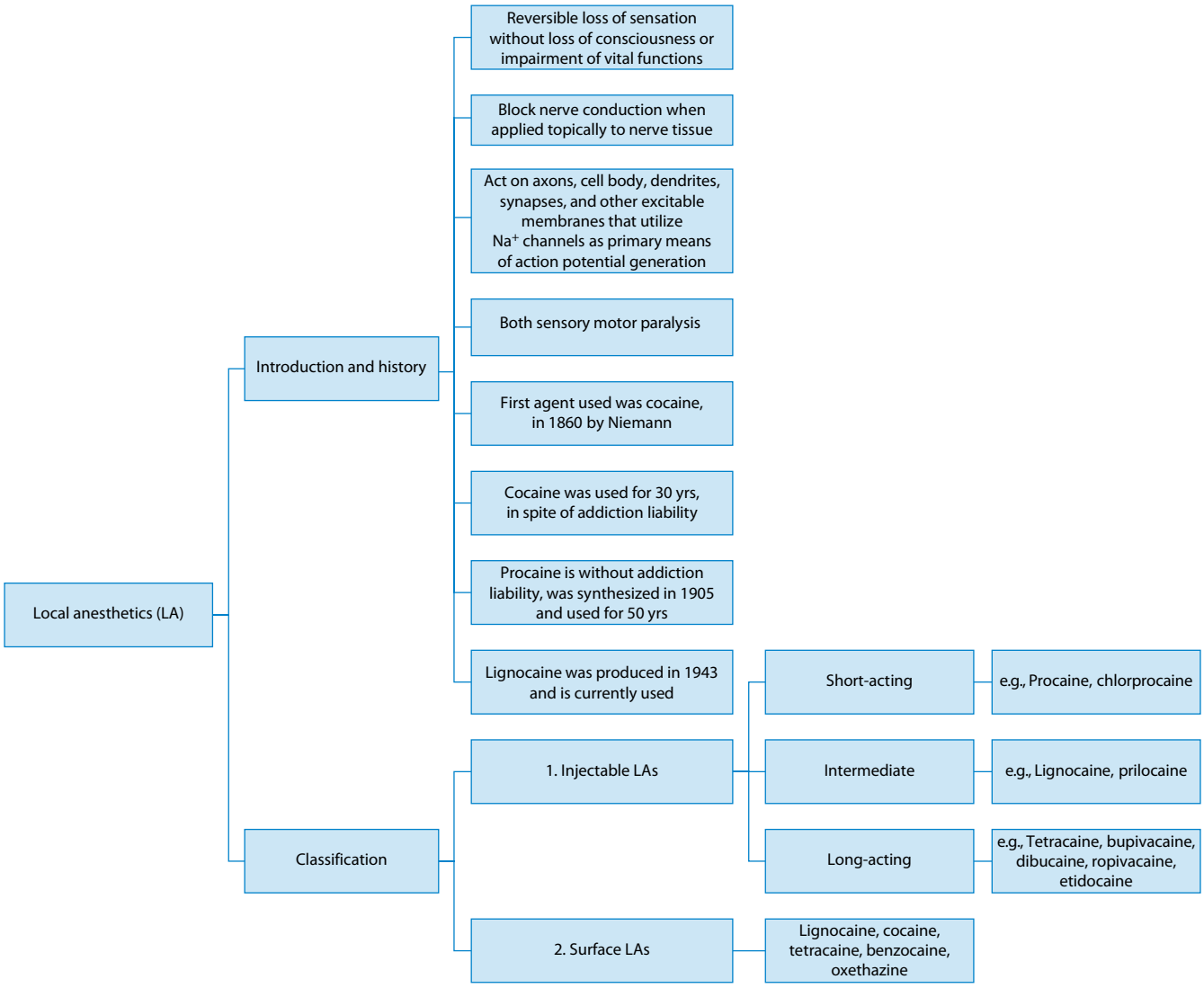
27.8 PREANESTHETIC MEDICATION AND BALANCED ANESTHESIA



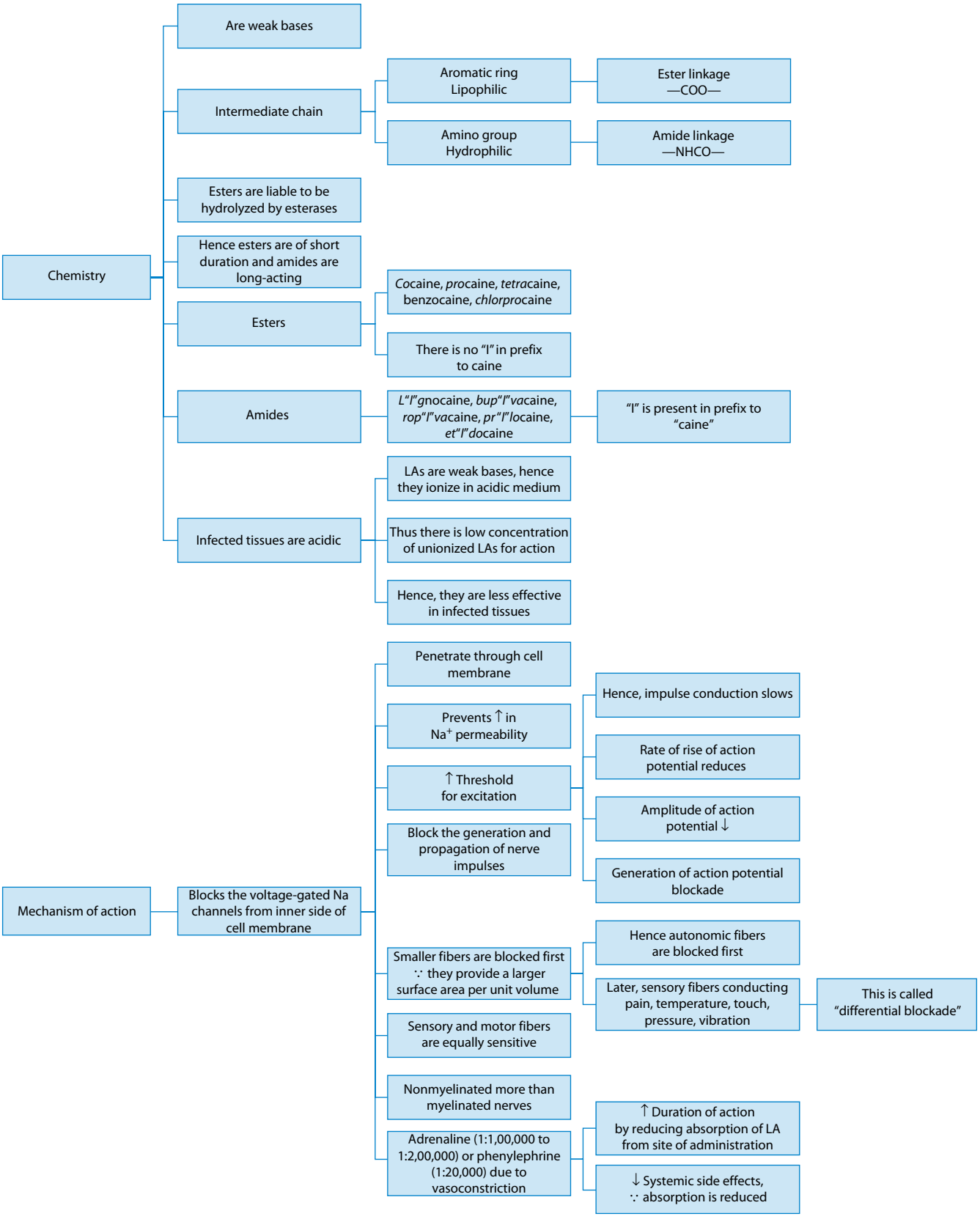
28

Local anesthetics (LA)

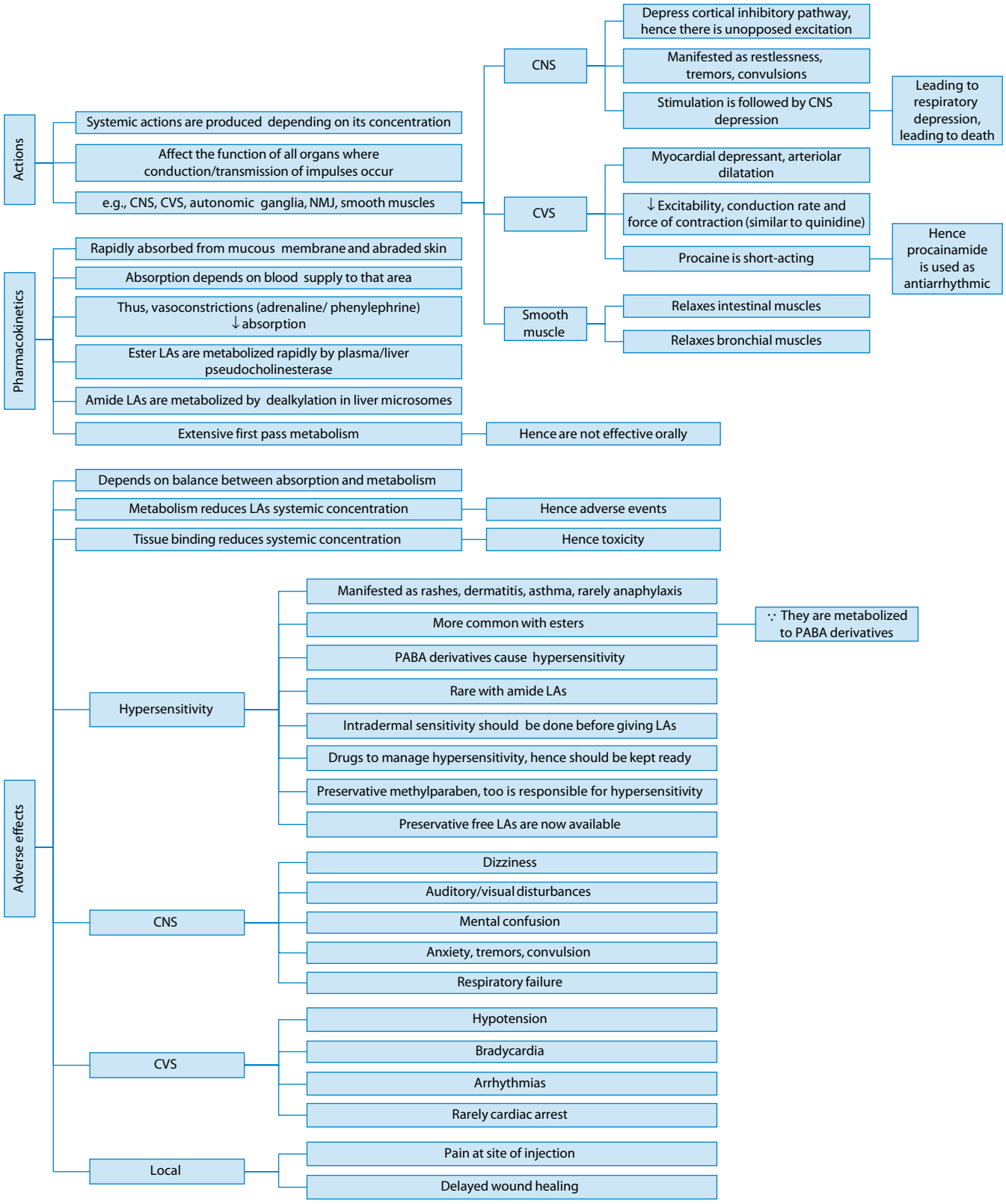
28.1 LOCAL ANESTHETICS (LA) – INTRODUCTION, HISTORY, AND CLASSIFICATION



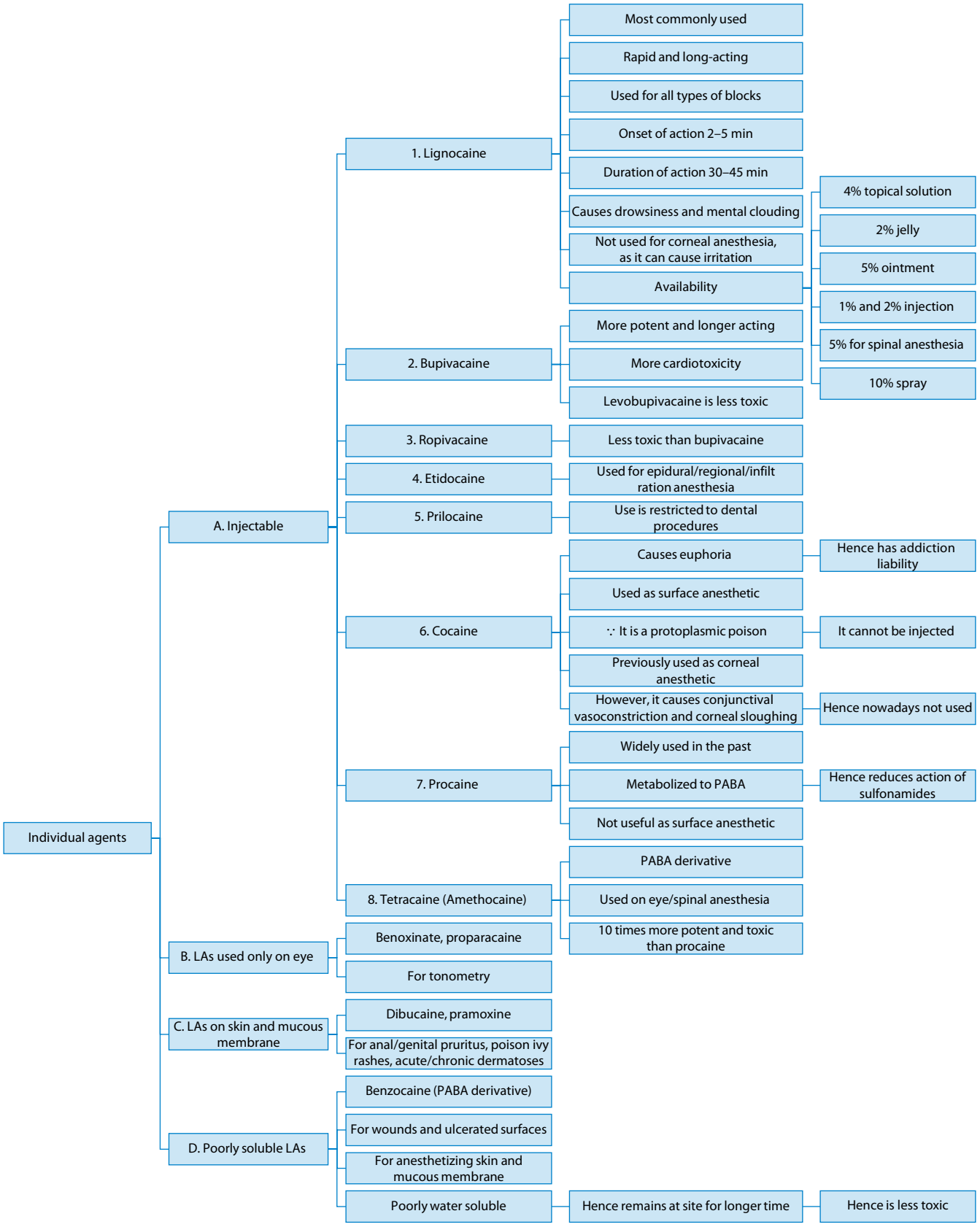
28.2 CHEMISTRY AND MECHANISM OF ACTION



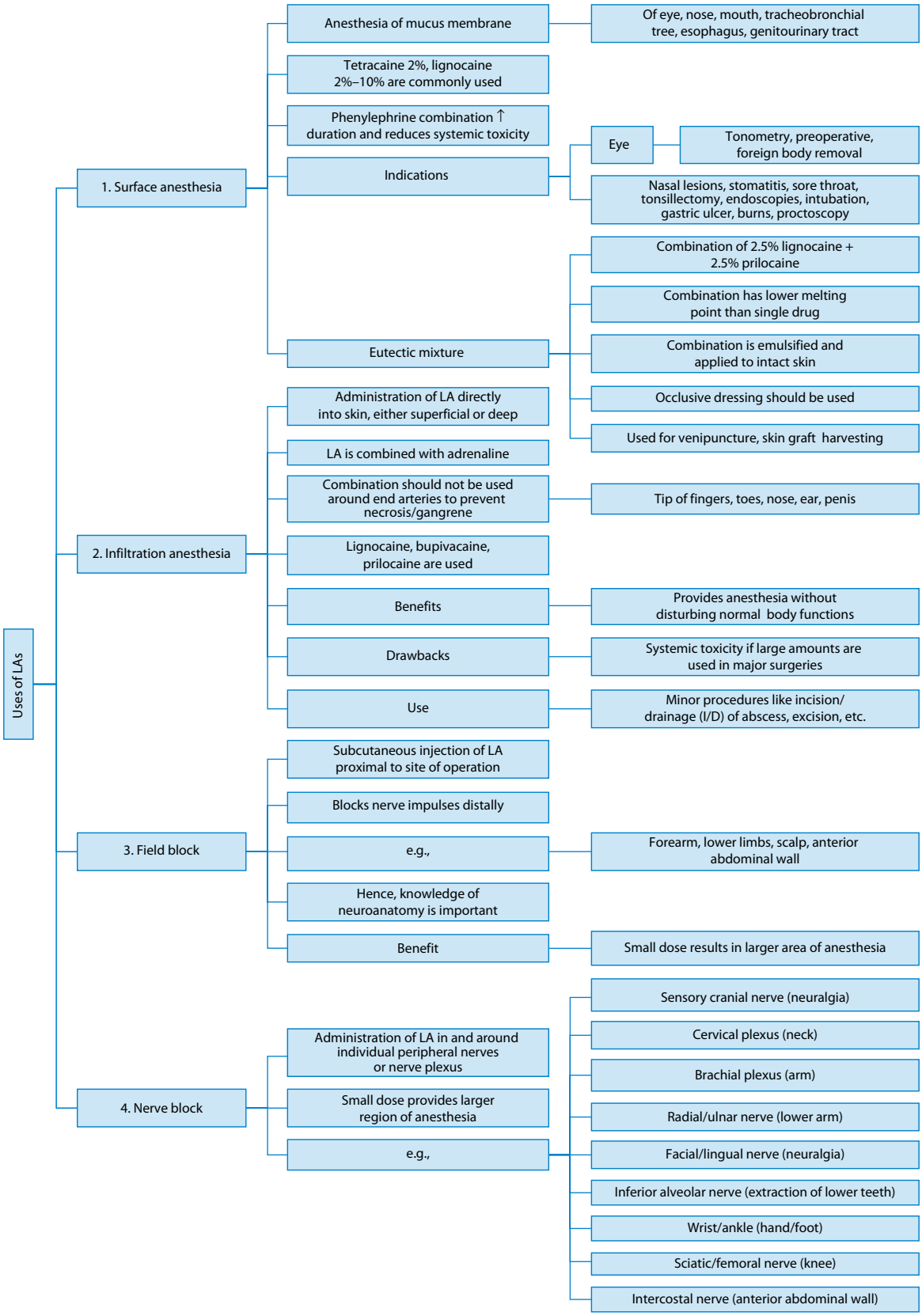
28.3 ACTIONS, PHARMACOKINETICS, AND ADRs



28.4 INDIVIDUAL AGENTS

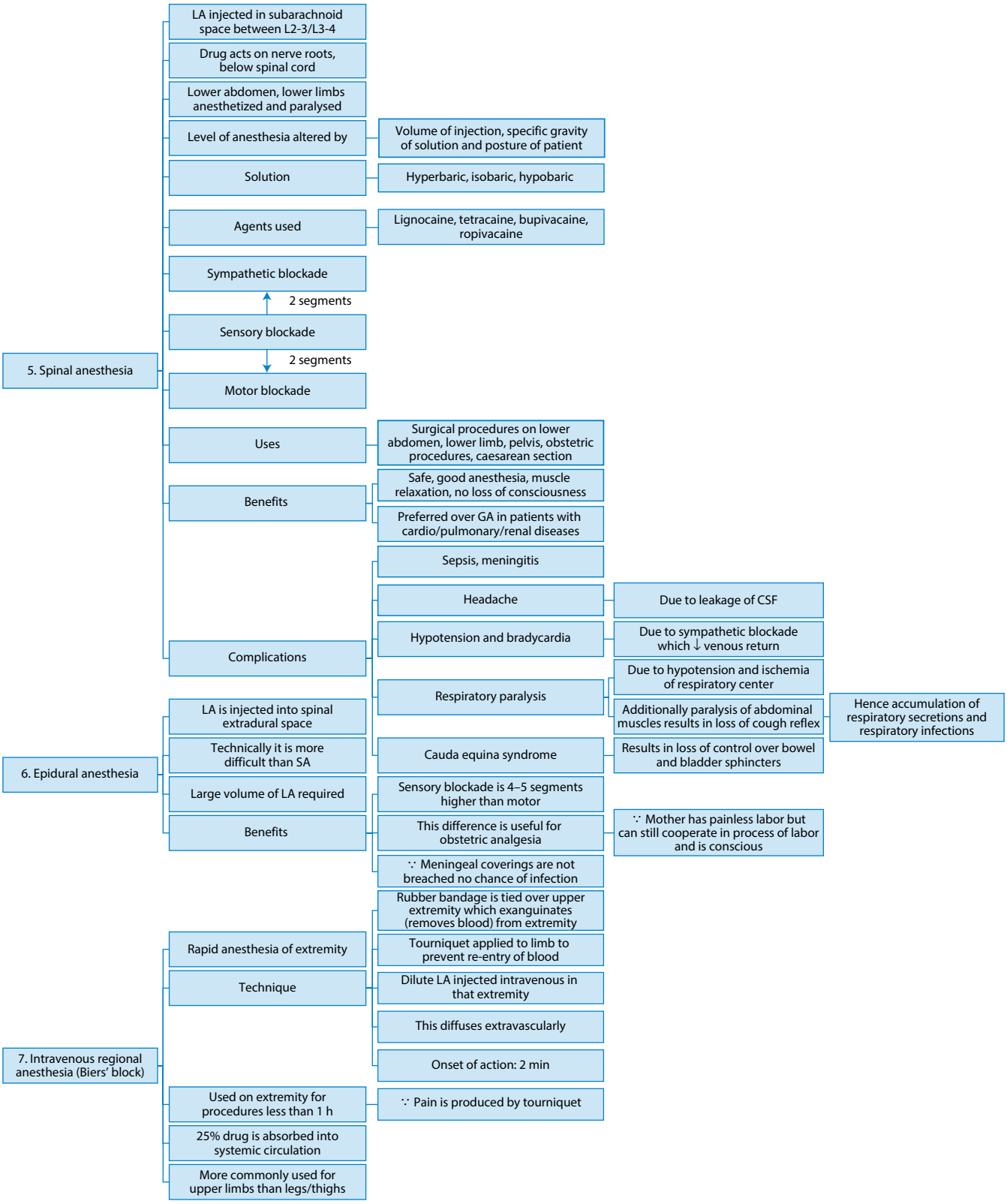


28.5 USES OF LAs



(*Continued*)

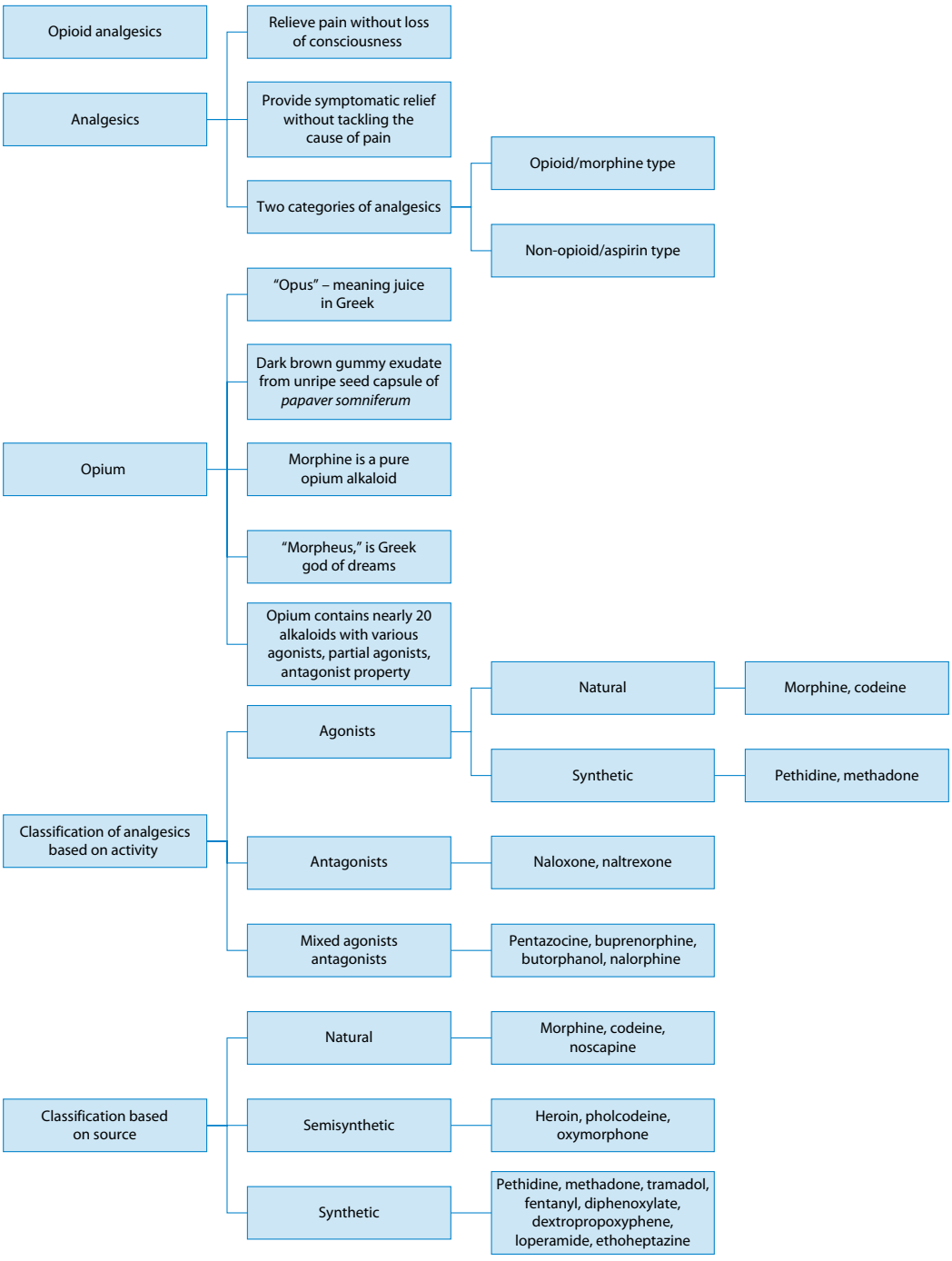
28.5 USES OF LAs (Continued)



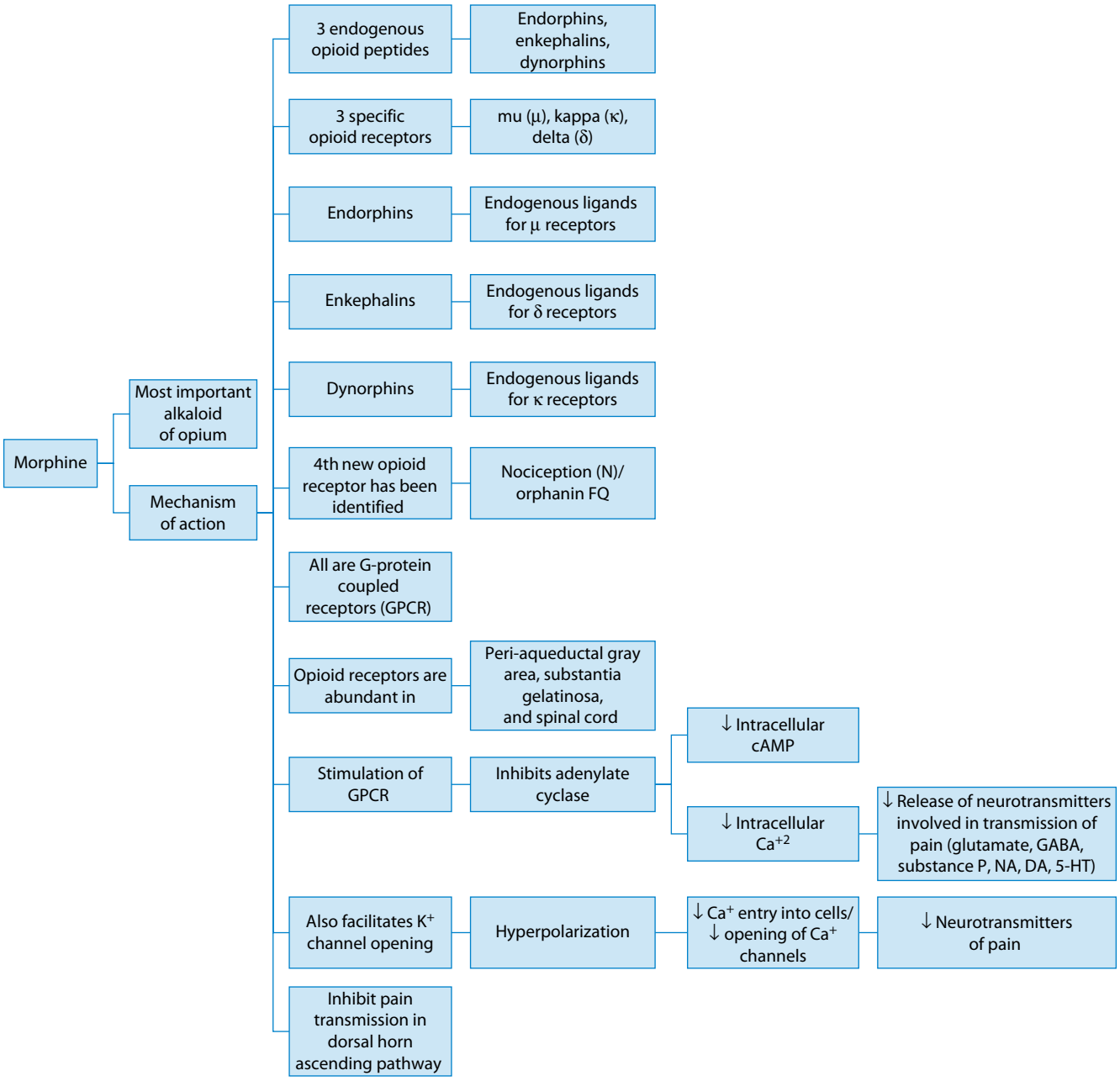
29

Opioid analgesics

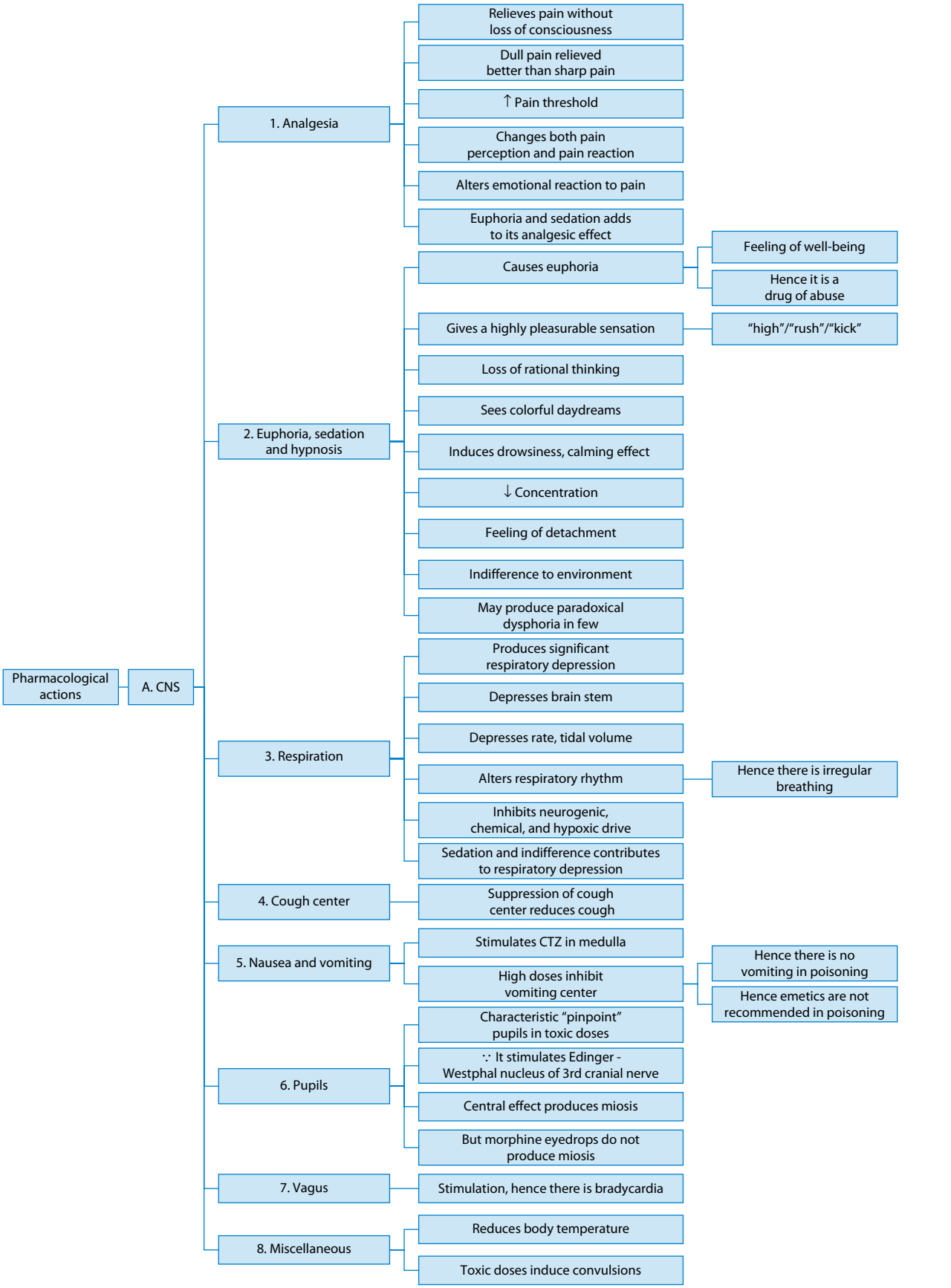
29.1 OPIOID ANALGESICS CLASSIFICATION



29.2 MORPHINE AND MECHANISM OF ACTION

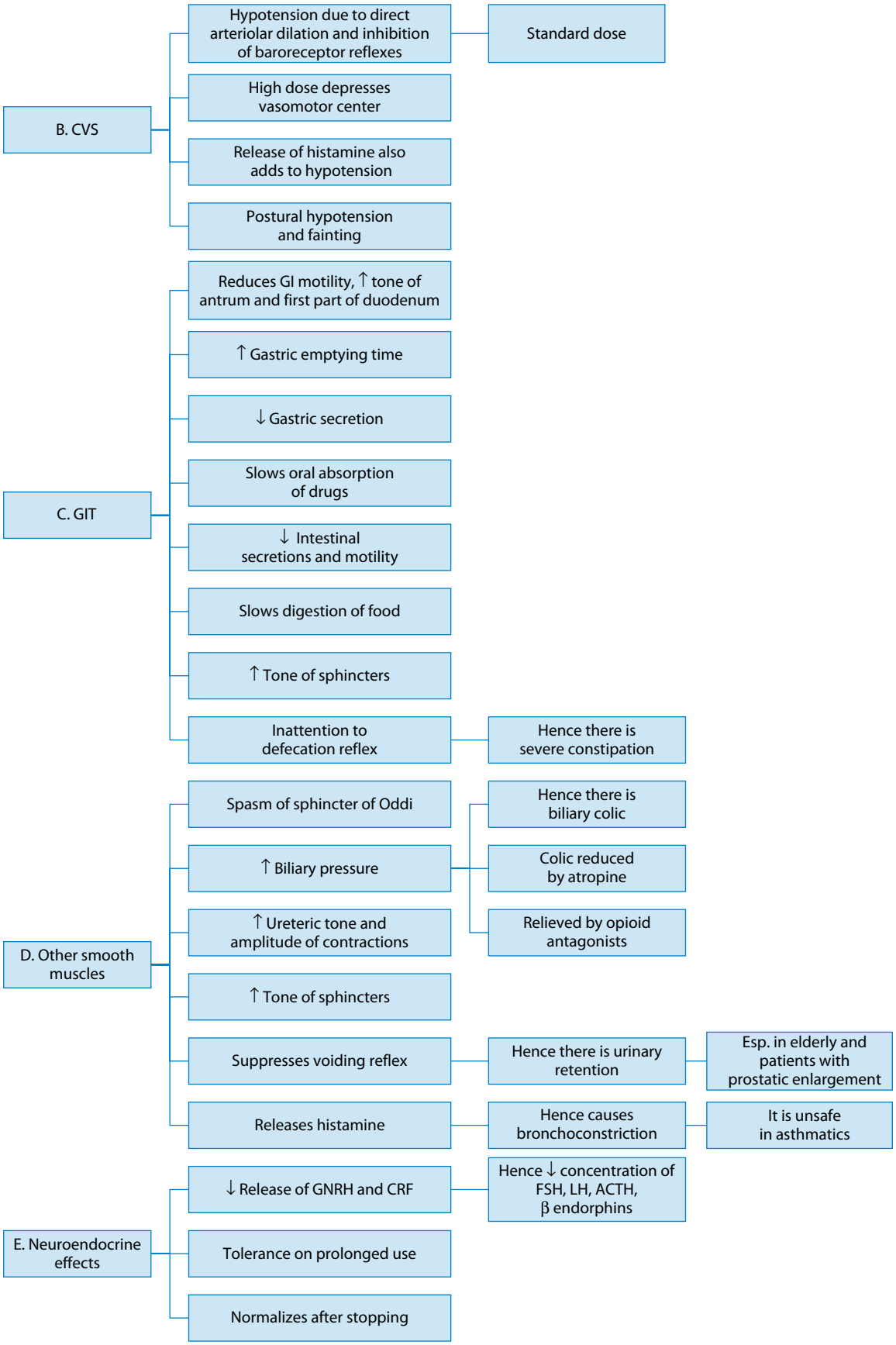


29.3 PHARMACOLOGICAL ACTIONS

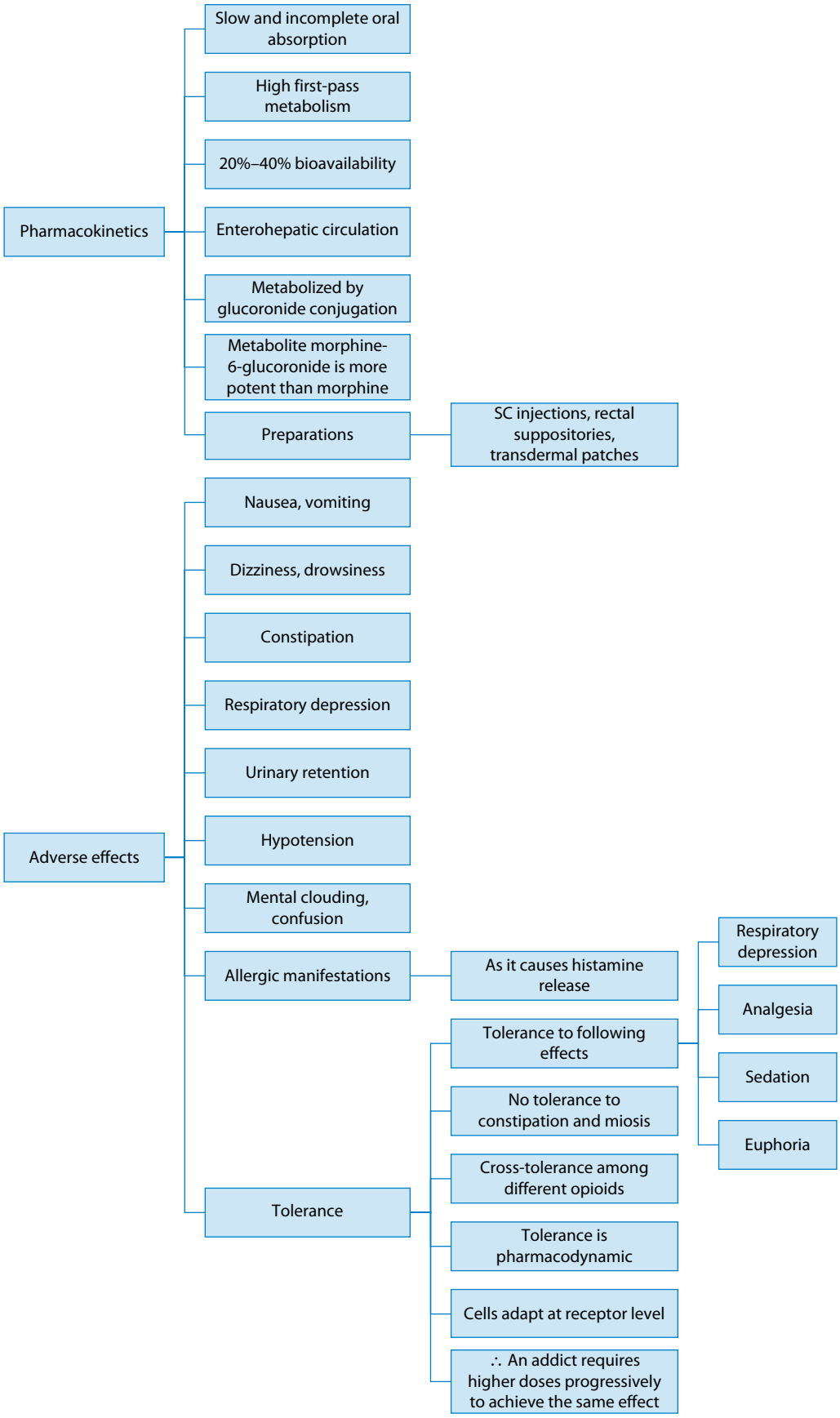


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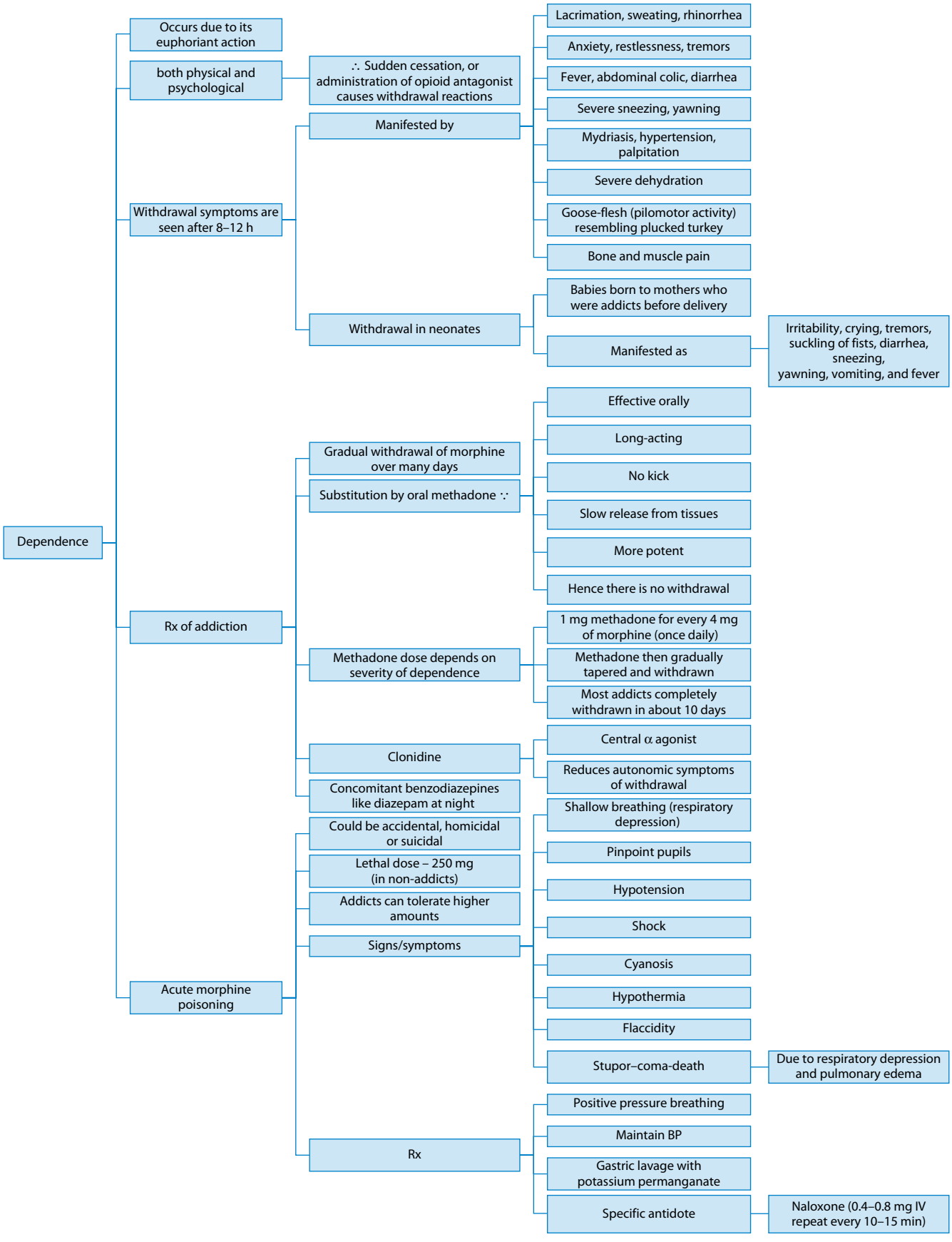
29.3 PHARMACOLOGICAL ACTIONS (Continued)



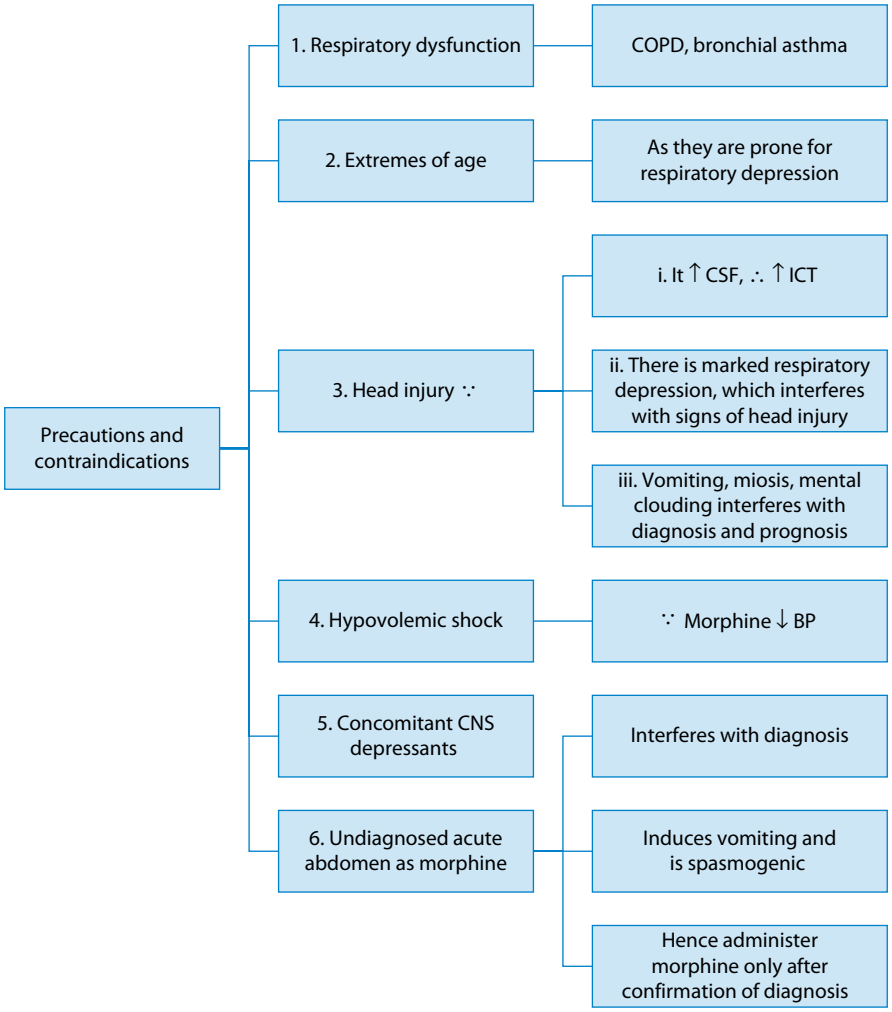
29.4 PHARMACOKINETICS AND ADVERSE EFFECTS



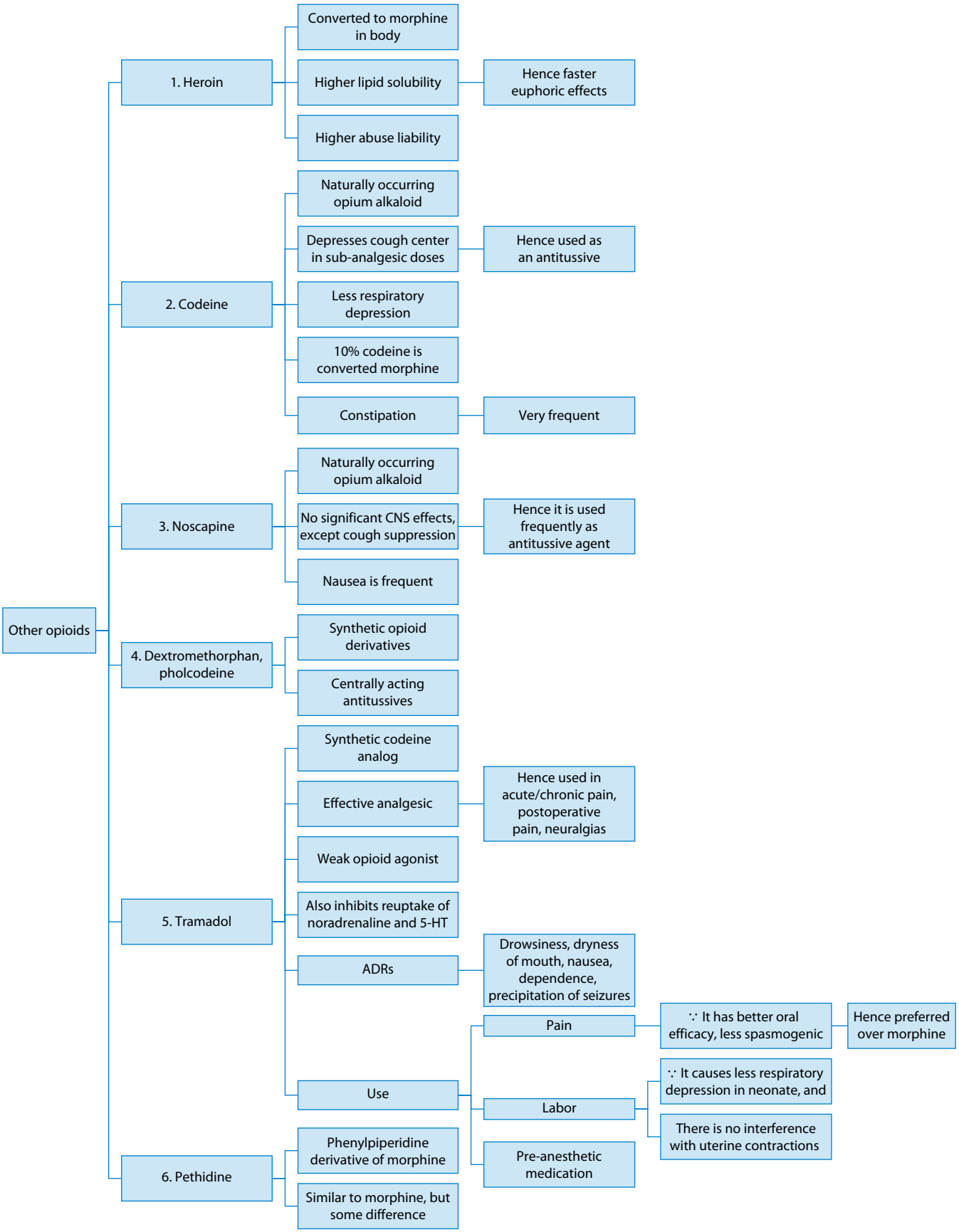
29.5 DEPENDENCE



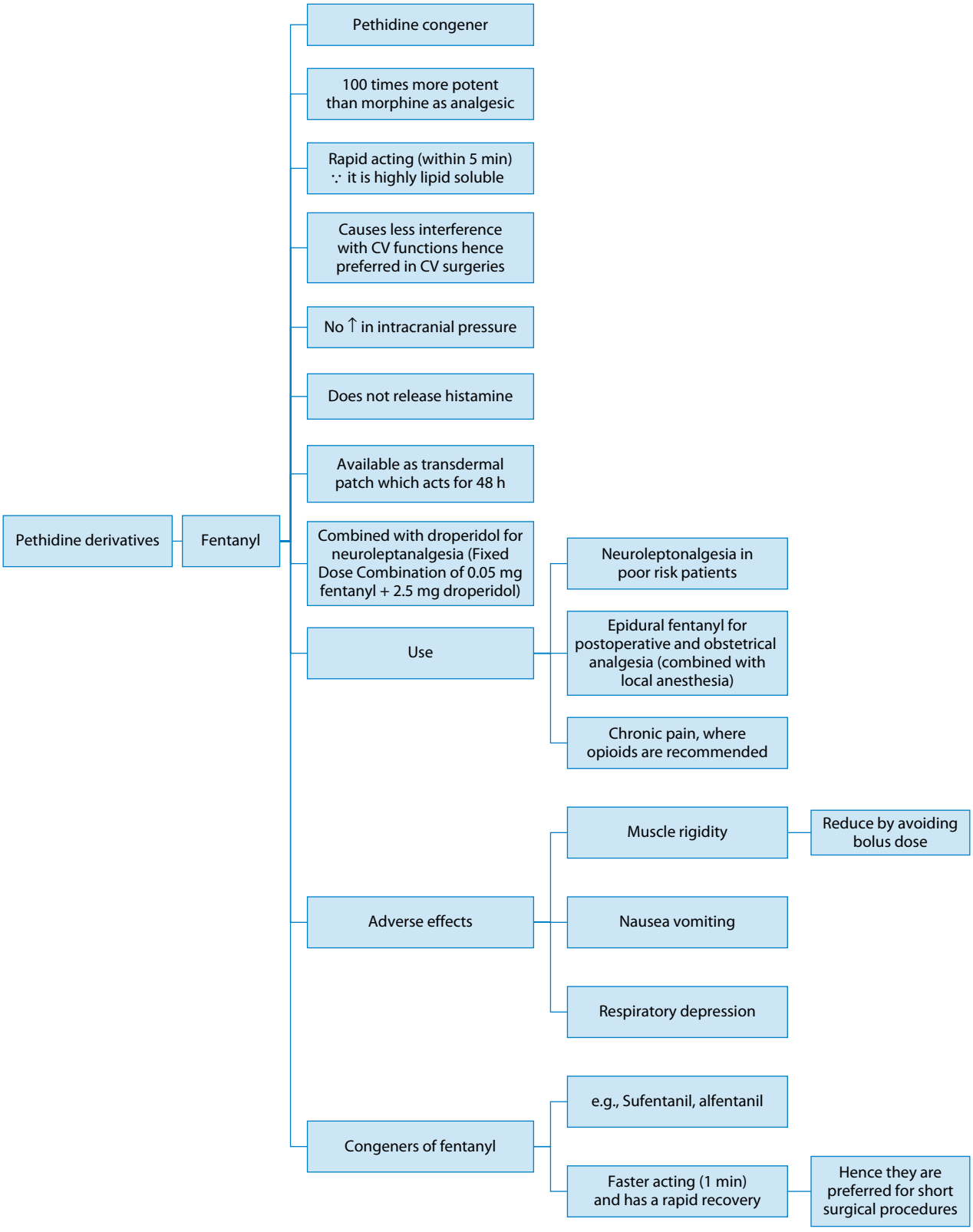
29.6 PRECAUTIONS AND CONTRAINDICATIONS



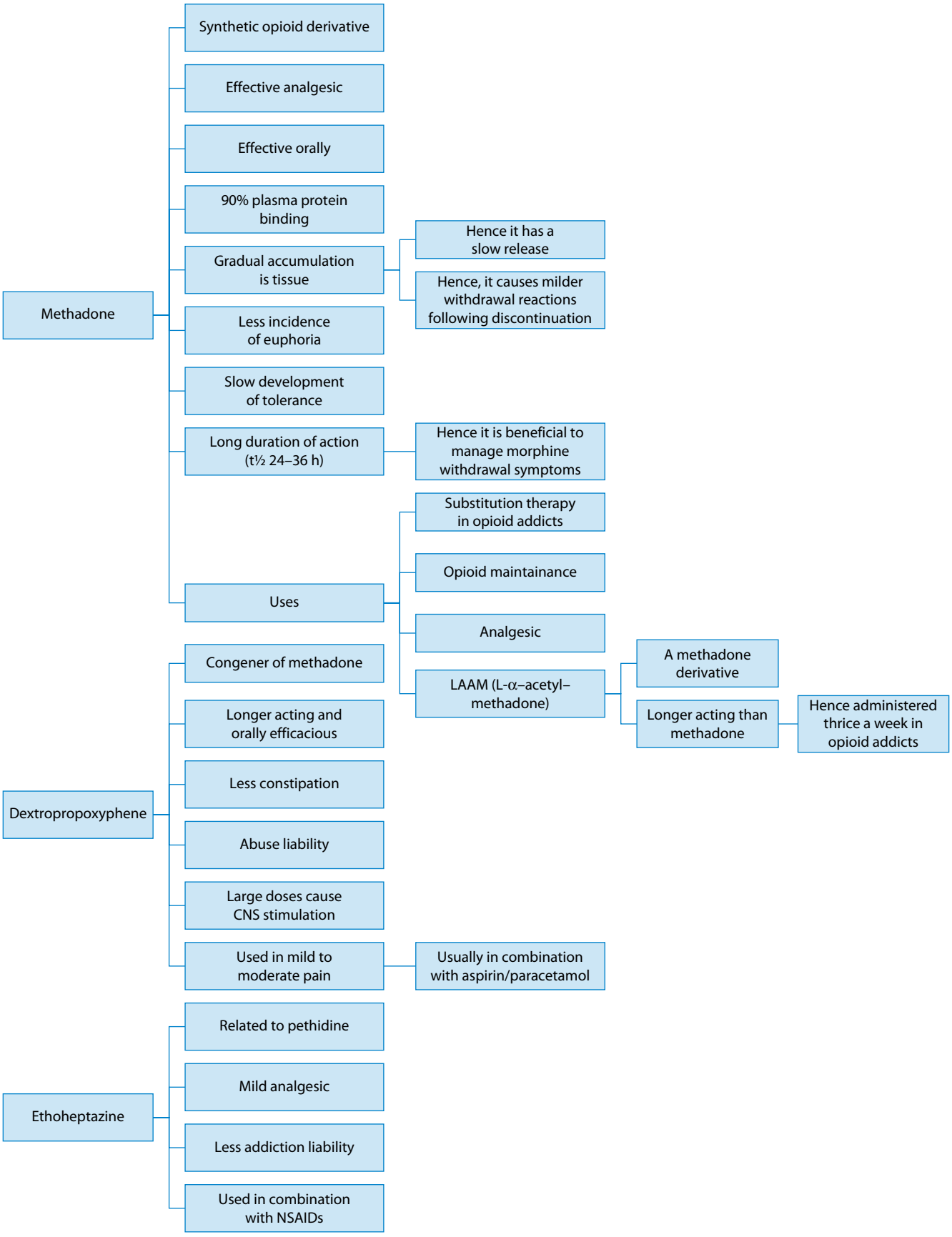
29.7 OTHER OPIOIDS



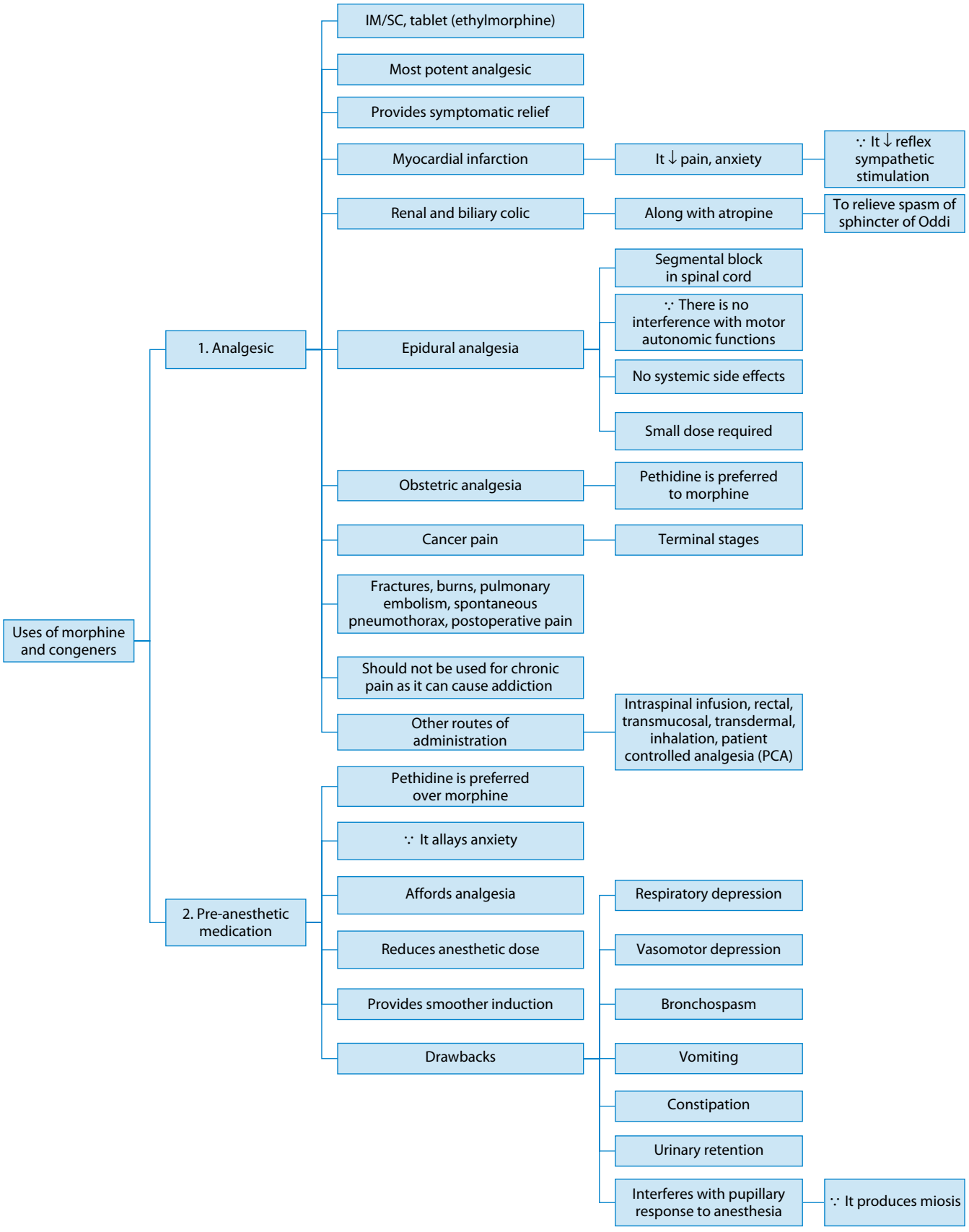
29.8 PETHIDINE DERIVATIVES – FENTANYL



29.9 METHADONE, DEXTROPROPOXYPHENE, AND ETHOHEPTAZINE

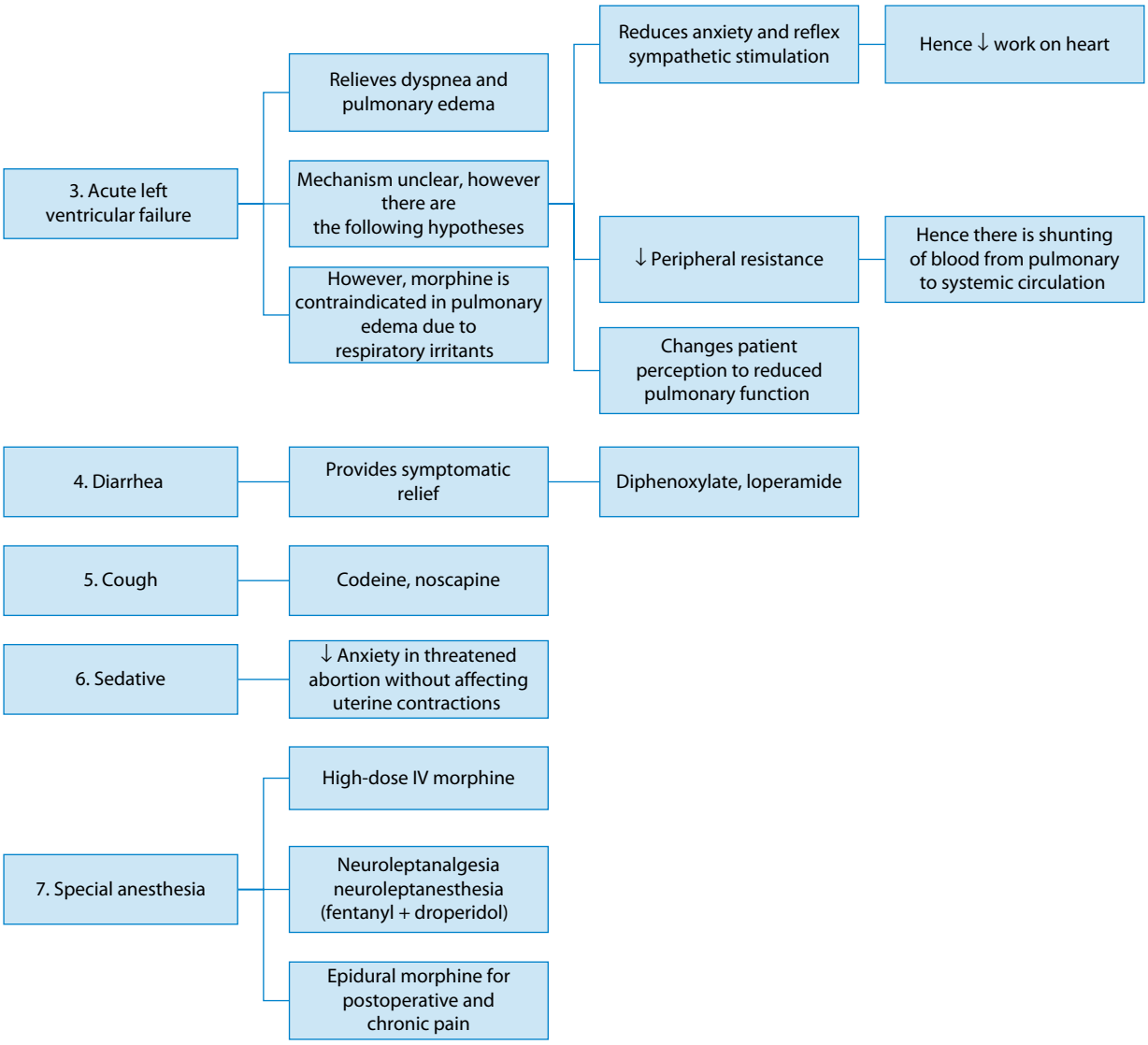


29.10 USES OF MORPHINE AND CONGENERS

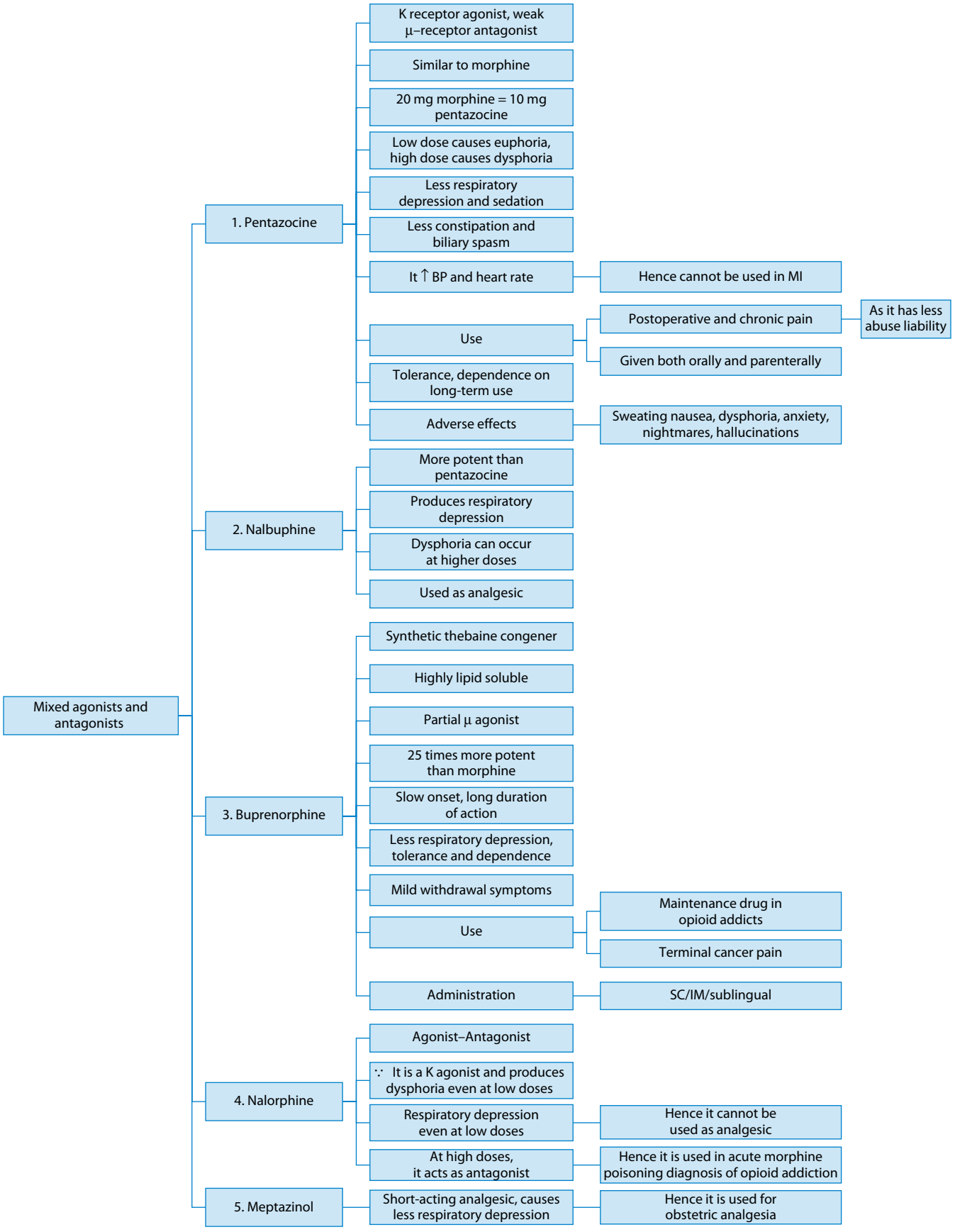


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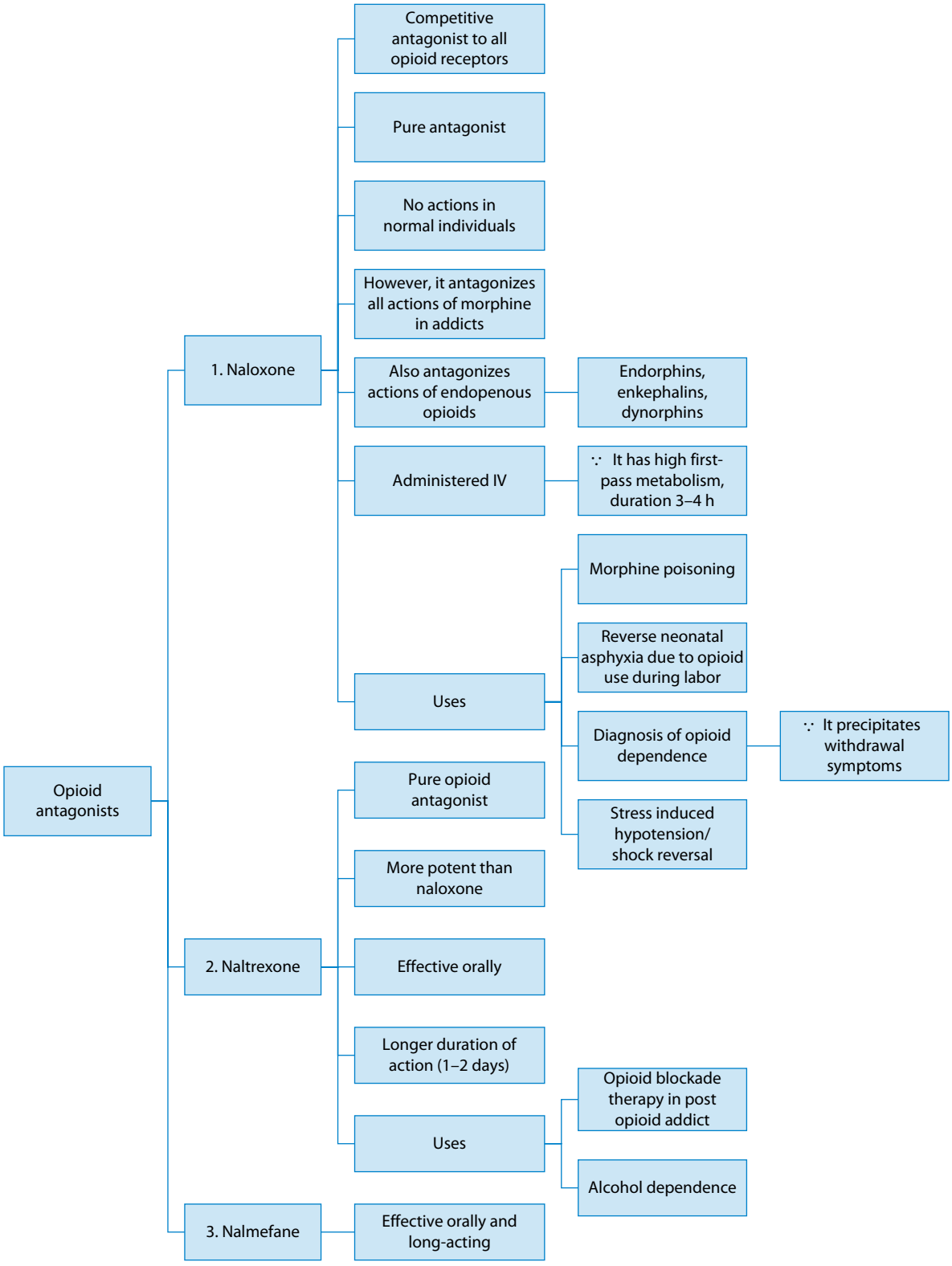
29.10 USES OF MORPHINE AND CONGENERS (Continued)



29.11 MIXED AGONISTS AND ANTAGONISTS



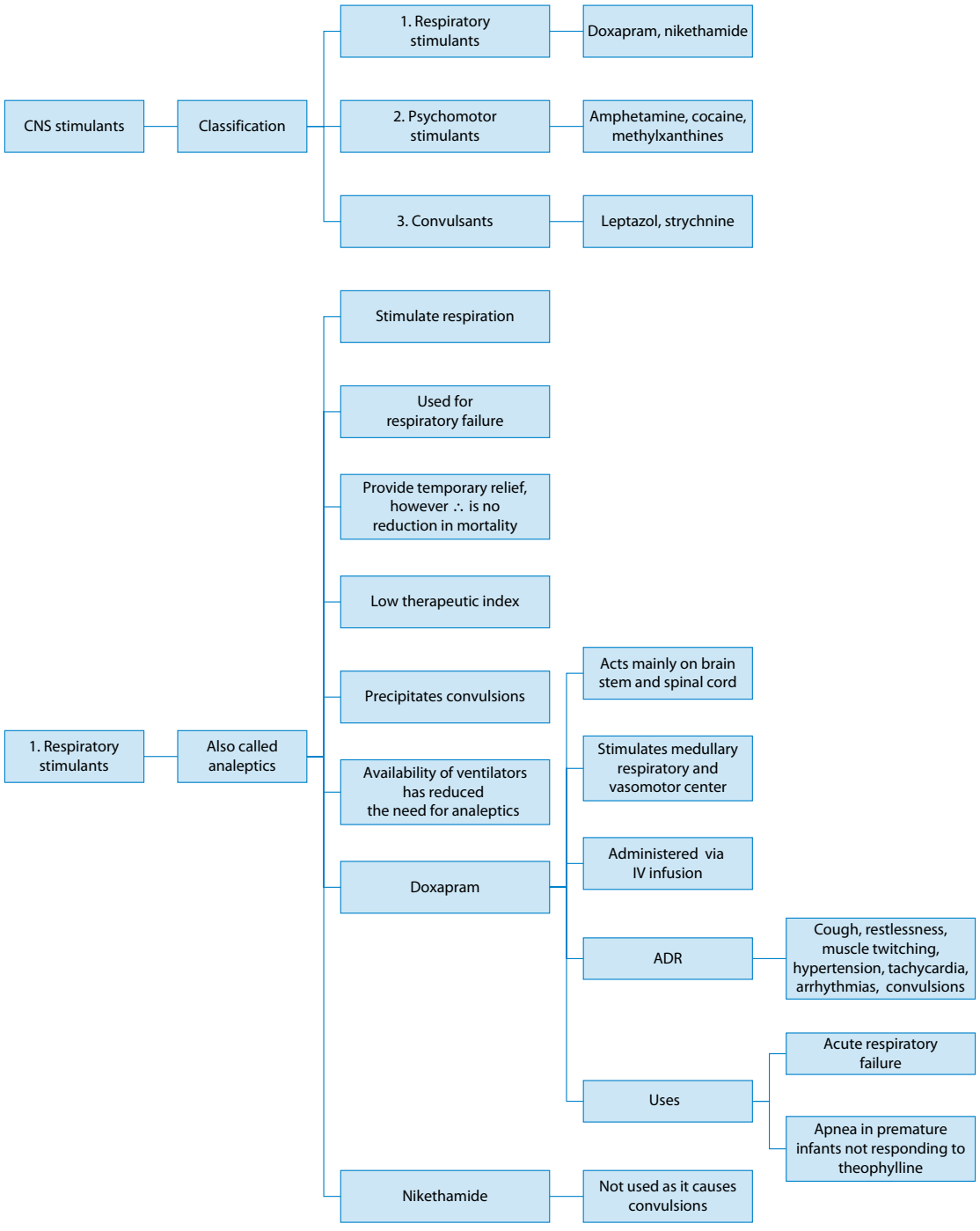
29.12 OPIOID ANTAGONISTS



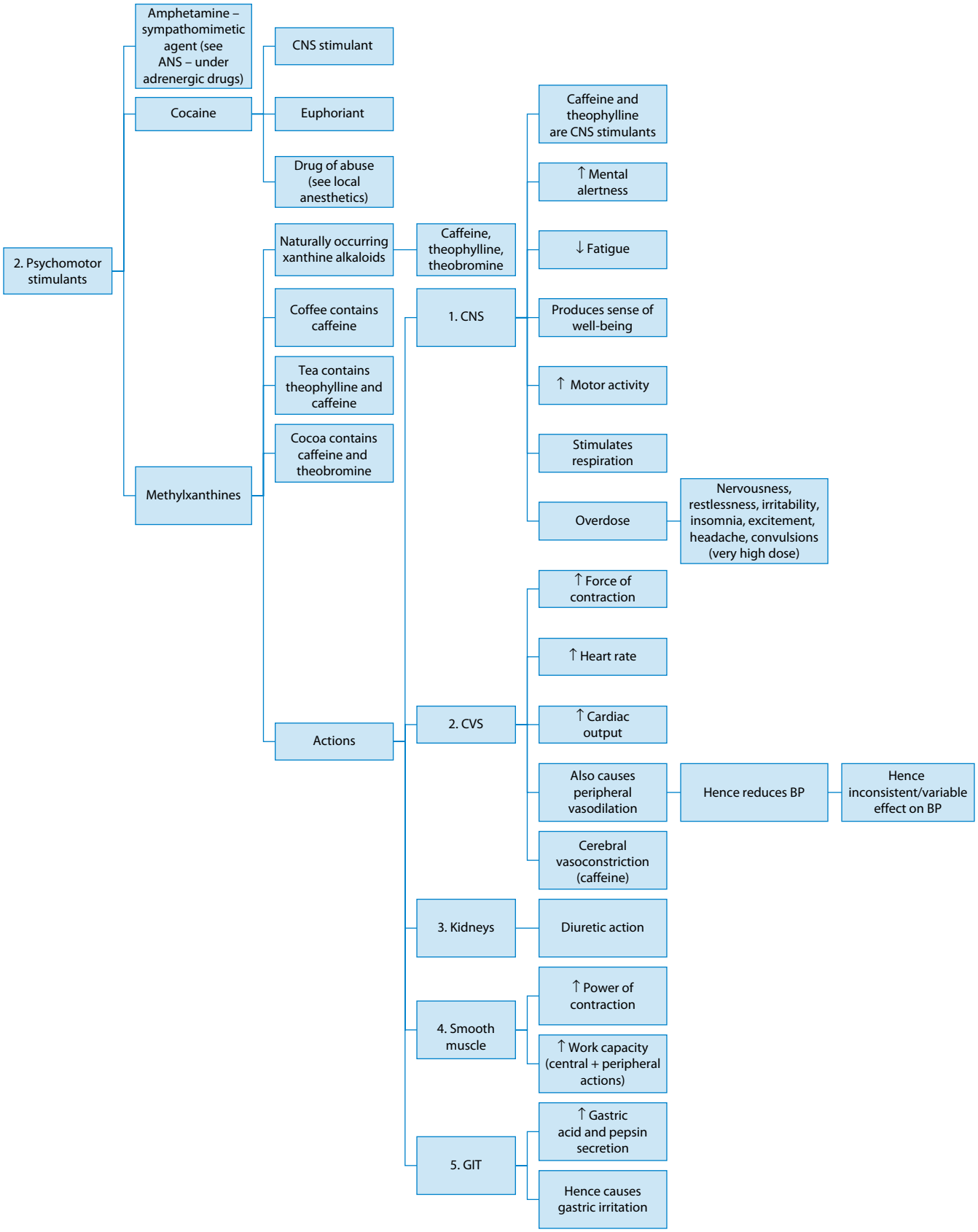
30

CNS stimulants/drugs of abuse

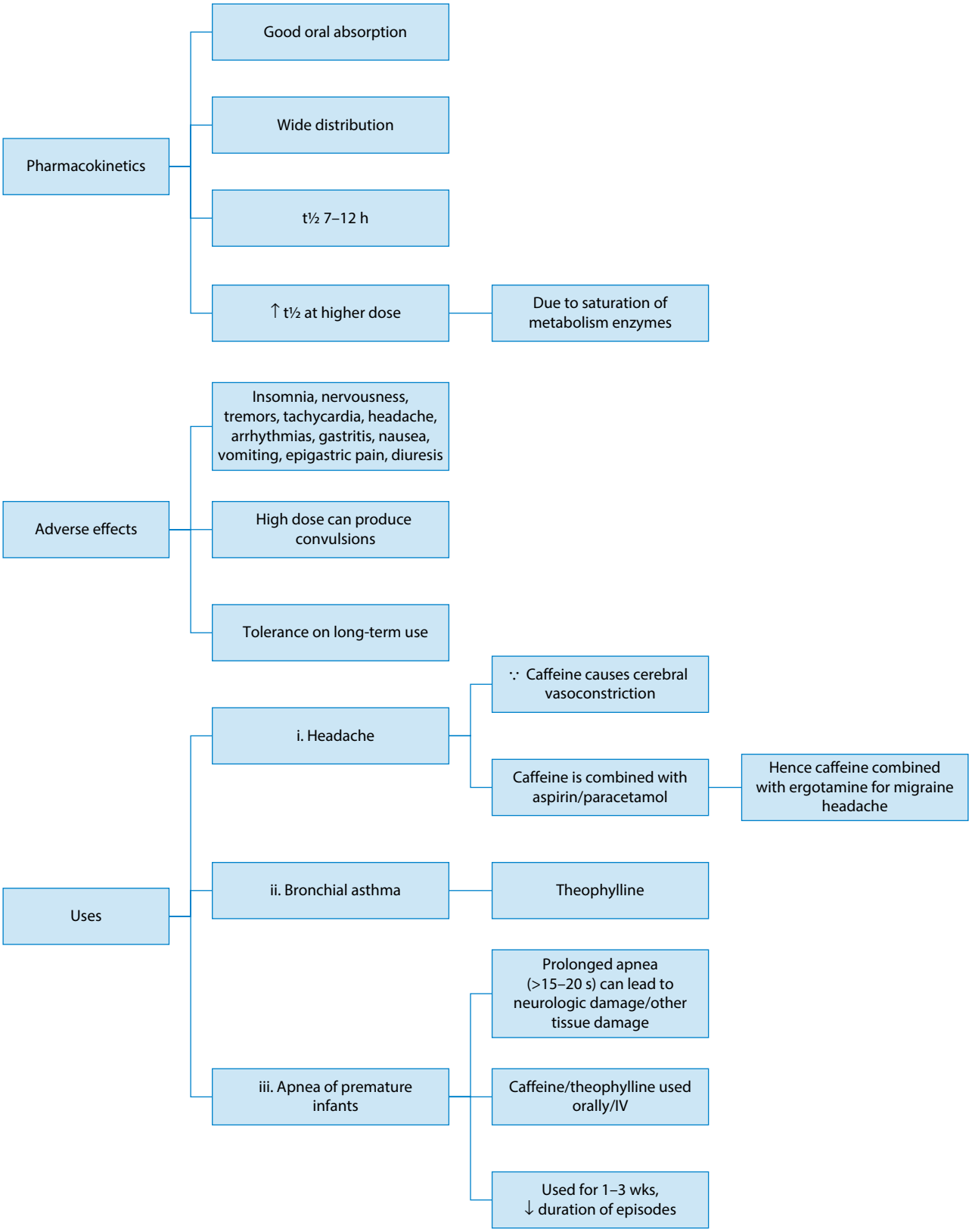
30.1 CNS STIMULANTS – CLASSIFICATION AND RESPIRATORY STIMULANTS



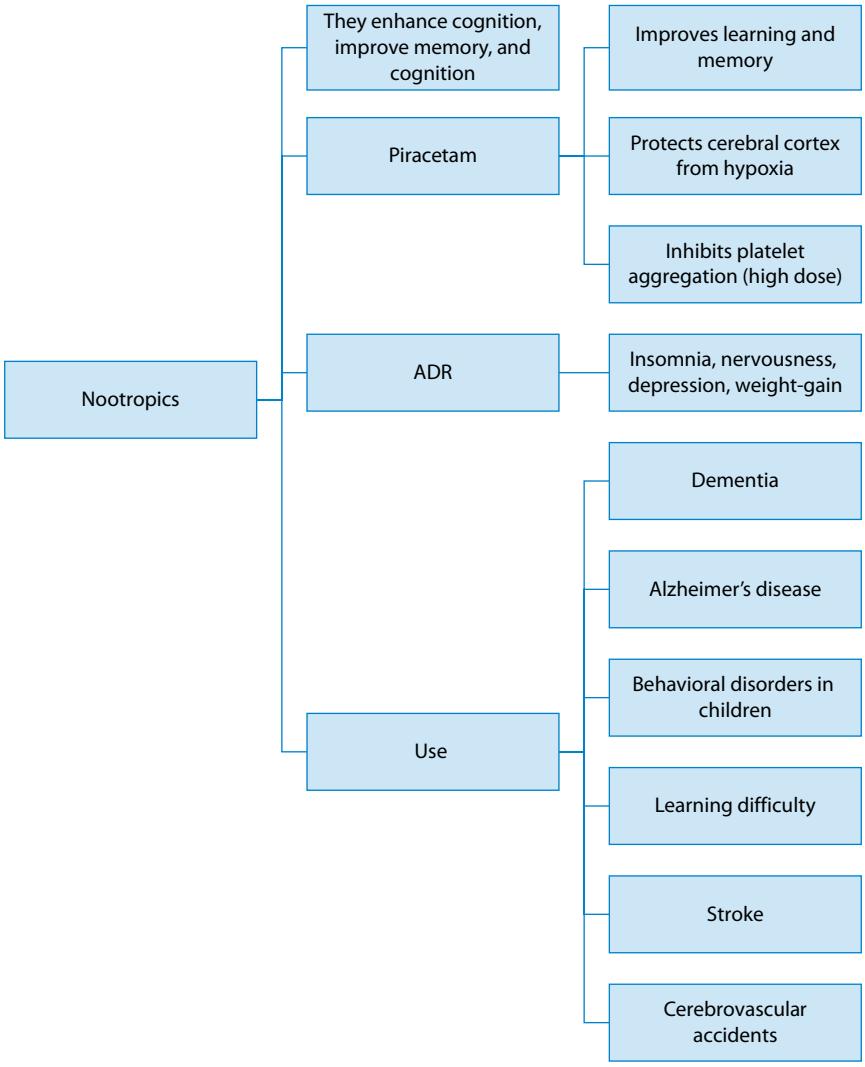
30.2 PSYCHOMOTOR STIMULANTS/METHYLXANTHINES – ACTIONS



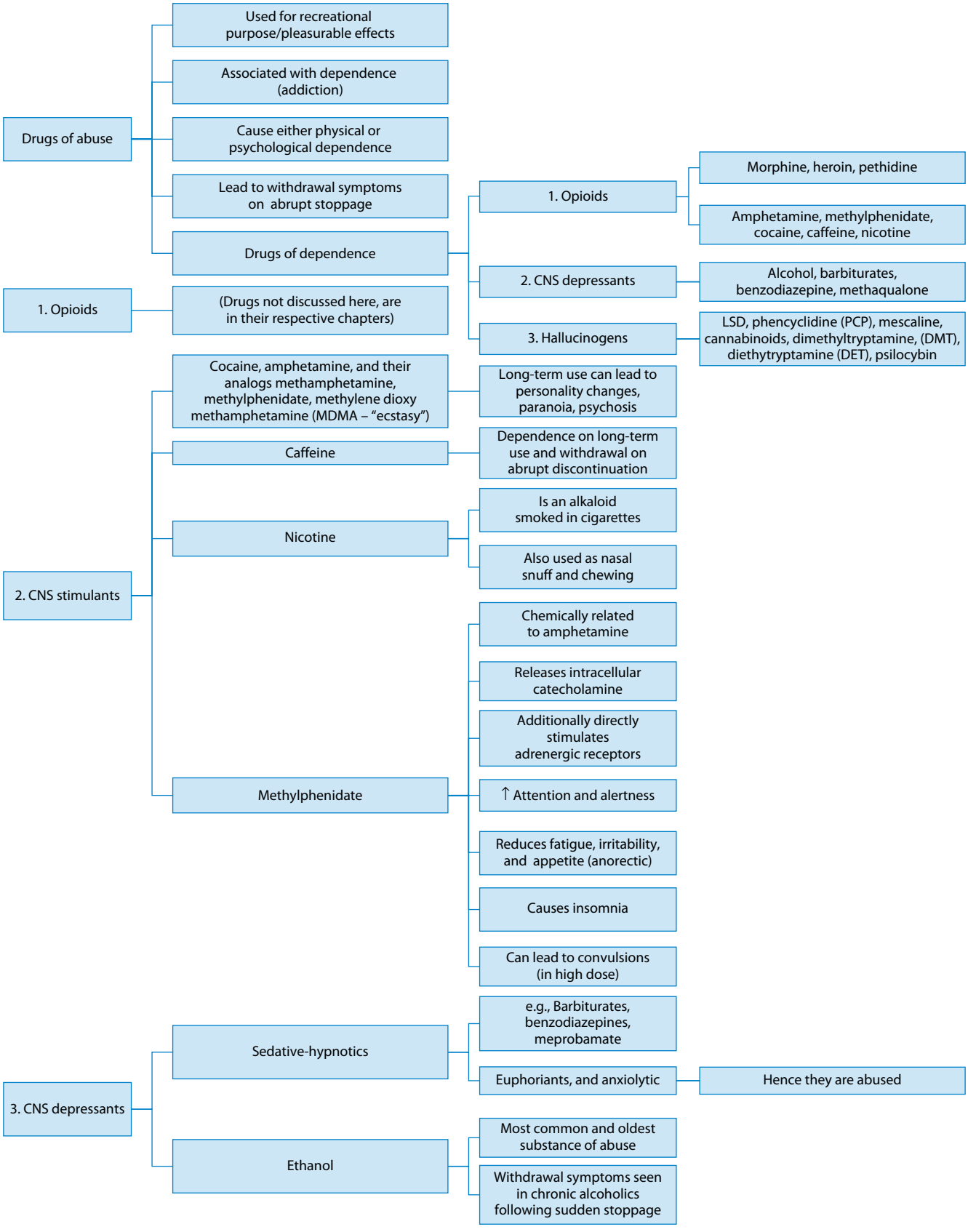
30.3 METHYLXANTHINES – PHARMACOKINETICS, ADVERSE EFFECTS, AND USES



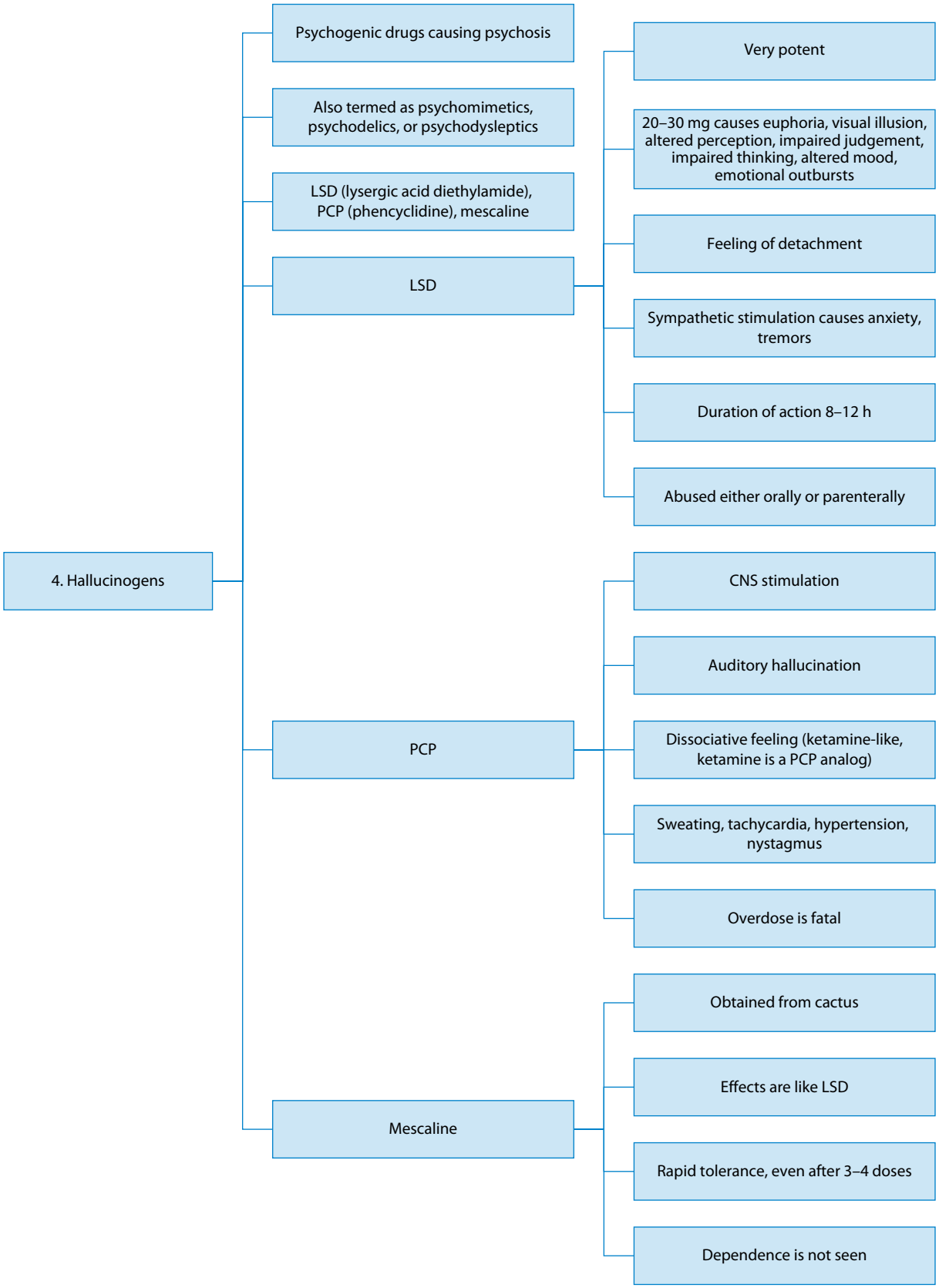
30.4 NOOTROPICS



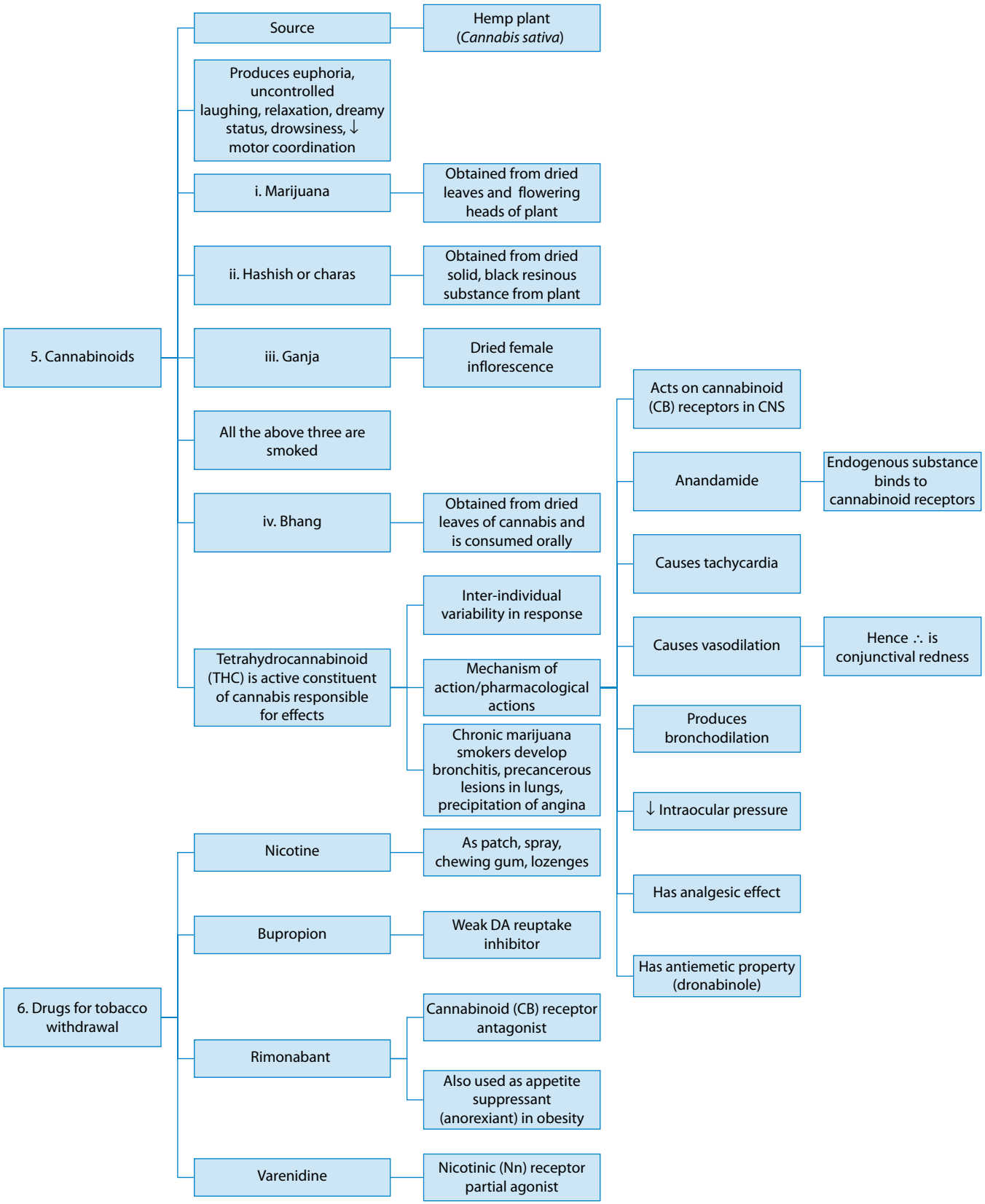
30.5 DRUGS OF ABUSE – OPIOIDS, CNS STIMULANTS, AND CNS DEPRESSANTS



30.6 HALLUCINOGENS



30.7 CANNABINOIDS AND DRUGS FOR TOBACCO WITHDRAWAL



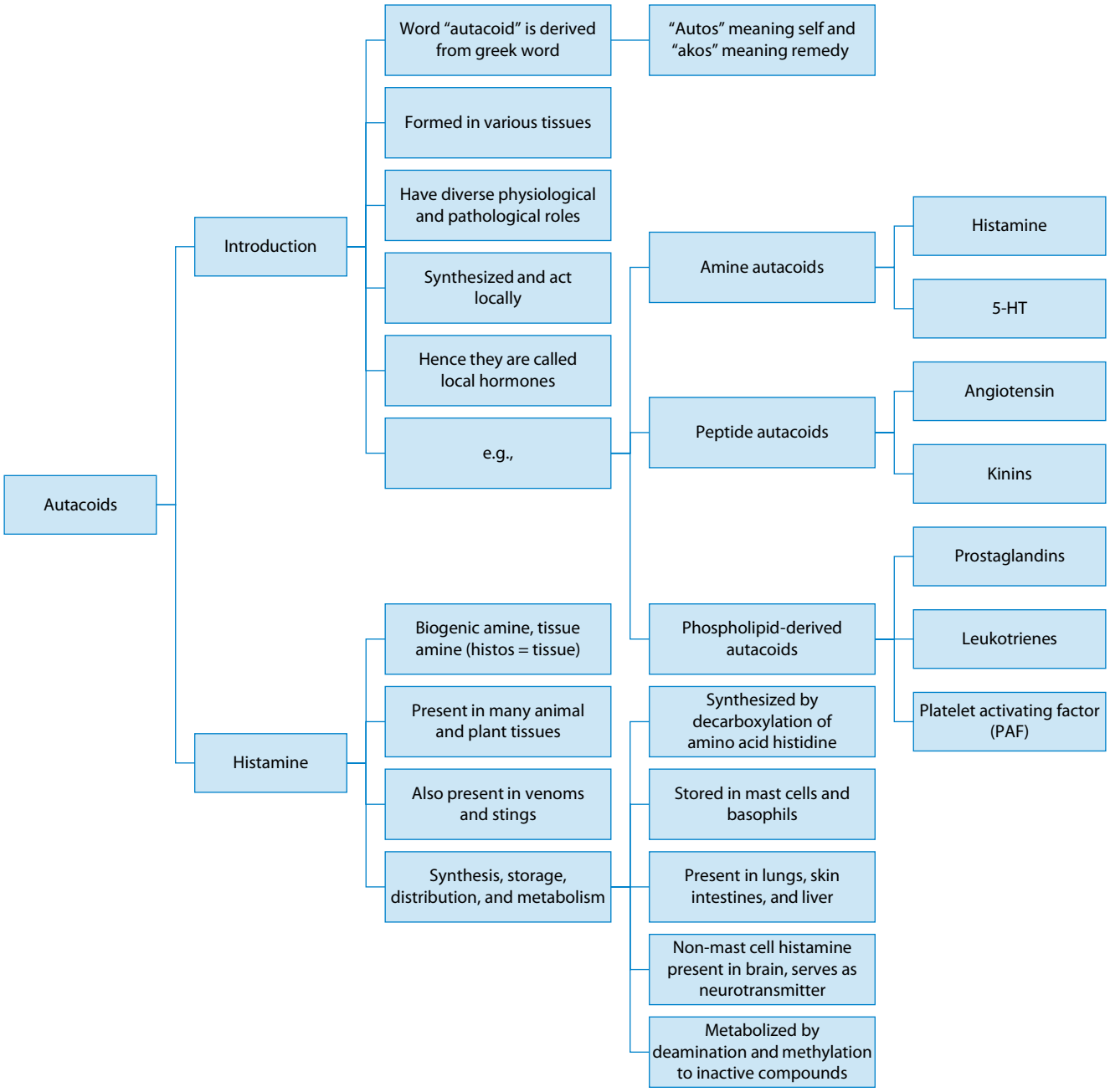
PART V

Autacoid pharmacology

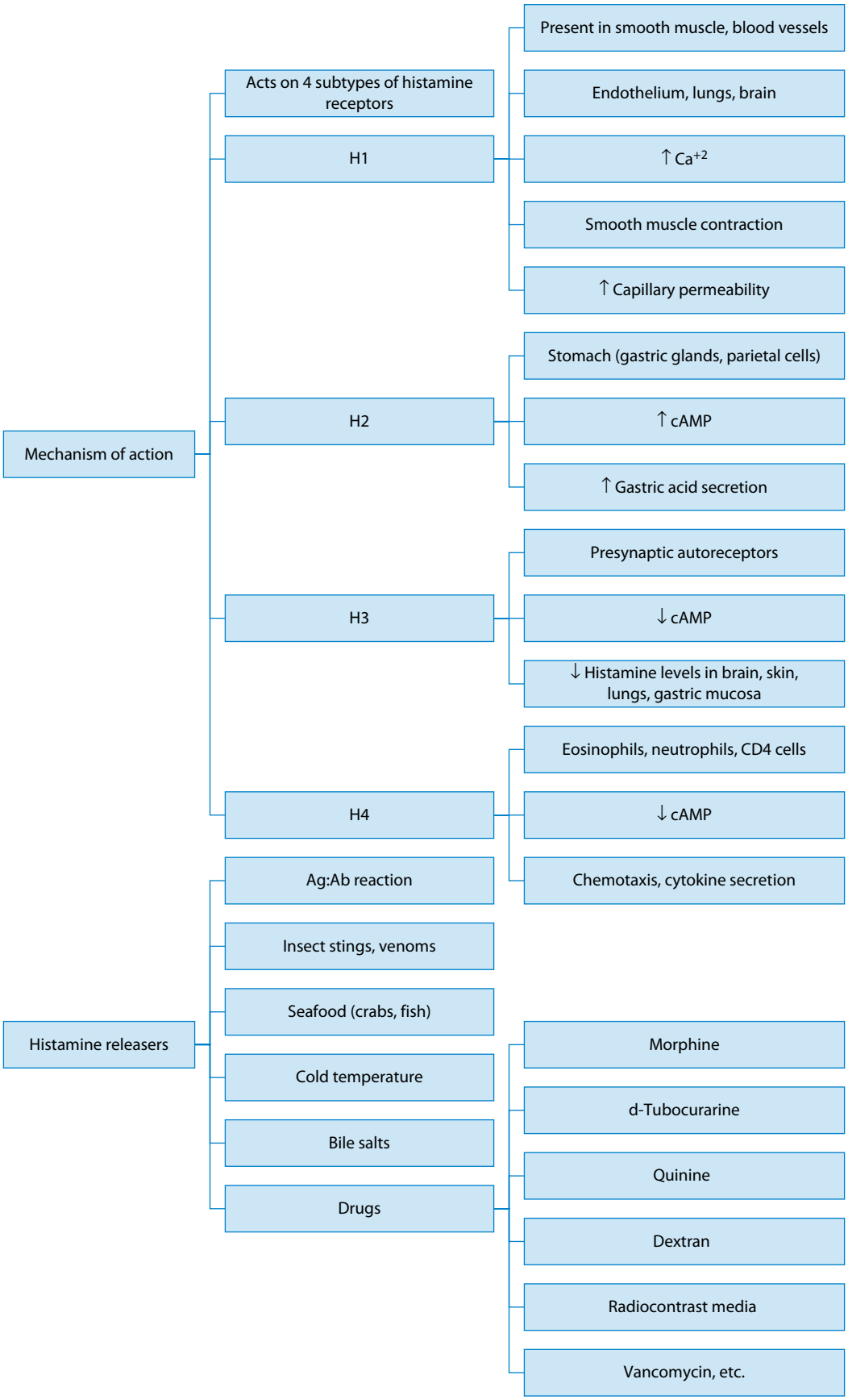
31

Autacoids, histamine and antihistaminics

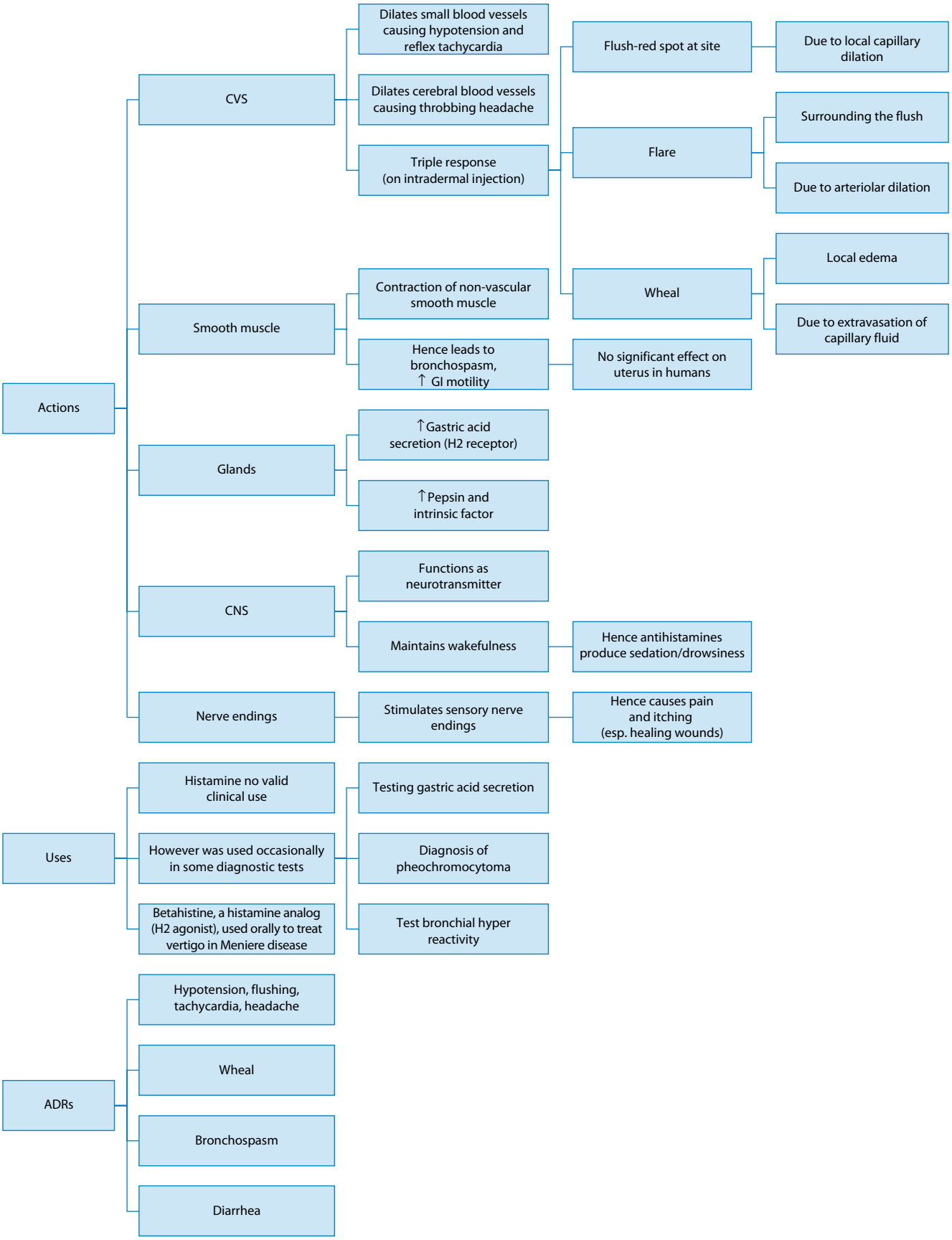
31.1 AUTACOIDS – INTRODUCTION, CLASSIFICATION OF AUTACOIDS AND HISTAMINE



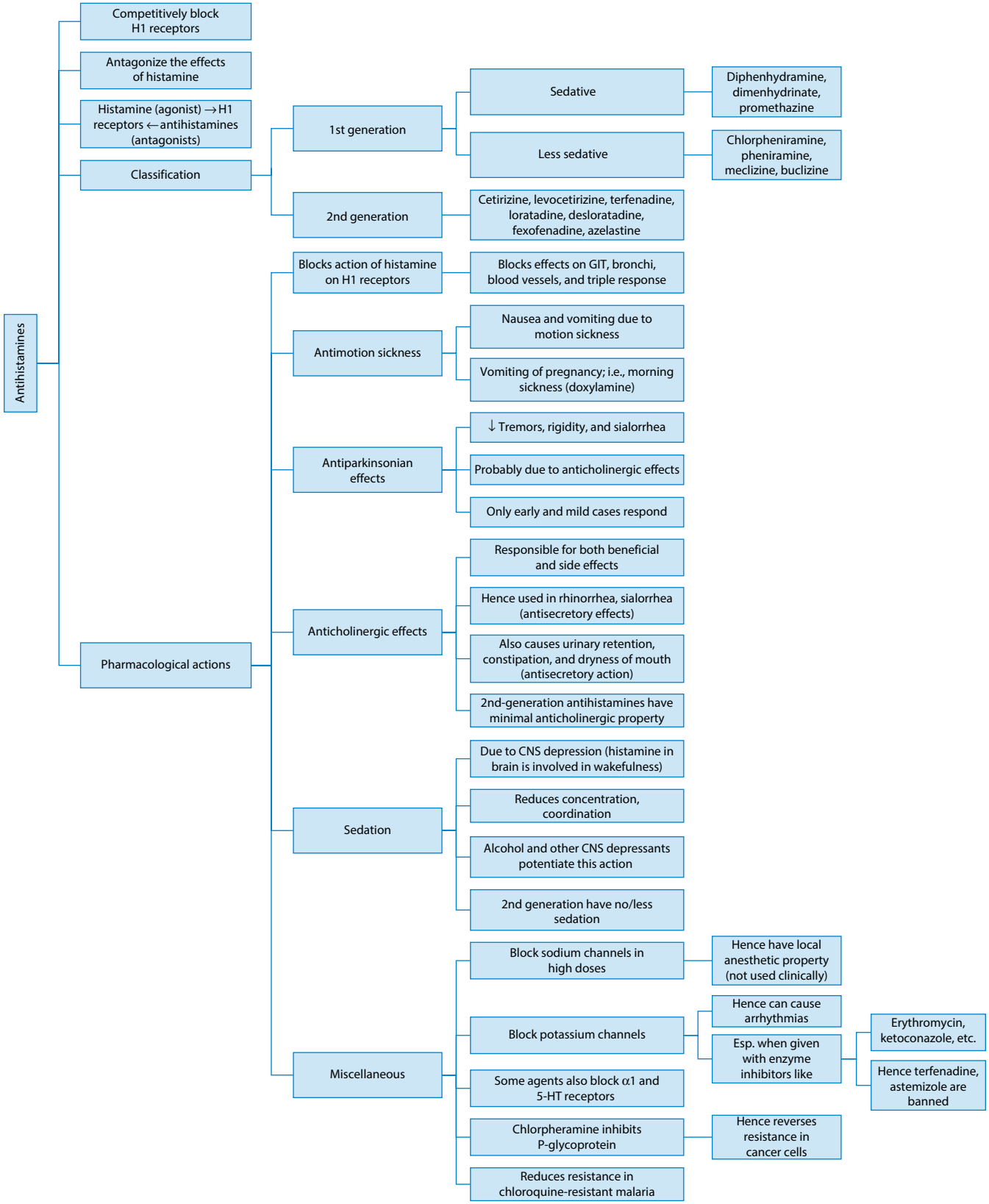
31.2 MECHANISM OF ACTION AND HISTAMINE RELEASERS



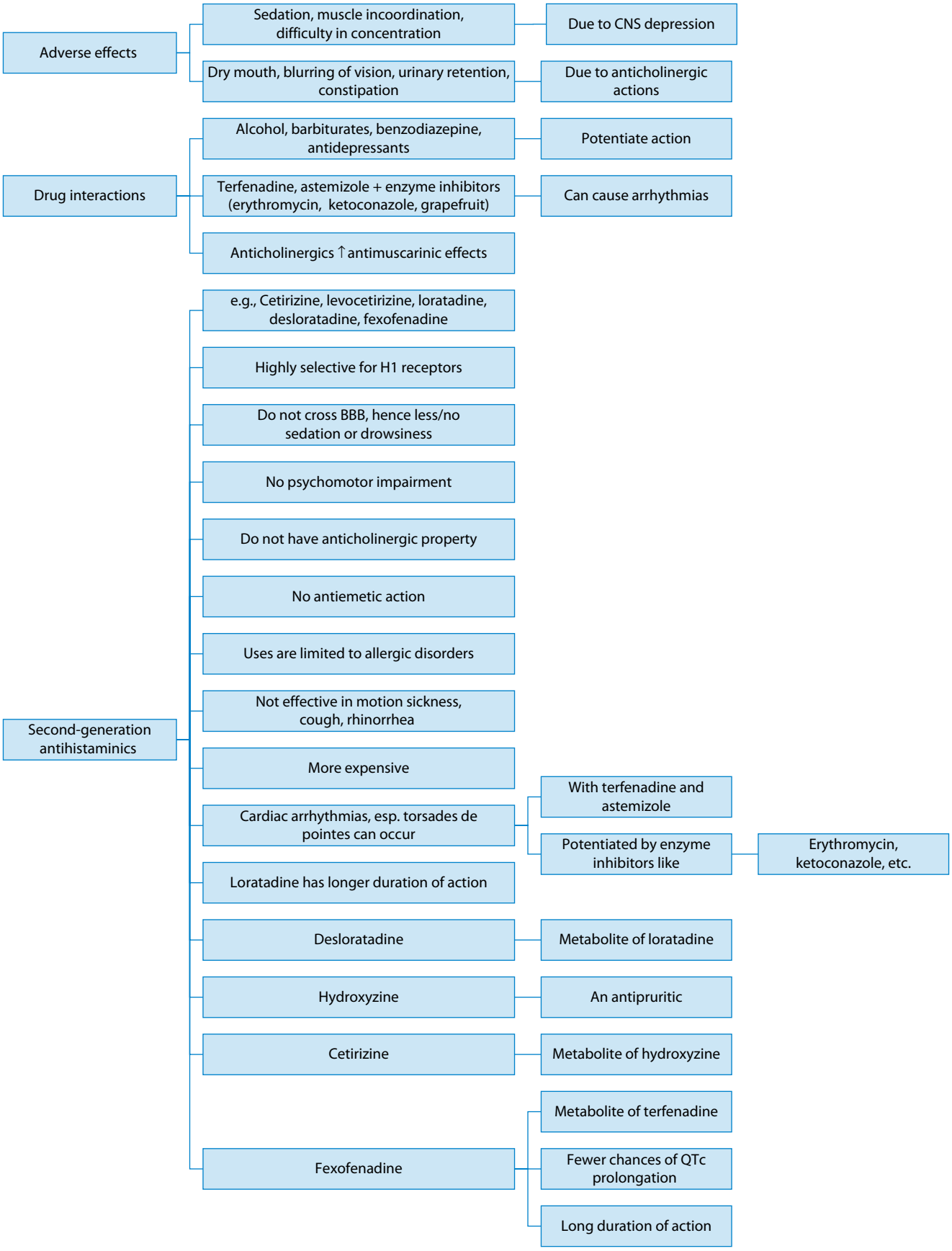
31.3 ACTIONS, USES, AND ADRs



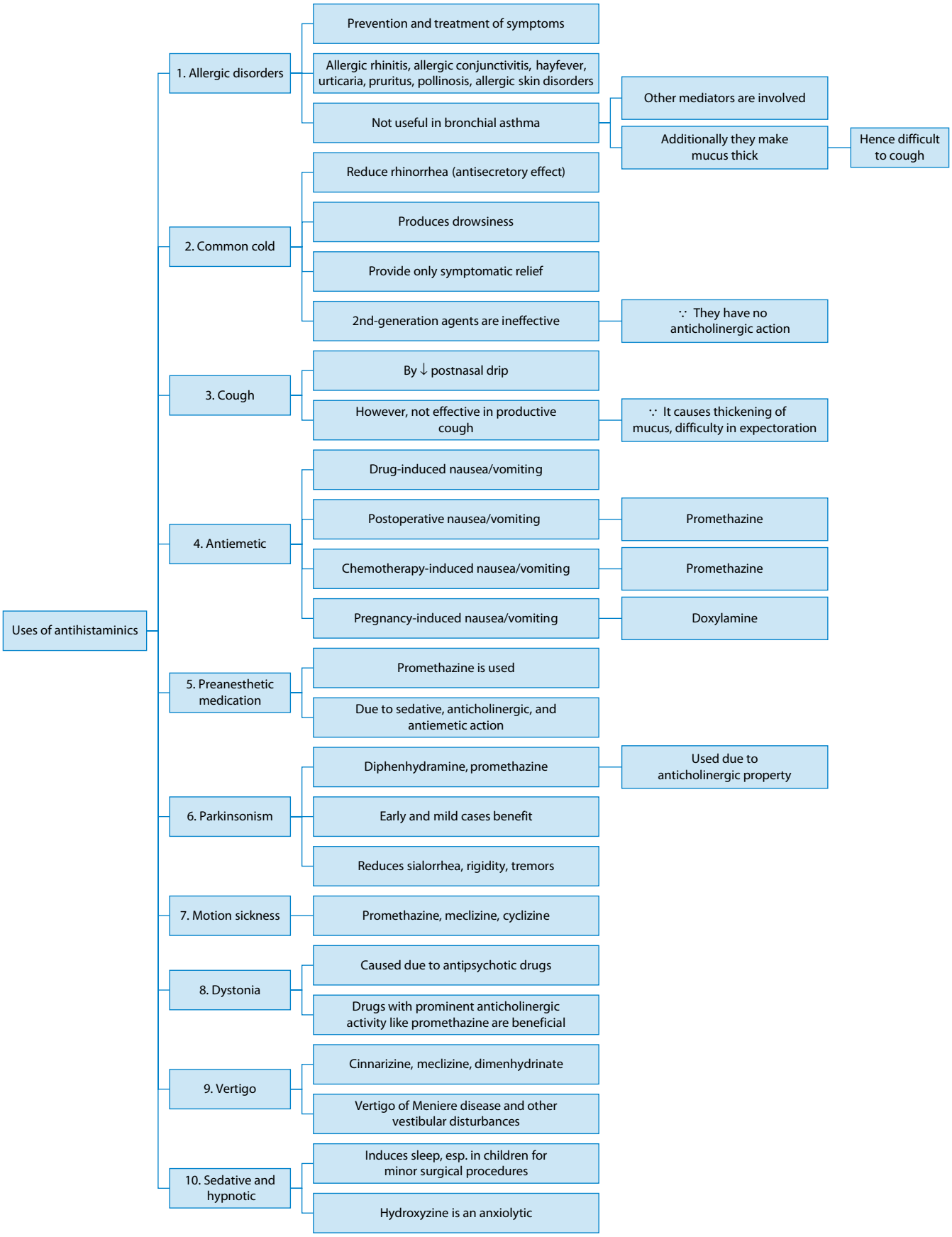
31.4 ANTIHISTAMINES – CLASSIFICATION AND PHARMACOLOGICAL ACTIONS



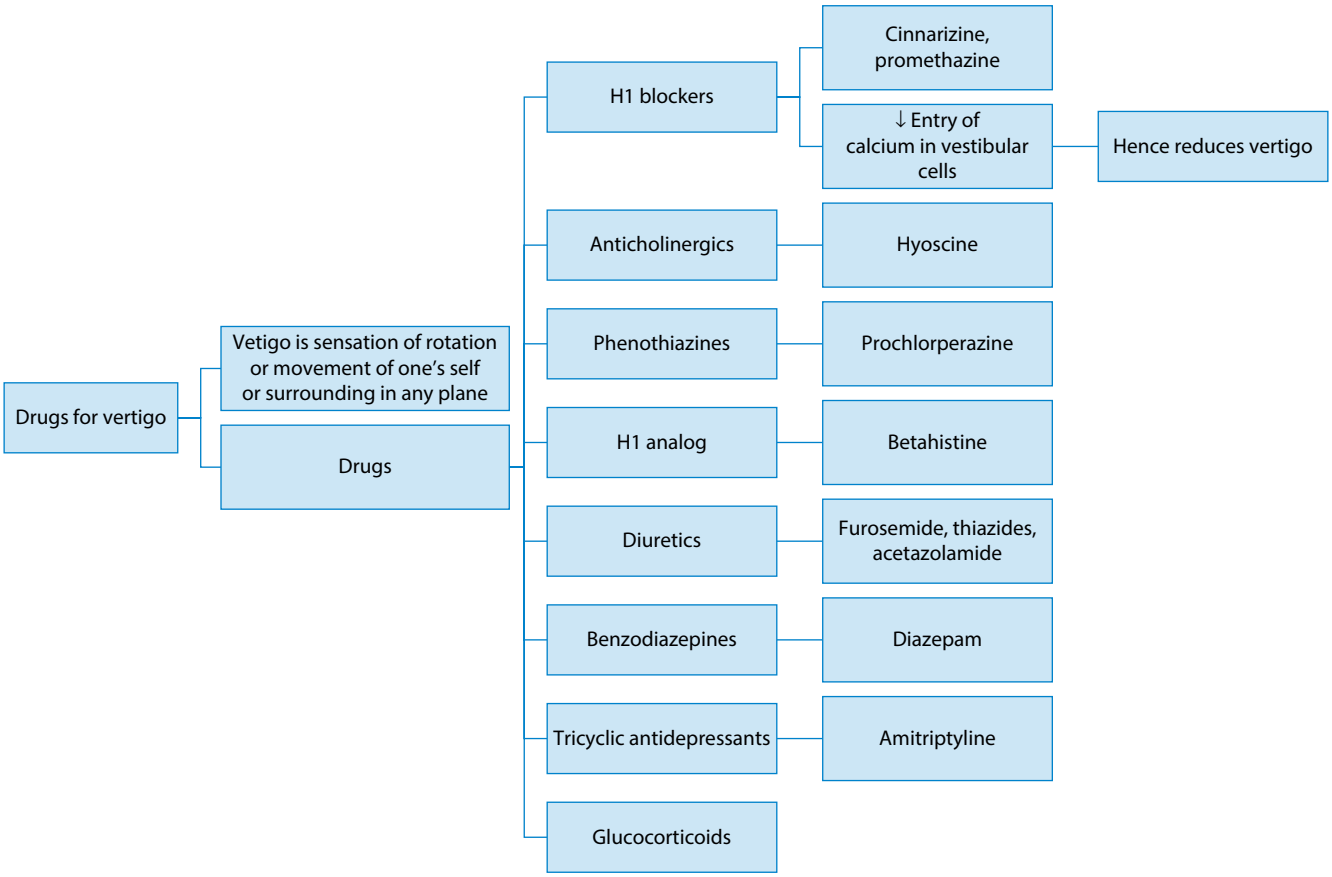
31.5 ADVERSE EFFECTS, DRUG INTERACTIONS, AND SECOND-GENERATION ANTIHISTAMINICS



31.6 USES OF ANTIHISTAMINICS



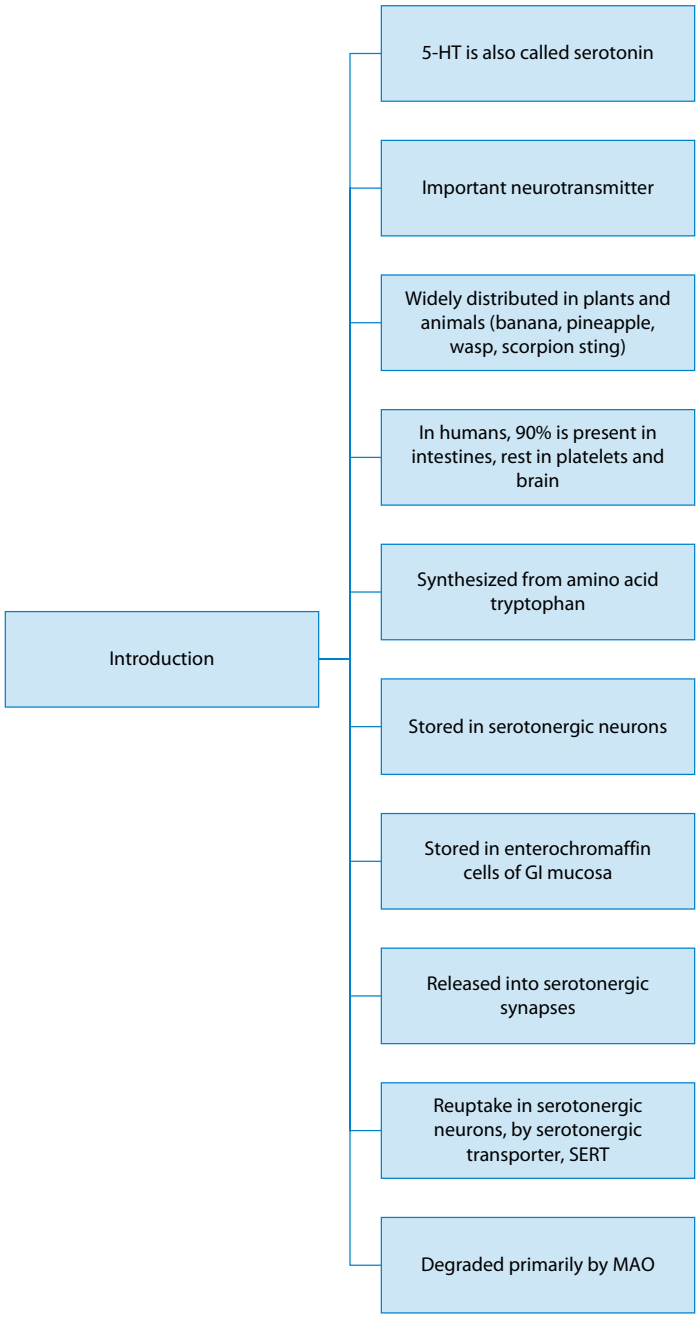
31.7 DRUGS FOR VERTIGO



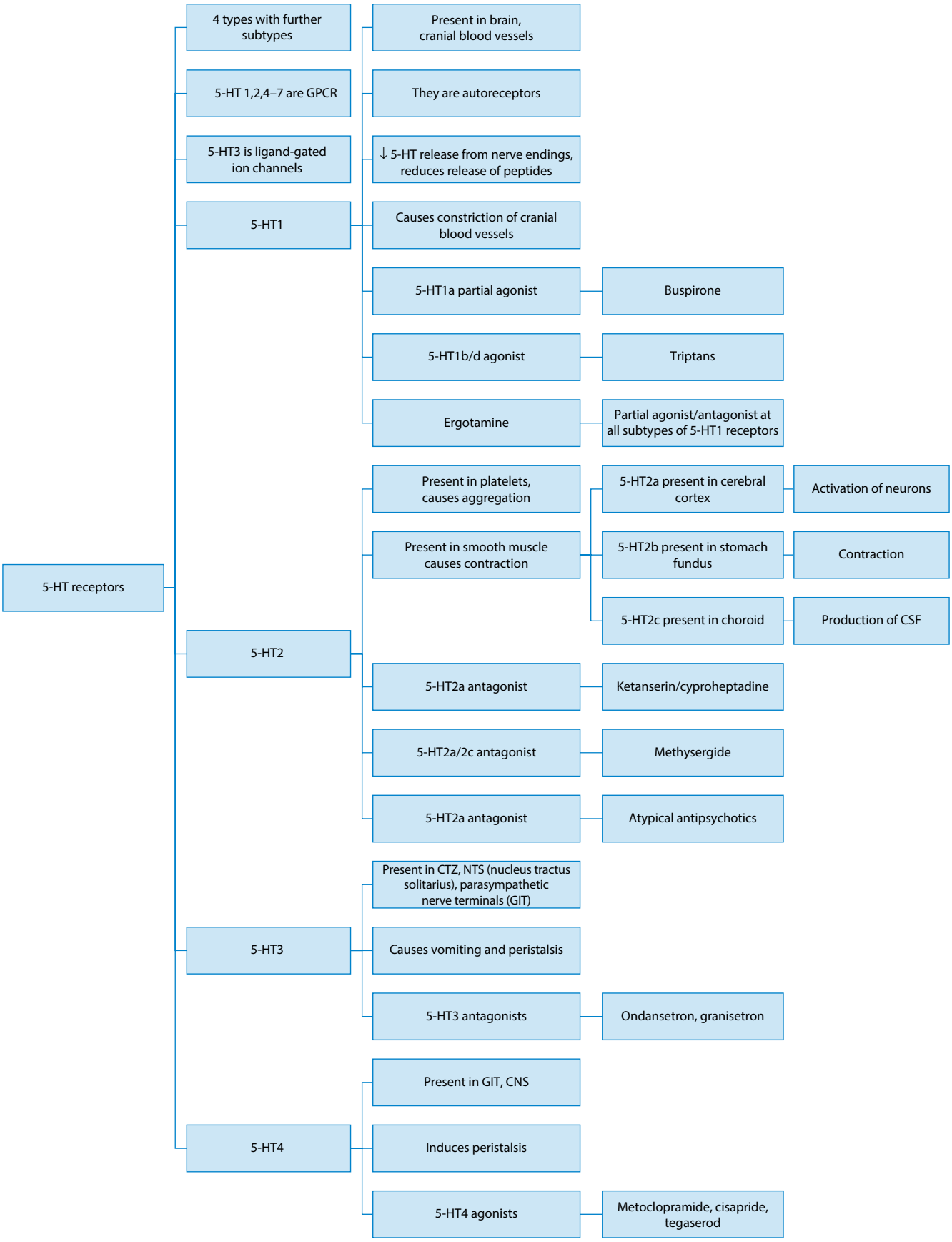
32

5-Hydroxytryptamine agonists and antagonists and drug treatment of migraine

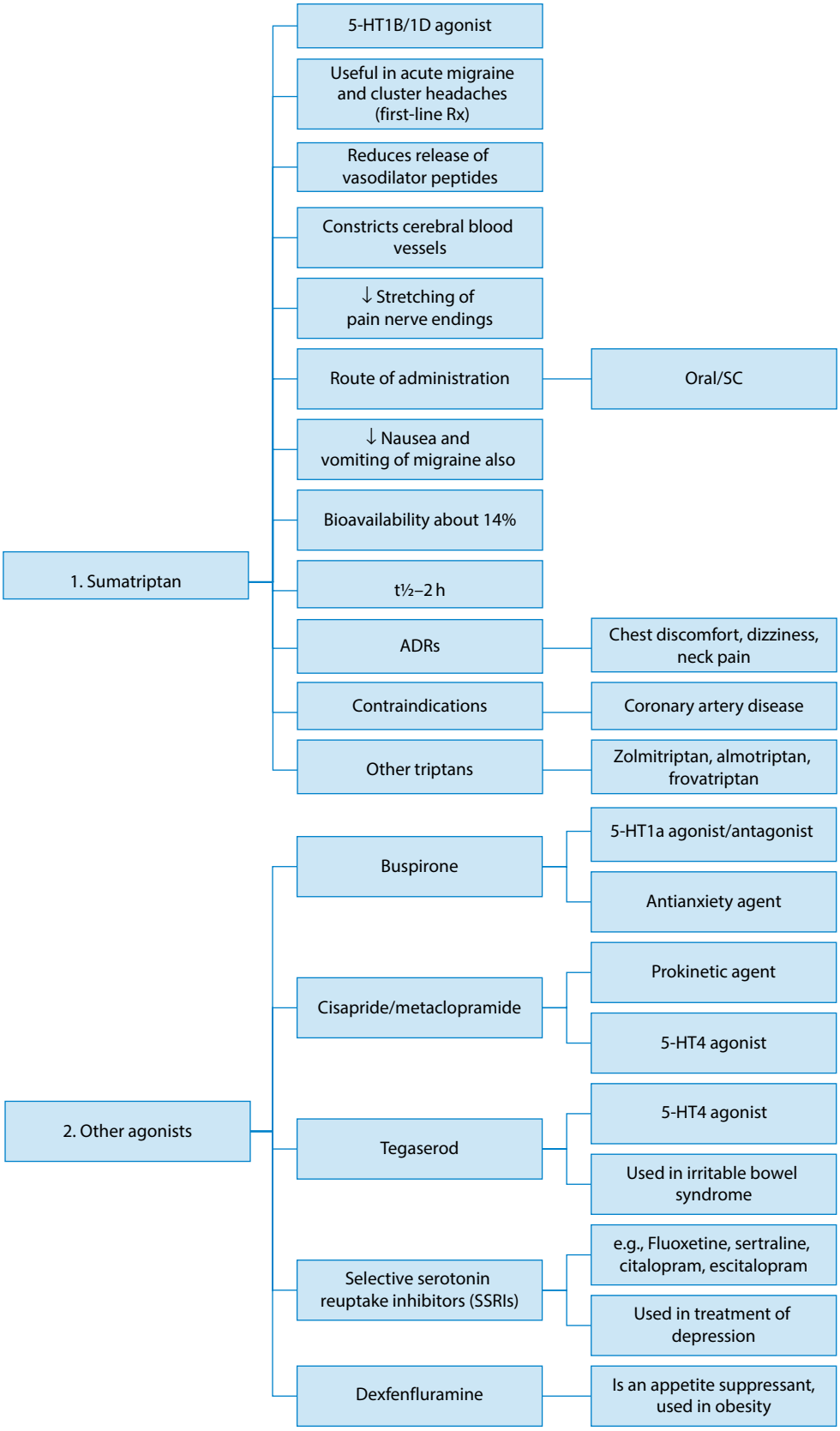
32.1 5-HYDROXYTRYPTAMINE – INTRODUCTION



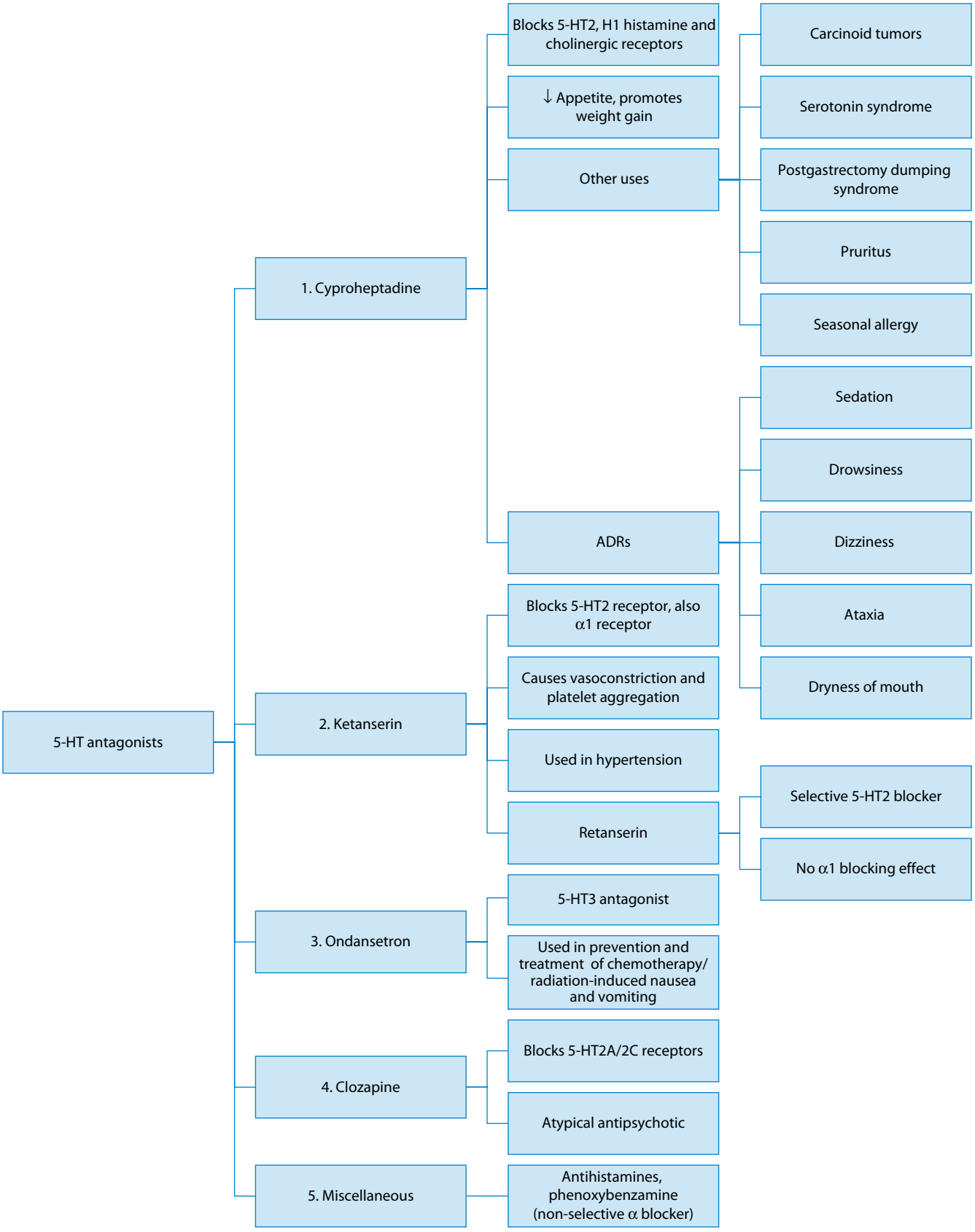
32.2 5-HT RECEPTORS



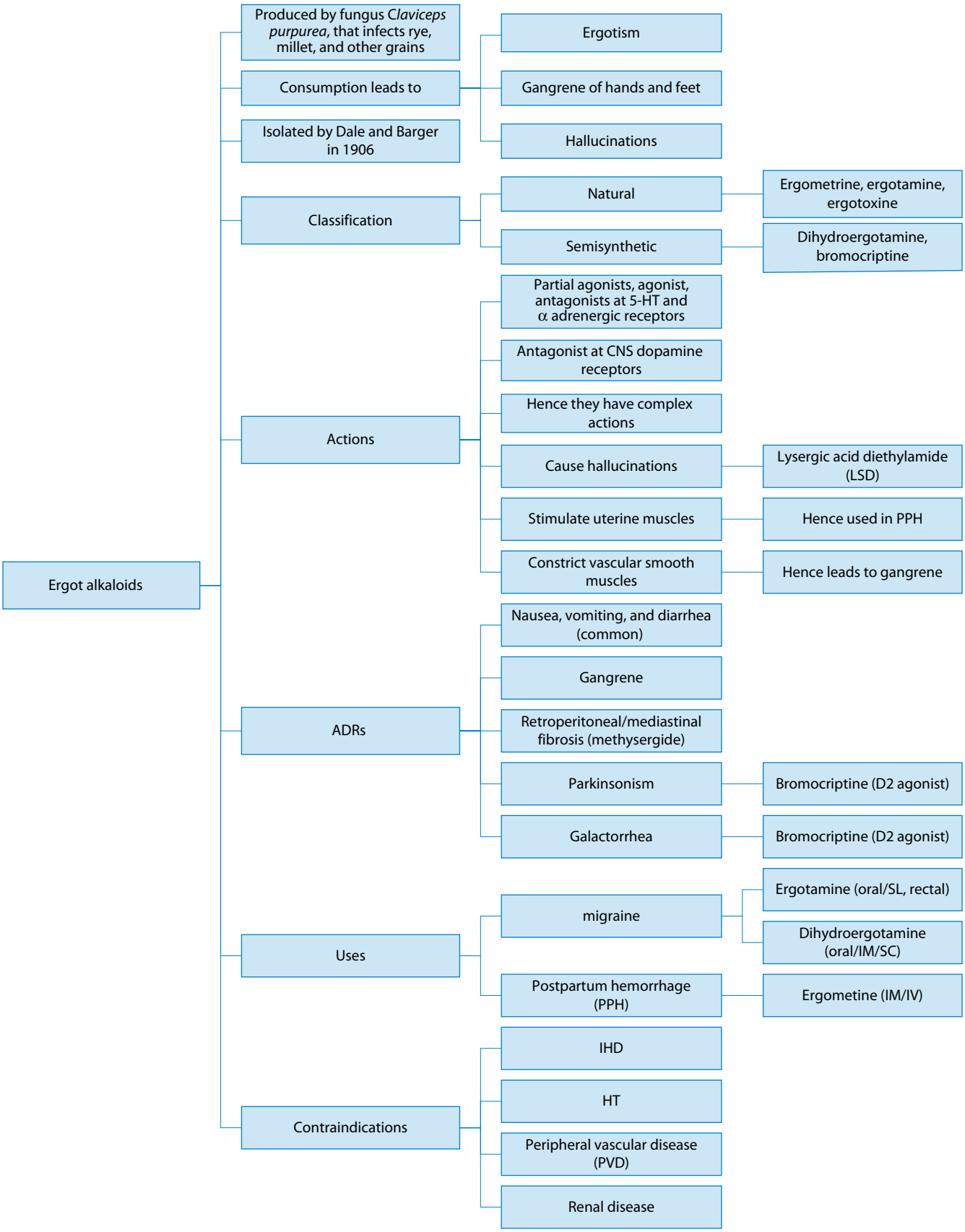
32.3 5-HT AGONISTS



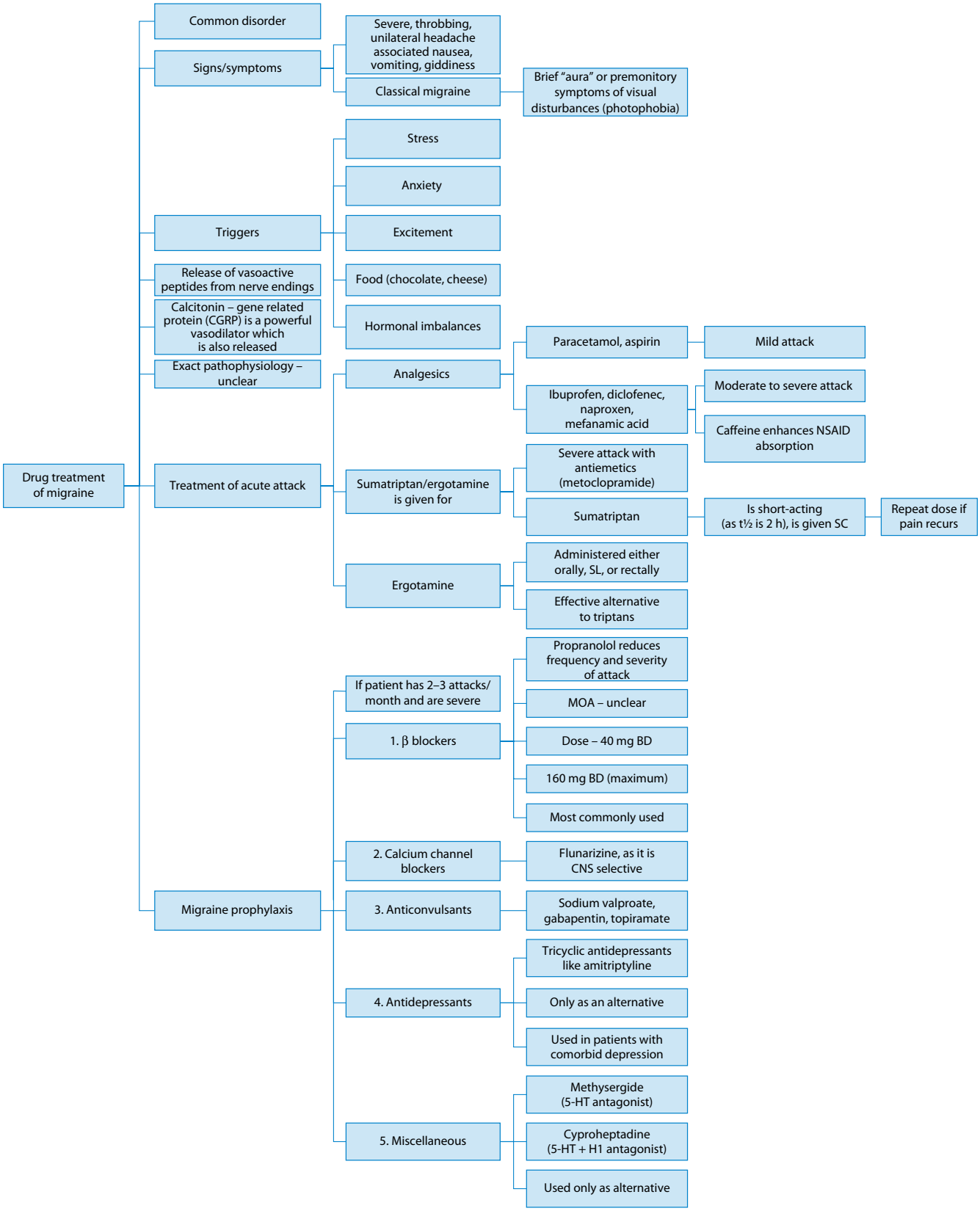
32.4 5-HT ANTAGONISTS



32.5 ERGOT ALKALOIDS



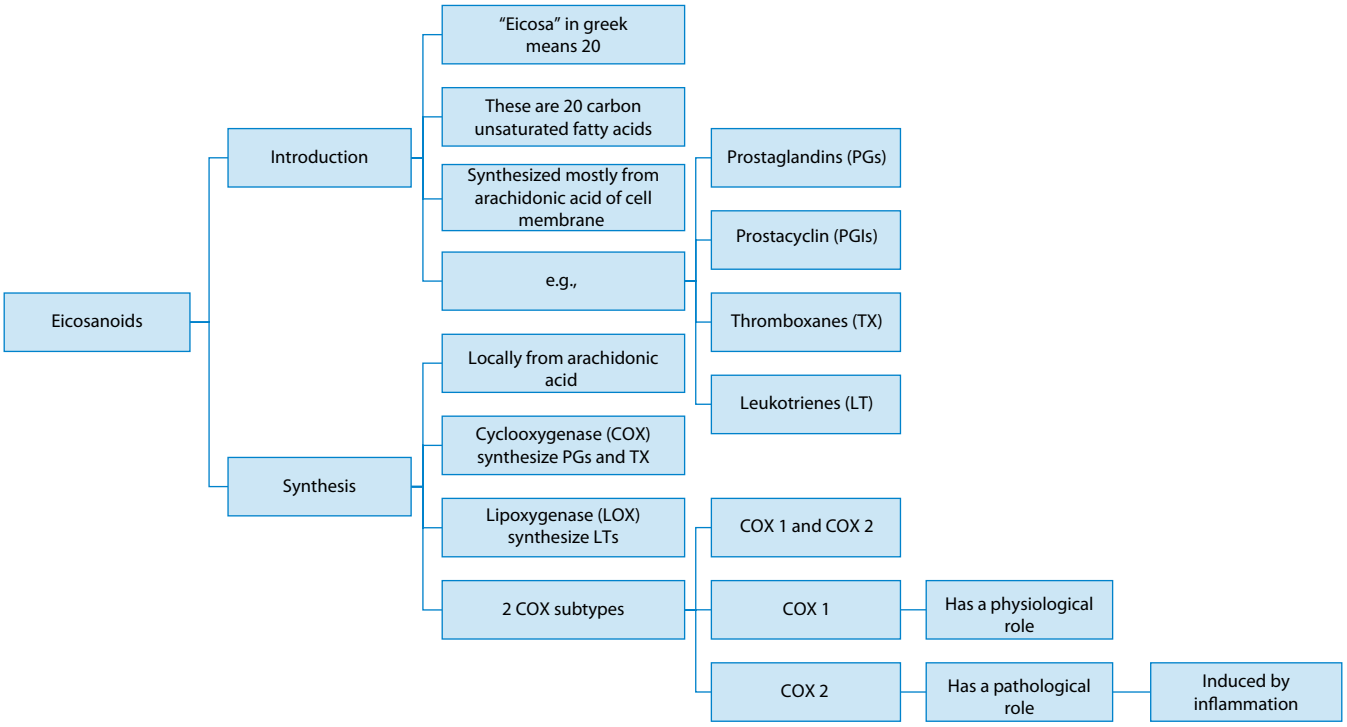
32.6 DRUG TREATMENT OF MIGRAINE



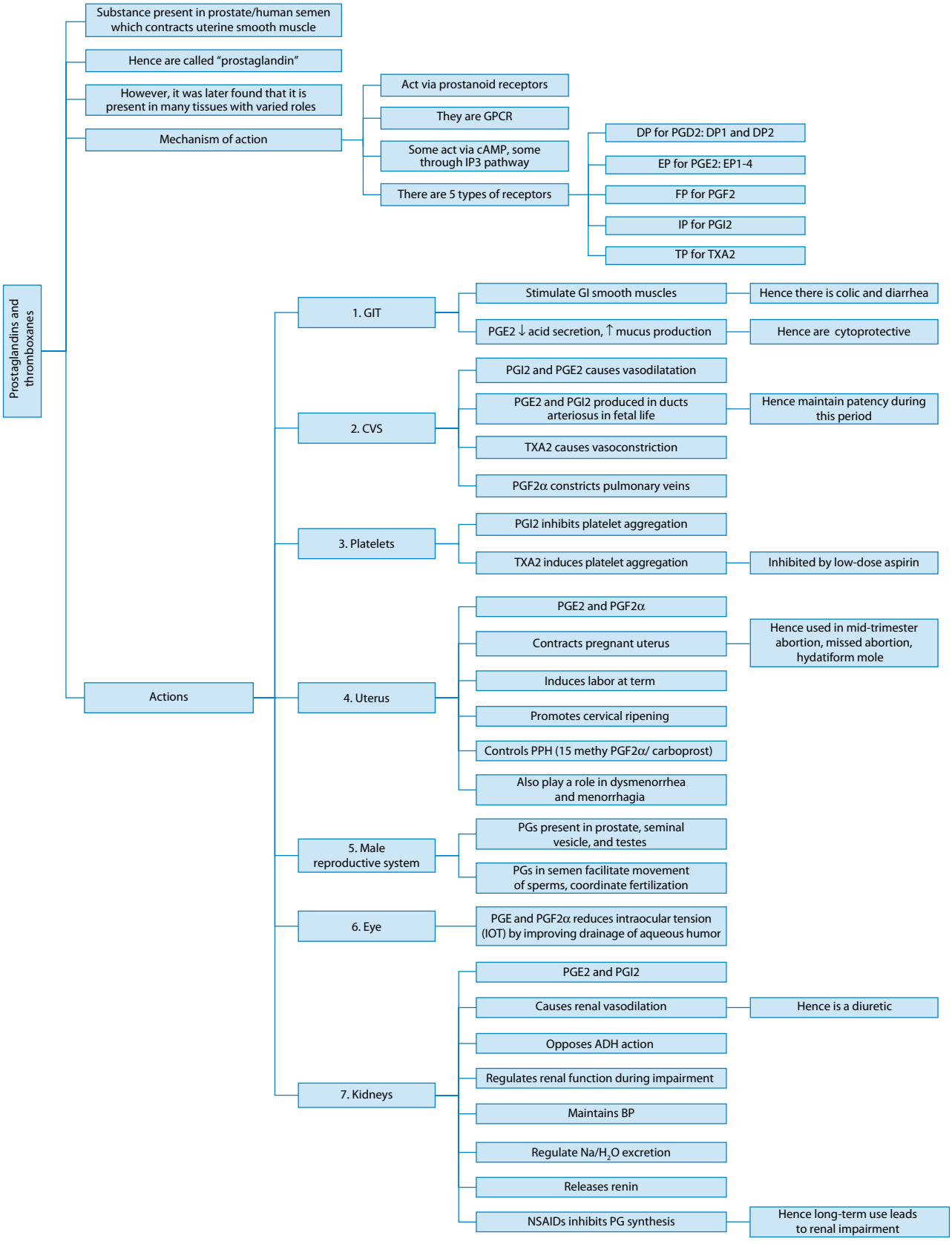
33

Eicosanoids and leukotrienes

33.1 EICOSANOIDS – INTRODUCTION AND SYNTHESIS

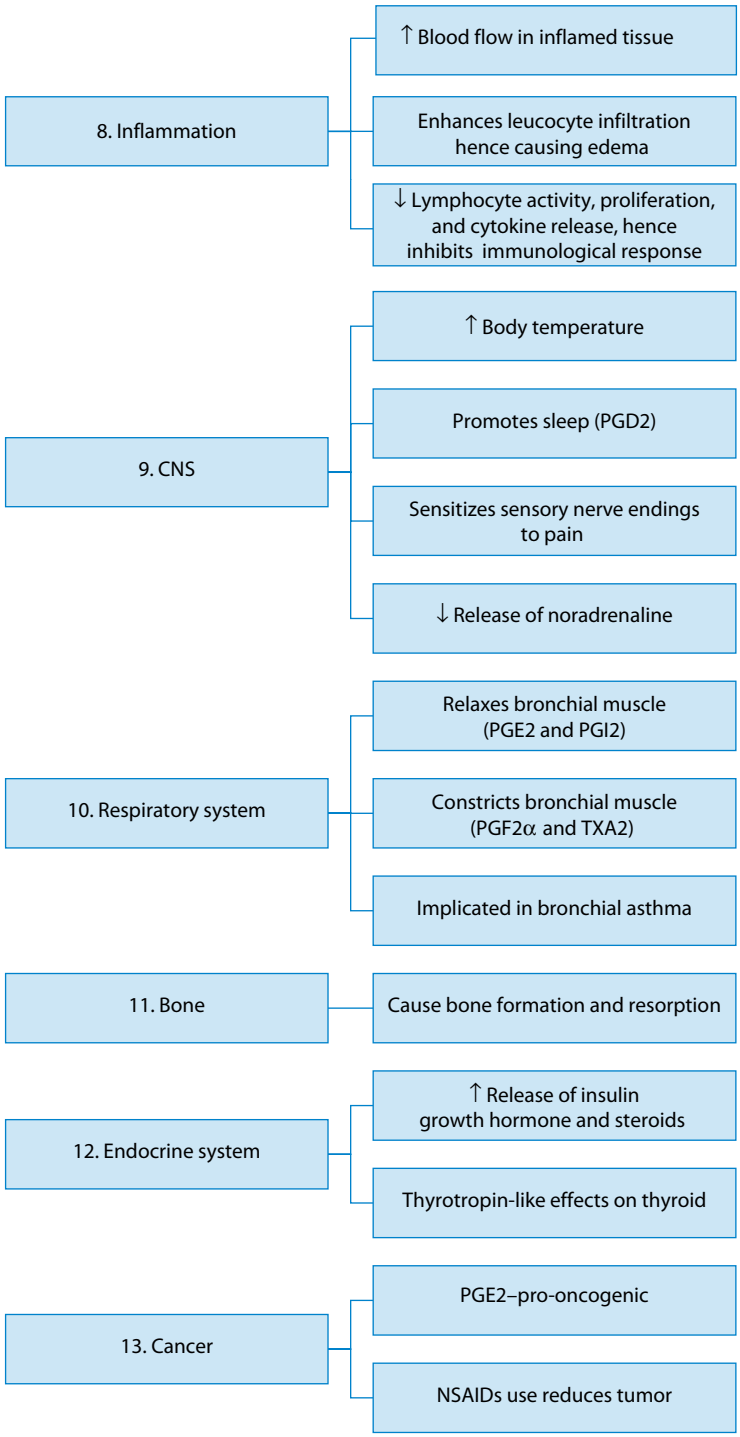


33.2 PROSTAGLANDINS AND THROMBOXANES – MECHANISM OF ACTION AND ACTIONS

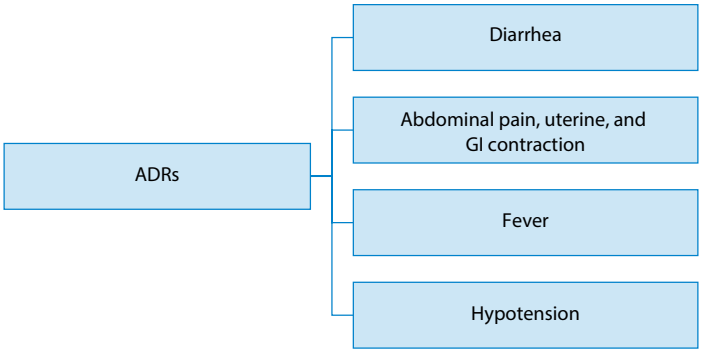


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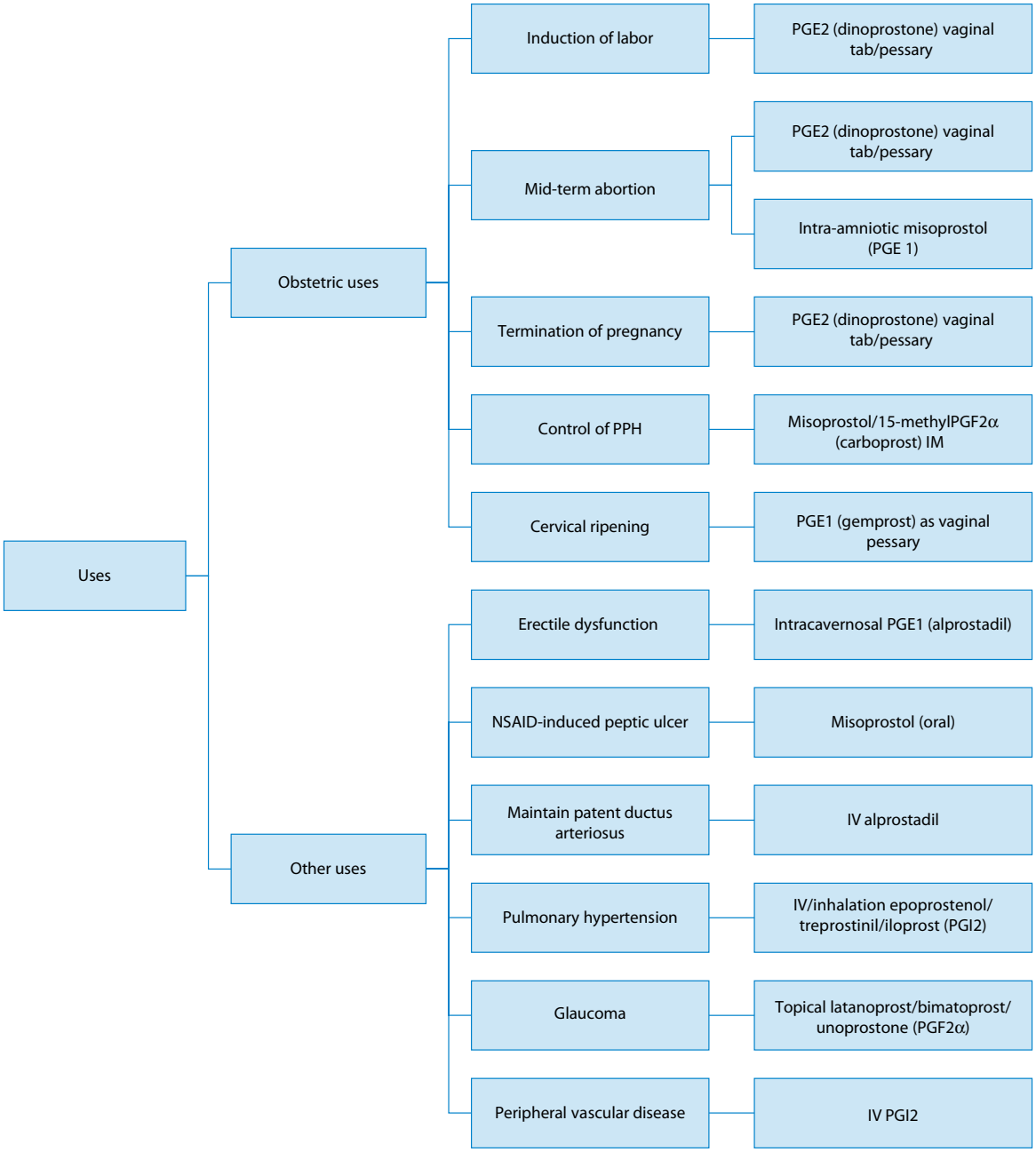
33.2 PROSTAGLANDINS AND THROMBOXANES – MECHANISM OF ACTION AND ACTIONS (Continued)



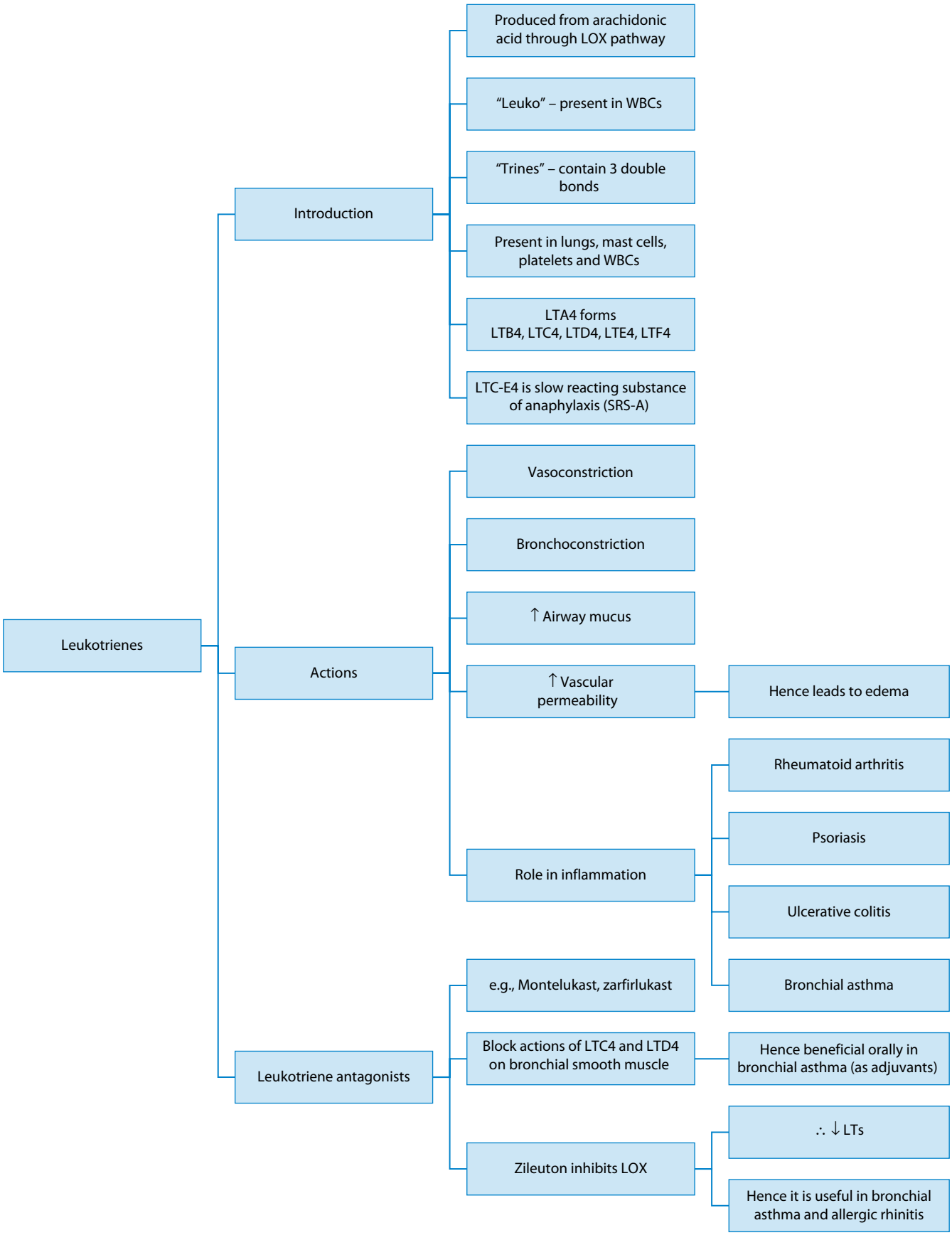
33.3 ADR



33.4 USES



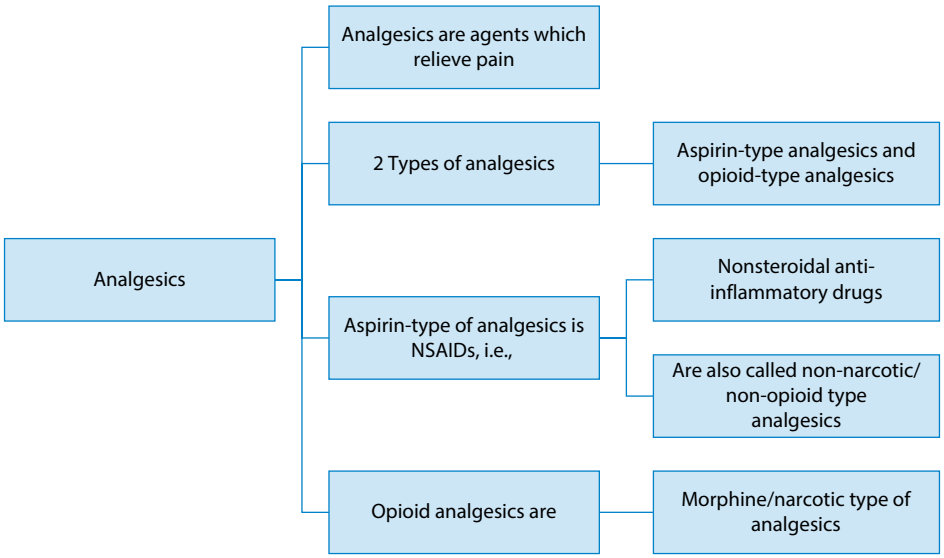
33.5 LEUKOTRIENES – INTRODUCTION, ACTIONS, LEUKOTRIENE ANTAGONISTS AND PLATELET-ACTIVATING FACTOR



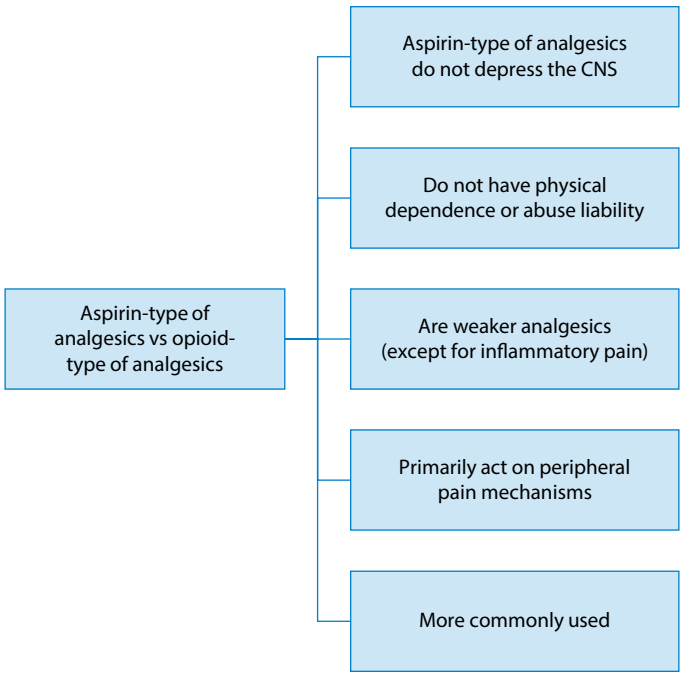
34

Nonsteroidal anti-inflammatory drugs (NSAIDs)

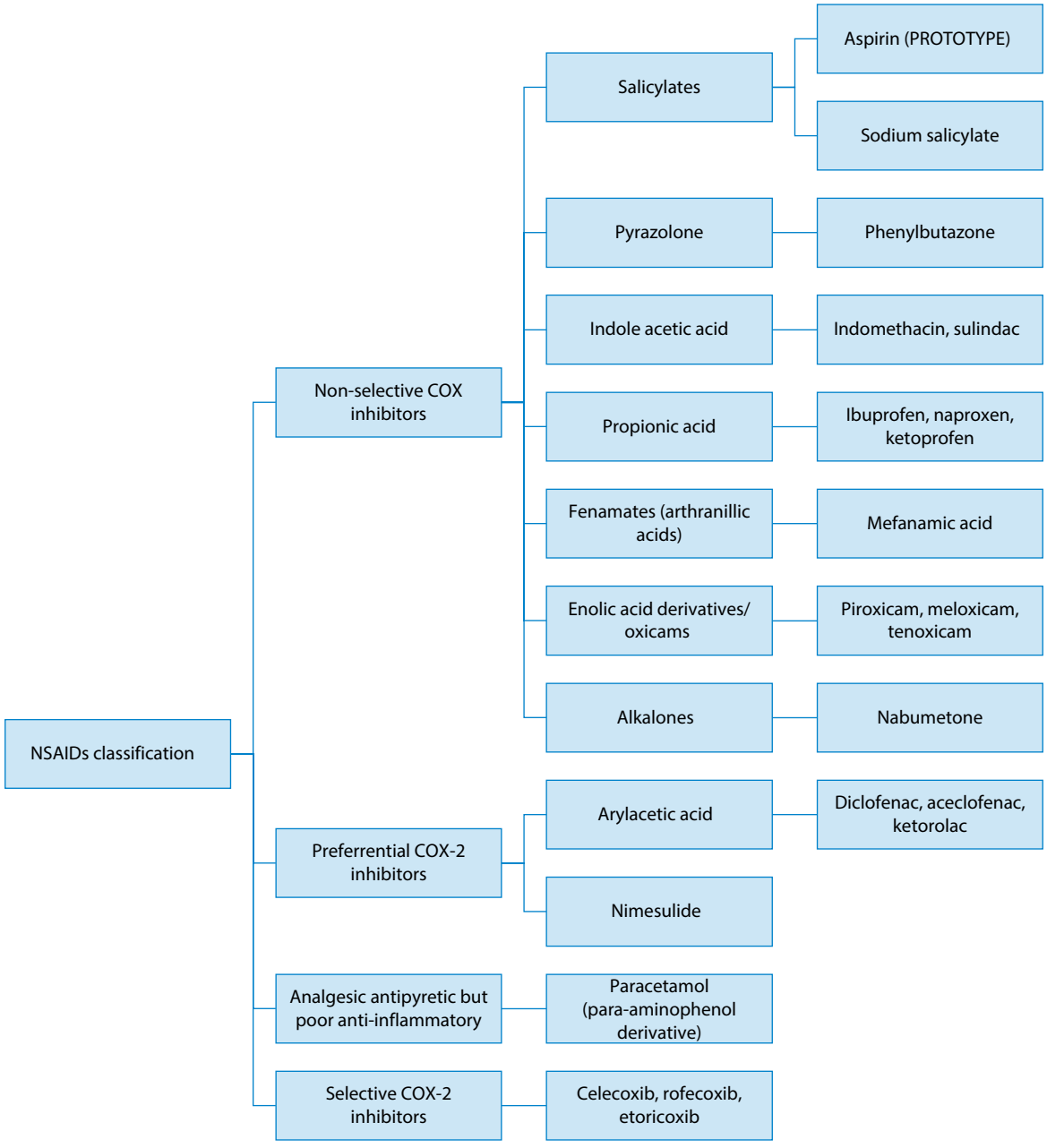
34.1 ANALGESICS



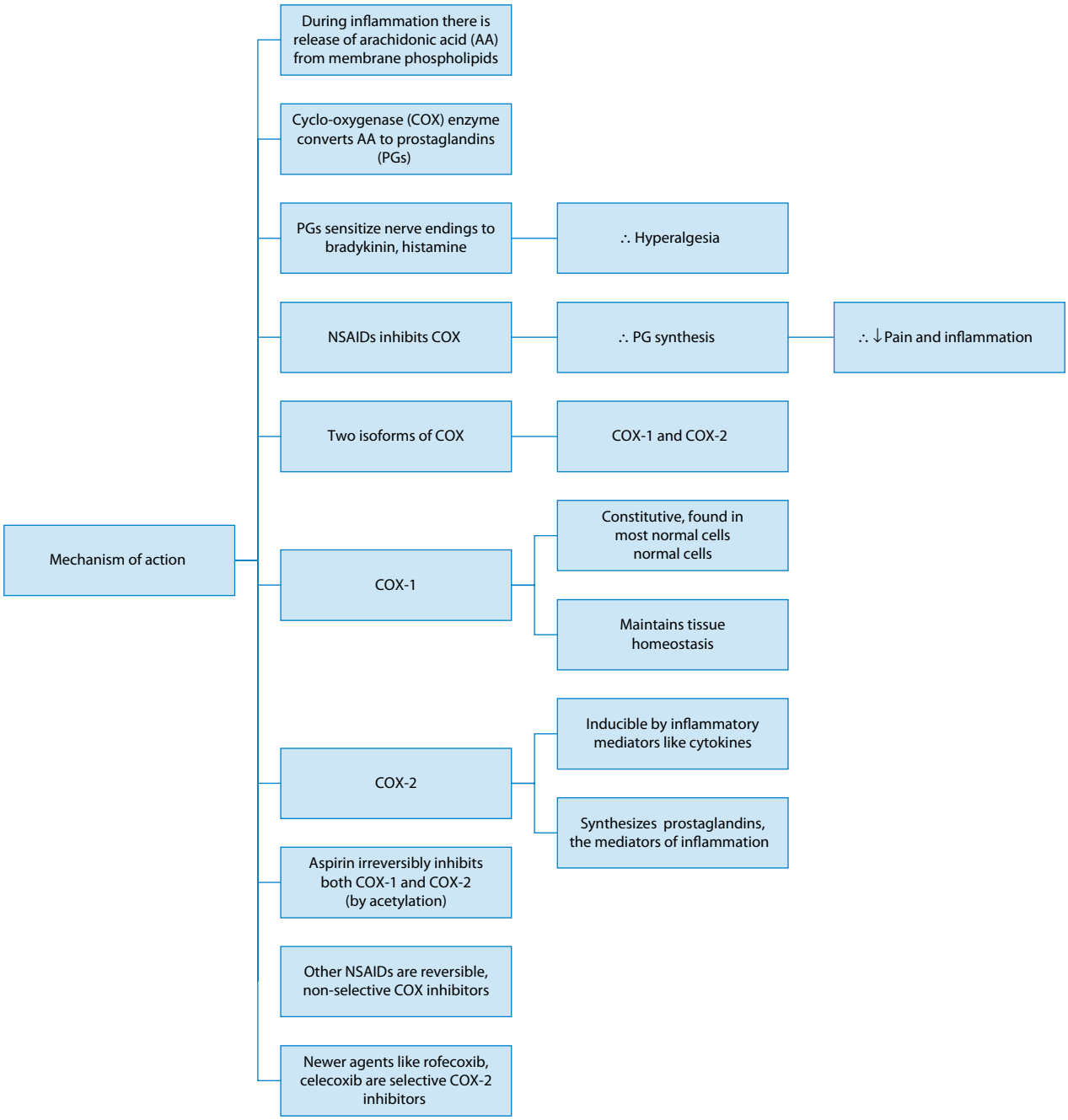
34.2 ASPIRIN-TYPE OF ANALGESICS vs. OPIOID-TYPE OF ANALGESICS



