Clinician’s Handbook of Oral and Maxillofacial Surgery

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This book is dedicated to Evie Laskin, my wife for over 63 years. She was always my best friend, my biggest supporter, my kindest critic, and my greatest love. —DML
Preface

Why develop a handbook of oral and maxillofacial surgery when there are already numerous texts available that can provide the clinician with extensive information about the various aspects of the specialty? The problem is that, in certain circumstances, textbooks can be too detailed. They are fine when one has the time to seek out the proper text and then sit and read through long chapters containing extensive information about specific problems or procedures; however, when one is faced with an urgent clinical situation and needs a quick answer, textbooks will not readily serve this purpose.

The main intent of this handbook is to provide important information in a concise and easily searchable format from areas of oral and maxillofacial surgery that can present situations in which immediate answers to clinical problems may be necessary. The authors of the various sections have been selected for their clinical expertise and therefore their ability to know the questions that may arise and the information that will answer these questions.

Although designed as a quick-reference source, this handbook can also serve many other functions. Reading the text in advance allows busy practitioners to easily review a considerable amount of clinically significant information. By doing so, they will not only increase their knowledge base, but also establish a familiarity with the text that will make it easier for them to find necessary information in an urgent situation. The book is also a handy compilation of relevant information for trainees in oral and maxillofacial surgery, as well as those in other hospital-based dental specialties, who are just beginning to learn this material. Finally, it is an organized resource for the review of important information pertinent to those preparing for the American Board of Oral and Maxillofacial Surgery.

I would like to express my sincere appreciation and thanks to all of the contributing authors who gave so freely of their time and effort. Without their willingness to share their knowledge and expertise, this book would not have been possible.
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chapter 1

Patient Evaluation

A comprehensive evaluation of a patient’s medical status involves history taking and physical examination. A standard method of performing and recording this examination has evolved. Because this examination is a significant event for most patients, the examiner must display a personal and professional attitude for the proper rapport to develop. Eliciting the patient’s cooperation is an important part of performing a high-quality examination. The examiner must also understand that it is sometimes necessary to compromise completeness in the interest of speed when dealing with an acutely ill or potentially unstable patient.

History

Patient Identification

Before recording the examination, note the patient’s full name, age, date of birth, sex, race, and marital status. When pertinent, record the occupation, place of birth, or religious affiliation; otherwise, these may be considered later as a part of the social history.

Source of Information

Include the sources of information used in compiling the history. Usual sources are the patient, a friend or relative, previous medical charts, or a referral letter from a physician, dentist, or institution. Also include an assessment of the reliability of this information.

Chief Complaint (CC)

Ascertain the principal reason the patient is seeking medical attention. This is
best recorded as a simple phrase stating the complaint and its duration. There is no obligation to use the patient’s own words, although they may be enlightening. A typical chief complaint is written as a list, eg:
- Abdominal pain for 1 month
- Vomiting blood for 1 day

History of Present Illness (HPI)

Question the patient in detail concerning complaints, and record the history in chronologic order. Unlike the chief complaint, the description of the present illness should be written in paragraph form.

Past Medical/Dental History (PMH)

Obtain information in the following areas:

General: Previous illnesses and response to therapy, known active medical problems, previous hospitalizations and surgery

Allergies: Drugs, contrast media, foods, etc

Immunizations: Tetanus, diphtheria, pneumonia, influenza, poliomyelitis, measles, rubella, mumps, varicella, human papilloma virus, hepatitis B, shingles

Trauma: Significant injuries, blood transfusions

Social History

Question the patient about:

Environment: Present and previous residences, occupations, and climatic exposures

Society: Education, religious affiliation, marital status, living arrangements, financial situation, hours of work, sleep, and daily exercise

Drugs: Caffeinated beverages, alcohol, tobacco products, illegal drugs

Sexual History

This is probably the most difficult area about which to obtain information
when conducting a medical history. Of extreme importance is determining whether the patient has engaged in high-risk sexual behavior, which may increase the risk for infectious diseases such as AIDS, hepatitis, or herpes. The interviewer can soften the impact of these delicate issues by asking, “Have you been in contact with anyone at high risk for any of the following: tuberculosis (TB), herpes, hepatitis, AIDS?” If there is a positive response, more specific questions may be asked, ie, sexual preference, kind of contact, etc.

Family History
Inquire about the age, medical problems, and, if applicable, cause of death of parents, spouse, siblings, and children.

Review of Systems
Present each major symptom to the patient and ask if he or she is experiencing or has experienced any of them. Asking about previous symptoms serves as a check on the accuracy and completeness of the patient’s history. Following is a list of the symptoms to be reviewed in this section of the history:

General: Fever, chills, sweating, weakness, fatigue, change in weight

Skin: Rash; itch; pigmentation; bruising; scars; nails—change in shape, brittleness, pitting; hair—excessive loss, change in texture or distribution

Head: Headache, trauma

Eyes: Decreased vision, transient or permanent loss of vision, double vision, spots, pain, redness, tearing, discharge, sensitivity to light, use of glasses or contact lenses, cataracts, most recent examination for glaucoma

Ears: Hearing loss, ringing, dizziness associated with subjective feeling of rotation, pain, discharge

Nose and sinuses: Bleeding, discharge, obstruction, colds, change in sense of smell, pain

Mouth: Pain; lesions; dryness; tongue—soreness, lesions or coating, enlargement; problems with taste; teeth—pain, extractions, most recent dental
examination; gums—bleeding, lesions, discoloration

Throat: Frequent sore throats or tonsillitis, hoarseness, problems with swallowing

Neck: Pain, stiffness, swelling, lumps, limitation of motion, thyroid enlargement

Breasts: Lumps, tenderness, discharge, change in nipple, changes on self-examination

Respiratory system: Cough, sputum, coughing up blood, night sweats, shortness of breath, wheezing, pain with breathing, exposure to TB, most recent TB skin test and chest radiograph

Cardiovascular system: Chest pain, shortness of breath with exertion or when lying down, swelling of legs or feet, pounding in chest, irregular or rapid heartbeat, heart murmur, high blood pressure

Vascular system: Lower extremity pain with exertion, leg cramps, blood clots, varicose veins, coldness or change in color of extremity

Gastrointestinal tract: Poor appetite; food intolerance; regurgitation; chest pain or fullness after eating or when lying down; pain or problems with swallowing; belching; nausea; vomiting; vomiting blood; abdominal pain; diarrhea; constipation; change in bowel habits or in color or character of stool; hemorrhoids; anal itch; gallstones; yellow color of eyes or skin; abdominal swelling; liver disease or hepatitis

Urinary tract: Pain on urination; frequent, urgent, repeated, or nocturnal need to void; bloody urine; loss of urine on clothes or bed; difficulty starting or stopping stream; change in size of stream or color of urine; kidney stones; infections; flank pain

Genitoreproductive system: Female—menses—age of onset, frequency, duration, pain, amount of flow, presence of clots, date of most recent period; menopause—age, associated symptoms, bleeding since menopause; vaginal pain, itch, discharge, and odor; date of most recent pelvic examination and Pap smear; venereal diseases; pregnancy; contraceptive history. Male—penile lesions, discharge, and pain; inability to achieve and maintain erection; scrotal or testicular pain, swelling, and lumps; venereal diseases

Joints: Pain, redness, warmth, swelling, stiffness, limitation of motion,
deformities
Lymph nodes: Enlargement, pain, tenderness
Blood: Anemia, easy bruising or bleeding, blood transfusions
Endocrine system: Thyroid enlargement or malfunction; heat or cold intolerance; change in skin or hair texture; diabetes—excessive eating, drinking, or urinating; change in skin pigmentation or hair distribution
Allergies: Hives, hay fever, allergic rashes, asthma, nonmedication allergies
Nervous system: Seizures, loss of consciousness, fainting, memory loss, confusion, speech impairment; cranial nerve function (see page 10); motor nerves—loss of strength, local paralysis, involuntary movements, loss of coordination; sensory nerves—numbness, tingling, pain
Psychiatric considerations: Depression; anxiety; worries; problems with family, friends, job, or financial matters; difficulty in sleeping

**Physical Examination**

**Methods**

Four methods of examination are available:

**Inspection:** Encompasses observing the total patient and then examining various areas of the body more closely. It is performed with the patient at rest and during certain maneuvers.

**Palpation:** Involves the use of fingertips for touch, the metacarpophalangeal area of the palms for detecting vibration, and the dorsal aspect of the hand for determining temperature.

**Percussion:** Indirect percussion consists of striking the distal interphalangeal joint of the middle finger of one hand with the tip of the middle finger of the other hand. It is used to define the position of certain organs and to analyze the density of tissues.

**Auscultation:** In most instances involves listening through the stethoscope. High-pitched sounds are best heard with the diaphragm and low-pitched sounds with the bellpiece.

Preliminary Statement
Recording of the physical examination should begin with a brief description of the patient’s overall appearance, including references to general state of health and nutrition and to any distress.

Specific Areas

Vital signs: Check blood pressure, pulse, respiratory rate, oxygen saturation, and temperature, and record height and weight.

Skin: Observe turgor, texture (cool, wet), pigmentation, lesions (distribution, configuration, morphology), scars, hair distribution, and nails. Measure turgor by squeezing some skin between the thumb and index finger. When released, the skin should return promptly to its usual place. This is customarily done over the sternum.

Head: Check for size, shape, lumps, depressions, and hair distribution.

Eyes: Examine position, color vision, and visual acuity. Check vision of each eye with a standard pocket or wall Snellen eye chart.

Visual fields: Position yourself facing the patient at a distance of about 50 cm. Have the patient stare into your left eye with his or her right eye; both of you should close your opposite eye. Bring a target object in from the periphery at different angles and have the patient tell you when it comes into view. Compare his or her performance with your own.

Lids: Check for motion, symmetry, swelling, and lesions.

Sclera and conjunctiva: Examine for injection, hemorrhage, or icterus.

Cornea and lens: Check for arcus senilis and for abrasions or opacities.

Pupils: Test for equality, roundness, regularity, reaction to light (direct and consensual), and accommodation. Shine a light obliquely into a pupil. Observe for constriction in the same (direct) and opposite (consensual) pupil. Repeat on the other pupil.
**Extraocular movements:** Have the patient focus on your finger as it traces an *H* in the air.

**Nystagmus:** Observe the eyes in primary position and with some lateral gaze for repetitive movements.

**Strabismus:** Cover, then uncover, each eye while the patient maintains focus on an object in the distance. Movement to maintain focus indicates strabismus.

**Ophthalmoscopic examination:** Check pupil and lens (red reflex, opacities), optic nerve (margins, size of physiologic cup), vessels (size, crossing changes), retina (hemorrhage, exudate), and macula (hemorrhage, exudate). With the lens of the ophthalmoscope set at 0 diopters, darken the room and have the patient stare straight into the distance. Examine the patient’s right eye with your right eye and with your right hand on the ophthalmoscope; reverse the procedure for the left eye. Beginning at a distance of about 30 cm, find the red reflex, and slowly move closer to the patient’s eye. Then adjust the lens setting until the retinal structures come into focus. Find the optic disc. The margins should be distinct, although there may be slight blurring medially. The physiologic cup is a pale area within the disc on its lateral side. Note its size in relation to the disc as a whole (ie, the cup-to-disc ratio). Next, follow the course of each major vessel from its emergence in the disc, through the disc margin (it should remain in focus), then distally as far as possible. An artery and a vein should run together, the vein being larger and a darker red. Compare the relative size of the vessels, and record this as a vein-to-artery ratio (usually about 5:4).

**Ears**

**External:** Check for deformities and if pain occurs with mild pressure or movement.

**Meatus:** Examine for cerumen and any discharge, swelling, redness, masses, or foreign bodies.
**Otoscopic examination:** Assess for light reflex, malleus, and drum perforation, bulging, or retraction. When examining the patient’s ear, grip the otoscope in one hand. With the other hand gently pull the auricle upward and backward. Using the largest speculum the patient’s canal will comfortably accommodate, insert it at a slightly anterior and downward angle. It will eventually be necessary to tilt or rotate the speculum slightly to see as much of the drum and middle ear structures as possible.

**Hearing tests:**
- *Spoken and whispered voice.* Test each ear separately, occluding the other with your or the patient’s finger. Test hearing using high-pitched (whisper, 512-cycle-per-second [cps] tuning fork) and low-pitched (spoken voice) sounds. Note that the 512-cps fork is used during the ear examination and the 125-cps fork is used to test vibration sense.
- *Weber test.* Touch the handle of the vibrating tuning fork against the patient’s forehead. The sound should be heard with equal intensity in both ears.
- *Rinne test.* Touch the handle of the vibrating tuning fork to the patient’s mastoid process. When the patient can no longer hear this sound, hold the vibrating end near the ear. This air-conducted sound should still be audible, although the bone-conducted sound is not.

**Nose:** Check for deformities and patency of nostrils. Note mucosal color or swelling, tenderness, septal deviation or perforation, and turbinate swelling or polyps. Compress each nostril and have the patient breathe through the other side. The internal structures of the nose are best examined with a nasal speculum, but an otoscope with a short, wide nasal attachment can be used.

**Mouth**

*Lips:* Check for color and lesions.

*Teeth:* Examine for missing or loose teeth, caries and fillings,
shape of teeth, tenderness, and malocclusion.

Gums: Note any recession, discoloration, bleeding, swelling, or inflammation.

Buccal mucosa: Assess for color, lesions, and parotid duct opening. Tongue (dorsum and undersurface): Check color, size, papillae, coating, tremors, lesions, and masses.

Palate: Note color, masses, and petechiae.

Tonsils: Examine pillars, size, and presence of exudate.

Pharynx: Check color, exudate, masses, and gag reflex.

Temporomandibular joint: Examine for range of mouth opening, lateral and protrusive movement, tenderness, clicking or crepitation, and condylar deformity.

Neck: Check for position, symmetry, and masses.

Muscles: Examine for hypertrophy, atrophy, and tenderness.

Nodes: Assess size, mobility, and tenderness. Palpate the entire neck for nodes, paying special attention to the area in front of and behind the ear; at the base of the skull; in front of (especially superiorly), under, and behind the sternocleidomastoid muscle; along the underside of the mandible; and behind the head of the clavicle.

Trachea: Check position.

Thyroid gland: Note size, shape, symmetry, nodules, tenderness, and bruits. With the thumb and index finger, begin at the base of the thyroid cartilage and palpate downward until the thyroid isthmus is detected. Ask the patient to swallow and feel the thyroid rise against your fingers.

Jugular venous distention: Observe the external jugular vessels as the patient changes from the sitting to the supine position. Stop at the point where the venous column is visible a few centimeters
above the clavicle. The internal jugular pulse is even more reliable, but is clinically more difficult to evaluate.

Breasts: Assess for size, symmetry, venous pattern, tenderness, and masses. With the patient sitting, observe the breasts for retraction or asymmetry, both at rest and while the patient performs specific maneuvers. Have the patient raise her arms above her head, then have her push her hands firmly against her hips and lean forward. Have the patient assume the supine position with the hand on the same side as the breast to be examined behind the head. Examine each quadrant and the tail of the breast into the axilla by pressing the breast against the chest wall with the tips of the fingers. Check the nipple and areola for color, lesions, retraction, discharge, and fissures. Squeeze the nipples and observe for discharge.

Axillae: Note any lesions, lumps, or nodes. Examine the patient’s axilla, controlling the patient’s arm position with one hand on the patient’s wrist. Push the other hand deep into the axilla and then let the patient’s arm drop lightly over your hand. Use your fingertips to compress the axillary contents against the ribs.

Thorax: Assess for chest size and shape, rib deformities, and tenderness.

Lungs

*Inspection*: Check respiratory rate and rhythm, respiratory excursions, retraction of interspaces with inspiration, and use of accessory muscles of respiration.

*Palpation*: Examine for tactile fremitus. The patient should be in the sitting position. Because vibration is being measured, use the metacarpophalangeal area of the palm. Ask the patient to repeat the word ninety-nine and compare symmetric areas of the chest.

*Percussion*: Check lung resonance, lower borders, and diaphragmatic movement. Percuss symmetric areas of the chest and compare the sounds.

*Auscultation*
- *Breath sounds*. Listen to the breath sounds as the patient
breathes deeply through the mouth. Note intensity and inspiratory/expiratory ratio. The inspiratory phase is normally longer than the expiratory phase.

- *Adventitious sounds.* Note location, timing, pitch, and persistence after coughing.
- *Voice sounds.* Note loudness, distinctness, and symmetry.

**Heart**

*Inspection:* Check for apical impulse and other impulses. With the patient in the supine position, the apical impulse is visible in about one-fifth of the normal population and is usually located about 1 cm medial to the midclavicular line on the left side in the fifth intercostal space.

*Palpation:* Note apical impulse, abnormal impulses, palpable heart sounds or rubs, and thrills. Use the palms to palpate over the heart because findings are being sought more through vibration than through touch. Palpate over visible impulses first and then palpate over the remainder of the pericardium. The normal location of the apical impulse, described earlier, is often palpable despite not being visible in the majority of people.

*Auscultation:* Auscultation of the heart is also usually performed with the patient in the supine position; changing to other positions brings out specific abnormalities, such as sitting and leaning forward to check for aortic disorders and assuming the left lateral decubitus position for mitral disorders. Check for rate, rhythm, heart sounds, gallops, murmurs, and rubs. Listen first over the apex for rate and rhythm. Then sequentially auscultate over the entire pericardium for normal heart sounds, extra heart sounds, and murmurs. Clearly identify the heart sounds before pursuing other sounds. Distinguish between extra heart sounds (S3, S4, click) and murmurs or rubs. Intensity is graded on a scale of 1 to 6. The subjectivity of this scale can be minimized by following these guidelines:
- **Grade 1.** Very faint and heard only when paying close attention
- **Grade 2.** Faint, but unmistakably present
- **Grade 3.** Clearly louder than grade 2, but not associated with a thrill
- **Grade 4.** Loud and associated with a thrill
- **Grade 5.** Very loud but requiring stethoscope partly on the chest to be heard
- **Grade 6.** Able to be heard with the stethoscope off the chest

Abdomen

*Inspection:* Check for contour, pulsations, venous dilation, peristalsis, scars, and masses. With the patient in the supine position, inspect the abdomen from the side, your head at a level only slightly above that of the abdominal surface.

*Auscultation:* Assess for bowel sounds, bruits, and friction rubs. Note that in the abdominal examination, auscultation should precede palpation and percussion.

*Palpation:* Examine for muscle spasm, tenderness, normal organs, organo-megaly, masses, and aortic pulsation. Using the fingertips of one hand, press lightly over all regions of the abdomen. This will help relax the anxious patient and will localize tender areas. Then repeat the examination using deeper palpation. Examine from lower to upper abdomen to avoid missing the liver or spleen edge. Examine the left side first unless there is a history of pain in the area or it is tender on light palpation. The quadrant in which symptoms exist should be examined last; if palpation there causes pain, it will be difficult for the patient to relax for palpation of the other abdominal areas. If tenderness is elicited, check for rebound tenderness by pushing in slowly, then quickly letting go.

*Percussion:* Estimate liver size. Note that liver palpation does not
provide any information regarding the location of the upper border of the liver. Therefore, percussion is essential to estimate liver size.

Male genitalia

**Penis:** Examine for circumcision status, lesions, urethral orifice stricture, and any discharge. Be sure to retract foreskin if the patient is uncircumcised. Squeeze the tip of the penis to try to express discharge.

**Scrotum:** Check for skin lesions, testicular size, tenderness or lumps, non-testicular masses, and hernia. Examine the testes simultaneously between the thumb and index finger; use both hands to examine each testicle individually. The normal adult testicle measures 5 cm in diameter. Have the patient stand and strain. Observe the inguinal areas for any bulges.

Rectal area: Check for hemorrhoids, lesions, sphincter tone, anal and rectal wall masses, tenderness and induration, stool color, and occult blood. Also examine prostate for size, consistency, or lumps. The rectal examination is usually performed with the patient in the left lateral decubitus position with the legs flexed. Insert the gloved index finger into the patient’s rectum with the flexor surface facing posteriorly. Examine 90 degrees in each direction and then rotate your hand so that the flexor surface of the finger faces anteriorly. Then examine that side of the bowel wall. After examining the anterior bowel wall, focus your attention on the prostate gland. The median furrow should be identified; it separates the lateral lobes. The finger should be able to identify the uppermost extent of the prostate. Palpate carefully the entire surface of the gland for irregularities.

Peripheral vascular system: Check for the presence of carotid, radial, femoral, dorsalis pedis, and posterior tibial pulses. Examine their equality, contour, and rhythm, and note any volume changes or bruits. Grading the strength of pulses is done by designating them normal, diminished, or absent. When examining the carotid pulse in the lower half of the neck, do not stimulate the
carotid sinus or palpate the carotid arteries simultaneously.

Extremities: Check for venous pattern, lesions, hair distribution, condition of nails, redness, cyanosis, edema, temperature, and nodes. Check the patient’s lower extremities for edema by pressing the thumb firmly over the dorsum of the foot and over the tibia at mid-ankle. If edema is found, check at higher levels as well. If the patient has been supine, check also over the sacrum. Estimate the depth of pitting in millimeters.

Spine: Examine for kyphosis, scoliosis, and lordosis; check for range of motion, spinal process tenderness, and local muscle spasm. Test cervical range of motion by observing the patient’s ability to extend his or her neck and to touch ear to shoulder, chin to shoulder, and chin to chest.

Peripheral joints: Check for redness, swelling, active range of motion, deformity, local muscle atrophy, tenderness, passive range of motion, warmth, and crepitation. Examine each joint individually.

Nervous system

**Cranial nerves:** Note that the evaluation of the cranial nerves involves many maneuvers already described. Evaluate these nerves by assessing the following functions related to the separate nerves:

I. Smell.
II. Visual acuity, color vision, visual fields.
III, IV, VI. Extraocular movements, pupil reactions.
V. Motor: Masseter muscle strength (have the patient bite down while you palpate the masseter muscle for strength and symmetry), lip tremor.
V. Sensory: Sensation over face, corneal reflex (touching the cornea with a piece of cotton should cause blinking).
VII. Motor: Facial asymmetry, tics (have the patient perform several facial movements, such as exposing the teeth, puffing the cheeks, closing the eyes tightly, and raising the eyebrows, and check for asymmetry).
VII. Sensory: Taste.
VIII. Cochlear: Hearing.
VIII. Vestibular: Nystagmus.
IX, X. Uvula movement, gag reflex.
XI. Trapezius and sternocleidomastoid strength (have the patient force his or her head from the side toward the midline against the resistance of your hand). Note the force generated and use your other hand to feel the tension in the sternocleidomastoid muscle.
XII. Tongue movement, tremor, atrophy.

Motor nerves: Check for muscle atrophy, symmetry, and abnormal movements. Assess strength of proximal and distal muscle groups of upper and lower extremities. Screen for proximal muscle weakness by having the patient hold the arm straight out laterally from the shoulder while you attempt to push the arm down.

Sensory nerves: Assess superficial pain, touch, proprioception, vibration, and stereognosis.
  - **Superficial pain.** Test ability to distinguish the sharp from the dull end of a safety pin or needle in symmetric areas.
  - **Touch.** Check ability to detect stimulation with cotton or gauze.
  - **Proprioception.** Holding a finger or toe by its sides (to avoid stimulating pressure sensation), see if the patient can differentiate the flexed from the extended position.
  - **Vibration.** Using a 125-cps tuning fork, compare symmetry and sensation (by comparison with yourself) of vibratory sense over superficial bones.
  - **Stereognosis.** Place common, small objects in the patient’s hand (coin, keys) and have him or her identify them by touch alone.

Deep tendon reflexes: Routine testing should include biceps, triceps, knee, and ankle reflexes. Reflexes can be arbitrarily graded as: 0 = no response; 1 = decreased; 2 = normal; 3 = increased; 4 = clonus.

Superficial reflexes: Check abdominal and cremasteric (male)
reflexes. *Abdominal reflex*: With the patient in the supine position, stroke his or her abdomen with a moderately sharp object, such as a key or a reflex hammer handle, from the lateral side toward the midline. There should normally be abdominal muscle contraction on the side of the stimulation, with movement of the umbilicus toward the stimulus.

*Pathologic reflexes*: With the patient in the supine position, test the Babinski sign using a moderately sharp stimulus. Lightly stroke the lateral aspect of the sole of the foot vertically from the heel to the base of the toes. As you approach the toes, change the course of the stimulation to a medially directed path along the base of the toes toward the great toe. The normal response is plantar flexion of the toes. Be careful not to confuse an abnormal response (dorsiflexion of the great toe, fanning of the other toes, dorsiflexion of the ankle, flexion of the knee and thigh) with a simple withdrawal response.

*Brainstem reflexes*: Test with the cranial nerves.

*Coordination*: Observe the patient’s gait and ability to perform rapidly alternating movements.

Mental status: Assess by noting the following:

*Appearance*: Dress, grooming

*Motor behavior*: Facial expression, posture, poise

*Verbal behavior*: Voice (pitch, intensity), relevance, vocabulary

*Mood*: Emotion, tones

*Thought processes*: Delusions, hallucinations

*Cognitive functions*: Orientation (person, place, time); memory (immediate, distant); general knowledge (name of current president, large cities); abstraction (proverb interpretation, similarities); calculations (serial sevens); judgment (test questions)
Assessment and Plan
Information gathered from the complete history and physical examination is analyzed to develop working hypotheses. Problem solving, using the scientific method, is begun at the time of the initial patient interview. Once symptoms are recorded and the patient is examined, several diagnostic possibilities may occur, although one or two might seem most likely. The most common diseases/diagnoses are considered first. The assessment is the formulation of a working diagnosis using cognitive and intellectual skills. Clinical judgment is used to weigh diagnostic possibilities in order to arrive at the most likely conclusion. When many diagnoses explain the symptoms and events of the chief complaint or present illness, a differential diagnosis is made. Once all of the diagnostic possibilities are recorded, a plan for each is initiated. These plans may include additional laboratory and other studies, consultations, and medications.
Hospital Protocol and Procedure

The role of today’s hospitals in health care delivery covers a wide range of options from ambulatory care to tertiary care. Community hospitals, highly specialized care facilities, level I trauma centers, and academic health centers provide a diversity of institutional orientation that can be confusing. The common thread that ties them all together is the goal of optimal quality patient care and a set of standards that provides guidance in achieving that goal. Hospital protocol is a system intended to standardize the processing of patient care. It covers the how, where, when, and by whom questions that relate to that processing.

The Joint Commission (formerly the Joint Commission on Accreditation of Healthcare Organizations) was formed in 1947 to establish standards of care for hospitals. Its current membership includes the American Medical Association, American Dental Association, American Hospital Association, American College of Surgeons, and American College of Physicians. It has become the principal accrediting body for hospitals and is a major force for change in the standards of care for patient care delivery systems.

Organization of Hospitals

Governing Body

Each hospital has a governance system that includes a group of individuals (e.g., board of directors) who are vested with ultimate responsibility for the hospital, including the care provided. This group has the final authority for hospital decisions. The group’s membership often includes individuals who represent the local community, health care providers, and investors, if applicable.

The governing body delegates responsibilities for day-to-day operations
and patient care issues to those actively involved in such areas. They retain final authority for decisions that are of major importance or represent substantial changes in policy for the hospital.

Administrative Staff

Hospital administration is responsible for the efficient operation of the hospital. Areas of authority include physical plant, personnel, fiscal affairs, and community relations.

There is a chief administrative officer (e.g., hospital director) who is in charge of hospital operations and is responsible to the governing body.

The nursing staff is part of hospital administration. There is a director of nursing who is responsible for the operation of the nursing service, including the management of nursing personnel. The director of nursing is responsible for the nursing care that is provided.

Medical Staff

The medical staff is composed of health care providers who have been granted the privilege to treat patients in that particular hospital. This includes physicians, dentists, and other health care providers duly licensed to provide care independently in their particular area. Categories of the medical staff include active and consulting status.

Active status: Includes those practitioners with appropriate state licenses who are approved for medical staff membership and who are eligible to vote, hold office, and serve on standing committees. They are required to attend medical staff meetings.

Consulting status: Includes those practitioners, duly licensed as health care providers and approved by the medical staff, who provide professional services on a consultative basis. They are not eligible to vote, hold office, or serve on standing committees. They are not required to attend meetings of the medical staff.

Other categories of medical staff membership may be established. Examples include honorary, associate, fellow, courtesy, and voluntary. Each category must be defined, including responsibilities and restrictions.
Bylaws, Rules, and Regulations

Each hospital is required to have bylaws, rules, and regulations that define the process by which the hospital conducts its business. These bylaws, rules, and regulations must be approved by the governing body, published, and available for review. They must include definitions; responsibilities; due process policy; appeals process; mechanisms for change; and other policies, procedures, and processes necessary for the hospital to achieve its mission of health care delivery.

Standing Committees

Standing committees are established under the bylaws, rules, and regulations and are responsible to the medical staff for review, monitoring, and recommendations in their respective areas of activity. The committees vary from institution to institution but usually include the following:

Executive committee: Implements actions of the medical staff (made up of elected officers)

Credentials committee: Reviews applications for medical staff membership and requests for clinical privileges

Medical records review committee: Reviews medical records to ensure appropriate documentation of health care

Infection control committee: Conducts surveillance of hospital-based infection potential and reviews and analyzes actual infections

Operating room committee: Supervises the proper utilization of and enforces the rules and regulations governing the operating rooms

Transfusion committee: Reviews blood and blood products usage

Attending Staff and Clinical Privileges

Attending Staff

This staff consists of those members who have been granted clinical privileges in accordance with the established procedures of the medical staff, as delineated by its bylaws, rules, and regulations.
Clinical Privileges

Privileges are based in part on licensure, specific training, experience, and demonstrated competence. Other reasonable criteria, uniformly applied to all applicants, may be required.

- The bylaws, rules, and regulations clearly define the application process by which attending staff are granted clinical privileges. The process and the criteria for the granting of clinical privileges are established by the medical staff and are uniformly applied to all those seeking privileges. An appeals process is available should the applicant be dissatisfied with the outcome of the privilege process.
- Patient care services that are provided must be within the scope of privileges granted to the attending staff providing the services. Limitations, if any, may also be incorporated into the privileges granted.

Medical Records

Purpose

The medical record serves as documentation of the course of the patient’s care, including evaluation, diagnosis, treatment, and observed responses. It facilitates communication between various health care personnel involved in that patient’s care and is considered to be a legal document that may be submitted as evidence in case of litigation.

Format

The medical staff determines the format and forms of the medical record. The format must include:
- Patient identification
- Comprehensive health history
- Physical examination findings
- Written orders for diagnostic and therapeutic studies
- Informed consent documentation
- Progress notes describing patient’s status and response to therapy
- Results of studies (eg, laboratory, radiographic)
- Summary of evaluation, treatment, patient’s response to treatment, and
current status

The 2009 federal economic stimulus package included incentives, in the form of increased Medicare reimbursement rates, for hospitals to adopt the use of electronic medical records (EMR). However, many factors have slowed its implementation, including the difficulty of facilitating communication among various information systems and software applications, assuring the confidentiality of individual patients’ information, and incorporating written records into the EMR; the cost of implementation; and legal considerations.

Rounds
Hospitalized Patients

Hospitalized patients should be seen on a daily basis to evaluate their progress and current status. Although the frequency and time of day may vary with the circumstances of each patient and the attending staff, patients are usually seen twice a day. Because of other responsibilities and operating schedules, rounds usually occur early in the morning and late in the afternoon or early evening.

House Staff

The house staff is expected to be knowledgeable about the patients. This includes the health history, diagnosis, procedures planned or performed, and the current status of the patient. All laboratory values, radiograph results, and current medications should be known. The house officer should be able to report the patient’s symptoms, vital signs, and overall progress. This information should be available to the attending staff and provides the basis for the day’s progress note, which is recorded in the patient’s medical record.

Admitting Note
Purpose

The admitting note is the initial entry in the progress note section of the patient record. It is a relatively brief synopsis of the circumstances justifying admission, what is thought to be the patient’s problem, and what is planned for the patient during the period of hospitalization.
Reason for admission

Diagnoses

Plans for treatment

Example Admitting note

“This 48-year-old cauc. man was involved in an altercation this early am, sustaining facial trauma without loss of consciousness. Brought to UCSD by ambulance and admitted to Oral & Maxillofacial Surgery Service for evaluation, repair of injuries. Admitting diagnoses include (L) ZMC Fx; nasal Fx; mandible Fxs, (R) angle, (L) parasymphysis.