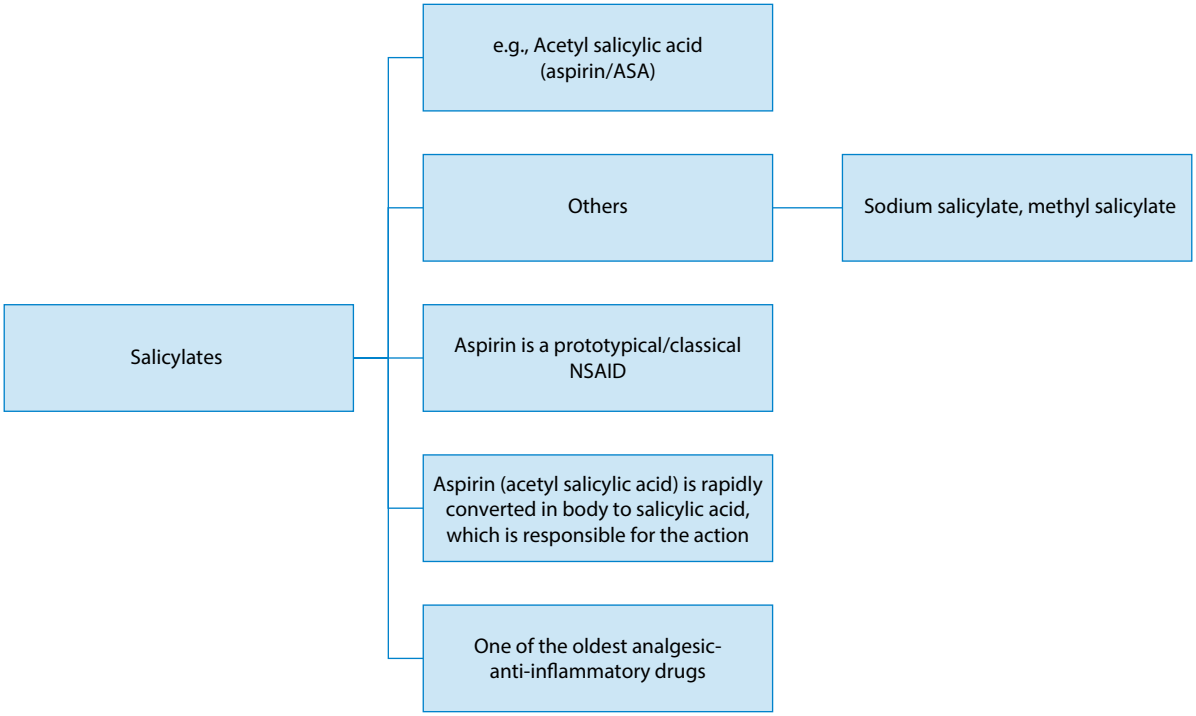
34.3 NSAIDs CLASSIFICATION



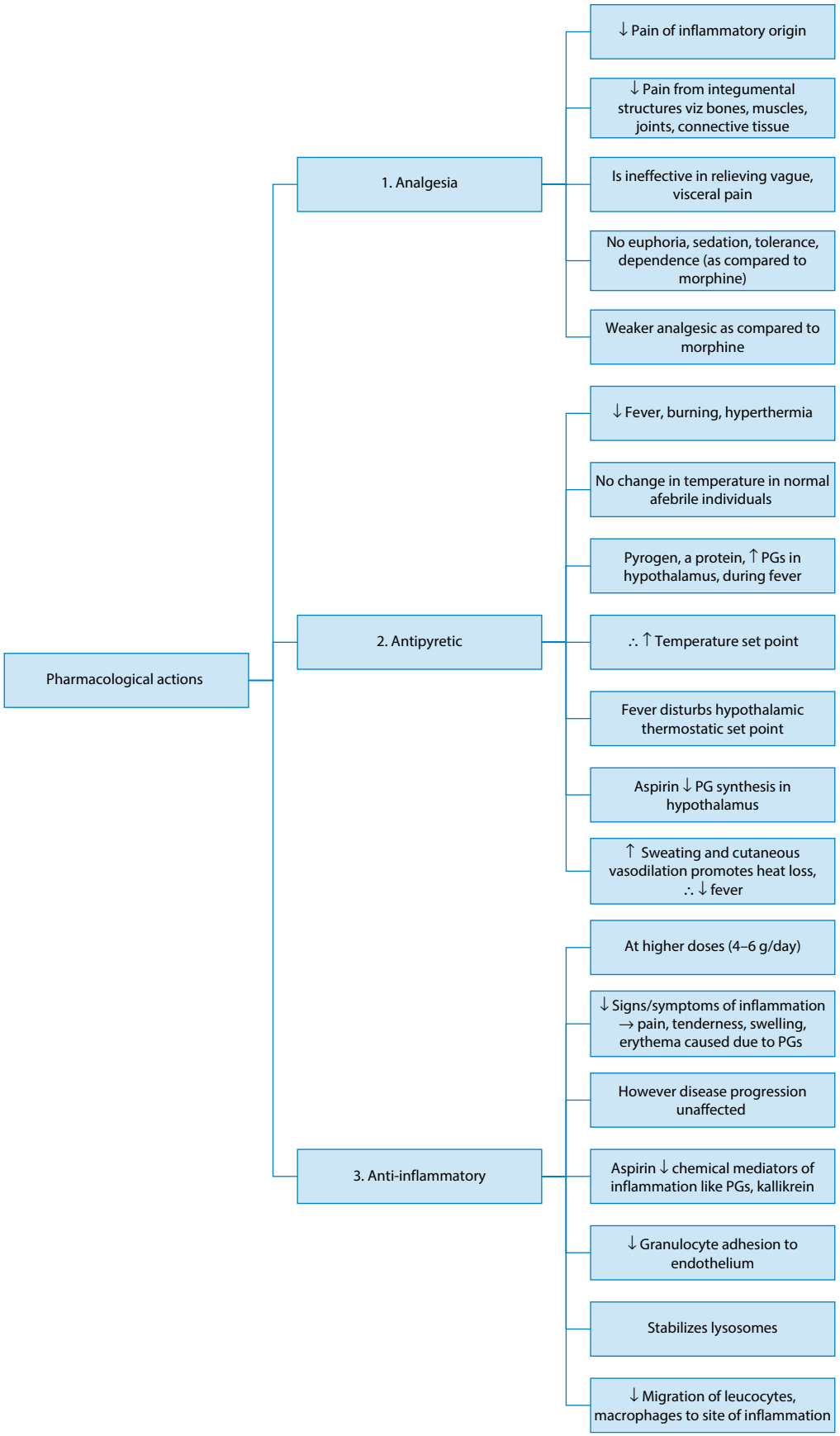
34.4 MECHANISM OF ACTION



34.5 SALICYLATES

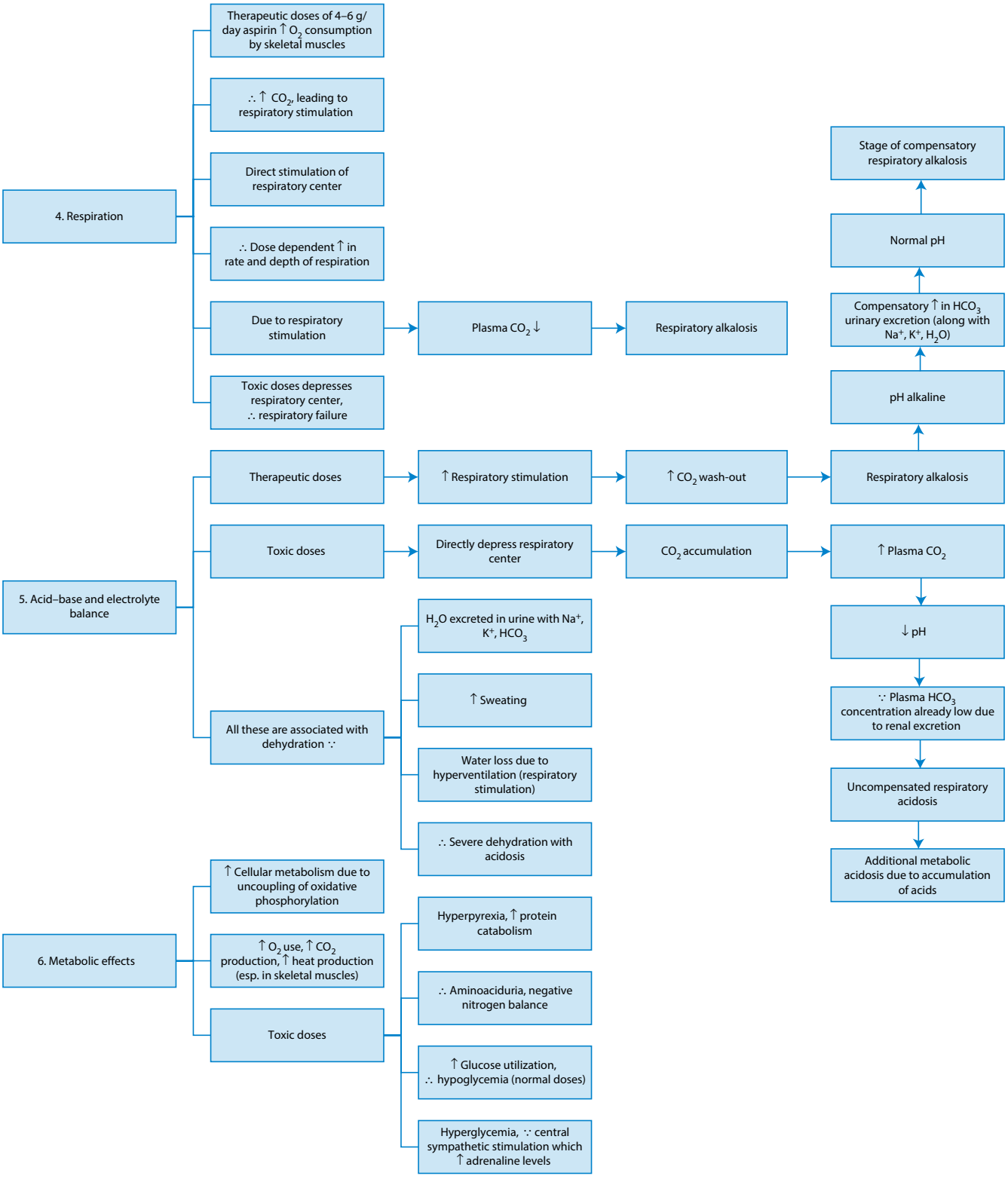


34.6 PHARMACOLOGICAL ACTIONS



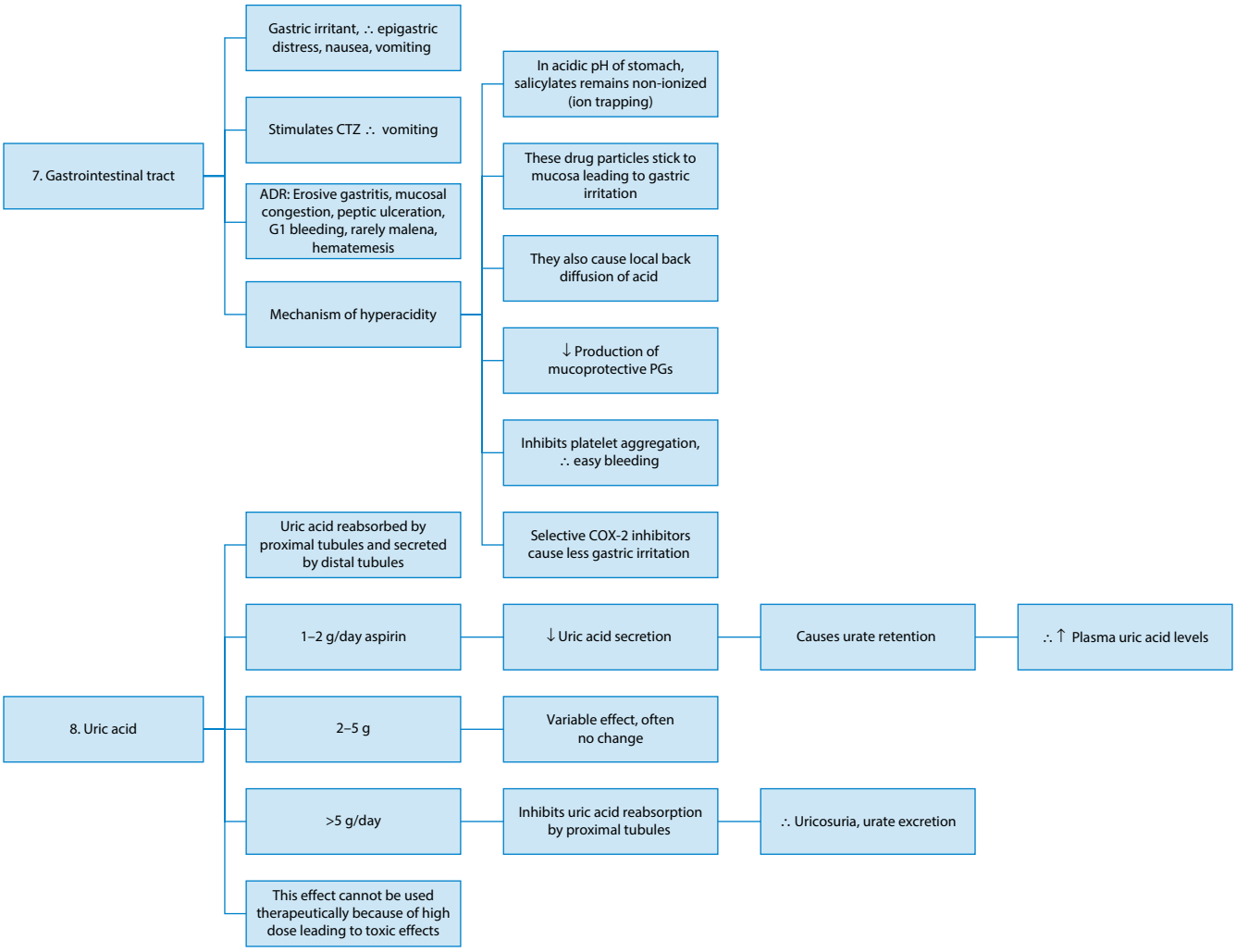
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34.6 PHARMACOLOGICAL ACTIONS (Continued)



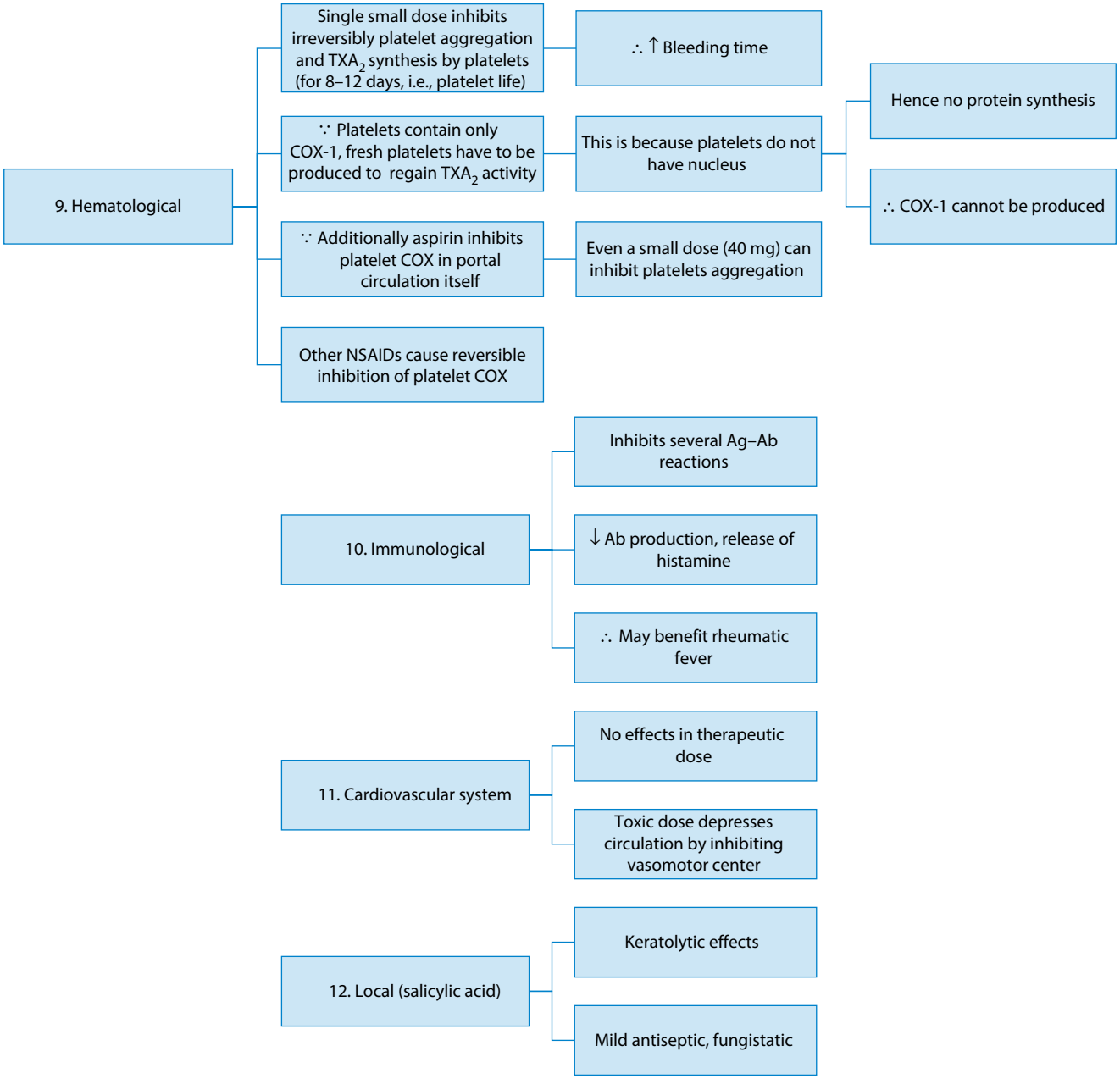
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34.6 PHARMACOLOGICAL ACTIONS (Continued)

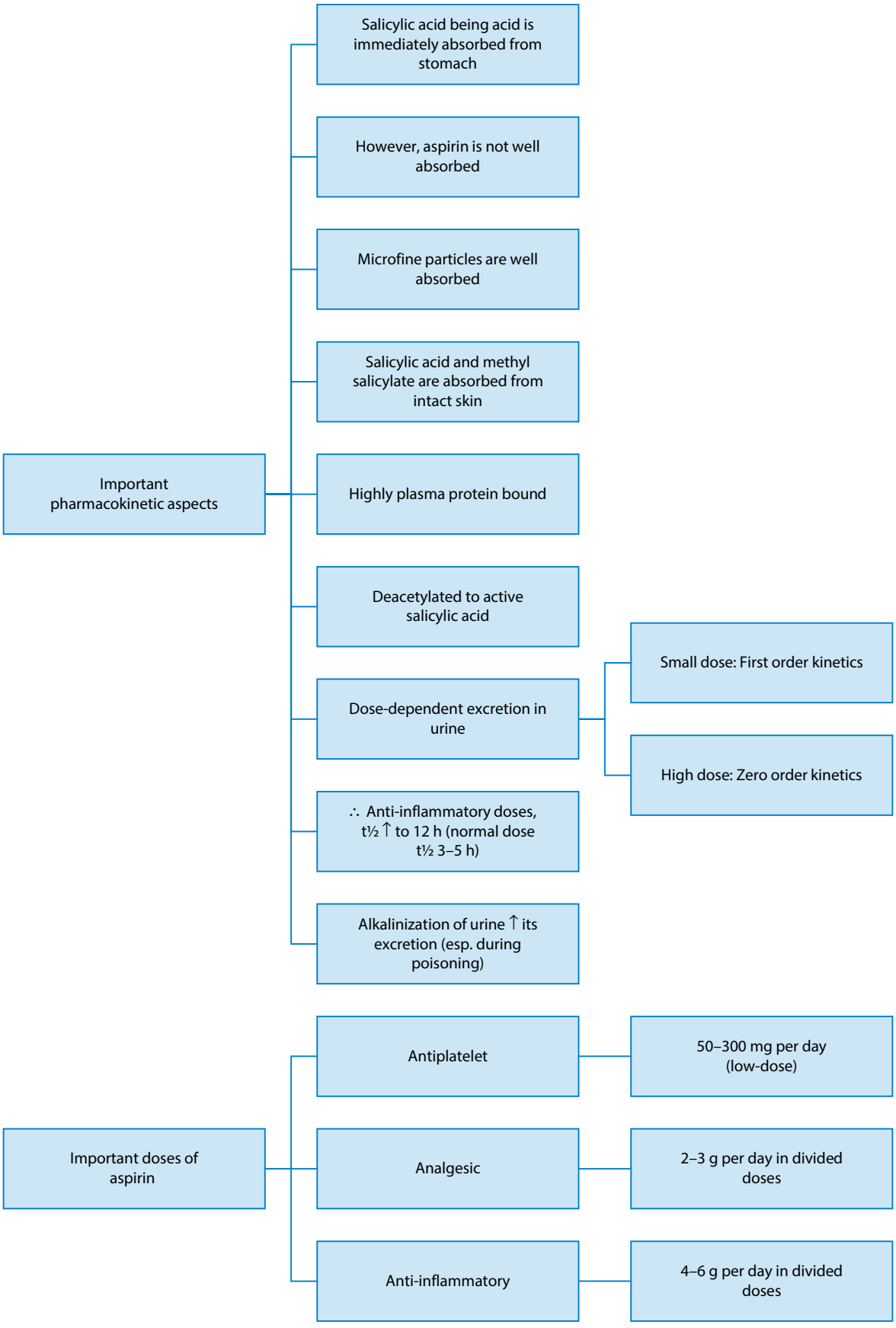


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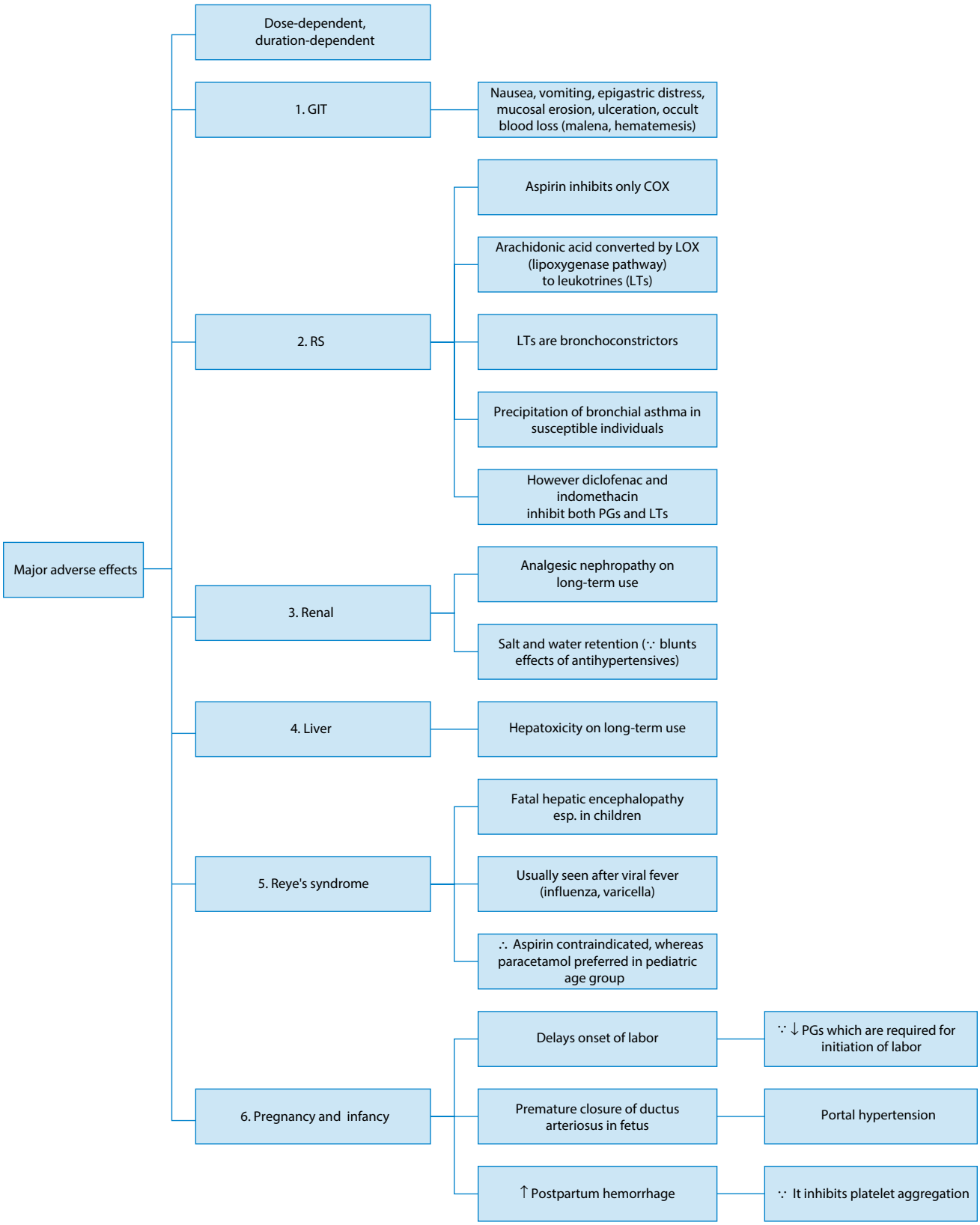
34.6 PHARMACOLOGICAL ACTIONS (Continued)



34.7 IMPORTANT PHARMACOKINETIC ASPECTS AND DOSES

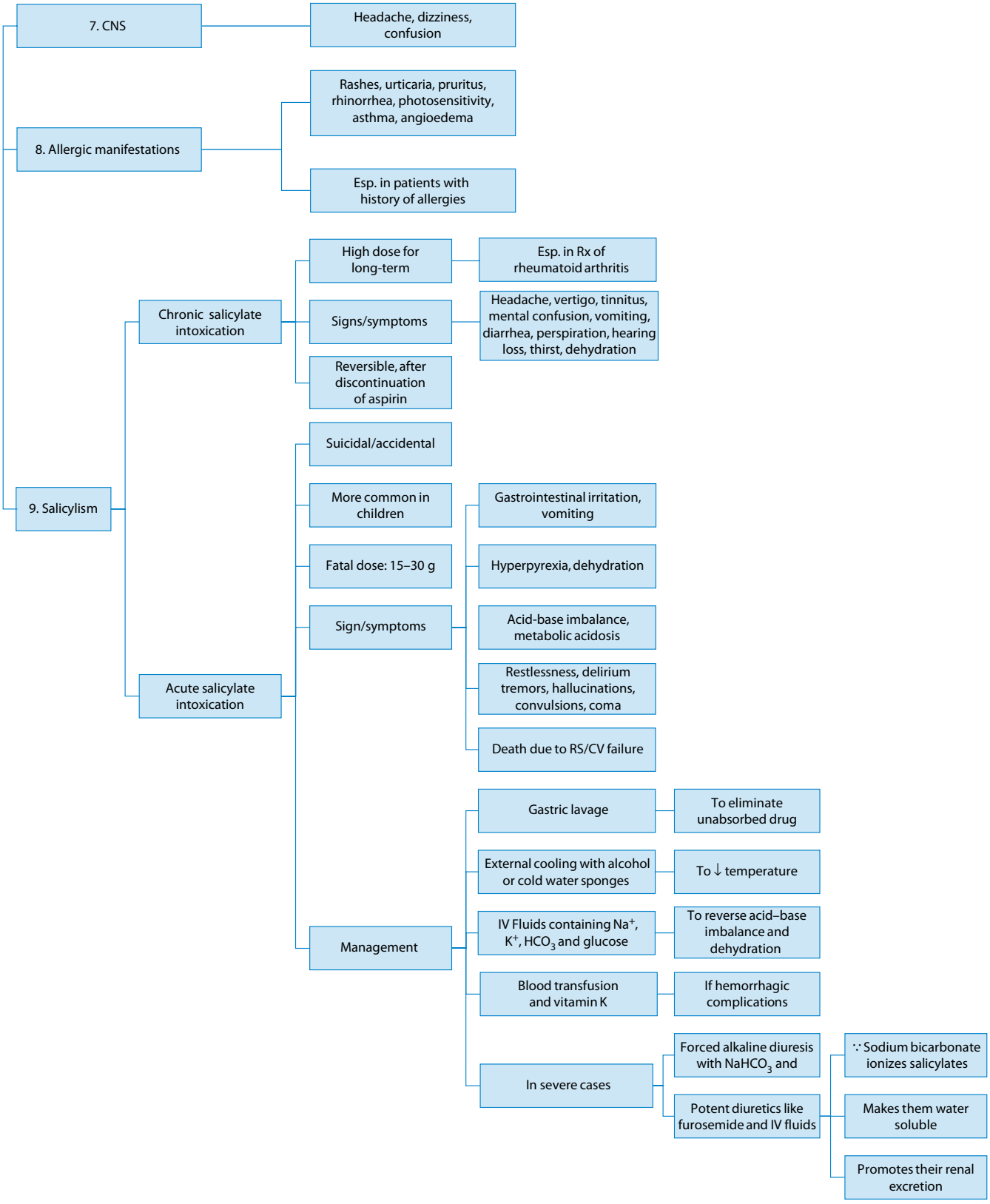


34.8 MAJOR ADVERSE EFFECTS

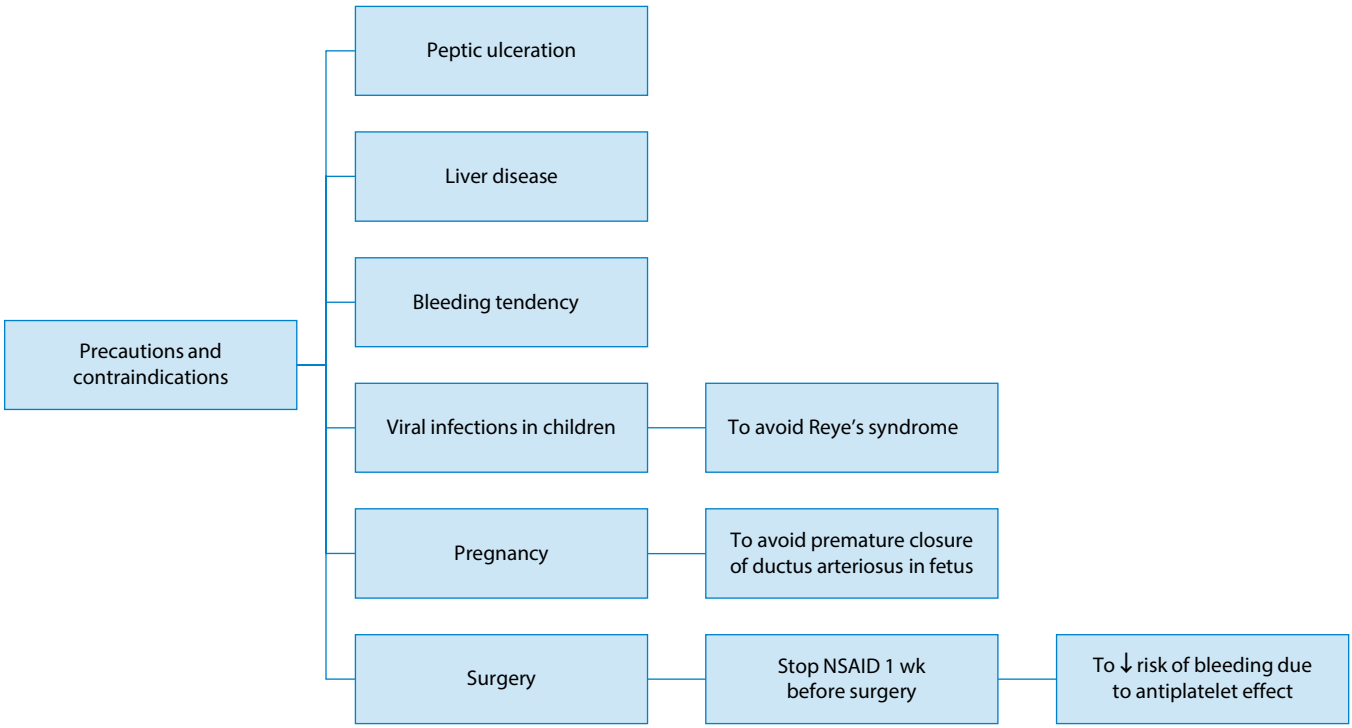


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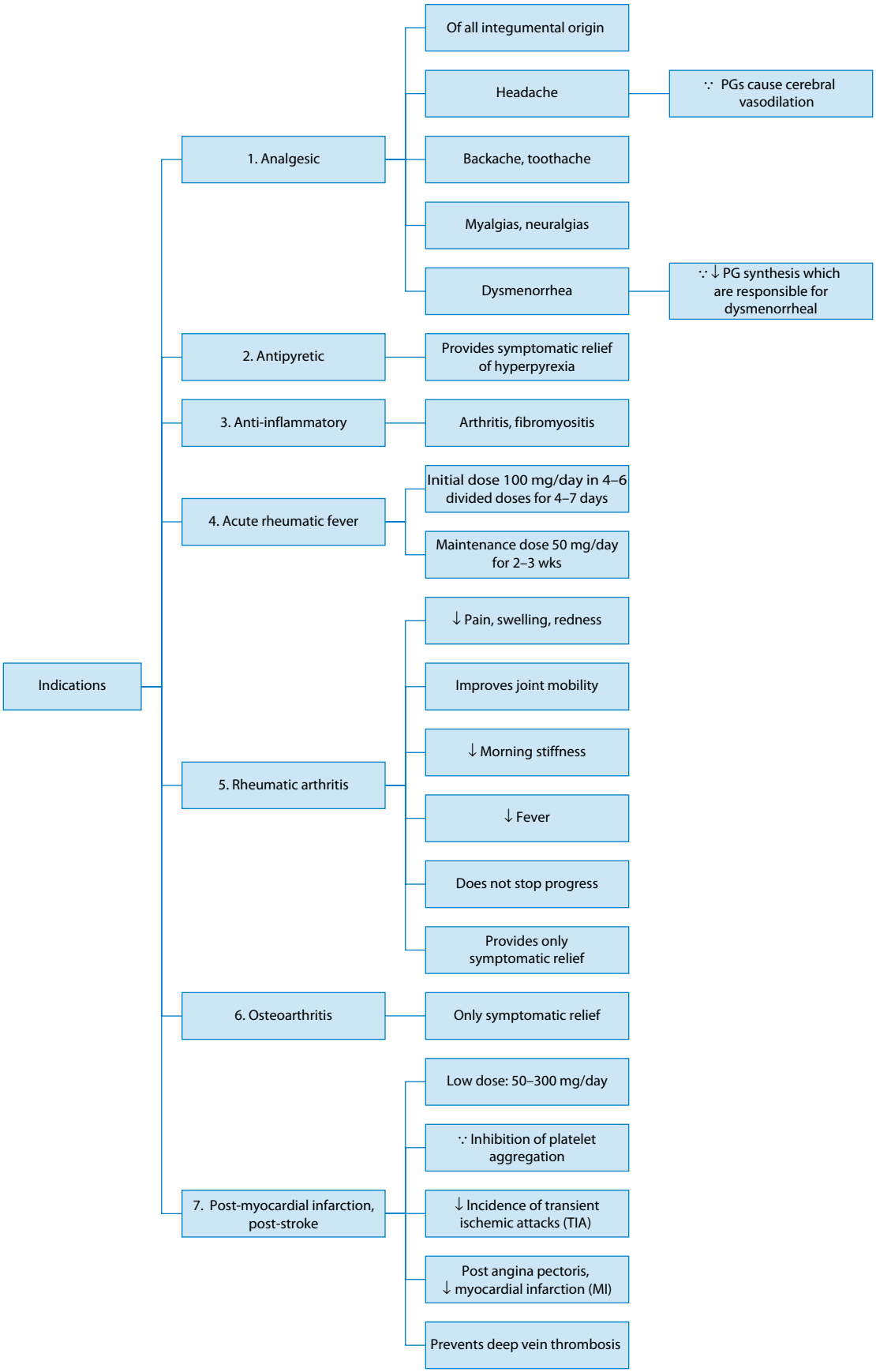
34.8 MAJOR ADVERSE EFFECTS (Continued)



34.9 PRECAUTIONS AND CONTRAINDICATIONS

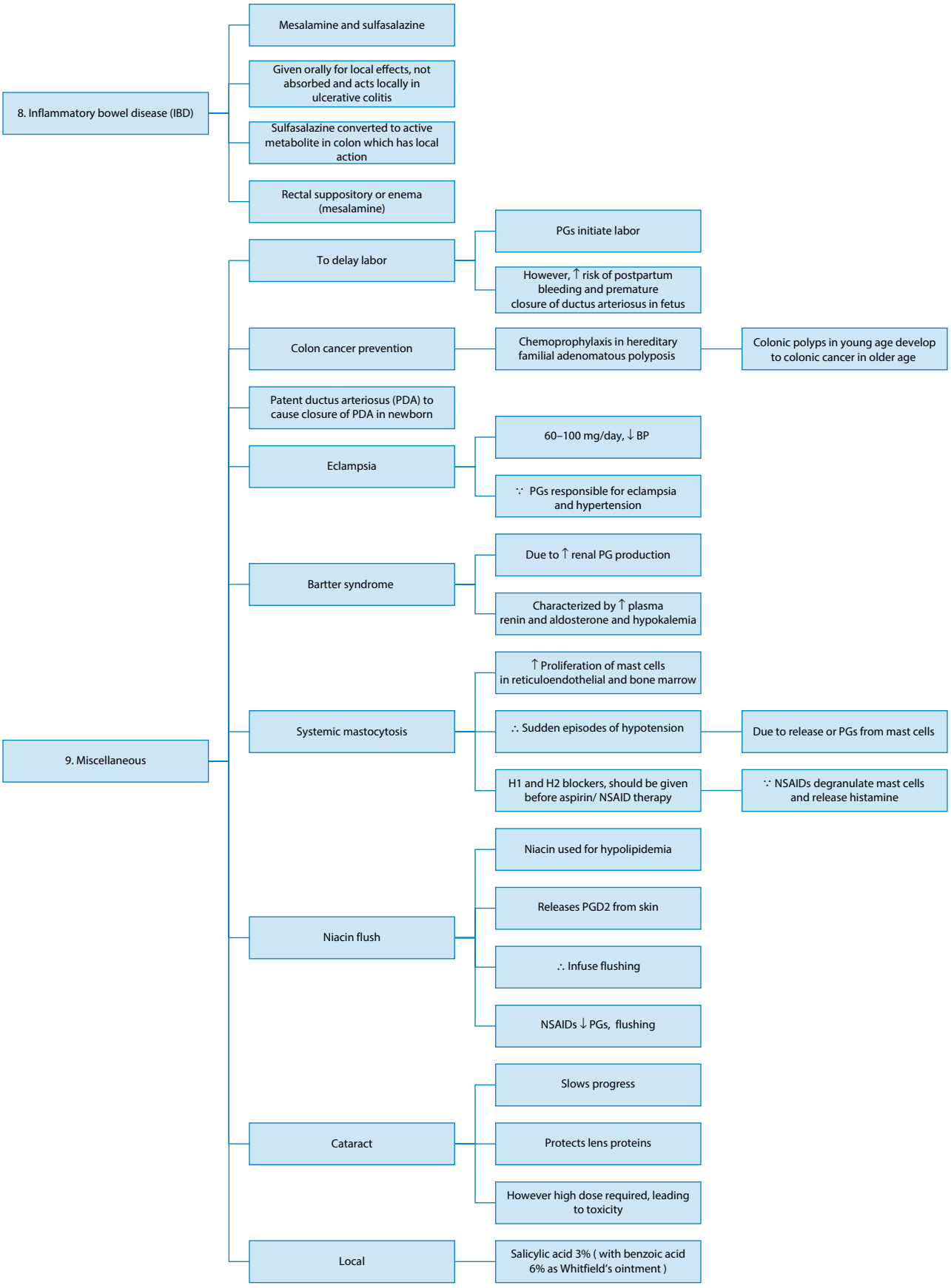


34.10 INDICATIONS

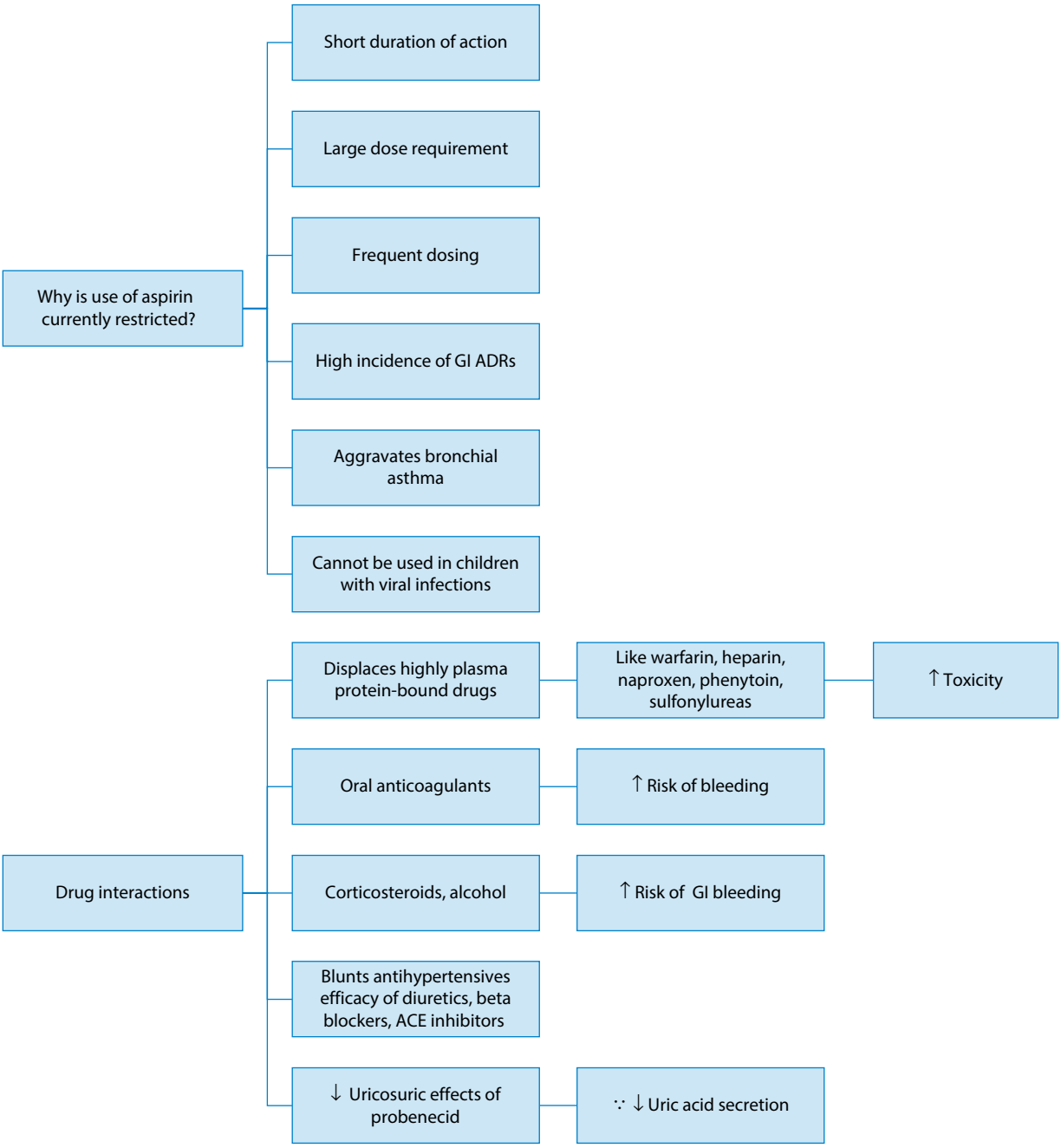


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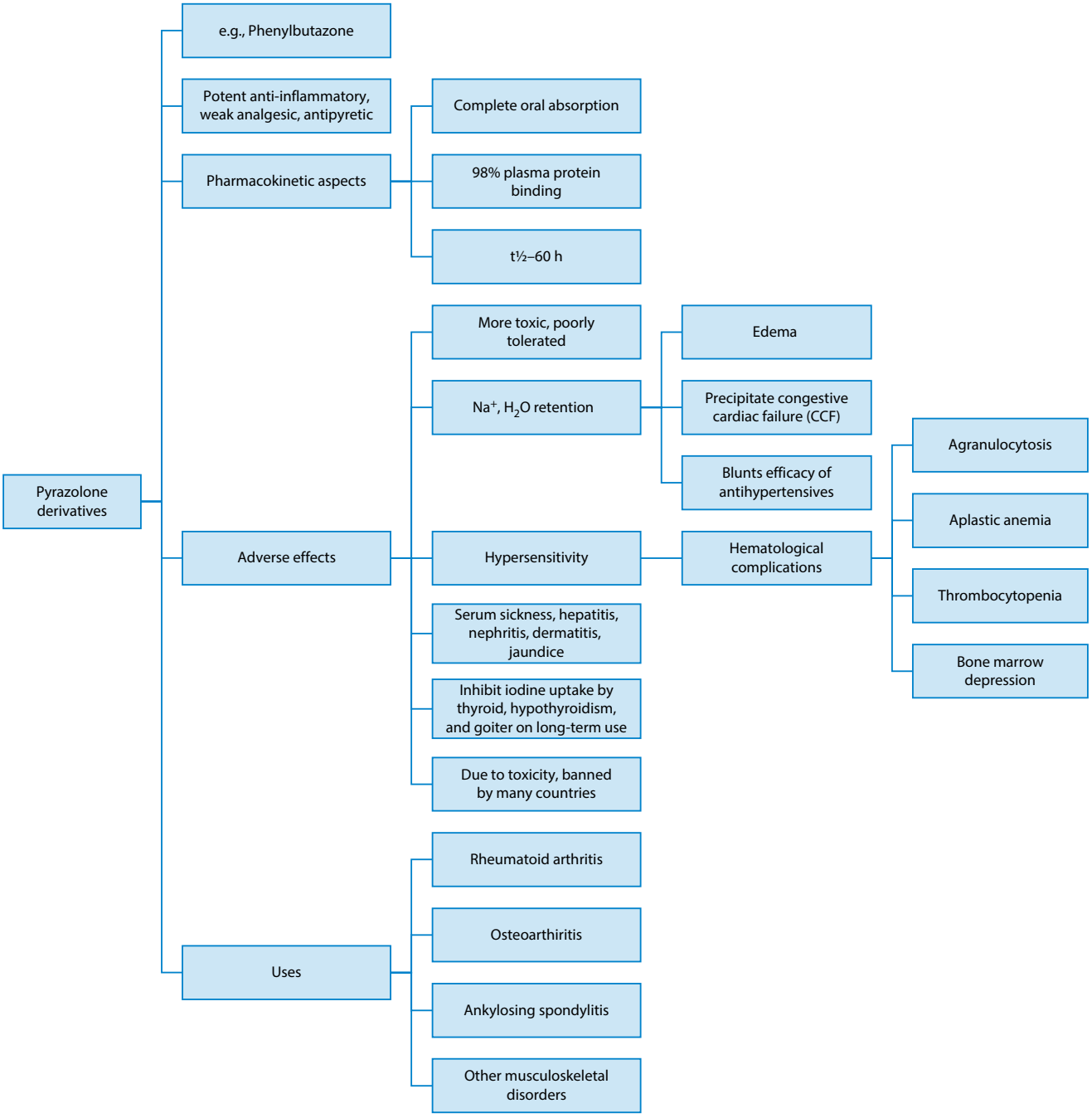
34.10 INDICATIONS (Continued)



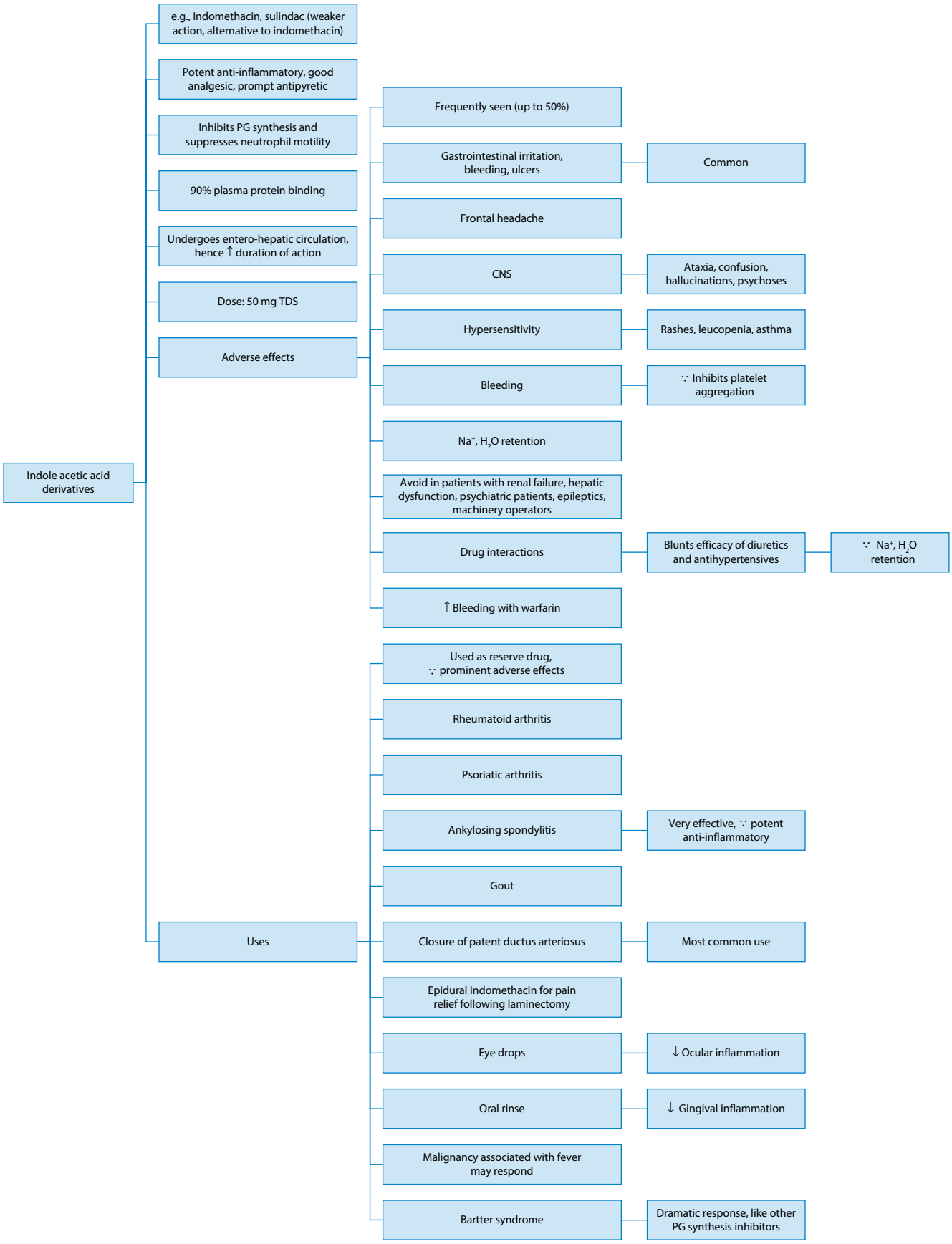
34.11 WHY USE OF ASPIRIN IS CURRENTLY RESTRICTED AND DRUG INTERACTIONS



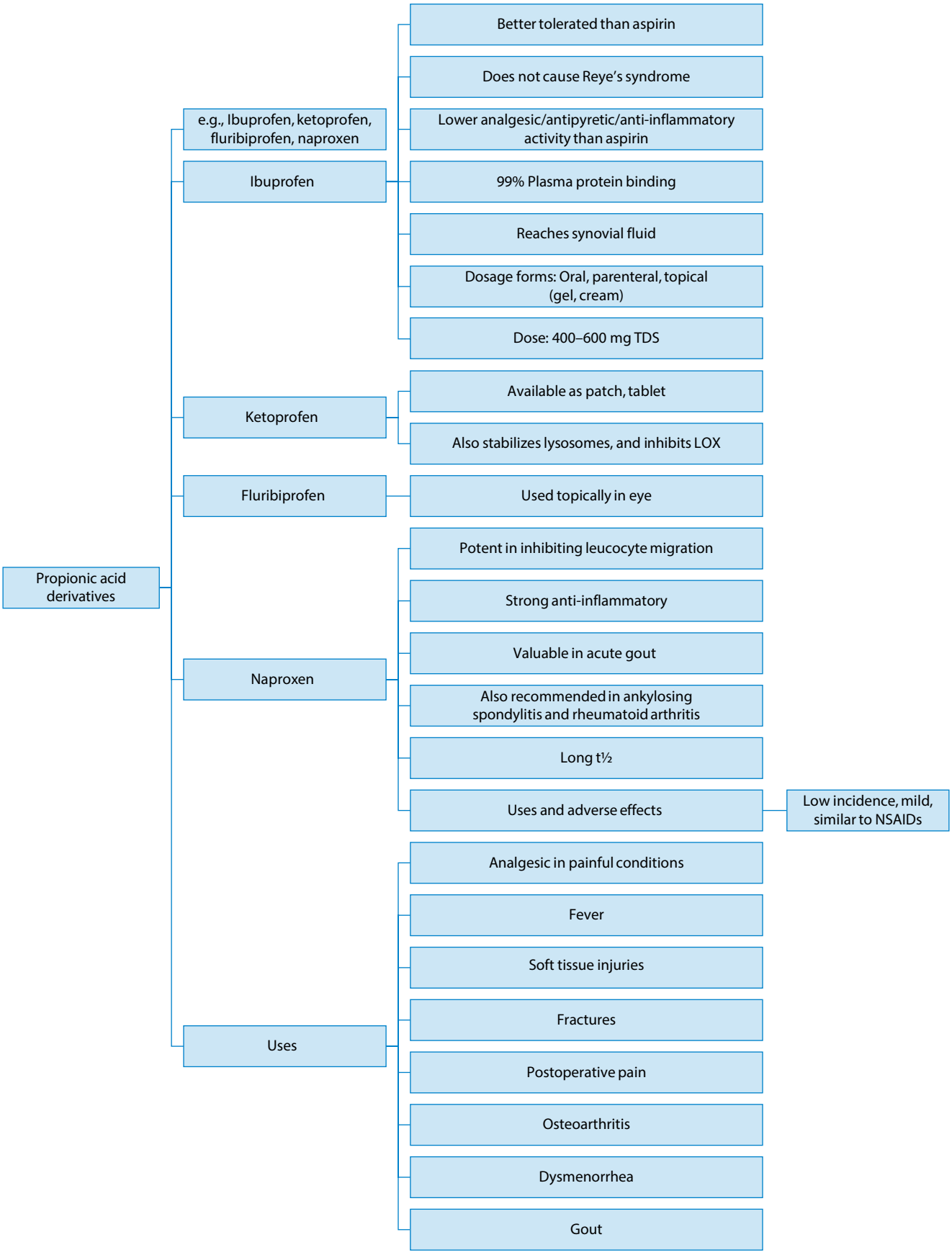
34.12 PYRAZOLONE DERIVATIVES



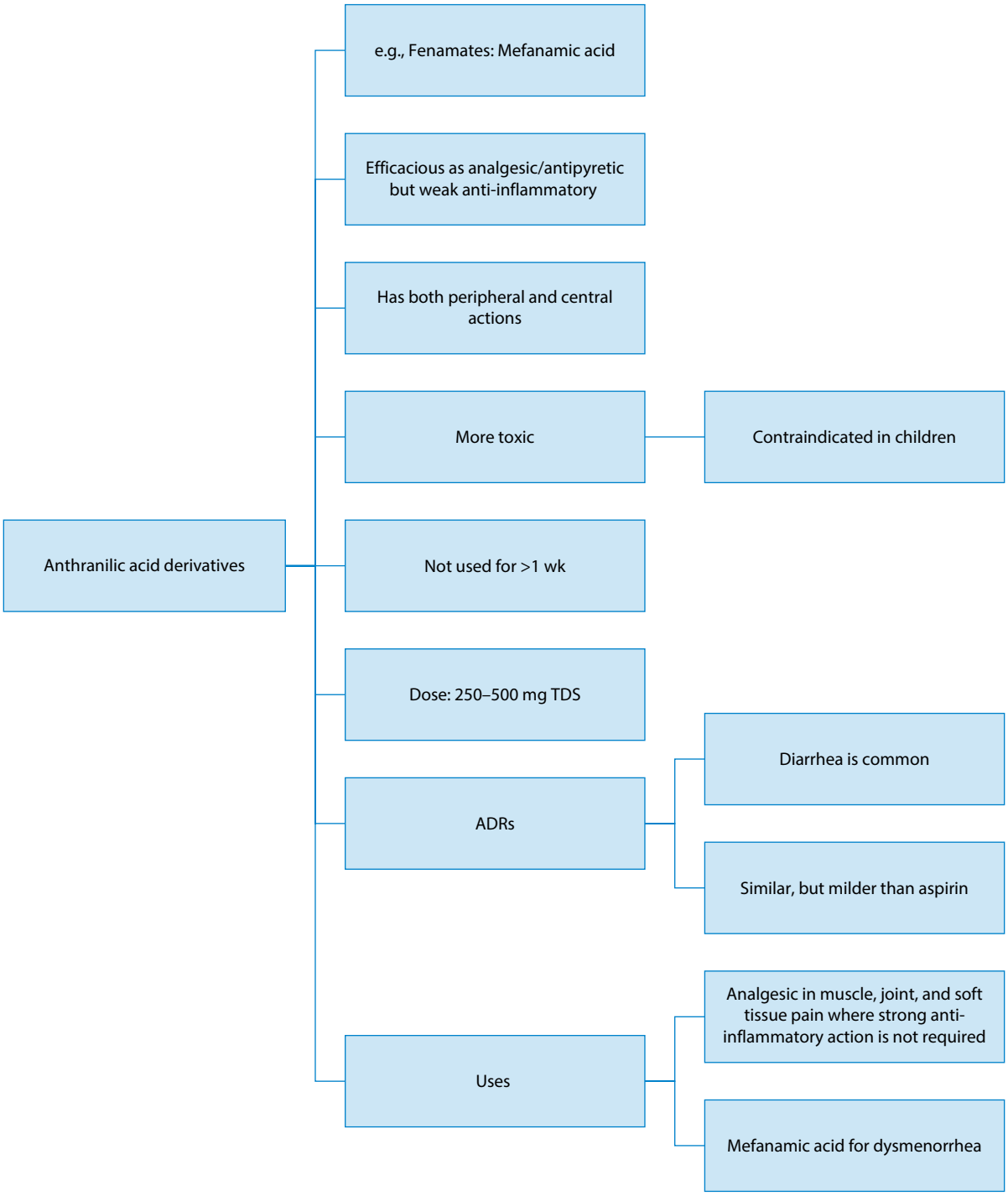
34.13 INDOLE ACETIC ACID DERIVATIVES



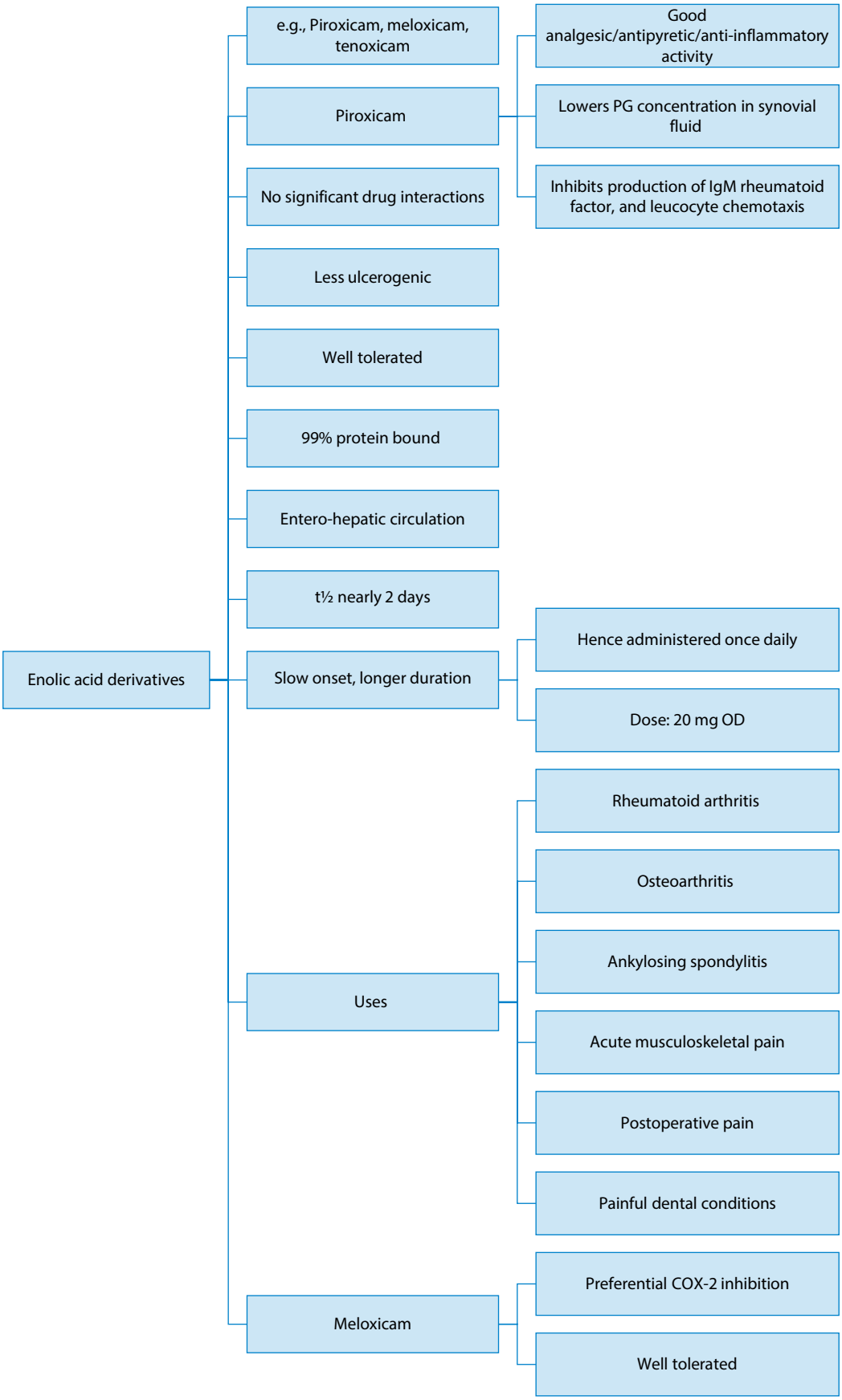
34.14 PROPIONIC ACID DERIVATIVES



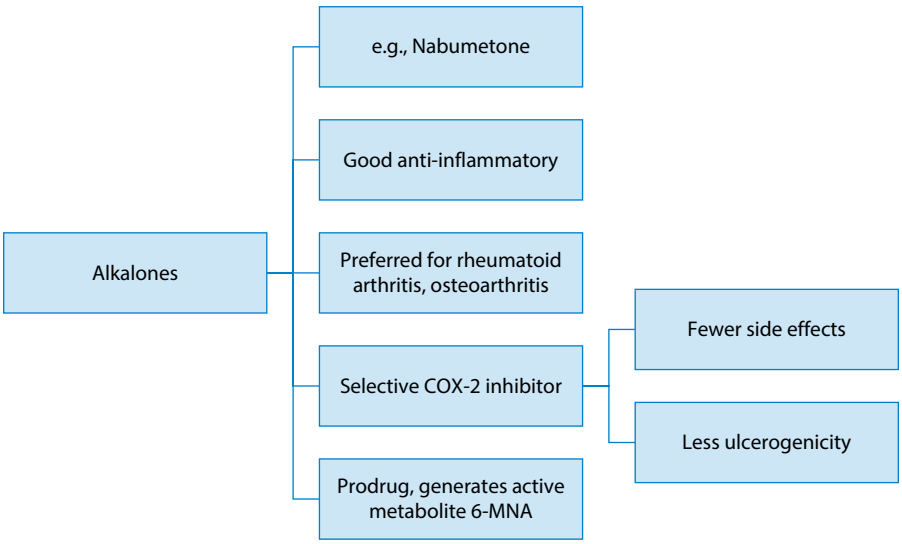
34.15 ANTHRANILIC ACID DERIVATIVES



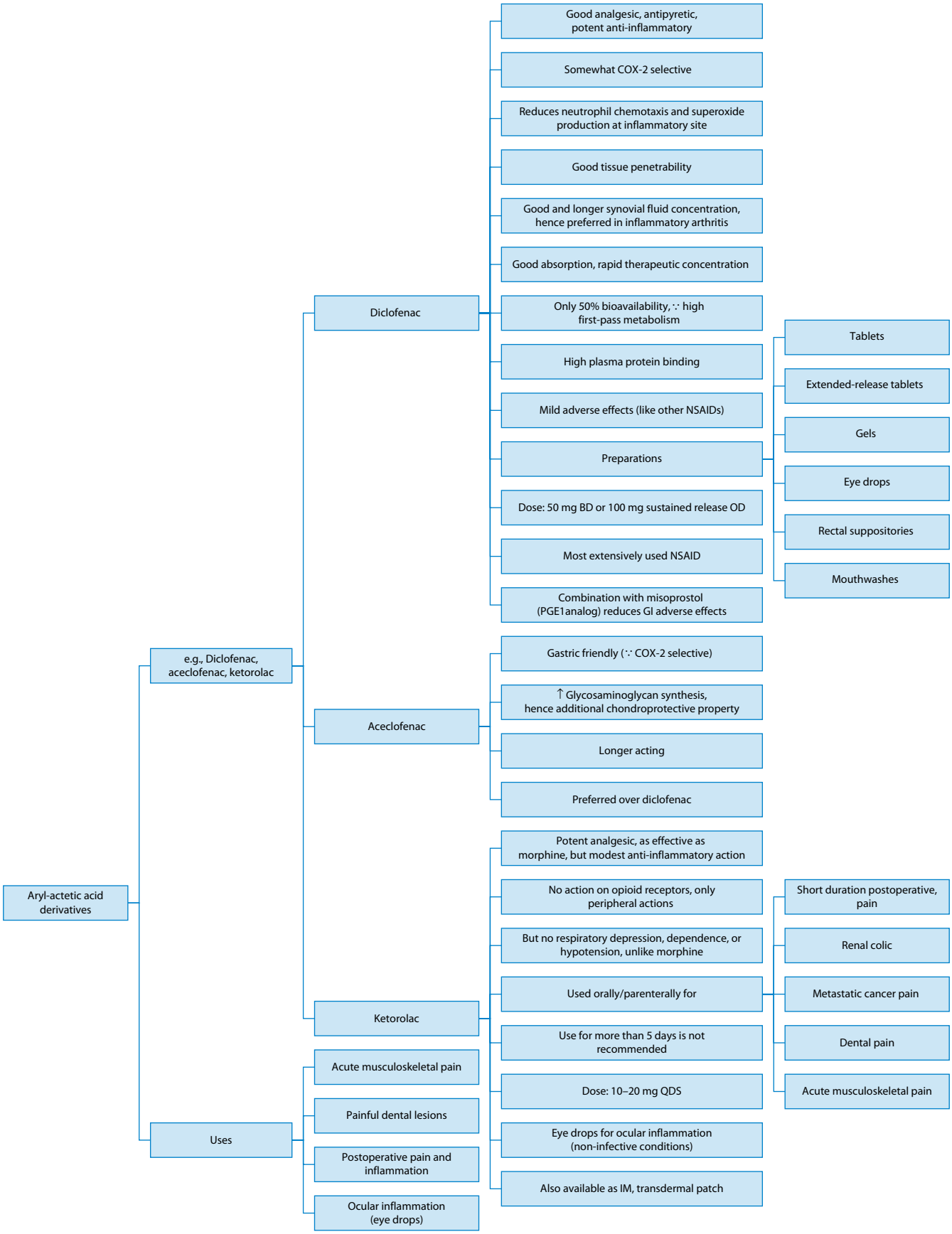
34.16 ENOLIC ACID DERIVATIVES



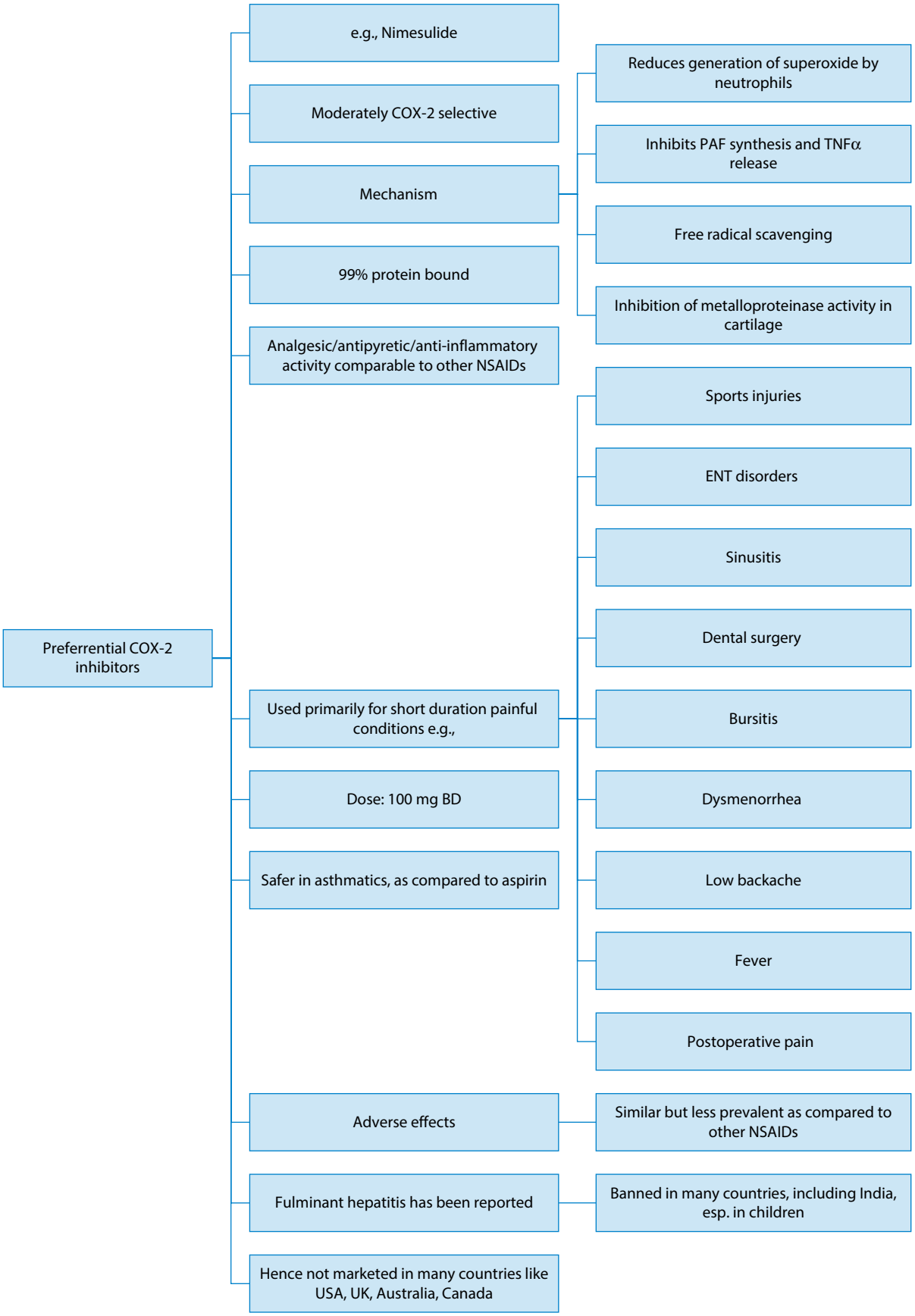
34.17 ALKALONES



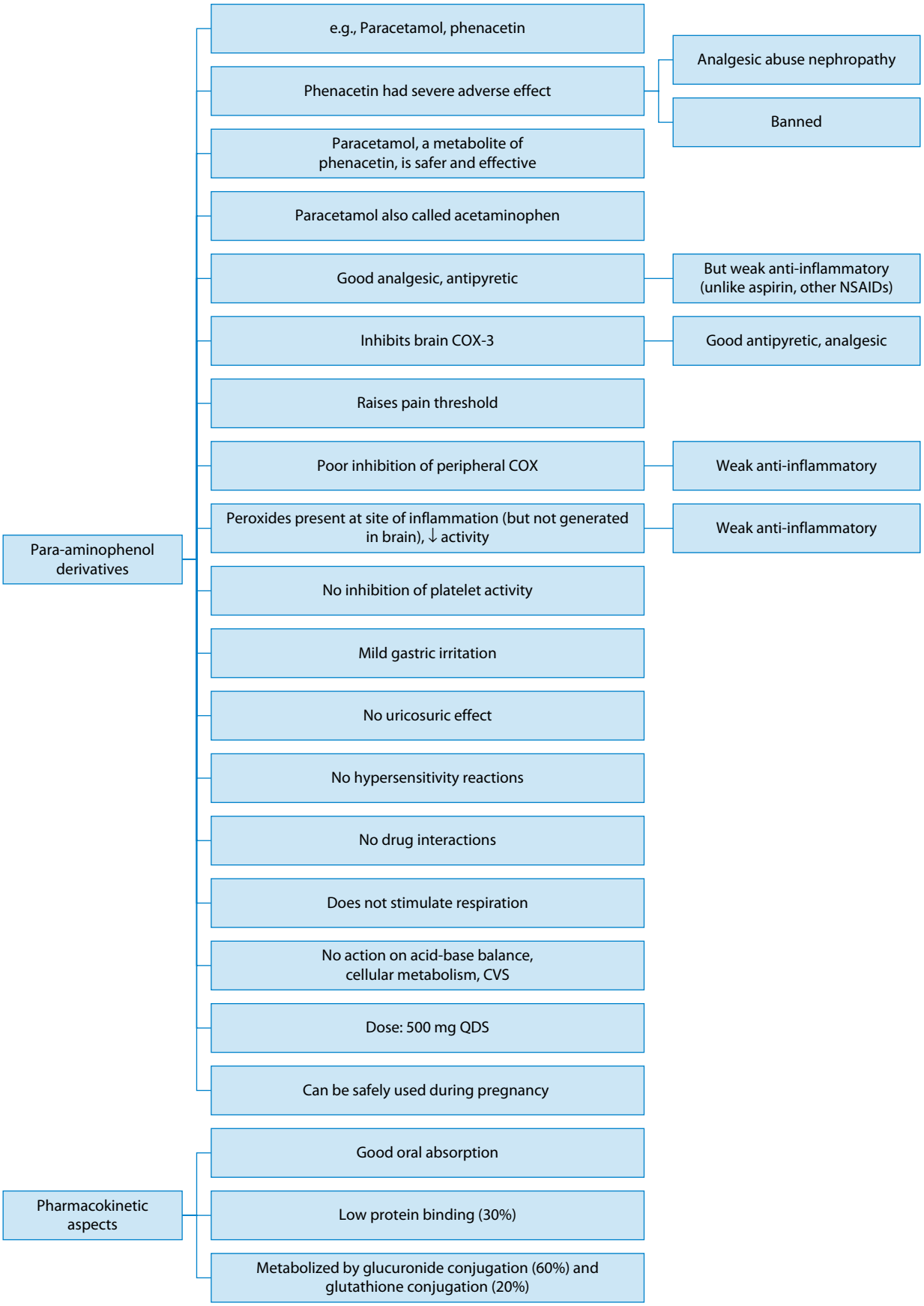
34.18 ARYL-ACTETIC ACID DERIVATIVES



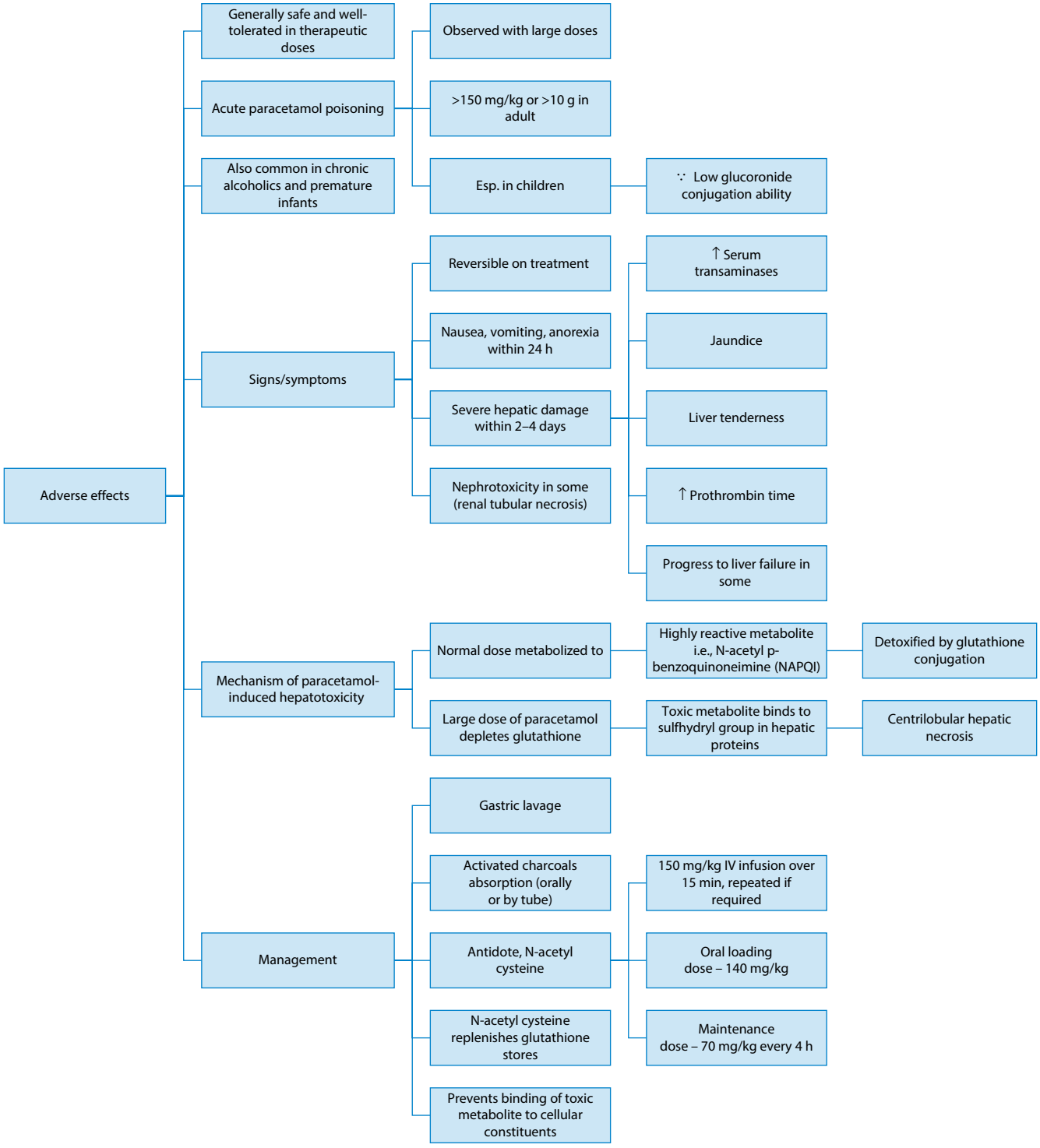
34.19 PREFERRENTIAL COX-2 INHIBITORS



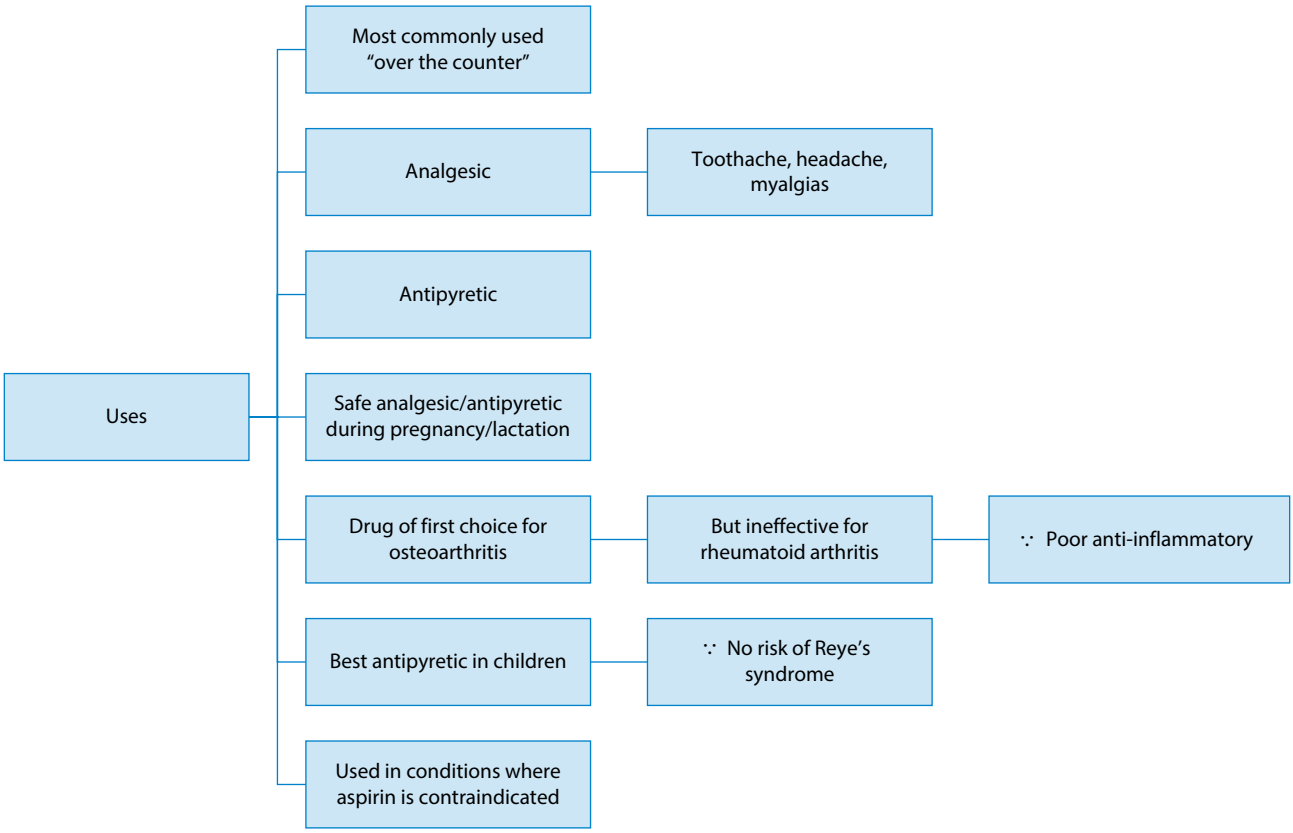
34.20 PARA–AMINOPHENOL DERIVATIVES, PARACETAMOL AND PHARMACOKINETIC ASPECTS



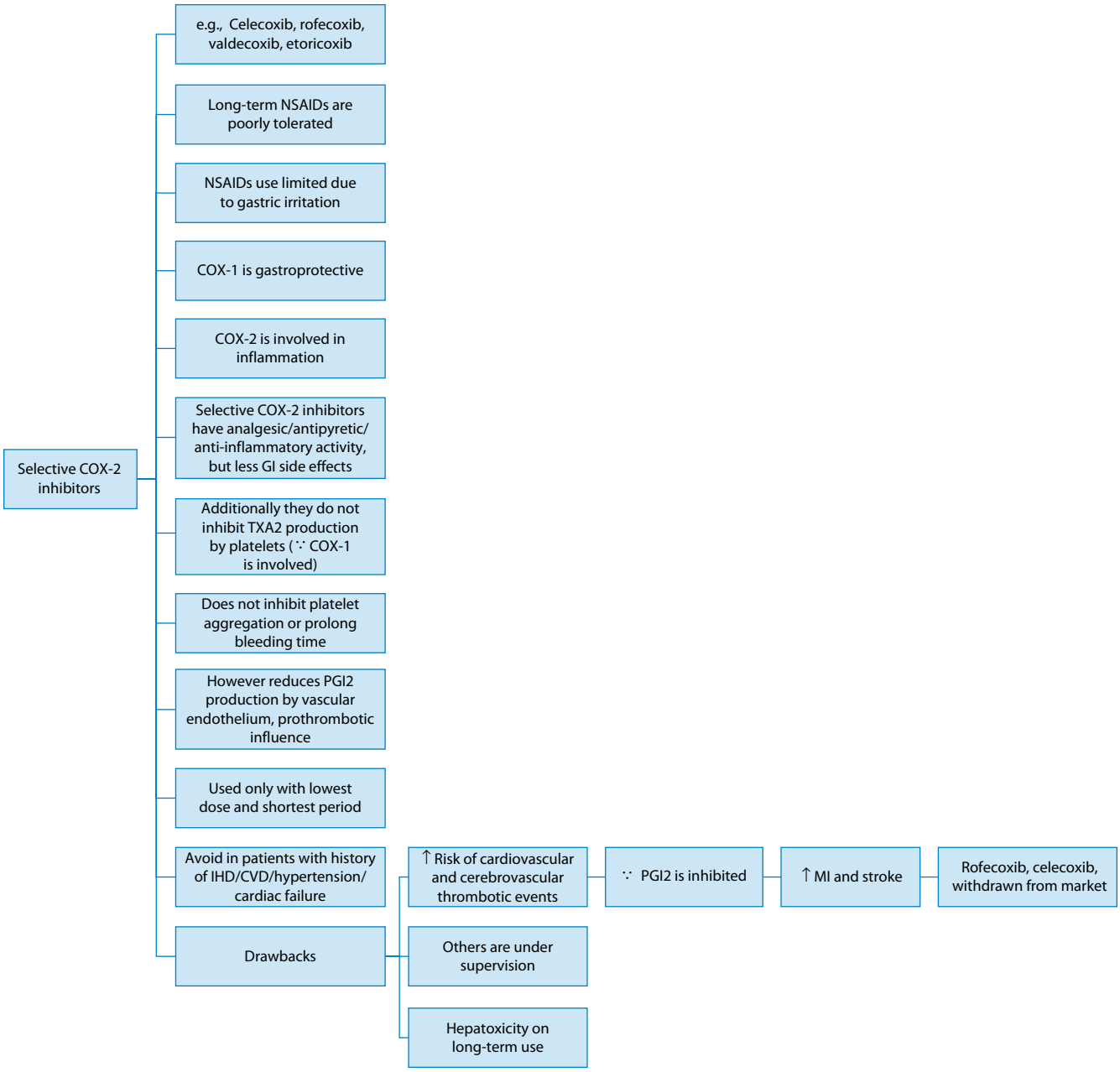
34.21 ADVERSE EFFECTS



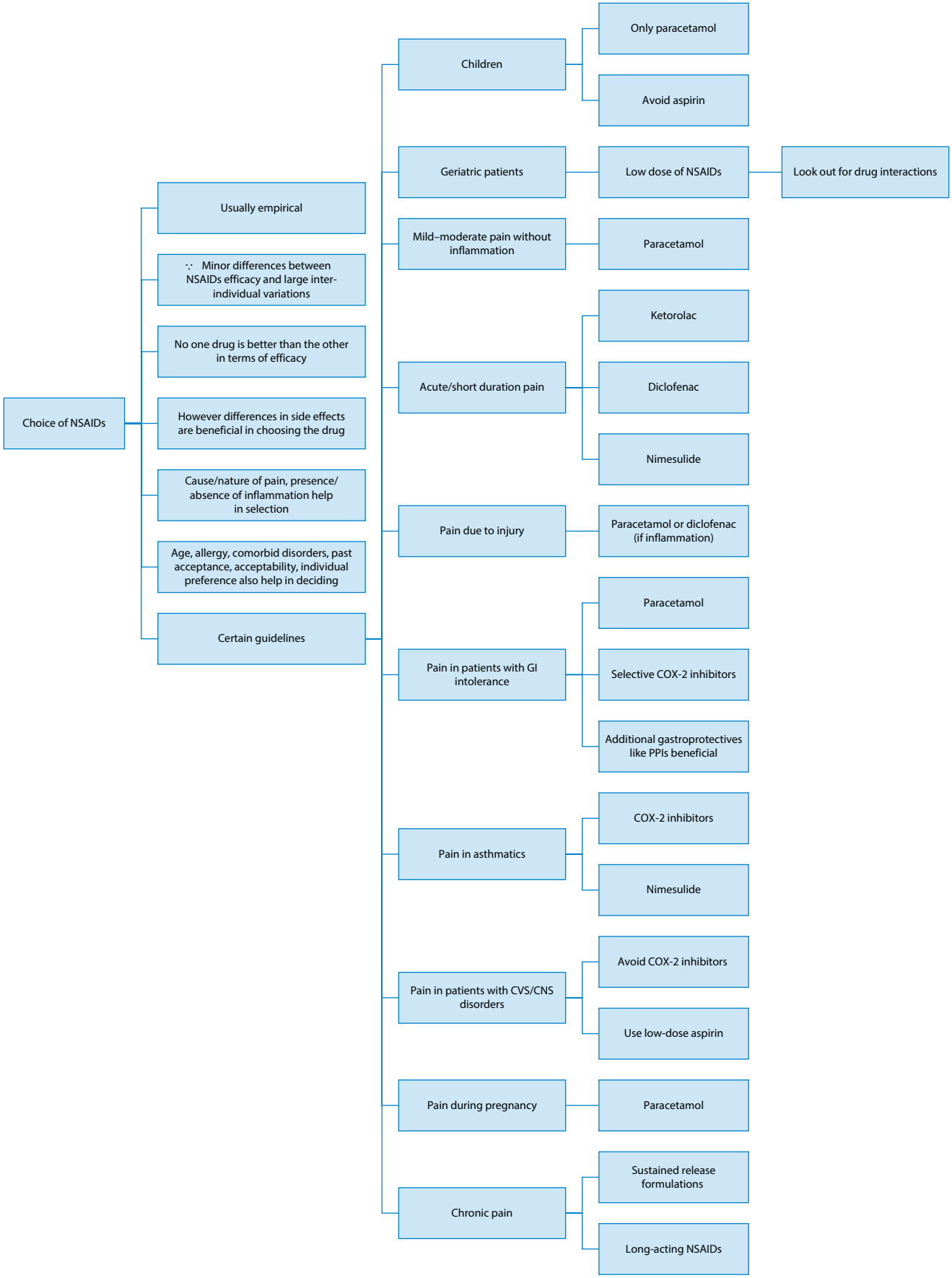
34.22 USES


Uses
Most commonly used "over the counter"
Analgesic
Toothache, headache, myalgias
Antipyretic
Safe analgesic/antipyretic during pregnancy/lactation
Drug of first choice for osteoarthritis
But ineffective for rheumatoid arthritis
∵ Poor anti-inflammatory
Best antipyretic in children
∵ No risk of Reye's syndrome
Used in conditions where aspirin is contraindicated

34.23 SELECTIVE COX-2 INHIBITORS



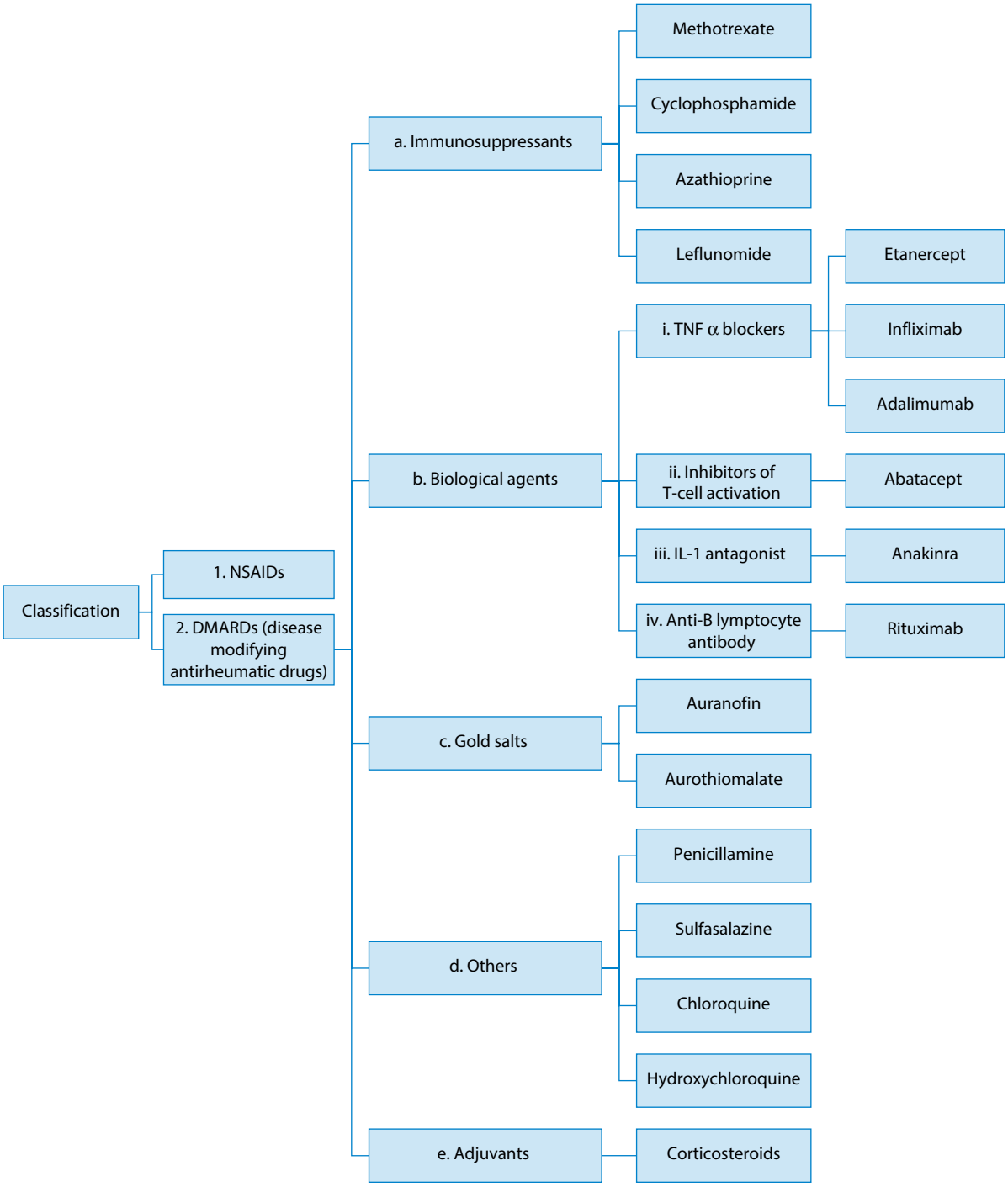
34.24 CHOICE OF NSAIDs



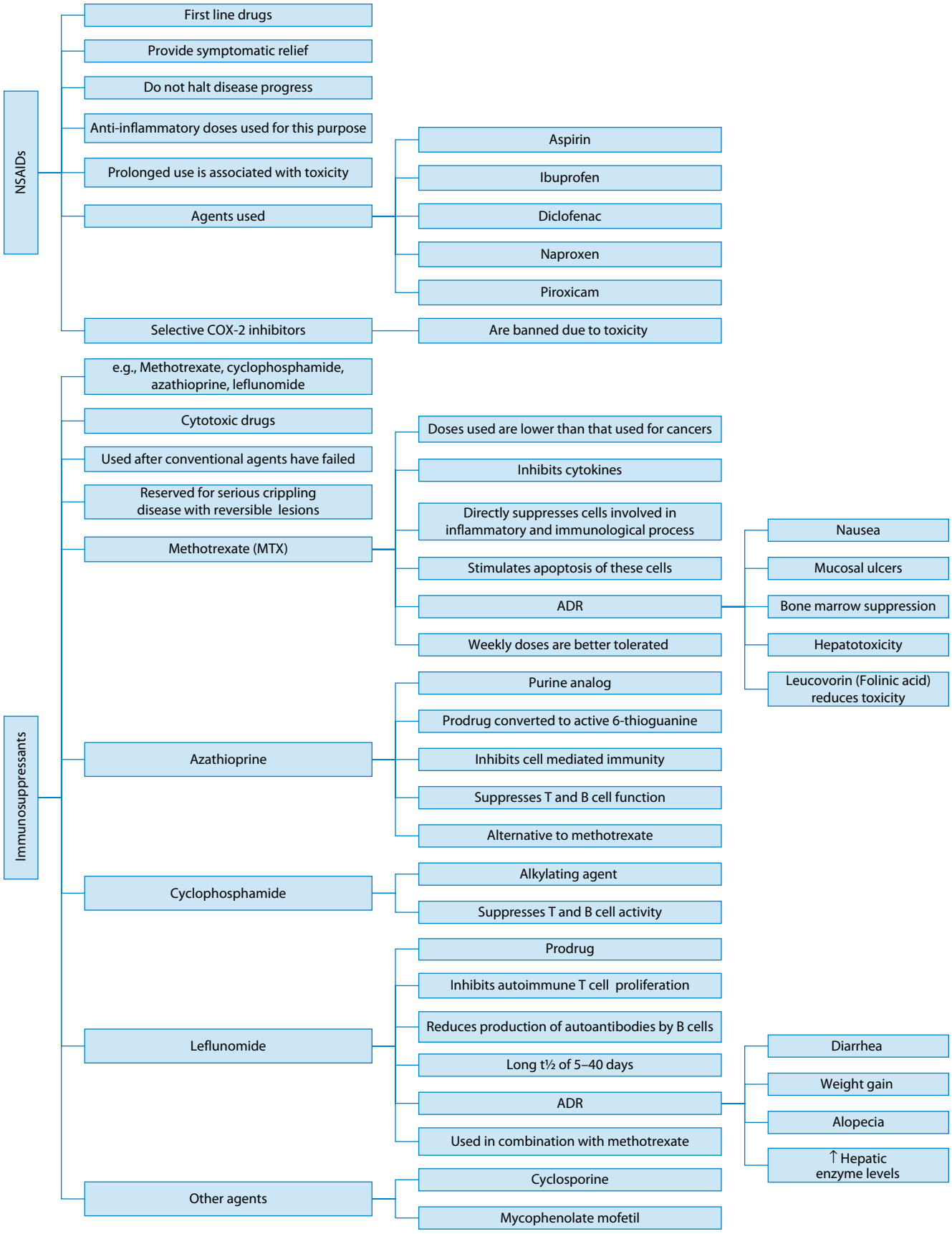
35

Drugs used in rheumtoid arthritis and gout

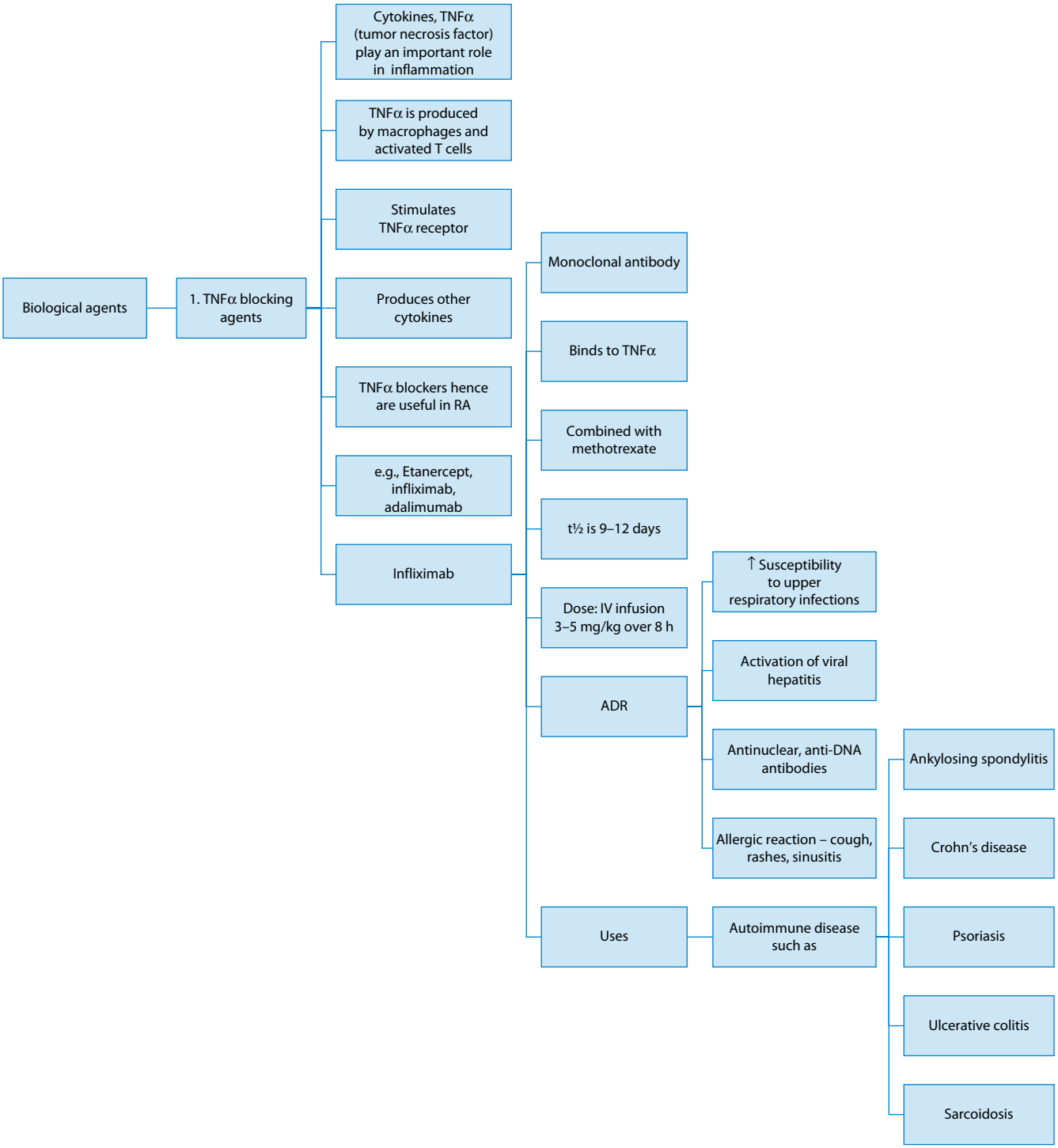
35.1 DRUGS USED IN RHEUMATOID ARTHRITIS – CLASSIFICATION



35.2 NSAIDs AND IMMUNOSUPPRESSANTS

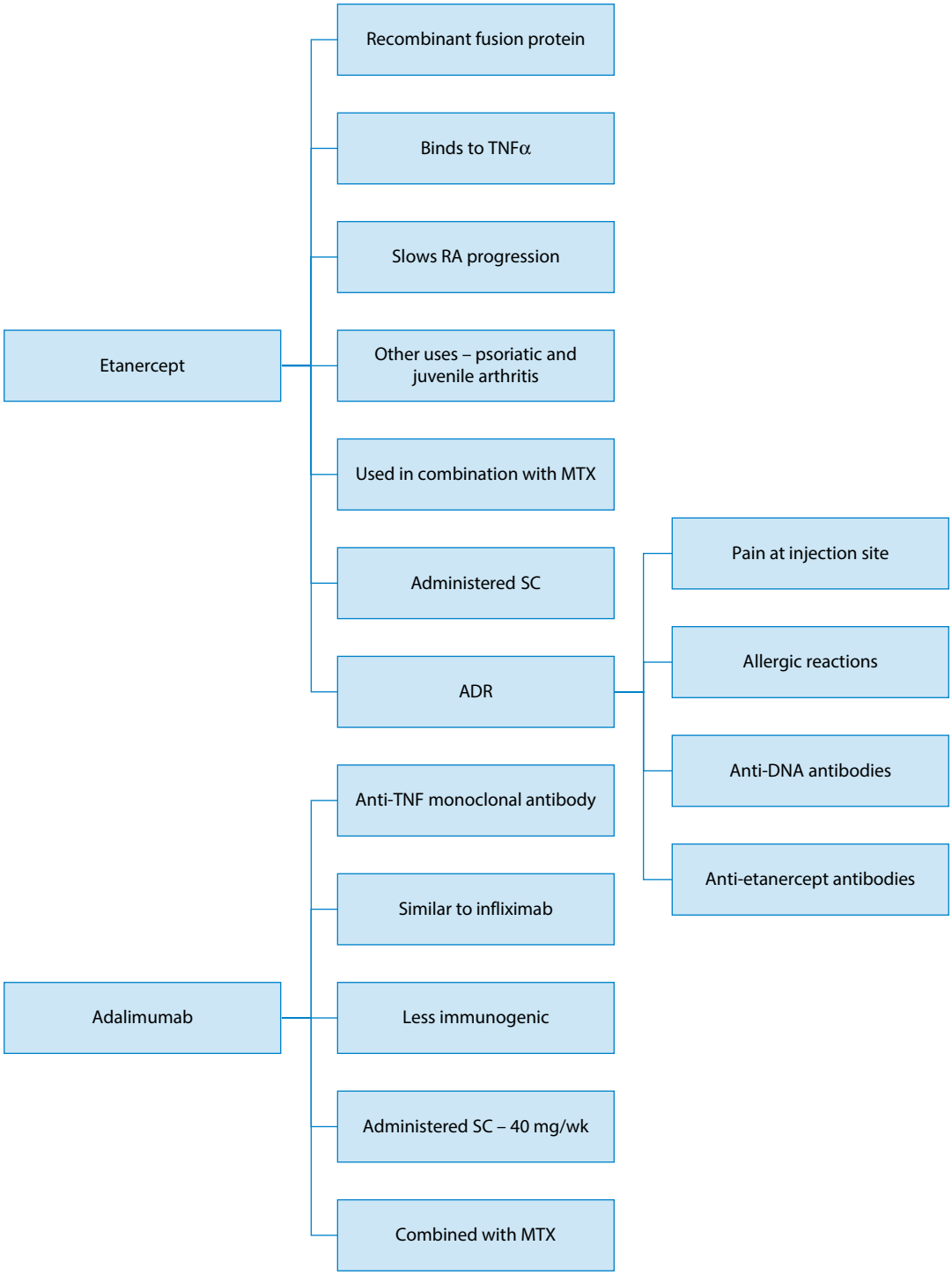


35.3 BIOLOGICAL AGENTS

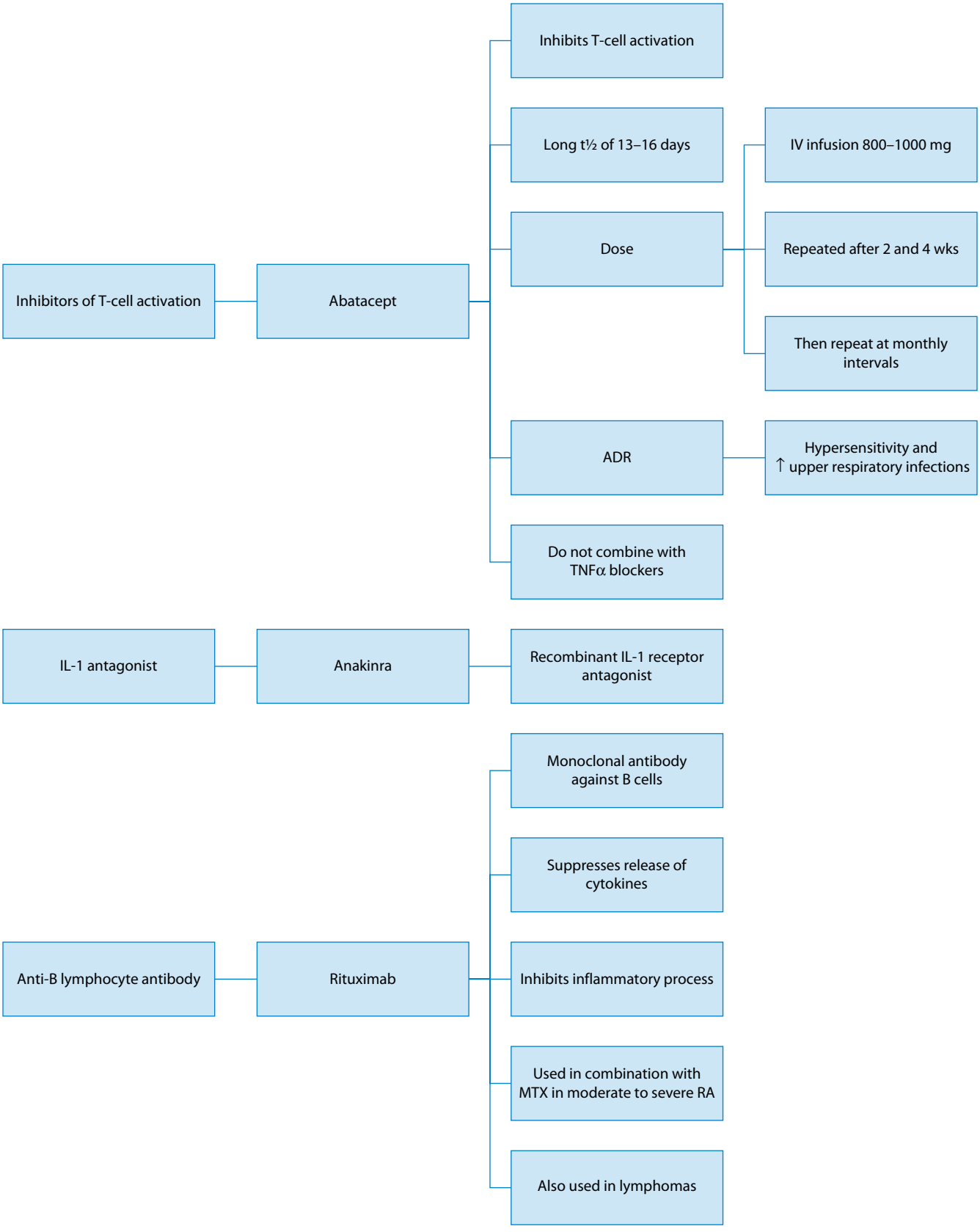


(*Continued*)

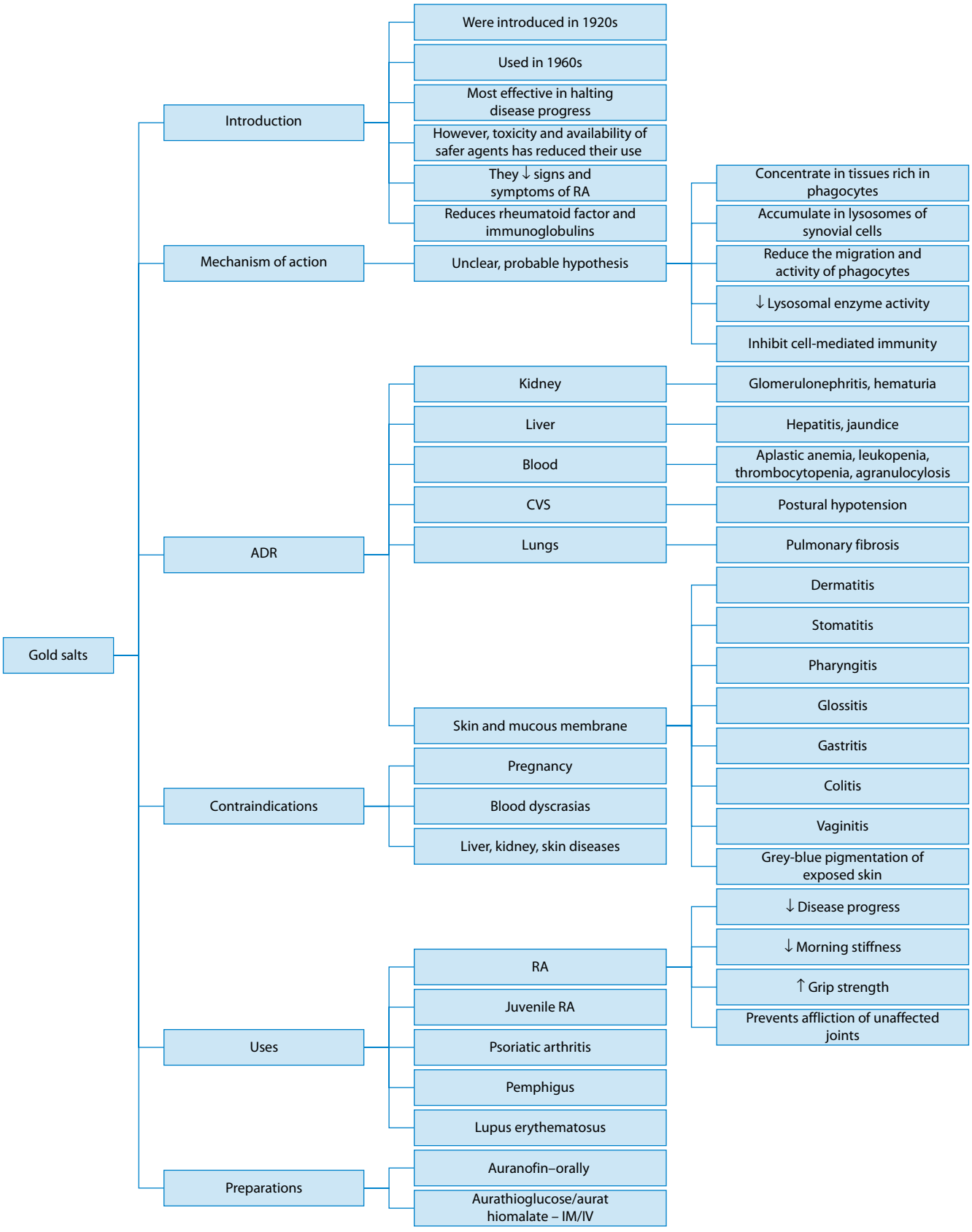
35.3 BIOLOGICAL AGENTS (Continued)



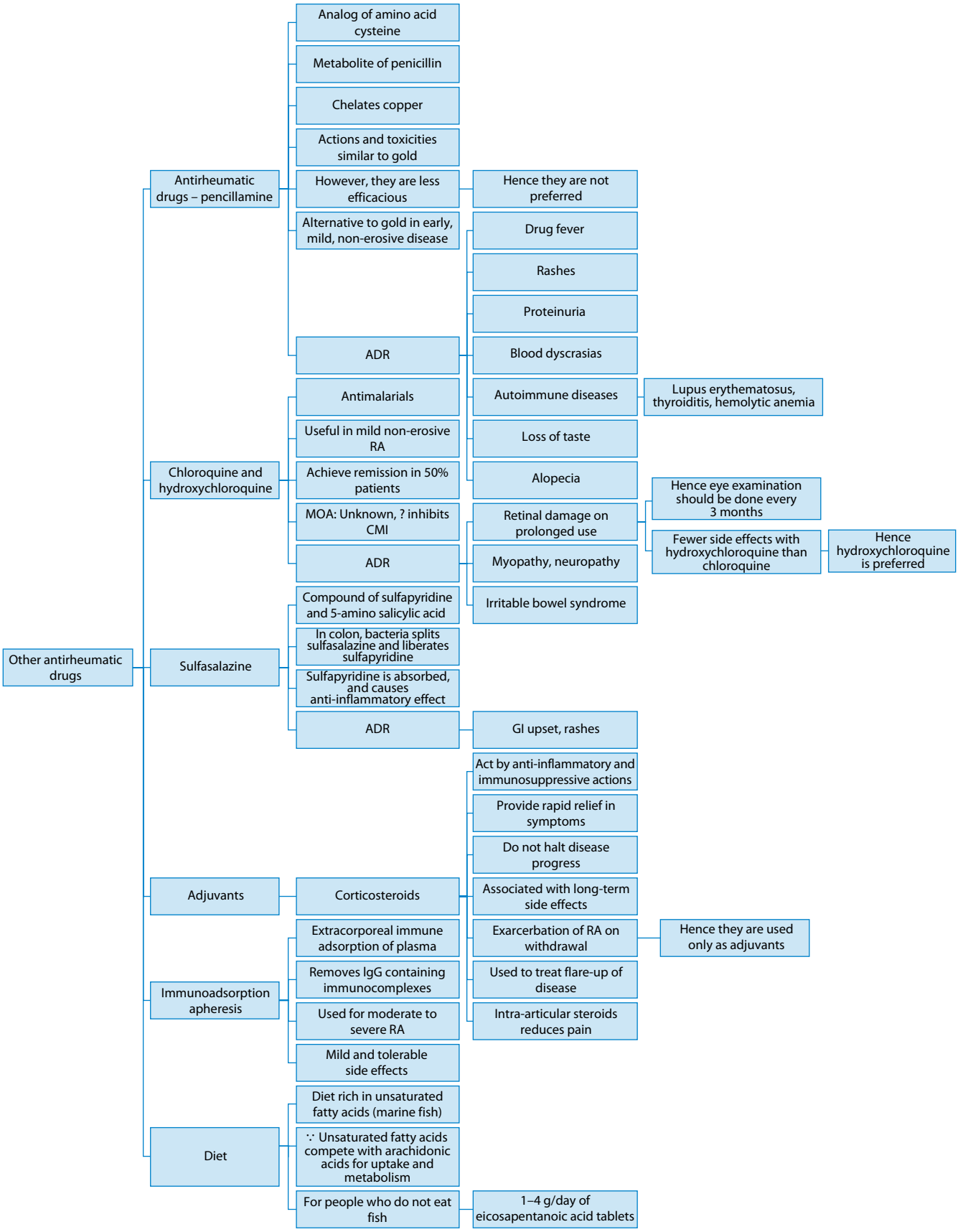
35.4 INHIBITORS OF T-CELL ACTIVATION, IL-1 ANTAGONIST, AND ANTI-B LYMPHOCYTE ANTIBODY



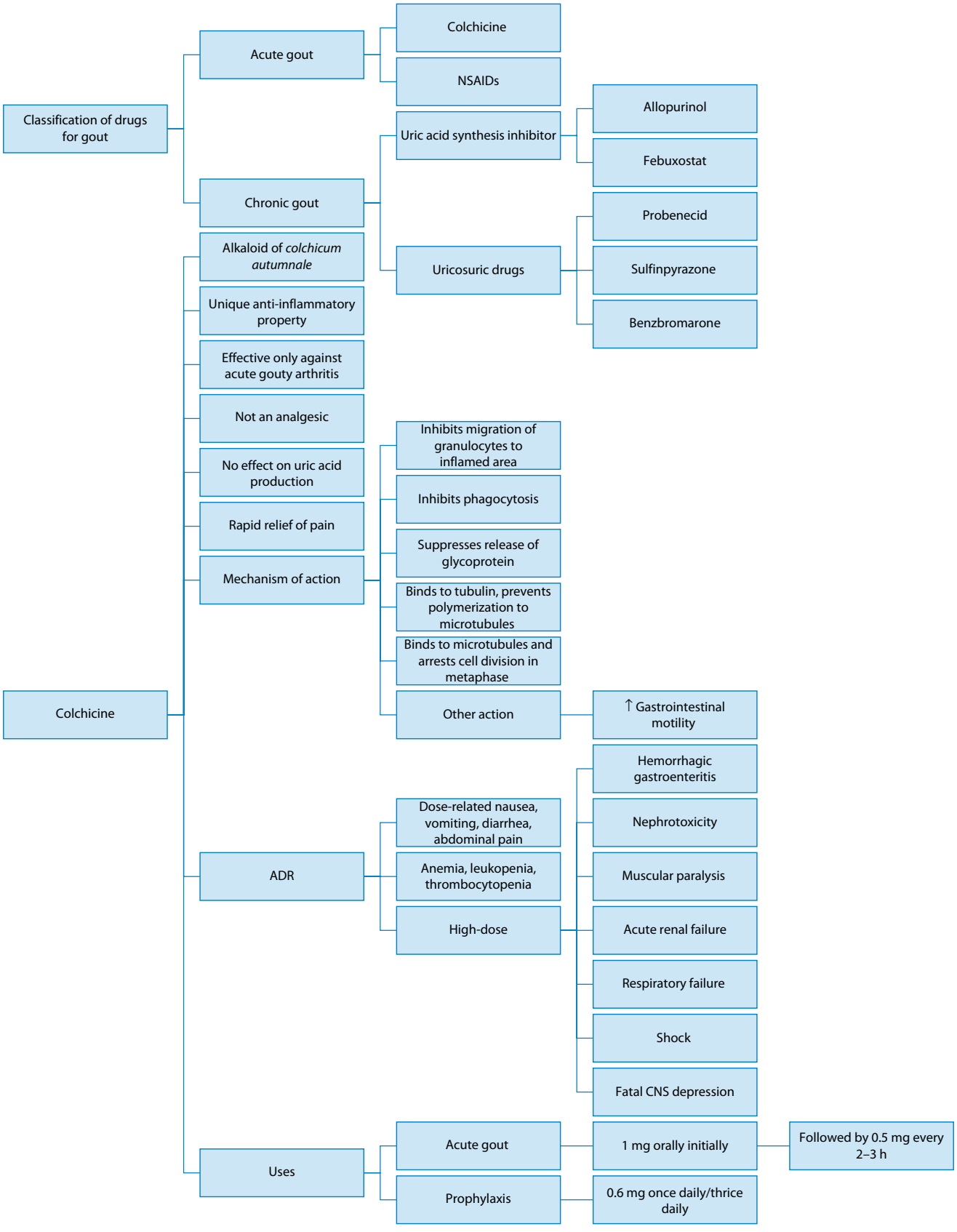
35.5 GOLD SALTS



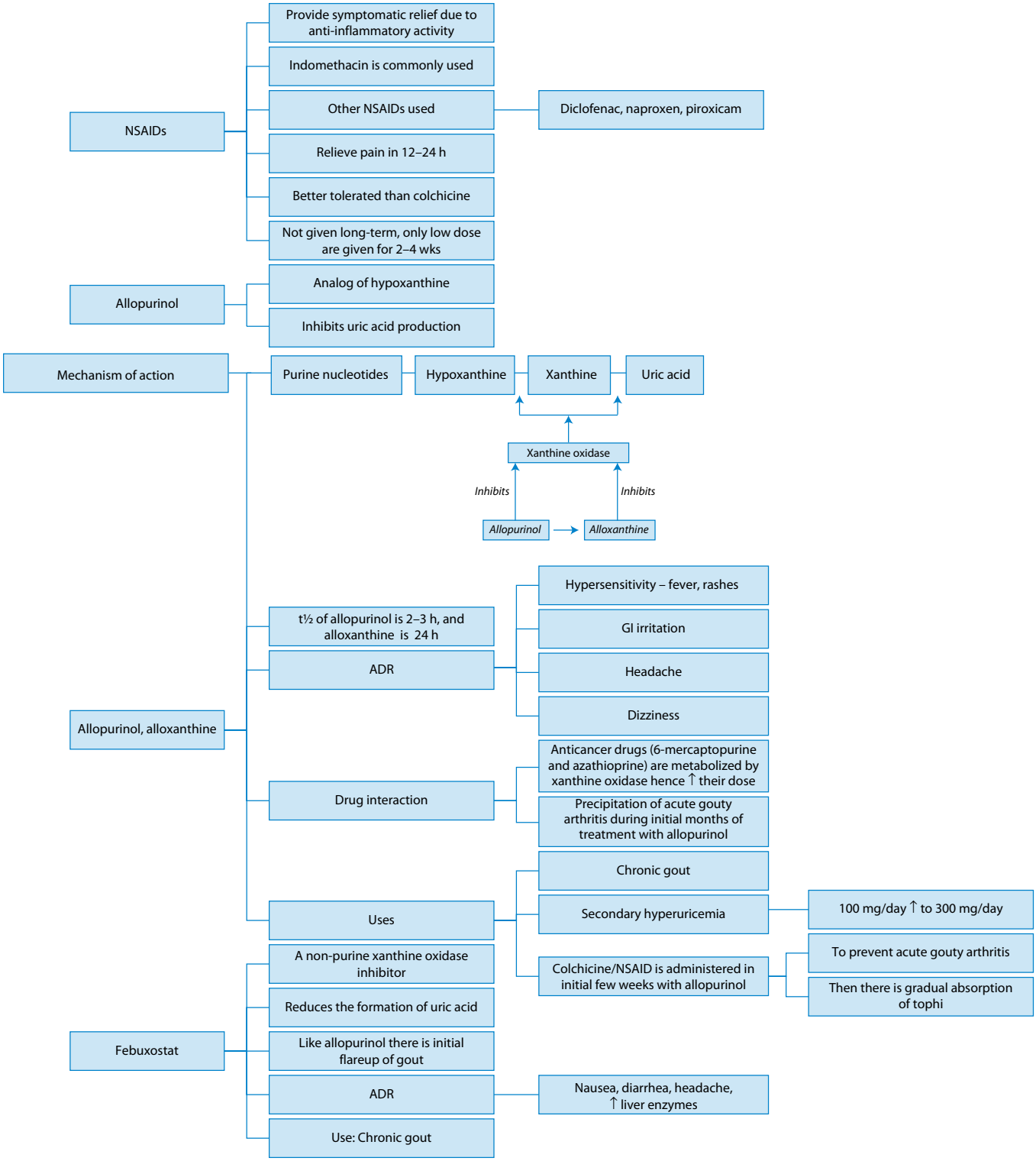
35.6 OTHER ANTIRHEUMATIC DRUGS



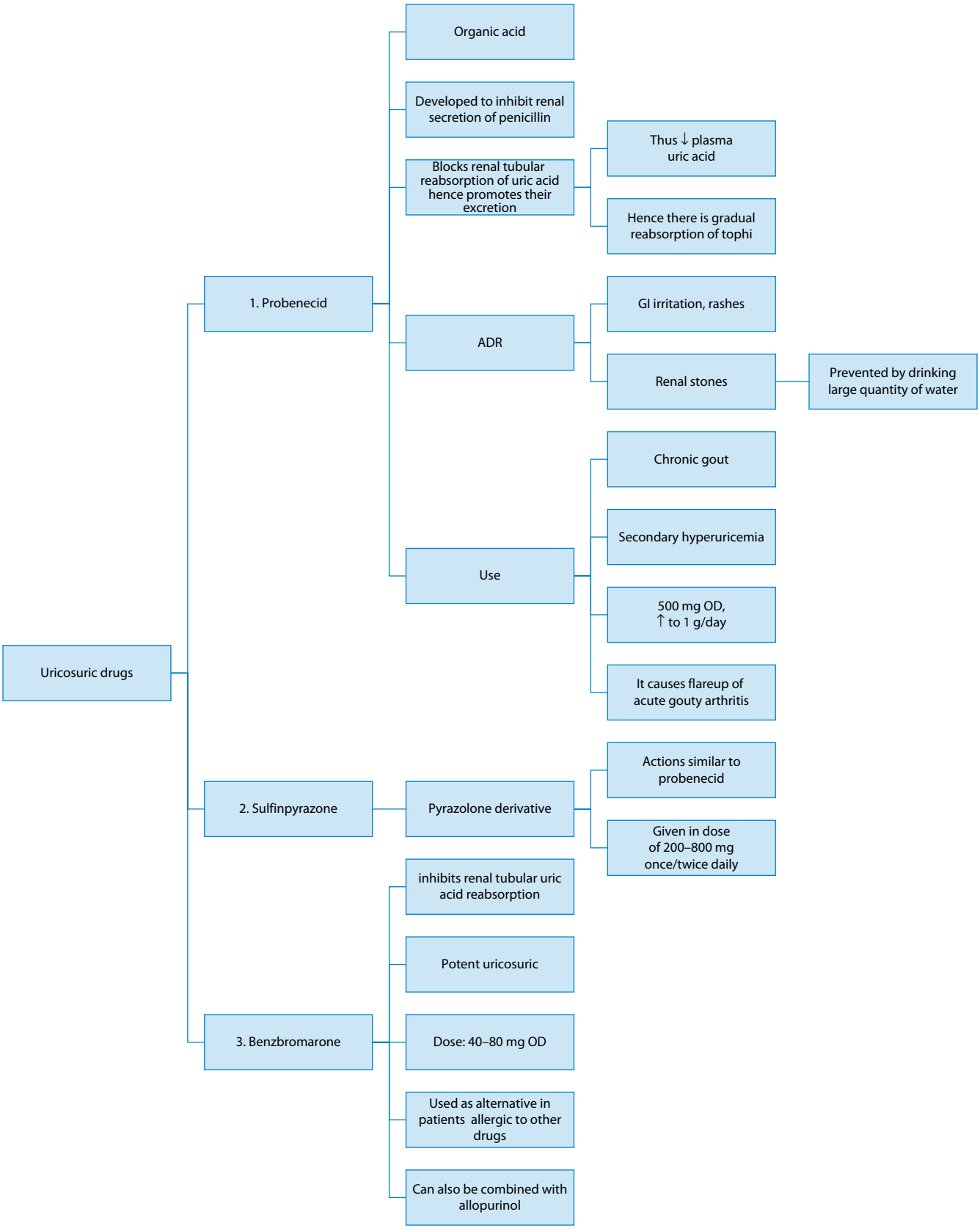
35.7 CLASSIFICATION OF DRUGS FOR GOUT AND COLCHICINE



35.8 NSAIDs, ALLOPURINOL, AND FEBUXOSTAT



35.9 URICOSURIC DRUGS





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http://taylorandfrancis.com

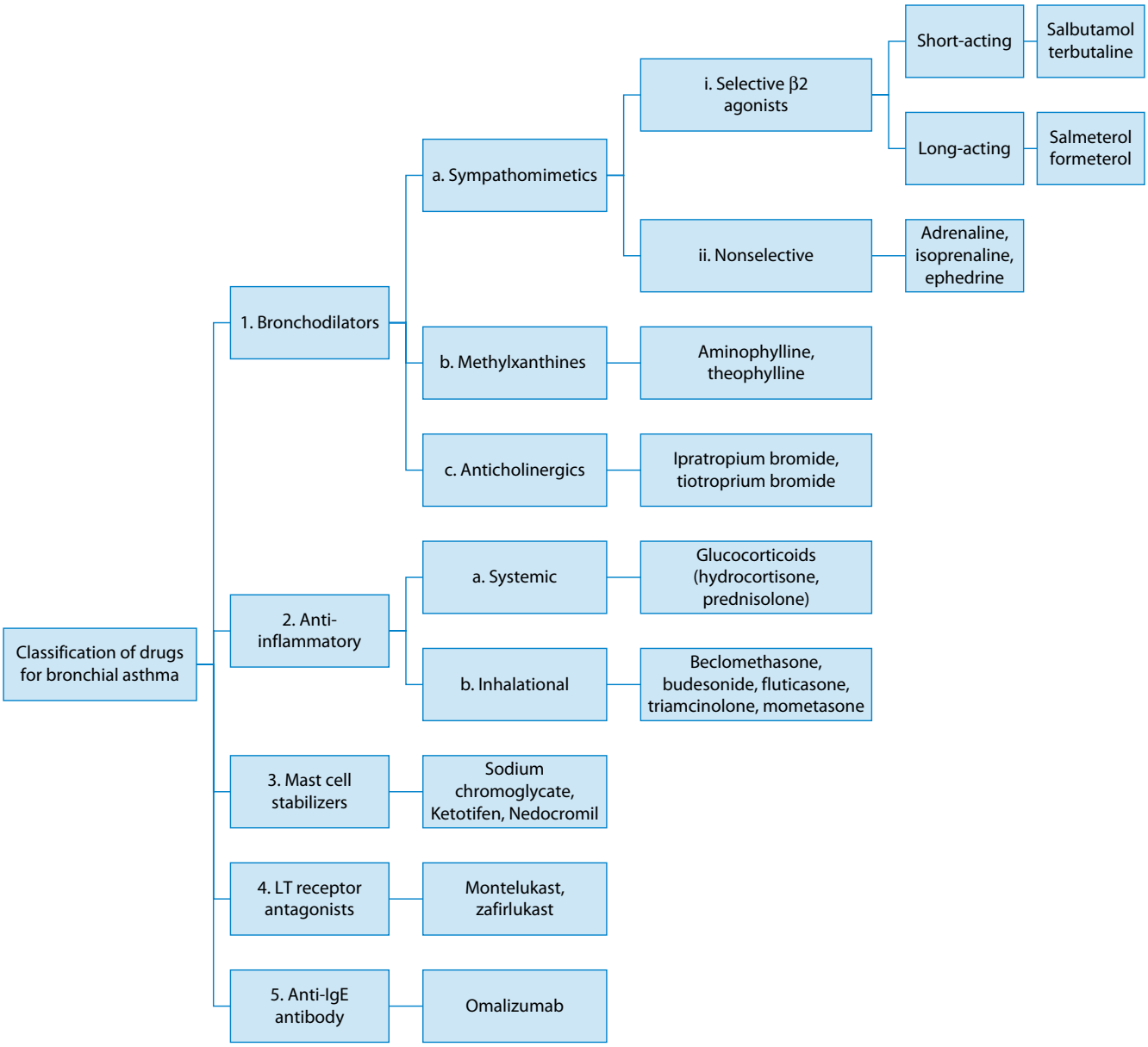
PART VI

Respiratory pharmacology

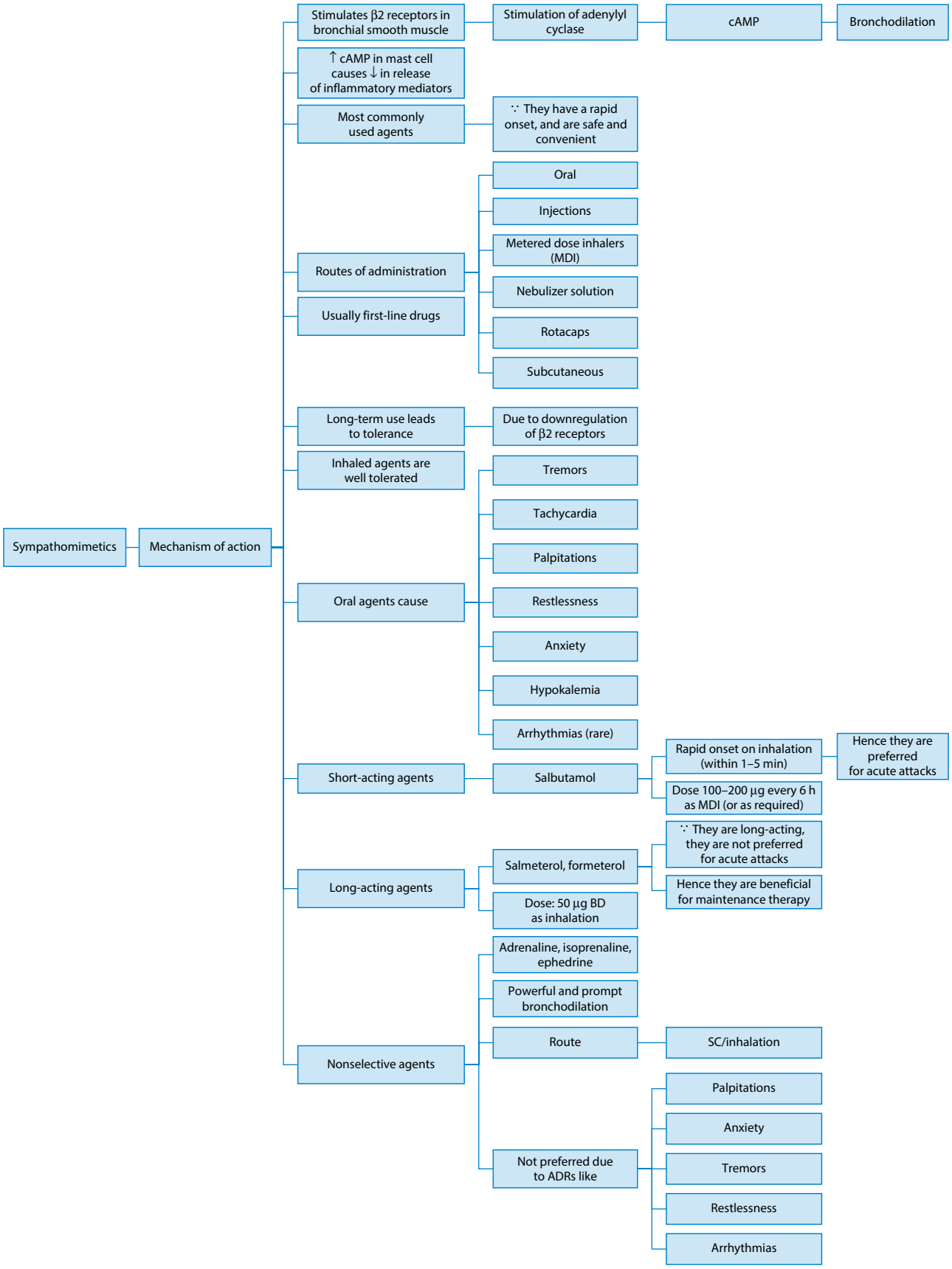
36

Drugs used in the treatment of bronchial asthma and chronic obstuctive pulmonary disorders (COPD)

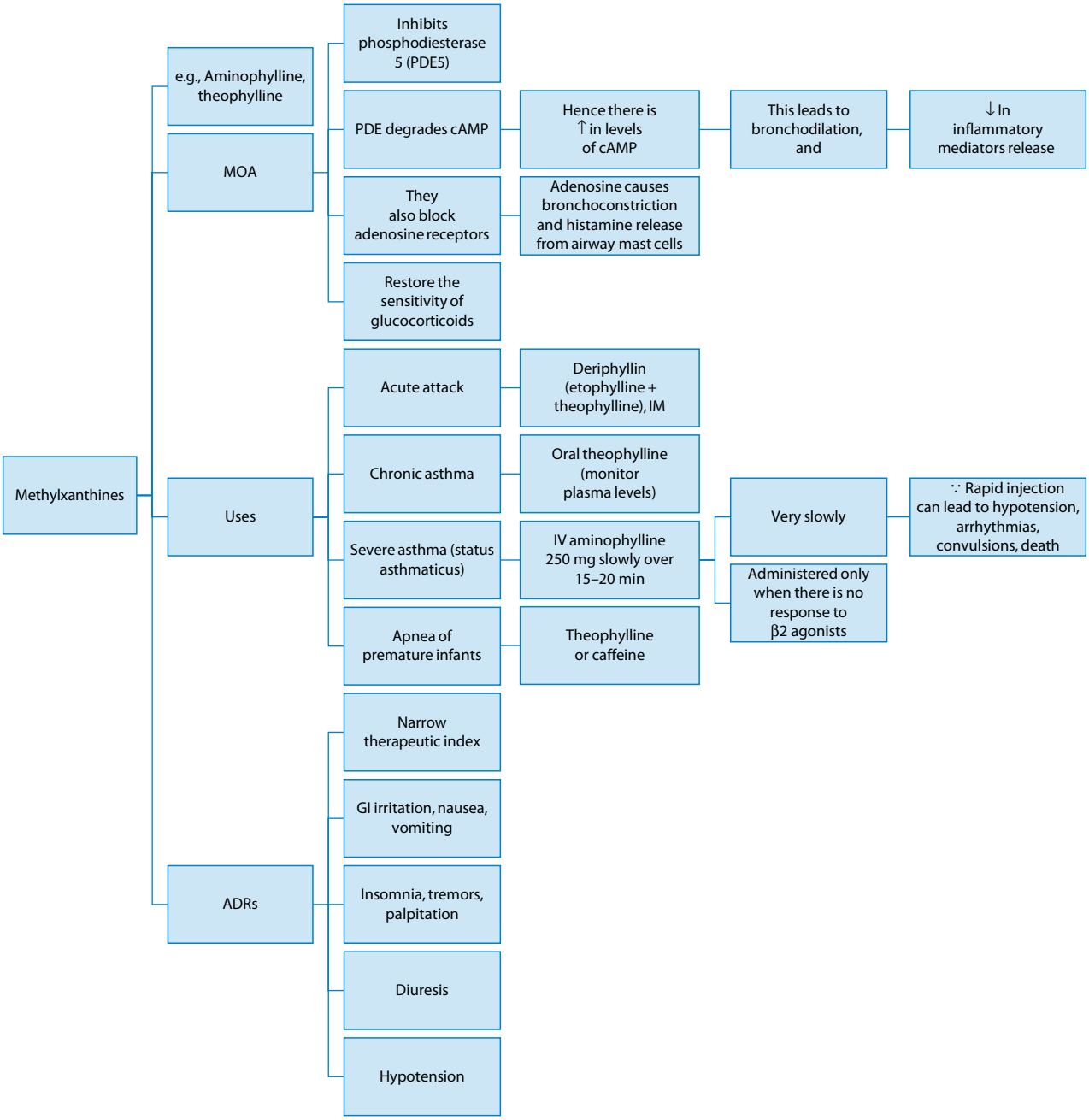
36.1 CLASSIFICATION OF DRUGS FOR BRONCHIAL ASTHMA



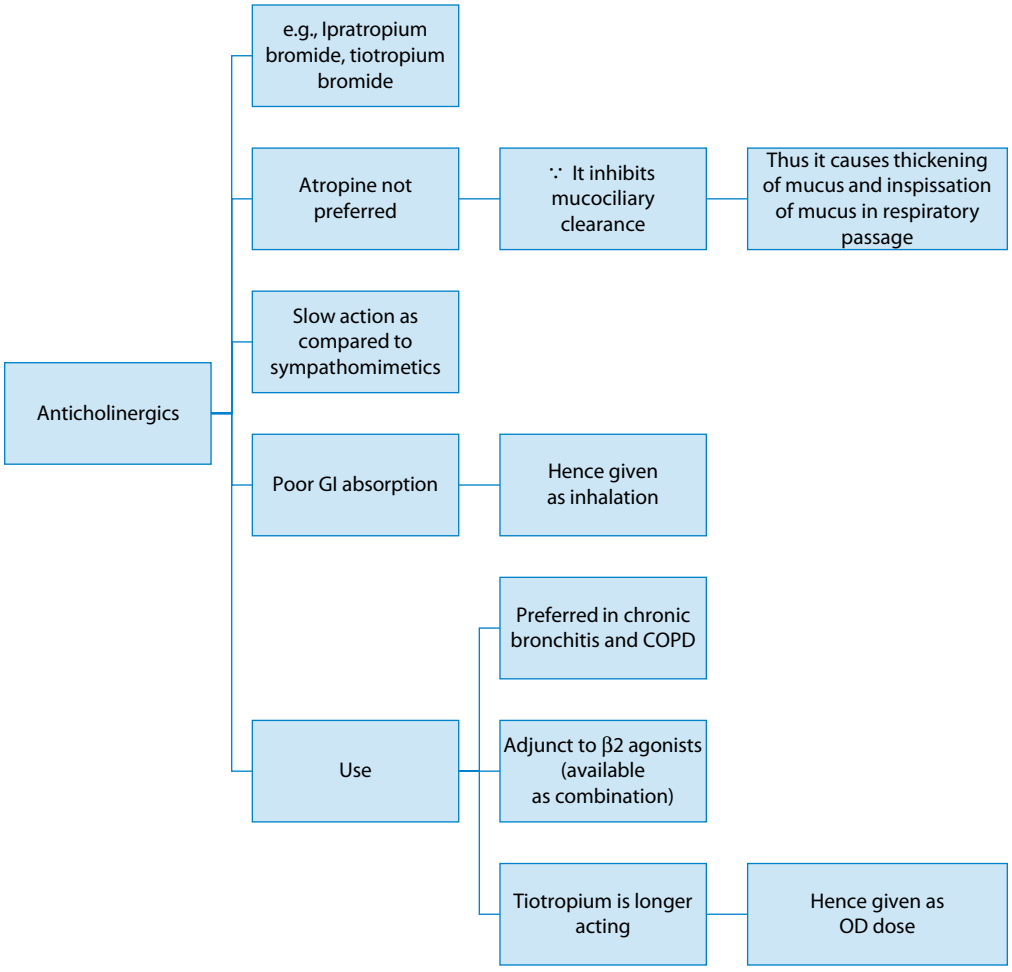
36.2 SYMPATHOMIMETICS



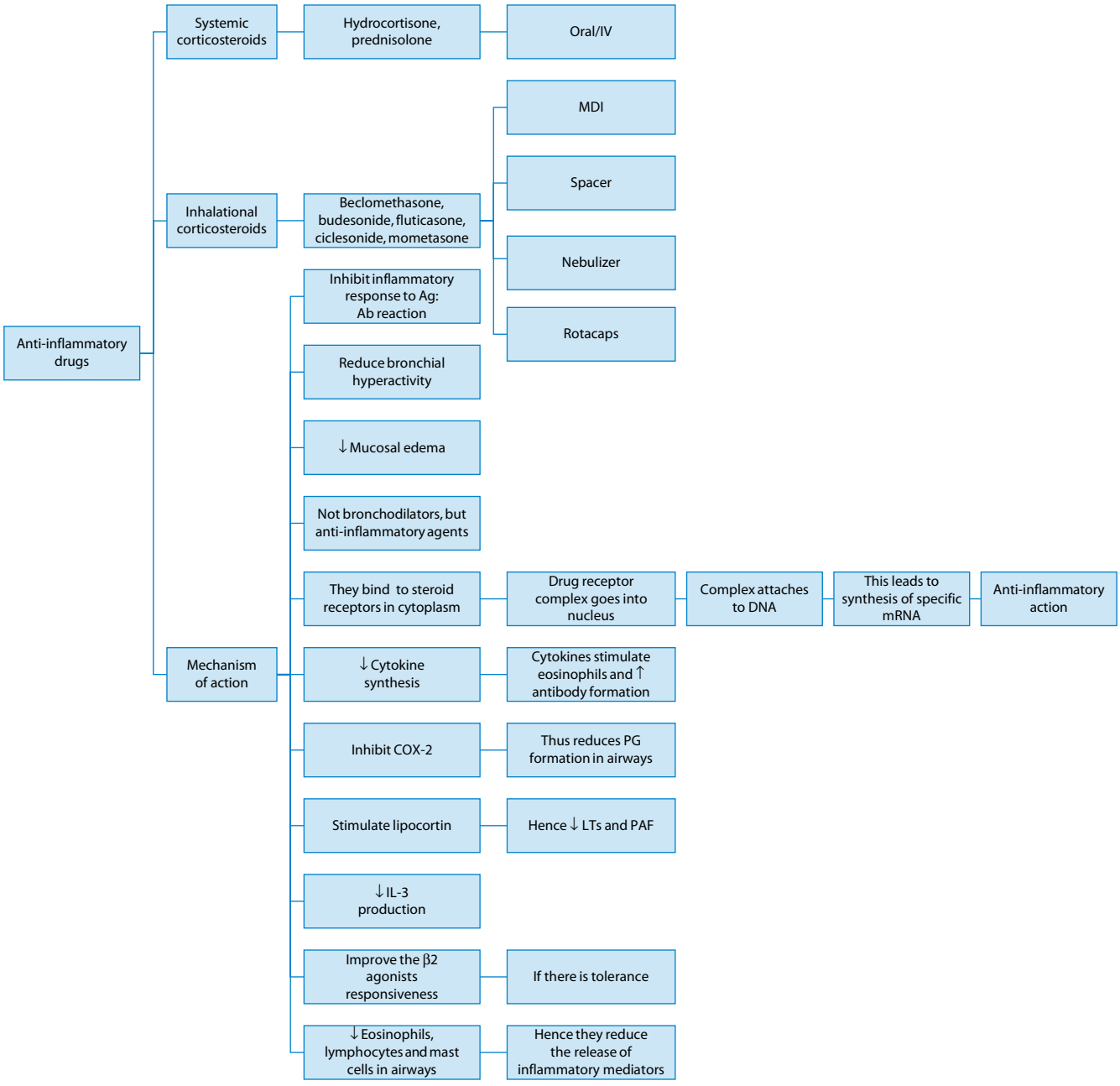
36.3 METHYLXANTHINES



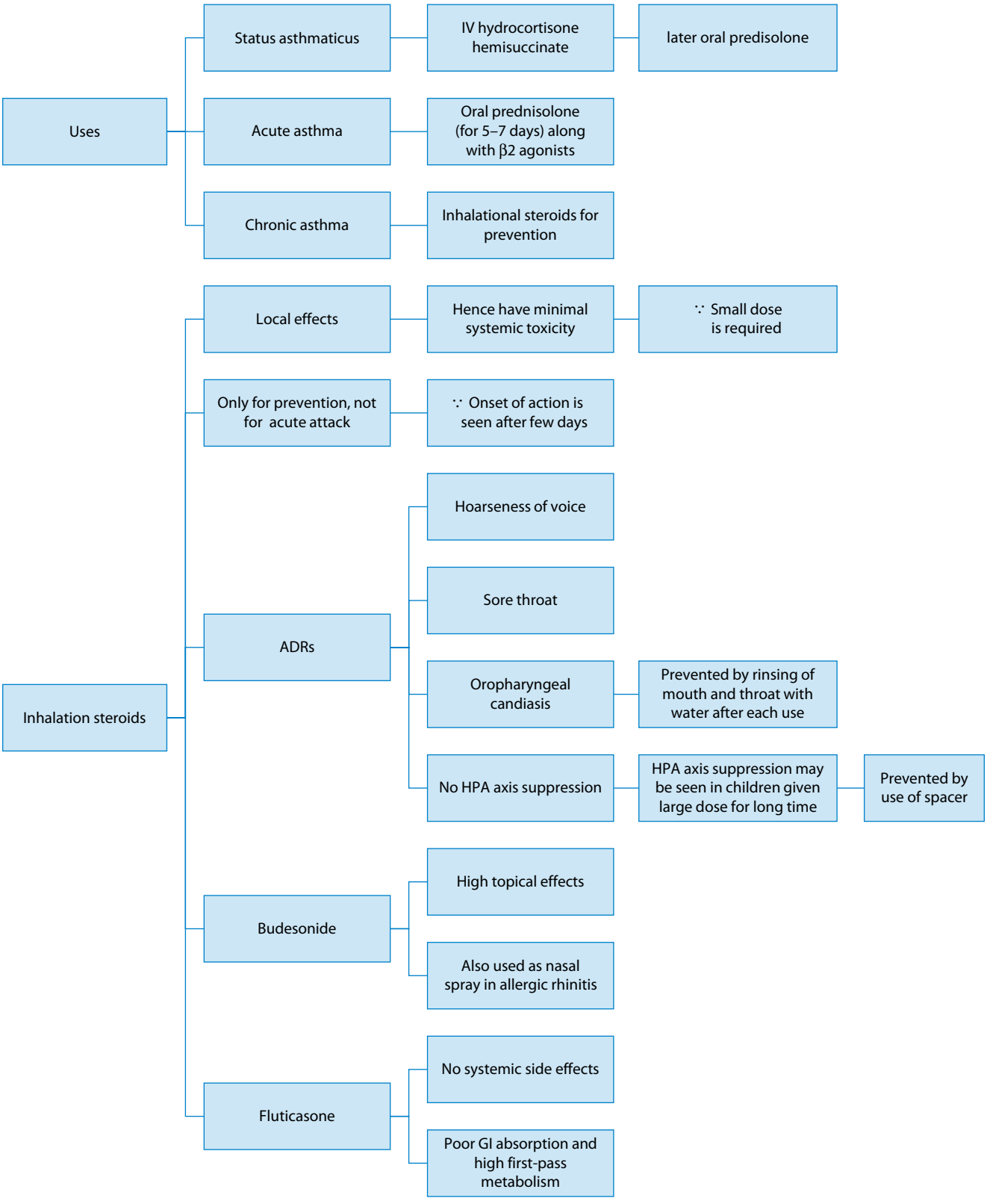
36.4 ANTICHOLINERGICS



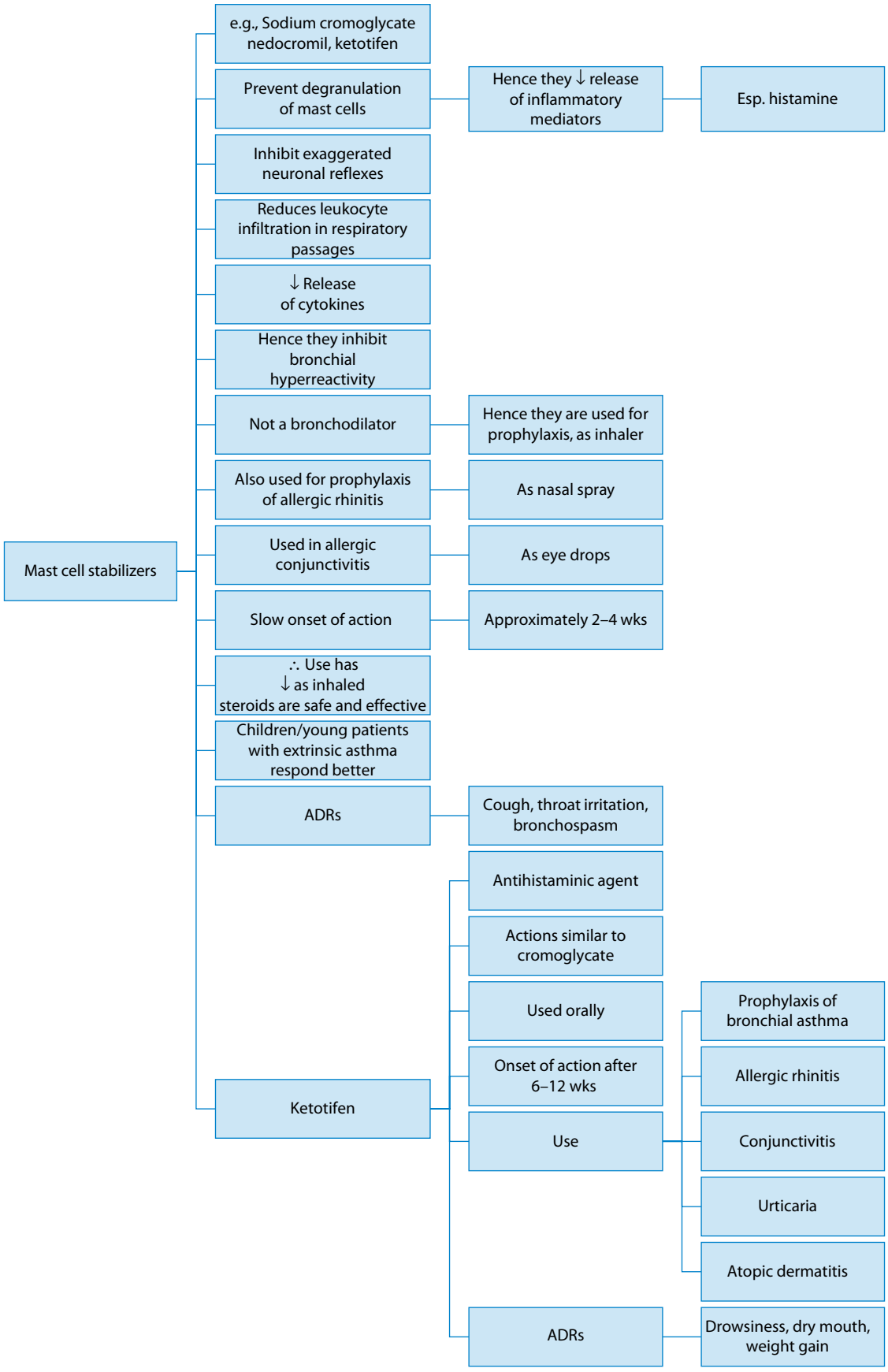
36.5 ANTI-INFLAMMATORY DRUGS



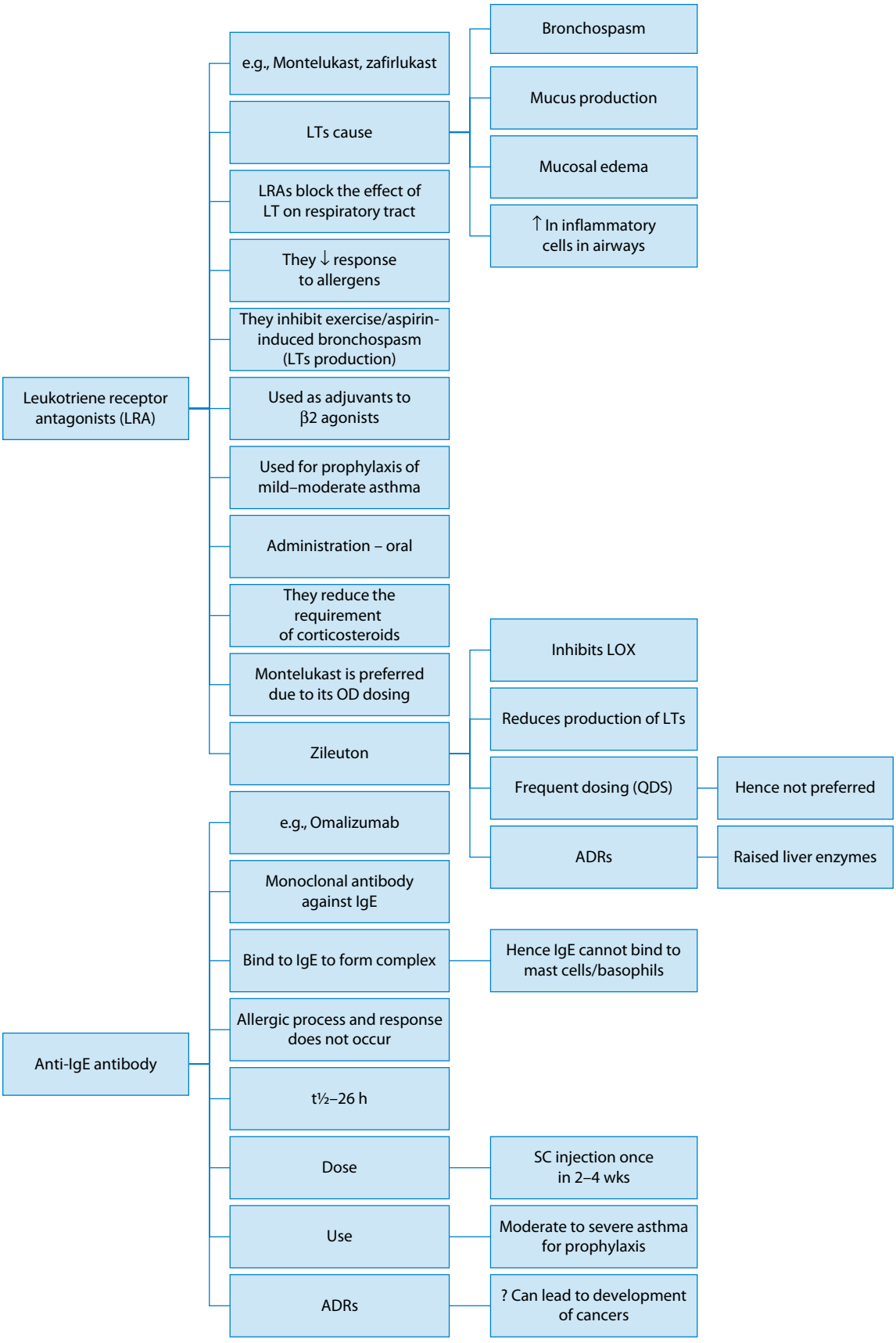
36.6 ANTI-INFLAMMATORY DRUGS – USES, INHALATION STEROIDS



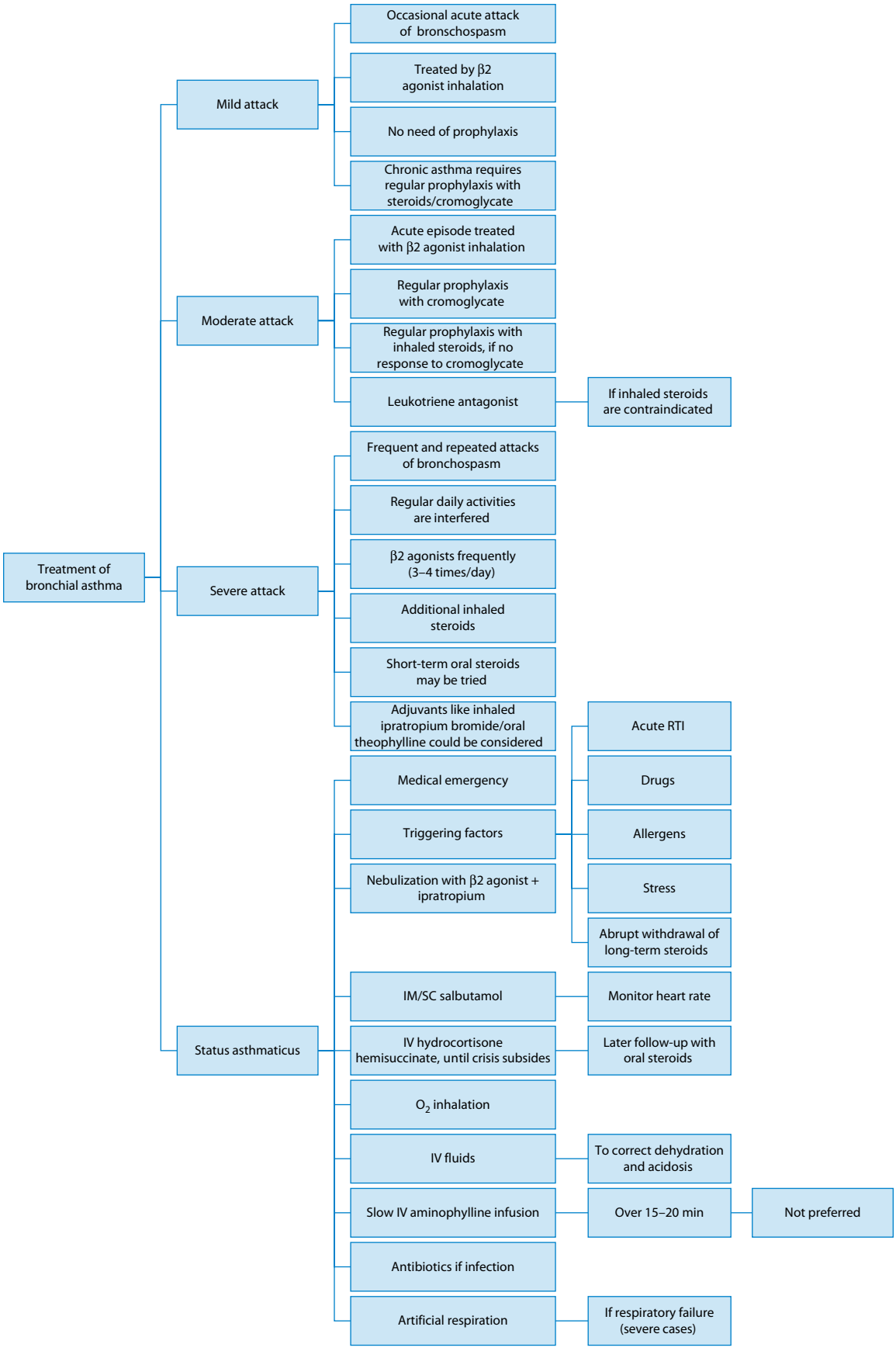
36.7 MAST CELL STABILIZERS



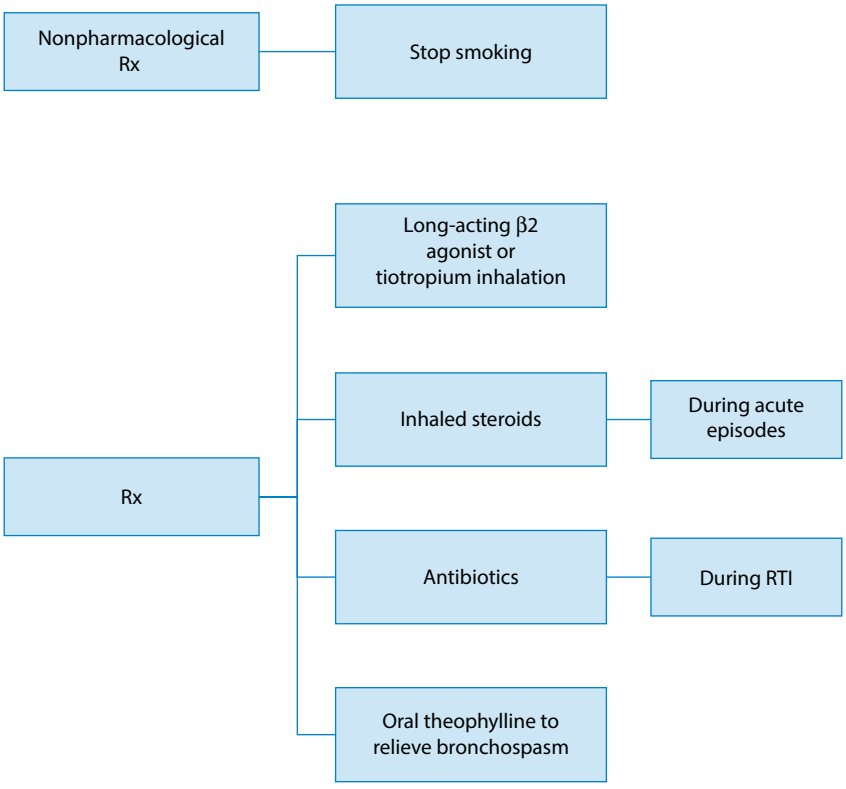
36.8 LEUKOTRIENE RECEPTOR ANTAGONISTS (LRA) AND ANTI-IgE ANTIBODY



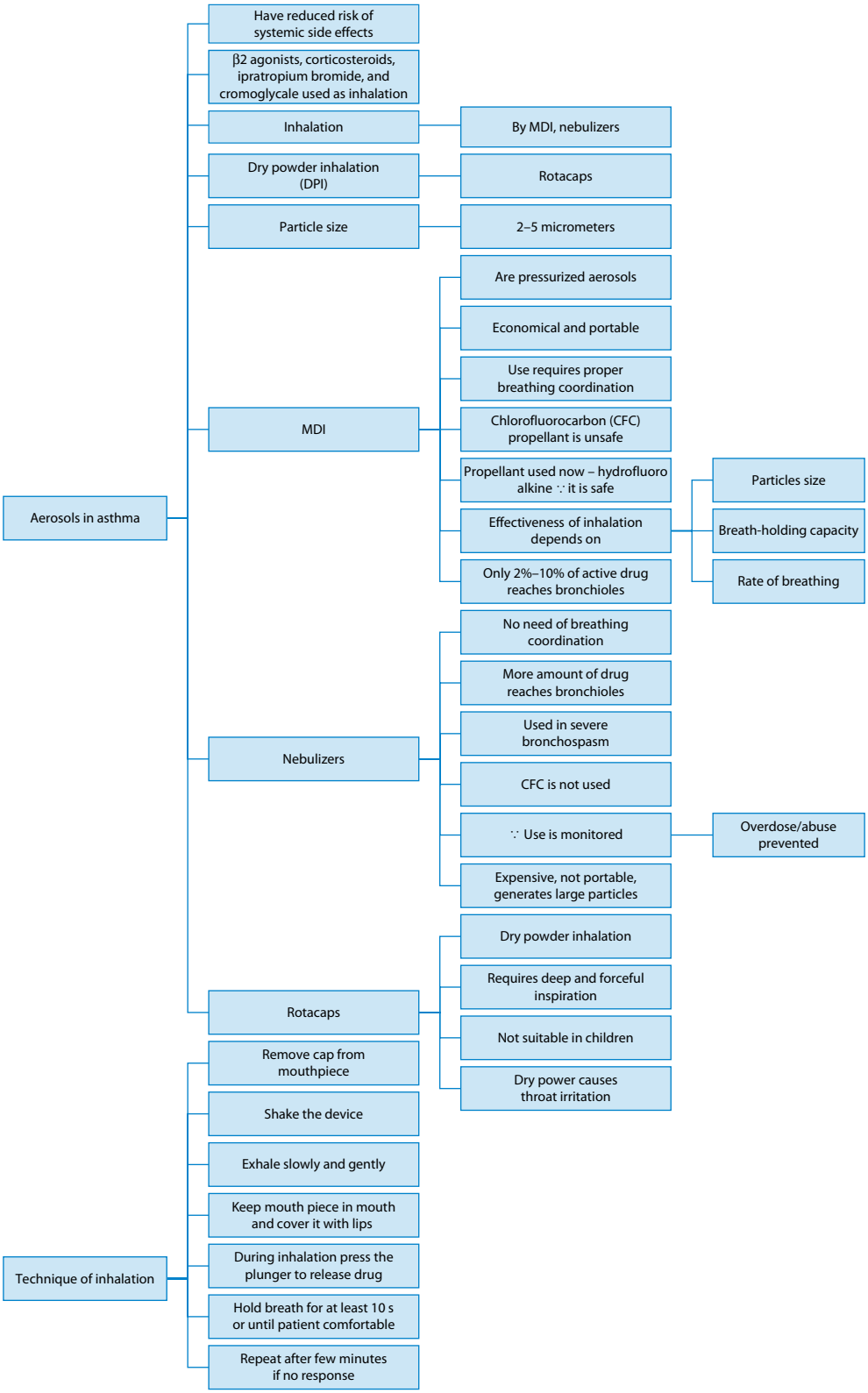
36.9 TREATMENT OF BRONCHIAL ASTHMA



36.10 MANAGEMENT OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)/CHRONIC OBSTRUCTIVE LUNG DISEASE (COLD)



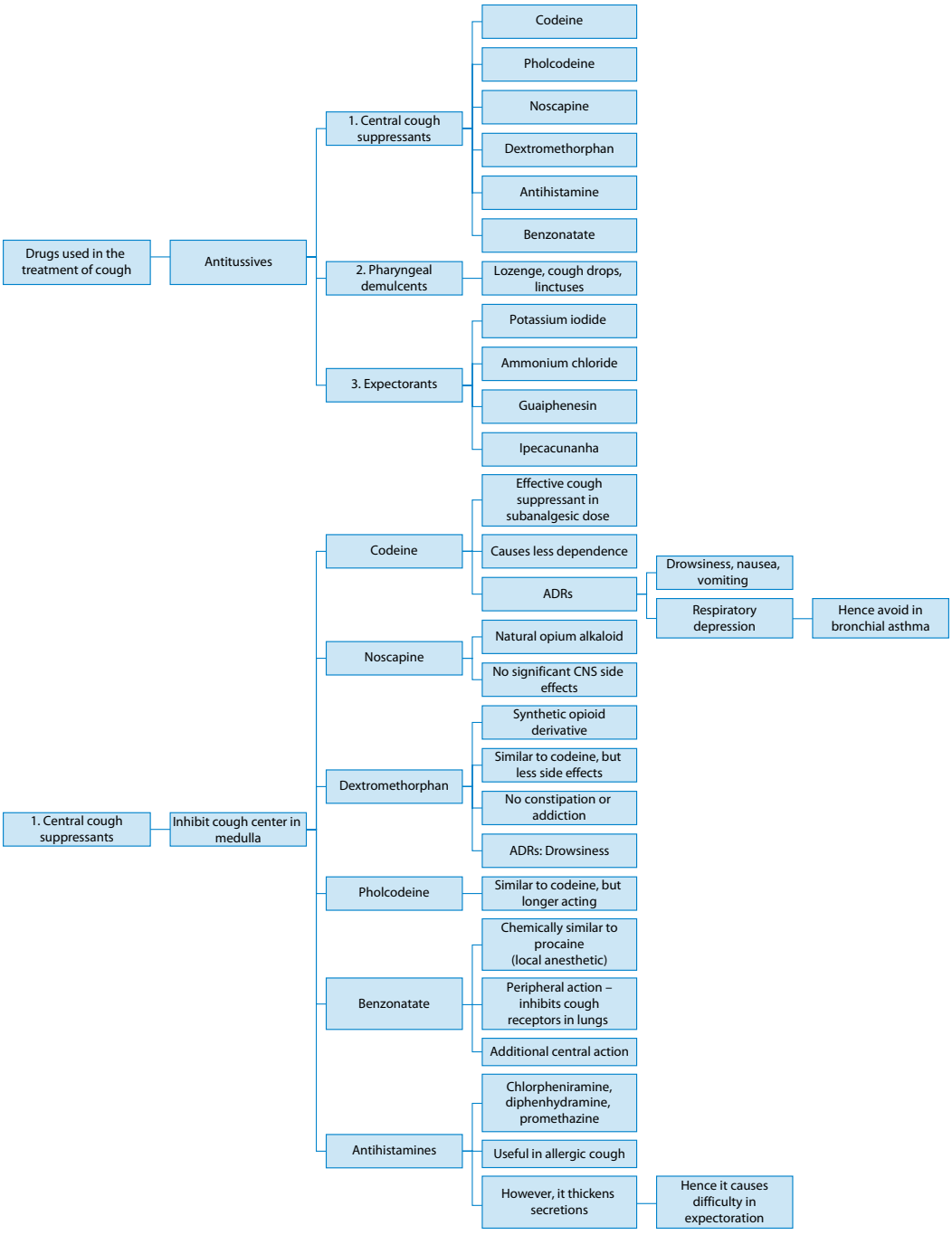
36.11 AEROSOLS IN ASTHMA



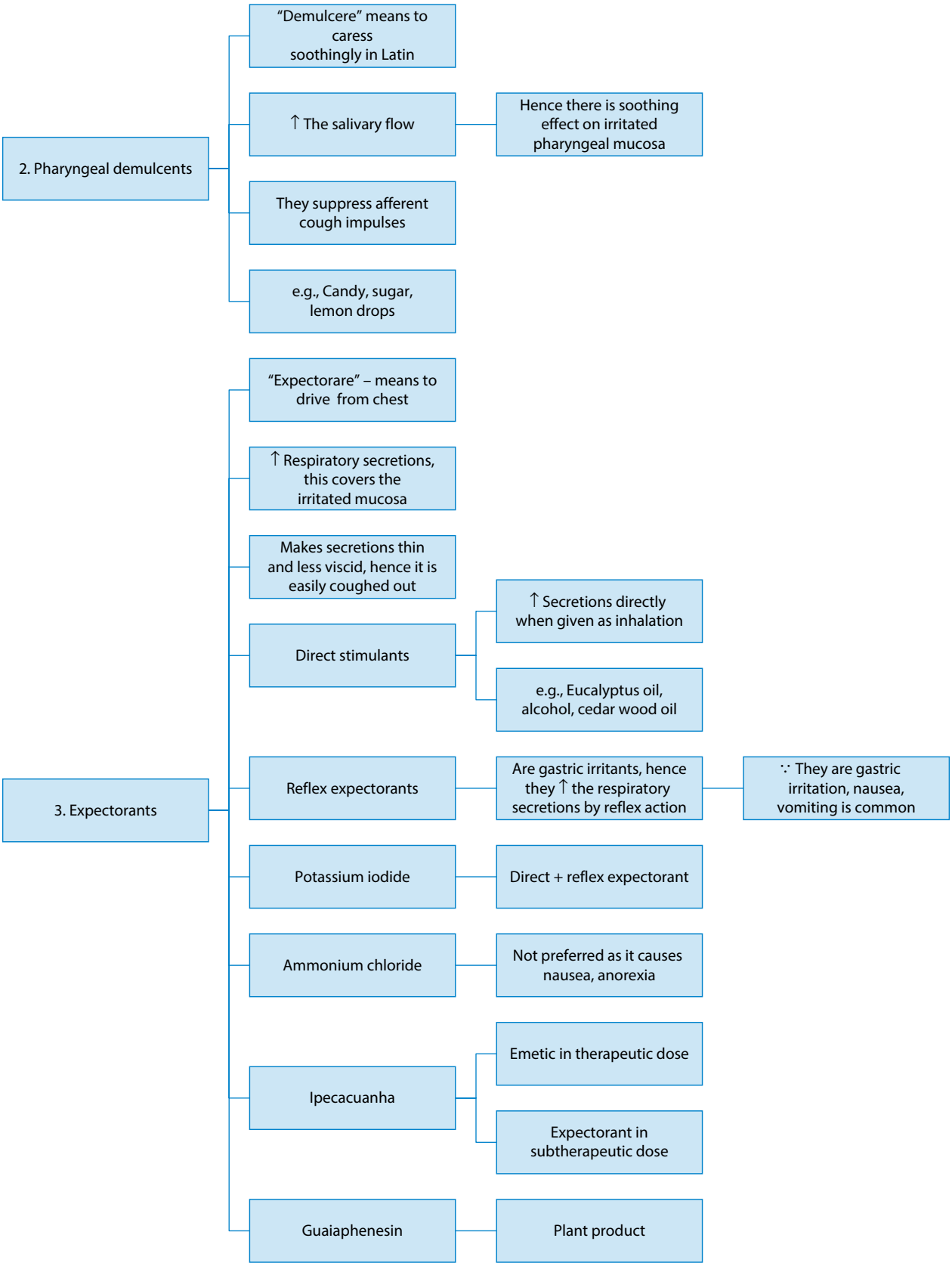
37

Drugs used in the treatment of cough

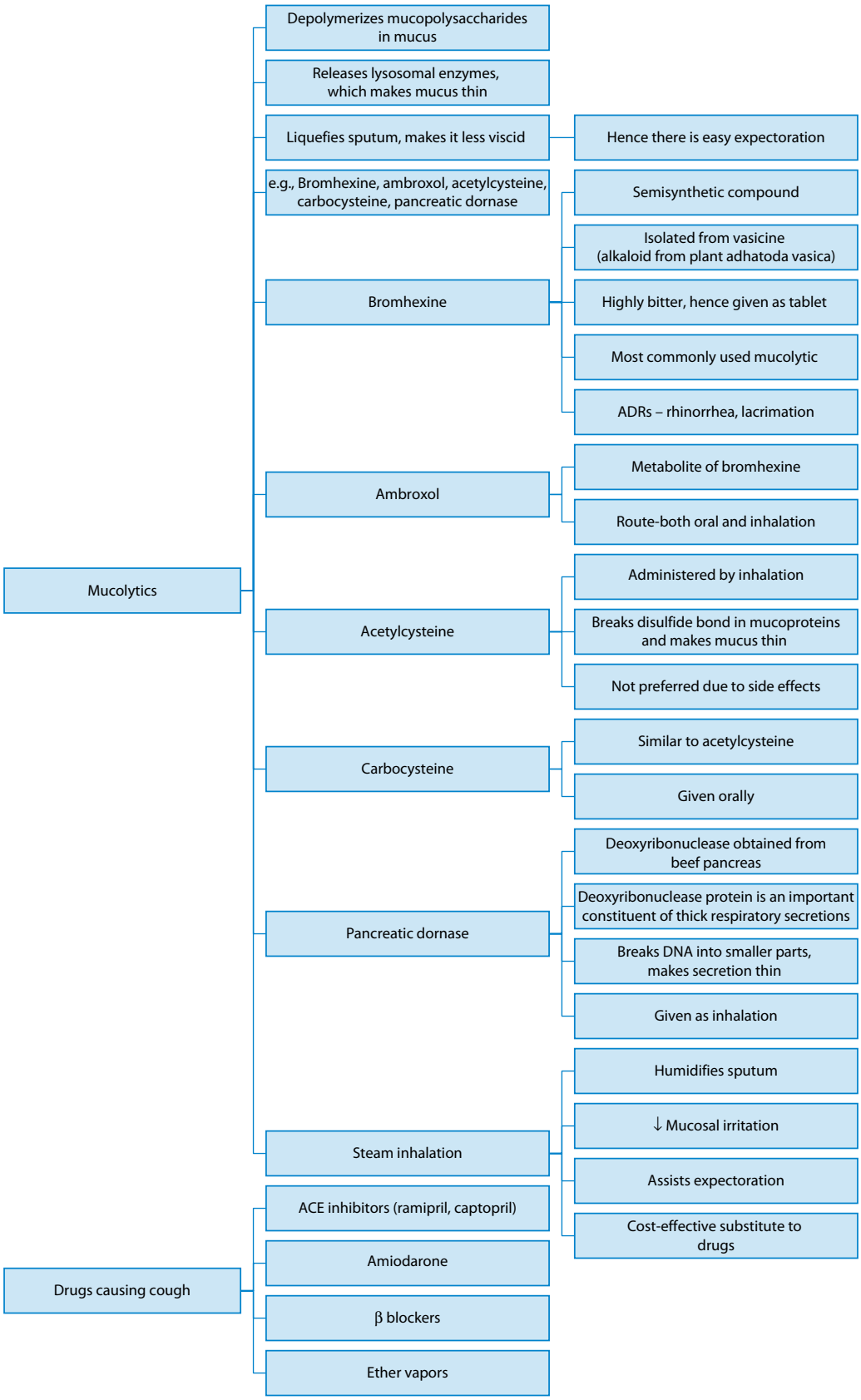
37.1 ANTITUSSIVES AND CENTRAL COUGH SUPPRESSANTS