Plan to take Pt to OR tomorrow am for ORIF (L) ZMC, (R) mand. angle/Fx; closed reduction, nasal Fx, (L) parasymphysis Fx; under gen. anes. Discussed Dx, Tx plan, potential complications & Pt and family.”

Admitting Orders

Purpose

The admitting orders are specific instructions to nursing personnel to admit the patient to the hospital, provide for medications, and begin the inpatient portion of diagnosis and/or therapy. It is helpful to have a standardized format that can be modified to the specific needs of each patient.

Content

• Admitting doctor, service
• Diagnosis
• Condition of patient
Example Admitting orders

Admit to: Oral and Maxillofacial Surgery Service

Attending: Dr M.W. Jones

Dx: Multiple facial fractures

Condition: Stable

Vital signs: Routine Q shift

Ambulation: Ad lib

Nursing: 1. NPO & MN
   2. Void on call to OR
   3. Shower, shave, shampoo this PM
   4. Start IV @ 18 g Jelco this evening. Run D5–1/2 NS @ KCl 20 mEq/L at 125 mL/h
   5. Ice packs to face
   6. Op permit for open reduction of facial fx's

Diet: Full liquid, as tolerated, until NPO order

Labs: 1. Astra 7
    2. CBC & diff.
    3. UA
    4. PT, PTT
    5. ECG
    6. PA & Lat. CXR
Consultation

Purpose

The consultation is a formal request for input from another doctor or support service. It permits the admitting doctor to gain advice and/or active participation from that other source regarding selected aspects of the patient’s evaluation and management. Responses are expected promptly (within 24 hours).

Content

The consultation may be initiated by direct contact, but is best managed by a hospital form that includes the patient’s name and number; the working diagnosis; the time, date, and name of the requesting doctor; and the specific request (eg, evaluate cardiac status, manage diabetic care, anesthetic considerations). The document has a space for written comments by the individual or service consulted.

Preoperative Note

Purpose

The preoperative note establishes what is thought to be wrong with the patient and what procedure is planned. It indicates that the appropriate preoperative tests have been performed and reviewed. It also confirms the presence of an informed consent document (operative permit).

Content

- Preoperative diagnosis
- Planned procedures
- Allergies
Example Preoperative note

Preop Dx: Mandibular retrognathia

Planned procedures: 1. Bilat. mand. ramus sagittal split osteotomies
                     2. Advancement genioplasty

Allergies: None known

Labs: CBC 12.9 12.8/39.2 140

<table>
<thead>
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<th></th>
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<th>107</th>
<th>8</th>
<th>87</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lytes</td>
<td>4.4</td>
<td>29</td>
<td>0.8</td>
<td></td>
</tr>
</tbody>
</table>

PT: 12.8/12.4

PTT: 22.6/26.9

Operative permit: Signed and on chart

NOTE: A standardized format for recording the laboratory results is useful in organizing the handwritten notations, eg:

<table>
<thead>
<tr>
<th></th>
<th>CBC: Hgb</th>
<th>WBC</th>
<th>Platelets</th>
<th>Electrolytes: Na</th>
<th>Cl</th>
<th>BUN</th>
<th>K CO2</th>
<th>Creatinine</th>
<th>Glucose</th>
</tr>
</thead>
</table>

Prothrombin time: Patient’s value/control value Partial thromboplastin time: Patient’s value/control value
Operative Permit (Informed Consent)

Purpose

Surgical care provided on an inpatient requires a written, signed, informed consent document. Often referred to as an op permit, it is an essential part of the medical record. It must be properly completed prior to any invasive, anesthetic, or other procedure that involves any significant risk to the patient.

Content

- The nature of the planned procedure, described in understandable lay terms.
- The possible alternative methods of treatment, if any.
- The name of the surgeon(s).
- The risks associated with the procedure, including those associated with the anesthetic.
- The patient’s signature; if the patient is a minor, incompetent, or unable to comprehend and/or sign, the parent, legal guardian, or next of kin may do so. In life-threatening emergencies where consent cannot be immediately obtained, full documentation, including justification for proceeding with treatment, should be recorded on the patient’s medical record.

Preoperative Orders

Purpose

Preoperative orders ensure that the patient is ready for surgery. All necessary laboratory tests, radiographs, consultations, and permits must be ordered in a timely manner to ensure their availability before surgery.

Content

- When the patient is to go to the operating room
- NPO status
- Operative permit
- Laboratory tests
Example Preoperative orders

1. To OR on call
2. NPO \* MN
3. Op permit signed and on chart
4. Preop meds per anesthesia
5. Lab results on chart

Signed______________________________

Operative Note

Purpose

The operative note is a handwritten note describing the operative procedure, who was involved, the findings, any complications, and the condition of the patient. A dictated version of the report is also required, usually within 24 hours of the procedure.

Content

- Diagnoses, both preoperative and postoperative
- Procedure(s) performed
- Surgeons, including assistant(s)
- Findings
- Anesthetics used
- Fluids administered
- Estimated blood loss
- Drains, if any
- Specimens, if any
- Complications, if any
- Condition of patient
Example Operative note

Preoperative Dx: Vertical maxillary excess
Postoperative Dx: Same
Procedure: (1) Le Fort I osteotomy with internal rigid fixation; (2) Bilat. mand. sag. split ramus osteotomies
Surgeons: (1) M.W. Jones, (2) J.A. Smith, (3) D.M. Brown
Findings: Consistent with diagnosis
Anesthetics: Suforane, oxygen, nasoendotracheal intubation (hypotensive technique)
Fluids: RL 600 mL
EBL: 150 mL
Drains: None
Specimens: None
Complications: None
Condition: Stable, transferred to recovery room

Postoperative Orders
Purpose
Postoperative orders ensure that the findings and effects of surgery are properly considered. All standing orders are automatically canceled when the patient goes to the operating room. Therefore, after surgery, all orders must be written anew, including, if indicated, those that were in effect before surgery.

Content
- Admitting doctor, service
- Diagnosis
Example Postoperative orders

Admit to PORR, then transfer to floor when extubated, stable

Attending: Dr Jones

Dx: S/P BSSO/Le Fort I osteotomy for mandibular prognathism and maxillary hypoplasia

Condition: Satisfactory

Vital signs: Routine PORRU

Allergies: NKDA

Ambulation: Bed rest

Nursing: 1. Pt to be extubated/anesthesia
   2. 40% O2 T-piece
   3. NT suction qh prn while intubated
   4. Vaseline to lips prn
   5. Ice bag to both cheeks 20 min on and 20 min off × 24 h
   6. Pen and pad at bedside
   7. Suction at HOB
   8. Wire cutter & hemostat at HOB
   9. Salem sump NG to low wall suction
   10. Foley to straight drain/gravity
   11. Elevate HOB to 30°
   12. Encourage deep breathing
Diet: NPO

Record I&O

Labs: AM labs: Astra 7, CBC & diff, postop x-rays when patient is transported to floor, facial & mandibular series

Meds:
1. Ancef 3 gm IVPB q8h
2. 8 mg Decadron IV q8h × 3 doses, then Depomedrol 40 mg IM
3. Demerol 50 mg/Phenergan 25 mg IM/IV q4–6h prn severe pain
4. Tylenol 650 mg supp q4h prn temp. > 100°F
5. Zofran 4 mg IM/IV q6h prn nausea or vomiting

Signed___________________________________

Postoperative Note

Purpose

The postoperative note considers the effects of surgery and anesthesia on the patient’s status. The night of surgery note (shown in the example below) is a postoperative note that is of special importance because it is the first progress note after surgery. Subsequent postoperative notes are modified to the more standardized progress note (SOAP method; see “Progress Notes”).

Content

• Procedure performed
• Level of consciousness
• Vital signs
• Ins and outs
• Laboratory results
• Results of clinical examination, including breath sounds, bowel sounds, and condition of wound
Example Oral and maxillofacial surgery night of surgery note

Procedure: BSSO/Le Fort I osteotomy
Level of consciousness: Easily arousable but drowsy, airway intact
Vital signs: BP 105/65, P 100, R 16
In and outs:
  - IV: 1,100 mL
  - Foley: 2,200 mL
  - NG: 200 mL
Labs: Hct: 29%
PE: RRR $ murmurs, gallops
Lungs: Bibasilar rales
Abdomen: BS × 4
Head and neck: Moderate facial edema, pressure bandage intact, minimal intraoral oozing, MMF stable.
Assessment: Satisfactory postop results and progress.
Plan: Will remove NG and Foley in AM. Begin clear liquids in AM.

Signed______________________________
The progress notes are comparable to a log book in that they are a daily written account of the patient’s progress. The SOAP method has become a very popular standard format for progress notes.

Content

S = Subjective (how the patient feels; symptoms)
O = Objective (the results of your observations and examination, eg, vital signs, laboratory results, clinical examination)
A = Assessment (based on the above, your opinion of the patient’s present status)
P = Plan (what is planned for the patient; change in medication; additional tests; discharge)

Example Progress note

S—c/o swelling, some nasal stuffiness. Reports tingling (L) lower lip; (R) side ok. Minimal pain, no need for pain meds. Walked up and down hall 6 assistance.
O—BP 105/85 P 92 R 20

Temp: 100.4° (R)
Intake: 850 mL IV

250 mL po

Output: 1,400 mL urine
Moderate facial swelling
Able to close teeth into occl. splint
Wounds dry

A—Satisfactory progress \( \frac{1}{3} \) day s/p Le Fort I, mand. osteotomies
P—Encourage ambulation, po intake

To XR today for postop films
Plan DC tomorrow if progress continues

Signed______________________________

**Discharge Summary**

**Purpose**

The discharge summary is a standardized accounting of the patient’s hospitalization. It is a capsuled outline of the patient’s progress from the time of admission to the time of discharge. A dictated version is required, usually within 24 hours of the date of discharge.

**Content**

- Date of admission
- Date of discharge
- Admitting diagnosis
- Discharge diagnosis
- Attending doctor and service
- Referring doctor
- Procedure
- History, clinical examination findings, and pertinent laboratory values
- Hospital course
- Condition of patient at discharge
Example Oral and maxillofacial surgery discharge summary note

Date of admission: 1/26/10
Date of discharge: 1/28/10
Admitting Dx: (1) Vertical maxillary hyperplasia, (2) mandibular prognathism
Discharge Dx: Same
Attending: Dr Jones
Service: Oral and maxillofacial surgery
Referring physician: Dr Jameson
Procedure: BSSO setback/Le Fort I osteotomy

Brief history, pertinent physical findings, and lab data: Patient has a long history of inability to masticate foods easily, inability to put teeth end to end, and a “bad bite.” S/P active ortho Tx × 1 year. Tx plan calls for surgical phase consisting of BSSO setback and a Le Fort I osteotomy. Physical findings consistent with maxillary hyperplasia and mandibular prognathism.

Lab data: All WNL

Hospital course: Pt admitted on 1/26/10 and underwent a BSSO/Le Fort I osteotomy. Patient had uneventful perioperative and postoperative course. Patient transferred from PORR to floor late afternoon 1/26/10. The following day the patient cont. to improve. The next day the patient was judged suitable for DC to home.
Condition at DC: Vital signs were stable, afebrile, tolerating a po diet well, ambulatory without assistance, surgical wounds healing $\$ signs of infection or complications. The patient’s overall condition was judged to be improved and suitable for DC.

Disposition: To home, improved

Discharge meds:
1. Keflex elixir 250 mg/5 mL—Disp: 200 mL; sig: 10 mL qid until gone
2. Tylenol $\&$ codeine elixir—Disp: 300 mL; sig: 15 mL PO q3–4h prn pain, or Roxicet—Disp: 300 mL; sig: 10 mL PO q4–6h prn pain
3. Dimetapp elixir—Disp: 300 mL; sig: 15 mL PO q4–6h prn congestion
4. Chlorhexidine rinse—Disp: 16 oz; sig: 10 mL PO swish × 2 min then expel; use bid
5. Multivitamins & Fe liquid—Disp: † month supply; sig: use daily as directed
6. Wire cutter and hemostat—sig: use only in emergency; to be carried by patient at all times

Discharge instructions and follow-up
1. Oral hygiene
2. Wound care
3. Activity level
4. Medications
5. Return to clinic

Signed______________________________________________

Discharge Orders

Purpose
Discharge orders are necessary to release the patient from the hospital. They indicate that the necessary medications and follow-up care will be arranged.
Content

- When to discharge
- Medications to be prescribed
- Home care instructions
- Follow-up care appointment

**Example Discharge orders**

1. DC IV
2. Disch. to home
3. Return to clinic on 1/31/10 @ 1:00 PM
4. Disch. meds:
   - Keflex elixir 250 mg/5 mL—Disp: 200 mL; sig: 10 mL PO qid until gone
   - Tylenol & codeine elixir—Disp: 300 mL; sig: 10 mL PO q3–4h prn pain
   - Dimetapp elixir—Disp: 300 mL; sig: 15 mL PO q4–6h prn congestion
   - Chlorhexidine rinse—Disp: 16 oz; sig: 10 mL PO swish × 2 min, then expel; use bid
   - Multivitamins and Fe Liquid—Disp: 1 month supply; sig: use daily as directed
   - Wire cutter & hemostat—sig: use only in emergency; to be carried at all times

Signed________________________________________
Laboratory Tests and Their Interpretation

Laboratory tests are an invaluable aid to the practicing oral and maxillofacial surgeon. In conjunction with a thorough history and physical examination, the myriad laboratory tests available can aid in the diagnosis of various diseases and allow the precise preoperative and postoperative management of patients with systemic disease. In addition, patients without overt disease can be screened before procedures that carry potentially serious complications, such as general anesthesia, are begun.

Hematology
Collection Techniques

Venipuncture equipment: Tourniquet; alcohol preparation pad; Vacutainer holder, needle and tubes, or appropriate-size syringe and 18- to 22-gauge needle; adhesive bandage

Collection sites: Antecubital fossa, dorsum of hand, forearm, saphenous vein, external jugular vein, femoral vein

Collection tube types:

<table>
<thead>
<tr>
<th>Tube color</th>
<th>Additive</th>
<th>Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red</td>
<td>None</td>
<td>Serum for blood chemistry, blood type and crossmatch, serology</td>
</tr>
<tr>
<td>Blue</td>
<td>Sodium</td>
<td>Coagulation studies</td>
</tr>
</tbody>
</table>
Evaluation Techniques

Blood smear analysis: Consists of differential white blood cell count, evaluation of size and morphology of red and white blood cells

**Wright stain technique**
- Place small drop of anticoagulated blood on clean glass slide.
- Touch second glass slide to first at 45-degree angle and draw back to touch drop of blood.
- Move second slide in opposite direction to spread blood over the slide in a thin film.
- Allow slide to air dry and label with patient’s name.
- Flood slide with Wright stain and allow to stand for 3 to 4 minutes.
- Rinse off stain with distilled water and allow slide to air-dry.
- Examine under oil immersion, counting 200 white blood cells in a strip running the entire length of the film. Classify cells as to type and record as a percentage of the total white blood cells.

Automated analysis
**Coulter counter**: Electronic cell counter with automatic chemical determination of hemoglobin concentration and measurement of hematocrit by conductance. The three red cell indices are calculated from these data.

**Three-cell differential count**: Automated separation of white blood cells into three groups based on size: large cells (stab neutrophils and band cells), middle-size cells (monocytes, eosinophils, large lymphocytes), and small cells (normal lymphocytes).

**Definitions of terms**

**Anisocytosis**: Variation in red blood cell (RBC) size

**Complete blood cell (CBC) count**: White blood cell (WBC) count, red blood cell (RBC) count, hemoglobin (HgB), hematocrit (Hct), red cell indices (mean corpuscular volume [MCV], mean corpuscular hemoglobin [MCH], mean corpuscular hemoglobin concentration [MCHC])

**Hemogram**: CBC + differential WBC count and platelet count

**Hyperchromic**: Abnormally high amounts of hemoglobin

**Hypochromic**: Deficient cell hemoglobin producing pale-appearing cell

**Macrocytic**: RBC larger than normal; diameter > 7.7 µm; MCV > 100 µ³

**Microcytic**: RBC smaller than normal; diameter < 6.7 µm; MCV < 80 µ³

**Normocytic**: RBC cell diameter 6.7 to 7.7 µm; MCV = 80 to 100 µ³

**Poikilocytosis**: RBCs of irregular shape (Burr cells, sickle cells)

**Polycythemia**: Increased RBC count in a unit volume of blood in
the presence of an increased total blood volume

*Red cell indices:*

\[
M_{CV} = \frac{\text{Hct} (\%) \times 10}{\text{RBC (millions/mm}^3)}
\]

\[
M_{CH} = \frac{\text{Hgb} (\text{g/100 mL}) \times 10}{\text{RBC (millions/mm}^3)}
\]

\[
M_{CHC} = \frac{\text{Hgb} (\text{g/100 mL})}{\text{Hct} (\%)}
\]

*Shift to left:* A predominance of immature polymorphonuclear neutrophils (PMNs) with only one or two nuclear lobes

*Shift to right:* A predominance of PMNs with four nuclear lobes

**Normal Values**

Hct: Men: 47% ± 7.0%

Women: 42% ± 5.0%

Hgb: Men: 14–18 g/100 mL

Women: 12–16 g/100 mL

MCV: 80–100 fL

MCH: 28–31 µg

MCHC: 30%–35%

RBC: Diameter: 6.7–7.7 µm

Men: 4.2–5.4 × 10^6 cells/mm^3

Women: 3.6–5.0 × 10^6 cells/mm^3
Reticulocytes: 0.5%–1.5%

WBC: Total: 4–11 × 10³ cells/mm³

**Differential**

- PMN: 40%–75%
- Lymphocytes: 15%–45%
- Eosinophils: 1%–6%
- Basophils: 0%–2%
- Monocytes: 1%–10%

Platelets: 145–375 × 10³/mm³

Prothrombin time (PT): Usually 12–14 seconds. Always given with control; should be within 2 seconds of control.

Partial thromboplastin time (PTT): Usually 25–45 seconds. Always given with control; should be within 4 seconds of control.

Erythrocyte sedimentation rate (ESR): Wintrobe:

- Men: 0–9 mm/h
- Women: 0–20 mm/h
- Child: 0–13 mm/h

Westergren:

- Men: 0–15 mm/h
- Women: 0–20 mm/h
- Child: 3–13 mm/h

**Differential Diagnosis**

RBC indices
Decreased MCV, decreased MCH (microcytic, hypochromic)
- Iron deficiency anemia
- Thalassemia
- Anemia of chronic disease

Increased MCV, increased MCH (macrocytic, hyperchromic)
- Megaloblastic anemia; B\textsubscript{12} or folate deficiency
- Early posthemorrhage period

Normal MCV and MCH (normocytic, normochromic)
- Blood loss
- Hemolysis
  - Marrow failure
  - Systemic disease

RBC morphology

Sickled cells: Sickle cell anemia

Howell-Jolly bodies: Megaloblastic anemia, postsplenectomy

Spherocytes: Autoimmune hemolytic anemia, hereditary spherocytosis

Basophilic stippling: Lead poisoning

Burr cells: Severe liver disease

Helmet cells: Uremia, malignant hypertension, hemolytic transfusion reaction

Reticulocytes: Hemorrhage, hemolysis, post-Fe\textsubscript{2} treatment

Hct
**Increased:** Heavy smokers, high altitude, dehydration, prolonged tourniquet stasis, primary or secondary polycytemia

**Decreased:** Volume overload, anemia (iron deficiency, megaloblastic, sickle cell), blood loss, hemolysis

**WBC**

**Increased:** Acute infections, uremia, steroids, hemorrhage, leukemia

**Decreased:** radiation, aplastic anemia, infectious mononucleosis, septicemia

**WBC (differential)**

**Neutrophils (PMNs)**

**Increased:** Infections, granulocytic leukemia, surgery, severe exercise

**Decreased:** Viral infections, aplastic anemia, drugs, radiation, dialysis

**Shift to left:** Hemorrhage, toxemia, bacterial infections

**Shift to right:** Iron deficiency anemia, liver disease, megaloblastic anemia

**Eosinophils**

**Increased:** Allergic disorders, parasitic infection, collagen vascular diseases, Addison disease, malignancy
Decreased: Steroids, stress, adrenocorticotropic hormone (ACTH) excess, Cushing syndrome

Basophils

Increased: Polycythemia, chronic myeloid leukemia
Decreased: Steroids, stress, acute rheumatic fever, thyrotoxicosis

Lymphocytes

Increased: Viral infections, tuberculosis, mononucleosis, acute and chronic lymphocytic leukemia
Decreased: Stress, uremia, steroids

Monocytes

Increased: Monocytic leukemia, tuberculosis, chronic inflammation or infection, collagen disease (rheumatoid arthritis, systemic lupus erythematosus), subacute bacterial endocarditis, protozoal infections
Decreased: Aplasia

WBC morphology

Döhle inclusion bodies in PMNs: Burns, infection
Auer bodies: Acute myelogenous leukemia
Hypersegmentation: Megaloblastic anemias

Toxic granulation: Infection or inflammatory disease

Platelet count

*Increased:* Malignancy, postsurgery or postsplenectomy, rheumatoid arthritis (RA), iron deficiency anemia, trauma, acute hemorrhage

*Decreased:* Idiopathic thrombocytopenic purpura (ITP), marrow invasion or aplasia, hypersplenism, disseminated intravascular coagulation (DIC), cirrhosis, quinidine toxicity (and many other drugs), massive transfusions, viral infections, infectious mononucleosis

**Blood Chemistry Tests**

Electrolytes

Sodium (136 to 145 mEq/L)

*Increased:* Dehydration, glycosuria, diabetes insipidus

*Decreased:* Diuretic use, congestive heart failure, hyperglycemia, renal failure, vomiting, diarrhea

Chloride (95 to 108 mEq/L)

*Increased:* Dehydration, nonanion gap metabolic acidosis, diarrhea, diabetes insipidus

*Decreased:* Vomiting, excess sweating, congestive heart failure, chronic renal failure, diuretic use, syndrome of inappropriate antidiuretic hormone (SIADH), diabetes mellitus with
ketoacidosis

Potassium (3.5 to 5.2 mEq/L)

*Increased:* Renal failure, adrenal insufficiency, acidosis, hemolysis, medications, iatrogenic

*Decreased:* Diuretic therapy, alkalosis, vomiting, nasogastric suctioning, mineralocorticoid excess

Bicarbonate (24 to 30 mEq/L)

*Increased:* Dehydration, respiratory acidosis, emphysema, vomiting, metabolic alkalosis

*Decreased:* Metabolic acidosis, respiratory alkalosis, renal failure, diarrhea

Anion gap (8 to 12 mEq): Difference in mEq between serum sodium and the sum of serum chloride and bicarbonate

*Normal:* Diarrhea, renal tubular acidosis

*Increased:* Renal failure, lactic acidosis, ketoacidosis, salicylate toxicity

*Decreased:* Disseminated intravascular coagulation, multiple myeloma

Renal Function

Blood urea nitrogen (BUN) (6 to 20 mg/dL)

*Increased:* Renal failure of all types, dehydration, gastrointestinal
bleeding, increased protein catabolism

*Decreased:* Liver damage, protein deficiency, starvation, overhydration

Creatinine (0.7 to 1.4 mg/dL)

*Increased:* Renal failure, muscle disease, false positives with diabetic ketoacidosis

*Decreased:* Pregnancy; rarely clinically significant

Liver Function

Albumin (3.5 to 5.0 g/dL)

*Increased:* Dehydration; rarely clinically significant

*Decreased:* Liver failure, starvation, hyperthyroidism, leukemia, nephrotic syndrome

Alkaline phosphatase (30 to 115 U/L)

*Increased:* Biliary tract obstruction, bone disease (Paget disease), hyperparathyroidism, osteoblastic bone tumors

*Decreased:* Hypophosphatasia, hypothyroidism, malnutrition

Bilirubin (0.2 to 1.2 mg/dL)

*Increased total:* Acute and chronic hepatitis, cirrhosis, biliary tract obstruction, hemolysis, fasting

*Increased direct (conjugated):* Obstructive liver disease, hepatitis, drug-induced cholestasis
Increased indirect (unconjugated): Hemolytic anemia, hepatocellular liver disease

Aspartate aminotransferase (AST) (formerly SGOT) (5 to 25 U/L)

Increased: Liver disease, acute myocardial infarction, pancreatitis, muscle trauma, congestive heart failure, hemolysis
Decreased: Rarely clinically significant

Alanine aminotransferase (ALT) (formerly SGPT) (5 to 30 U/L)

Increased: Liver disease (more specific than AST), pancreatitis, biliary obstruction

Total protein (6.0 to 8.5 g/dL)

Increased: Multiple myeloma, dehydration, sarcoidosis
Decreased: Liver failure, starvation, inflammatory bowel disease

Acute Myocardial Infarction
Creatinine phosphokinase (CPK) (female 50 to 60 IU/L; male 50 to 180 IU/L)

Increased total: Myocardial infarction, striated muscle necrosis, cerebrovascular accident, hypothyroidism, malignant hyperthermia

Isoenzyme fractionation of CPK
- MM band (striated muscle): Crush injury, seizures, malignant hyperthermia, Intramuscular (IM) injections
- MB band (cardiac muscle): Acute myocardial infarction, myocarditis
- BB band (brain): Cerebrovascular accident, malignant hyperthermia

Lactate dehydrogenase (LDH) (45 to 100 U/L)

*Increased:* Acute myocardial infarction, hepatitis, malignant tumors, muscle disease, pulmonary embolus, hemolysis

*LDH isoenzymes:* LDH I to LDH V; elevated LDH I, II—myocardial origin; LDH V > LDH IV—liver disease

AST (SGOT) (8 to 20 U/L) (see page 31)

Metabolic Bone Disease
Alkaline phosphatase (30 to 115 U/L)

*Increased:* Bone, liver, pancreas, lung diseases

Calcium (8.5 to 10.5 mg/dL)

*Increased:* Hyperparathyroidism, hypervitaminosis D, metastatic bone tumors, Paget disease, multiple myeloma, sarcoidosis, chronic renal failure

*Decreased:* Hypoparathyroidism, hypoalbuminemia, renal failure, alkalosis, acute pancreatitis, convulsions, vitamin D deficiency

Phosphorus (2.3 to 4.7 mg/dL)
**Increased:** Hypoparathyroidism, chronic renal failure, acidosis, hypervitaminosis D, Addison disease

**Decreased:** Hyperparathyroidism, alcoholism, hypokalemia, vitamin D deficiency, alkalosis, diabetes mellitus

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Other Tests

**Acid phosphatase (< 0.8 IU/L)**

**Increased:** Prostate carcinoma, prostatitis

**Amylase (5 to 75 IU/L)**

**Increased:** Pancreatitis, parotitis (can fractionate to determine parotid versus pancreatic origin), duodenal ulcer, diabetic ketoacidosis, peritonitis, blunt abdominal trauma

**Decreased:** Pancreatic destruction, liver damage

**Cholesterol (140 to 260 mg/dL)**

**Increased:** Hypercholesterolemia-hyperlipidemia, biliary tract obstruction, pancreatitis, hypothyroidism, diabetes mellitus

**Decreased:** Starvation, chronic disease, hyperthyroidism, liver disease, steroid therapy

**C-reactive protein (CRP) (10 mg/L)**

**Increased:** Inflammation, bacterial and viral infection, burns, slightly with age
Glucose (80 to 110 g/dL)

*Increased:* Diabetes mellitus, stress, hyperthyroidism, pregnancy, pancreatic disease, steroid therapy, Cushing syndrome

*Decreased:* Reactive hypoglycemia, pancreatic disorders, starvation, liver disease, hyperinsulinism, hypothyroidism, hypopituitarism, Addison disease, sepsis

Magnesium (1.6 to 2.6 mg/dL)

*Increased:* Renal failure, diabetic coma, severe dehydration, lithium intoxication, Addison disease, hypothyroidism

*Decreased:* Alcoholism, diuretics, acute pancreatitis, nasogastric suctioning, malabsorption, renal tubular acidosis

Serum osmolality (278 to 298 mOsm/kg)

*Increased:* Alcohol ingestion, hyperglycemia, water loss, administration of mannitol

*Decreased:* Syndrome of inappropriate antidiuretic hormone, diuretics, Addison disease, low serum sodium

Uric acid (1.5 to 8.0 mg/dL)

*Increased:* Renal disease, gout, thiazide diuretics, myeloproliferative diseases of bone

*Decreased:* Wilson disease, allopurinol use, aspirin ingestion, Hodgkin disease
Coagulation
Bleeding time (Duke, Ivy methods < 6 minutes; Template < 10 minutes)

*Increased:* Thrombocytopenia, von Willibrand disease, aspirin therapy

Clotting time (Lee White, 6 to 7 minutes)

*Increased:* Heparin therapy, clotting factor deficiency

Fibrinogen (150 to 450 mg/dL)

*Increased:* DIC, severe bleeding, burns, hematologic disorders

Thrombin time (10 to 14 seconds)

*Increased:* Heparin, DIC, fibrinogen deficiency

Partial thromboplastin time (PTT) (25 to 45 seconds)

*Increased:* Heparin, defects in intrinsic clotting mechanism, hemophilia A and B, prolonged use of tourniquet before drawing blood

Prothrombin time (PT) (12 to 14 seconds)

*Increased:* Sodium warfarin, vitamin K deficiency, liver disease, DIC, prolonged use of tourniquet before drawing blood
International normalized ratio (INR): The international normalized ratio (INR) was developed to standardize PT results, which can vary for a normal individual because of inconsistencies in the reagent used to perform the test. This system affords a more uniform interlaboratory assessment of the Coumadin effect, thus enabling a direct comparison between PT results, regardless of the reagent/instrument variables. Manufacturers assign each lot of thromboplastin an international sensitivity index (ISI) value calibrated from World Health Organization reference material.

The INR is calculated using the formula:

\[
\text{INR} = \left( \frac{\text{Patient protime}}{\text{Mean of normal range}} \right) \times \text{ISI}
\]

Interpretation of PT and PTT in patients with bleeding disorders
- PT time prolonged, PTT time normal: Liver disease, decreased vitamin K, decreased or defective factor VII
- PT time normal, PTT time prolonged: Decreased or defective factor VIII, IX, or XI or lupus anticoagulant present
- PT time and PTT time prolonged: Decreased or defective factor I (fibrinogen), II (prothrombin), V, or X, von Willebrand disease, liver disease, DIC
- PT time and PTT time normal: Decreased platelet function, thrombocytopenia, factor XIII (prekallikrein) deficiency, mild deficiencies in other factors, mild form of von Willebrand disease

Urinalysis
Normal Values
- Appearance: Straw yellow; clear
- Specific gravity
Infant: 1.002 to 1.006
Child and adult: 1.001 to 1.035

pH: 4.6 to 8.0
Negative: Glucose, protein, bilirubin, blood, acetone, nitrite
Trace: Urobilinogen
RBC count: Male, 0 to 3/high-power field (HPF); female, 0 to 5/HPF
WBC count: 0 to 4/HPF
Epithelial cells: Occasional
Hyaline casts: Occasional
Bacteria: None
Urine output: Adult, 0.5 to 1.0 mL/kg/h (1,000 to 1,600 mL/d)

Differential Diagnosis
Appearance

Pink/red: Blood, Hgb, food coloring
Brown/black: Bile pigments, iron, melanin
Orange: Pyridium, bile pigments
Cloudy: Pyuria, blood, mucus, bilirubin
Foamy: Proteinuria, bile salts

pH

Acidic: Ketoacidosis, chronic obstructive pulmonary disease (COPD), high protein diet
Basic: Urinary tract infection, renal tubular acidosis, vomiting, metabolic alkalosis

Specific gravity (value > 1.023 indicates normal renal concentrating ability)

*Increased:* Volume depletion, congestive heart failure, diabetes mellitus, adrenal insufficiency

*Decreased:* Fluid overload, glomerulonephritis, diabetes insipidus

Bilirubin: Positive indicates hepatitis, obstructive jaundice

Blood: Positive indicates trauma, infection, menses, hemolytic anemia, transfusion reaction, renal stones

Glucose: Positive indicates diabetes mellitus, pancreatitis, shock, steroids, hyperthyroidism, Cushing disease

Ketones: Positive indicates starvation, diabetic ketoacidosis, vomiting, diarrhea, hyperthyroidism, pregnancy

Nitrite: Positive indicates infection

Protein: Positive indicates fever, stress, malignant hypertension, congestive heart failure, pyelonephritis

Urobilinogen: Positive indicates congestive heart failure, hepatitis, hyperthyroidism, cirrhosis

RBCs: Trauma, cystitis, prostatitis, coagulopathy, tumors

WBCs: Infection, tuberculosis, renal tumors, acute glomerulonephritis

Urine electrolytes

- Sodium < 10 mEq/L = volume depletion, hyponatremia, congestive heart failure
- Sodium > 30 mEq/L = acute tubular necrosis
- Chloride < 10 mEq/L = volume depletion, vomiting, diuretic use
- Potassium < 10 mEq/L = hypokalemia
Osmolality (500 to 1,200 mOsm/L)

*Increased:* Dehydration, SIADH, adrenal insufficiency

*Decreased:* Acute renal failure, diabetes insipidus, excessive fluid intake

Creatinine clearance

*Normal:* Male total creatinine—18 to 25 mg/kg/24 h/clearance—100 to 125 mL/min; female total creatinine—12 to 20 mg/kg/24 h/clearance—85 to 105 mL/min

*Decreased:* Renal insufficiency

*Increased:* Pregnancy, early diabetes mellitus

24-hour urine studies

- Calcium (100 to 300 mg/24 h urine)

  *Increased:* Bone metastasis, Paget disease, sarcoidosis, hyperparathyroidism, glucocorticoid excess

  *Decreased:* Hypothyroidism, renal failure, rickets, thiazide diuretics

- 17-ketosteroids (male, 9 to 22 mg/dL; female, 6 to 15 mg/dL)

  *Increased:* Severe stress, ACTH excess, Cushing syndrome

  *Decreased:* Addison disease

**Microbiology**
Gram Stain Procedure

- Smear material to be stained on a clean glass slide; allow to air-dry; fix slide by passing through a flame several times.
- Stain by dipping the slide in the following sequence of stains: crystal violet, 1 minute, rinse under tap water; Gram iodine, 1 minute, rinse; dip in each of the three jars of ethanol, rinse; safranin, 1 minute, rinse, blot, and air-dry.
- Scan slide at low power (×100). Final examination is made under oil immersion (×1,000).