37.2 PHARYNGEAL DEMULCENTS AND EXPECTORANTS



37.3 MUCOLYTICS AND DRUGS CAUSING COUGH





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http://taylorandfrancis.com

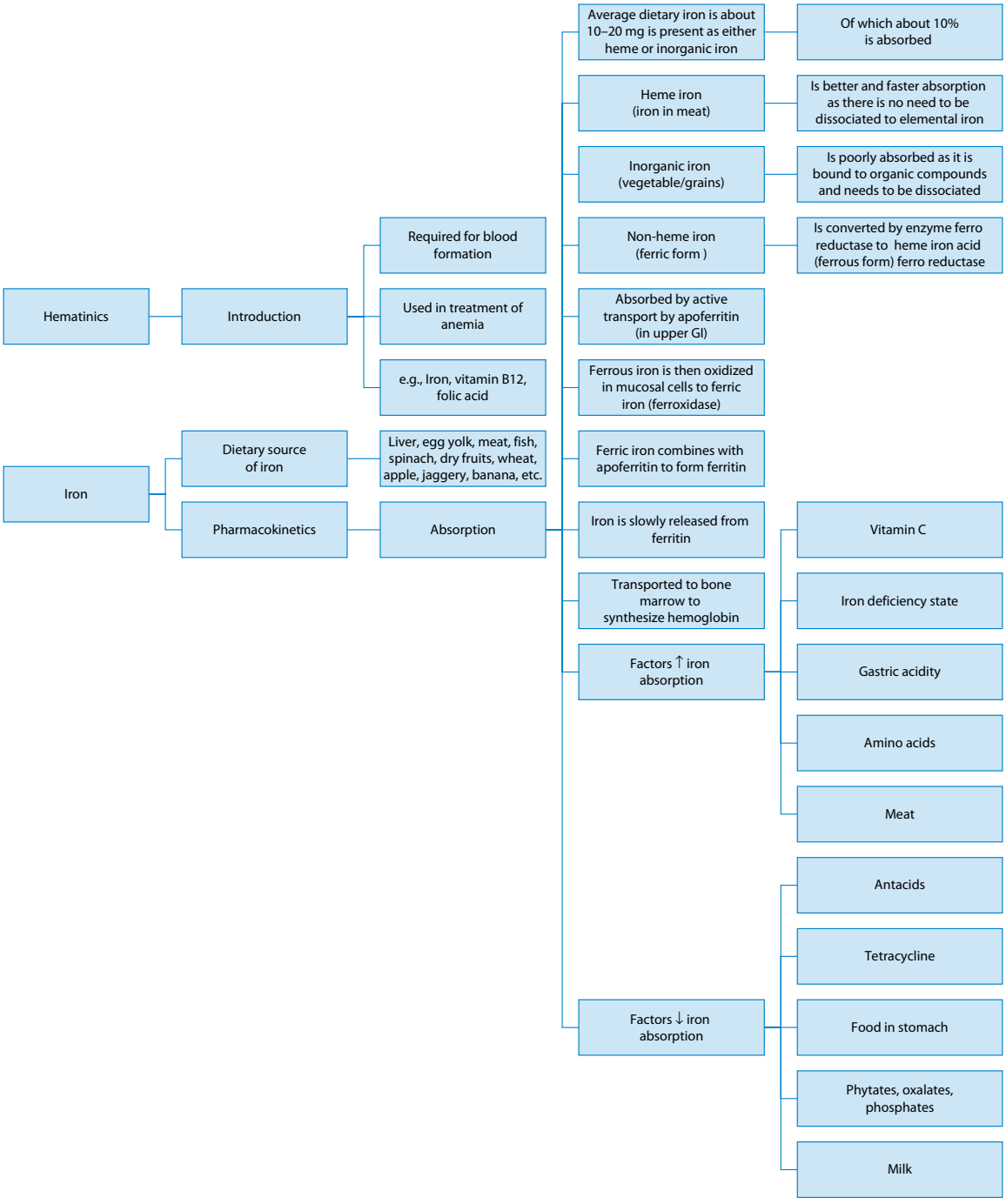
PART VII

Hematological pharmacology

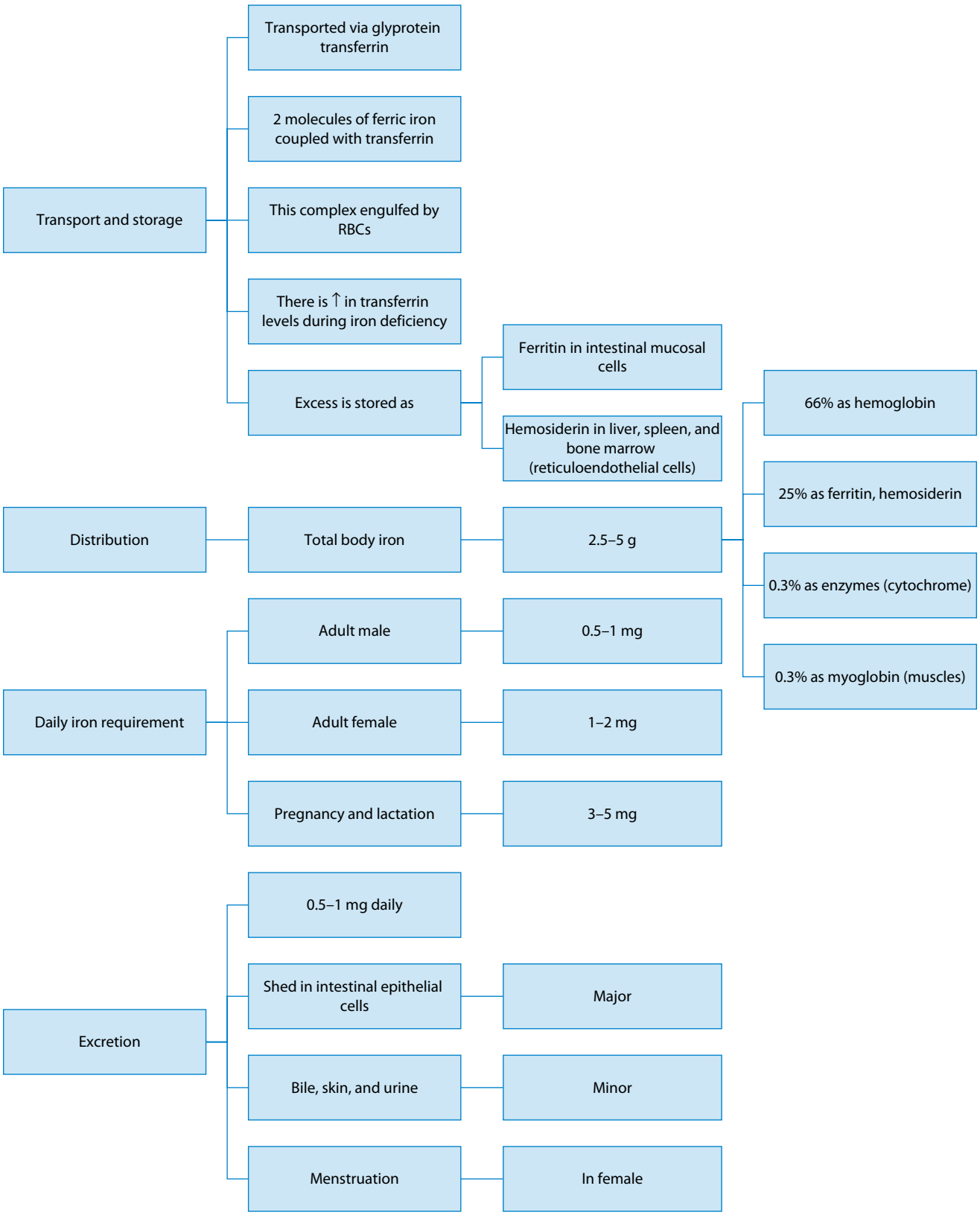
38

Hematinics

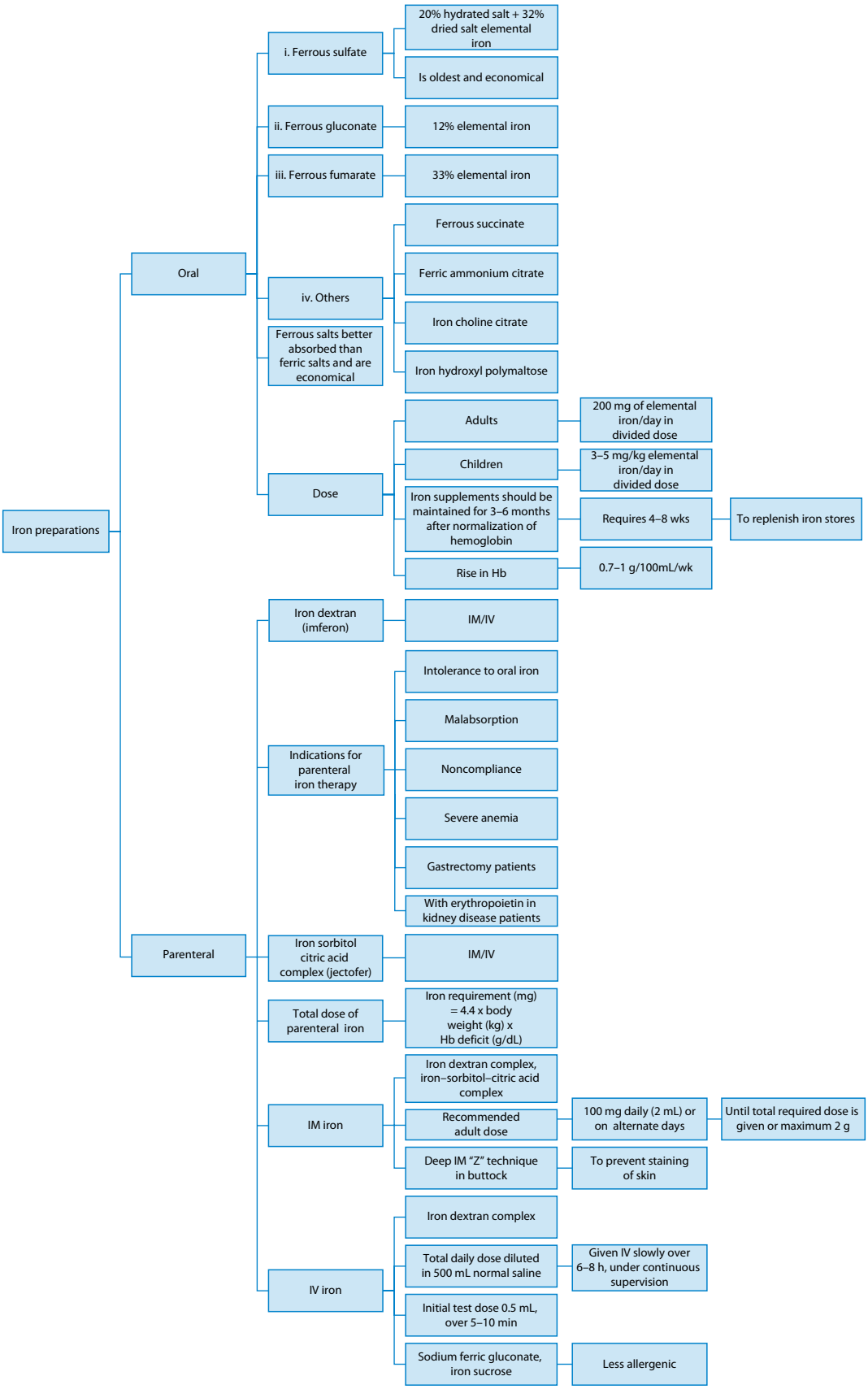
38.1 INTRODUCTION AND IRON ABSORPTION



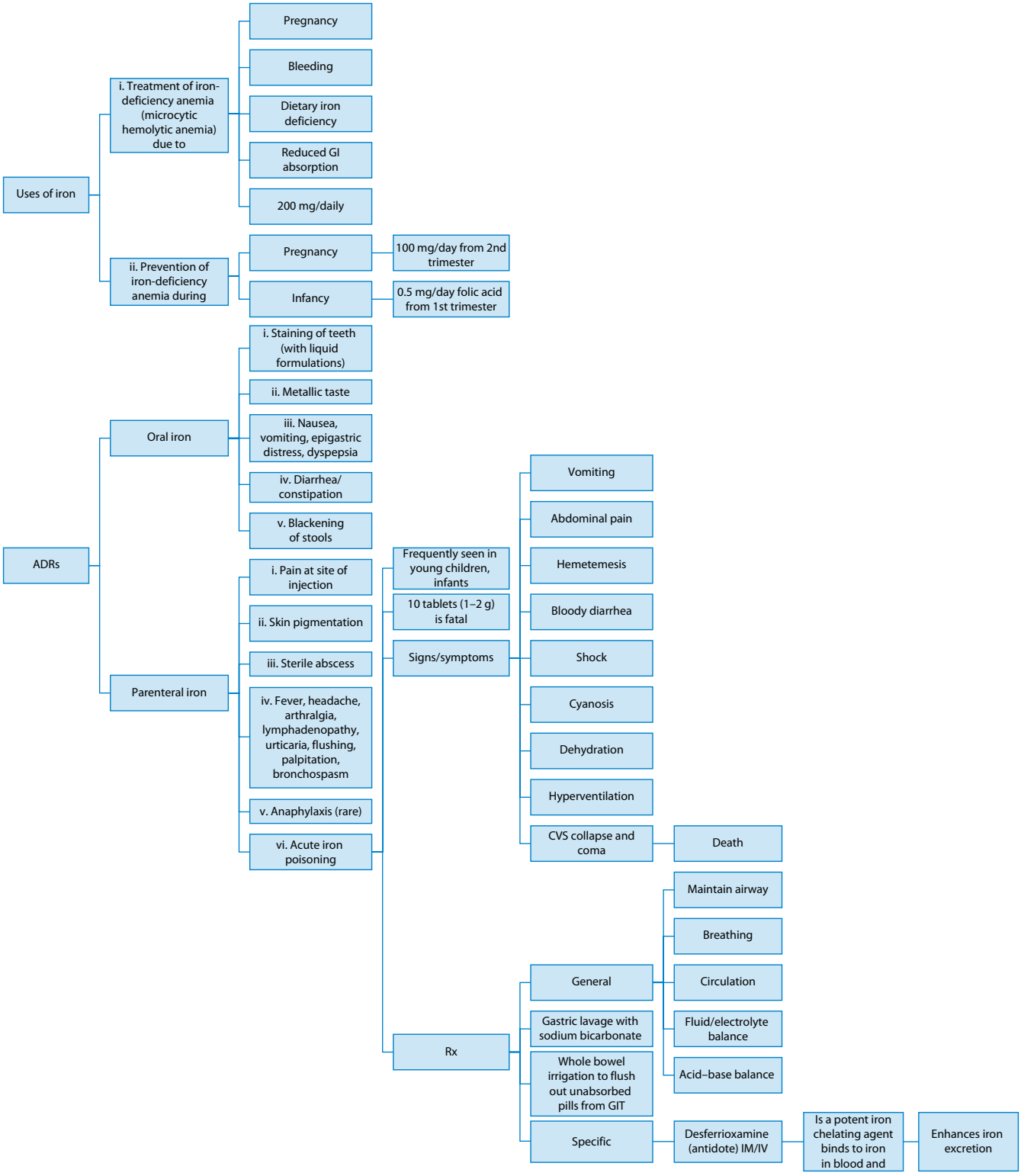
38.2 IRON METABOLISM AND REQUIREMENTS



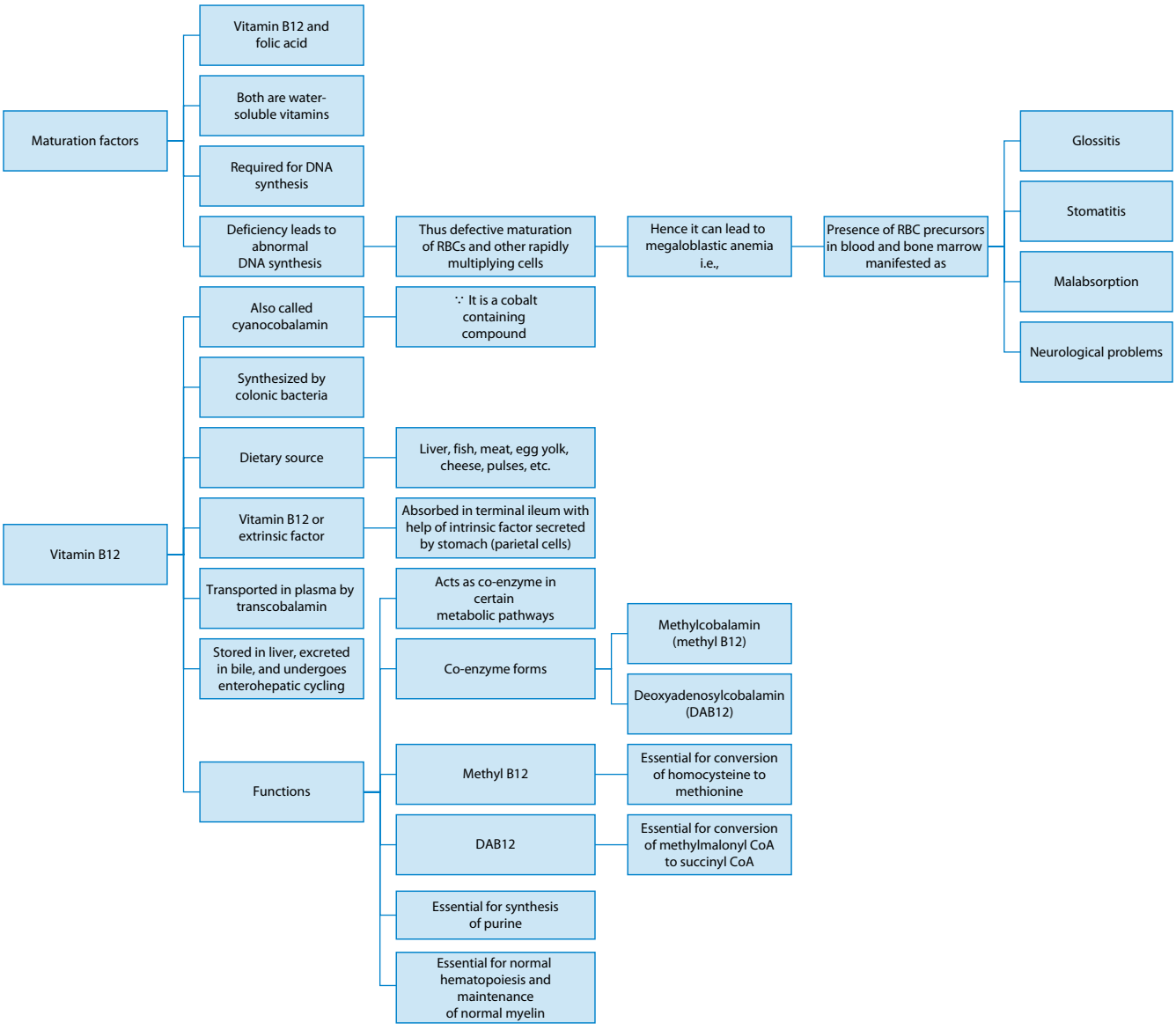
38.3 IRON PREPARATIONS – ORAL AND PARENTERAL



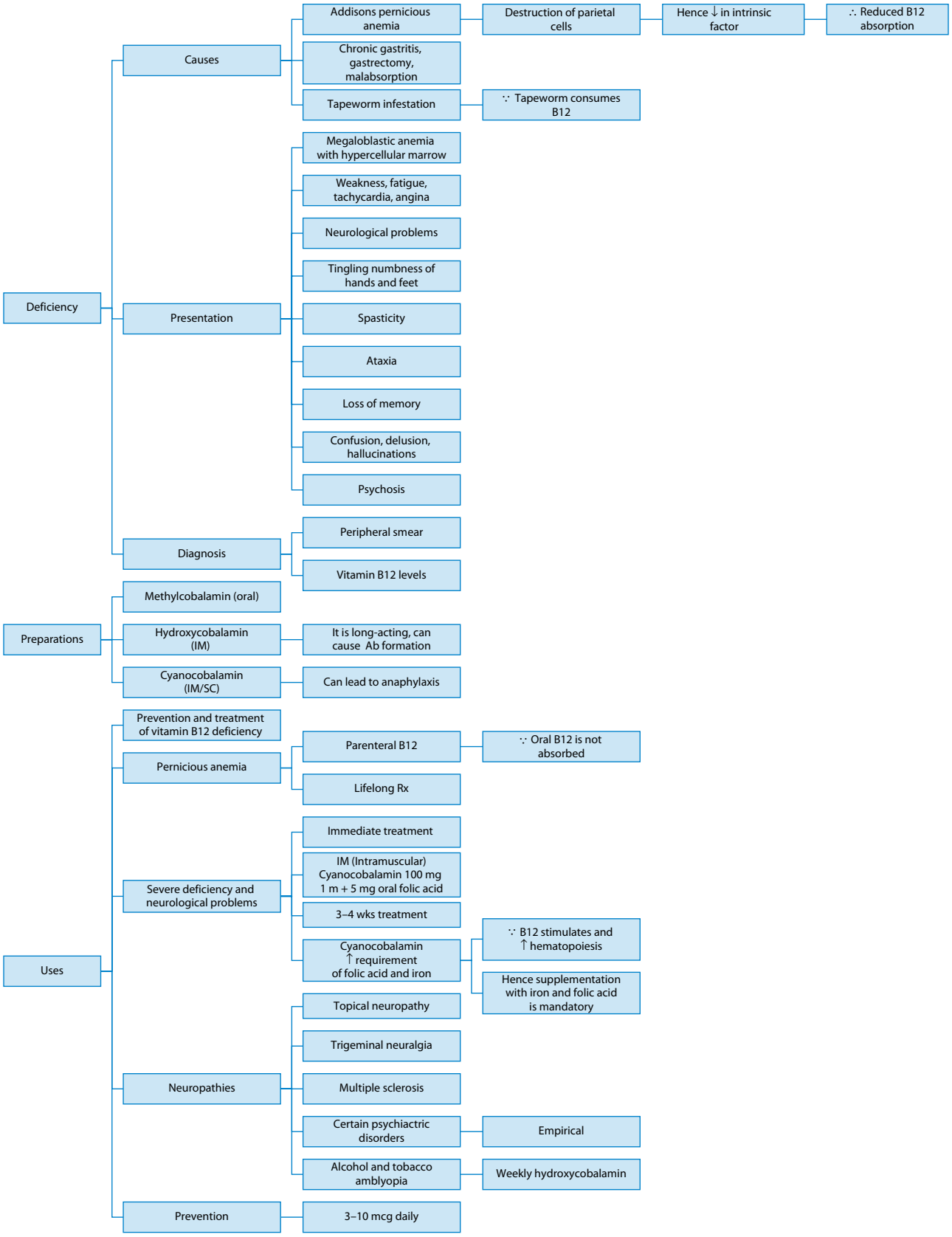
38.4 USES OF IRON AND ADRs



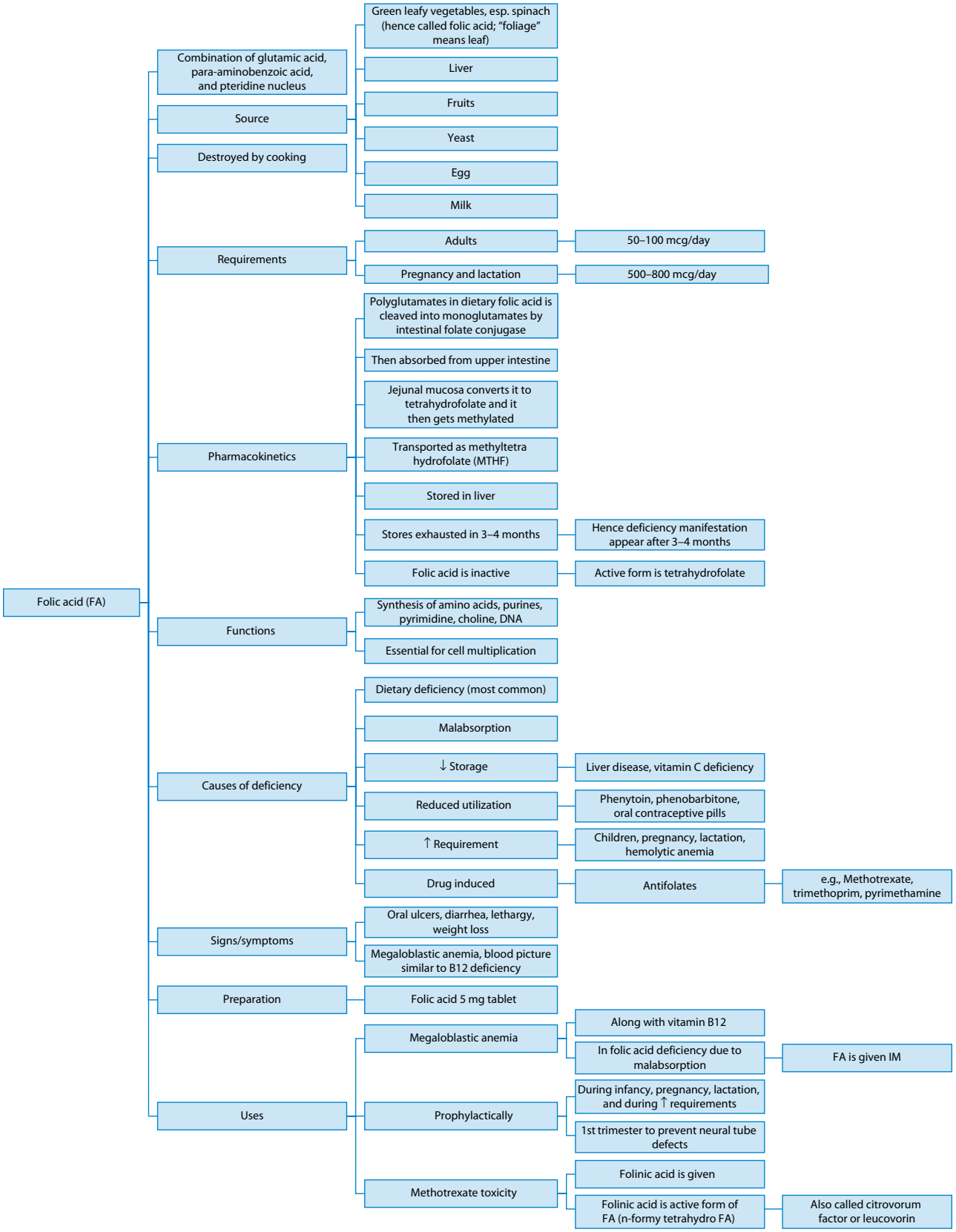
38.5 MATURATION FACTORS AND VITAMIN B12



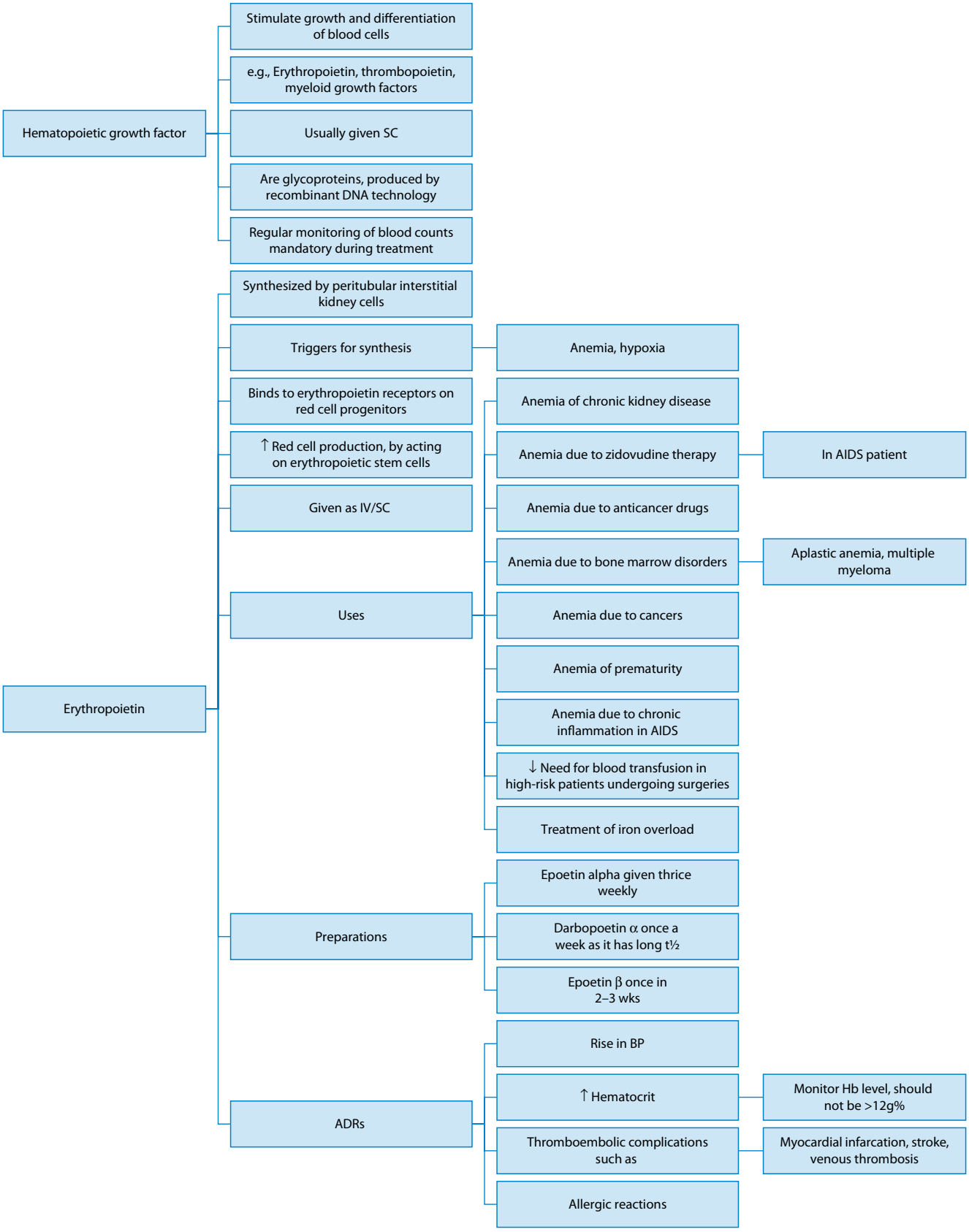
38.6 DEFICIENCY, PREPARATIONS, AND USES



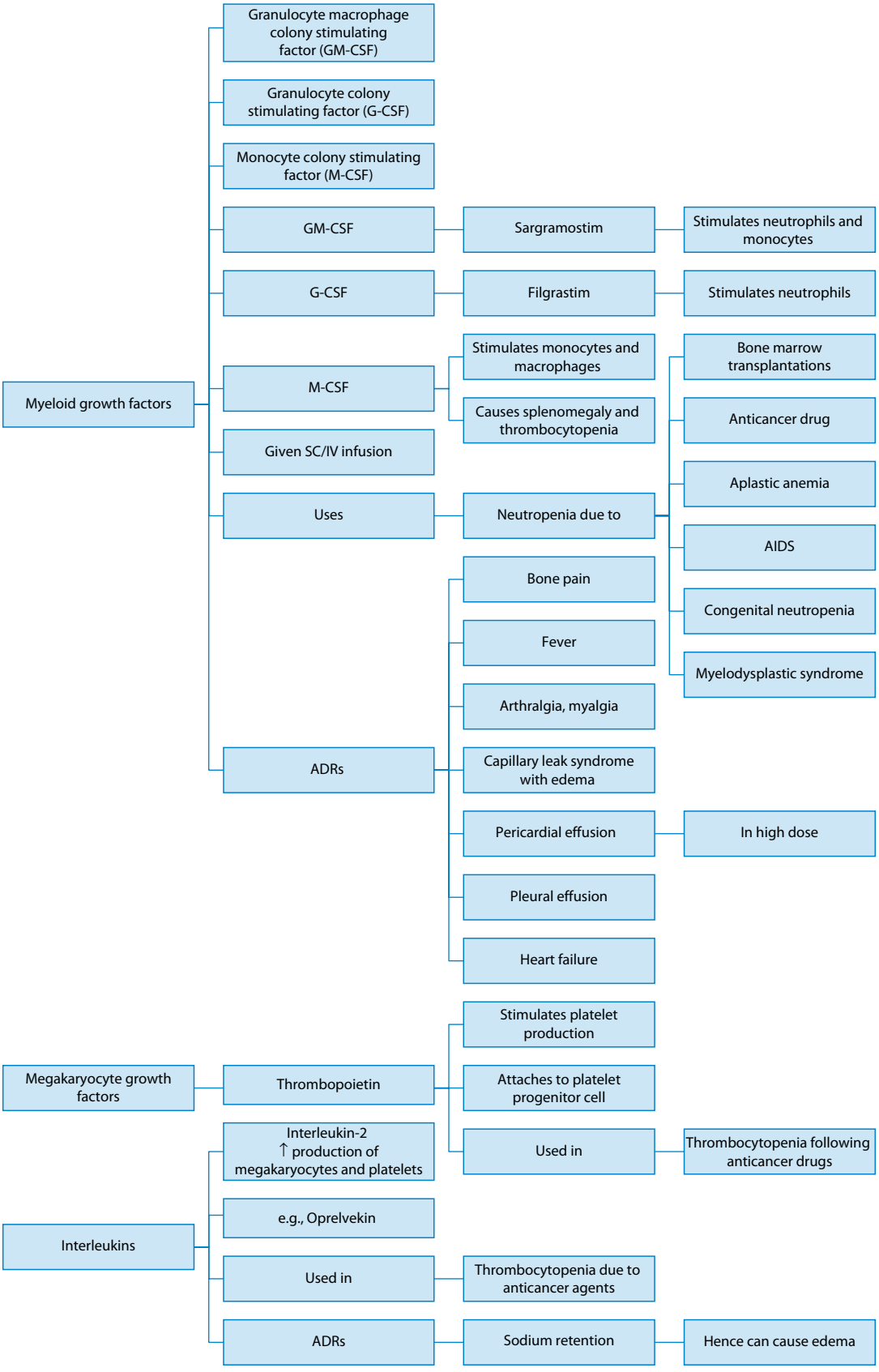
38.7 FOLIC ACID (FA)



38.8 HEMATOPOIETIC GROWTH FACTOR AND ERYTHROPOIETIN



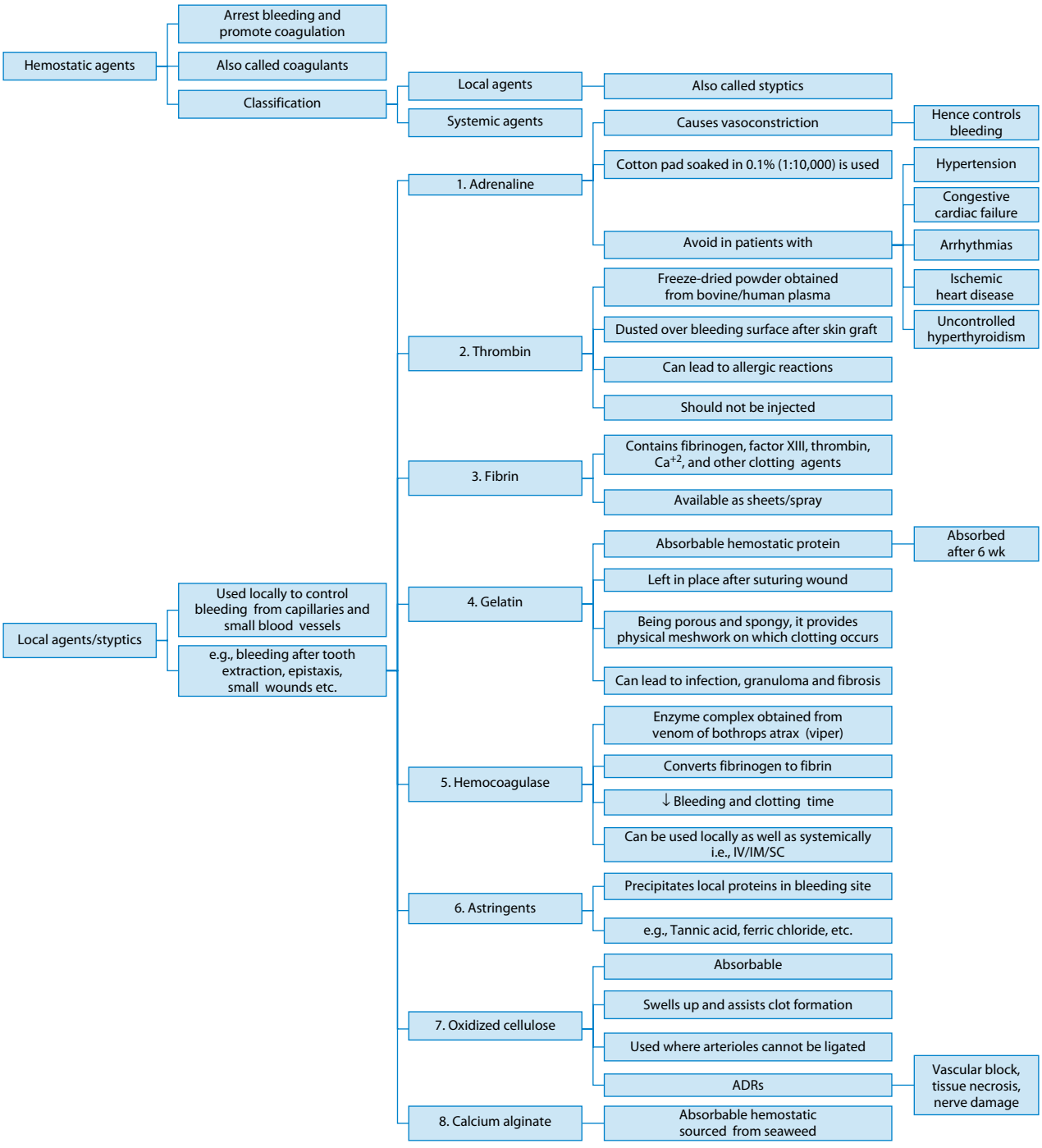
38.9 MYELOID GROWTH FACTORS, MEGAKARYOCYTE GROWTH FACTORS, AND INTERLEUKINS



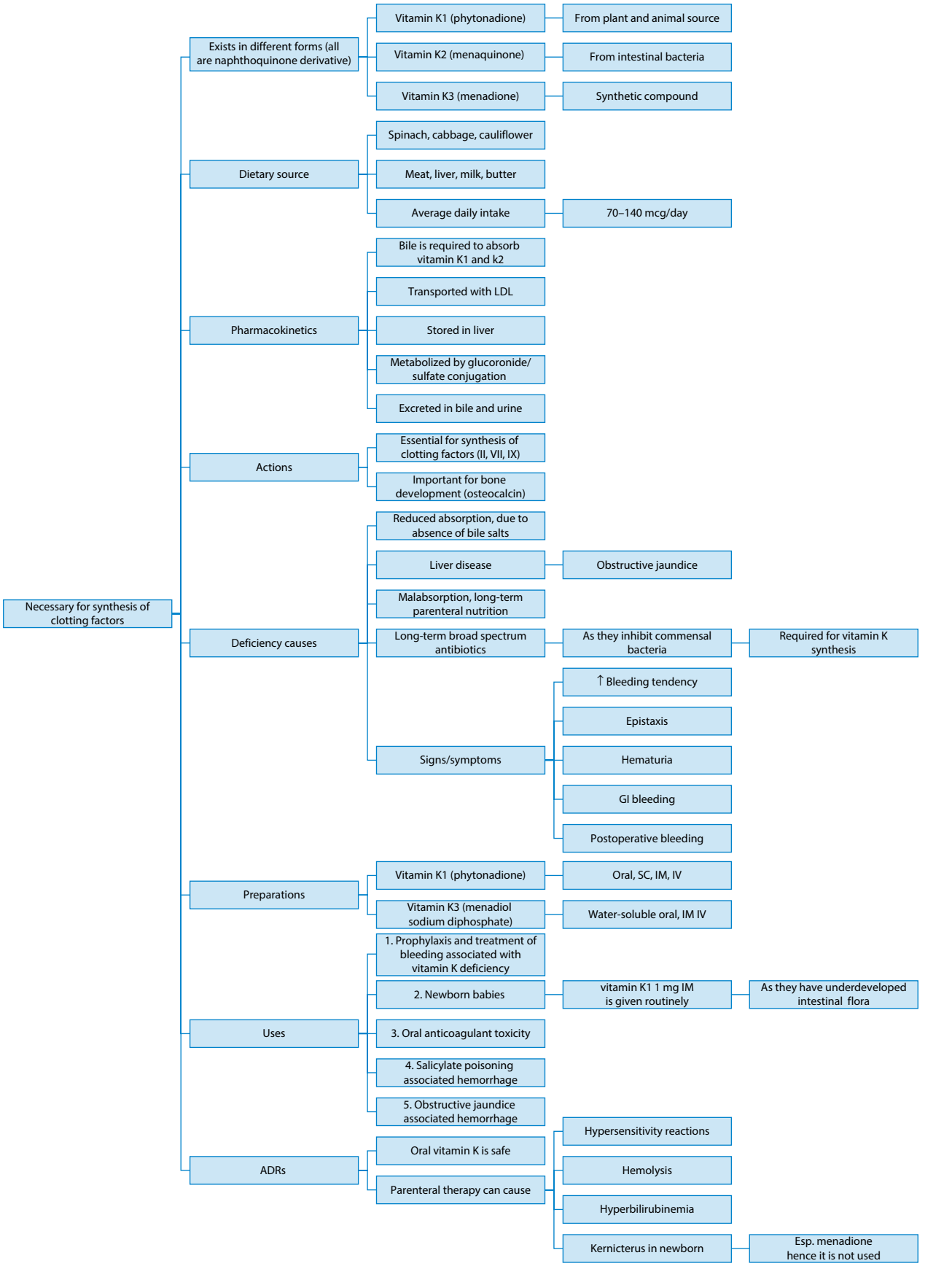
39

Hemostatic agents

39.1 LOCAL AGENTS/STYPTICS

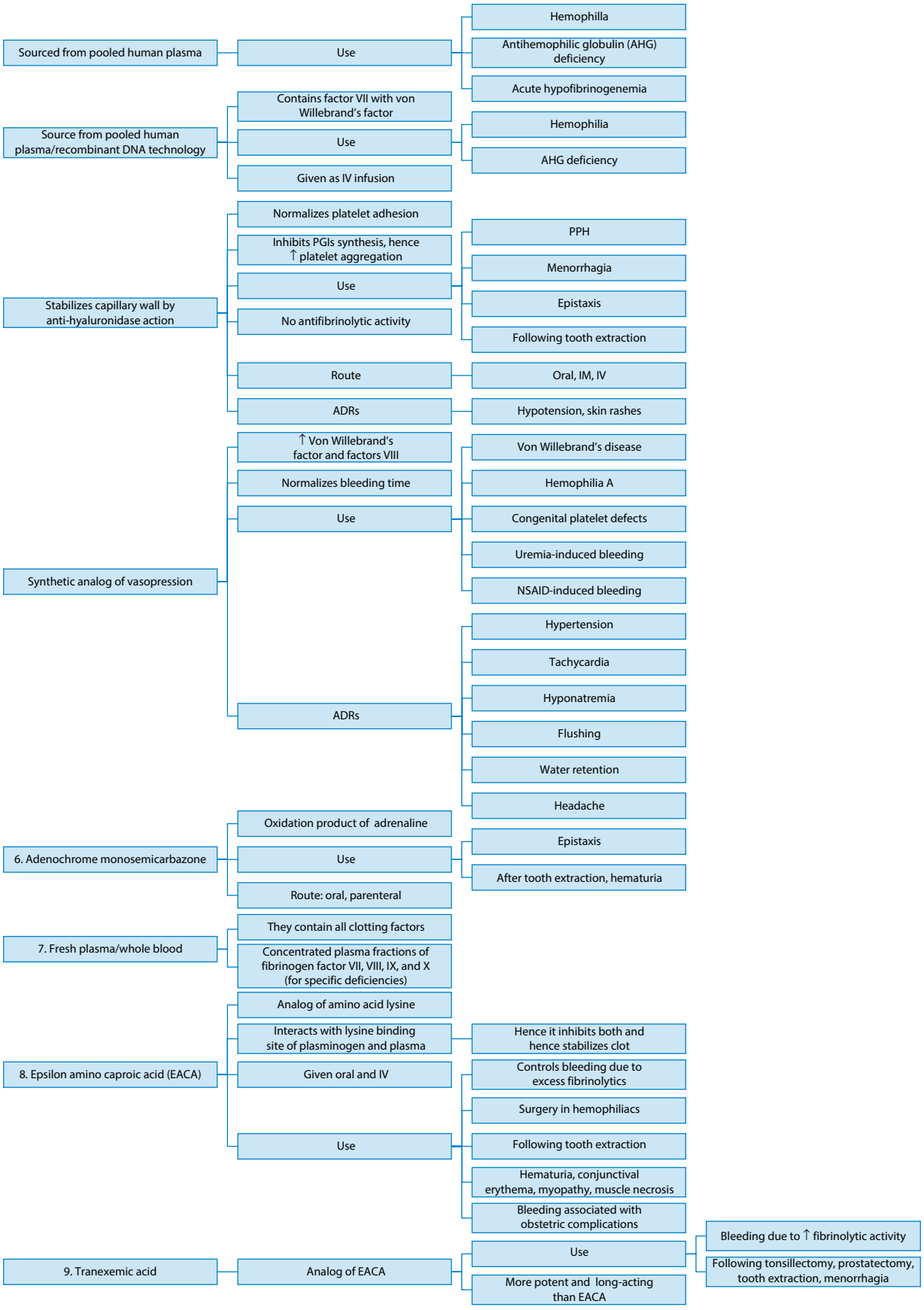


39.2 SYSTEMIC AGENTS

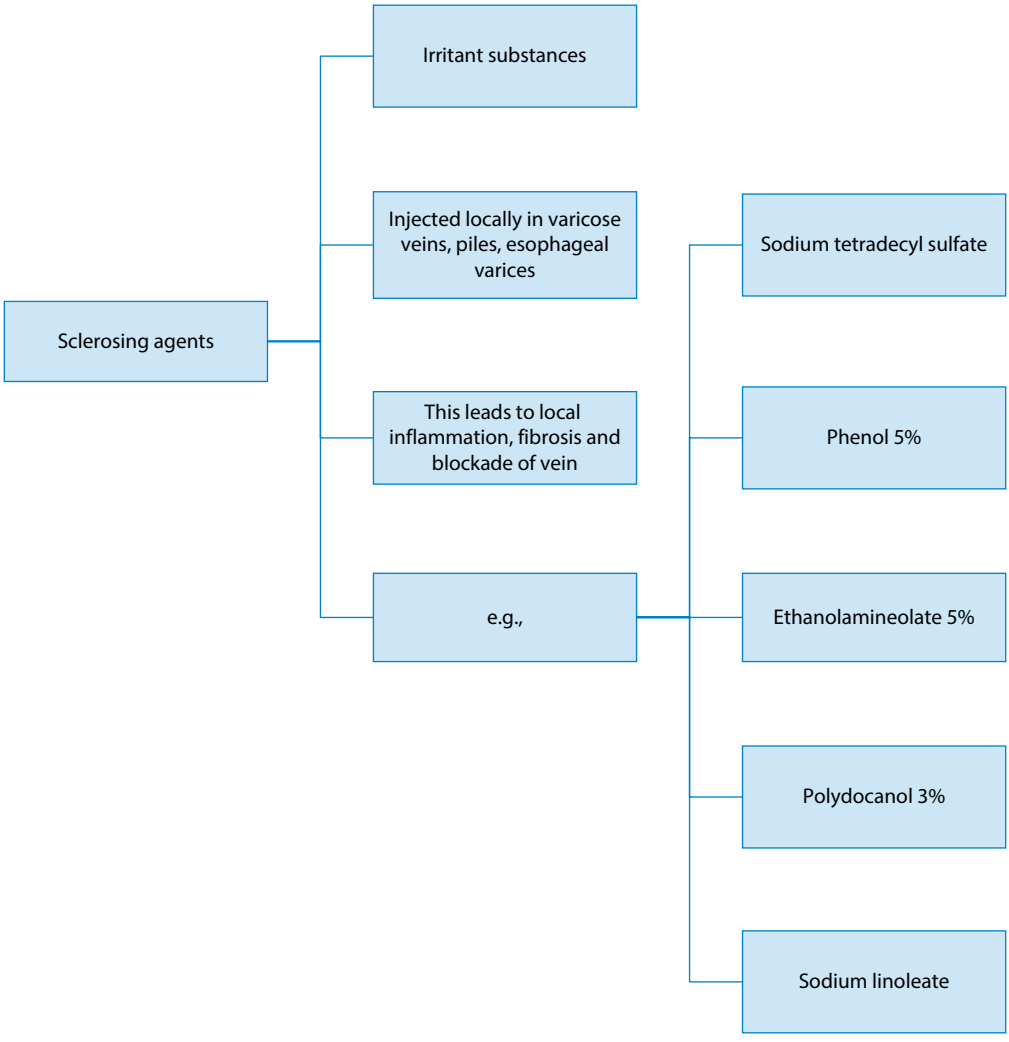


(*Continued*)

39.2 SYSTEMIC AGENTS (Continued)



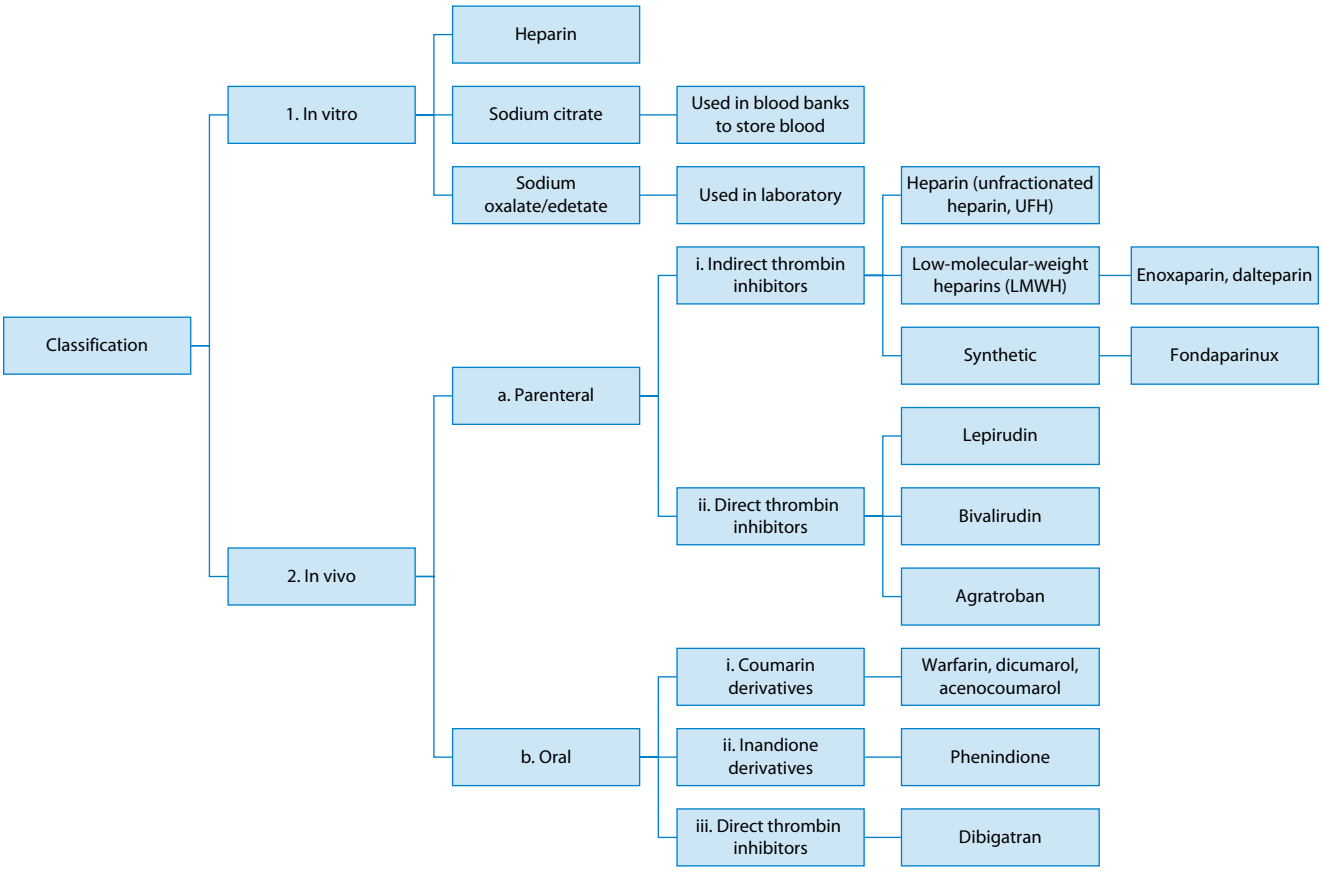
39.3 SCLEROSING AGENTS



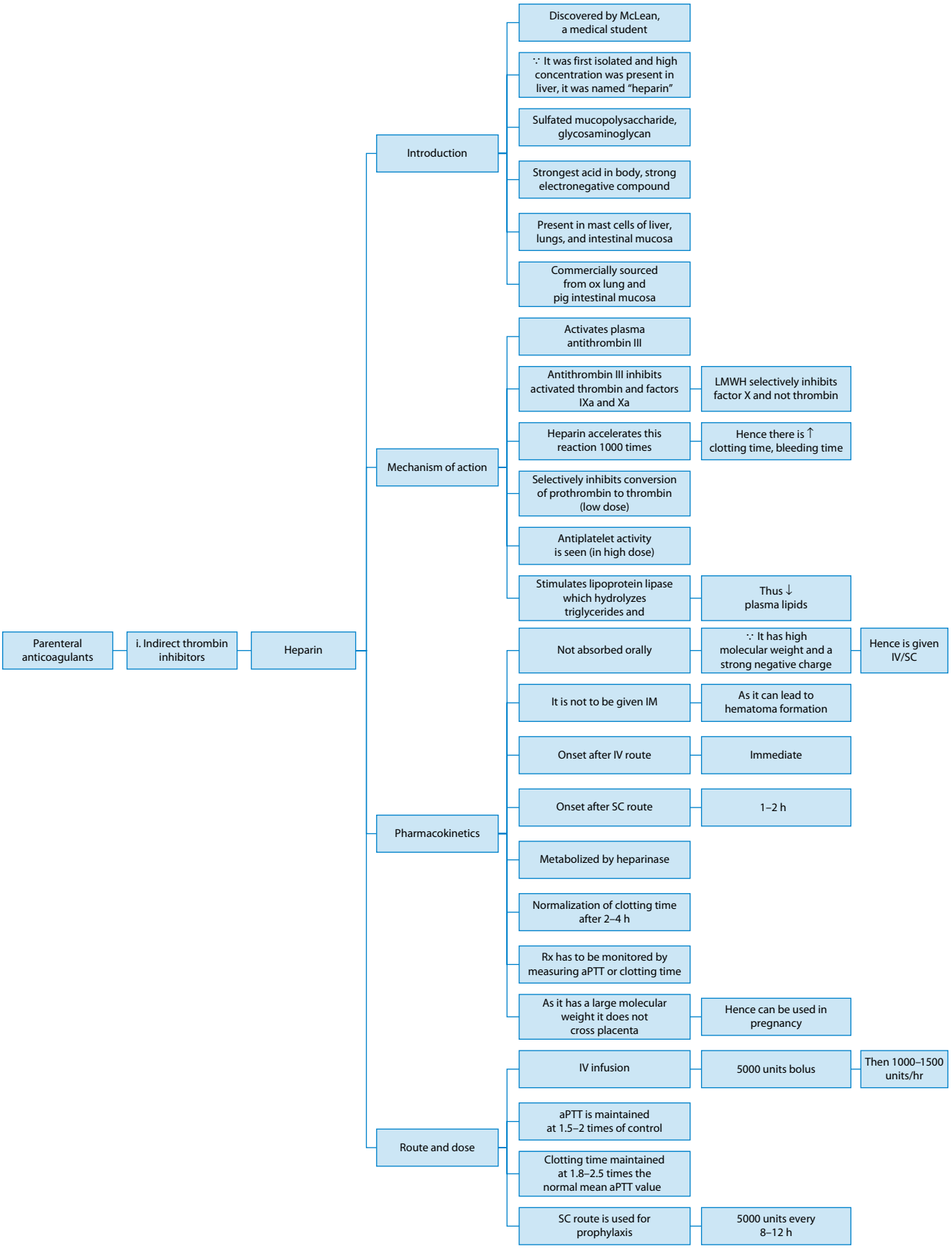
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Anticoagulants

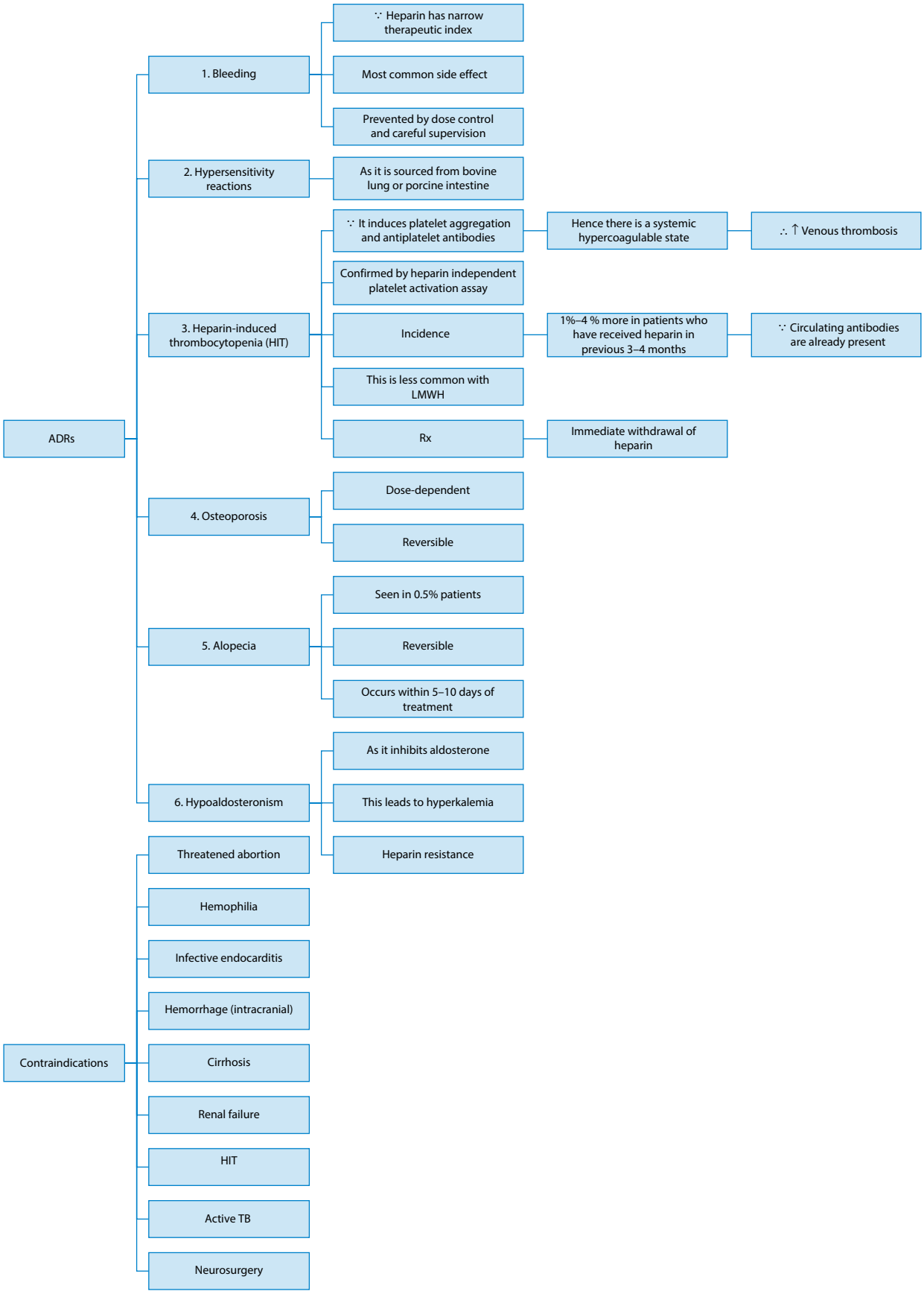
40.1 ANTICOAGULANTS – CLASSIFICATION



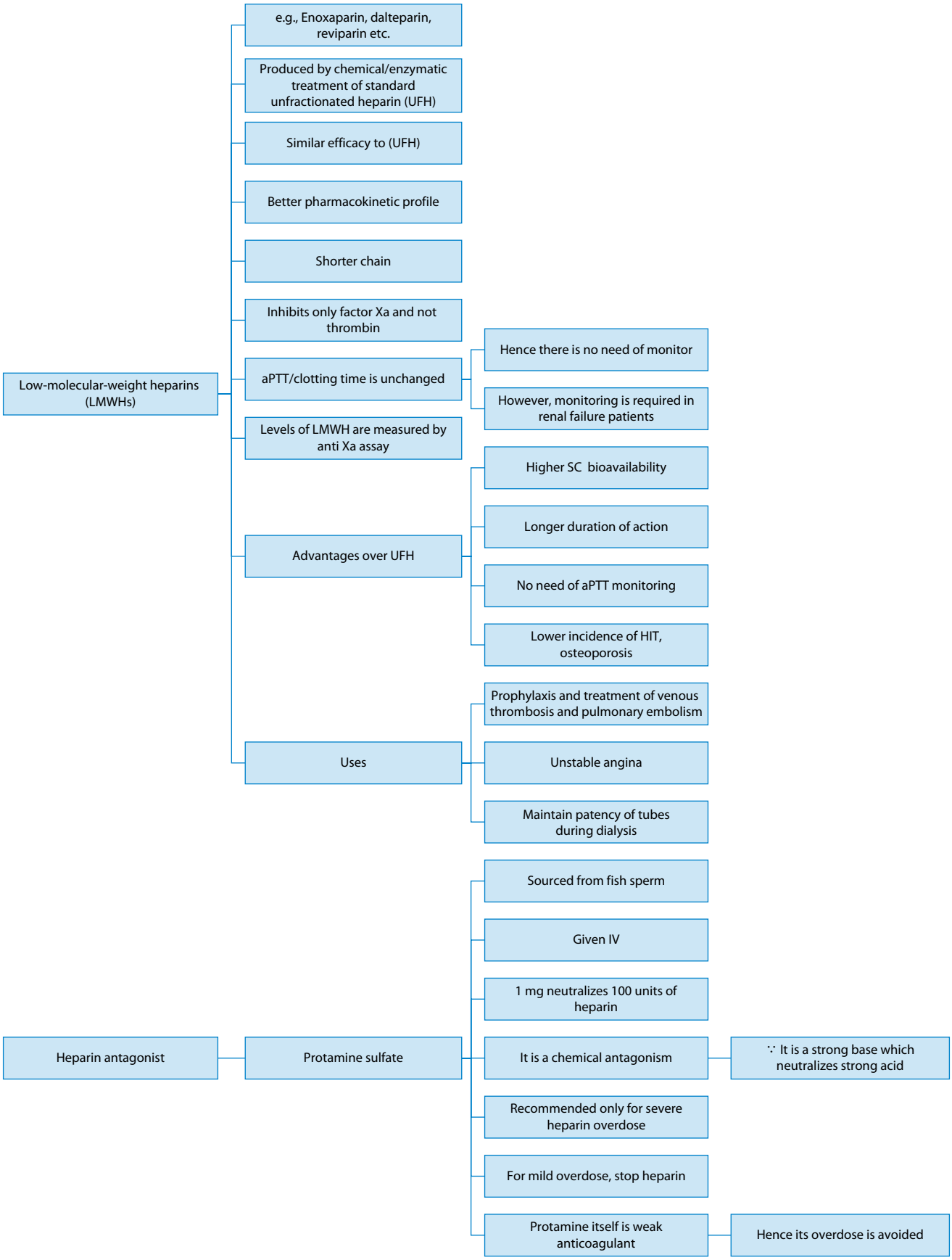
40.2 PARENTERAL ANTICOAGULANTS – HEPARIN



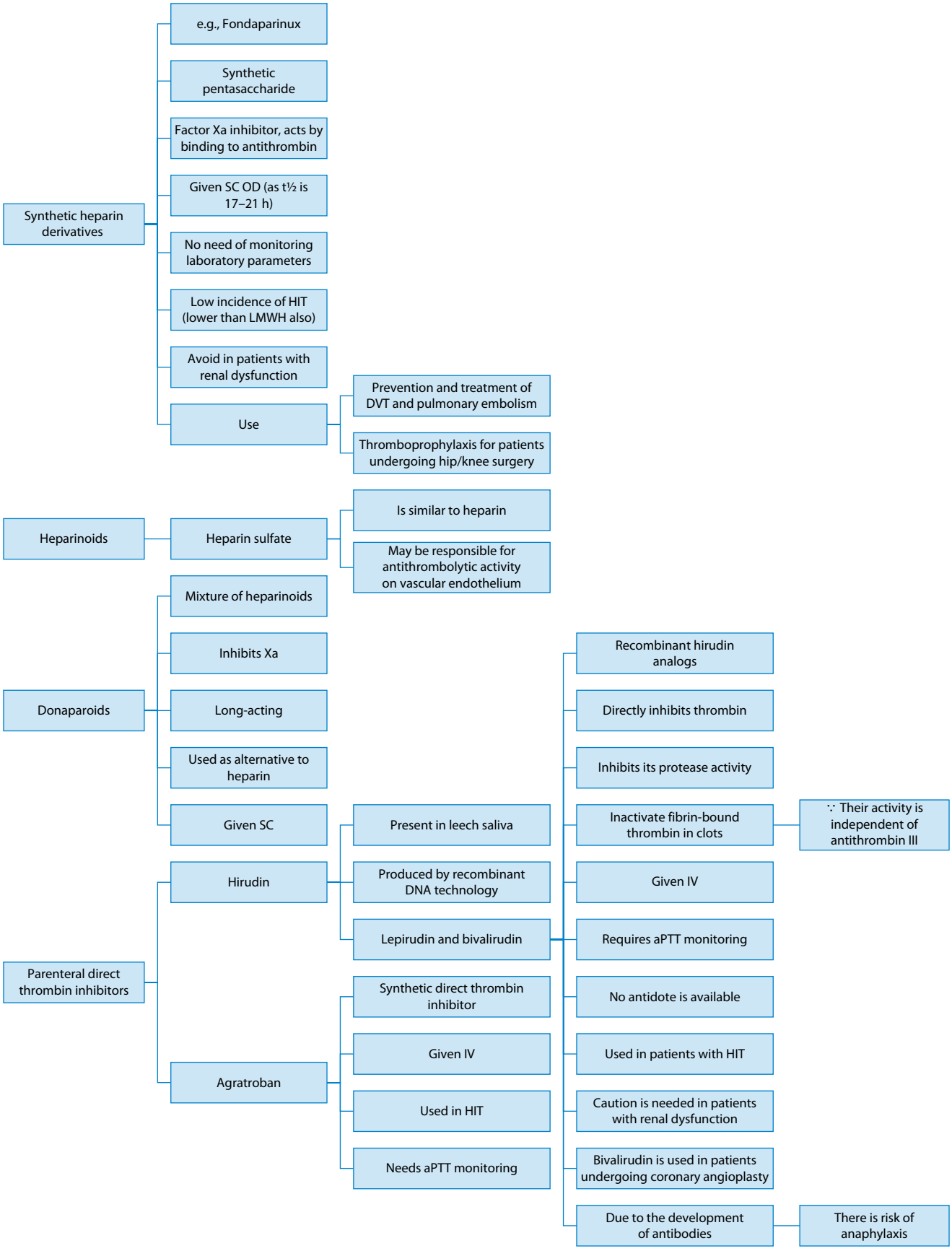
40.3 ADRs AND CONTRAINDICATIONS OF HEPARIN



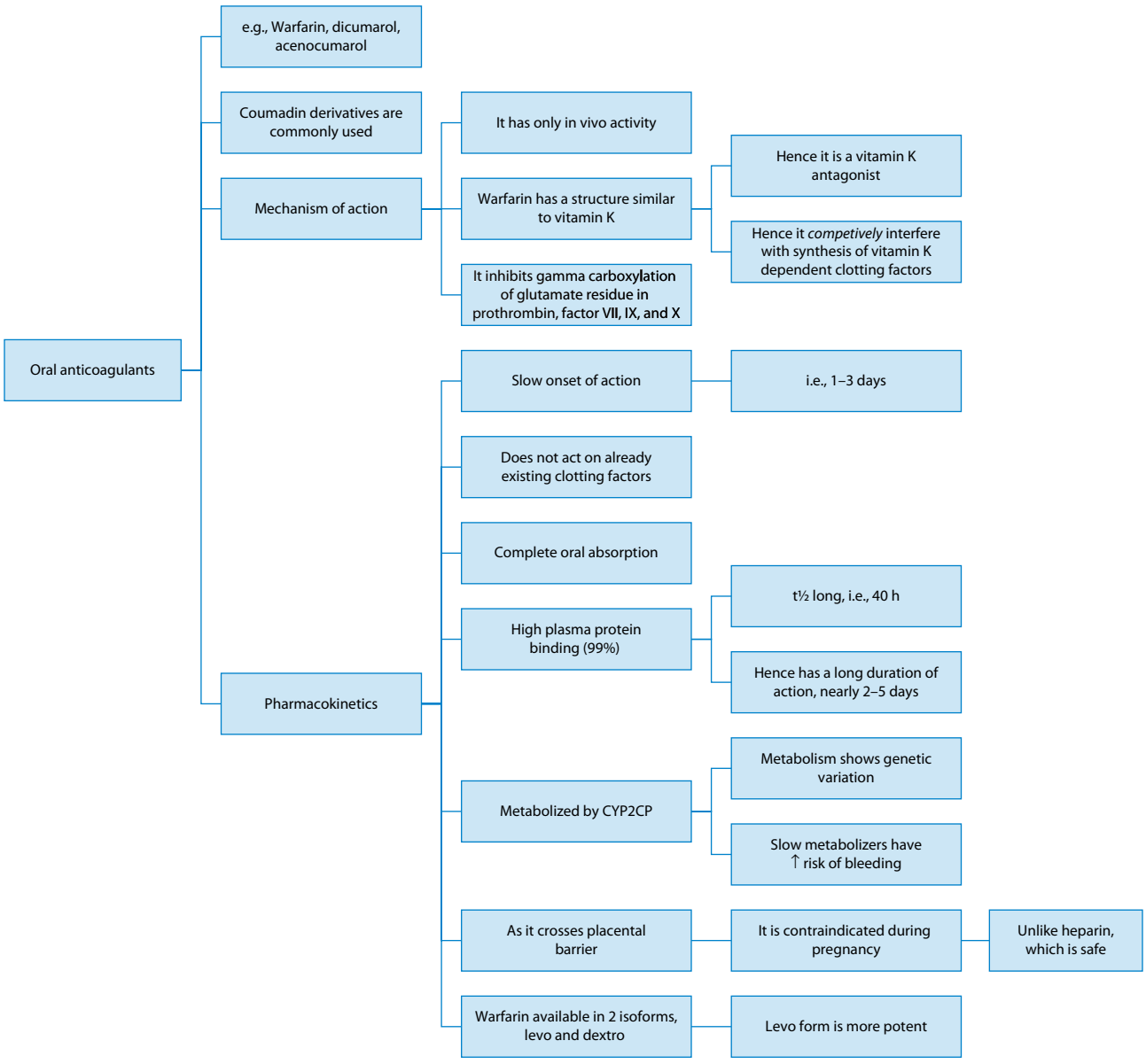
40.4 LOW-MOLECULAR-WEIGHT HEPARINS (LMWHs) AND HEPARIN ANTAGONIST



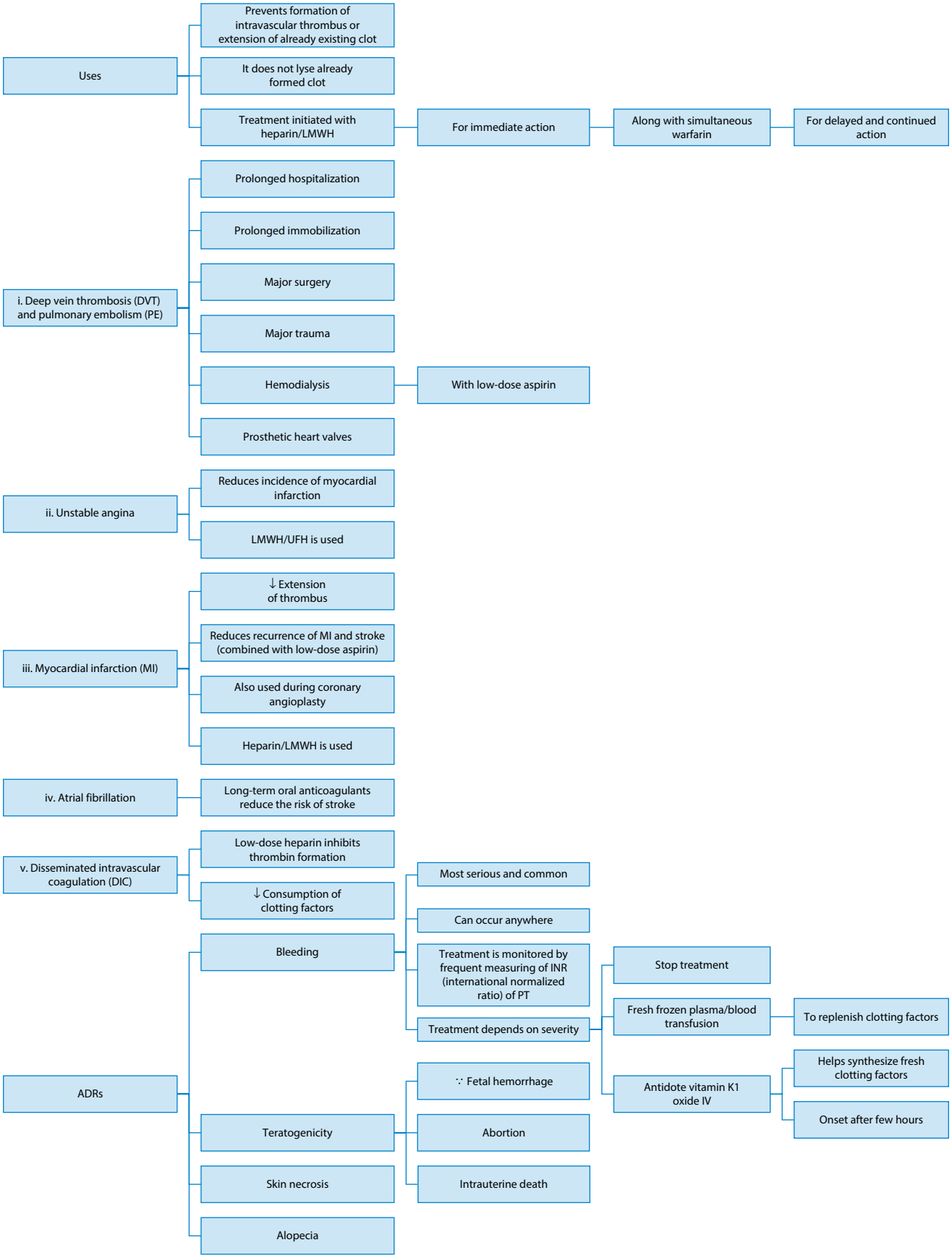
40.5 SYNTHETIC HEPARIN DERIVATIVES, HEPARINOIDS, AND PARENTERAL DIRECT THROMBIN INHIBITORS



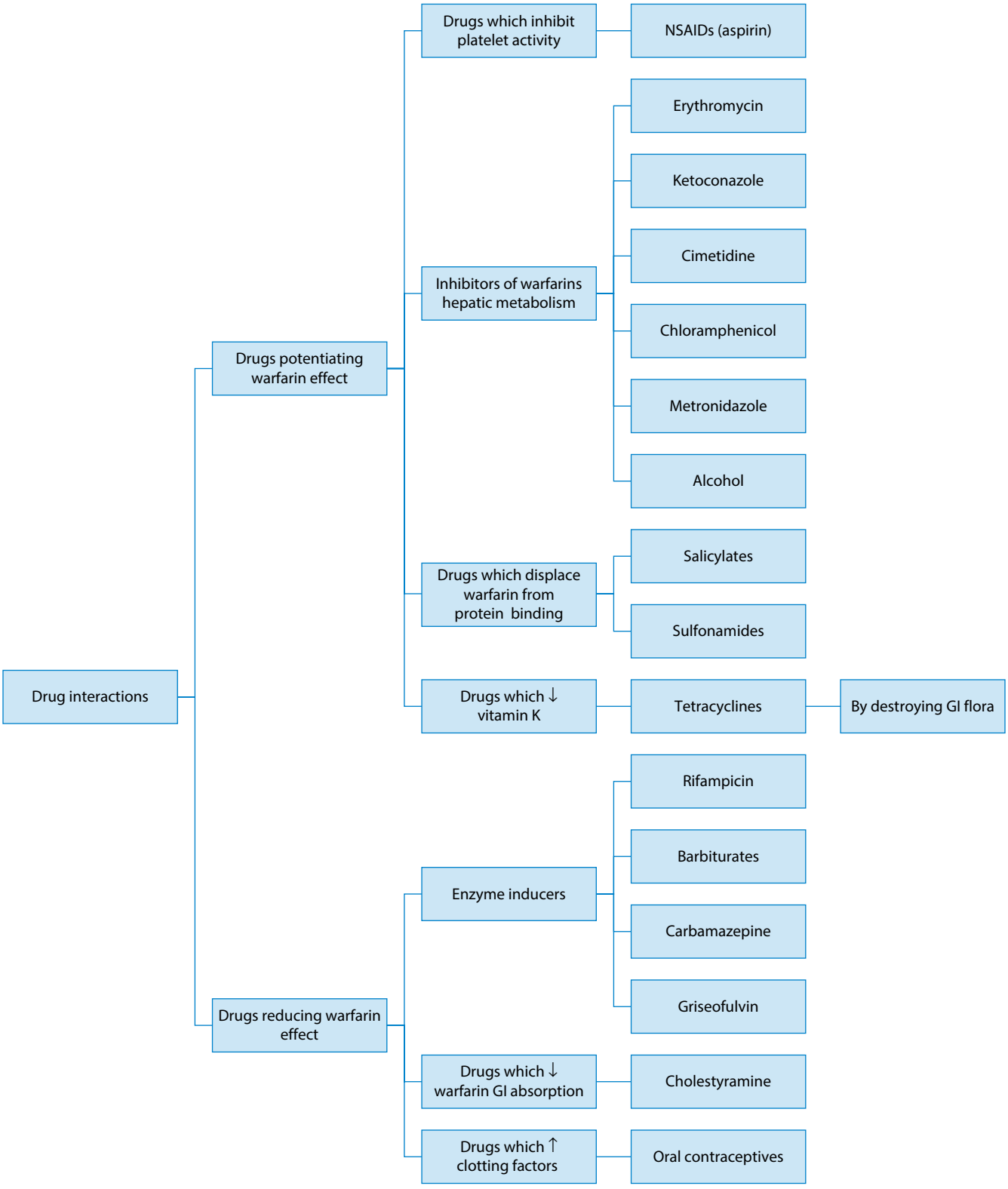
40.6 ORAL ANTICOAGULANTS – MECHANISM OF ACTION AND PHARMACOKINETICS (WARFARIN)



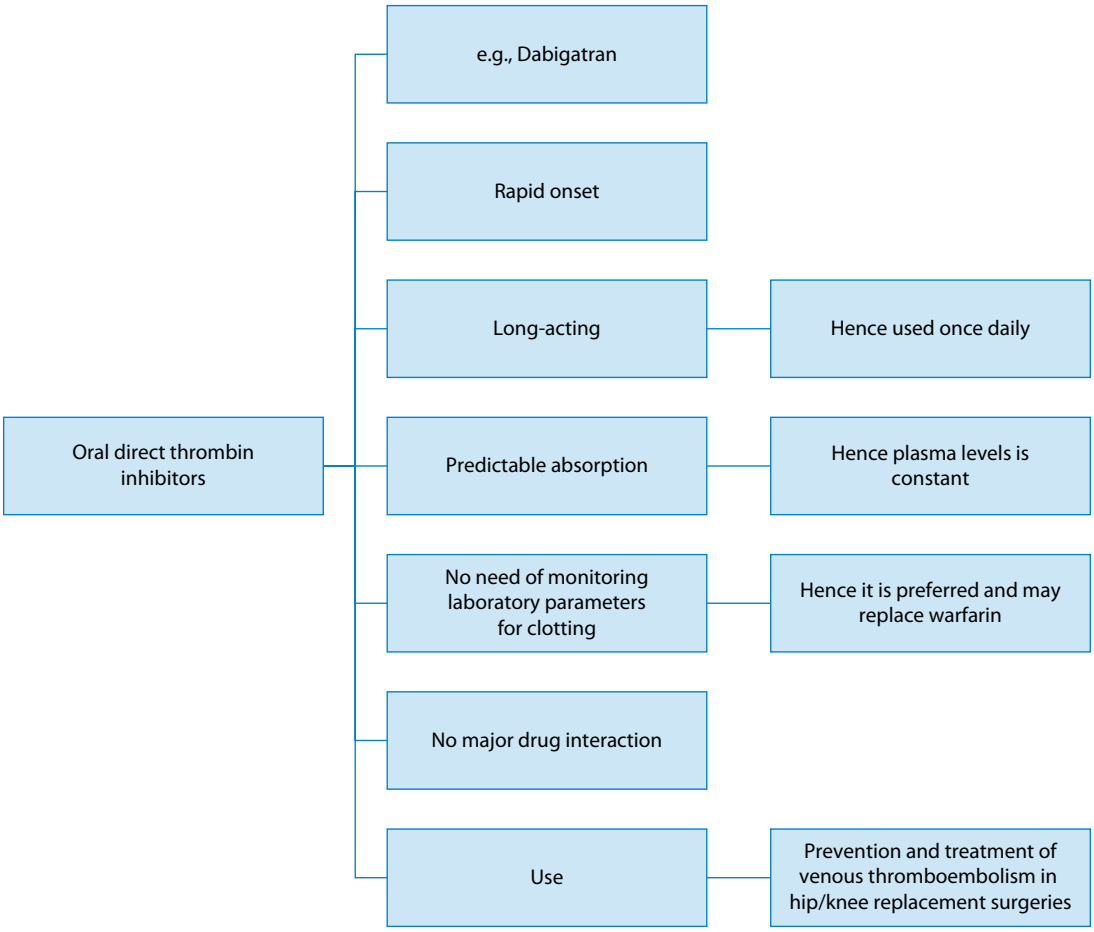
40.7 USES AND ADRs OF WARFARIN



40.8 DRUG INTERACTIONS OF WARFARIN



40.9 ORAL DIRECT THROMBIN INHIBITORS



40.10 DIFFERENCES BETWEEN HEPARIN vs. LMW HEPARIN

	Heparin	**Low-molecular-weight heparin**
1. Mol wt	High	Low
2. Source	Natural	Semi-synthetic
3. Thrombin inhibition	Present	Absent
4. Clotting parameters	Effected	Not effected
5. Laboratory monitoring	Needed	Not needed
6. SC bioavailability	Low	High
7. Duration of action	2–4 h (Short)	18–24 h (long)
8. Dose	4–6/day	Once daily
9. Bleeding complications	High	Minimal
10. Thrombocytopenia	High	Low

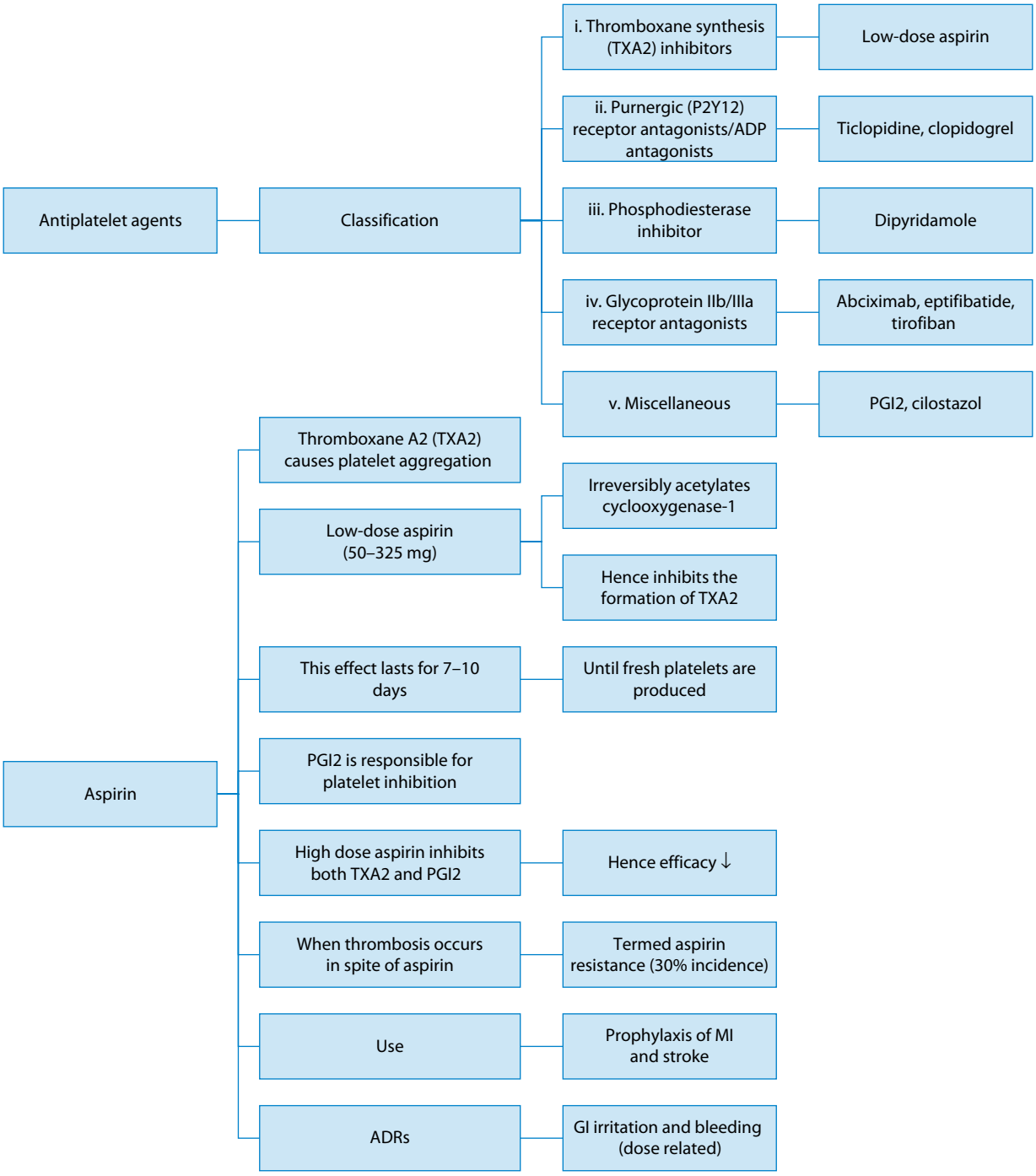
40.11 DIFFERENCES BETWEEN HEPARIN AND DICUMAROL/WARFARIN

	Heparin	**Dicaumarol/warfarin**
1. Source	Natural	Synthetic
2. Chemistry	Mucopolysaccharide	Coumarin
3. Action	In vitro and in vivo	Only in vivo
4. Administration	Parenteral (IV/SC)	Oral
5. Onset	Rapid (3–6 h)	Slow (1–3 days)
6. Duration	Short (2–4 h)	Long (4–7 days)
7. Mechanism	Stimulates antithrombin III	Inhibits clotting factors
8. Antidote	Protamine sulfate	Vitamin K1 oxide
9. Usage	For initiation	For maintenance
10. Usage in pregnancy	Used	Not used as it is teratogenic
11. Cost	Expensive	Economical
12. Monitoring	Measuring aPTT/clotting time	Synthetic

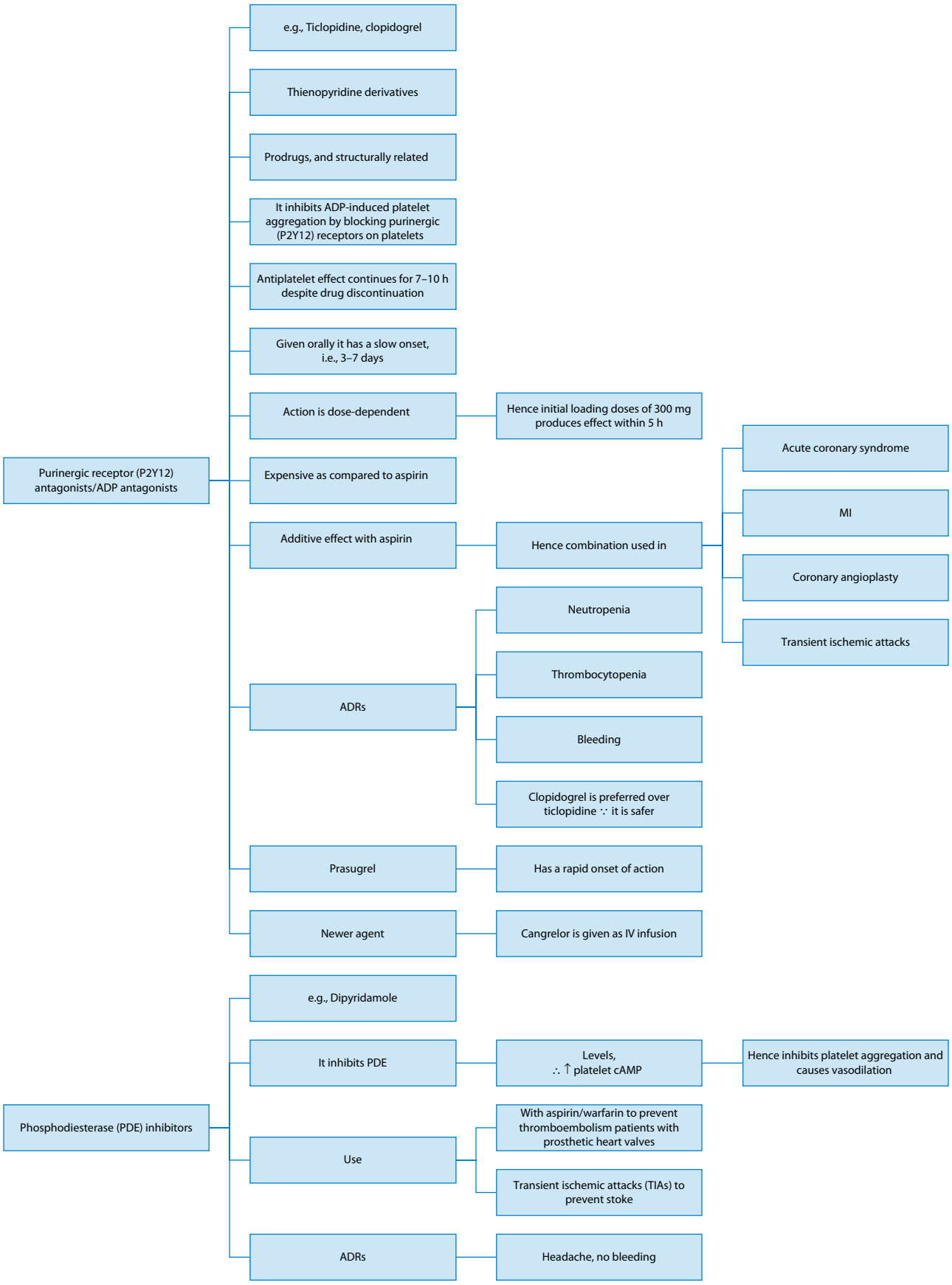
41

Antiplatelet agents

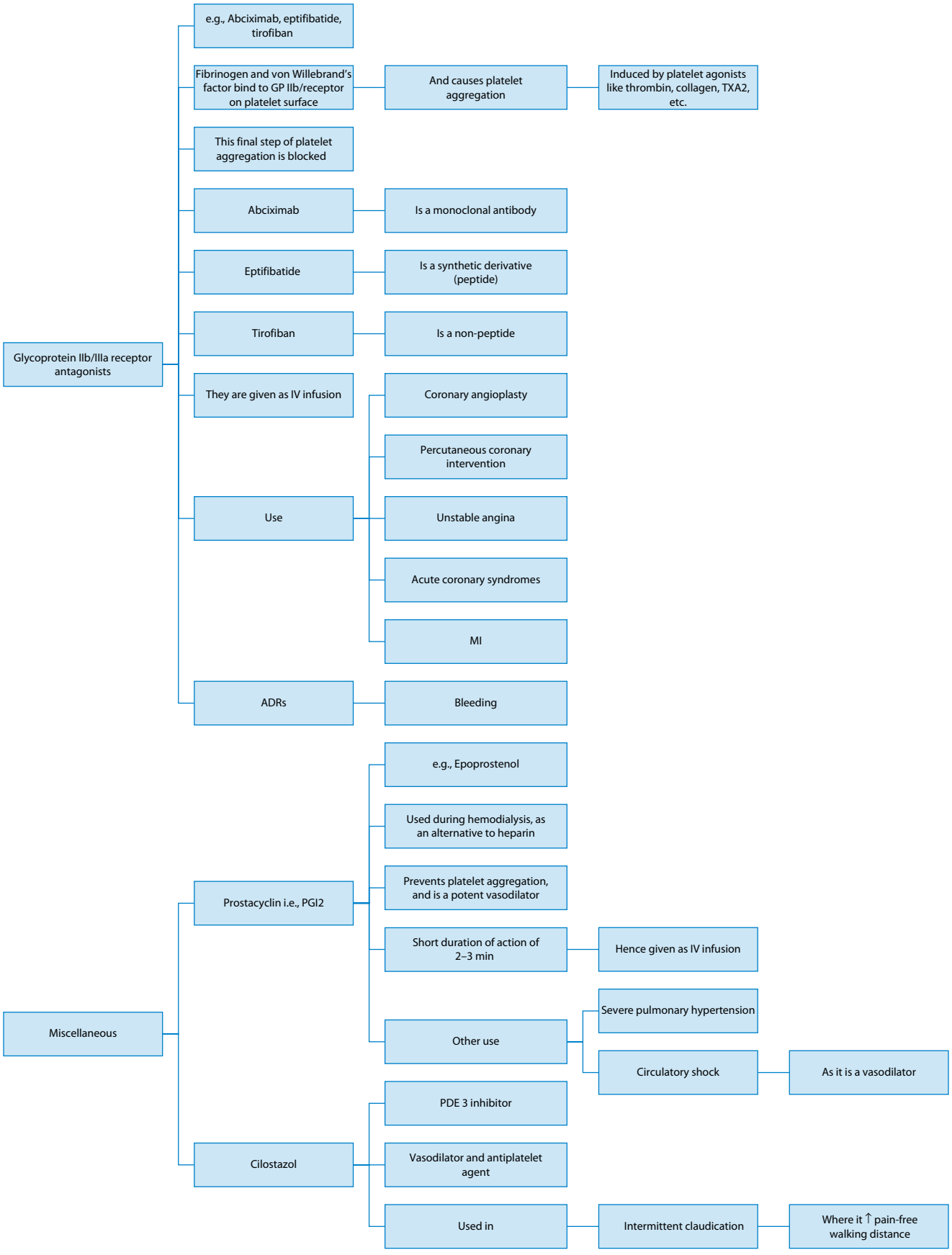
41.1 CLASSIFICATION AND ASPIRIN



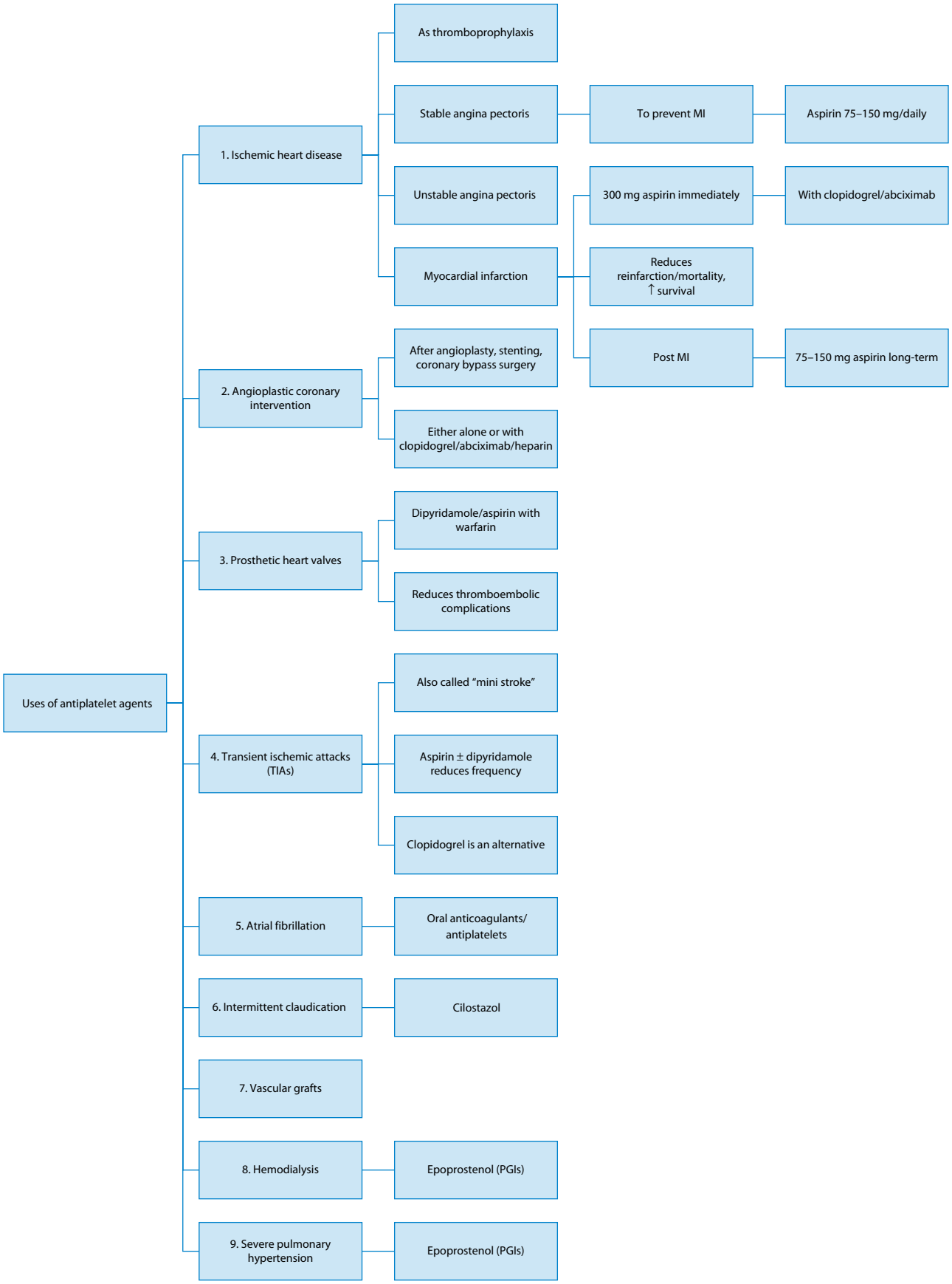
41.2 PURINERGIC RECEPTOR (P2Y12) ANTAGONISTS/ADP ANTIGONISTS AND PHOSPHODIESTERASE (PDE) INHIBITORS



41.3 GLYCOPROTEIN IIB/IIIA RECEPTOR ANTAGONISTS AND MISCELLANEOUS



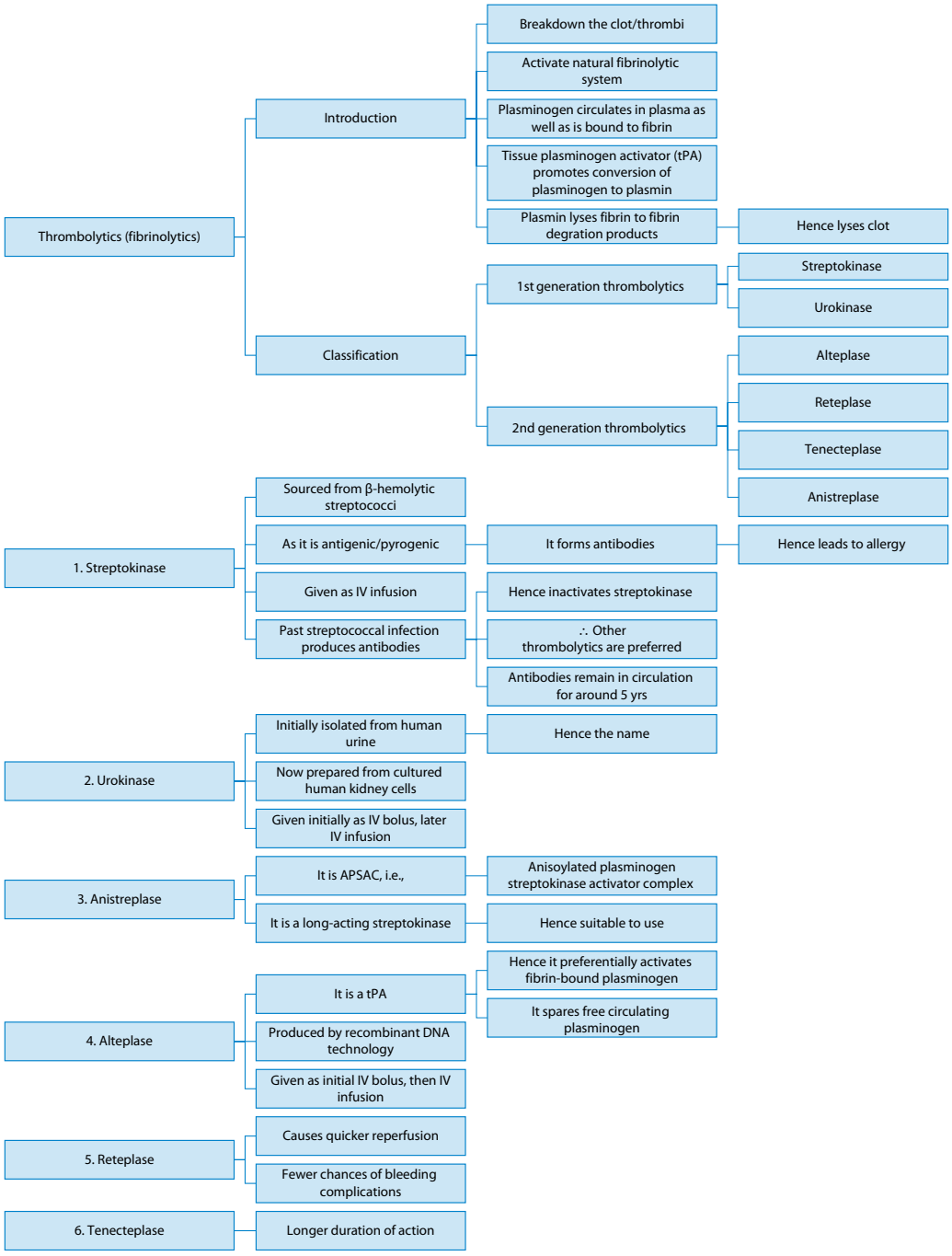
41.4 USES OF ANTIPLATELET AGENTS



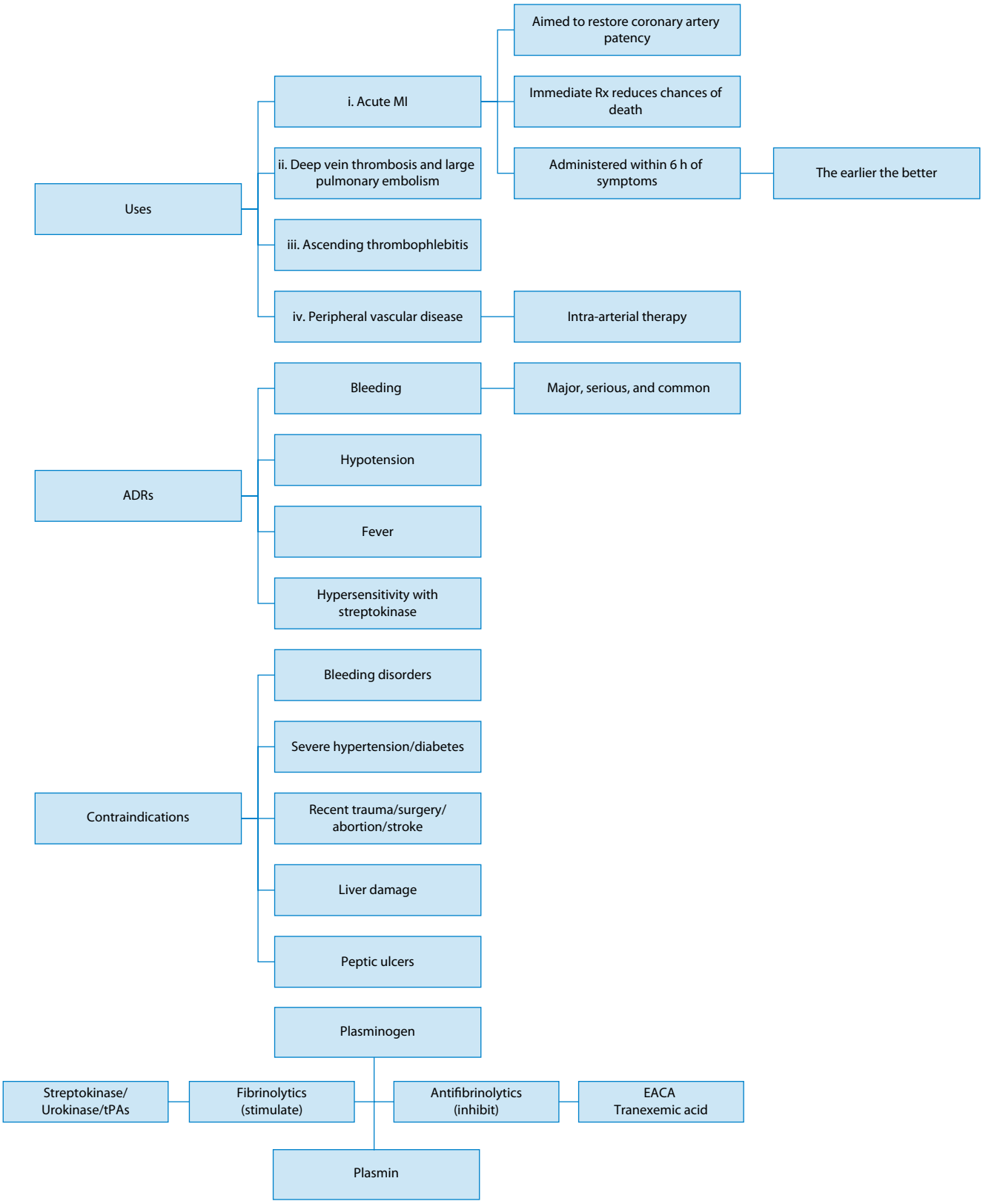
42

Thrombolytics (fibrinolytics) and antifibrinolytics

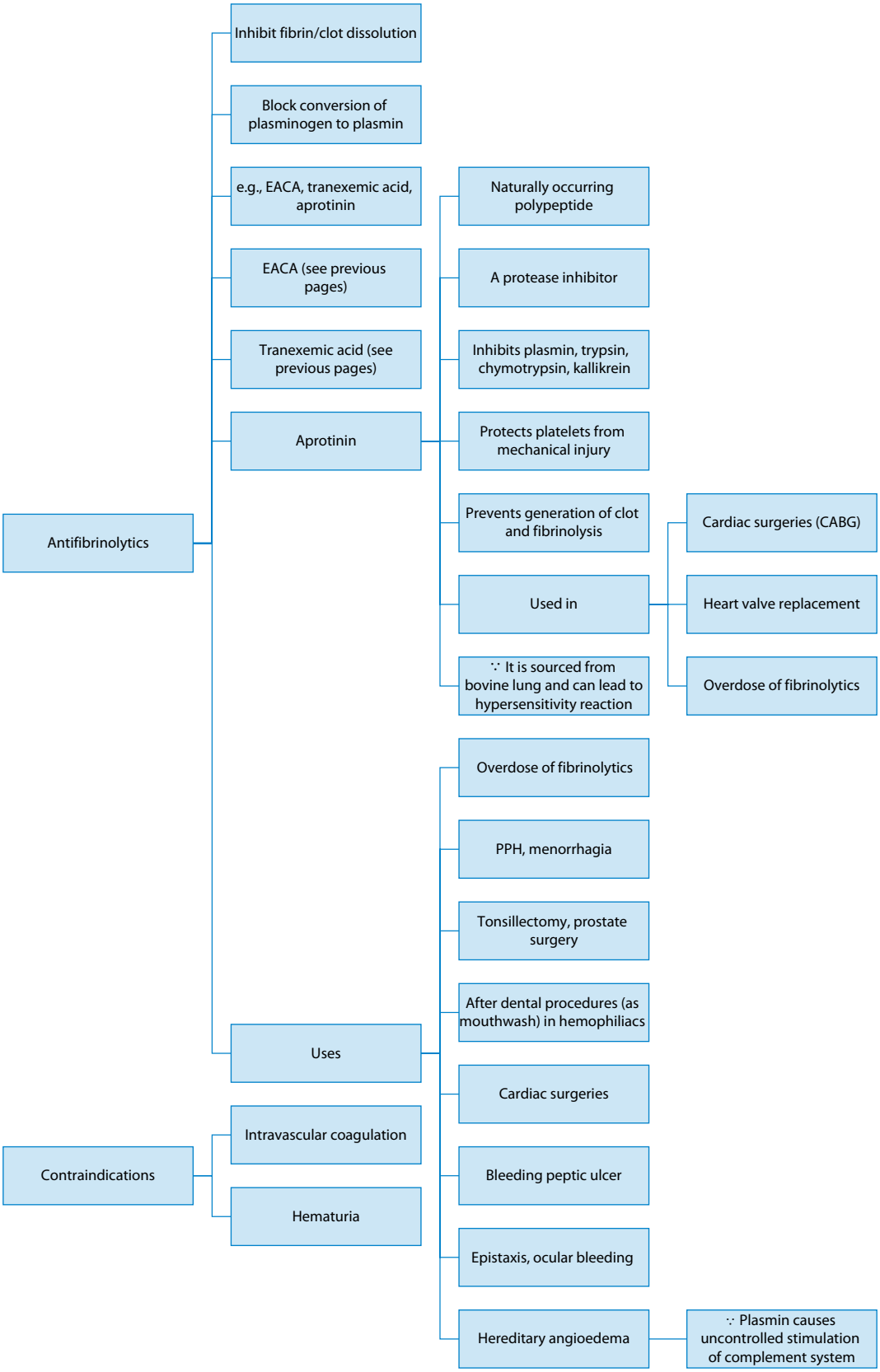
42.1 THROMBOLYTICS (FIBRINOLYTICS) – INTRODUCTION, CLASSIFICATION, AND INDIVIDUAL AGENTS



42.2 USES, ADRs, AND CONTRAINDICATIONS



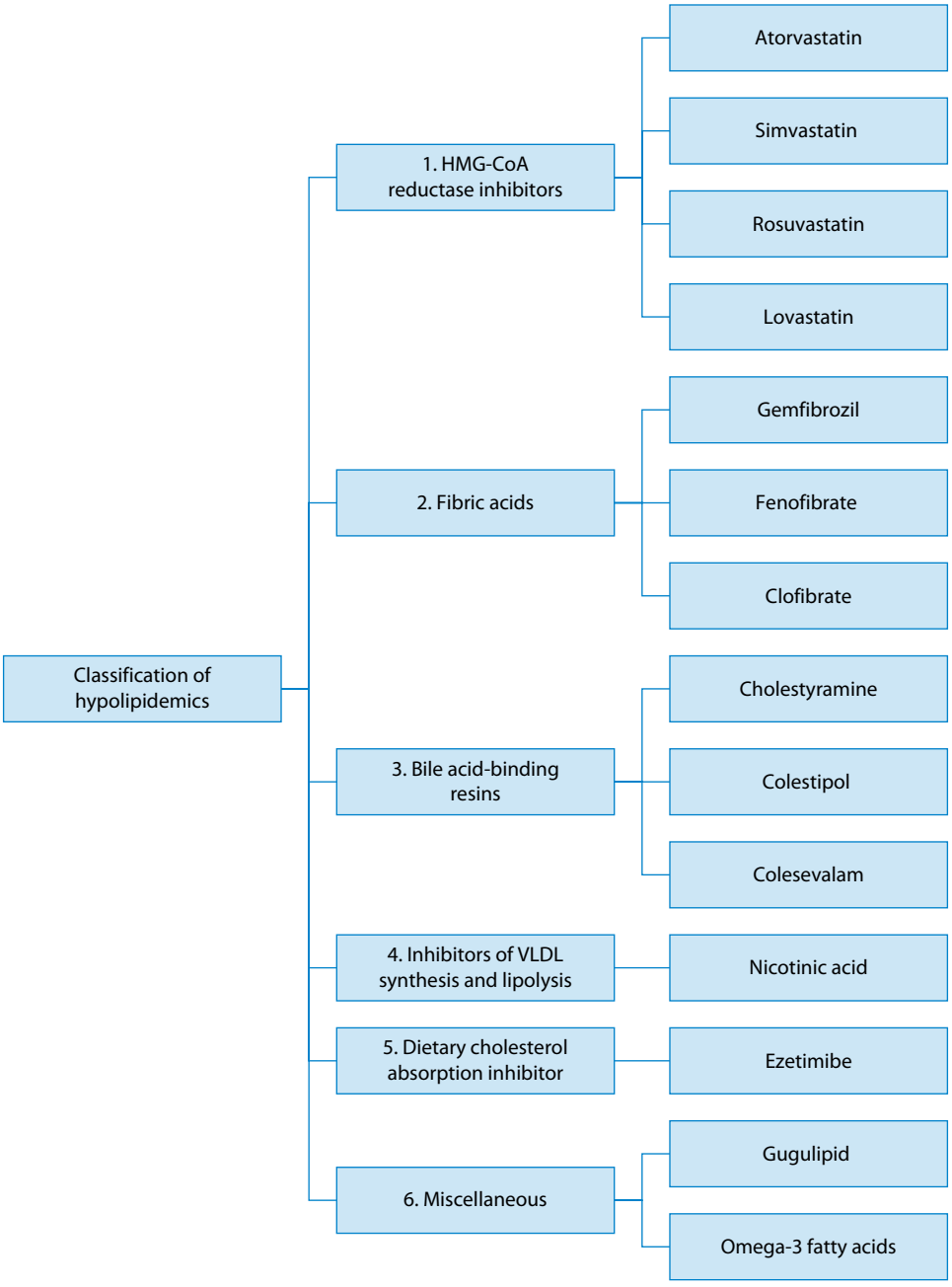
42.3 ANTIFIBRINOLYTICS – USES AND CONTRAINDICATIONS



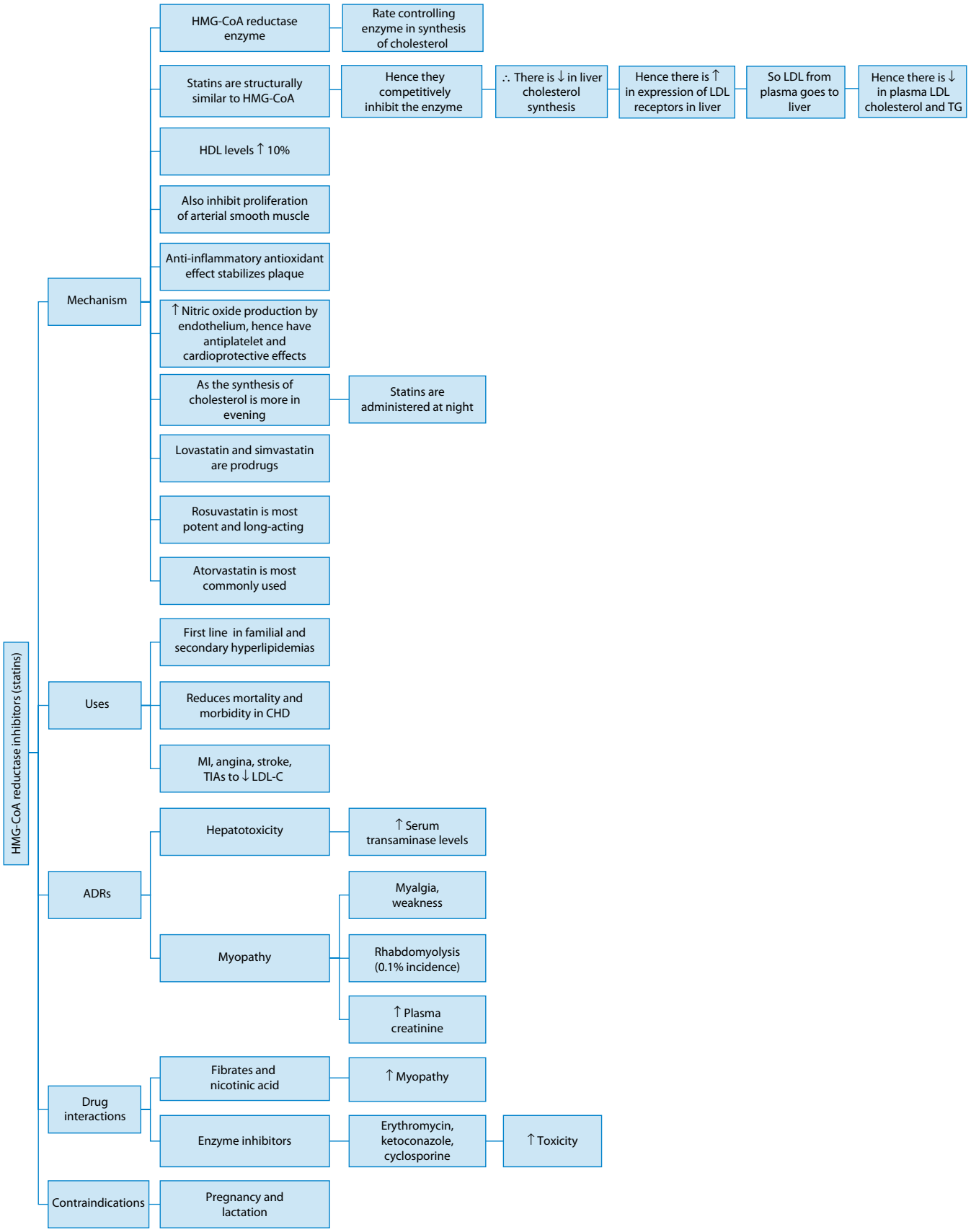
43

Hypolipidemic drugs

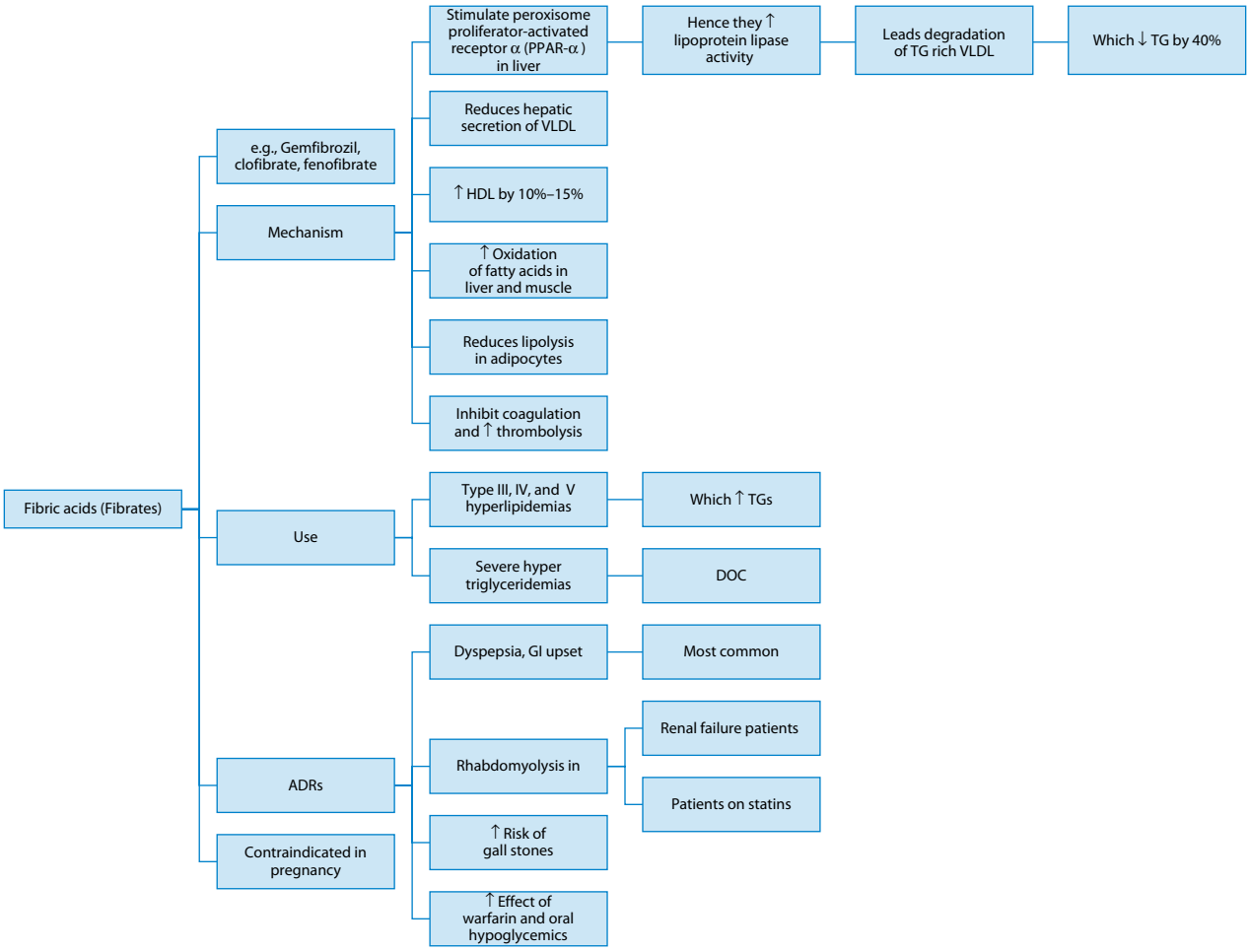
43.1 CLASSIFICATION OF HYPOLIPIDEMICS



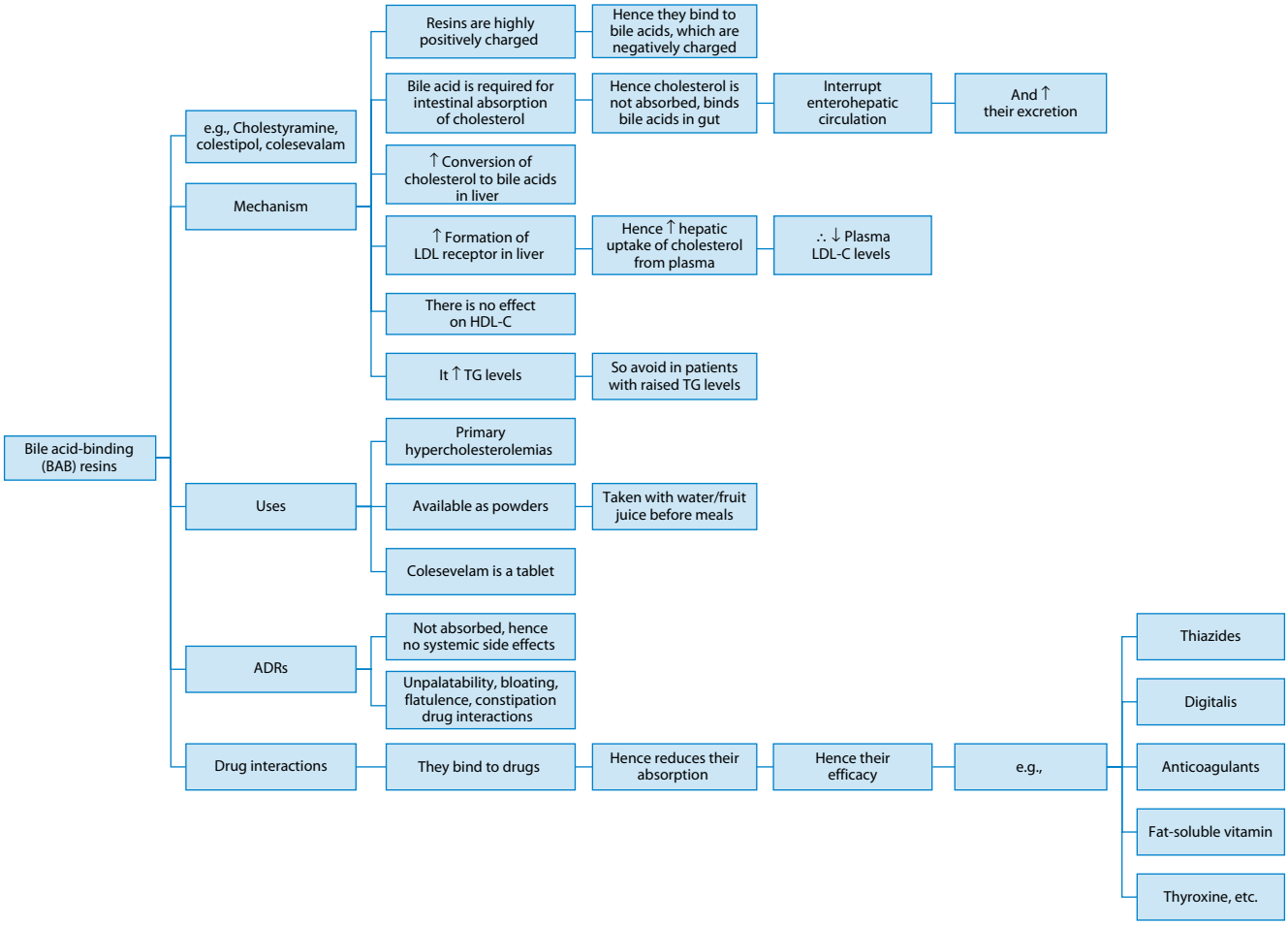
43.2 HMG-CoA REDUCTASE INHIBITORS (STATINS)



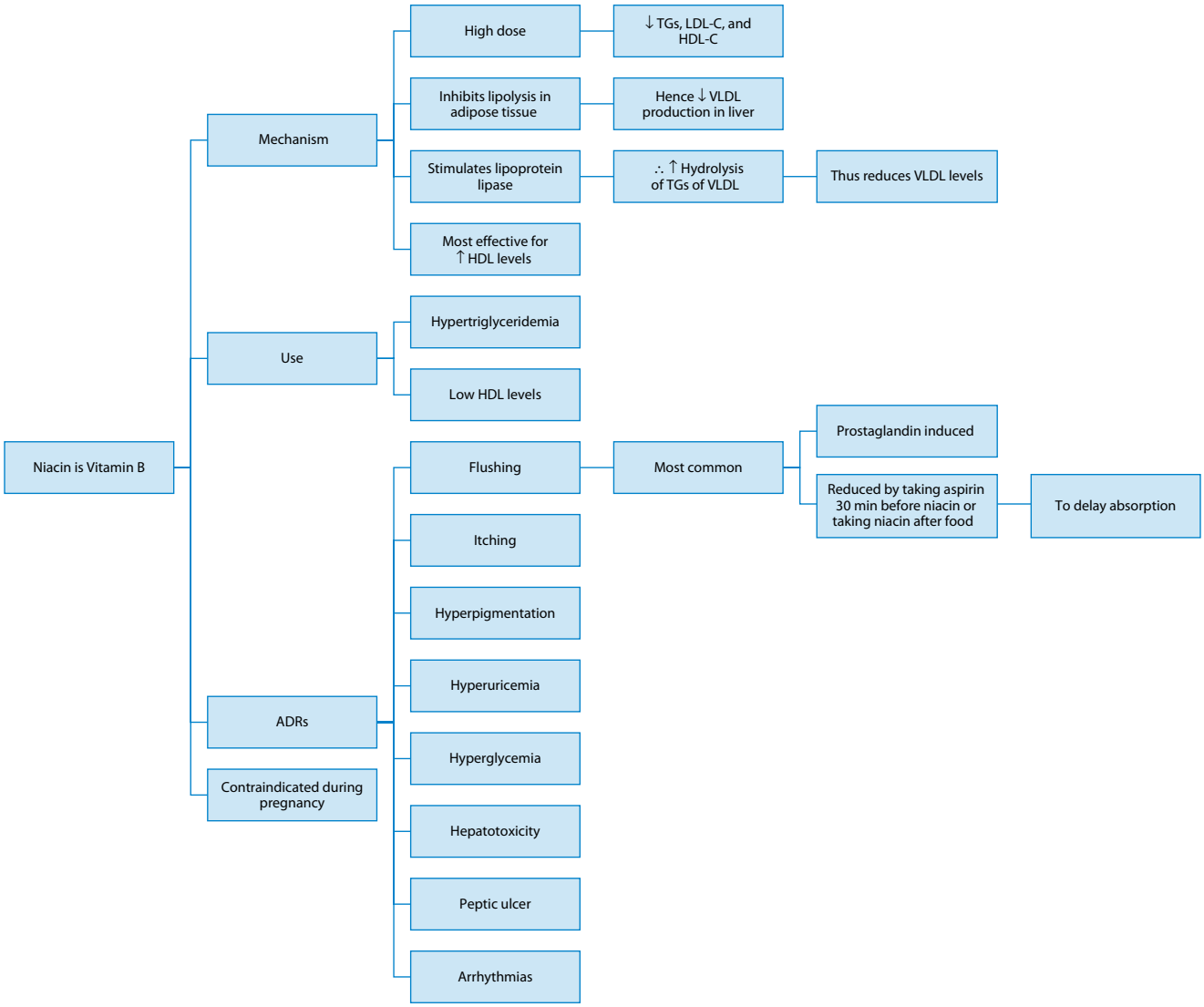
43.3 FIBRIC ACIDS (FIBRATES)



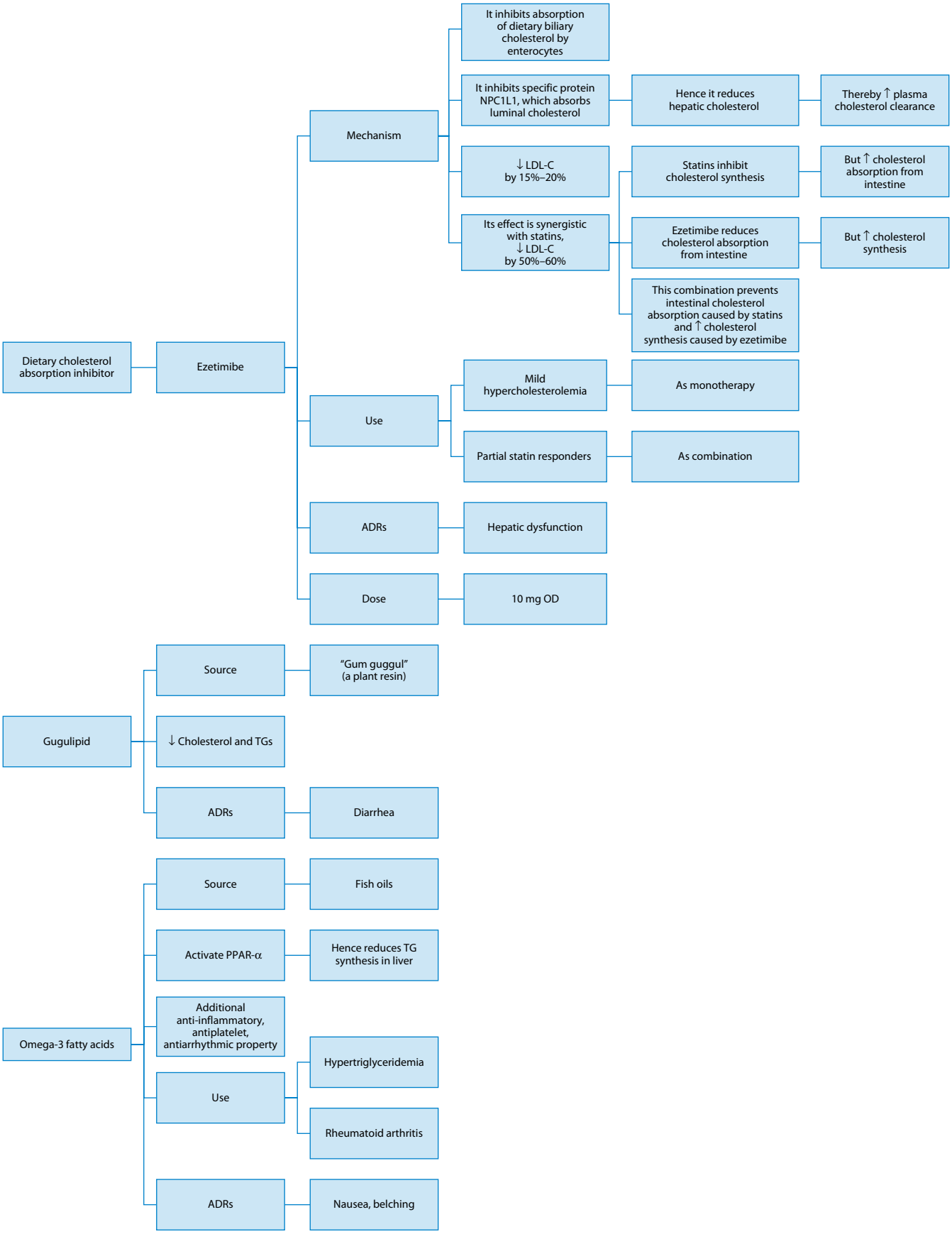
43.4 BILE ACID-BINDING RESINS (BAB – RESINS)



43.5 NICOTINIC ACID OR NIACIN



43.6 DIETARY CHOLESTEROL ABSORPTION INHIBITOR, GUGULIPID, AND OMEGA-3 FATTY ACIDS





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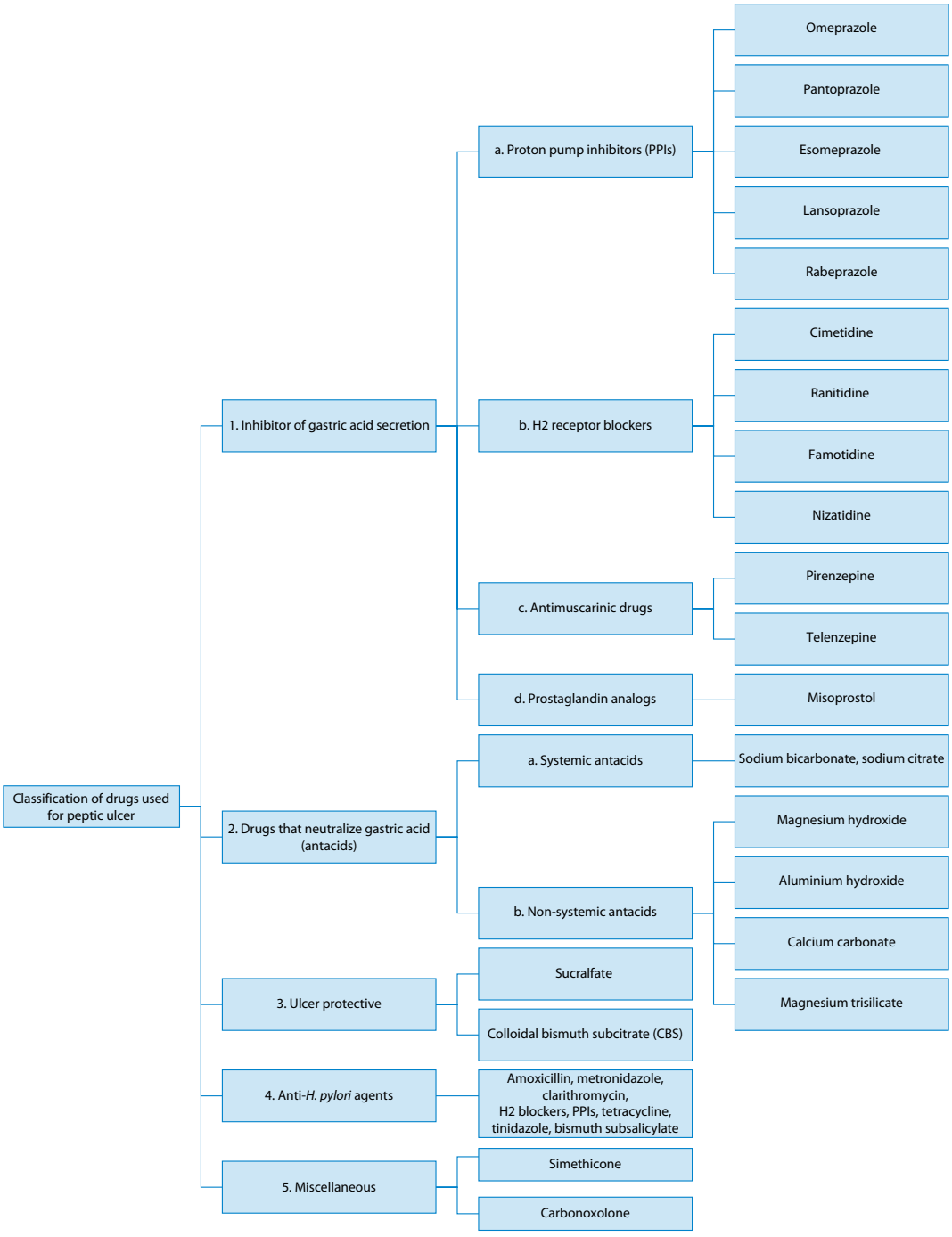
PART VIII

Gastrointestinal pharmacology

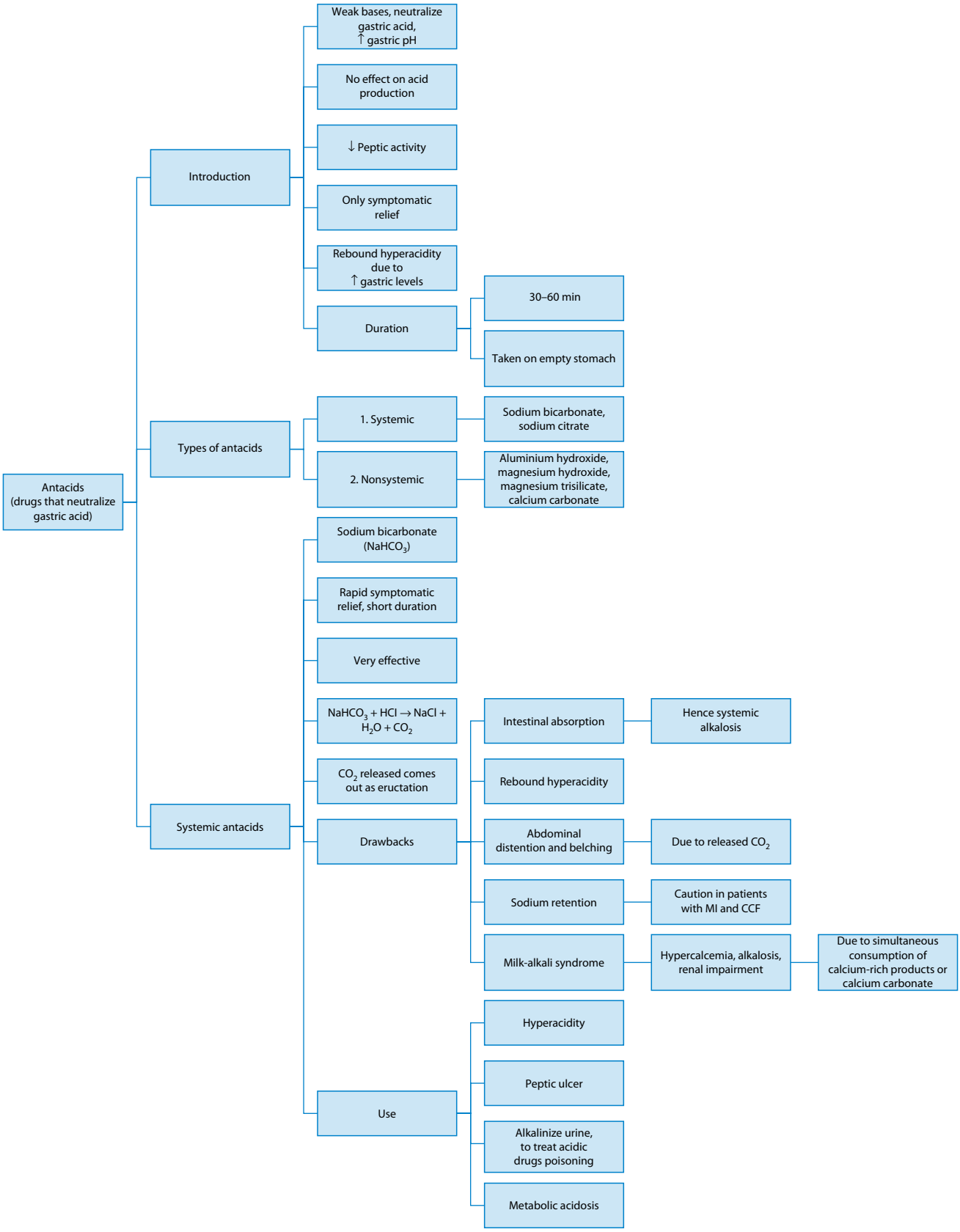
44

Drug therapy of peptic ulcer and GERD

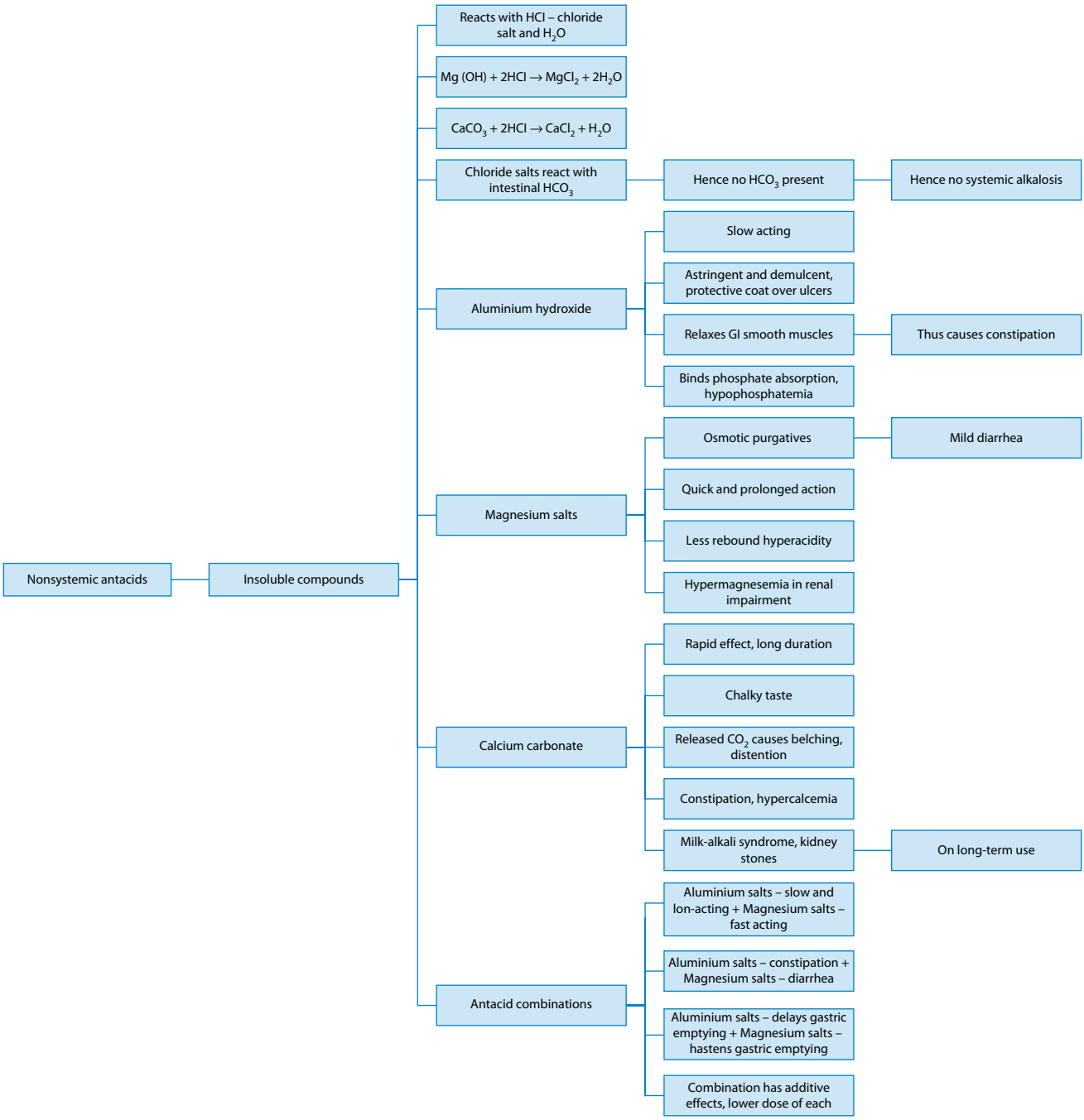
44.1 CLASSIFICATION OF DRUGS USED FOR PEPTIC ULCER



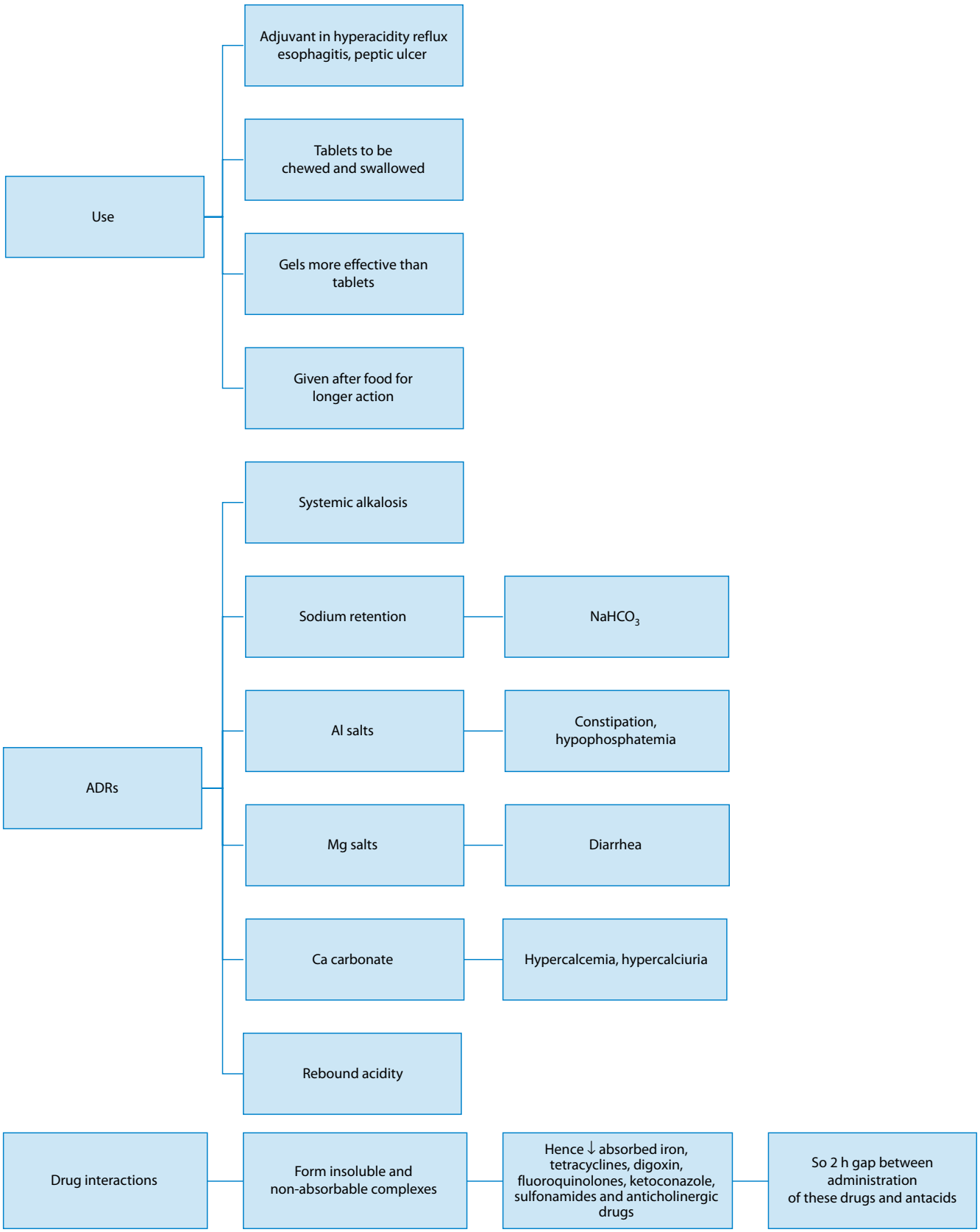
44.2 ANTACIDS – INTRODUCTION, TYPES, AND SYSTEMIC ANTACIDS



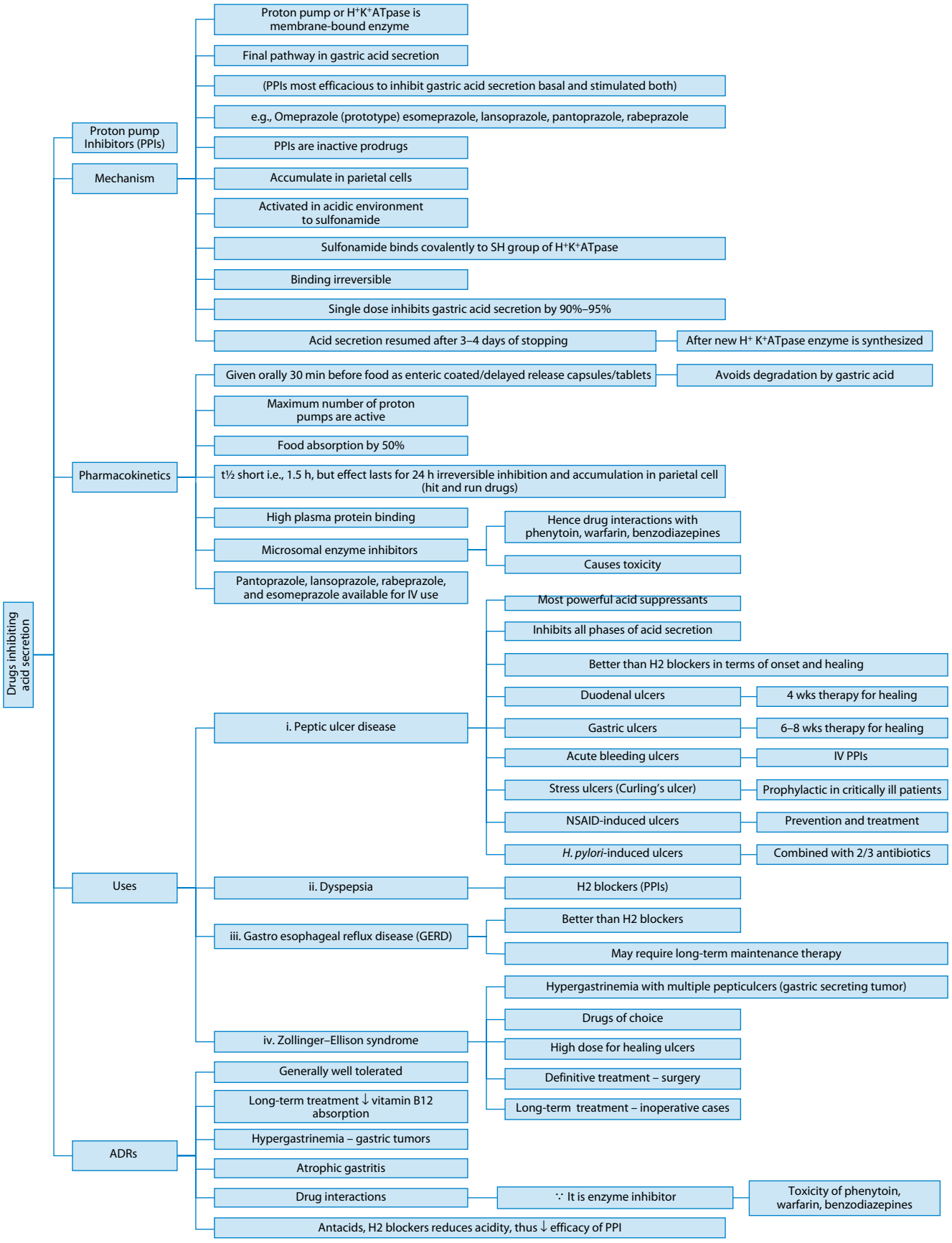
44.3 NONSYSTEMIC ANTACIDS



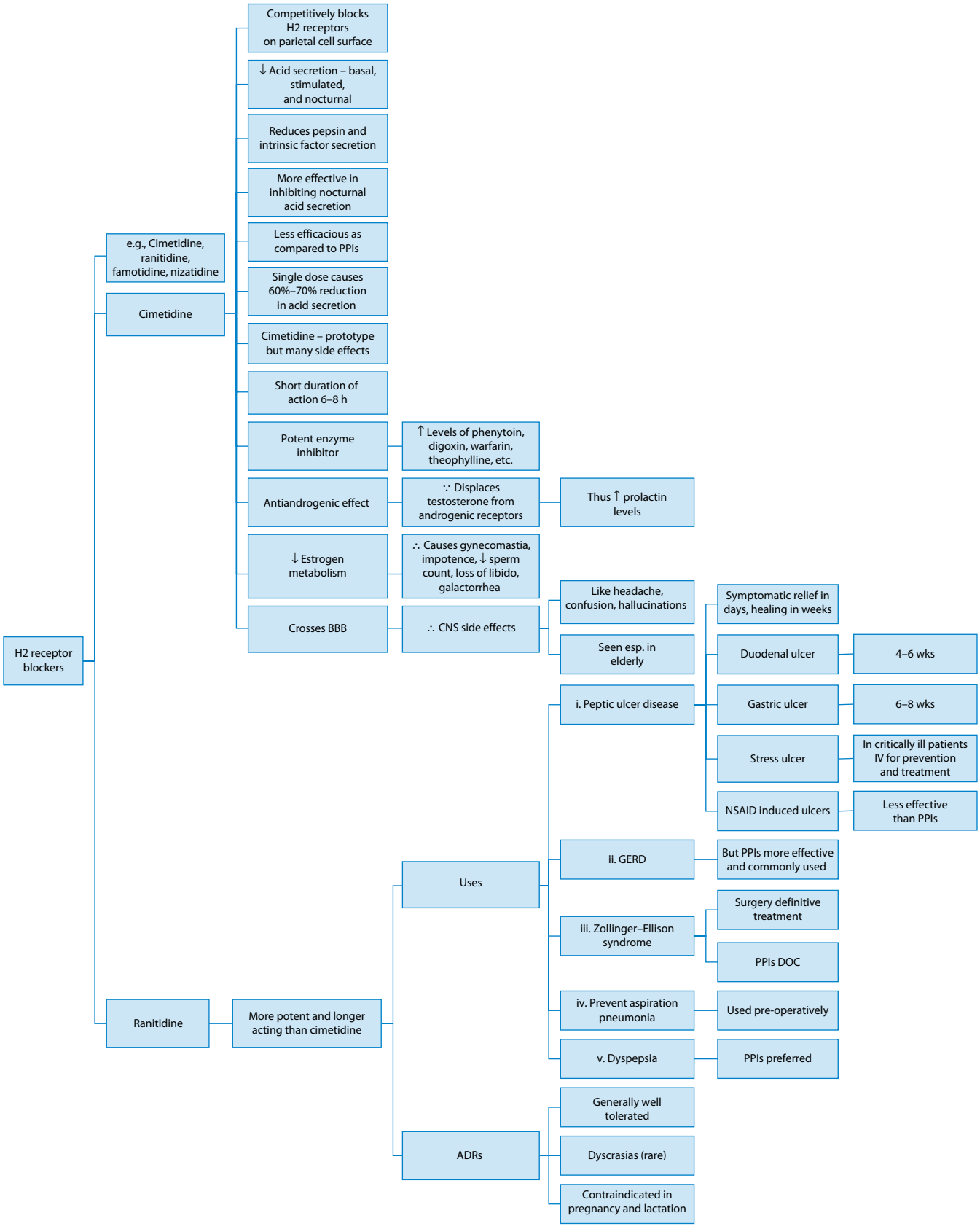
44.4 USE, ADRs, AND DRUG INTERACTIONS



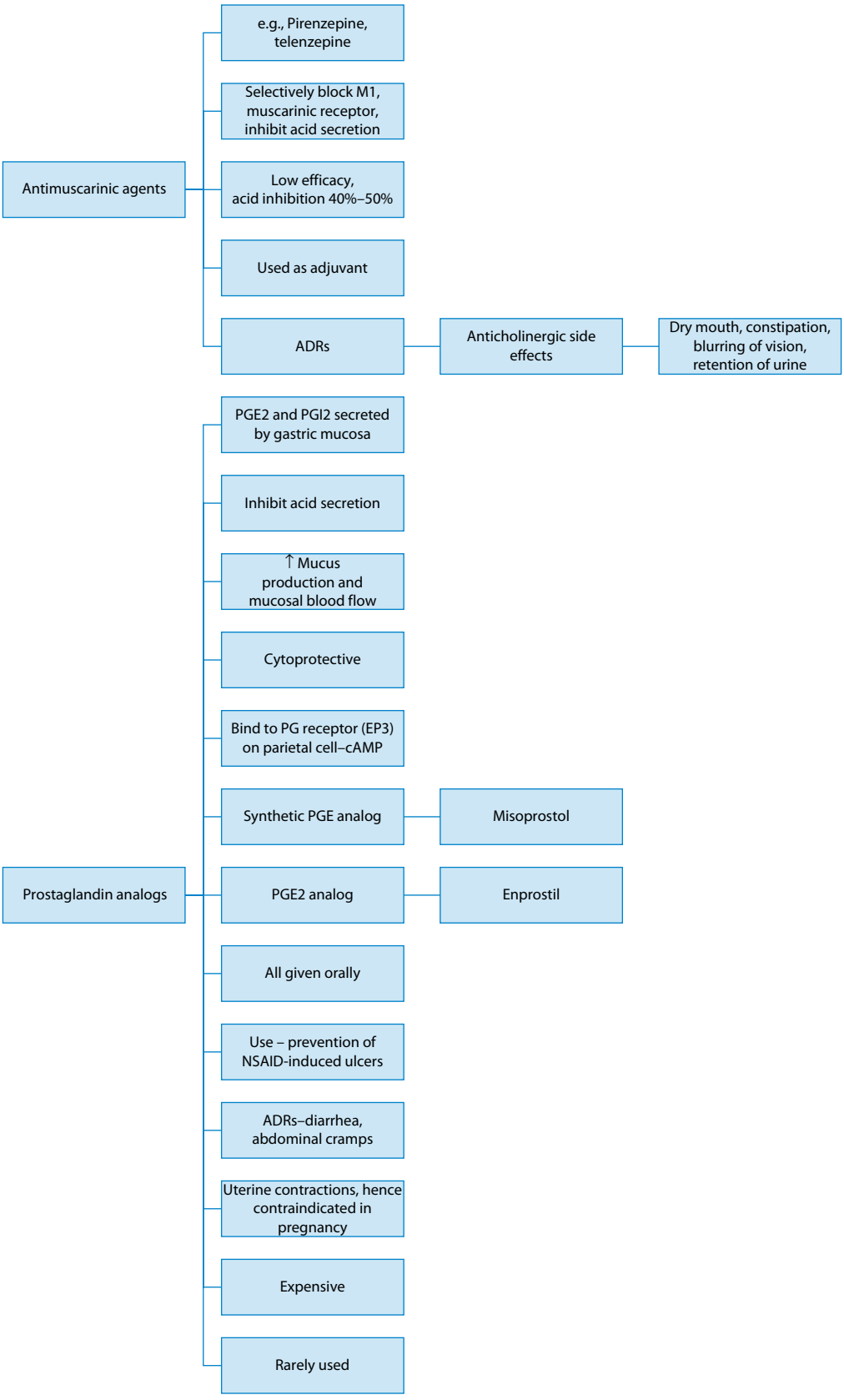
44.5 PROTON PUMP INHIBITORS (PPIs)



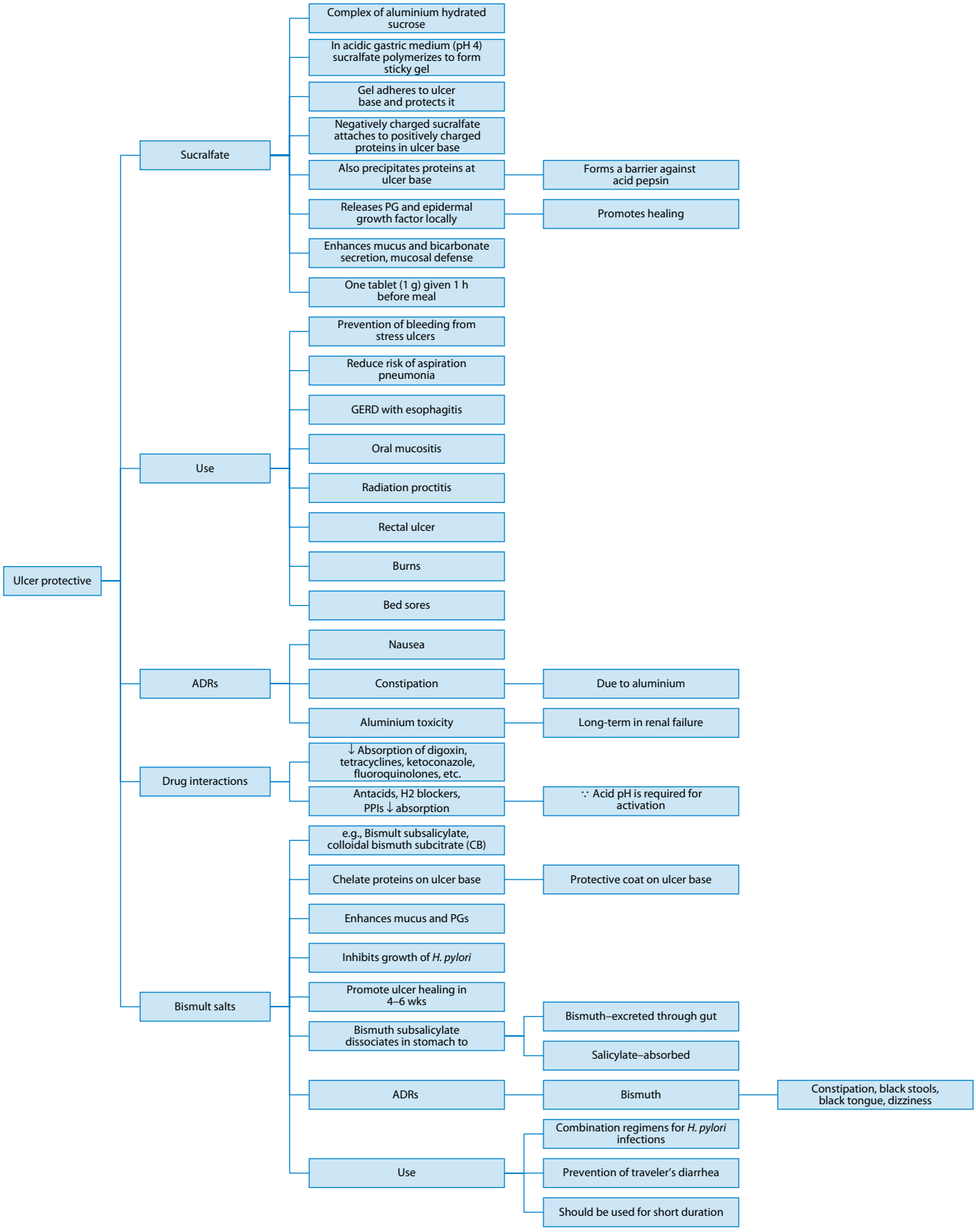
44.6 H2-RECEPTOR BLOCKERS



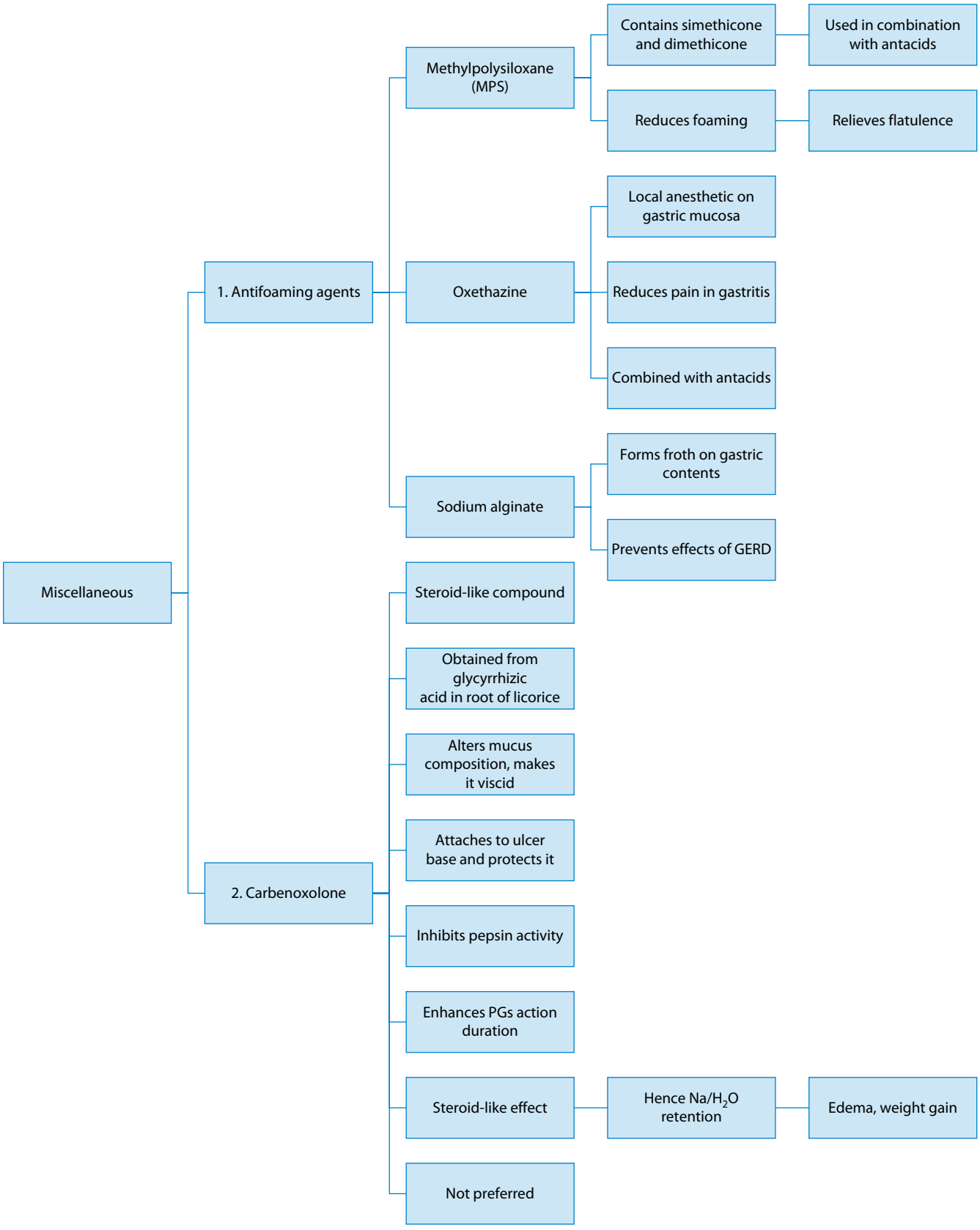
44.7 ANTIMUSCARINIC AGENTS AND PROSTAGLANDIN ANALOGS



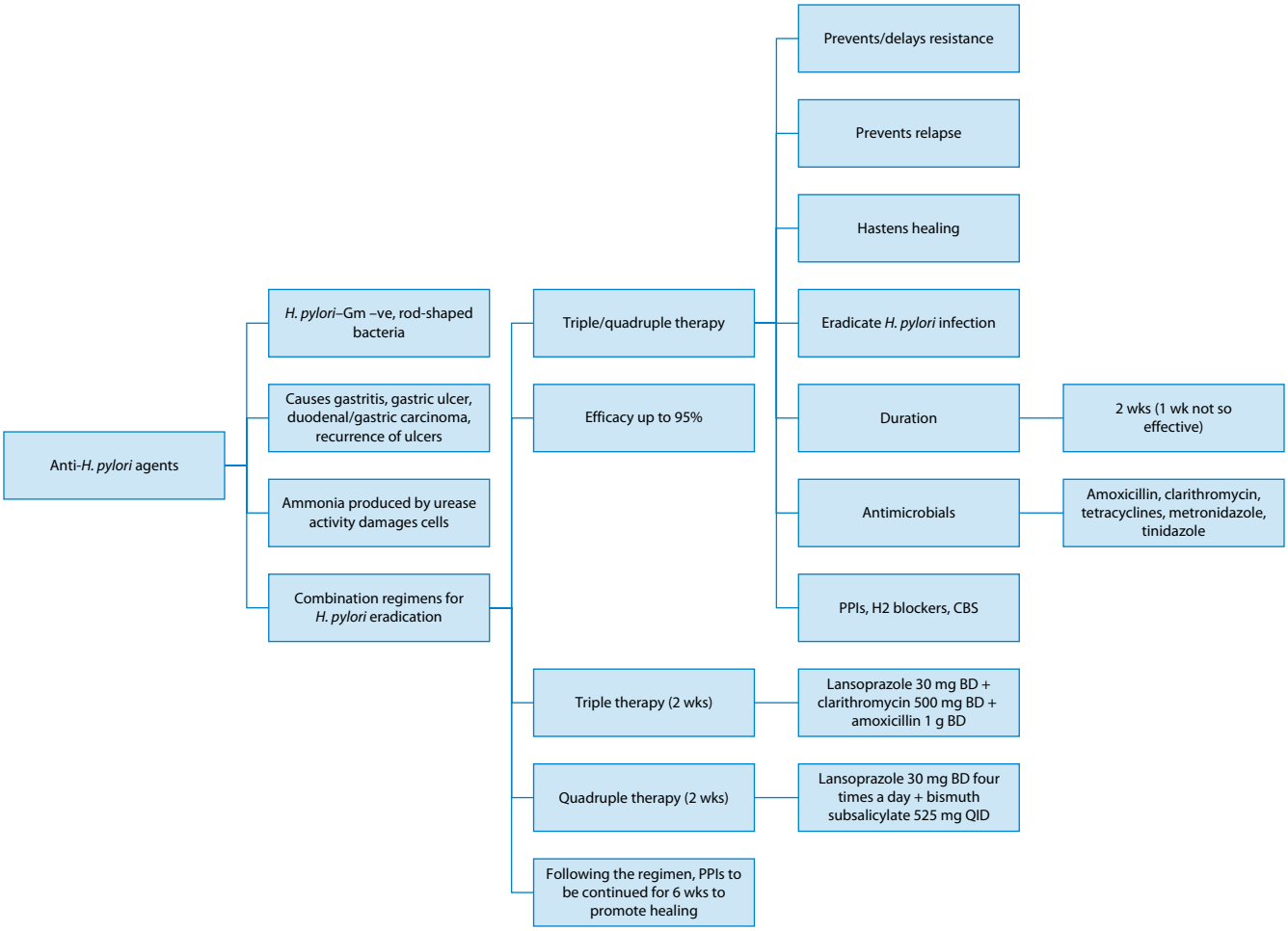
44.8 ULCER PROTECTIVES



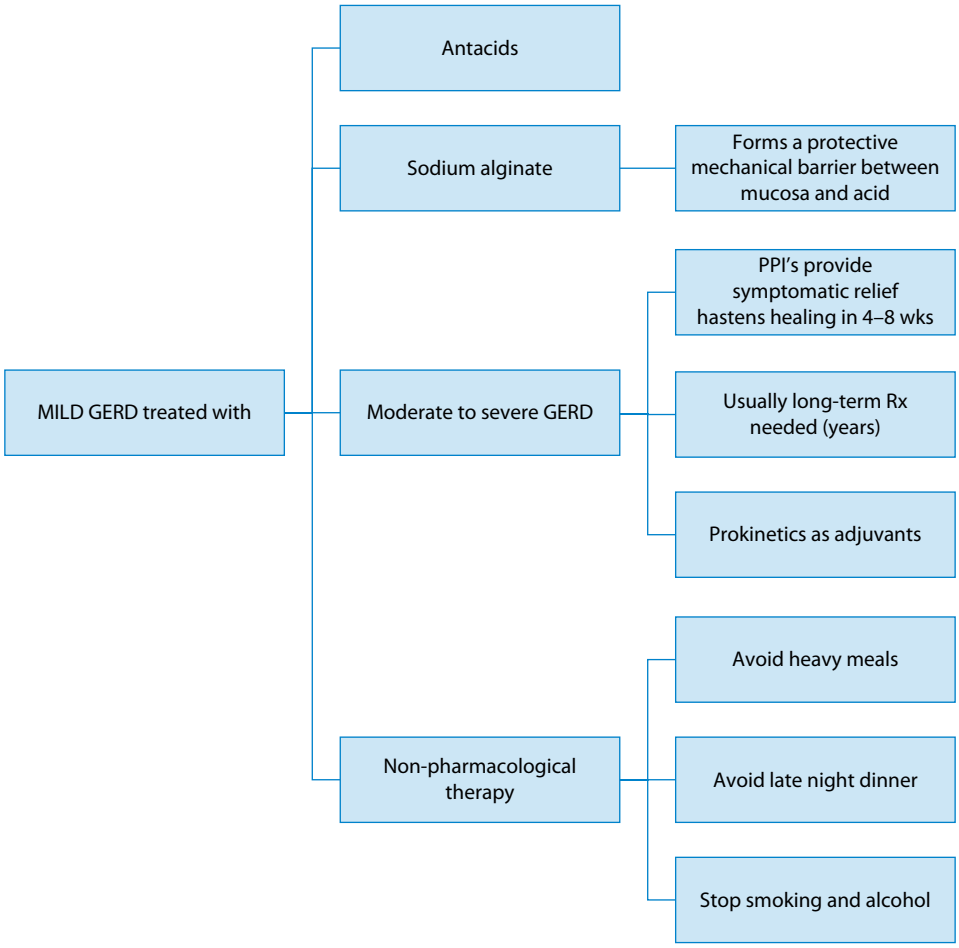
44.9 MISCELLANEOUS AGENTS



44.10 ANTI-*H. PYLORI* AGENTS



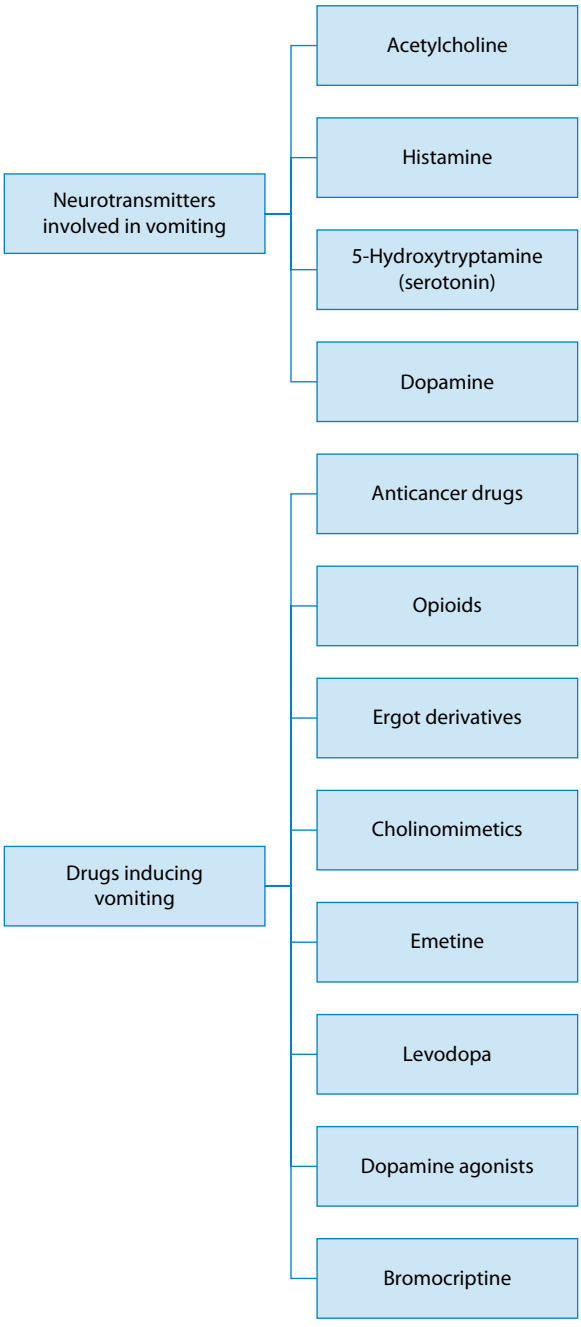
44.11 GASTROESOPHAGEAL REFLUX DISEASE (GERD) – MANAGEMENT



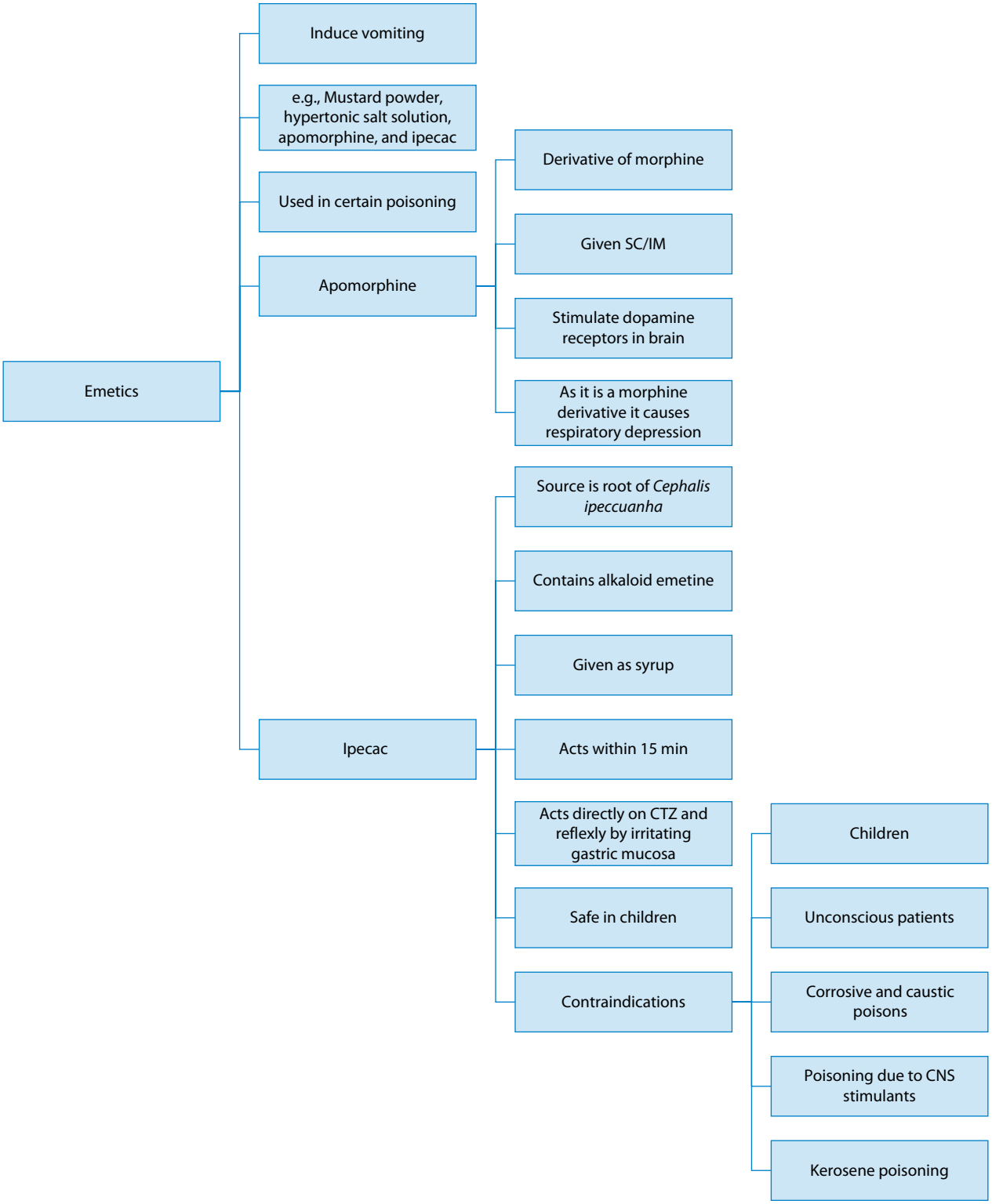
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Emetics and antiemetics

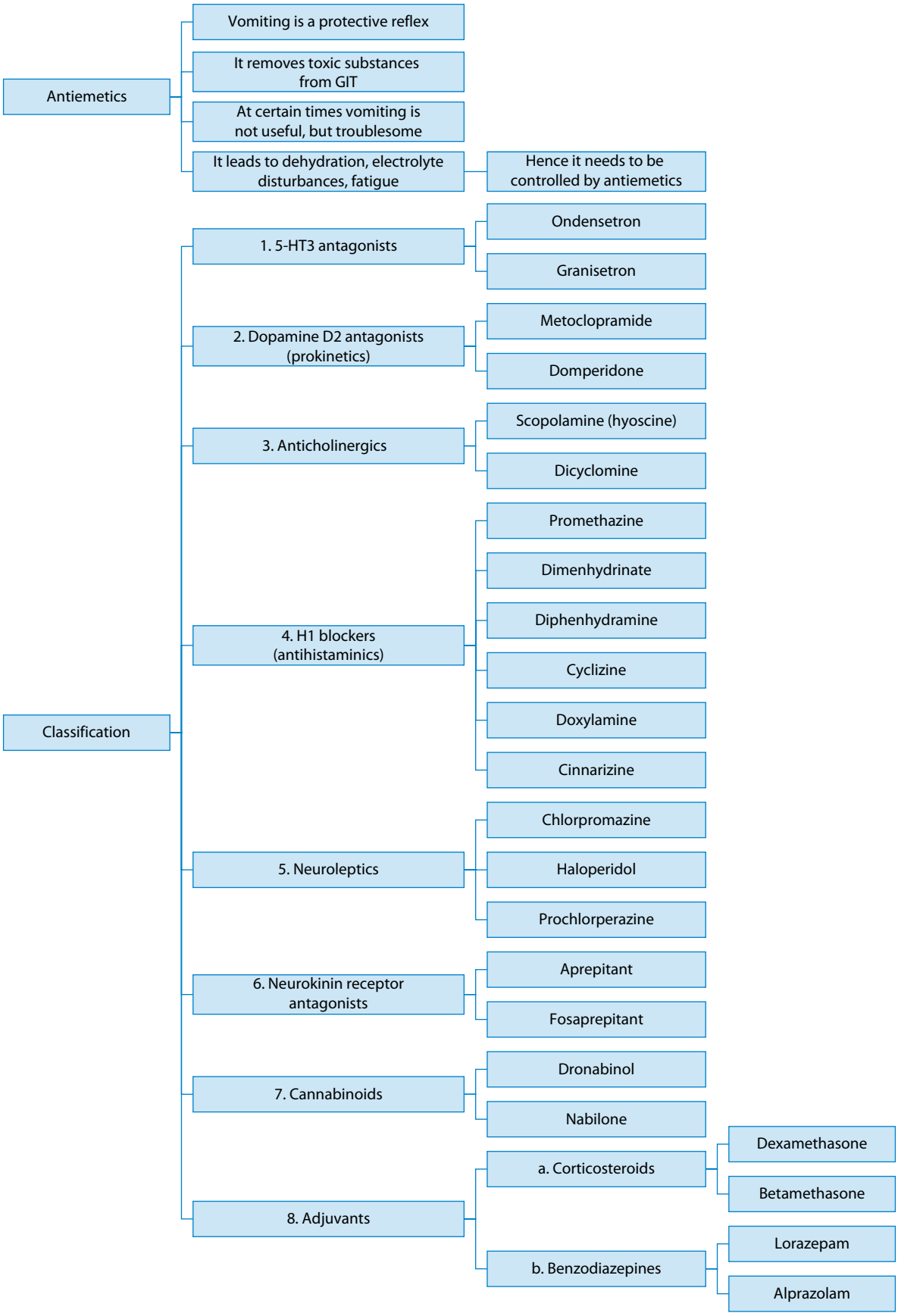
45.1 NEUROTRANSMITTERS AND DRUGS INVOLVED IN VOMITING



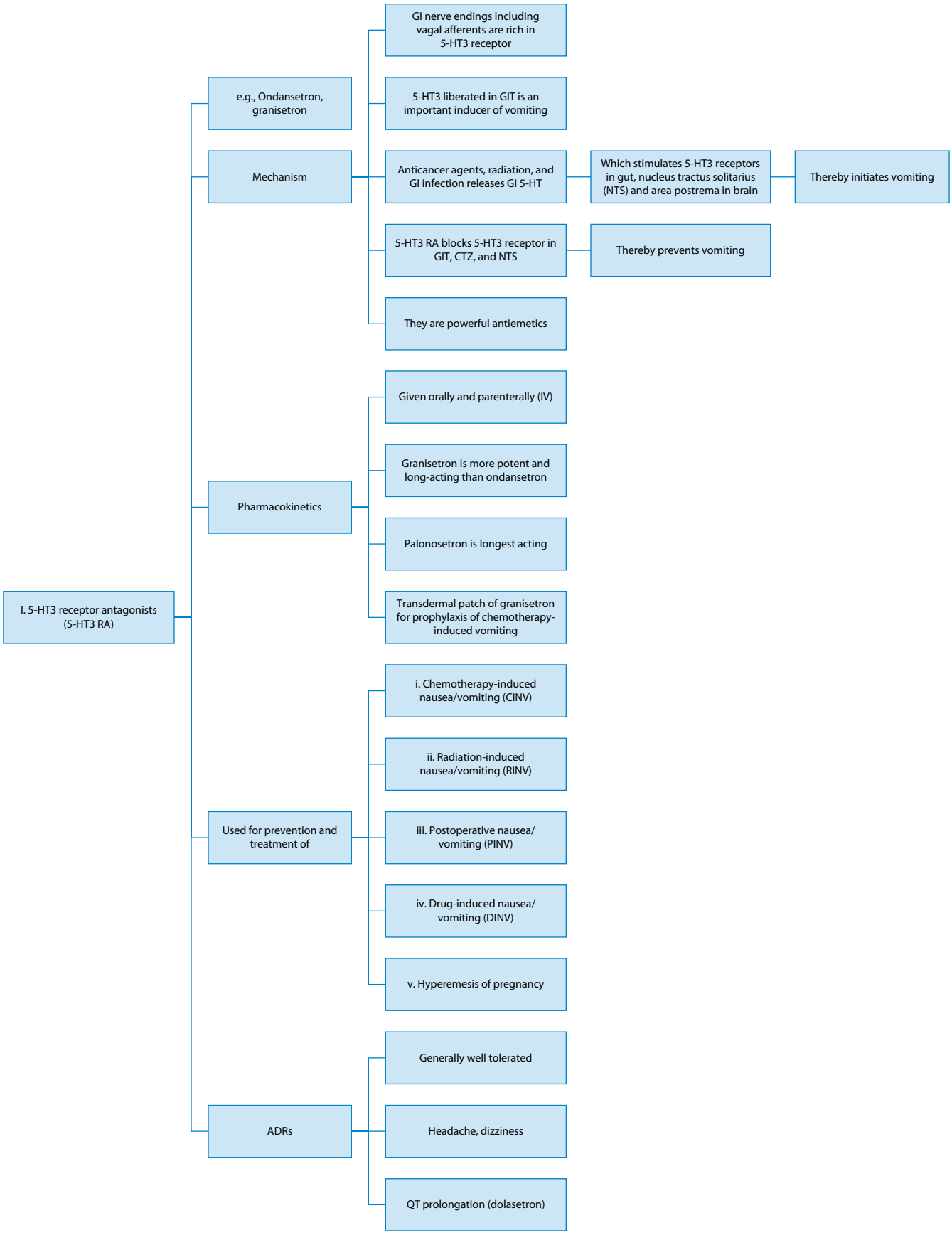
45.2 EMETICS


Emetics
Induce vomiting
e.g., Mustard powder, hypertonic salt solution, apomorphine, and ipecac
Used in certain poisoning
Apomorphine
Derivative of morphine
Given SC/IM
Stimulate dopamine receptors in brain
As it is a morphine derivative it causes respiratory depression
Ipecac
Source is root of Cephalis ipeccuanha
Contains alkaloid emetine
Given as syrup
Acts within 15 min
Acts directly on CTZ and reflexly by irritating gastric mucosa
Safe in children
Contraindications
Children
Unconscious patients
Corrosive and caustic poisons
Poisoning due to CNS stimulants
Kerosene poisoning

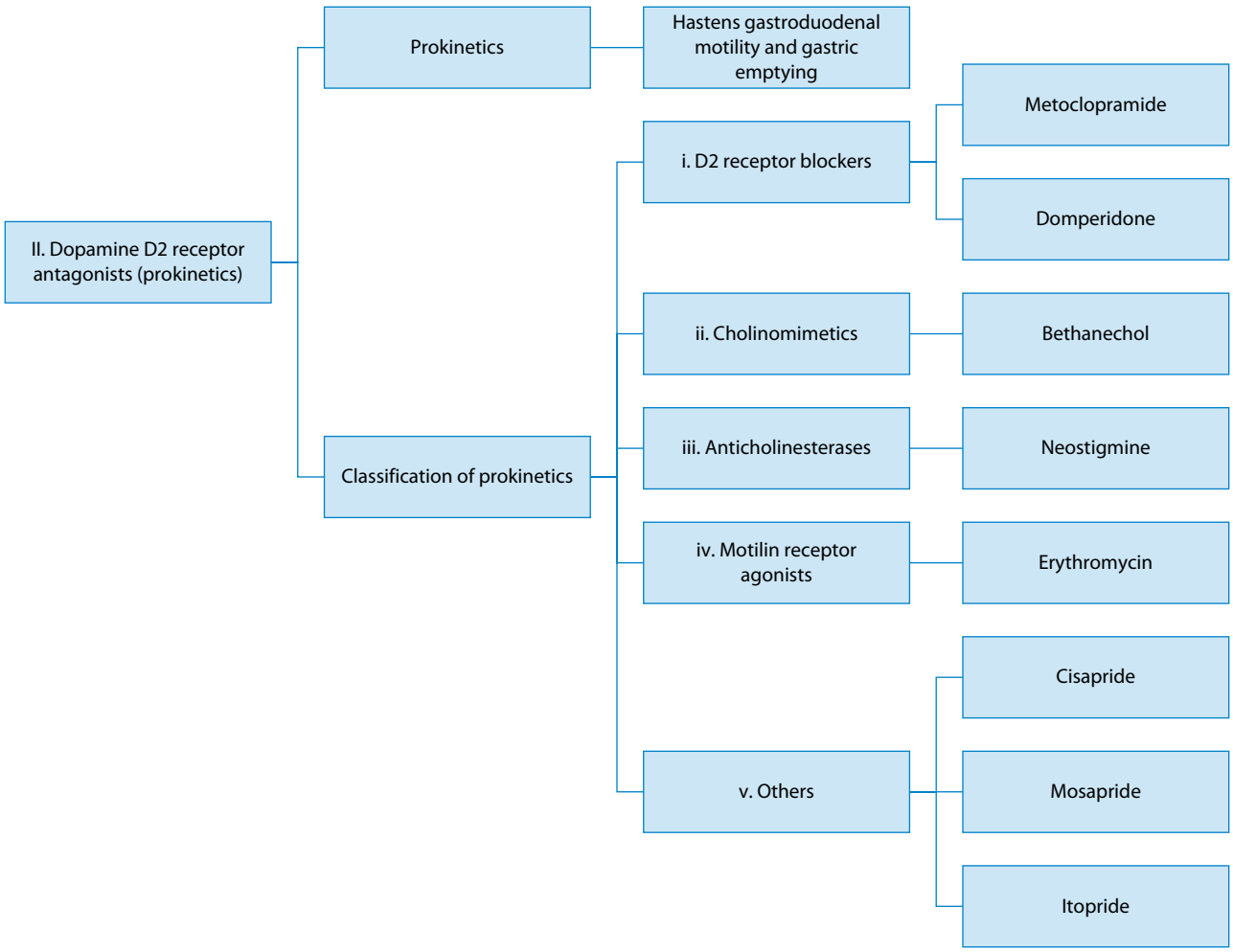
45.3 CLASSIFICATIONS OF ANTIEMETICS



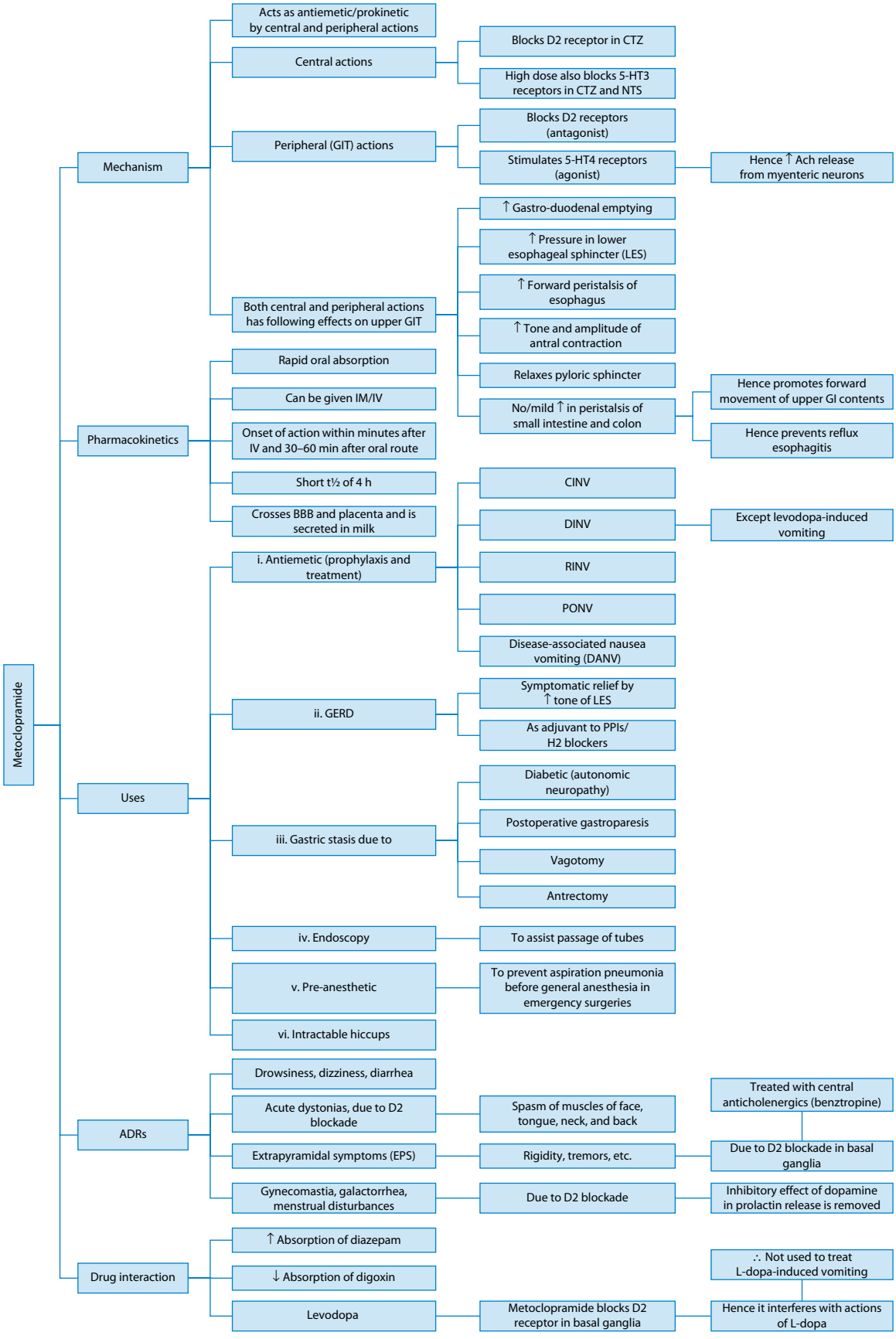
45.4 5-HT3 RECEPTOR ANTAGONISTS (5-HT3RA)



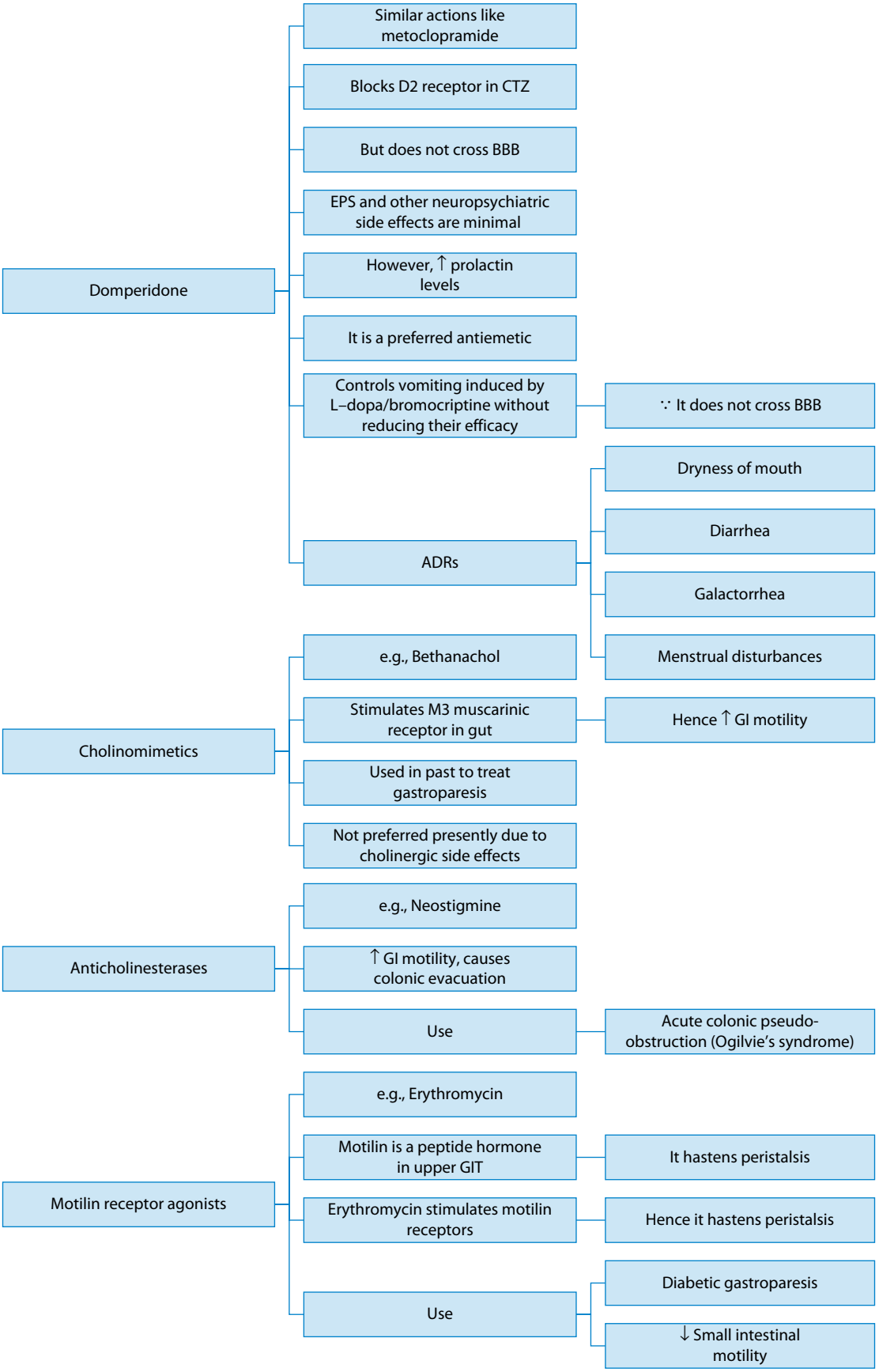
45.5 DOPAMINE D2 RECEPTOR ANTAGONISTS (PROKINETICS)



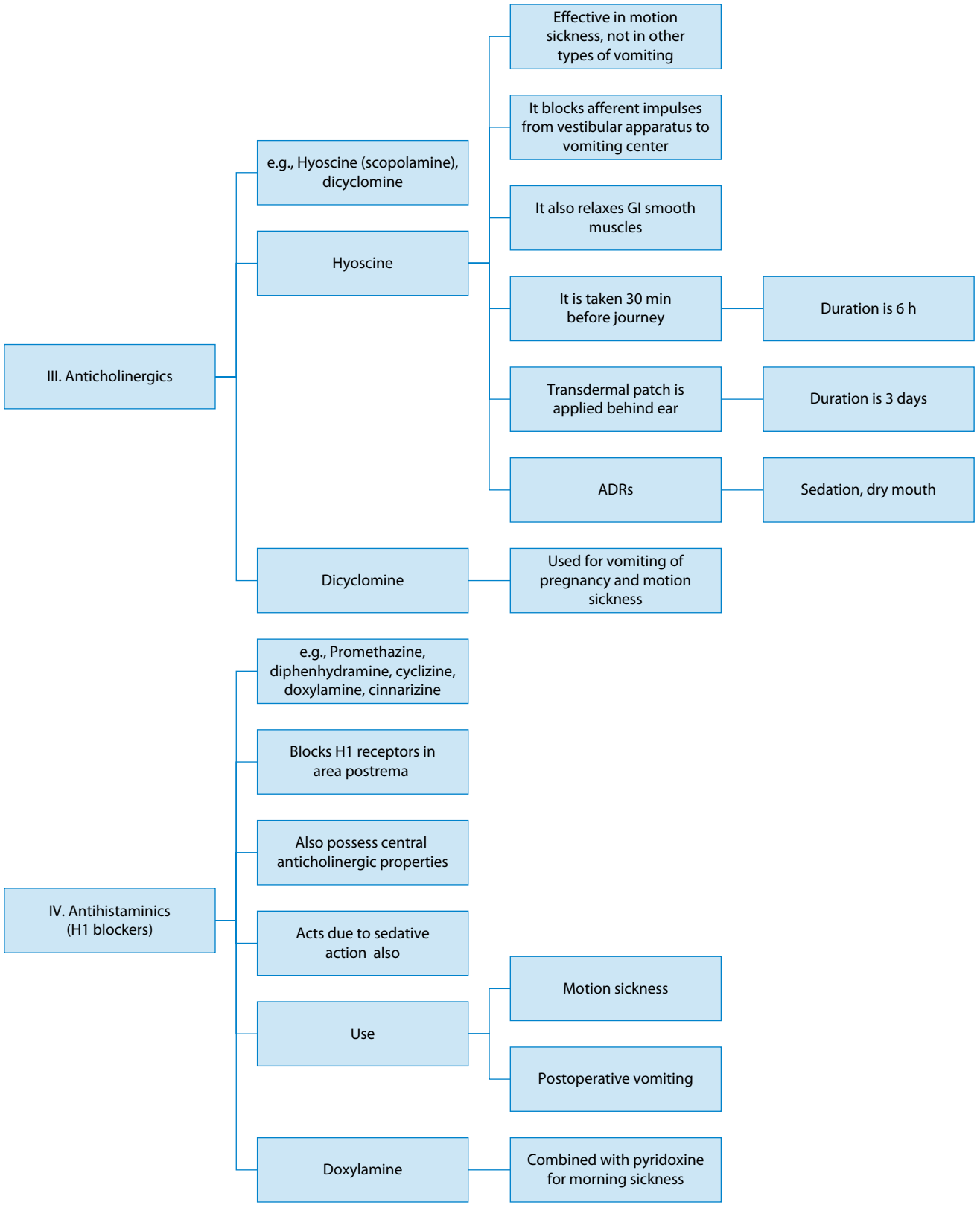
45.6 METOCLOPRAMIDE



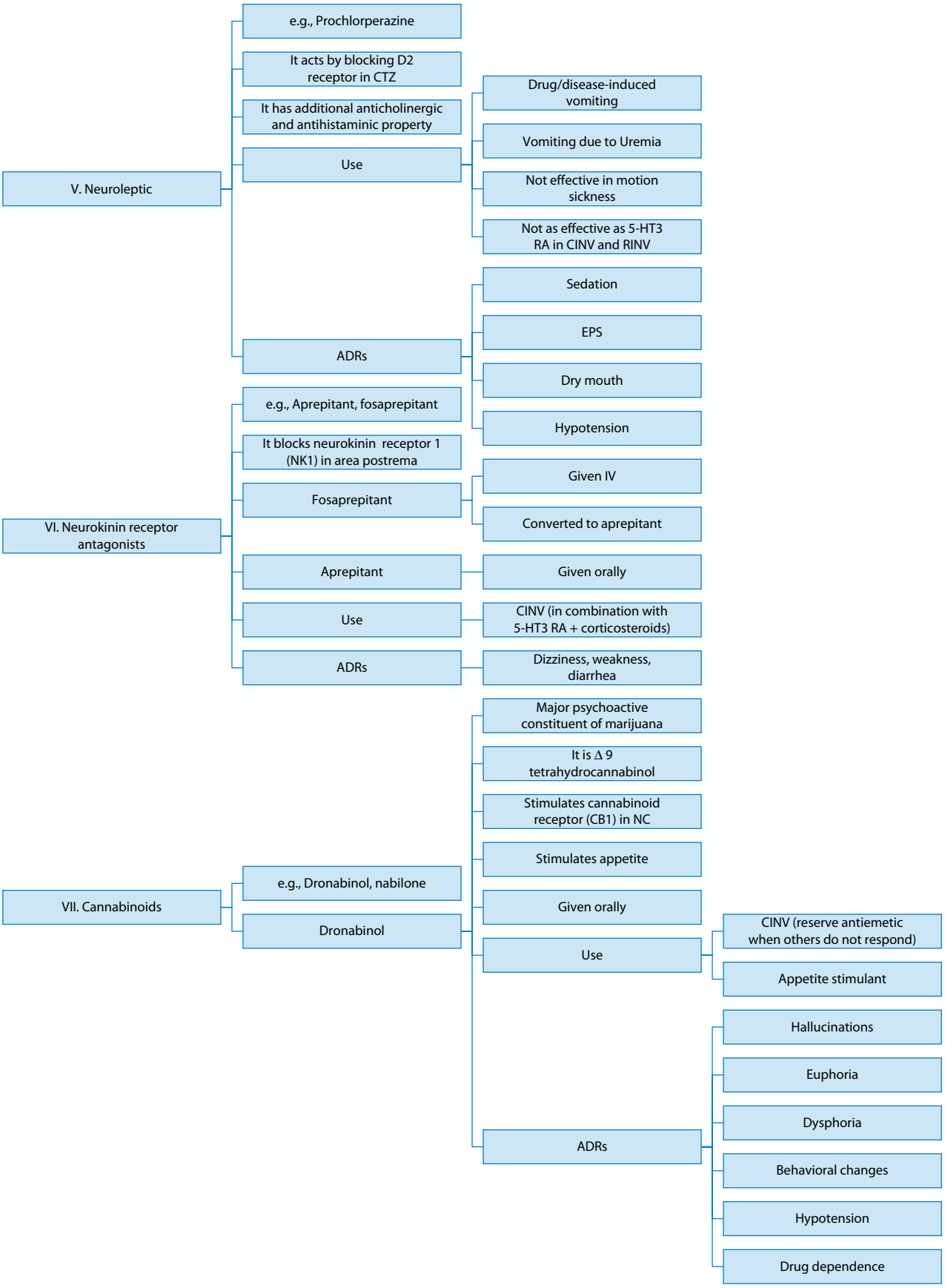
45.7 DOMPERIDONE, CHOLINOMIMETICS, ANTICHOLINESTERASES, AND MOTILIN RECEPTOR AGONISTS



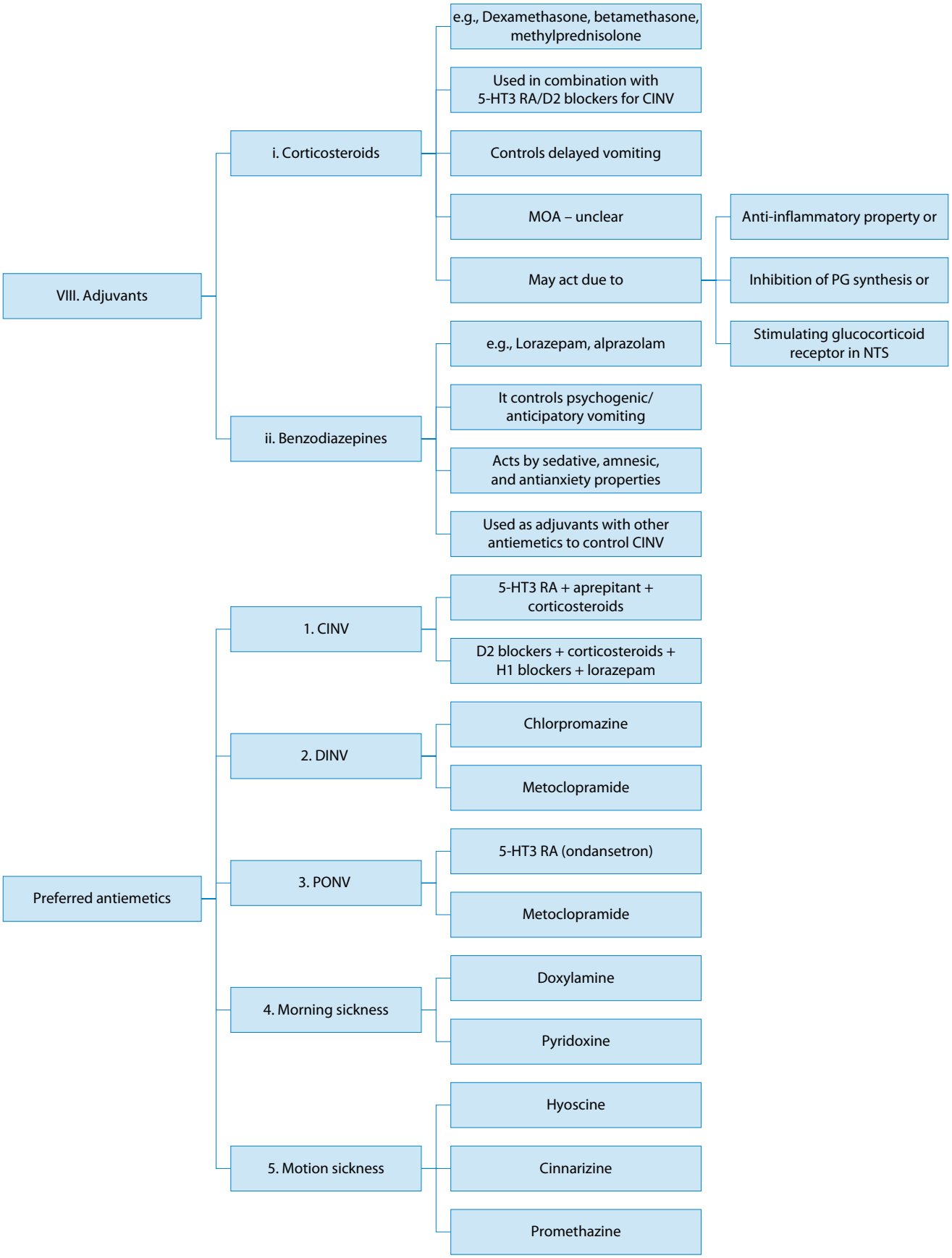
45.8 ANTICHOLINERGICS AND ANTIHISTAMINICS (H1 BLOCKERS)



45.9 NEUROLEPTIC, NEUROKININ RECEPTOR ANTAGONISTS, AND CANNABINOIDS



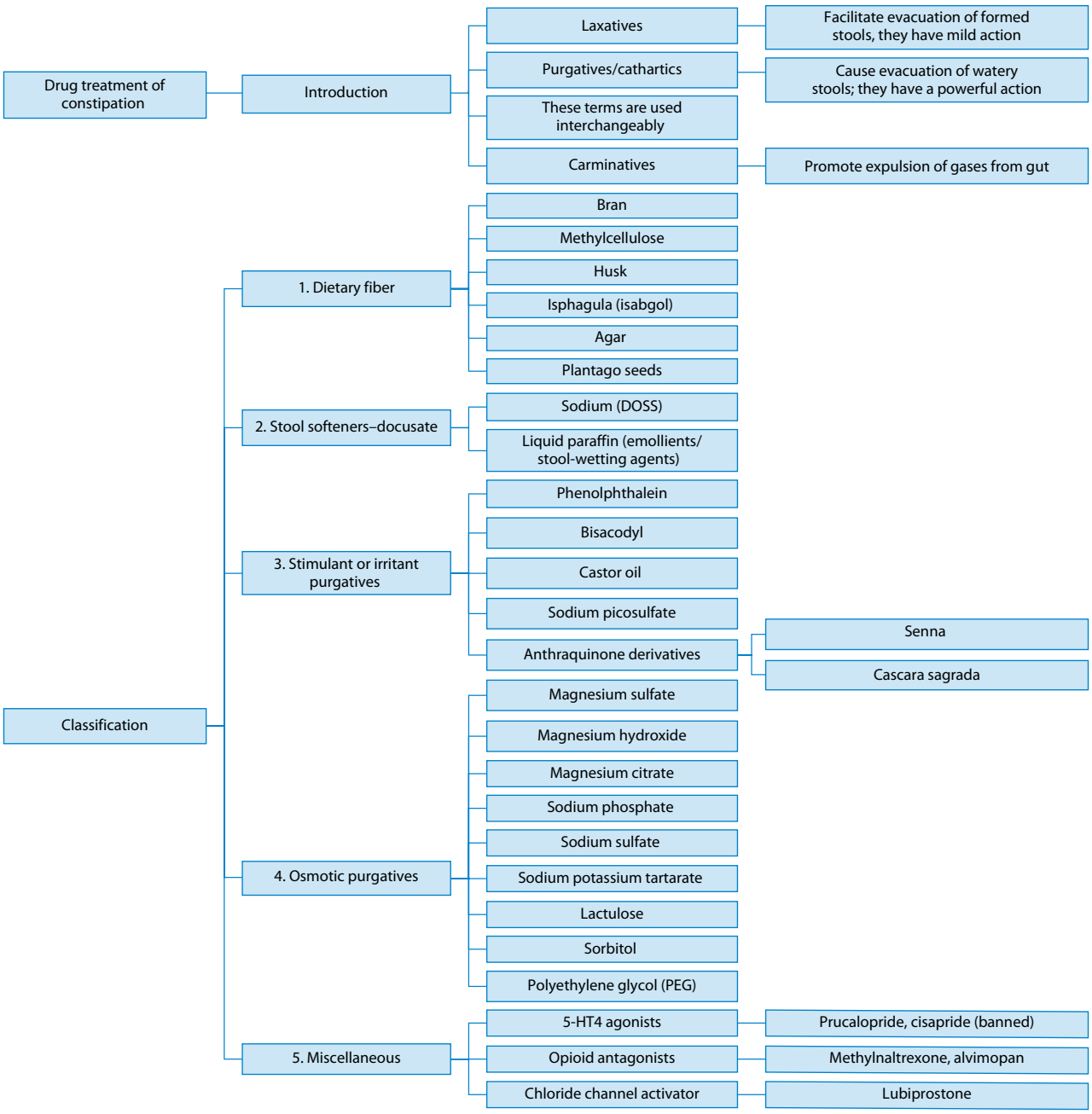
45.10 ADJUVANTS AND PREFERRED ANTIEMETICS



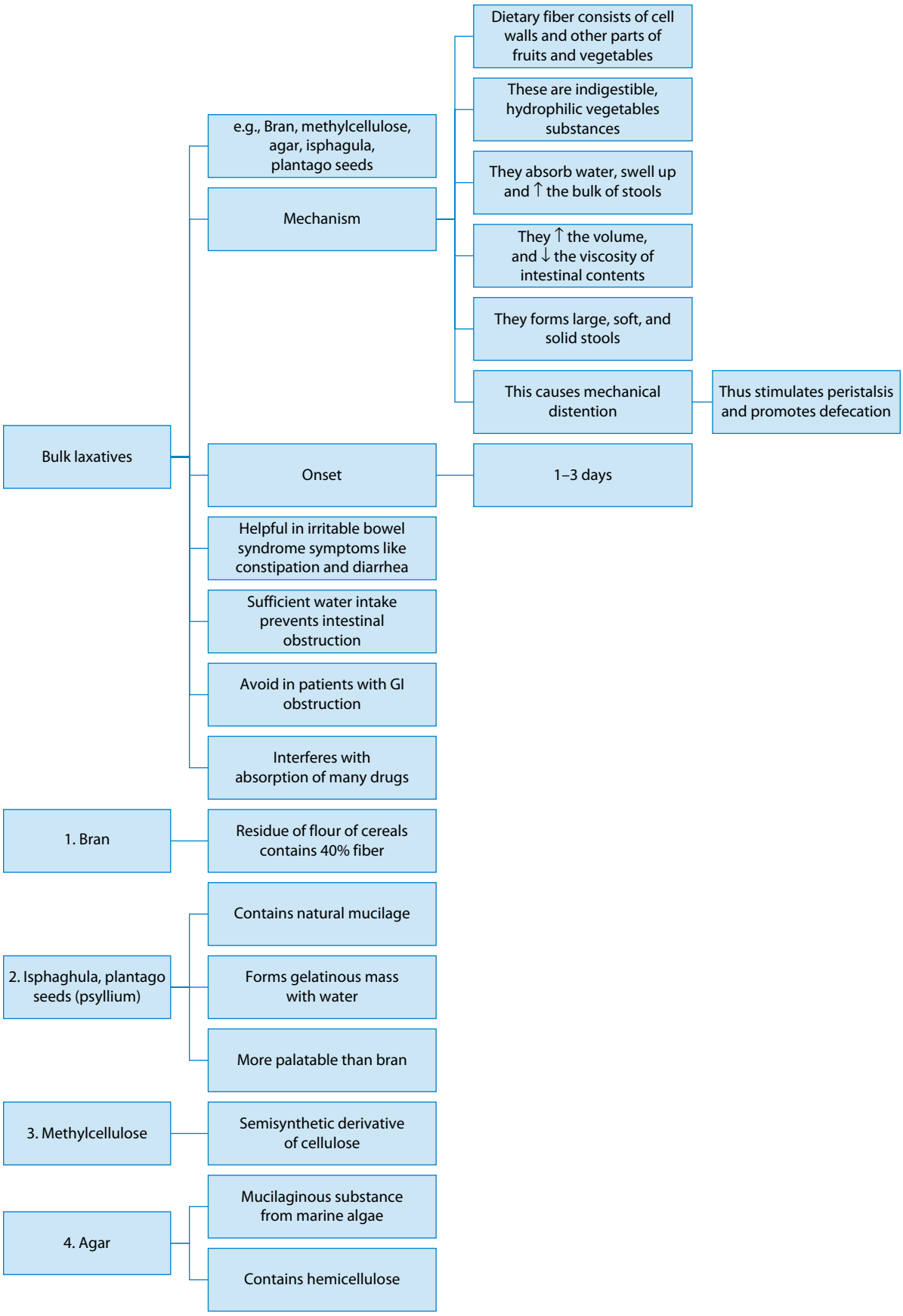
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Drug treatment of constipation, treatment of IBS, and IBD

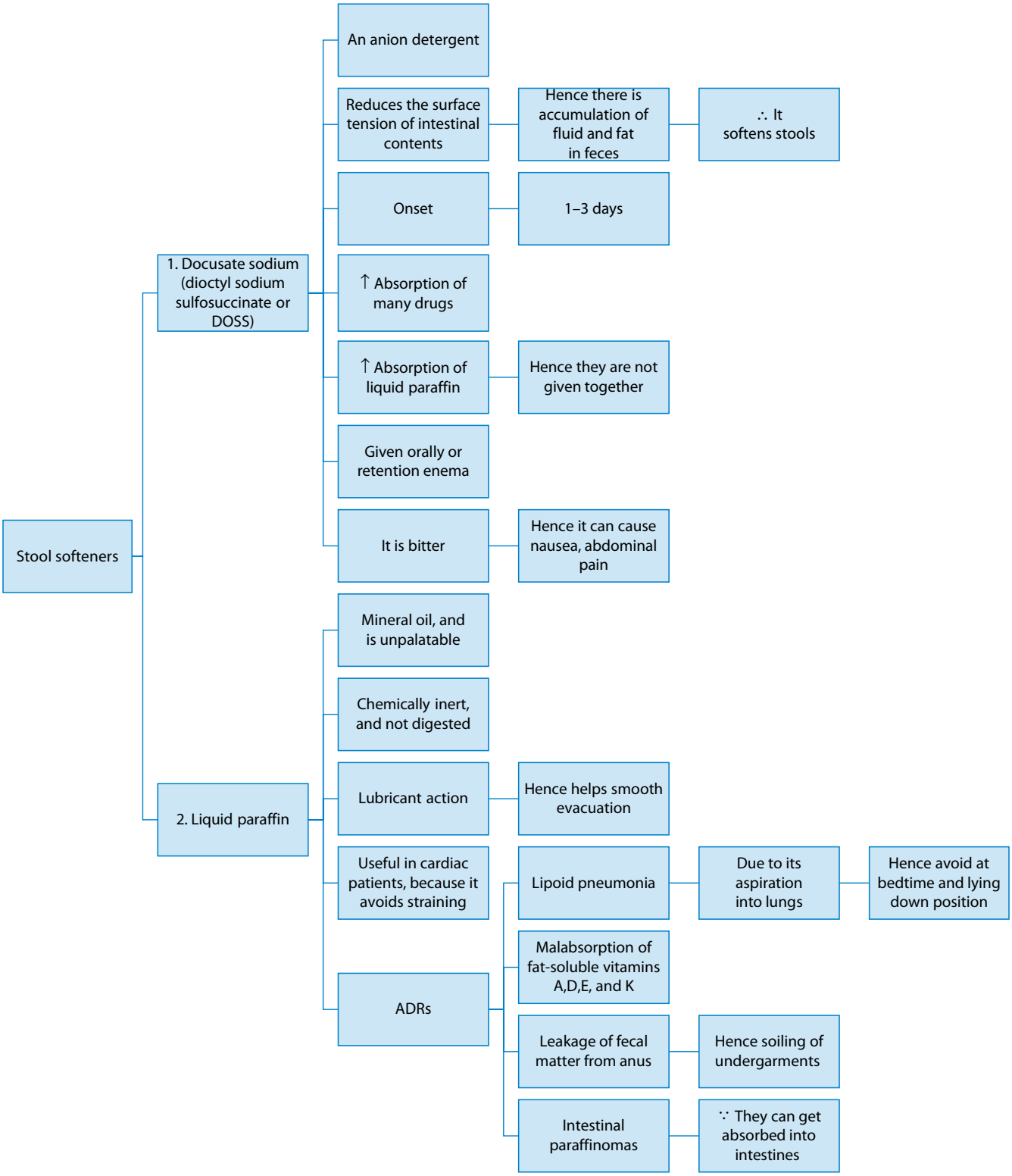
46.1 INTRODUCTION AND CLASSIFICATION



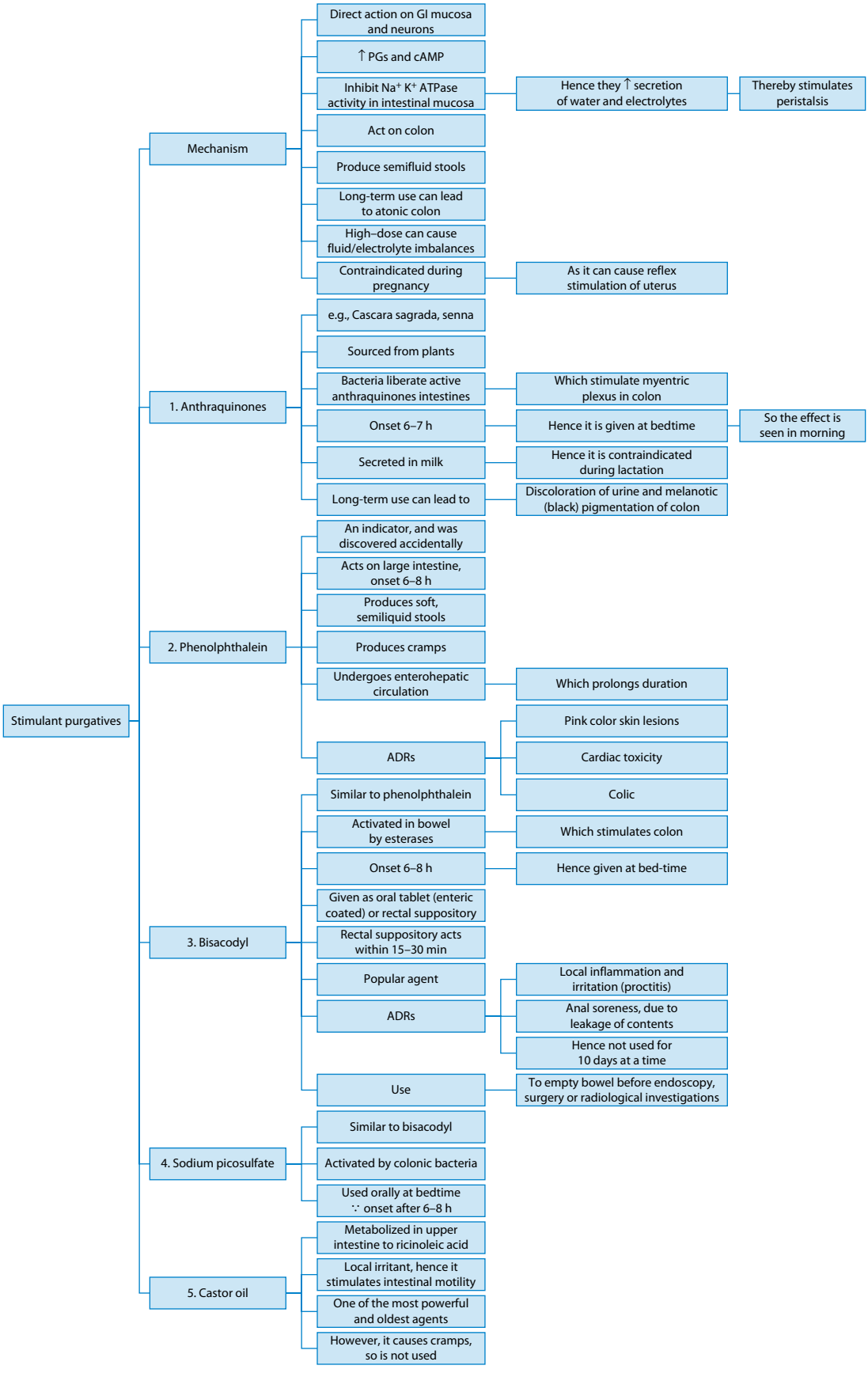
46.2 BULK LAXATIVES



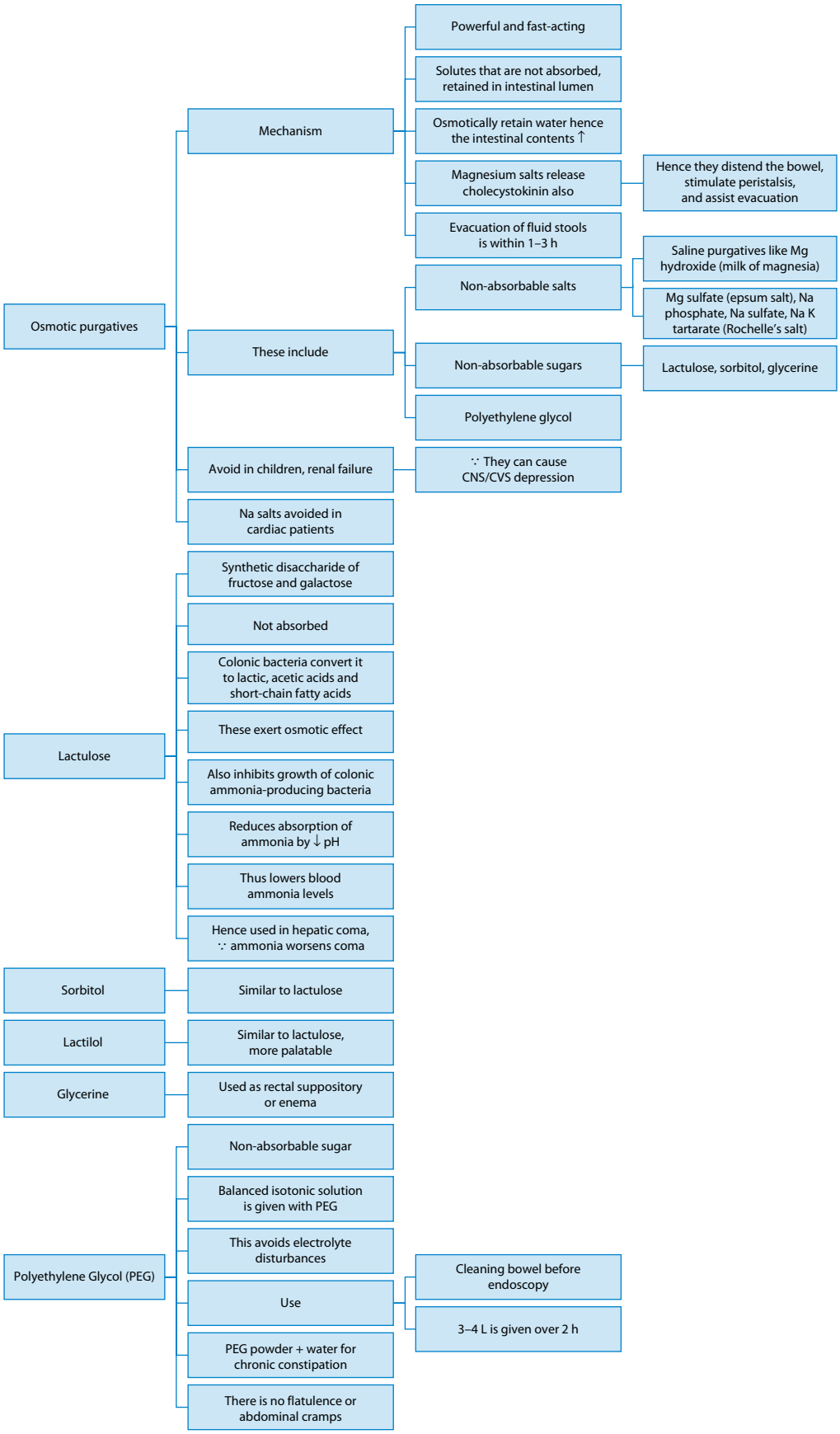
46.3 STOOL SOFTENERS



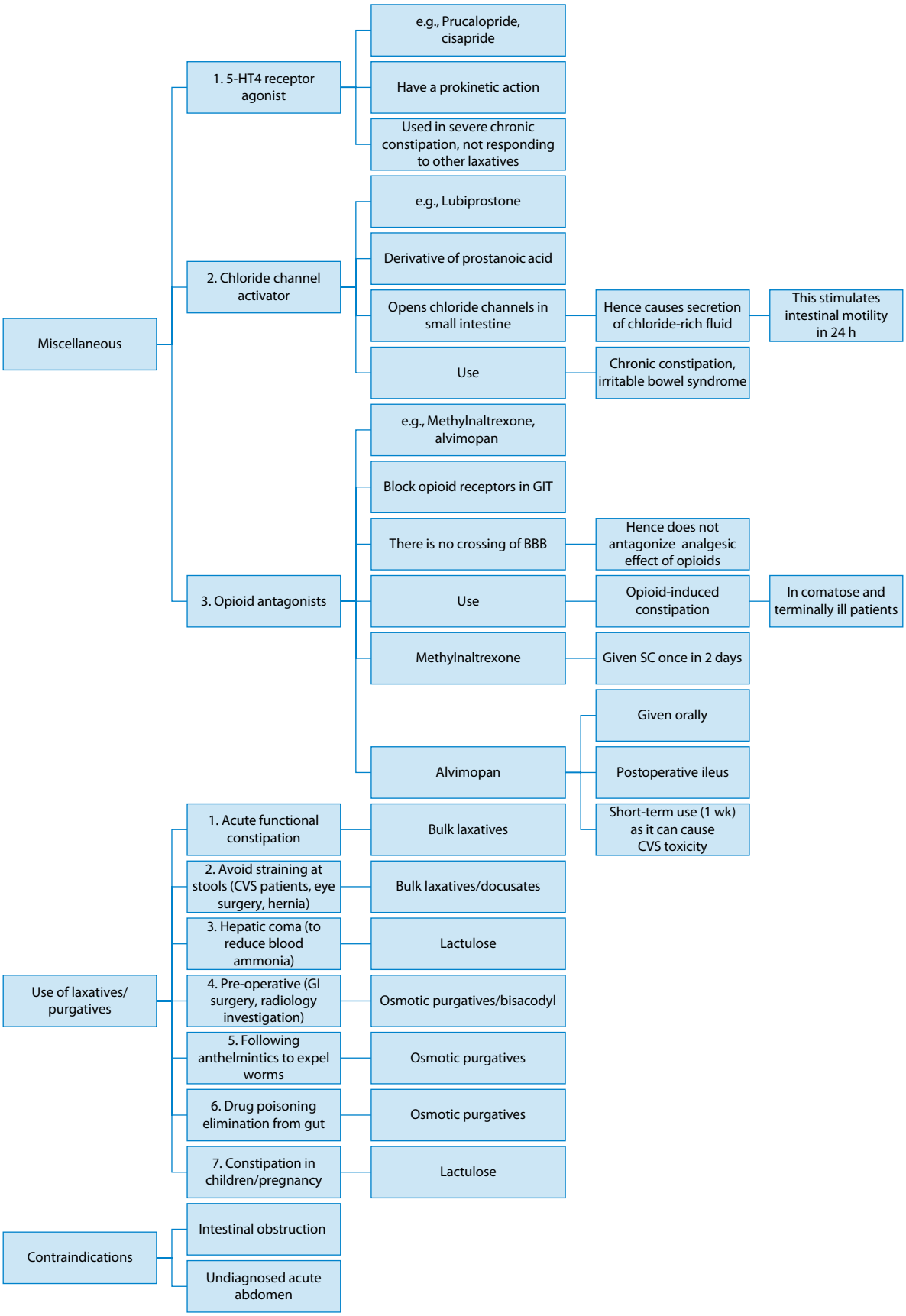
46.4 STIMULANT PURGATIVES



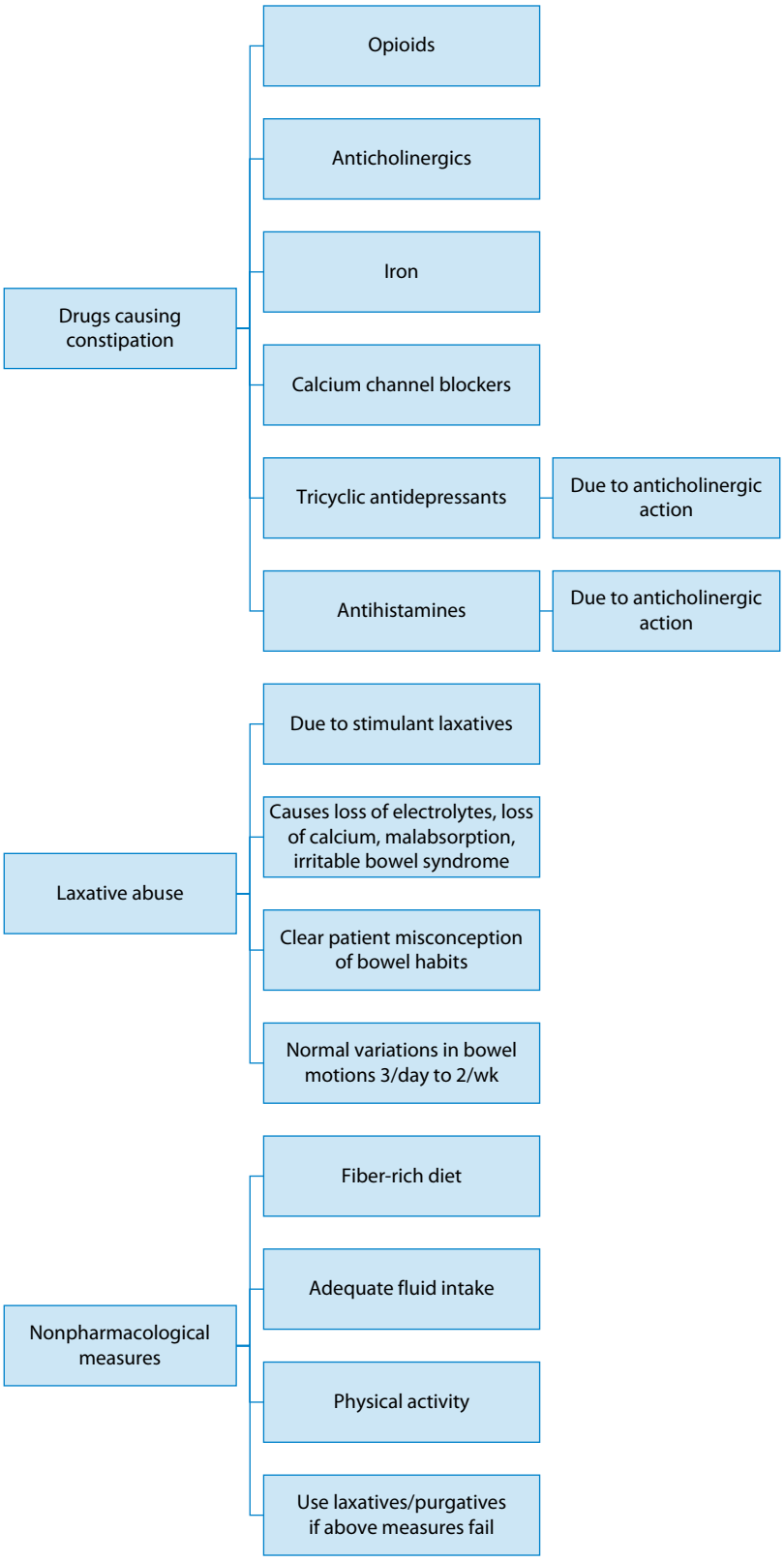
46.5 OSMOTIC PURGATIVES



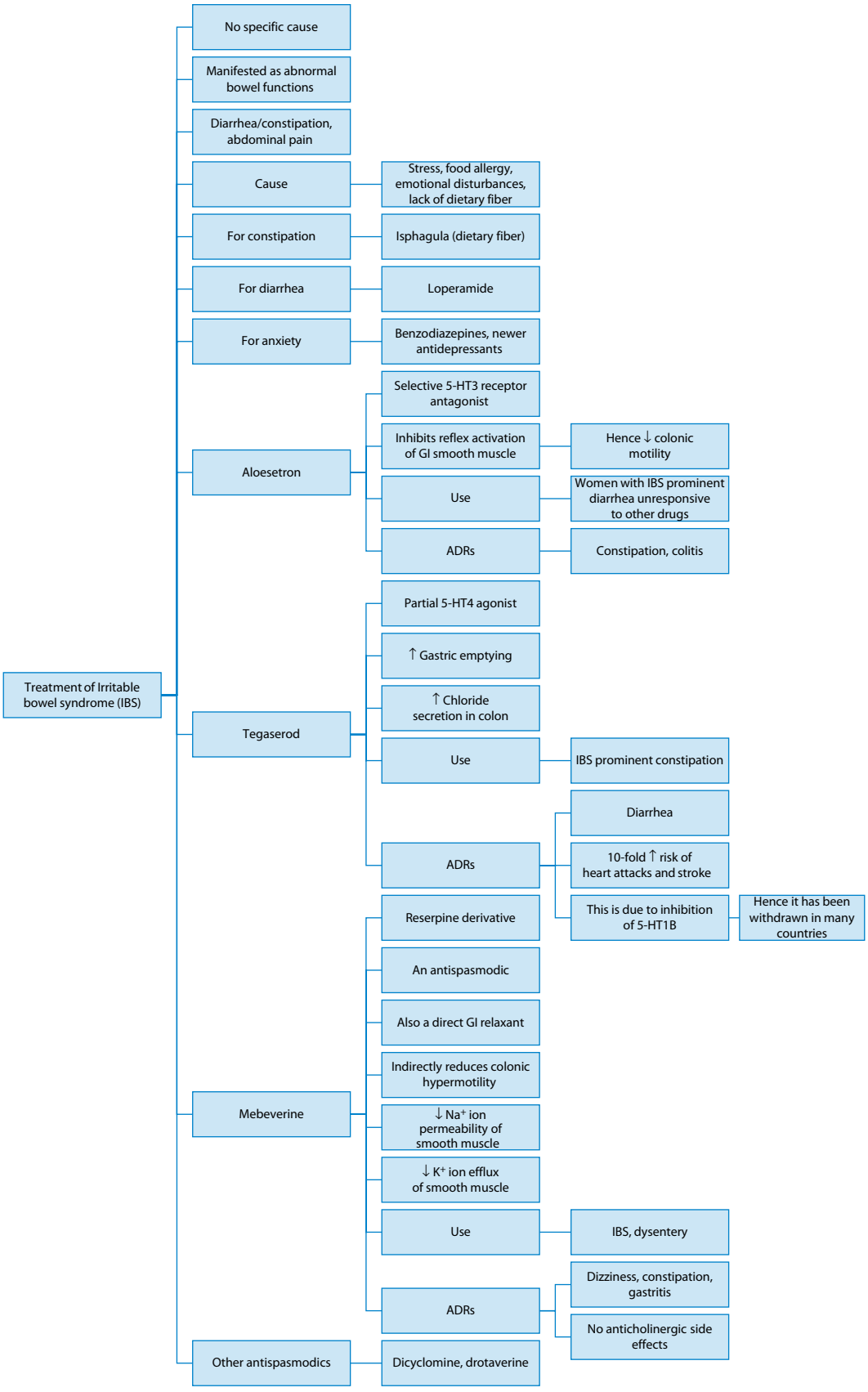
46.6 MISCELLANEOUS AGENTS AND USE OF LAXATIVES/PURGATIVES



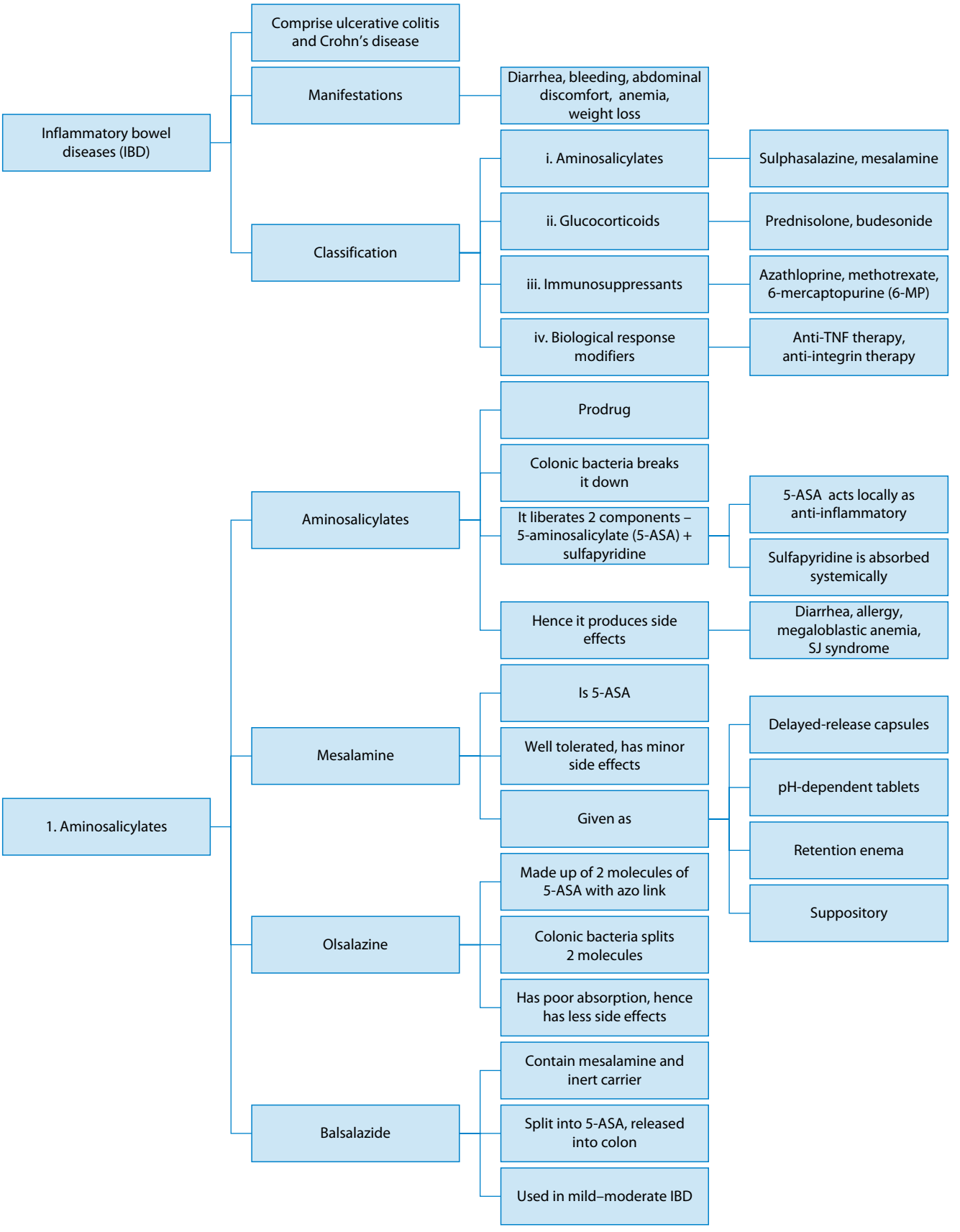
46.7 DRUGS CAUSING CONSTIPATION, LAXATIVE ABUSE, AND NONPHARMACOLOGICAL MEASURES



46.8 TREATMENT OF IRRITABLE BOWEL SYNDROME (IBS)

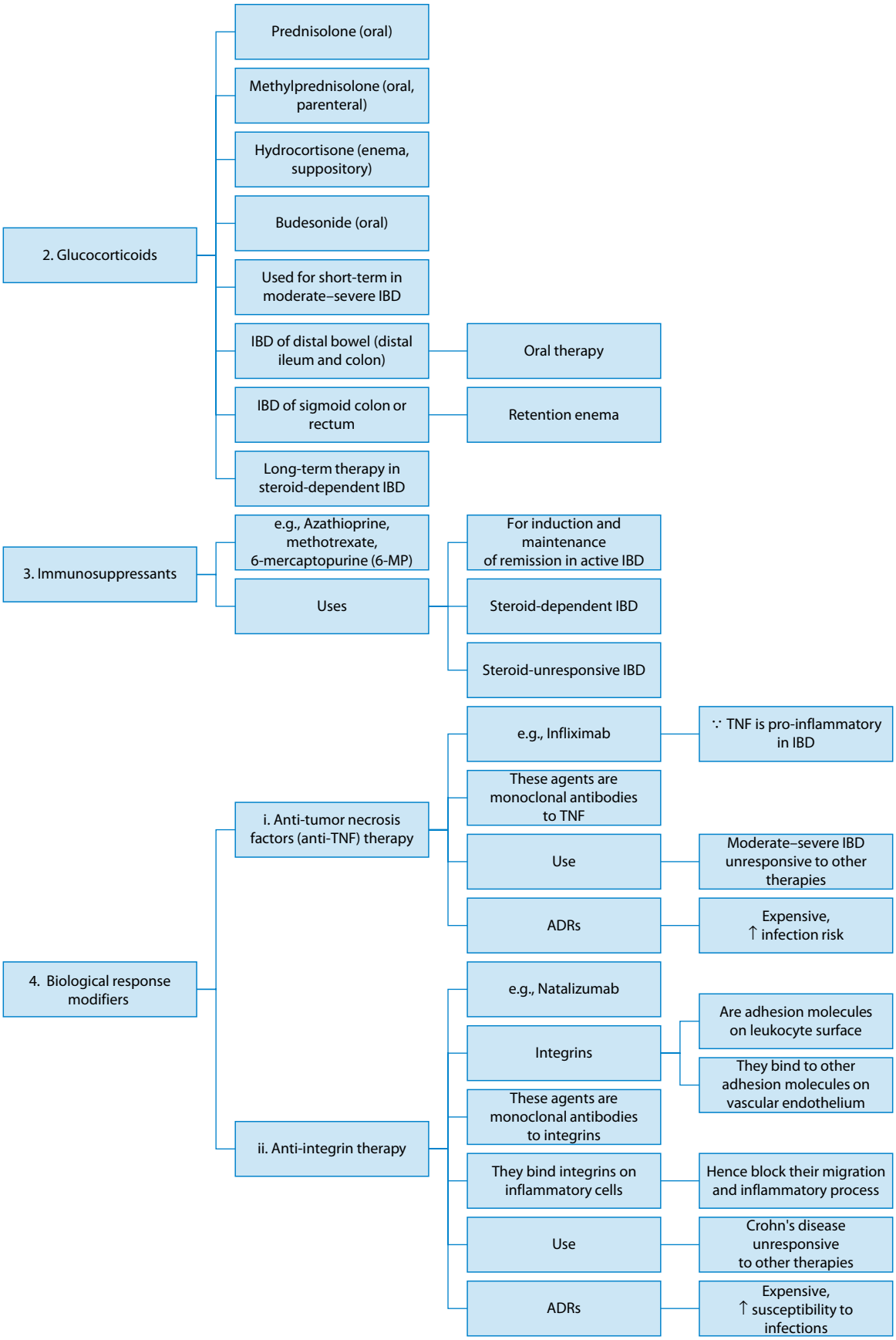


46.9 INFLAMMATORY BOWEL DISEASES (IBD) AND TREATMENT



(*Continued*)

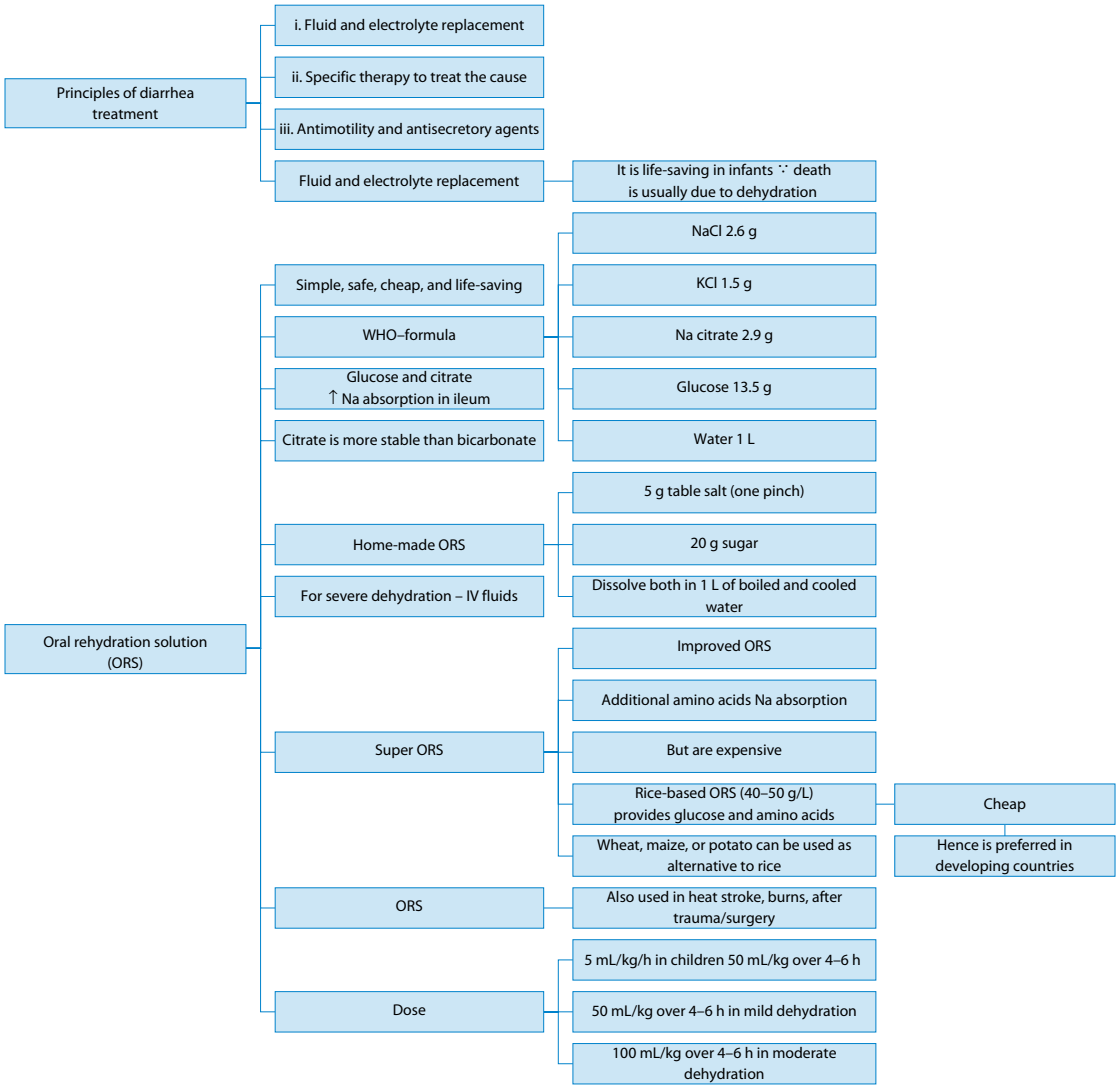
46.9 INFLAMMATORY BOWEL DISEASES (IBD) AND TREATMENT (Continued)



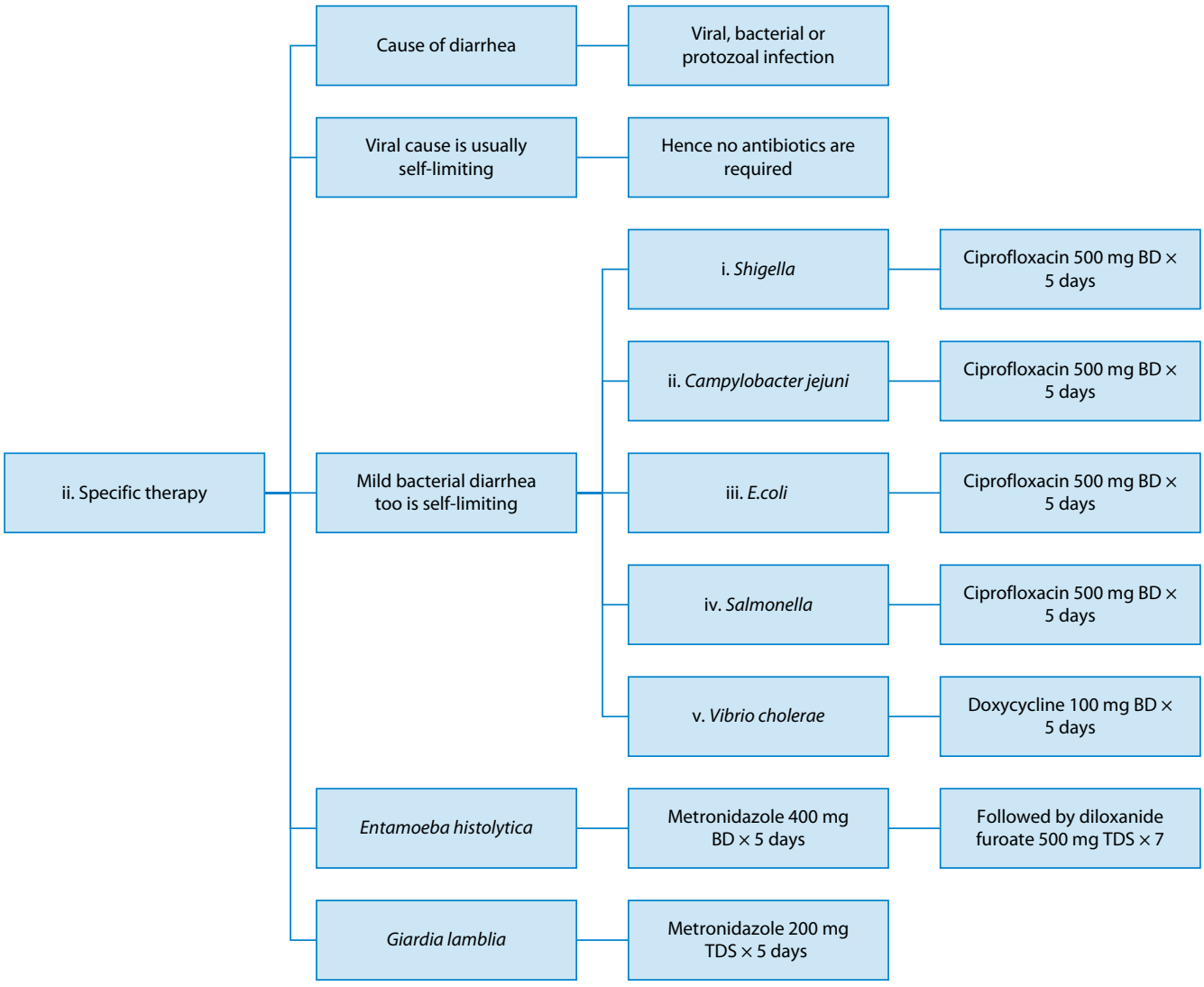
47

Drug treatment of diarrhea

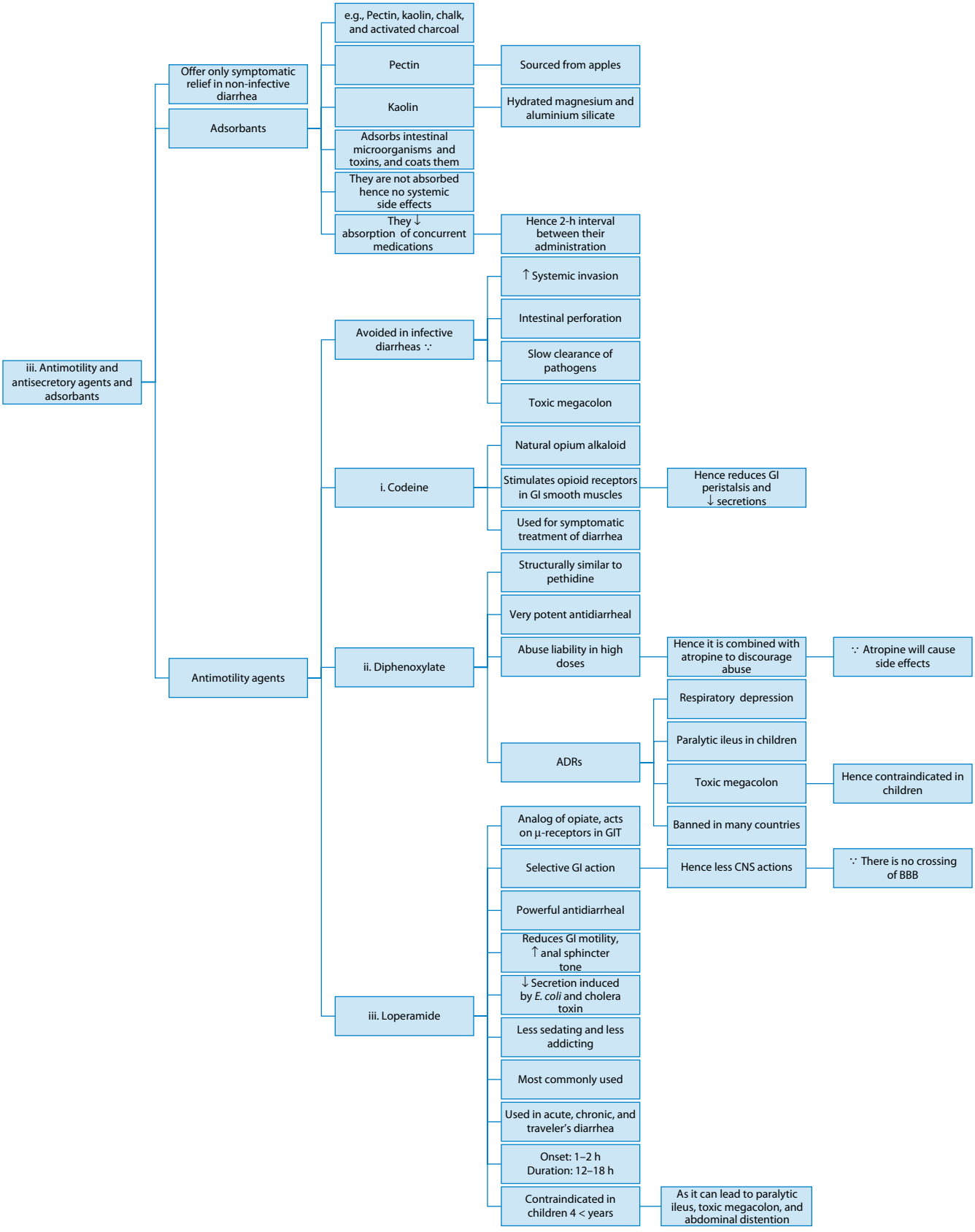
47.1 PRINCIPLES OF DIARRHEA TREATMENT AND ORS



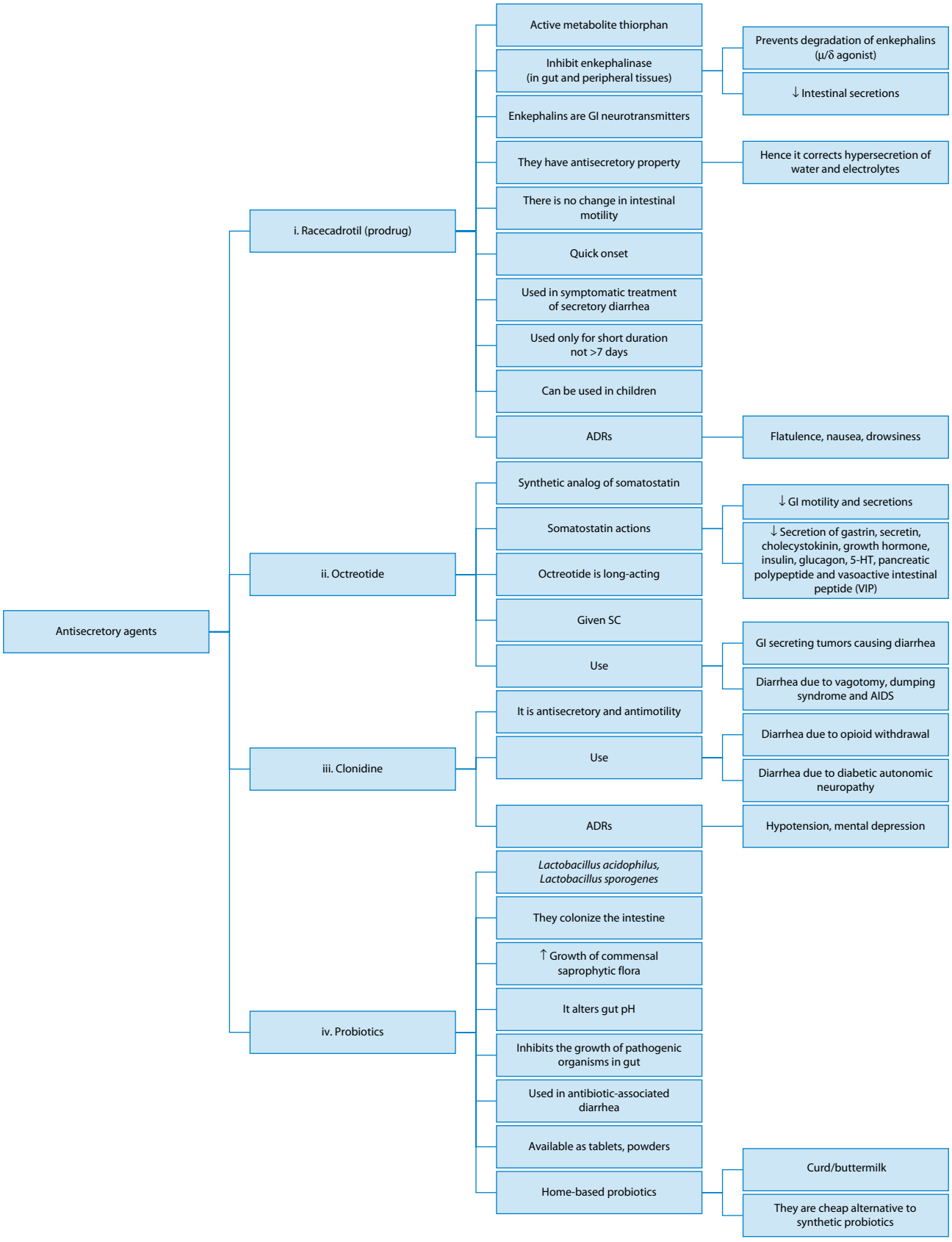
47.2 SPECIFIC THERAPY



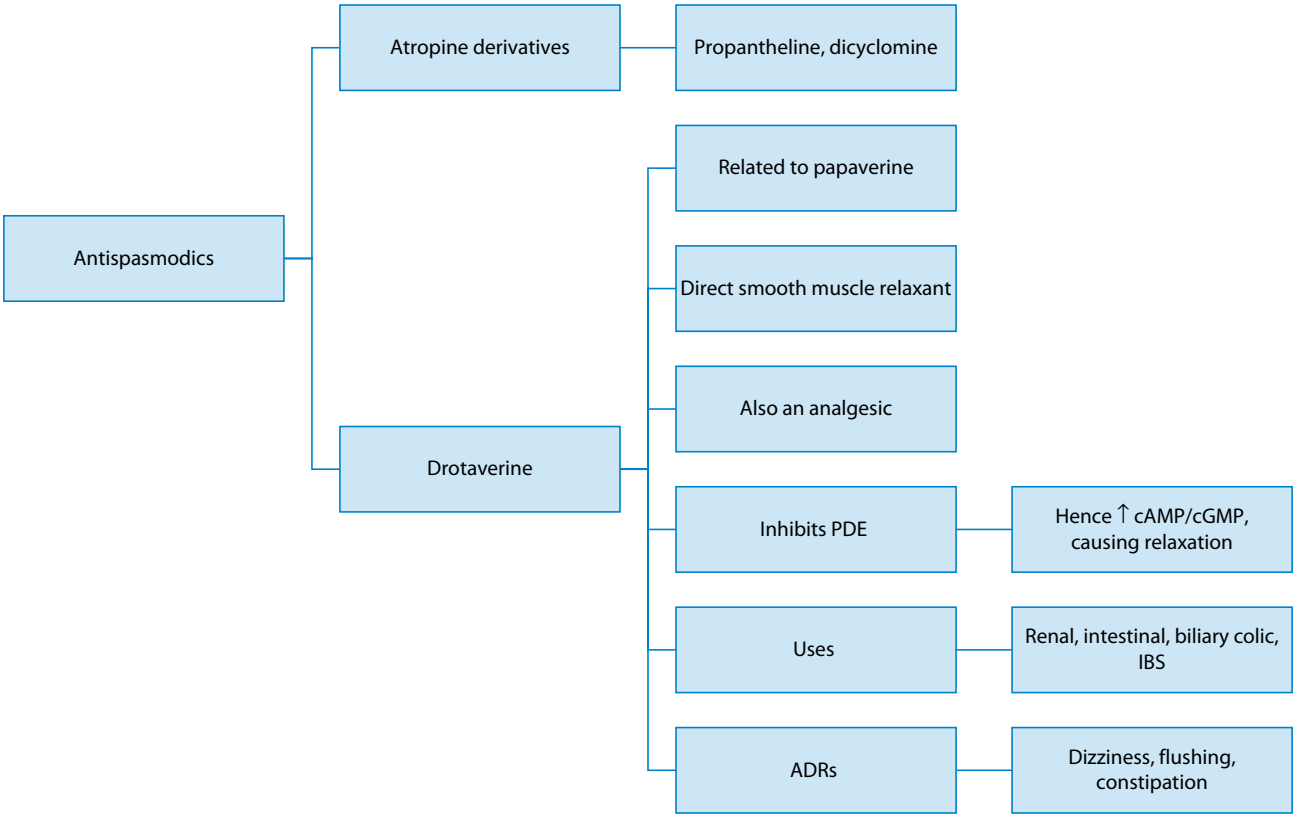
47.3 ANTIMOTILITY AND ANTISECRETORY AGENTS AND ADSORBANTS



47.4 ANTISECRETORY AGENTS AND PROBIOTICS



47.5 ANTISPASMODICS





Taylor & Francis
Taylor & Francis Group
http://taylorandfrancis.com

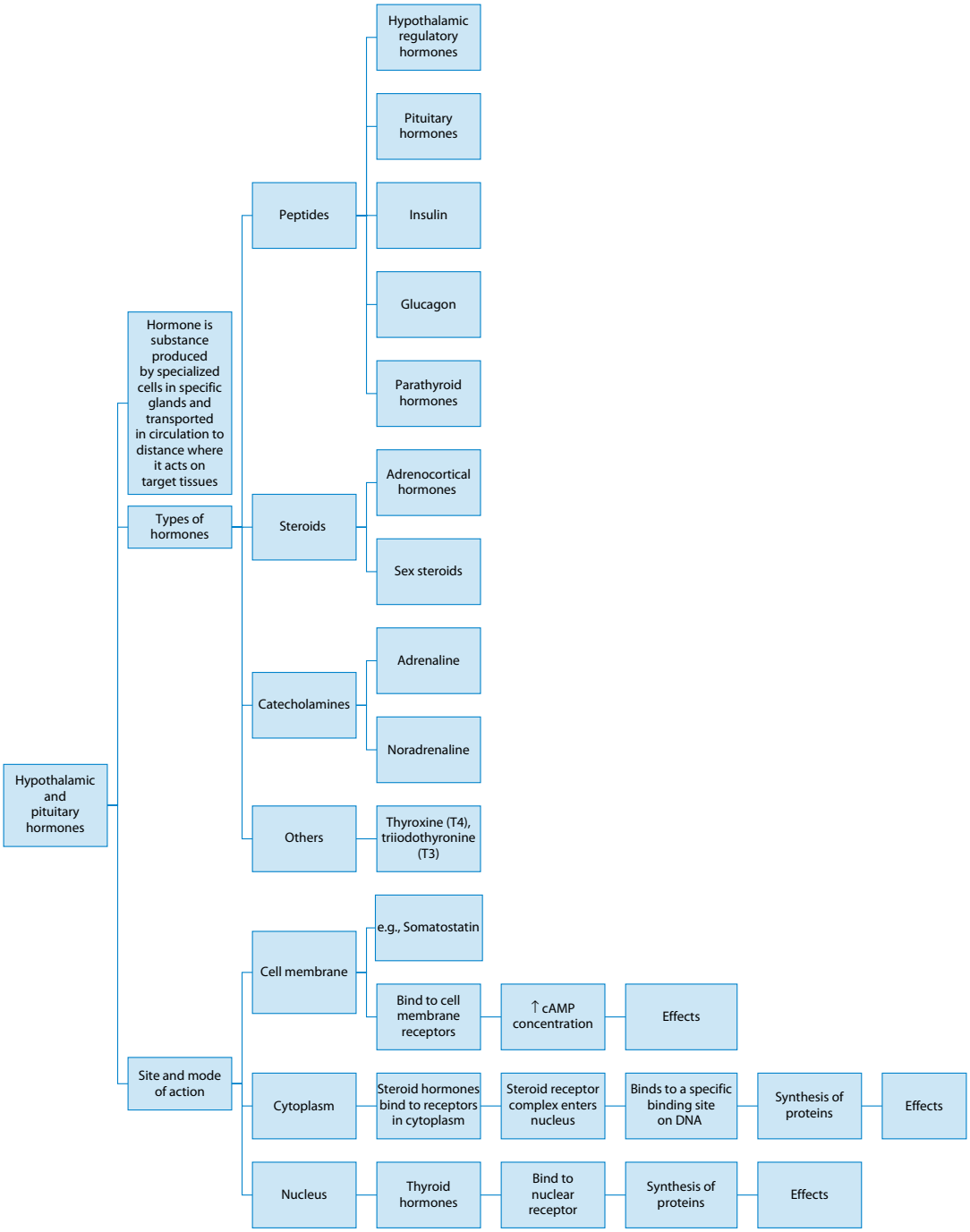
PART IX

Endocrine pharmacology

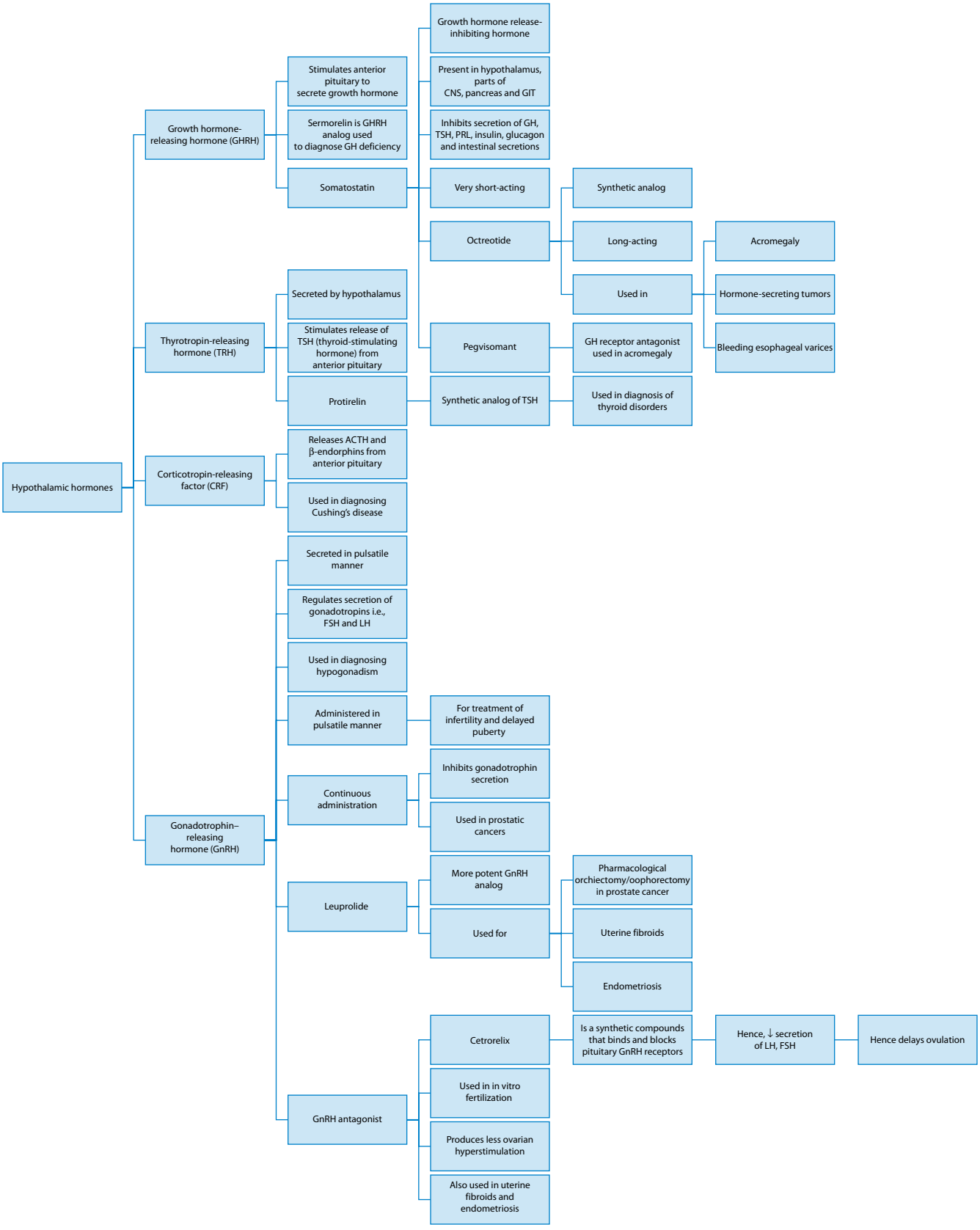
48

Hypothalamic and pituitary hormones

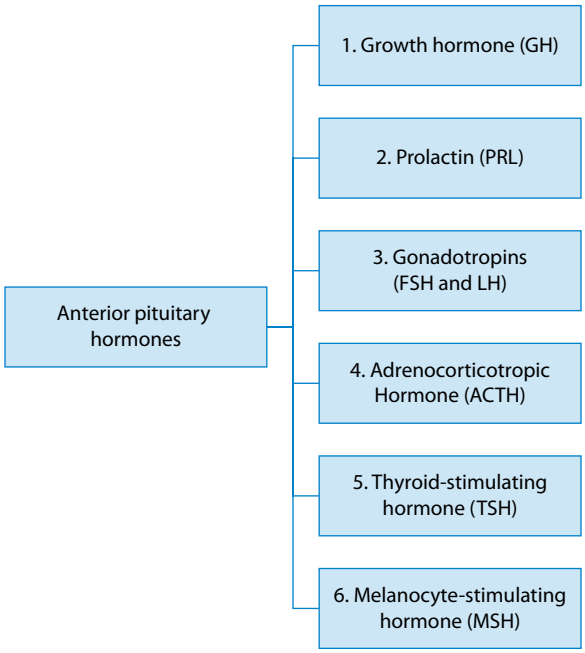
48.1 HYPOTHALAMIC AND PITUITARY HORMONES – TYPES, MODES, AND MECHANISM OF ACTION



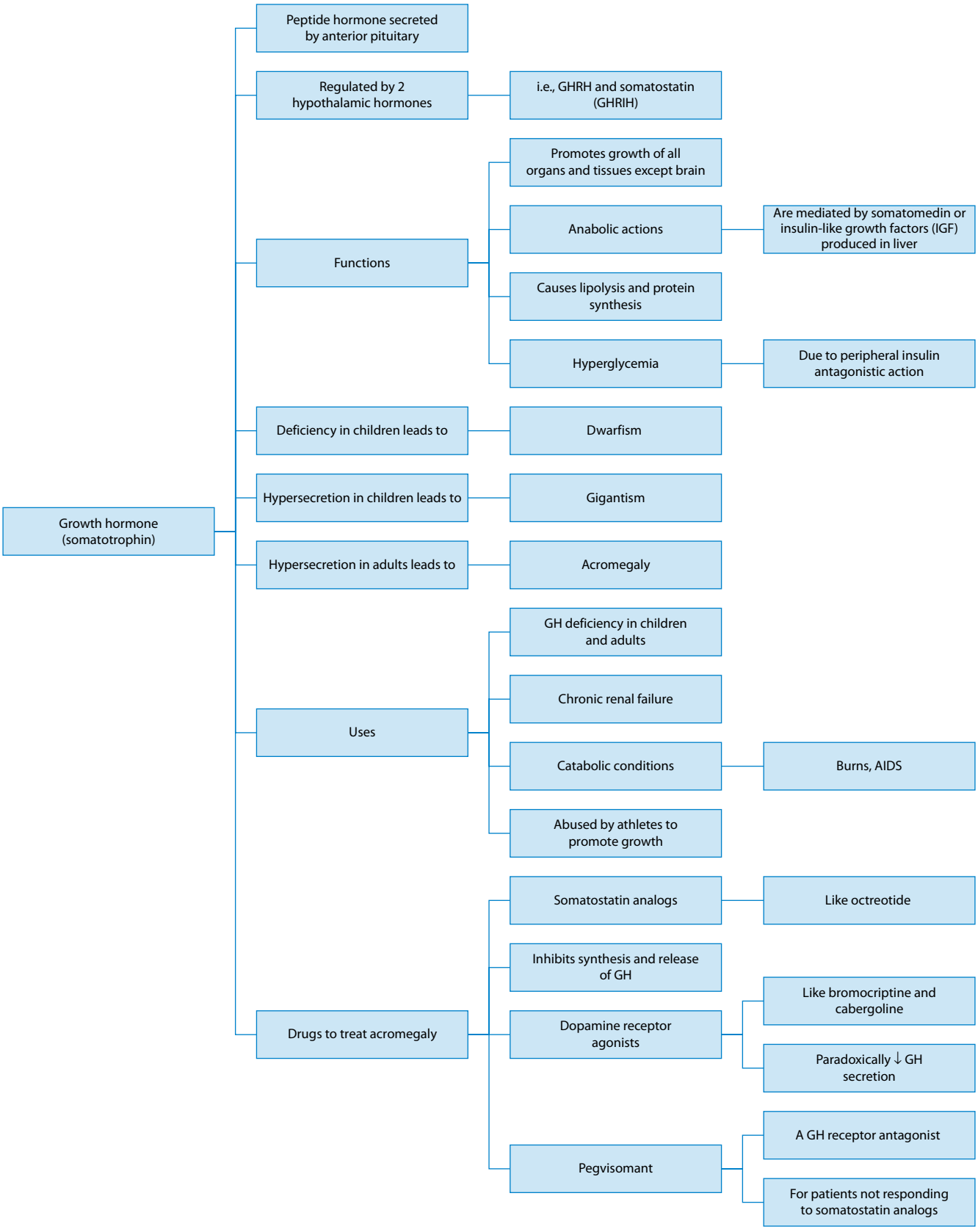
48.2 HYPOTHALAMIC HORMONES



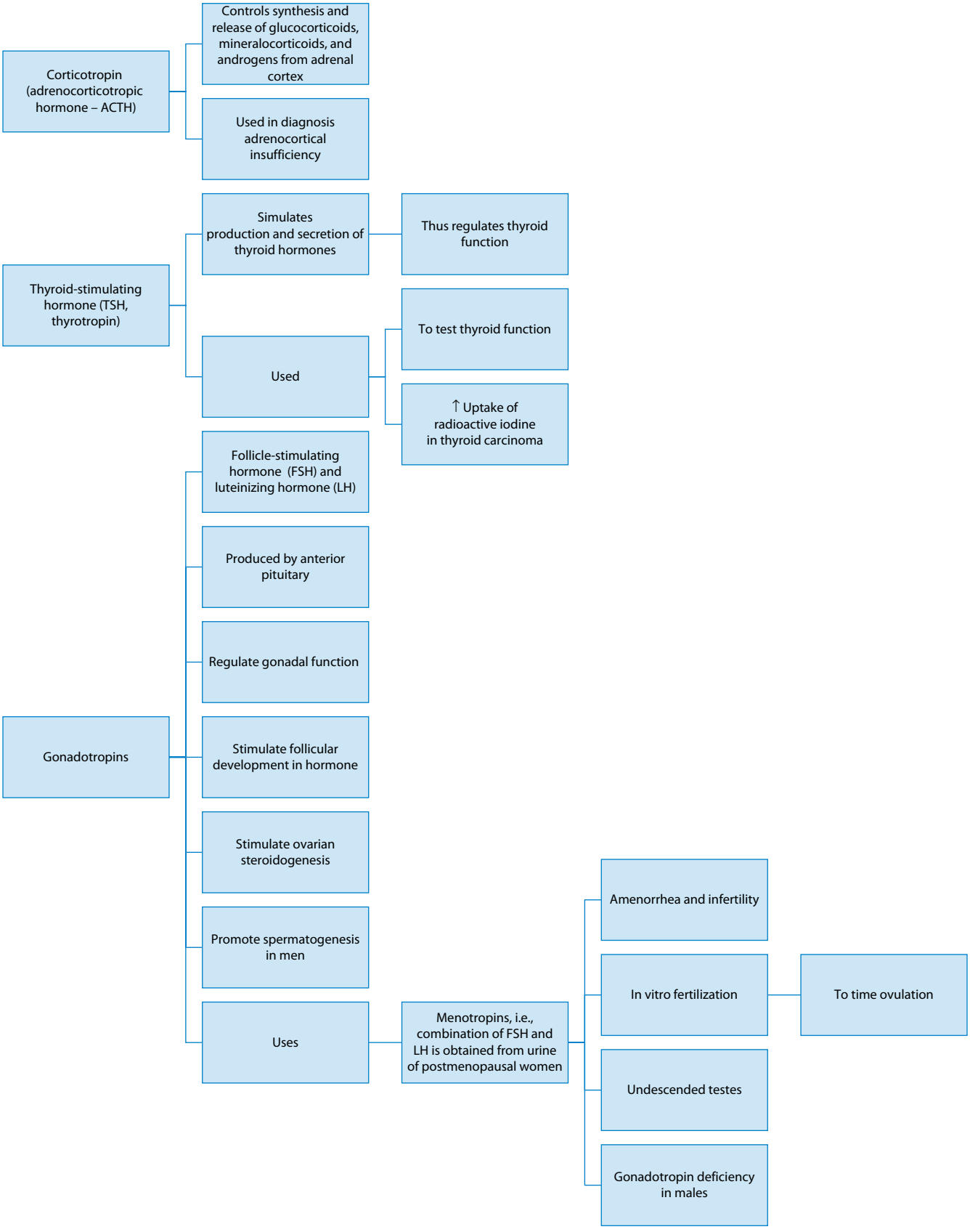
48.3 ANTERIOR PITUITARY HORMONES



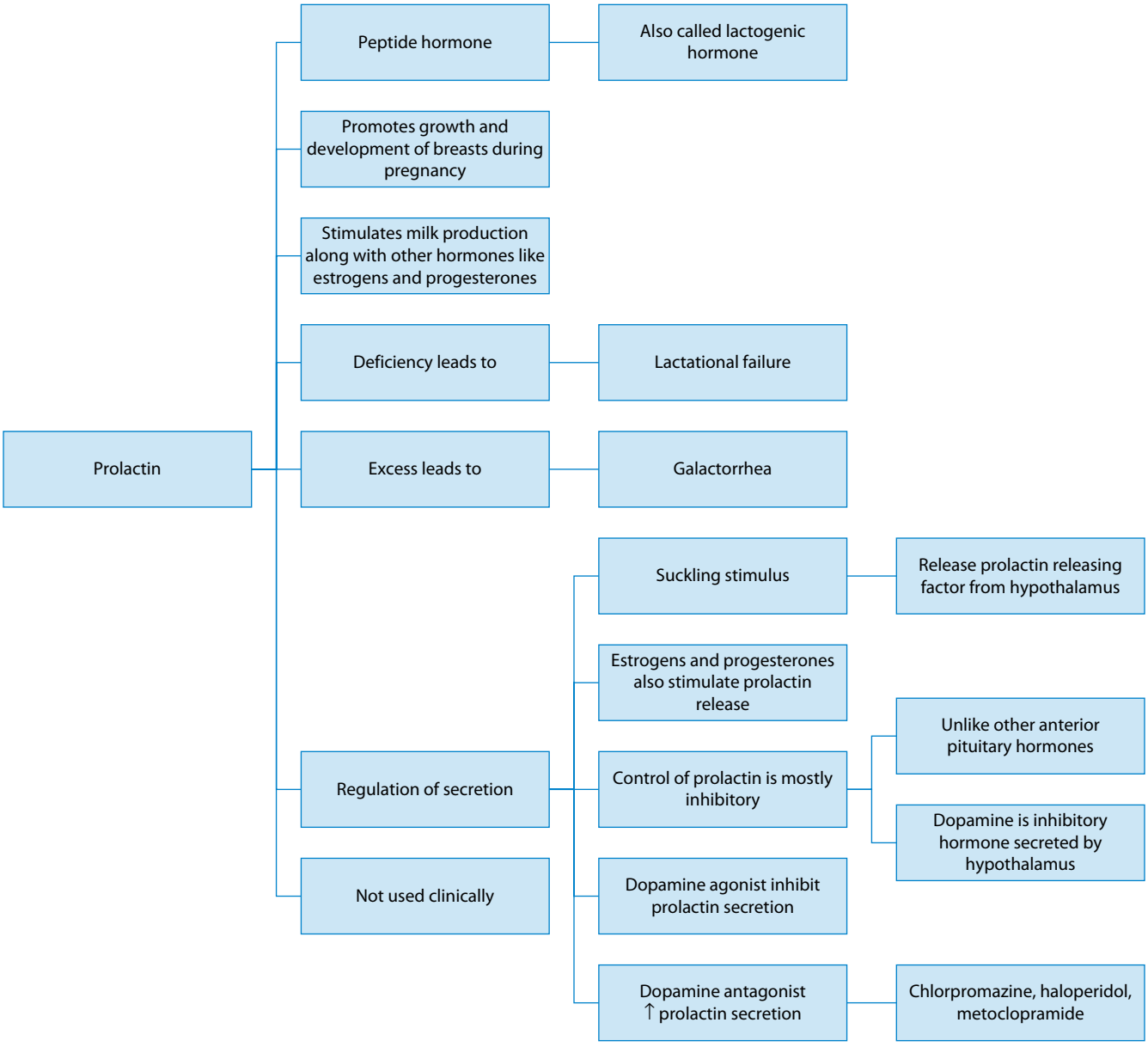
48.4 GROWTH HORMONE (SOMATOTROPHIN)



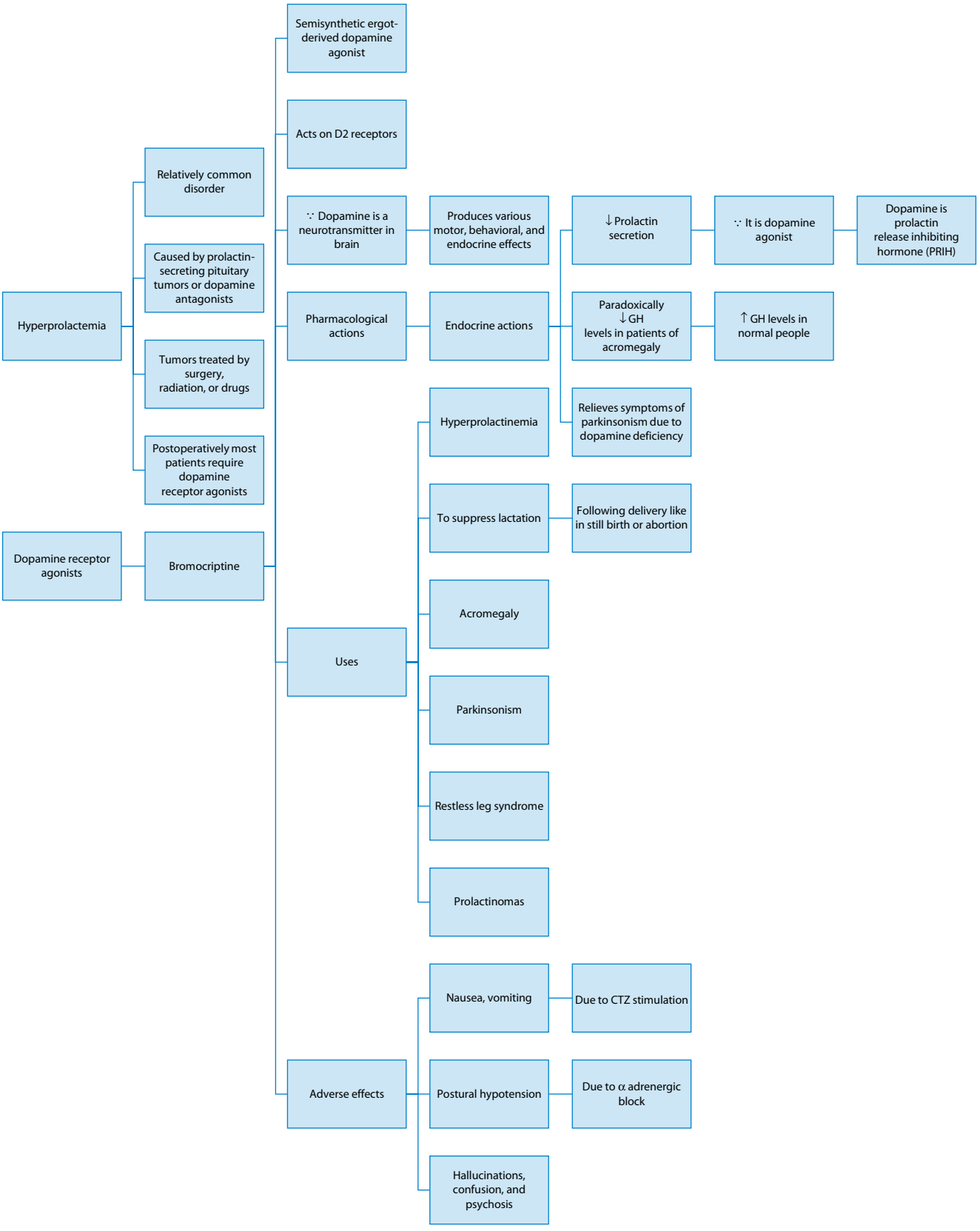
48.5 CORTICOTROPIN (ADRENOCORTICOTROPIC HORMONE – ACTH), THYROID-STIMULATING HORMONE (TSH, THYROTROPHIN), AND GONADOTROPINS



48.6 PROLACTIN



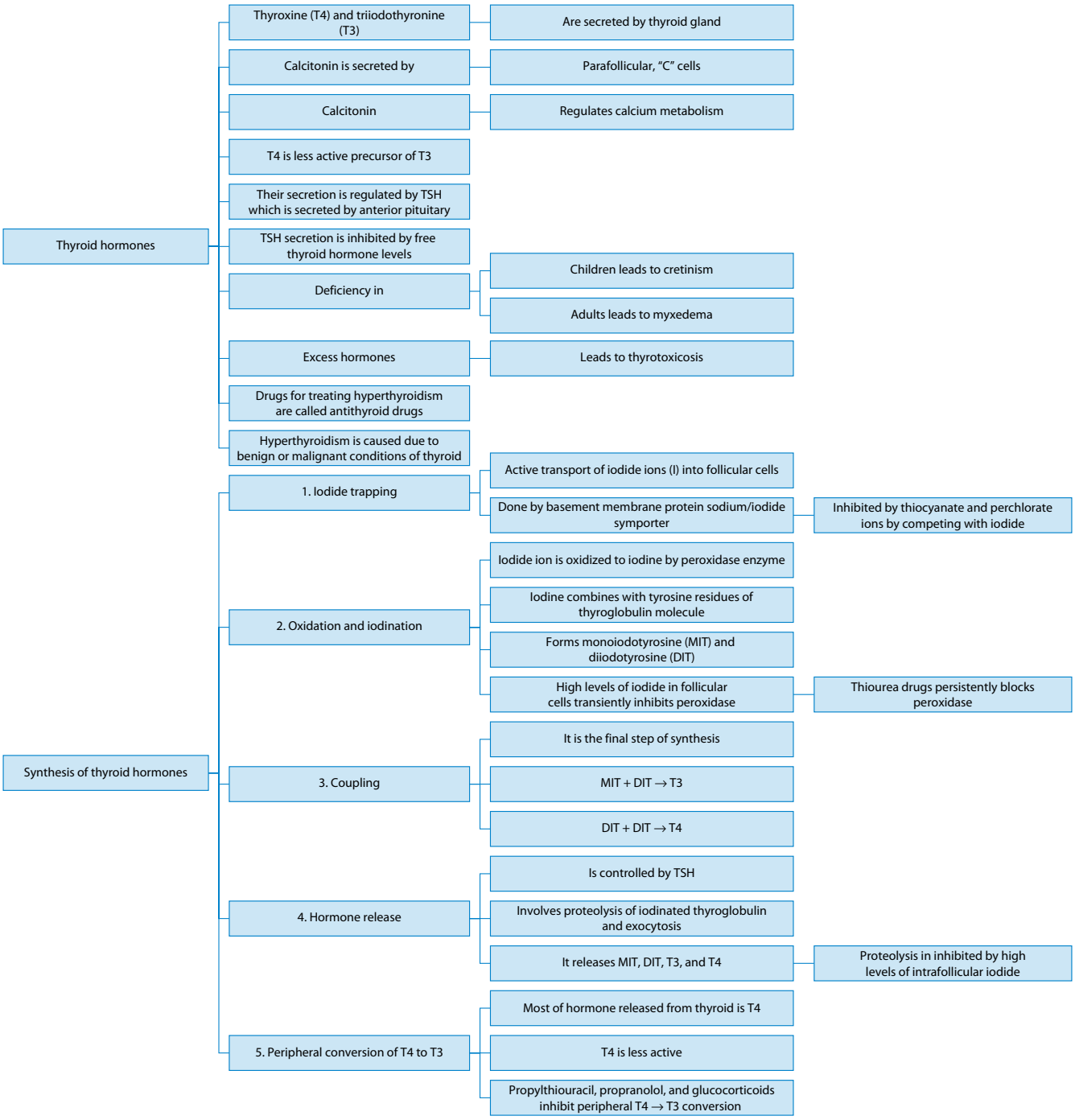
48.7 HYPERPROLACTEMIA AND DOPAMINE RECEPTOR AGONISTS



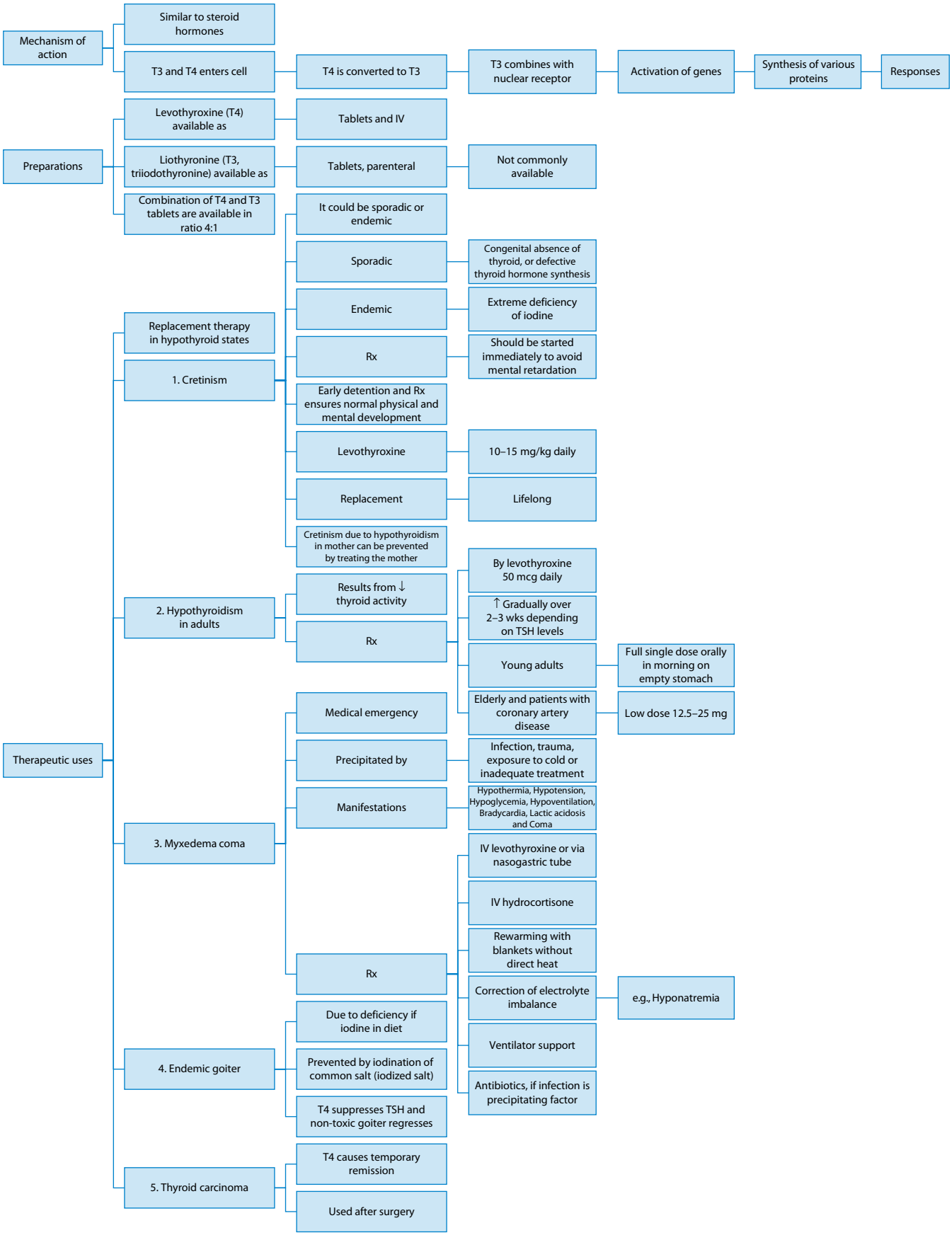
49

Thyroid hormones and antithyroid agents

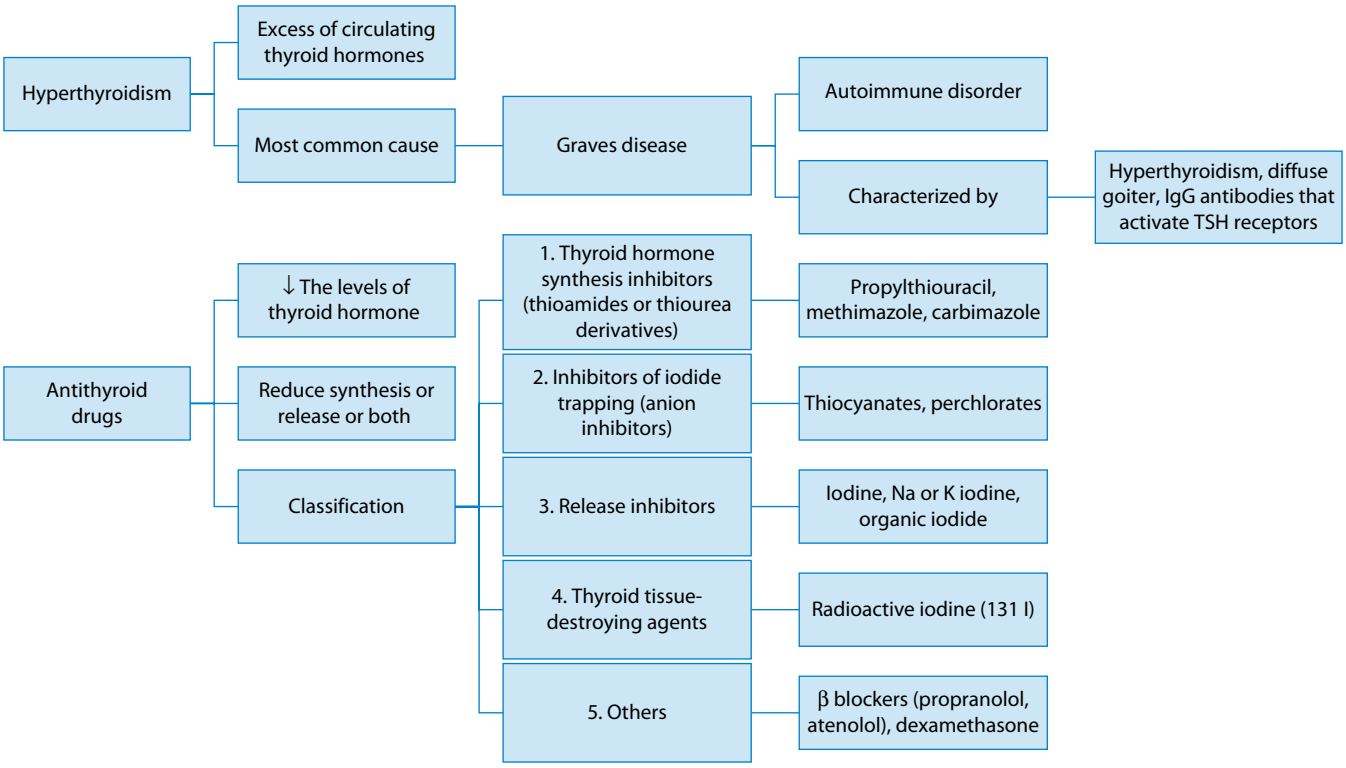
49.1 THYROID HORMONES – REGULATION AND SYNTHESIS



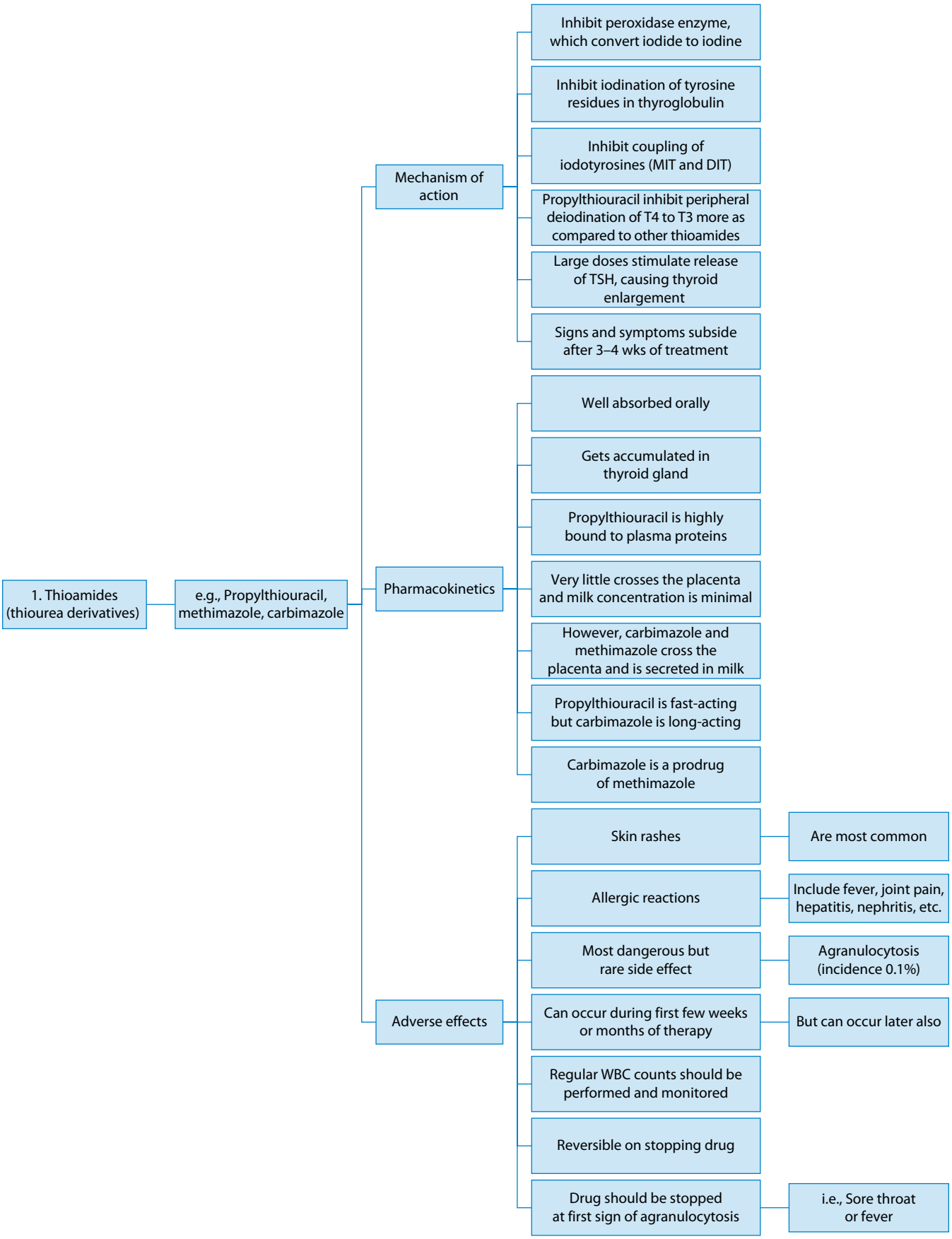
49.2 MECHANISM OF ACTION, PREPARATIONS, AND THERAPEUTIC USES



49.3 HYPERTHYROIDISM AND CLASSIFICATION OF ANTITHYROID DRUGS

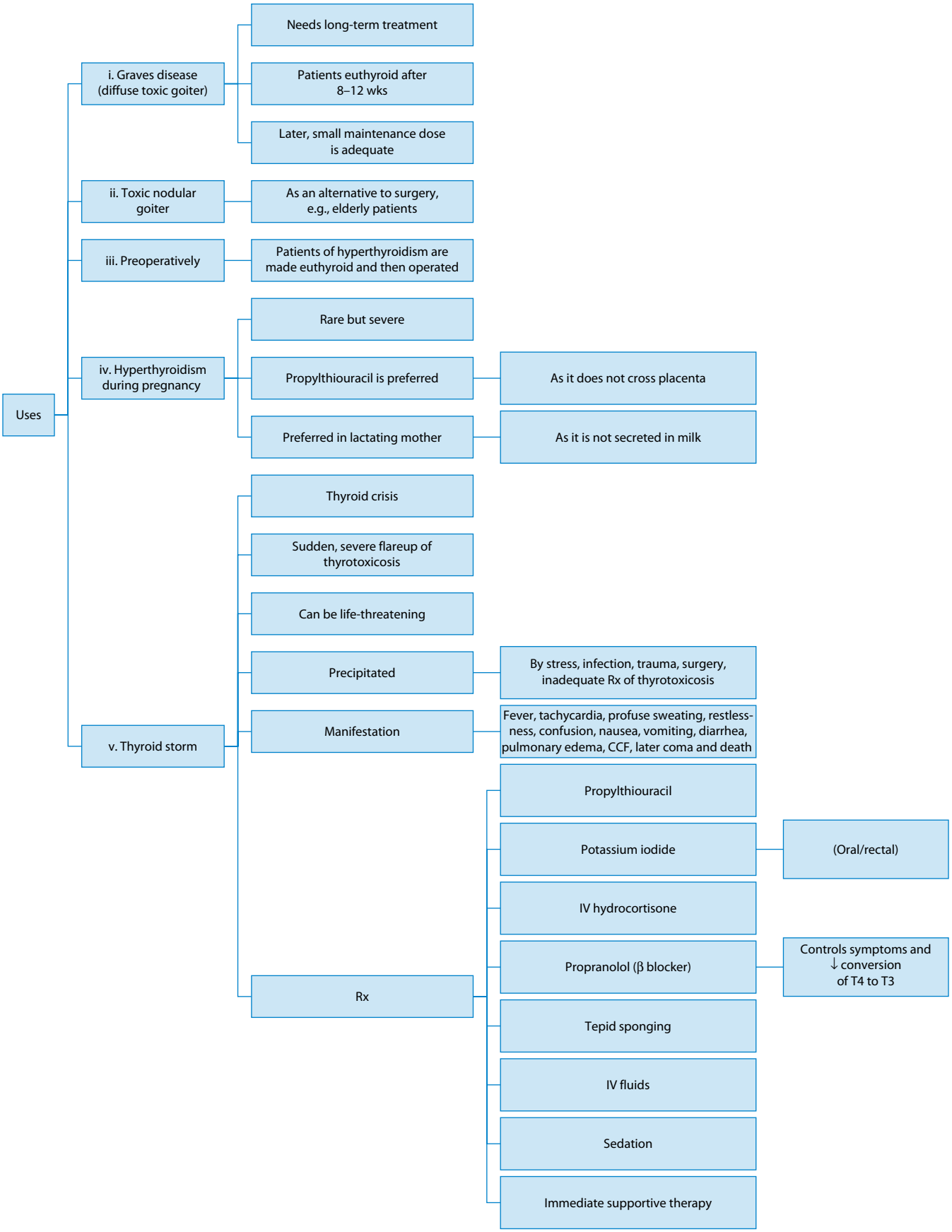


49.4 THIOAMIDES (THIOUREA DERIVATIVES)

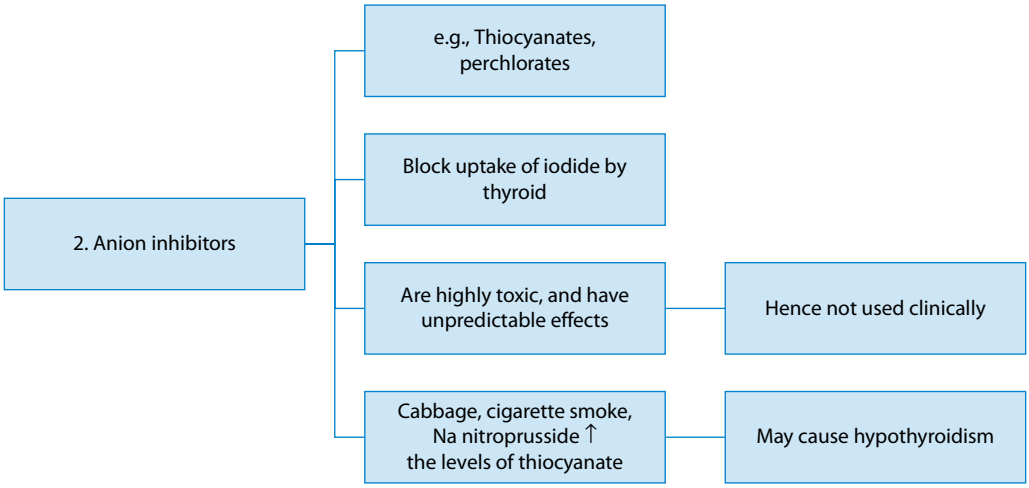


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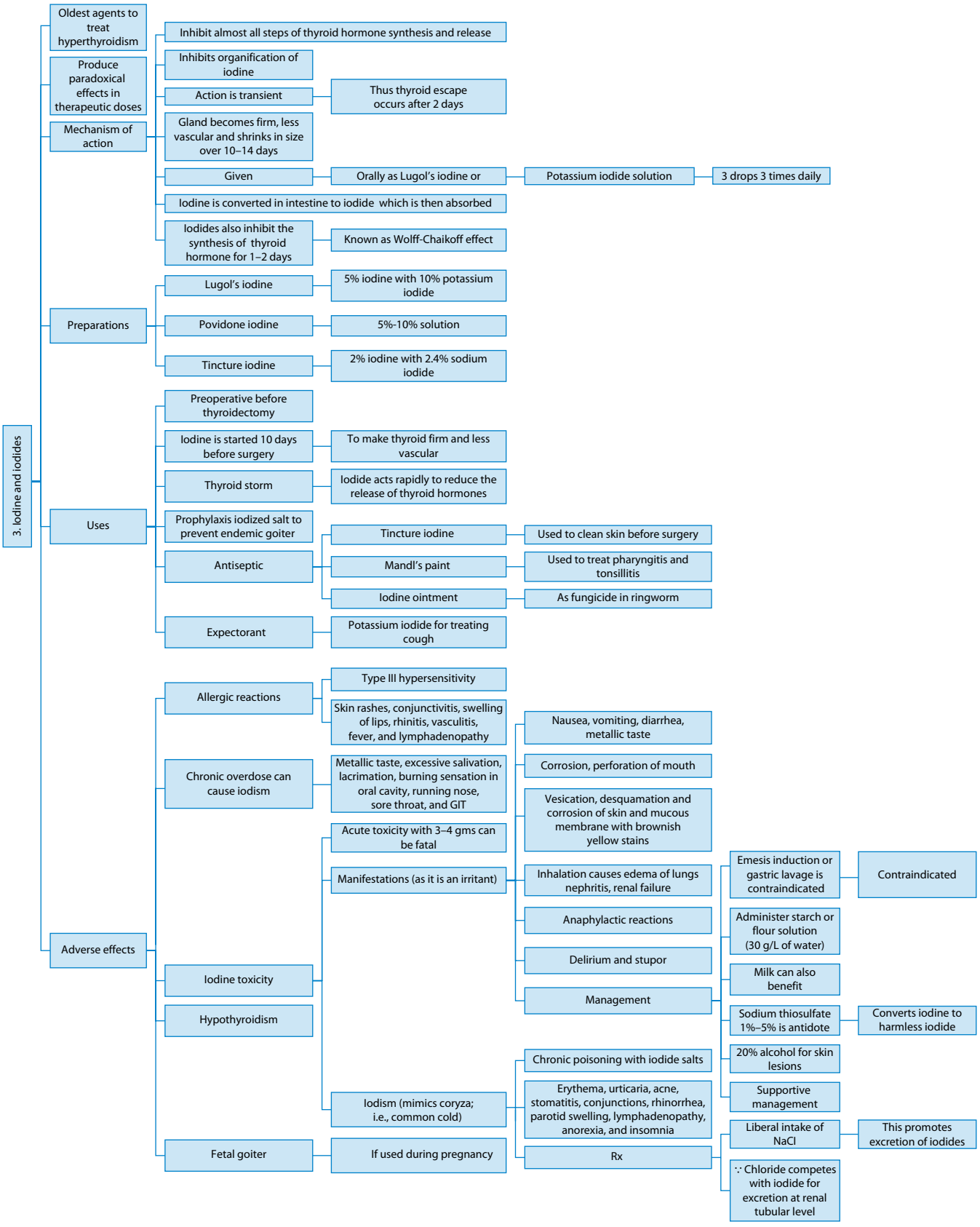
49.4 THIOAMIDES (THIOUREA DERIVATIVES) – USES (Continued)



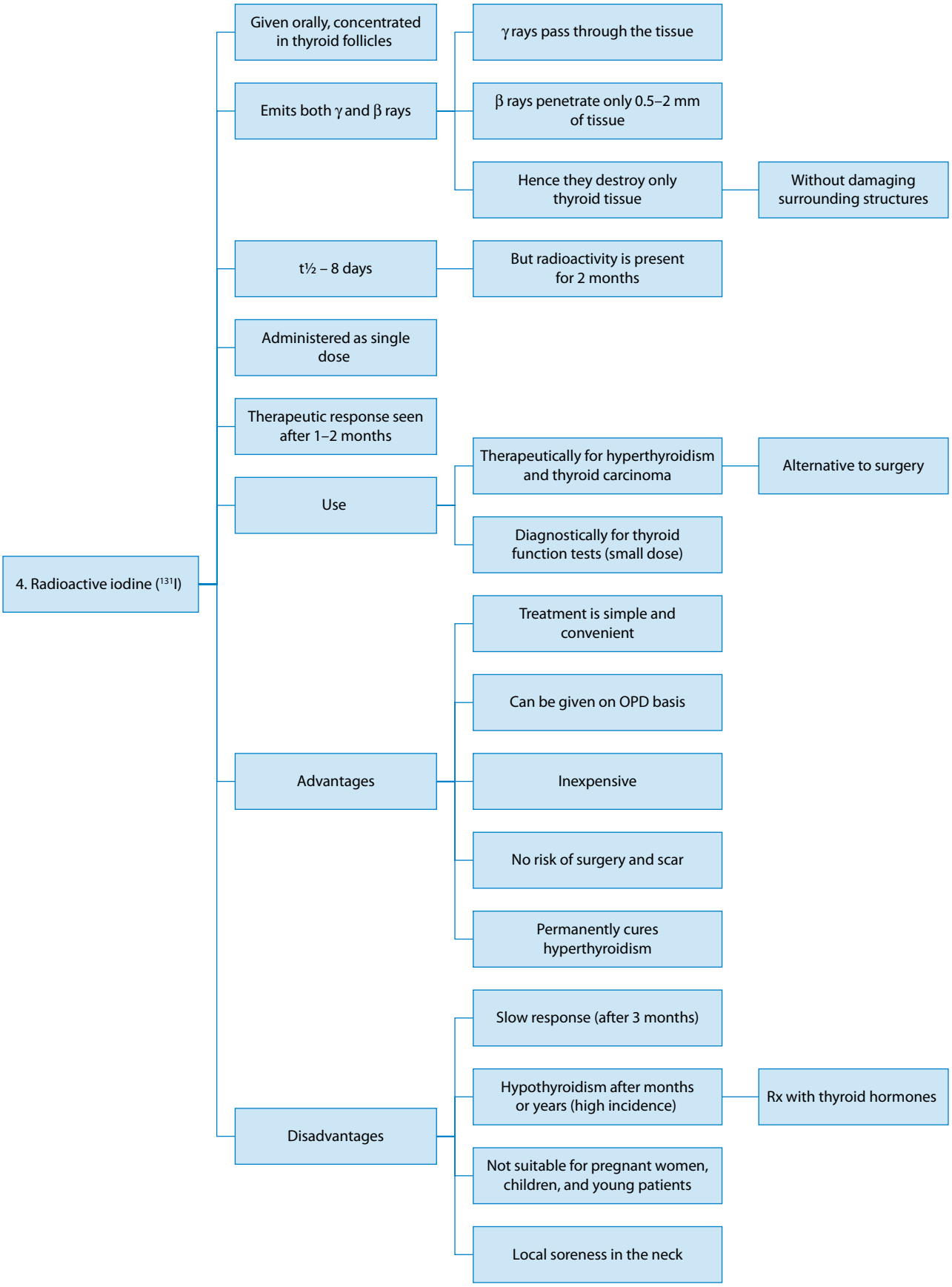
49.5 ANION INHIBITORS



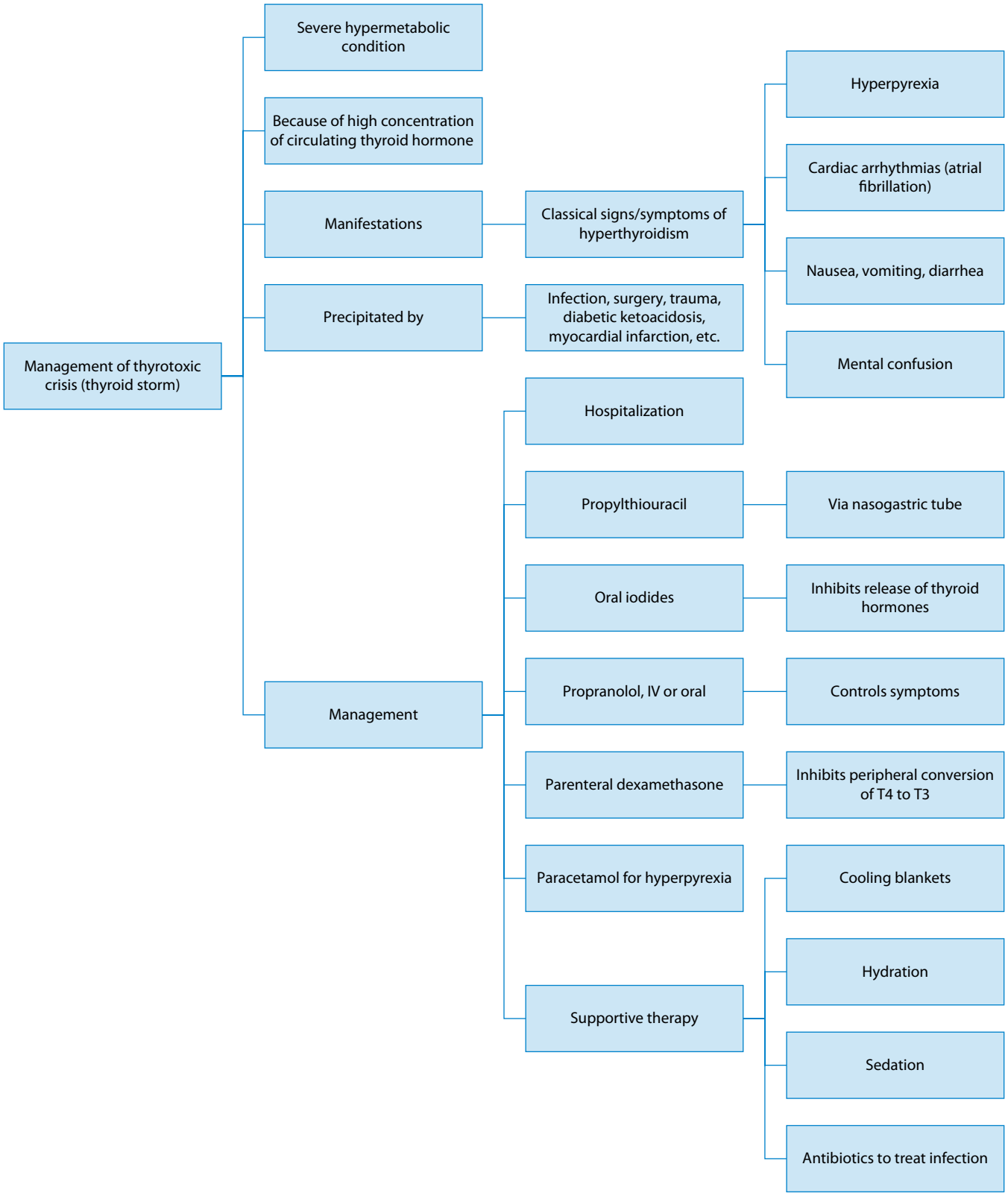
49.6 IODINE AND IODIDES



49.7 RADIOACTIVE IODINE (^{131}I)



49.8 MANAGEMENT OF THYROTOXIC CRISIS (THYROID STORM)



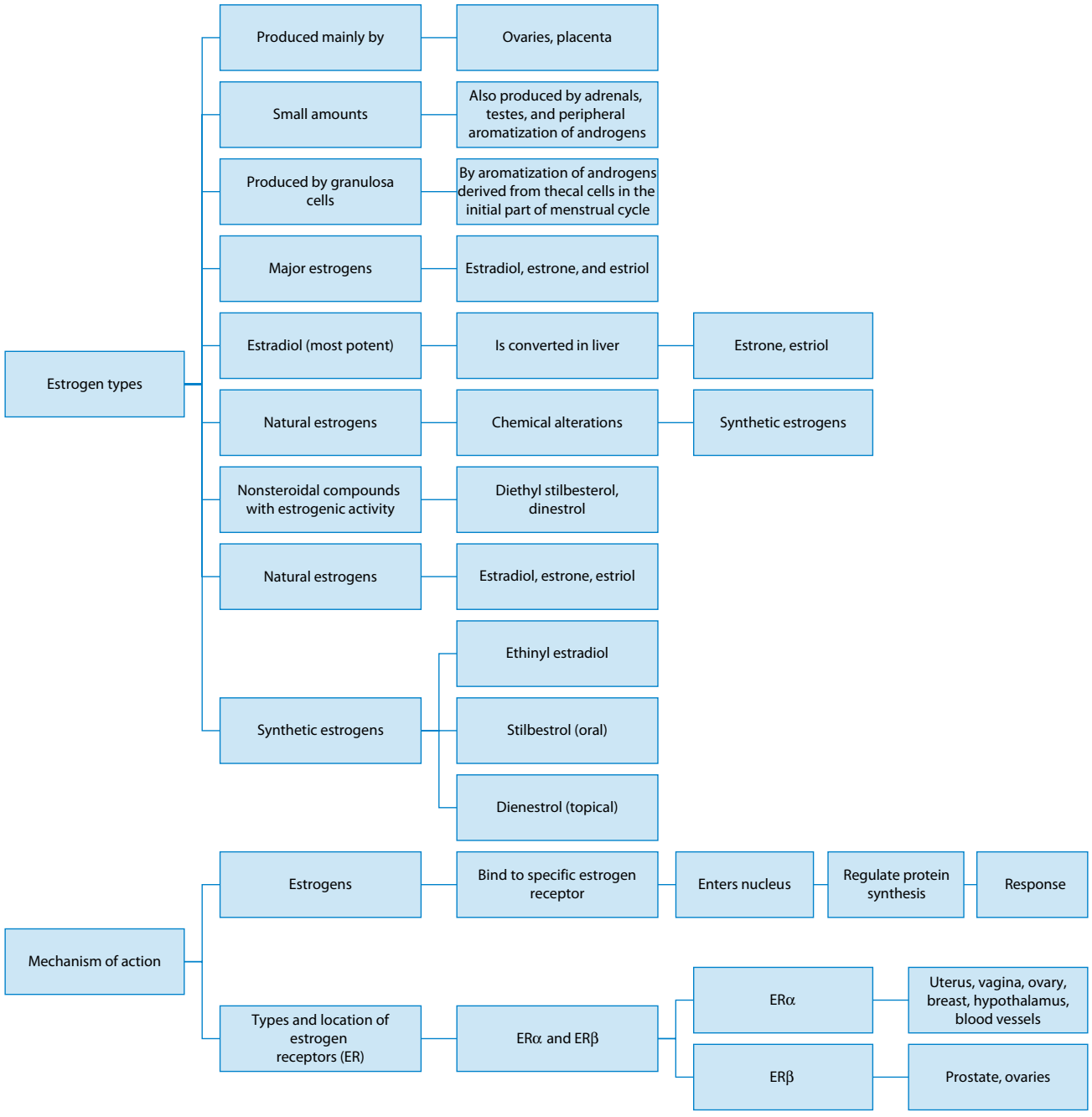
49.9 DIFFERENCES BETWEEN PROPYLTHIOURACIL AND METHIMAZOLE (CARBIMAZOLE)

Features	Propylthiouracil	Methimazole
i. Onset	Fast	Slow
ii. Potency	Less	More
iii. t½	1–2 h	6 h
iv. Protein binding	High	Low
v. Duration of action	Short (4–8 h)	Long (12–24 h)
vi. Placenta transfer	Negligible	Easy
vii. Secretion in milk	Negligible	Significant
viii. Pregnancy	Preferred	Not Preferred
ix. Lactation	Preferred	Not Preferred
x. Dose	Tid – qid	Od – bid

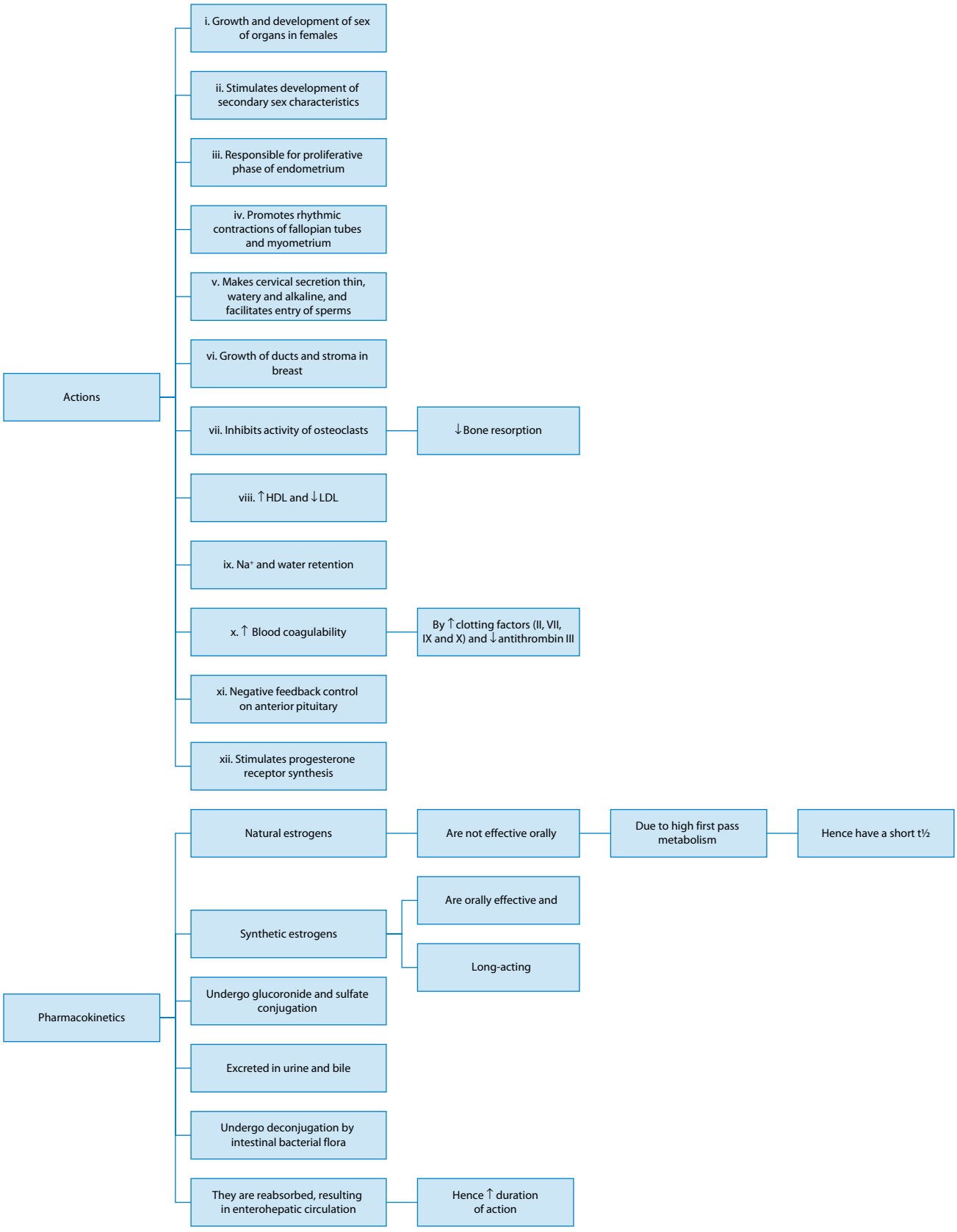
50

Estrogen, progestins, and hormonal contraceptives

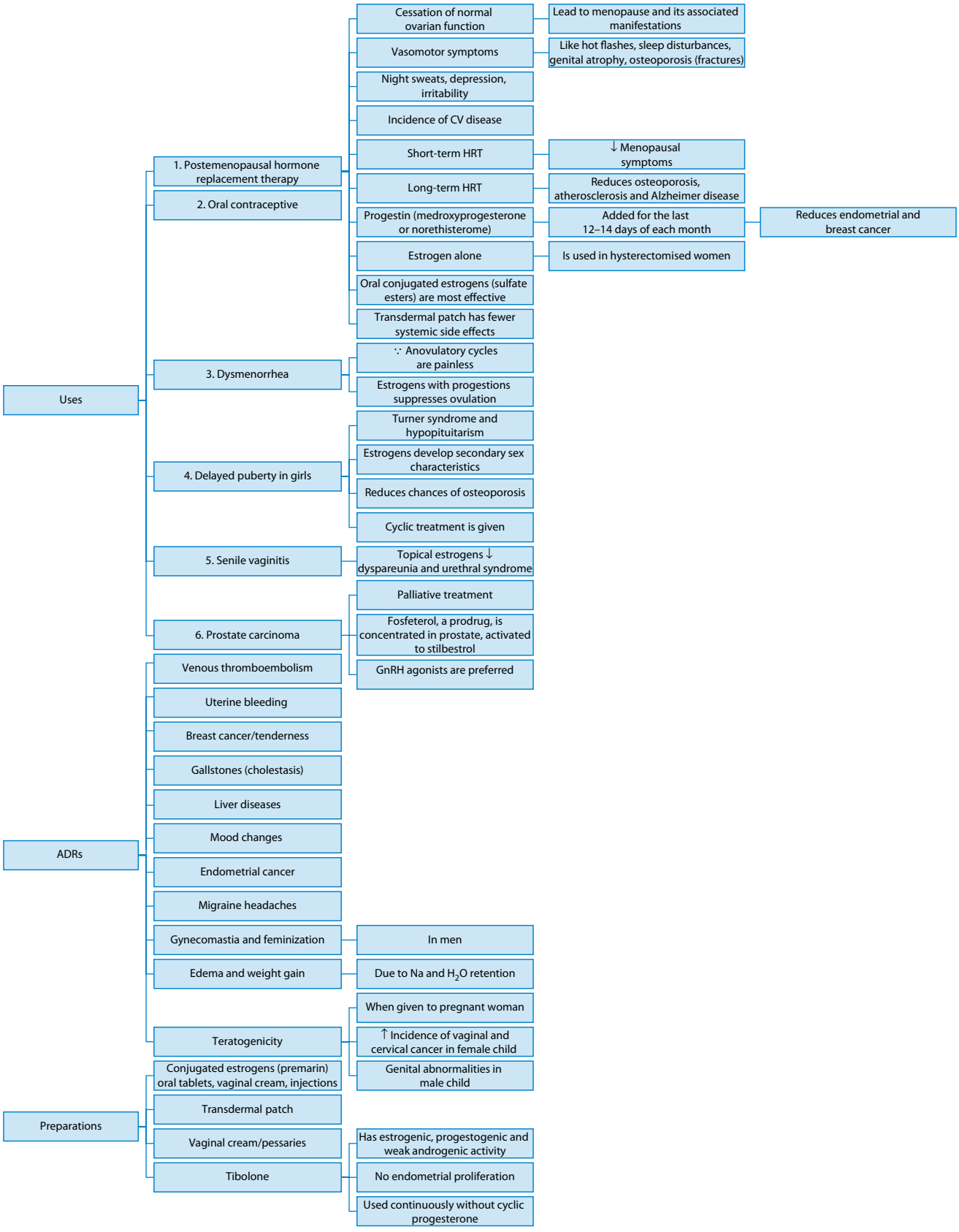
50.1 ESTROGENS – TYPES AND MECHANISM OF ACTION



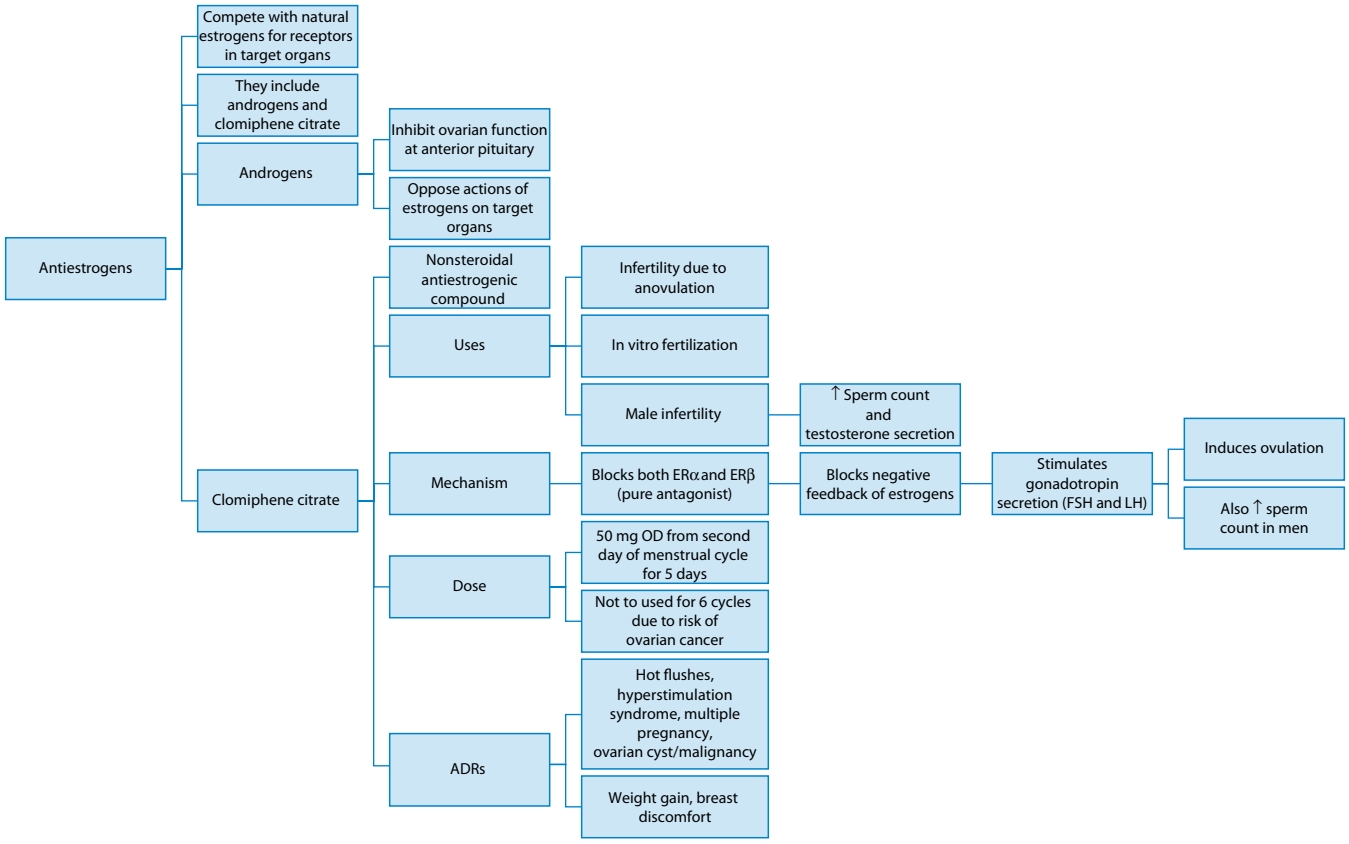
50.2 ACTIONS AND PHARMACOKINETICS



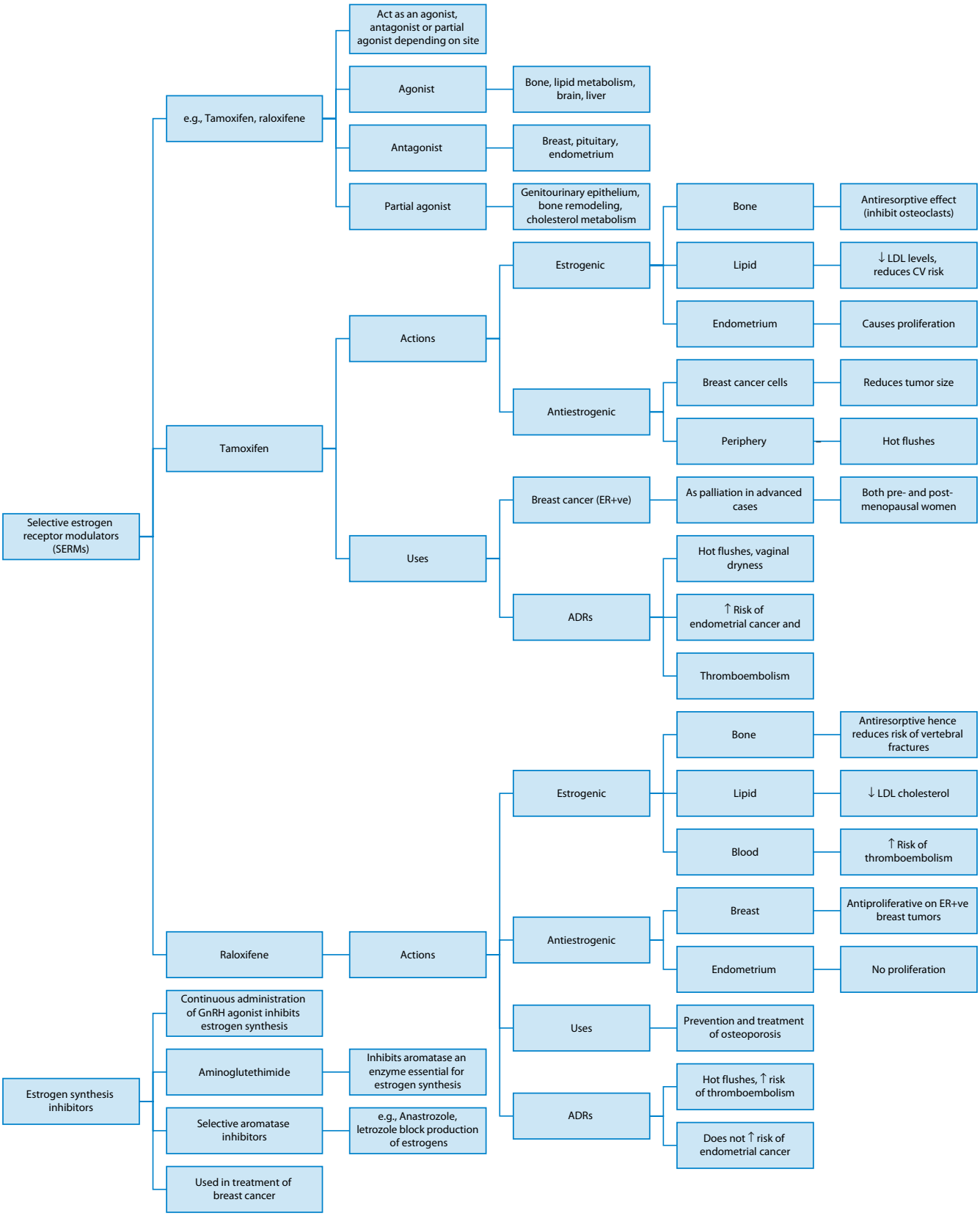
50.3 USES, ADRs, AND PREPARATIONS



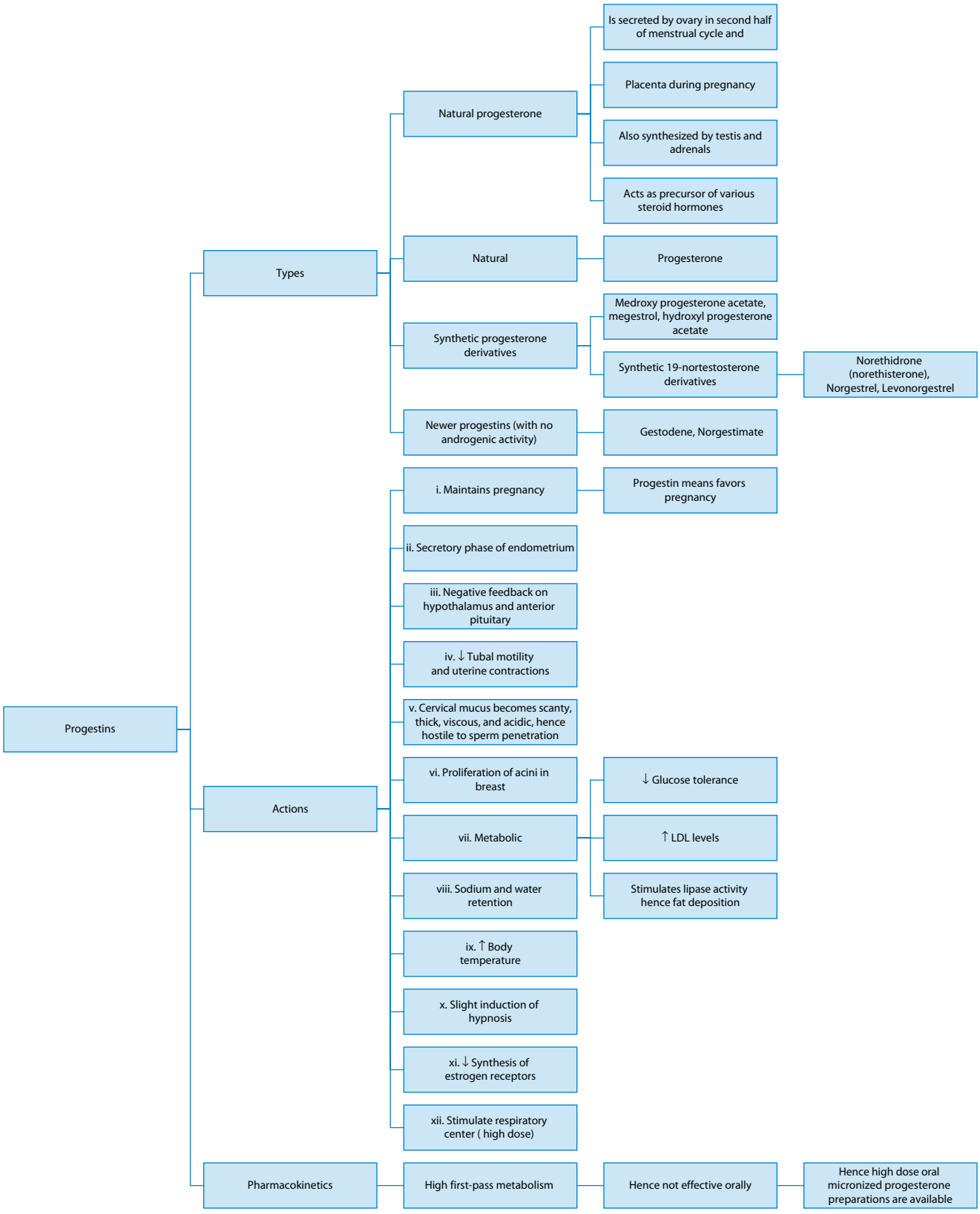
50.4 ANTIESTROGENS



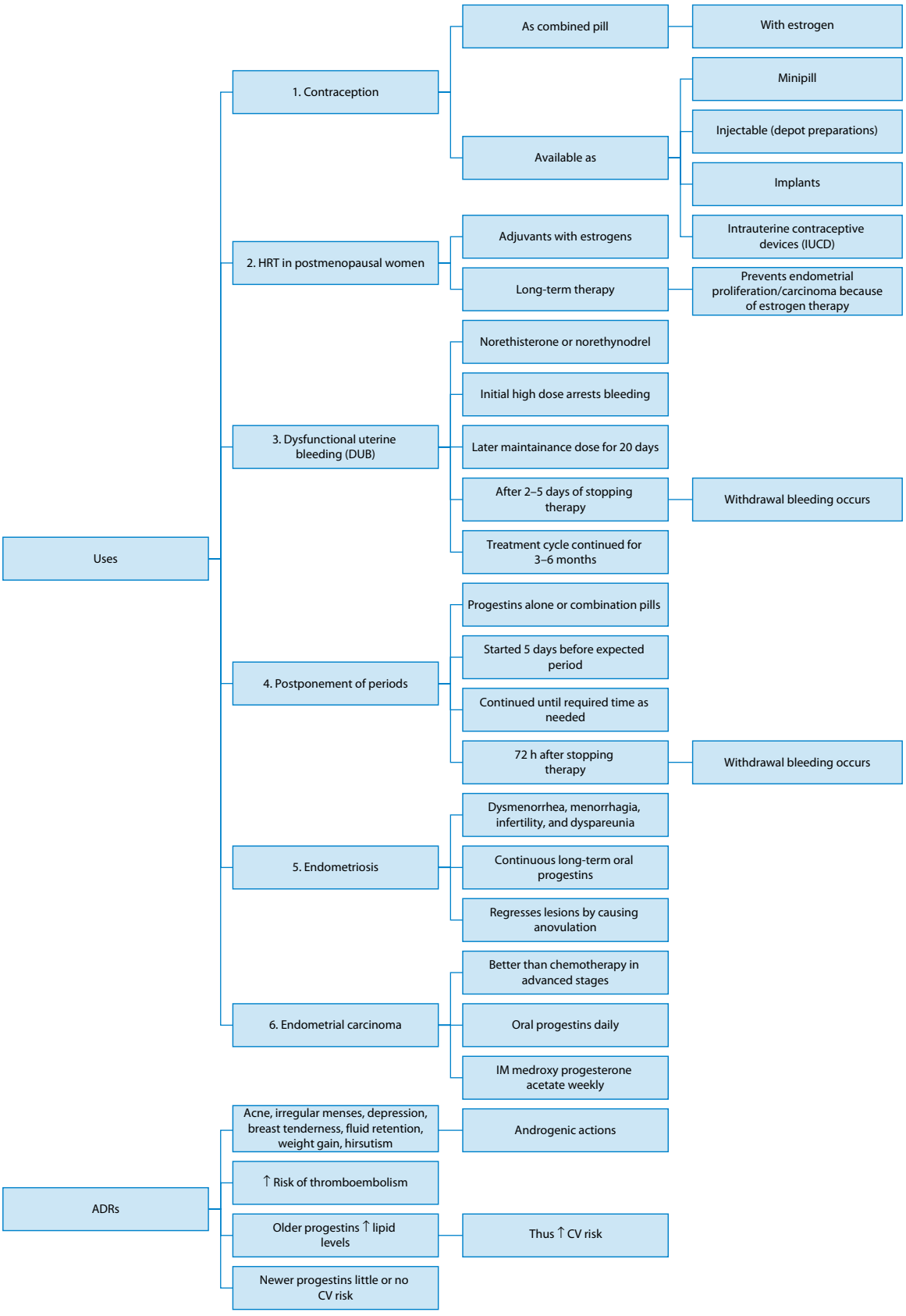
50.5 SELECTIVE ESTROGEN RECEPTOR MODULATORS (SERMs) AND ESTROGEN SYNTHESIS INHIBITORS



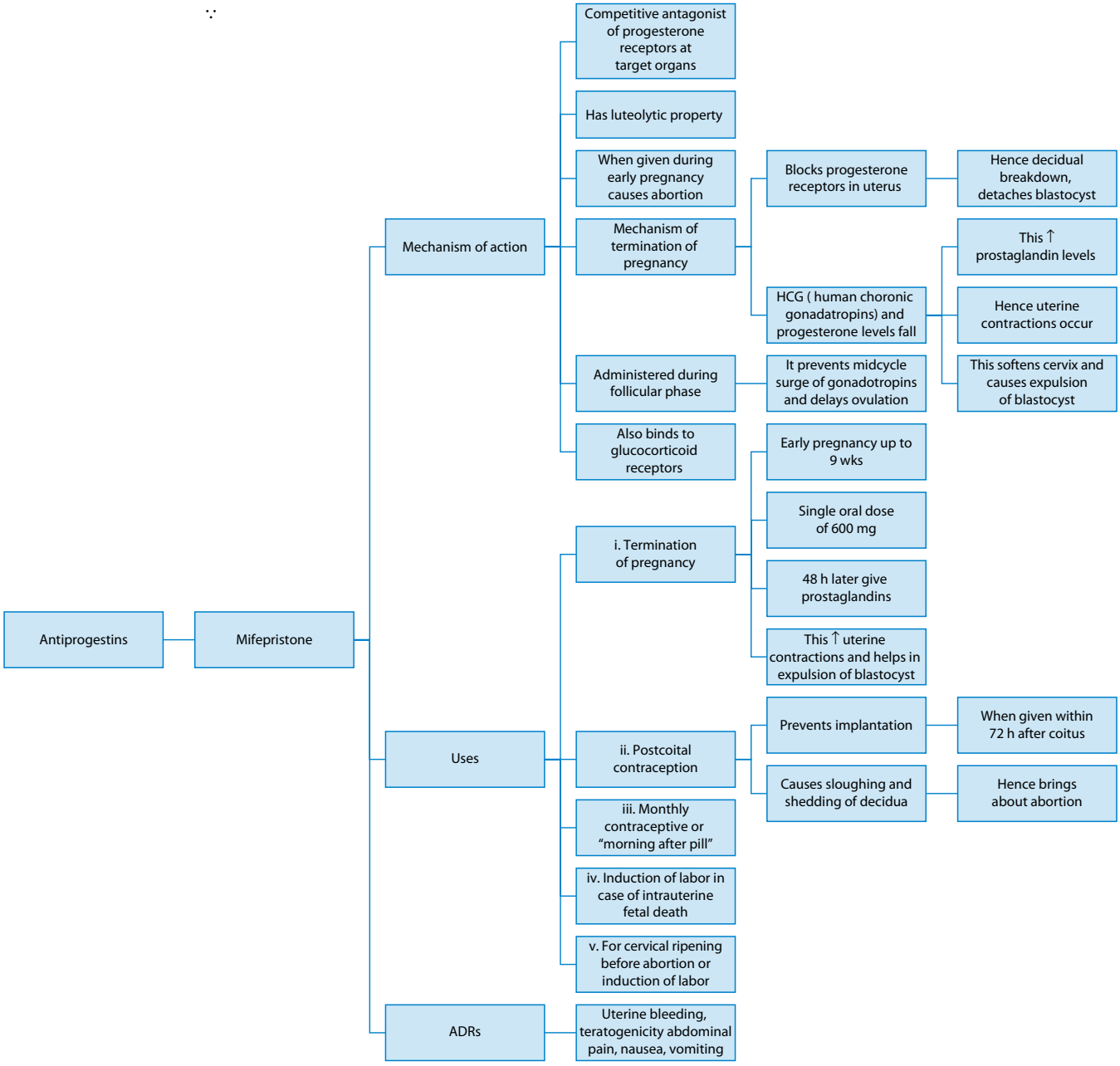
50.6 PROGESTINS – TYPES, ACTIONS, AND PHARMACOKINETICS