Gram Stain Characteristics of Common Pathogens

Gram-positive cocci

* Aerobic: *Staphylococcus*, *Streptococcus*, *Diplococcus*
* Anaerobic: *Peptococcus*, *Peptostreptococcus*

Gram-positive rods

* Aerobic: *Lactobacillus*
* Anaerobic: *Clostridium*, *Eubacterium*, *Actinomyces*, *Propionibacterium*

Gram-negative cocci

* Aerobic: *Neisseria*
* Anaerobic: *Veillonella*

Gram-negative rods

* Aerobic: *Haemophilus*, *Enterobacter*
Anaerobic: *Bacteroides, Fusobacterium*

Acid-Fast Stain
Stains acid-fast bacilli red (*Mycobacterium tuberculosis*).

Potassium Hydroxide (KOH) Preparation
Used for the diagnosis of fungal infections. KOH destroys most elements other than the fungus.

Wayson Stain
Good stain to scan for bacteria because it colors most bacteria.

India Ink Preparation
Used to identify fungal organisms (*Cryptococci*) in cerebrospinal fluid.

Cultures
Throat culture: Used to differentiate viral pharyngitis from bacterial pharyngitis (group A beta-hemolytic streptococci, *Bordetella pertussis, Neisseria gonorrhoeae, Corynebacterium diphtheriae*).

Sputum culture: Most useful in diagnosis of acute pneumonia, but most clinicians rely on the Gram stain and clinical findings.

Urine culture: Satisfactory specimens for urine culture include the midstream clean-catch voided specimen, sterile aspirate from a closed-system catheter tubing, or sterile suprapubic aspirate.

Stool culture: Fresh sample grown on standard media will isolate common pathogens such as *Shigella* and *Salmonella*. *Clostridium difficile* best diagnosed by *C difficile* toxin determination. Parasitic diseases diagnosed by evaluation for ova and parasites.

Antibiotic Sensitivity Testing
Evaluates susceptibility of pathogenic organisms to various antibiotics.
Agar-disc diffusion: Standardized concentration of organisms mixed in agar and poured into a Petri dish. Small paper discs impregnated with standard concentrations of antibiotics are placed on the agar. Clear zone of inhibited bacterial growth around each disc is measured as an indicator of the organism’s sensitivity to each antibiotic.

Serial dilution method: Standardized suspension of the organism is inoculated into tubes containing serial dilutions of various antibiotics. The least concentration of the antibiotic that will inhibit growth of the organisms is reported as the minimum inhibitory concentration (MIC).

Common Pathogens in Various Infections
Osteomyelitis: *Staphylococcus aureus*
Sinusitis: *Streptococcus pneumoniae, Haemophilus influenzae, S aureus*
Skin: *S aureus, Streptococcus pyogenes*
Pneumonia: *S pneumoniae, M tuberculosis, Mycoplasma pneumoniae, Klebsiella, Enterobacter,* respiratory viruses, *Legionella pneumophila*
Bacterial endocarditis: *Streptococcus viridans, S aureus, Enterococcus*
Meningitis: *S pneumoniae, H influenzae, Neisseria meningitides, Cryptococcus*
Urinary infections: *Escherichia coli, Proteus mirabilis, Pseudomonas, Klebsiella, Enterobacter*
Urethritis: *Gonococcus, Chlamydia trachomatis*

**Special Tests**
Antinuclear antibody (ANA) (normally negative)

*Positive*: Collagen-vascular disease, RA, systemic lupus erythematosus (SLE), scleroderma, lupus, drug reactions

ESR (Wintrobe; males 0 to 9 mm/h, females 0 to 20 mm/h)
*Increased:* Infection, inflammation, rheumatic fever, endocarditis, neoplasm, acute myocardial infarction, temporal arteritis

Fluorescent treponemal antibody (FTA) (normally nonreactive)

*Positive:* Syphilis

Glycohemoglobin (4.6% to 7.1%)

*Increased:* Uncontrolled diabetes mellitus; reflects levels over preceding 3 to 4 months

HIV antibody (normally negative)

*Enzyme-linked immunosorbent assay (ELISA)* (screening test)

*Western blot:* Used for confirming presence of HIV antibody

*Positive:* AIDS, AIDS-related complex

SLE cell preparation (no cells seen is normal)

*Positive:* SLE, RA, scleroderma, lupus, drug reactions

Monospot (normal is negative)

*Positive:* Infectious mononucleosis; rarely in leukemia, RA, viral hepatitis

Myoglobin (qualitative negative)
**Positive**: Acute myocardial infarction, malignant hyperthermia, skeletal muscle disorders

Rheumatoid factor (< 1.40 by latex fixation test)

**Increased**: RA, SLE, syphilis, subacute bacterial endocarditis (SBE), chronic inflammation

Venereal Disease Research Laboratories (VDRL) (normal is nonreactive)

**Positive**: syphilis; confirm with FTA test (see above)

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**Blood Gases**

**Indications for Measurement**
- Altered ventilatory status: stroke, asthma, COPD
- Hypoxemia: pneumonia
- Hypocapnia: hyperventilation
- Hypercapnia: COPD
- pH disturbance: ketoacidosis

**Normal Values—Arterial Blood Sample**
- Partial pressure of oxygen: PO$_2$ 80 to 95 mm Hg
- Oxygen saturation: SaO$_2$ 93% to 98%
- Partial pressure of CO$_2$: PCO$_2$ 36 to 43 mm Hg
  - Bicarbonate (HCO$_3^-$) content: 20 to 30 mEq/L
  - Arterial pH: 7.35 to 7.45

**Interpretation of Arterial Blood Gas Results**

**Arterial pH**
- $< 7.35 = $ acidosis
7.35 to 7.45 = normal, compensated, or mixed disorder
> 7.45 = alkalosis

Acidosis—Respiratory or metabolic?

**Respiratory**
- $\text{PCO}_2 > 45 \text{ mm Hg} = \text{respiratory acidosis}; \text{HCO}_3 \text{ usually normal in early stages of simple respiratory acidosis but will increase in compensated chronic acidosis.}
- Etiology of respiratory acidosis: Secondary to $\text{CO}_2$ buildup and can involve COPD, pulmonary edema, cardiac arrest, pneumonia, chest wall or airway injury, drug effects, CNS depression.

**Metabolic**
- $\text{HCO}_3 < 22 \text{ mEq/L} = \text{metabolic acidosis}; \text{PCO}_2$ will usually fall as patient hyperventilates to correct acidosis by blowing off $\text{CO}_2$. An example of this would be Kussmaul respiration seen in diabetic ketoacidosis.
- Etiology of metabolic acidosis: Normal anion gap—diarrhea, renal tubular acidosis; increased anion gap—aspirin toxicity, hyperalimentation, renal failure, diabetic ketoacidosis, lactic acidosis (normal value, see page 30).

Alkalosis—Respiratory or metabolic?

**Respiratory**
- $\text{PCO}_2 < 35 \text{ mm Hg} = \text{respiratory alkalosis, compensatory decrease in plasma HCO}_3$
- Etiology of respiratory alkalosis: Hyperventilation, CNS injury, fever, pulmonary embolism, compensation for metabolic acidosis, excessive mechanical ventilation
Metabolic
- HCO$_3^-$ > 26 mEq/L = metabolic alkalosis
- Etiology of metabolic alkalosis: Iatrogenic causes include diuretic therapy, HCO$_3^-$ treatment in dialysis patients, nasogastric suctioning, severe vomiting, severe potassium depletion, disease states such as Cushing syndrome, hypoparathyroidism

Compensated acid-based disorders

Acidosis
- Acute respiratory acidosis: HCO$_3^-$ increased by 1 mmol/L for each 10 mm Hg increase in PCO$_2$
- Chronic respiratory acidosis: HCO$_3^-$ increased by 4 mmol/L for each 10 mm Hg increase in PCO$_2$
- Metabolic acidosis: PCO$_2$ decreased 1.0 to 1.5 mm Hg for every 1 mmol/L decrease in HCO$_3^-$

Alkalosis
- Acute respiratory alkalosis: HCO$_3^-$ decreased by 2 to 4 mmol/L for each 10 mm Hg decrease in PCO$_2$
- Chronic respiratory alkalosis: HCO$_3^-$ decreased by 2 to 5 mmol/L for each 10 mm Hg decrease in PCO$_2$
- Metabolic alkalosis: PCO$_2$ increased 0.25 to 1.0 mm Hg for each 1 mmol/L increase in HCO$_3^-$
chapter 4

Diagnostic Imaging

Oral and maxillofacial surgeons encounter patients with medical problems beyond the head and neck and therefore need to be aware of the myriad radiologic studies available for imaging the entire body. This chapter discusses some of the more common studies, their indications, and special information needed when ordering them. Radiologic studies that have more direct application to evaluation of the head and neck are also discussed.

Plain Film Studies
Noncontrast Studies

Chest radiographs: These radiographs should be ordered only for patients in whom there is suspicion of chest pathology or in whom the presence of occult chest pathology would compromise their immediate medical care.

Posteroanterior (PA) film: The following are guidelines for assessing the structures shown in the PA film (Fig 4-1a).

- Soft tissues: Check for asymmetry, loss of soft tissue planes, and air in improper locations. In a woman, check for breast shadows; their absence may indicate that the patient has had a mastectomy.
- Bony structures: Examine the ribs, clavicles, scapulae, proximal humeri, and vertebrae to look for osteolytic or osteoblastic lesions as well as fractures, arthritic changes, or other abnormalities.
- Diaphragm: Both sides should be of equal height, or the right side should be slightly higher than the left. The costophrenic angles should be sharp; blunting of the angles suggests either pleural scar or effusion. It is important to check below the diaphragm to look for subdiaphragmatic free air and other abnormalities.
- Heart and mediastinum: Transverse length of the heart should measure no more than one-half of the thoracic diameter in an adult. It may be
slightly larger in children. The aortic knob should be distinctly visible. The mediastinum should be checked for widening, which can occur with traumatic disruption of the aorta as well as tumor/adenopathy. The trachea should be straight and visualized down to the carina; if it is deviated, pathology such as a tumor, lung collapse, or a tension pneumothorax should be considered. In the child, the mediastinum may appear widened secondary to the normal thymic silhouette.

- **Hila:** Left hilum should appear 2 to 3 cm higher than the right. Look for pulmonary vascular hypertension or hilar adenopathy.

- **Lung fields:** Lung fields and mediastinum should be examined for the presence of lines and tubes (e.g., endotracheal tube, nasogastric tube, Swan-Ganz catheter). The lung fields should be clear of both interstitial disease and air space disease. Vessels should extend to the periphery of the lungs and should gradually taper centrally to peripherally. The vessels in the lower lung fields should be slightly larger than those in the upper lung fields. If the reverse is true, congestive heart failure should be considered. Other findings associated with congestive heart failure include an enlarged heart and Kerley B lines (small lines seen at the periphery of the lungs).

  — **Pneumothorax:** Lung fields should be examined closely for the presence of a pneumothorax. This is usually represented by a thin linear density paralleling the chest wall. No lung markings should be seen peripheral to this line.

  — **Changes of chronic obstructive pulmonary disease (COPD):** These include hyperexpanded lung fields, flattening of the diaphragm, a large lucent zone behind the sternum, and an elongated heart.

  — **Masses:** Masses within the lung fields can be divided into cavitary and noncavitary lesions. Either can represent tumor or infection. Masses also can be caused by other things such as vascular malformations, but other causes are far less common than tumor and infection.
**Fig 4-1a** Structures typically seen in a PA chest radiograph.

*Lateral film:* The structures typically seen on a lateral radiograph of the chest are shown in Fig 4-1b. The lateral chest film should be used to localize lesions to a specific area of the lungs or mediastinum. The lateral chest film is particularly helpful in examining for small pleural effusions (blunting of the posterior costophrenic angles) and vertebral or sternal abnormalities.

*Portable chest radiograph:* Used on incapacitated patients to detect pneumonia, pulmonary edema, line and tube placement, pneumothoraces, and effusions. Heart size cannot be accurately evaluated.
**Fig 4-1b** Structures usually seen in a lateral chest radiograph.

**Lordotic chest radiograph:** Permits best visualization of the apices of the lung and is ordered when there is a questionable lesion seen in these areas on the standard chest radiograph.

**Lateral decubitus chest radiograph:** Detects small amounts of free-flowing pleural effusion (approximately 150 mL) and distinguishes pleural scar/loculated pleural effusion from free-flowing pleural effusion.

**Expiratory chest radiograph:** Used to accentuate and better visualize a small pneumothorax.

Acute abdominal series: Includes supine and upright abdominal films (to view the kidneys, ureter, and bladder) and a chest radiograph. Used for the initial evaluation of acute abdominal pain or trauma.

KUB: Stands for “kidneys, ureter, and bladder” and is also known as a supine abdominal radiograph. It is useful in the initial work-up of abdominal pain, distension of the bowel, or change of bowel habits. It is also used for evaluation of urinary tract problems. The majority of renal stones and 10% to 20% of gallstones are visualized by a KUB. Free intraperitoneal air is more difficult to observe on the KUB than on the acute abdominal series.
Evaluation of the KUB involves examining the bowel gas pattern and looking for unusual calcifications. The following are also examined: the psoas muscles, kidneys, liver, splenic shadows; flank stripes; vertebral bodies; and pelvic bones. Radiopaque foreign bodies can also be seen.

Abdominal decubitus radiograph: Used in the debilitated patient in place of an upright abdominal film. The patient’s left side is usually down. This study is used to determine air-fluid levels within the bowel and free intraperitoneal air. It is usually not as easy to detect free intraperitoneal air on this film as on a standard chest radiograph.

Cervical spine: Usually includes PA, lateral, and odontoid views of the cervical spine. Occasionally both oblique views are included. It is useful for evaluating traumatic injury (although CT of the cervical spine also is frequently used for this indication), neck pain, and neurologic symptoms referable to the upper extremities. It is imperative that all seven vertebrae be seen for an examination to be considered acceptable.

Thoracic spine radiograph: Usually includes anteroposterior (AP) and lateral views of the thoracic spine and is used to evaluate trauma or pain referable to the thoracic spine. If these films are negative, a swimmer’s view (ie, superior oblique view) may be obtained to evaluate the upper thoracic spine.

Lumbar spine: This study is used for the initial evaluation of low back pain and trauma to the lumbar spine. Usually includes AP and lateral views of the lumbar spine. If the L5–S1 disc spaces are not well visualized, a coned-down lateral view should also be included. Frequently, both oblique views are also included.

Mammography: Used to detect cancers of the breast. Can visualize lesions that are smaller than 5 mm. A negative mammogram should not exclude further work-up of a palpable mass because mammography can miss approximately 15% of malignancies.

Contrast Studies

An oral agent, such as barium or diatrizoate meglumine and diatrizoate sodium solution, or an intravenous (IV) contrast agent is used in these studies. The choice of a contrast agent should be left to the radiologist. However, clinical information provided by the clinician is essential in making
this choice. Such information includes, but is not limited to, a history of allergies and renal function and the specific information sought from the study.

Air contrast barium enema (BE): This study is infrequently used, with clinicians opting either for colonoscopy or virtual colonoscopy. Indications can include diarrhea, heme-positive stools, crampy abdominal pain, change in bowel habits, or unexplained weight loss. Consult the radiologist for patient preparation.

Virtual colonoscopy: This is a computed tomography (CT)–mediated procedure used to create an animated, three-dimensional (3D) view of the inside of the colon. Consult the radiologist for patient preparation. Advantages of this procedure include: doesn’t require sedation (which is needed for colonoscopy); provides more detailed images compared with a barium enema; takes less time than either colonoscopy or barium enema; allows visualization of the inside of a colon that is narrowed due to inflammation or the presence of an abnormal growth. Disadvantages include: requires bowel preparation and insertion of a tube into the rectum for expanding the large intestine with gas or liquid; does not allow removal of tissue samples or polyps; does not reliably detect precancerous polyps smaller than 10 mm.

Upper gastrointestinal series (UGI): Includes an esophagram (see “Barium swallow” below) in addition to a study of the stomach and duodenum. It is a double-contrast study using barium and air and is useful for detection of gastritis, ulcers, masses, hiatal hernias, and gastroesophageal reflux. It is also an important part of the work-up of heme-positive stools and upper abdominal pain. Consult the radiologist for patient preparation.

Small bowel follow-through: Usually a direct extension of a UGI; represents a single-contrast examination of the small bowel. This is used in the work-up of diarrhea, malabsorption, upper gastrointestinal (GI) bleeding, and abdominal cramps.

Barium swallow (esophagram): Usually performed with barium, although a water-soluble contrast agent can be used to evaluate the swallowing mechanism and to look for esophageal lesions or abnormal peristalsis. No preparation is usually required for this study.
Intravenous pyelogram (IVP): Uses IV contrast medium to evaluate the kidneys, ureters, and bladder. Indications include hematuria, kidney stones, urinary tract infection, suspected malignancy, and flank pain. This is usually preceded by a KUB. Consult the radiologist for proper patient preparation. Currently, this study is being replaced by CT IVP (noncontrast), CT with contrast, or magnetic resonance imaging (MRI).

Cystogram: Bladder is filled and emptied with a Foley catheter in place. This study is used to evaluate for filling defects in the bladder and bladder perforation. No patient preparation is required.

Voiding cystourethrogram: Bladder is filled with contrast medium through a Foley catheter. The catheter is then removed, and the patient is asked to void. This study is used to look for abnormalities of the urethra and vesico-urethral reflux. No patient preparation is usually required for this study.

Retrograde urethrogram: Used to demonstrate urethral strictures and traumatic disruption of the urethra.

Retrograde pyelogram: Usually performed by a urologist. Contrast medium is injected into the ureters and collecting systems of the kidneys under cystoscopic guidance. Indications include a kidney and ureter that cannot be visualized by an IVP, filling defects in the collecting system, renal masses, urethral obstruction, or allergy to IV contrast medium.

T-tube cholangiogram: Some patients who have undergone gallbladder and biliary surgery are left with a T-tube in place in the common bile duct for drainage until postoperative swelling decreases. A cholangiogram can be performed through this T-tube to look for the patency of the biliary system.

Percutaneous transhepatic cholangiogram: Involves an invasive procedure, usually performed by the radiologist, who places a percutaneous needle into a dilated biliary duct under fluoroscopic control. This study is used to visualize the biliary tree in patients with an elevated bilirubin level (greater than 3 mg/dL).

Endoscopic retrograde cholangiopancreatogram (ERCP): Usually performed jointly by a gastroenterologist and radiologist. Contrast medium is endoscopically injected into the ampulla of Vater to visualize the common bile duct and the pancreatic duct.
Nuclear Scans
Cardiovascular disease: There have been remarkable advances in cardiovascular nuclear medicine over the past 40 years, with evidence-based outcomes validating its usefulness. Techniques to measure and quantify myocardial perfusion and viability, left ventricular ejection fraction (LVEF), and wall motion are widely used for the diagnosis of coronary artery disease (CAD) and significant anatomical stenosis and the determination of myocardial prognosis.

Pulmonary system and thromboembolism: Pulmonary embolism (PE) is a major health problem, with considerable controversy surrounding diagnostic approaches and their interpretation. The radionuclide lung scan is used because it is safe, readily available, easily performed, and sensitive for the diagnosis of PE. Although CT has become the major imaging procedure for PE, the radionuclide ventilation/perfusion lung scan (also called the V/Q scan) is still used frequently. Quantitation of pulmonary perfusion and ventilation is also helpful, especially when a right-to-left shunt is suspected.

Genitourinary system: Radionuclide renal scintigraphy provides important functional data to assist in the diagnosis and management of patients with a variety of suspected genitourinary problems. There are five different renal radiopharmaceuticals available and several different imaging protocols that look at flow, function, and urodynamics. Consultation with the nuclear medicine physician may help the referring physician order the most appropriate examination.

Hepatobiliary system: Nuclear medicine studies look at biliary dynamics. Thus, patency of the cystic duct in acute cholecystitis and biliary obstruction, leak, and atresia can be evaluated. Ultrasound, MRI, and CT are anatomical imaging modalities that diagnose gallstones easily and effectively.

Gastrointestinal (GI) system: Nuclear imaging of the GI system is most commonly used to localize acute lower tract hemorrhage and to quantify GI motility (gastric emptying). Ectopic gastric mucosa (Meckel diverticulum) can also be evaluated.

Infection imaging: Inflammatory and infectious processes can be occult and difficult to detect or localize clinically. MRI is very sensitive for detecting acute and, to a lesser extent, chronic infections in both bone and soft tissues.
and should be used as the primary imaging modality when the question is whether or not an infection is present. However, MRI is not very specific, particularly when imaging bone. For instance, it frequently cannot distinguish infection from infarct or a Charcot joint. When specificity in bone imaging is needed, a radiopharmaceutically tagged white blood cell (WBC) study is recommended for detecting acute osteomyelitis outside of the spine, and a gallium scan is recommended for detecting chronic osteomyelitis throughout the body as well as acute osteomyelitis of the spine.

Central nervous system: Nuclear medicine adds important information to the evaluation of cerebrovascular disease, seizures, dementia, brain death, brain tumors, and cerebrospinal fluid flow dynamics. Both positron emission tomography (PET) and single photon emission computed tomography (SPECT) have the resolution to be a powerful tool in such evaluations. The fusion of functional (nuclear medicine) images with anatomical images (ie, CT and MRI) enhances the diagnostic accuracy of the tests.

Skeletal system: Bone scanning evaluates both bone physiology and skeletal anatomy and is one of the most common applications of nuclear imaging. SPECT provides increased sensitivity and, combined with anatomical imaging detail, improves resolution and accurately helps in diagnosis. With continued advances in PET-CT and SPECT-CT, the role of nuclear imaging has become complementary to plain films, CT, and MRI in disease detection and patient management.

Endocrine imaging: Radionuclide evaluation of the thyroid can often contribute to the management of patients with diagnosed or suspected thyroid disease, both benign and malignant. The functional and structural information provided by a thyroid scan and uptake, combined with clinical information, allows for accurate diagnosis and a logical approach to therapy. Parathyroid imaging is used to localize a hyperfunctioning adenoma once the diagnosis has been confirmed biochemically. A variety of neuroendocrine tumors also accumulate different radiotracers and can be detected and localized by nuclear scans.

Oncologic imaging: Radioactive glucose (called FDG) is taken up by many tumors, including lymphoma, head and neck cancer, lung cancer, breast cancer, colorectal cancer, melanoma, and esophageal cancer; the list continues to grow. PET using FDG provides true molecular and functional
imaging. Its primary indications include accurate staging and monitoring the response to therapy.

**Ultrasound**

Ultrasound is noninvasive and relatively inexpensive. In addition, it eliminates radiation exposure. The following represent areas in which ultrasound has been used extensively.

**Abdomen**

*Kidneys:* Ultrasound is particularly helpful in detecting hydronephrosis and loss of renal parenchyma. Frequently, the cause of the hydronephrosis also can be determined.

*Gallbladder:* The first line in the evaluation of gallstones is currently ultrasound. Not only can this modality detect stones in the gallbladder, but it also can frequently detect stones elsewhere in the biliary system. Ultrasound is also useful in determining other forms of gallbladder disease, including acute cholecystitis.

*Pancreas:* Ultrasound can be used in evaluating for tumor, pancreatitis, and pseudocysts of the pancreas.

*Aorta:* Ultrasound can be used to grossly evaluate for aneurysm.

**Pelvis:** Ultrasound is principally used for evaluating urinary tract problems, including tumor and infection. It is also quite helpful in the evaluation of gynecologic problems such as ovarian and uterine masses, abscesses, and ectopic pregnancies. In most examinations of the pelvis it is helpful to have a full bladder.

**Chest:** Although not particularly helpful in evaluating lung fields, ultrasound can be used to localize a pleural effusion prior to thoracentesis.

**Obstetrics:** Ultrasound can be used for fetal dating, guiding amniocentesis, detection of fetal anomalies, localization of the placenta, and diagnosis of multiple gestations.

**Cardiac:** Currently echocardiography is the principal means of evaluating for
pericardial effusion, congenital heart abnormalities, and acquired valvular abnormalities.

Deep venous thrombosis (DVT): Doppler ultrasound currently is the modality of choice for detecting DVT of the lower extremities. However, it is inaccurate below the popliteal veins.

Musculoskeletal system: Ultrasound is being used more widely in the musculoskeletal system for detection of problems such as rotator cuff tears, tendinopathy, and joint effusions. It is also being used to guide joint aspiration and injections.

**Computed Tomography (CT) Scans**

In addition to its use in imaging the head and neck (see section “Imaging of the Head and Neck,” page 50), CT scanning has been used to evaluate just about every area of the body. It can be used with or without IV contrast medium; the radiologist in consultation with the clinician should decide when IV contrast medium is beneficial. In addition, when examining areas in the vicinity of the GI tract, dilute oral contrast medium can be used. Not only does CT scanning provide excellent anatomical information concerning most body parts, but it can also determine the vascularity of specific lesions.

Head: CT is used as one of the first studies in the evaluation of severe head trauma. It can detect bleeding, contusion, and fracture. It can also be used to evaluate intracranial tumors, hydrocephalus, or other abnormalities within the brain and surrounding structures. CT is the first imaging study that can be used in the evaluation of acute stroke, and CT whole brain perfusion studies are currently used to pinpoint major vessel occlusive disease.

Spine: CT can be used for evaluating disc abnormalities such as tumors and infections or hypertrophic bony changes such as spinal stenosis, although MRI is considered a better option. However, it is currently used as the imaging modality of choice in the evaluation of spine trauma, particularly the cervical spine.

Chest: CT is used for the work-up of abnormalities detected on the routine chest radiograph. In particular, it is excellent for evaluating and staging tumors. It is also useful for evaluating unusual infections, mediastinal abnormalities, and bony thoracic abnormalities. Today, CT is the imaging modality of choice for detecting pulmonary embolism. Only patients with a
contraindication to IV contrast agents undergo ventilation/perfusion nuclear medicine scans. Recently there have been studies looking at the efficacy of chest CT for the screening of lung cancer, but to date these studies have failed to prove its efficacy.

Cardiac: Noncontrast cardiac CT can be used as a screening examination for coronary artery disease. By measuring the amount of calcium in the arteries, radiologists can determine the patient’s relative risk for such disease. The “triple-rule-out” chest CT is quickly becoming a mainstay in the work-up of patients presenting to the emergency room with chest pain. This study helps rule out pulmonary embolism, coronary artery disease, and aortic dissection.

Abdomen: CT is the imaging modality of choice in evaluating almost all structures within the abdomen. However, MRI has made some inroads in evaluation of the abdomen, particularly the liver and the biliary system.

Retroperitoneum: CT is very helpful in evaluating the pancreas, especially in instances where ultrasound cannot visualize the pancreas because of overlying bowel gas. It has also been extremely helpful in evaluating the kidneys for abnormalities such as tumors.

Pelvis: CT is used extensively in the work-up and staging of cancer of the bladder, prostate, ovaries, cervix, and rectum.

Musculoskeletal systems: CT scanning has myriad uses within the musculoskeletal system, including delineation of complex fractures, CT arthrography (used when MRI is contraindicated), and evaluation of soft tissue masses (although MRI has generally supplanted CT in this area). In particular, the introduction of 3D reconstruction has proved to be very useful in the musculoskeletal system as well as in the head and neck.

**Magnetic Resonance Imaging (MRI)**

MRI has assumed a role of unparalleled importance in diagnostic medicine. MRI is unquestionably the imaging modality of choice for much of the pathology encountered in the central nervous system, including the spinal cord; the musculoskeletal system, including the spine and major joints; and the cardiovascular system. Use of MRI in other areas of the body is also rapidly advancing. Not only does MRI provide excellent anatomical detail without exposing the patient to harmful radiation, but also, with its varying pulse sequences, it permits some physiologic evaluation of pathologic
processes throughout the body.

Central nervous system/spine: MRI has revolutionized diagnosis within the central nervous system. It is now the imaging modality of choice for ruling out multiple sclerosis, evaluating central nervous system tumors and infections, and evaluating intervertebral discs and syringomyelia. However, CT is still preferred for evaluating bony abnormalities of the spine, including spinal stenosis.

Cardiac: MRI is efficacious in evaluating aortic dissections, aneurysms, and congenital heart disease and is finding new uses in the evaluation of ischemic heart disease.

Musculoskeletal: MRI is the imaging modality of choice in evaluating avascular necrosis; the extent of bone and soft tissue tumors; and the internal structures of joints, including the knee, shoulder, ankle, temporomandibular joint (TMJ), wrist, and hip. It is also useful in evaluating soft tissue infections, osteomyelitis, and other abnormalities of bone marrow.

## Imaging of the Head and Neck

### Plain Films

Plain films have been almost completely supplanted by CT. Only rarely are such views still used, especially if CT is readily available.

### Computed Tomography (CT)

Even with the availability of MRI, multidetector CT (MDCT) still plays a major role in diagnostic imaging of the head and neck. The major disadvantage of CT is the exposure to ionizing radiation. This is particularly problematic in patients who are receiving multiple CT studies. It is also problematic in the pediatric population because of the increased risk of radiation-induced cancers and cataracts. In certain instances, the CT study requires intravenous iodinated contrast administration; therefore, allergy to iodine and impaired renal function are concerns in certain patients.

Trauma: CT is the gold standard imaging modality for facial trauma. CT for facial trauma is performed without the use of IV contrast medium. CT, performed with high-resolution protocols and bone algorithm acquisition, offers exquisite detail of the osseous structures of the face and neck.
Multiplanar reformatted images, usually in the sagittal and coronal planes, allow visualization of fractures, TMJ luxation, ossicular chain luxation, and orbital extraocular muscle entrapment. Two-dimensional (2D) and 3D reconstructions provide surgeons with easily understandable road maps for the planning of reconstructive surgery in complex facial trauma. Additionally, CT is capable of demonstrating hemosinus, hemotympanum, and conditions associated with soft tissue trauma, such as edema, hematomas, soft tissue emphysema, and air within the orbit. CT of the face can usually be performed at the same time as the head and cervical spine study when the patient is admitted to the emergency room. CT following intrathecal injection of contrast agent (termed CT-CYSTERNOGRAPHY and usually achieved via lumbar puncture) can be performed to evaluate for cerebrospinal fluid (CSF) leaks from dural and bony defects of the skull base, mastoids, ethmoid plate, and sellar floor.