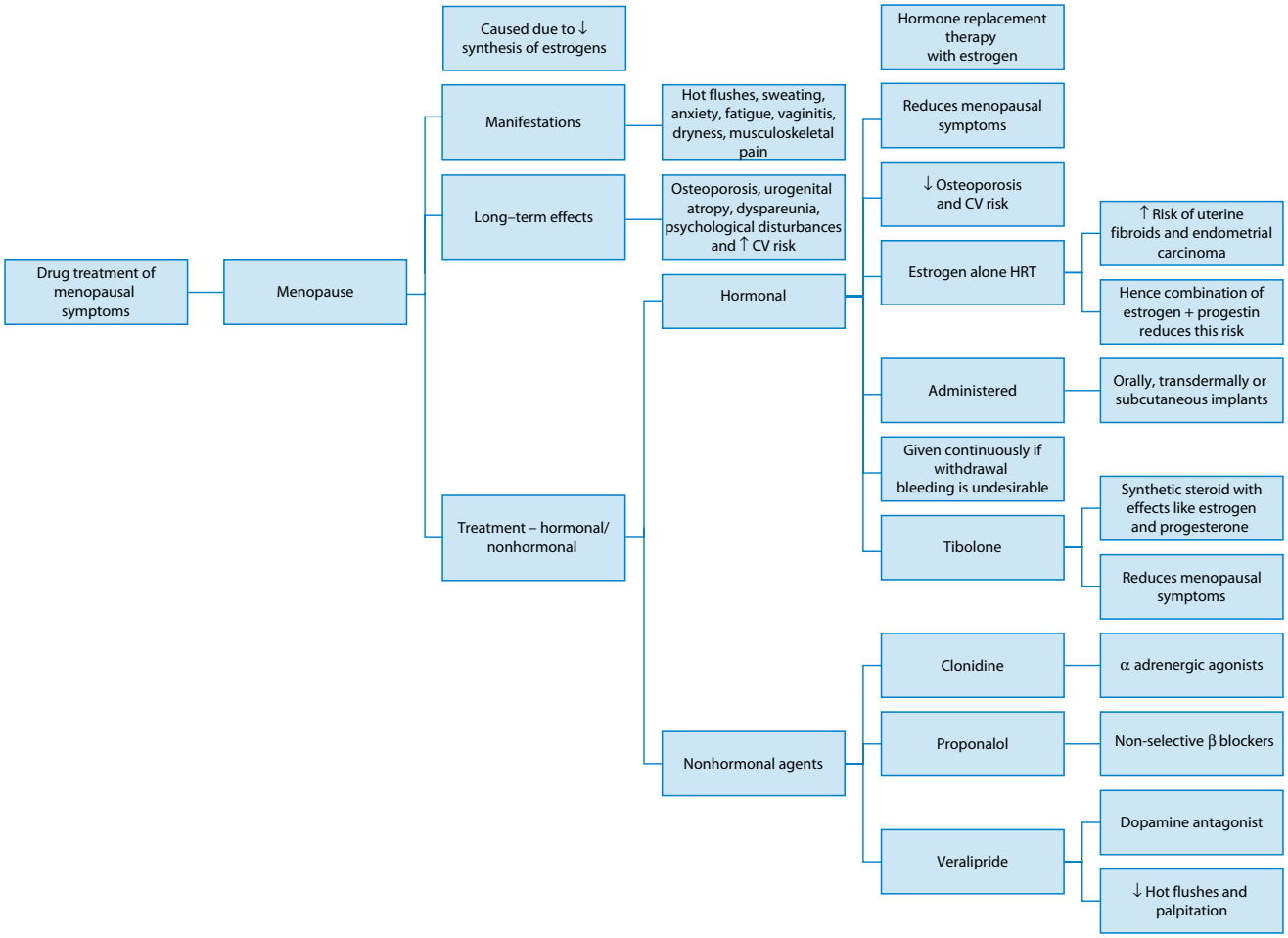
50.7 USES AND ADRs OF PROGESTINS



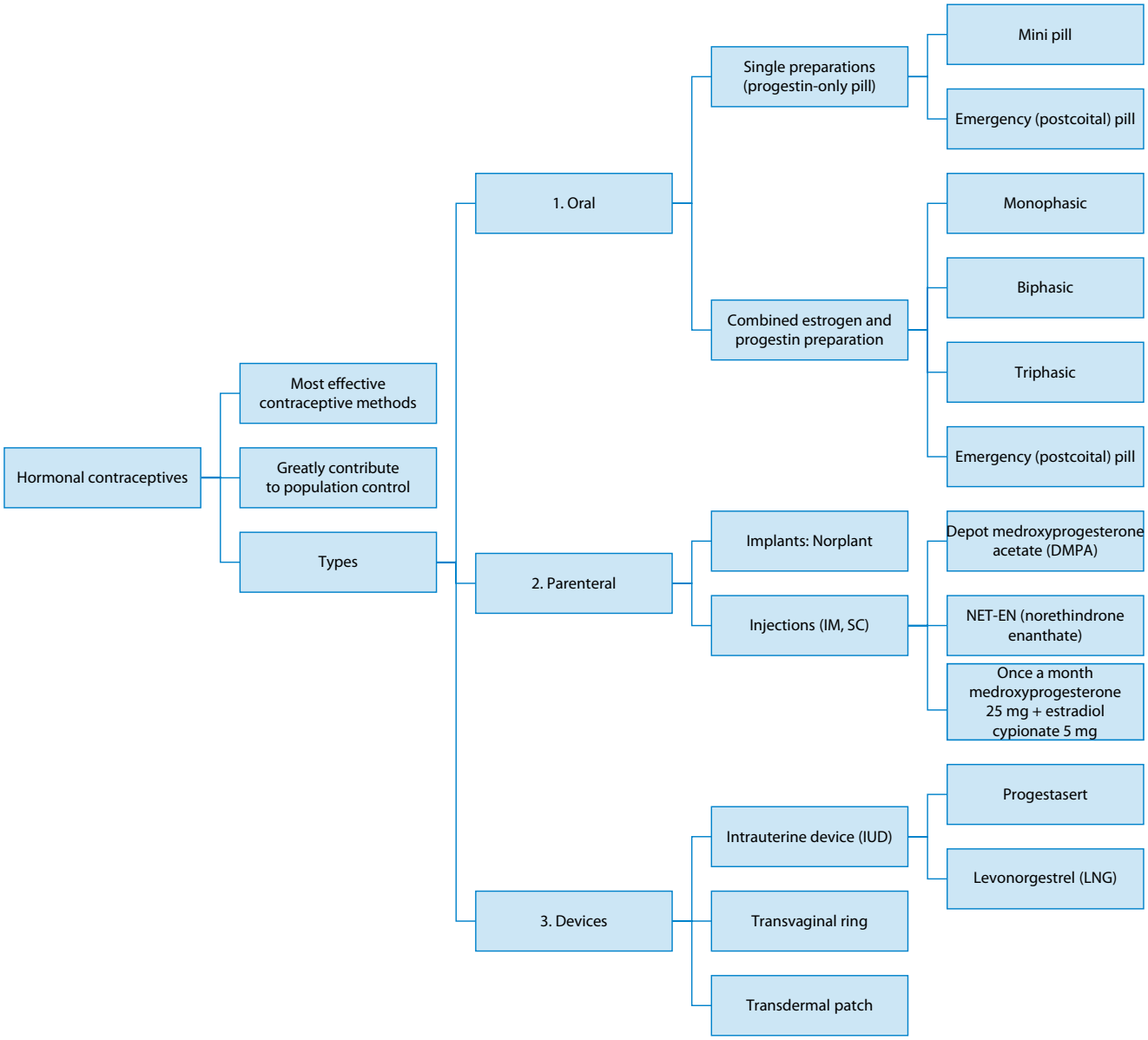
50.8 ANTIPROGESTINS



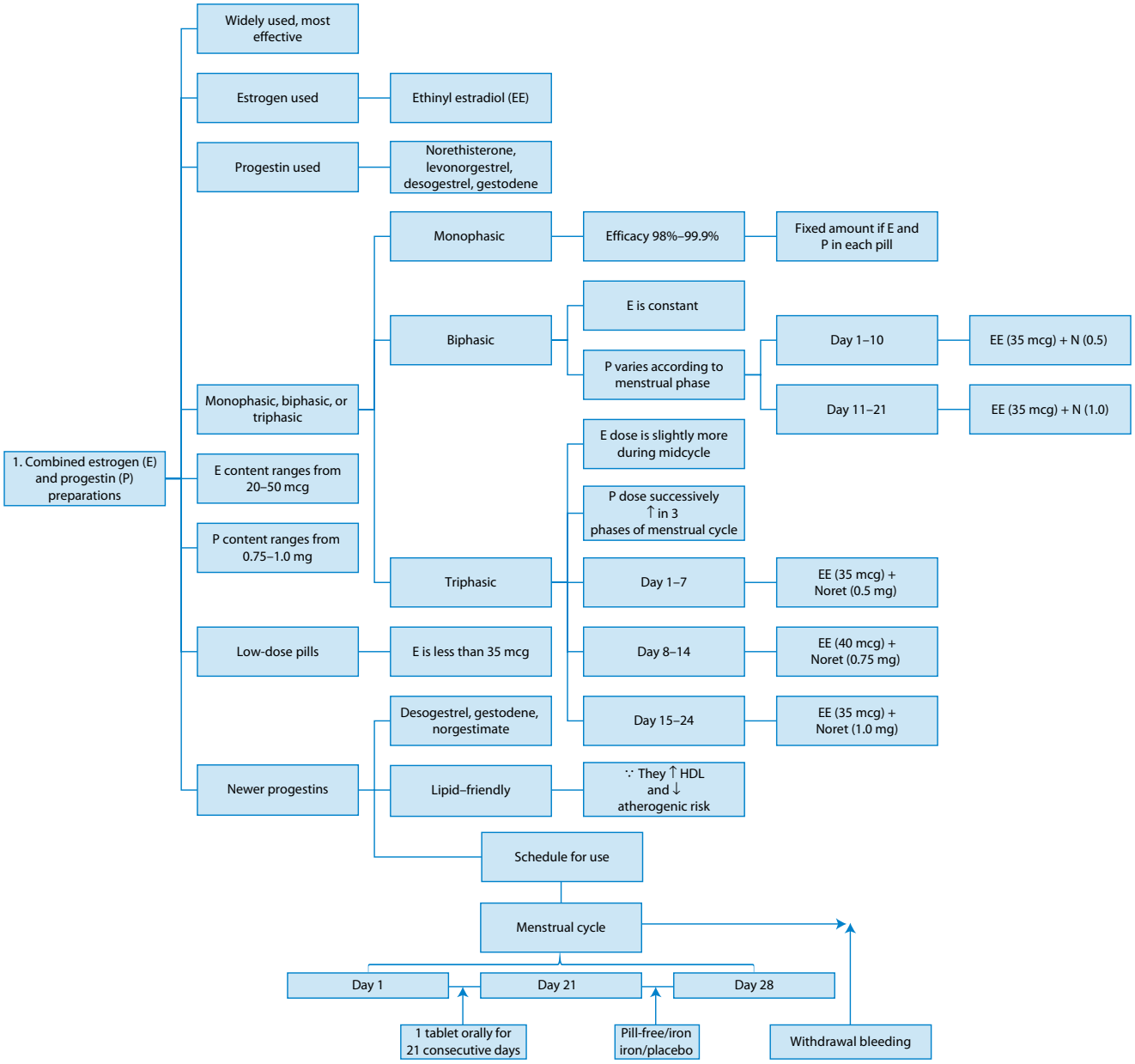
50.9 DRUG TREATMENT OF MENOPAUSAL SYMPTOMS



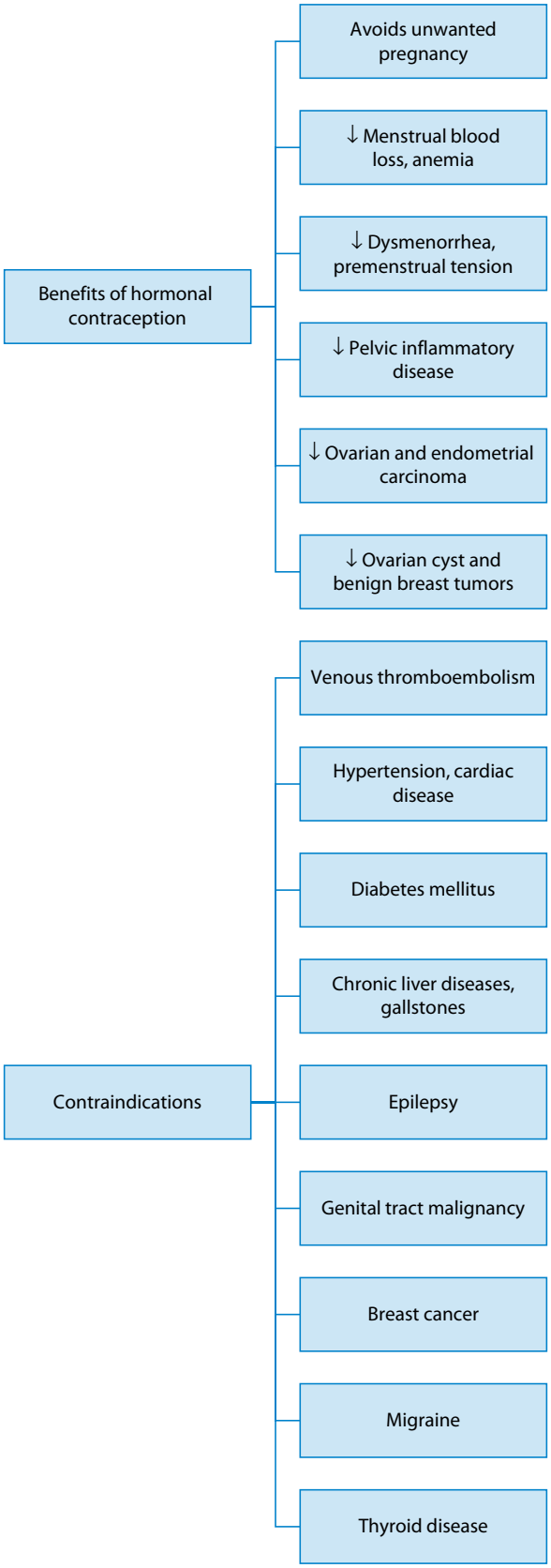
50.10 TYPES OF HORMONAL CONTRACEPTIVES



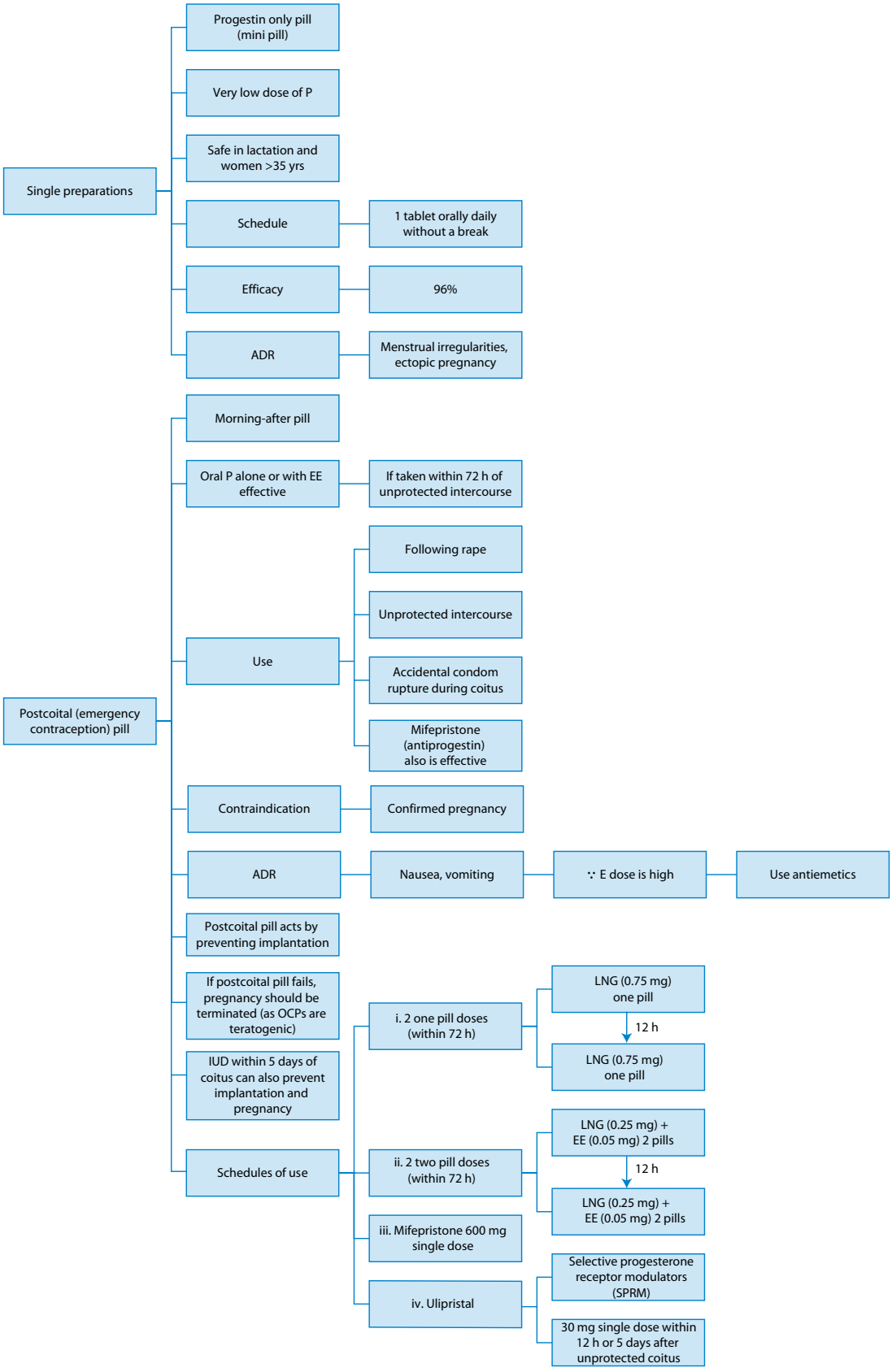
50.11 COMBINED ESTROGEN (E) AND PROGESTIN (P) PREPARATIONS



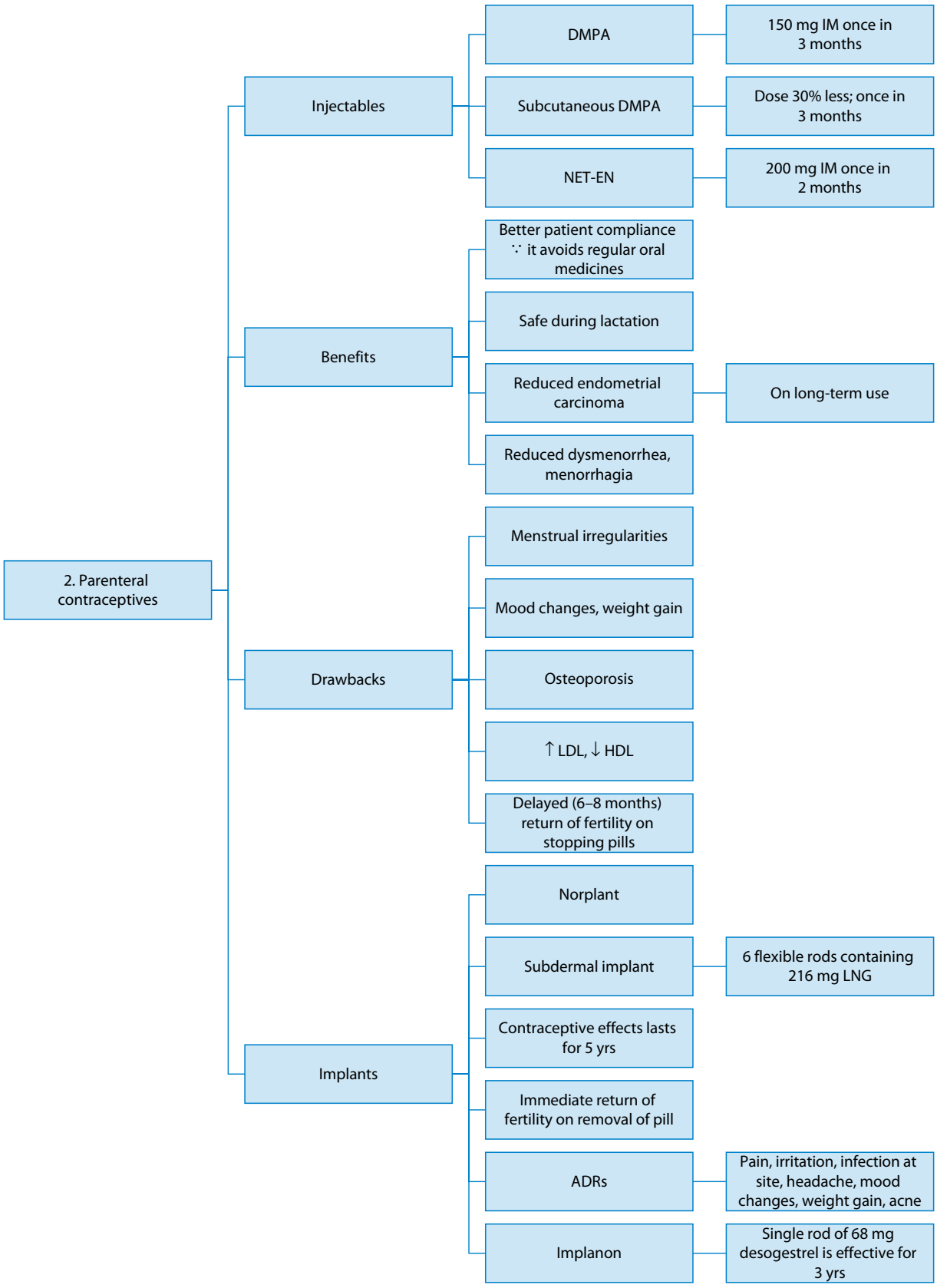
50.12 BENEFITS OF HORMONAL CONTRACEPTION AND CONTRAINDICATIONS



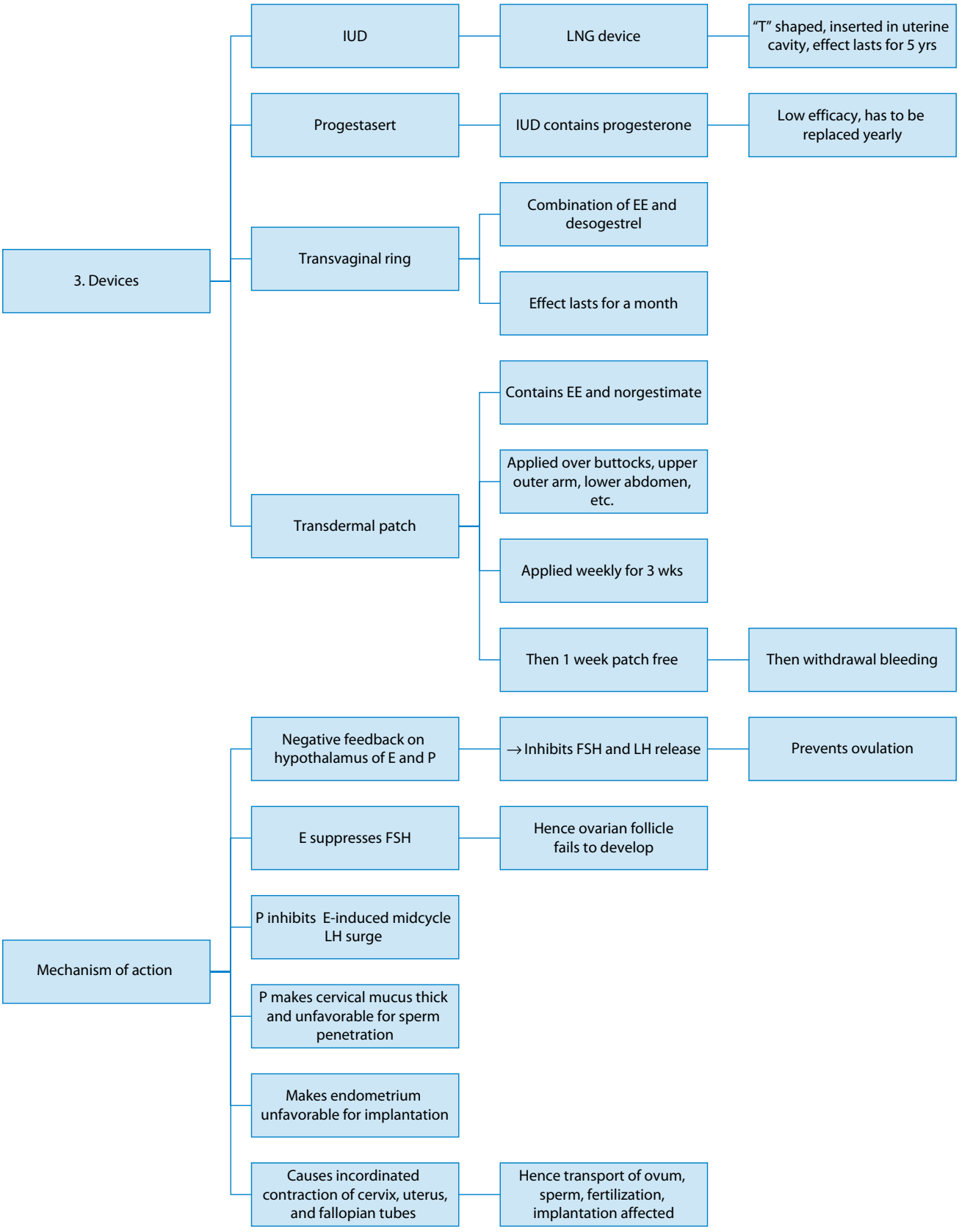
50.13 SINGLE PREPARATIONS AND POSTCOITAL (EMERGENCY CONTRACEPTION) PILL



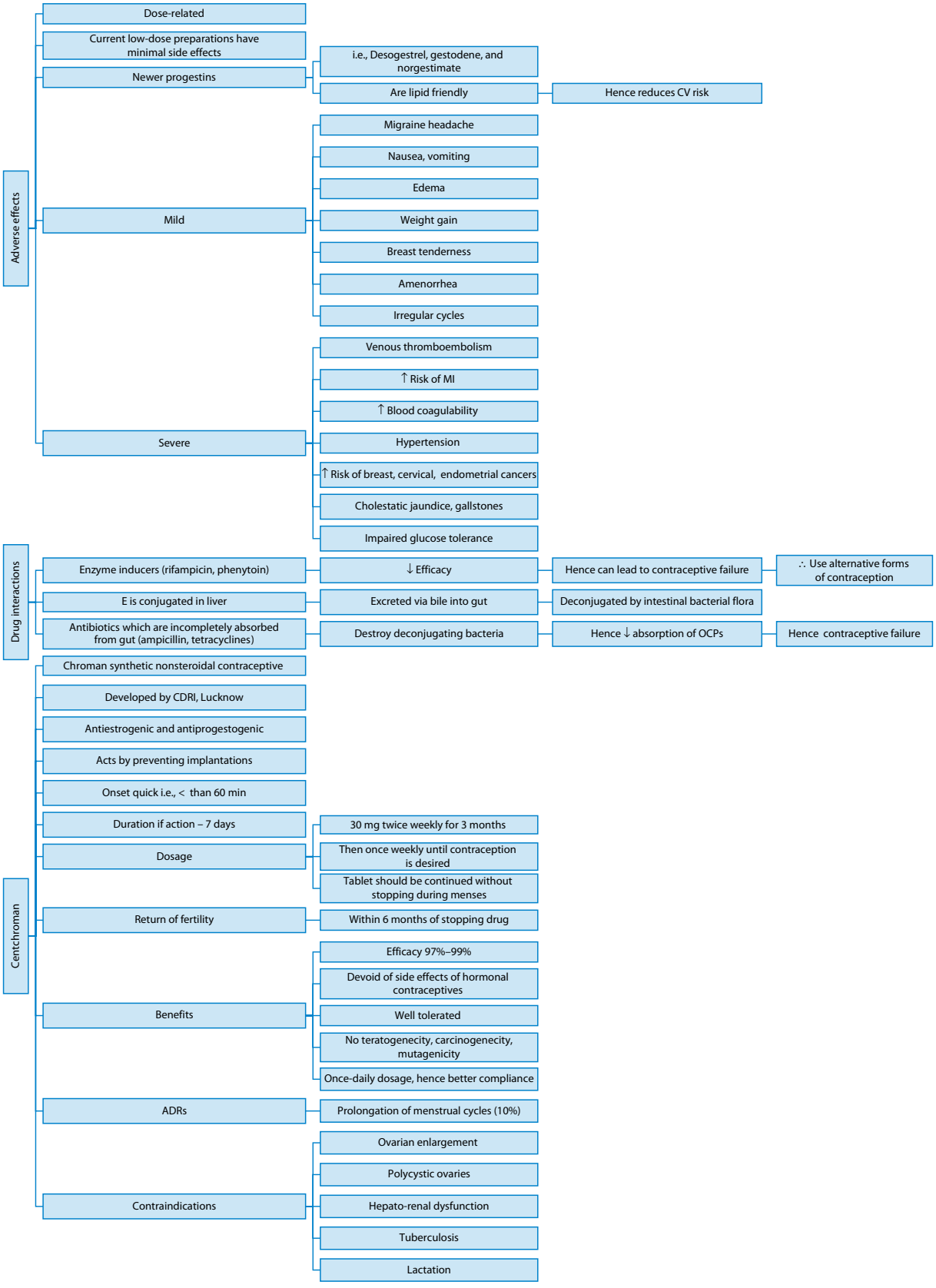
50.14 PARENTERAL CONTRACEPTIVES



50.15 DEVICES AND MECHANISM OF ACTION OF CONTRACEPTIVES



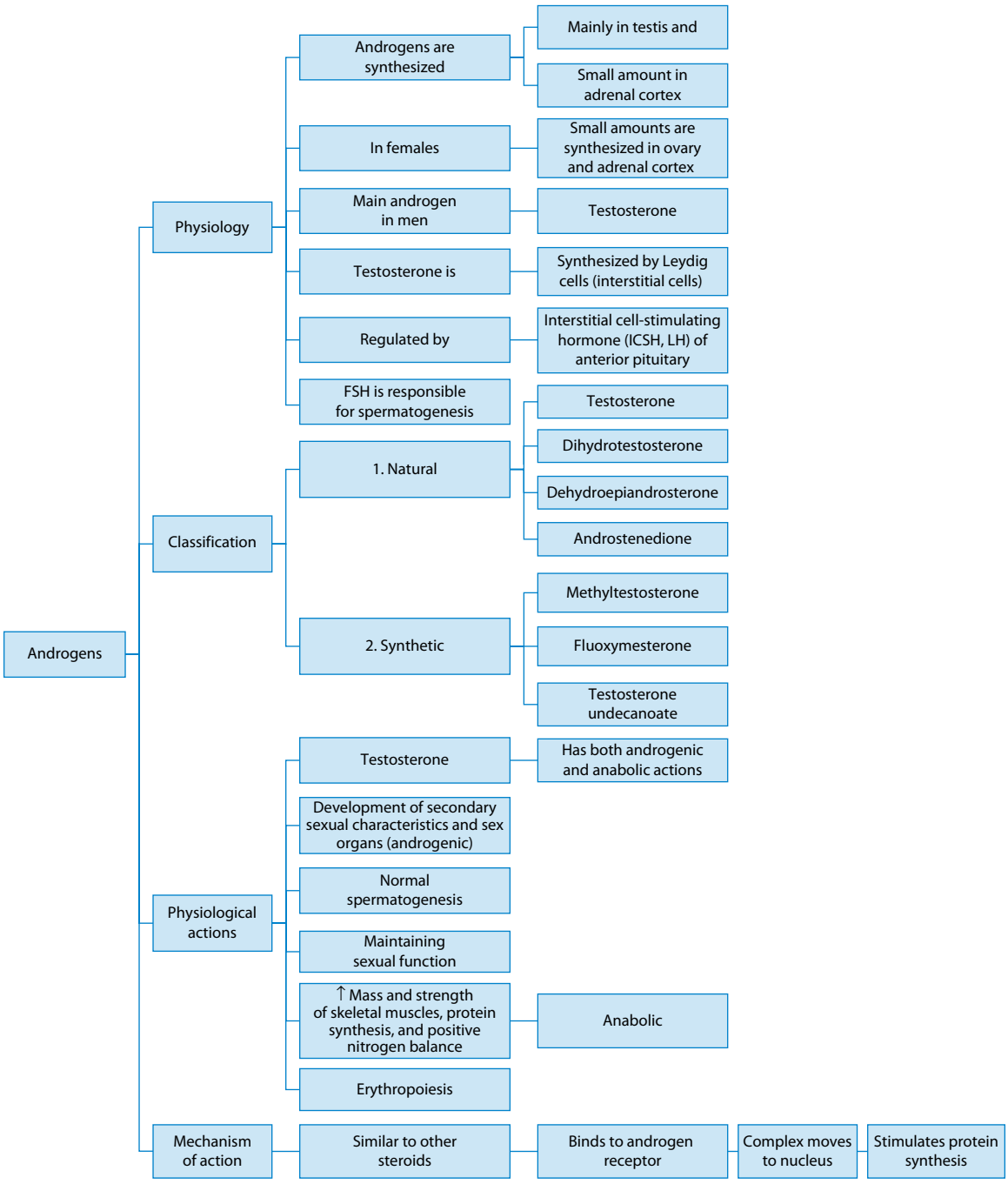
50.16 ADVERSE EFFECTS, DRUG-INTERACTIONS, AND CENTCHROMAN



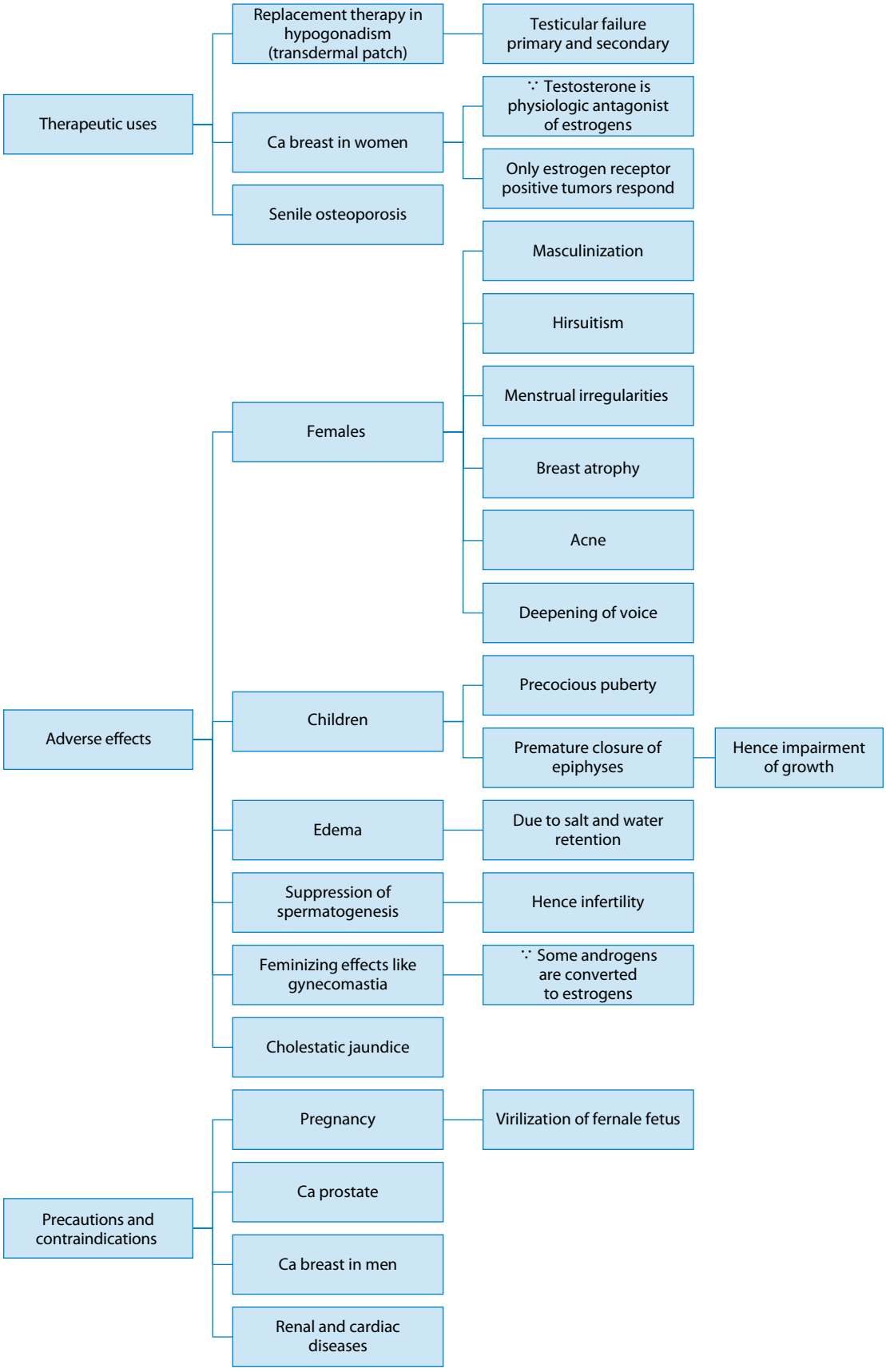
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Androgens and anabolic steroids

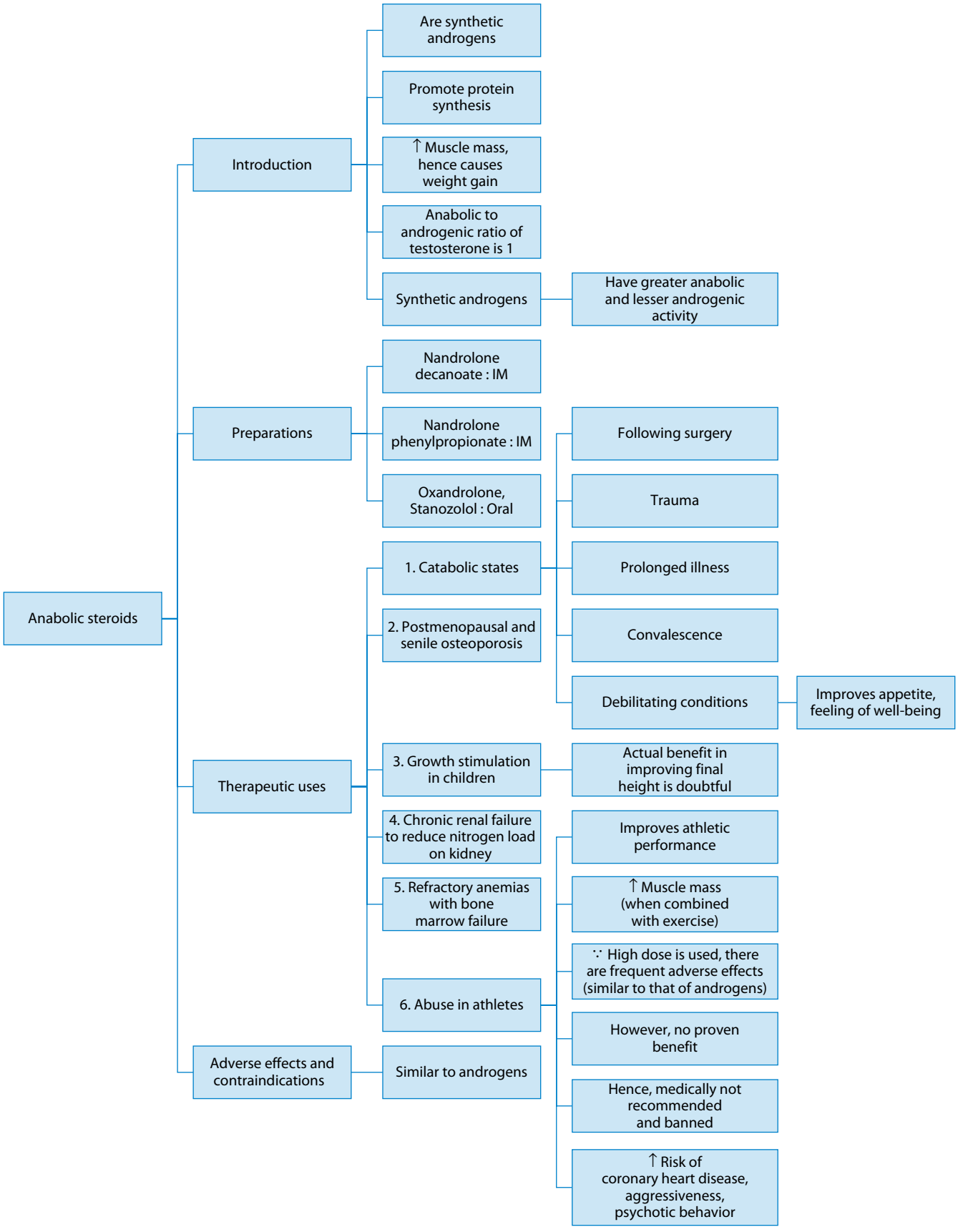
51.1 ANDROGENS – PHYSIOLOGY, CLASSIFICATION, ACTIONS, AND MECHANISM OF ACTION



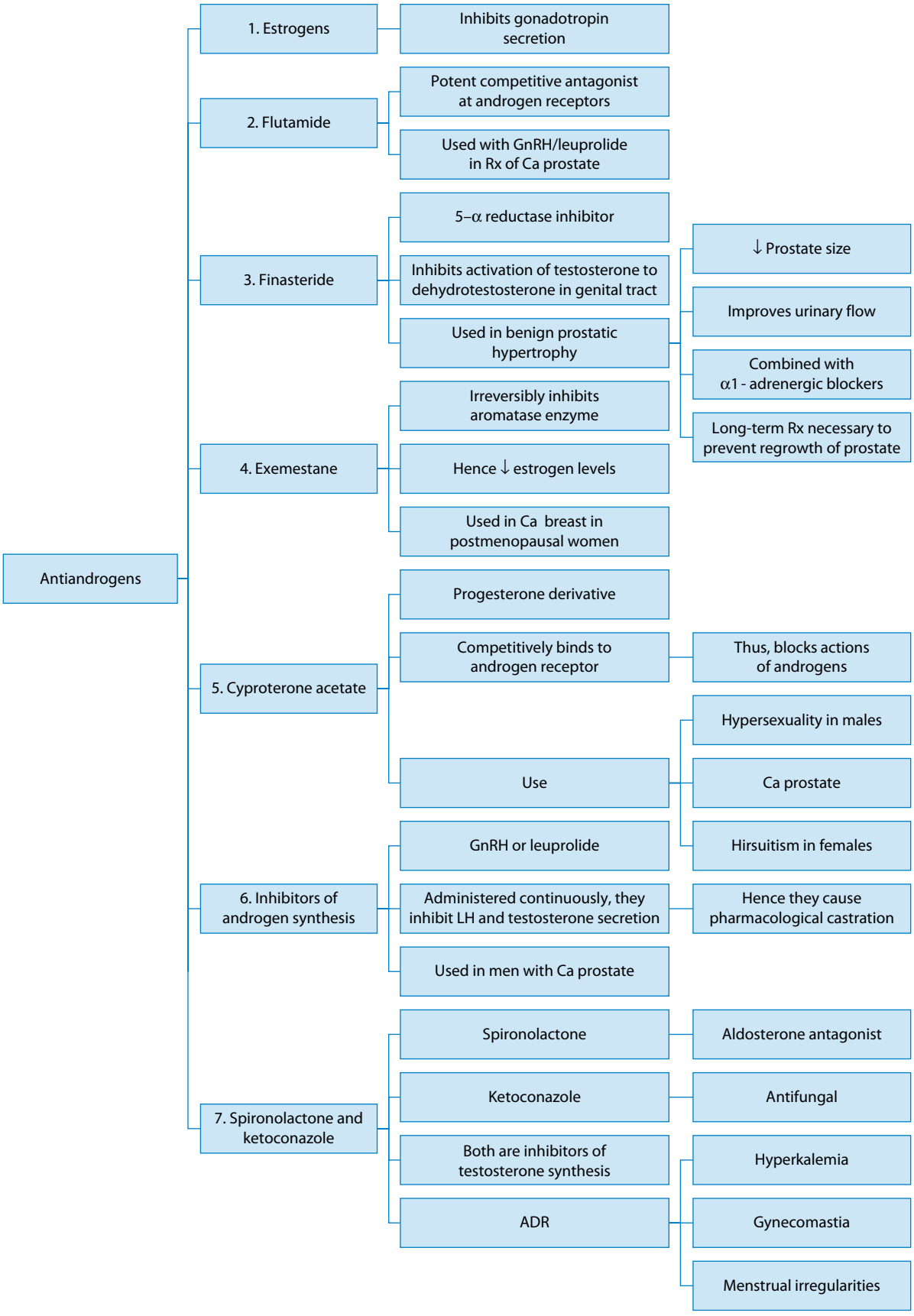
51.2 THERAPEUTIC USES, ADVERSE EFFECTS, AND PRECAUTIONS AND CONTRAINDICATIONS



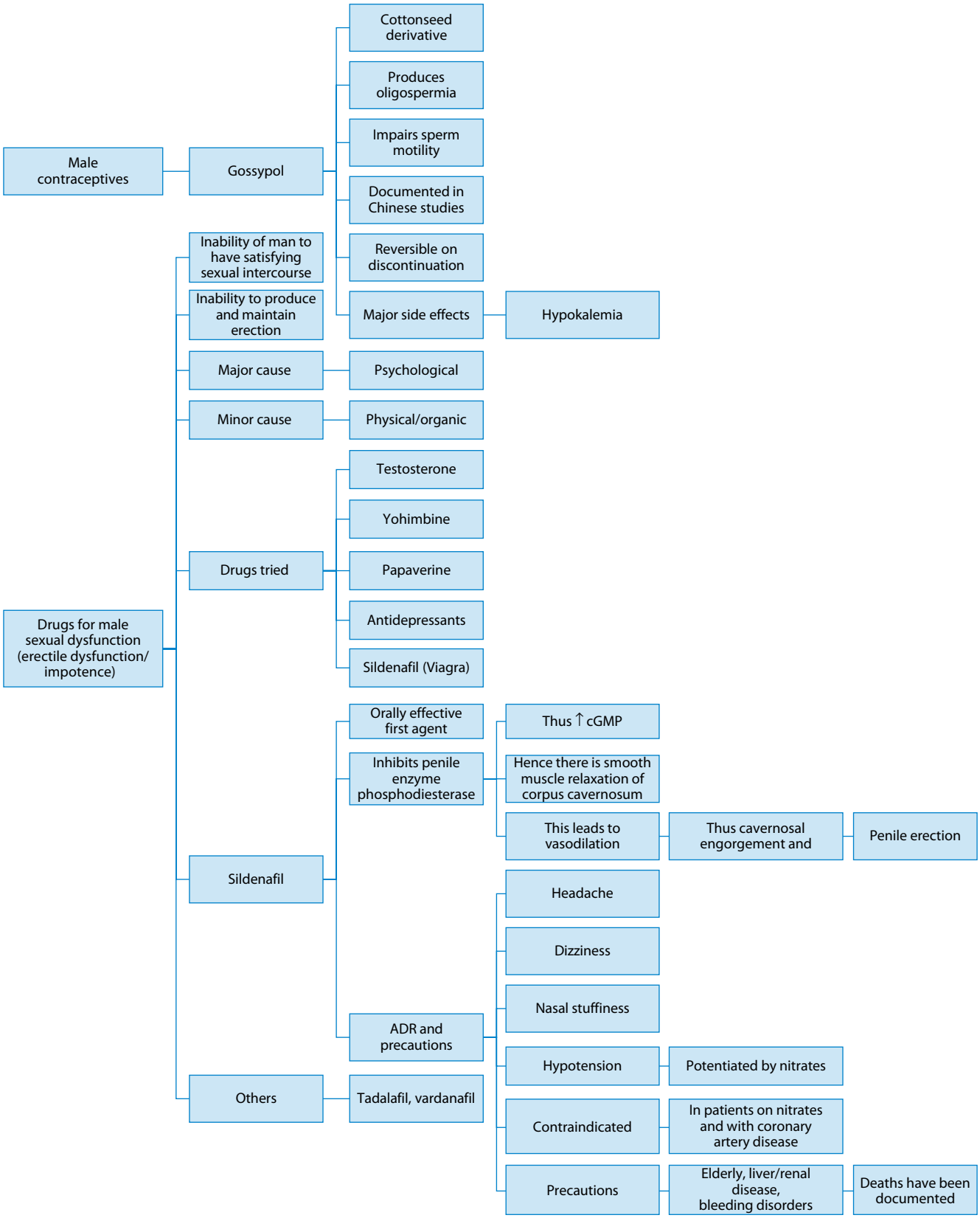
51.3 ANABOLIC STEROIDS



51.4 ANTIANDROGENS



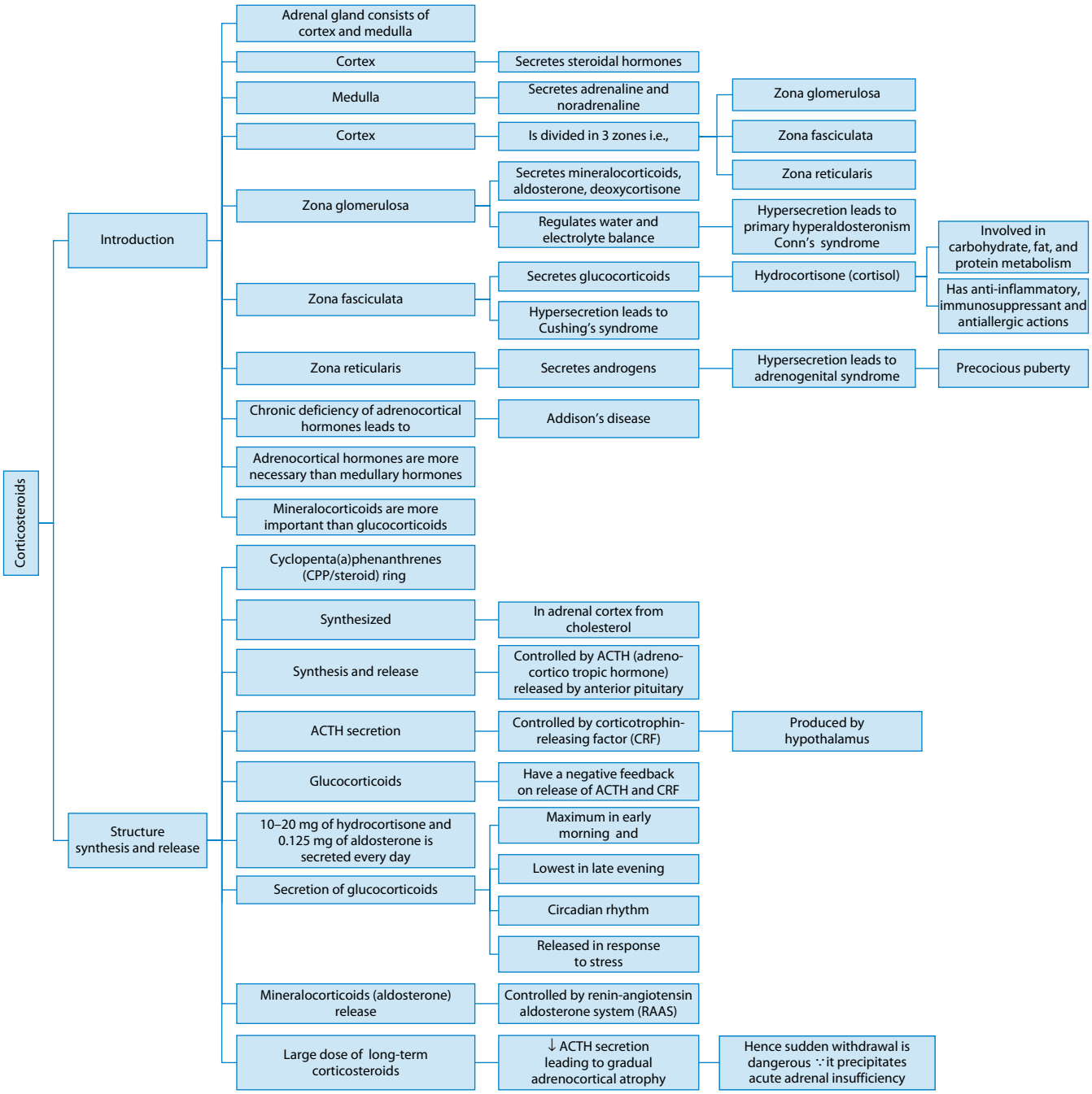
51.5 MALE CONTRACEPTIVES AND DRUGS FOR MALE SEXUAL DYSFUNCTION (ERECTILE DYSFUNCTION/IMPOTENCE)



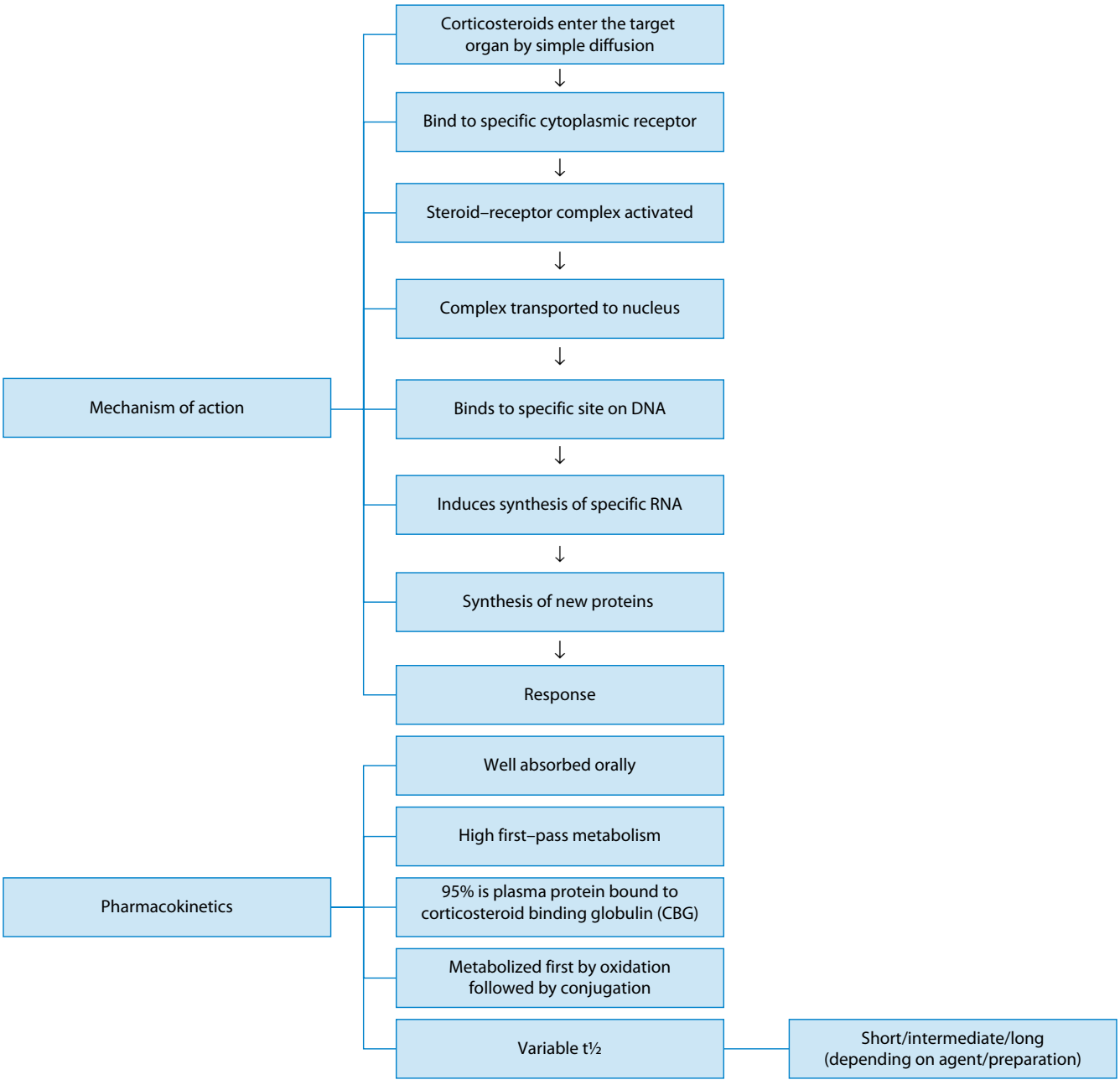
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Corticosteroids

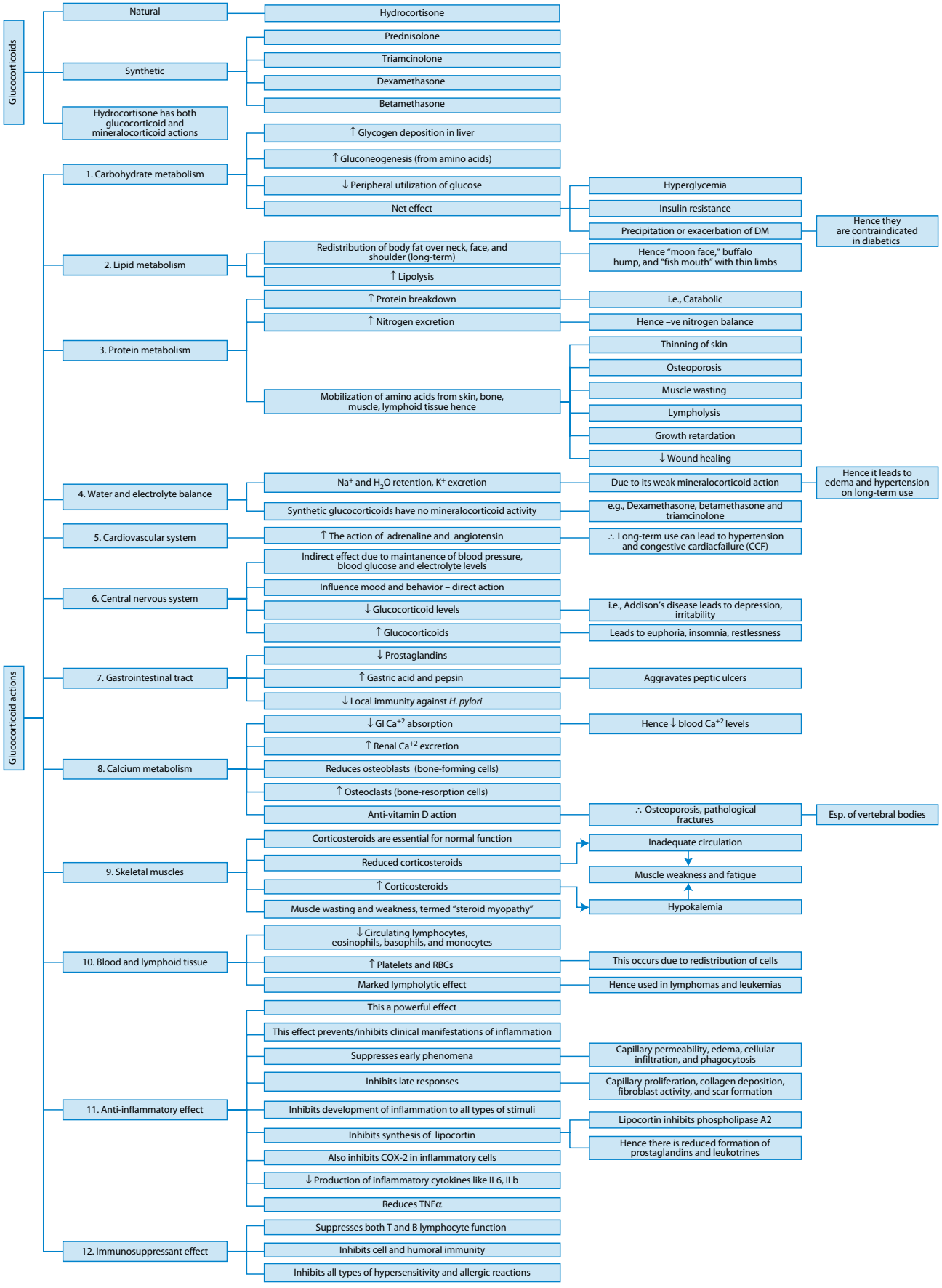
52.1 CORTICOSTEROIDS – INTRODUCTION, STRUCTURE SYNTHESIS, AND RELEASE



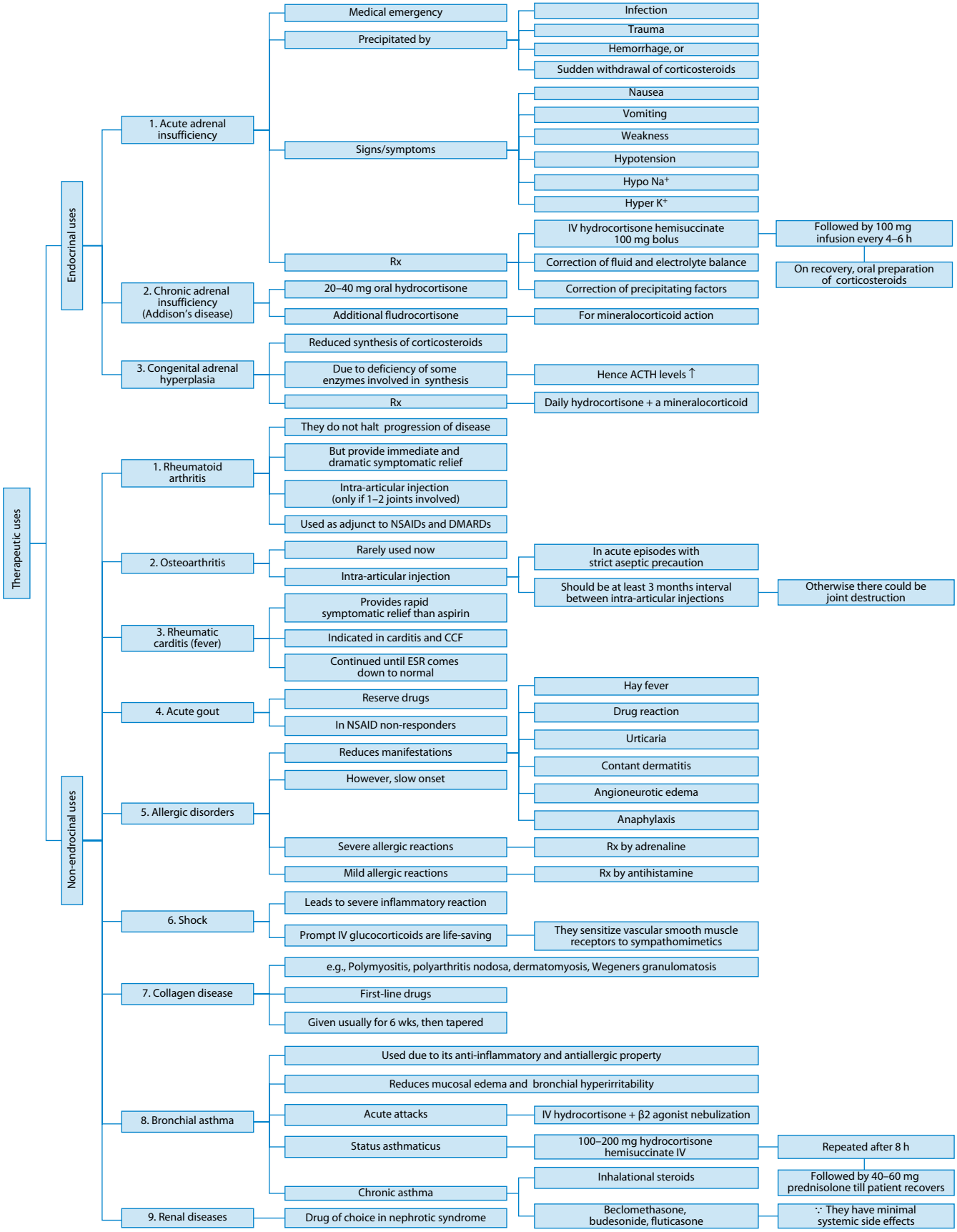
52.2 MECHANISM OF ACTION AND PHARMACOKINETICS



52.3 GLUCOCORTICOID ACTIONS

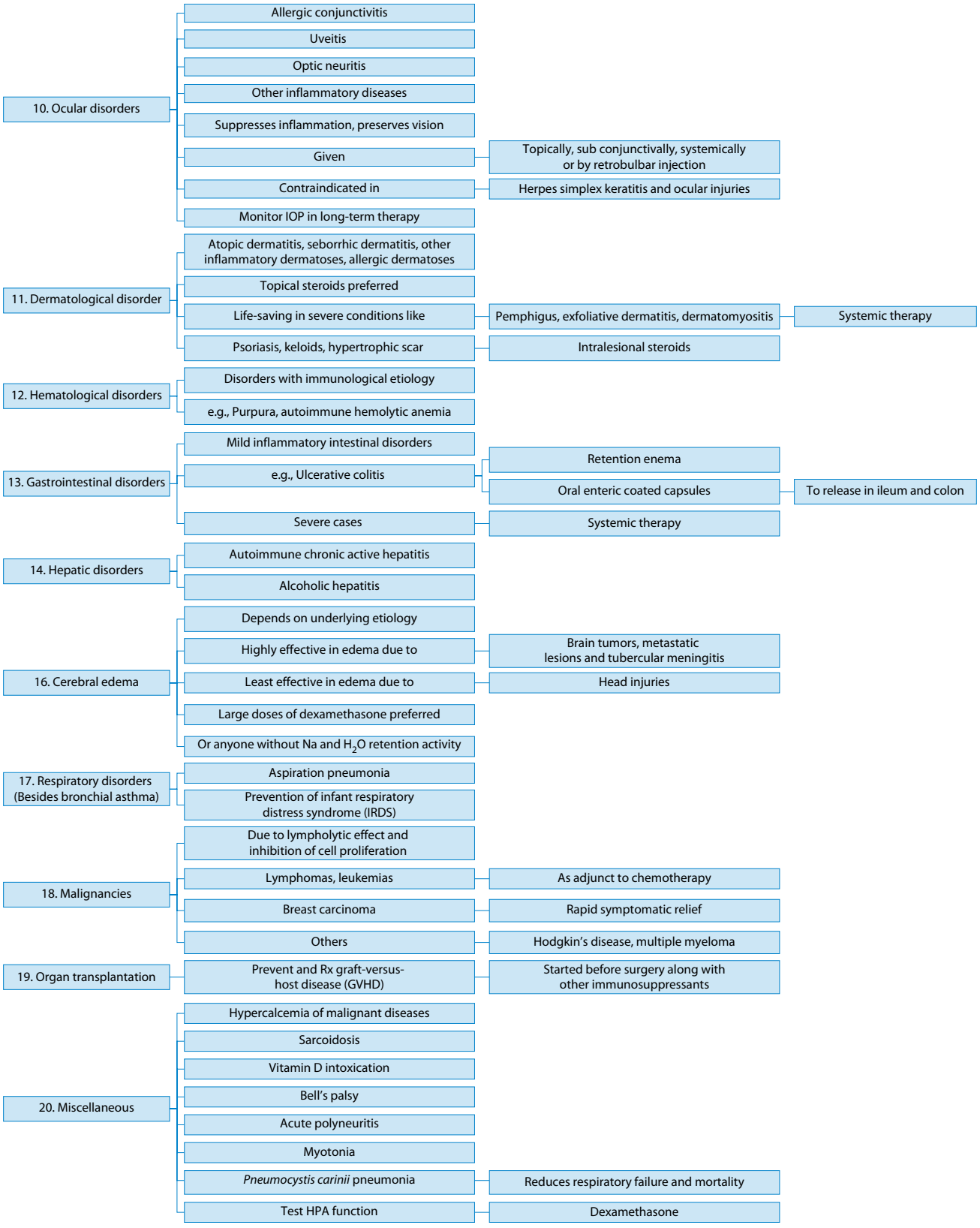


52.4 THERAPEUTIC USES

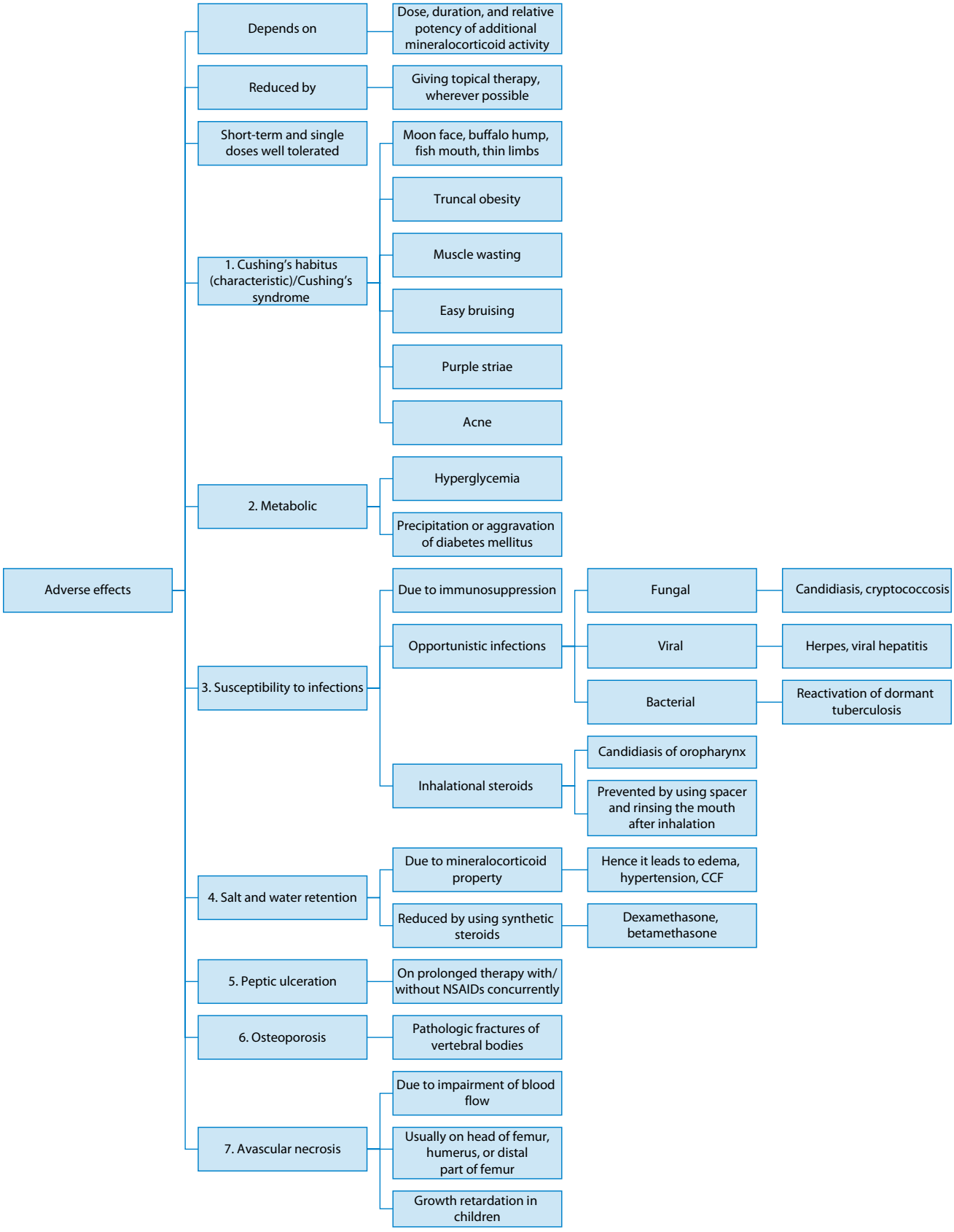


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52.4 THERAPEUTIC USES (Continued)

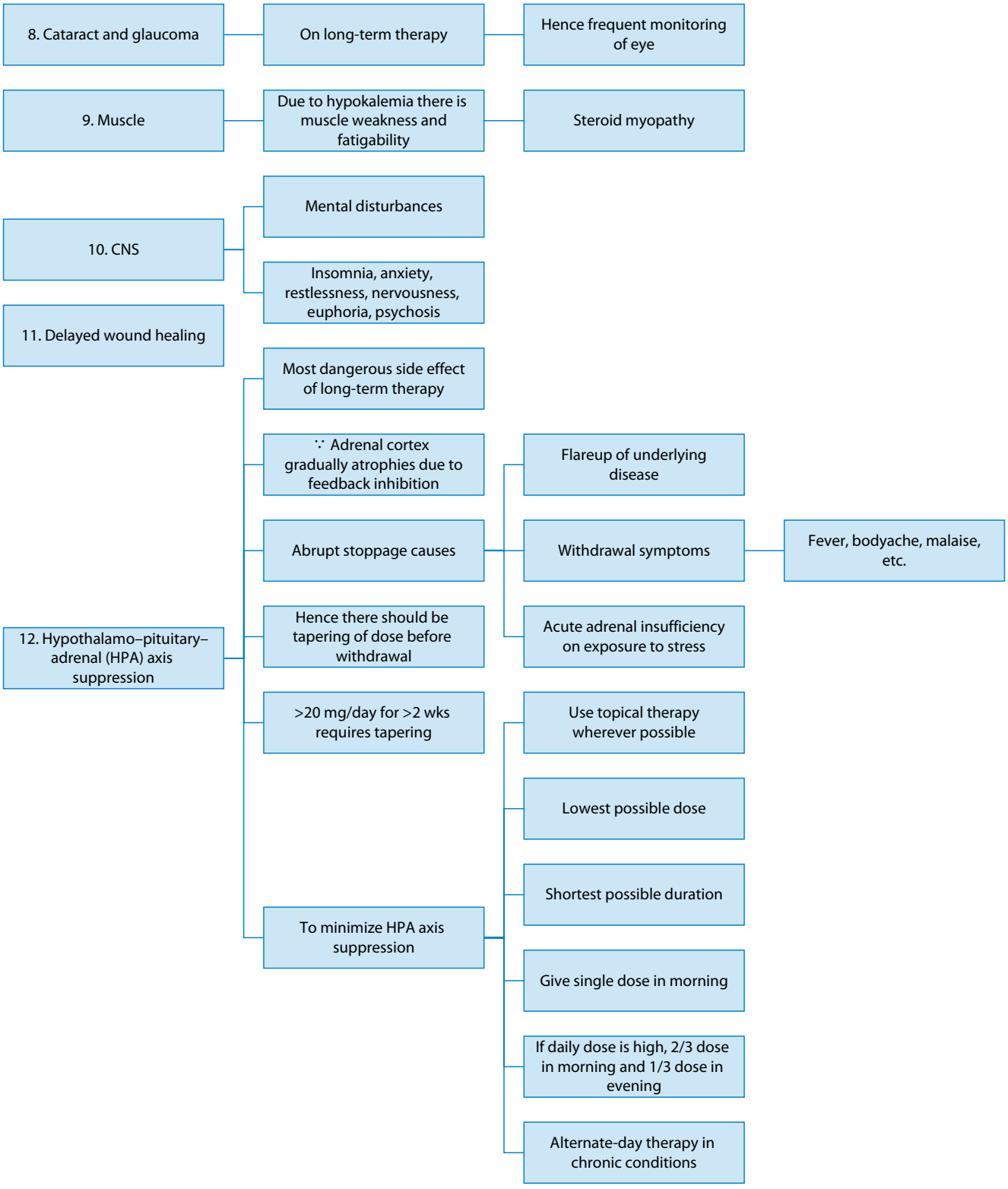


52.5 ADVERSE EFFECTS

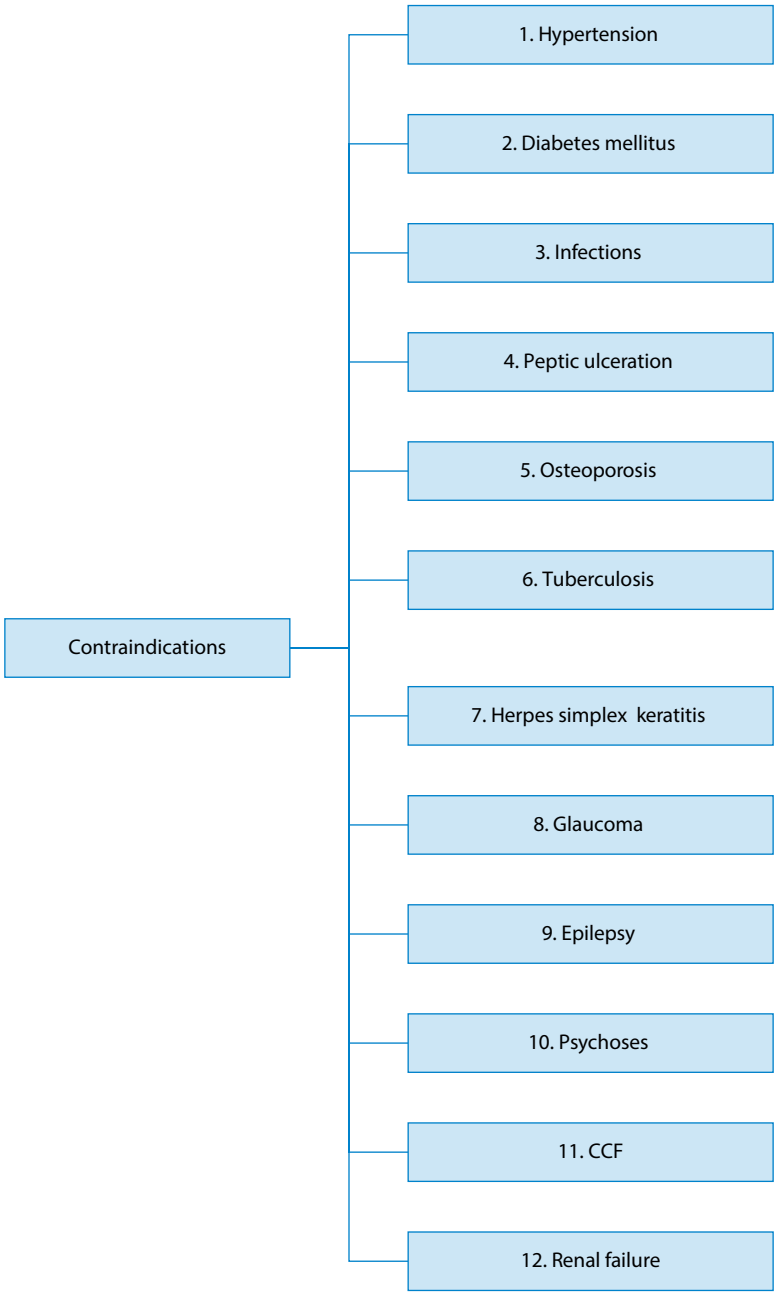


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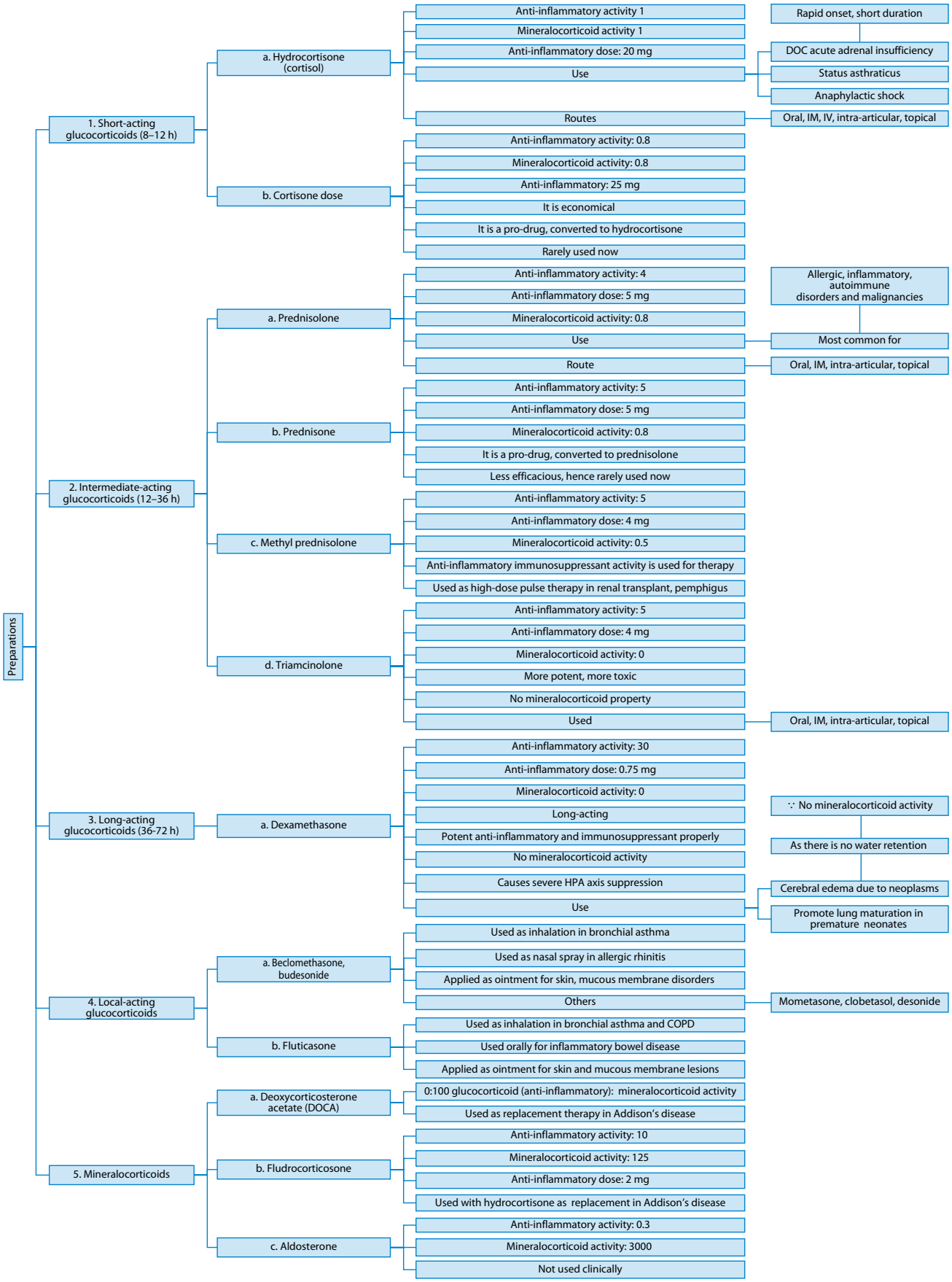
52.5 ADVERSE EFFECTS (Continued)



52.6 CONTRAINDICATIONS



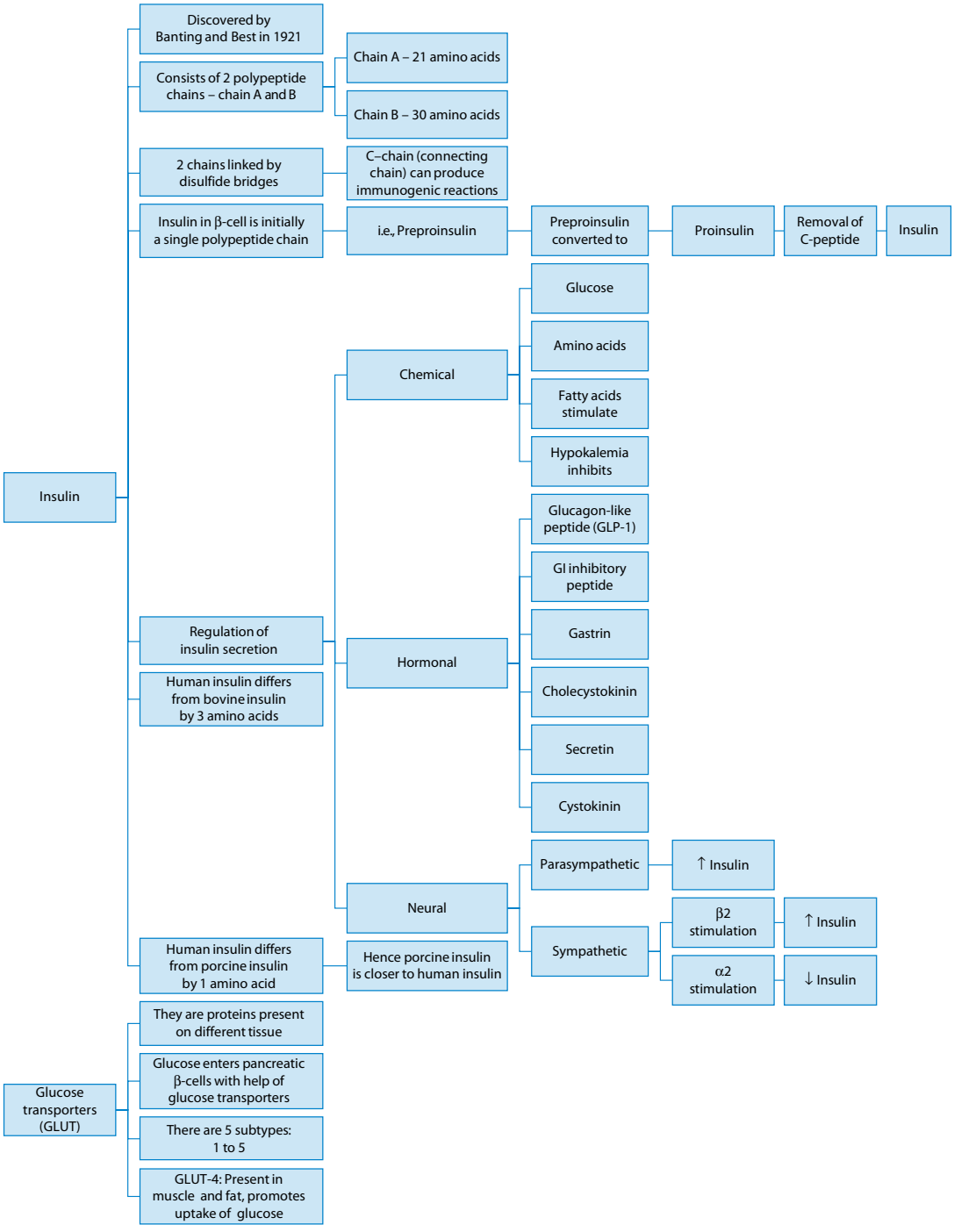
52.7 PREPARATIONS AND CLASSIFICATIONS



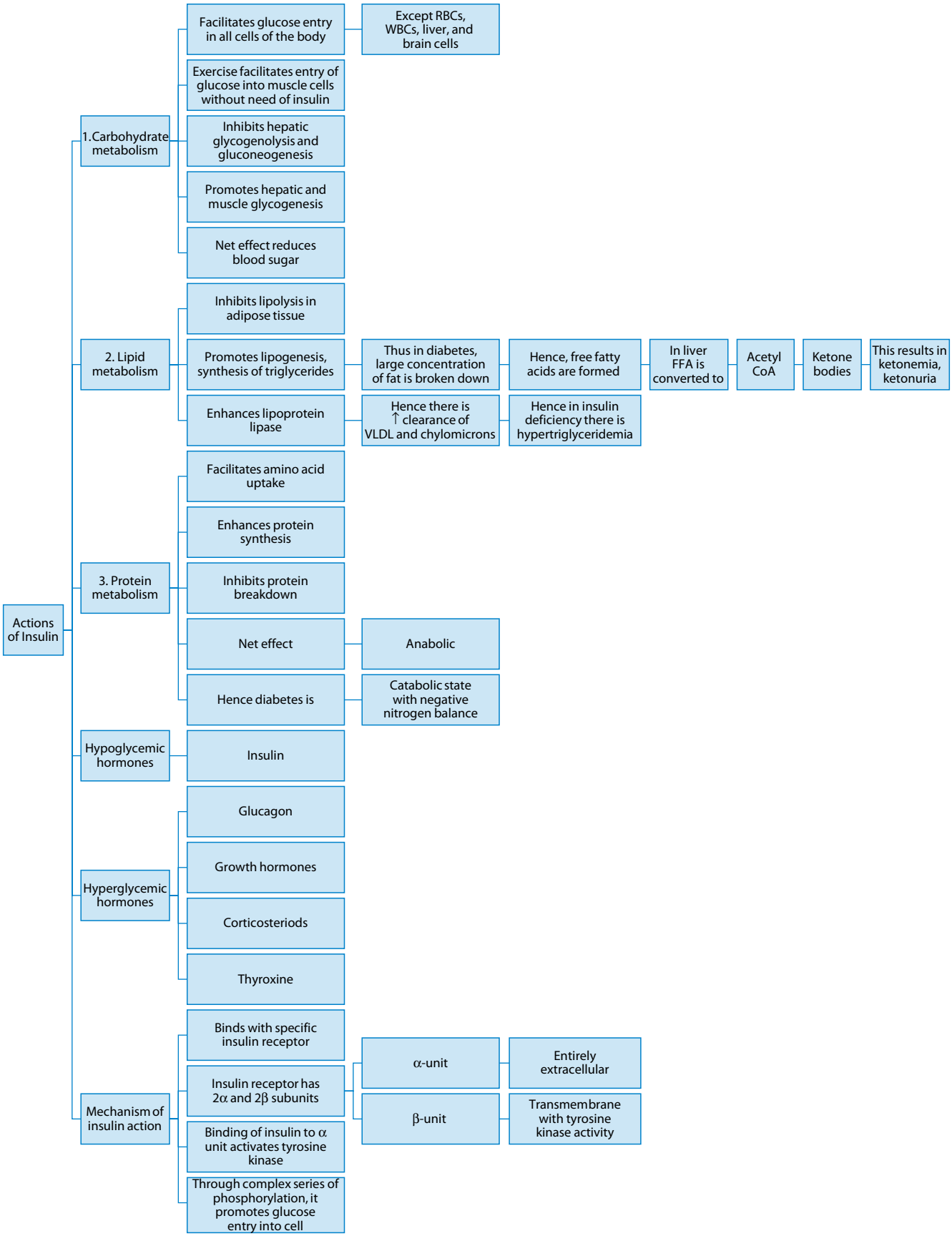
53

Insulin and oral antidiabetic agents

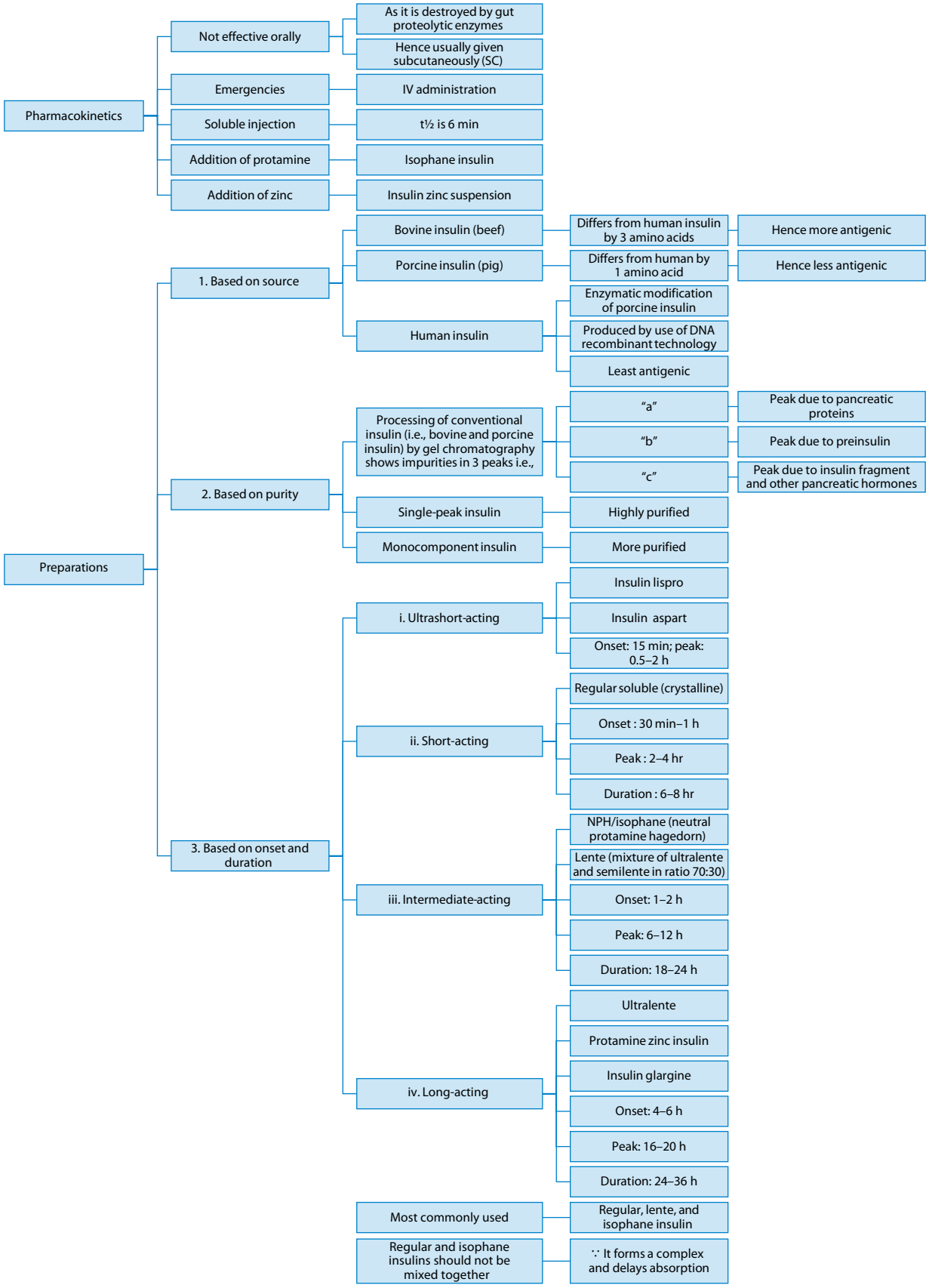
53.1 INSULIN REGULATION AND GLUCOSE TRANSPORTERS (GLUT)



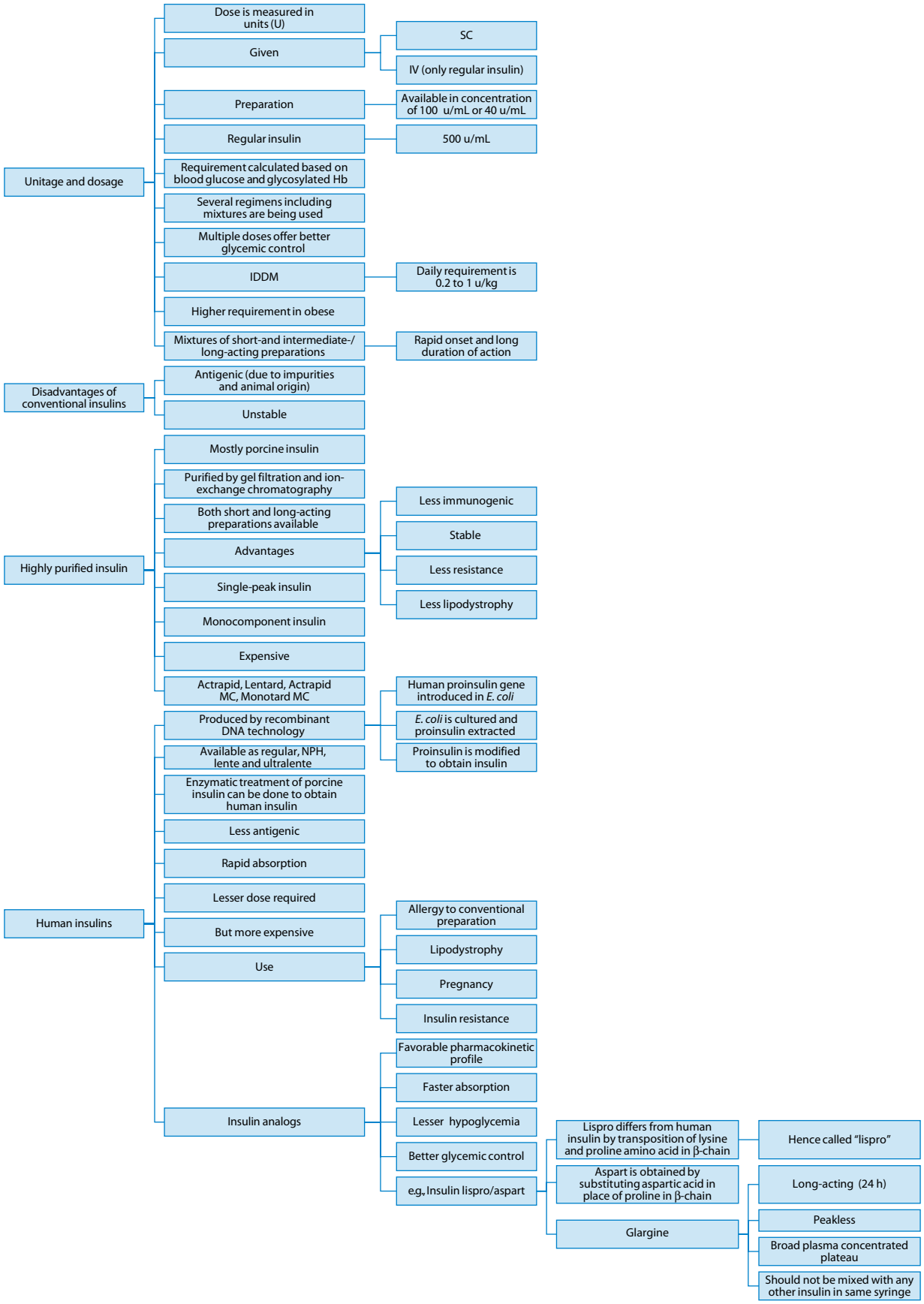
53.2 ACTIONS OF INSULIN AND MECHANISM OF ACTION



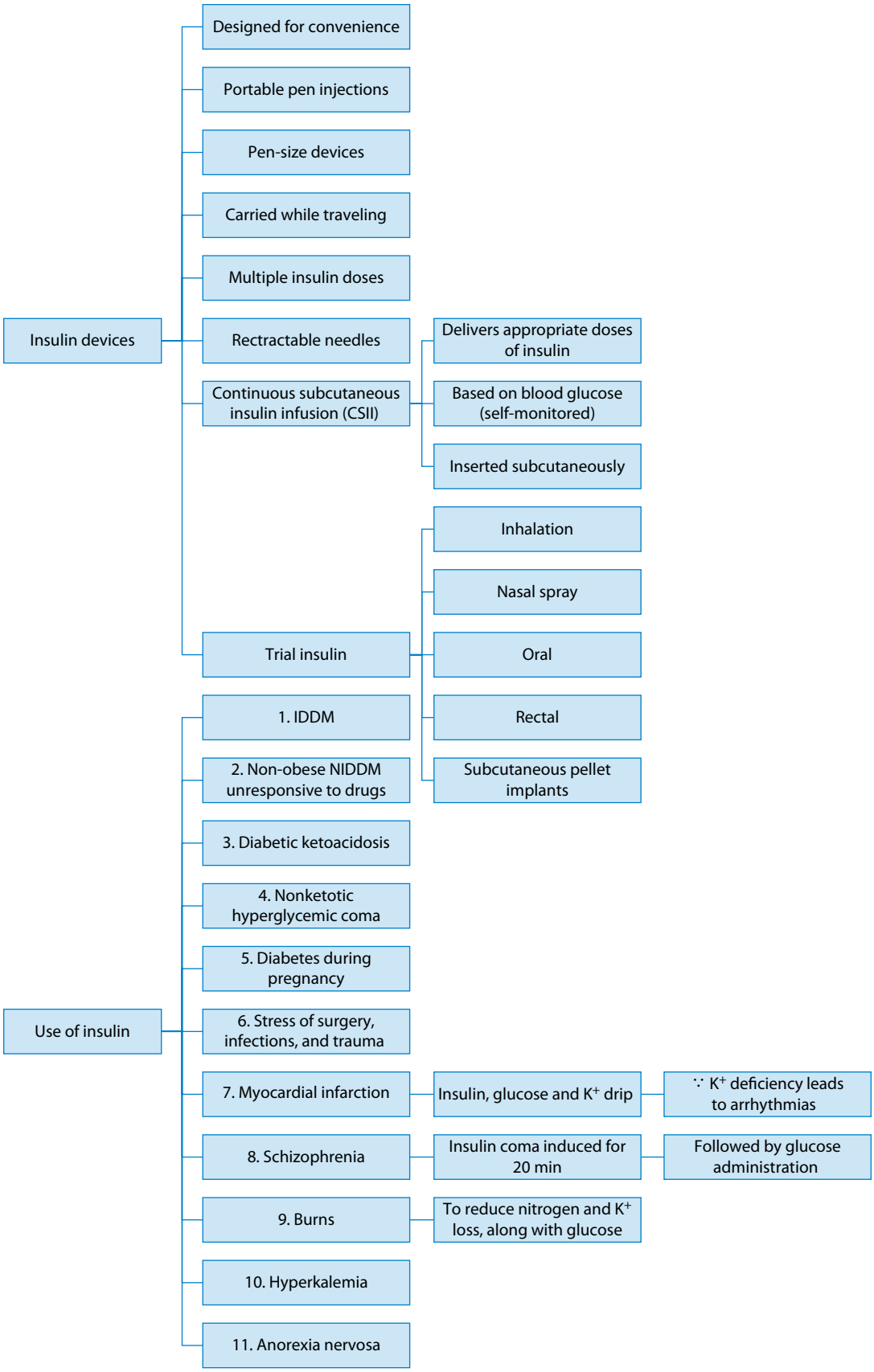
53.3 PHARMACOKINETICS AND PREPARATIONS



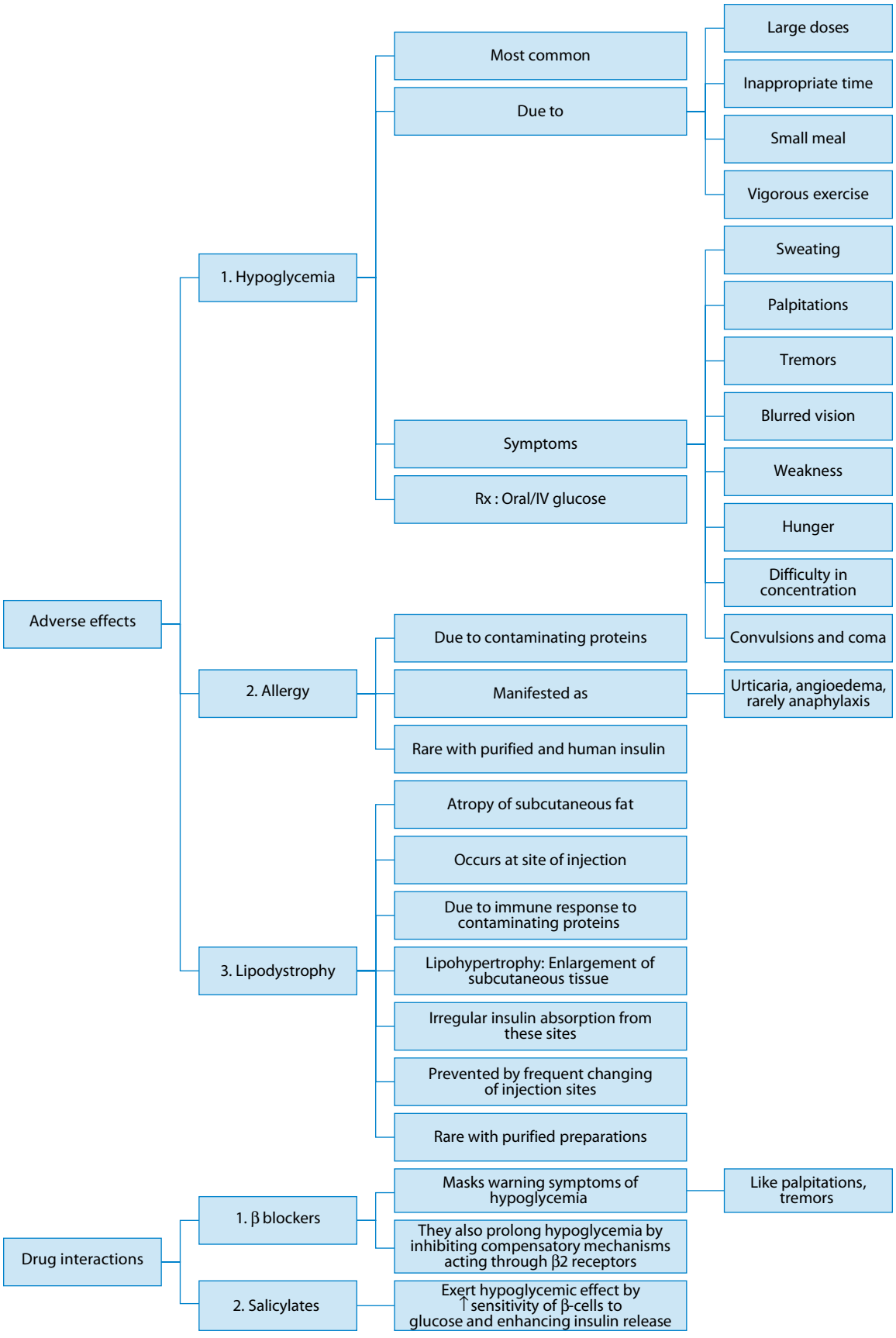
53.4 UNITAGE AND DOSAGE, HUMAN INSULINS, AND INSULIN ANALOGS



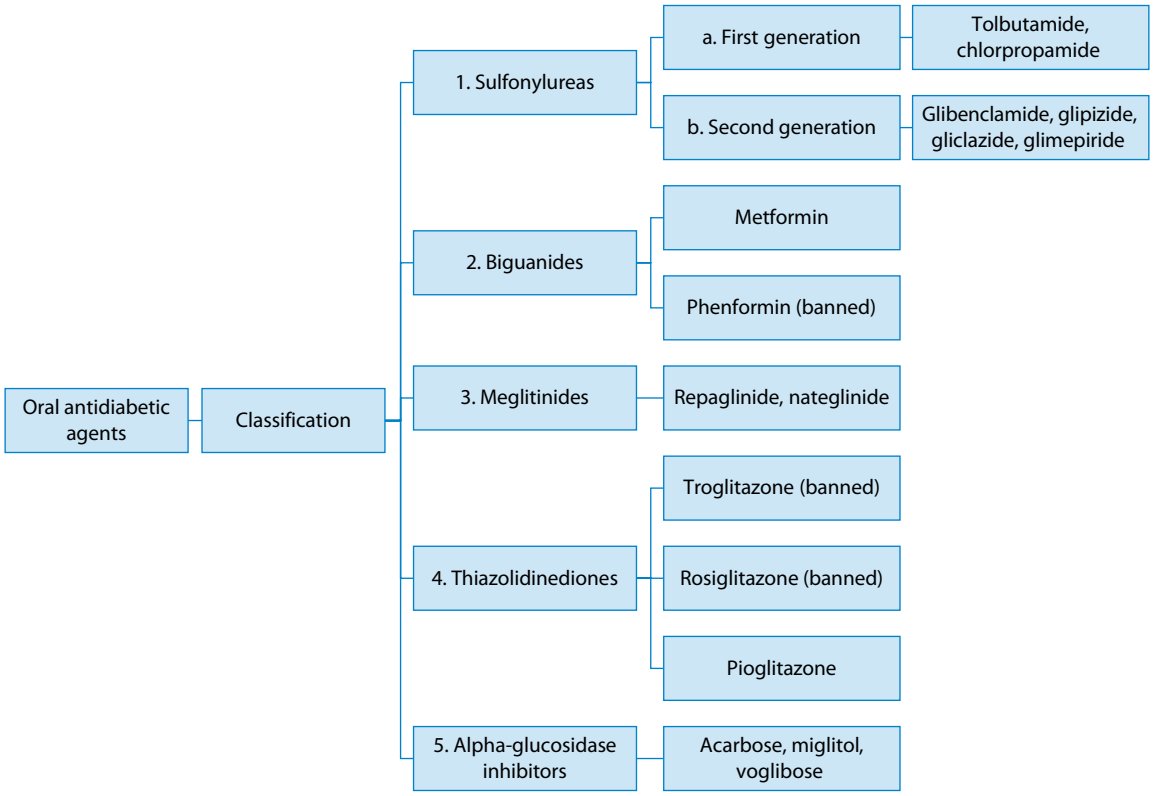
53.5 INSULIN DEVICES AND USE OF INSULIN



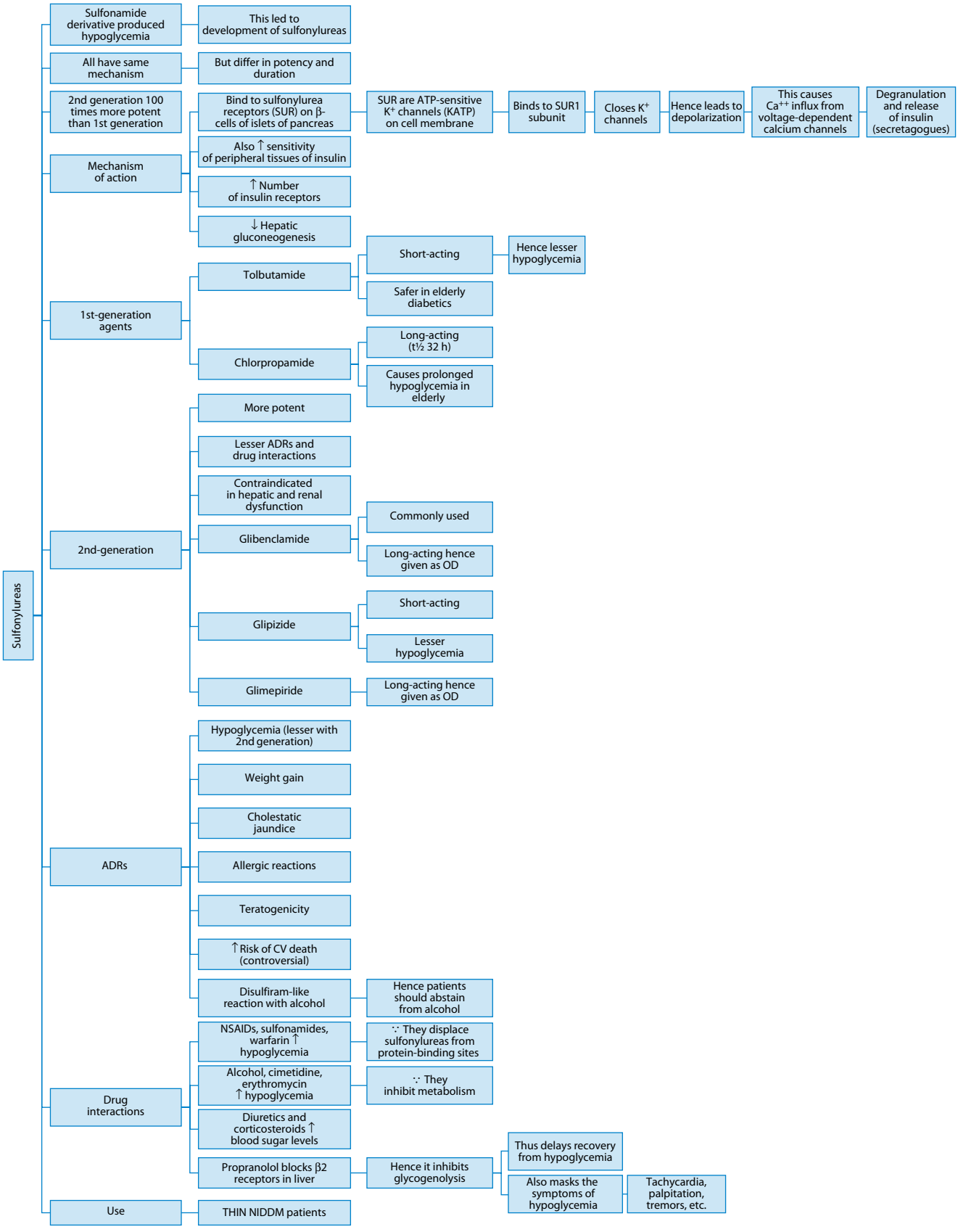
53.6 ADVERSE EFFECTS AND DRUG INTERACTIONS



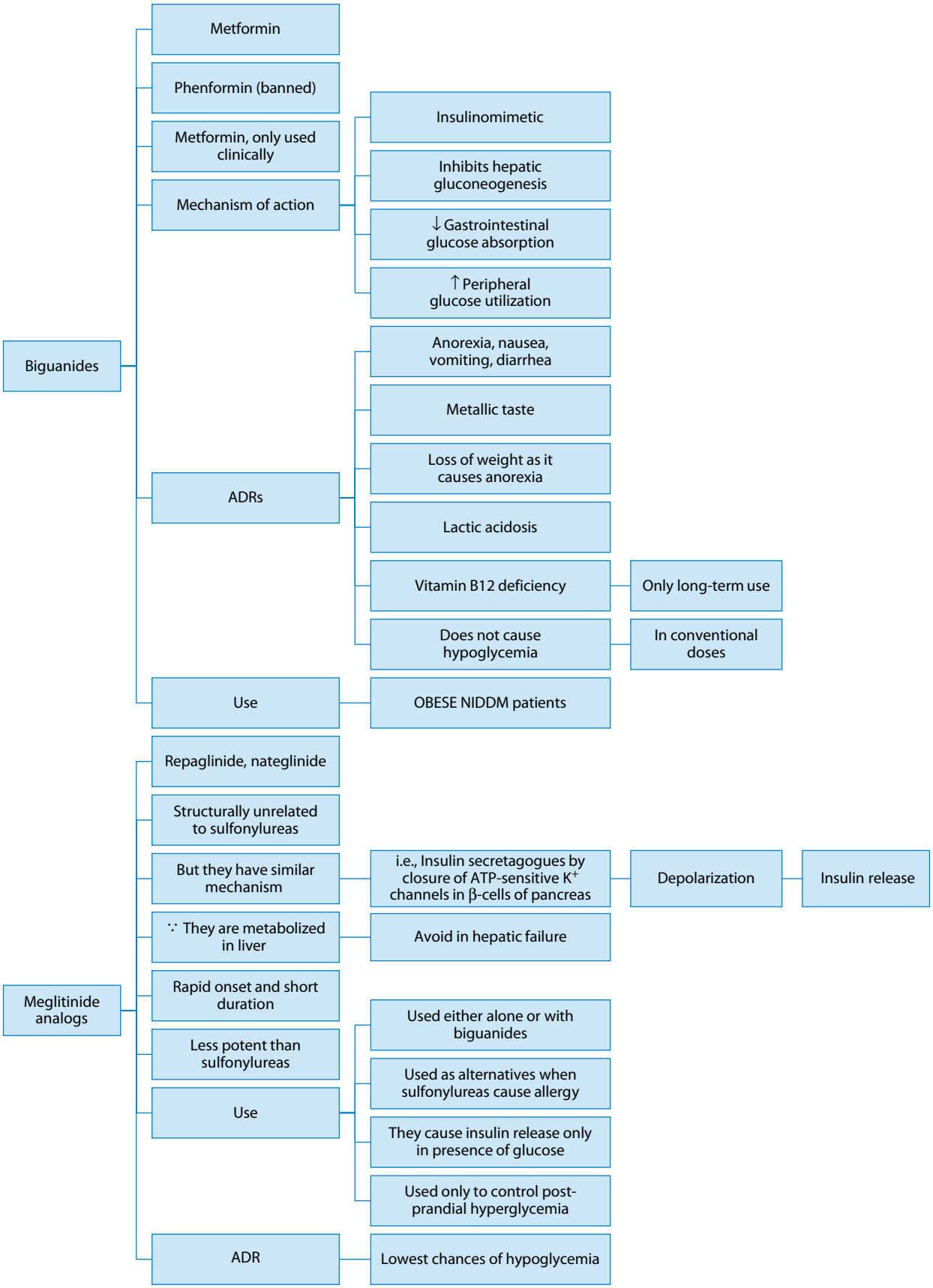
53.7 ORAL ANTIDIABETIC AGENTS – CLASSIFICATION



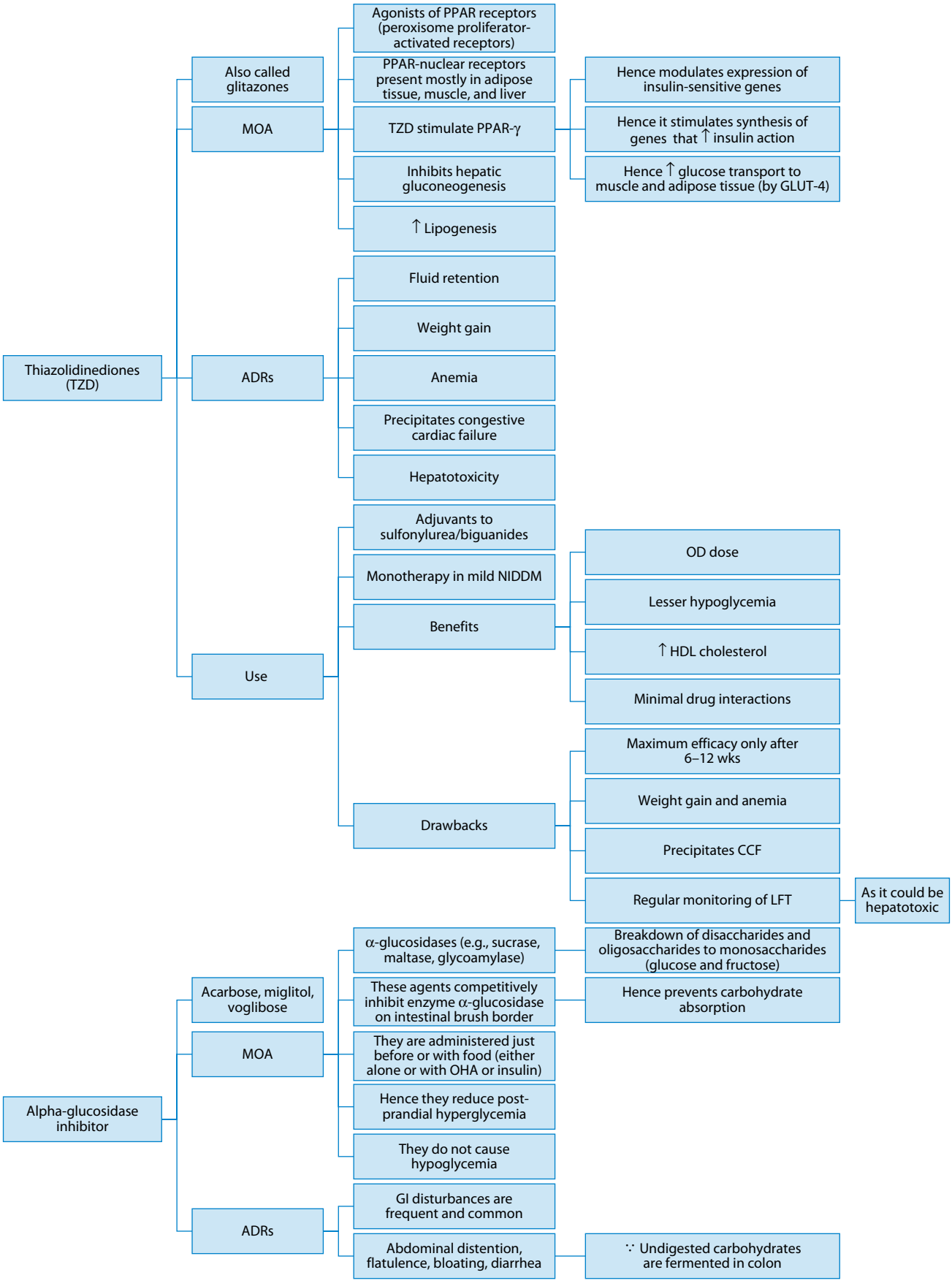
53.8 SULFONYLUREAS



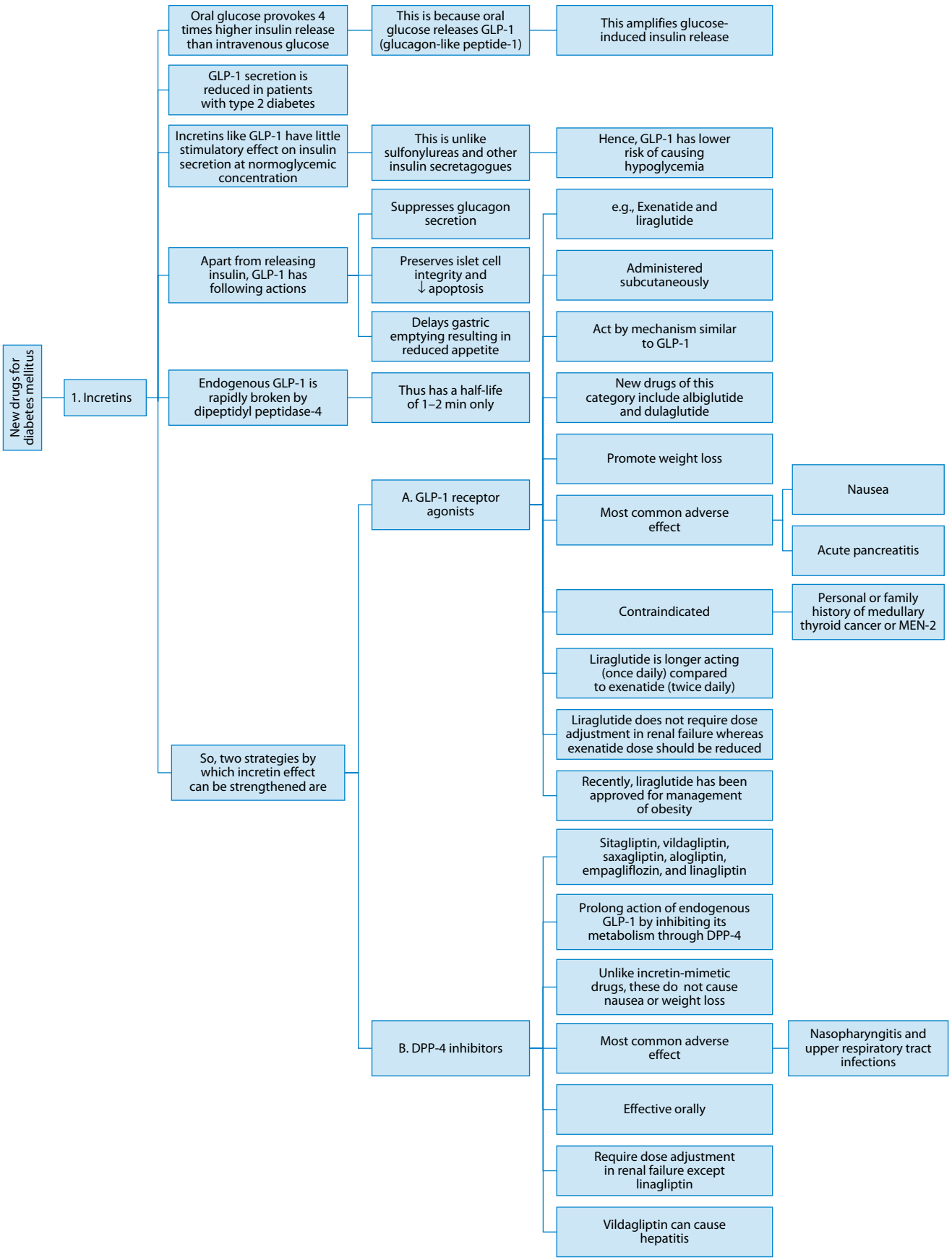
53.9 BIGUANIDES AND MEGLITINIDE ANALOGS



53.10 THIAZOLIDINEDIONES (TZD) AND ALPHA-GLUCOSIDASE INHIBITOR

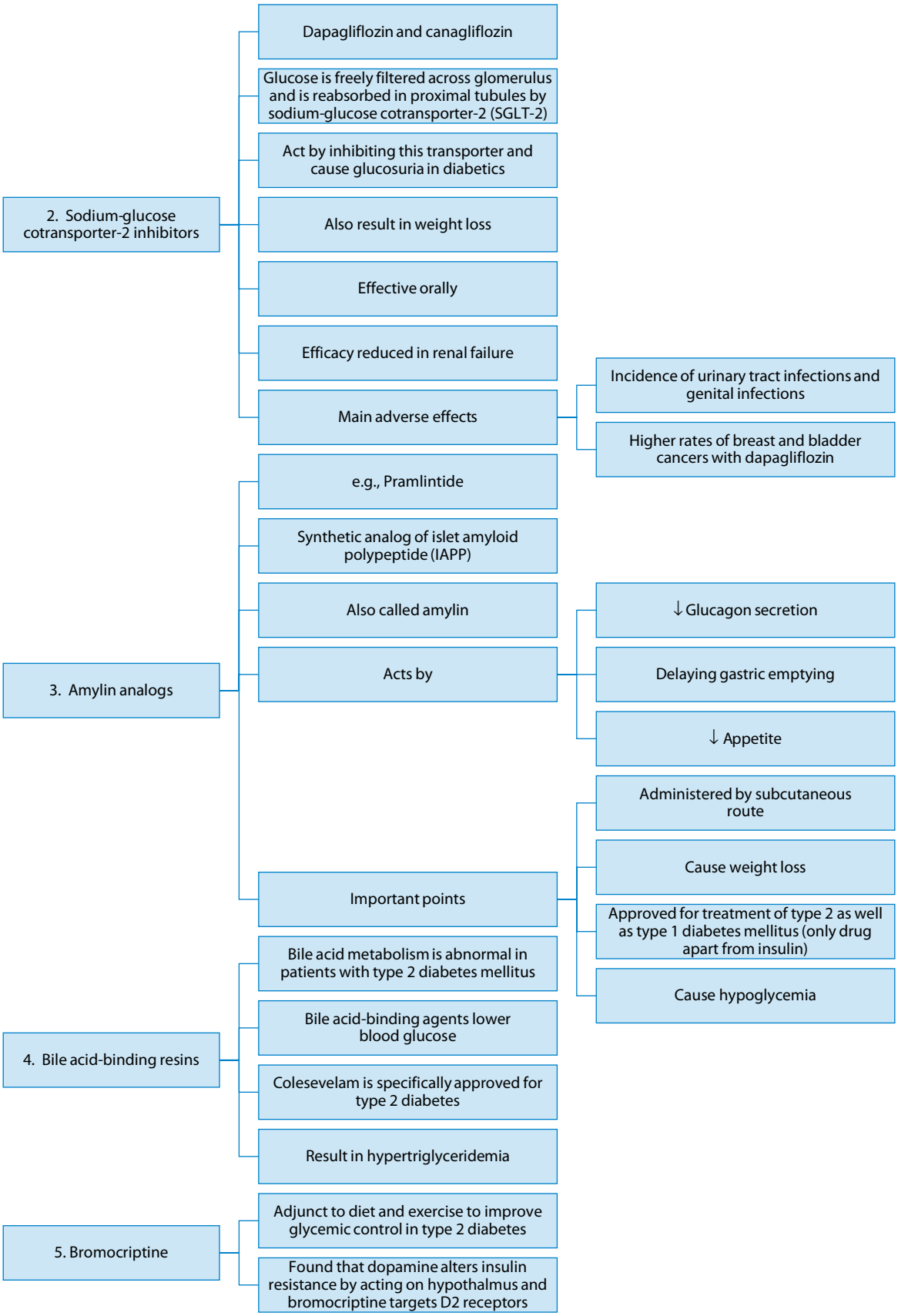


53.11 NEW DRUGS FOR DIABETES MELLITUS



(*Continued*)

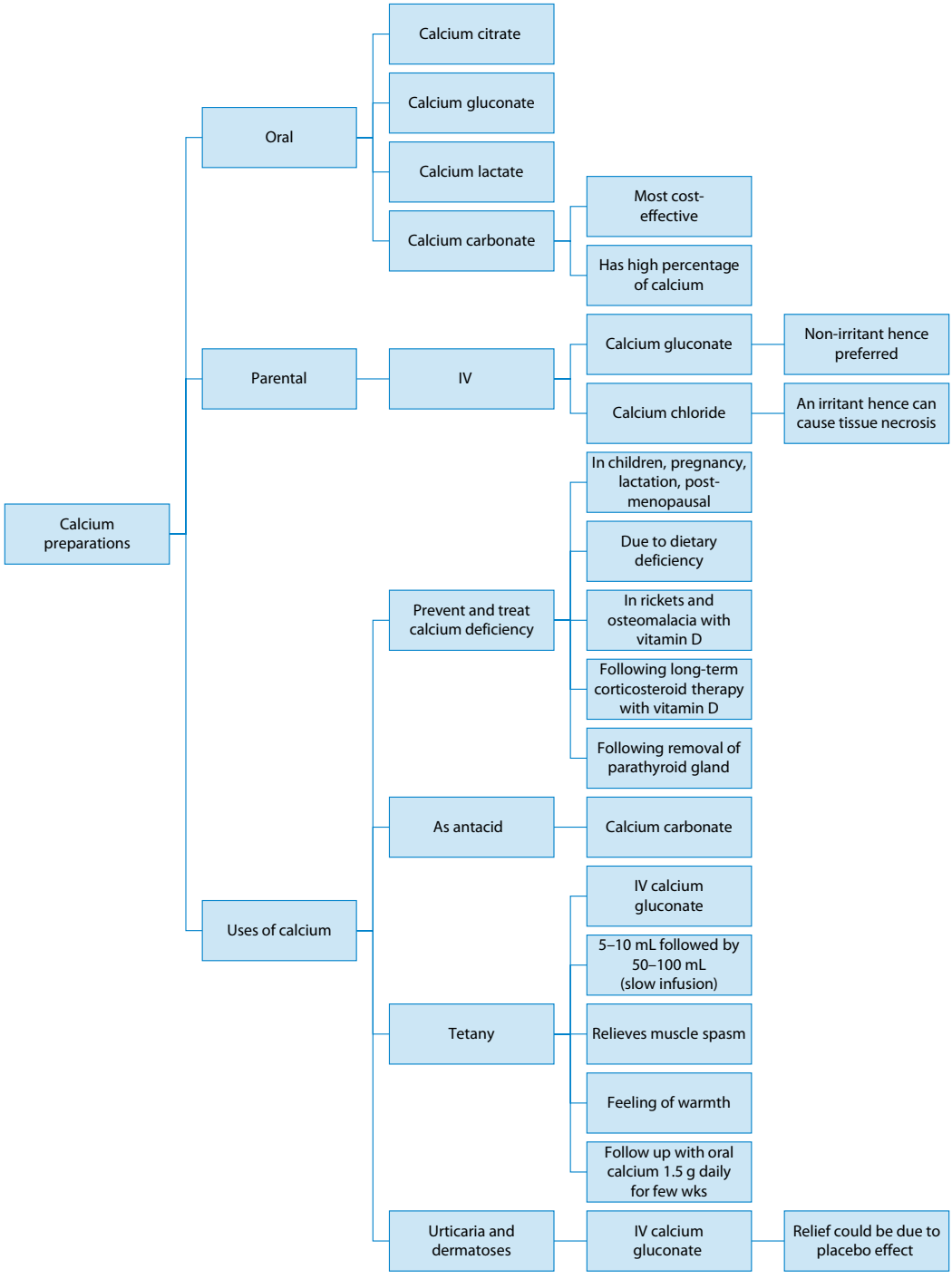
53.11 NEW DRUGS FOR DIABETES MELLITUS (Continued)



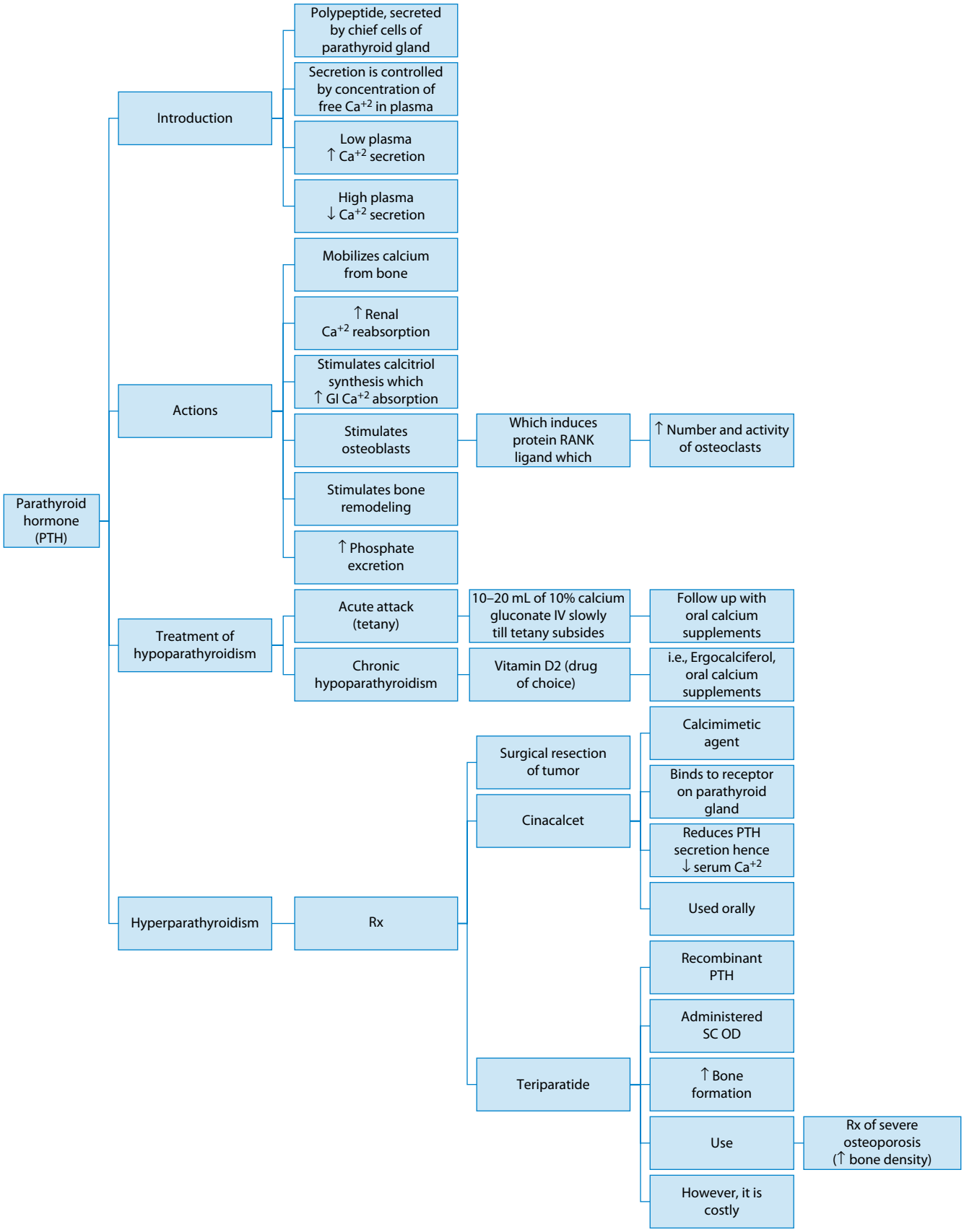
54

Agents affecting calcium balance

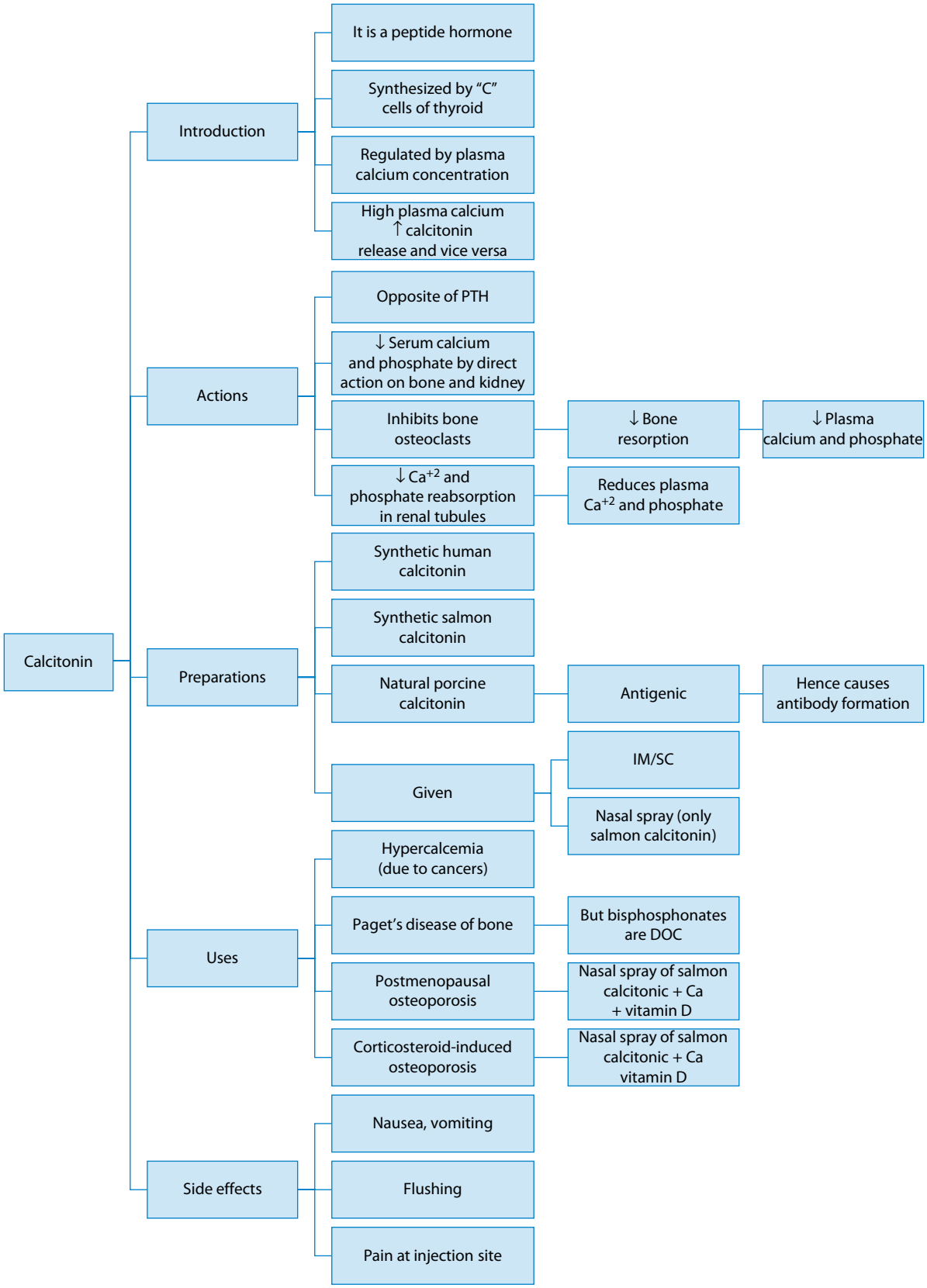
54.1 CALCIUM PREPARATIONS AND USES



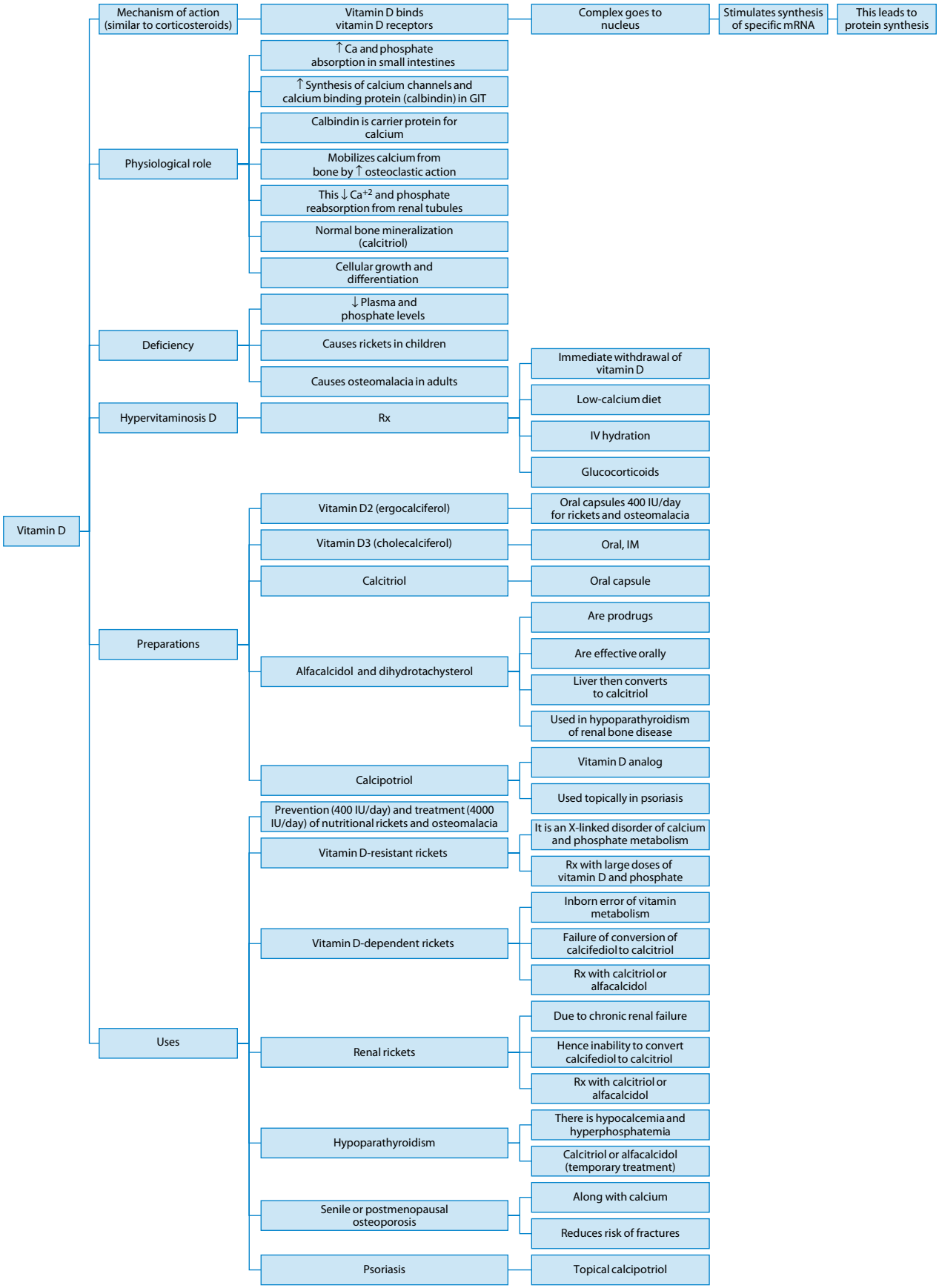
54.2 PARATHYROID HORMONE (PTH)



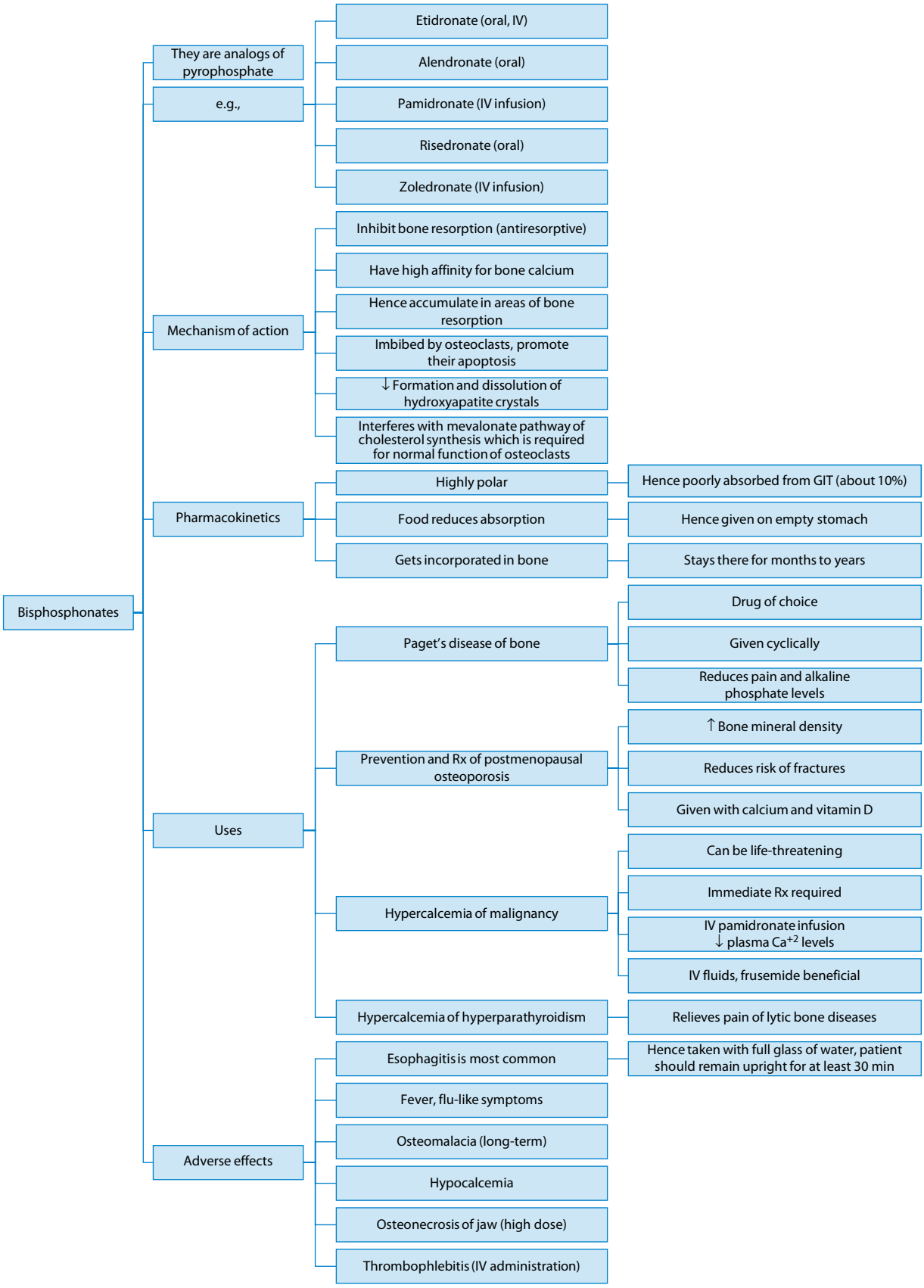
54.3 CALCITONIN



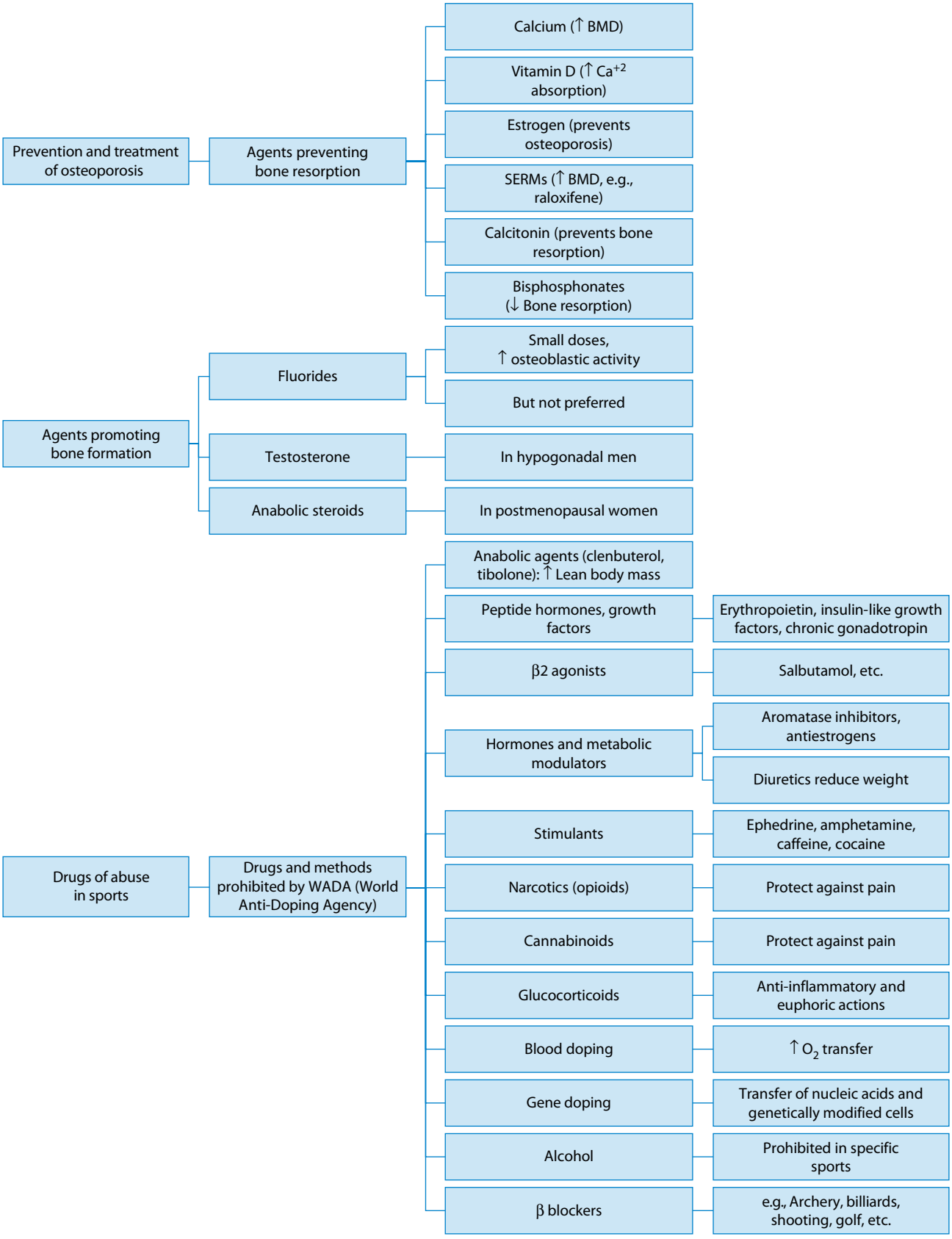
54.4 VITAMIN D



54.5 BISPHOSPHONATES



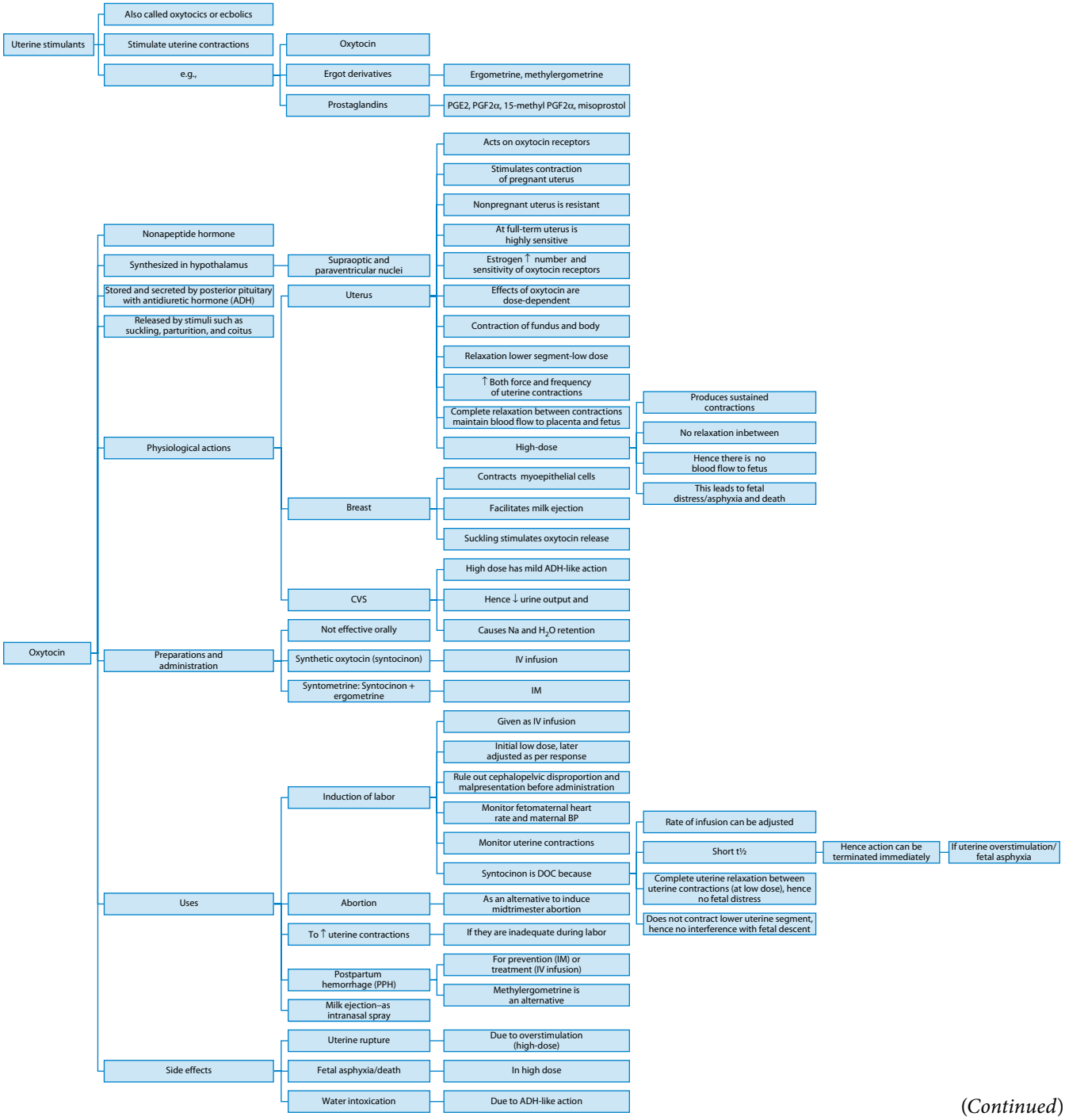
54.6 PREVENTION AND TREATMENT OF OSTEOPOROSIS, AND DRUGS OF ABUSE IN SPORTS



55

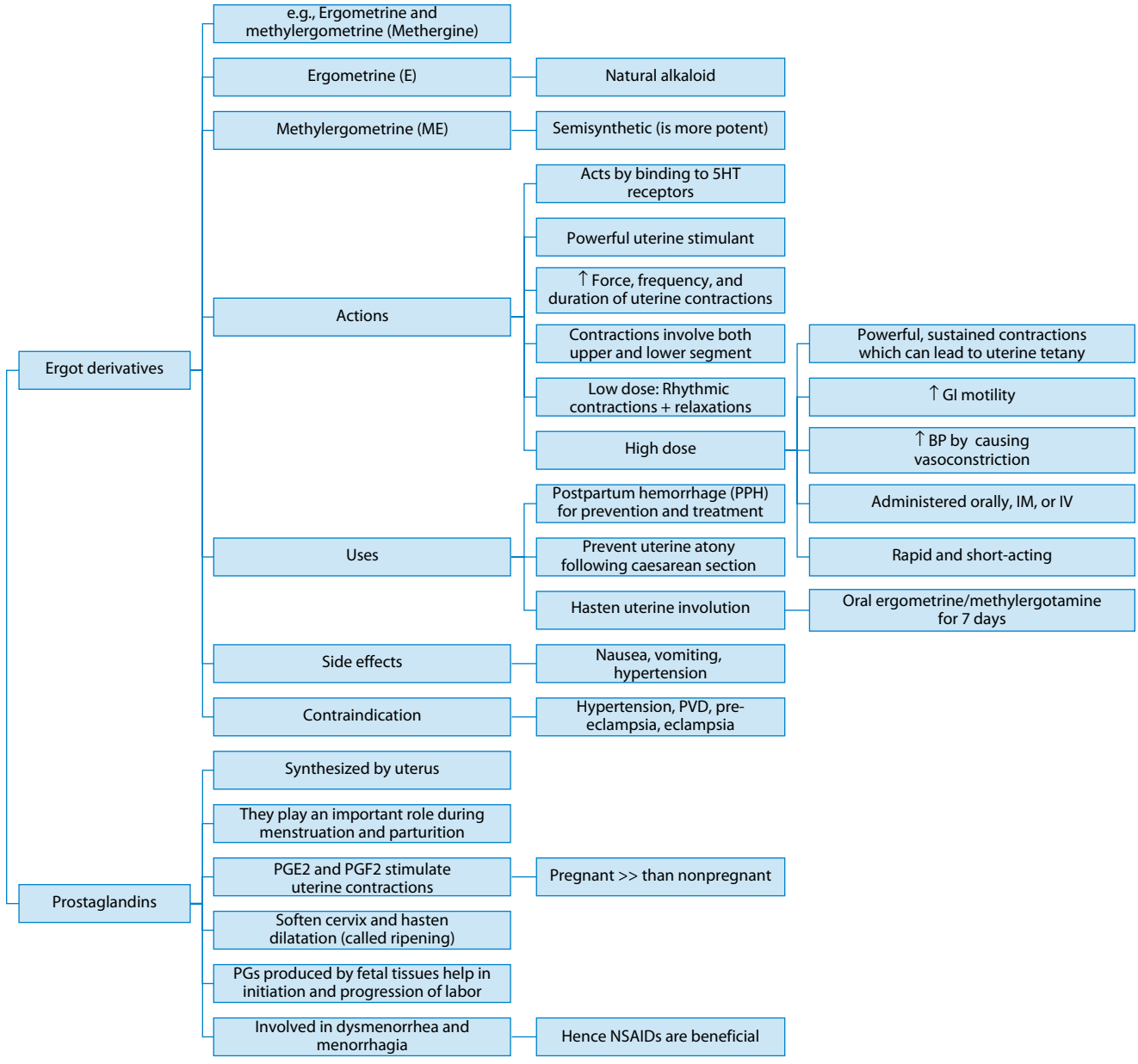
Drugs acting on uterus

55.1 UTERINE STIMULANTS



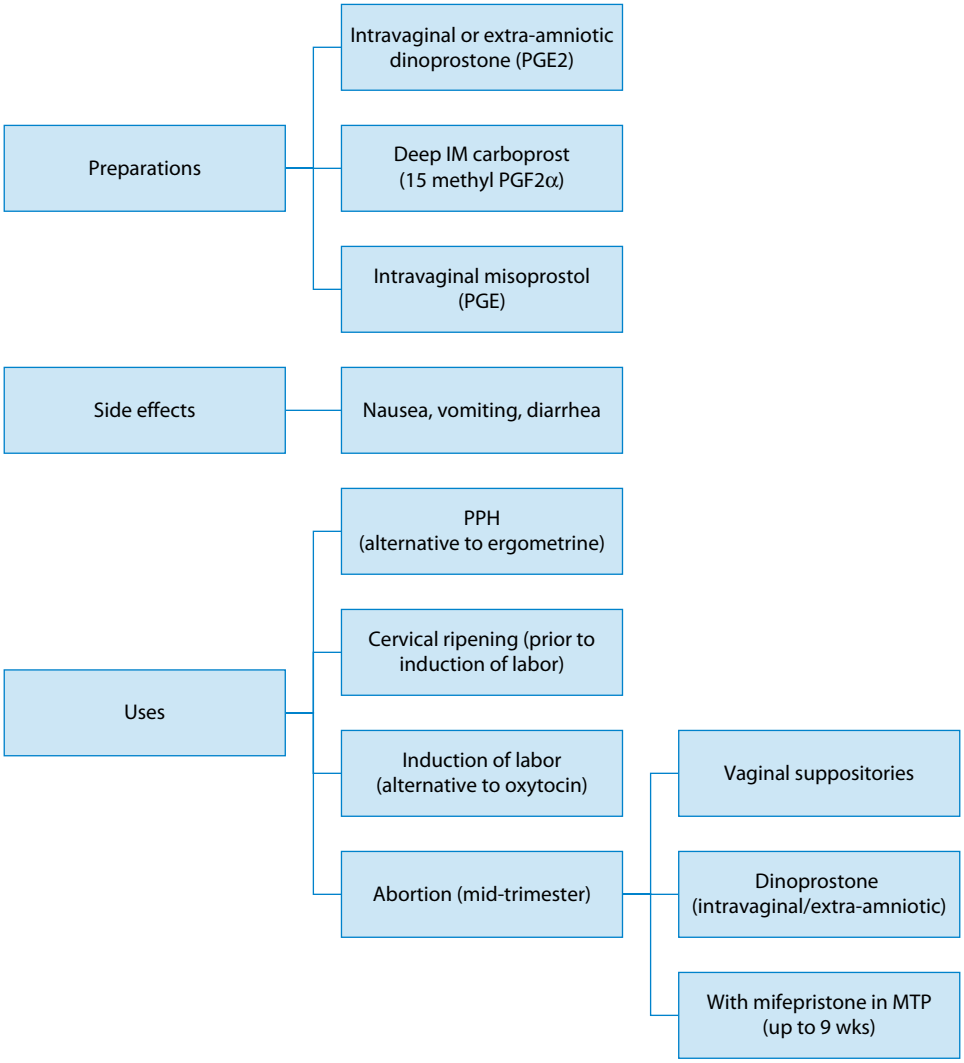
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55.1 UTERINE STIMULANTS (Continued)

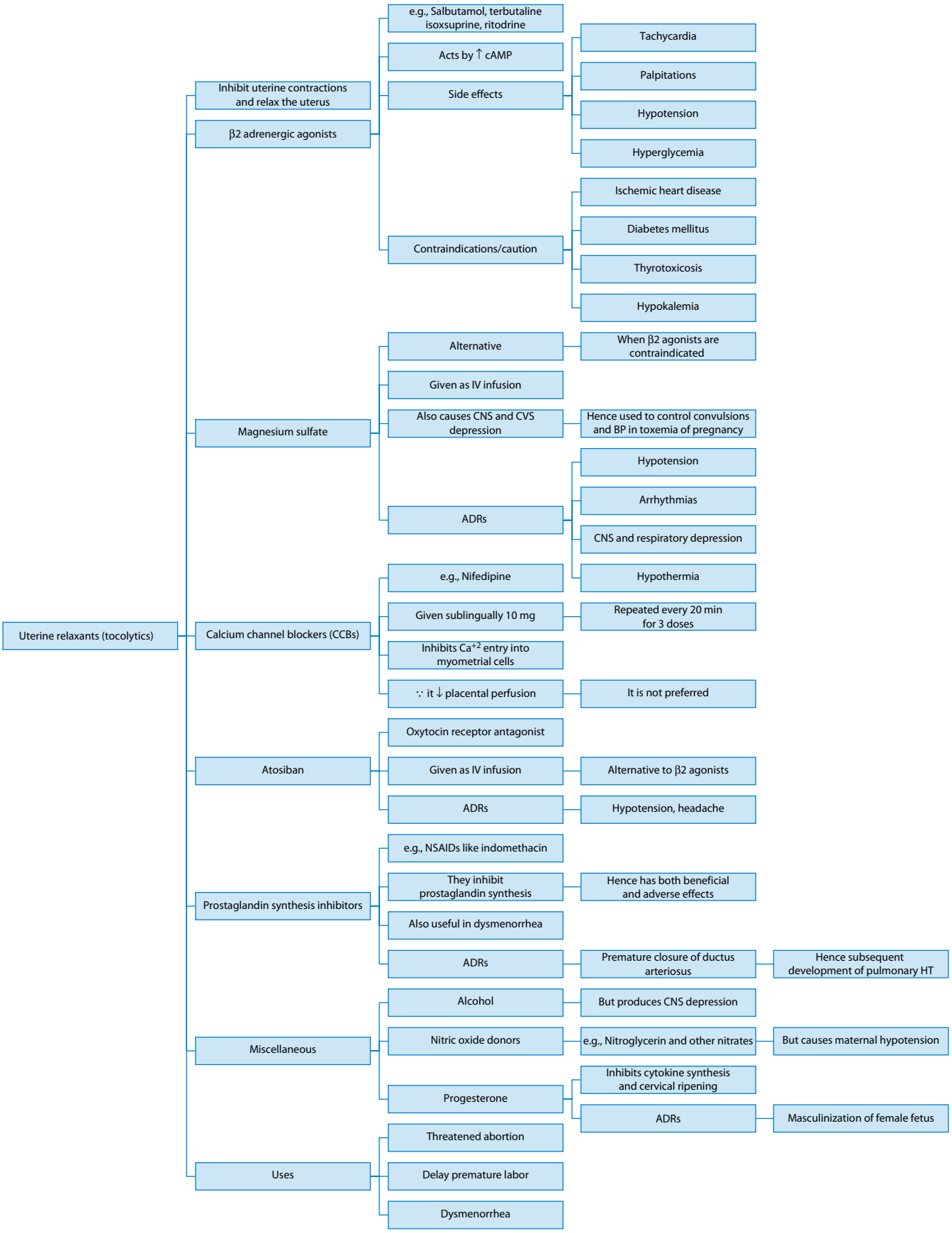


(*Continued*)

55.1 UTERINE STIMULANTS (Continued)



55.2 UTERINE RELAXANTS (TOCOLYTICS)



55.3 DIFFERENCES BETWEEN OXYTOCIN AND ERGOMETRINE

Oxytocin	**Ergometrine**
1. Synthetic (commercial)	Natural
2. Peptide	Alkaloid
3. Acts on oxytocin receptors	5-HT receptors
4. Endogenous	Exogenous
5. Only IV	Oral, IM and IV
6. Short duration t½ 15 min	Long duration t½ 2 h
7. Contracts body & fundus	Contracts whole uterus
8. Relaxes lower segment	No relaxation
9. Induces labor, milk ejection	PPH, uterine involution



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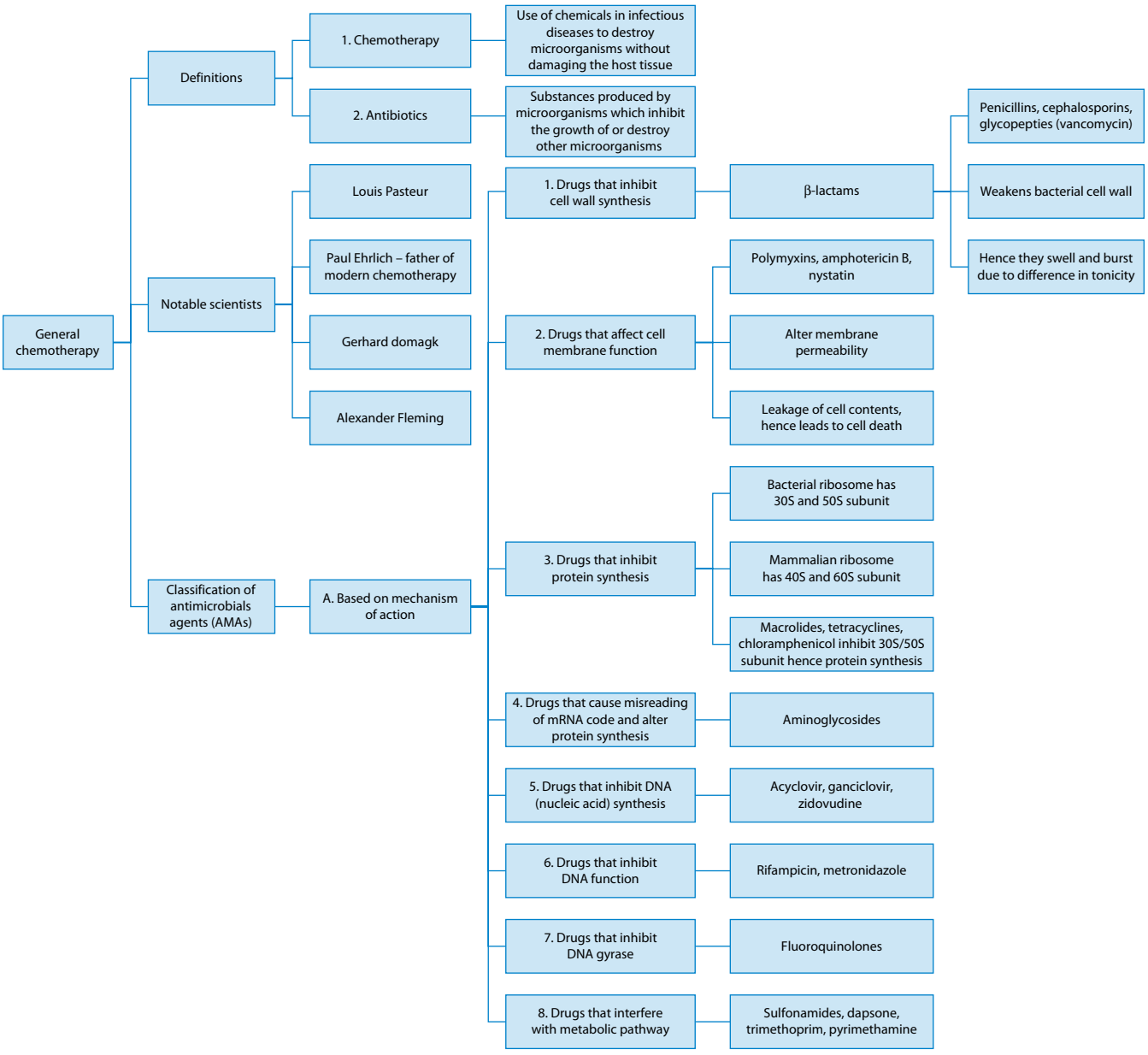
PART X

Chemotherapy

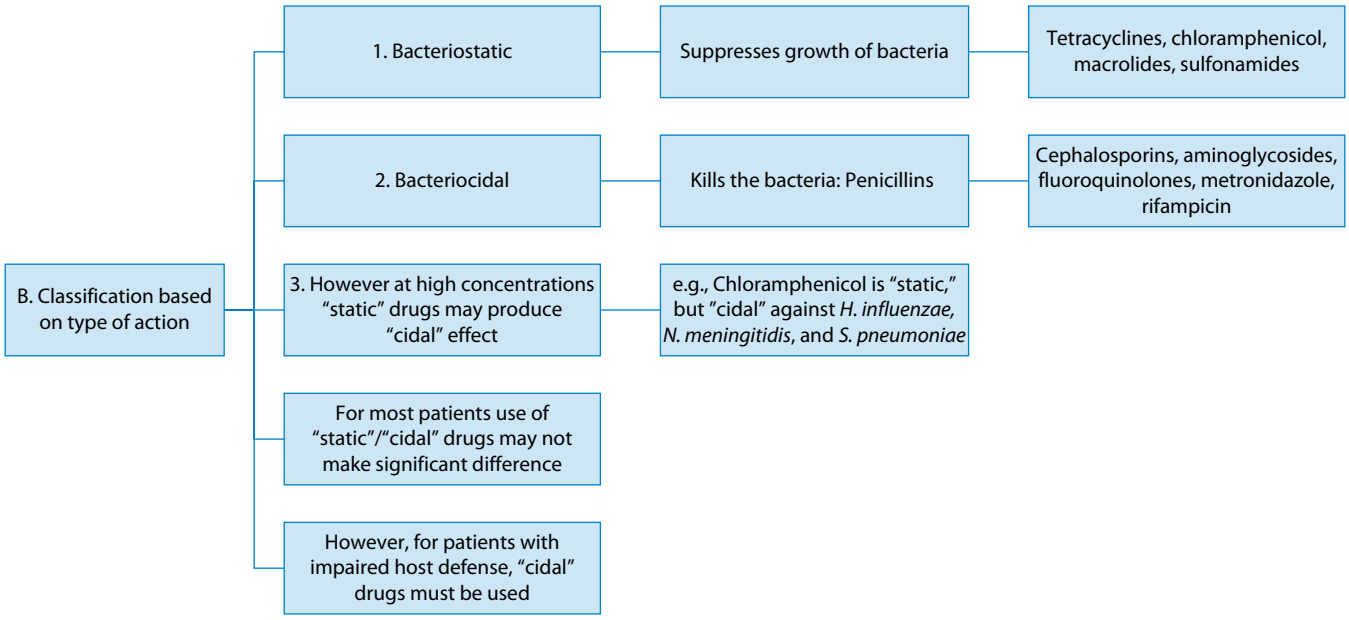
56

General chemotherapy

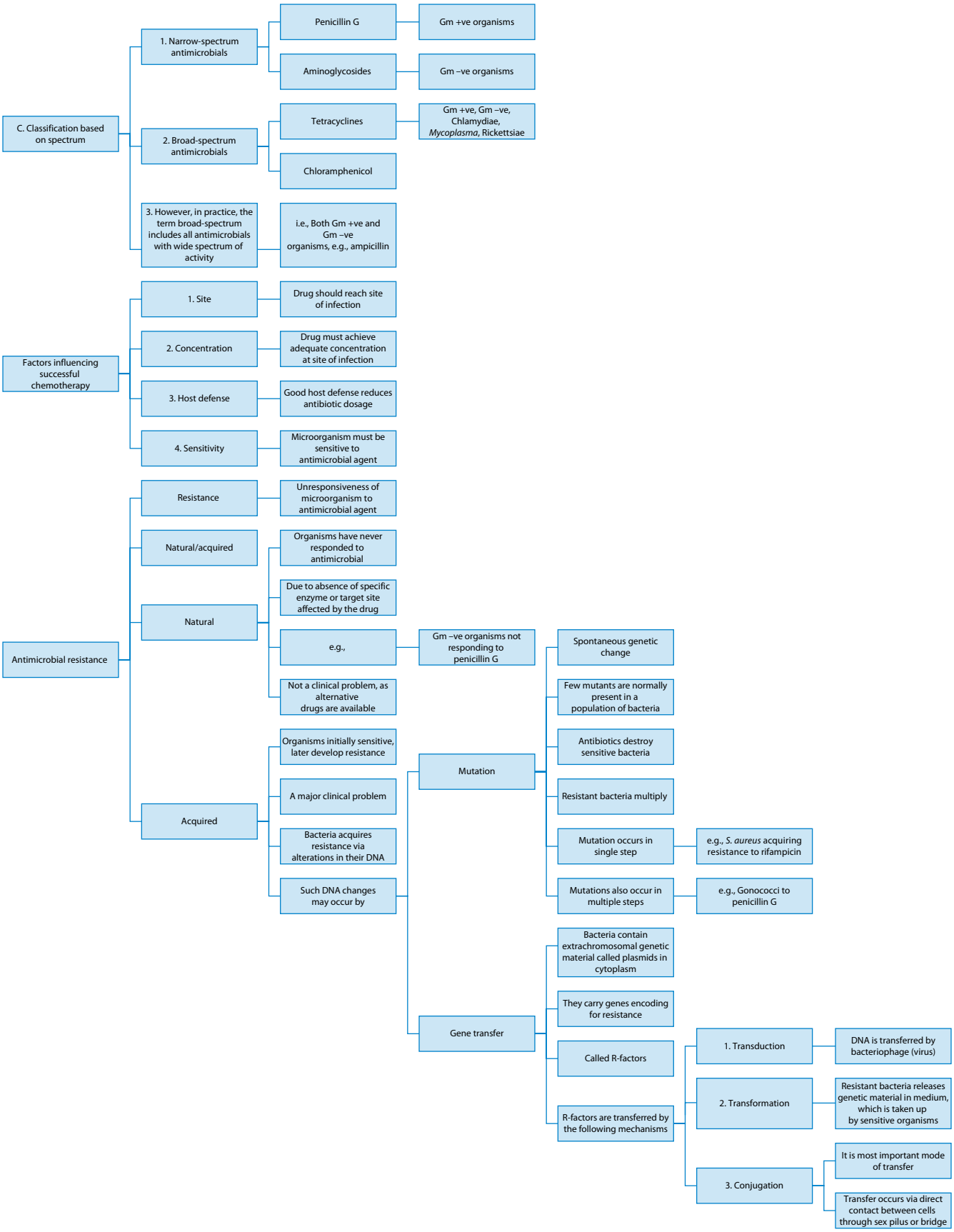
56.1 DEFINITIONS AND CLASSIFICATIONS



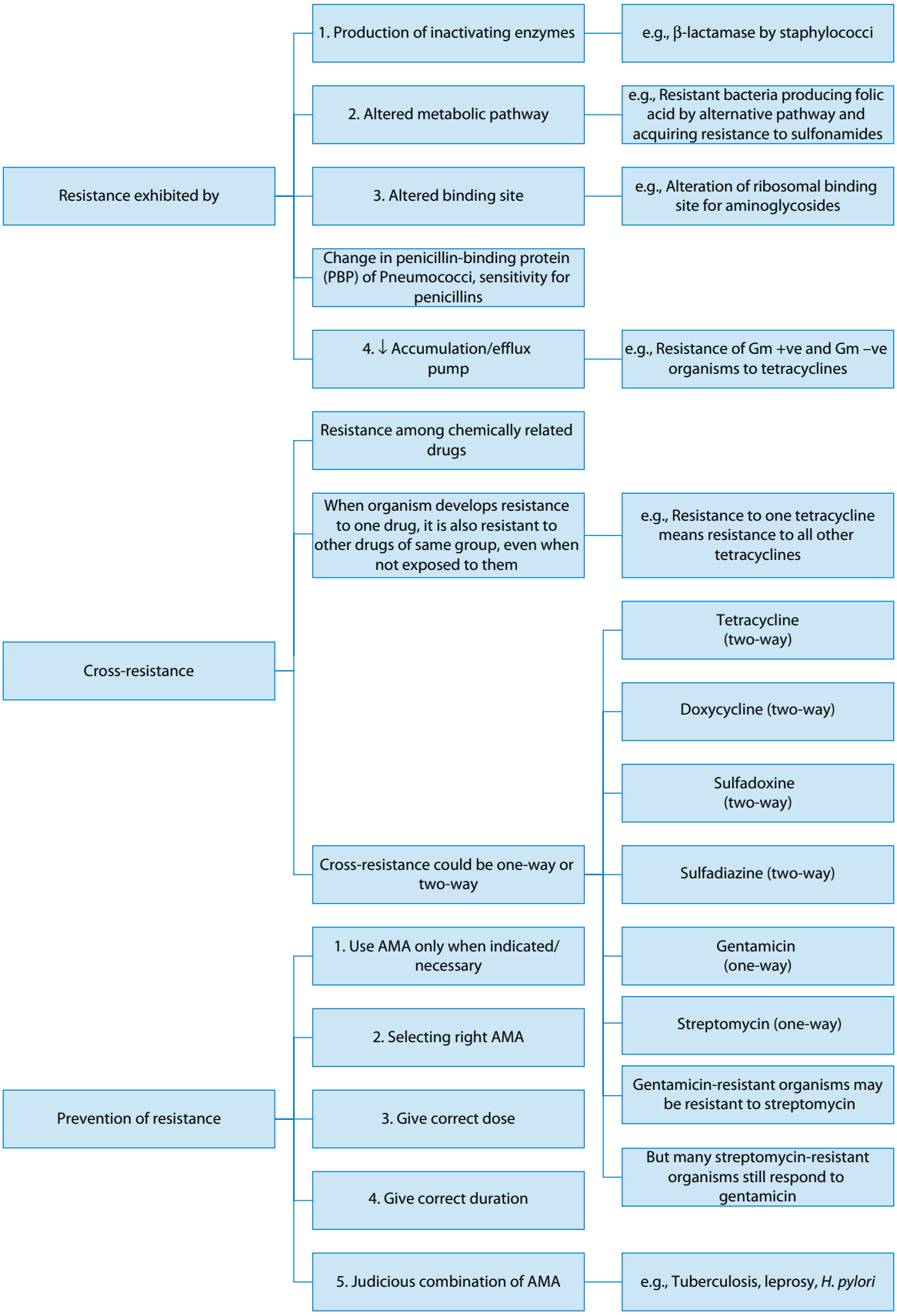
56.2 CLASSIFICATIONS



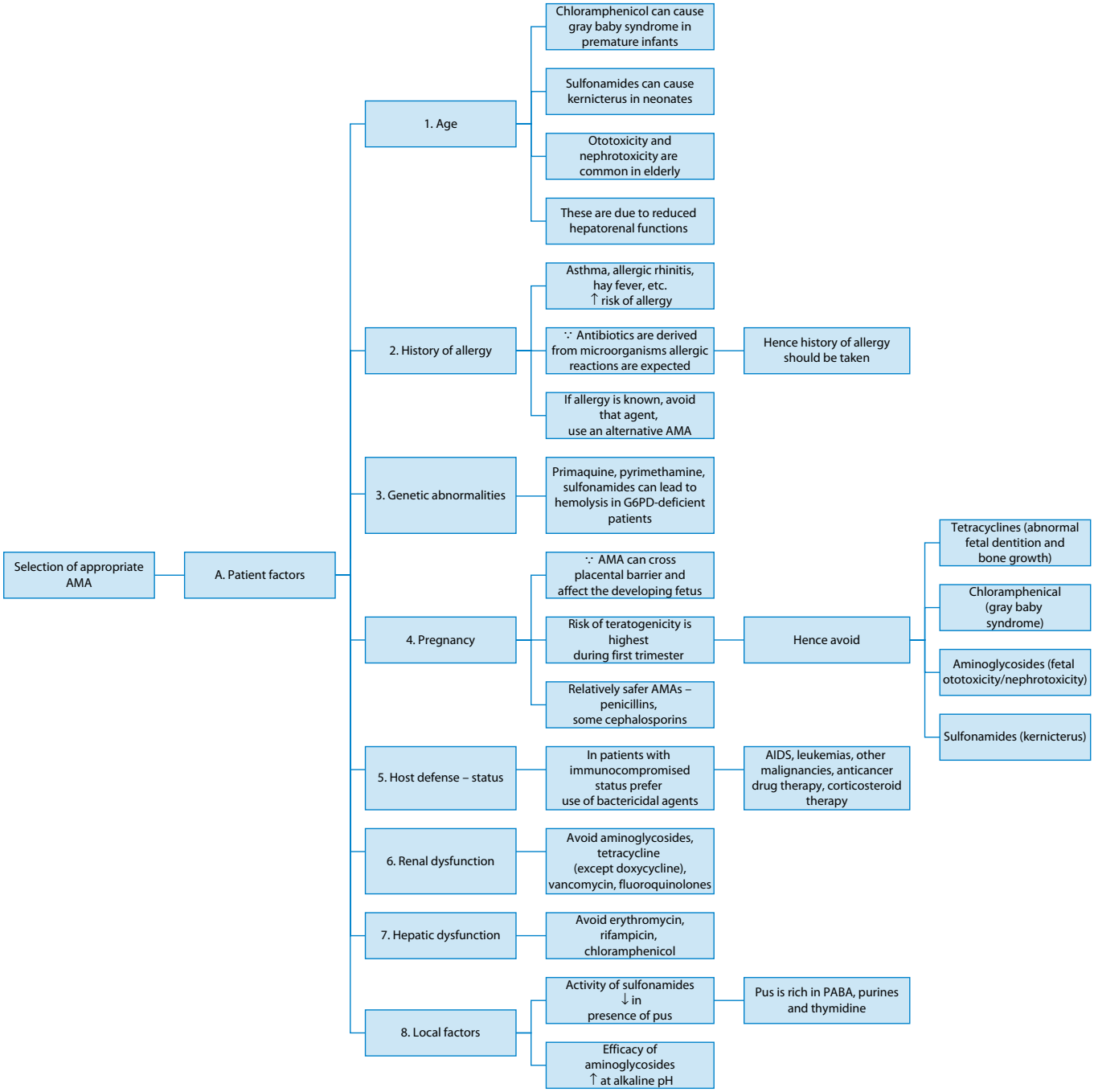
56.3 CLASSIFICATION, FACTORS INFLUENCING SUCCESSFUL CHEMOTHERAPY, AND ANTIMICROBIAL RESISTANCE



56.4 ANTIMICROBIAL RESISTANCE

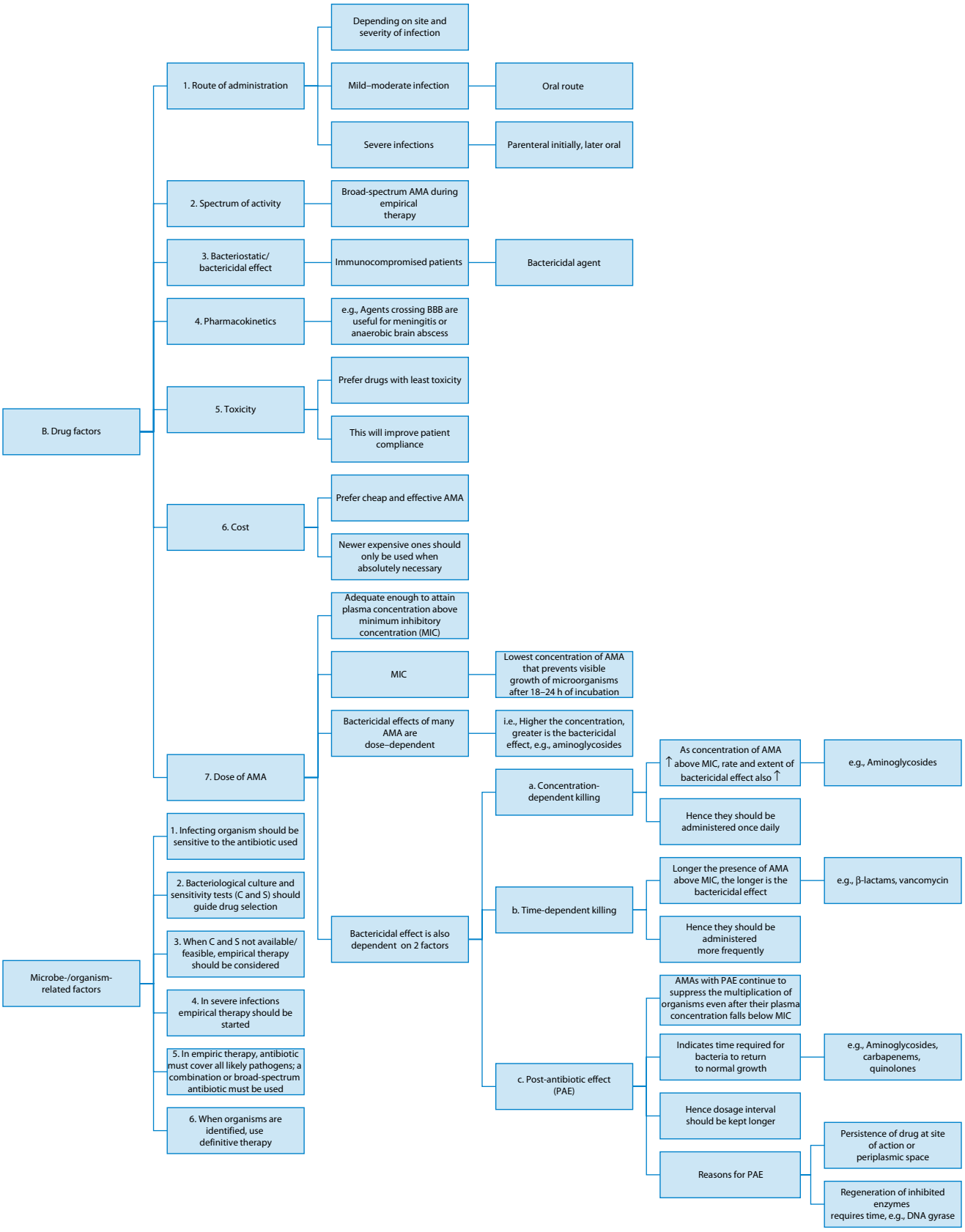


56.5 SELECTION OF APPROPRIATE AMA

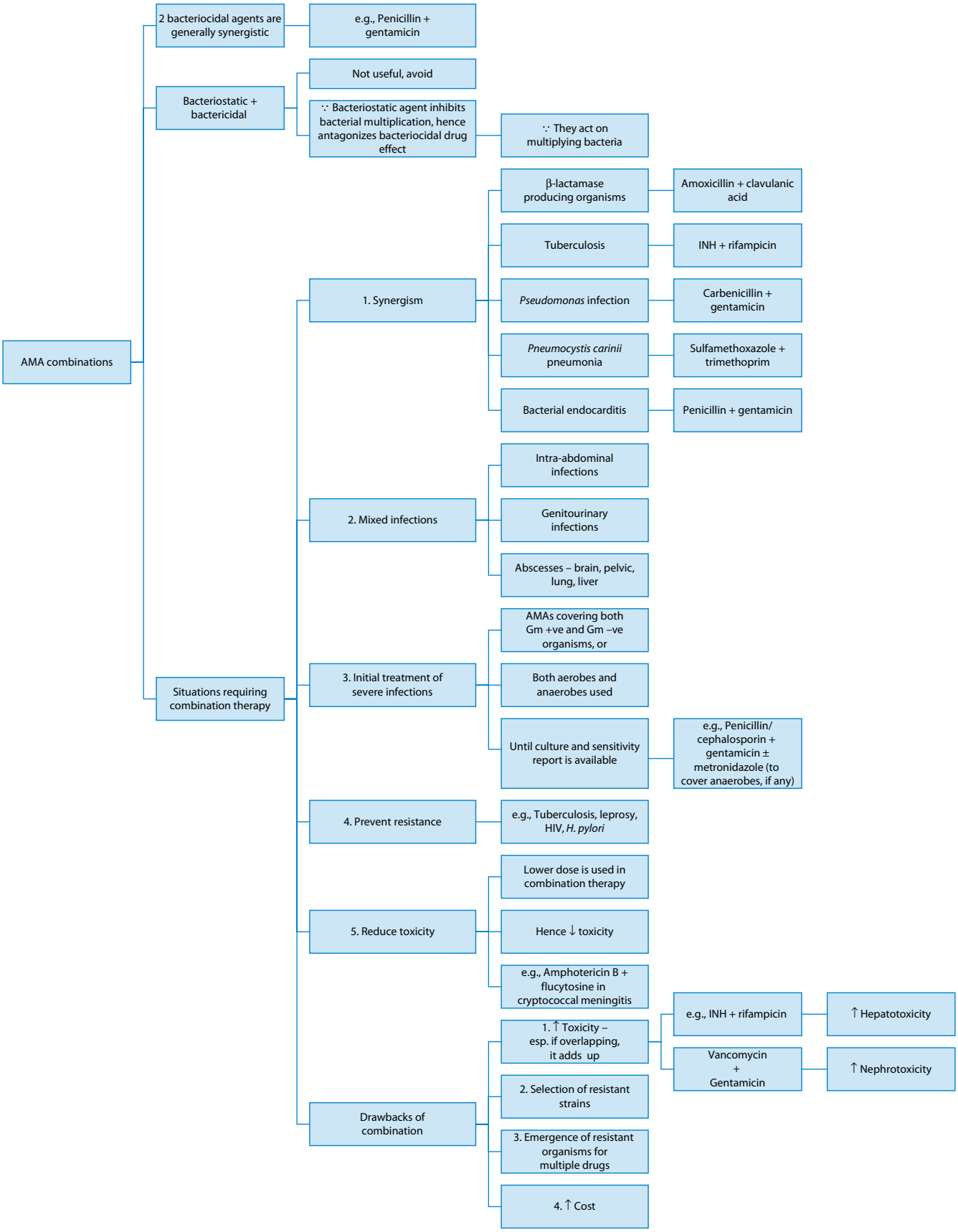


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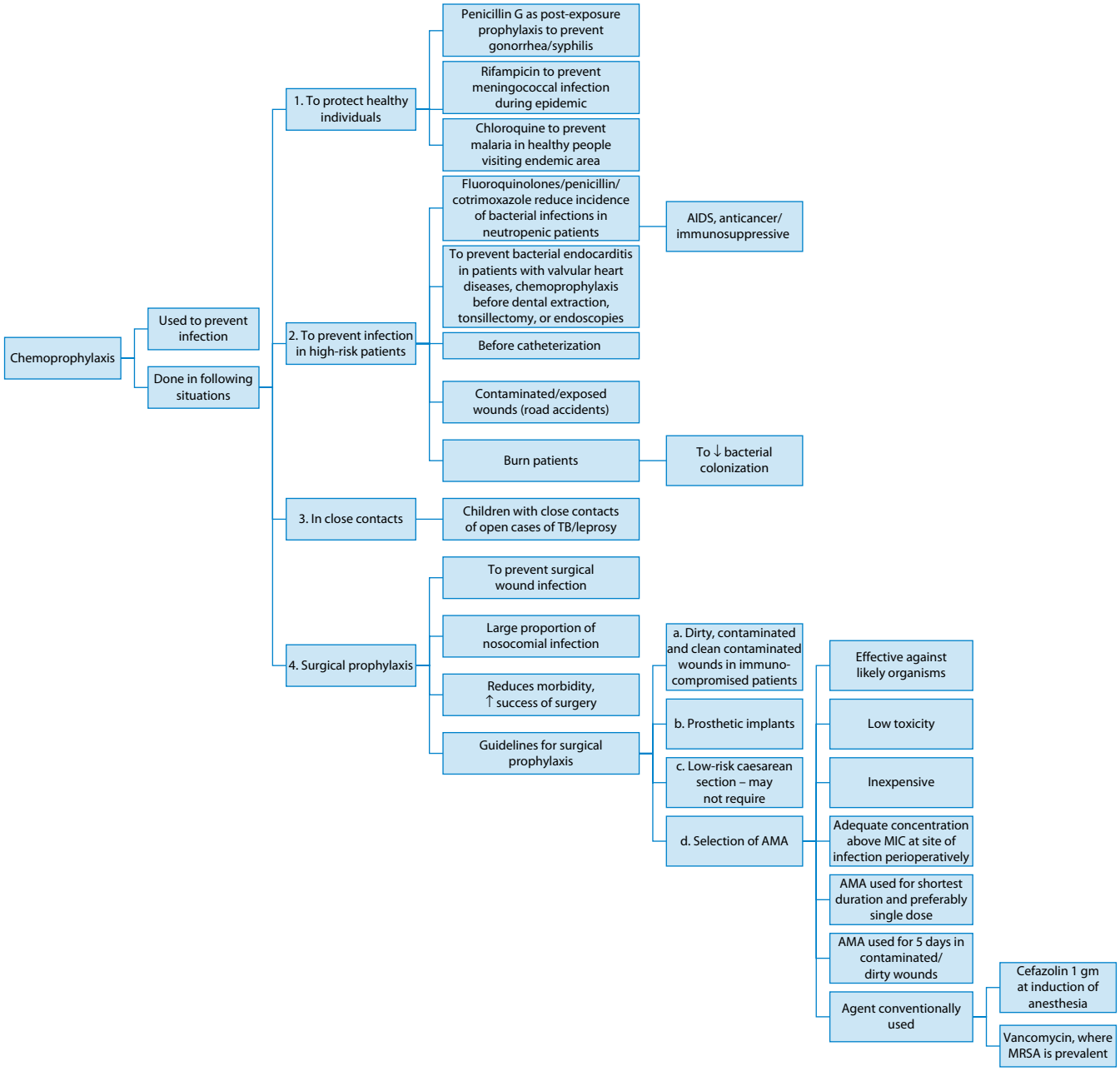
56.5 SELECTION OF APPROPRIATE AMA (Continued)



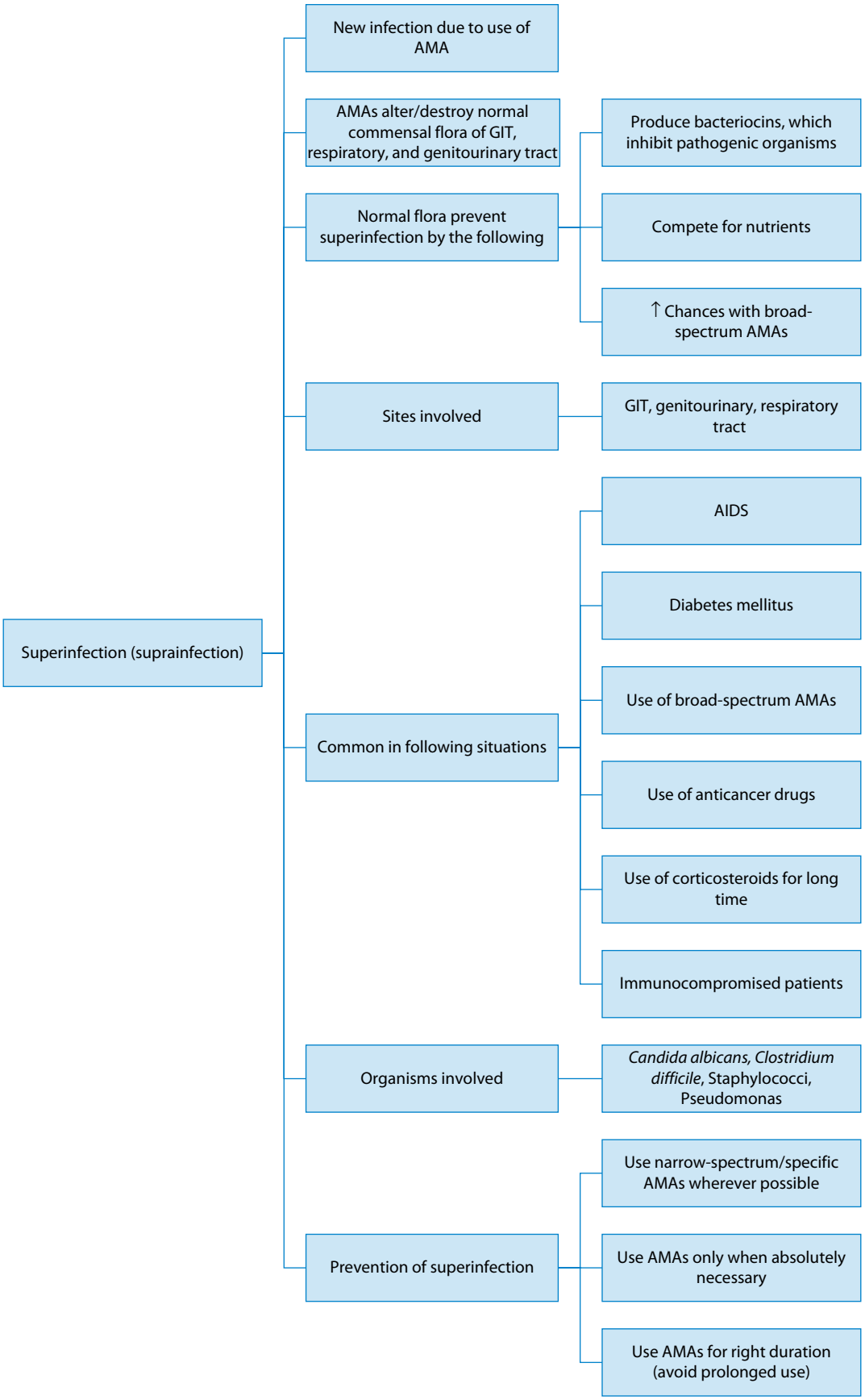
56.6 AMA COMBINATIONS



56.7 CHEMOPROPHYLAXIS



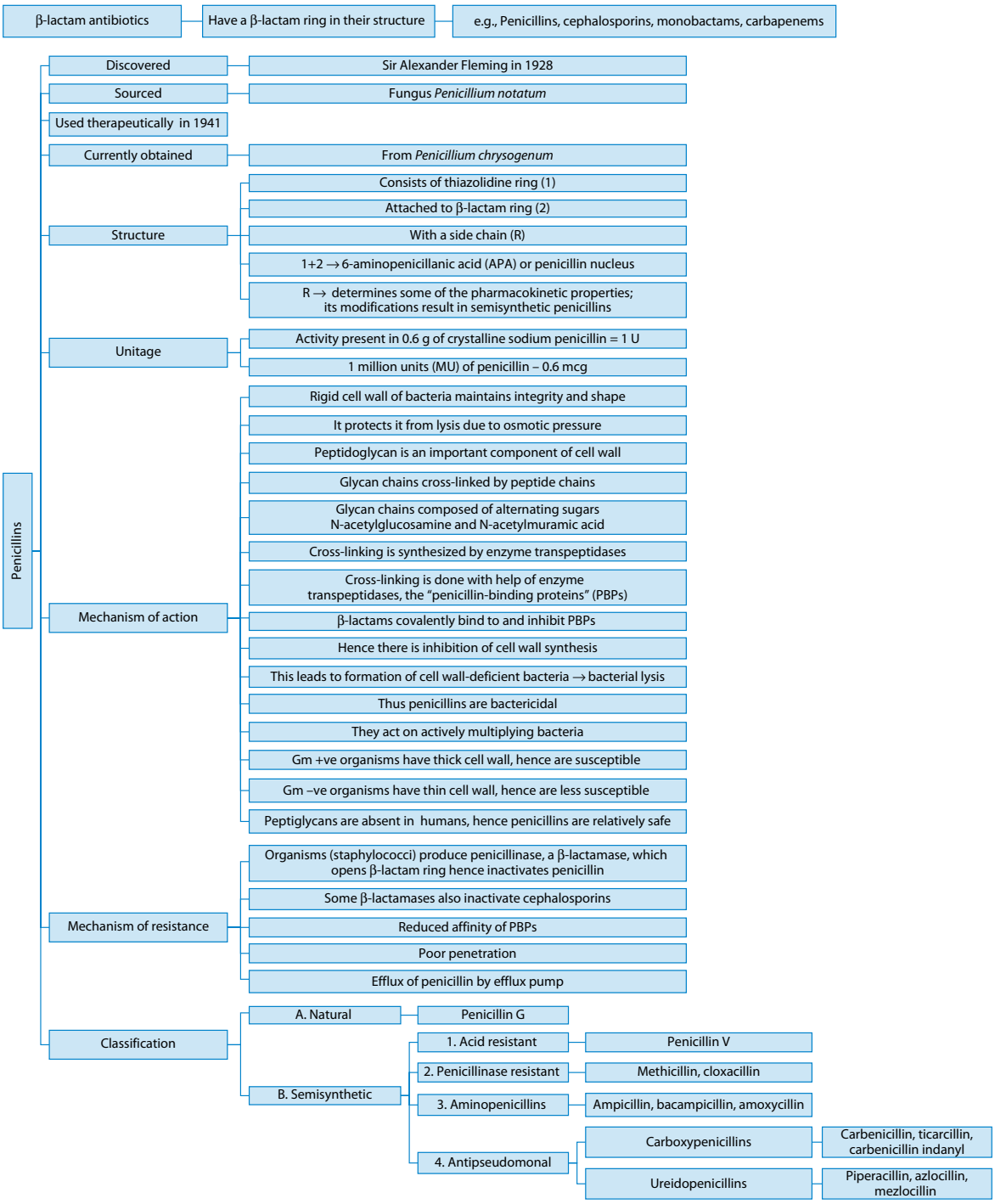
56.8 SUPERINFECTION (SUPRAINFECTION)



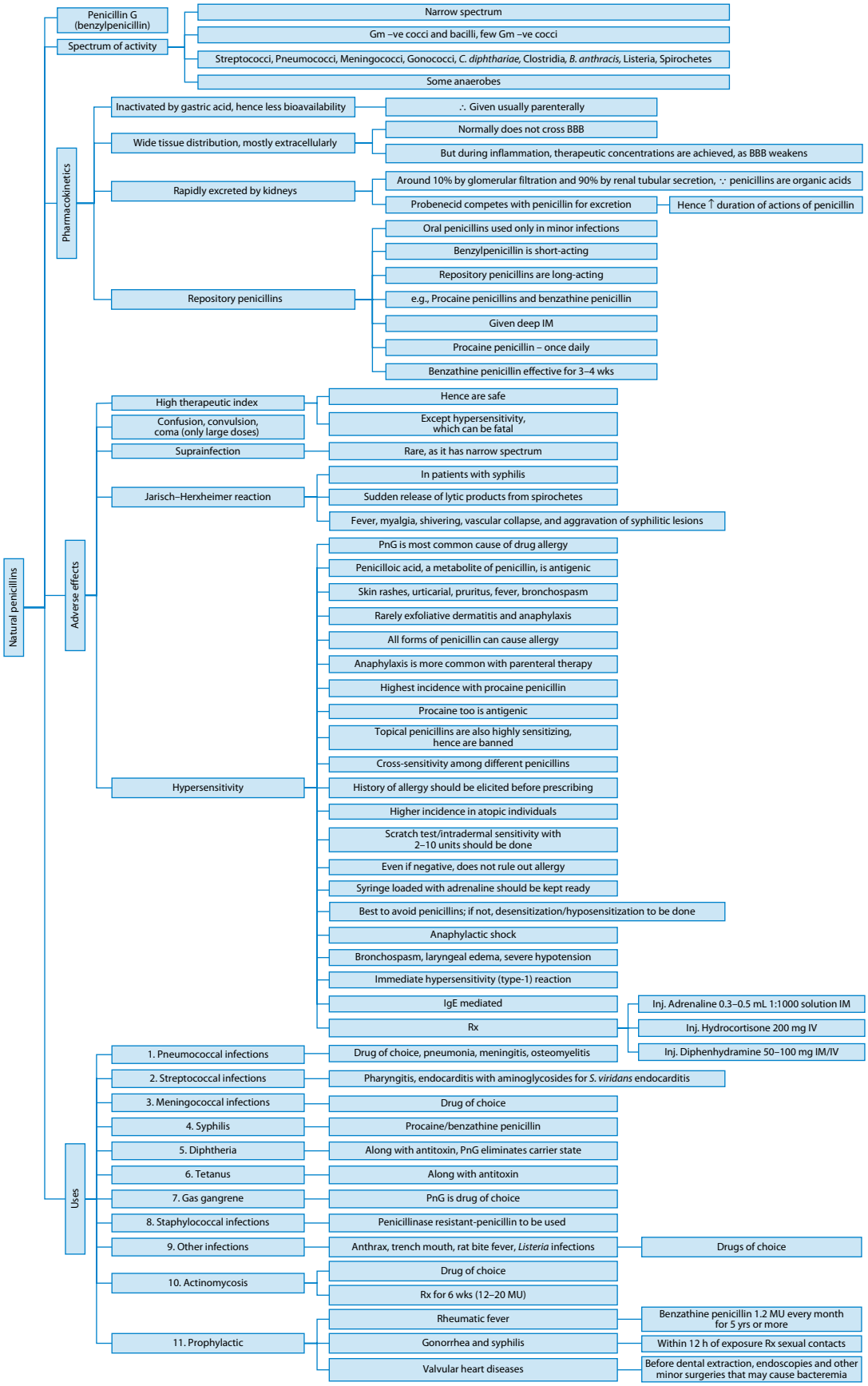
57

Beta-lactam antibiotics

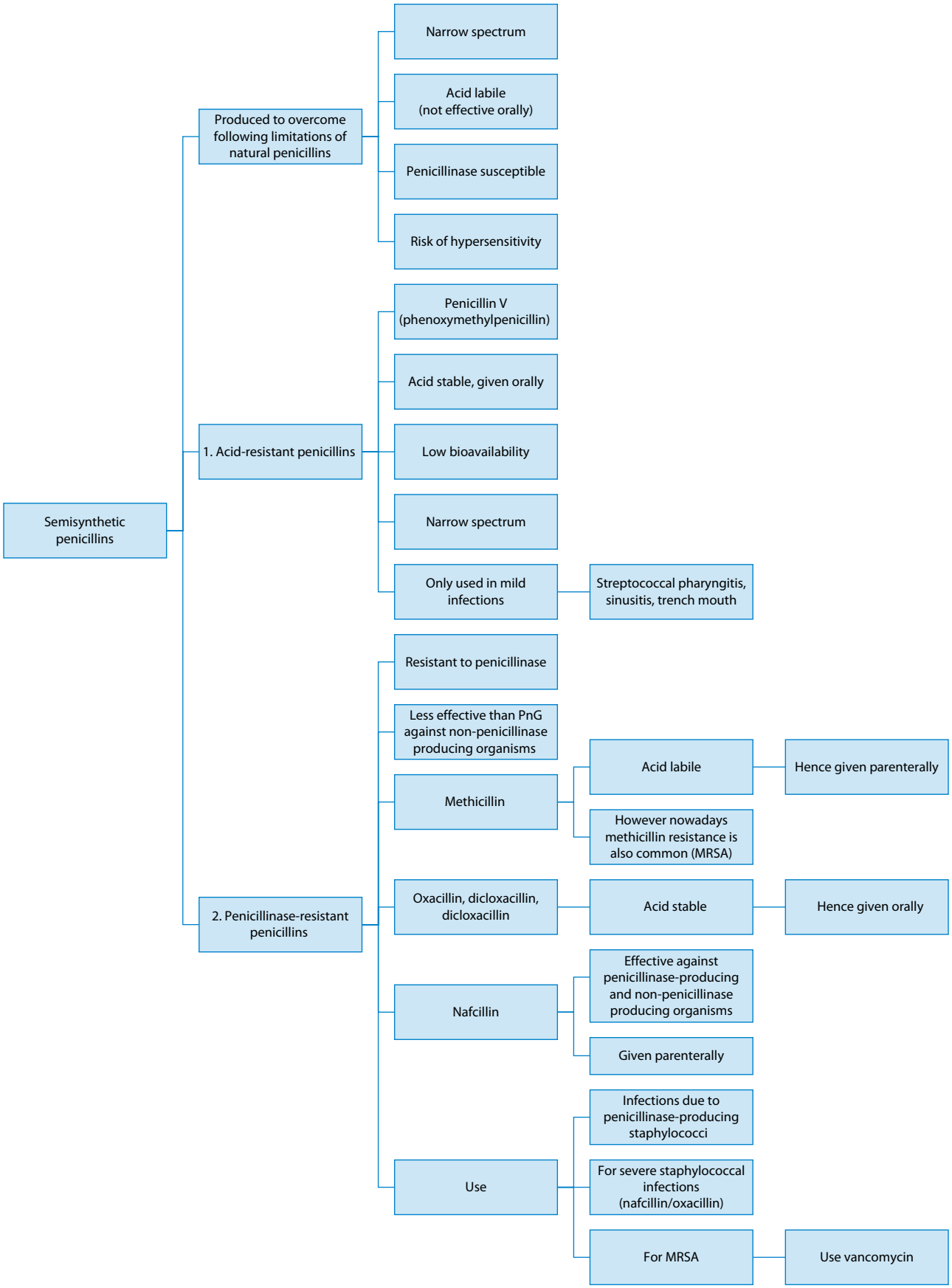
57.1 PENICILLINS



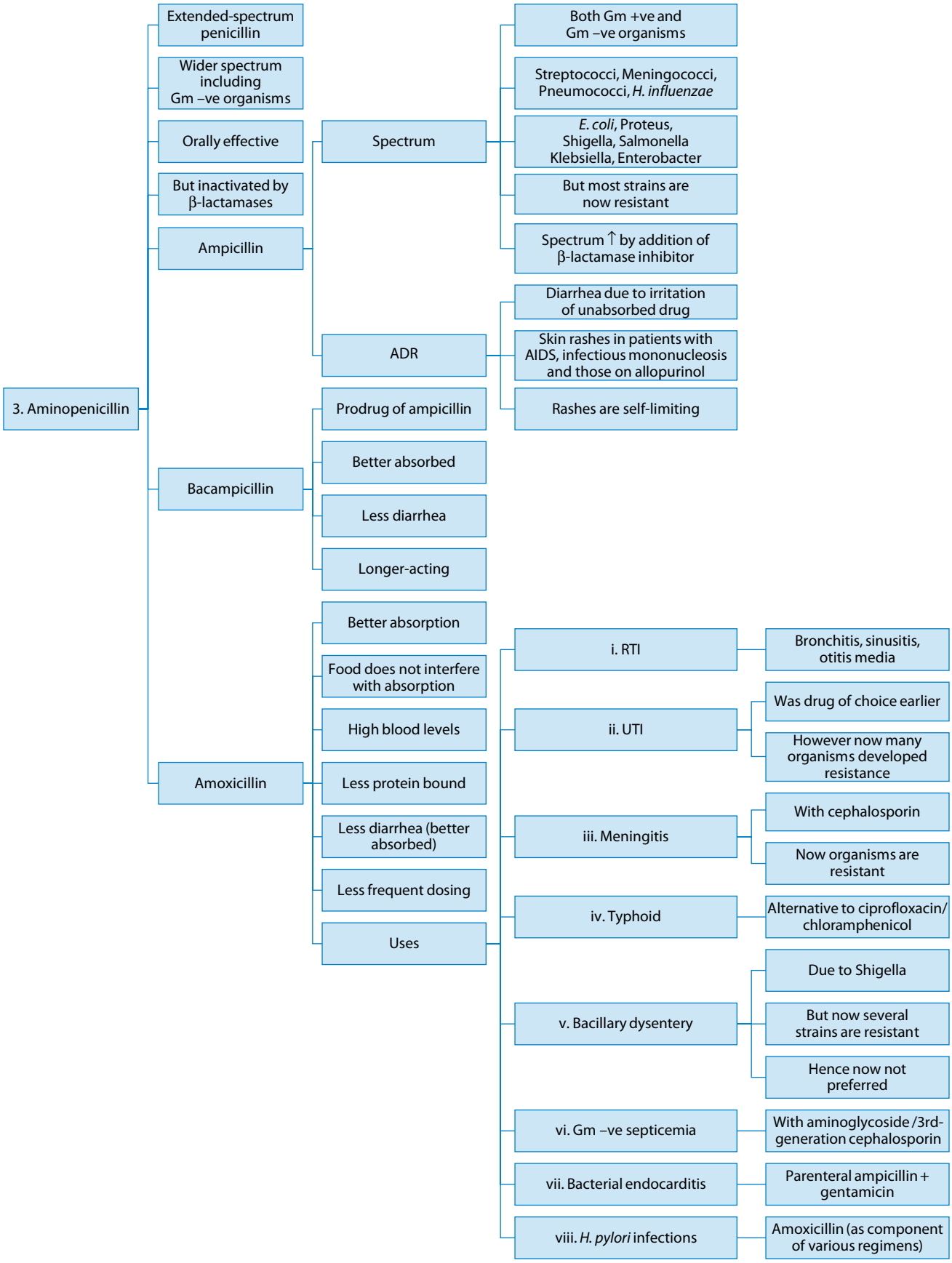
57.2 NATURAL PENICILLINS



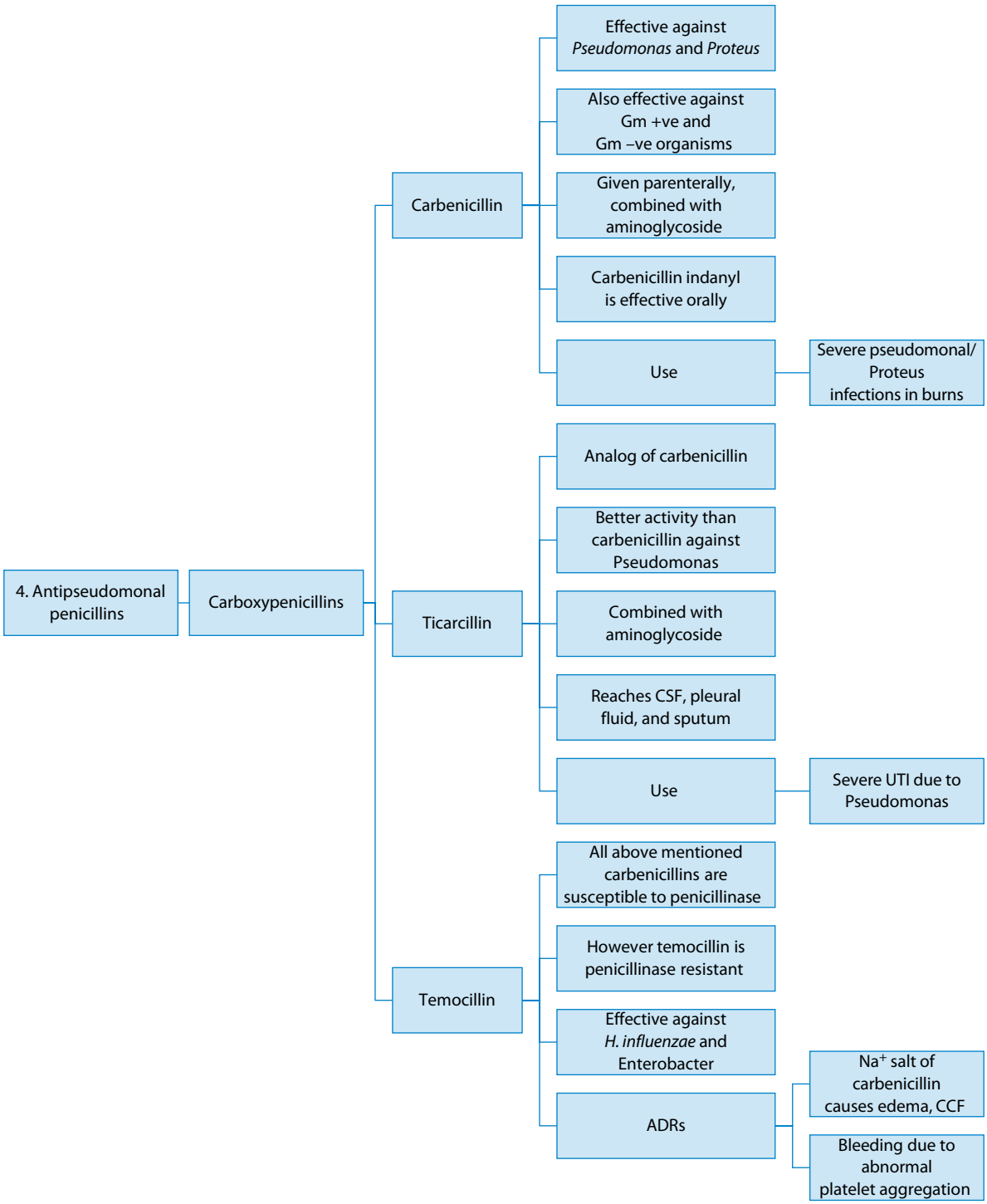
57.3 SEMISYNTHETIC PENICILLINS



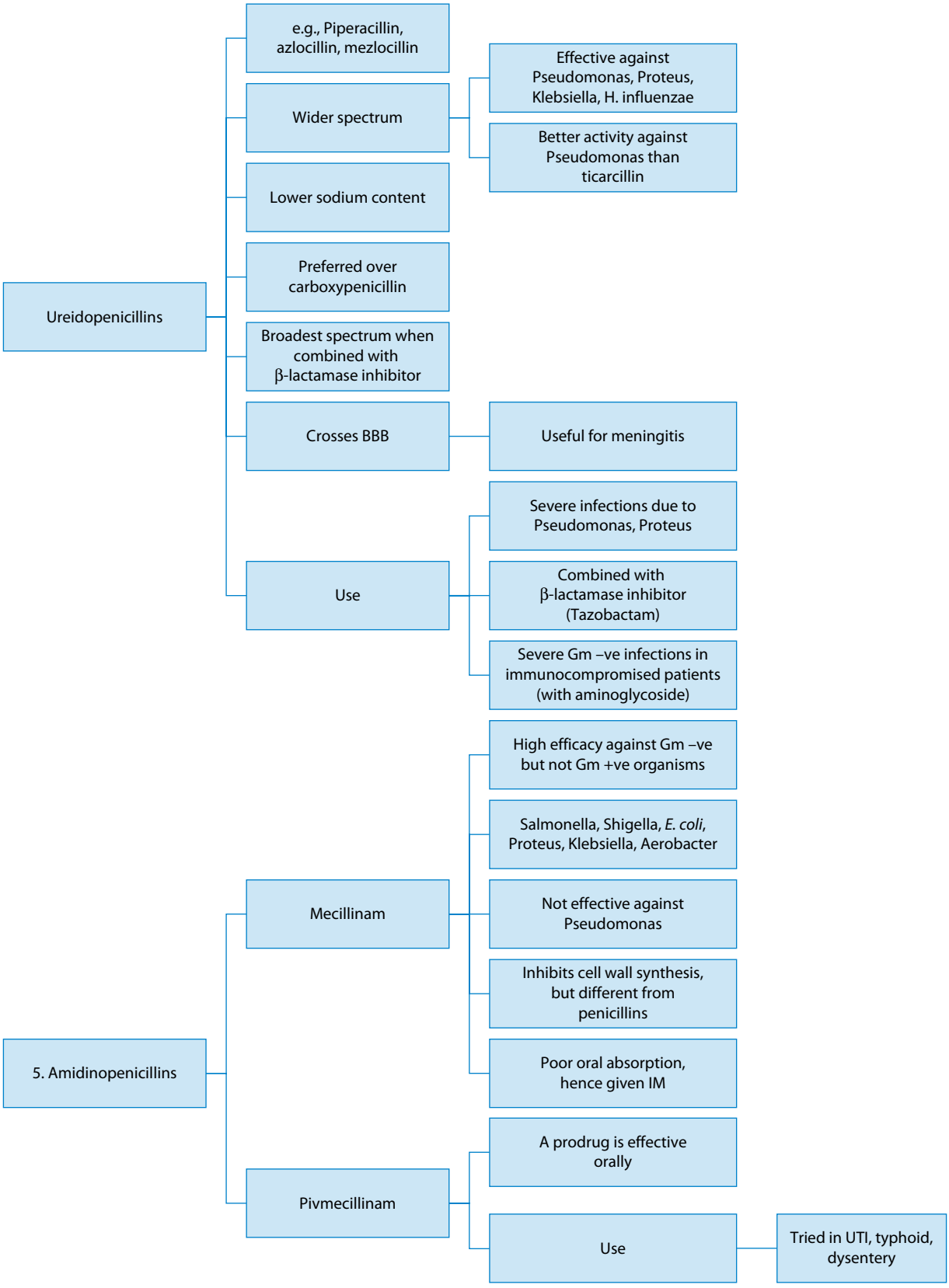
57.4 AMINOPENICILLIN



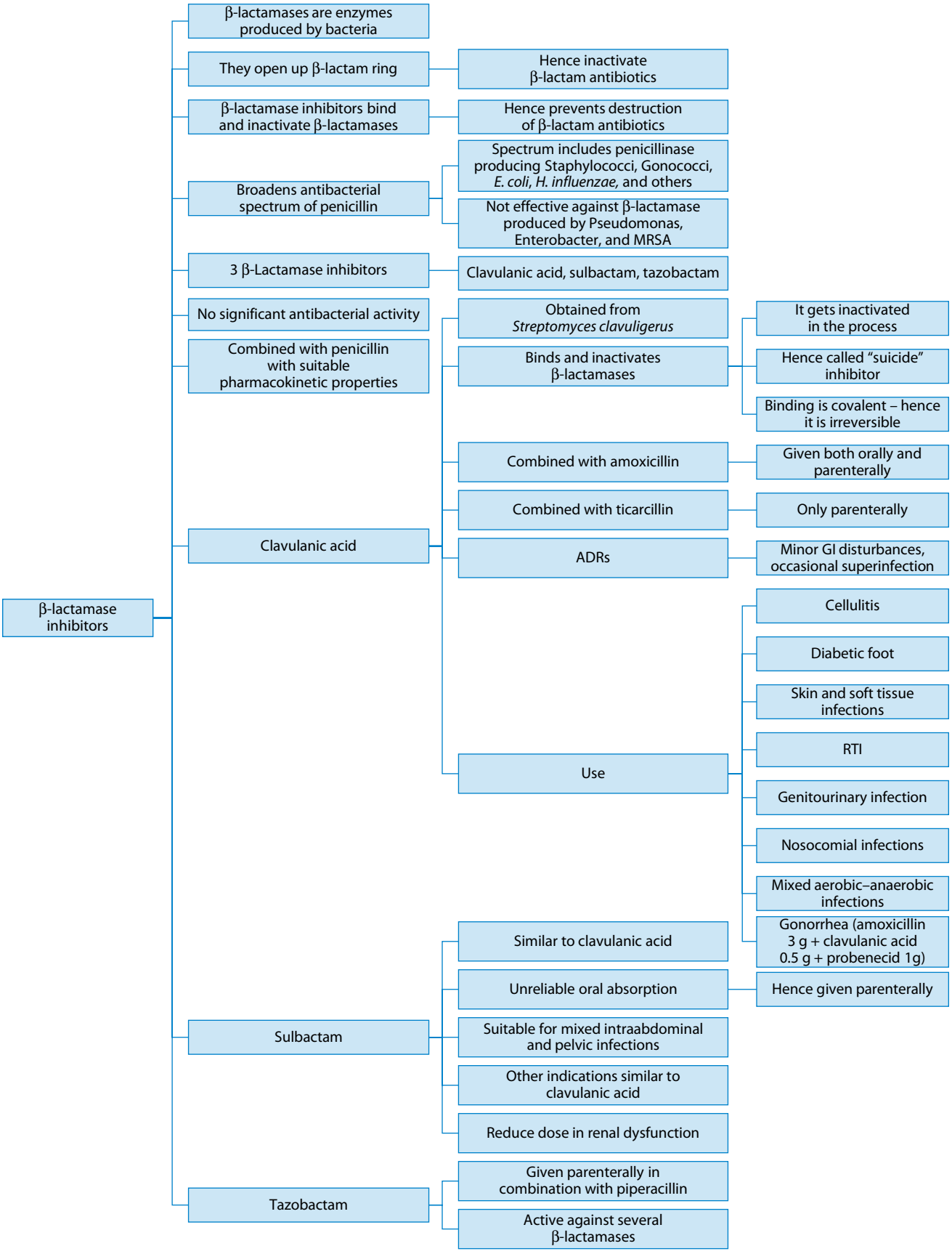
57.5 ANTIPSEUDOMONAL PENICILLINS



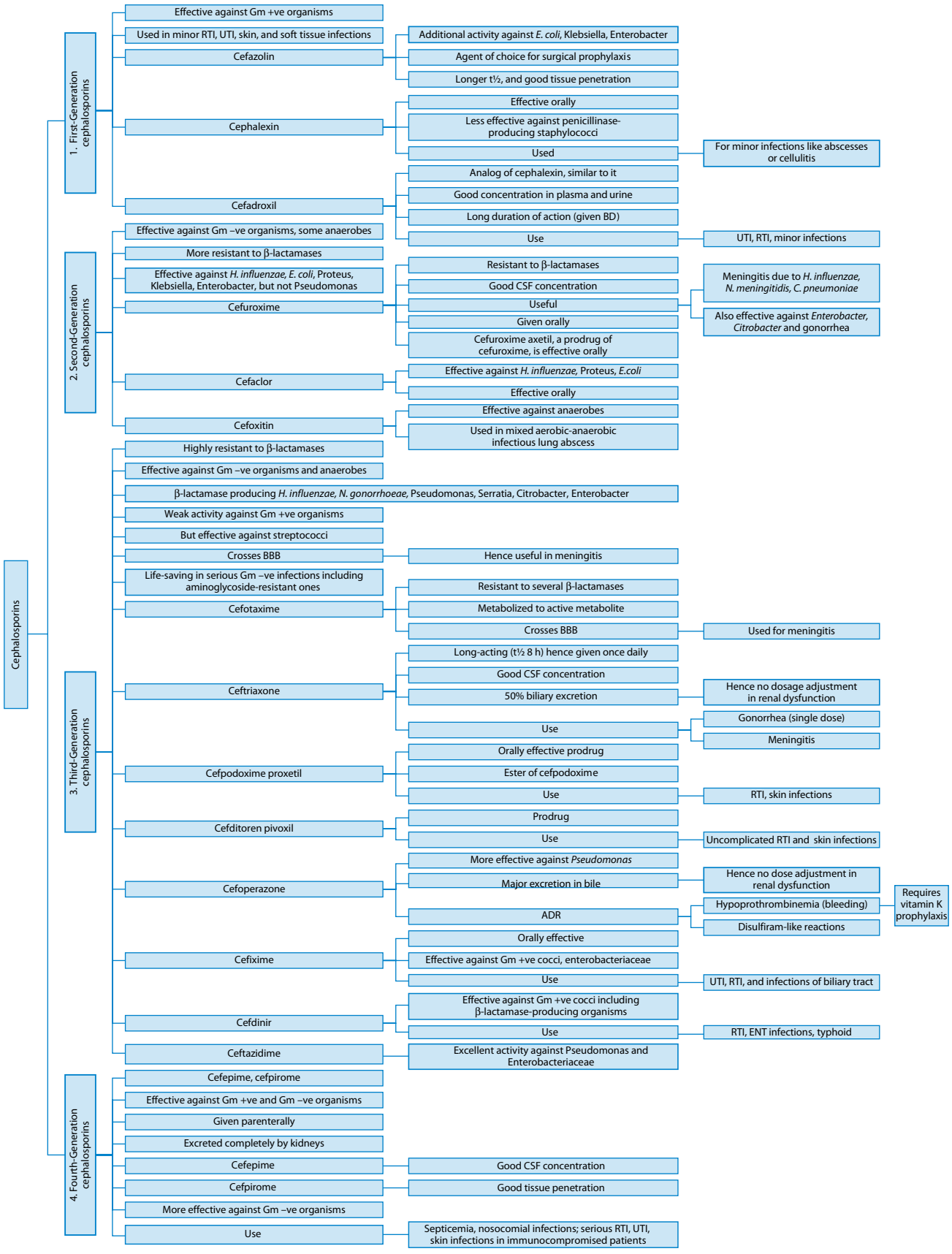
57.6 UREIDOPENICILLINS AND AMIDINOPENICILLINS



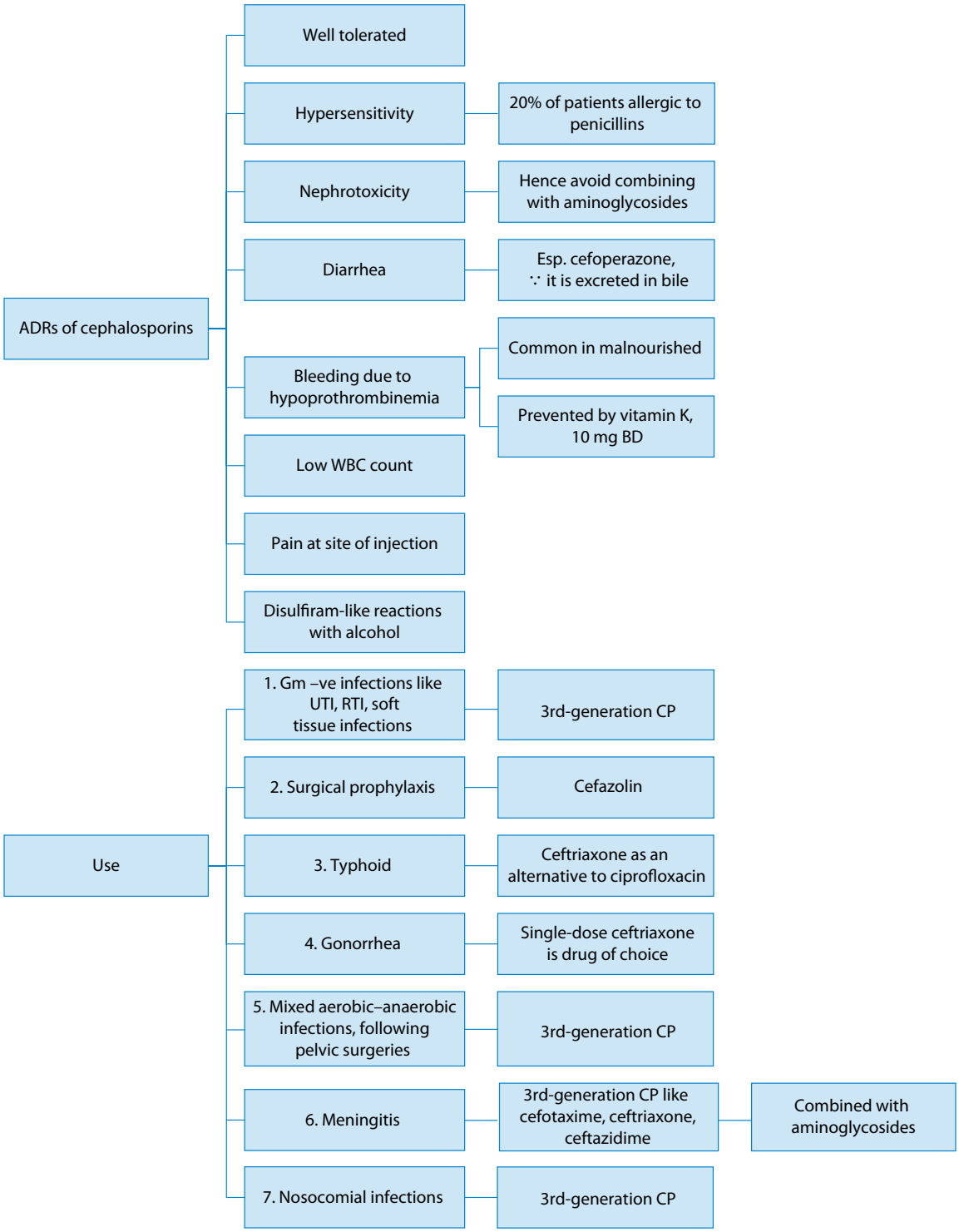
57.7 β-LACTAMASE INHIBITORS



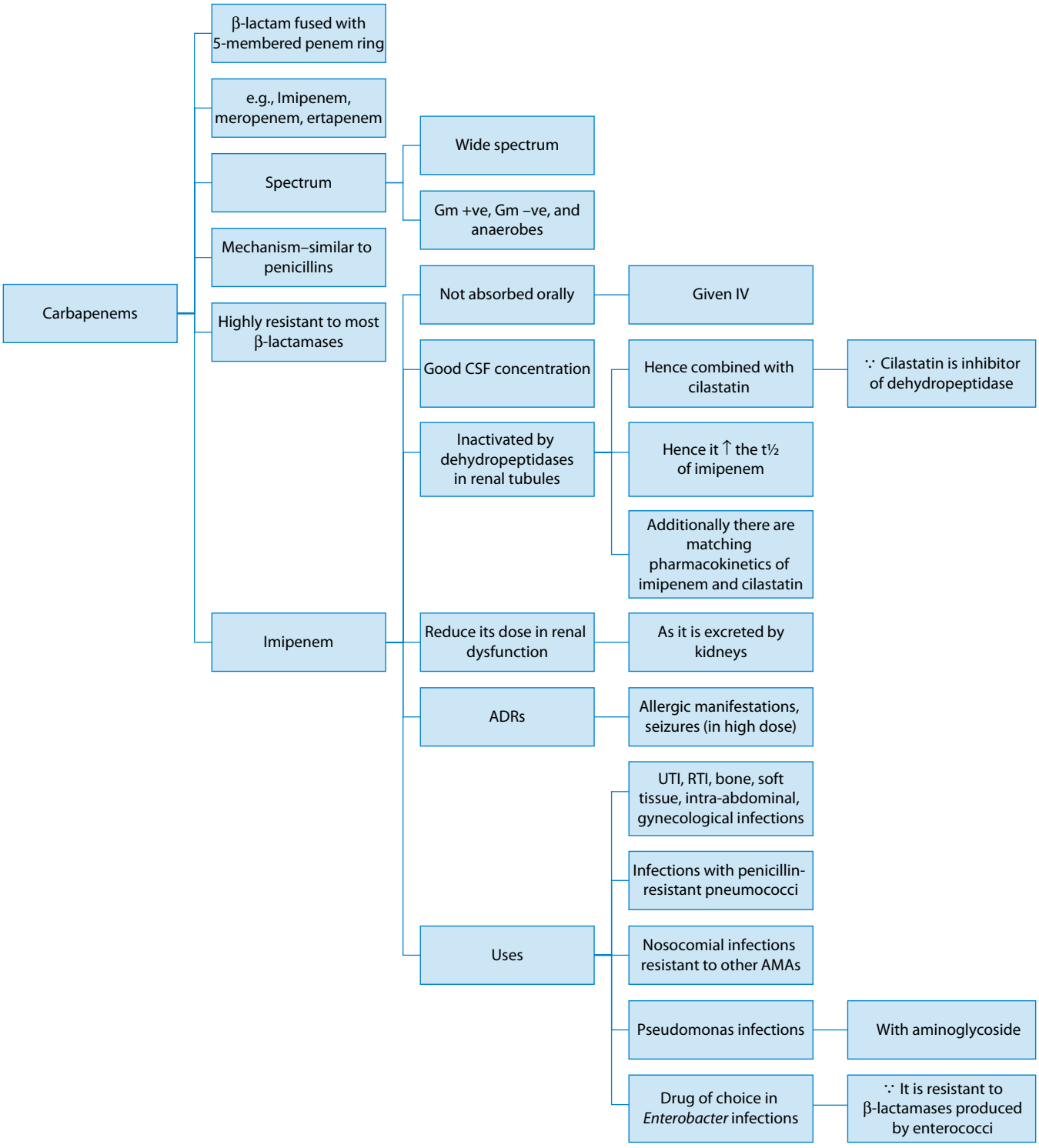
57.8 CEPHALOSPORINS



57.9 CEPHALOSPORINS – ADRs AND USE

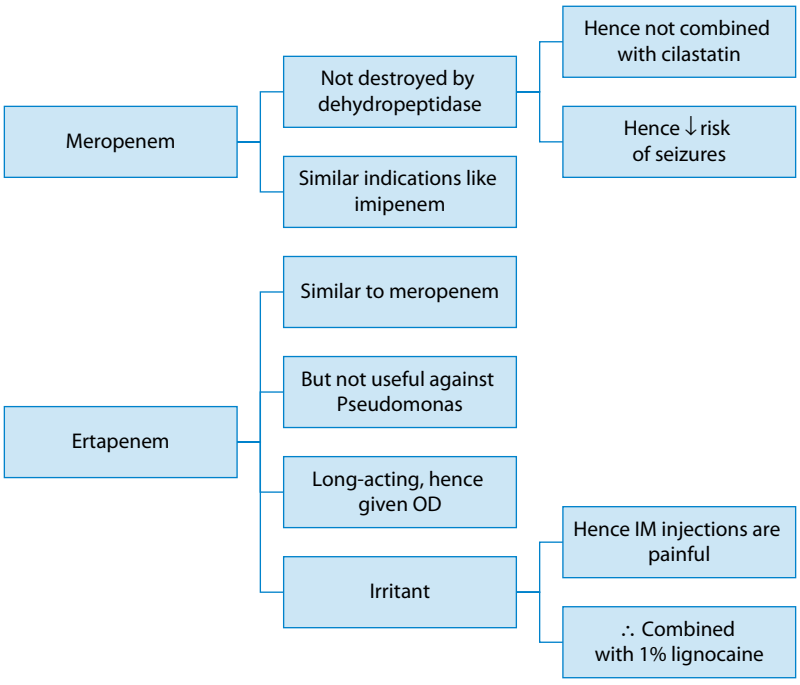


57.10 CARBAPENEMS

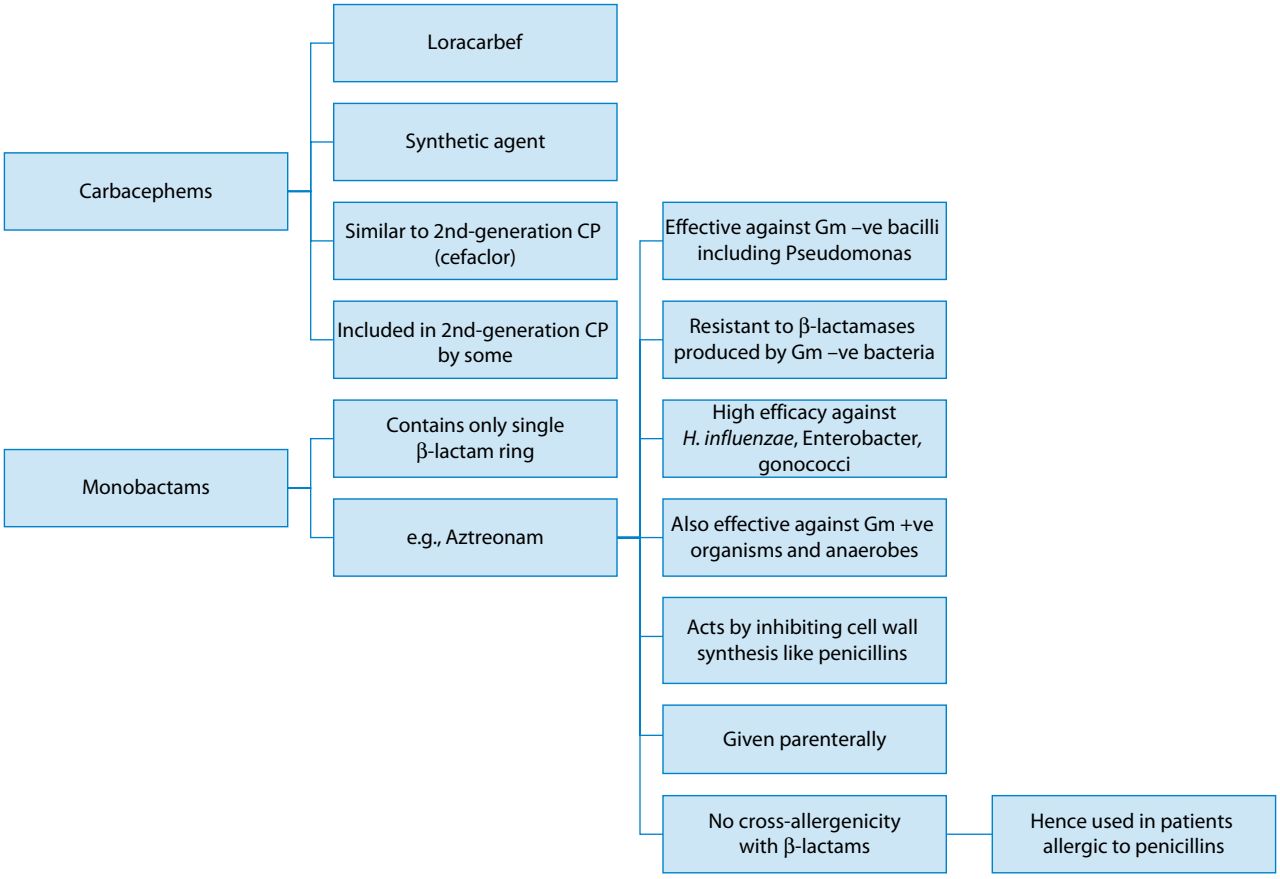


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57.10 CARBAPENEMS (Continued)



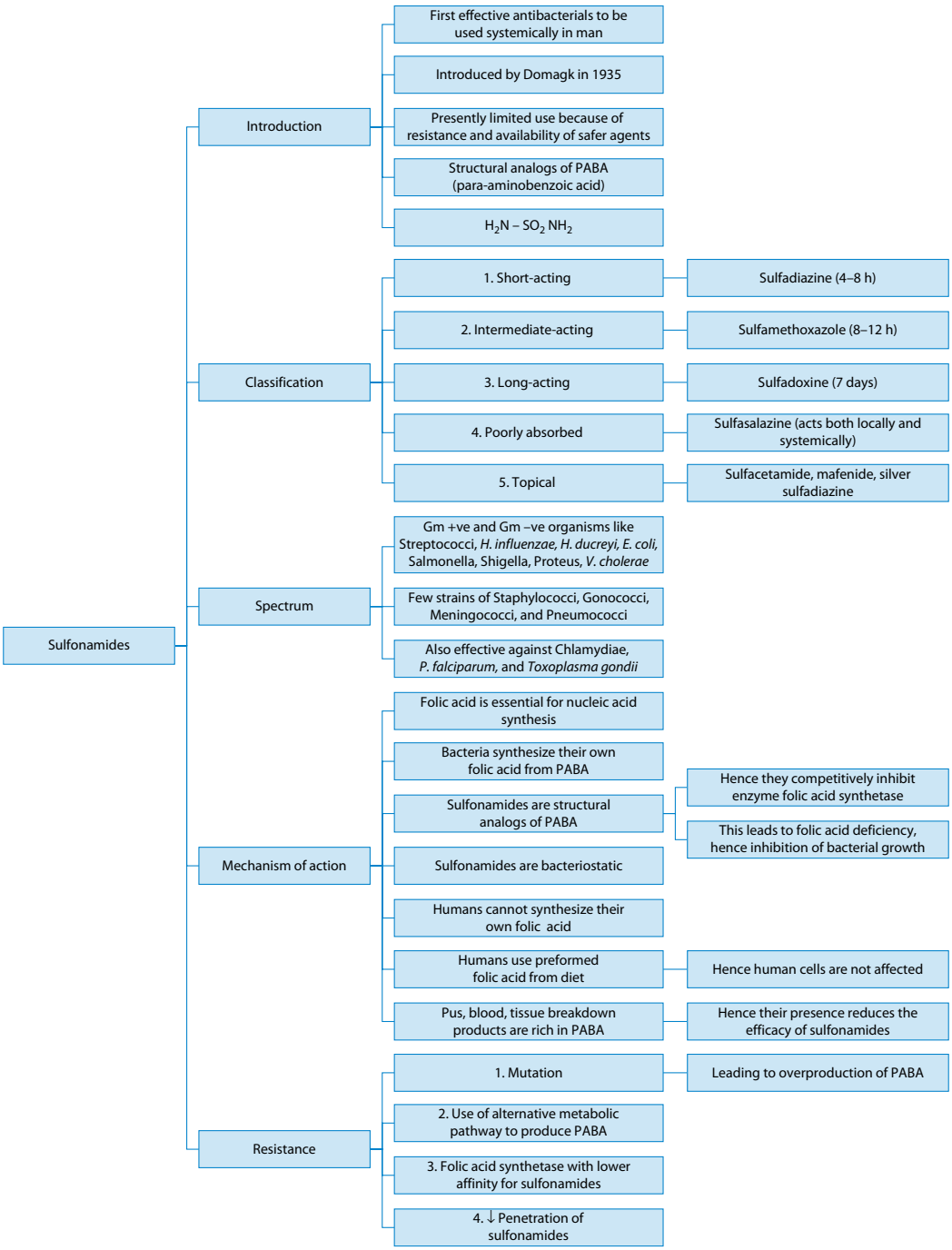
57.11 CARBACEPHEMS AND MONOBACTAMS



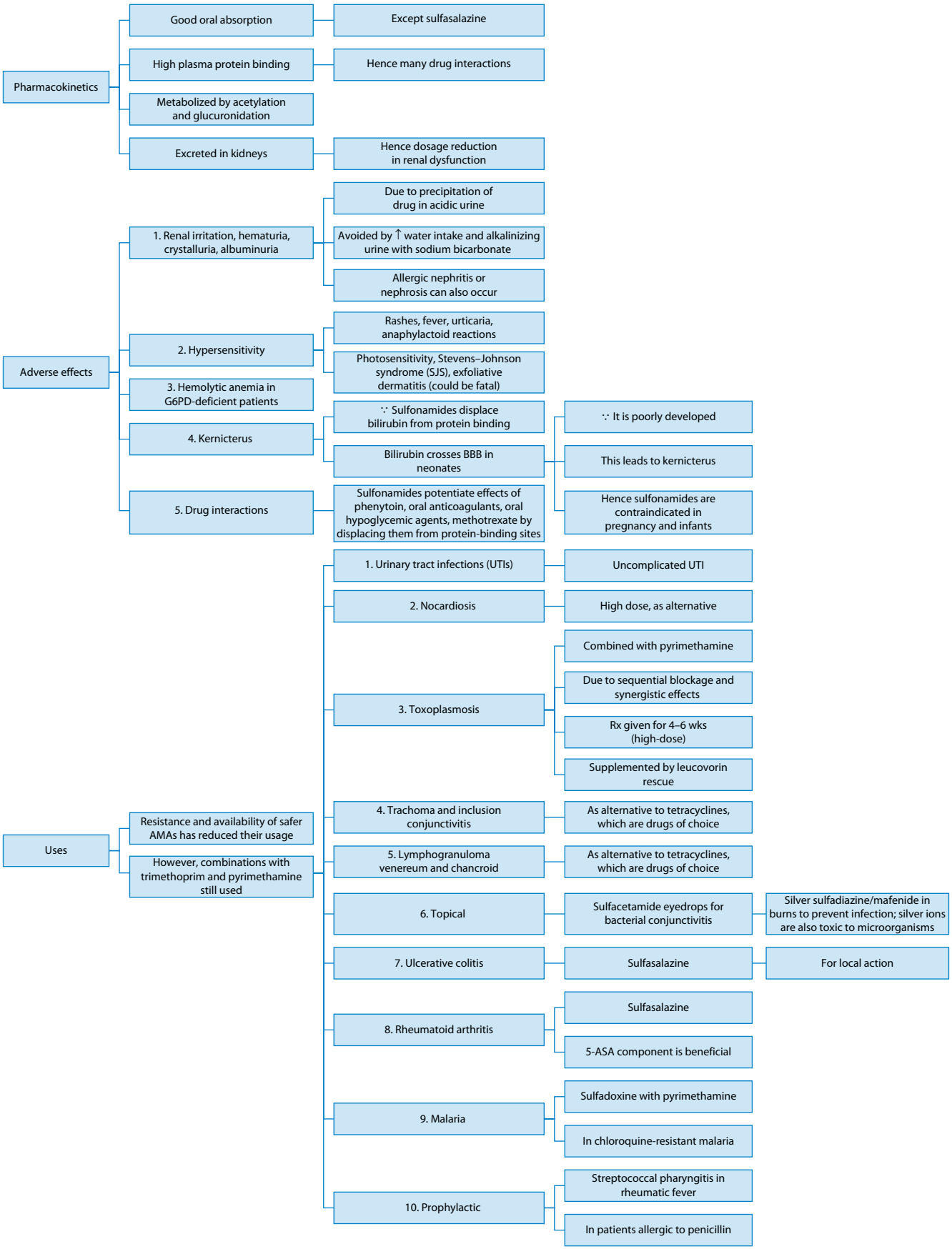
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Sulfonamides

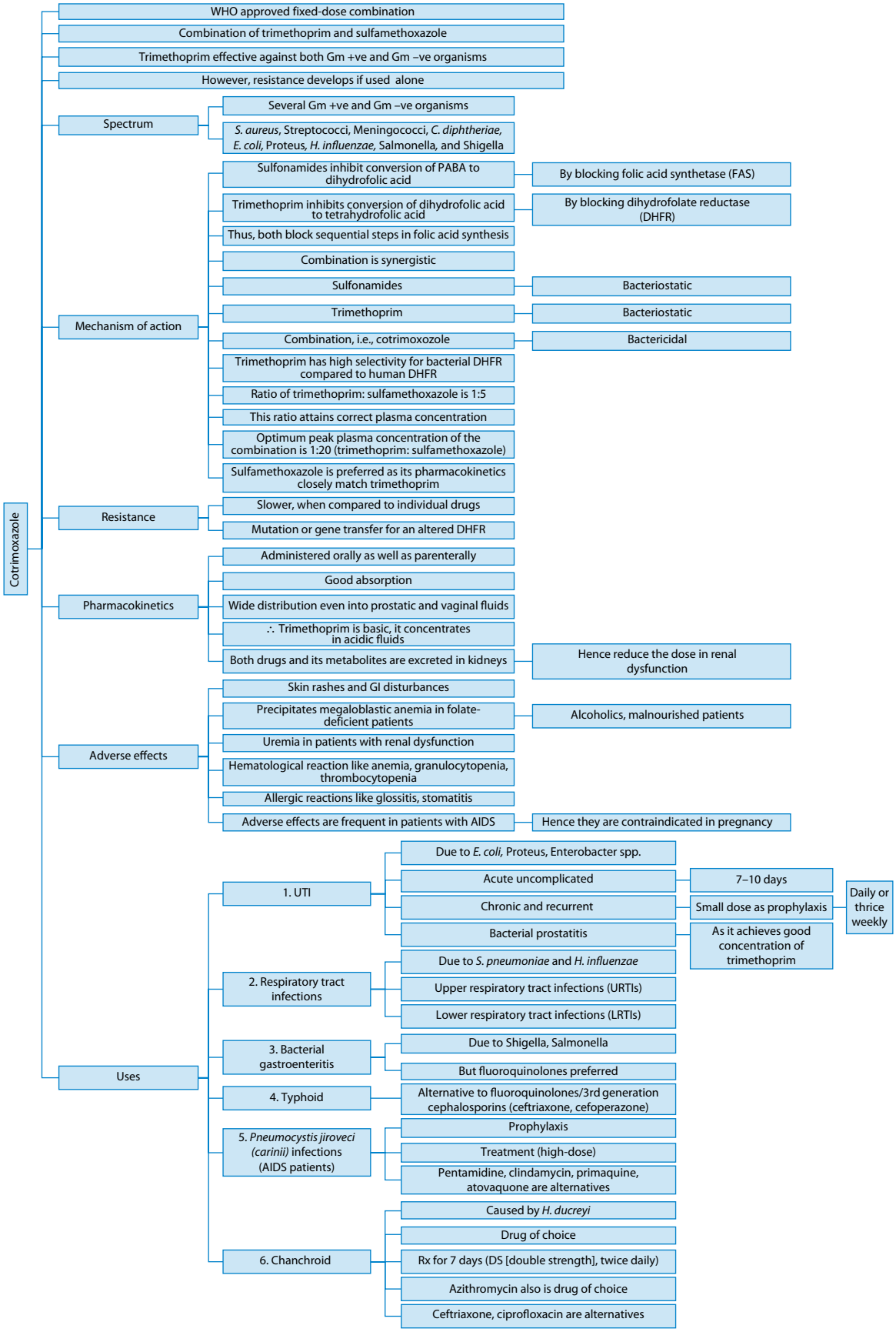
58.1 SULFONAMIDES – INTRODUCTION, CLASSIFICATION, SPECTRUM, MECHANISM OF ACTION, AND RESISTANCE



58.2 SULFONAMIDES – PHARMACOKINETICS, ADVERSE EFFECTS, AND USES



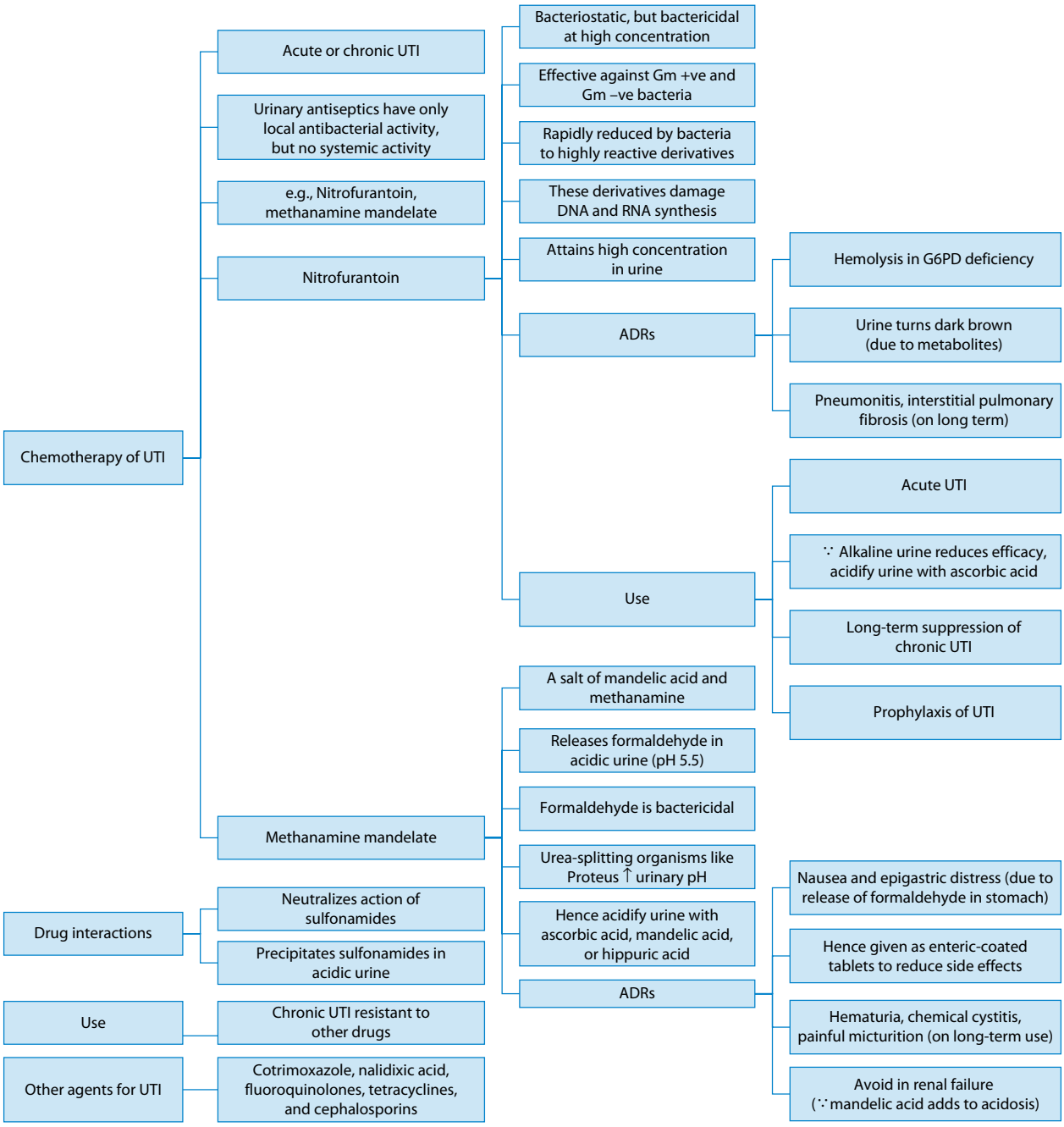
58.3 COTRIMOXAZOLE



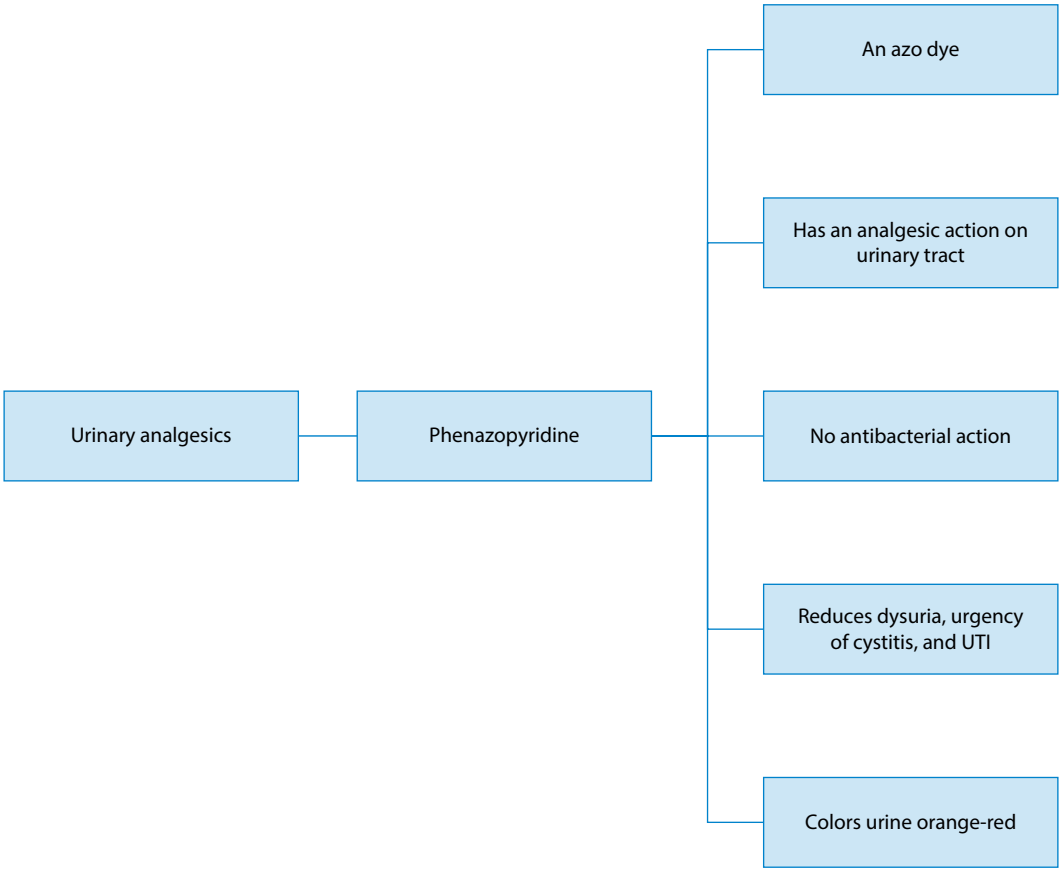
59

Chemotherapy of urinary tract infections and sexually transmitted diseases

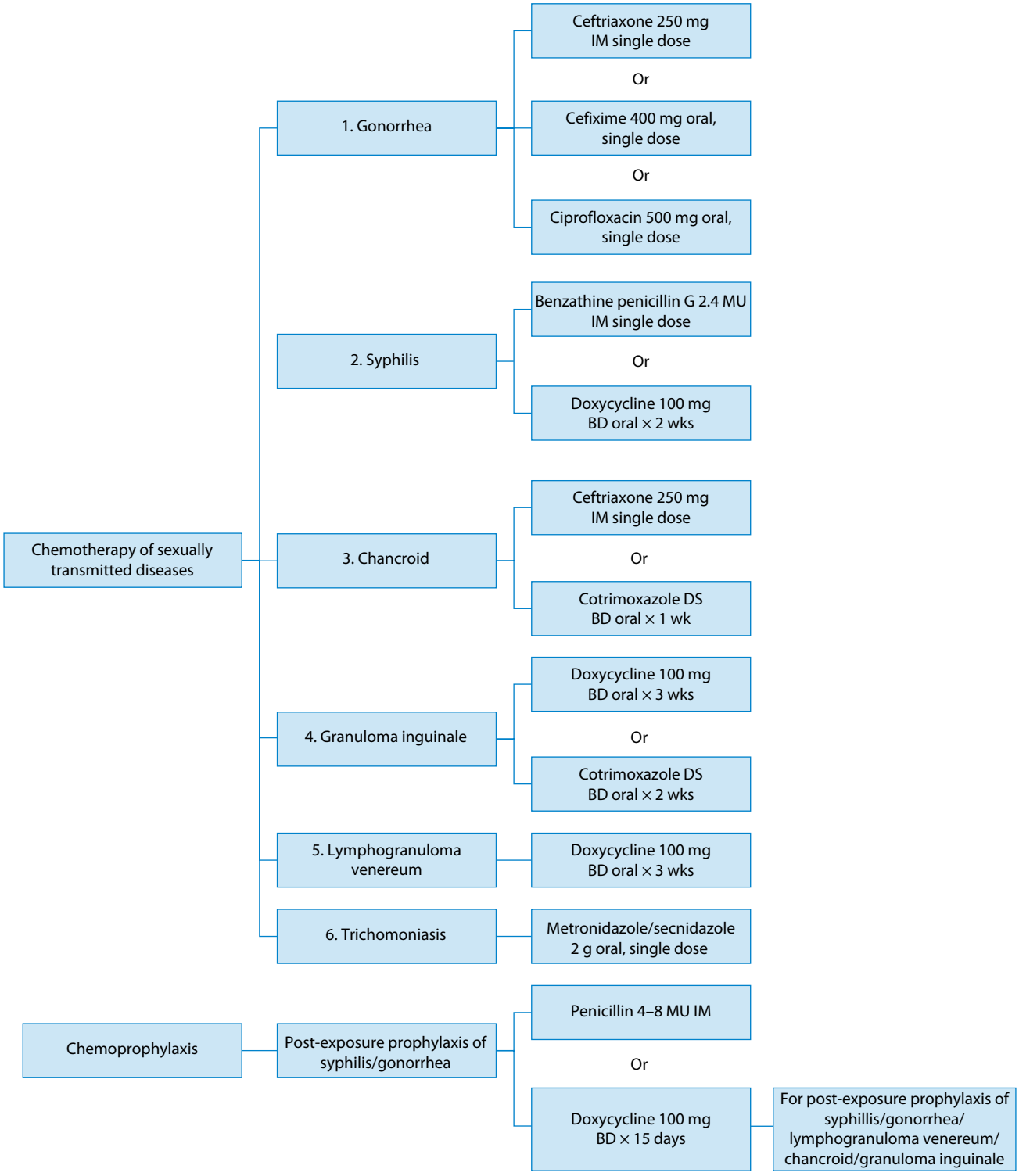
59.1 CHEMOTHERAPY OF UTI – ANTIMICROBIALS



59.2 URINARY ANALGESICS



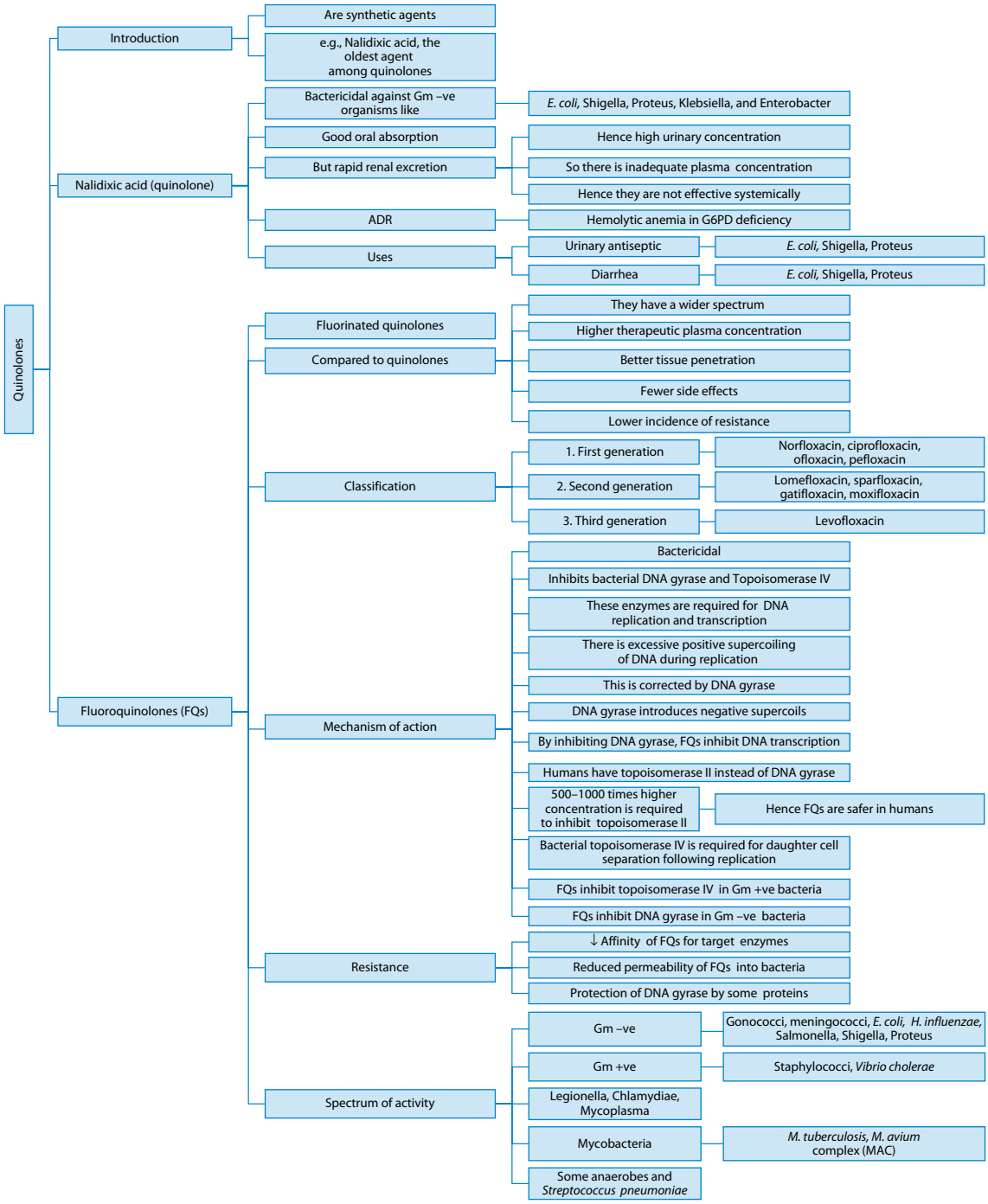
59.3 CHEMOTHERAPY OF SEXUALLY TRANSMITTED DISEASES



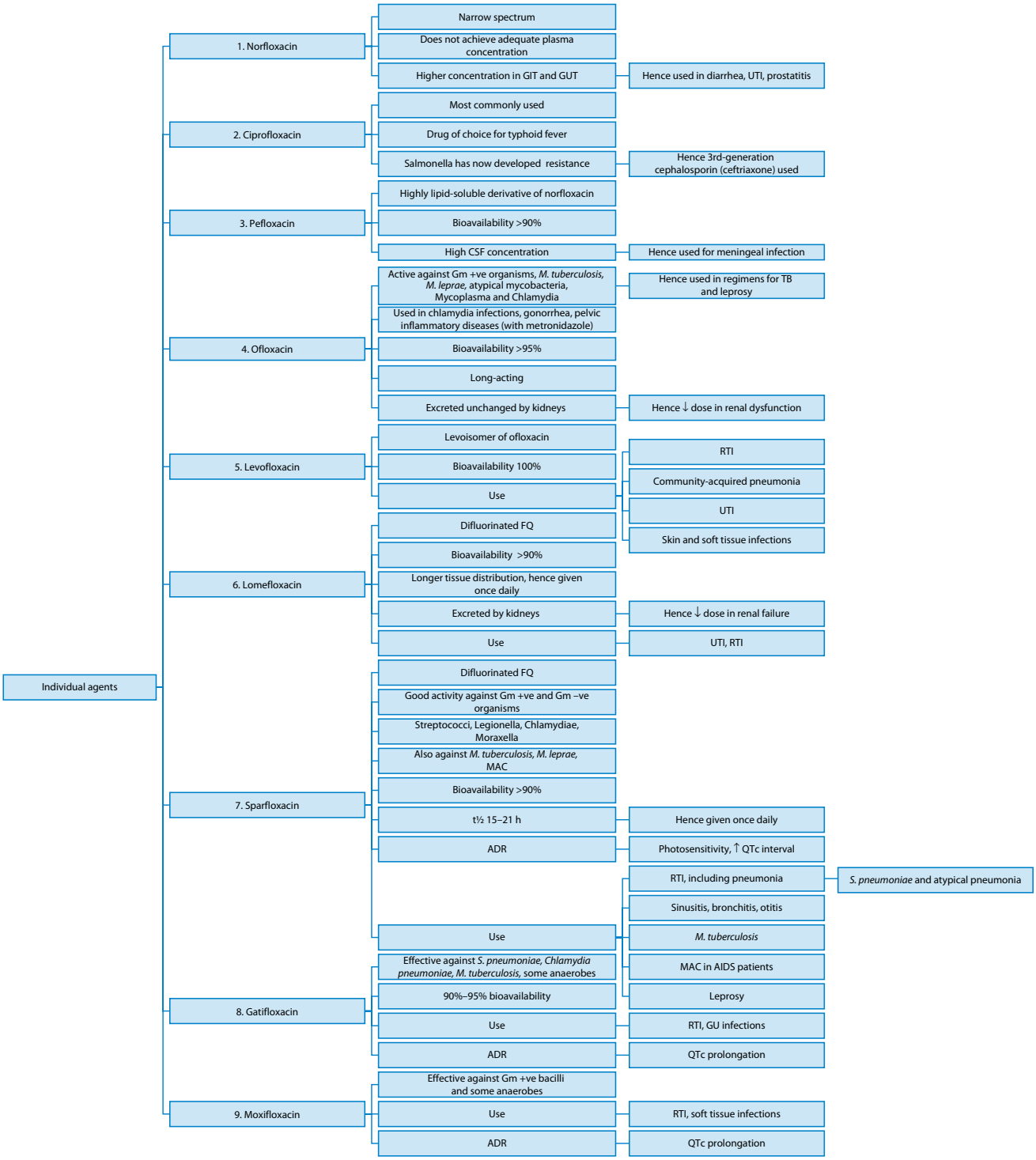
60

Quinolones

60.1 FLUOROQUINOLONES (CIPROFLOXACIN)



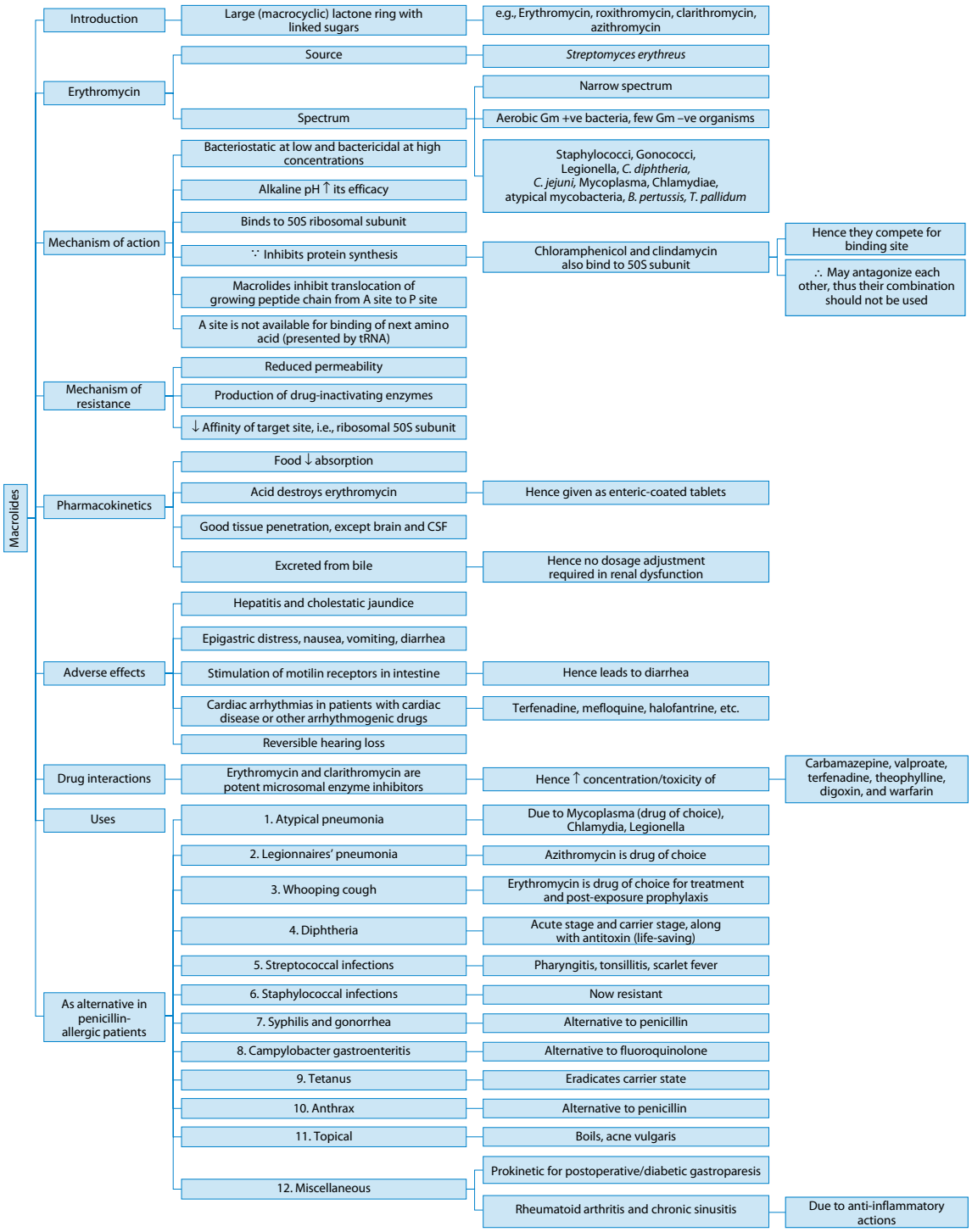
60.2 INDIVIDUAL AGENTS



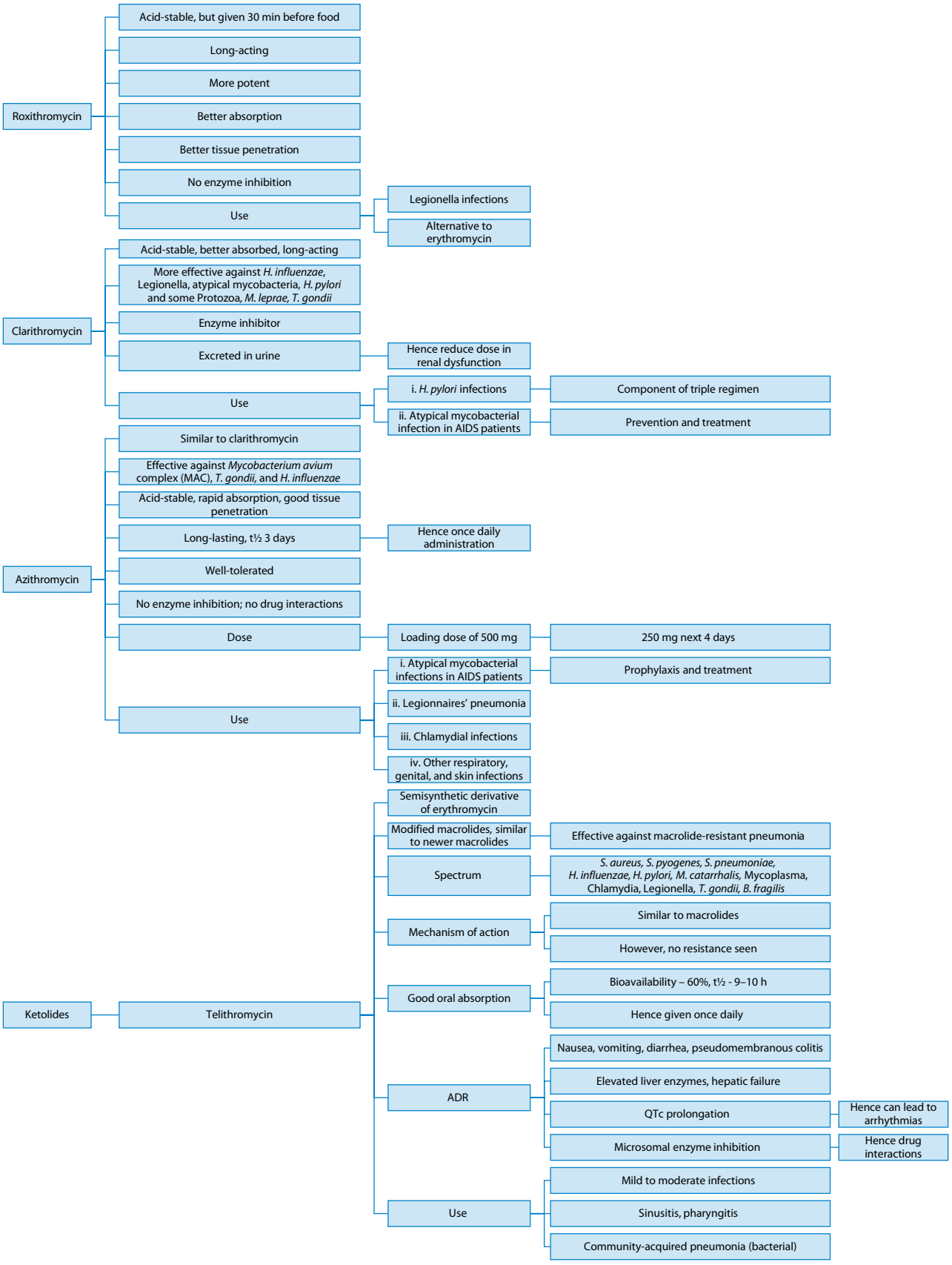
61

Macrolides

61.1 MACROLIDES



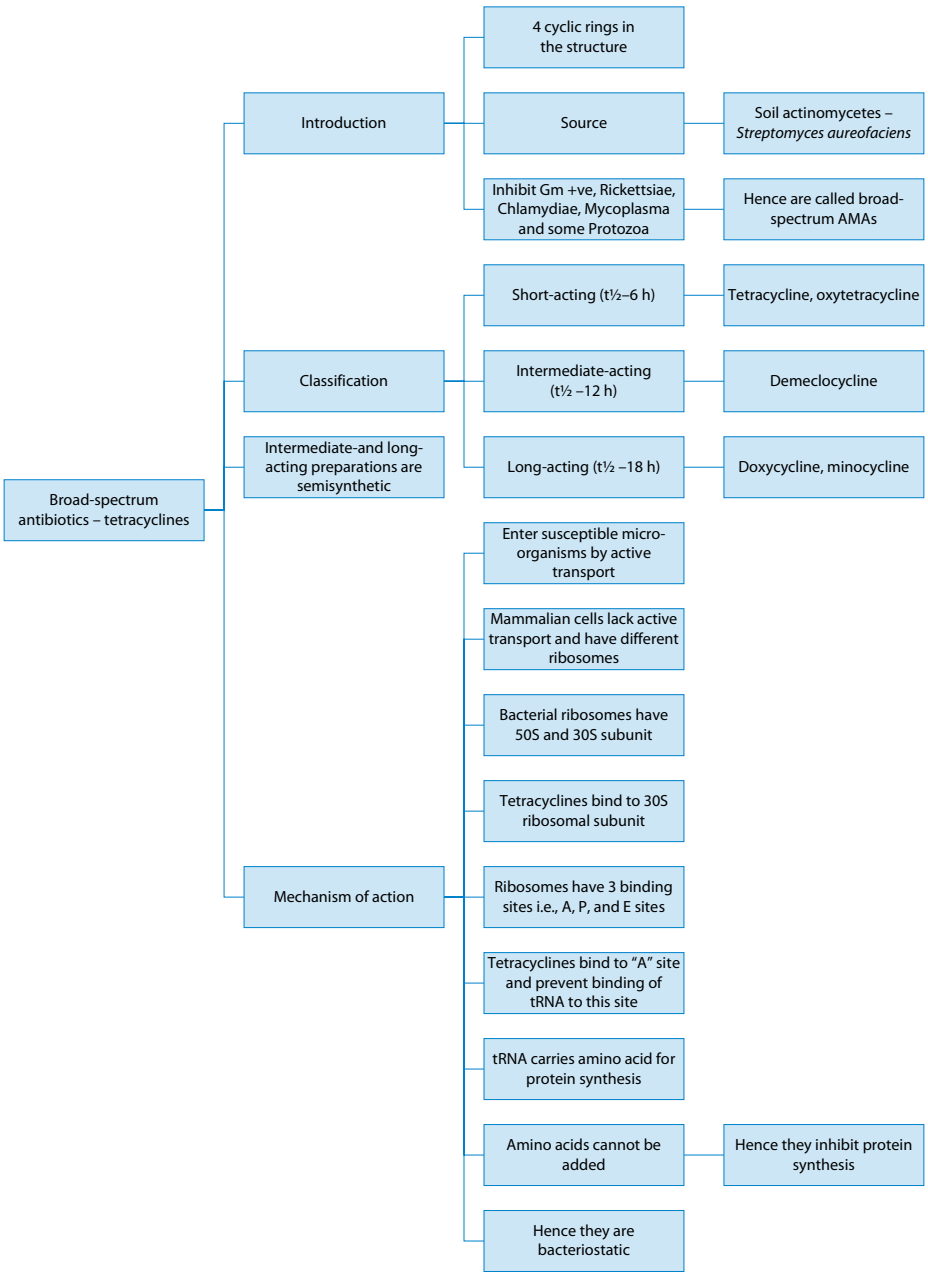
61.2 INDIVIDUAL MACROLIDES AND COMPARISON



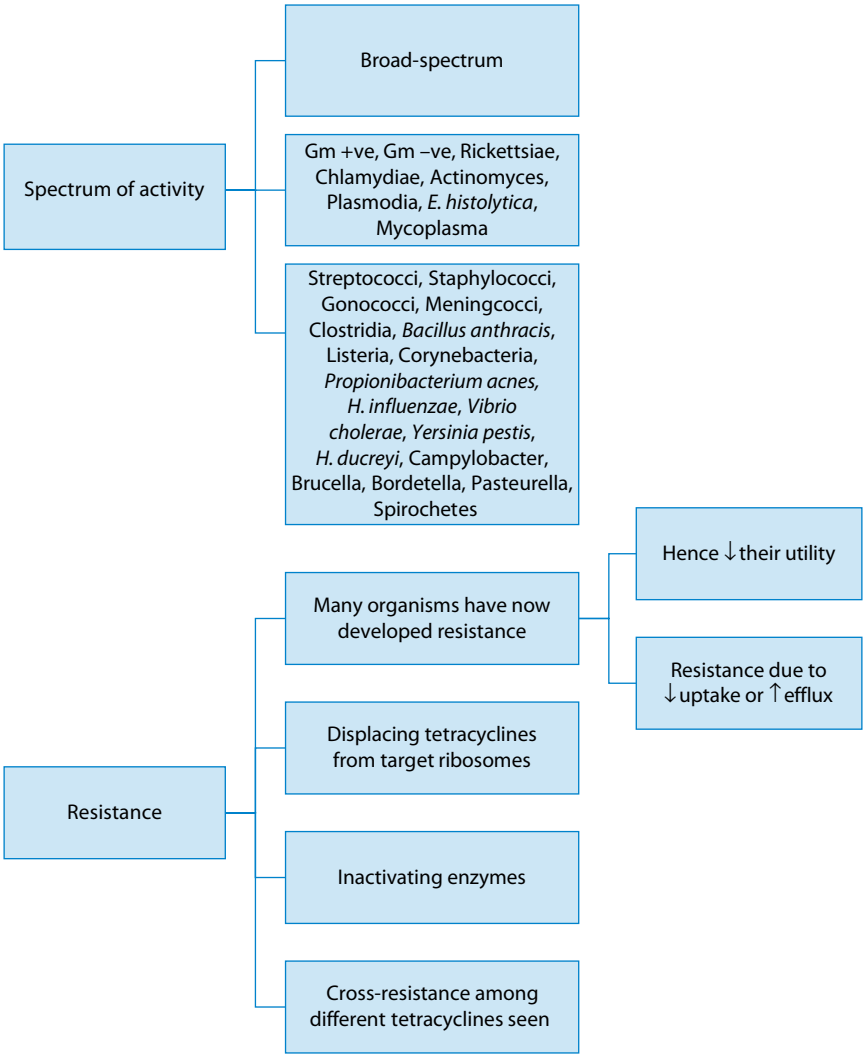
62

Broad-spectrum antibiotics – Tetracyclines and chloramphenicol

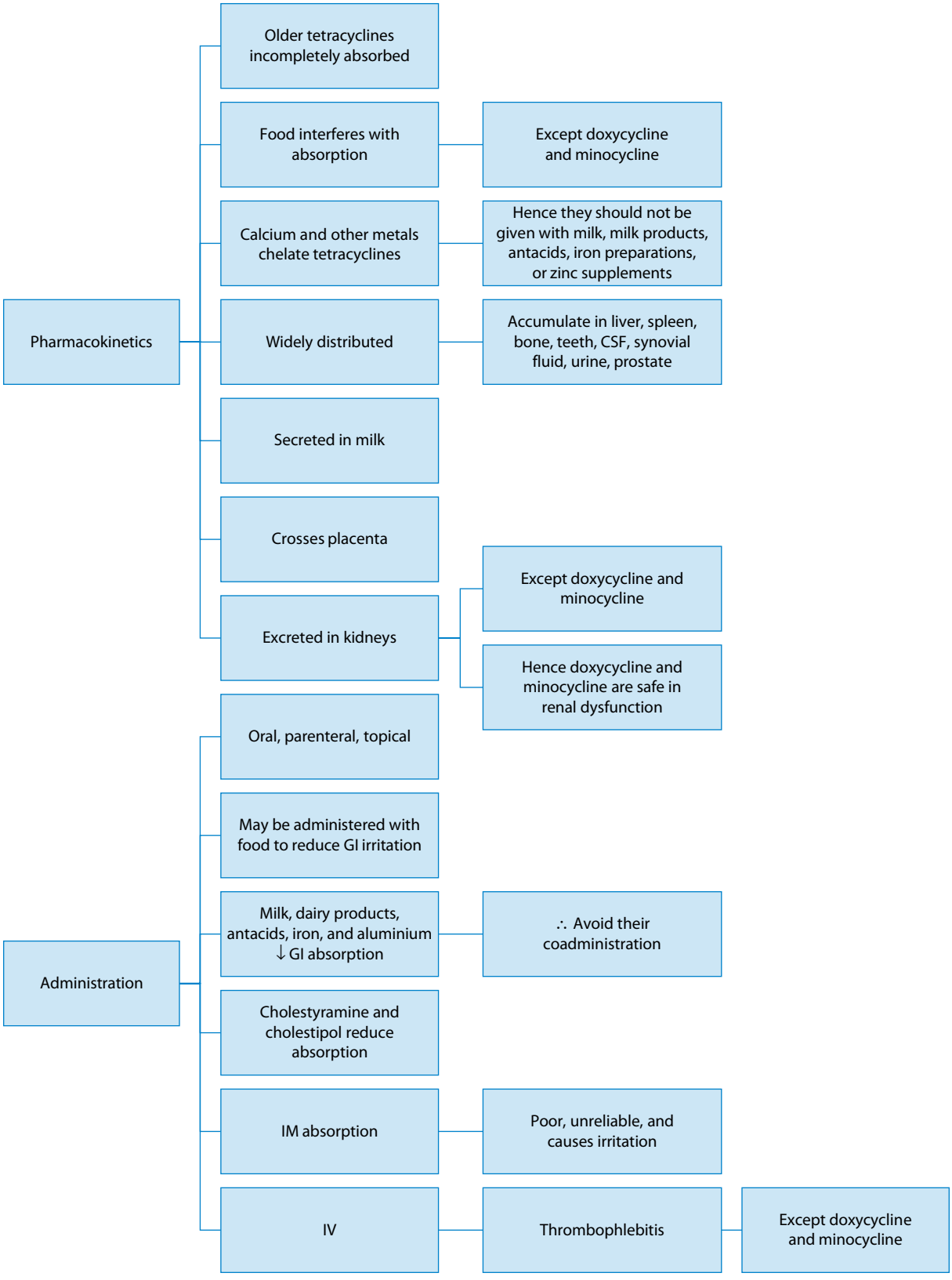
62.1 TETRACYCLINES – INTRODUCTION, CLASSIFICATION, AND MECHANISM OF ACTION



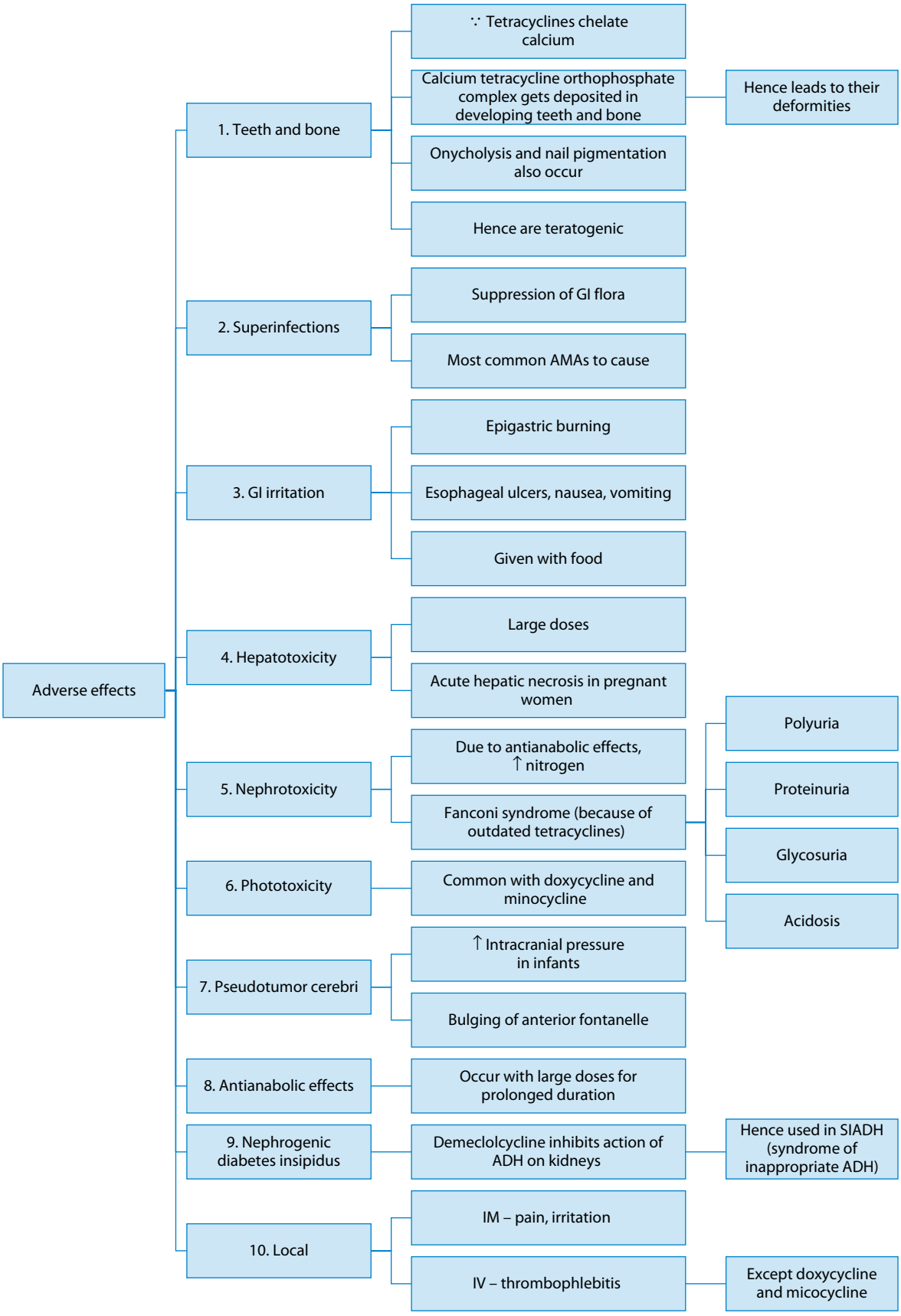
62.2 SPECTRUM OF ACTIVITY AND RESISTANCE



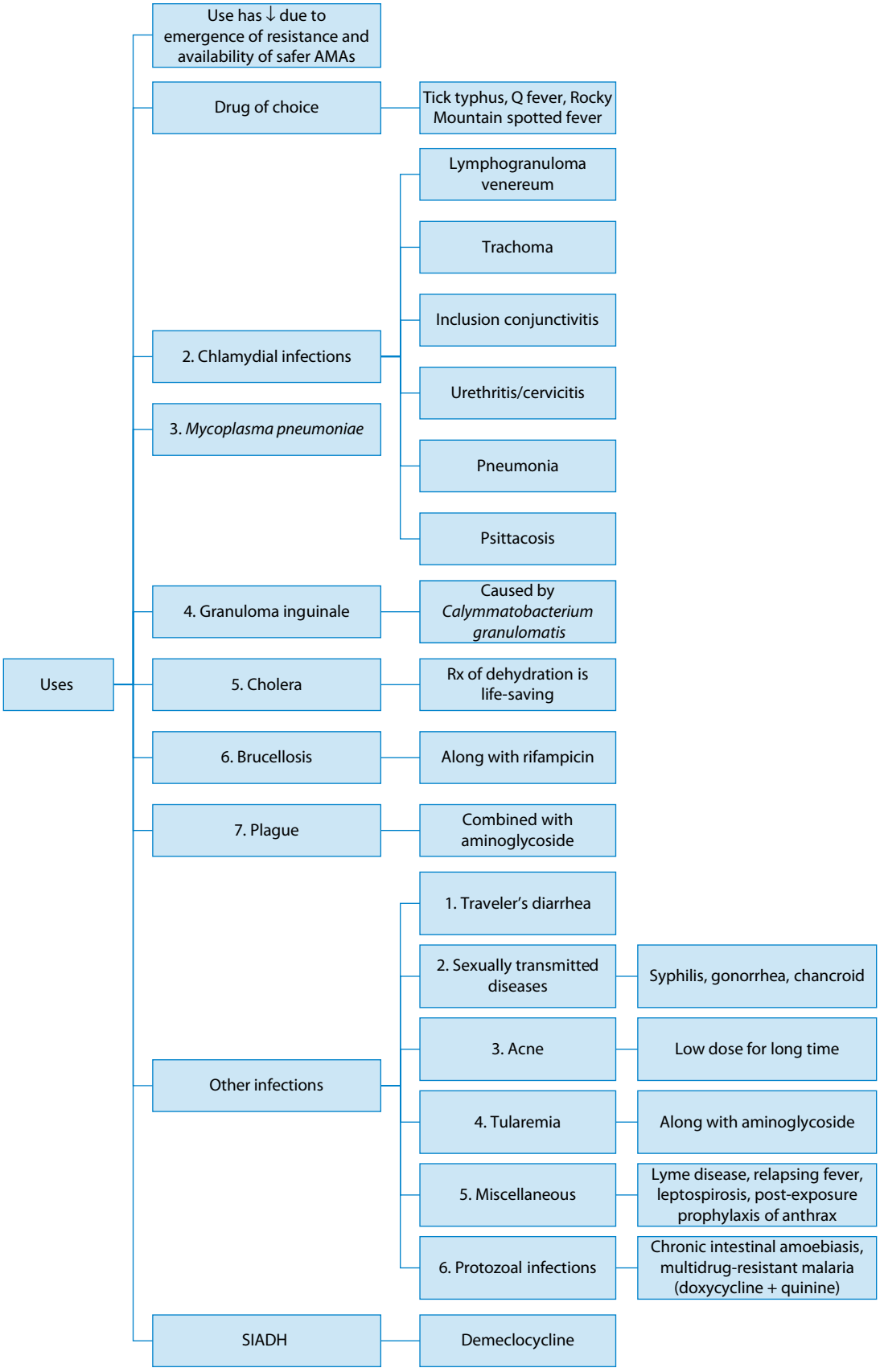
62.3 PHARMACOKINETICS AND ADMINISTRATION



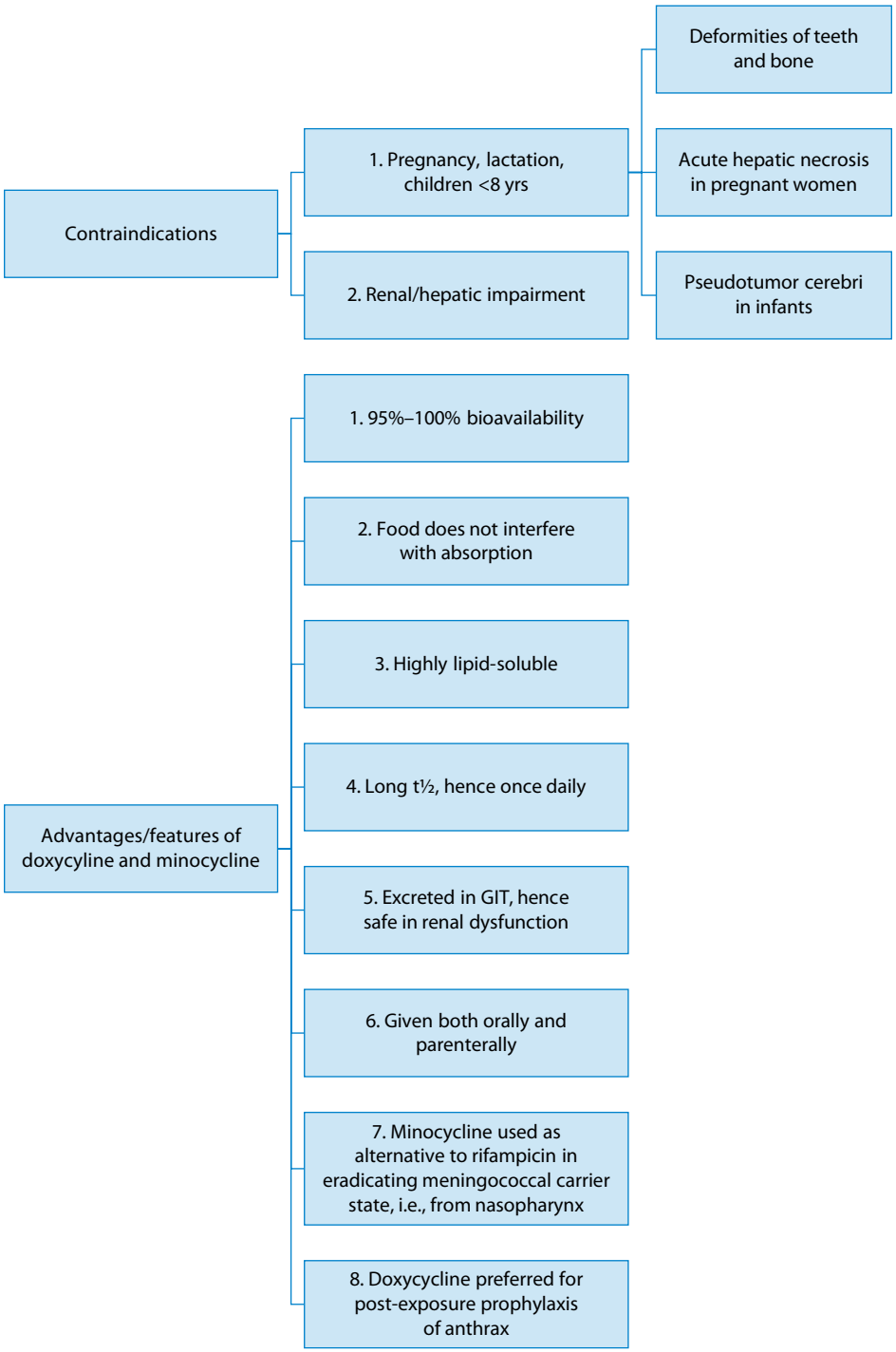
62.4 ADVERSE EFFECTS



62.5 USES


Uses
Use has ↓ due to emergence of resistance and availability of safer AMAs
Drug of choice
Tick typhus, Q fever, Rocky Mountain spotted fever
2. Chlamydial infections
Lymphogranuloma venereum
Trachoma
Inclusion conjunctivitis
Urethritis/cervicitis
Pneumonia
Psittacosis
3. *Mycoplasma pneumoniae*
4. Granuloma inguinale
Caused by *Calymmatobacterium granulomatis*
5. Cholera
Rx of dehydration is life-saving
6. Brucellosis
Along with rifampicin
7. Plague
Combined with aminoglycoside
Other infections
1. Traveler's diarrhea
2. Sexually transmitted diseases
Syphilis, gonorrhea, chancroid
3. Acne
Low dose for long time
4. Tularemia
Along with aminoglycoside
5. Miscellaneous
Lyme disease, relapsing fever, leptospirosis, post-exposure prophylaxis of anthrax
6. Protozoal infections
Chronic intestinal amoebiasis, multidrug-resistant malaria (doxycycline + quinine)
SIADH
Demeclocycline

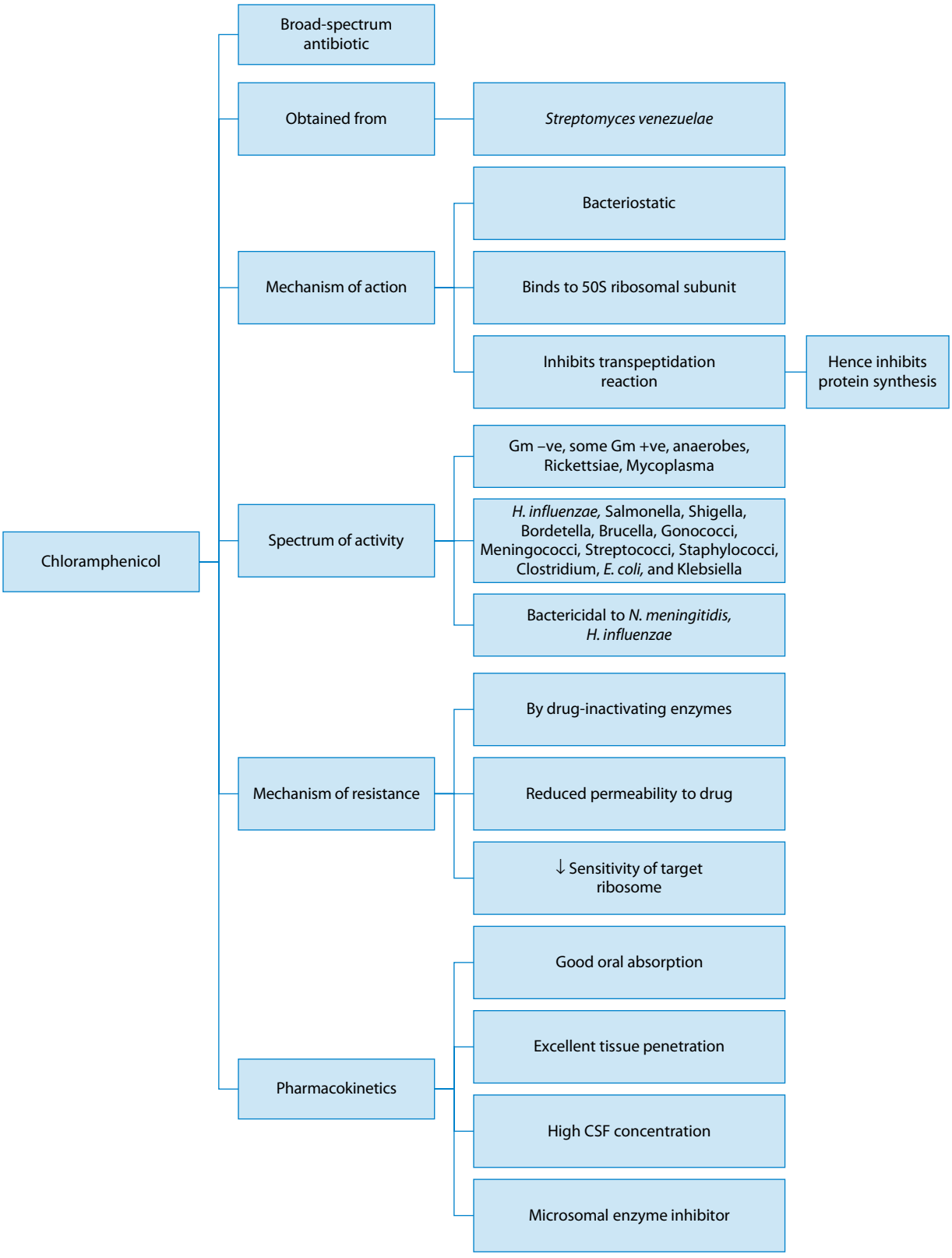
62.6 CONTRAINDICATIONS AND ADVANTAGES/FEATURES OF DOXYCYCLINE AND MINOCYCLINE



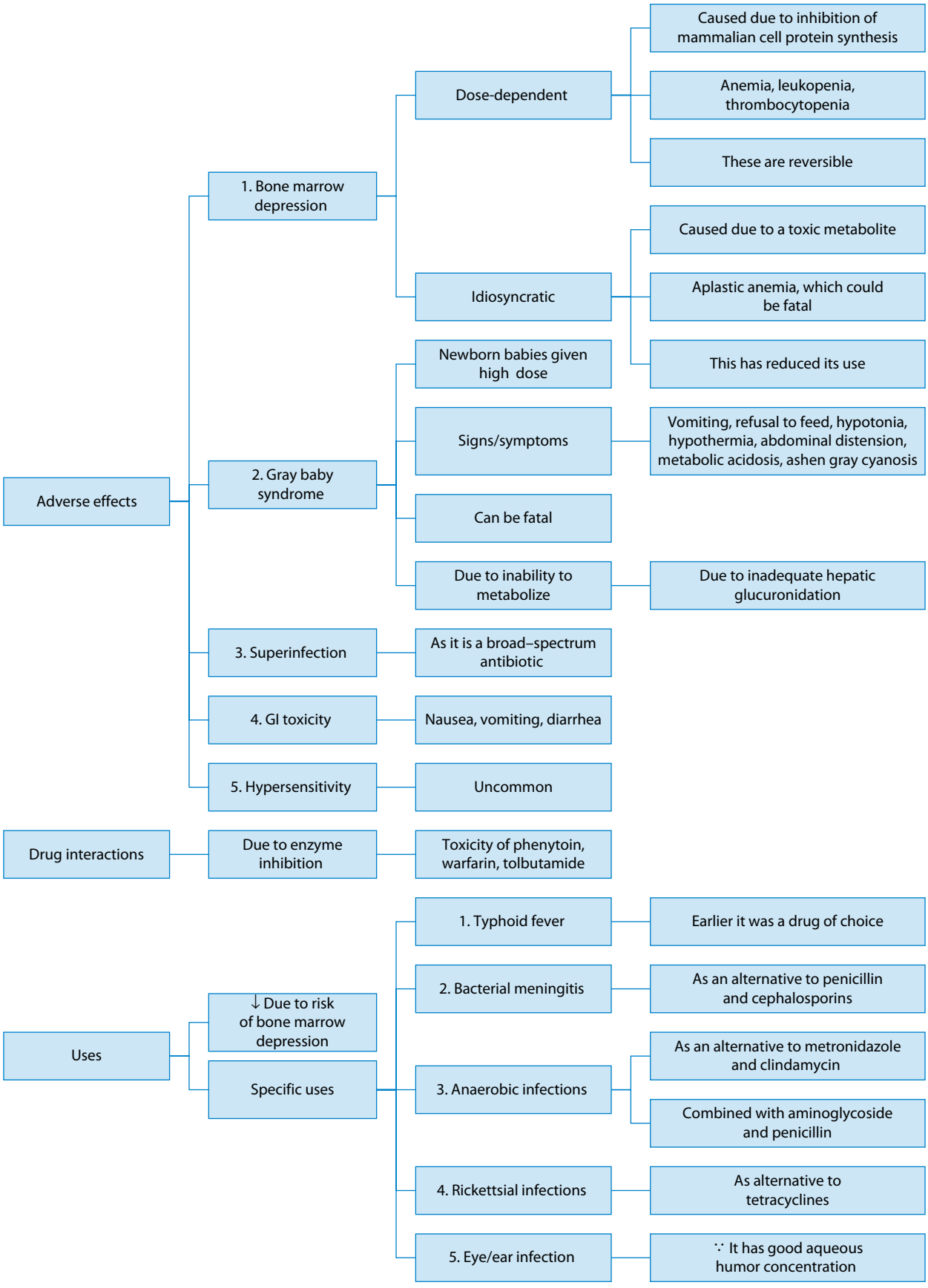
62.7 COMPARE/CONTRAST – TETRACYCLINE vs. DOXYCYCLINE

	Tetracycline	**Doxycycline**
1. Source	Semisynthetic	Semisynthetic
2. GI absorption	Incomplete	Complete
3. Bioavailability	75%	95%
4. t½	8–10 h	18–24 h
5. Lipid solubility	Low	High
6. Dosing	QID	OD
7. Excretion	Kidney	GIT
8. Safety in renal impairment	Not safe	Safe
9. GI flora	High suppression	Low suppression
10. Phototoxicity	Low	High

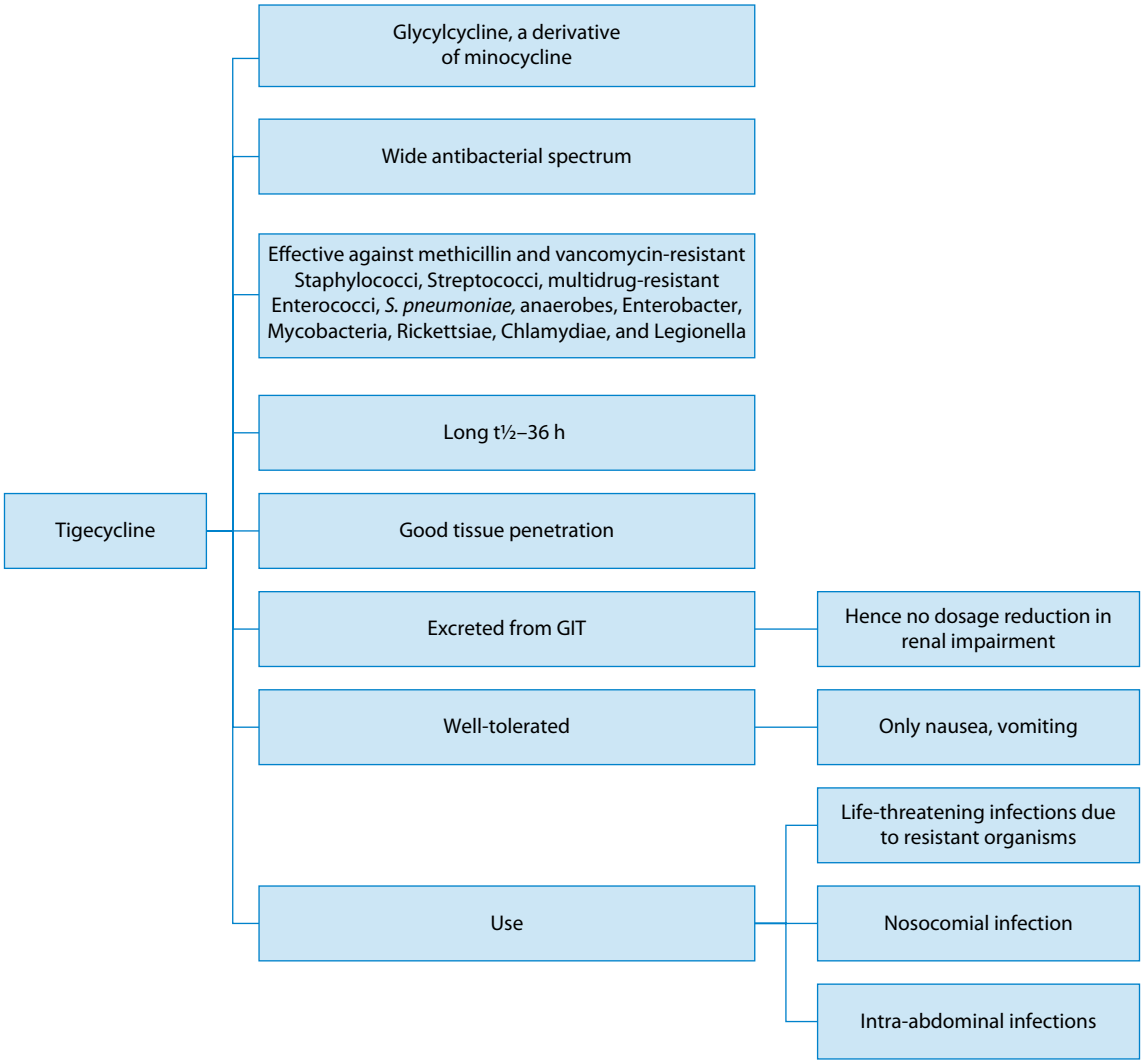
62.8 CHLORAMPHENICOL – MECHANISM OF ACTION, SPECTRUM OF ACTIVITY, MECHANISM OF RESISTANCE, AND PHARMACOKINETICS



62.9 ADVERSE EFFECTS, DRUG INTERACTIONS, AND USES



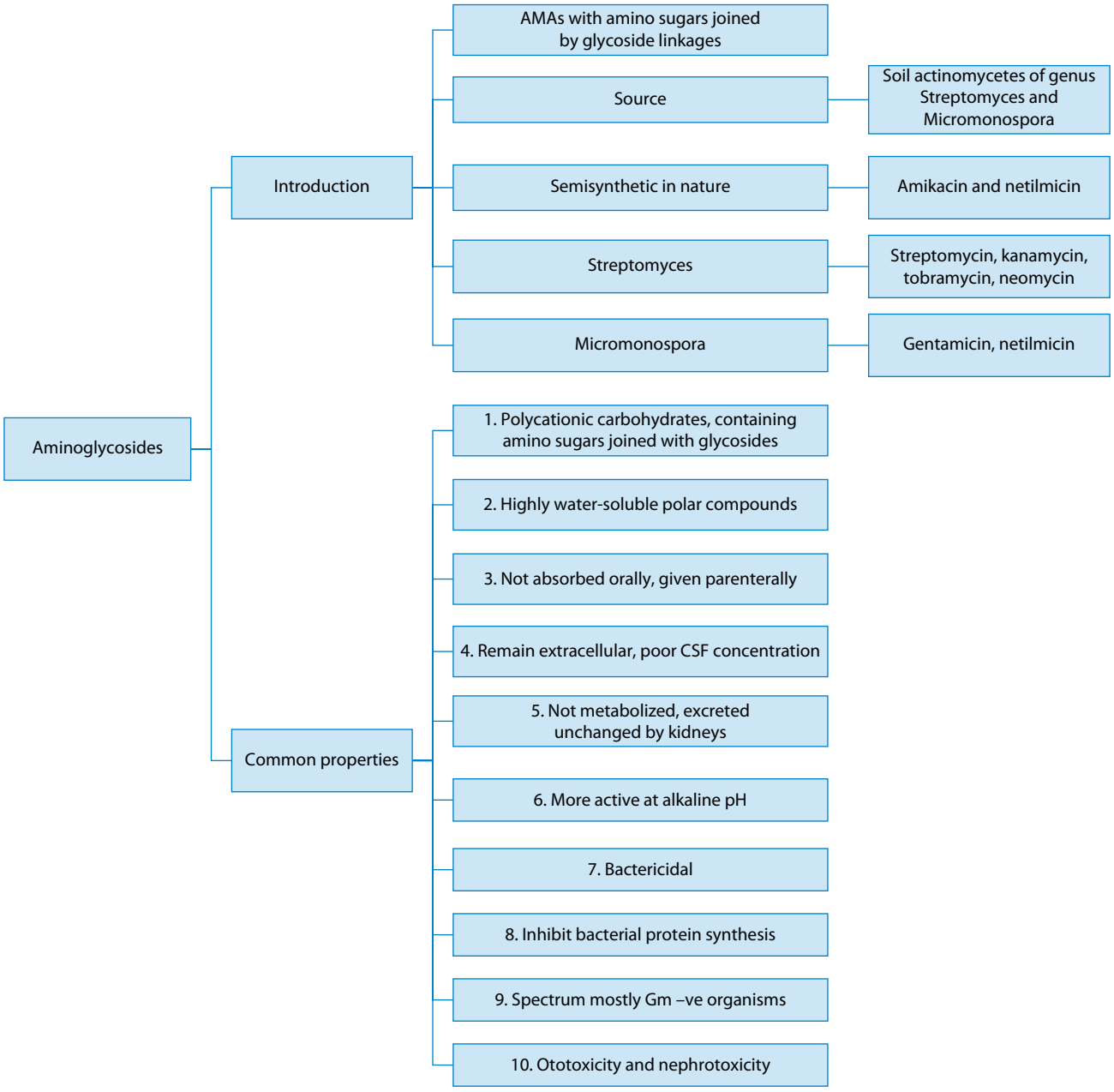
62.10 TIGECYCLINE



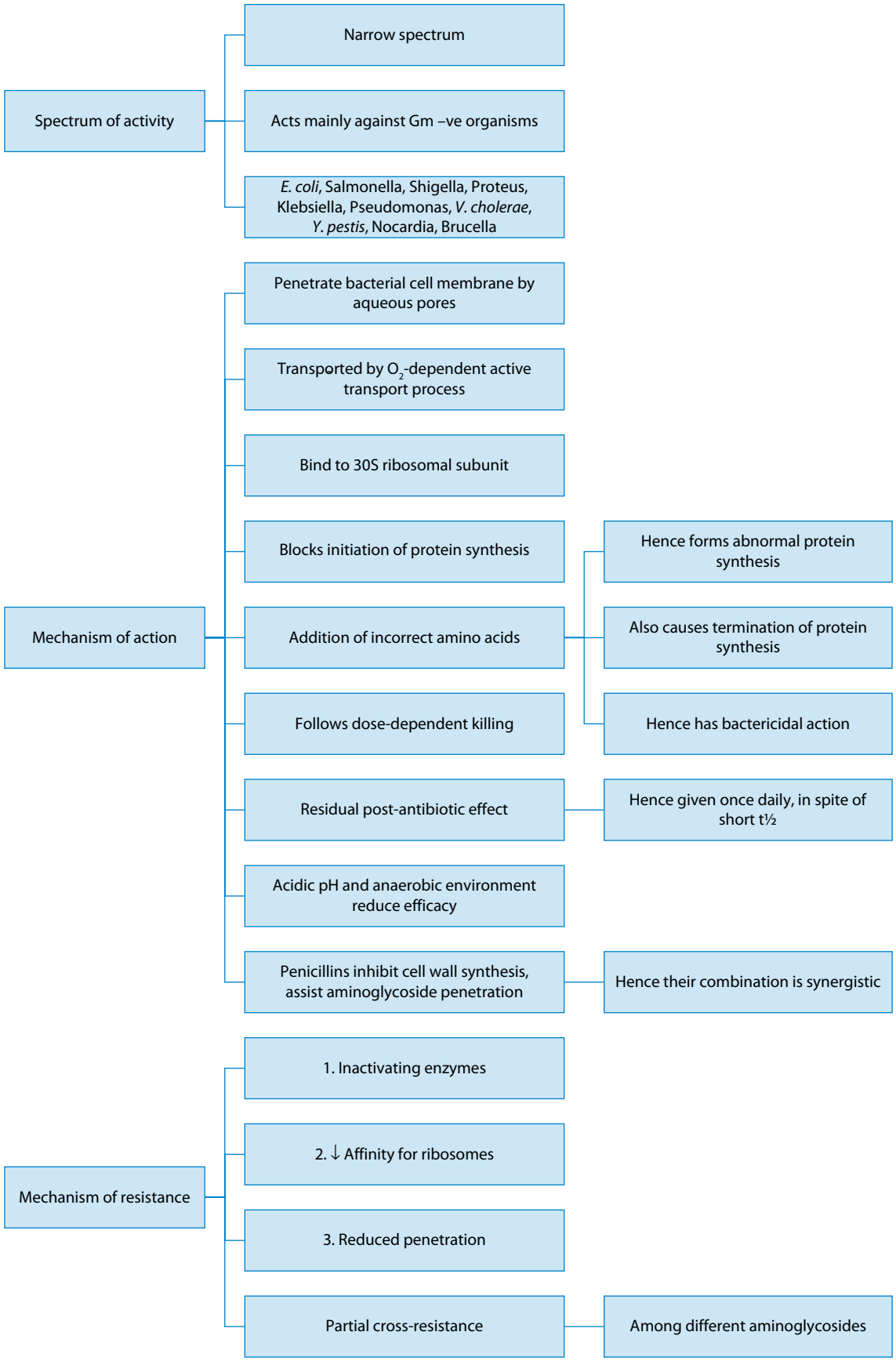
63

Aminoglycosides

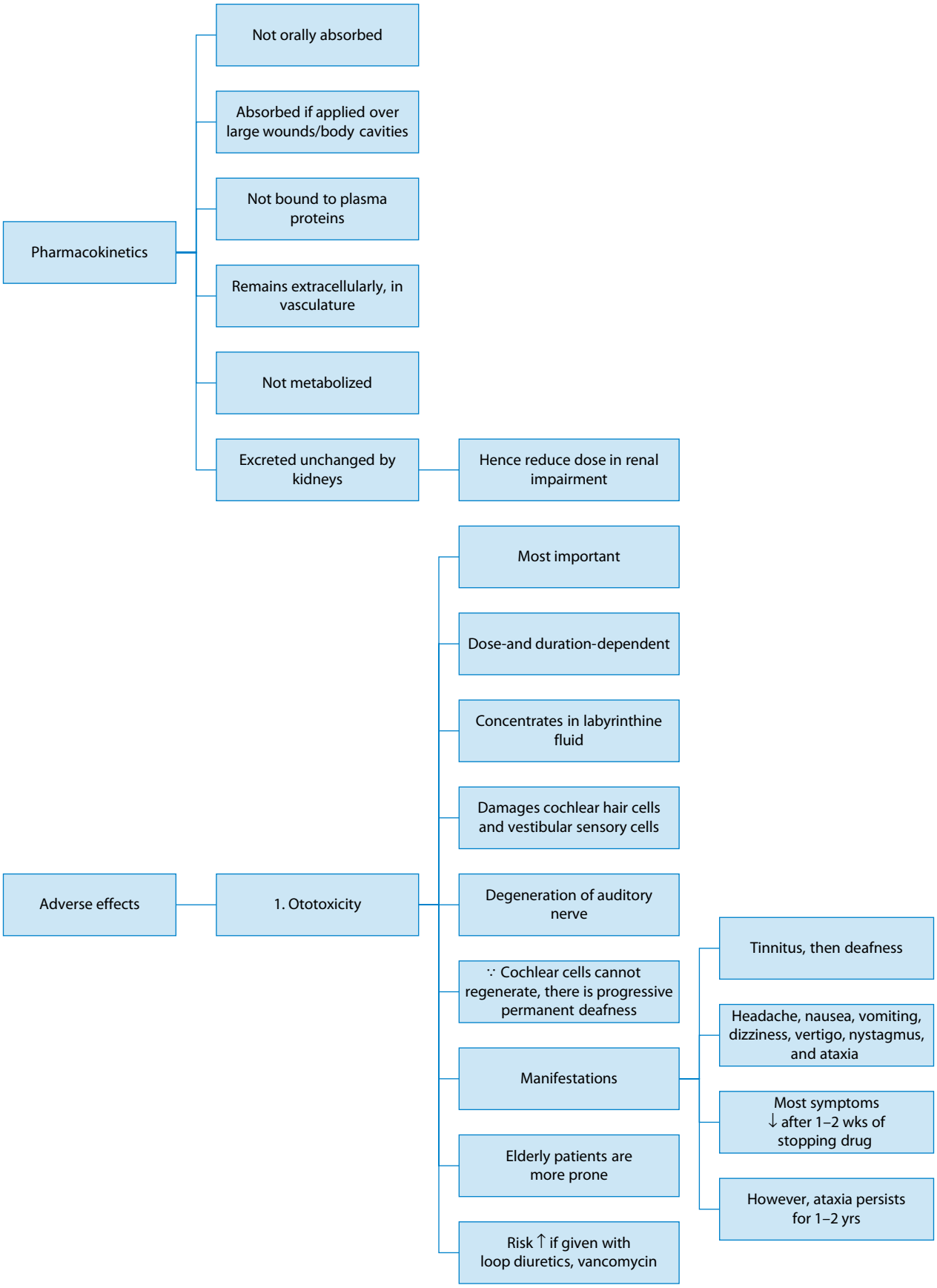
63.1 INTRODUCTION AND COMMON PROPERTIES



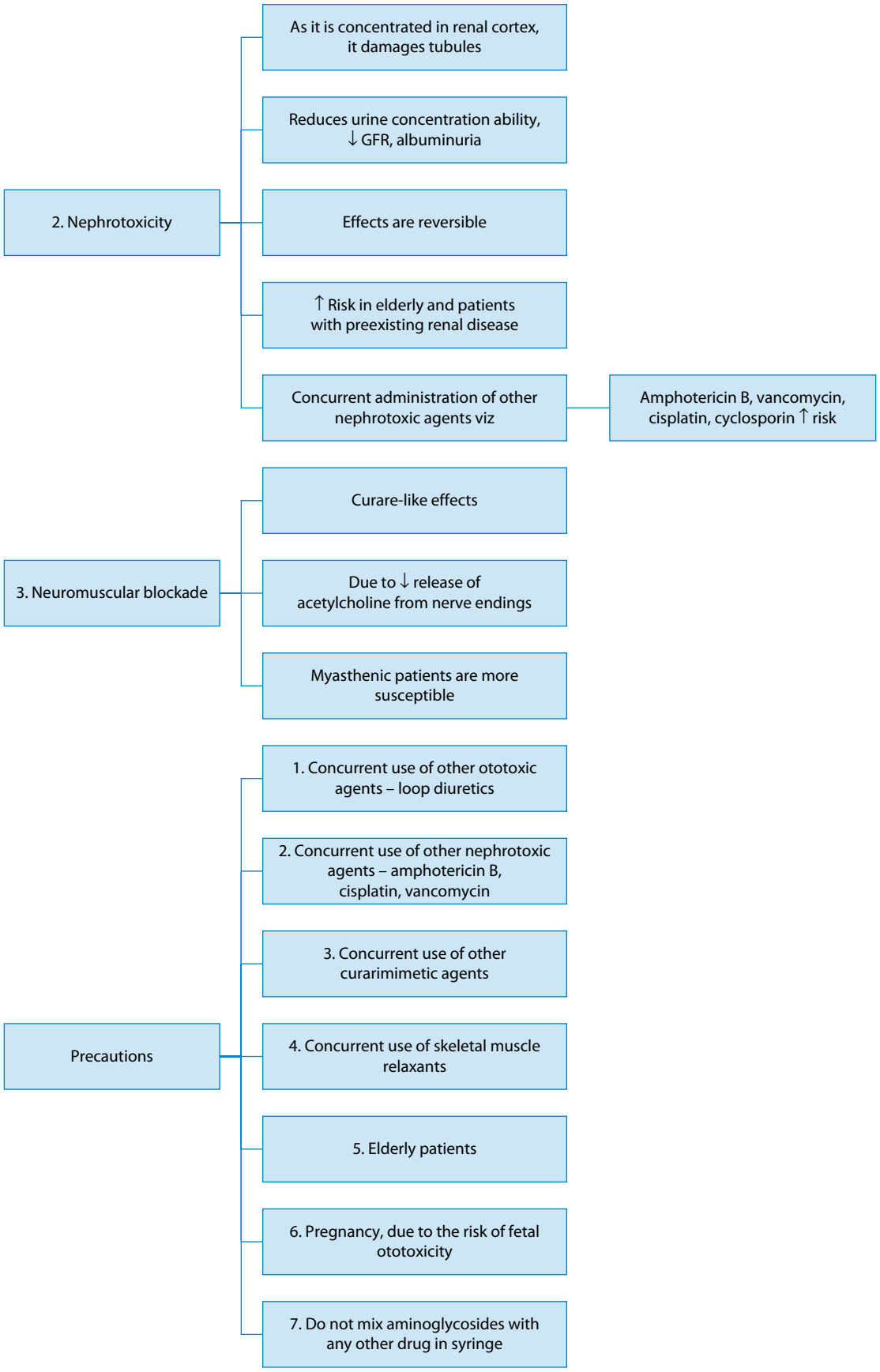
63.2 SPECTRUM, MECHANISM OF ACTION, AND MECHANISM OF RESISTANCE



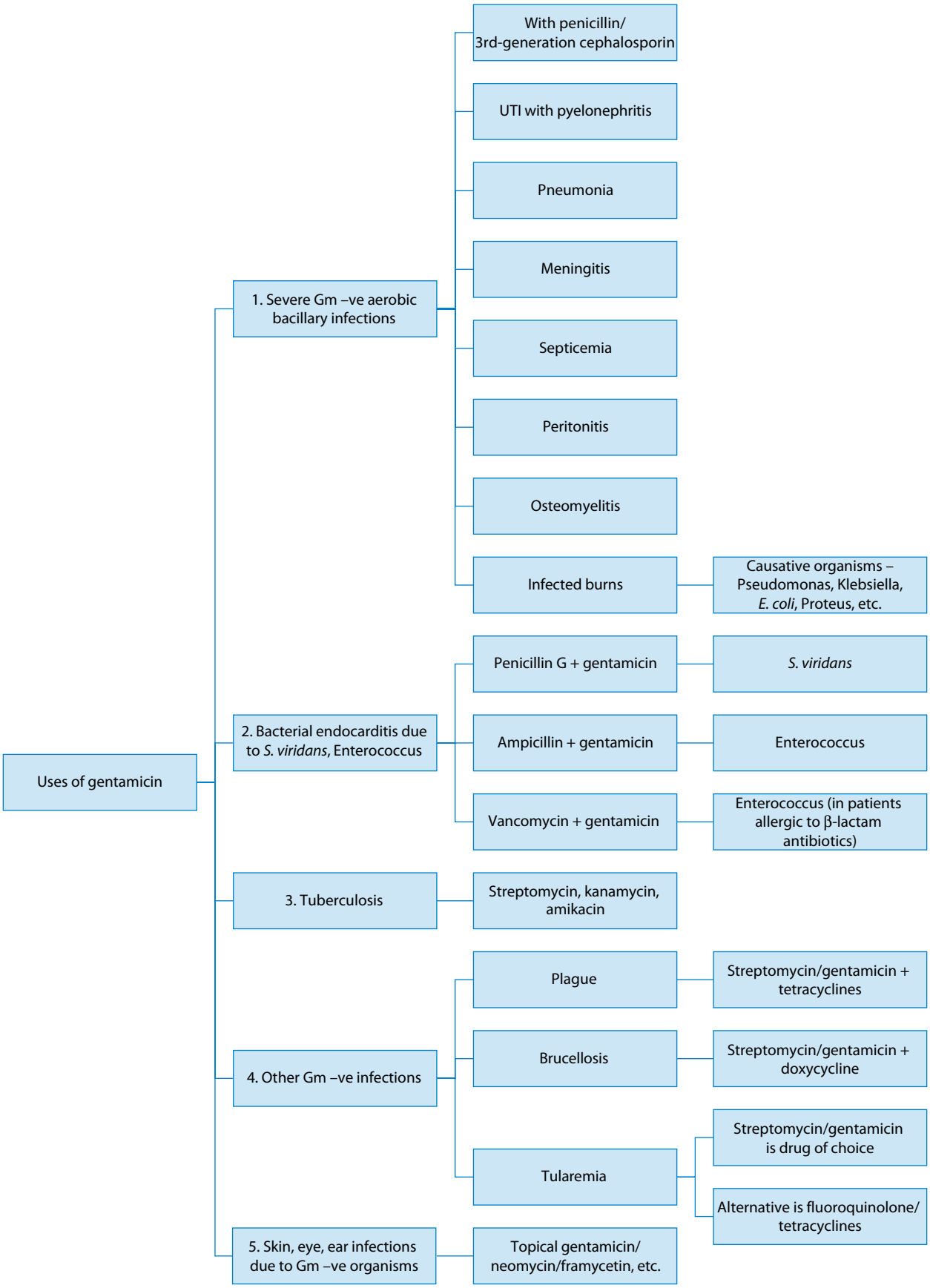
63.3 PHARMACOKINETICS AND ADRs



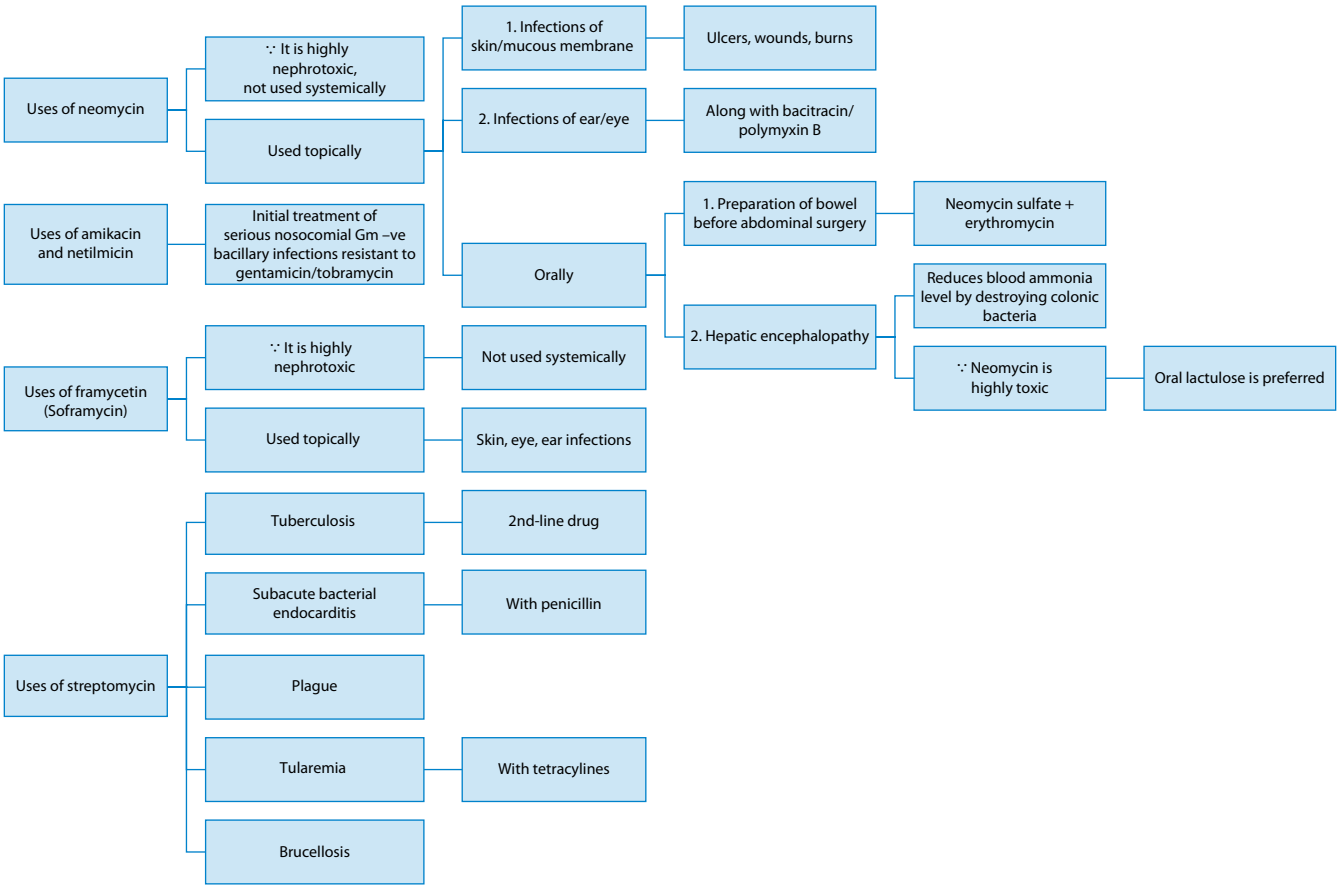
63.4 ADRs AND PRECAUTIONS



63.5 USES OF GENTAMICIN



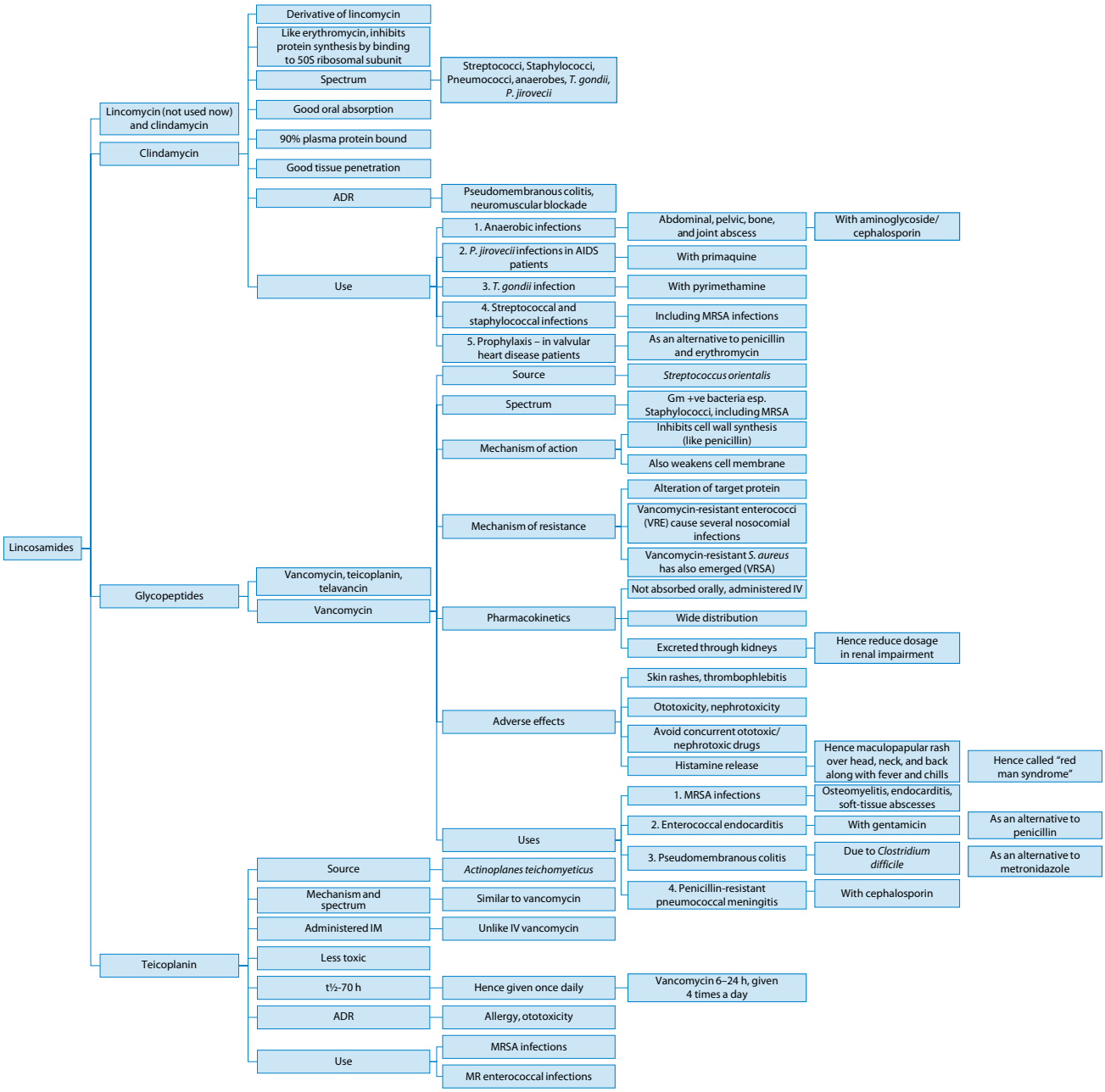
63.6 OTHER AMINOGLYCOSIDES



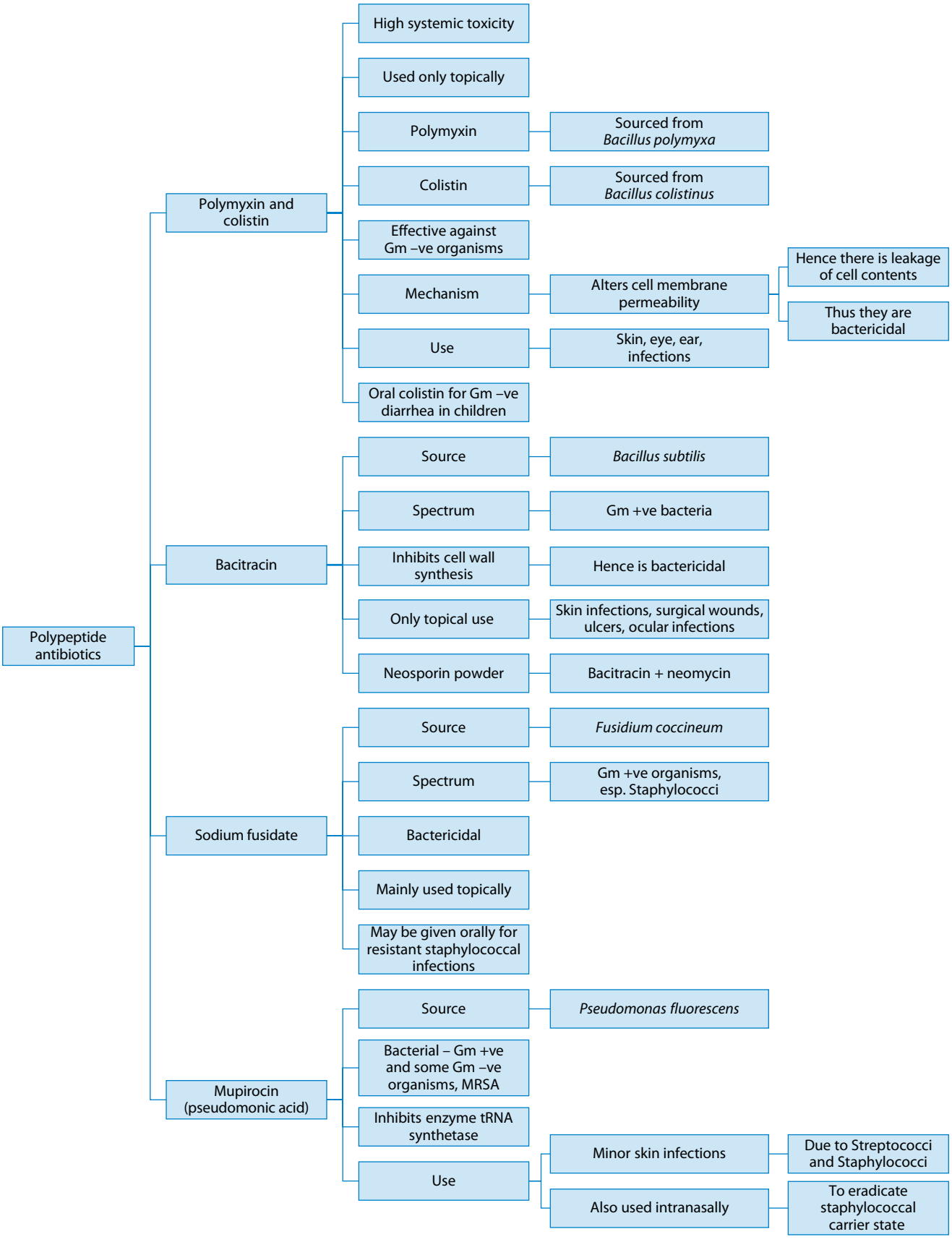
64

Miscellaneous antibiotics

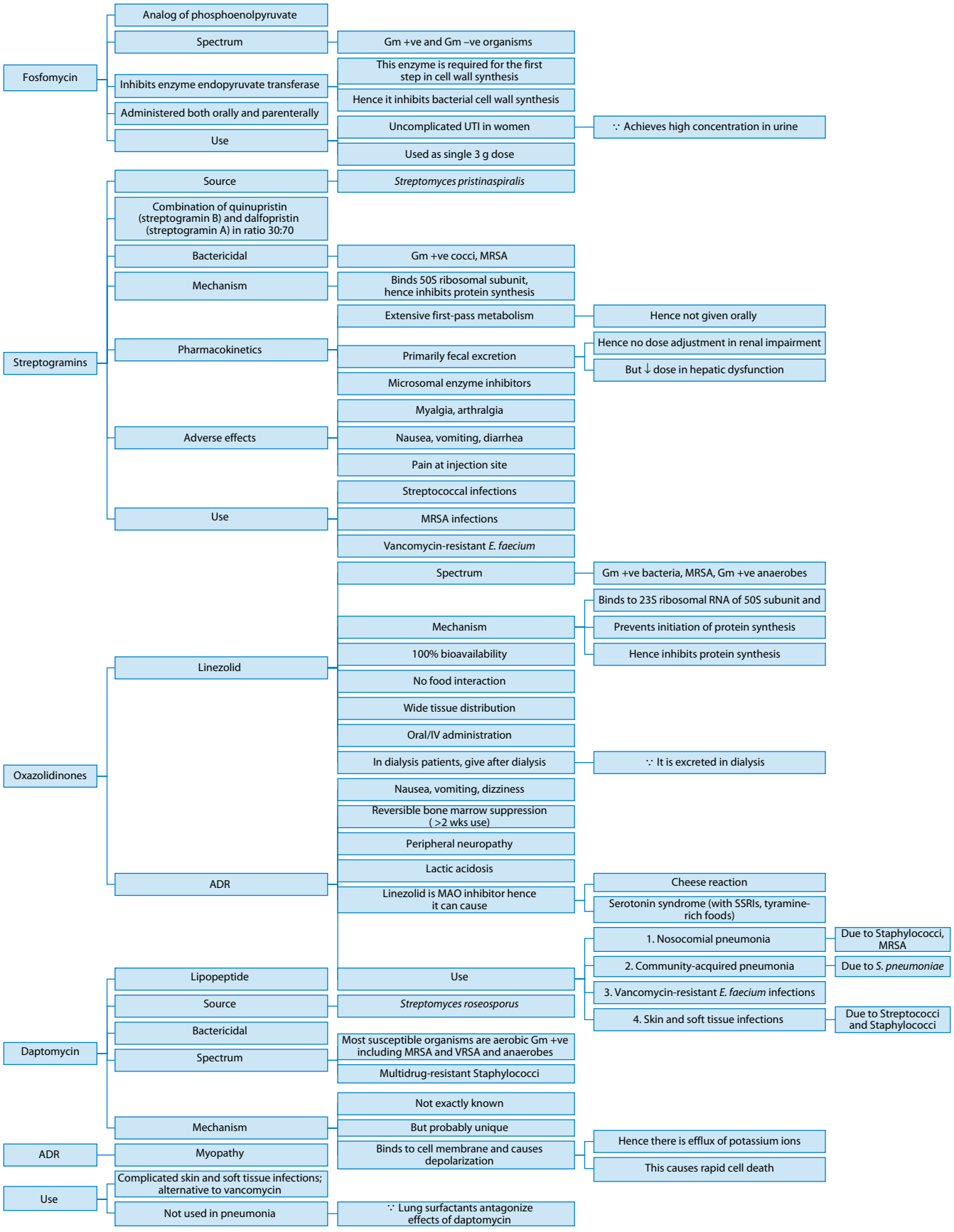
64.1 LINCOSAMIDES, GLYCOPEPTIDES, AND TEICOPLANIN



64.2 POLYPEPTIDE ANTIBIOTICS



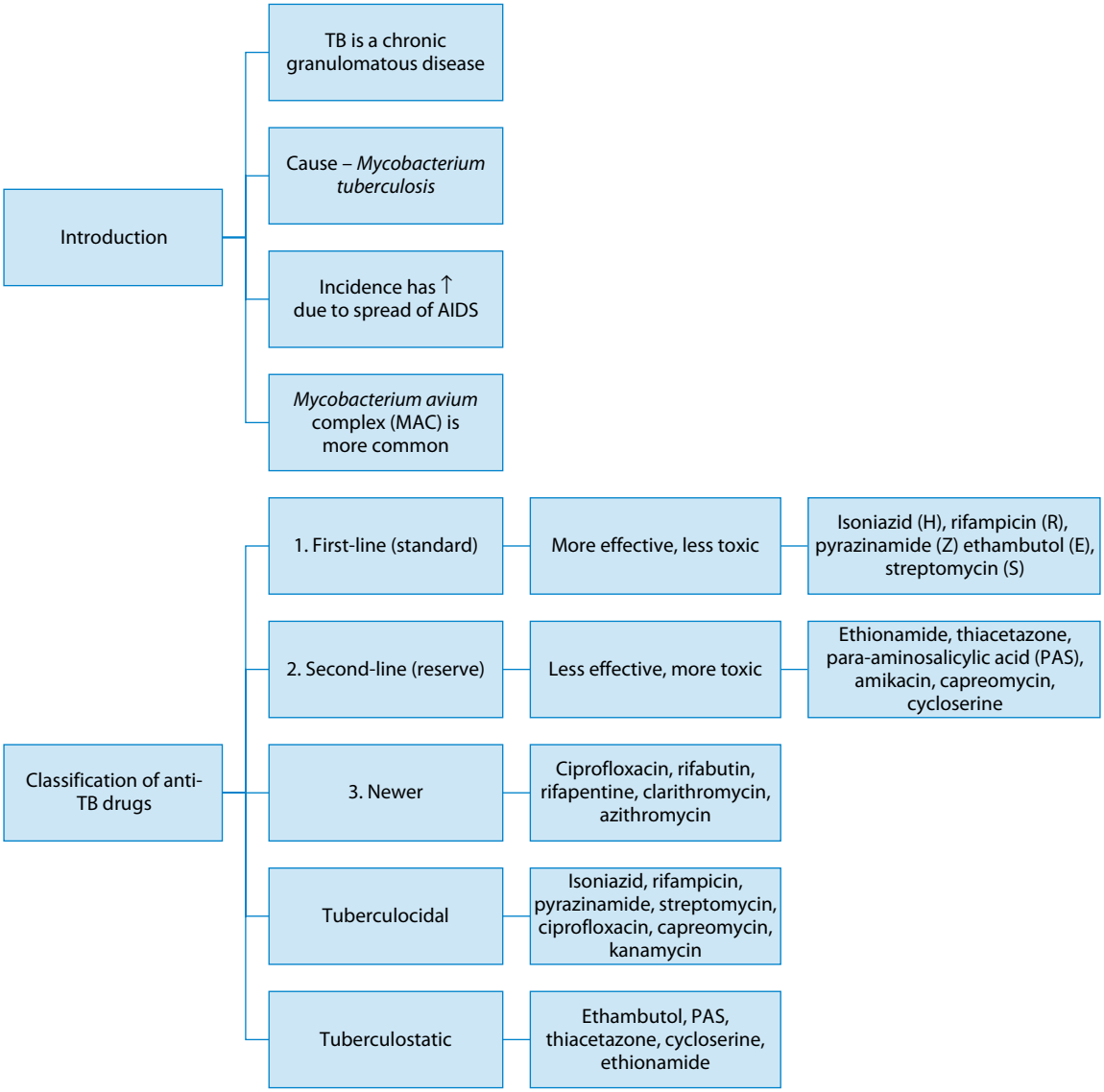
64.3 FOSFOMYCIN, STREPTOGRAMINS, OXAZOLIDINONES, AND DAPTOMYCIN



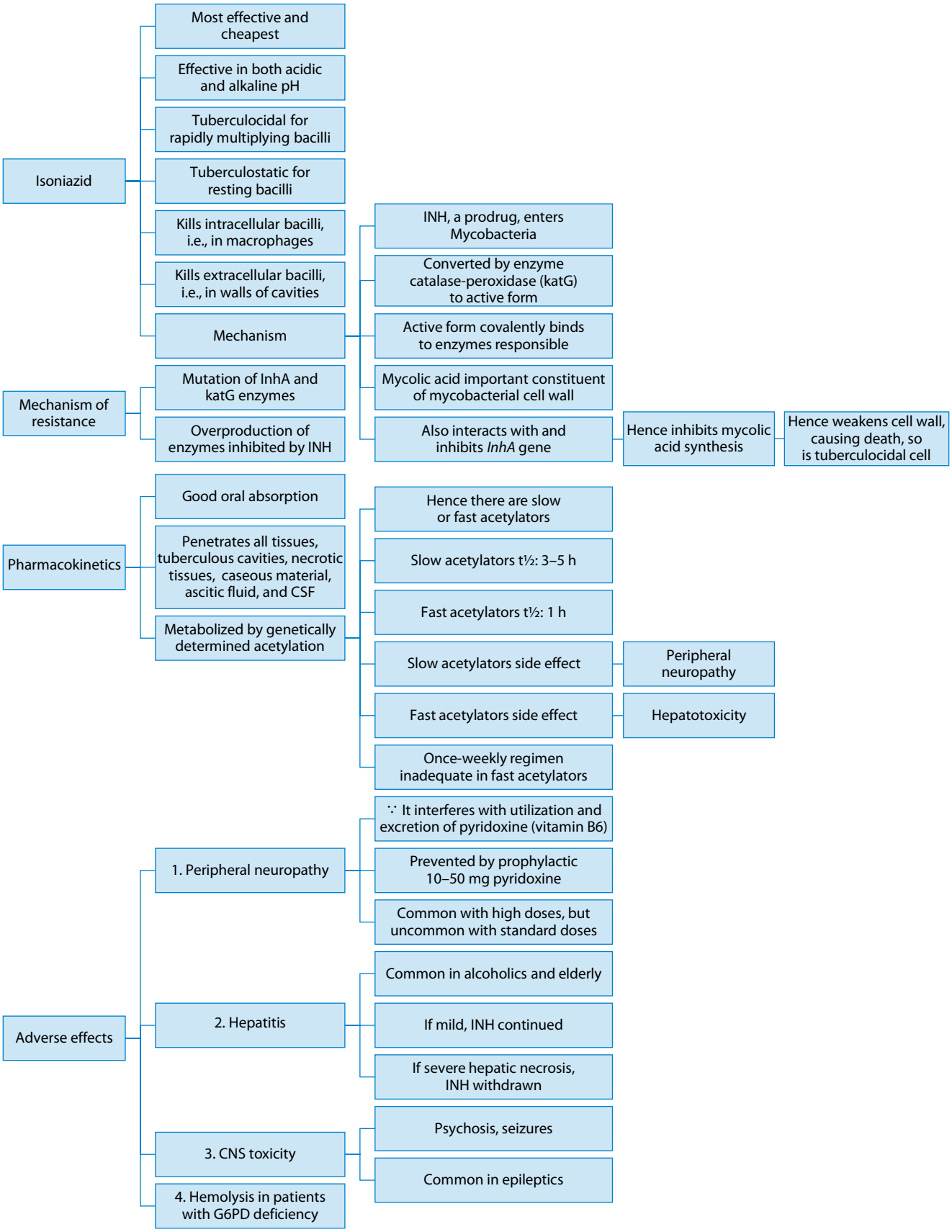
65

Chemotherapy of tuberculosis (TB)

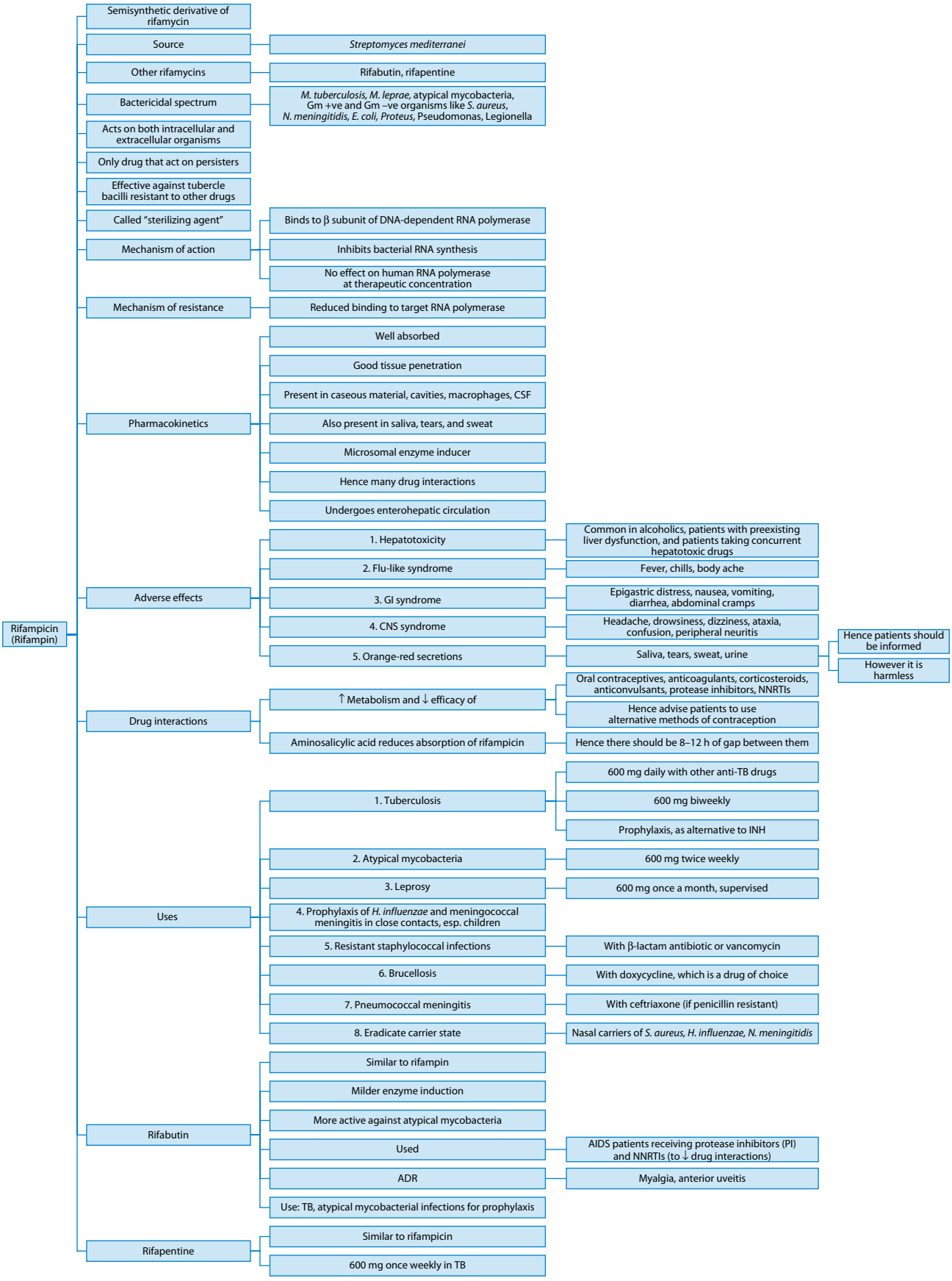
65.1 INTRODUCTION AND CLASSIFICATION



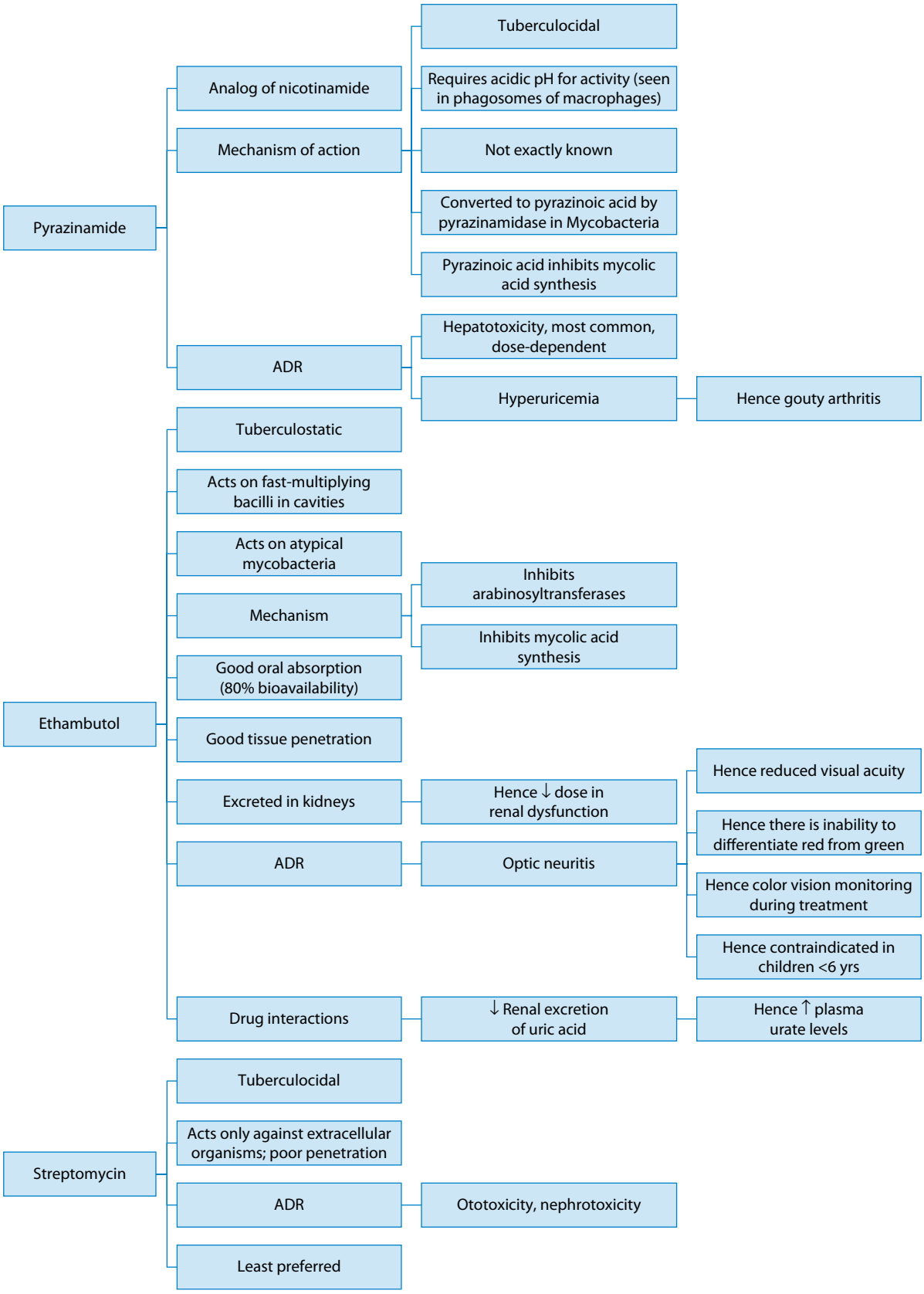
65.2 FIRST-LINE DRUGS – ISONIAZID



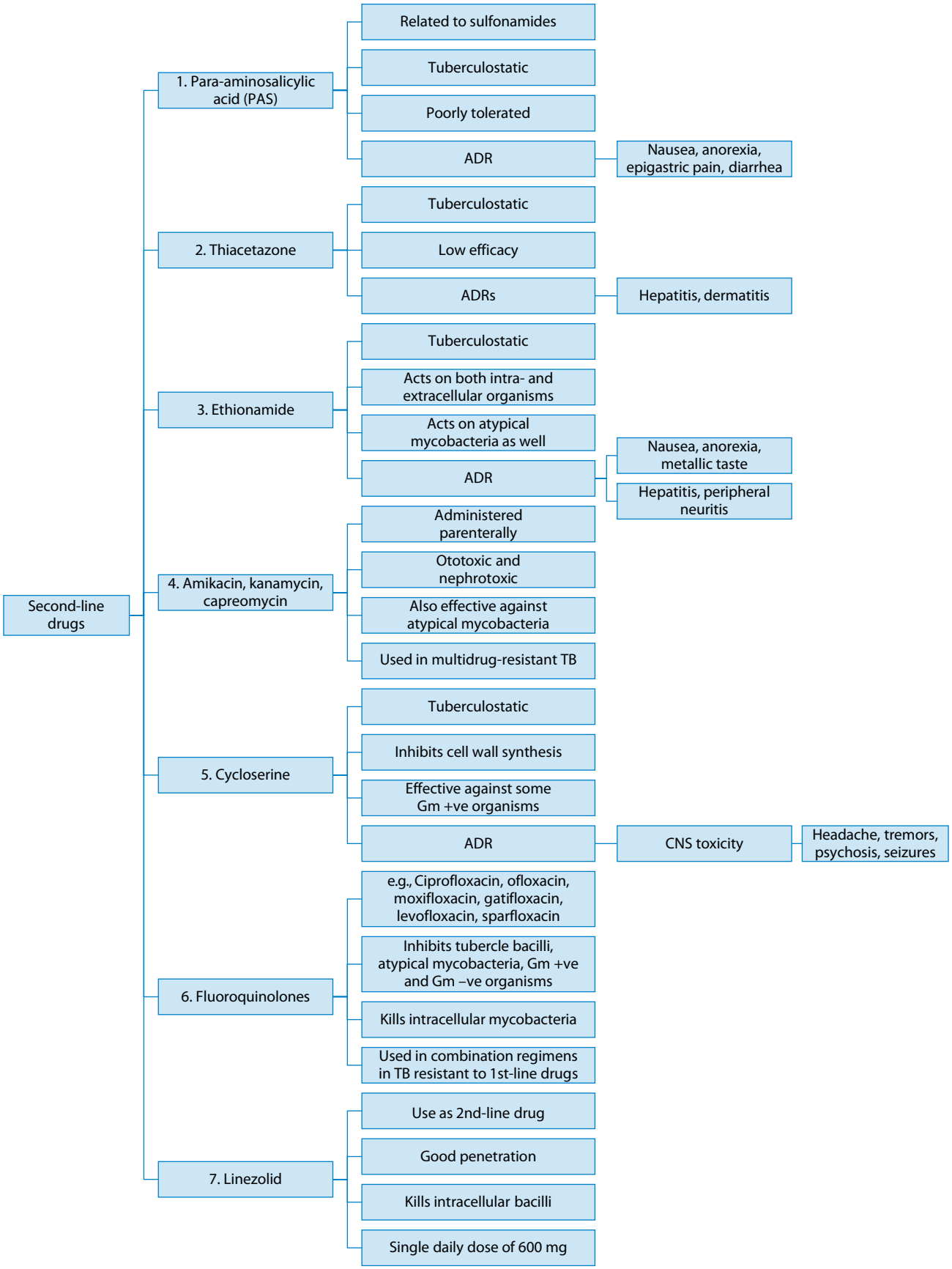
65.3 RIFAMPICIN (RIFAMPIN)