Foreign bodies: CT is the gold standard for visualizing foreign bodies in the soft tissues of the head and neck, in the orbits, and in the airways. Most foreign bodies are either radiopaque (hyperdense) or radiolucent (hypodense). However, some foreign bodies remain undetectable by CT.

Neoplasms and infections: CT is the mainstay in the diagnosis and follow-up of head and neck cancer. It similarly can be used for the diagnosis of infectious processes within this region. Scans are usually performed during or immediately following IV iodine contrast administration. However, the natural contrast offered in the head and neck region by the hypodense fat planes allows depiction of expansile and infiltrative masses and nodal disease. It also helps in the evaluation of major vascular and osseous structures as well as the airways. Osseous involvement is characterized on CT by cortical erosion or a periosteal reaction, which can be a relatively late phenomenon. MRI is more sensitive to early bone marrow infiltration. Finally, the conjoined use of CT and PET (PET-CT) has significantly increased the sensitivity for detection of areas of primary and nodal neoplastic disease in the head and neck.

After surgery and/or radiation treatment, the regional anatomy is often profoundly altered, and distinguishing between normal posttreatment changes and residual or recurrent disease can be particularly challenging. A baseline study after treatment serves as a useful comparison for further follow-ups.
Vascular changes: CT angiography (CTA) is a very accurate diagnostic modality to assess patency, caliber, and course of the major neck arterial and venous structures; for surgical planning; and to rule out traumatic dissection, critical stenoses, neoplastic vascular compression, encasement, and/or occlusion.

Salivary glands: CT without IV contrast is useful for depiction of sialolithiasis. Other pathology involving the ductal system requires preliminary cannulation of the duct and injection of a radiopaque contrast agent. CT with IV contrast administration is commonly used for the evaluation of salivary gland tumors, although MRI is considered the imaging modality of choice for this purpose.

TMJ: CT offers detailed evaluation of the bony parts of the TMJ. CT is not accurate for evaluation of the TMJ soft tissues.

Magnetic Resonance Imaging (MRI)

MRI has true multiplanar cross-sectional and multiparametric capabilities, which provide superior soft tissue resolution. MRI of the head and neck region can be reliably performed only with high-field-strength magnets (1.0 T or higher) and fat-saturation sequences, which enhance the signal from pathologic processes. Usually, MRI studies of the head and neck require the use of IV gadolinium contrast agents. MRI has the advantage of using nonionizing energies, but it cannot be performed in patients with electronic devices such as pacemakers or ferromagnetic implants and/or foreign bodies, which have the potential to dislodge and cause injury. Gadolinium-based contrast agents have a much better safety profile with respect to allergic reactions than do the iodinated agents used in CT imaging. However, they have been found to produce a devastating disease known as nephrogenic systemic fibrosis (NSF) if used in patients with severely impaired renal function. Therefore, these agents should not be administered to patients with glomerular filtration rates less than 15 mL/min/1.73m², unless they are absolutely necessary for the patient’s work-up. While, in principle, MRI is the gold standard for the evaluation of the head and neck soft tissues, it is a technically challenging examination. It requires immobility and cooperation from the patient for the duration of the examination (20 to 45 minutes), and its diagnostic value can be impaired by frequent swallowing and heavy
breathing.

Trauma: MRI is not the modality of choice in evaluation of head and neck trauma but can be used as a complementary tool to CT in very selected cases such as posttraumatic nasal encephaloceles, mastoid fractures with CSF fistulas, and optic nerve injuries.

Neoplasms and infections: MRI with IV contrast administration is the gold standard imaging modality for the assessment of soft tissue tumors of the head and neck. It offers superior characterization of infiltration of surrounding structures, nodal disease, perineural spread of disease, intracranial extension, and early infiltration of the bone marrow of the skull base. It can be used to image infectious processes as well. In the assessment of bony involvement, MRI is more sensitive than CT and bone scintigraphy but requires rigorous technique. In practical terms CT is more commonly used for patient follow-up because of its lower cost, greater speed and availability, and its ability to produce standardized reproducible results.

Vascular changes: MR angiography (MRA), like CTA, is a very accurate diagnostic modality to assess patency, caliber, and course of the major neck arterial and venous structures. It is performed during a power-injected, IV bolus of a gadolinium-based contrast agent.

TMJ: MRI is considered the imaging modality of choice in the overall evaluation of TMJ pathology. In particular, it provides exquisite visualization of the disc and allows for detection of effusion within the joint. It also allows for evaluation of the medullary cavity of the mandibular condyle and permits detection of avascular necrosis. Finally, with use of gradient echo sequences, pseudodynamic depiction of TMJ movement during mouth opening is possible. However, MRI is not as effective as CT in the evaluation of degenerative bony changes in the joint.
Interpretation of the Electrocardiogram

The interpretation of data derived from an electrocardiogram (ECG) is essential in the management of medical emergencies, the performance of anesthesia, and the proper presurgical work-up of patients. This chapter discusses an orderly method of rapid ECG interpretation and provides examples of the most common abnormalities.

Electrical Pathway
Impulses begin in the sinoatrial (SA) node high in the posterior wall of the right atrium. The resultant stimulus causes a wave of atrial contraction. The impulse continues into the atrioventricular (AV) node where there is an approximate one-tenth-second delay before the AV node is stimulated. The impulse passes down the bundle of His (common bundle) and the right and left bundle branches. This begins the wave of ventricular contraction. The impulse then terminates in the Purkinje fibers, causing rapid depolarization and complete contraction of the ventricle (Fig 5-1).
Fig 5-1 Cardiac anatomy. RA, right atrium; LA, left atrium; RV, right ventricle; LV, left ventricle.

**Normal ECG Wave (Fig 5-2)**

P Wave
- Represents electrical activity of atrial depolarization and contraction
- Originates in the SA node
- Is characterized by repolarization that is usually very small or obscured by other waves
Fig 5-2 Normal ECG wave.

PR Interval
- Represents the lag in electrical conduction through the AV node
- Is normally less than 0.2 seconds in duration
- Allows time for ventricular filling
- If elongated, indicates AV node disease

QRS Complex
- Represents the electrical activity of ventricular depolarization and contraction
- Is normally less than 0.12 seconds in duration
- If widened, indicates (in most cases) conduction abnormalities of the bundle of His or its divisions (ie, bundle branch block)

ST Segment
- Represents a period of apparent electrical inactivity between ventricular depolarization and repolarization
- Is usually isoelectric
- If baseline is elevated or depressed, usually indicates injury to the myocardium

T Wave
- Represents electrical repolarization of the ventricles
- Is not associated with any physical event

QT Interval
- Represents total time of ventricular depolarization, contraction, and repolarization
- If shortened or prolonged, may represent electrolyte disturbances or drug effects

Lead Placement
The purpose of the ECG is to measure electrical potential differences between two leads. By measuring the electrical potentials of the cardiac
impulse using several leads at different angles, the axis (or vector) of the impulse and many disease states of the heart can be determined. The leads are divided into limb leads and chest leads.

Limb Leads
These measure the impulse in the frontal plane.

Placement (Fig 5-3)
- Right arm
- Left arm
- Left leg

Significance
- By using these three leads (I, II, III, with the polarity as shown by the triangle in Fig 5-3), the impulse can be defined relative to three known vectors. Impulses that travel parallel to a lead will have a positive deflection in that lead. Impulses that travel opposite to the direction of a lead will have a negative deflection.
- By averaging two leads with a common ground (done automatically by the ECG machine), three more vectors using the same extremity electrodes can be used. These are termed aVR, aVL, and aVF and are perpendicular to leads I, II, and III (Fig 5-4).
- These vectors allow determination of the direction of the cardiac impulse in the frontal plane. The more positive the QRS deflection in any lead, the more closely the impulse follows the direction of that lead.
Fig 5-3 Limb lead placement.

Fig 5-4 Frontal (limb) leads.

Figs 5-5a and 5-5b Chest leads.
Chest Leads (Figs 5-5a and 5-5b)

Placement
- $V_1$: Right of sternum in fourth intercostal space (ICS)
- $V_2$: Left of sternum in fourth ICS
- $V_3$: Midway between $V_2$ and $V_4$
- $V_4$: Midclavicular line in fifth ICS
- $V_5$: Midway between $V_4$ and $V_6$
- $V_6$: Lateral chest in fifth ICS

Significance
- By using the chest leads, the direction of the impulse in the horizontal (or axial) plane may be determined.

**ECG Abnormalities**
There are five factors to consider:
- Rate
- Rhythm
- Axis
- Hypertrophy
- Infarction
Rate
Techniques for determination

*Grid method (Fig 5-6)*
- Dark lines = 0.2 seconds
- Light lines = 0.04 seconds

*Fig 5-6 Grid method for determining rate. Note that next R wave falls between dark lines representing 75 BPM and 100 BPM (or approximately 80 BPM).*

*Fig 5-7 Scan method for determining rate.*

- Find R wave near dark line.
- Count dark lines until next R wave.
- Use logarithmic progression for each dark line starting with 300, then 150, 100, 75, 60, 50 (ie, \( \frac{300}{2}, \frac{150}{2}, \frac{75}{2}, \frac{60}{2}, \frac{50}{2} \) etc).
- Interpolate rate if R wave falls between two dark lines.
Scan method
- Note 3-second markers on ECG strip (Fig 5-7).
- Measure number of complexes (or RR intervals) between two markers (6 seconds) and multiply by 10.
- Advantages
  - More accurate at extremely fast or slow rates
  - More accurate in irregular rhythms

Normal rates: Normal impulses originate in the SA node and are regulated by the autonomic nervous system. However, each tissue has its own intrinsic rate, which will take over if no impulse occurs from a more proximal tissue.
- SA node: 60 to 100 beats per minute (BPM)
- AV node: 60 BPM
- Ventricles: 30 to 40 BPM

Abnormalities of rate
- Sinus tachycardia: Rate > 100 BPM
- Sinus bradycardia: Rate < 60 BPM
- Emergency rates (atrial, ventricular): 150 to 250 BPM

Fig 5-8 Normal sinus rhythm.

Rhythm
Sinus rhythm (Fig 5-8)
**ECG characteristics**
- Rate: 60 to 100 BPM (higher in children)
- P wave: Consistent configuration
- PR interval: Normal (0.12 to 0.20 seconds)
- QRS complex: Normal (0.04 to 0.12 seconds)
- Rhythm: Regular
- Origin: Sinus node

**Pertinent data/clinical relevance**
- Normal sinus rhythm (NSR) may not exclude underlying heart disease
- Clinical relevance: Normal state

*Treatment:* None

Sinus arrhythmia (Fig 5-9)

**ECG characteristics**
- Rate: Normal (60 to 100 BPM)
- P wave: Normal, consistent
- PR interval: Normal
- QRS complex: Normal
- Rhythm: Irregular
- Origin: Sinus node

**Pertinent data/clinical relevance**
- May or may not be associated with respiration
- Usually considered a normal variant
- May, however, occur in diseased hearts (especially immediately postmyocardial infarction)

*Treatment:* None usually needed
Premature atrial contractions (PACs) (Fig 5-10)

**ECG characteristics**
- Rate: Varies due to varying number of PACs, but usually normal (50 to 100 BPM).
- P wave: Differs from usual P wave. Altered wave may be upright, inverted, or biphasic.
- PR interval: Differs from normal PR interval. Usually less than normal.
- QRS complex: Normal.
- Rhythm: Basic rhythm is normal sinus rhythm, except for occasional prematurity (irregularly irregular). Frequency varies.
- Origin: Ectopic focus in atria.

**Pertinent data/clinical relevance**
- May be unifocal or multifocal (with different P waves for each focus).
- Usually see a noncompensatory pause following the ectopic beat.
- May occur in healthy hearts when stimulated by caffeine, drugs, stress, alcohol, hypoxia, and metabolic abnormalities.
- May occur in patients with coronary or rheumatic heart disease, lung disease, or mitral stenosis.
- Conduction through AV node and ventricle usually normal, but may be nonconducted or blocked.

_Treatment:_ If treatment is necessary, atropine or another anticholinergic drug may be given.

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**Fig 5-11 Sinus tachycardia.**

Sinus tachycardia (Fig 5-11)

**ECG characteristics**
- Rate: Greater than 100 BPM
- P wave: Normal
- PR interval: Normal
- QRS complex: Normal
- Rhythm: Regular sinus rhythm except for fast rate
- Origin: SA node

**Pertinent data/clinical relevance**
A normal physiologic response to endogenous catecholamine release or exogenous sympathomimetic amines (eg, epinephrine).

Also seen with anxiety and emotional states, exercise, febrile conditions, anemia, hypotension, and myocardial infarction (MI).

Usually a benign condition. May be dangerous in patients with coronary heart disease because of increased demand for myocardial oxygen and decreased diastolic perfusion time.

May cause dyspnea, angina, and palpitations in some patients.

_Treatment:_ When treatment is necessary, it is aimed at eliminating the underlying cause.

Supraventricular tachycardia (SVT) (**Fig 5-12**)

**ECG characteristics**

- **Rate:** Greater than 100 BPM, usually 150 to 250 BPM.
- **P wave:** Differs from usual P wave. May be obscured in previous T wave or absent altogether.
- **PR interval:** Variable. Usually not measurable due to high rate.
- **QRS complex:** Normal.
- **Rhythm:** Rapid, regular rhythm. Usually the tachycardia is paroxysmal and interposed with sinus rhythm.
- **Origin:** Atrial ectopic focus (unifocal or multifocal) or AV node.

**Pertinent data/clinical relevance**

- Two types: Tachycardia may be atrial or nodal, but often it is impossible to distinguish between the two, and therefore the term _supraventricular tachycardia_ is used.
Supraventricular tachycardia.

- Often associated with congenital, coronary, and rheumatic heart disease; mitral valve prolapse; and hypertension.
- Digitalis toxicity may manifest as SVT with AV block.
- May cause angina, palpitation, dizziness, and weakness.

Treatment
- Vagal maneuvers, including yawning, Valsalva maneuver, breath holding, and carotid massage
- Medical therapy includes beta-blockers (eg, propranolol), and calcium channel antagonists (eg, verapamil, adenosine)
- Synchronized cardioversion

Atrial fibrillation (Fig 5-13)

ECG characteristics
• Rate: Variable, but most patients commonly have an atrial rate of 300 to 500 BPM with a ventricular capture rate of 150 to 180 BPM.
• P wave: Not well visualized. P waves are seen as constant coarse or fine undulations in the baseline between complexes. Each P wave differs from the next.
• PR interval: Variable, or more commonly, impossible to distinguish.
• QRS complex: Normal.
• Rhythm: Ventricular capture rate is irregularly irregular.
• Origin: Multiple atrial ectopic foci.

**Pertinent data/clinical relevance**

• May be paroxysmal or chronic
• Chronic form usually caused by heart disease, including coronary heart disease, hypertension, cardiomyopathy, thyroid disease, and mitral valve stenosis
• Usually have loss of atrial contraction (“atrial kick”) with associated loss of cardiac output
• Stasis of blood often leads to pulmonary and systemic emboli

**Treatment:** Generally aimed at decreasing the ventricular capture rate or converting the rhythm to normal sinus rhythm

• Drugs to slow the rate to less than approximately 100 BPM (eg, digoxin, propranolol, calcium channel blockers such as diltiazem)
• Drugs to suppress the fibrillation (eg, amiodarone, sotalol, quinidine)
• Synchronized cardioversion
• Anticoagulation
ECG characteristics

- Rate: Atrial rate variable, averaging 220 to 350 BPM. The ventricular rate is also variable, depending on the conduction through the AV node, and usually ranges from 100 to 220 BPM (the faster rates often are associated with 2:1, 3:1, or 4:1 AV blocks).
- P wave: The P waves are visualized as multiple “sawtooth” flutter waves (“F” waves). Depending on the degree of AV conduction, several may be seen before conduction to the ventricles.
- PR interval: Usually regular, but may vary if AV conduction varies.
- QRS complex: Usually normal.
- Rhythm: The atrial rhythm is usually regular. The ventricular rate may be regular or irregular depending on the variability of the ventricular conduction.
- Origin: A solitary ectopic focus in the atria.

Pertinent data/clinical significance

- Seldom occurs in the absence of heart disease
- Commonly seen in association with rheumatic heart disease (especially mitral and tricuspid valvular disease), coronary heart disease, acute or chronic cor pulmonale, and hypertension
- Rarely induced by digitalis intoxication
- May degenerate into atrial fibrillation

Treatment: Usually aimed at converting the rhythm to a more stable one or decreasing the ventricular capture rate
- Drug therapy (verapamil, digitalis, propranolol, diltiazem)
- Synchronized cardioversion if associated with hypotension or congestive heart failure

*Fig 5-15 Nodal rhythm. Arrow indicates retrograde P wave.*

Junctional or nodal rhythm *(Fig 5-15)*

**ECG characteristics**
- Rate: 40 to 60 BPM (may also see accelerated nodal rhythm at 60 to 100 BPM).
- P wave: Usually absent but occasionally will be retrograde, inverted, or follow QRS complex.
- PR interval: Usually not applicable, but if a P wave is present and inverted, then PR interval will be less than normal.
- QRS complex: Normal.
- Rhythm: Very regular.
- Origin: AV node.

**Pertinent data/clinical relevance**
When impulse from SA node or atrium does not reach the AV node in time, it will take over as the primary pacemaker. Causes include MI, metabolic abnormalities, vagal stimulation, and digitalis intoxication. Loss of “atrial kick” decreases cardiac output. Often seen in healthy heart during anesthesia.

Treatment
- Remove underlying cause.
- Atropine to increase rate if symptoms occur.

First-degree AV block (Fig 5-16)

**ECG characteristics**
- Rate: Normal
- P wave: Normal
- PR interval: Greater than normal (> 0.2 seconds); usually constant but occasionally varies

*Fig 5-16 First-degree AV block. Arrow indicates prolonged PR interval.*

- QRS complex: Normal
- Rhythm: Normal, regular (unless PR interval varies)
- Origin: SA node

**Pertinent data/clinical relevance**
- Common conduction abnormality.
May be seen in healthy heart because of vagal tone, digitalis intoxication, or advancing age. May also be associated with MI, atrial septal defects, or valvular heart disease.

Rarely progresses to higher-degree block.

**Treatment:** Determine underlying cause.

**Fig 5-17** Second-degree AV block, type I. Brackets indicate increasing PR intervals; arrow indicates P wave that is not conducted to ventricles ("dropped beat").

Second-degree AV block, Mobitz type I (Wenckebach phenomenon) (**Fig 5-17**)

**ECG characteristics**

- Rate: May be normal or often slow (< 50 BPM).
- P wave: Normal; however, not all P waves are conducted.
- PR interval: Progressive prolongation of PR interval until a QRS complex is dropped.
- QRS complex: Normal except that occasional complexes are dropped.
- Rhythm: May be regularly irregular (if conduction block is fixed) or irregularly irregular (if variable block is present).
- Origin: SA node.

**Pertinent data/clinical relevance**

- Basic blockage is in AV node.
May be seen in normal hearts because of vagal stimulation, medications (digitalis, propranolol), infection, uremia, or metabolic abnormalities. Also seen in MI and ischemia.

May progress to bradycardia or higher-degree AV block.

Produces symptoms when bradycardia occurs.

**Treatment**

- Atropine to correct bradycardia, if needed
- Pacemaker

**Fig 5-18** Second-degree AV block, type II. Arrows indicate intermittent nonconducted P waves.

Second-degree AV block, Mobitz type II (Fig 5-18)

**ECG characteristics**

- Rate: Atrial rate usually normal, but ventricular rate slower by 2:1 or 3:1 margin because of conduction block; quite commonly less than 50 BPM.
- P wave: Normal except for high degree of nonconduction.
- PR interval: When conducted to the ventricles, the PR interval is usually normal or slightly prolonged.
- QRS complex: When present, the QRS is usually normal or sometimes shows evidence of bundle branch block.
- Rhythm: Usually regular but may be irregularly irregular if the block varies.
- Origin: SA node.
**Pertinent data/clinical relevance**

- Indicates severe conduction system disease, which may progress into complete heart block (third degree).
- When block is 3:1 or greater, it is often called “advanced” or “high-degree” AV block.
- Because of the slow rates, premature ectopic beats are common.
- Symptoms are common and are related to the slow rate and diminished cardiac output (syncope, dizziness).

**Treatment**

- Atropine to increase rate, if needed
- Pacemaker usually required

---

**Fig 5-19** Third-degree AV block. Note regular pattern of P waves (some of which are buried in other waves), but independent pattern of QRS complexes.

Third-degree AV block (complete heart block) (Fig 5-19)

**ECG characteristics**

- Rate: Atrial rate usually normal. Ventricular rate may vary between 20 to 60 BPM (depending on whether pacing is from AV node or ventricle itself).
- P wave: Normal appearance but independent of QRS (ie, no conduction to ventricles).
- PR interval: Because there is no conduction to the ventricles, there is technically no PR interval. It will appear that there is a variable PR interval, however, when QRS complexes occur near P waves.
- QRS complex: May be normal if nodal escape rhythm, or wide and bizarre if ventricular escape rhythm.
- Rhythm: Usually slow and regular.
- Origin: P wave from SA node. QRS may come from AV node (if nodal escape) or ventricles (if ventricular escape).

**Pertinent data/clinical relevance**
- Almost always indicates significant conduction system disease, especially if block is infranodal.
- Causes include coronary heart disease, MI, infection, metabolic disturbance, trauma, or drug effect (phenytoin, digitalis, quinidine).
- Symptoms (syncope, hypotension) are caused by the slow rate and diminished cardiac output.
- An unstable rhythm. Tachyarrhythmias and episodes of asystole are common.

**Treatment**
- Atropine to increase rate if needed; however, this may be ineffective in wide-complex blocks and should be used with caution in patients with an MI.
- Pacemaker almost always required.
Bundle branch block (BBB) (interventricular conduction defect) (Fig 5-20)

**ECG characteristics**
- Rate: Normal.
- P wave: Normal.
- PR interval: Normal.
- QRS complex: Widened, greater than 0.12 seconds, with two peaks to the R wave, termed R and R’ (“R prime”).
- Rhythm: Usually regular unless combined with another arrhythmia.
- Origin: SA node.

**Pertinent data/clinical relevance**
- Impulse normal to level of bundle of His. Impulse must then travel around blockage to ventricles, causing wide QRS complex.
- RBBB more common than LBBB.
- May occur in healthy patients (R > L) but usually associated with organic heart disease, MI, systemic and pulmonary hypertension, valvular disease, pulmonary embolism, or chronic obstructive pulmonary disease (COPD).

**Treatment**
- Treat underlying medical problem.
- Pacemaker occasionally necessary.

Premature ventricular contractions (PVCs) (Fig 5-21)

**ECG characteristics**
- Rate: Usually normal.
- P wave: Usually indistinguishable or hidden by QRS.
PR interval: Not measurable.
QRS complex: Prolonged, greater than 0.12 seconds, and often showing LBBB pattern. Often bizarre and inverted.
Rhythm: Usually irregularly irregular because of extra systoles. May be regularly irregular if bigeminy or trigeminy pattern.
Origin: Ventricles.

![Fig 5-21](image) *Premature ventricular contractions. Arrows indicate premature complexes.*

**Pertinent data/clinical relevance**
- May occur in healthy individuals or from underlying organic heart disease.
- Causes include caffeine ingestion; smoking; anxiety; ischemic, valvular, or hypertensive heart disease; drug toxicity; and metabolic abnormalities.
- May be unifocal or multifocal.
- Main symptom is palpitation.

**Treatment**
- Treating the underlying cause often suffices if there is no association with an MI.
- In the face of an acute MI, the indications for treatment include: more than five PVCs per minute; multifocal PVCs; couplets (two PCVs in a row); a PVC that begins on the preceding T wave (“R on T” phenomenon).
- Treatment includes intravenous lidocaine, procainamide, or amiodarone. Successful bolus injection of one of these drugs
should be followed by an intravenous drip of the same drug to maintain suppressive therapy.

**Fig 5-22 Ventricular tachycardia.**

Ventricular tachycardia (Fig 5-22)

**ECG characteristics**
- Rate: Variable, but usually in the range of 100 to 220 BPM.
- P wave: Usually indistinguishable.
- PR interval: Not measurable.
- QRS complex: Wide (> 0.12 seconds), bizarre, often inverted; may have LBBB pattern.
- Rhythm: Usually regular and fast.
- Origin: Ventriles.

**Pertinent data/clinical relevance**
- May be paroxysmal or sustained.
- Almost always a sign of significant heart disease (particularly MI).
- Signs and symptoms range from dizziness and palpitations to severe hypotensive and cardiovascular collapse.
- An unstable rhythm, which can degenerate into ventricular fibrillation.

**Treatment**
If pulse is present and stable, antiarrhythmics such as lidocaine, procainamide, amiodarone, or sotalol can be given.
If refractory or unstable, use cardioversion or amiodarone.
If no pulse is present, administer cardiopulmonary resuscitation (CPR), defibrillation, and antiarrhythmic therapy when converted.

Fig 5-23 Ventricular fibrillation.

Ventricular fibrillation (Fig 5-23)

**ECG characteristics**
- Rate: Not calculable.
- P wave: Missing.
- PR interval: Missing.
- QRS complex: No actual complex. The only wave forms visible are random undulations in the baseline.
- Rhythm: Irregularly irregular and without a pulse.
- Origin: Ventricles.

**Pertinent data/clinical relevance**
- May be fine or coarse. Must differentiate from 60-cycle interference on ECG.
- There is no effective cardiac output (ie, cardiac arrest/sudden death).
• May arise de novo or from degeneration of another rhythm.

_Treatment_ (see also chapter 11)
- CPR to begin advanced cardiac life support [ACLS] protocol
- Defibrillation
- Epinephrine or vasopressin
- Lidocaine, amiodarone as antiarrythmics

*Fig 5-24 Asystole.*

Ventricular standstill (asystole) (*Fig 5-24)*

**ECG characteristics**
- Rate: Either nonexistent or only rare, occasional complexes
- P wave: Only rarely present and nonconducted
- PR interval: Not present
- QRS complex: Not present
- Rhythm: No rhythm seen; no pulse present
- Origin: None

**Pertinent data/clinical relevance**
- Results when intrinsic pacemakers fail.
- May be short term or terminal.
- Causes include hypoxia, severe bleeding, anaphylaxis, anesthetic/drug overdose.
- No cardiac output (true cardiac arrest).
- Asystole is a very difficult rhythm to convert.

**Treatment**
- CPR (to begin ACLS protocol)
- Atropine
- Epinephrine or vasopressin
- Pacemaker

**Axis**

The axis of the heart is the summation of the vectors of the electrical impulse. Essentially, this provides the orientation of the impulse and the heart within the body. Because of the large size of the left ventricle, the axis is normally downward and to the left.

Determination: Use positive and negative deflections of leads I and aVF to determine lateral and vertical vectors, respectively. The more positive the deflection in each of these leads, the more the axis follows the direction of that lead (ie, right to left for lead I and superior to inferior for lead aVF). Further specificity of the axis may be obtained using other leads (Fig 5-25).

*Fig 5-25* Axis of the cardiac vector. Normal vector should be between 0 degrees and +90 degrees. RAD, right axis deviation; LAD, left axis deviation.
Normal axis

- QRS deflection: Positive in leads I and aVF
- Clinical correlation: Normal

Left axis deviation

- QRS deflection: Positive in lead I and negative in aVF
- Clinical correlation: Left ventricular hypertrophy or bundle branch block

Right axis deviation

- QRS deflection: Negative in lead I and positive in aVF
- Clinical correlation: Right ventricular hypertrophy or bundle branch block

Extreme right axis deviation

- QRS deflection: Negative in lead I and in aVF

Hypertrophy

Left atrial hypertrophy

- Biphasic P wave in V₁ with wide, large terminal portion
- Notched P wave in leads I and II

Clinical correlation: May indicate effects of left ventricular hypertrophy or mitral stenosis

Right atrial hypertrophy

- Biphasic P wave in V₁ with wide, large initial portion
- Tall, peaked P waves in leads II and III

Clinical correlation: COPD, pulmonary hypertension
Left ventricular hypertrophy
- Deep S wave in V₁ and tall R wave in V₅ totaling greater than 35 mm.
- T waves in V₅ or V₆ may be inverted with gentle down slope and rapid up slope.

  Clinical correlation: Hypertension, aortic valvular disease, congenital heart defects

Right ventricular hypertrophy
- S wave is smaller than the R wave in V₁.
- R wave grows progressively smaller from V₁ to V₆.
- T wave inversion and ST segment depression in V₁ to V₃.

  Clinical correlation: COPD, mitral stenosis, congenital heart defects

Infarction
Ischemia: Symmetrically inverted T waves in the leads nearest the part of the heart affected, most commonly in the chest leads. May also see ST segment depression.

  Clinical correlation: Inadequate myocardial oxygen supply

Injury: Injury to the myocardium is indicated by ST segment elevation and tall, positive T waves.

  Clinical correlation: Aids in determination of age of an infarction and therefore whether acute treatment is necessary

Infarction: Significant Q waves that are 0.04 seconds or longer (one small
box on ECG strip), or greater than one-third the size of the entire QRS complex, indicate infarction. Specific leads indicate where the infarction has occurred:

- Anterior: Q waves in $V_1$, $V_2$
- Inferior: Q waves in II, III, aVF
- Lateral: Q waves in I, aVL, $V_5$, $V_6$
- Posterior: Q waves in $V_6$; large R waves in $V_1$, $V_2$; ST segment depression in $V_1$, $V_2$

**Clinical correlation:** Infarction indicates actual damage to the heart muscle. Specific areas and specific feeding coronary arteries can be isolated.

Miscellaneous Effects

Electrolytes

*Hypokalemia:* Presence of U waves (small positive deflection following the T wave). May also see ST depression and flat T wave.

*Hyperkalemia:* Tall peaked T waves, QRS widening, P wave flattening.

*Hypocalcemia:* Prolonged QT interval.

*Hypercalcemia:* Shortened QT interval.

Drugs

*Digitalis toxicity:* SA and AV node blocks, tachycardias, PVCs, ventricular tachycardia, and atrial fibrillation. There will be a slow downward sloping of the ST segment.

*Quinidine:* Wide notched P waves, prolonged QT intervals,
widened QRS complexes, ST depression, flat T waves, and the presence of V waves.

Pericarditis: ST segment elevation that is flat or concave. Elevated T waves.
chapter 6 Management of Fluids and Electrolytes

Perioperative management of fluids and electrolytes is one of the most basic and essential components of patient care. It is imperative to provide adequate preoperative stabilization before general anesthesia to prevent hypotension, renal failure, cardiac dysrhythmias, and other potential intraoperative complications. Postoperative fluid and electrolyte levels must be closely monitored, and management should be modified, sometimes on an hourly basis, if complications such as sepsis, hypotension, urinary retention, or endocrine disorders develop and alter the expected postoperative course. A thorough understanding of fluid and electrolyte balance is necessary for the proper management of the surgical patient.

Basic Considerations

Definitions

Mole (mol): The gram molecular weight of a substance (the molecular weight in grams). One millimole (mmol) is equal to 1/1,000 mol.

Equivalent (Eq): The amount of a substance that will combine with one atomic weight of hydrogen. One equivalent is the atomic weight or molecular weight divided by the ionic valance. A milliequivalent (mEq) is one thousandth of an equivalent.

Osmole (Osm): A measure of osmotic pressure; the drawing power a substance exerts on water. An osmotic effect of a substance in solution depends on the number of particles dissolved, independent of weight, electrical charge, valence, or chemical formula.

1 mol = 1 Osm
1 mmol = 1 mOsm

Concentrations of Solutes in Solution
Percent: Per hundred = units per 100 mL of solution. Unless otherwise specified, units refer to grams. Many laboratories report values in mg%, redefining units to mg/100 mL.

Molality: Concentration of a solute expressed as mol/1,000 g of solvent (a measure of mass per mass).

Molarity: Number of moles of solute per liter of solution at a specified temperature (a measure of mass per volume).

The difference between molal and molar concentrations is negligible in the range of concentrations and temperatures of body fluids; therefore, for all practical purposes molal = molar and osmolality = osmolarity.

Total Body Water
Total body water (TBW) (average):

- Adult man       60%
- Adult woman     55%
- Child           65%
- Infant          65%
- Newborn         75%

Most clinical calculations use 60% TBW for adults and 75% for children and infants. TBW decreases with age and obesity.