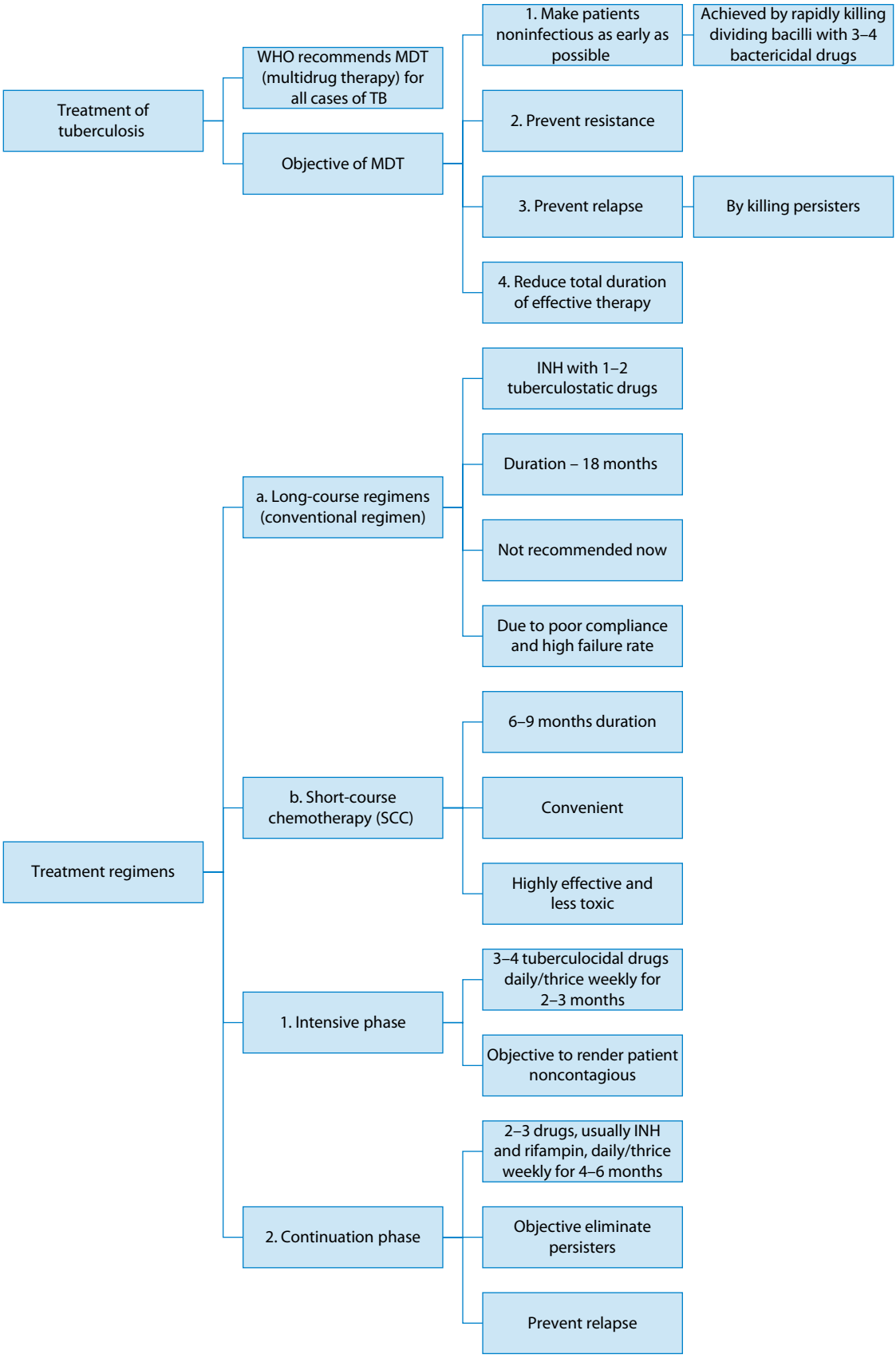
65.4 PYRAZINAMIDE, ETHAMBUTOL, AND STREPTOMYCIN



65.5 SECOND-LINE DRUGS



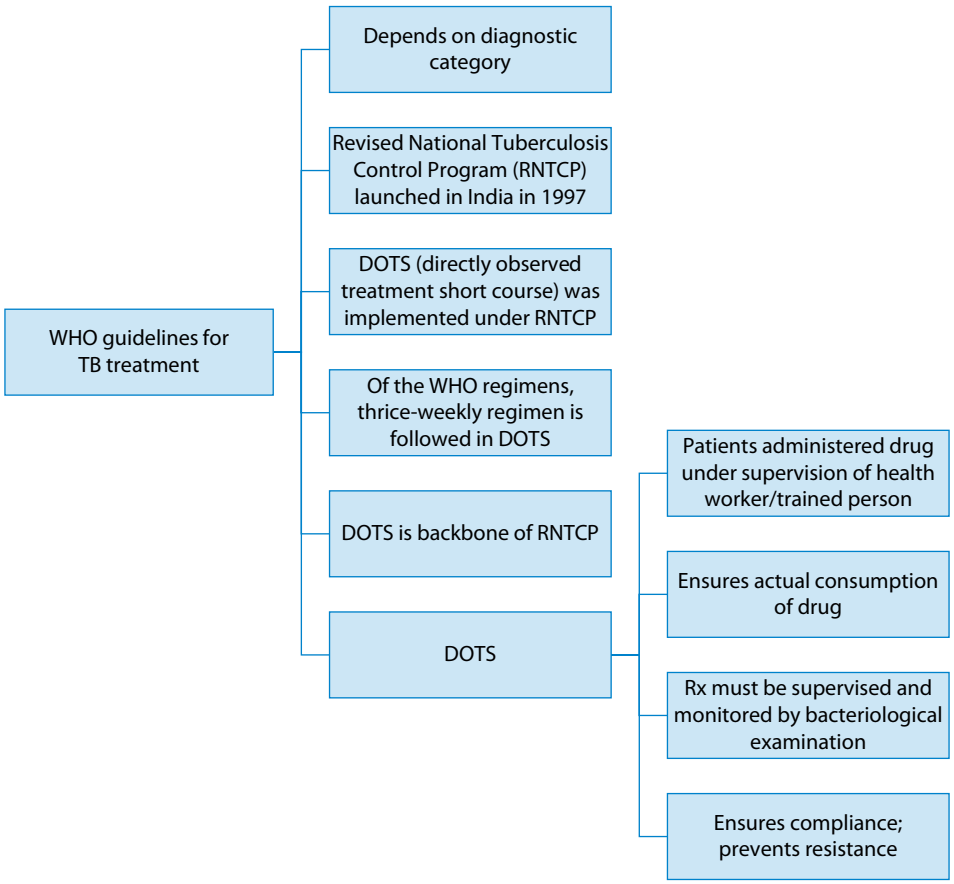
65.6 TREATMENT OF TUBERCULOSIS – OBJECTIVES AND REGIMENS



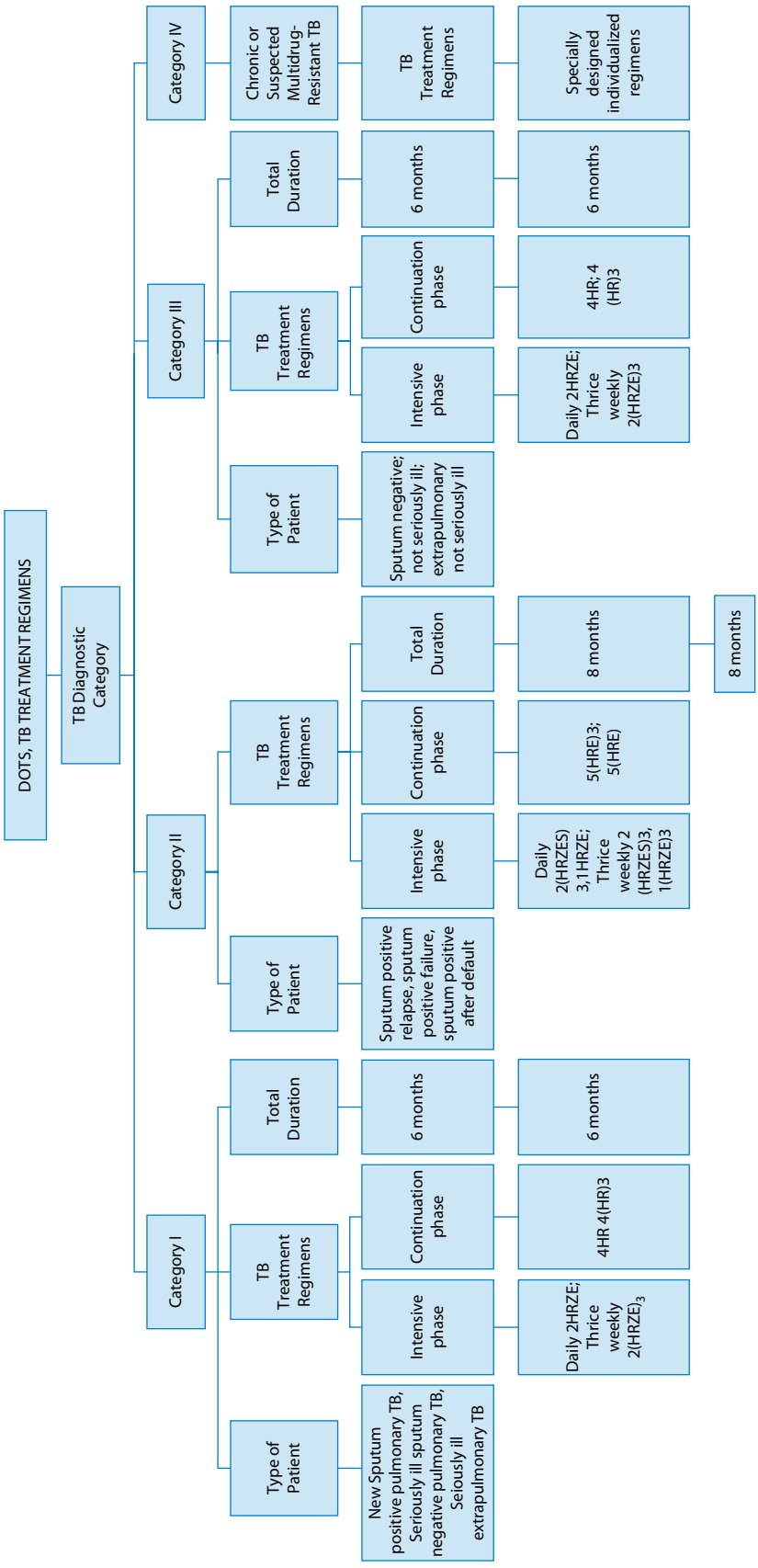
65.7 DOSES OF COMMONLY USED ANTI-TB DRUGS

Recommended dosage	Daily mg/kg	50 kg	Thrice weekly mg/kg	50 kg
1. Isoniazid (H)	5 (4–6)	300 mg	10 (8–12)	600 mg
2. Rifampicin (R)	10 (8–12)	600 mg	10 (8–12)	600 mg
3. Pyrazinamide (Z)	25 (20–30)	1500 mg	35 (35–40)	2000 mg
4. Ethambutol (E)	15 (15–20)	1000 mg	30 (20–30)	1000 mg
5. Streptomycin (S)	15 (12–18)	1000 mg	15 (12–18)	1000 mg

65.8 WHO GUIDELINES FOR TB TREATMENT

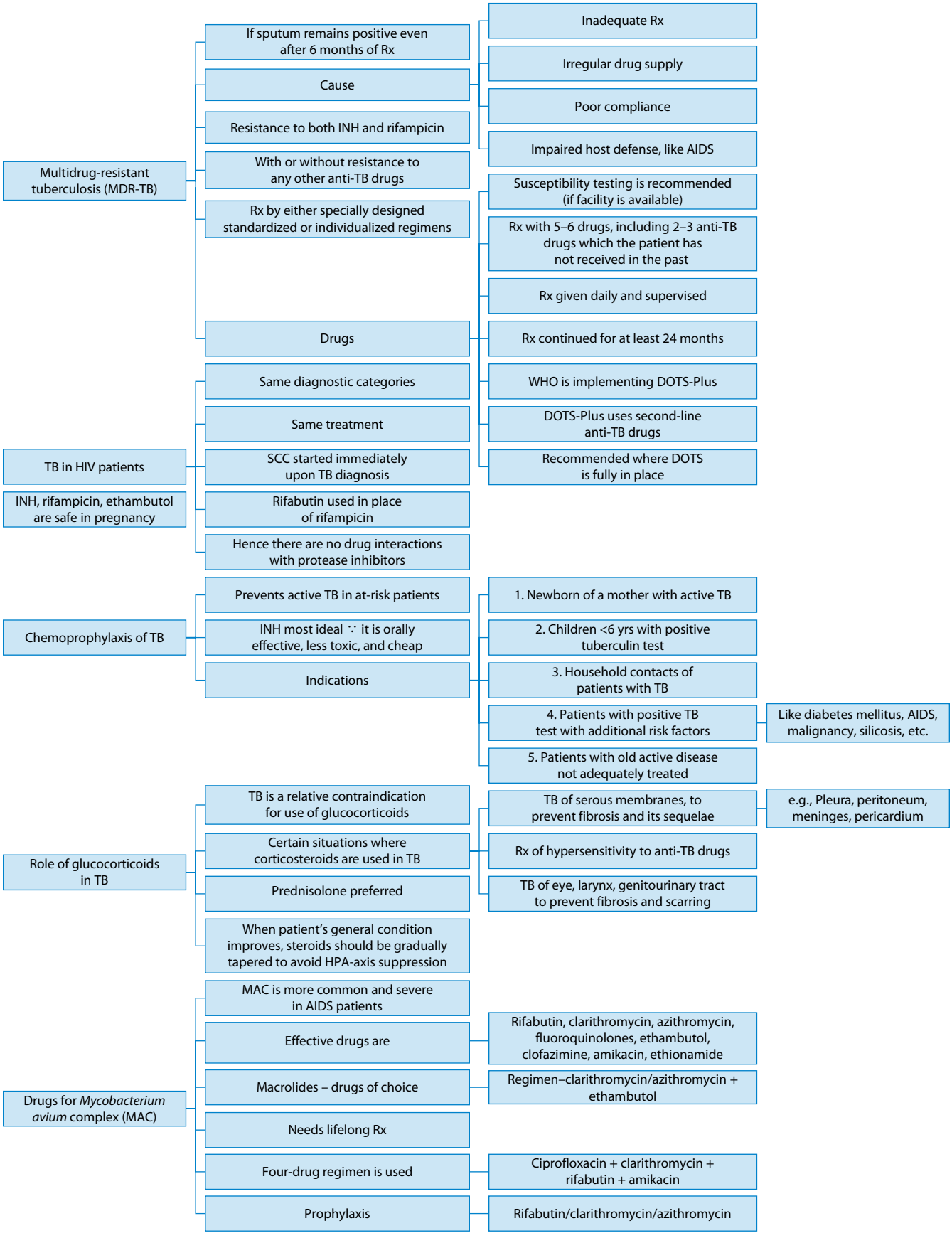


65.9 DOTS, TB TREATMENT REGIMENS



- Prefix before number indicates months of treatment
- Subscript after regimen indicates number of doses per week
- No subscript means regimen is given daily
- H - isoniazid, R - rifampicin, Z - pyrazinamide, E - ethambutol, S - streptomycin
- HRZ are given orally
- S is given IM

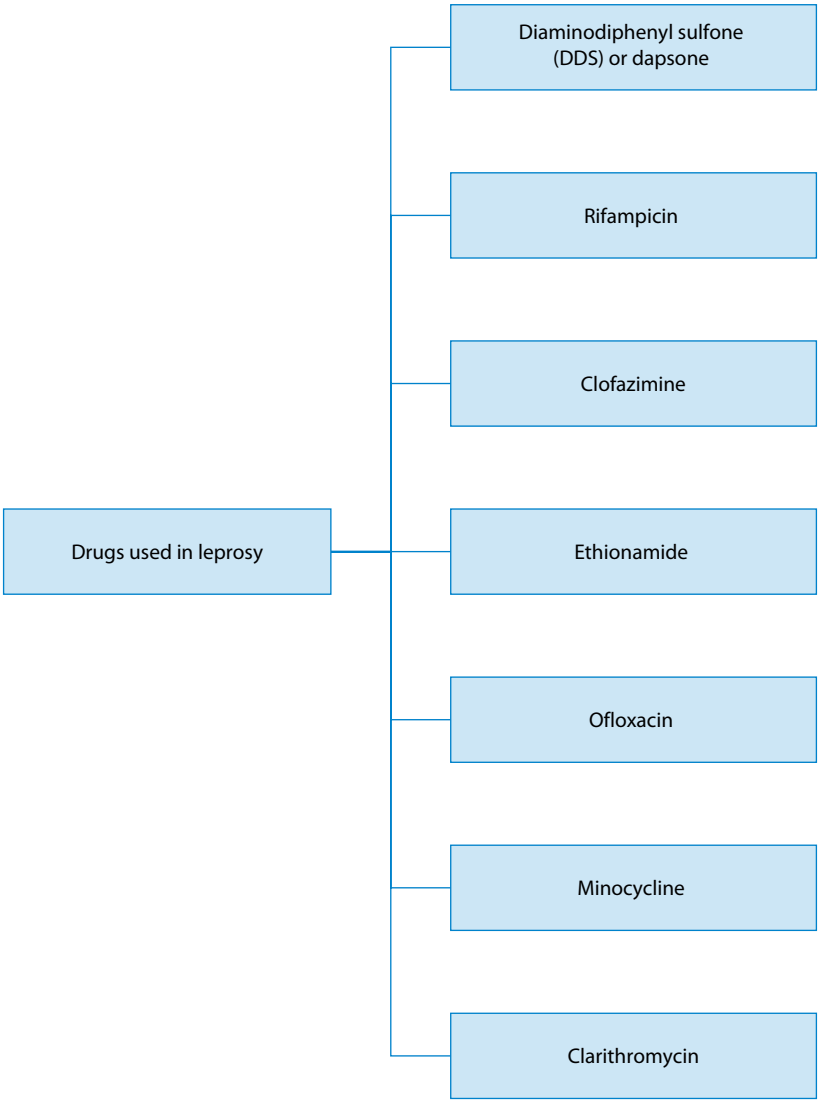
65.10 MULTIDRUG-RESISTANT TUBERCULOSIS (MDR-TB), TB IN HIV PATIENTS, TB IN PREGNANCY, CHEMOPROPHYLAXIS OF TB, ROLE OF GLUCOCORTICOIDS IN TB, AND DRUGS FOR *MYCOBACTERIUM AVIUM* COMPLEX (MAC)



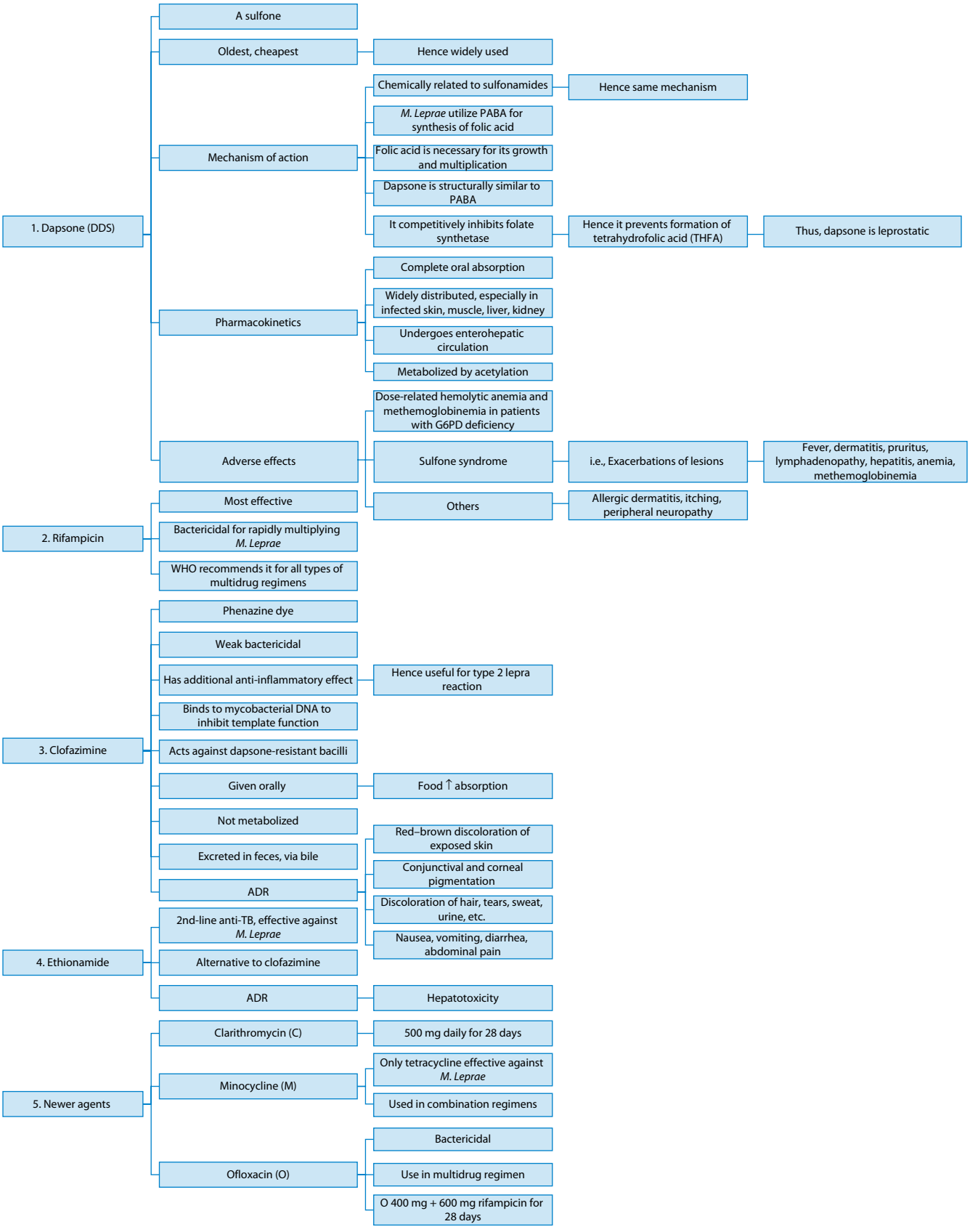
66

Chemotherapy of leprosy

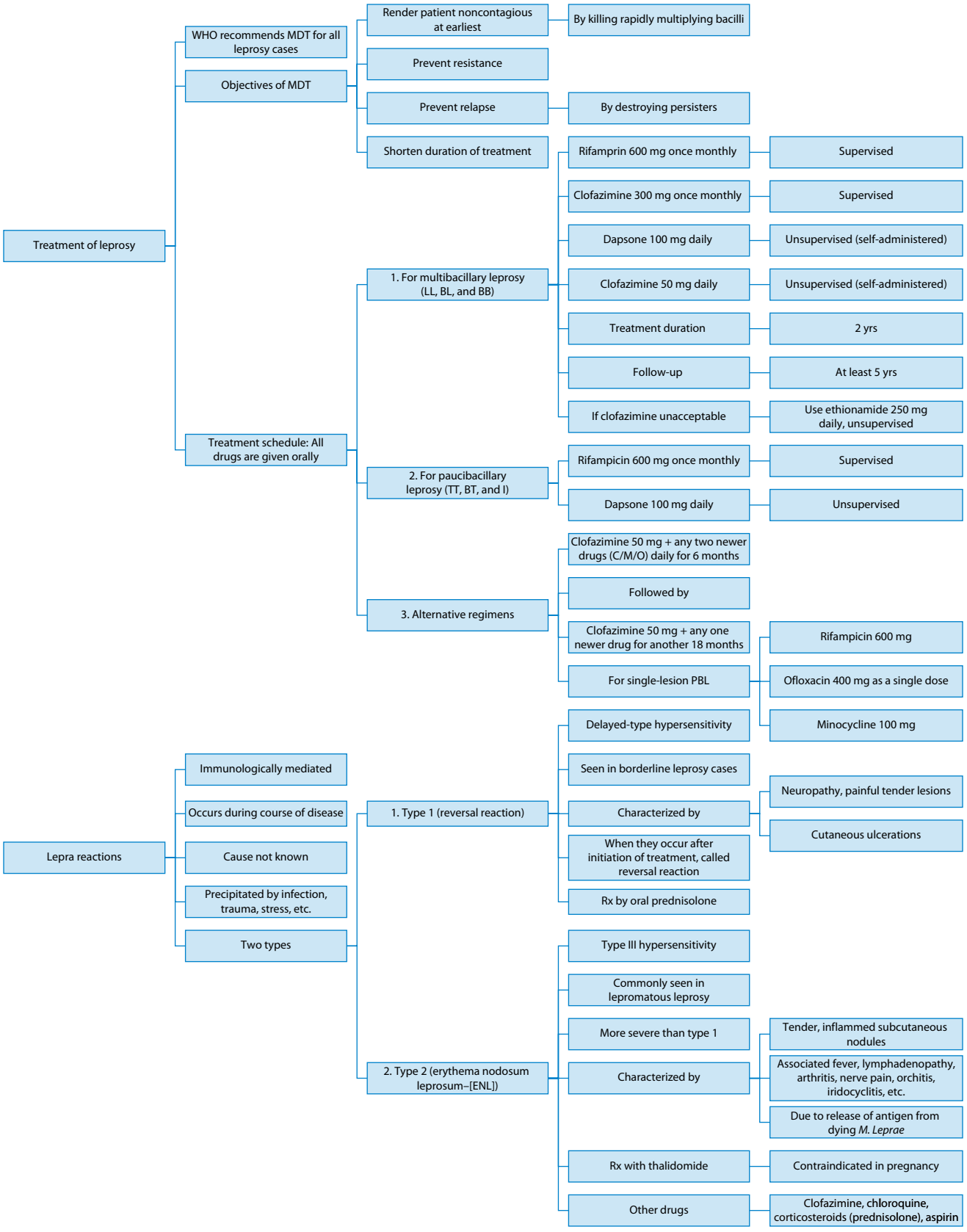
66.1 DRUGS USED IN LEPROSY



66.2 DAPSONE (DDS), RIFAMPICIN, CLOFAZIMINE, ETHIONAMIDE, AND NEWER AGENTS



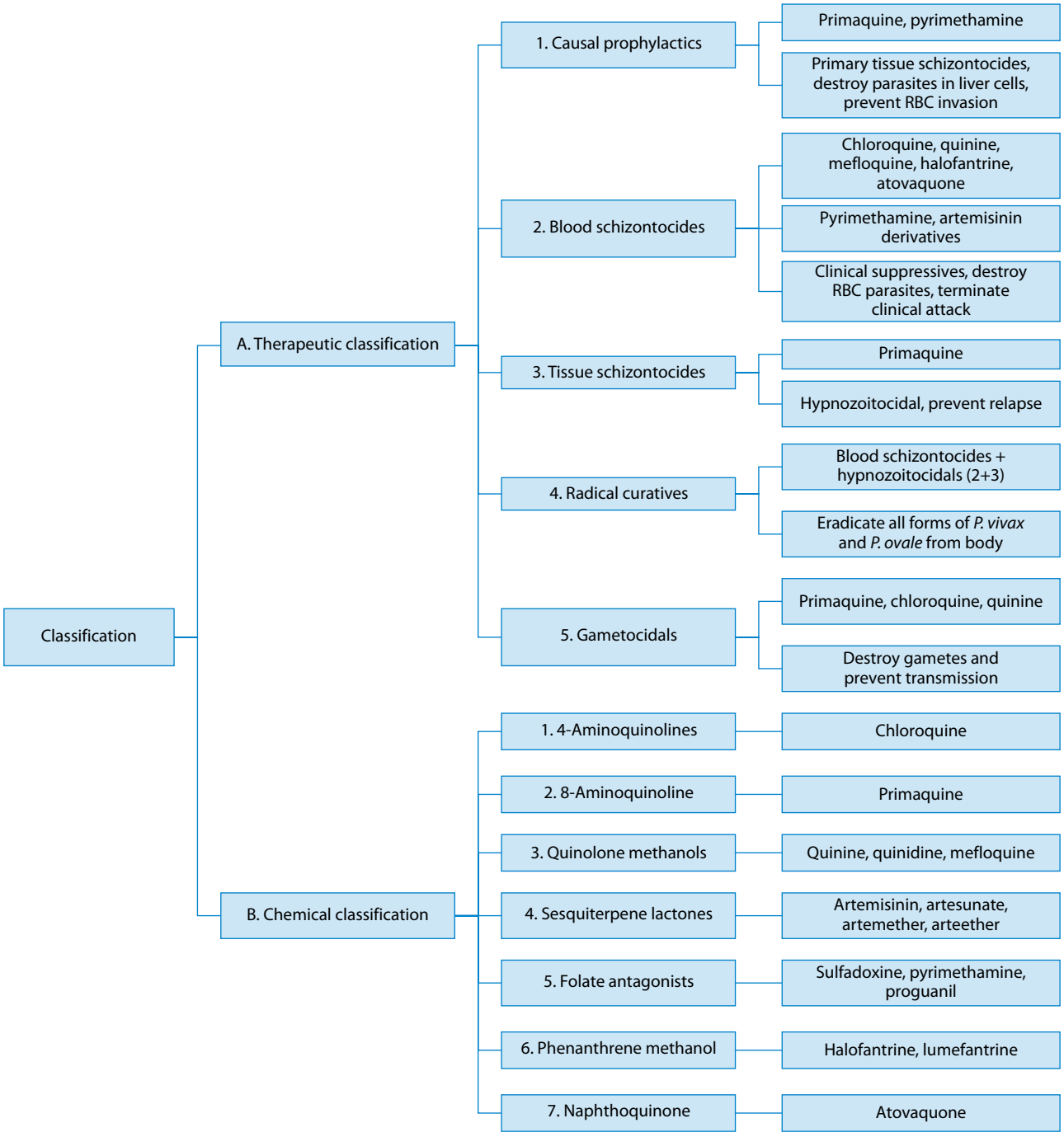
66.3 TREATMENT OF LEPROSY AND LEPRA REACTIONS



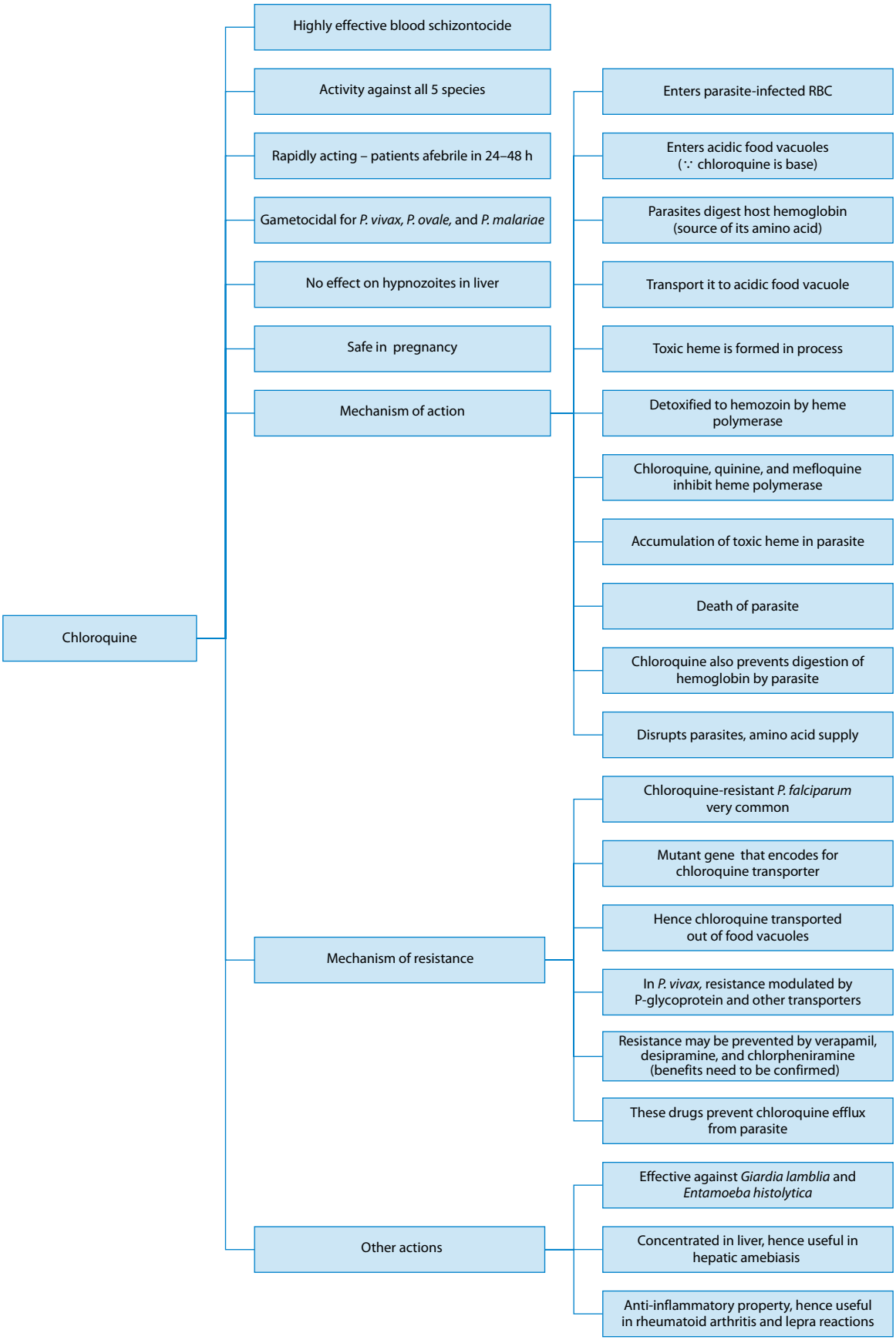
67

Chemotherapy of malaria

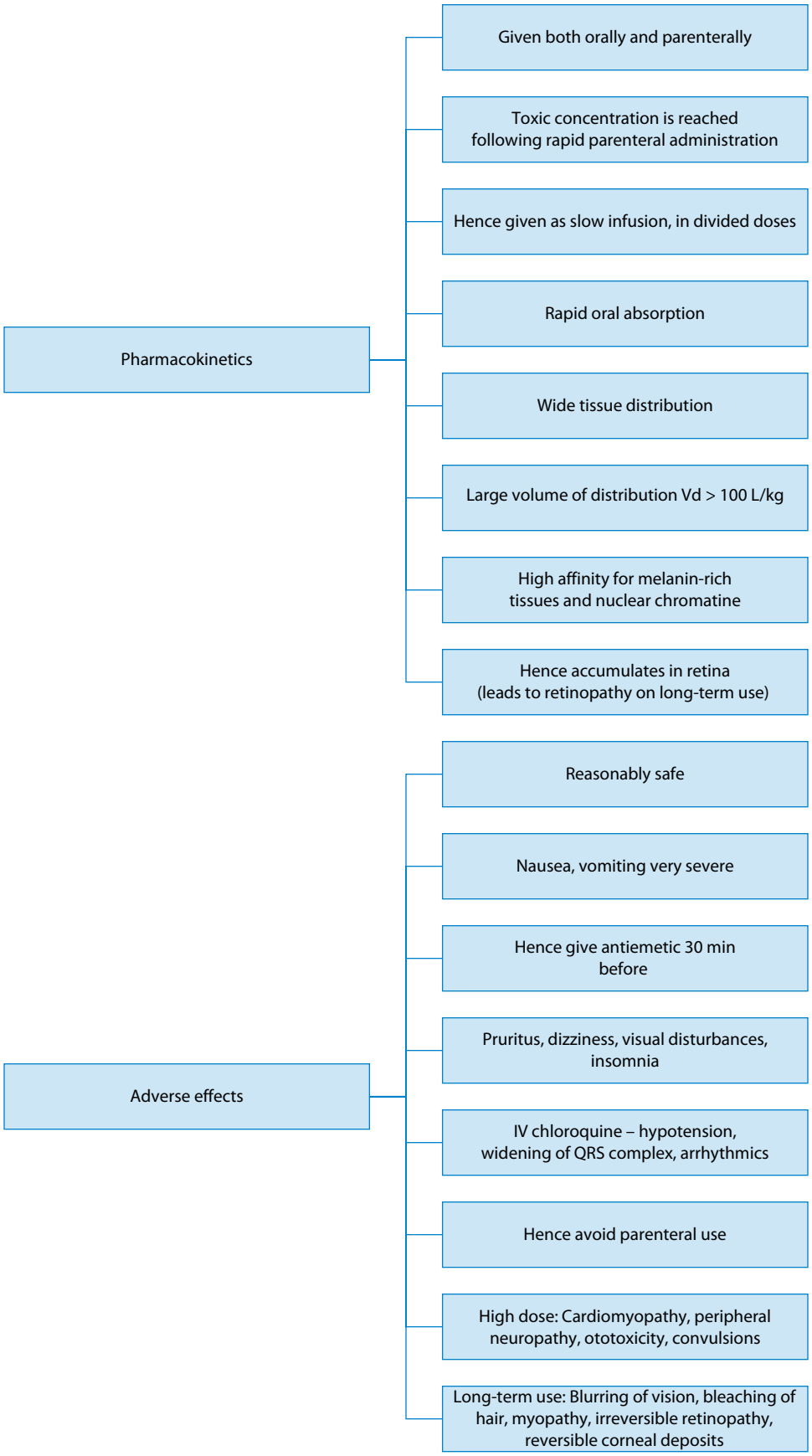
67.1 CLASSIFICATION OF ANTIMALARIALS



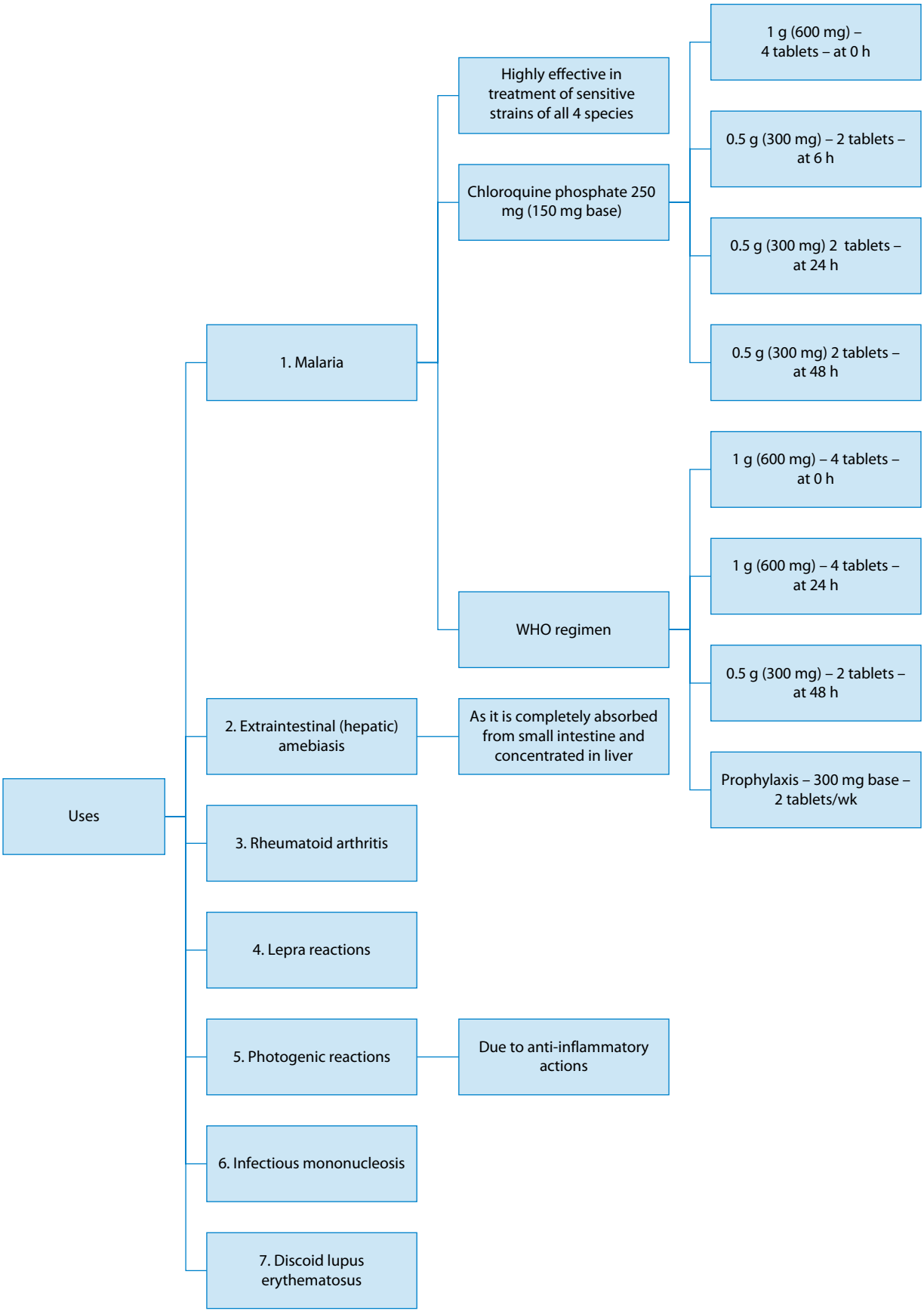
67.2 CHLOROQUINE – MECHANISM OF ACTION AND RESISTANCE



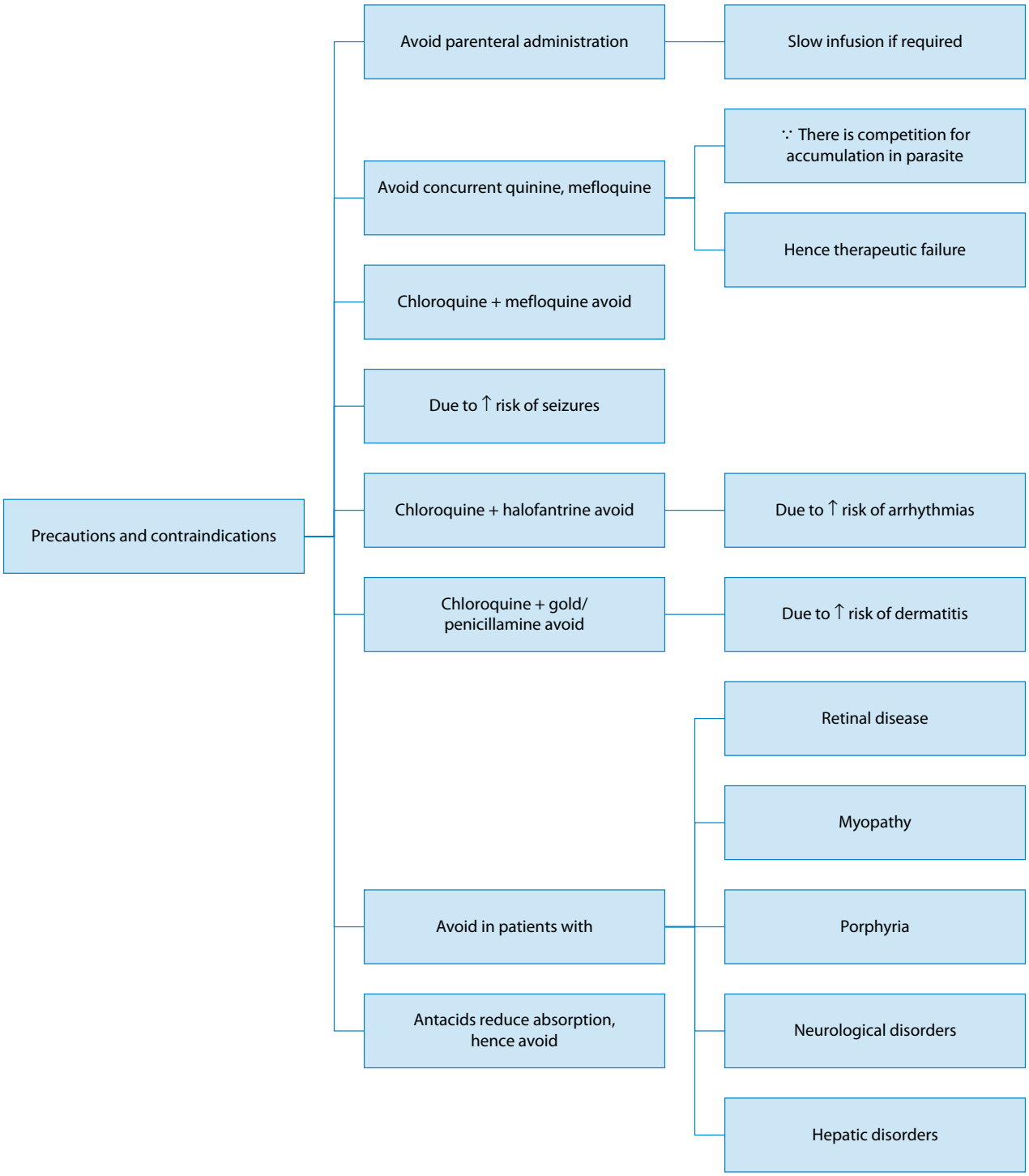
67.3 PHARMACOKINETICS AND ADVERSE EFFECTS



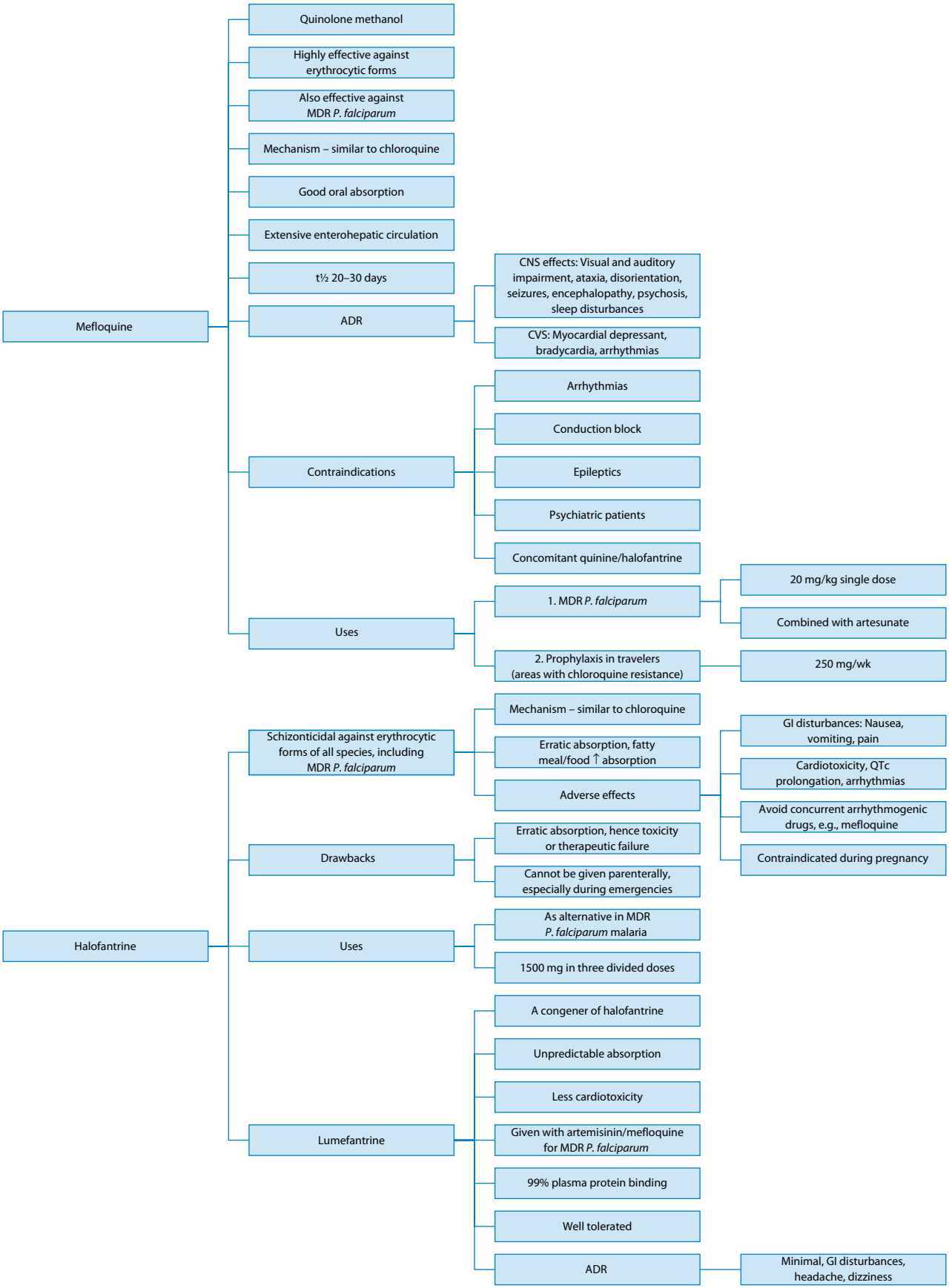
67.4 USES


Uses
1. Malaria
Highly effective in treatment of sensitive strains of all 4 species
Chloroquine phosphate 250 mg (150 mg base)
1 g (600 mg) – 4 tablets – at 0 h
0.5 g (300 mg) – 2 tablets – at 6 h
0.5 g (300 mg) 2 tablets – at 24 h
0.5 g (300 mg) 2 tablets – at 48 h
WHO regimen
1 g (600 mg) – 4 tablets – at 0 h
1 g (600 mg) – 4 tablets – at 24 h
0.5 g (300 mg) – 2 tablets – at 48 h
Prophylaxis – 300 mg base – 2 tablets/wk
2. Extraintestinal (hepatic) amebiasis
As it is completely absorbed from small intestine and concentrated in liver
3. Rheumatoid arthritis
4. Lepra reactions
5. Photogenic reactions
Due to anti-inflammatory actions
6. Infectious mononucleosis
7. Discoid lupus erythematosus

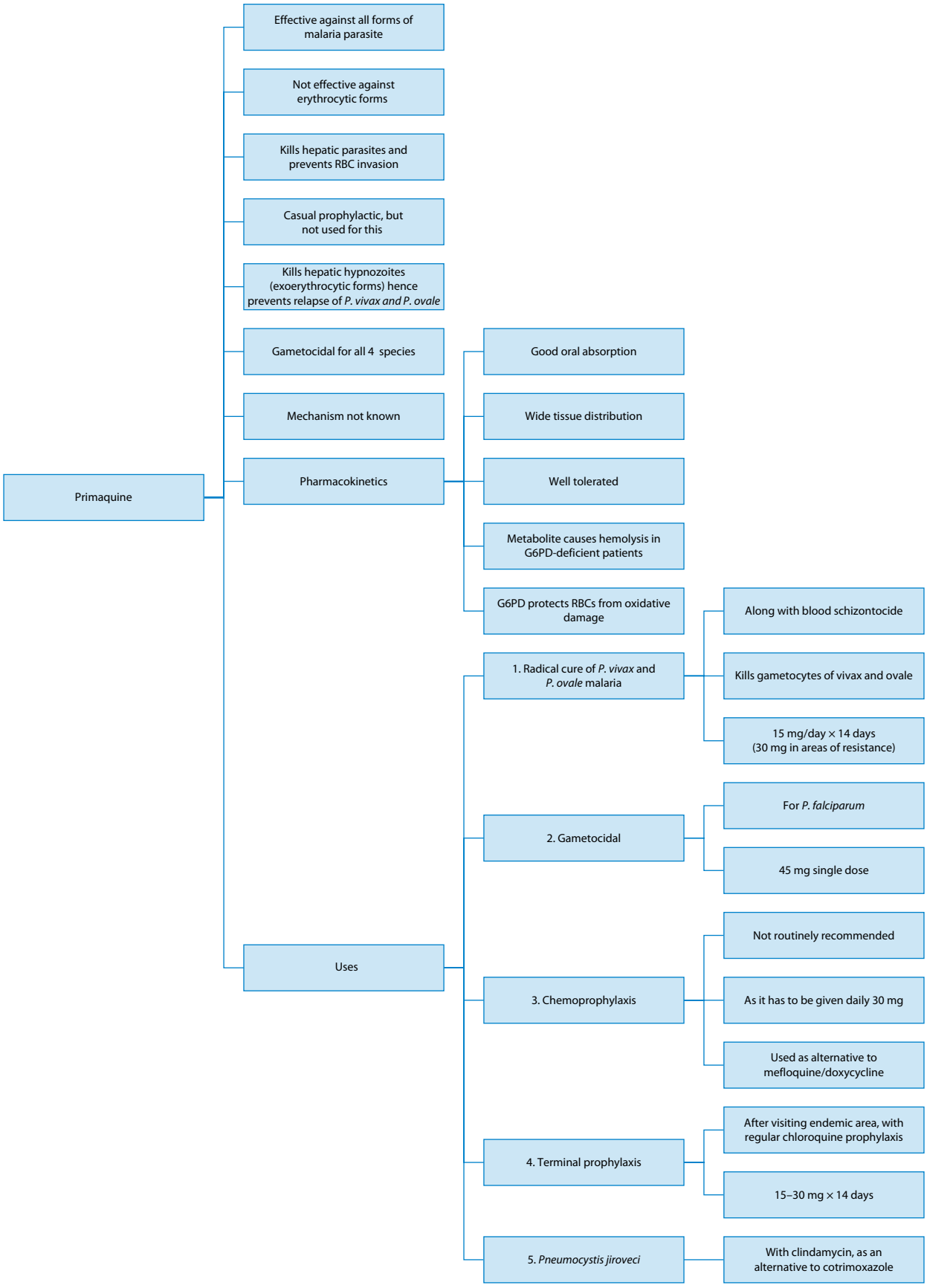
67.5 PRECAUTIONS AND CONTRAINDICATIONS



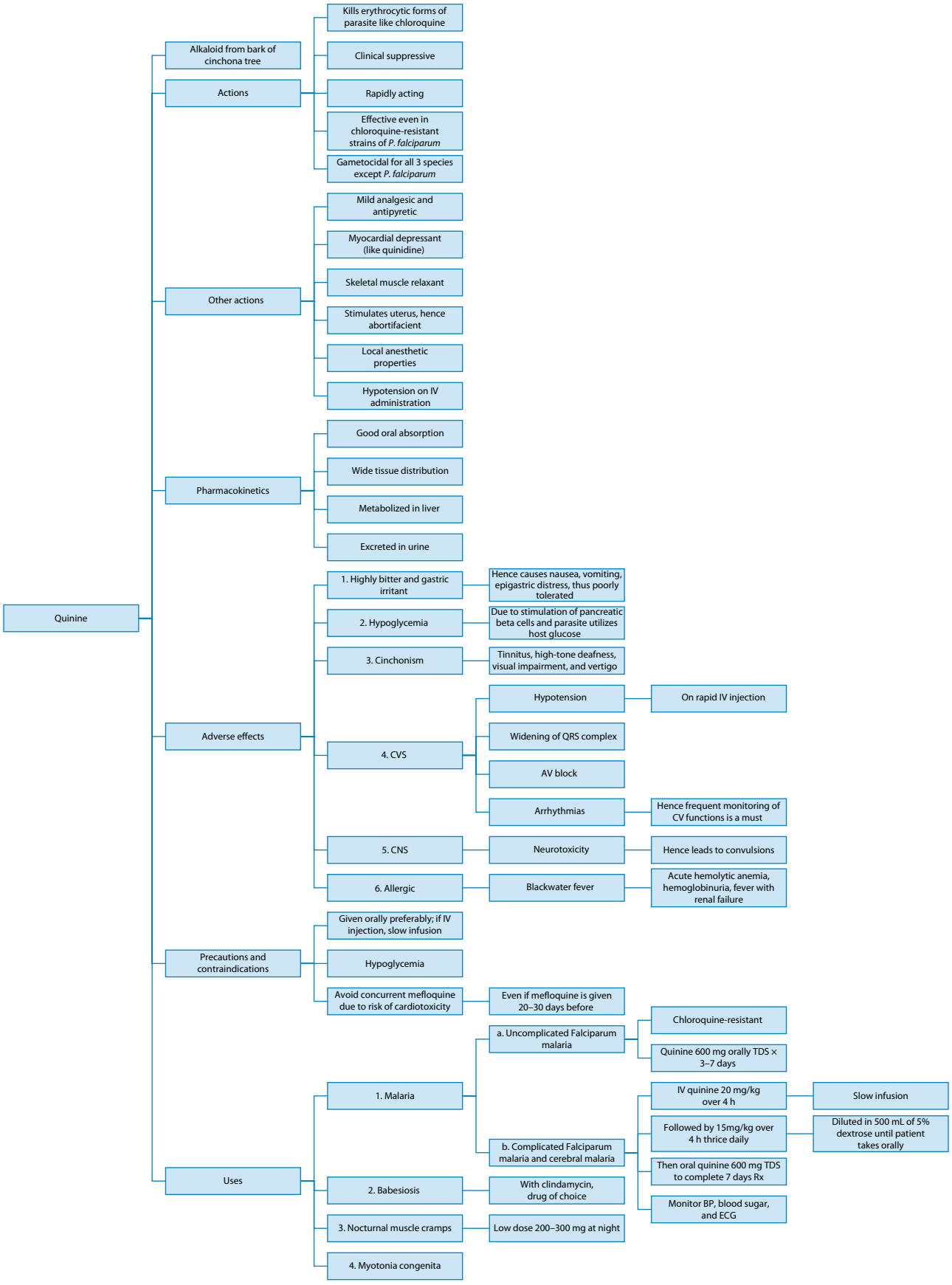
67.6 MEFLOQUINE AND HALOFANTRINE



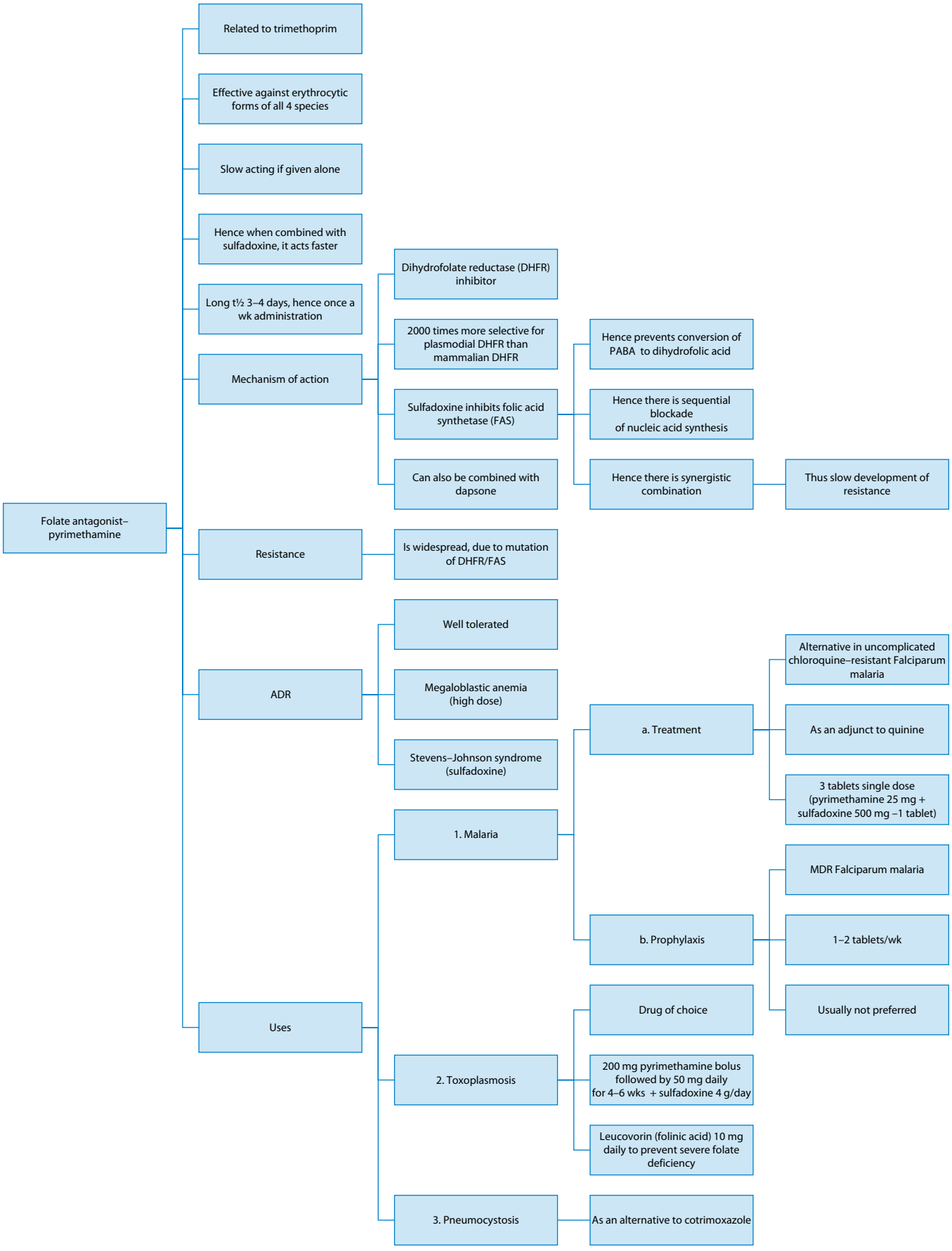
67.7 PRIMAQUINE



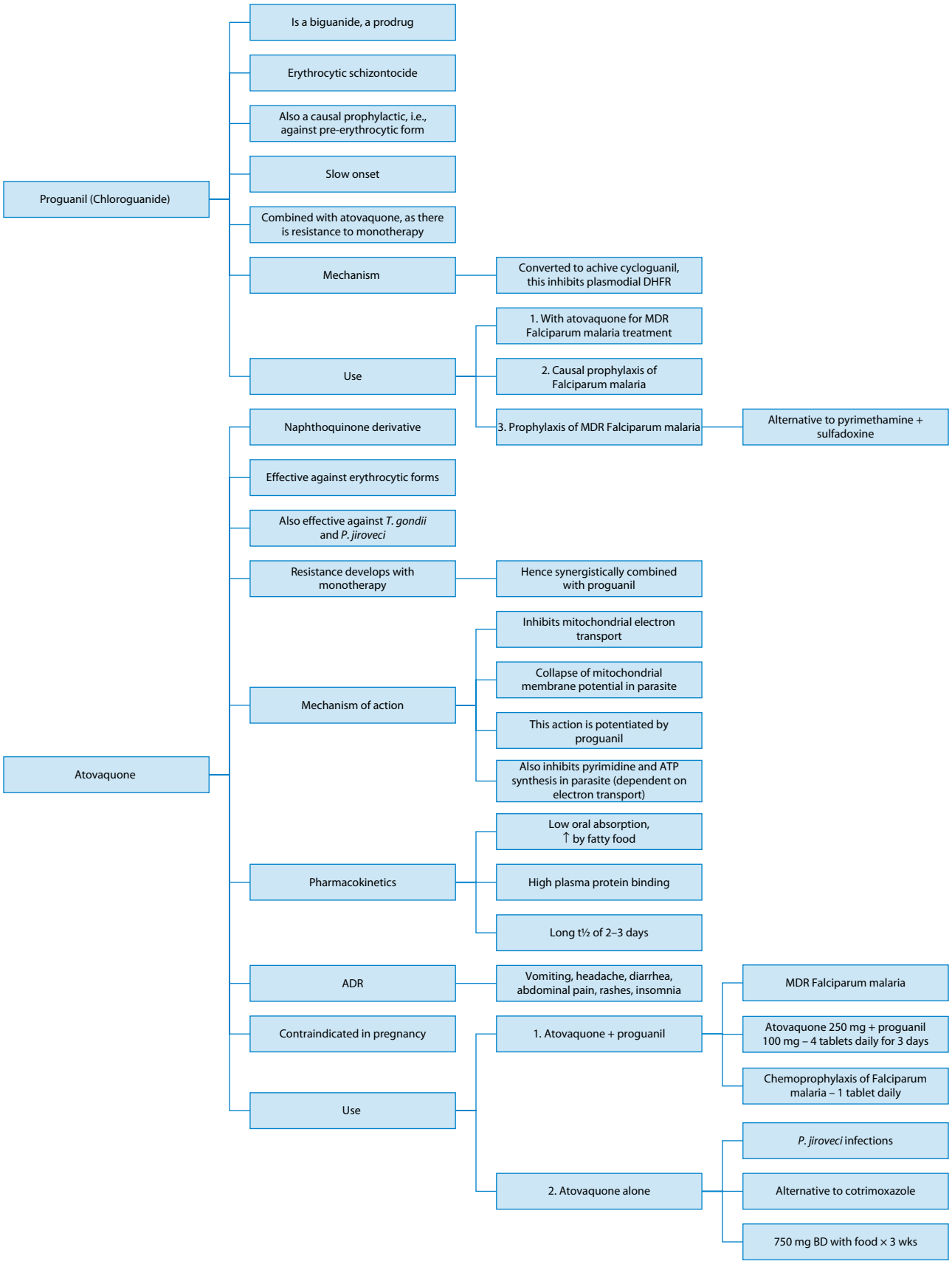
67.8 QUININE


Quinine
Alkaloid from bark of cinchona tree
Actions
Kills erythrocytic forms of parasite like chloroquine
Clinical suppressive
Rapidly acting
Effective even in chloroquine-resistant strains of P. falciparum
Gametocidal for all 3 species except P. falciparum
Other actions
Mild analgesic and antipyretic
Myocardial depressant (like quinidine)
Skeletal muscle relaxant
Stimulates uterus, hence abortifacient
Local anesthetic properties
Hypotension on IV administration
Pharmacokinetics
Good oral absorption
Wide tissue distribution
Metabolized in liver
Excreted in urine
Adverse effects
1. Highly bitter and gastric irritant
Hence causes nausea, vomiting, epigastric distress, thus poorly tolerated
2. Hypoglycemia
Due to stimulation of pancreatic beta cells and parasite utilizes host glucose
3. Cinchonism
Tinnitus, high-tone deafness, visual impairment, and vertigo
4. CVS
Hypotension
On rapid IV injection
Widening of QRS complex
AV block
Arrhythmias
Hence frequent monitoring of CV functions is a must
5. CNS
Neurotoxicity
Hence leads to convulsions
6. Allergic
Blackwater fever
Acute hemolytic anemia, hemoglobinuria, fever with renal failure
Precautions and contraindications
Given orally preferably; if IV injection, slow infusion
Hypoglycemia
Avoid concurrent mefloquine due to risk of cardiotoxicity
Even if mefloquine is given 20–30 days before
Uses
1. Malaria
a. Uncomplicated Falciparum malaria
Chloroquine-resistant
Quinine 600 mg orally TDS × 3–7 days
b. Complicated Falciparum malaria and cerebral malaria
IV quinine 20 mg/kg over 4 h
Slow infusion
Followed by 15mg/kg over 4 h thrice daily
Diluted in 500 mL of 5% dextrose until patient takes orally
Then oral quinine 600 mg TDS to complete 7 days Rx
Monitor BP, blood sugar, and ECG
2. Babesiosis
With clindamycin, drug of choice
3. Nocturnal muscle cramps
Low dose 200–300 mg at night
4. Myotonia congenita

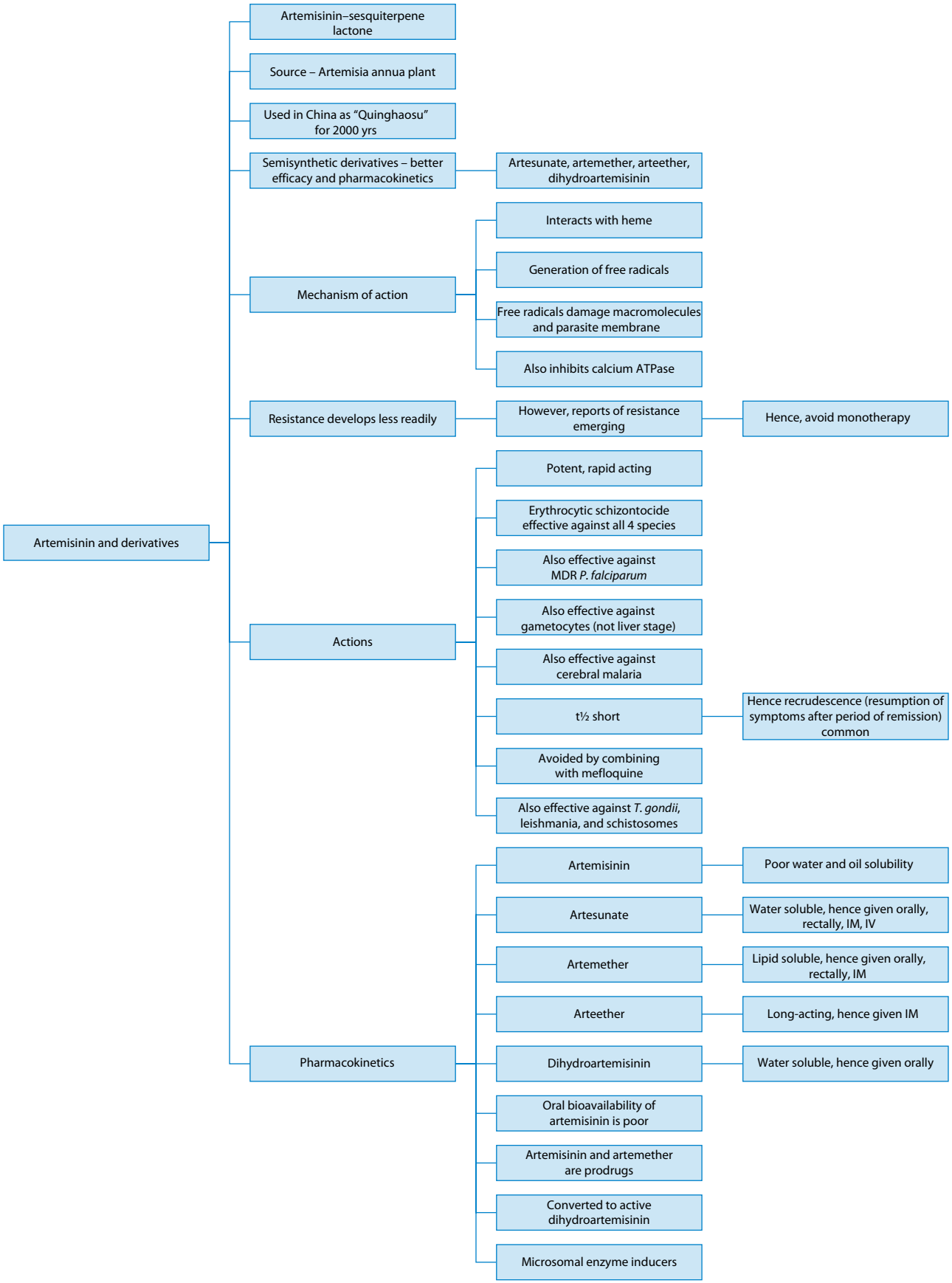
67.9 FOLATE ANTAGONIST – PYRIMETHAMINE



67.10 PROGUANIL (CHLOROGUANIDE) AND ATOVAQUONE

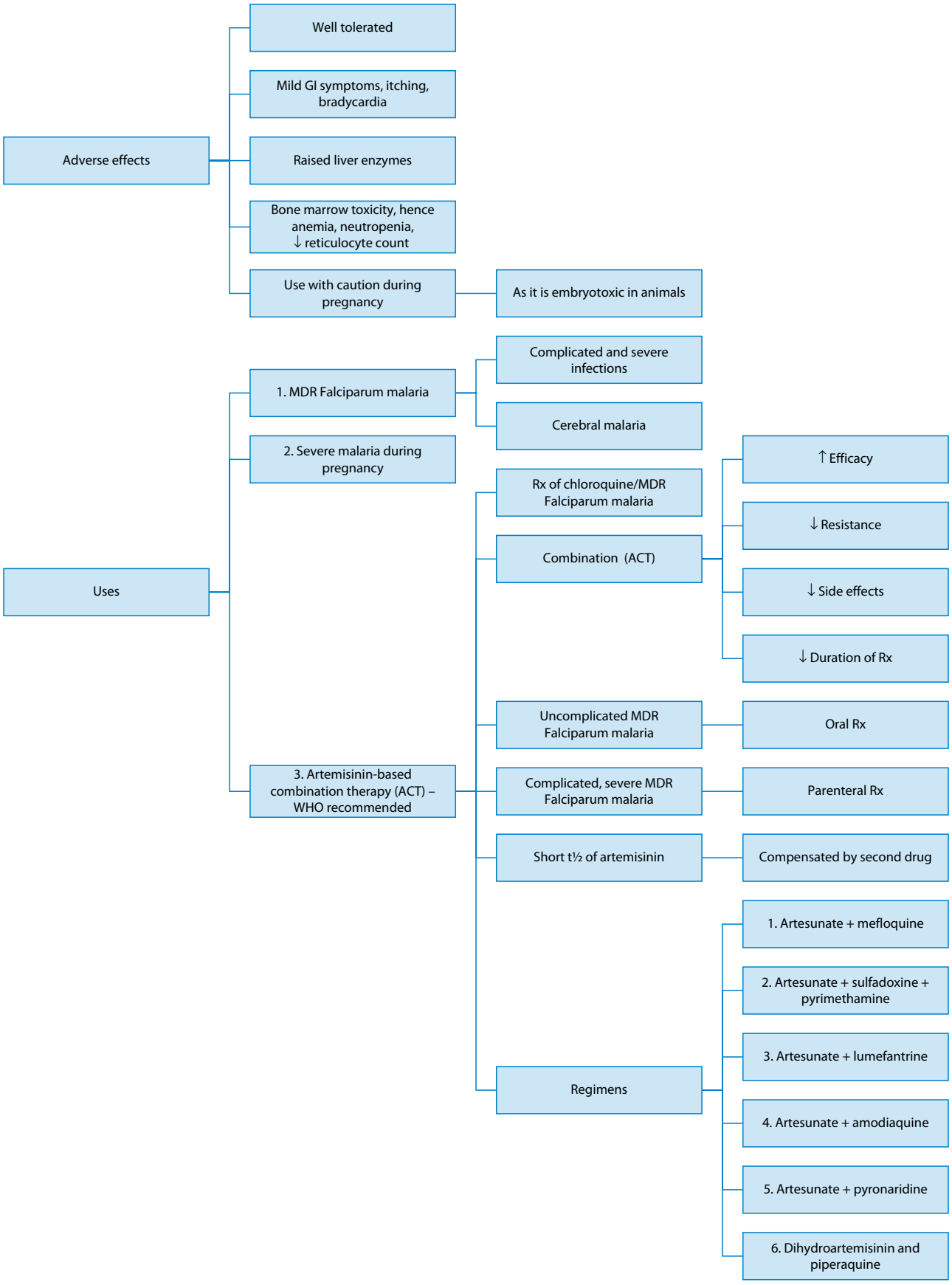


67.11 ARTEMISININ AND DERIVATIVES

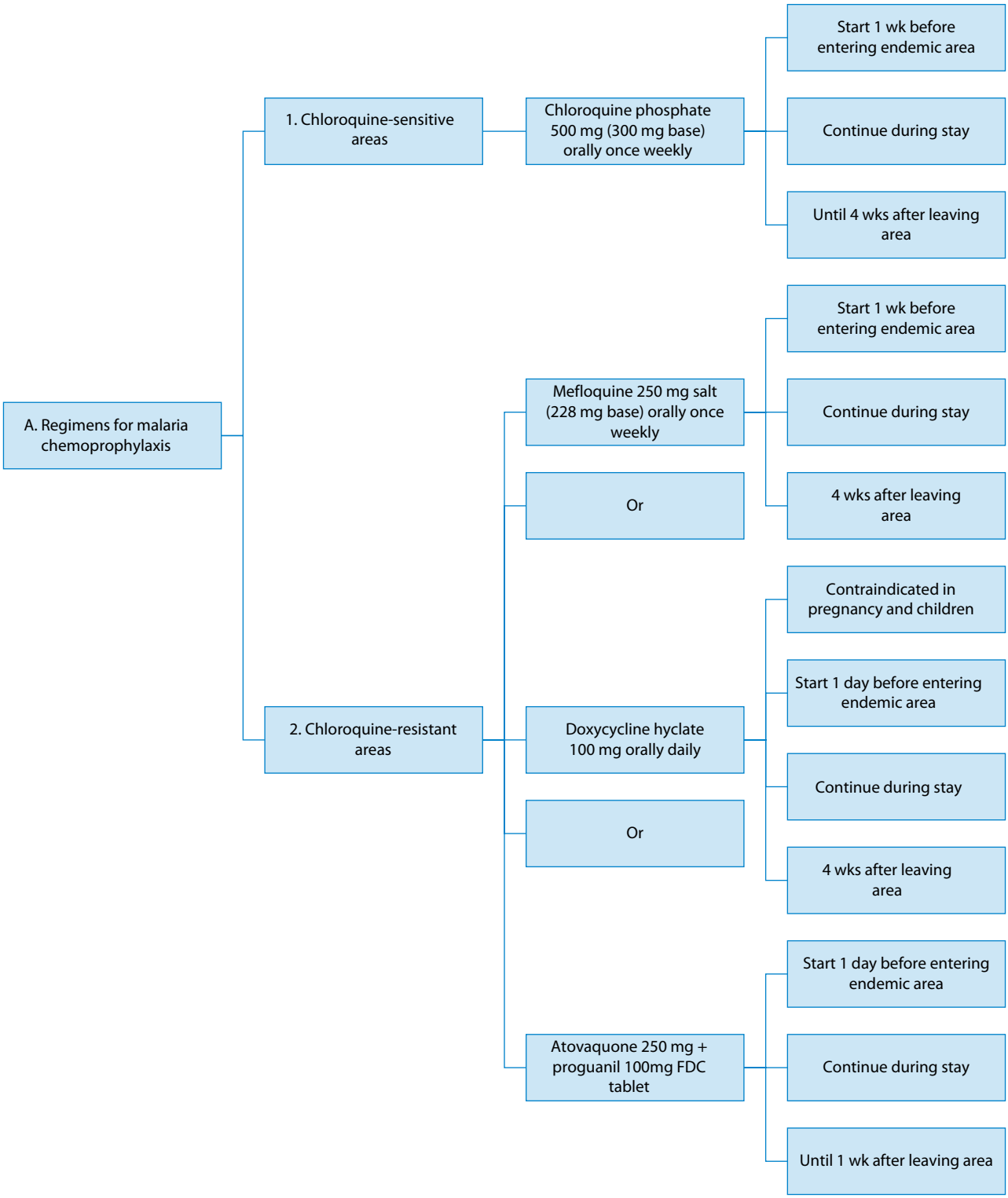


(*Continued*)

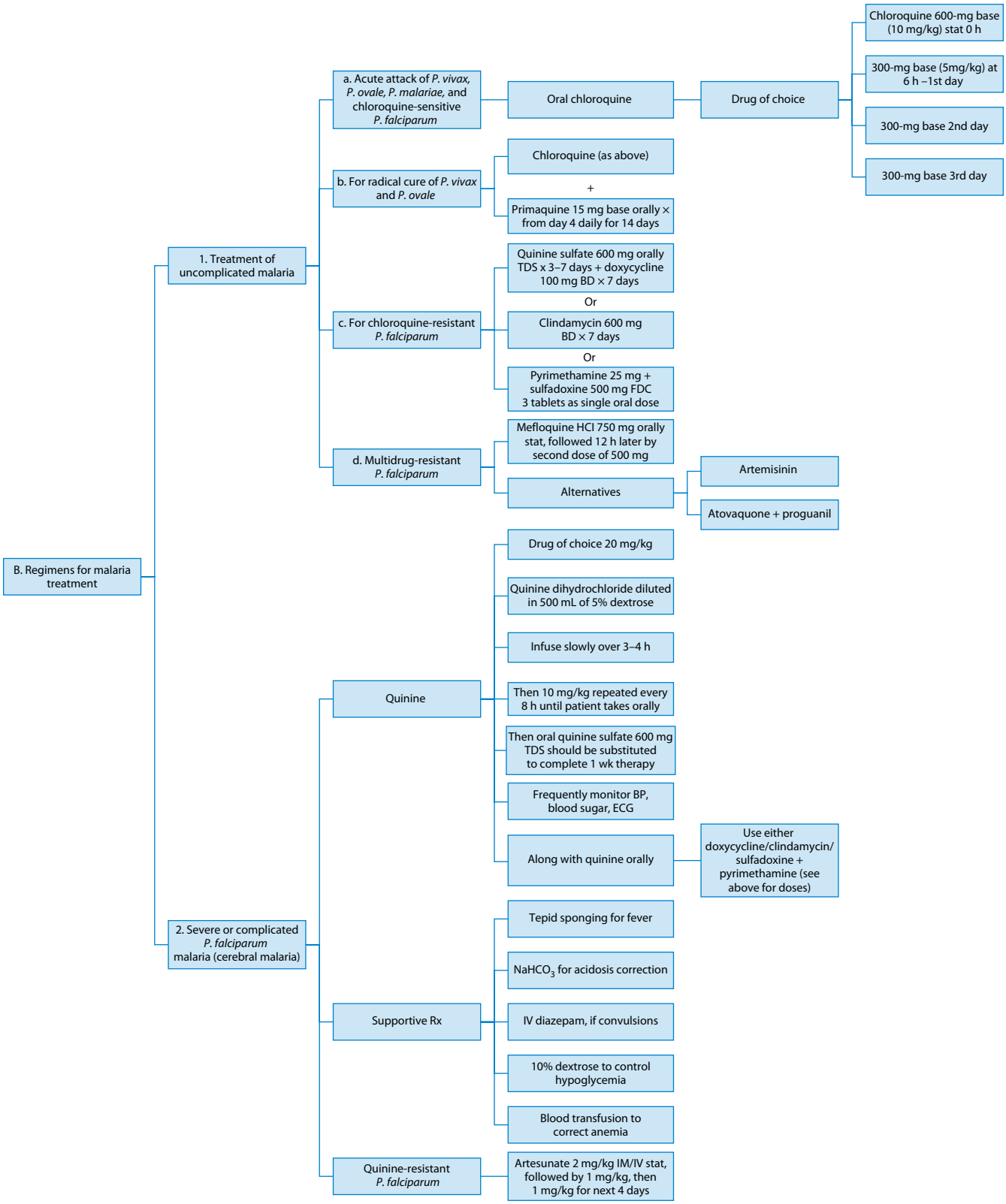
67.11 ARTEMISININ AND DERIVATIVES (Continued)



67.12 REGIMENS FOR MALARIA CHEMOPROPHYLAXIS



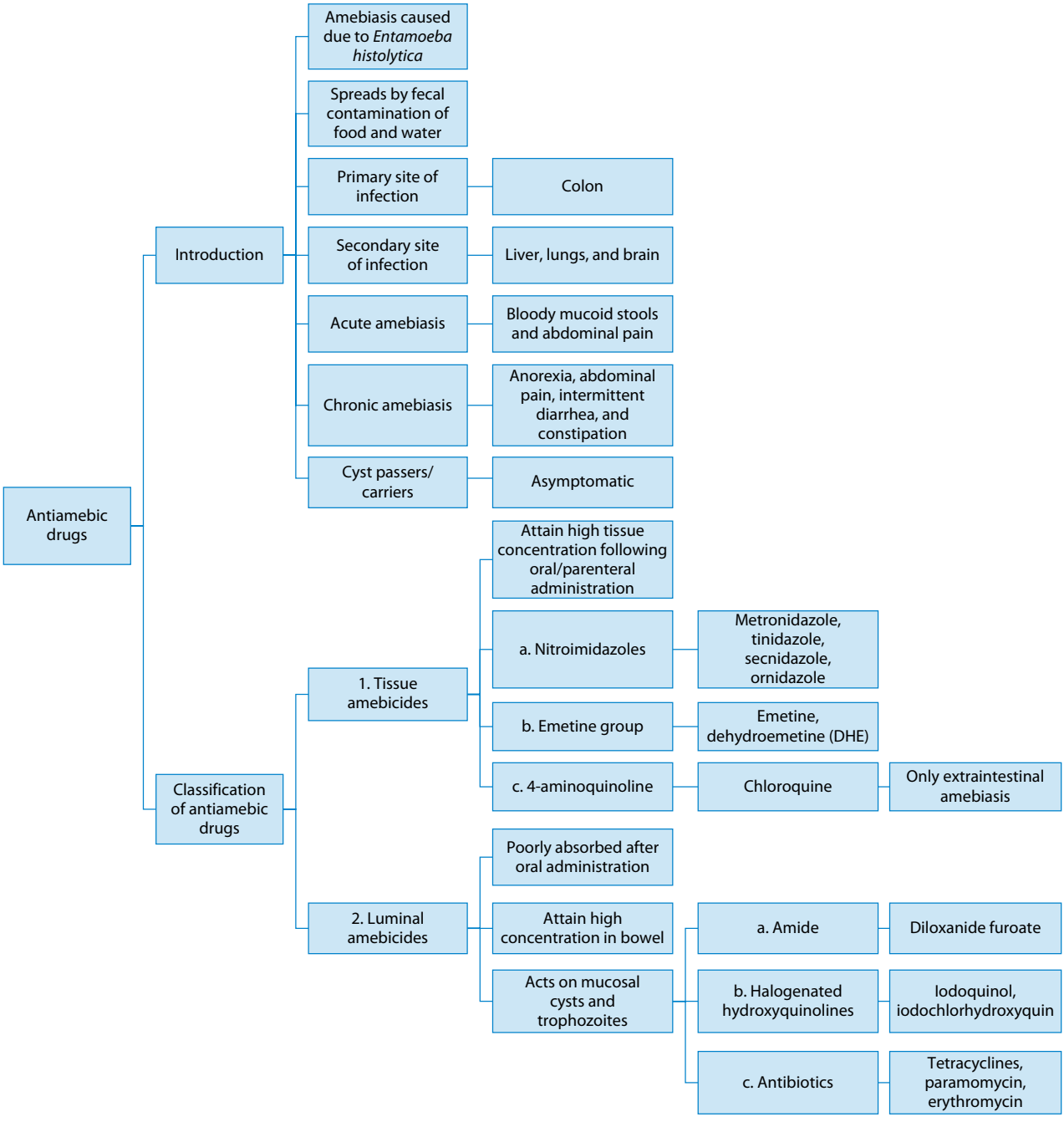
67.13 REGIMENS FOR MALARIA TREATMENT



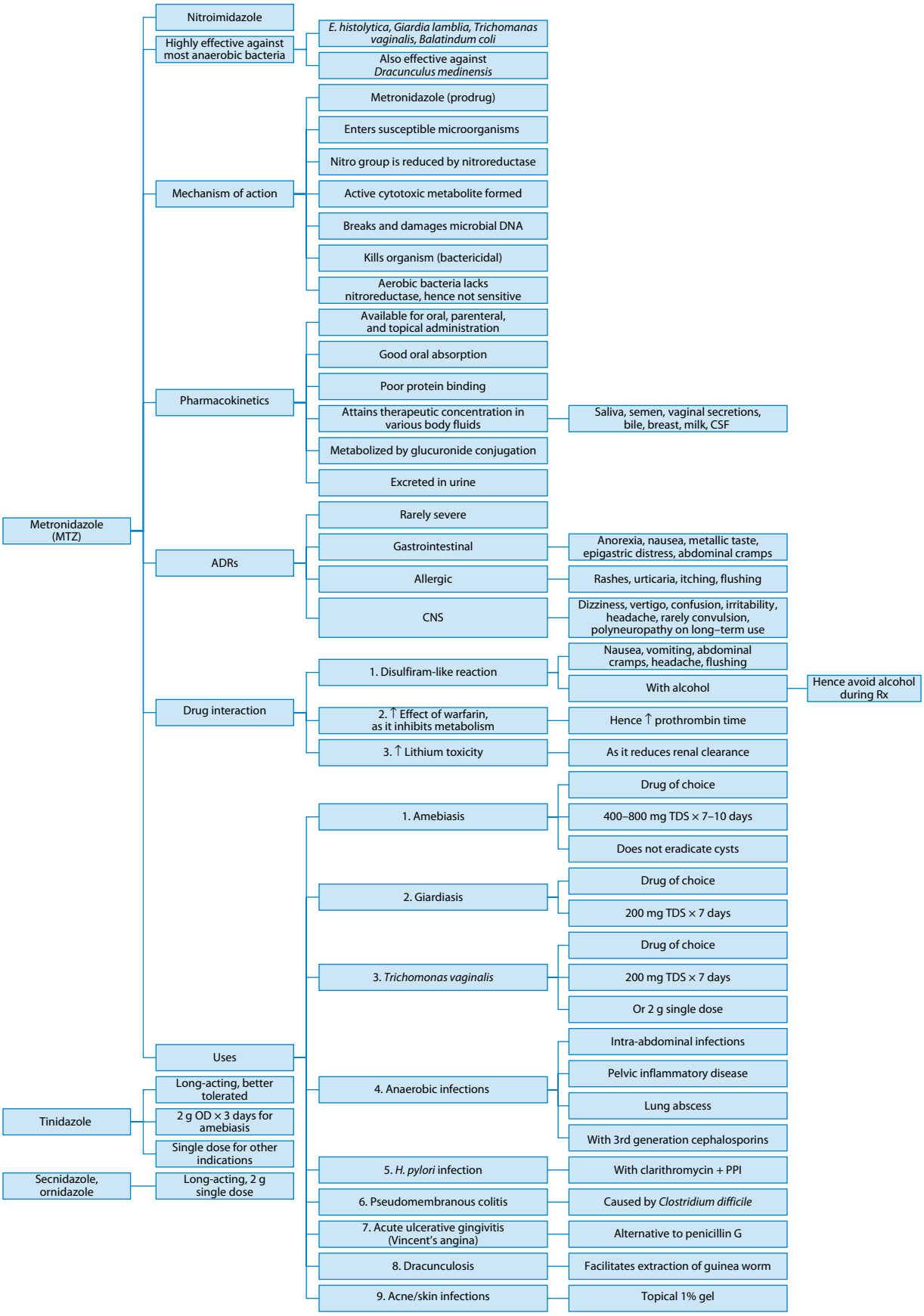
68

Drugs for amebiasis/pneumocystosis/ leishmaniasis/trypanosomiasis

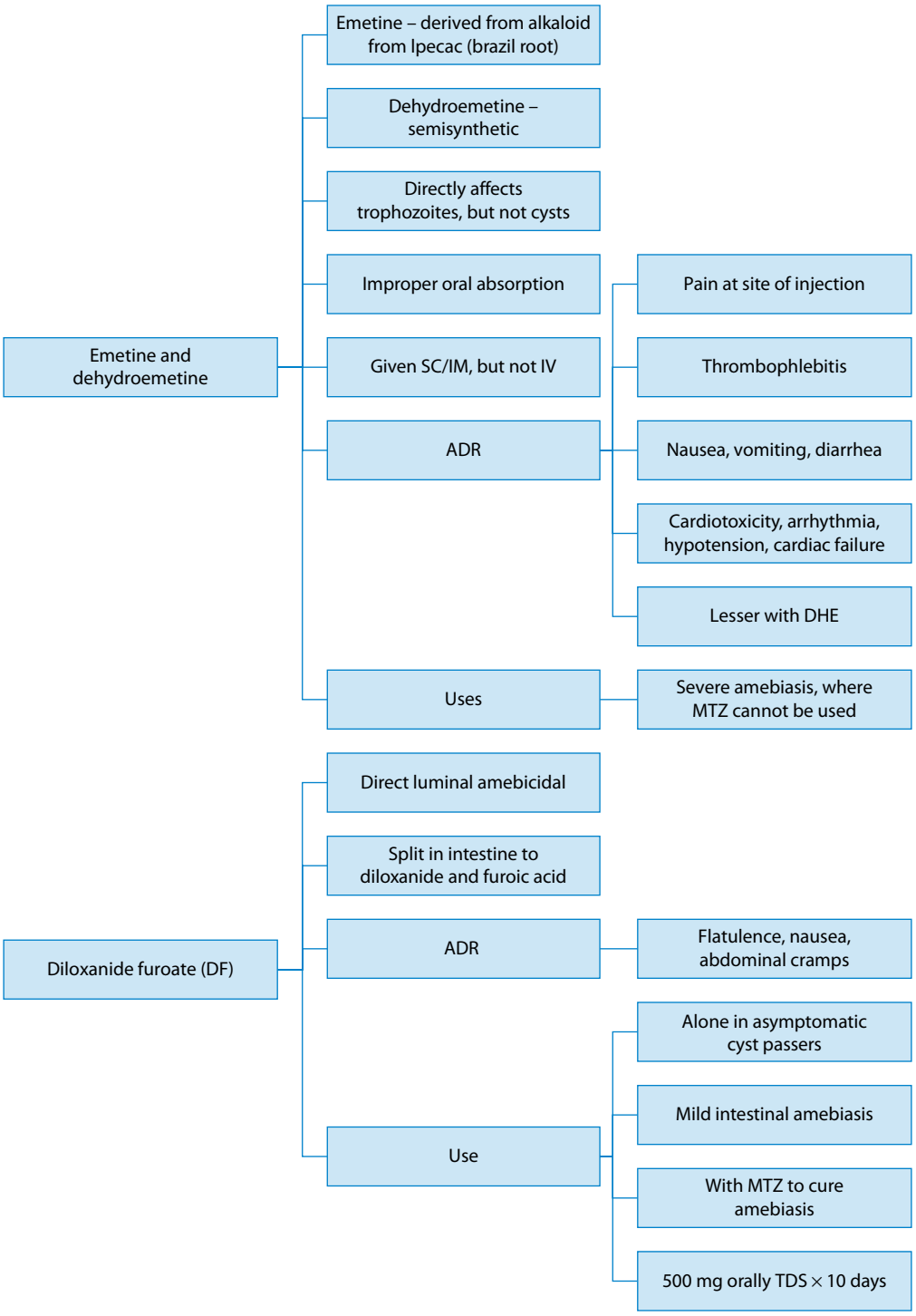
68.1 INTRODUCTION AND DRUGS – CLASSIFICATION



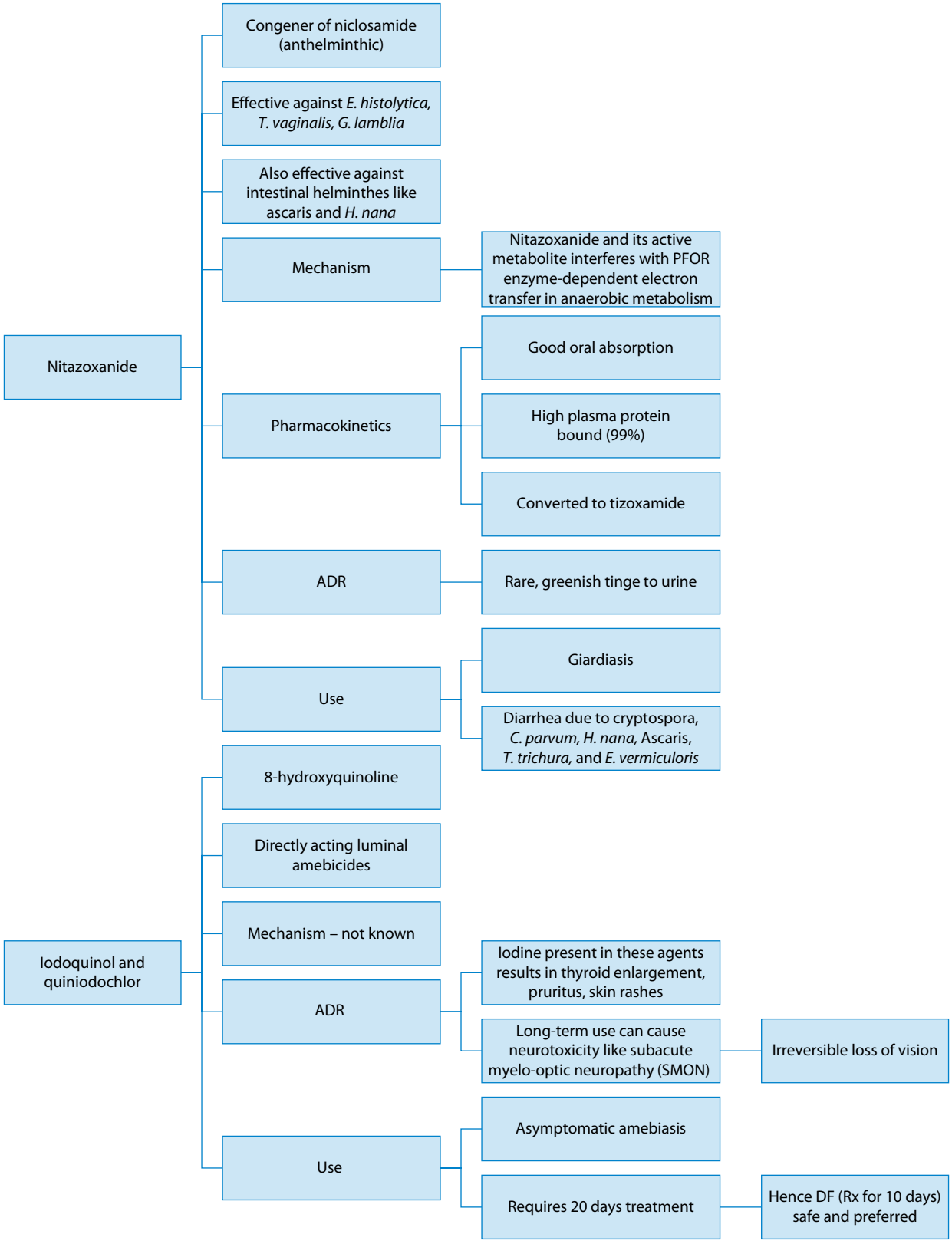
68.2 METRONIDAZOLE (MTZ)



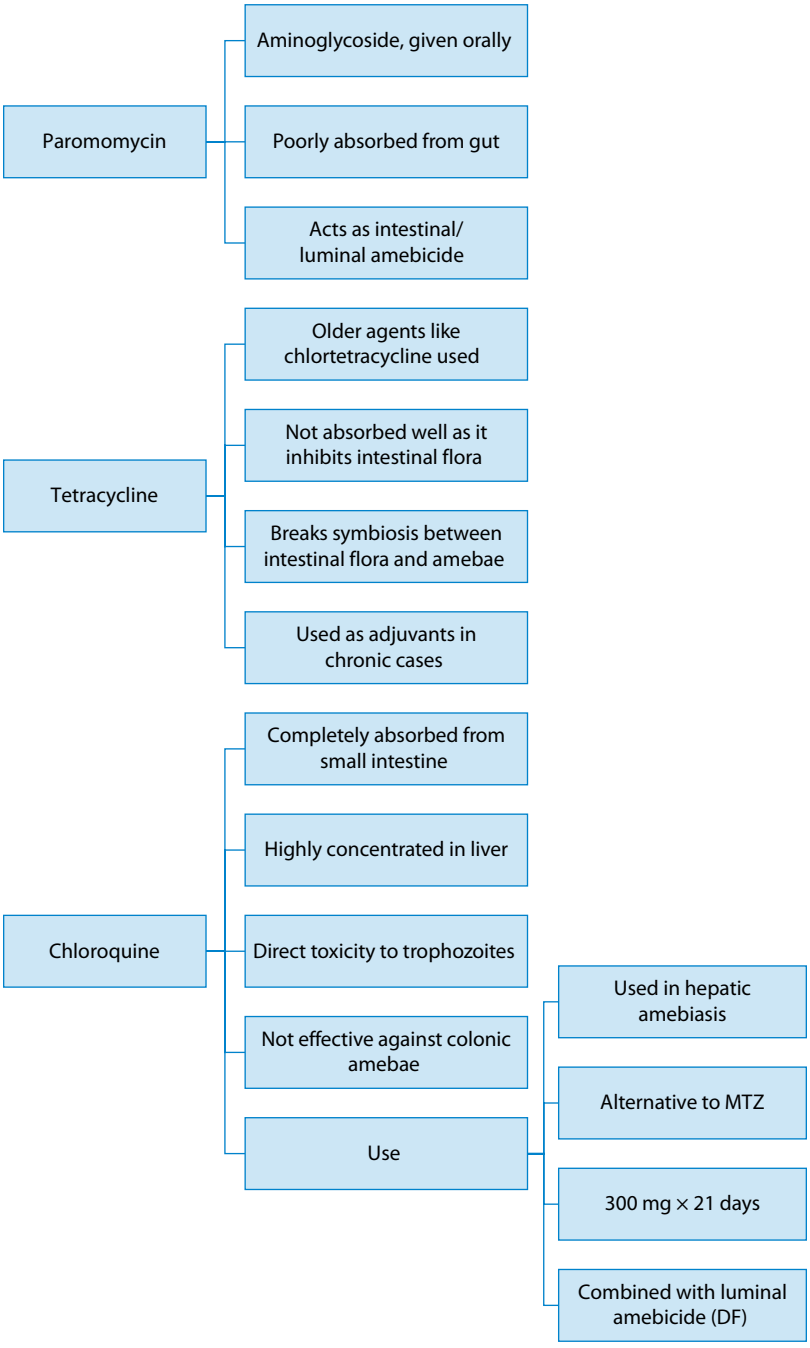
68.3 EMETINE AND DEHYDROEMETINE, DILOXANIDE FUROATE (DF)



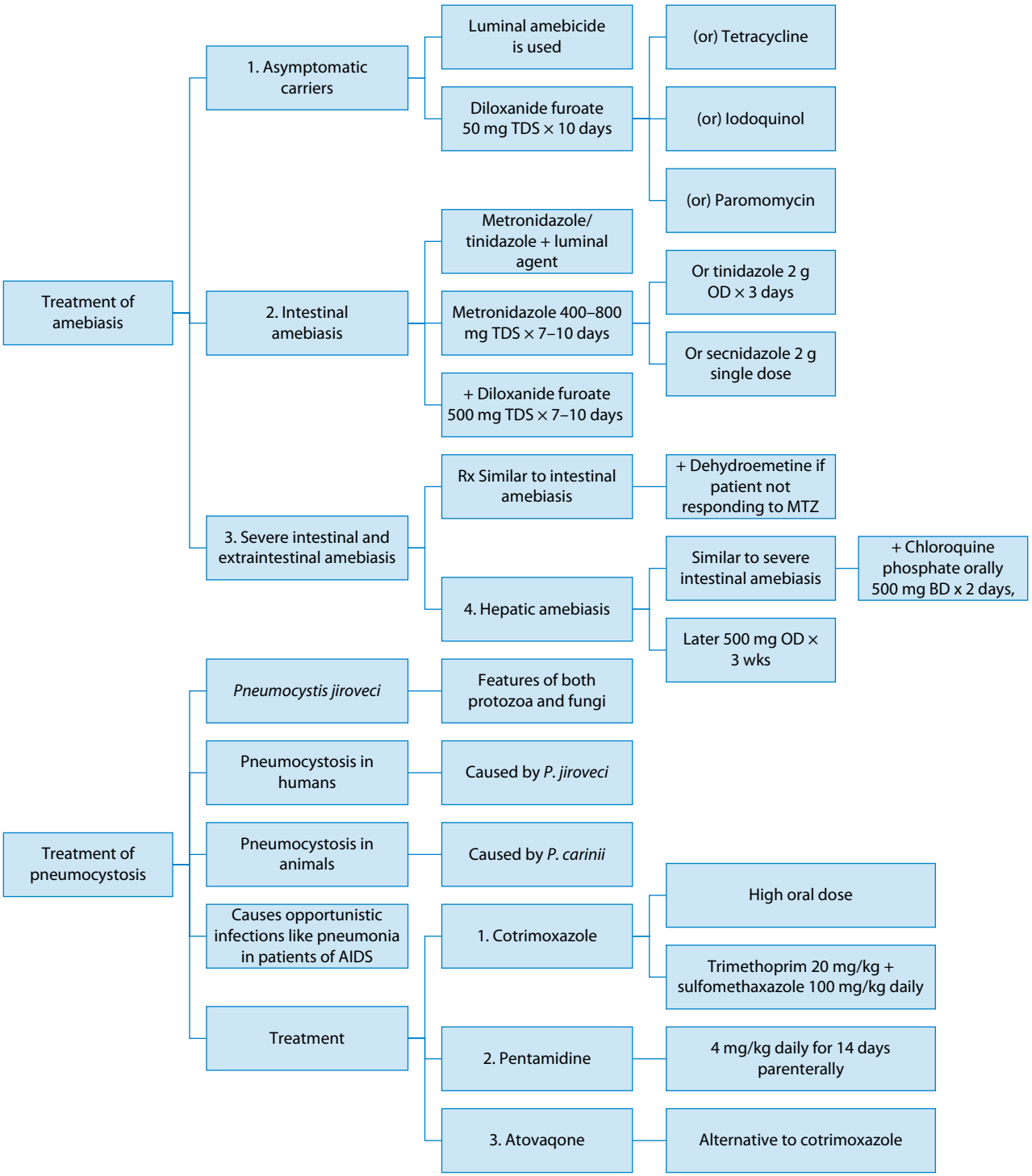
68.4 NITAZOXANIDE, IODOQUINOL, AND QUINIODOCHLOR



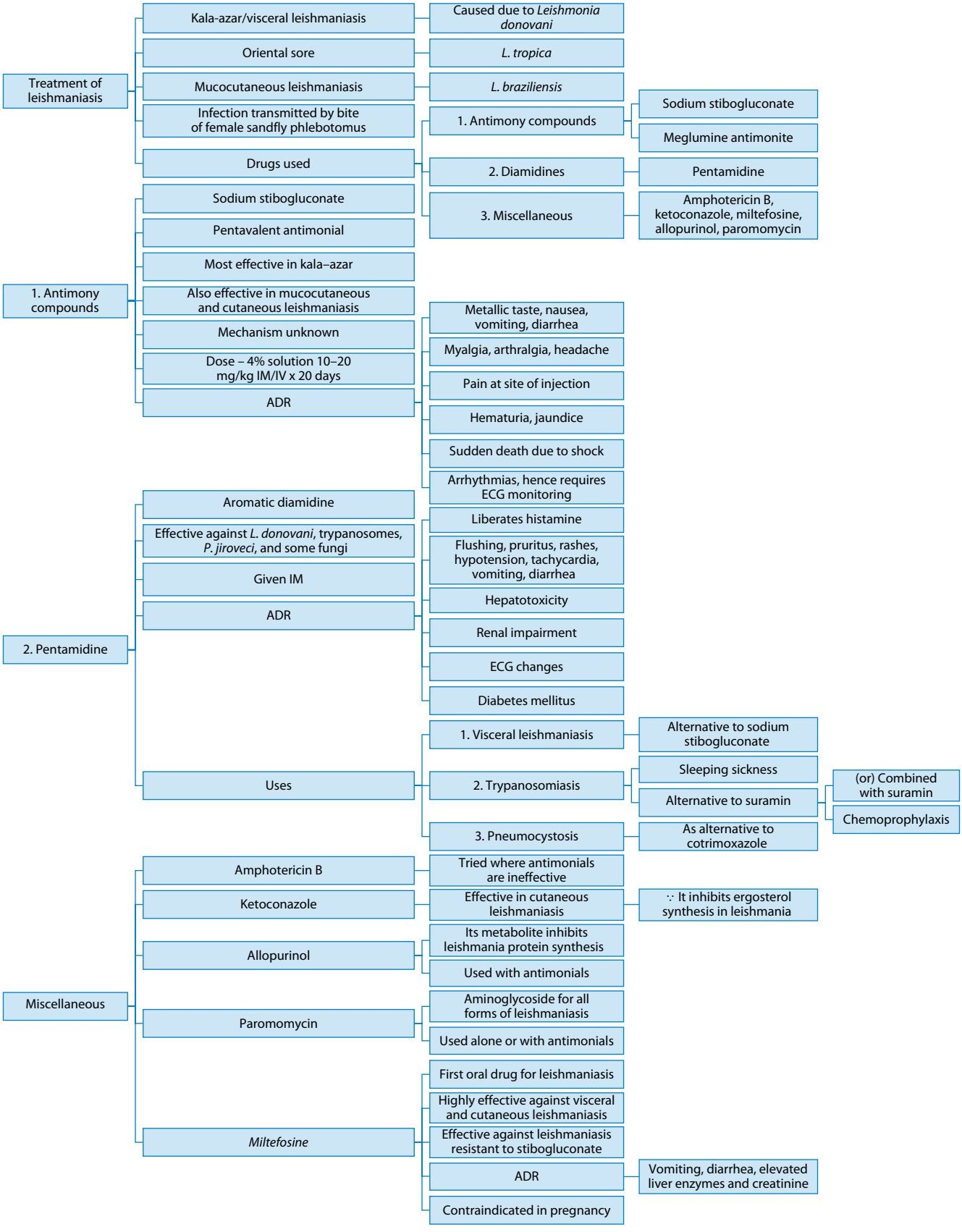
68.5 PAROMOMYCIN, TETRACYCLINE, AND CHLOROQUINE



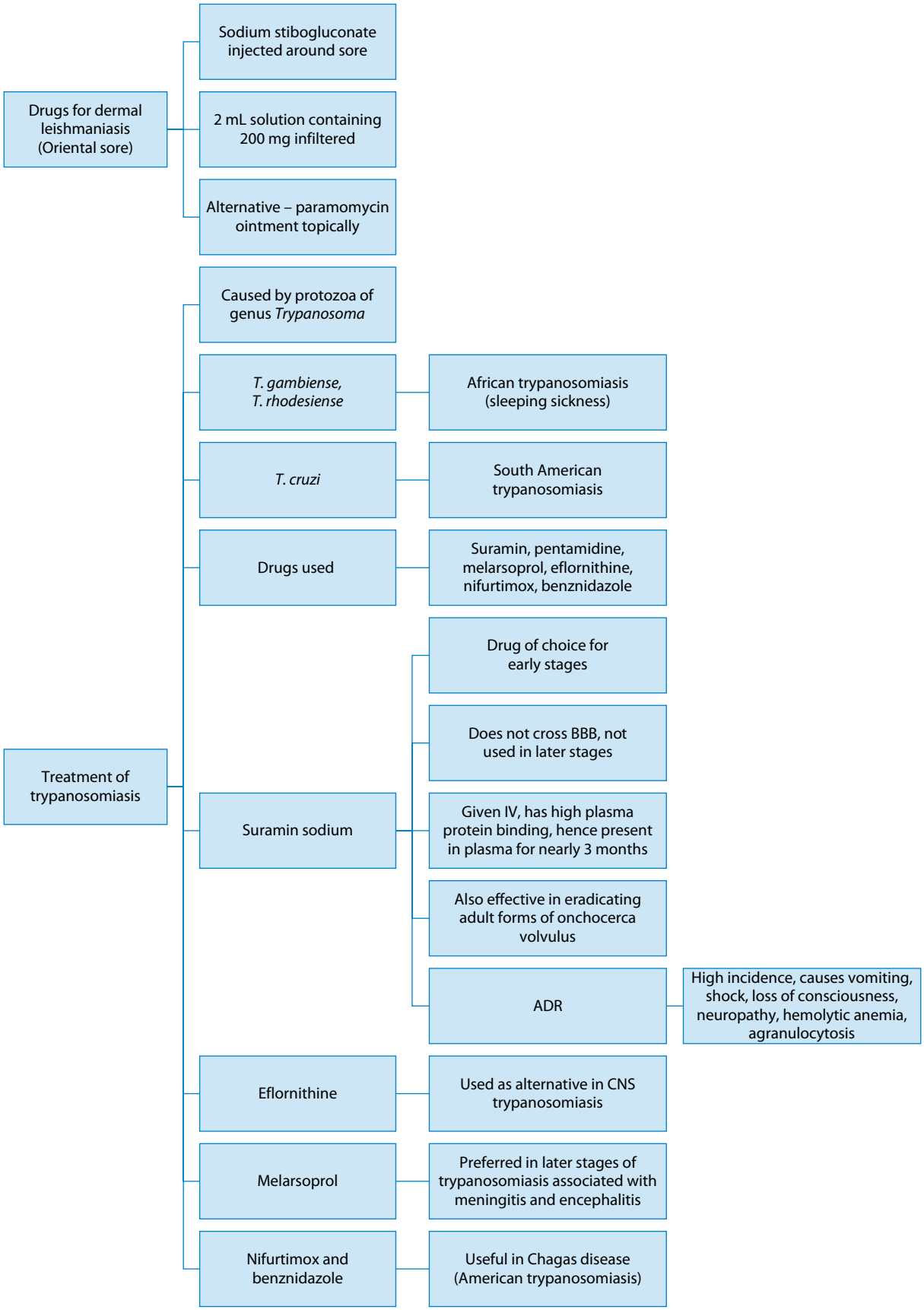
68.6 TREATMENT OF AMEBIASIS, TREATMENT OF PNEUMOCYSTOSIS



68.7 TREATMENT OF LEISHMANIASIS



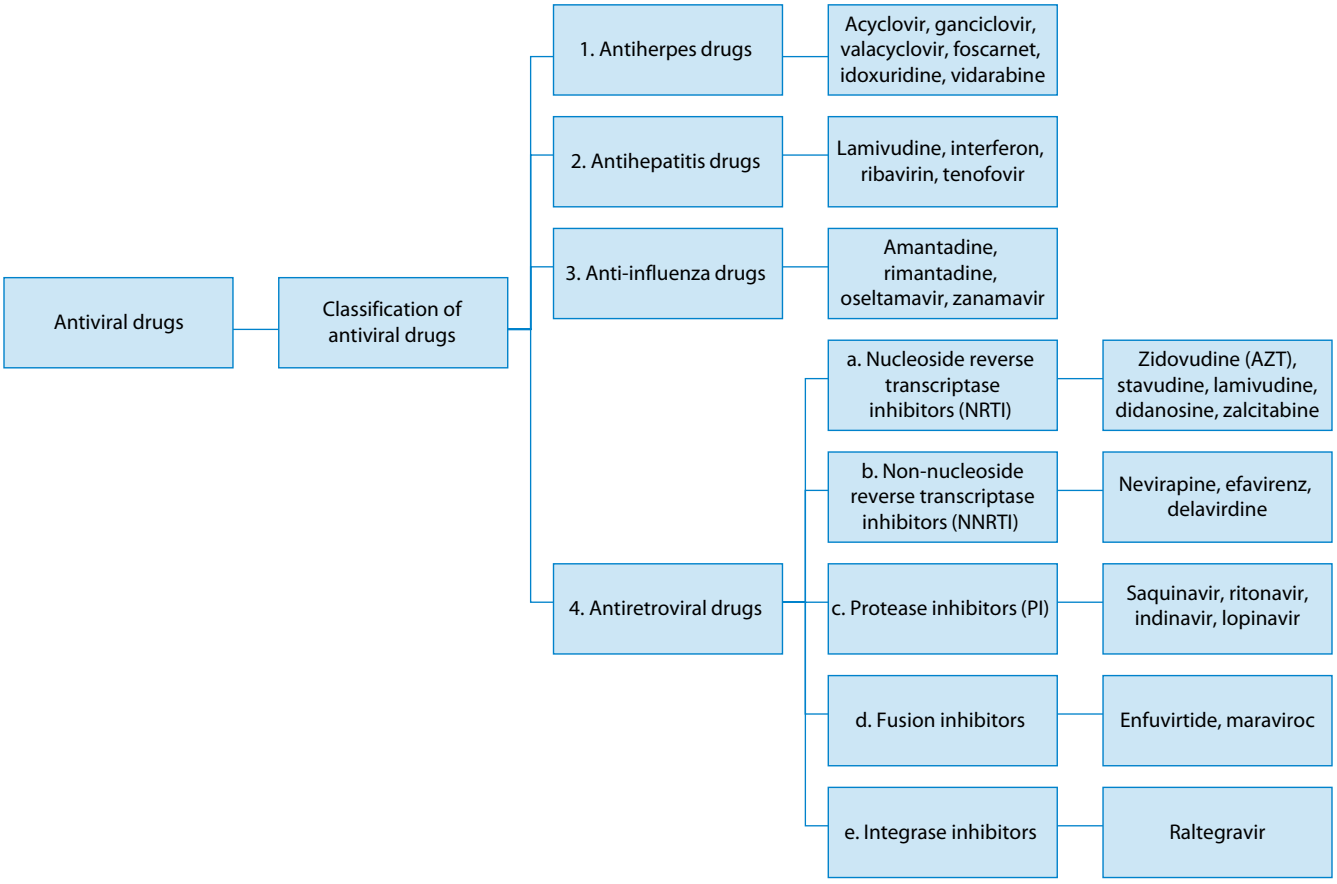
68.8 DRUGS FOR DERMAL LEISHMANIASIS (ORIENTAL SORE), TREATMENT OF TRYPANOSOMIASIS



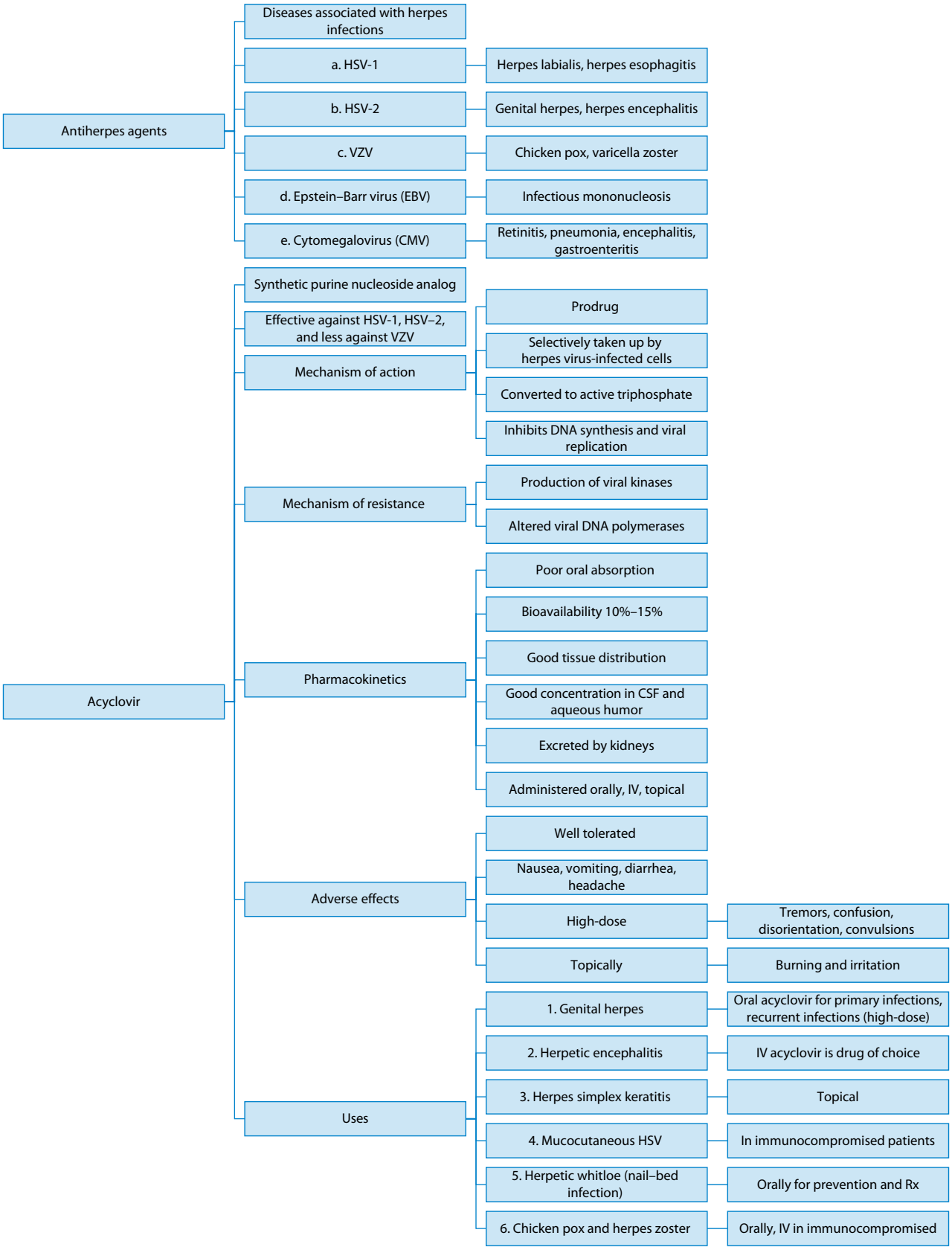
69

Antiviral drugs

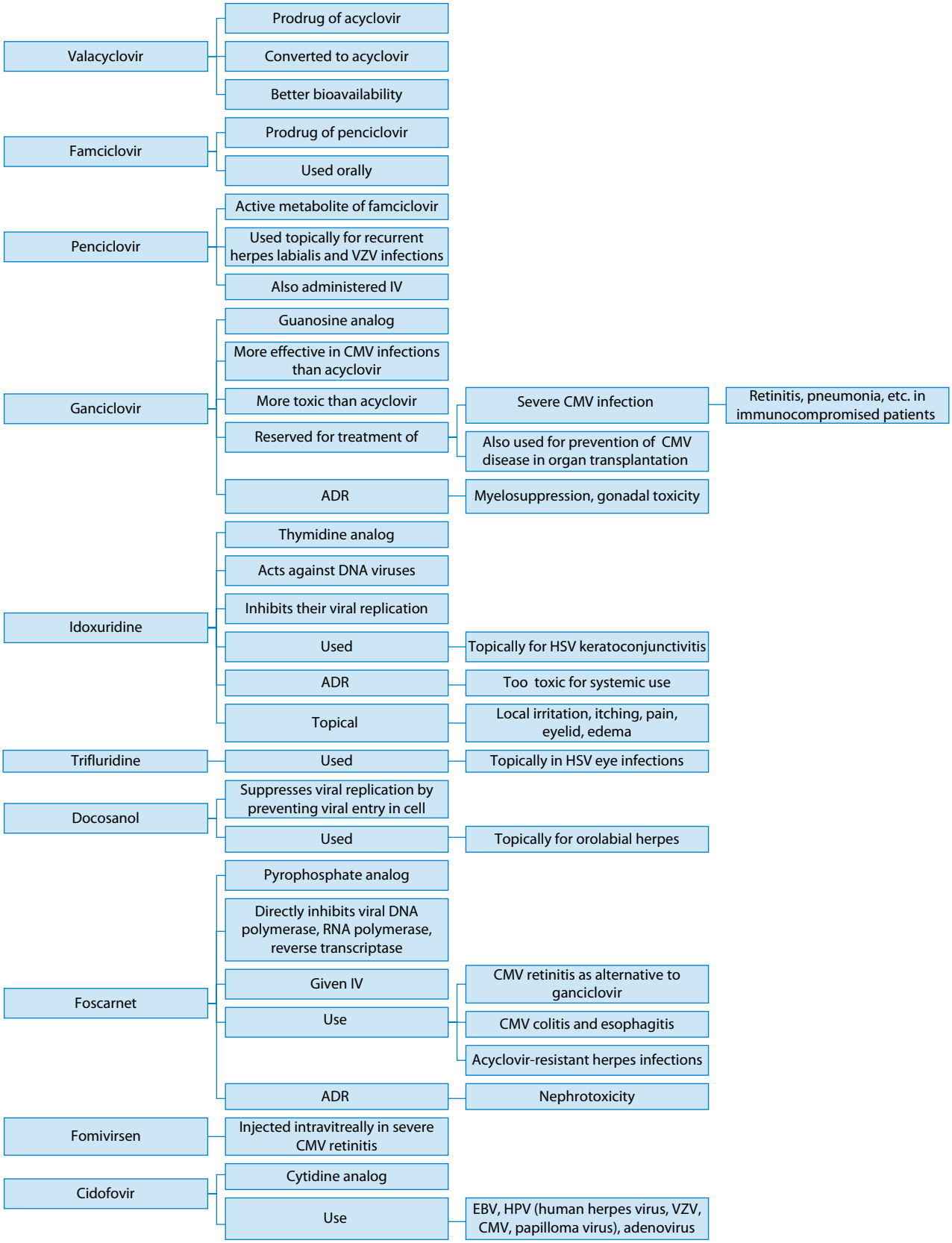
69.1 ANTIVIRAL DRUGS – CLASSIFICATION



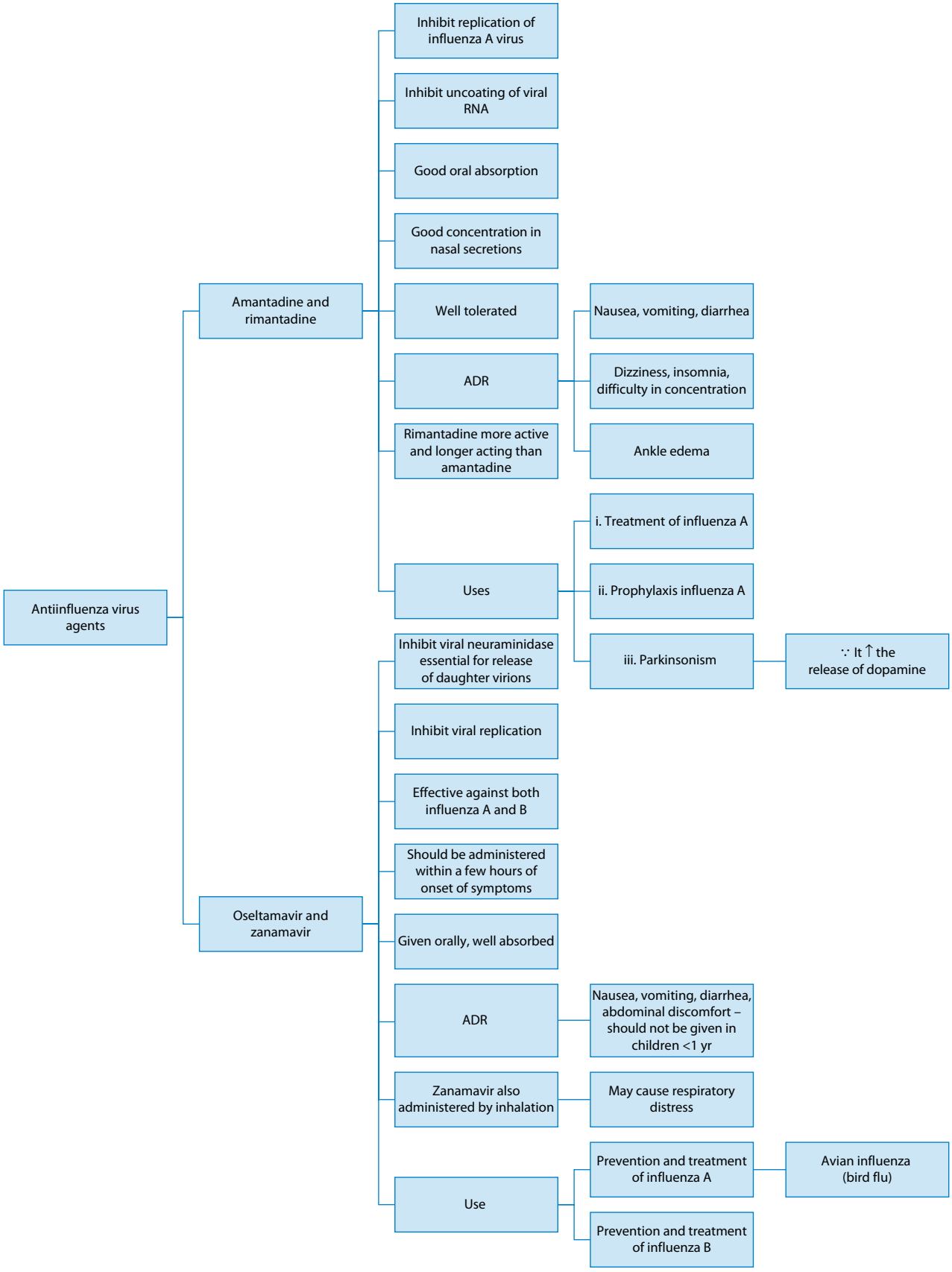
69.2 ANTIHERPES AGENT – ACYCLOVIR



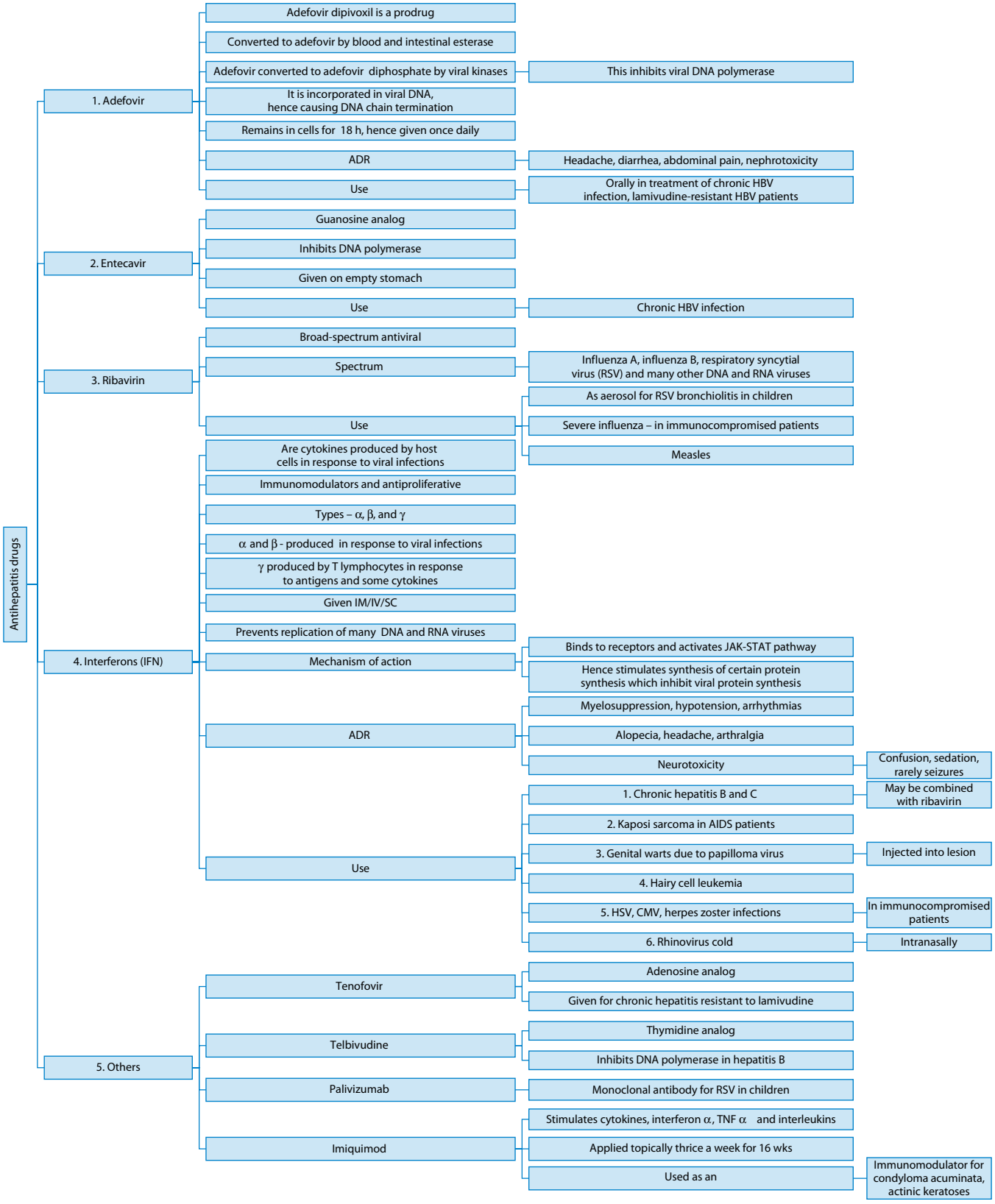
69.3 OTHER ANTIHERPES DRUGS



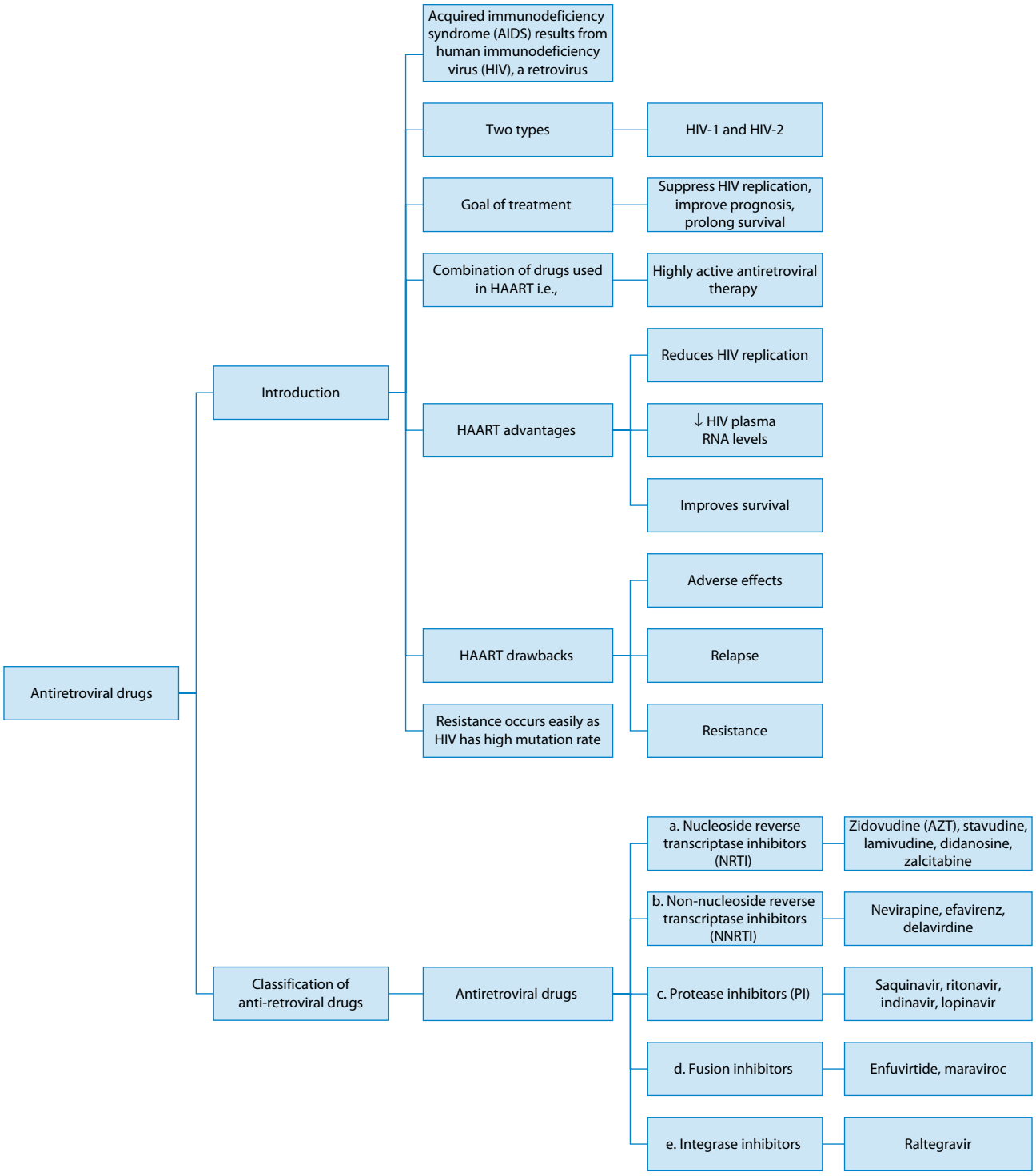
69.4 ANTIINFLUENZA VIRUS AGENTS



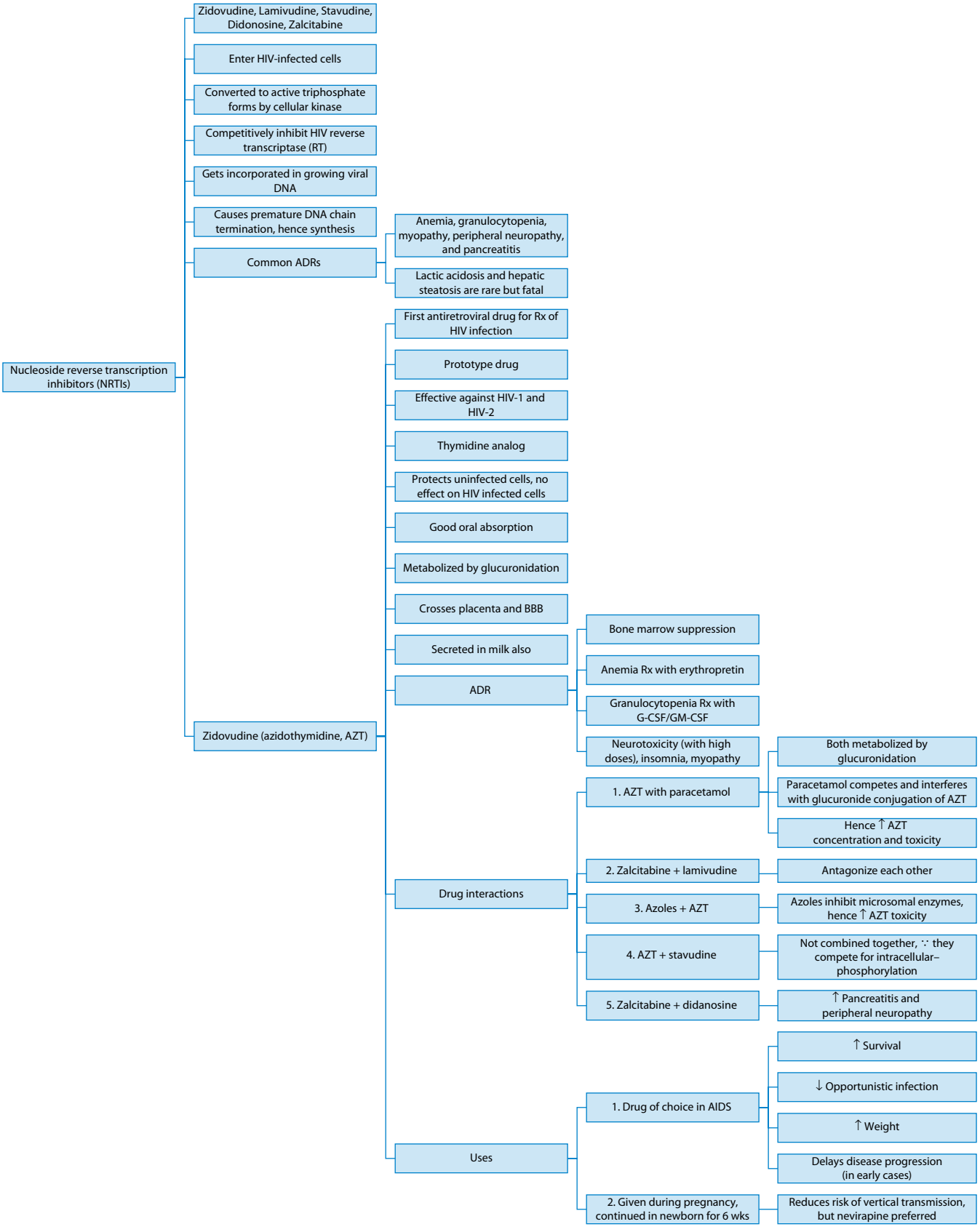
69.5 ANTIHEPATITIS DRUGS



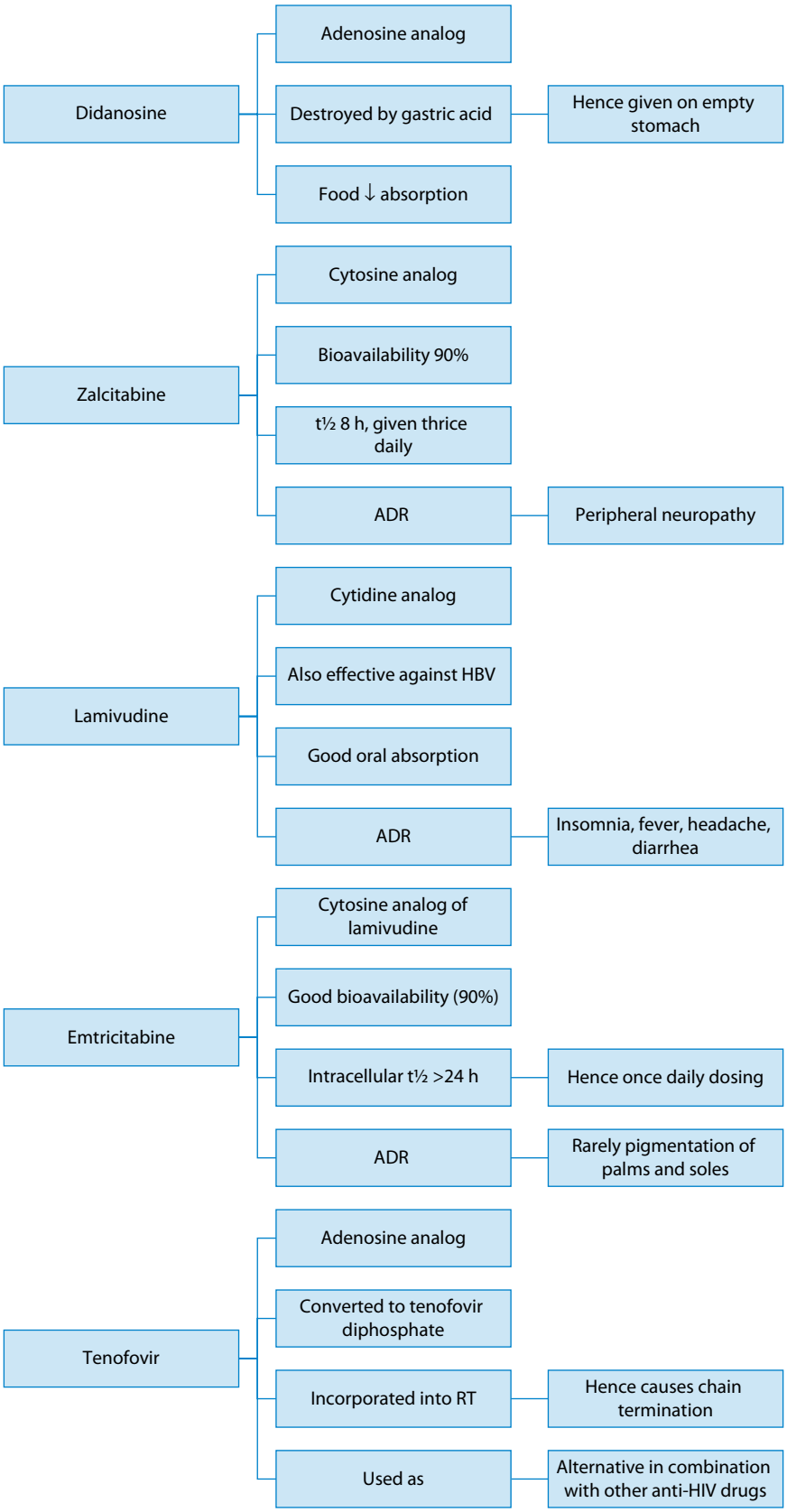
69.6 ANTIRETROVIRAL DRUGS – INTRODUCTION AND CLASSIFICATION



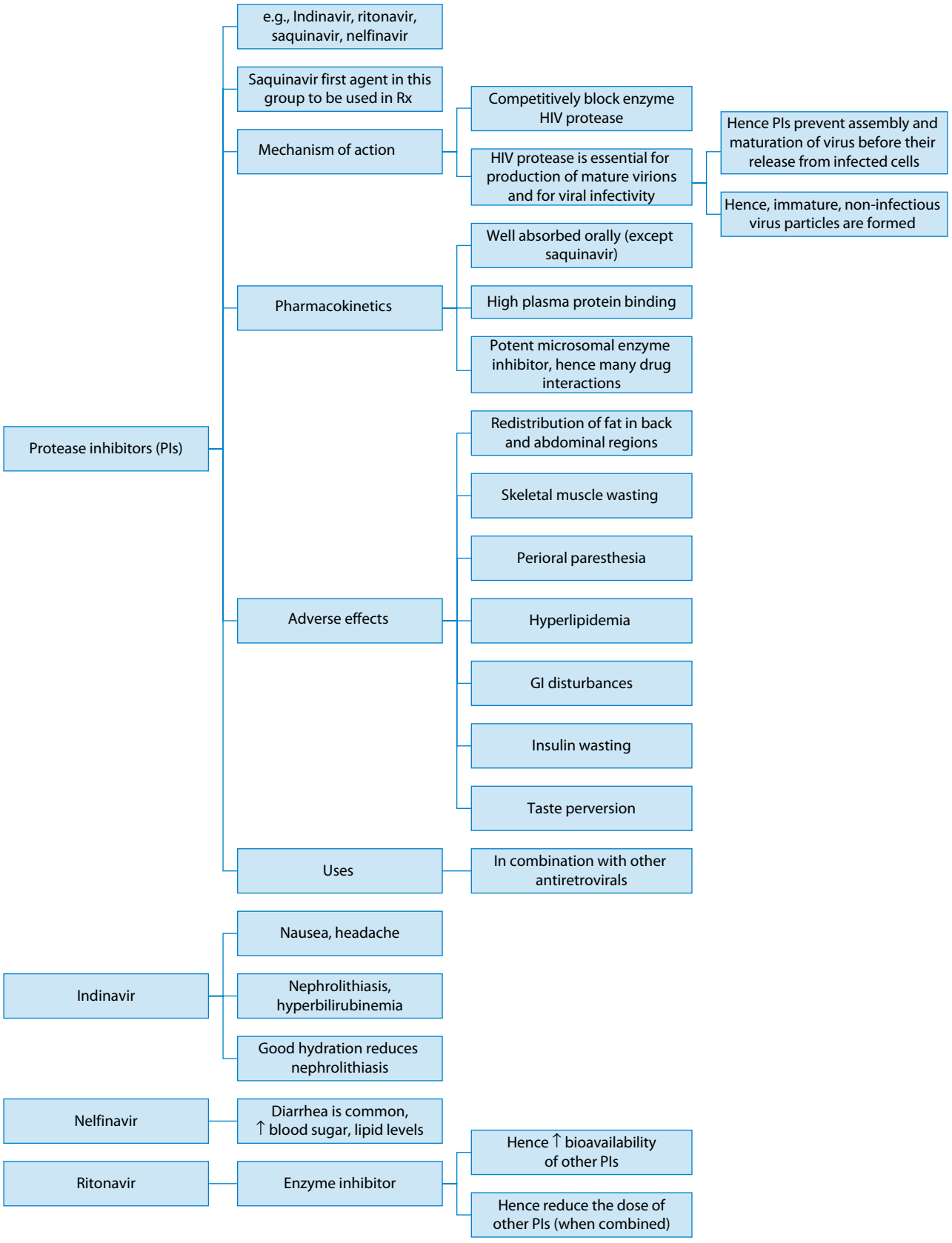
69.7 NUCLEOSIDE REVERSE TRANSCRIPTION INHIBITORS (NRTIs)



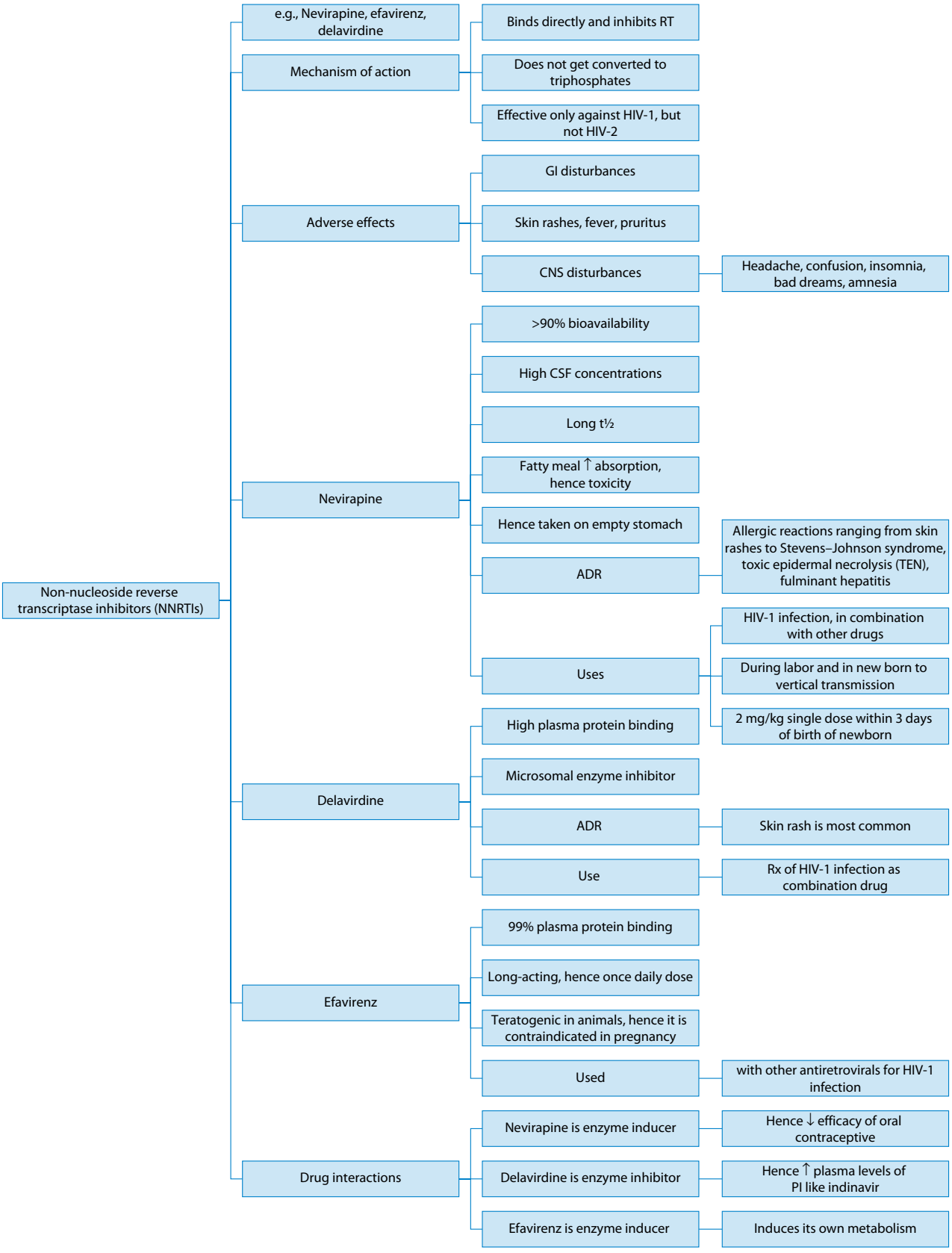
69.8 OTHER NRTIs



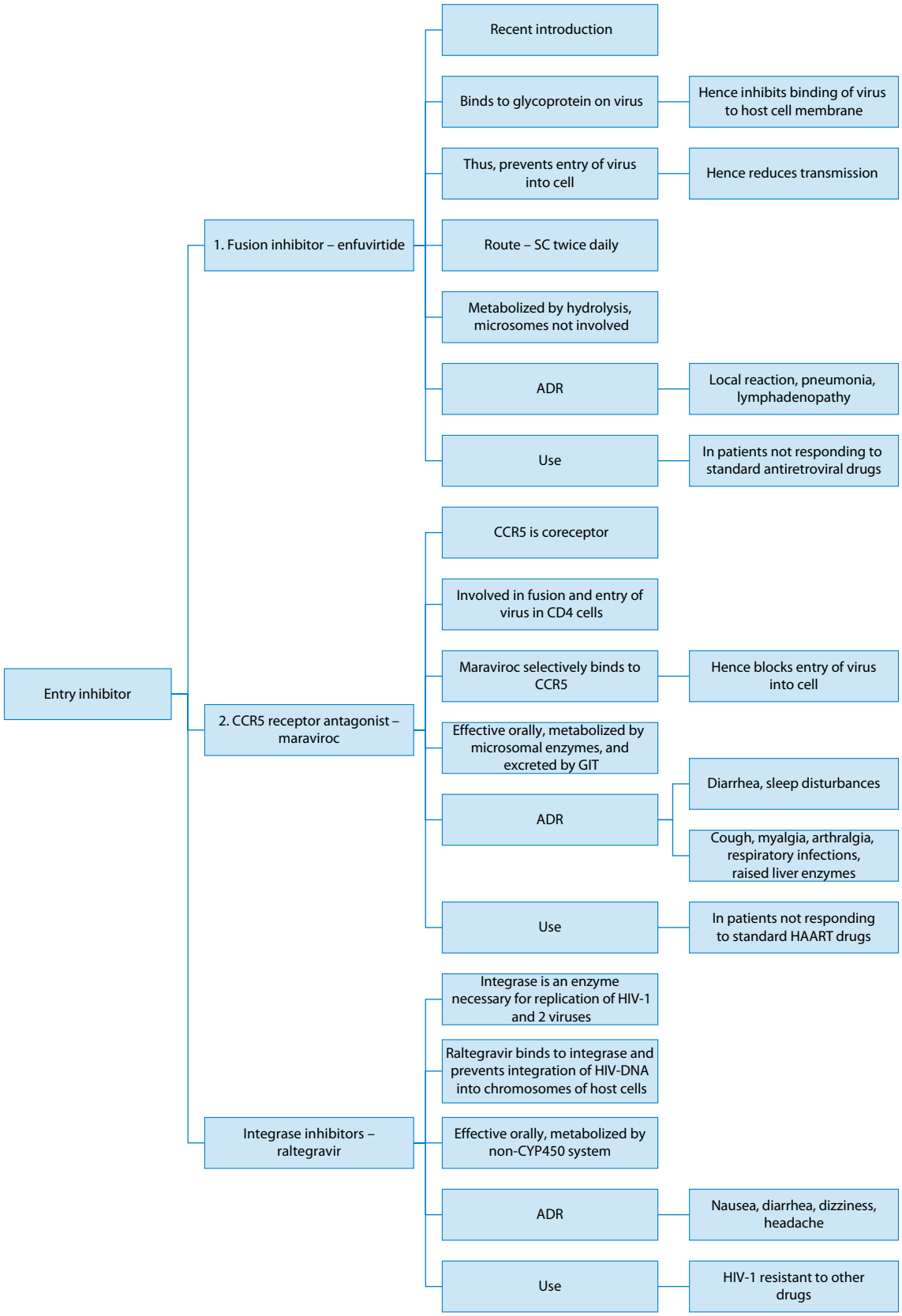
69.9 PROTEASE INHIBITORS (PIs)



69.10 NON-NUCLEOSIDE REVERSE TRANSCRIPTASE INHIBITORS (NNRTIs)



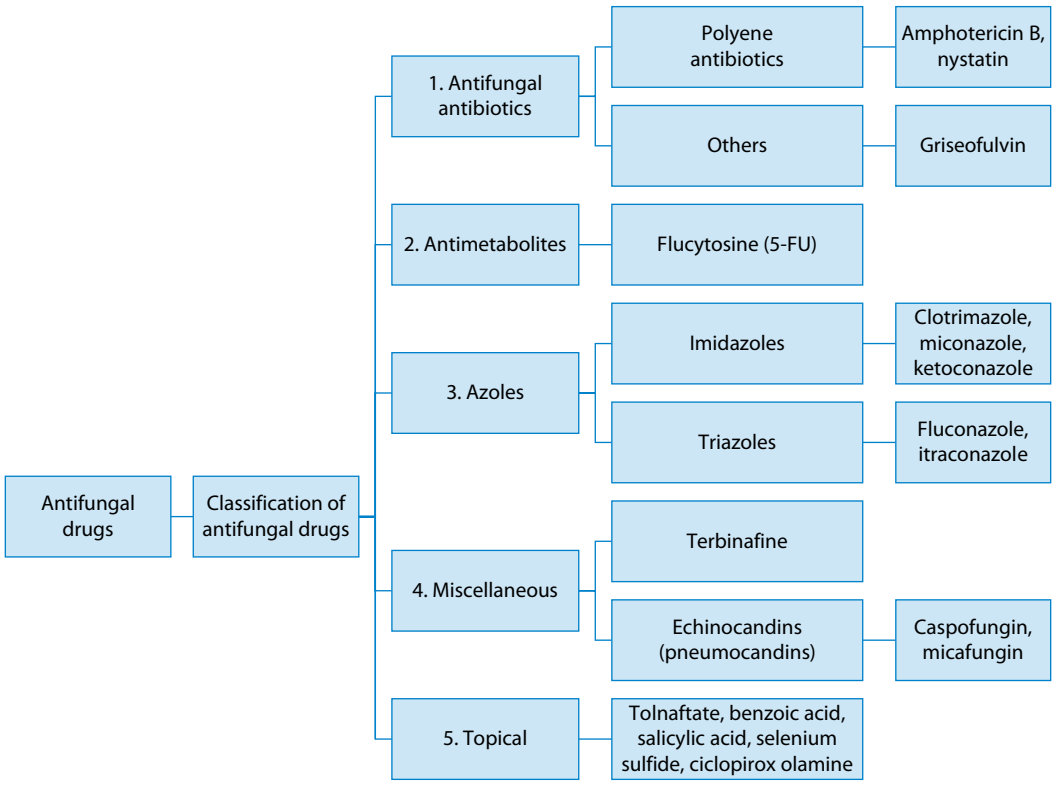
69.11 ENTRY INHIBITOR



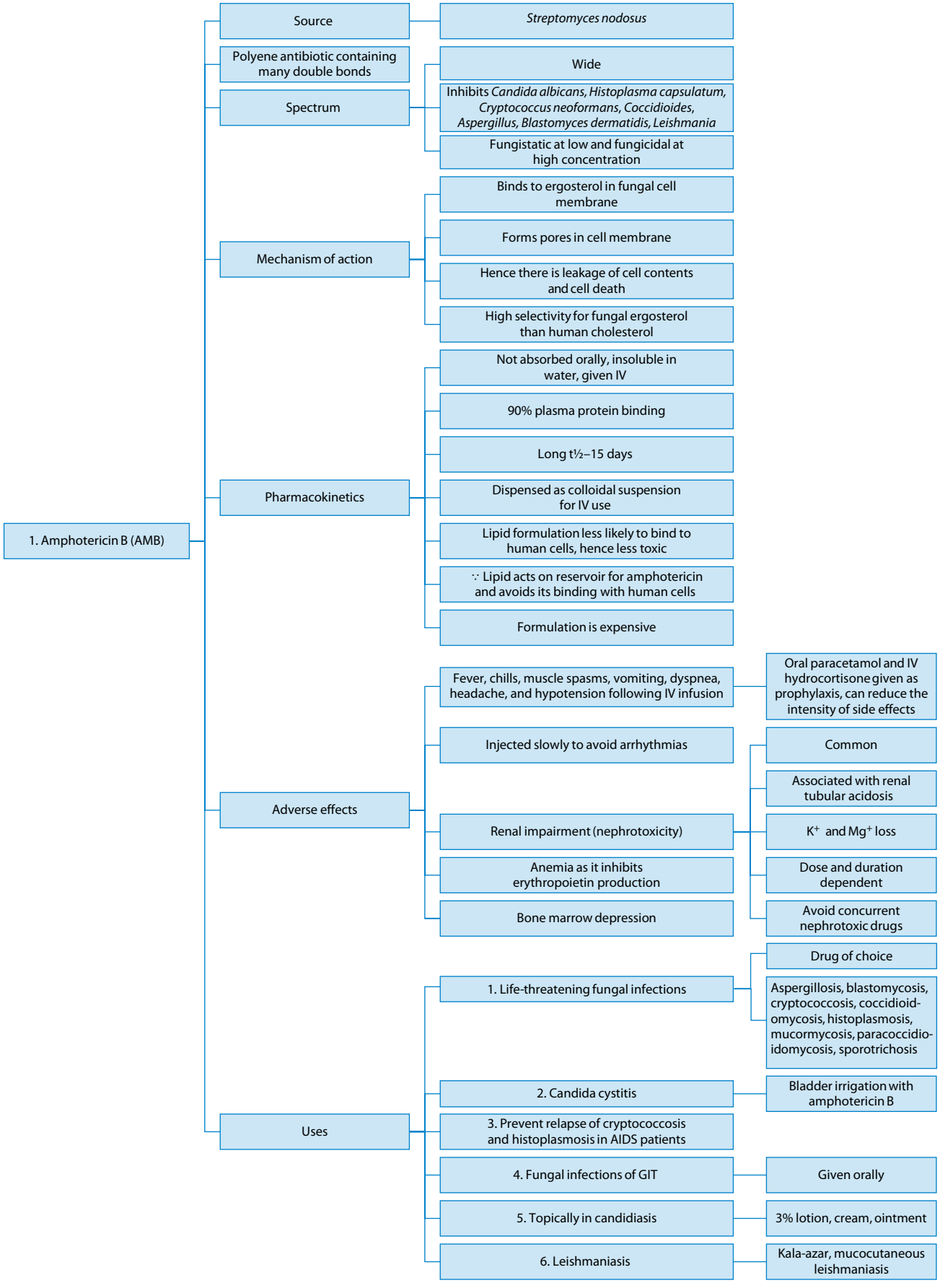
70

Antifungal drugs

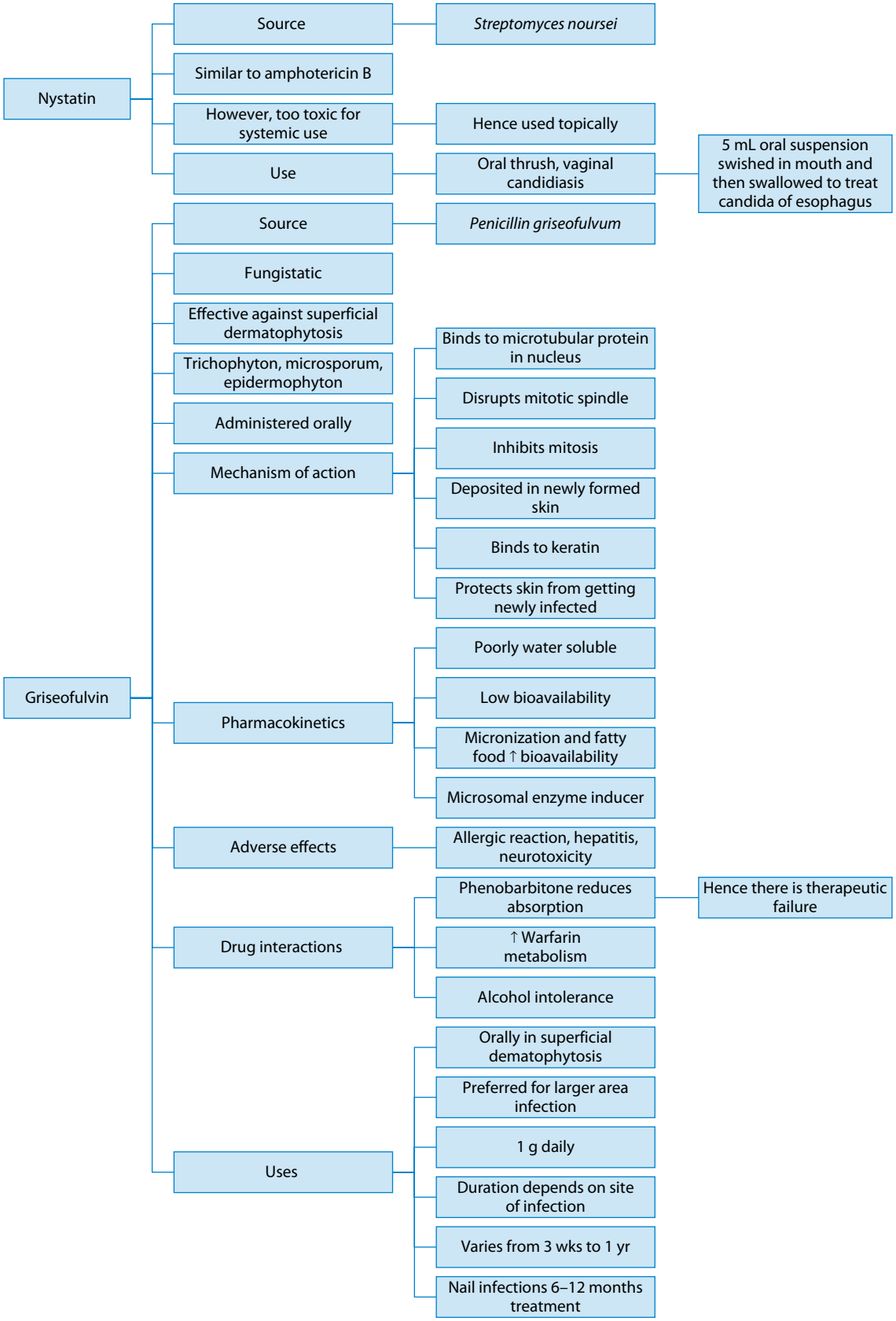
70.1 CLASSIFICATION OF ANTIFUNGAL DRUGS



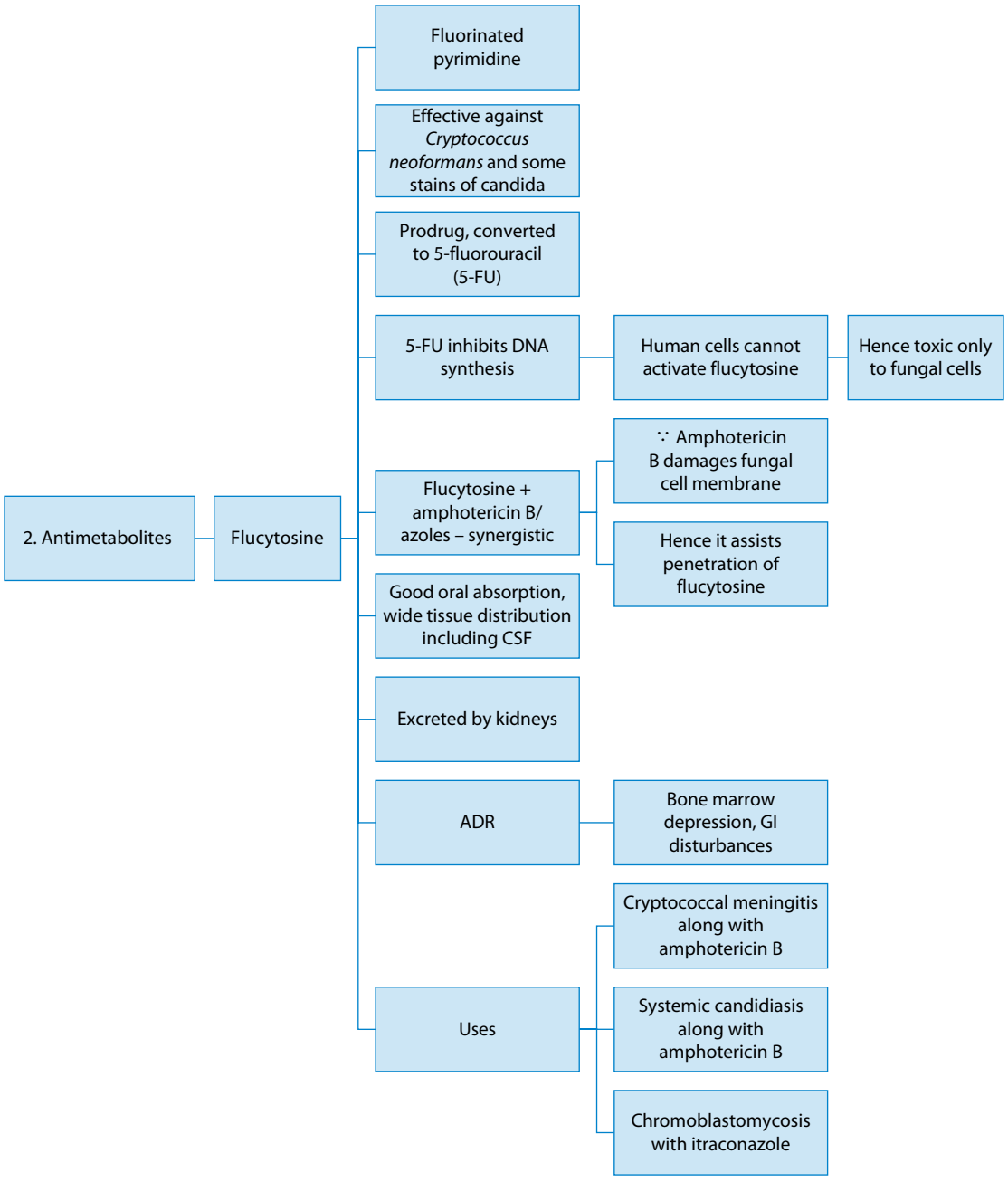
70.2 ANTIFUNGAL ANTIBIOTICS – AMPHOTERICIN B (AMB)



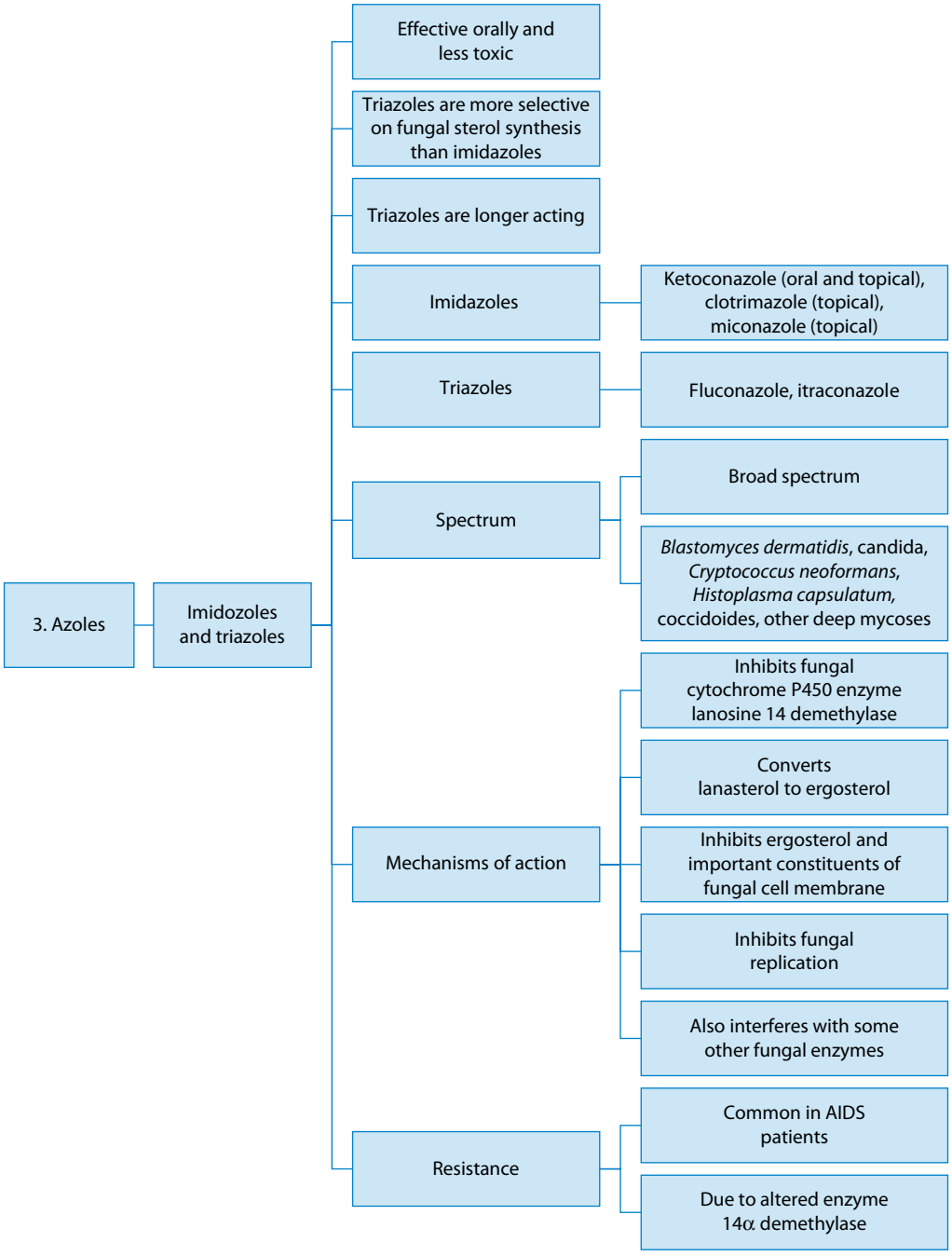
70.3 NYSTATIN, GRISEOFULVIN



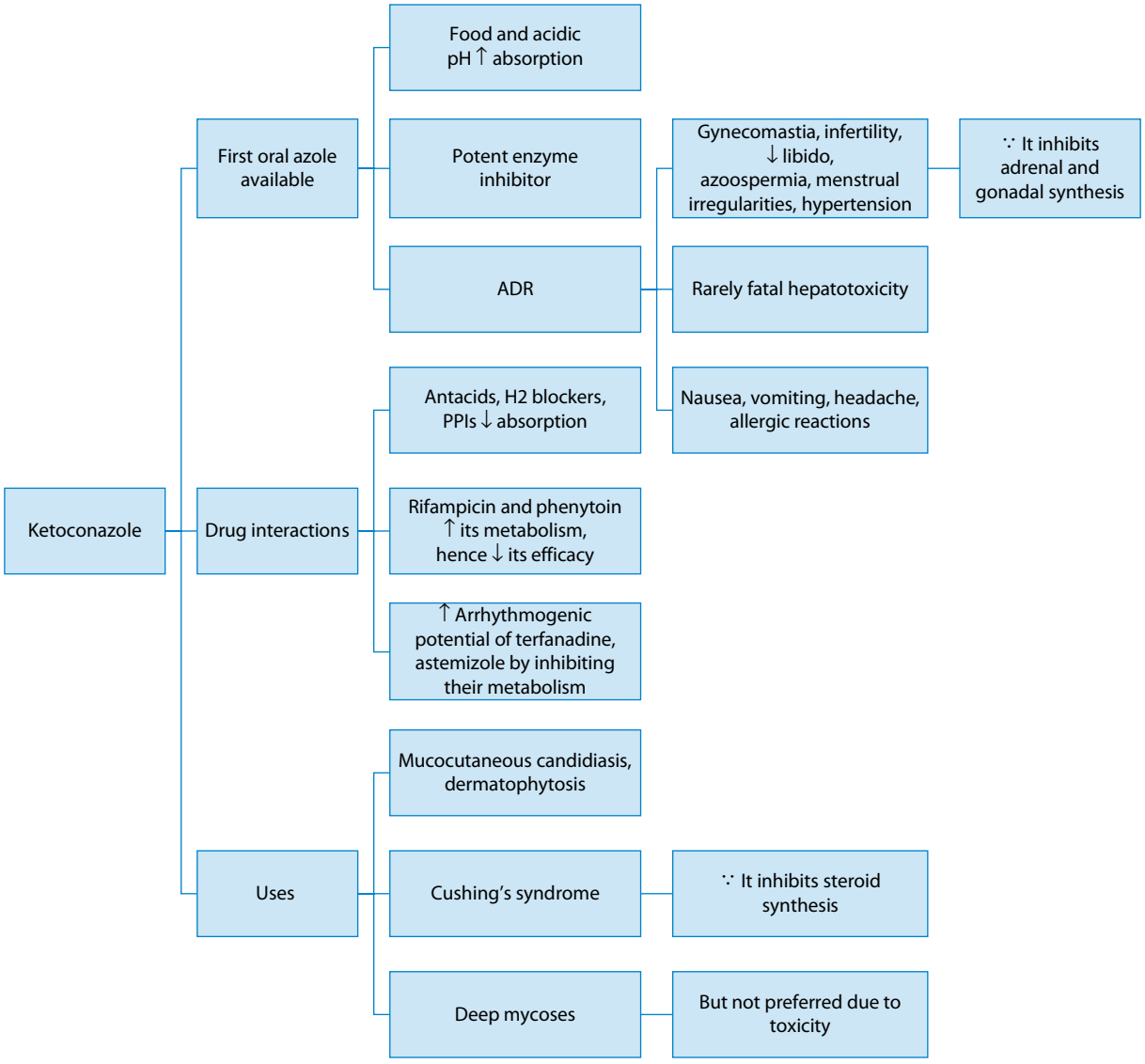
70.4 ANTIMETABOLITES



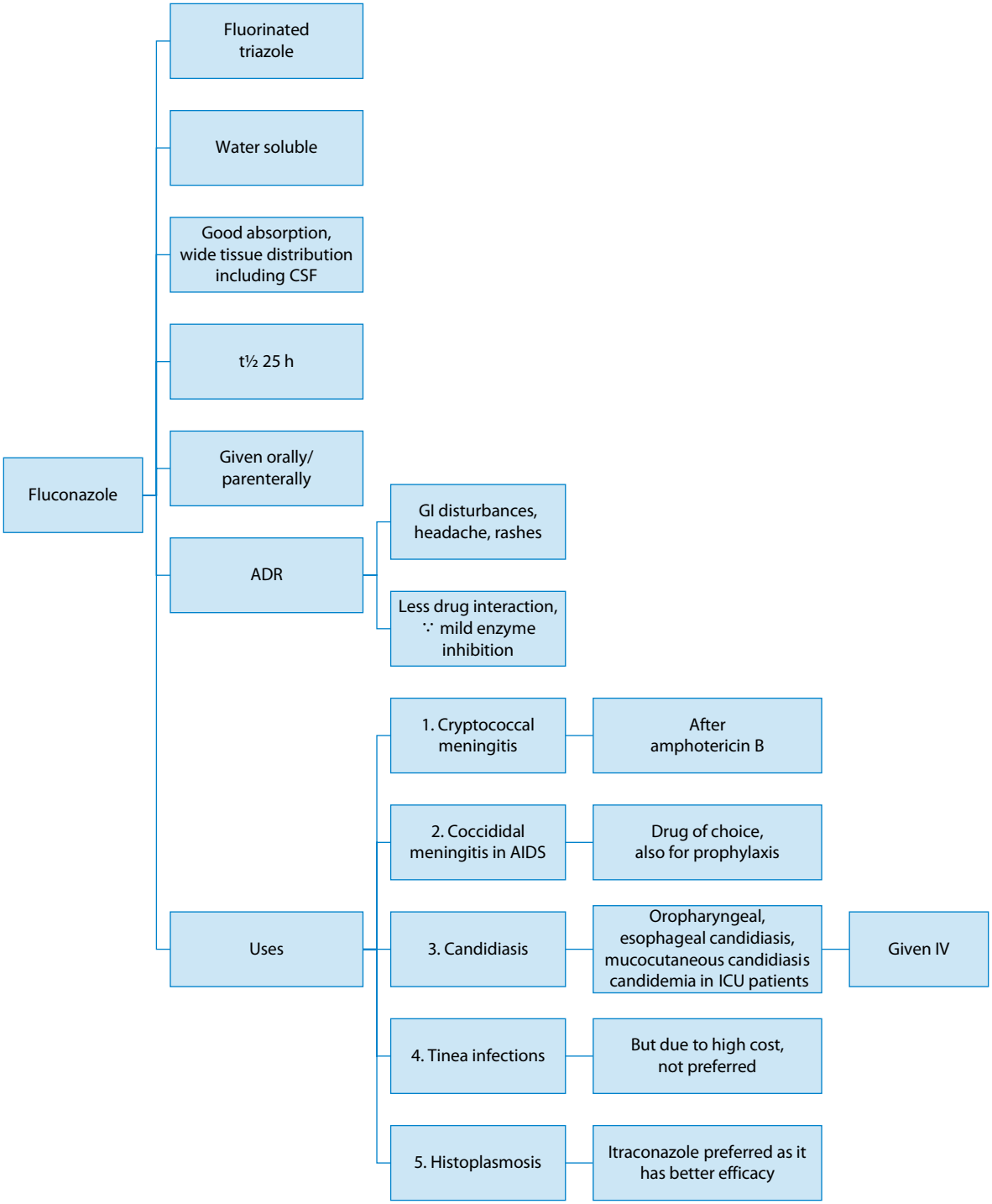
70.5 AZOLES


3. Azoles
Imidozoles and triazoles
Effective orally and less toxic
Triazoles are more selective on fungal sterol synthesis than imidazoles
Triazoles are longer acting
Imidazoles
Ketoconazole (oral and topical), clotrimazole (topical), miconazole (topical)
Triazoles
Fluconazole, itraconazole
Spectrum
Broad spectrum
Blastomyces dermatidis, candida, *Cryptococcus neoformans*, *Histoplasma capsulatum*, coccidoides, other deep mycoses
Mechanisms of action
Inhibits fungal cytochrome P450 enzyme lanosine 14 demethylase
Converts lanasterol to ergosterol
Inhibits ergosterol and important constituents of fungal cell membrane
Inhibits fungal replication
Also interferes with some other fungal enzymes
Resistance
Common in AIDS patients
Due to altered enzyme 14α demethylase