Total body water is separated into multiple compartments:
In a 70-kg male patient:

- **TBW** = 60% of body wt (kg) or 42 L
- **ICF** = 40% of body wt (kg) or 28 L
- **ECF** = 20% of body wt (kg) or 14 L
  - Extravascular fluid = 15% of body wt (kg) or 10.5 L
  - Intravascular fluid = 5% of body wt (kg) or 3.5 L

**Blood Volume**

- Blood volume (BV) = Plasma volume + Red blood cell volume
  - = 8% of body wt
  - = 5.6 L (approximately)

In each compartment, electrical neutrality exists; however, the total number of positive and negative ions within the compartment may be different (Table 6-1).

The difference between interstitial and intravascular fluid (both are ECFs) is the presence of formed elements (red blood cells, white blood cells, platelets) and a higher concentration of proteins in intravascular fluids. This produces a difference in the colloid osmotic pressure, which maintains the plasma fluid within the intravascular compartment.
Fluids
Goals of Fluid Management
1. Attain and maintain normal body composition and homeostasis
2. Correct life-threatening imbalances
3. Avoid complications of too-rapid correction
4. Integrate fluid and electrolyte therapy with nutritional therapy

Table 6-1 Composition of ECF and ICF

<table>
<thead>
<tr>
<th>Substance</th>
<th>ECF</th>
<th>ICF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na(^+) (mEq/L)</td>
<td>142</td>
<td>10</td>
</tr>
<tr>
<td>K(^+) (mEq/L)</td>
<td>5</td>
<td>141</td>
</tr>
<tr>
<td>Ca(^{++}) (mEq/L)</td>
<td>5</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Mg(^{++}) (mEq/L)</td>
<td>3</td>
<td>58</td>
</tr>
<tr>
<td>Cl(^-) (mEq/L)</td>
<td>103</td>
<td>4</td>
</tr>
<tr>
<td>HCO(_3^{-}) (mEq/L)</td>
<td>28</td>
<td>10</td>
</tr>
<tr>
<td>Phosphate (mEq/L)</td>
<td>4</td>
<td>75</td>
</tr>
<tr>
<td>Glucose (mg%)</td>
<td>90</td>
<td>0–20</td>
</tr>
</tbody>
</table>

5. Keep fluid orders clear and simple regarding:
   a. Type of fluid
   b. Rate and duration of infusion
   c. Added electrolytes
6. Minimize expense by:
   a. Ordering stock solutions whenever possible
   b. Ordering larger sizes whenever possible

Clinical Approach
1. Identify fluid and electrolyte imbalances and their magnitude.
2. Determine which problems need correction prior to surgery.
3. Determine the daily maintenance of fluids and electrolytes.
5. Evaluate for renal, cardiac, endocrine, and hepatic dysfunction.

To address these questions requires an understanding of a patient’s normal daily fluid gains and losses (Table 6-2), determination of ongoing fluid and electrolyte losses from various sources (Table 6-3), a thorough history and clinical evaluation, and laboratory testing.

Cardiovascular, renal, endocrine, and hepatic disease may significantly alter fluid and electrolyte replacement.

Water losses and gains are regulated by osmoreceptors of the central nervous system (CNS) that stimulate thirst and by antidiuretic hormone (ADH) secreted by the pituitary gland. Thirst occurs when the patient’s serum sodium increases by 4 mEq/L. ADH levels become elevated in states of pain, stress, injury, or surgery, and this increases water reabsorption by the kidney.

**Table 6-2 Normal sensible and insensible losses and gains of water**

<table>
<thead>
<tr>
<th>Route</th>
<th>Average daily volume (mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water gain</td>
<td></td>
</tr>
<tr>
<td>Sensible</td>
<td></td>
</tr>
<tr>
<td>Oral fluids</td>
<td>800–1500</td>
</tr>
</tbody>
</table>
Solid foods 500–700

Insensible

Water of oxidation 250

Water of solution Varies

Water loss

Sensible

Urine 800–1500

Intestinal 0–250

Sweat Varies

Insensible

Lung and skin 600

Table 6-3 Fluid and electrolyte losses from various sources

<table>
<thead>
<tr>
<th>Fluid</th>
<th>Electrolytes*</th>
<th>Average volume (mL/24 h)</th>
<th>Replacement fluid (mL/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salivary</td>
<td>10 K: 26 Cl: 10 HCO$_3$: 30</td>
<td>1500</td>
<td>NS or LR</td>
</tr>
<tr>
<td>Stomach</td>
<td>70 K: 20 Cl: 100 HCO$_3$: 0</td>
<td>1500</td>
<td>0.45 NS with 20 mEq KCL</td>
</tr>
<tr>
<td>Small bowel</td>
<td>150 K: 5 Cl: 100 HCO$_3$: 40</td>
<td>3000</td>
<td></td>
</tr>
<tr>
<td>Biliary</td>
<td>150 K: 5 Cl: 100 HCO$_3$: 40</td>
<td>20–800</td>
<td>LR</td>
</tr>
<tr>
<td>Pancreas</td>
<td>150 K: 5 — HCO$_3$: 55</td>
<td>100–800</td>
<td></td>
</tr>
<tr>
<td>Colon</td>
<td>60 K: 30 Cl: 40 —</td>
<td>—</td>
<td></td>
</tr>
</tbody>
</table>

*Values in mEq/L of fluid.
NS = normal saline; LR = lactated Ringer solution.

Physical Evaluation
Clinical signs and symptoms of fluid imbalance by system

**Central nervous system**
- **Deficit:** Sleepiness, apathy, slow responses, anorexia, vomiting, decreased tendon reflexes, anesthetic distal extremities, stupor, coma
- **Excess:** None

**Gastrointestinal**
- **Deficit:** Progressive decrease in food consumption, nausea, vomiting, refusal to eat, ileus and distention, diarrhea
- **Excess:** Edema of tissues at surgery

**Cardiovascular**
- **Deficit:** Orthostatic hypotension, tachycardia, collapsed peripheral and central veins, weak pulse, distant heart sounds, cold extremities, absent peripheral pulses
- **Excess:** Elevated venous pressure, distention of peripheral and central veins, increased cardiac output, loud heart sounds, functional murmurs, high pulse pressure, increased pulmonary second sound, gallop, pulmonary edema

**Tissue**
- **Deficit:** Decreased skin turgor, atonic muscles, sunken eyes, decreased tongue size with longitudinal wrinkles
- **Excess:** Subcutaneous pitting, edema, pulmonary crackles

**Metabolic**
- **Deficit:** Decreased temperature
- **Excess:** None

Changes in body weight from dehydration: Body weight is an accurate test for determining fluid excess or deficiency if done repeatedly and recorded accurately on a day-to-day basis. Weight loss greater than 300–500 g/d
indicates dehydration secondary to decreased fluid intake and/or increased water losses.

<table>
<thead>
<tr>
<th>Weight loss</th>
<th>Degree of dehydration</th>
</tr>
</thead>
<tbody>
<tr>
<td>4% of body wt</td>
<td>Mild</td>
</tr>
<tr>
<td>6% of body wt</td>
<td>Moderate</td>
</tr>
<tr>
<td>8% of body wt</td>
<td>Severe</td>
</tr>
</tbody>
</table>

Monitoring urine output, heart rate, and blood pressure on a repeated basis and comparing them to measured fluid intake assists in determining fluid requirements. The normal adult should have an hourly urine output between 0.5–2.0 cc/kg/h (average, 1.0 cc/kg/h). Less than 0.5 cc/kg/h is inadequate and implies the patient requires additional oral or intravenous (IV) fluids. Normal urinary output in children and infants should be 2.0 cc/kg/h. Urinary output less than 1.0 cc/kg/h indicates the patient needs additional fluids.

Laboratory Evaluation

Certain laboratory tests correlate with dehydration and overhydration. Elevation of serum Na\(^+\) levels accompanied by elevations of the blood urea nitrogen (BUN), serum osmolarity, hematocrit, and creatinine suggest dehydration. A urine specific gravity greater than 1.030 (normal = 1.010) indicates dehydration. Urine electrolyte concentrations indicate renal conserving values:

<table>
<thead>
<tr>
<th>Tubular activity</th>
<th>Sodium (mEq/L)</th>
<th>Potassium (mEq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&gt; 40</td>
<td>&gt; 40</td>
</tr>
<tr>
<td>Conserving (early dehydration)</td>
<td>10–30</td>
<td>20–30</td>
</tr>
</tbody>
</table>
Maximal (severe dehydration)

Fractional excretion of sodium is an important test to determine the cause of decreased urinary output. It can be calculated when urine sodium and urine and plasma creatinine values are known.

Fractional excretion of sodium

\[
\frac{\text{Urine Na} \times \text{Plasma Cr}}{\text{Urine Cr}}
\]

- < 1 = Prerenal azotemia (dehydration or decreased renal perfusion)
- > 1 = Renal failure

Decreased serum Na\(^+\), BUN, creatinine, hematocrit, and serum osmolarity and a urine specific gravity < 1.003 indicates overhydration. Edema, a sign of overhydration, occurs when 2–4 kg or more of extra fluid is retained.

Fluid Types

Fluid replacement is categorized by the clinical situation and type of fluid used. Fluid types are divided into crystalloids (molecular weight < 8,000 daltons) and colloids (molecular weight > 8,000 daltons). Table 6-4 shows the glucose and electrolyte concentrations of various crystalloid solutions. Colloid solutions include serum albumin (hepatitis-free, expensive) and 6% hetastarch.

Table 6-4 Glucose and electrolyte concentrations of various crystalloid solutions*
Maintenance fluids are used to meet normal daily requirements in patients unable to consume sufficient fluids. The most common maintenance fluid is 5% dextrose in half-strength normal saline (D$_5$½NS) with 20 mEq/L of KCl. Replacement fluids are formulated to correct body fluid deficits caused by loss or sequestration of nearly isotonic, polyionic body fluids. Replacement fluids are used to acutely replace volume deficits in patients with dehydration, trauma, and sepsis. Normal saline or lactated Ringer solution are the two primary replacement solutions. Replacement fluids can be given rapidly (boluses of 500 mL to 1 L) to correct for major fluid deficits. Most maintenance and replacement fluids should have an osmolality close to normal to avoid hemolysis.

Crystalloid solutions replace mainly extracellular volume, with one-fourth remaining intravascularly and three-fourths remaining in the interstitium. Colloids, due to their larger molecules, remain intravascular. Administration of 3 mL of crystalloid is equivalent to 1 mL of colloid or blood for intravascular replacement.

**Electrolyte Abnormalities**

Although most oral and maxillofacial surgery patients are relatively healthy, a significant number have coexisting surgical and/or medical conditions that

<table>
<thead>
<tr>
<th>Solution</th>
<th>G</th>
<th>Na</th>
<th>Cl</th>
<th>K</th>
<th>Ca</th>
<th>L</th>
<th>Osmoles (Osm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LR</td>
<td>—</td>
<td>130</td>
<td>110</td>
<td>4</td>
<td>2.7</td>
<td>23</td>
<td>276</td>
</tr>
<tr>
<td>D$_5$LR</td>
<td>50</td>
<td>130</td>
<td>102</td>
<td>4</td>
<td>2.7</td>
<td>23</td>
<td>536</td>
</tr>
<tr>
<td>NS (0.9%)</td>
<td>—</td>
<td>154</td>
<td>154</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>308</td>
</tr>
<tr>
<td>% NS (0.45%)</td>
<td>—</td>
<td>77</td>
<td>77</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>% NS (0.2%)</td>
<td>—</td>
<td>34</td>
<td>34</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>D$_5$W</td>
<td>50</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>260</td>
</tr>
<tr>
<td>D$_5$NS</td>
<td>50</td>
<td>154</td>
<td>154</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>560</td>
</tr>
<tr>
<td>D$_5$½NS</td>
<td>50</td>
<td>77</td>
<td>77</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>280</td>
</tr>
</tbody>
</table>

G, glucose; LR, lactated Ringer solution; D$_5$%, 5% dextrose solution; NS, normal saline; W, water; —, not applicable.

*Glucose (G) in g/L; others in mEq/L.
alter their fluid and electrolyte balance. The majority of these patients can be
treated with minor adjustments in fluids, diet, or medications. Occasionally
patients present with or develop severe fluid and electrolyte abnormalities
(eg, trauma, diabetic, elderly, oncologic, and postoperative patients), which
must be correctly diagnosed and treated to avoid major morbidity and even
mortality.

Sodium
Serum Na$^+$
- Normal 135–145 mEq/L
- Hypernatremia > 140 mEq/L
- Hyponatremia < 135 mEq/L
- Normal daily intake: 135–145 mg/day (adult)
  
  2.4 mg/kg/day (infant/child < 20 kg)

Serum Na$^+$ reflects the measurement of total body osmolarity (normal:
280–300 mOsm). In order to determine the significance and treatment of a
serum Na$^+$ excess or deficit, the patient’s volume status must be evaluated.
Determination of serum osmolarity and urine sodium levels will assist in
evaluation and treatment.

Hyponatremia: Proper diagnosis of hyponatremia is based on clinical
evaluation of the extracellular fluid volume, comparison of calculated and
measured plasma osmolality, and urinary Na$^+$ determination.

Calculated osmolality = 2[Na$^+$ + K$^+$] + [BUN/2.8] + [Glucose/18]

If the measured osmolality is 10 mOsm/kg greater than the calculated
osmolality, significant unmeasured osmotically active solutes are present.

Causes
- Hyponatremia with normal or elevated osmolarity
—Hyperglycemia
—Hyperlipidemia
—Hyperproteinemia

- Hyponatremia with increased extracellular fluid volume
  —Congestive heart failure
  —Cirrhosis
  —Nephrotic syndrome
  —Chronic renal failure

- Hyponatremia with normal extracellular fluid volume
  —Glucocorticoid deficiency
  —Vomiting
  —Drugs
  —Hypothyroidism
  —Hypokalemia
  —Syndrome of inappropriate antidiuretic hormone (SIADH; other para-neoplastic syndromes, CNS disorders)

- Hyponatremia with decreased extracellular fluid volume
  —Third spacing
  —Gastrointestinal (GI) losses
  —Mineralocorticoid deficiency
  —Diuretics
  —Salt-losing nephritis

Clinical signs and symptoms
- CNS: Lethargy, confusion, seizures
- GI: Nausea, vomiting, anorexia
- Musculoskeletal: Muscle cramps
- Metabolic: Hypothermia

Symptoms are more severe with acute hyponatremia than with chronic hyponatremia. Symptoms of hyponatremia rarely develop until the serum Na⁺
Treatment: First, determine if the hyponatremia is acute or chronic. Serum Na⁺ correction should usually be gradual (over 48–72 hours) unless symptoms dictate rapid correction (110–115 mEq/L with seizures, stupor, or coma). Rapid correction of serum Na⁺ levels below 120 mEq/L can produce CNS osmotic shifts and central pontine myelinolysis. Correction can be rapid until the patient becomes asymptomatic (120–125 mEq/L) and then slowly corrected to 135–140 mEq/L over the next 2 to 3 days. Serial serum Na⁺ determination and clinical and physiologic monitoring should be repeated frequently.

- **Chronic**
  - Correct slowly (48–72 h)
  - Normal or increased ECF volume: Water restriction (800–1,200 mL/d)
  - Expanded ECF volume: Diuretics + fluid restriction
  - ECF depletion: Normal saline

- **Acute**
  1. Determine TBW excess.

\[
\text{Actual TBW (L)} = \text{Body weight (kg)} \times 60%
\]

\[
\text{Desired TBW} = \frac{\text{Actual plasma Na}^+}{\text{Desired plasma Na}^+} \times \text{Actual TBW}
\]

\[
\text{Free water excess (L)} = \text{Actual TBW} - \text{Desired TBW}
\]

2. Calculate Na⁺ deficit (for decreased ECF volume).

\[
\text{Na}^+ \text{ deficit (mEq)} = (\text{Desired plasma Na}^+ - \text{Actual plasma Na}^+) \times \text{TBW}
\]

3. Determine rate of correction.
The rate of correction can vary from 0.3 to 2.5 mEq/L/h and depends on the severity of the symptoms. Rapid correction (1 to 2.5 mEq/L/h) in symptomatic patients requires close monitoring because osmotic shifts in the CNS can produce central pontine myelinolysis.

• Correction of hypovolemic hyponatremia
  1. Determine the total Na\(^+\) deficit.
  2. Determine volume of 0.9% saline required to correct the deficit.

\[
\text{Total Na}^+ \text{ deficit} \times \frac{1 \text{ L} \times 0.9\% \text{ saline}}{154 \text{ mEq/L}} = \text{Volume of 0.9\% saline required (L)}
\]

3. Select the rate of correction (mEq/L/h) and determine the time period over which the deficit should be corrected. Correct to 120 mEq/L first, if necessary, and then recalculate to correct to 140 mEq/L.

\[
(120 \text{ mEq/L} - \text{serum Na}^+/\text{L}) \times (\text{Rate of correction [h/mEq/L]}) = \text{Time period (h)}
\]

4. Determine the volume required/h to achieve appropriate serum Na\(^+\). (Volume of 0.9% saline [mL] / Time [h] required) = mL/h.

• Correction of hypervolemic hyponatremia:
  1. Determine free water excess.
     Actual TBW — Desired TBW = Free water excess

\[
\text{Current serum Na}^+ \times \text{wt (kg)} \times 0.6 = \text{Desired TBW}
\]

\[
\text{Desired serum Na}^+ \times \text{wt (kg)} = \text{Desired TBW}
\]
2. Select rate of Na\(^+\) replacement (mEq/L/h) and determine correction time (in hours).
3. Determine the average hourly free water loss required. 
   \[
   \text{Volume of excess free water (mL)} / \text{Time required (h)} = \text{mL/h}
   \]
4. Treatment is usually accomplished with furosemide, with or without the use of 0.9% saline or 3.0% saline.
5. Monitoring of serum K\(^+\) is required with the use of furosemide.

Hypernatremia

*Causes of hypernatremia*
- Loss of free water
  - Inadequate free water intake
  - Skin loss (sweating, fever, burn)
  - GI loss
  - Renal loss (high output renal failure, osmotic diuresis, diabetes insipidus)
- Solute loading
  - Inappropriate IV replacement
  - Tube feedings
  - Brainstem injuries

*Clinical signs and symptoms*
- CNS: Restlessness, tremors, weakness, delirium, maniacal behavior, confusion, ataxia, seizures, coma
- Tissue: Decreased saliva and tears; dry, sticky, and red mucous membranes; red, swollen tongue; flushed skin
- Renal: Oliguria
- Metabolic: Fever

*Treatment:* The treatment of hypernatremia is dependent on the
etiology. Diabetes insipidus requires special treatment and determination of whether the cause is nephrogenic or central. Patients with serum Na\(^+\) levels > 160 mEq/L, serum osmolarity > 350 mOsm, and/or rapid elevation of serum Na\(^+\) develop symptoms. For asymptomatic or minimally symptomatic patients, a solution of 5% dextrose in water (D\(_5\)W) is a satisfactory solution. In symptomatic patients, 0.9% saline is still hypotonic (154 mEq/L) and should be used to correct volume deficits. Once the volume has been restored, changing to a hypotonic IV fluid is indicated.

1. Calculate free water deficit (FWD).

\[
\text{FWD (L)} = \frac{\text{Serum Na}^+}{140} - 140 \times \frac{\text{wt in kg}}{2}
\]

or

Desired TBW — Actual TBW = FWD (L)

2. Select rate of correction (usually < 1 mEq/L/h) and determine the number of hours required to correct the hypernatremia.

\[
(\text{Actual serum Na}^+ — 145) \times \text{Rate of correction} = \text{Time required (h)}
\]

3. Determine the hourly replacement.

\[
\frac{\text{FWD (mL)}}{\text{Time required (h)}} = \text{mL/h}
\]

or

Administer D\(_5\)W, replacing half of the total deficit over 24 hours and the remaining half over the following 48 hours.

4. Monitor and adjust replacement based on serial serum Na\(^+\).
Nephrogenic diabetes insipidus is treated with salt restriction and thiazide diuretics after correction of hypercalcemia or hypokalemia. Any causative medications must be stopped. Central diabetes insipidus requires treatment with desmopressin acetate (DDAVP).

Potassium

Serum $K^+$
- Normal: 3.5 to 5.2 mEq/L
- Hyperkalemia: $> 5.2$ mEq/L
- Hypokalemia: $< 3.5$ mEq/L
- Normal daily intake: 40 to 60 mEq/L
- Total body $K^+$: 3,000 to 4,000 mEq

Serum $K^+$ levels reflect approximately 1% to 2% of total body $K^+$ and are an indirect measure of total body $K^+$. A 1-mEq/L change in the serum $K^+$ indicates a 100- to 400-mEq/L change in total body $K^+$.

Hypokalemia

*Causes of hypokalemia*  *Clinical signs and symptoms*
- Decreased dietary intake  · Cardiac
  - Alcoholism  · Flattened T waves
  - Starvation  · Prominent U waves
  - Malnutrition  · ST depression
- Increased loss  · Dysrhythmias (especially with concomitant use of digitalis)
  - Diarrhea (multiple causes)
—Diuretics
—Hypotension

—Vomiting
• Neuromuscular

—Nasogastric suction
—Weakness

—Mineralocorticoid excess
—Respiratory failure

—Interstitial nephritis
—Rhabdomyolysis

—Hypomagnesemia
—Hyporeflexia

—Aminoglycosides
• Renal

—High-dose penicillin/carbenicillin
—Polyuria

• Increased intracellular shift
—Concentrating defect

—Alkalosis
—Metabolic alkalosis

—Insulin
—Decreased glomerular filtration rate (GFR)

—β-Agonist

—B₁₂
• Metabolic

—IV glucose
—Glucose intolerance secondary to decreased insulin release

—Potentiation of hypercalcemia and hypomagnesemia

Diagnosis
• Serum K⁺ level
• Review medical history for signs, symptoms, and medications

_Treatment:_ Correction of hypokalemia requires simultaneous correction of any alkalosis or acidosis, volume deficits, other electrolyte disturbances (eg, Mg²⁺), discontinuation of any offending medications, and potassium replacement. Adequate renal function must be confirmed.

Oral supplements represent the safest method of K⁺ replacement. Different formulations exist and can be used depending on the acid-base imbalance (metabolic alkalosis: potassium chloride; metabolic acidosis: potassium citrate, bicarbonate, or gluconate) and gastrointestinal tolerance. Dosage varies depending on the degree of hypokalemia. Alternative therapy for patients with high renal losses include the potassium-sparing diuretics.

Intravenous therapy is used when oral therapy is not possible or rapid replacement is required (serum K⁺ < 2.5 mEq/L in the absence of cardiac dysrhythmias). The usual replacement rate is 10 mEq/h in nonemergent situations. In emergent situations, 20 to 40 mEq/h can be infused, but cardiac monitoring is required due to the risk of dysrhythmias and frequent serum K⁺ level determination must be performed. Higher concentrations of K⁺ should be administered peripherally to avoid direct cardiac effects, but there is a risk of phlebitis secondary to venous irritation. Glucose-free solutions should be used because glucose can drive K⁺ intracellularly and further exacerbate the hypokalemia.

**Hyperkalemia**

<table>
<thead>
<tr>
<th>Causes of hyperkalemia</th>
<th>Clinical signs and symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Pseudohyperkalemia (spurious)</td>
<td>• Cardiac</td>
</tr>
<tr>
<td>—Phlebotomy</td>
<td>—Peaked T waves</td>
</tr>
<tr>
<td>—Hemolysis</td>
<td>—QRS widening</td>
</tr>
</tbody>
</table>
—Thrombocytosis
—Leukocytosis

• Increased intake
  —Iatrogenic $K^+$ administration

• Decreased renal excretion
  —Renal failure
  —$K^+$-sparing diuretics

• Neuromuscular
  —Weakness
  —Paresthesia
  —Areflexia
  —Respiratory failure

• Redistribution to extracellular fluid
  —Acidosis
  —Muscle damage
  —Hyperglycemia
  —Insulin deficit
  —$\beta$-Antagonist
  —Digitalis overdose
  —Succinylcholine

*Diagnosis*
• Serum K\(^+\) level
• EKG analysis
• Additional evaluations
  —Continuous cardiac monitoring
  —Evaluation of medications
  —Evaluation of renal function
  —Arterial blood gases

**Treatment**

• Acute: Potassium levels greater than 7.0 mEq/L, electrocardiographic (ECG) changes, or severe symptoms constitute a need for emergent therapy. Frequent serial serum K\(^+\) measurements are necessary.

  1. 10% calcium gluconate IV (10 mL) given for 2 to 3 minutes will decrease K\(^+\) levels for approximately 1 hour. A second dose can be given in 5 minutes if there is no response. However, this is only a temporary measure, and additional therapy is required. Caution must be used in patients with digitalis intoxication.

  2. Sodium bicarbonate 44 mEq (1 amp) IV will reduce serum K\(^+\) levels and can be given with D\(_5\)W and insulin. Intracellular shifts begin approximately 15 minutes after administration and last approximately 1 to 2 hours. A repeated dosage may be given 15 minutes later if ECG changes are still present. Administration may aggravate hypocalcemia and lead to seizures and tetany. If hypocalcemia is present, calcium should be given first.

  3. 50 g of D\(_5\)W IV administered with 10 U of regular insulin IV for 5 minutes will decrease serum K\(^+\) levels in approximately 30 minutes and work for several hours.

  4. IV furosemide, 40 mg or more, may be useful when no contraindications exist (eg, hypotension or hypovolemia).

  5. Kayexalate, a cation-exchange resin, can be given
orally or rectally. Each gram of Kayexalate binds approximately 1 mEq of $K^+$. One to 2 g of sodium are exchanged for each mEq of $K^+$. Caution must be used in patients with renal insufficiency or congestive heart failure because sodium overload may occur.

—Oral: 15 to 40 g is combined with 100 mL of 20% sorbitol to prevent constipation. It may be administered four to six times daily.
—Rectal: 50 mg is combined with 200 mL of 20% sorbitol and given as a 1- to 2-hour retention enema. It may be repeated every 4 hours as necessary.

- Chronic: Treatment of chronic hyperkalemia is directed at correction of the underlying cause. Elimination of drugs, reduction of dietary potassium, loop diuretics, and mineralocorticoid replacement are used for the appropriate conditions.

Chloride

An isolated change in serum chloride tells very little about a patient’s condition unless it can be evaluated as part of an overall fluid and electrolyte pattern.

Normal level: 95 to 108 mEq/L
Normal intake: 80 to 140 mEq/24 h

<table>
<thead>
<tr>
<th>Hyperchloremia</th>
<th>Hypochloremia</th>
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</thead>
<tbody>
<tr>
<td>Causes of hyperchloremia</td>
<td>Causes of hypochloremia</td>
</tr>
<tr>
<td>• Hyperchloremic acidosis</td>
<td>• Metabolic alkalosis</td>
</tr>
</tbody>
</table>
• Respiratory alkalosis  
• Dehydration  
• Diabetes insipidus  
• Medications (acetazolamide, ammonium chloride)  
• Renal tubular acidosis

Table 6-5 Method for determining water, sodium, and potassium replacement

<table>
<thead>
<tr>
<th>Patient weight (kg)</th>
<th>$H_2O$ (mL/kg/d)</th>
<th>Na⁺ (mEq/kg/d)</th>
<th>K⁺ (mEq/kg/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>First 10 kg</td>
<td>100</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Second 10 kg</td>
<td>50</td>
<td>1.5</td>
<td>1</td>
</tr>
<tr>
<td>Each kg thereafter</td>
<td>15–20</td>
<td>0.6</td>
<td>0.4</td>
</tr>
<tr>
<td>Total for 70-kg patient</td>
<td>2500 mL/d</td>
<td>75 mEq/d</td>
<td>50 mEq/d</td>
</tr>
</tbody>
</table>

Table 6-6 Simplified method for determining maintenance fluids

<table>
<thead>
<tr>
<th>Patient weight (kg)</th>
<th>Amount of fluid/h (mL/kg)</th>
<th>Amount of fluid/h for a 70-kg patient (mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>First 10 kg</td>
<td>4</td>
<td>40</td>
</tr>
<tr>
<td>Second 10 kg</td>
<td>2</td>
<td>20</td>
</tr>
<tr>
<td>Each kg thereafter (50 kg)</td>
<td>1</td>
<td>50</td>
</tr>
<tr>
<td>Total</td>
<td>110 mL/h</td>
<td></td>
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</tbody>
</table>

Postoperative Fluid and Electrolyte Management

1. Assess losses and gains.
2. Correct existing deficiencies. For head and neck procedures, provide 3 to 5 mL/kg/h times the duration of the operation added to the duration
the patient was NPO.

3. Provide for maintenance. Water, Na\(^+\), and K\(^+\) are the substances most necessary to replace in the short-term management of a patient’s fluid balance. The necessary maintenance doses can be determined as shown in Tables 6-5 and 6-6 or calculated using body surface area (Fig 6-1). The preceding is most useful for children.

**Transfusion Guidelines**

For more information, see section “Transfusion of Blood and Blood Components” in chapter 8.

**Clinical Decision Basis**

Concomitant disease states may dictate early use of blood products rather than crystalloid or colloid replacement in traumatized and surgical patients. The decision to use blood is no longer based solely on hemoglobin levels. Hemoglobin and hematocrit have been shown to be poor indicators of blood loss in patients with acute hemorrhage. In the past, blood transfusions were often given for hemoglobin values < 10 g/dL. Today, unless concomitant medical conditions or symptoms dictate, a hemoglobin of 7 g/dL or less is a more appropriate guide-line for transfusion. However, without clinical signs and/or symptoms (ongoing blood loss, failure of crystalloid therapy to maintain tissue perfusion, chest pain, labored respirations, orthostatic hypotension), the need for transfusion is in doubt even if the hemoglobin is below 7 g/dL.
### Samples of daily maintenance requirements

Water/ Na / K (mL) (mEq)

<table>
<thead>
<tr>
<th>Height (cm)</th>
<th>Body surface (m²)</th>
<th>Mass (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>195</td>
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<td>190</td>
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<td>10</td>
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</tbody>
</table>

### Height (cm)

- 200 cm: 79 in.
- 195 cm: 77 in.
- 190 cm: 75 in.
- 185 cm: 73 in.
- 180 cm: 71 in.
- 175 cm: 69 in.
- 170 cm: 67 in.
- 165 cm: 65 in.
- 160 cm: 63 in.
- 155 cm: 61 in.
- 150 cm: 59 in.
- 145 cm: 57 in.
- 140 cm: 55 in.
- 135 cm: 53 in.
- 130 cm: 51 in.
- 125 cm: 49 in.
- 120 cm: 47 in.
- 115 cm: 45 in.
- 110 cm: 43 in.
- 105 cm: 41 in.
- 100 cm: 39 in.

### Body surface (m²)

- 2.80 m²
- 2.70 m²
- 2.60 m²
- 2.50 m²
- 2.40 m²
- 2.30 m²
- 2.20 m²
- 2.10 m²
- 2.00 m²
- 1.95 m²
- 1.90 m²
- 1.85 m²
- 1.80 m²
- 1.75 m²
- 1.70 m²
- 1.65 m²
- 1.60 m²
- 1.55 m²
- 1.50 m²
- 1.45 m²
- 1.40 m²
- 1.35 m²
- 1.30 m²
- 1.25 m²
- 1.20 m²
- 1.15 m²
- 1.10 m²
- 1.05 m²
- 1.00 m²
- 0.95 m²
- 0.90 m²
- 0.86 m²

### Mass (kg)

- 150 kg: 330 lb
- 145 kg: 320 lb
- 140 kg: 310 lb
- 135 kg: 300 lb
- 130 kg: 290 lb
- 125 kg: 280 lb
- 120 kg: 270 lb
- 115 kg: 260 lb
- 110 kg: 250 lb
- 105 kg: 240 lb
- 100 kg: 230 lb
- 95 kg: 220 lb
- 90 kg: 210 lb
- 85 kg: 200 lb
- 80 kg: 190 lb
- 75 kg: 180 lb
- 70 kg: 170 lb
- 65 kg: 160 lb
- 60 kg: 150 lb
- 55 kg: 140 lb
- 50 kg: 130 lb
- 45 kg: 120 lb
- 40 kg: 110 lb
- 35 kg: 100 lb
- 30 kg: 95 lb
- 25 kg: 90 lb
- 20 kg: 85 lb
- 15 kg: 80 lb
- 10 kg: 75 lb
- 5 kg: 70 lb
With regard to surgical patients, maximum blood loss during a procedure before transfusion is based on percent loss of the total estimated blood volume (EBV; limit: infant—10%, children—15%, adults—20%). The EBV is determined as follows:

- Adults 70 mL/kg body wt
- Children 75 mL/kg body wt
- Infants 80 mL/kg body wt

or

Minimal allowable hematocrit (eg, 21%):

\[
\text{EBV} = \left( \frac{\text{Preop Hct} - \text{Allowable Hct}}{\text{Preop Hct}} \right) \times \text{Blood loss tolerable prior to transfusion}
\]

**Underestimation:** Remember that surgeons generally underestimate blood loss by as much as 15% to 40%.

**Crossmatched, Type-Specific, and Type O Blood**

Fully crossmatched blood is preferable when there is sufficient time. The risk of transfusion reaction is lowest with such blood. However, it normally takes the laboratory approximately 1 hour to complete the crossmatch and have blood available, and therefore whole blood is not the first choice in patients with life-threatening hemorrhage. When the patient is stabilized by initial crystalloid therapy, crossmatched blood is preferred.
Type-specific blood can be provided within 20 minutes by most laboratories. ABO and Rh compatibilities are checked, while minor incompatibilities are not evaluated and may exist. The risk of transfusion reaction is greater than with crossmatched blood; however, the risk is usually acceptable.

Type O blood is used whenever type-specific blood is not available. Rh-negative type O blood is preferred to prevent Rh sensitization and future complications.
Nutrition for the Surgical Patient

General Considerations
Whether it is to supplement or completely replace normal feeding, the main goal of surgical nutrition is the recognition and correction of perioperative nutritional deficiencies and the maintenance of an optimal nutritional status. This results in better wound healing, a lower infection rate, and increased overall survival rate.

Nutrients
Carbohydrates
Basic characteristics
- Most efficient source of calories.
- Major energy source, providing at least 45% to 65% of daily caloric diet.
- Stored as glycogen in the liver.
- Glycogen is depleted 12 hours after cessation of food intake; fats and proteins are then used as a source of calories.
- To prevent protein metabolism and avoid ketosis, a minimum of 50 g of carbohydrates should be included in the daily diet.

Caloric value: 4 cal/g parenterally adds 3.4 cal/g (Table 7-1)

Fats
Basic characteristics
- The most concentrated source of calories.
- Essential fatty acids are required in the diet.
The brain and central nervous system will not accept fat as a fuel source, only sugars. Fats cannot be converted into sugars (proteins can); therefore, without carbohydrate intake, proteins will be metabolized.

Caloric value: 9 cal/g

**Table 7-1** *Caloric values of nutrients*

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Cal*/g</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate</td>
<td>4</td>
</tr>
<tr>
<td>Protein</td>
<td>4</td>
</tr>
<tr>
<td>Fat</td>
<td>9</td>
</tr>
</tbody>
</table>

* One calorie (4.2 J) is the amount of heat required to raise 1 kg of water 1°C.

Proteins

Basic characteristics

- Necessary for the formation and maintenance of tissue structure, as well as immunologic, contractile, and enzymatic function.
- Essential amino acids are required in the diet.
- Serve as energy source for the brain in starvation situations by conversion to simple sugars (glyconeogenesis).

Caloric value: 4 cal/g

Micronutrients

Micronutrients are substances that cannot be made by the body and are required in very small quantities for proper biochemical function. Usually serve as coenzymes.
Vitamins

*Fat soluble: A, D, E, and K*
- Stored in the liver.
- Deficiency can occur after prolonged malnutrition or secondary to liver disease.

*Water soluble: B vitamins (“B complex”) and vitamin C*
- Facilitate reactions involved in generation and transfer of energy.
- Scantly stored, but rapidly absorbed from the gastrointestinal tract.
- Deficiencies develop quickly in the malnourished state.

Trace elements
- Function in metabolic and immunologic processes.
- Are important for proper wound healing.
- Subclinical deficiencies commonly occur in hospitalized patients.

**Nutritional Assessment**
Recognition of the nutritionally compromised patient is the primary step in nutritional care. The nutritional requirements of healthy patients are considerably different than those of presurgical and postsurgical patients with nutritional deficits. It is important to appreciate these differences when planning nutritional support.

**History**

Weight loss
Unintentional weight loss is an important consideration when determining nutritional status. The percentage of the body weight lost is measured over time to determine the severity of the process (*Table 7-2*).
Table 7-2 Severity of weight loss based on percentage of body weight lost in a specific time period*

<table>
<thead>
<tr>
<th>Time period</th>
<th>Significant weight loss (%)</th>
<th>Severe weight loss (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 week</td>
<td>1–2</td>
<td>&gt; 2</td>
</tr>
<tr>
<td>1 month</td>
<td>5</td>
<td>&gt; 5</td>
</tr>
<tr>
<td>3 months</td>
<td>7.5</td>
<td>&gt; 7.5</td>
</tr>
<tr>
<td>6 months</td>
<td>10</td>
<td>&gt; 10</td>
</tr>
</tbody>
</table>


Prior medical history findings that could predispose to malnutrition include:
- Liver disease
- Renal failure
- Inflammatory bowel disease
- Neoplastic disease, chemotherapy
- AIDS

Physical Examination

Integument: Altered skin texture, rash, nail deformities, changed hair quality

Oral: Cheilosis, glossitis, mucosal atrophy, gingivitis, loss of periodontal integrity

Abdomen: Liver enlargement, abdominal mass

Extremities: Reduced muscle size and strength, edema

Neurologic: Mental status changes, neuropathy
Anthropometric Measurements

- Used to assess the degree of depletion of body fat and protein stores by comparing to normal values for age and gender.
- Triceps skin fold (TSF) measurement, determined with calipers, is used to evaluate body fat.
- Protein, stored in skeletal muscle, can be appraised by measuring the mid–upper arm circumference and adjusting this value to reflect the amount of subcutaneous fat present. The resultant value is called the arm muscle circumference (AMC) (Table 7-3).

\[ \text{AMC} = \frac{\text{Midarm circumference} - (\pi)(\text{TSF})}{10} \]

- Body mass index (BMI) is a useful tool for estimating an individual’s body fat. It is calculated by dividing the body weight in kilograms by the height in meters squared:

  Normal: 18.5–24.9
  Overweight: 25–29.9
  Obese: > 30

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triceps skin fold</td>
<td>11</td>
<td>19</td>
</tr>
<tr>
<td>Arm muscle circumference</td>
<td>270</td>
<td>213</td>
</tr>
</tbody>
</table>

Table 7-3 Normal anthropometric values for adults (mm)

Laboratory Evaluation
Serum albumin
- Normal = 3.5 to 4.4 g/dL
- Moderate malnutrition = 2.5 to 3.5 g/dL
- Severe malnutrition = < 2.5 g/dL

Total lymphocyte count
- Normal = 1,500 to 2,000/mm$$^3$$
- Moderate malnutrition = 1,000 to 1,500/mm$$^3$$
- Severe malnutrition = < 1,000/mm$$^3$$

Creatinine height index (CHI)
- Used to estimate the degree of protein loss from body cell mass

$$\text{CHI} = \frac{\text{24-hour urine creatinine excretion}}{\text{Normal creatinine excretion for height}}$$

Other plasma proteins
- Transferrin: Normal = 200 mg/dL; mild malnutrition = < 175 mg/dL; severe malnutrition = < 147 mg/dL
- Retinol-binding protein = < 40 g/mL
- Thyroxine-binding protein = < 200 g/mL

Immunologic testing
- Skin testing that evaluates cell-mediated immunity (recall antigen skin testing)
- Anergy seen in malnourished, hypoproteinemic states

**Normal Daily Requirements**
Protein Requirements
Protein balance: Under constant homeostatic control. The body alters protein stores to compensate for changes in activity and levels of physical stress. Therefore, the daily protein requirement varies with changing levels of stress and activity.
- Requirement for adult male = 0.8 g of protein/kg weight/d
• Requirement for children = 2.0 to 2.5 g of protein/kg weight/d
• Requirement in acute illness = 1.5 g of protein/kg weight/d
• Severely stressed patients, such as burn patients or patients in intensive care, may require 2 g of protein/kg weight/d

Nitrogen balance: Calculations can confirm the effectiveness of nutritional support. Positive nitrogen balance signifies that more nitrogen is being taken in than excreted. This is consistent with an anabolic state. In negative nitrogen balance, losses exceed intake and a catabolic state prevails:
• 1 g of nitrogen = 6.25 g of protein
• Calculation of nitrogen balance:

\[
N \text{ balance} = \frac{\text{Protein intake (g)}}{6.25} - (\text{24-h urine urea} + X)
\]

where \(X\) is a factor that accounts for N loss via skin, feces, and respiratory tract and usually equals 2 to 4.
• Ratio of calories to nitrogen. The energy required for protein synthesis is expressed by a calories-to-nitrogen ratio of 150–200:1.

This means that ideal diets should have 150 to 200 nonprotein calories per gram of nitrogen. This ratio increases when the body is stressed. It can be as high as 400:1 in uremic patients.

Caloric Requirements

Total energy expenditure for healthy adults can be divided into three main components:
• Basal or resting metabolic rate
• Energy required for the thermic effects of digestion
• Energy required for exercise

During illness this is also affected by hypermetabolism, hypercatabolism, or significant insulin resistance, all of which can increase resting metabolic rates by:
• 110% to 120% in elective surgery and medical patients
• 135% to 150% in trauma patients
• 150% to 170% in septic patients

The daily caloric requirement may be determined by a variety of methods.
• The patient’s body weight may be used to estimate the daily caloric requirement. This calculation is rapid, but it is only a crude approximation.

- Adult = 21 to 30 cal/kg weight/d
- Infant = 90 to 120 cal/kg weight/d
- Geriatric = 18 cal/kg weight/d

• A more accurate method to ascertain the daily caloric requirement involves determining the basal energy expenditure (BEE). Three formulas can be used to determine the BEE.
  1. The Harris-Benedict equation, proposed in 1919, can be used to estimate this value, but due to changes in lifestyle this equation tends to overestimate the caloric needs by about 5%.

    BEE (men) = 13.7(weight) + 5(height) — 5(age) + 666
    BEE (women) = 10(weight) + 1.8(height) — 5(age) + 655
    with weight in kilograms, height in centimeters, age in years.

    To determine daily caloric requirements, the BEE may be multiplied by a value for different levels of injury and stress:

    Healthy patient: 1.20
    Elective surgery: 1.44
    Trauma: 1.53
    Trauma and steroids: 1.80
Sepsis, burns: 1.90

2. The Mifflin–St Jeor formula was found to provide another accurate determination of BEE in a study published by the American Dietetic Association.

\[
\text{BEE (men): BMR} = 10(\text{weight}) + 6.25(\text{height}) - 5(\text{age}) + 5
\]
\[
\text{BEE (women): BMR} = 10(\text{weight}) + 6.25(\text{height}) - 5(\text{age}) - 161
\]
with weight in kilograms, height in centimeters, age in years.

3. In critical illness, the gold standard for measuring the BEE is indirect calorimetry using the Weir formula, which involves the oxygen used and the carbon dioxide produced by the patient, along with the nitrogen excreted in the urine, to measure the BEE.

\[
\text{BEE (kcal/min)} = 3.9(\text{VO}_2) + 1.1(\text{VCO}_2) - 2.2(\text{urine nitrogen})
\]
where \(\text{VO}_2\) = oxygen uptake (mL/min), \(\text{VCO}_2\) = carbon dioxide output (mL/min).

Requirements for Micronutrients
The recommended vitamin requirements are summarized in Table 7-4, and elemental requirements are listed in Table 7-5.

*Table 7-4* Daily requirements for vitamins (recommended daily allowance...
for adults)

<table>
<thead>
<tr>
<th>Vitamin</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>800–1000 µg</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>1300 µg</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>8–10 mg</td>
</tr>
<tr>
<td>Vitamin K</td>
<td>70–80 µg</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vitamin</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiamine (B₁)</td>
<td>1.1–1.2 mg</td>
</tr>
<tr>
<td>Riboflavin (B₂)</td>
<td>1.3–1.7 mg</td>
</tr>
<tr>
<td>Pantothenic acid (B₃)</td>
<td>4–7 mg</td>
</tr>
<tr>
<td>Pyridoxine (B₆)</td>
<td>1.6–2.0 mg</td>
</tr>
<tr>
<td>Cyanocobalamin (B₁₂)</td>
<td>2 µg</td>
</tr>
<tr>
<td>Niacin</td>
<td>13–19 mg</td>
</tr>
<tr>
<td>Folate</td>
<td>400 µg</td>
</tr>
<tr>
<td>Biotin</td>
<td>30–100 µg</td>
</tr>
<tr>
<td>Ascorbic acid (C)</td>
<td>100–200 mg</td>
</tr>
</tbody>
</table>

**Table 7-5 Daily trace element requirements**
Chromium 50–200 µg
Copper 2–3 mg
Fluoride 4 mg
Iodine 150 µg
Iron 15 mg women, 10 mg men
Manganese 2–5 mg
Molybdenum 75–250 µg
Selenium 55–70 µg
Zinc 12–15 mg

Guidelines for Hospital Nutrition
Hospital diets are available in various consistencies, compositions, and caloric contents. A balanced diet contains:

- Carbohydrate = 50% to 70%
- Fats = 20% to 30%
- Protein = 10% to 20%

General Diet
- Also called house diet or regular diet.
- Designed to maintain or attain optimal nutritional status. Individual requirements may vary based on sex, height, weight, and level of activity.
- Indicated for adults who do not require specific nutrient alterations for a preexisting deficit or disorder.
- Composition: Protein—118 g; fat—68 g; carbohydrate—282 g; calories—2, 200.

Altered Consistency Diets
Clear liquid diet
- Intended for minimal fluid and energy needs in a consistency that requires little digestion and no chewing. This diet does not meet the recommended dietary allowances for any nutrient except vitamin C. If the clear liquid diet is used more than 3 days, the rationale for continuing the diet should be reviewed and revised as necessary.
- Indicated as an initial feeding following surgery or a period of intravenous feedings.
- Composition varies greatly. Usually only supplies 10 g of protein and 300 calories.

Full liquid diet
- Includes thicker fluids that are liquid or become liquid at body temperature.
- Indicated postoperatively as a transition between clear liquids and solid foods. It is also useful in patients who are unable to chew after oral or head and neck surgery.
- Composition: Protein—76 g; fat—91 g; carbohydrate—312 g; calories—2,300.

Mechanically soft diet
- Formulated to minimize the amount of chewing necessary to ingest food.
- Indicated for patients who have had oral, head, and neck surgery and who may be having difficulty chewing or swallowing.
- Composition: Protein—100 g; fat—60 g; carbohydrate—290 g; calories—2,080.

Pureed diet (wired-jaw diet)
- Provides full nutrition in a smooth consistency that can be swallowed with no chewing. This diet is based on soft food that is pureed and strained to achieve the appropriate consistency to facilitate swallowing.
- Indicated for alert patients who do not have proper masticatory function. This includes patients in maxillomandibular fixation.
- Composition: Same as for mechanically soft diets.

Altered Composition Diets
There are many types of diets, and their compositions are changed to help manage various disease processes. Listed below are only a few of the more common altered diets. Dietary consultation should be obtained when planning to administer these or other restricted and modified diets.

Diabetic diet: The American Diabetes Association no longer recommends a specific “Diabetic Diet” or “ADA Diet.”
- Diets are individualized by the treating physician or dietitian.
- Goals of diet therapy:
  1. Maintenance of near-normal blood glucose levels
  2. Achievement of optimum serum lipids
  3. Provision of adequate calories for maintaining or attaining reasonable weight

- Total protein should be 10% to 20% of daily caloric intake.
- The remaining caloric intake is less than 10% from saturated fat and 10% from polyunsaturated fats.
- Diets are individually modified based on periodic monitoring of metabolic parameters, blood glucose, glycosylated hemoglobin, lipids, body weight, and blood pressure.

Sodium-restricted diet: Used in the management of essential hypertension, liver disease, renal disease, and cardiac failure. Available sodium restricted diets include:
- No added salt (NAS) diet
- 4 g/d (174 mEq)
- 2 g/d (87 mEq)
- 1 g/d (45 mEq) (Compliance with this diet may be difficult for some patients.)

Protein-controlled and potassium-restricted diets: Used for patients in acute or chronic renal failure who are on dialysis

Fat-modified diets: Important in the management of hyperlipidemia states, diabetes mellitus, nephrotic syndrome, and hypothyroidism

High-fiber and low-fiber diets: Used for patients with various gastrointestinal and metabolic disorders
Feeding Methods

Enteral Feedings

Preferred if the gastrointestinal tract is functionally able to digest and absorb nutrients. Enteral feedings can be administered by one of two routes.

- The oral route is obviously the most physiologic and best tolerated.
- Tube feedings are administered in situations where the oral cavity cannot be used (eg, postsurgery of the pharynx or esophagus) and needs to be bypassed.

Formulas

- Two basic types of enteral formulas are available:
  - Elemental (also called monomeric or residue-free)
  - Nonelemental (polymeric)

- Monomeric formulas are absorbed directly from the gastrointestinal tract, usually by simple diffusion, with only minimal stimulation of intestinal or pancreatic enzymes. These formulas are most often indicated for patients with gastrointestinal tracts that are unable to enzymatically break down and actively transport complex nutrients (eg, patients with inflammatory bowel disease, short bowel syndrome, or pancreatic insufficiency).
- Polymeric diets consist of complex proteins, polysaccharides, long chain triglycerides, electrolytes, minerals, and vitamins. A normal digestive, proteolytic, and lipolytic capacity is required.
- Most commercially available polymeric and monomeric formulas supply 1 cal/mL. They differ in their osmolarity and protein sources. Table 7-6 lists the components for two common enteral formulas. There are also formulations available that supply 1.5 and 2.0 cal/mL for patients requiring additional caloric support.

Equipment

- Enteral nutrition may be delivered via nasal, gastric, or jejunostomy tubes. Nasal tubes may be passed into the stomach or duodenum.
- Gastric tubes may be placed via open surgery or percutaneously (ie, percutaneous endoscopic gastrostomy [PEG]).
- Nasogastric tube feedings should only be used in alert, conscious patients. In patients unable to maintain protective airway reflexes, aspiration may lead to life-threatening pulmonary complications. Nasoduodenal or gastric tubes should be used in patients at risk for aspiration.
- Fine-bore (10 French) silastic and polyurethane nasal tubes with metal-weighted tips are better tolerated than rigid Levine and Salem Sump tubes.

**Table 7-6 Enteral diet formulation**

<table>
<thead>
<tr>
<th></th>
<th>Monomeric</th>
<th>Polymeric</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cal/mL</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Cal-nitrogen ratio</td>
<td>149:1</td>
<td>153:1</td>
</tr>
<tr>
<td>mOsm/kg H₂O</td>
<td>630</td>
<td>450</td>
</tr>
<tr>
<td>Carbohydrate (g/L)</td>
<td>210</td>
<td>145</td>
</tr>
<tr>
<td>Protein (g/L)</td>
<td>38</td>
<td>37</td>
</tr>
<tr>
<td>Fat (g/L)</td>
<td>2.8</td>
<td>37</td>
</tr>
<tr>
<td>Na⁺ (mEq/L)</td>
<td>20</td>
<td>32</td>
</tr>
<tr>
<td>K⁺ (mEq/L)</td>
<td>20</td>
<td>32</td>
</tr>
<tr>
<td>Cl⁻ (mEq/L)</td>
<td>23</td>
<td>30</td>
</tr>
<tr>
<td>(mmol/L)</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Calcium (mEq/L)</td>
<td>25</td>
<td>6</td>
</tr>
<tr>
<td>Copper (mg/L)</td>
<td>1.0</td>
<td>1</td>
</tr>
<tr>
<td>Nutrient</td>
<td>Value 1</td>
<td>Value 2</td>
</tr>
<tr>
<td>---------------</td>
<td>---------</td>
<td>---------</td>
</tr>
<tr>
<td>Iodine (µg/L)</td>
<td>80</td>
<td>75</td>
</tr>
<tr>
<td>Iron (mg/L)</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Magnesium (mg/L)</td>
<td>6.5</td>
<td>4</td>
</tr>
<tr>
<td>Manganese (mg/L)</td>
<td>0.9</td>
<td>2</td>
</tr>
<tr>
<td>Zinc (mg/L)</td>
<td>10</td>
<td>15</td>
</tr>
</tbody>
</table>

### Tube feeding methods
- The position of the tube tip should be radiographically documented prior to feeding.
- With hyperosmolar formulas, feedings are started with a dilute, one-fourth–strength solution. The concentration may be gradually increased every 2 to 3 days.
- The initial rate of feeding should be about half of the final rate to permit the gastrointestinal tract to accommodate to the osmotic load. The diet is advanced by first increasing the volume and then the concentration.
- Tube feedings may be administered by three basic methods:
  - **Continuous drip (preferred method):** Using an infusion pump to ensure a constant rate over a 24-hour period.
  - **Bolus feedings:** Giving a large volume of formula at timed intervals (eg, 400 mL over 30 minutes every 6 hours).
  - **Timed feedings:** Administering a drip between set time intervals, usually during daytime when the patient is alert.

### Complications
- **Diarrhea:** The most common complication connected with tube feedings. It is usually caused by rapid administration of a high
osmotic formula. Decreasing the rate or the strength may remedy this situation. Stool should be assayed for Clostridium difficile and fat malabsorption in refractory cases.

Tube trauma: May cause ulceration of the gastrointestinal mucosa. This can be minimized by the use of soft tubes.

Inadvertent placement of the tube into the trachea or esophagus: May result in aspiration. Tube position should be confirmed radiographically before feedings are started.

Aspiration: May also be caused by poor patient position and regurgitation of gastric contents. The patient’s head should always be elevated to a 30-degree angle to prevent this.

Dehydration, hypernatremia, and azotemia: The most common metabolic derangements that occur as a result of osmotic variations. Excess water must be added to prevent these complications. Thirst is an early indication of a hyperosmolarity. Close monitoring of fluid balance, serum electrolytes, and urine specific gravity is essential.

Parenteral Nutrition

Used when the gastrointestinal tract is functionally or mechanically impaired. Parenteral nutrition is administered via a peripheral or central vein. Parenteral nutrition is more costly than enteral feeding and has a higher infection and complication rate.

General

- Parenteral alimentation involves the continuous infusion of a hyperosmolar solution containing sources of carbohydrate, protein, fat, vitamins, and trace elements. These nutrients are administered through an indwelling catheter.
- Parenteral alimentation is indicated for feeding critically ill patients who suffer from malnutrition, sepsis, trauma, or surgical stress when their gastrointestinal tract cannot be used.
- Parenteral nutrition may be given in quantities and concentrations to provide complete requirements. This is called total parenteral nutrition (TPN). Intravenous alimentation may also be used in a less concentrated
form to supplement inadequate oral intake. Most solutions used for TPN are extremely hyperosmolar (> 1,100 mOsm). These fluids are administered via a central line to minimize damage to the vein. Solutions for supplemental nutrition may be given through a peripheral vein. The osmolarity should be < 900 mOsm for peripheral administration.

Components of parenteral nutrition (Table 7-7)

Protein: Supplied by a variety of commercially available amino acid solutions. The concentration of these solutions ranges from 7% to 11.4%. The standard central vein alimentation usually contains an 8.5% or a 10% amino acid solution. A 7% concentration is used in peripheral vein solutions. There are special solutions available for patients in renal, hepatic, or cardiac failure.

**Table 7-7 Composition of parenteral nutrition formulas**

<table>
<thead>
<tr>
<th>Base solution</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose 50%</td>
<td>250 g/500 mL</td>
</tr>
<tr>
<td>Protein 8.5%</td>
<td>42.5 g/500 mL</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Additives to each unit</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium chloride</td>
<td>40 mEq</td>
</tr>
<tr>
<td>Potassium chloride</td>
<td>30 mEq</td>
</tr>
<tr>
<td>Potassium phosphate</td>
<td>15–30 mEq</td>
</tr>
<tr>
<td>Magnesium sulfate</td>
<td>8 mEq</td>
</tr>
</tbody>
</table>
Acetate 25 mEq

Additives to one unit daily

Calcium gluconate 10% 4.5 mEq

Multivitamin infusion (MVI) 10 mL

Trace elements

ZnSO₄ 5 mg

CuSO₄ 1–2 mg

CrCl 1.5 µg

MnCl 0.5 µg

NaSeO₃ 60 µg

Intravenous lipid emulsion (piggyback) daily

20% 100 g/500 mL

Added weekly (intramuscularly)

Folic acid 5 mg

Vitamin K 10 mg

Iron dextran 10 mg

Added monthly (intramuscularly)

Vitamin B₁₂ 1 mg
Carbohydrate sources: Intravenous solutions of 50% and 10% dextrose in water are used for central and peripheral vein administration, respectively; 500 mL of these solutions are mixed with 500 mL of amino acid solution, yielding final glucose concentrations of 25% and 5% per liter, respectively. Lipids: Given in 20% and 10% emulsions via a “piggyback” into the central or peripheral line.

Concentrations of electrolytes: These differ in the various stock solutions. Based on current laboratory values, additional electrolytes should be included daily to meet the specific needs of the patient.

Vitamin and trace element requirements: These are supplied in ready-mixed solutions that can be added to the intravenous solution daily. Vitamin K, 5 to 10 mg, is added to the solution once a week.

Regular insulin: May be added to each liter of solution as needed to prevent hyperglycemia.

Administration of parenteral nutrition

- Confirm central line placement radiographically before infusing alimentation.
- The starting infusion rate should be 50 to 100 mL/h. The rate can be slowly increased in 25-mL/h increments every day to allow for adjustment to the increased osmotic load. The final rate and volume infused is determined by the amount of calories desired.
- The patient should be carefully monitored. The volume and composition of the solution are adjusted as needed based on physical findings and laboratory data. Vital signs and intake/output volumes should be recorded regularly. Weight is checked daily. Electrolytes are measured daily until stable, then are checked every 2 to 3 days. Complete blood cell count, liver enzymes, blood urea nitrogen, and magnesium and phosphate levels are measured once a week.
- Hyperglycemia may occur after parenteral alimentation is started. Blood
glucose levels should be checked daily during the first few days of infusion. Regular insulin may be added to each unit of solution as necessary. As the carbohydrate tolerance stabilizes, glucose levels need only be checked every 2 to 3 days.

Complications

<table>
<thead>
<tr>
<th><strong>Metabolic</strong></th>
<th><strong>Catheter</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Hypoglycemia</td>
<td>• Pneumothorax</td>
</tr>
<tr>
<td>• Hyperglycemia</td>
<td>• Thrombosis</td>
</tr>
<tr>
<td>• Hyperosmolar nonketotic hyperglycemia</td>
<td>• Sepsis</td>
</tr>
<tr>
<td>• Metabolic acidosis</td>
<td></td>
</tr>
<tr>
<td>• Hypokalemia</td>
<td></td>
</tr>
<tr>
<td>• Anemia, vitamin B\textsubscript{12} deficiency</td>
<td></td>
</tr>
<tr>
<td>• Liver enzyme elevation</td>
<td></td>
</tr>
<tr>
<td>• Fluid overload</td>
<td></td>
</tr>
</tbody>
</table>
chapter 8

Use of Blood and Blood Products

Approximately 15 million blood transfusions are administered annually to sick patients in the United States, and around 4.9 million patients receive blood each year. The incidence of posttransfusion infection is: hepatitis B, 1 in 205,000; hepatitis C, 1 in 2,000,000; and HIV, 1 in 2,135,000.

Selection of Donors
Criteria

Potential donors must:

- Be 17 years or older in most states; minimum age in some states is 16 years.
- Be in good health. *Healthy* means that they feel well and can perform normal activities. If they have a chronic condition such as diabetes or high blood pressure, *healthy* also means that they are being treated and the condition is under control.
- Have not donated whole blood within the past 8 weeks (56 days).
- Have not undergone double red blood cell (RBC) donations in the past 16 weeks (112 days).
- Have not donated platelets in the past 3 days.
- Not have hemochromatosis.
- Not have had hepatitis caused by a virus, or unexplained jaundice, since age 11 years. This includes those who had hepatitis with cytomegalovirus (CMV) or Epstein-Barr virus (EBV).
- Not have AIDS, ever had a positive HIV test, or have done something that puts them at risk for becoming infected with HIV.
- Not have used intravenous (IV) drugs that were not prescribed by a physician.
- Not be pregnant.
• Have a systolic blood pressure of no more than 180 mm Hg, a diastolic blood pressure of no more than 100 mm Hg, an oral temperature less than 99.5°F, weight of at least 110 lb, and minimum hemoglobin of 12.5 g/dL.
• Wait for 3 days after having an oral surgery procedure and not be on antibiotics for infection.
• Not have a first-degree blood relative who had Creutzfeld-Jacob disease.
• Not have received a dura mater transplant or human pituitary growth hormone.
• Not have vascular insufficiency, including coronary insufficiency; congestive heart failure; or a cardiac arrhythmia.

**Autologous Transfusions**

Preoperative phlebotomy: A technique in which a patient’s blood is drawn prior to elective surgery and the blood or blood components are reinfused during or following the procedure. Autologous blood donation is considered a medical procedure that requires a written prescription, and the rules of eligibility are less strict than for regular volunteer donations. Whole blood can be stored for 35 days when collected with citrate phosphate dextrose adenine (CPDA-1) anticoagulant. RBCs collected with an additive solution can be stored for 42 days. Frozen RBCs and plasma can be stored for many years.

Advantages: No risk of transfusion-transmitted infections, elimination of technical errors in the obtaining and crossmatching of blood, absence of the risk of alloimmunization to both cellular and plasma protein antigen, increased stimulation to bone marrow erythropoiesis, and decreased cost to the patient.

Disadvantages: Anemia, hypovolemia, and possible loss of the donated blood when surgery is delayed or cancelled

• All autologous donors should receive iron supplementation until the hemoglobin level is normal. Ferrous gluconate, 320-mg tablet every 8 hours, is well tolerated, but may cause constipation.
• The lower limit for autologous transfusion is a hematocrit of 34% when donations are to be given at weekly intervals. The final donation should be given at least a week before surgery.
Only ABO and Rh typing have to be determined prior to administration of an autologous transfusion. However, it is safer for both the patient and the staff handling the blood if full laboratory processing takes place.

**Changes During Storage of Whole Blood**

All blood products that are stored undergo changes. Citrate phosphate dextrose (CPD) is the standard preservative. A unit of whole blood (450 to 500 mL) contains 63 mL of CPD anticoagulant.

Platelets are rendered inadequate after 3 days of preservation in CPD and, when blood is stored at 4°C, the acidity of the blood mixed with CPD gradually falls to a pH of 6.5 after 2 weeks of storage. The level of 2,3-diphosphoglyceric acid (2,3-DPG) gradually declines until it is virtually gone at the end of 3 weeks. This is of critical importance because 2,3-DPG regulates the association and disassociation of oxygen from hemoglobin. In the absence of 2,3-DPG, oxygen is not available for tissue perfusion. With storage, there is also a loss of factors V and VIII, and the levels of ammonia, potassium, and lactate rise.

**Metabolic Changes Caused by Transfusions**

Hyperkalemia and alkalosis: Citrate in the blood preservative contributes to the hyperkalemia seen with use of stored blood. Citrate is also metabolized to yield bicarbonate, which contributes to the alkalosis noted.

Hypocalcemia: Results from the presence of the citrate preservative. Because citrate is a chelating agent, it binds the ionized calcium. The resultant hypocalcemia can cause cardiac arrhythmias. A normal adult can metabolize the citrate in one unit of blood in 5 minutes. It is recommended that 13.5 mEq of calcium be given with every five units of blood when that amount is given within 30 minutes.

Hypothermia: Hypothermia is an expected finding with multiple, rapid transfusions of stored blood. Because blood is stored at 5°C, patients have increased difficulty in metabolizing potassium and the citrate in the preservative if the administered blood is cold. There is also an increased affinity for oxygen by the hemoglobin, and thus a lessened ability of the blood to oxygenate the tissues. Blood, therefore, must be warmed.
Coagulopathy: As already stated, factors V and VIII decay during storage. The absence of functional platelets is also a major contributor to the coagulopathy noted with massive transfusions. A platelet concentrate should be given when a patient receives 10 or more units of blood within 1 hour.