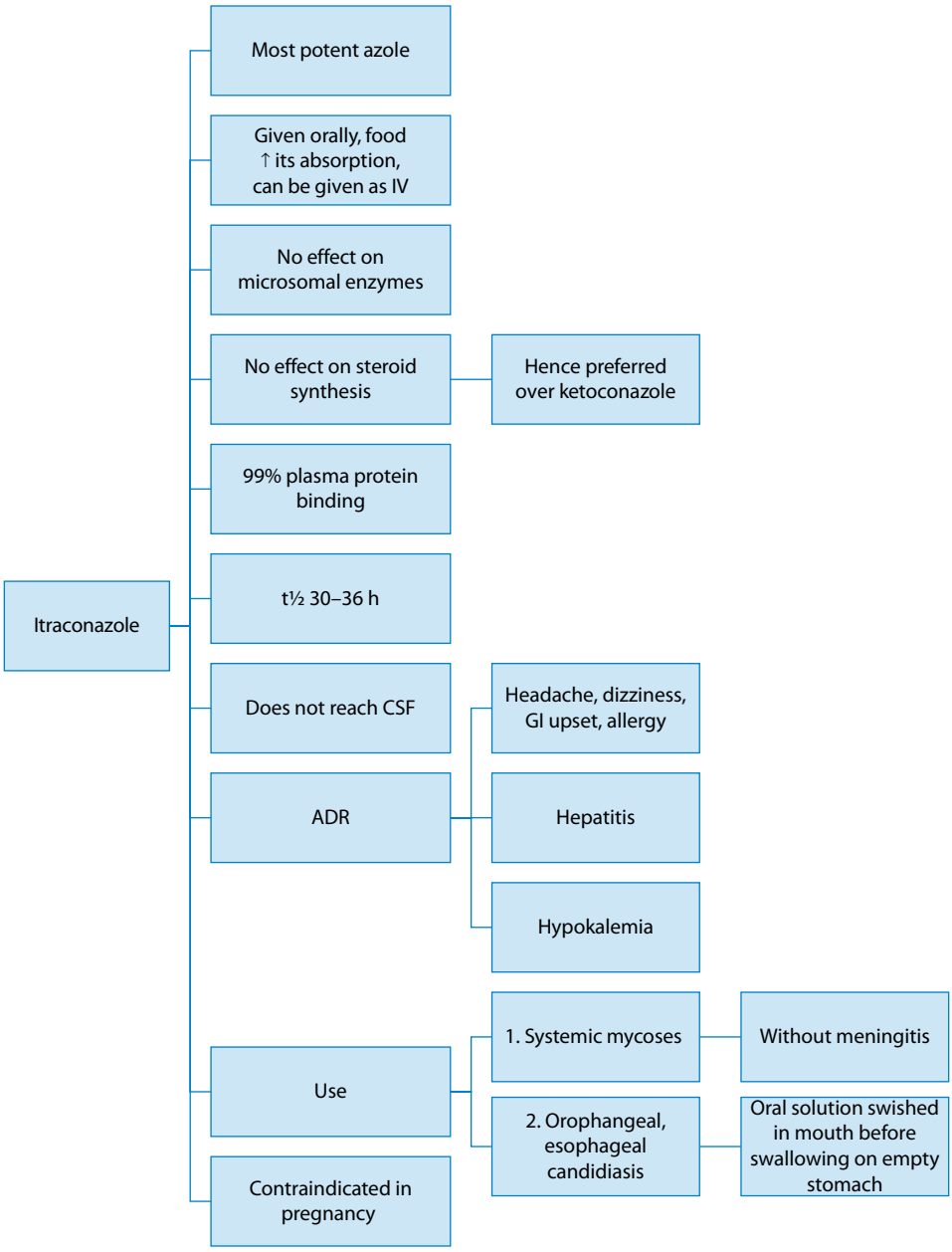
70.6 KETOCONAZOLE



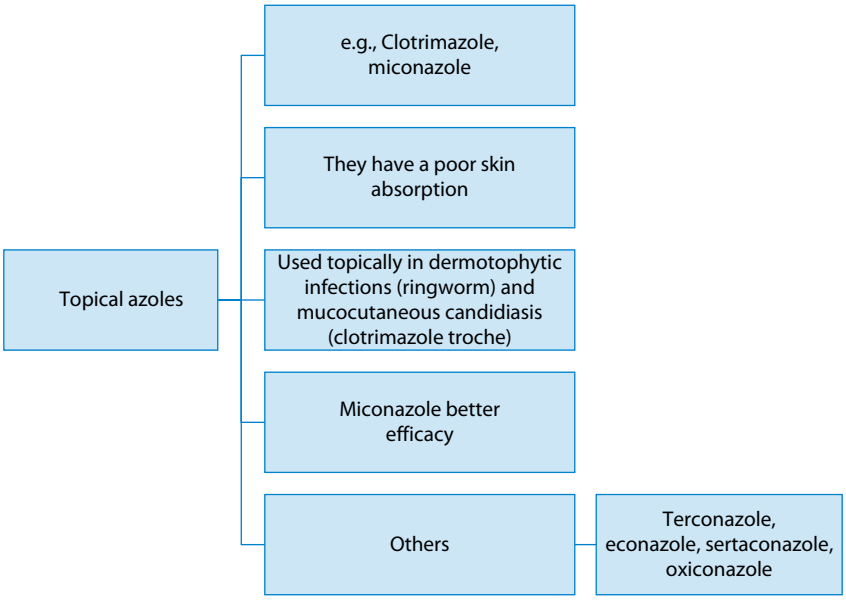
70.7 FLUCONAZOLE



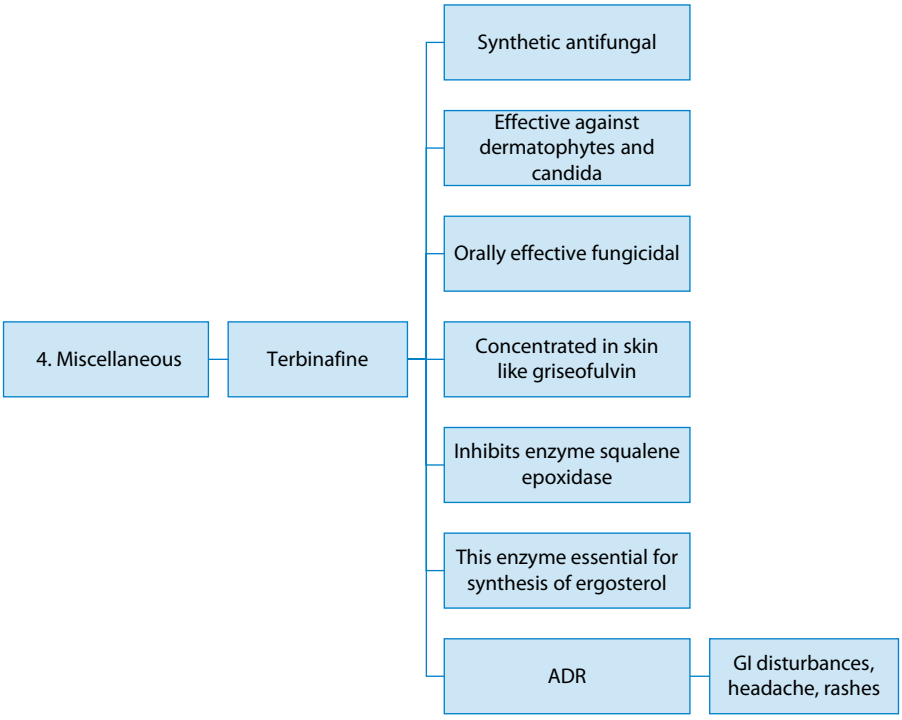
70.8 ITRACONAZOLE



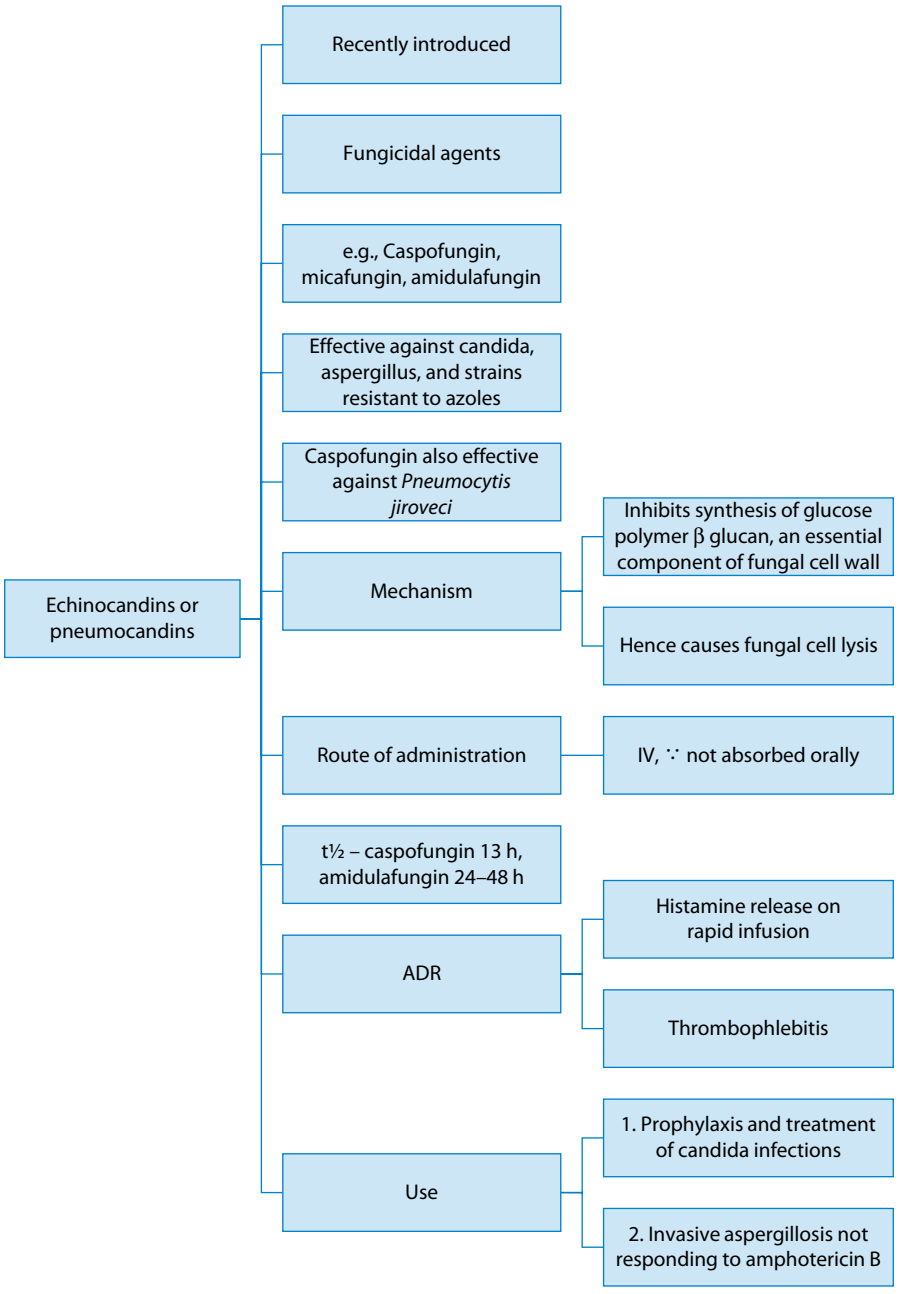
70.9 TOPICAL AZOLES



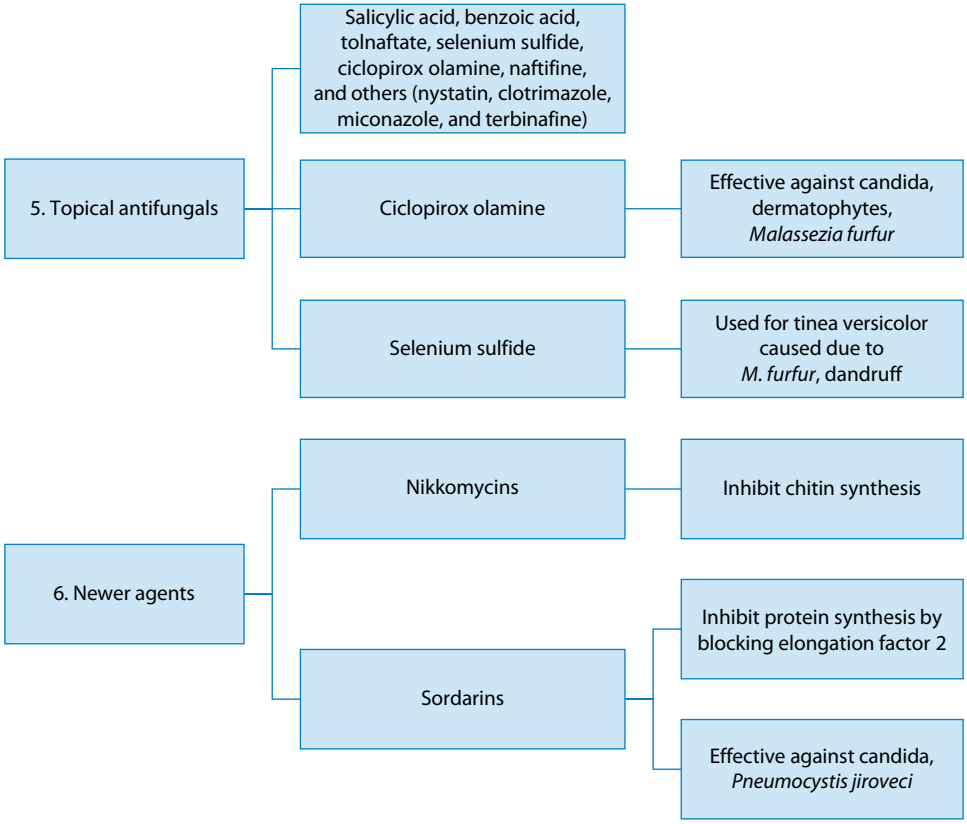
70.10 MISCELLANEOUS – TERBINAFINE



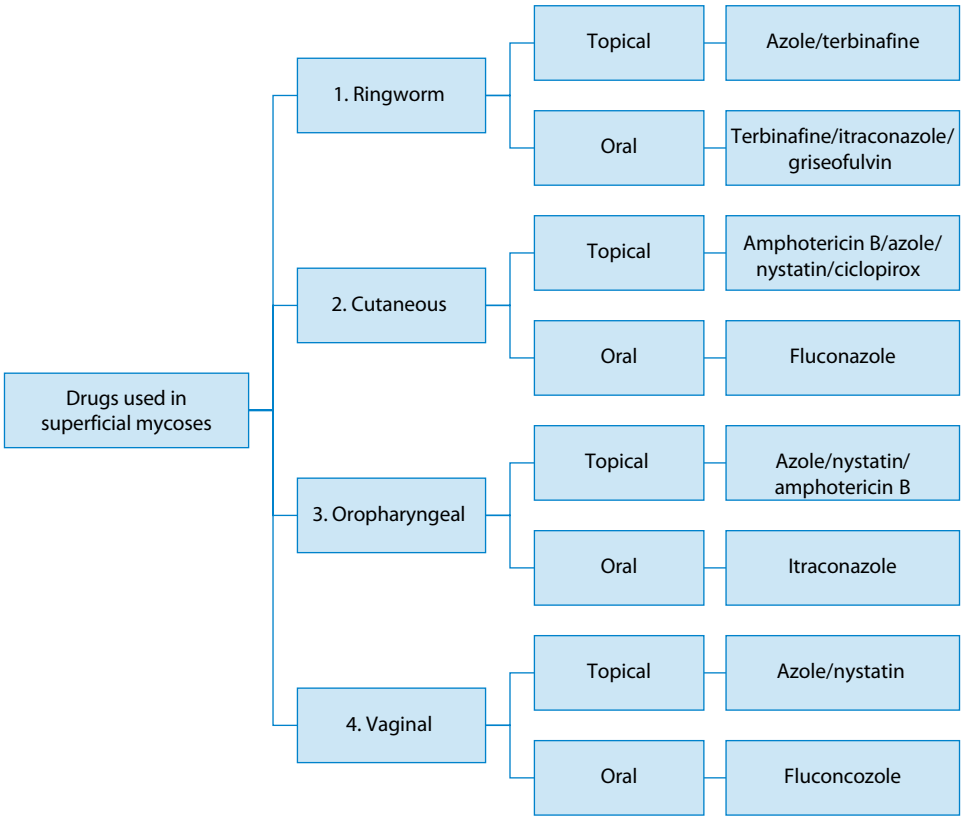
70.11 ECHINOCANDINS OR PNEUMOCANDINS



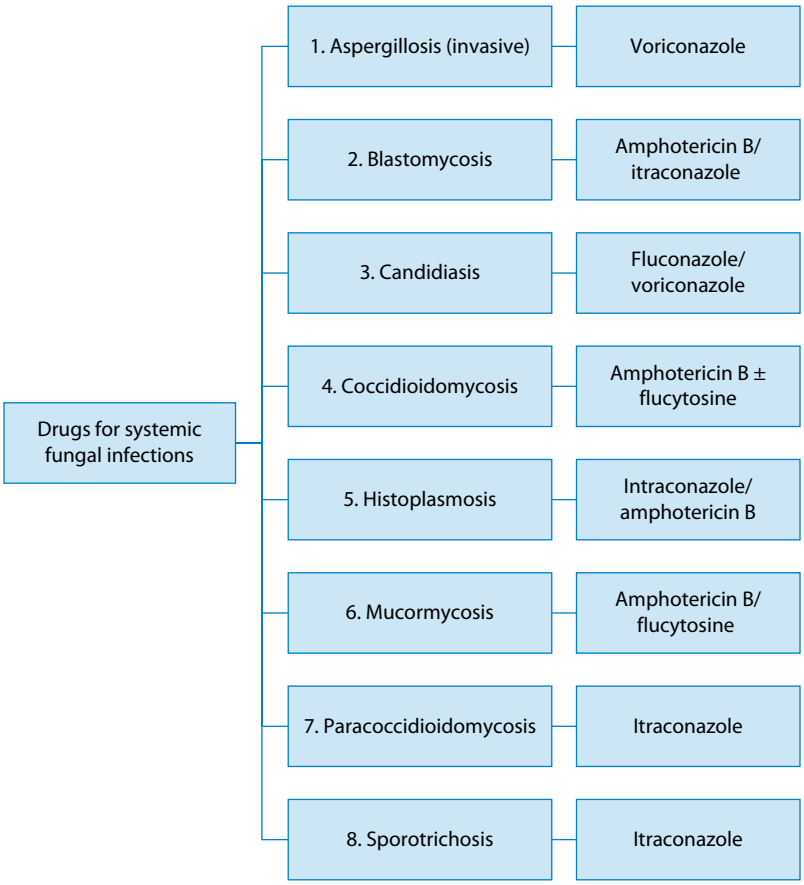
70.12 TOPICAL ANTIFUNGALS AND NEWER AGENTS



70.13 DRUGS USED IN SUPERFICIAL MYCOSES



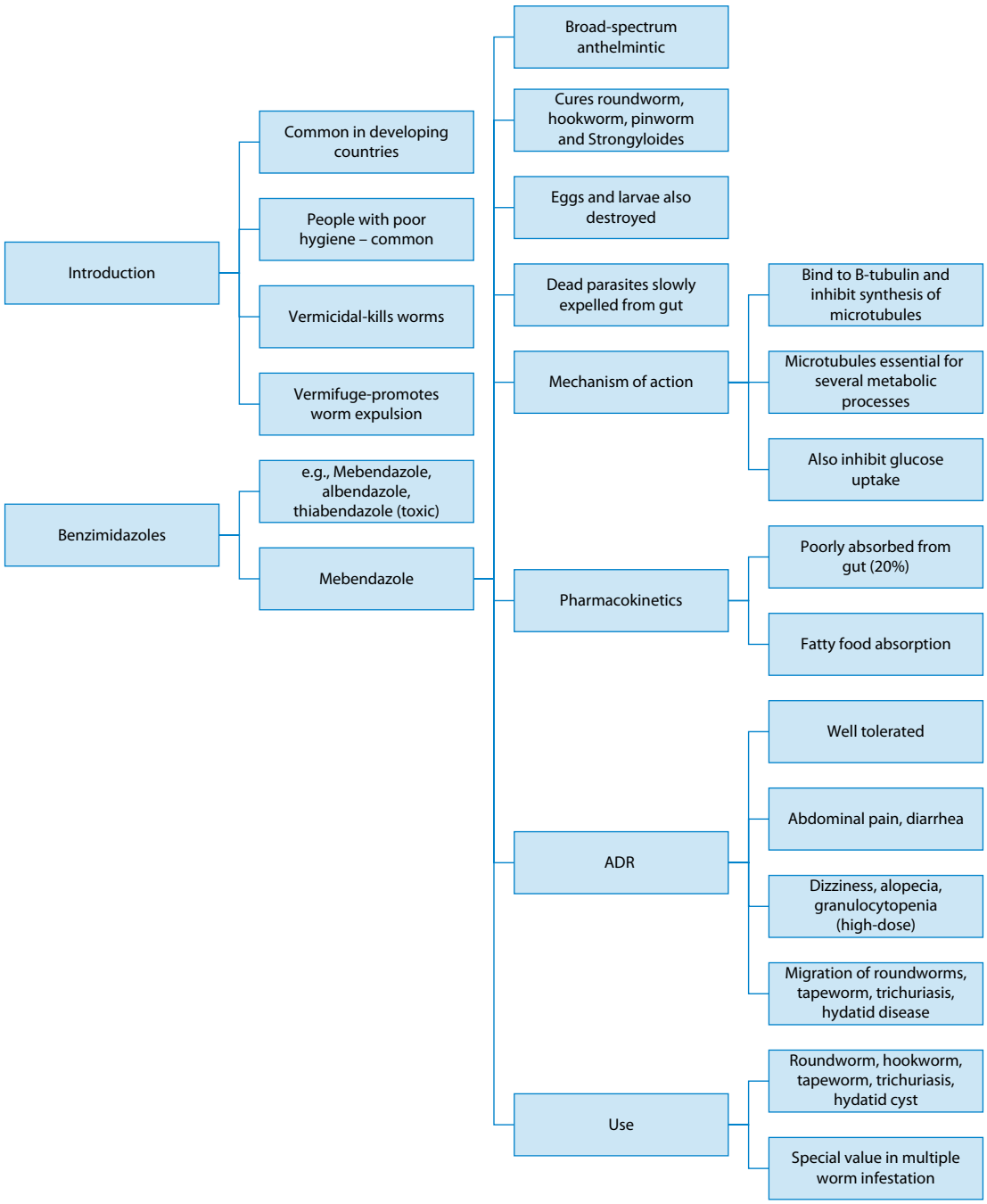
70.14 DRUGS FOR SYSTEMIC FUNGAL INFECTIONS



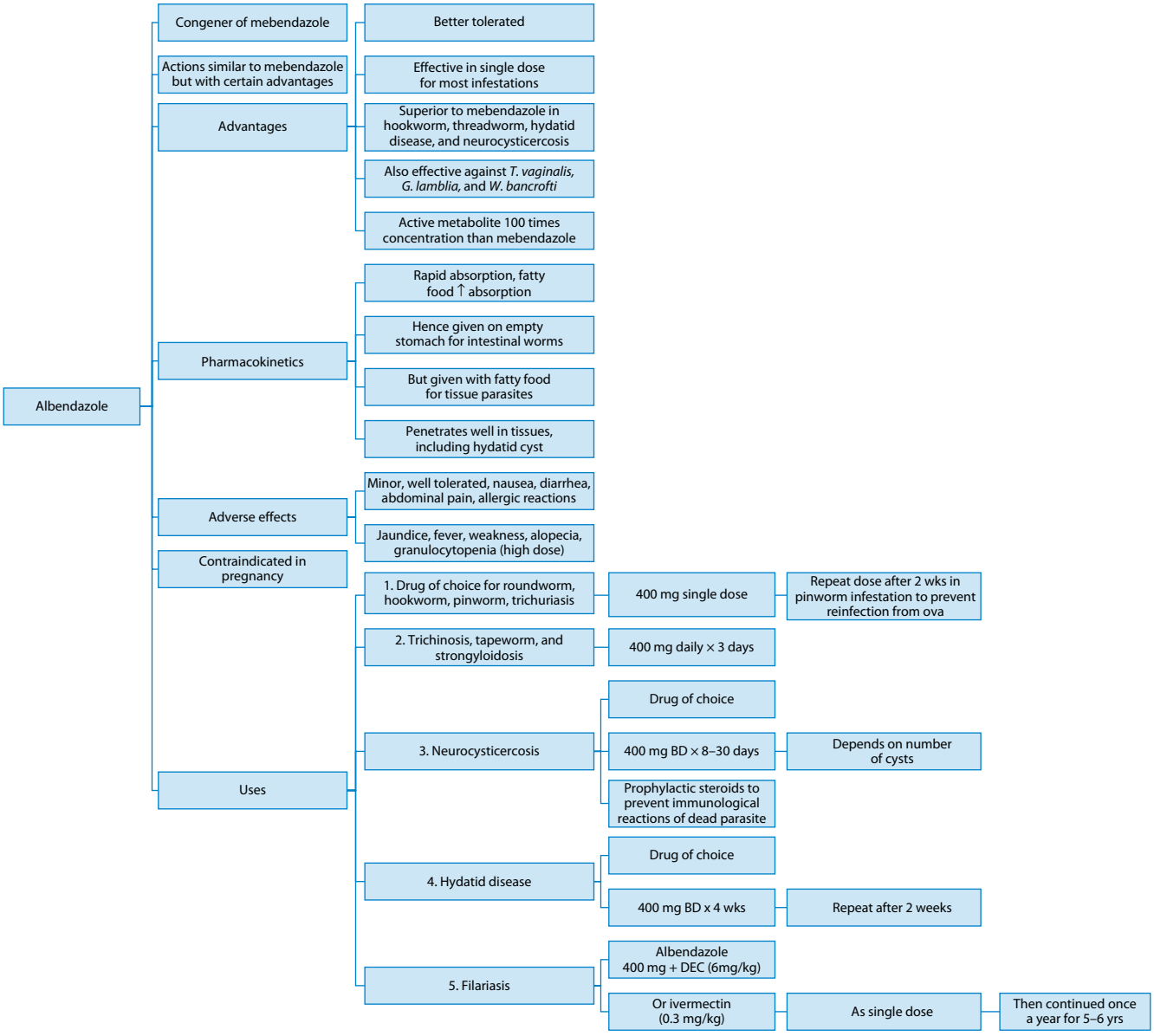
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Anthelmintics

71.1 MEBENDAZOLE

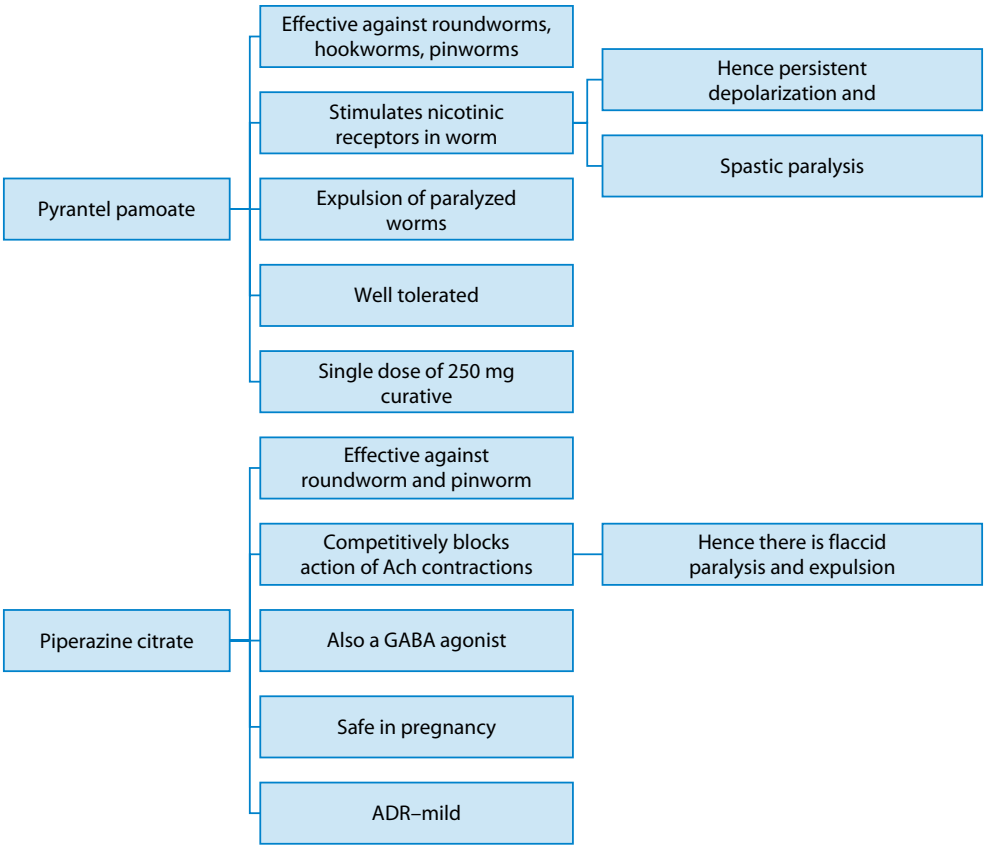


71.2 ALBENDAZOLE, PYRANTEL PAMOATE, PIPERAZINE CITRATE

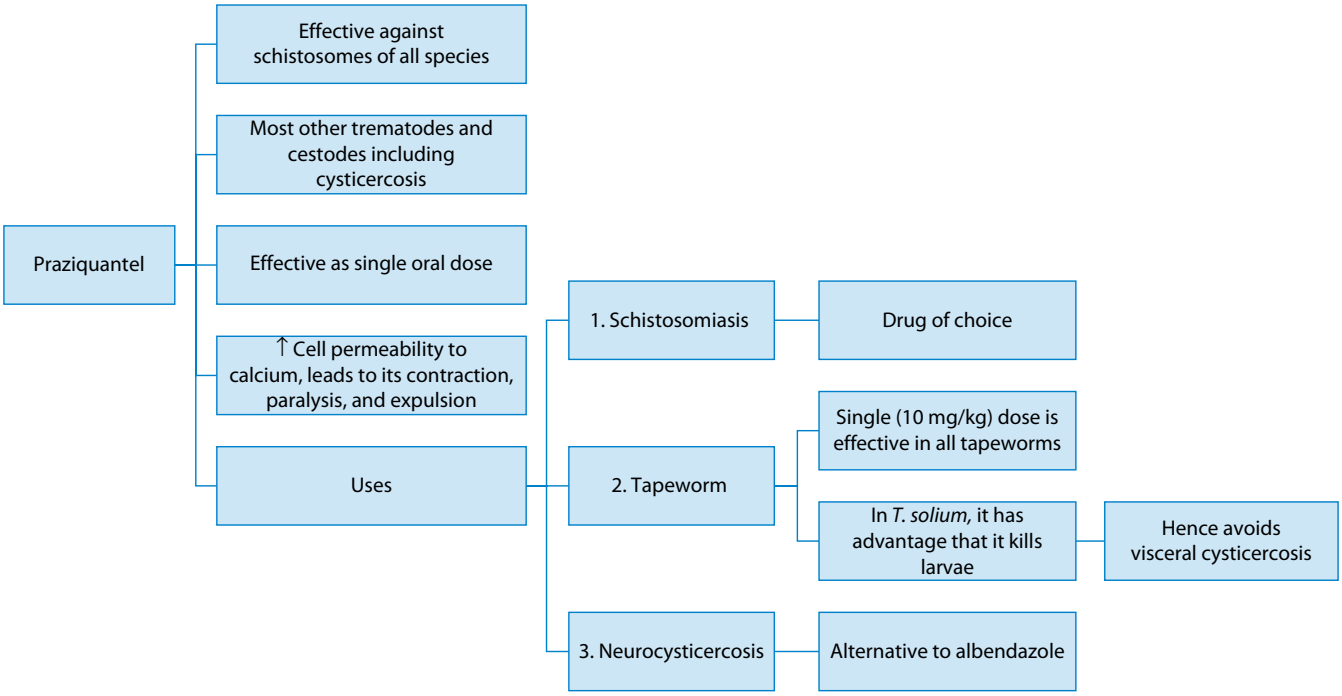


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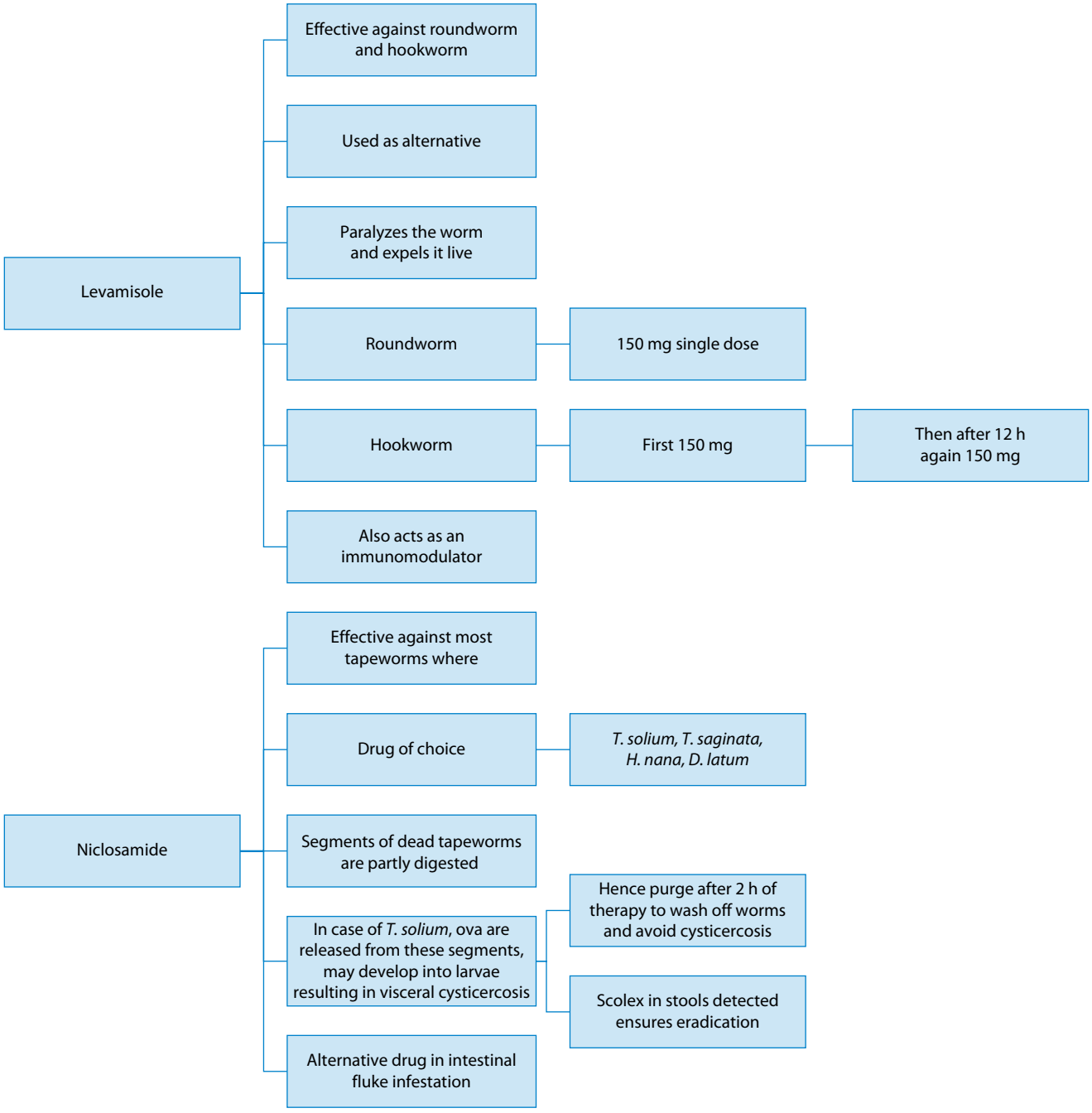
71.2 ALBENDAZOLE, PYRANTEL PAMOATE, PIPERAZINE CITRATE (Continued)



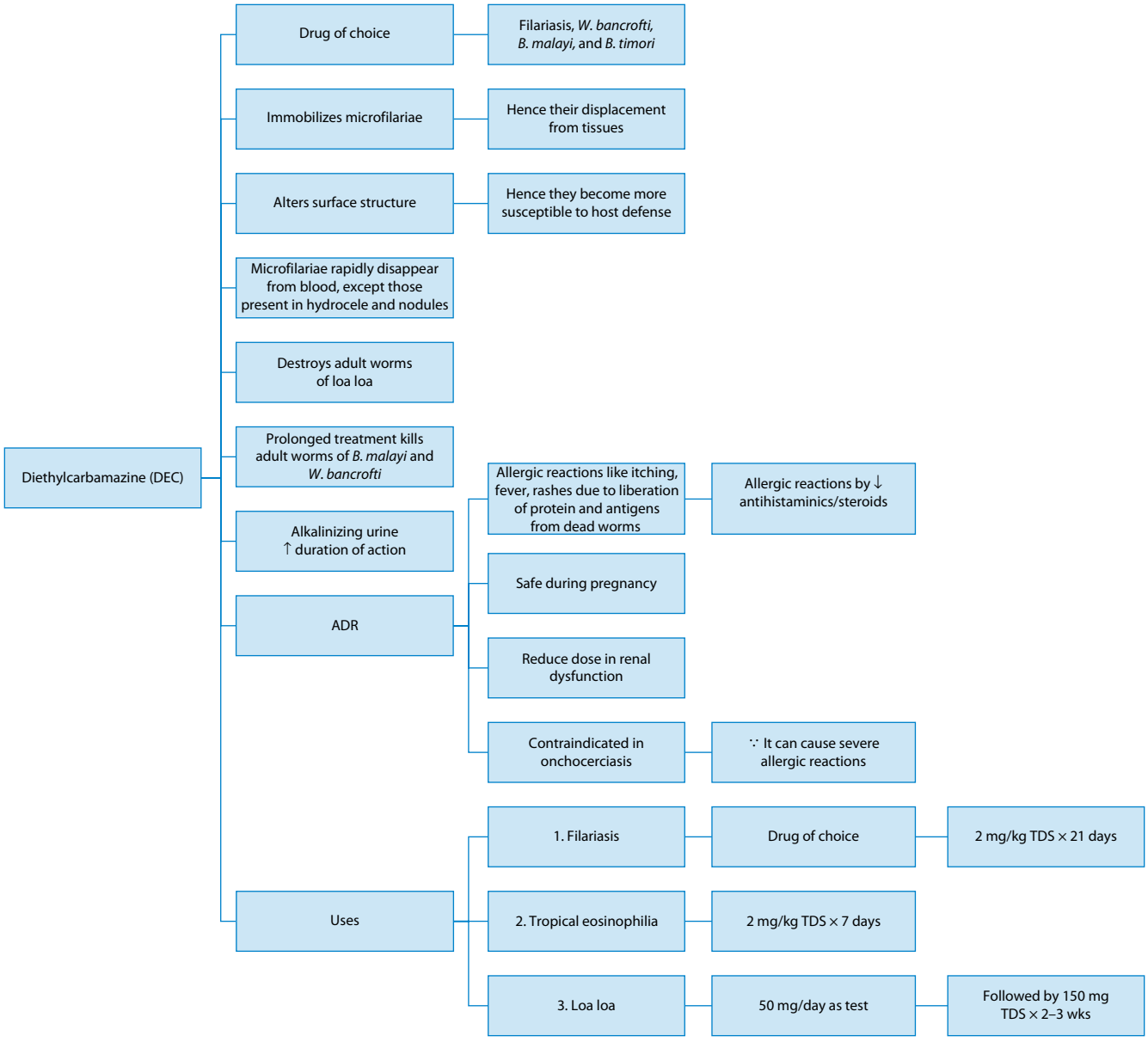
71.3 PRAZIQUANTEL



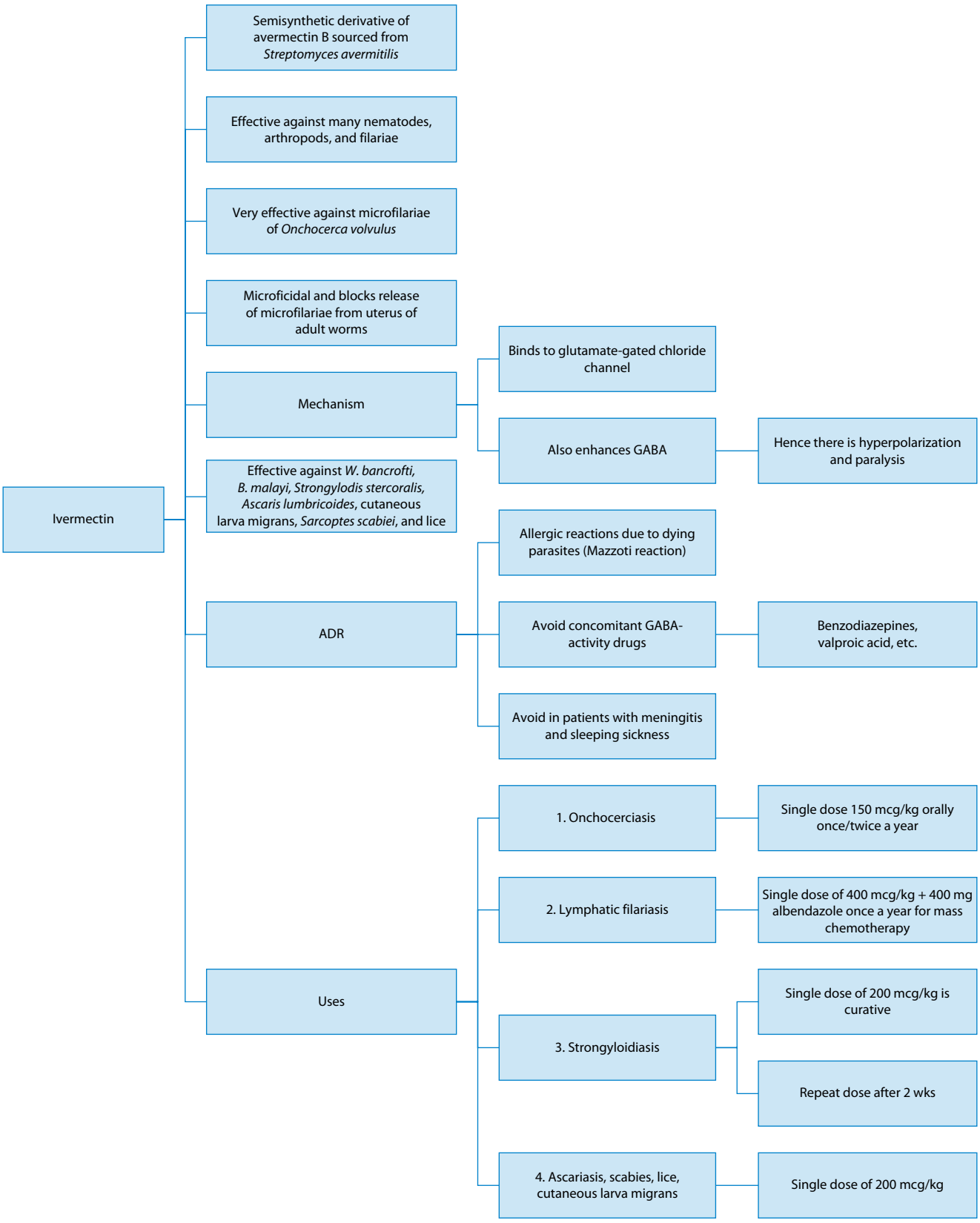
71.4 LEVAMISOLE AND NICLOSAMIDE



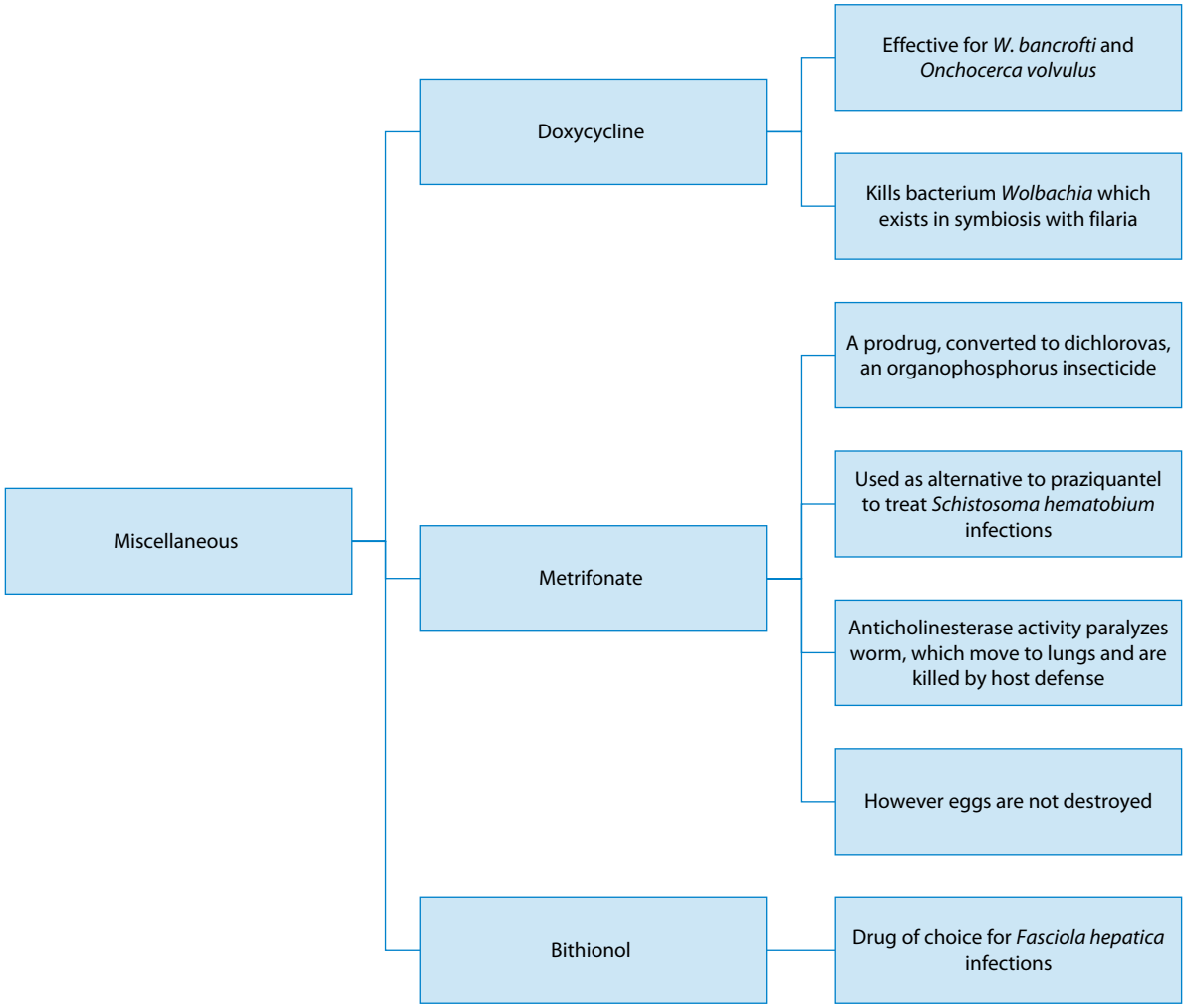
71.5 DIETHYLCARBAMAZINE (DEC)



71.6 IVERMECTIN



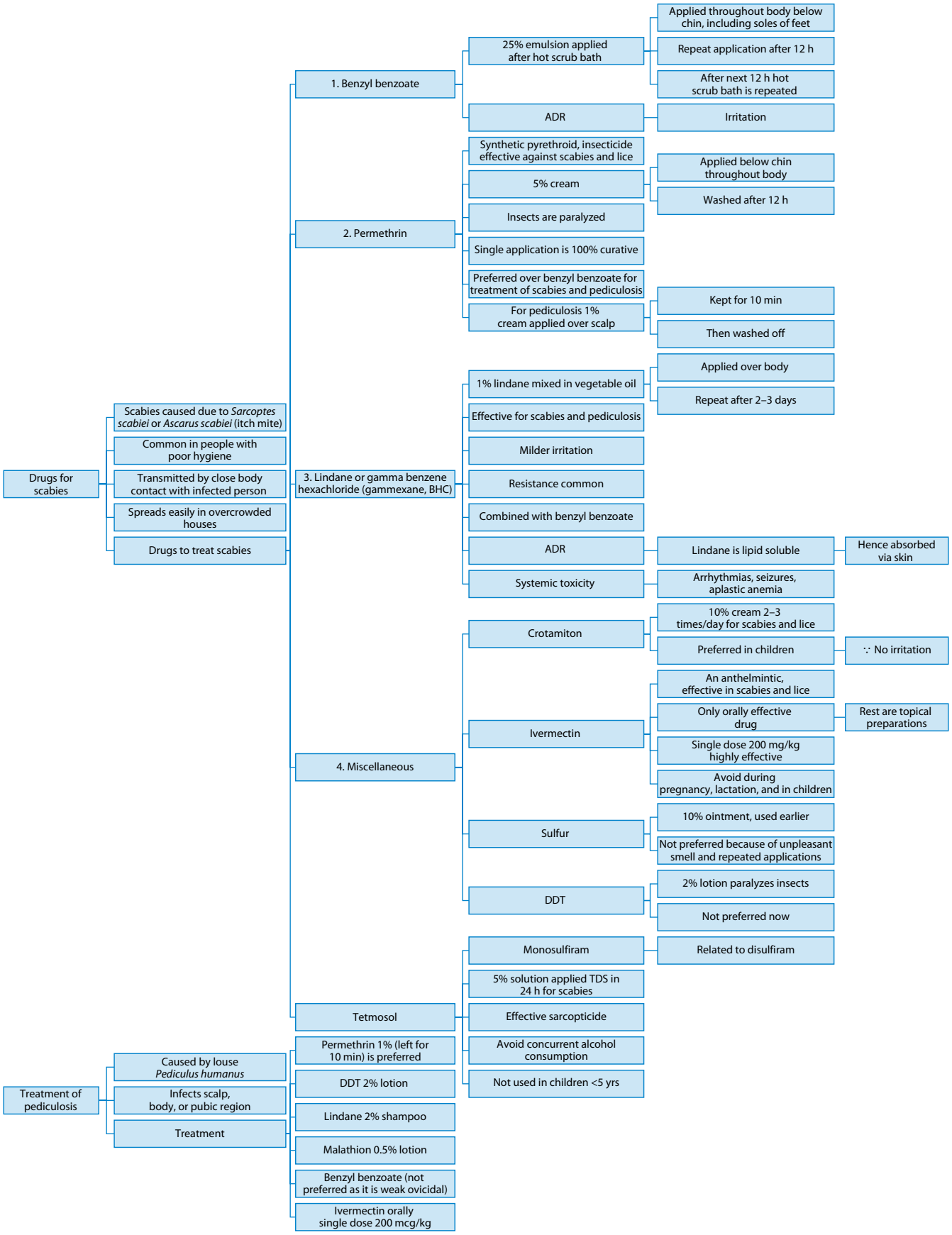
71.7 MISCELLANEOUS



71.8 PREFERRED DRUGS FOR HELMINTIC INFESTATIONS

Worm	Drugs of choice	Alternative
1. *Ascaris lumbricoides* (roundworm)	Mebendazole (M)/albendazole (A) piperazine (Pp)	Pyrantel (P)
2. *Ankylostoma duodenale* (hookworm) *Necator americanus*	M/A	Pyrantel (P)
3. *Enterobius vermicularis* (pinworm)	M/A/P	Pp
4. *Trichuris trichura* (whipworm)	M	A
5. *Strongyloides stercoralis*	A	Thiabendazole
6. *Dracunculus medinensis* (guineaworm)	Metronidazole	M
7. *Neurocysticercosis* (tapeworms)	Niclosamide/praziquantel A A	Praziquantel
8. Hydatid disease	A	M
9. Filaria	DEC + A	Ivermectin + A
10. Schistosomes	Praziquantel	–
11. *Onchocerca volvulus*	Ivermectin	–
12. *Fasciola hepatica* (sheep liver fluke)	Bithionol	–

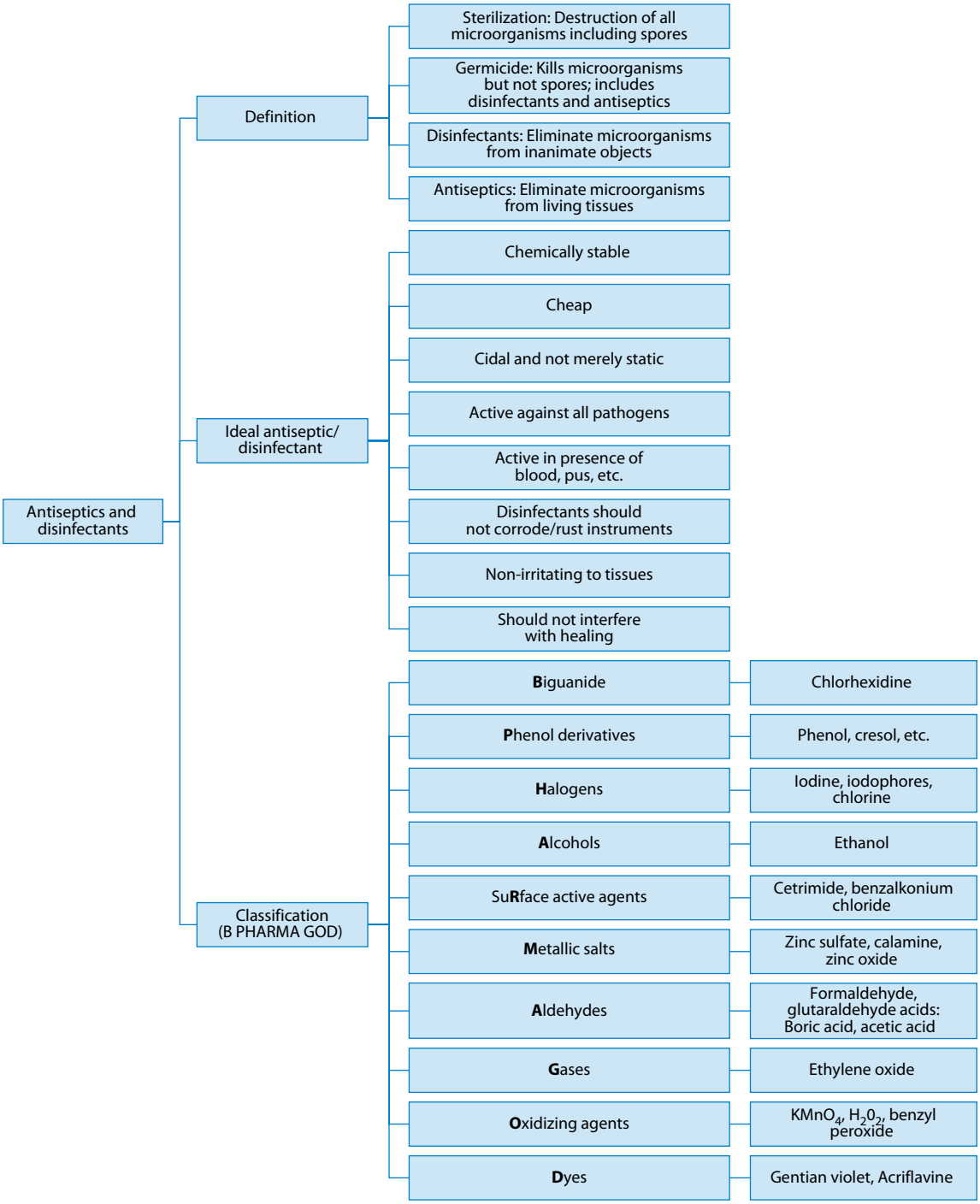
71.9 DRUGS FOR SCABIES AND TREATMENT OF PEDICULOSIS



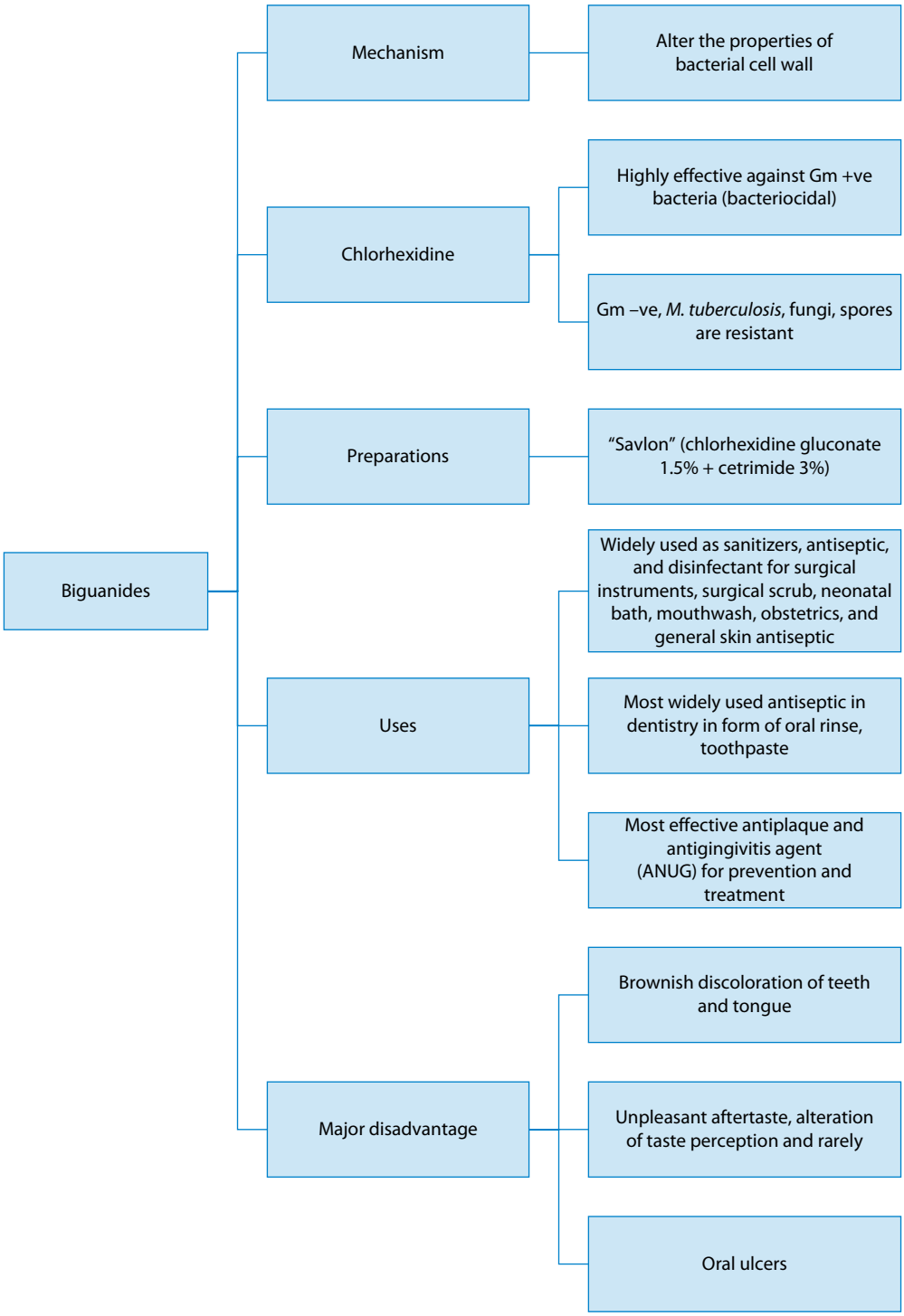
72

Antiseptics and disinfectants

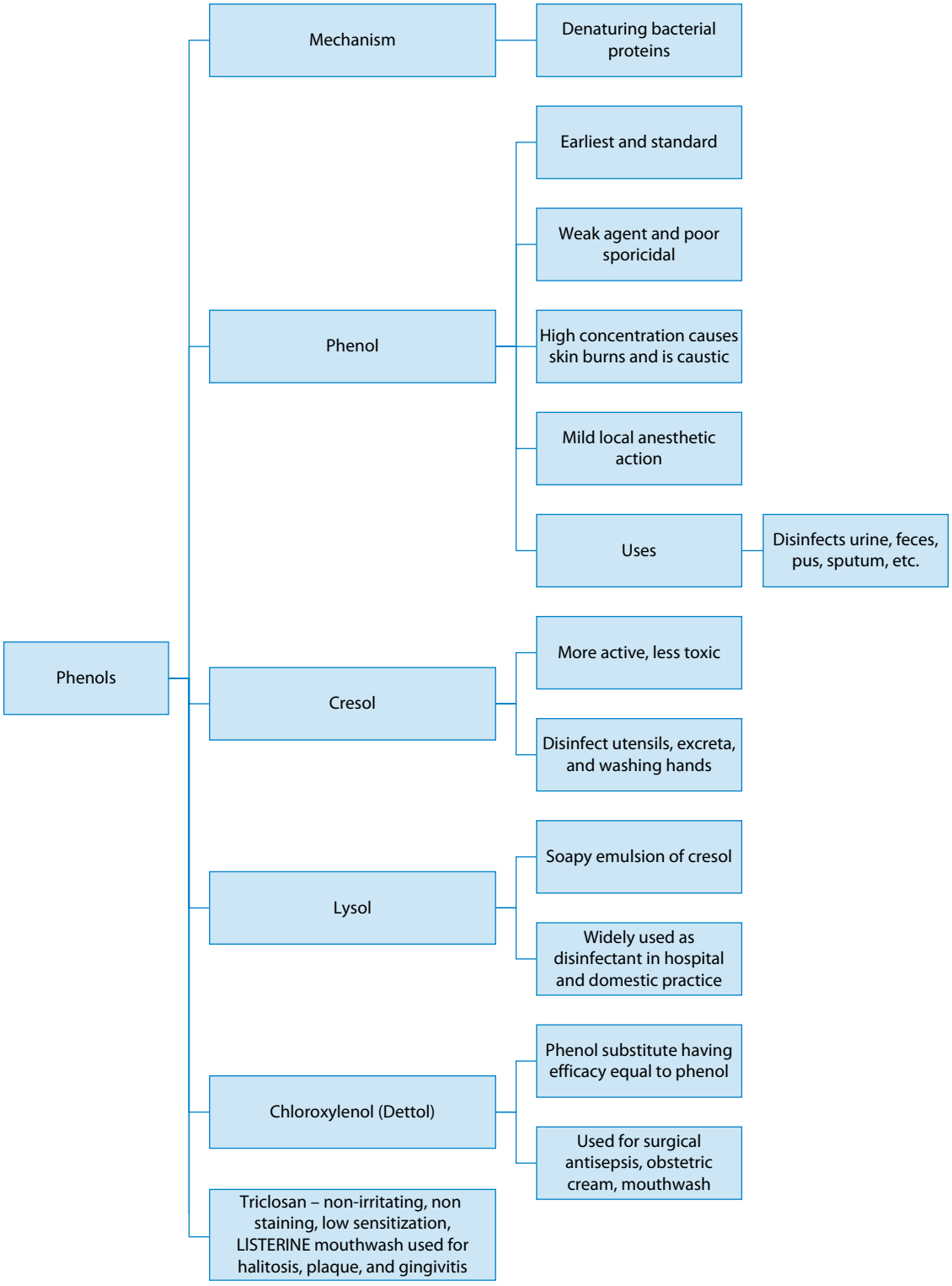
72.1 DEFINITION AND CLASSIFICATION



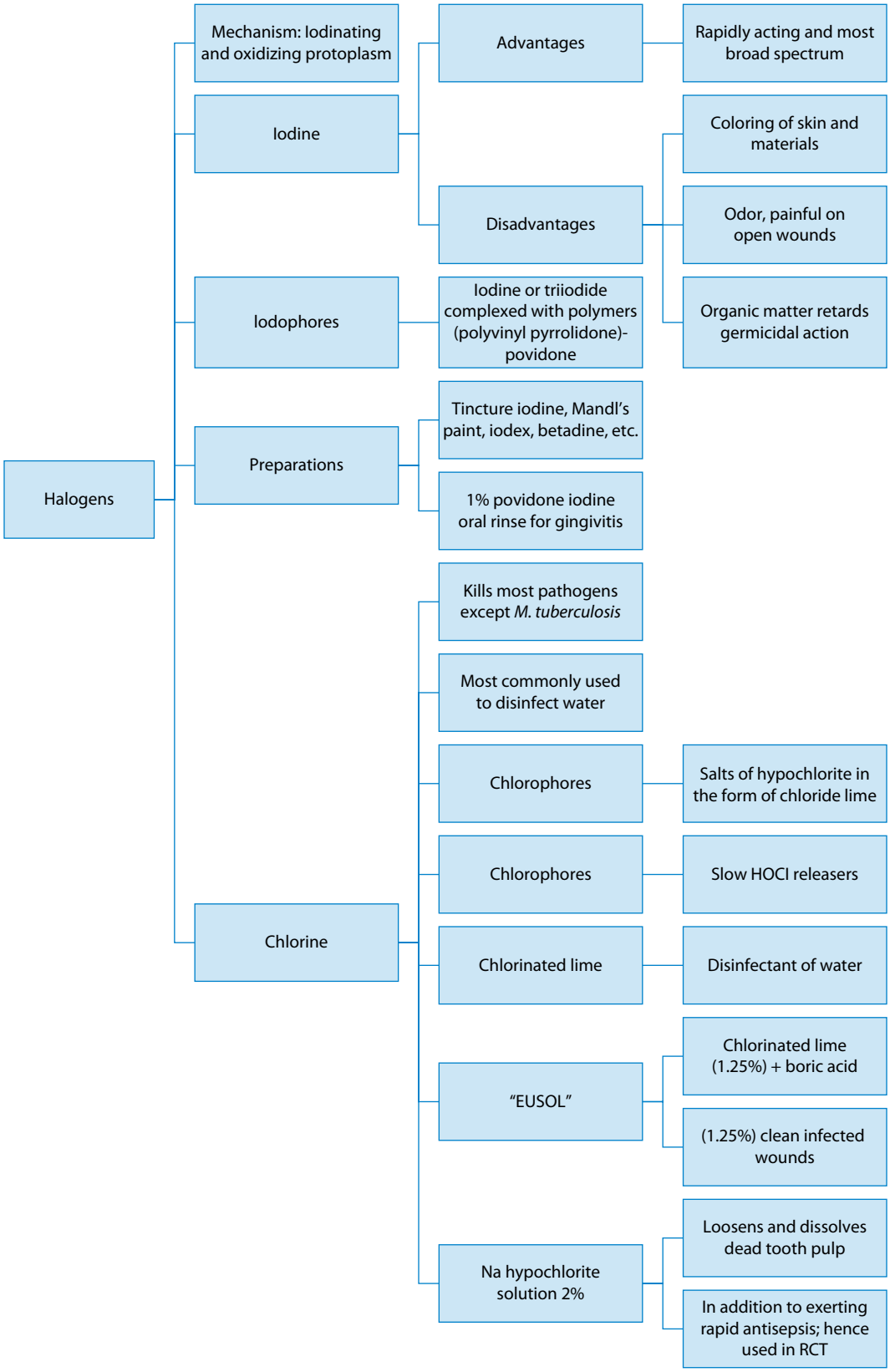
72.2 BIGUANIDES



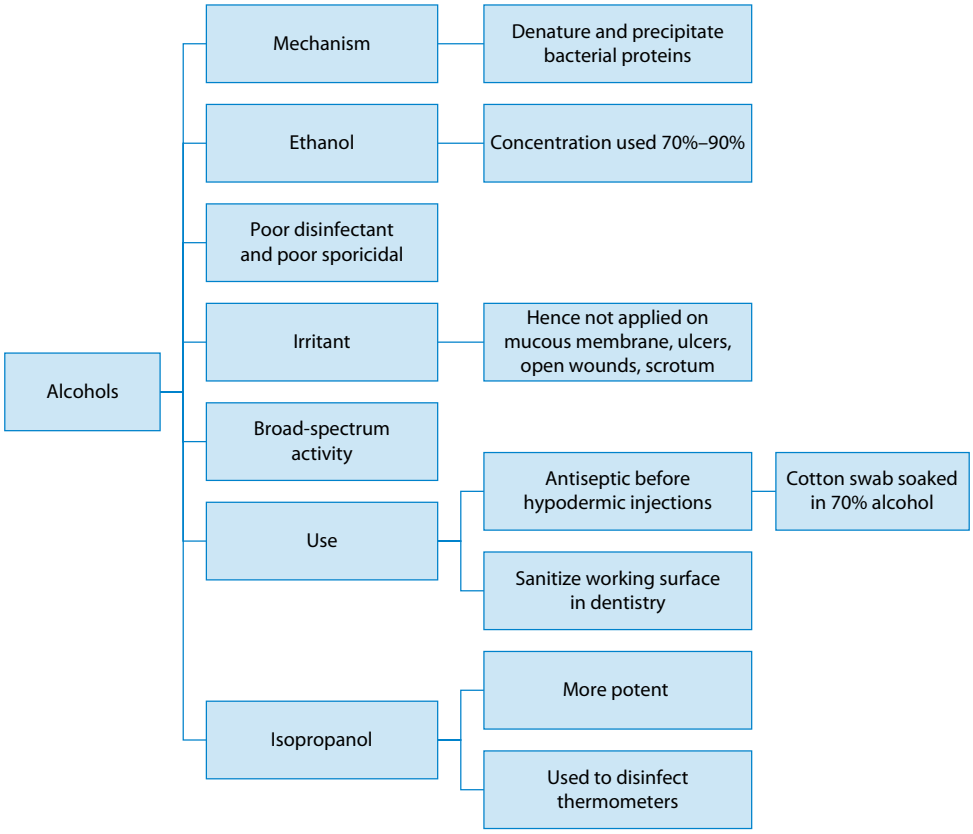
72.3 PHENOLS


Phenols
Mechanism
Denaturing bacterial proteins
Phenol
Earliest and standard
Weak agent and poor sporicidal
High concentration causes skin burns and is caustic
Mild local anesthetic action
Uses
Disinfects urine, feces, pus, sputum, etc.
Cresol
More active, less toxic
Disinfect utensils, excreta, and washing hands
Lysol
Soapy emulsion of cresol
Widely used as disinfectant in hospital and domestic practice
Chloroxylenol (Dettol)
Phenol substitute having efficacy equal to phenol
Used for surgical antisepsis, obstetric cream, mouthwash
Triclosan – non-irritating, non staining, low sensitization, LISTERINE mouthwash used for halitosis, plaque, and gingivitis

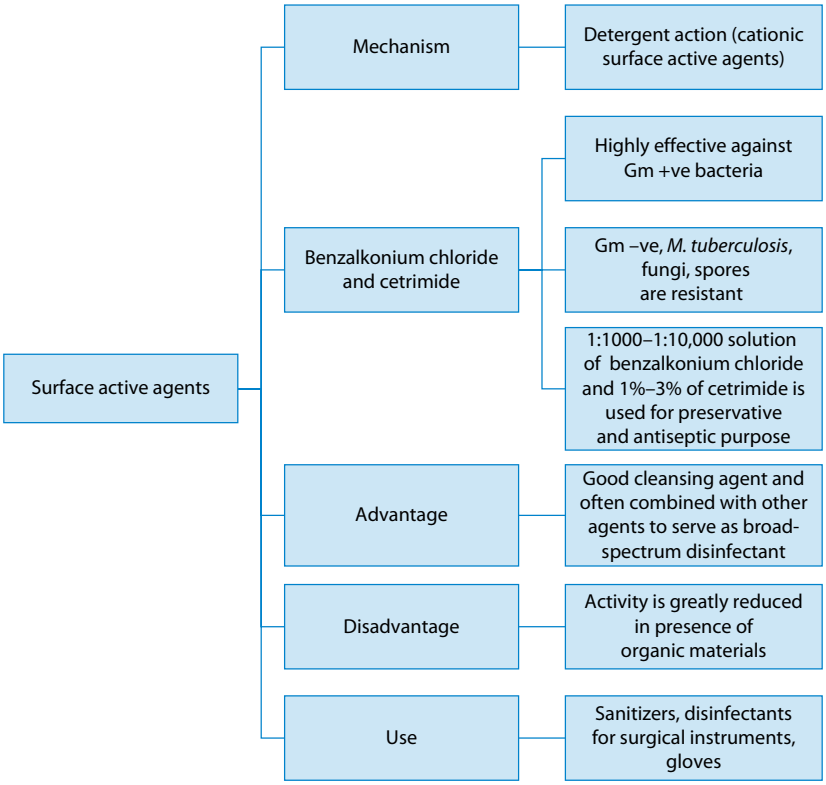
72.4 HALOGENS



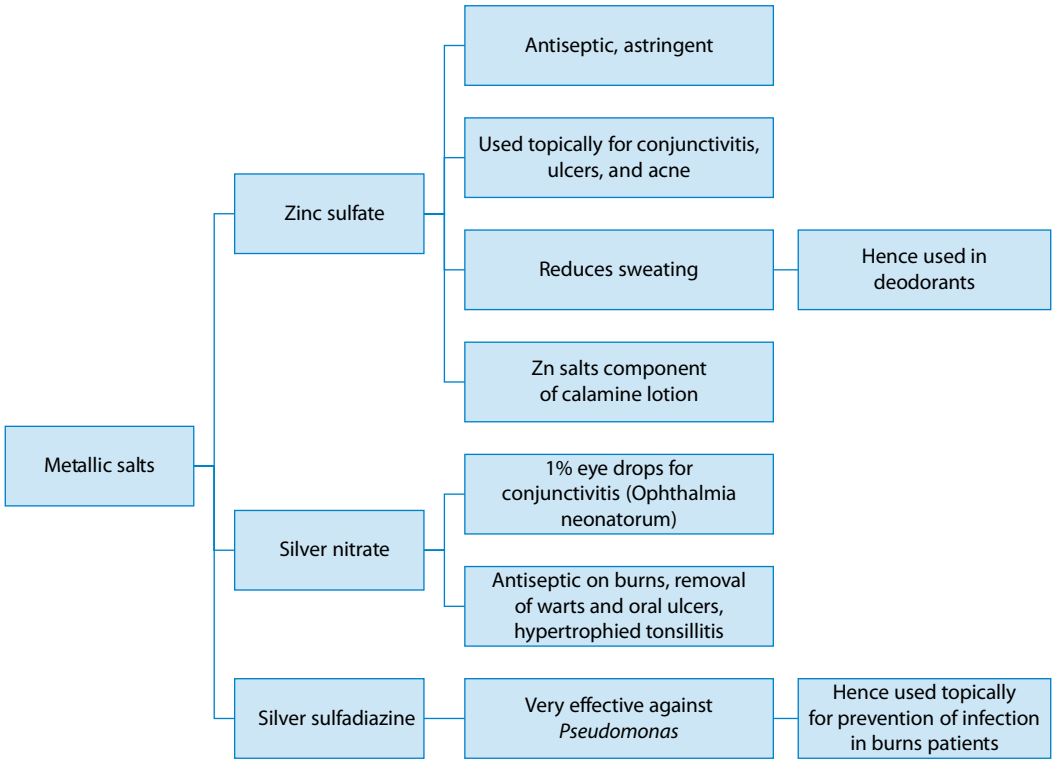
72.5 ALCOHOLS



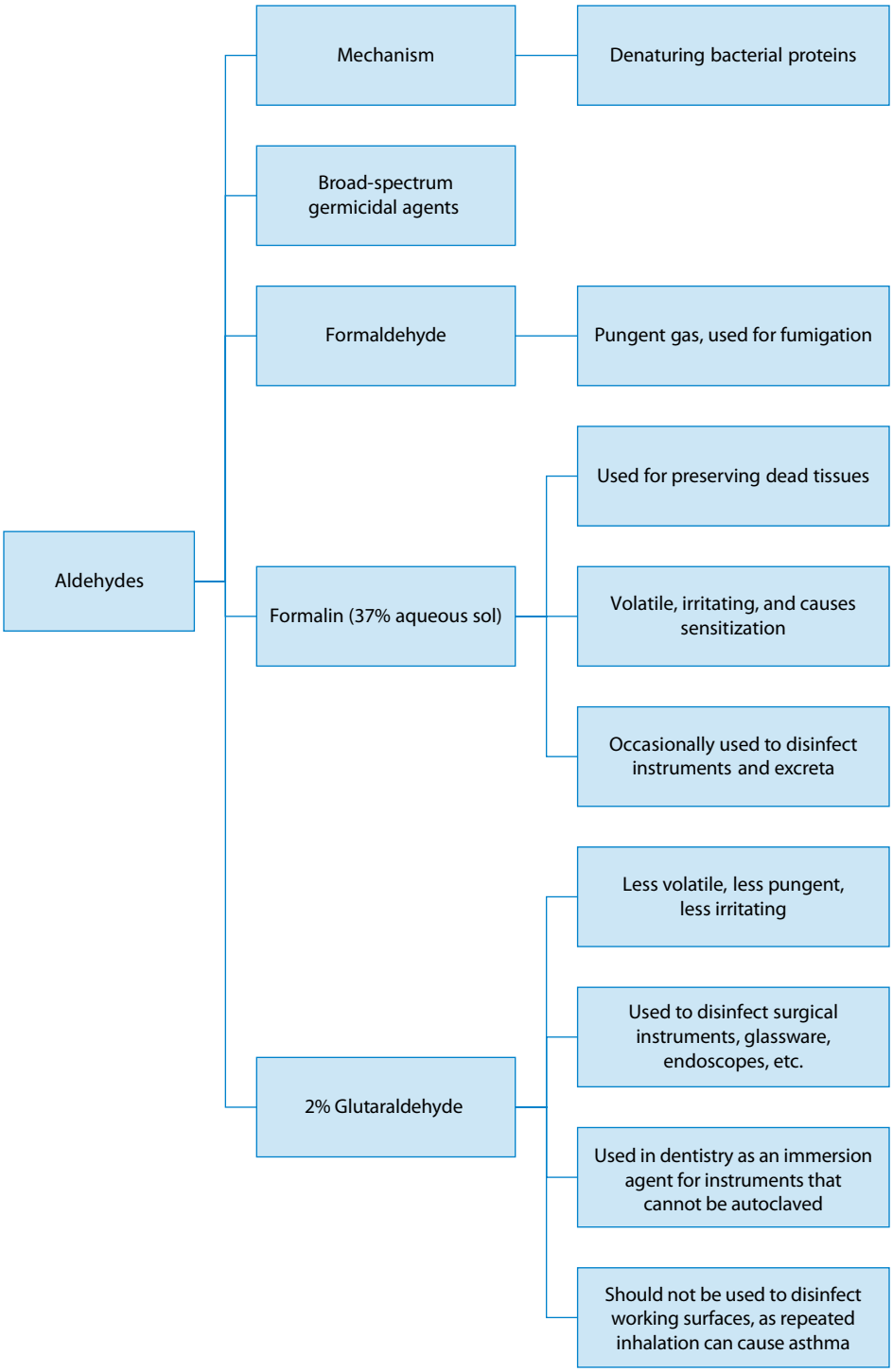
72.6 SURFACE ACTIVE AGENTS



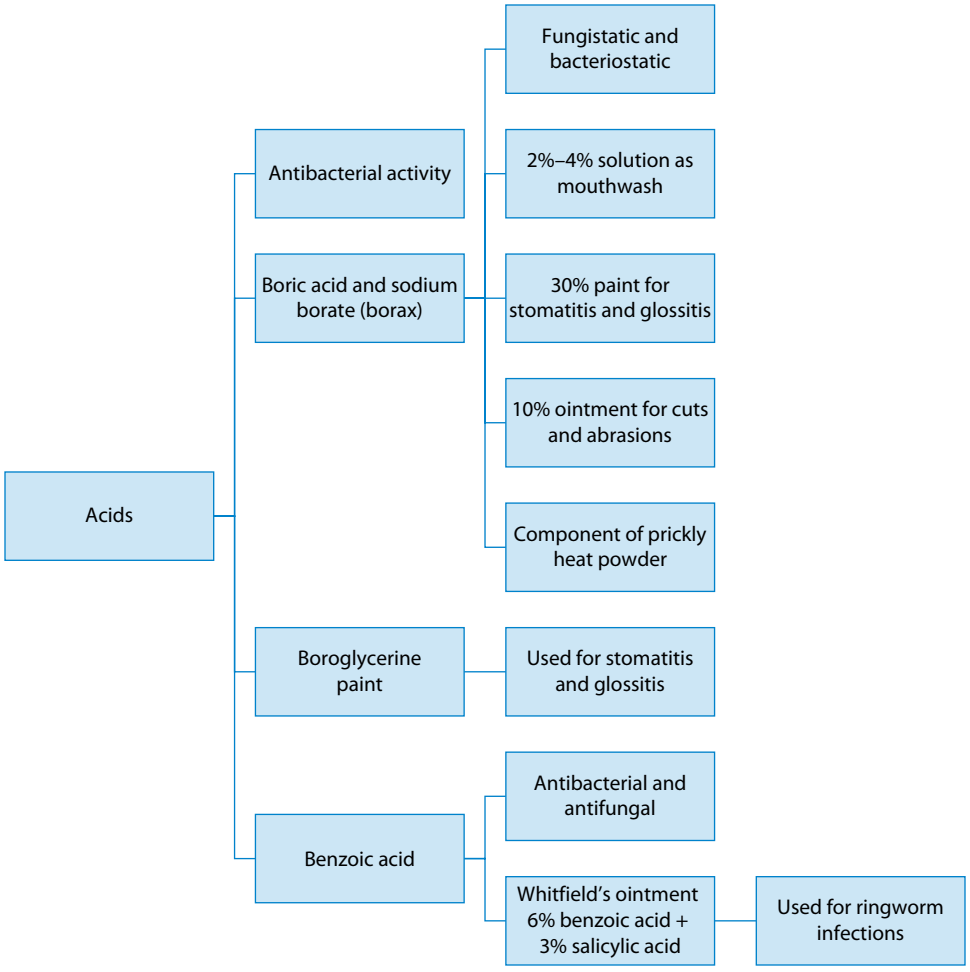
72.7 METALLIC SALTS



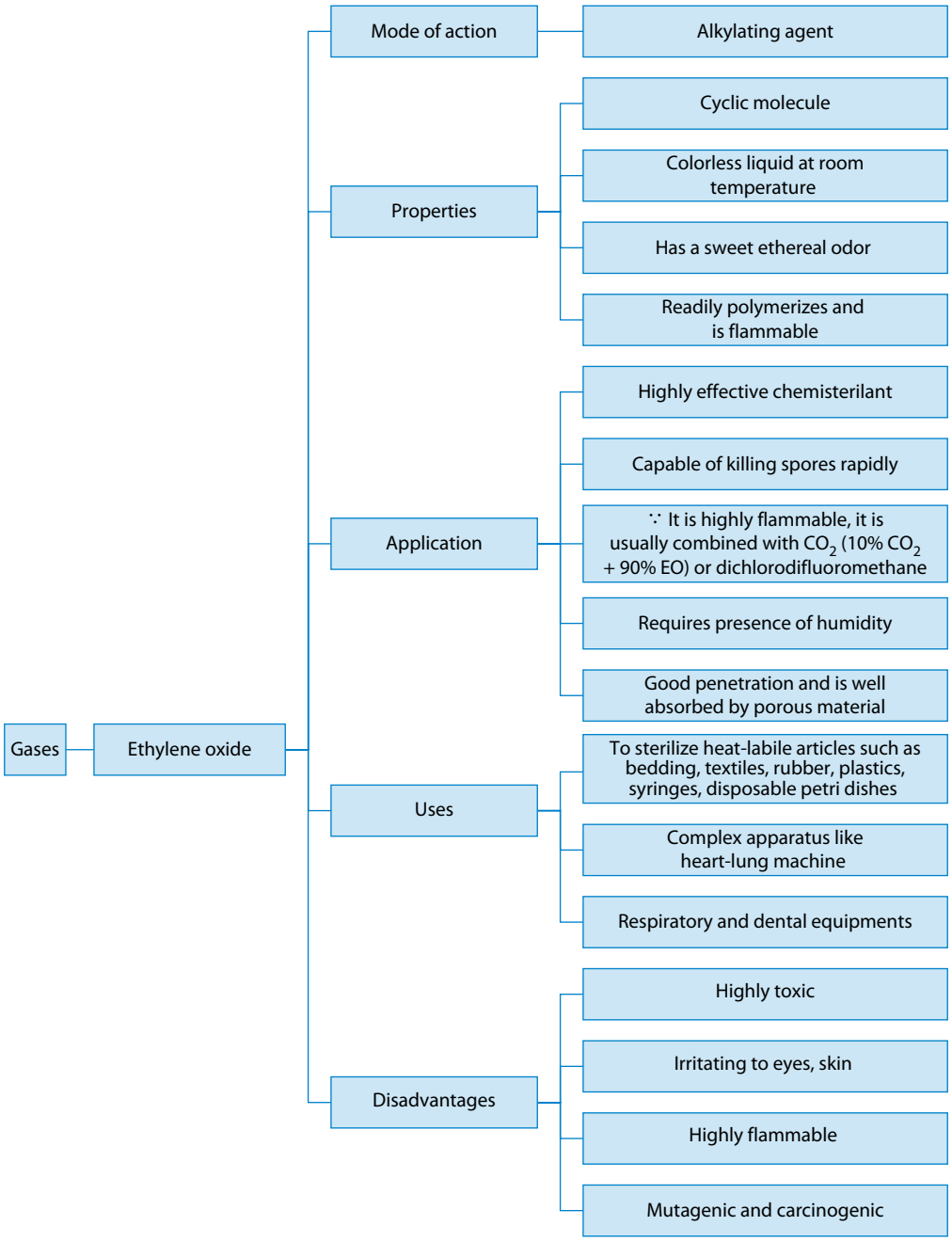
72.8 ALDEHYDES



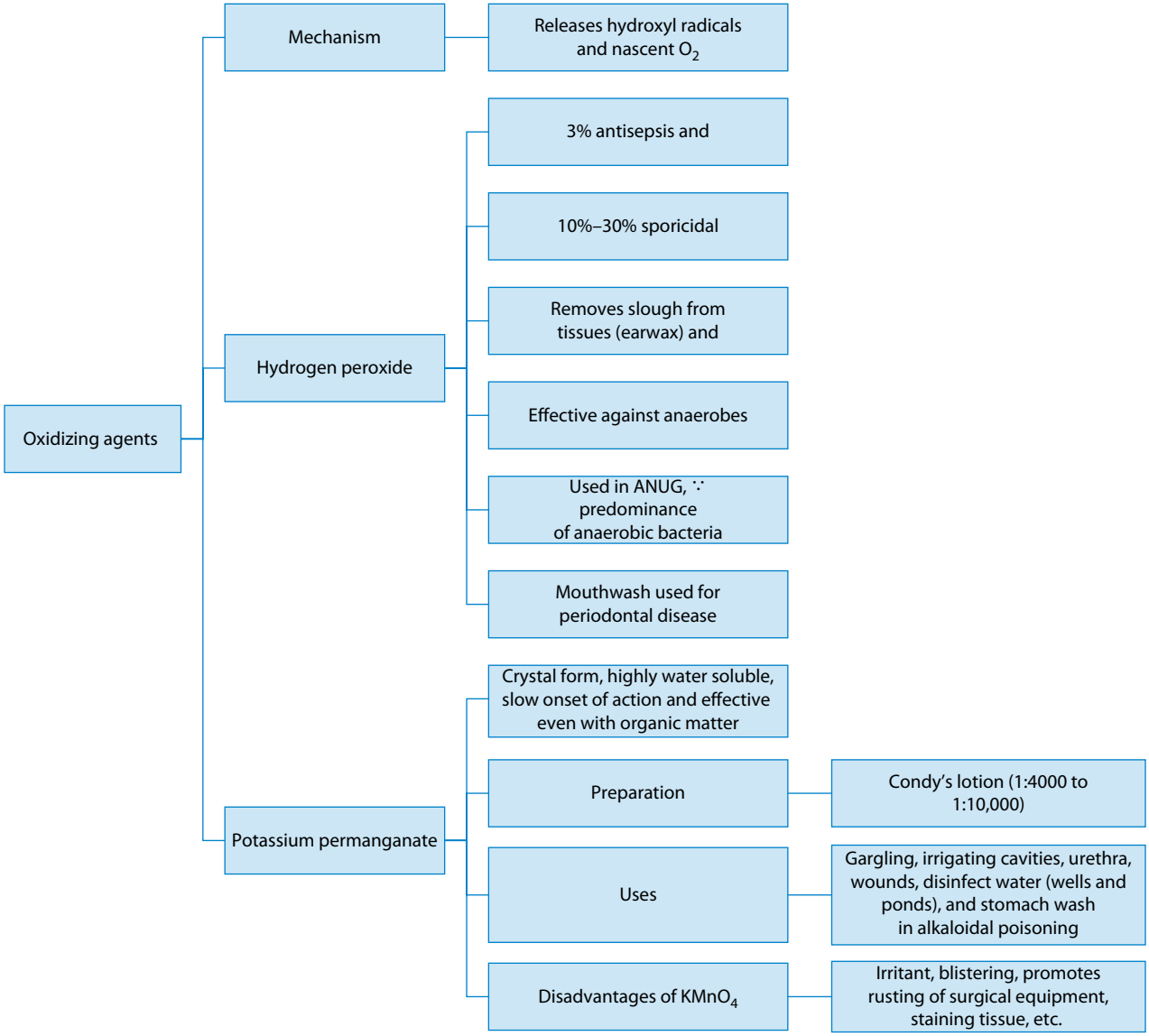
72.9 ACIDS


Acids
Antibacterial activity
Boric acid and sodium borate (borax)
Fungistatic and bacteriostatic
2%–4% solution as mouthwash
30% paint for stomatitis and glossitis
10% ointment for cuts and abrasions
Component of prickly heat powder
Boroglycerine paint
Used for stomatitis and glossitis
Benzoic acid
Antibacterial and antifungal
Whitfield's ointment 6% benzoic acid + 3% salicylic acid
Used for ringworm infections

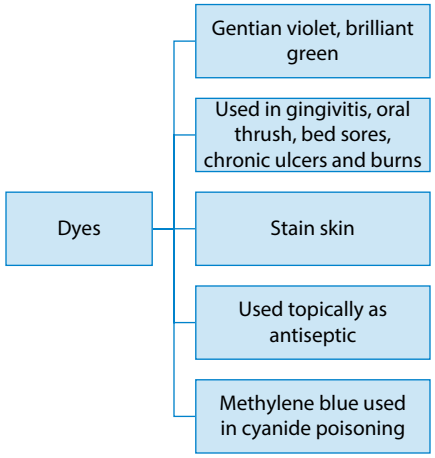
72.10 GASES


Gases
Ethylene oxide
Mode of action
Alkylating agent
Properties
Cyclic molecule
Colorless liquid at room temperature
Has a sweet ethereal odor
Readily polymerizes and is flammable
Application
Highly effective chemisterilant
Capable of killing spores rapidly
∵ It is highly flammable, it is usually combined with CO_2 (10% CO_2 + 90% EO) or dichlorodifluoromethane
Requires presence of humidity
Good penetration and is well absorbed by porous material
Uses
To sterilize heat-labile articles such as bedding, textiles, rubber, plastics, syringes, disposable petri dishes
Complex apparatus like heart-lung machine
Respiratory and dental equipments
Disadvantages
Highly toxic
Irritating to eyes, skin
Highly flammable
Mutagenic and carcinogenic

72.11 OXIDIZING AGENTS



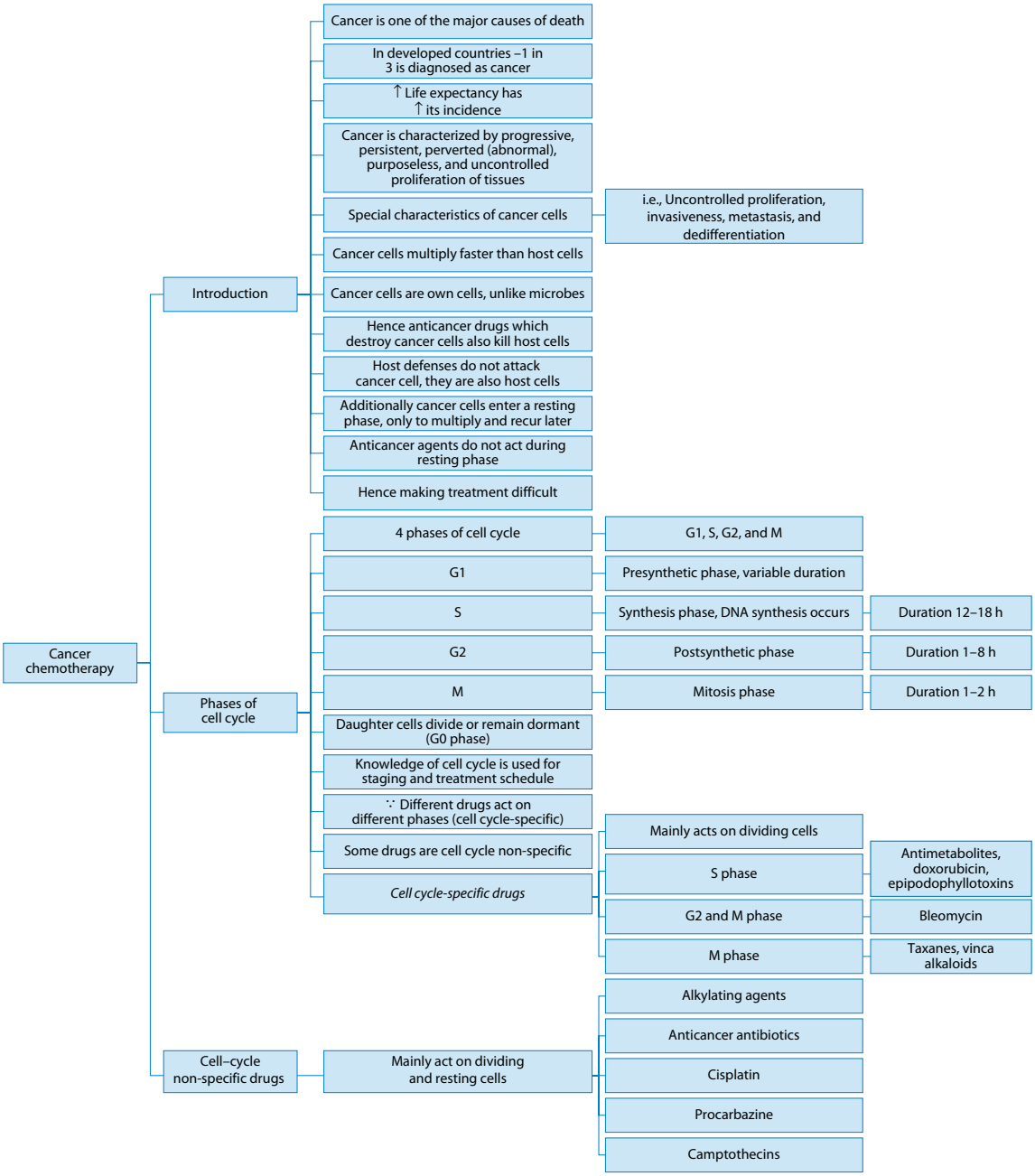
72.12 DYES



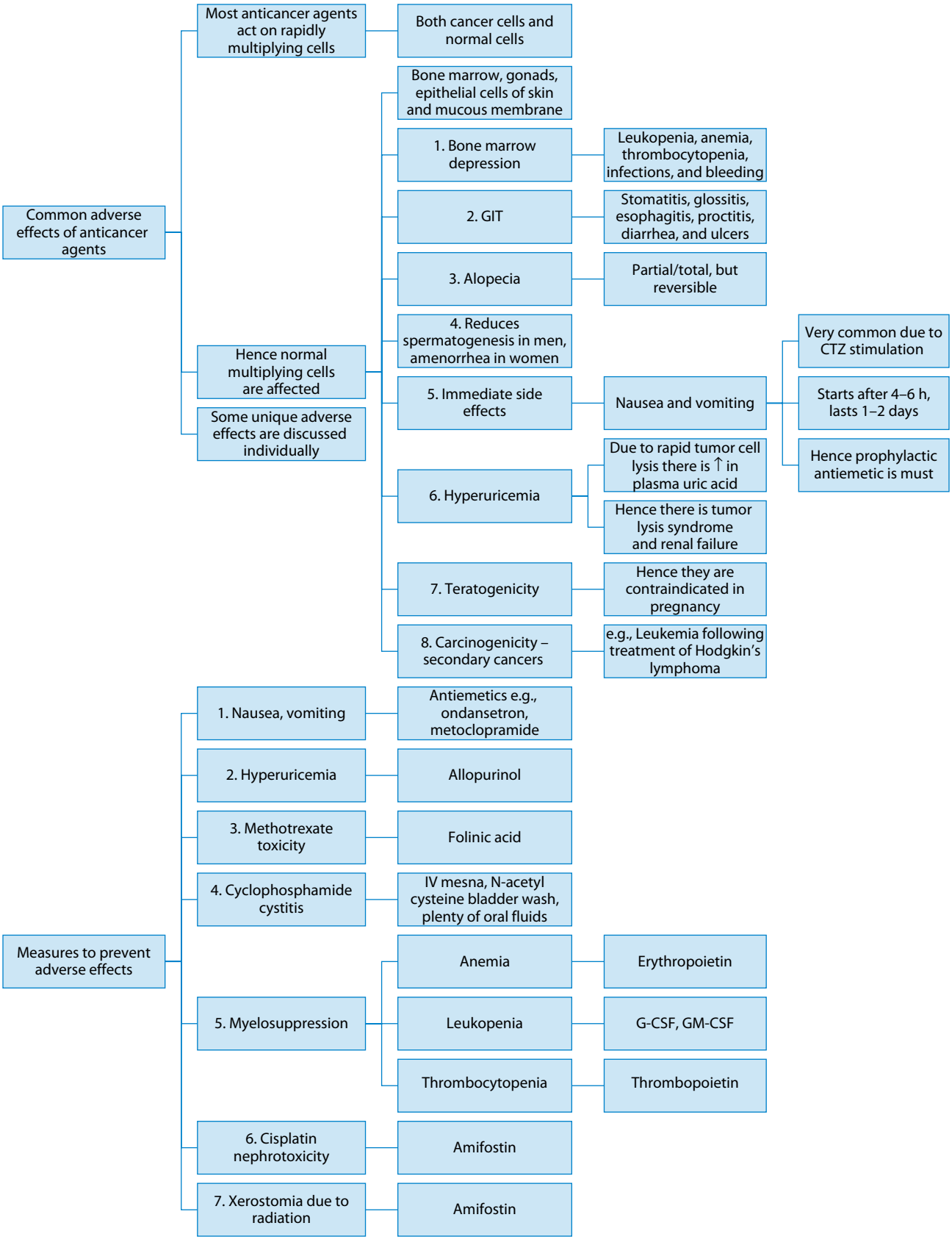
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Cancer chemotherapy

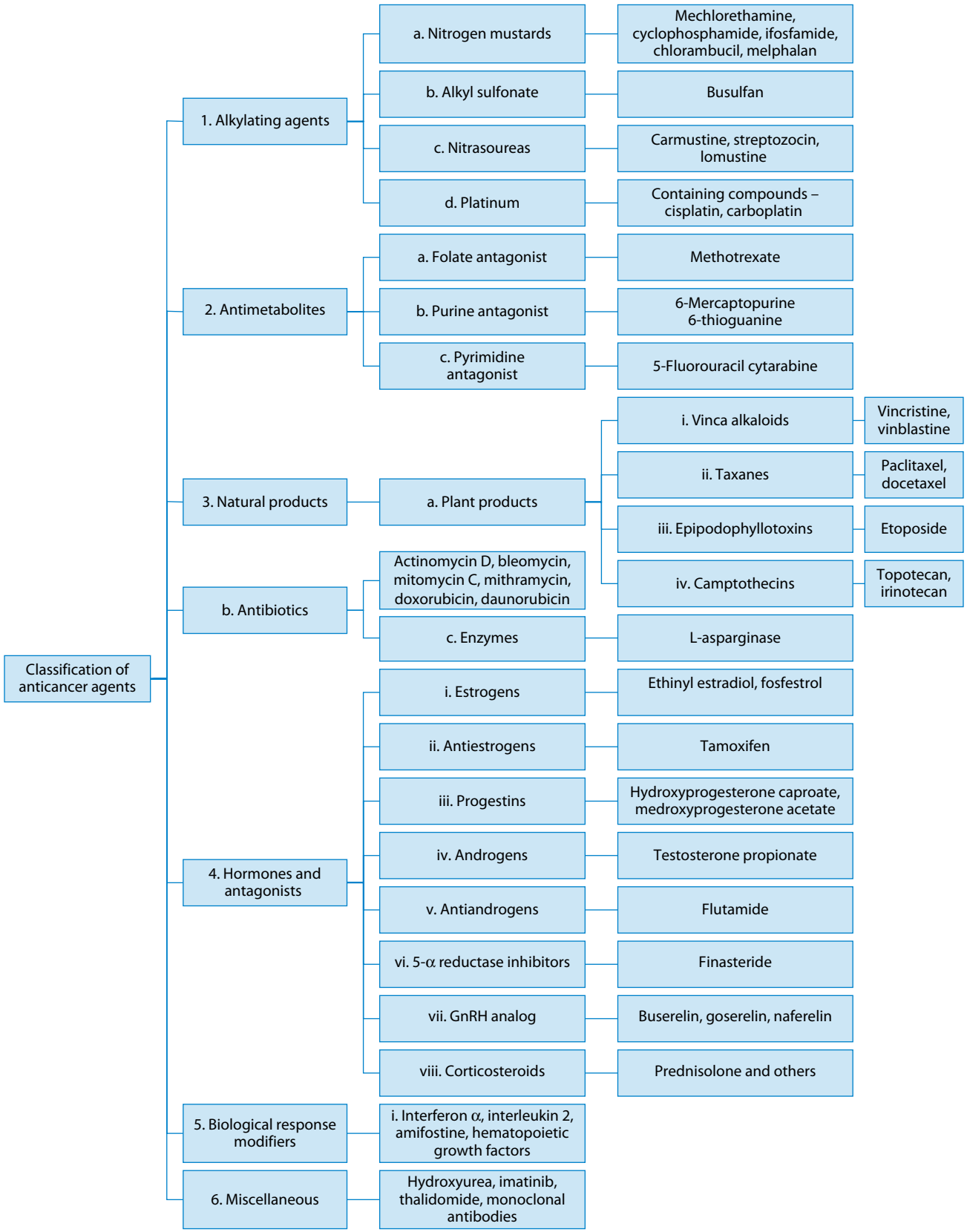
73.1 INTRODUCTION AND PHASES OF CELL CYCLES



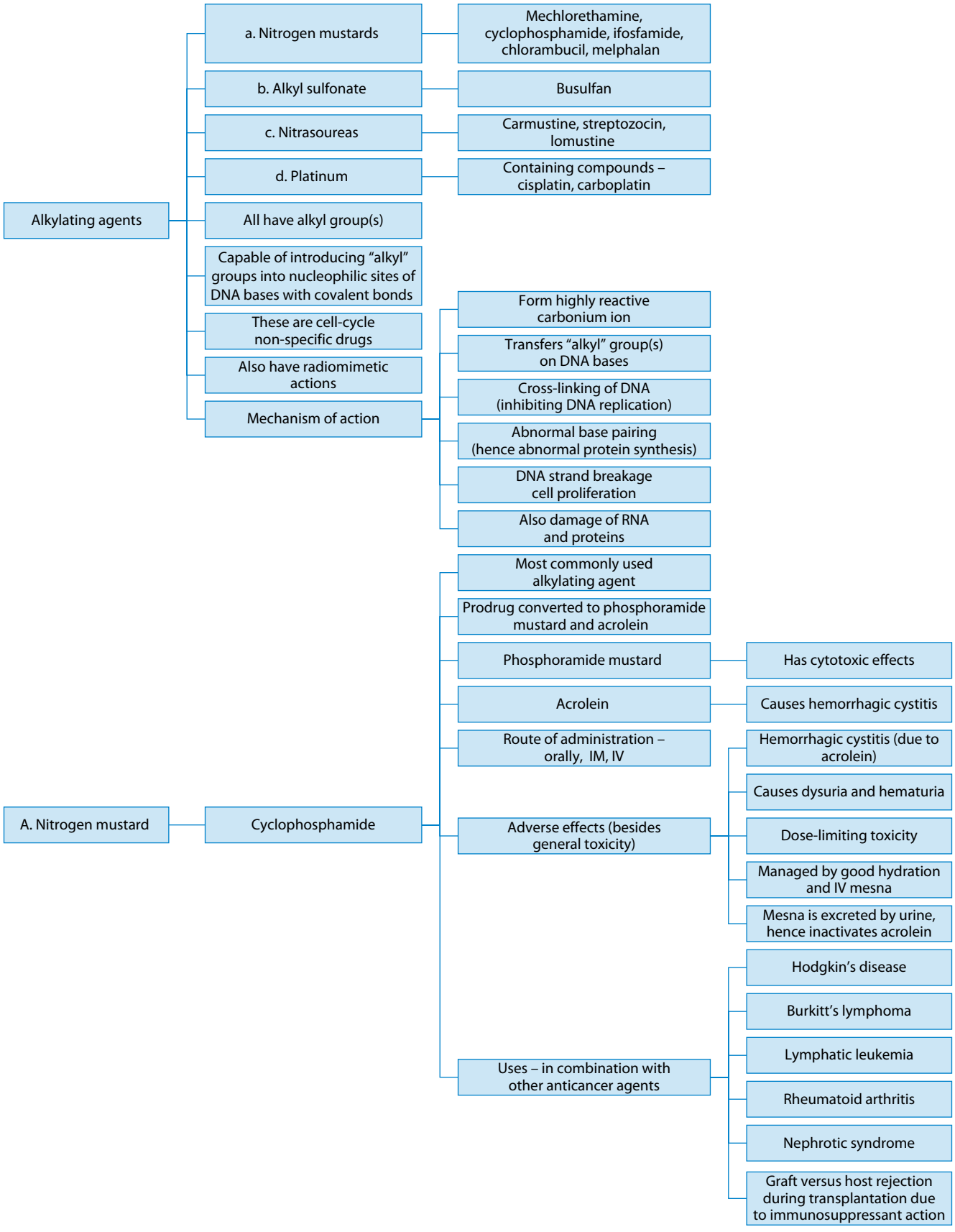
73.2 COMMON ADVERSE EFFECTS OF ANTICANCER AGENTS AND MEASURES TO PREVENT ADVERSE EFFECTS



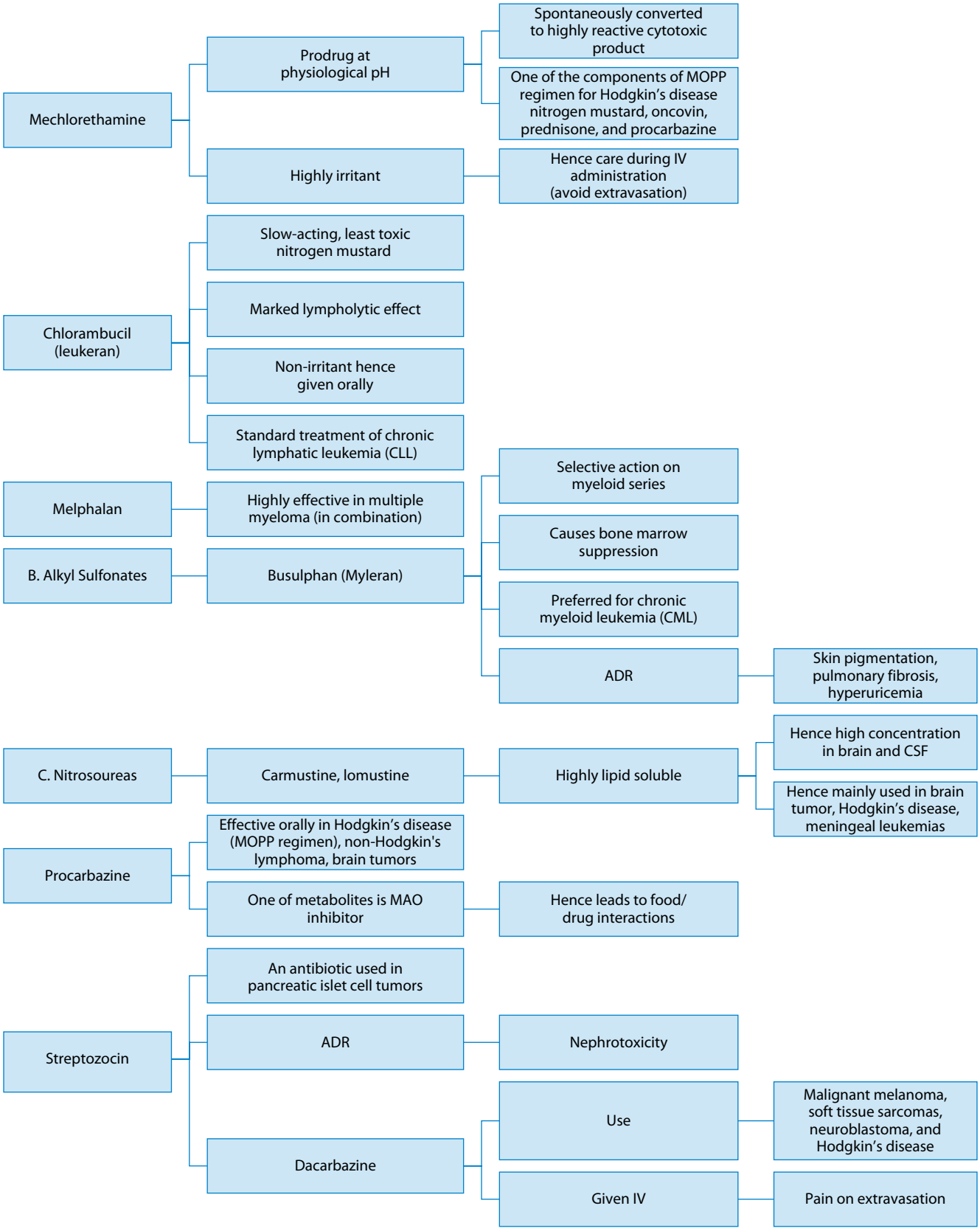
73.3 CLASSIFICATION OF ANTICANCER AGENTS



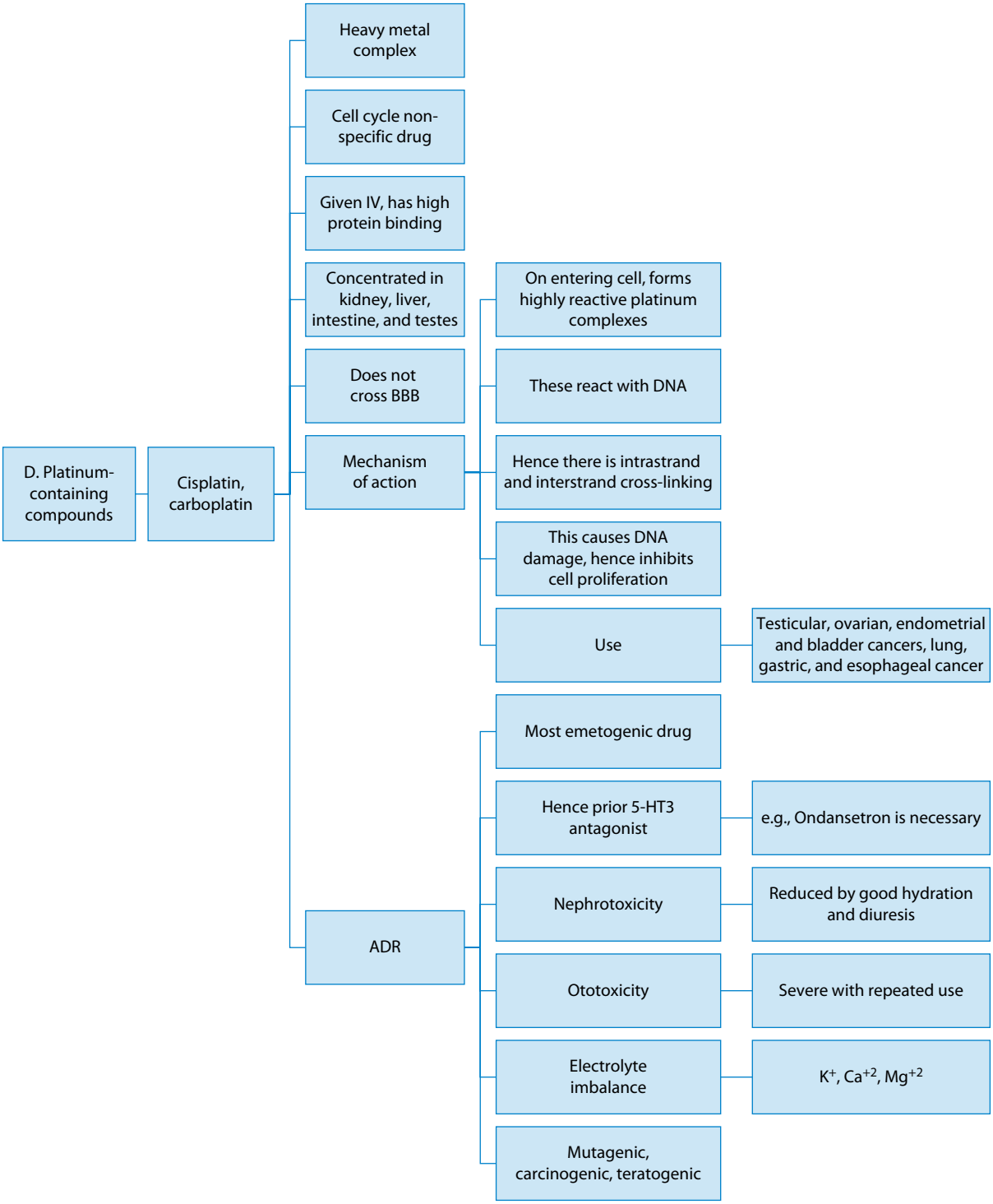
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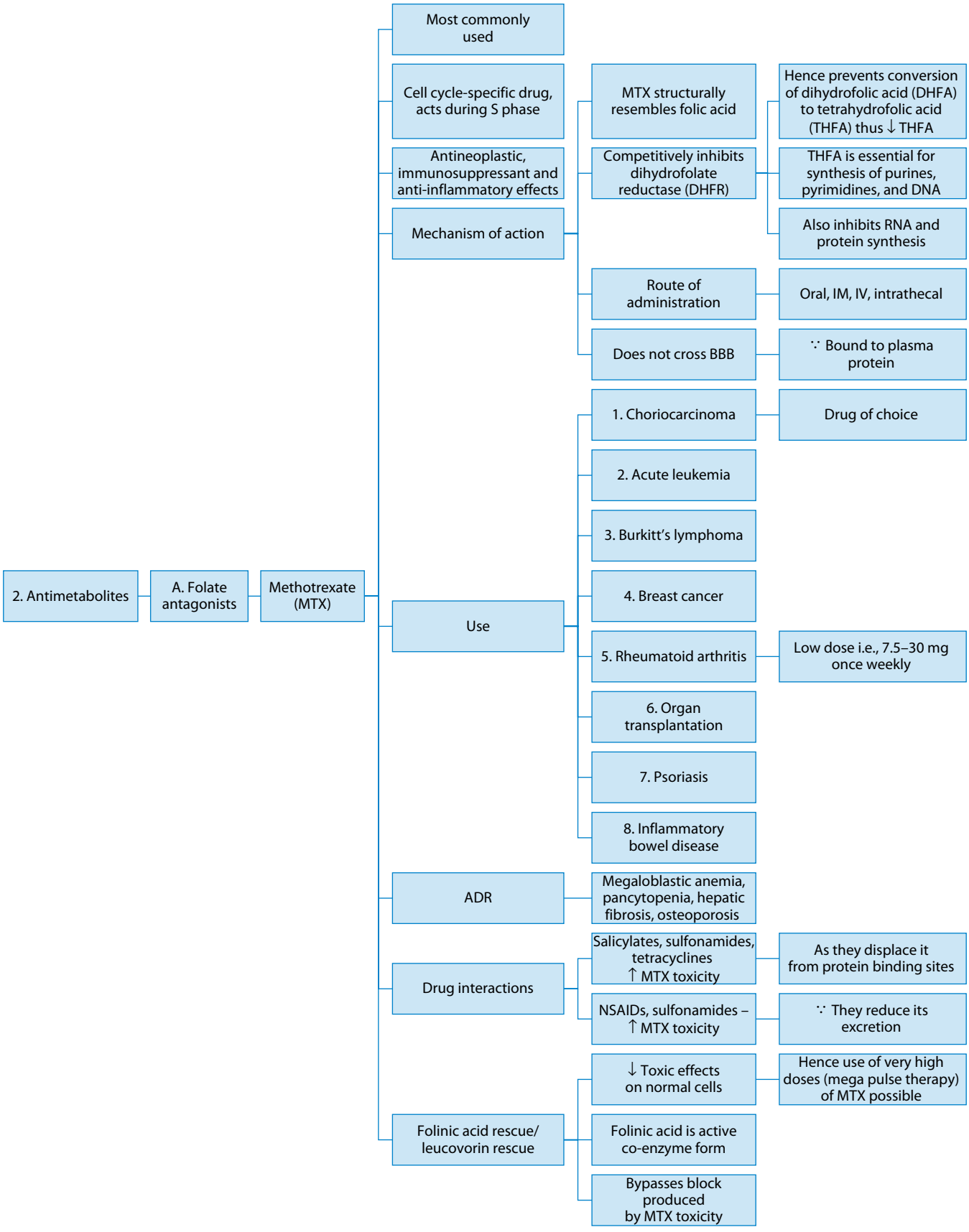
73.5 OTHER ALKYLATING AGENTS, ALKYL SULFONES, AND NITROSUREAS



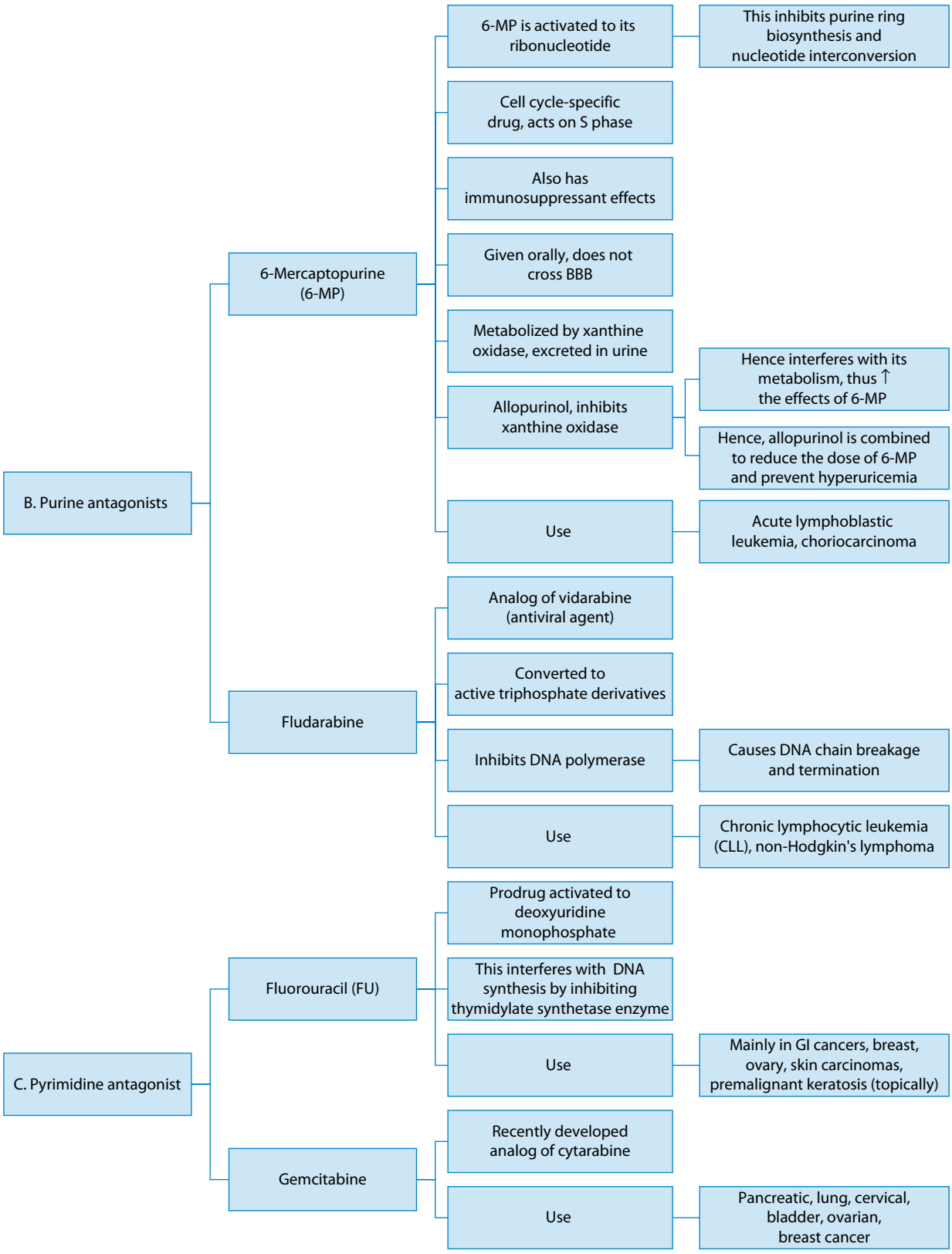
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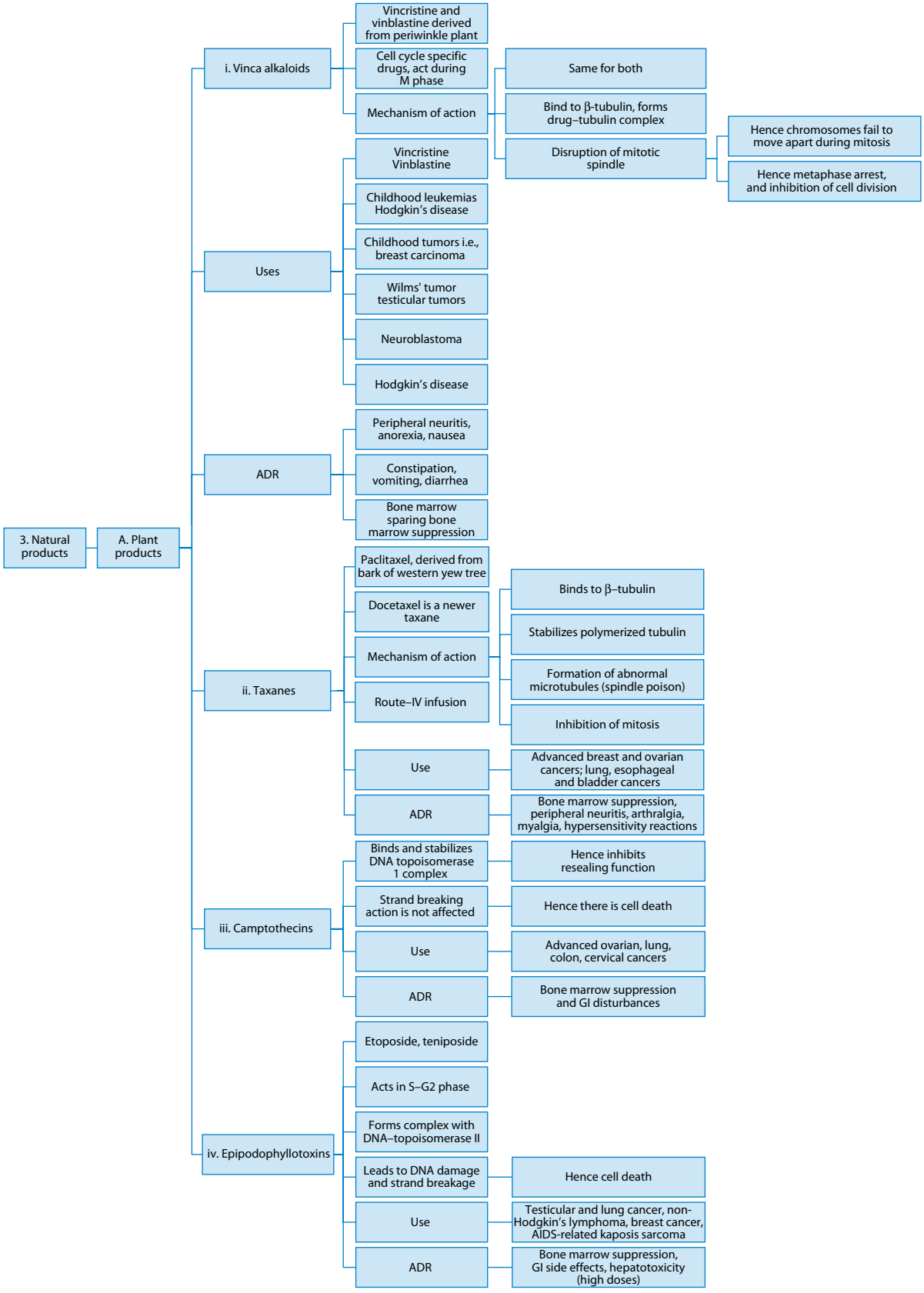
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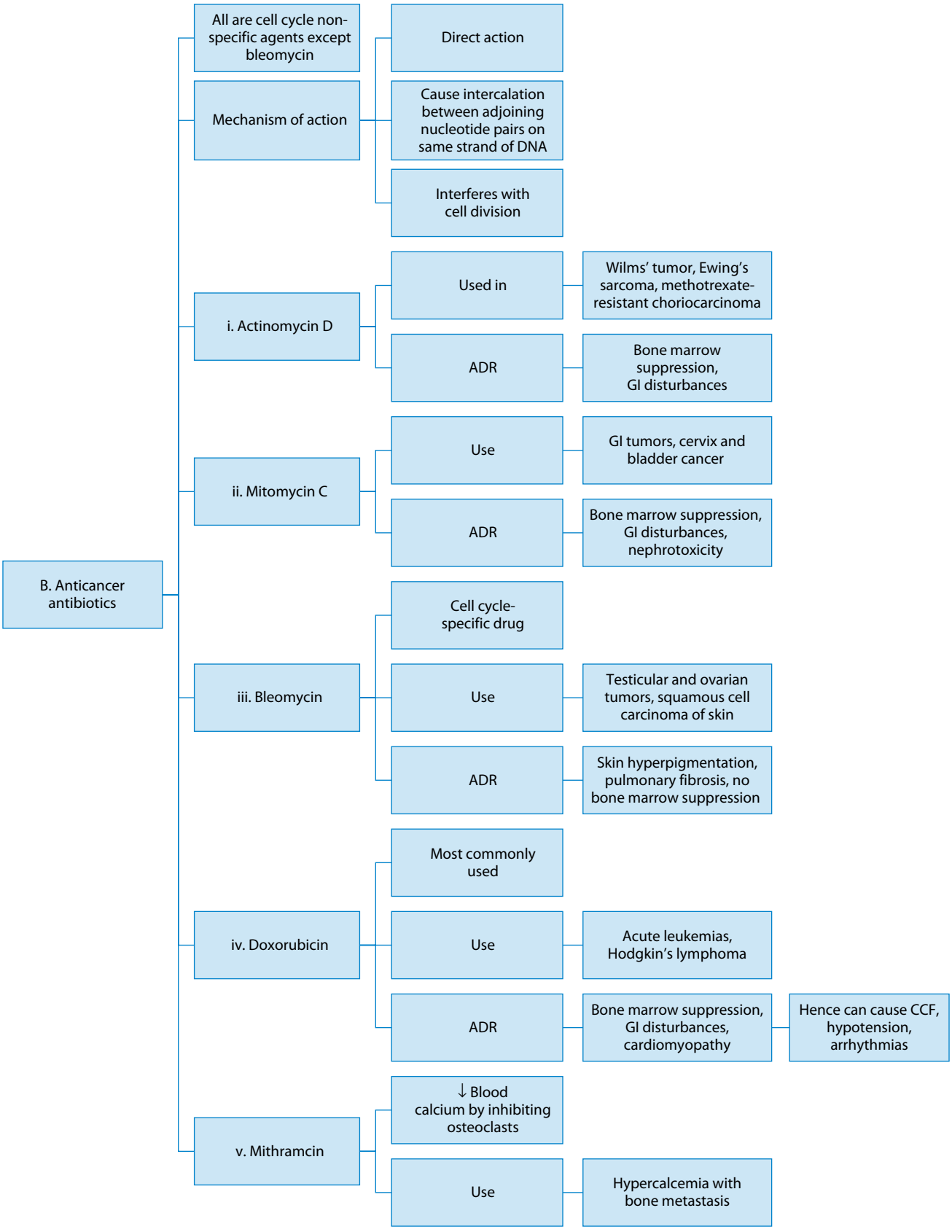
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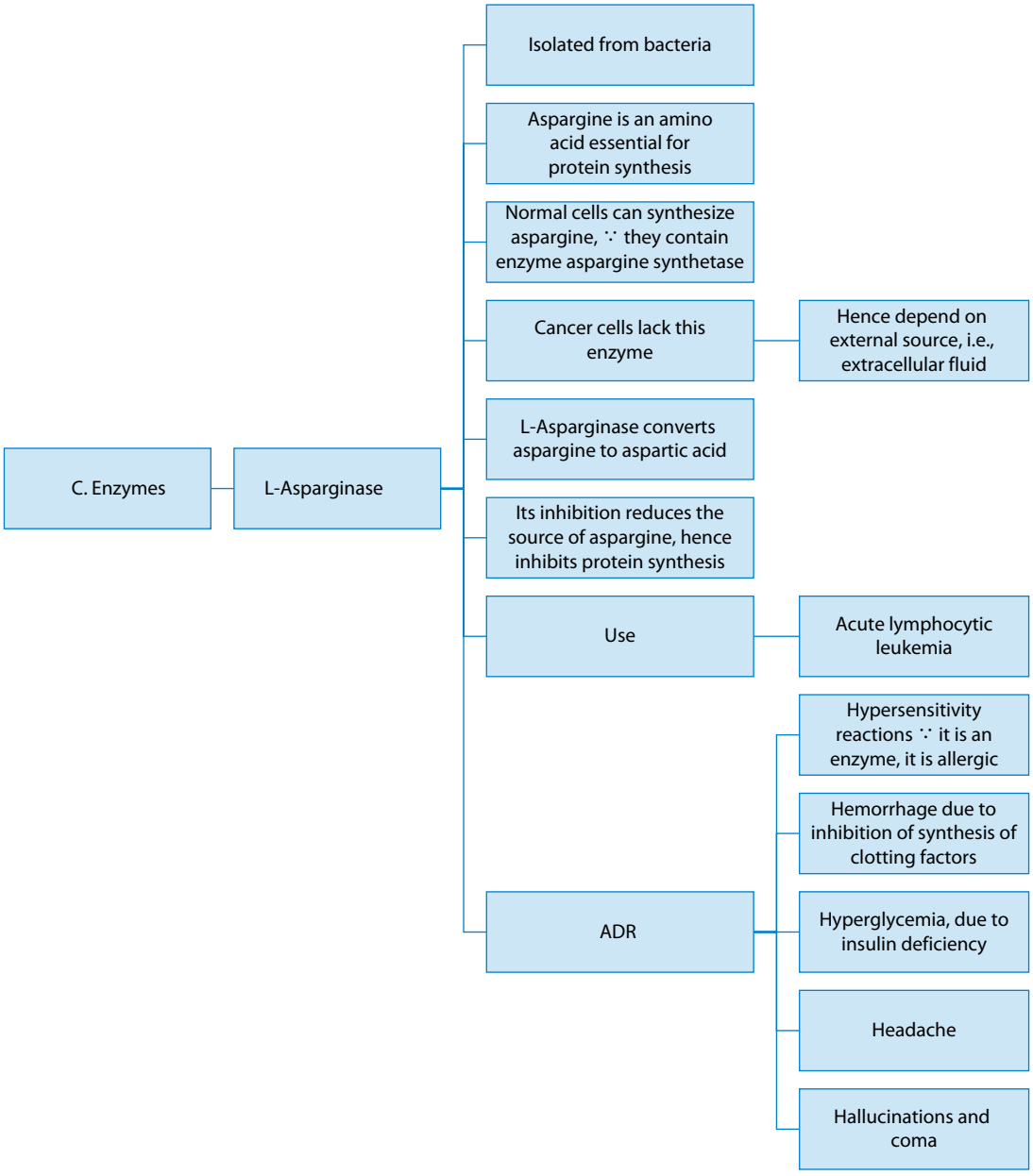
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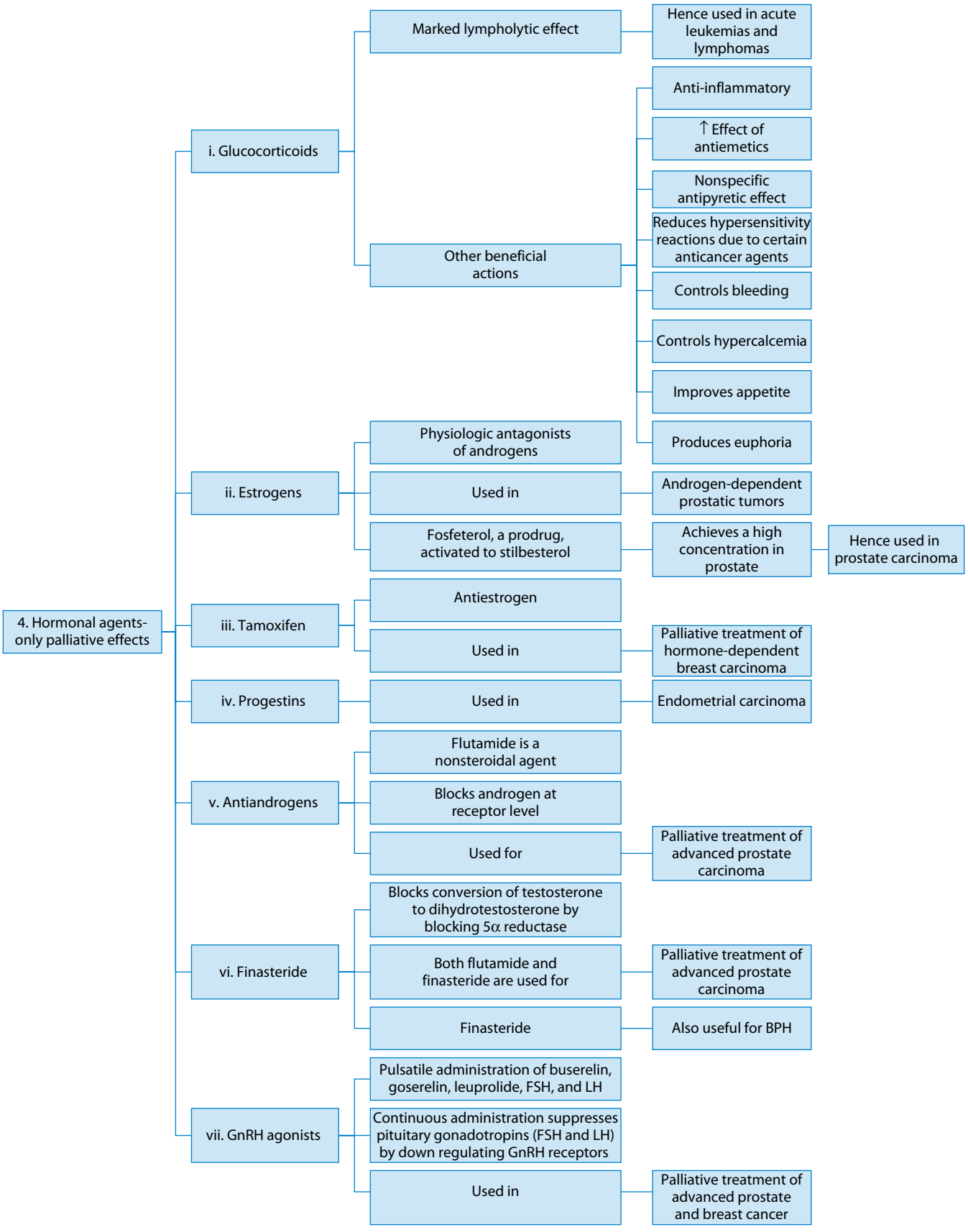
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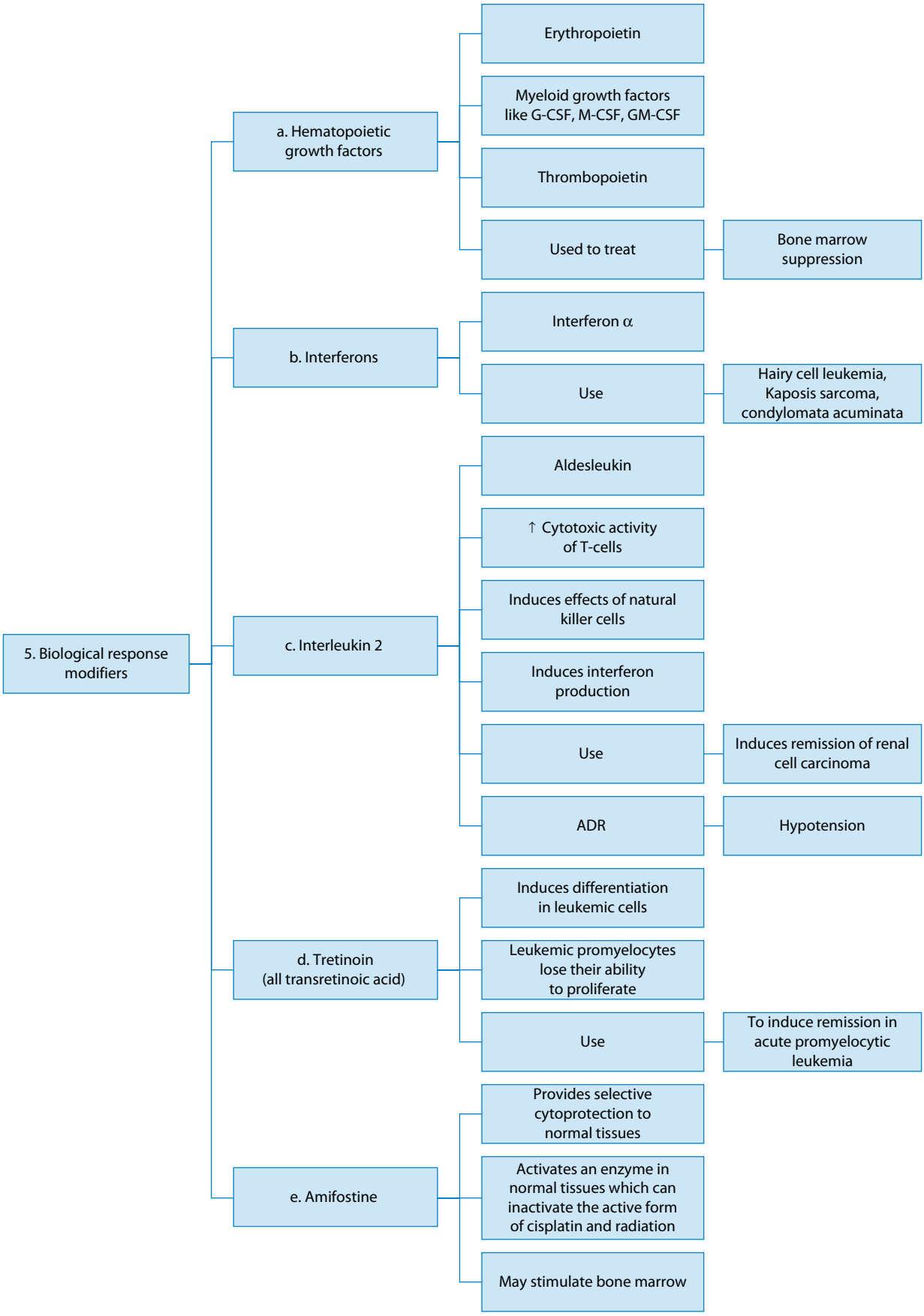
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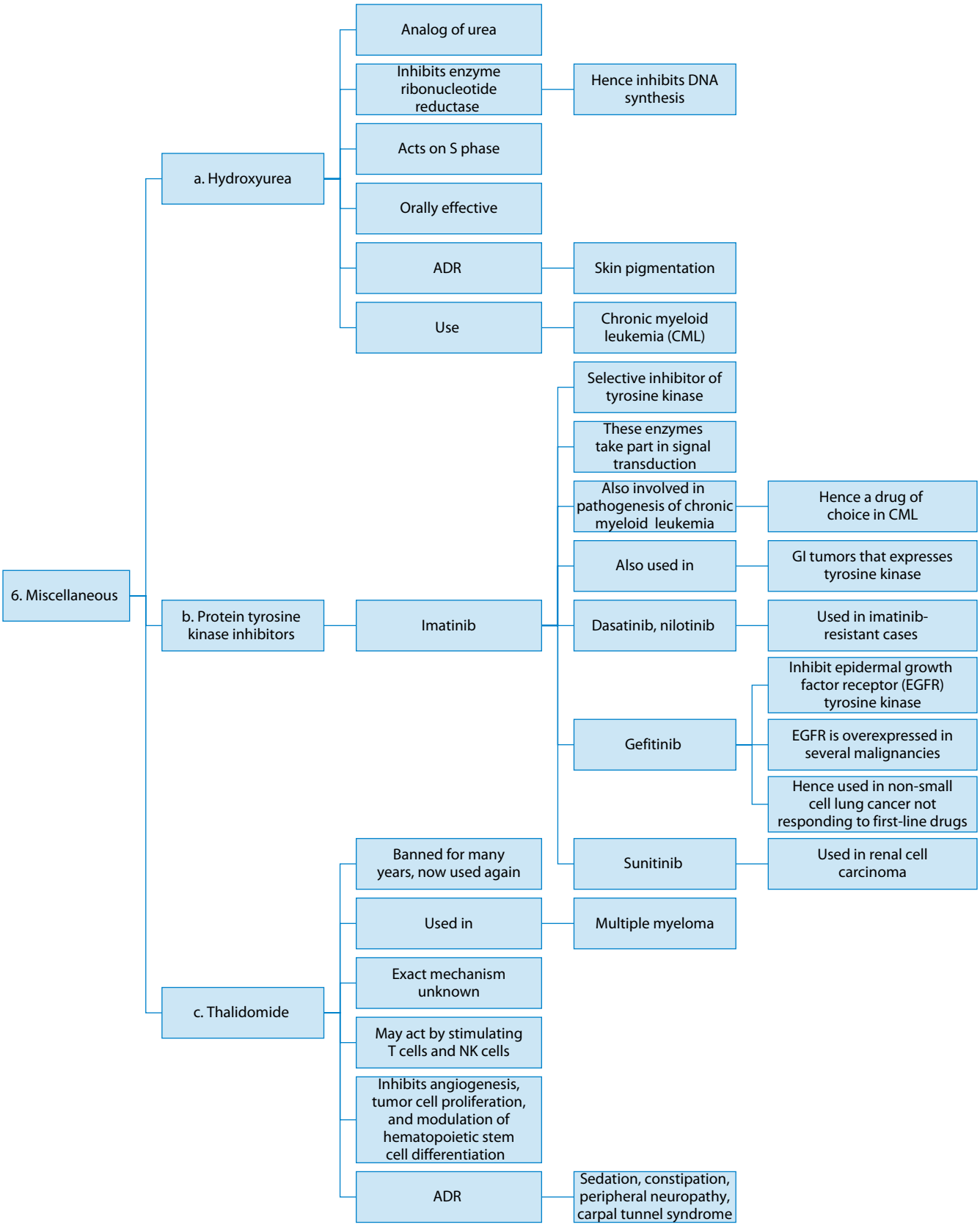
73.12 HORMONAL AGENTS



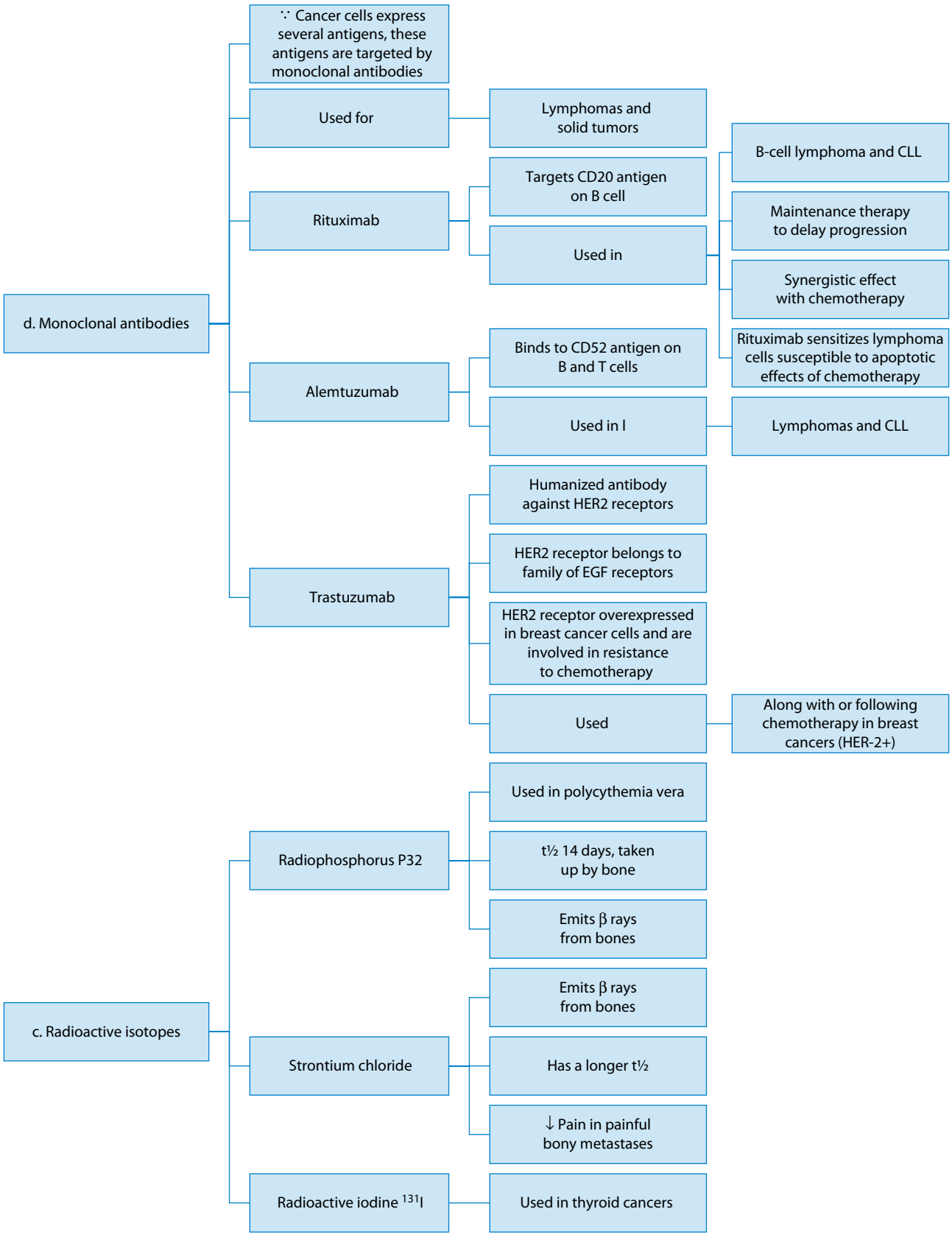
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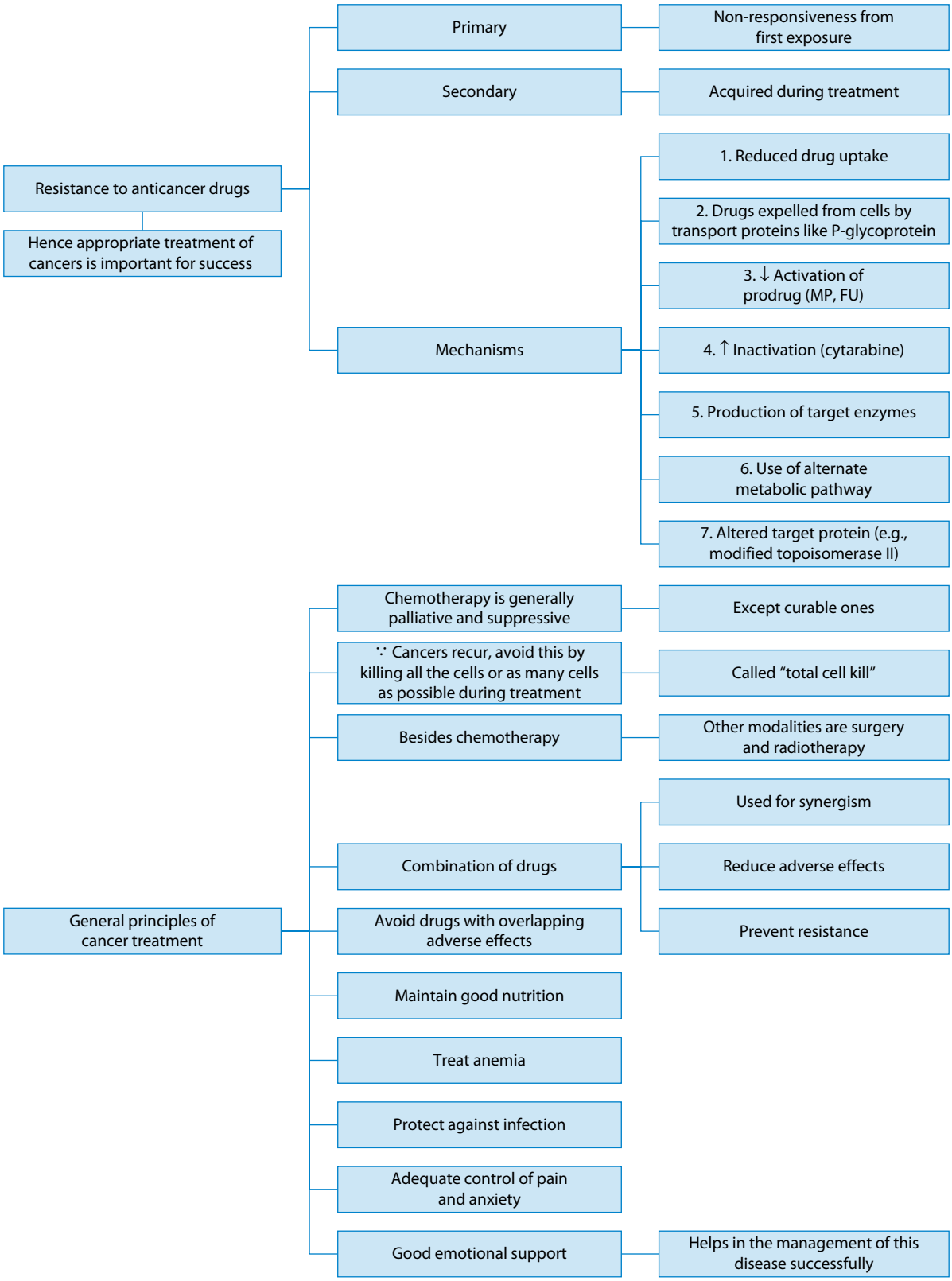
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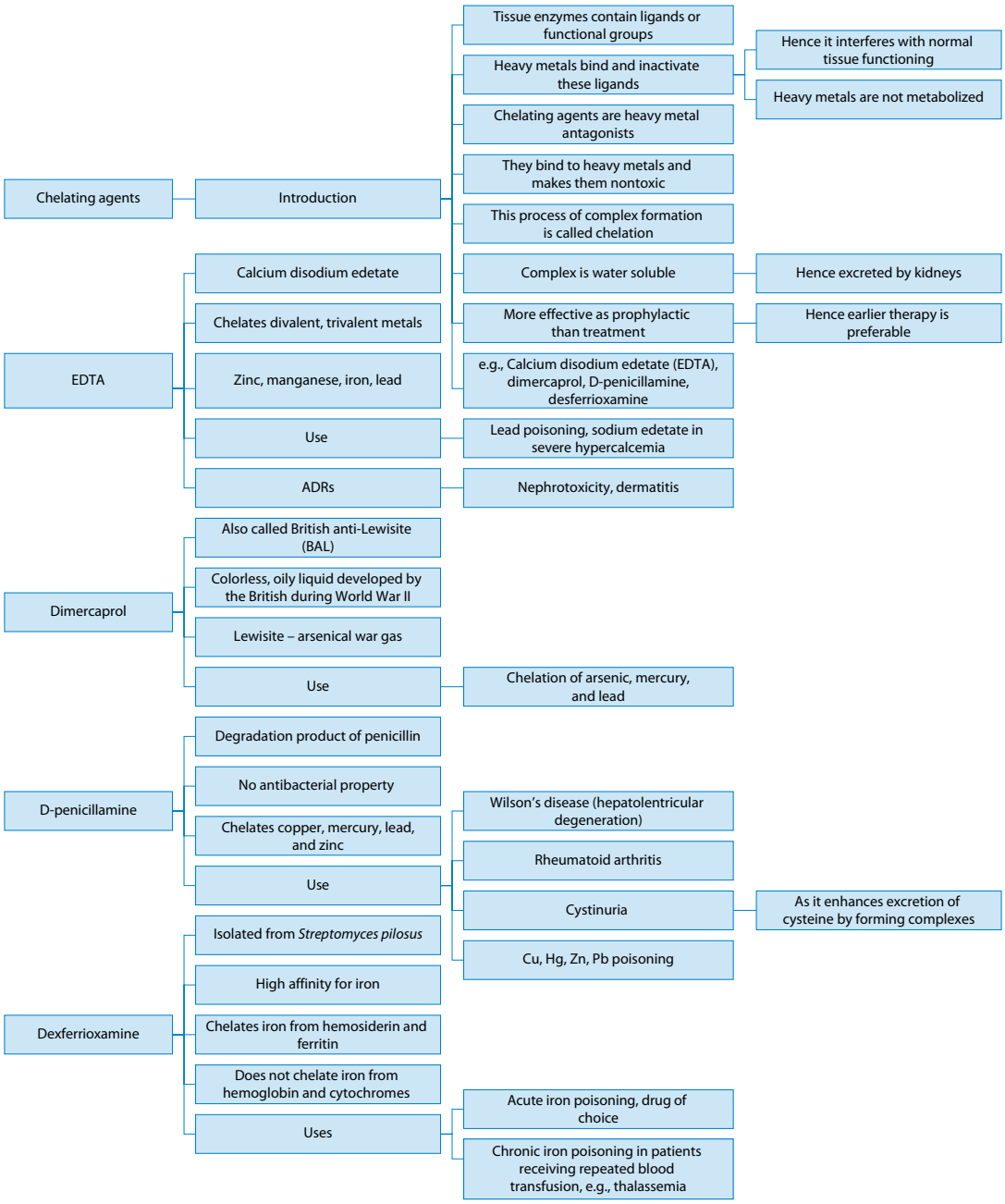
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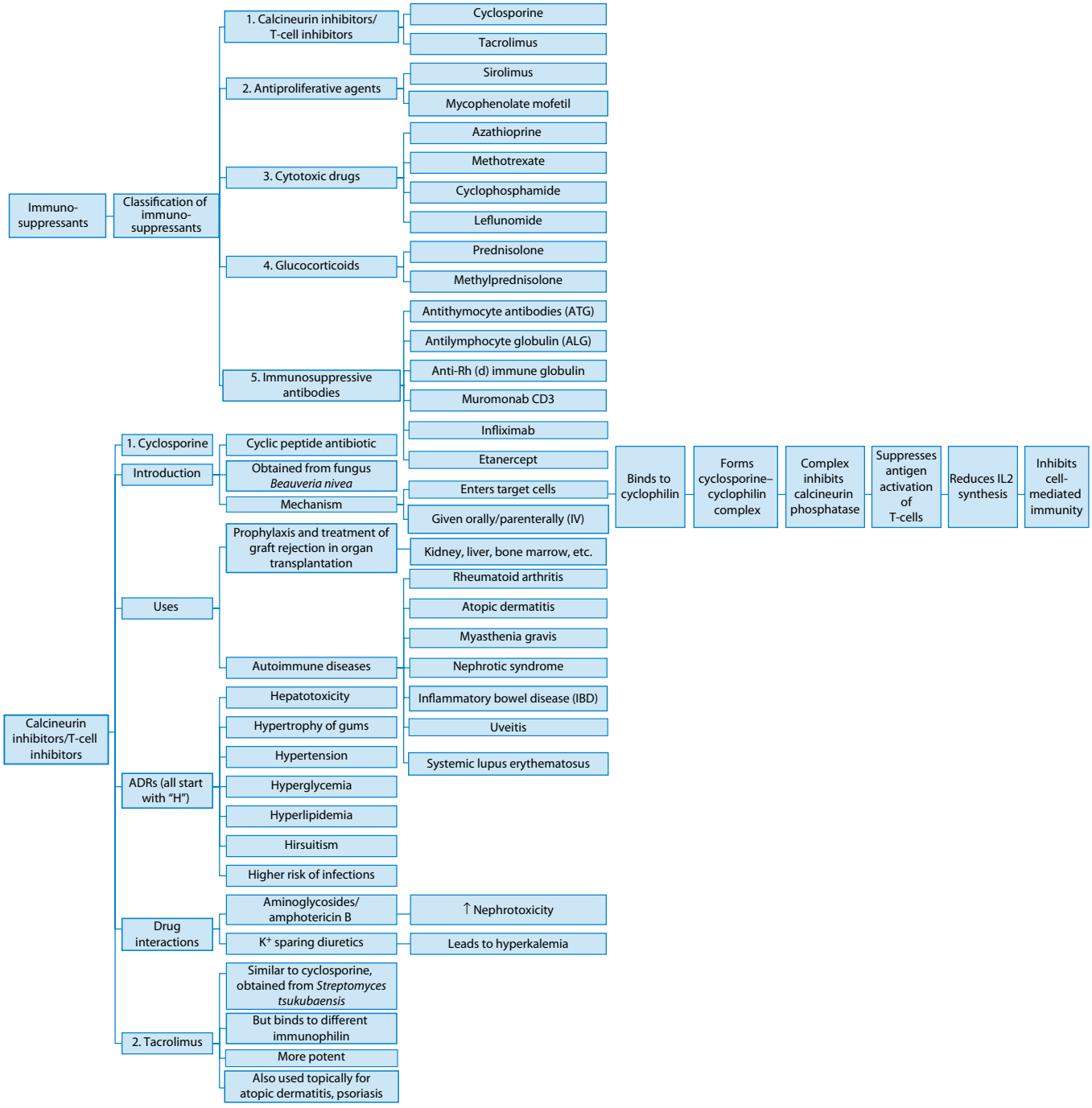
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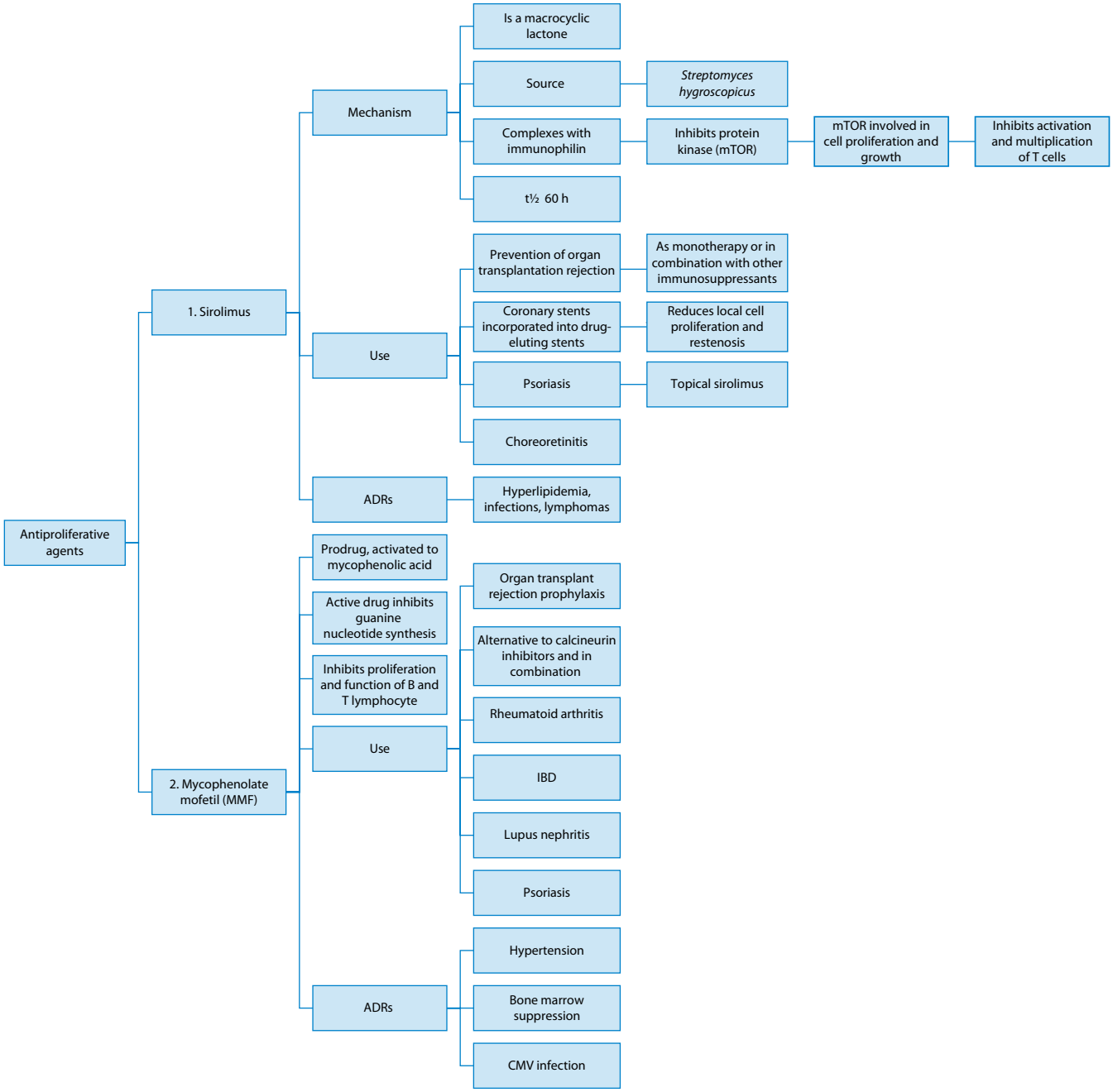
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Immunosuppressants and immunostimulants

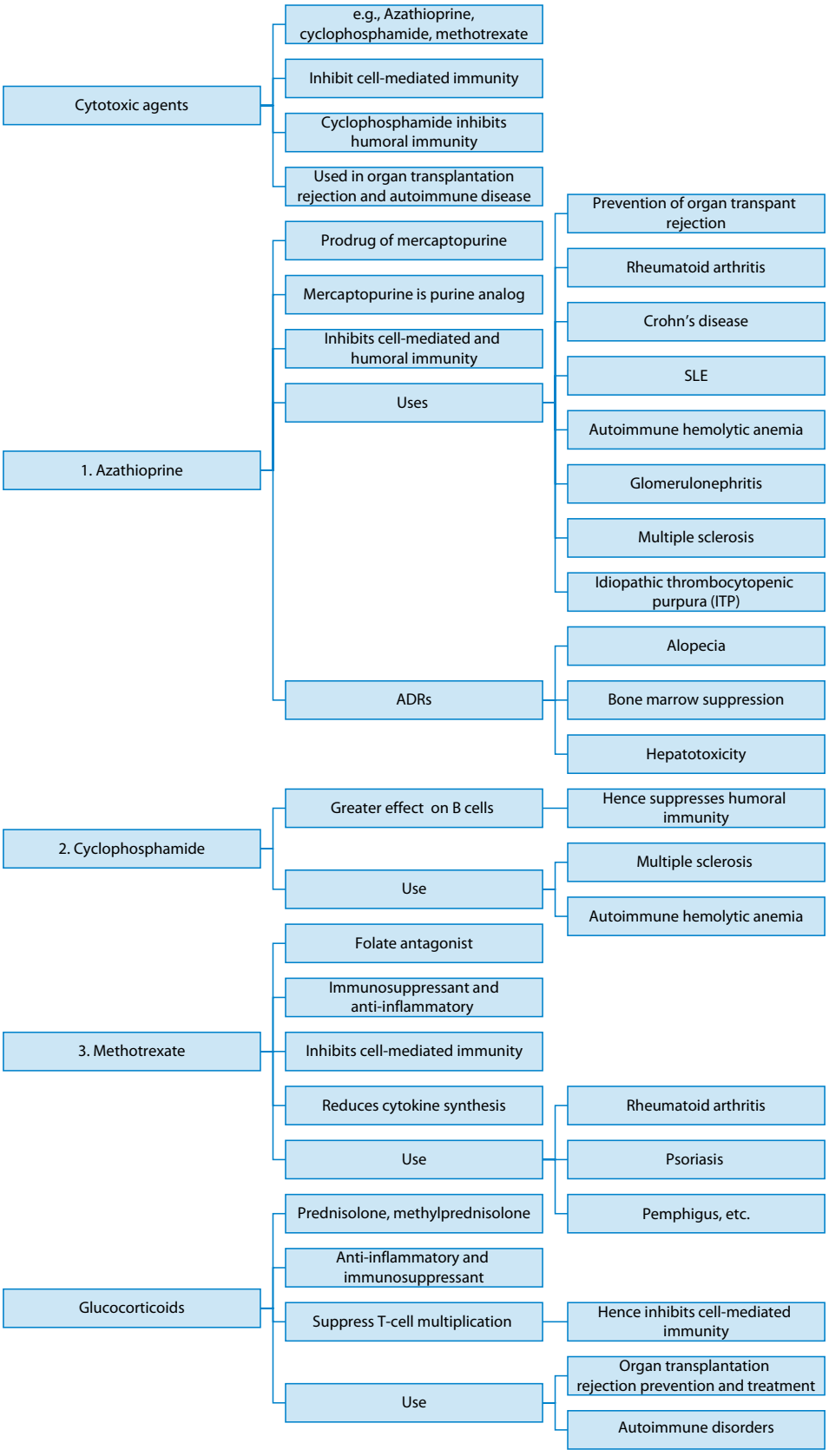
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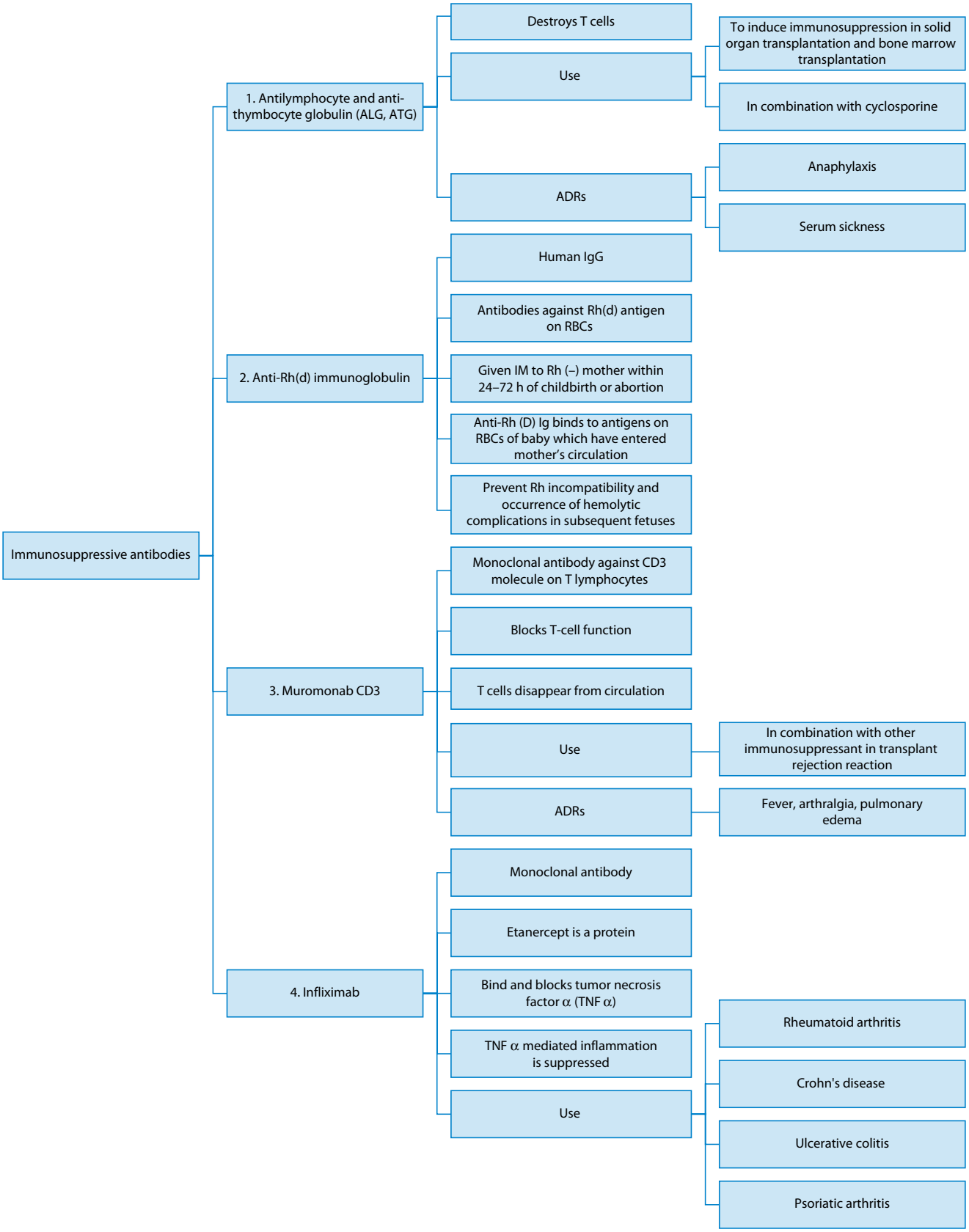
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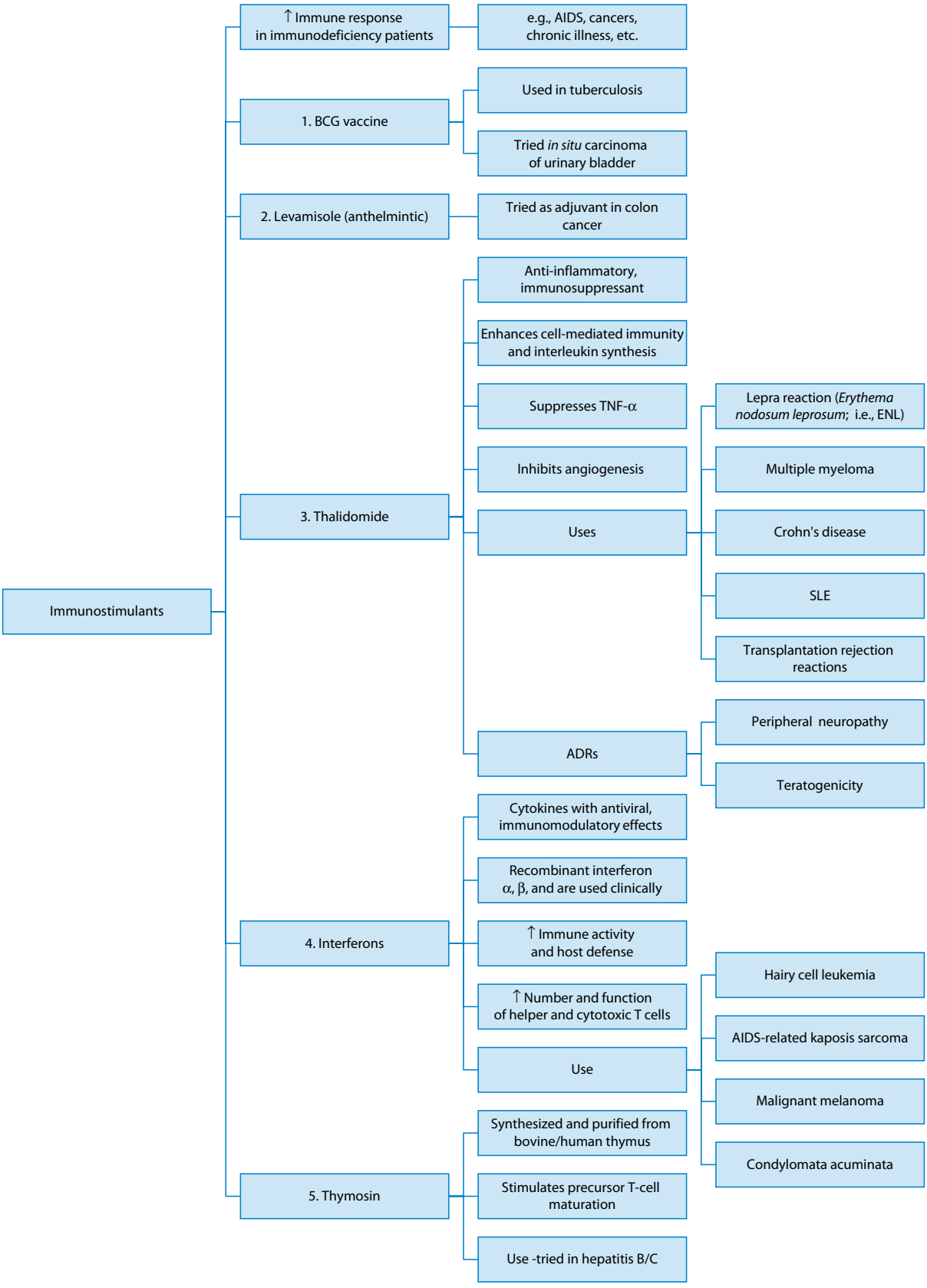
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