**Complications Associated with Blood Transfusions**

Any unexpected symptom occurring during or shortly after a blood or blood product transfusion should be considered to be due to the transfused product until proven otherwise. The most dreaded reaction is a hemolytic transfusion reaction, which occurs once in 3,000 transfusions and only when whole blood or packed cells are given. It is most commonly caused by improper identification of the patient and donor blood. The reaction results from the agglutination of the incompatible donor cells by the antibodies in the recipient’s plasma. There are two types of hemolytic reaction. The most common is the delayed type, which is marked by jaundice appearing hours or days after transfusion. The less common is the acute type, which is marked by immediate symptoms.

**Symptoms of Hemolytic Transfusion Reaction**

- Back pain, chills, fever, flushing, hypotension, and occipital headache.
- In severe cases, there may also be disseminated intravascular coagulation (DIC), hemoglobinuria, oliguria or anuria, chest pain, dyspnea, and shock.
- When transfusion occurs under general anesthesia, most of these symptoms will be absent.
- The first signs of a transfusion reaction may be oliguria, hemoglobinuria, and renal failure.
- Risk of an acute hemolytic transfusion reaction is 1 to 4 per million units transfused, with a mortality rate of approximately 50%.
- Delayed hemolysis most commonly occurs in the repeatedly transfused patient 5 to 10 days after the transfusion and is marked by a fall in the hematocrit together with jaundice or a rise in bilirubin.

**Diagnosis of Hemolytic Transfusion Reaction**

When a suspected transfusion reaction occurs:
- Stop the transfusion but keep the IV line open with slow infusion of 0.9% sodium chloride. The risk of a severe reaction is much greater if more than 200 mL of incompatible blood has been transfused in an adult.
- See if the patient was receiving the blood intended for him or her.
- Confirm the reaction by drawing an anticoagulated blood sample, centrifuging it, and observing the supernatant for free hemoglobin in the plasma.
- Perform a direct Coombs test on the patient’s RBCs.
- Compare pretransfusion and posttransfusion levels of hepatoglobin.
- If free hemoglobin is detected in the plasma and/or the Coombs test is positive, dispatch the transfused blood unit with the patient’s clotted blood sample to the blood bank for ABO incompatibility studies.
- Finally, check to see if the hemoglobin and hematocrit have risen after the transfusion. In a normal 70-kg adult there should be a 3% rise in the hematocrit after the transfusion of one unit of packed cells or whole blood.

Management of Incompatible Blood Transfusion Reaction

To prevent renal failure and wash out the tubules
- Stop the transfusion, but keep the IV port open.
- Give 1,000 mL of 0.9% sodium chloride over 1 hour.
- Give supplemental oxygen.
- Continue IV fluids to maintain urine flow over 100 mL/h, and remove the remaining free hemoglobin from the tubules.
- If unable to maintain urine output greater than 100 mL/h, give furosemide, 20 to 80 mg IV, to achieve prompt diuresis.
- Place a central venous line to guard against overhydration.

If bleeding occurs due to DIC
- Perform prothrombin time (PT)/international normalized ratio (INR), partial thromboplastin time (PTT), and complete blood cell (CBC) with platelet count.
- The use of heparin is controversial. When indicated, doses of 500 to 750 units are usually adequate.
- In replacement therapy, platelets can be given to replete to 30,000 or 50,000 per microliter, if possible.
• Correct appropriate clotting factor deficiencies with cryoprecipitate or fresh frozen plasma.
• If cause of mismatch reaction is solved, resume blood transfusion, if necessary.

Anaphylactoid Reaction

This reaction to blood or blood products is extremely rare but potentially lethal. Symptoms are nausea, abdominal cramps, diarrhea, and emesis, ending in severe hypotension. Fever is absent, but chills are common. The most common cause of an anaphylactoid reaction is the presence of antibodies to immunoglobulin A (IgA).

Treatment

• Stop transfusion and do not restart.
• Infuse 0.9% sodium chloride solution IV to counteract hypotension.
• Give high-flow oxygen supplementation and support the airway as needed.
• Give epinephrine, 0.4 mL of a 1:1,000 solution subcutaneously, or 0.1 mL of a 1:1,000 solution diluted in 10 mL of normal saline IV slowly.

To prevent an anaphylactic transfusion reaction in sensitized IgA-deficient patients, use RBCs that have been freed of IgA intake plasma or, alternatively, use autologous blood.

Fever Without Hemolysis

This is a common transfusion reaction. It usually does not occur until 1 or 2 hours after completion of the transfusion. It is not accompanied by hemolysis. The reason for the fever is the reaction of the patient’s antibodies to the granulocytes in the donor’s blood. Transfusion of more than 250 mL of infused blood or blood cells is usually required to cause symptoms.

Treatment

• Stop transfusion and rule out the possibility of hemolysis.
• Do not restart transfusion.
• Administer acetaminophen, 650 mg orally.
• Use white cell pooled blood if another transfusion is needed.
Urticaria
This occurs in about 1% or 2% of transfusions.

Treatment
- If this is the only finding, the transfusion can be continued (the only reaction where this can be done).
- Slow down the transfusion.
- Give diphenhydramine, 50 mg IV or intramuscularly (IM).
- Resume transfusion slowly after hives have disappeared.

Heart Failure
Too rapid a transfusion can result in fluid overloading, with resultant congestive heart failure. This is especially true in the very young, in the elderly, and in patients with chronic anemia or heart disease. The transfusion rate should be well below 200 mL/h. Consider oxygen supplementation and diuresis as needed.

Infections
Any infectious agent present in the blood of the donor during donation is potentially transferable. Bacterial contamination is rare; the problem is almost exclusively viruses. Human hepatitis viruses are the most commonly transmitted.

Transfusion of Blood and Blood Components
The decision to transfuse a patient should take into consideration the following factors: intravascular volume, duration of the anemia and the operation, probability of extended blood loss, and physiologic condition of the patient (ie, impaired pulmonary or myocardial function, cerebrovascular or peripheral circulatory disease). As a broad guideline, healthy patients with hemoglobin values greater than 10 g/dL rarely require perioperative transfusion. Those with values less than 6 g/dL due to acute anemia will require RBC transfusion.

Whole Blood
Rarely given and only indicated where there is an acute need to replace both RBCs and plasma.
RBC Transfusions

Also referred to as packed cells, red cells, packed red blood cells, and RBCs.

- If possible, reconstituted packed RBCs are preferred to whole blood. It is specifically indicated when hypervolemia is a problem.
- With packed cells, more erythrocytes can be transferred than with an equal volume of whole blood.
- A RBC transfusion should be used in patients with chronic anemia and in patients who are not in danger of having hypovolemic hypotension. It is also indicated when other blood products are needed, such as platelet concentrates and plasma derivatives. It can be used when compatible blood is unavailable. Packed cells minimize the risk of a “minor” incompatibility reaction.

Washed RBCs

In this process, the majority of the plasma fraction is removed and the product consists mainly of RBCs suspended in saline. It is indicated in patients with a history of an allergic transfusion reaction to a component of the plasma, which occurs in 1% to 3% of blood recipients. Washed RBCs contain 10% to 20% fewer RBCs than the original unit.

Frozen Deglycerolized RBCs

The RBCs are frozen at −30°C or less in glycerol, a cryoprotective agent. Glycerol allows the RBCs to be stored for many years without losing their characteristics. It is therefore indicated for the preservation of rare blood and autologous transfusions. The glycerol has to be washed out after thawing. Depending on the freeze-thaw technique used, 7% to 30% of the original red cell mass is lost. Following thawing, the RBCs must be used within 24 hours.

Platelet Concentrate

- Also referred to as platelets, pooled platelets, and plateletpheresis. Pooled platelets (random-donor platelets [RDP]) refers to the shared donation of 4 to 10 donors and contains approximately $8.0 \times 10^{10}$ platelets per 50-mL bag. Plateletpheresis (single-donor platelets [SDP]) is a single-donor automated collection and contains 3.5 to $4.0 \times 10^{11}$ platelets per 250-mL bag.
Platelets are highly immunogenic and form potent platelet-destroying alloantibodies. Seventy percent of patients become refractory after repeated platelet transfusions. Patients who have a platelet count below 50,000 per microliter need a transfusion if they are to undergo surgery. Patients with a platelet count of 60,000 to 100,000 per microliter should be transfused only if there is excessive bleeding during surgery.

Platelet transfusion is also often indicated in conditions of low platelet count and decreased rate of platelet production such as in acute leukemia, aplastic anemia, and bone marrow aplasia caused by chemotherapy. It is also useful at times in conditions marked by a low platelet count and normal or increased platelet production such as after massive transfusions, and with splenomegaly, thrombocytopenic purpura, idiopathic thrombocytopenic purpura, and DIC.

One unit of platelets is administered for each 10 kg of body mass. In the average adult, one unit increases the platelet count by at least 7,000 to 10,000/mm$^3$ for each RDP given or 30,000 to 60,000/mm$^3$ for each SDP given. The half-life of the transfused platelets is approximately 36 hours. The bleeding time should be measured immediately before and 3 hours after transfusion.

**Fresh Frozen Plasma (FFP)**

- Also referred to as *plasma* or *cryo-poor plasma*.
- Fresh frozen plasma is used as a replacement for deficiencies of factors II, V, VII, IX, X, and XI when specific component therapy is neither available nor desirable. In the average adult, each unit of FFP increases the level of all clotting factors by 2% to 3%. Bleeding can generally be controlled with FFP in a dosage of 10 mL/kg of body weight.
- A unit of FFP will contain approximately 200 units of activity of each of the clotting factors. A unit of clotting factor activity is the amount present in 1 mL of fresh normal plasma. Fifteen percent of clotting activity is lost in the process of freezing and thawing. FFP transfusion is seldom indicated if the PT and PTT are less than 1.5 times normal.
- Patients who are anticoagulated with warfarin are deficient in the vitamin K–dependent factors II, VII, IX, and X, as well as proteins C and S. If the patient is bleeding acutely, vitamin K, the usual means of therapy, cannot be used and plasma is used to achieve prompt hemostasis.
FFP is a source of the inhibitor antithrombin III. It is given to patients with this deficiency when undergoing surgery or to those who require heparin for the treatment of thrombosis.

• FFP is also a source of immunoglobulin and can be given to infants and adults with a humoral immunodeficiency.

• The risks of using FFP are the same as those associated with whole blood transfusion, with the exception of a mismatch reaction.

Cryoprecipitated Antihemophilic Factor

• Also referred to as cryoprecipitate or cryo.
• Cryoprecipitate contains concentrated levels of fibrinogen, factor VIII, von Willebrand factor (vWF), factor XIII, and fibronectin.
• Cryoprecipitate is considered an acellular blood component. Compatibility testing is unnecessary.
• Each unit of cryoprecipitate should contain at least 80 IU of factor VIII and 150 mg of fibrinogen.
• One unit of cryoprecipitate per 10 kg of body weight raises plasma fibrinogen concentration by about 50 mg/dL in the absence of continued consumption.
• Cryoprecipitate is indicated for bleeding associated with fibrinogen deficiencies and factor XIII deficiency.

Coagulation Disorders

Human Blood Coagulation

• The clotting cascade consists of intrinsic and extrinsic systems, with a final common pathway starting at factor X (Figs 8-1 and 8-2). The extrinsic system depends on tissue thromboplastin to activate factor VII for coagulation to occur. For the intrinsic system coagulation pathway to be initiated, only a prolonged incubation of the blood with a surface is necessary for clot formation.

• Both the extrinsic and intrinsic systems lead into the final pathway via the activation of factor X and the formation of thrombin. Thrombin converts fibrinogen to fibrin, which polymerizes to form a clot.
**Fig 8-1** Intrinsic and extrinsic pathways. HMWK, high molecular weight kininogen; α, activated form of the circulating zymogen.
**Evaluation of Blood Coagulation**

**Required Tests**
- Platelet count and evaluation of platelet quality
- Smear
- PT/INR and PTT to determine a defect in the intrinsic or extrinsic pathways
- Measurement of concentration of blood factors and thrombin and determination of reptilase time to evaluate the ability of fibrinogen to polymerize

**Prothrombin time**
- Measures factors II, V, VII, X, and fibrinogen. Normal value is less than 13.5 seconds.
- A doubling of the PT indicates a factor activity of 20%.
- A PT less than 14 seconds is necessary for adequate hemostasis.
- INR: PT can vary from hospital to hospital or from batch to batch, depending on the prothrombin reagent that is used. With INR, all results
are standardized using the international sensitivity index for the particular thromboplastin reagent and instrument combination used to perform the test. A normal INR is 1.0. The therapeutic anticoagulation range for patients on warfarin is generally between 2.0 to 2.5.

Partial thromboplastin time

- If the clotting time exceeds 50 seconds, tests for specific factors are indicated. Normal time is < 39 seconds. The test measures the integrity of the intrinsic pathway prior to the activation of factor X.
- Depends on the following factors:
  — Prekallikrein
  — Kininogen
  — Factors II, V, VIII, IX, X, XI, XII, and fibrinogen
- The preferred test for patients on heparin therapy.

Fibrinogen level: Normal is > 150 to 450 mg/dL; bleeding occurs with levels < 50 mg/dL.

Fibrinogen degradation products: Normal is < 10 µg/mL. This is an immunologic measurement of the breakdown products of fibrinogen or fibrin. This value will be elevated:

- Whenever plasmin degrades fibrin or fibrinogen
- In cases of DIC
- When the patient is receiving streptokinase
- Postoperatively

Note: High levels of fibrinogen or heparin may cause false-positive results.

Platelet count: Normal value is 150,000 to 400,000 mm$^3$. A spuriously low platelet count is seen:

- If clumping occurs on the smear
- In CBC tubes with ethylenediaminetetra acetic acid (EDTA) as an anticoagulant

If the count is low, confirm by studying smear at ×100. Get average count of six fields. Multiply number by 18,000 to get an estimated count. If not in agreement with the CBC, repeat count, especially if clumping is seen.
Platelet function tests

_Ristocetin test:_ Most sensitive. It is abnormal in 75% of all patients with von Willebrand disease. The test is also abnormal in patients with Bernard-Soullier giant platelet syndrome. In this condition you will see only giant platelets on a smear.

_Thrombin test:_ Normal value within 3 seconds of control. The test measures the ability of fibrinogen to polymerize. Performed by adding exogenous thrombin to the patient’s plasma and measuring the clotting time. Factors that cause a prolonged thrombin time include:
- Hypofibrinogenemia
- Dysfibrinogenemia
- Heparin
- Fibrinogen degradation products

_Reptilase time:_ Based on an enzyme that converts fibrinogen to fibrin. Normal value within 5 seconds of the control. Prolongation will occur for the same reasons that thrombin time is prolonged, with the exception that it will be normal in the presence of heparin. It is also a more sensitive test than the thrombin time. Fibrin degradation products have less effect on reptilase time than on thrombin time.

Interpretation of Coagulation Tests

Abnormal PT/INR, normal PTT
- Early vitamin K deficiency
- Early effect of sodium warfarin (Coumadin)
- Factor VII deficiency or inhibitor

Normal PT/INR, abnormal PTT

Deficiency or inhibition of:
- Factor XI
- Factor IX
• Factor VIII
• Lupus anticoagulant
• Heparin in low doses

Abnormal PT/INR, abnormal PTT
• Vitamin K deficiency
• Deficiency or inhibition of:
  —Factor X
  —Factor V
  —Factor II

• Early DIC
• Deficiency of protein C inhibitor, causing deficiency of both factors V and VIII
• Mild liver disease

Abnormal thrombin time
• DIC
• Dysfibrinogenemia
• Hypofibrinogenemia
• Elevated fibrinogen degradation products
• Heparin therapy
• Severe liver disease

Tests for Disseminated Intravascular Coagulation

DIC occurs when both procoagulants and fibrinolysins are activated. During the clotting and lysis that occur in the small vessels, platelets and clotting factors are consumed with resultant lowering of their concentration in the plasma. Fibrin split products (FSPs) are also released. Normal FSP concentration in serum is less than 10 mg/mL. The most common conditions causing DIC are shock, tissue necrosis, and amniotic fluid embolisms during labor.

Laboratory findings noted during acute DIC
• Decreased levels of clotting factors
• Decreased number of platelets
• Prolonged PT/INR, PTT
• Decreased levels of fibrinogen and factors VIII and V
• Laboratory findings may not be abnormal in chronic DIC

Fibrinogen and fibrin split products: Fibrin split products interfere with the PT. Blood concentration of fibrinogen degradation products increases in DIC, with lysis of large clots, liver disease, shock, transplant rejection, and thrombotic and thrombocytopenic purpura.

**Hemophilia A**

Classic hemophilia is due to deficiency of coagulation factor VIII in 85% of afflicted patients. The disorder is categorized according to its clinical severity. Most patients with severe hemophilia have less than 1% of factor VIII, moderately severe between 1% and 5%, and mild more than 5%.

• Clinical diagnosis: Family history and spontaneous hemarthrosis
• Laboratory diagnosis:
  — Prolonged PTT
  — Prolonged thrombin time
  — Normal bleeding time and fibrinogen levels

Management

Treatment for moderately and severely afflicted patients is infusion of factor VIII plasma concentrate, pretreated to eliminate viruses. Recombinant factor VIII is such a product.

Mild condition: Raise factor VIII to 25% level via single infusion.

Moderate condition: Initially raise factor VIII to 50% level. Repeat infusion, if necessary. Maintain the level at 25% for 2 to 3 days.

Severe condition: Raise factor VIII to 100% level and maintain at that level for several days.

Examples: For severe hemophiliacs undergoing multiple tooth extractions, levels should be raised to 100% and maintained at a minimum level of 50% for 5 to 7 days. Epsilon-aminocaproic acid 100 mg/kg should be given 4 hours before surgery to inhibit conversion of plasminogen to plasmin. It should be continued every 6 hours for 7 days postoperatively. Alternatively, instead of aminocaproic acid, tranexamic acid 10 mg/kg IV can be used 4 hours before surgery and 2 mg every 8 hours for 7 days postoperatively.
For patients with mild hemophilia who are undergoing minor surgery, accepted therapy is desmopressin acetate (DDAVP) 0.3 to 0.4 mcg/kg IV 2 hours preoperatively. Epsilon-aminocaproic acid is given 100 mg/kg orally every 6 hours for 7 days. Alternatively, tranexamic acid can be given 2 g every 8 hours for 7 days postoperatively.

**Hemophilia B (Christmas Disease)**
This is an X-linked recessive disorder that primarily affects males. The defective protein is factor IX, which is present in 15% of patients with hemophilia.

Clinical diagnosis
- Family history
- Spontaneous hemarthrosis

Laboratory diagnosis
- Prolonged PTT
- Prolonged thrombin time
- Reduced levels of factor IX

Treatment: Protocol for infusion of factor IX is identical to that for factor VIII. The use of aminocaproic acid or tranexamic acid is also the same. Factor IX concentrates have a half-life of 24 hours.

**von Willebrand Disease**
von Willebrand disease is hereditary and predominantly autosomal dominant. The condition affects approximately 1 in 8,000 individuals. Nearly 1% of the population has asymptomatic deficiencies. The primary deficiency in the disorder is the defective protein vWF, which prevents proper platelet adhesion. The secondary coagulation abnormality is due to a decreased level of factor VIII. There are six diagnostic categories of von Willebrand disease. Type I is the most common, constituting 80% of cases. It is marked by a decrease in the concentration of the multimers (high molecular weight protein chains), although they appear normal in function and structure.

Laboratory findings are:
- Prolonged bleeding time
- Reduced factor VIII coagulant activity
- Reduced ristocetin cofactor
- Increased factor VIII antigen

Treatment

For mild type I disorders: Desmopressin acetate (DDAVP) 0.3 to 0.4 mg/kg IV diluted in 50 mL of normal saline given over 15 to 30 minutes. DDAVP increases vWF and factor VIII levels threefold to sixfold in 90 to 120 minutes. The level is maintained for 4 to 8 hours. Tachyphylaxis is a limitation. Repeated doses can be given after at least 12 hours to ensure adequate endothelium cell stores.

Side effects (eg, mild headache, tachycardia, hypertension, and facial flushing) are mainly due to vasodilatation.

For types IIa and IIb: DDAVP is contraindicated in both vWF type IIa and IIb because thrombocytopenia and an increased incidence of bleeding can result. For more serious disorders, commercial factor VIII concentrates are available (human P) for IV use.

For oral and maxillofacial surgery procedures: Transfuse with FFP, cryoprecipitate, recombinant factor VIII, or porcine factor VIII to achieve factor VIII and vWF levels of 1 U/mL. Supplement with epsilon-aminocaproic acid starting 12 to 24 hours before surgery and continuing for 12 days postoperatively.
Basic Patient Management Techniques

Peripheral Venous Cannulation

Indications
- To gain access to the venous system for administration of drugs and fluids
- To obtain venous blood for various laboratory tests

Materials
- Intravenous (IV) cannula (21-gauge needle or larger, butterfly needle, catheter-over-needle, or catheter-through-needle devices) and syringe
- Sterile IV tubing and IV fluids
- Tourniquet
- Local anesthetic (3-mL syringe with 1% lidocaine, 25- or 27-gauge needle)
- Surface antiseptic

Technique
- Select a suitable site in upper extremity (eg, antecubital fossa, dorsum of hand).
- Apply tourniquet proximally.
- Cleanse skin with surface antiseptic.
- If large-bore (16-gauge or greater) cannula is used, anesthetize skin with local anesthetic.
- Stabilize vein (to prevent rolling and sliding) by applying traction distally with thumb on skin.
- With the bevel facing up, pierce skin with needle at about 20 or 30 degrees to entry point, directly over vein.
• Puncture vein and advance needle until blood returns; then advance an additional 1 to 2 mm. Remove the tourniquet.
• If catheter-over-needle device is used, rotate catheter and advance into vein, withdraw needle and discard.
• Connect IV tubing.
• Apply sterile dressing, and stabilize needle with adhesive tape.

Complications
• Infiltration
• Hematoma
• Phlebitis
• Cellulitis/sepsis
• Thrombosis

**Venous Cutdown**

Indications
• When peripheral cannulation is not technically possible

Materials and Supplies
• Venous cutdown tray
• IV cannula
• Sterile IV tubing
• Local anesthetic
• Surface antiseptic
• Sterile gloves and gown, mask, protective eyewear
• Tourniquet

Technique
• Select site:
  — Antecubital vein
  — Saphenous vein, ankle

• Place tourniquet around proximal leg or around proximal arm.
• Prepare and drape area; anesthetize area with local anesthetic.
• Make small transverse skin incision over vein.
• Approach vein by blunt dissection.
Identify and isolate vein.
Place silk ligature proximal and distal to venous access site.
Tie distal ligature.
Place IV cannula device through skin at separate entry point distally.
Incise vein with scalpel.
Release tourniquet.
Insert cannula into vein and advance it.
Connect IV tubing.
Tie proximal ligature around vein and cannula.
Close wound with interrupted 4-0 nylon sutures.
Apply antibiotic ointment to wound and skin entry point.
Apply sterile dressing and tape cannula in place.

Complications
- Wound infection
- Hematoma
- Phlebitis
- Cellulitis/sepsis
- Thrombosis

Central Venous Cannulation (Subclavian Vein)

Indications
- Emergency administration of IV fluids/drugs
- Measurement of central venous pressure
- Parenteral nutrition (hyperalimentation)

Materials
- Central line tray
- Sterile gloves and gown, mask, protective eyewear
- IV cannula, 10-mL syringe
- Sterile IV tubing
- Local anesthetic
- Surface antiseptic

Technique
- Place patient in supine position with legs slightly elevated
(Trendelenburg position).
- Place rolled towel under shoulders to hyperextend neck.
- Palpate landmarks: suprasternal notch, clavicle.
- Anesthetize skin inferior to junction of middle and medial third of clavicle.
- Use 14-gauge needle attached to 10-mL syringe containing 2 mL of normal saline.
- Puncture skin about 1 to 2 cm below junction of middle and medial third of clavicle.
- Advance needle, aiming for sternal notch, directing it in a medial, superior, and cephalad direction.
- Confirm entry into subclavian vein by aspiration.
- Rotate bevel of needle caudally toward superior vena cava.
- Disconnect syringe and place thumb over cannula hub to prevent bleeding or air entry.
- Advance catheter through needle into superior vena cava (approximately 15 cm).
- Turn head toward ipsilateral side during advancement.
- Stop advancement if resistance is encountered; do not pull back catheter — movement may shear off tip and embolize it.
- Withdraw needle and cover the needle guard.
- Aspirate and flush cannula with saline-filled syringe.
- Attach sterile IV tubing and begin infusion.
- Suture cannula and needle guard to skin.
- Apply antibiotic ointment to puncture site.
- Cover with sterile transparent dressing.
- Obtain chest radiograph to observe cannula position in superior vena cava and check for hemothorax or pneumothorax.

Complications
- Pneumothorax
- Hemothorax
- Hematoma
- Internal jugular vein cannulation
- Air embolism
- Catheter tip embolization
- Hydrothorax/hydromediastinum
Intraosseous Vascular Access

Indications

• Immediate access for emergency delivery of fluids, drugs, or blood products when venous access is not obtainable, especially in children

Materials

• Sterile gloves, gown and mask, protective eyewear
• Surface anesthetic
• IV solution and tubing
• Disposable 16- or 18-gauge intraosseous needle, 16- or 18-gauge spinal needle with stylet, or battery-powered needle inserter
• Lidocaine 1% with 25- or 27-gauge needle and 3-mL syringe
• Gauze and adhesive tape

Technique

• Select site:
  — Proximal tibia
  — Distal tibia
  — Proximal humerus
  — Iliac crest

• Cleanse and drape site.
• Infiltrate local anesthetic.
• Holding the needle in the palm of the hand with the index finger 1 to 1.5 cm from the tip of the needle, insert it through the skin and advance it through the cortex with a screwing motion. A sudden “give” is felt when the needle enters the marrow cavity.
• Withdraw stylet and attach the IV tubing.
• Apply antibiotic ointment and sterile dressing, and stabilize the needle with tape.

Complications
- Infiltration
- Hematoma
- Rarely osteomyelitis

**Arterial Line (A-Line) Placement**

**Indications**
- Blood gas measurement
- Continuous blood pressure measurement in hemodynamically unstable patient

**Contraindications**
- Infection at site of insertion
- Traumatic injury proximal to site of insertion

**Materials**
- Arterial line kit or 20- to 22-gauge angiocatheter and needle
- Surface antiseptic
- Lidocaine 1%
- Sterile dressing and tape

**Sites**
- **Radial artery:** Palpate in distal part of the forearm between radius and flexor carpi radialis muscle. When using this vessel, do the Allen test first to check for collateral circulation. Compress the radial and ulnar arteries while having the patient rapidly clench and unclench the fist, or while the patient’s arm is raised above the heart level for several seconds, to force blood from hand. Release pressure on the radial artery and observe the palmar surface of the hand. An erythematous blush indicates ulnar patency. Repeat procedure with release of pressure on the ulnar artery to ensure patency of the radial artery.
- **Dorsalis pedis artery:** Palpate on the arch of foot between the first and second metatarsal.
- **Femoral artery:** Palpate below the inguinal ligament midway between the anterior superior spine of the ilium and the pubic symphysis.
- **Brachial artery:** With arm extended and palm up, palpate as it courses medial to the biceps muscle and tendon into the antecubital fossa.
• **Axillary artery:** Palpate in axillary space with the arm abducted and rotated externally.

**Technique**
- Prepare injection site.
- Inject local anesthetic if patient is awake.
- Pass needle with the bevel up through the skin and into the vessel. Insert needle at 45 to 60 degrees for radial artery and at 90 degrees for femoral artery.
- Look for flash of blood. Once in artery, slide the angiocatheter into the vessel and remove the needle.
- Connect tubing, cover area with dressing, and secure catheter.
- Attach to monitoring equipment.

**Complications**
- Kinking of catheter
- Blockage of catheter
- Air embolism
- Hematoma
- Thrombosis
- Infection

**Nasogastric Tube Placement**

**Indications**
- To minimize nausea and vomiting secondary to swallowed blood after orthognathic or other extensive intraoral surgery
- Adynamic ileus

**Contraindications**
- Patients with head trauma, maxillofacial injury, or anterior fossa skull fracture (to avoid passing the tube through a fractured cribriform plate)
- Comatose patients who may be stimulated to vomit into an unprotected airway

**Materials**
- Nasogastric tube
• Water-soluble lubricant
• Catheter-tip syringe
• Normal saline
• Cup of water and straw

Technique
• Place patient in sitting position.
• Estimate tube length to be passed (distance of nose to earlobe plus distance of earlobe to xiphoid process—approximately 50 cm in adults).
  Mark distance on tube with tape.
  • Lubricate end of tube.
  • Elevate nasal tip and advance tube along nasal floor into nasopharynx.
  • Once the tube is in the pharynx, have the patient sip water through a straw to help swallow the nasogastric tube. In a conscious patient, placing the chin to the chest facilitates passage of the tube.
  • Advance tube to appropriate distance as patient swallows.
  • Inject air into tube with catheter-tip syringe and auscultate epigastrium.
    There should be an audible bubbling sound if it is correctly placed.
  • Tape tube to nose.
  • Connect to low, intermittent suction device.

Complications
• Gastric/esophageal erosion
• Epistaxis
• Tracheal intubation
• Otitis media
• Aspiration pneumonia secondary to vomiting

Chest Tube Placement
Indications
• Pneumothorax
• Hemothorax
• Pleural effusion
Materials

- Chest tube tray
- Chest tube (28 to 32 Fr for adult or teen male; 28 Fr for adult or teen female; 18 Fr for child) or Fuhrman catheter
- Local anesthetic
- Sterile gloves and gown, mask, protective eyewear
- Surface antiseptic
- Three-bottle water-seal suction or pleurovac system

Technique

Chest tube

- Elevate head of bed.
- Place patient in lateral decubitus position.
- Prepare and drape area.
- For pneumothorax, use second, third, or fourth intercostal space and advance tube to lung apex.
- For fluid removal (hemothorax or effusion), use sixth or seventh intercostal space and advance tube posteriorly and basally.
- Palpate intercostal space in either the anterior axillary or midaxillary line.
- Infiltrate skin and subcutaneous tissues down to pleura in the surgical area with local anesthetic.
- Make a horizontal skin incision parallel to the rib margin about 2 to 3 cm in length one interspace inferior to chosen entry site.
- Bluntly dissect superiorly to superior border of rib.
- Perforate pleura and enter pleural space.
- Spread with a clamp (Kelly or curved 6-inch Peon) to enlarge space to accommodate chest tube.
- Remove clamp and explore pleural space with your fifth finger to make sure you feel lung or a space and not liver or spleen.
- Place clamp on distal end of chest tube.
- Place another clamp on proximal end of chest tube and use it to advance the tube through the incision and over the superior edge of the rib into the pleural space.
- Remove the proximal clamp and connect distal end of tube to three-bottle water-seal suction apparatus or pleurovac system.
• Remove distal clamp.
• Tape all tube connections.
• Suture skin.
• Securely suture and tape chest tube in place.
• Cover area with sterile dressing and seal periphery with waterproof tape.
• Obtain postoperative chest radiograph to determine proper tube placement

Fuhrman catheter
• Insert introducer needle into desired interspace and aspirate air to confirm proper placement.
• Thread introducer wire through the needle into chest, and then remove the needle.
• Thread dilator over the wire and advance into chest to create a path for the catheter.
• Remove dilator and thread catheter over the wire into the chest.
• Remove the wire and tape or suture the catheter in place.
• Attach catheter to the suction unit.

Complications
• Incorrect placement
• Chest wall bleeding
• Lung laceration
• Puncture of liver or spleen
• Air leak
• Subcutaneous emphysema
• Chest tube occlusion
• Persistent pneumothorax

**Urinary Catheterization**

Indications
• To drain urine and quantify output (eg, during anesthesia to monitor volume status and renal perfusion, for a comatose patient, or in cases of shock)
• To manage urinary retention
• To obtain clean sample for detecting bacterial infection
Materials
- Foley catheter
- Catheterization kit (contains catheter of desired size such as 16 to 18 Fr, sterile drapes, antiseptic soap, lubricant, syringe to inflate cuff on catheter; may contain sterile gloves, mask)
- Drainage system (collection bag)

Technique

Male patient
- Put on sterile gloves.
- Isolate field with sterile drapes.
- Inflate 5-mL balloon cuff with sterile water in syringe to check for leaks, then deflate cuff; lubricate catheter with sterile water-soluble jelly.
- Retract foreskin.
- Stretch penis with left hand and prepare with right hand using antiseptic soap.
- Right hand now remains the sterile hand.
- With right hand, insert catheter all the way to sidearm.
- Confirm urine return (have assistant press on bladder).
- Inflate balloon with 5 mL of sterile water using syringe.
- Draw back on catheter to check for fixation at bladder neck.
- Attach catheter to drainage system (collection bag and tubing).
- Tape tubing to thigh.

Female patient
- Put on sterile gloves.
- Isolate field with sterile drapes.
- Inflate 5-mL balloon cuff with sterile water in syringe to check for leaks, then deflate cuff; lubricate catheter with sterile water-soluble jelly.
- Elevate the patient’s legs by bending at the knees.
- With left hand retract labia laterally to reveal the urethral orifice.
- With right hand prepare area from meatus toward anus using antiseptic soap.
- Right hand now remains the sterile hand.
- With right hand, insert catheter with syringe attached.
- Advance catheter 4 to 6 inches.
• Confirm urine return.
• Inflate balloon with 5 mL of sterile water using syringe.
• Draw back on catheter to check for fixation at bladder neck.
• Attach catheter to drainage system (collection bag and tubing).
• Tape tubing to thigh.

Contraindications

Male patient
• Blood at the meatus (pelvic fracture, suspected urethral transsection)
• Scrotal hematoma
• Acute infection
• Stricture or prostatic hypertrophy

Female patient
• Blood at the meatus (pelvic fracture, suspected urethral transsection)
• Acute infection

Complications
• Injury to urethra or bladder; rarely, bladder can be punctured
• Infection from bacterial contamination of urinary tract
• Vaginal insertion in females

Lumbar Puncture

Indications
• To obtain cerebrospinal fluid for diagnostic studies
• To administer intrathecal medications
• Therapeutic reduction of cerebrospinal fluid pressure

Contraindications
• Increased intracranial pressure (eg, papilledema), bleeding diathesis or infection near puncture site

Materials
• Lumbar puncture kit
• Local anesthetic
• Sterile gloves and gown, mask, protective eyewear

**Technique**

• Place patient in lateral decubitus position with back at edge of bed and legs and neck maximally flexed or in seated position with back flexed and leaning over the bedside tray table.
• Prepare and drape area (L4-5 interspace). In obese patients locate the sacral promontory. The end of this structure marks the L5-S1 space.
• Infiltrate skin with local anesthetic.
• Insert spinal needle through skin and subcutaneous tissue with bevel up.
• Advance needle slightly cephalad, aiming for the subarachnoid space. If bone is contacted, partially withdraw the needle, reposition, and readvance.
• Puncture dura. A slight pop or “give” is felt when this occurs.
• Remove stylet and advance needle very slowly, 1 to 2 mm at a time; pause and check for cerebrospinal fluid return.
• Straighten patient’s legs and neck.
• Attach manometer and measure opening pressure (should be 20 cm or less).
• Collect separate samples for cell count, protein and glucose, culture and sensitivity/Gram stain, special tests.
• Measure closing pressure.
• Apply dressing.
• Prescribe flat bed rest for 12 to 24 hours to minimize cerebrospinal fluid leakage and headache.

**Complications**

• Headache
• Backache
• Radicular pain
• Meningitis
• Transient paresis of sixth cranial nerve

**Tracheotomy**

**Indications**

• Long-term airway management
Materials
- Tracheotomy kit
- Sterile gloves and gown, mask, protective eyewear

Technique
- Place patient in supine position.
- Place rolled towel under shoulders to hyperextend neck.
- Check tracheotomy tube.
  - Inflate pilot cuff with 5 to 10 mL of air to check for leaks.
  - Deflate cuff.
  - Insert obturator.
- Identify landmarks: superior border of thyroid cartilage above and sternal notch below.
- Make transverse skin incision about halfway between the two landmarks (use vertical skin incision in emergency).
- Make vertical incision through fascia in midline of neck after retracting strap muscles laterally.
- Expose the pretracheal fascia (covering trachea and thyroid isthmus).
- Retract thyroid isthmus superiorly.
- Stabilize trachea with tracheal hook.
- Make an inverted U-shaped, inferiorly pedicled flap by incising through second and third tracheal rings.
- Retract flap anteriorly and inferiorly.
- Place two individual 2-0 silk sutures laterally in the flap to help identify the opening if accidental extubation should occur. These can be fastened to the skin.
- Insert dilator.
- Insert tracheotomy tube with obturator.
- Remove obturator.
- Inflate cuff.
- Attach tube to resuscitator bag and oxygen source.
- Ventilate manually.
- Suture outer cannula to skin.
- Tie umbilical tape holding tube around patient’s neck.
- Dress wound lightly with sterile tracheotomy dressing.
- Perform frequent endotracheal suctioning.
Complications
- Bleeding
- Tracheoesophageal fistula
- Tracheoinnominate artery fistula
- Tracheal stenosis

Control of Epistaxis
Indication
- Persistent nasal bleeding

Materials
- Sterile gown, gloves, mask, protective eyewear
- Headlight
- Suction apparatus
- Epistaxis tray
- Topical anesthetic and vasoconstrictor
- Silver nitrate sticks, ¼-inch strip gauze coated in petroleum jelly or antibiotic ointment
- Nasal tampon, sponge, or anterior epistaxis balloon
- Posterior and anterior epistaxis balloons or 10- to 14-Fr Foley catheter with 30-mL balloon

Technique
Anterior bleeding
- Remove blood and clots with suction.
- Identify bleeding site (Kiesselbach plexus on anterior septum is frequent site).
- Attempt to control by pressure. Insert cotton ball with 4% cocaine, oxymetazoline, or phenylephrine and firmly compress the alae for 5 minutes.
- Remove the cotton ball. If still bleeding, apply silver nitrate stick to area for 5 to 10 seconds.
- If bleeding persists, insert an anterior nasal pack.
- Place strip gauze, starting with floor of nose and layering to roof of the nose.
• Alternatively, a nasal tampon, sponge, or anterior nasal balloon can be used.
• Remove packing in 2 to 3 days.

Posterior bleeding (double balloon technique)
• Anesthetize posterior pharynx and soft palate with topical anesthetic spray.
• Pass posterior epistaxis balloon through naris into pharynx
• Inflate balloon with 7 to 10 mL of sterile water and pull it forward snugly to fit the posterior choana.
• Place the anterior balloon and inflate with 15 to 30 mL of sterile water.

Posterior bleeding (Foley catheter technique)
• Insert the catheter through the naris into the pharynx.
• Inflate balloon with up to 10 mL of sterile water and withdraw catheter until balloon is seated posteriorly.
• Pack the anterior nasal cavity with a balloon device, a nasal tampon, or layered gauze.

Complications
• Nasal septal necrosis
• Septal hematoma
• Sinusitis
• Infection
• Neurogenic syncope
• Toxic shock syndrome
chapter 10

Management of the Medically Compromised Surgical Patient

Patients needing oral and maxillofacial surgery often have medical conditions that may require treatment prior to or concomitant with such surgery. In some instances the procedure itself also may need to be modified because of the medical problem. This chapter discusses the various conditions that may be commonly encountered and their diagnosis and management.

Cardiovascular Disease
Coronary Artery Disease

The most common risk factors for coronary artery disease (CAD) are diabetes, hypertension, smoking, hypercholesterolemia, and a positive family history. CAD may result in stable angina or one of the acute coronary syndromes. Patients with stable angina often present with precordial pain radiating to the left arm, neck, and jaw on exertion. It is relieved by rest or the use of sublingual nitroglycerine. Acute coronary syndrome (ACS) includes unstable angina, non-ST-elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI). Symptoms are similar to stable angina but occur with less exertion or at rest and do not abate with rest. The history surrounding the onset of chest pain has a diagnostic sensitivity of 90% when the symptoms are classic. An electrocardiogram may show ST segment depression or inverted T waves indicating ischemia. ST segment elevation indicates frank myocardial infarction (MI). The treatment of any patient suspected of having ACS begins with the correct diagnosis. The diagnosis can be confirmed with:

- 12-lead electrocardiogram (ECG) (ST elevation, inverted T waves, Q waves)
• Elevated cardiac enzymes (creatine kinase-MB, troponins)

The initial treatment should include:
• Oxygen via facemask
• Nitrates administered sublingually (or intravenously IV)
• Morphine for pain and to decrease sympathetic output
• Aspirin, 325 mg chewed and swallowed

Additional treatment may include IV heparin and/or platelet glycoprotein IIa/IIIb antagonists to impair clotting and beta blockers to decrease myocardial oxygen demand. Definitive treatment may include the administration of thrombolytic agents, percutaneous angioplasty with stenting, or coronary artery bypass grafting.

All patients with CAD should be stratified according to risk prior to any proposed surgical procedure. Patients with ACSs should not undergo noncardiac surgery. Stratification begins with an assessment of the patient’s functional limitations. The ability to climb a flight of stairs without chest pain or shortness of breath suggests satisfactory cardiac function. Additional tests to consider include:
• Chest radiograph to evaluate for cardiomegaly, pulmonary edema, or pleural effusion
• ECG to evaluate for left ventricular hypertrophy, ST segment changes, inverted T waves, abnormal Q waves, and arrhythmias
• Transthoracic Doppler echocardiography for wall motion abnormalities, ejection fraction, and chamber pressures
• Stress test (eg, Bruce protocol) to assess for functional cardiac ischemia (can be combined with echocardiography and nuclear imaging to increase the sensitivity)
• Perfusion nuclear imaging to assess cardiac perfusion at rest and with function
• Cardiac angiography

The use of perioperative beta blockers has been shown to reduce the likelihood of cardiac events, including MI. Patients with a recent MI may be at risk for reinfarction following the initial infarct. This has led to the suggestion that elective surgery be avoided for a period of 6 months. However, the management of MI has radically changed over the past 20 years.
and, given the number of possible cardiac interventions following an MI, the risk of reinfarction may be much less than was originally thought. This may allow elective surgery to be performed within a much shorter period after an MI, perhaps after only 1 month.

Congestive Heart Failure

Congestive heart failure (CHF) is a result of inadequate cardiac output. Causes include MI, valvular heart disease, hypertension, anemia, pulmonary embolism, cardiomyopathy, thyrotoxicosis, and endocarditis. Heart failure can be left-sided, right-sided, or both. Patients with left-sided failure present with exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea, cardiomegaly, rales, and an S3 gallop. Right-sided heart failure results in elevated jugular venous pressure, peripheral edema (especially in the lower extremities), and atrial fibrillation. The diagnosis is best made with a transthoracic echocardiograph, although the measurement of brain natriuretic peptide (BNP) can be used to help diagnose and monitor CHF progression. Treatment is primarily aimed at reducing the after-load and increasing the cardiac output. Treatment may include:

- Angiotensin-converting enzyme (ACE) inhibitor (eg, captopril, lisinopril, ramipril)
- Diuretic (eg, furosemide or hydrochlorothiazide)
- Beta blocker (metoprolol or carvedilol)
- Digoxin

Valvular Heart Disease

- **Mitral stenosis (MS):** Often due to preexisting rheumatic heart disease. Patients may present with dyspnea, orthopnea, pulmonary edema, atrial fibrillation, and a right ventricular heave. Auscultation of an opening snap and a mid-diastolic low-pitched rumble at the apex are consistent with MS.
- **Aortic stenosis (AS):** May be congenital or secondary to rheumatic fever or dystrophic calcification. It may also be a result of obstructive hypertrophic subaortic stenosis. The classic presentation is syncope, angina, and exertional dyspnea. Patients are intolerant to changes in heart rate and peripheral vascular resistance. Auscultation of a high-pitched midsystolic crescendo-decrescendo murmur at the right upper
sternal border is consistent with AS.

- **Mitral regurgitation (MR):** May be secondary to mitral valve prolapse, rheumatic heart disease, endocarditis, or MI. Patients may present with pulmonary edema, hypotension, and dyspnea on exertion. Auscultation of a holosystolic high-pitched murmur at the apex is consistent with MR.

- **Aortic regurgitation (AR):** May be secondary to rheumatic heart disease, bicuspid aortic valve, endocarditis, or aortic root disease. Patients may present with pulmonary edema, hypotension, and CHF. Auscultation of a diastolic decrescendo murmur at the right upper sternal border, a high-pitched crescendo-decrescendo murmur at the right upper sternal border, and diastolic rumble at the apex are consistent with AR.

Prosthetic Valve Replacement

Prosthetic valves can be alloplastic or biologic. A biologic valve can be a heterograft or a xenograft. Valve function is best evaluated with an assessment of the functional capacity of the patient and a transthoracic echocardiograph. Mechanical valves always require anticoagulation, whereas biologic valves may not require anticoagulation after 3 months, although this depends on other risk factors. Patients should discontinue warfarin use prior to surgery if bleeding is likely to be a problem. Depending on the risk for thromboembolism and cerebrovascular accident, patients may have to be bridged with low-molecular-weight heparin (LMWH) or unfractionated heparin (UFH).

Antibiotic Prophylaxis

(see appendix, page 380)

Arrhythmias

The distinction between supraventricular and ventricular arrhythmias must be made early. The latter requires prompt attention and due concern. Arrhythmias can also be divided into bradyarrhythmias and tachyarrhythmias.

Bradyarrhythmias
- First-degree heart block with prolonged PR interval requires no treatment.
- Second-degree type I heart block with progressive prolonged PR interval and an occasional blocked impulse requires no treatment.
- Second-degree type II heart block with fixed prolonged PR interval and an occasional blocked impulse (4:1, 3:1, 2:1) may progress to third-degree heart block and may require pacing.
- Third-degree heart block with complete atrioventricular (AV) dissociation usually requires pacing.

Tachyarrhythmias

- **Supraventricular tachycardia (SVT):** Develops above the ventricles and presents with a normal narrow QRS complex unless conduction is abnormal. This group of arrhythmias includes atrial tachycardia, multifocal atrial tachycardia, atrial fibrillation, atrial flutter, and AV junctional tachycardia. Treatment of SVT involves cardioversion when the rhythm is unstable. Additionally, vagal maneuvers and carotid massage can be attempted. Adenosine can also be used to treat some SVT. Beta blockers, calcium channel blockers, amiodarone, and digoxin may also be used for rate control.

- **Ventricular dysrhythmias:** More concerning than SVT because of the potential for ventricular tachycardia and ventricular fibrillation. Elective surgery should be deferred in all patients with ventricular dysrhythmias. The initial treatment of ventricular dysrhythmias should follow the advanced cardiac life support (ACLS) protocol (see chapter 11). Long-term therapy may involve the use of antiarrhythmic drugs, radiofrequency ablation, or an implantable cardiac defibrillator.

Hypertension

Hypertension is defined as blood pressure in excess of 140/90 mm Hg measured on two separate occasions. It is one of the most common reasons for postponing elective surgery. Most hypertension is of unknown etiology and is referred to as essential hypertension. Secondary hypertension is a result of known pathology, including renal disease, Cushing syndrome, Conn syndrome, pheochromocytoma, hyperthyroidism, aortic regurgitation, and side effect of medication. Elevated blood pressure can be classified as shown in Table 10-1.
Hypertensive urgency, as defined by a blood pressure greater than 180/110, warrants treatment prior to any elective surgical procedure, and the patient should be seen by a primary care physician or internist within 24 hours.

Hypertensive emergency is defined as any elevated blood pressure associated with end organ damage (eg, encephalopathy, heart failure, pulmonary edema, renal failure). The diagnosis of hypertension should not be made for the first time in the postoperative setting because many perioperative factors, including pain, may elevate blood pressure in an otherwise normotensive patient. All patients with a history of hypertension should be maintained on their usual blood pressure medication during the perioperative period. Several antihypertensive medications reduce the patient’s ability to compensate for anesthesia-induced hypotension. On occasion, therefore, it may be necessary to discontinue a medication.

### Table 10-1 Hypertensive state according to blood pressure readings

<table>
<thead>
<tr>
<th></th>
<th>Systolic (mm Hg)</th>
<th>Diastolic (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt; 120</td>
<td>&lt; 80</td>
</tr>
<tr>
<td>Pre-hypertension</td>
<td>120–139</td>
<td>80–89</td>
</tr>
<tr>
<td>Stage I hypertension</td>
<td>140–159</td>
<td>90–99</td>
</tr>
<tr>
<td>Stage II hypertension</td>
<td>&gt; 160</td>
<td>&gt; 100</td>
</tr>
<tr>
<td>Hypertension urgency</td>
<td>&gt; 180</td>
<td>&gt; 110</td>
</tr>
</tbody>
</table>

The signs and symptoms of hypertension are rare unless a hypertensive emergency develops. In a hypertensive emergency the patient may present with severe headache, shortness of breath, chest pain or acute renal failure. An S4 gallop is often present because of the left ventricular hypertrophy, which may also be evident on the ECG. The treatment of a hypertensive emergency may include:
- Alpha-1, beta-1, and beta-2 receptor antagonists (labetalol, 20 to 40 mg IV every 10 minutes up to a maximum of 300 mg)
- ACE inhibitor (enalapril, 1.25 to 5 mg IV every 6 hours)
- Alpha-1 receptor antagonist (hydralazine, 10 to 20 mg IV every 6 hours; avoid in the elderly and patients with CAD)

The treatment of patients with stage I and stage II hypertension includes:
- Diuretic (eg, hydrochlorothiazide, 12.5 to 25 mg orally every day)
- Selective beta-1 receptor antagonist (eg, metoprolol 25 to 50 mg orally twice a day; especially patients with a history of MI)
- ACE inhibitor (captopril, 6.25 to 25 mg orally four times a day; change to long-acting agent, eg, lisinopril, 20 to 40 mg orally every day on discharge; especially in patients with diabetes and chronic heart failure)
- Angiotensin receptor blocker
- Calcium channel blocker

**Respiratory Disease**
Respiratory disease is a common diagnosis among surgical patients. The net result of most respiratory diseases is hypoxemia, which, in broad terms, may develop from one of four mechanisms.

- **Hypoventilation** (decreased respiratory rate or vital capacity)
  - Chest trauma
  - Pneumonia
  - Narcotics
  - Neurologic disease
  - Obstructive sleep apnea
  - Asthma
  - Chronic obstructive pulmonary disease (COPD)
  - Idiopathic pulmonary fibrosis

- **Diffusion impairment**
  - Idiopathic pulmonary fibrosis
  - Acute respiratory distress syndrome

- **Ventilation perfusion mismatch**
—Pulmonary embolism

- **Shunting**
  —Pulmonary edema
  —Pneumonia
  —Atelectasis
  —Cardiac septal defects
  —Chronic liver disease

The management of these patients initially requires a generalized approach while the cause for the hypoxemia is sought. Treatment involves the administration of oxygen using one of several methods.

- Nasal cannula (1 L/min, increases fraction of inspired oxygen \([\text{FIO}_2]\) to 40% at 6 L/min)
- Rebreathing facemask (increases \([\text{FIO}_2]\) to 60% at 10 L/min)
- Nonrebreathing facemask (increases \([\text{FIO}_2]\) to 90% at 10 L/min)
- CPAP/BiPAP with positive pressure (increases \([\text{FIO}_2]\) to 80%)
- Intubation/tracheotomy (increases \([\text{FIO}_2]\) to 100%)

Several diseases should be considered in any patient with signs or symptoms of disordered respiration. The diagnosis and, ultimately, the treatment depend on thorough history taking and physical examination as well as appropriate studies.

**Asthma**

Asthma is classically defined as bronchial hyper-responsiveness and reversible bronchoconstriction due to smooth muscle contraction leading to diminished ventilation and hypoxemia. Signs and symptoms of asthma include shortness of breath, nocturnal awakenings, limitation in activity, wheezing that is predominantly expiratory, cough, chest tightness, chest pain, and status asthmaticus (an acute attack that does not respond to the usual medications).

The diagnosis of asthma can be made with the following:

- **Pulmonary function tests (spirometry)**
Decrease in forced expiratory volume at 1 second (FEV\textsubscript{1})
(proportional to severity)
—No change in vital capacity
—Decrease in ratio of FEV\textsubscript{1} to forced vital capacity (FVC)
(proportional to severity)
—Increase in FEV\textsubscript{1} by 10% with bronchodilator treatment

- **Peak expiratory flow rate (PEFR)**
  —Decrease in PEFR to less than 80% of the normal value for the individual (based on height and age) suggests exacerbation

The goal of treating the asthmatic patient is to maintain all preoperative medications. The choice of medications is usually determined by classifying asthma as intermittent, mild persistent, moderate persistent, or severe persistent. The choice of medication is individualized, but often progresses through the following algorithm:

1. Short-acting beta agonist (SABA) (eg, albuterol metered dose inhaler [MDI] as needed)
2. Corticosteroid (eg, fluticasone MDI twice a day)
3. Long-acting beta agonist (LABA) (eg, salmeterol MDI twice a day)
4. Leukotriene receptor antagonist (eg, montelukast orally daily)

Perioperative exacerbations of asthma may be serious. The treatment of exacerbations includes:

1. SABA such as albuterol metered dose inhaler (MDI) or nebulizer (2.5 mg/5 mL normal saline [NS]) every 4 to 6 hours as needed
2. Anticholinergic medication (eg, ipratropium MDI or nebulizer (0.5 mg/5 mL NS) every 6 hours as needed
3. Corticosteroid burst (prednisolone 1 mg/kg/d orally for 5 days)

Chronic Obstructive Pulmonary Disease

Emphysema and chronic bronchitis are the two major respiratory diseases included under the heading of COPD. Emphysema is characterized by dilated and collapsed airways with alveolar destruction secondary to alpha\textsubscript{1}-antitrypsin deficiency as a result of smoking or a congenital deficiency. Chronic bronchitis is characterized by increased airway secretions and
mucous production. The net result of both diseases is retention of CO$_2$ and eventually hypoxemia. The signs and symptoms of COPD are hyperventilation, barrel chest, pursed lips, and decreased breath sounds. A patient with emphysema is often described as a “pink puffer,” whereas a patient with chronic bronchitis is often described as a “blue bloater.”

The diagnosis of COPD can be made with:

- **Pulmonary function tests (spirometry)**
  - Decrease in FEV$_1$ (proportional to severity)
  - No change in vital capacity
  - Decrease in ratio of FEV$_1$ to FVC to less than 70% of predicted value

- **Arterial blood gas**
  - Increase in PCO$_2$ and possible decrease in PO$_2$, respiratory alkalosis

- **Chest radiograph**
  - Loss of lung markings, hyperinflation, and flattened diaphragm

The treatment of a patient with COPD aims to maintain the preoperative medication regimen, which is typically determined by classifying COPD as mild, moderate, severe, or very severe. Despite maintaining normal preoperative medications, patients with COPD may sometimes experience perioperative exacerbations that require further treatment. This usually includes:

1. Ambulatory oxygen (including home O$_2$) at 2 to 6 L/min with nasal cannula or 6 to 10 L/min with facemask
2. SABA such as albuterol MDI or nebulizer (2.5 mg/5 ML NS) every 4 to 6 hours as needed
3. Anticholinergic medication (eg, ipratropium MDI or nebulizer (0.5 mg/5 mL NS) every 6 hours as needed
4. Corticosteroid burst (prednisolone 1 mg/kg/d orally for 5 days)
5. Possible intubation for patients who are doing poorly (although extubation in this group is particularly challenging)
Pneumonia

Nosocomial pneumonia may occur in the postoperative period. It can be associated with the use of mechanical ventilation, at which time it is referred to as ventilator-associated pneumonia. The development of pneumonia may be influenced by the use of perioperative antibiotics and the placement of nasogastric feeding tubes. The most common causative organisms associated with nosocomial pneumonia are Streptococcus pneumoniae, Staphylococcus aureus, Pseudomonas aeruginosa, Acinetobacter species, and occasionally anaerobic organisms. The net result is hypoxemia and sepsis. Signs and symptoms include shortness of breath, fever, chest pain, decreased breath sounds, and fremitus. The diagnosis is best made with:

- **Chest radiograph or computed tomography (CT) scan**
  - Infiltration or consolidation of lung parenchyma
  - Pleural effusion

- **Bronchoalveolar lavage**
  - Cultured organisms

- **Arterial blood gas**
  - Decrease in PO\(_2\)

- **Complete and differential blood cell count**
  - Increase in white blood cell count

- **Positive blood culture**

  The treatment of pneumonia includes the use of parenteral antibiotics, which may involve either monotherapy or combination therapy, depending on the organisms identified.

Pulmonary Embolism

More than 95% of pulmonary emboli (PE) are from the deep veins of the legs. These travel to the lungs, leading to respiratory and cardiovascular compromise. Most emboli are clinically silent due to their small size. Risk factors include smoking; contraceptive use; pregnancy; advanced age;
tumors; abdominal and orthopedic surgery; and hereditary coagulation disorders such as antithrombin III deficiency, factor V Leiden, and protein C and S deficiency. Prevention with sequential calf compressors, thromboembolic deterrent stockings, perioperative unfractionated heparin (UFH), or low molecular weight heparin (LMWH) are important. Signs and symptoms include chest pain, shortness of breath, sinus tachycardia, hemoptysis, leg pain and swelling, and pain on dorsiflexion of the foot (Homans sign). The workup for possible PE includes:

- **CT scan of the chest with PE protocol**
- **Duplex ultrasound of the leg veins**
- **Arterial blood gas measurement**
  - Alveolar-arterial (A-a) gradient
- **Chest radiograph**
  - Hampton hump (shallow, wedge-shaped consolidation in the lung periphery with the base against the pleural surface)
- **ECG**
  - S wave in lead I
  - Q wave and inverted T wave in lead III
- **D-dimer assay**
  - Normal D-dimer assay virtually eliminates PE and deep venous thrombosis (DVT)
- **Pulmonary angiography**

The treatment of suspected PE may include one of the following:

1. Heparin IV with 50 mg/kg bolus or 12 mg/kg/h drip
2. Enoxaparin (1 to 1.5 mg/kg subcutaneously [SC] twice a day)
3. Fondaparinux (5 to 10 mg SC every day)

Long-term anticoagulation should also be simultaneously initiated with warfarin, with a goal international normalized ratio (INR) of 2.5 to 3.0. Massive PE may lead to right ventricular strain and chemical thrombolysis, or surgical thrombectomy may then be required. Patients unable to be anticoagulated and patients with recurrent PE despite anticoagulation may
Atelectasis
Atelectasis is a common postoperative complication characterized by a segmental collapse of the lung alveoli. It leads to a progressive decline in lung compliance, impaired segmental ventilation, retained secretions, and a decrease in functional residual capacity. The signs and symptoms of atelectasis include decreased breath sounds, inspiratory crackles at the bases, labored breathing, and low-grade fever. The diagnosis is based on the clinical picture and temporal relationship of the symptoms to the immediate postoperative period and supported, when indicated, by the chest radiograph. Treatment includes incentive spirometry and early ambulation.

Pulmonary Edema
Pulmonary edema can develop from cardiac and noncardiac causes. When present with normal cardiac function, it may be the result of fluid overload, or it can follow extubation as a result of upper airway obstruction leading to negative pressure pulmonary edema (NPPE). The latter is more common in young males. The net result is significant alveolar transudation and hypoxemia. Risk factors include obesity, obstructive sleep apnea, and maxillofacial surgical procedures. Signs and symptoms include increased labored breathing, shortness of breath, decreased breath sounds, and bilateral crackles. The diagnosis is based on the clinical suspicion, the development of NPPE in the immediate postoperative period, and the chest radiograph findings.

Treatment usually requires supplemental oxygen, support of the airway (for NPPE) and possible reintubation and a short period of mechanical ventilation. Fluid restriction and/or diuretics are typically not required in NPPE but may be used in pulmonary edema.

Renal Disease
Hypertension, diabetes, polycystic kidney disease, pyelonephritis, and autoimmune kidney disease can all result in diminished renal function. The normal glomerular filtration rate (GFR) is approximately 120 mL/min. Generally the GFR needs to decrease to about 30 mL/min before a rise in
serum creatinine or urea is seen. This represents a 75% loss in renal function. As renal disease progresses, the normal urine production declines from 0.5 to 1 mL/kg/h until the patient eventually becomes oliguric or anuric. As renal function declines, the patient may become anemic and hypoalbuminemic, and the regulation of serum electrolytes (eg, Na\(^+\), K\(^+\), Ca\(^{2+}\), Mg\(^{2+}\)) is lost. Furthermore, the ability to regulate acid-base homeostasis is impaired as the kidney can no longer effectively excrete H\(^+\) or conserve bicarbonate. The effective removal of metabolic waste products and urea is also impaired. The basic assessment of the patient with renal disease should include:

- Serum creatinine and urea determination
- Serum electrolytes evaluation
- Bicarbonate measurement
- Hemoglobin and hematocrit determination

Serum creatinine and urea levels provide indirect evidence of renal function by correlating roughly with GFR. Calculating the GFR is the only accurate method to reliably quantitate renal function. This is typically done by calculating the creatinine clearance. Urinalysis with microscopy is only useful in the initial diagnosis of renal disease, when the presence of protein, red blood cells (RBCs), white blood cells, casts, and crystals can be identified. Urinalysis is infrequently used to monitor disease progression.

Perioperative renal impairment can occur in a patient with preexisting renal disease or in otherwise healthy patients. Abnormalities in renal function should be classified according to whether the cause is prerenal, renal, or postrenal:

- **Prerenal causes**
  - Hypovolemia
  - Shock
  - Hypotension
  - Heart failure

- **Renal causes**
  - Glomerulonephritis (GN): May be secondary to autoimmune disease, diabetes, HIV, or amyloidosis; readily identified by RBC casts in the urine
  - Acute tubular necrosis (ATN): May be secondary to renal ischemia,
hypotension, rhabdomyolysis, drugs, and intravenous contrast media; readily identified by muddy brown casts
—Interstitial nephritis (IN): Most often drug-related; readily identified by eosinophiluria
—Pyelonephritis

**Postrenal causes:** Usually obstructive in nature secondary to prostate hypertrophy/malignancy and renal stones

The assessment of a patient with new-onset renal failure should include measurement of urea, creatinine, and serum electrolytes; urinalysis with microscopy; and the fractional excretion of sodium (FENa). The latter helps to distinguish prerenal, renal, and postrenal causes. Urine osmolality, urine sodium, and the ratio of blood urea nitrogen (BUN) to creatinine can also be used to help distinguish among the three broad causes of renal failure. The FENa is calculated as follows:

\[
FENa = \frac{\text{Urine Na/Plasma Cr}}{\text{Urine Cr/Plasma Na}}
\]

A FENa less than 1% indicates prerenal causes, a FENa above 2% is consistent with renal causes, and a FENa above 4% suggests postrenal causes.

Perioperative management of the renal patient should focus on the following:

- Volume status, because patients may become quickly overloaded by fluid. IV fluid replacement should be used conservatively and without the addition of potassium.
- Clinically significant anemia should be managed acutely with packed red blood cells, although the potential for significant fluid shifts exists. Chronic management usually mandates erythropoietin SC or IV. Long-term iron supplementation is also warranted.
- Hyperkalemia can develop quickly and can result in cardiac arrhythmias. Hyperkalemia above 5.5 mEq/L mandates an ECG. A wide QRS complex, loss of P waves, and peaked T waves require urgent treatment with calcium gluconate to stabilize the cardiac membrane,
kayexalate to bind gastrointestinal potassium, and dextrose/insulin to drive the intracellular movement of potassium. Dialysis may be needed when the hyperkalemia is excessively high or ventricular cardiac arrhythmias develop.

- Many medications require discontinuation if they are nephrotoxic (eg, nonsteroidal anti-inflammatory drugs, some antibiotics) whereas others need to be “renally dosed” if they are renally excreted. This typically means a reduced frequency of drug administration.

**Liver Disease**

Liver disease can have a significant impact during the perioperative period. Careful history taking will often reveal patients who are at risk for liver disease, including those who have a history of alcoholism, substance abuse, prior blood transfusions, hepatitis, tattoos, and sexual promiscuity. Signs and symptoms may be conspicuous by their absence, although fatigue, pruritus, jaundice, palmar erythema, spider telangiectasia, splenomegaly, gynecomastia, testicular atrophy, and increased abdominal girth are all suggestive. Screening for liver disease in an otherwise healthy patient is of little benefit. The end stage of liver disease is cirrhosis, which results in portal hypertension with esophageal varices and hemorrhoids with the inherent potential for severe gastroesophageal bleeding. Perioperative morbidity and mortality are significantly greater in this patient population.

The liver is an important organ for the metabolism of many drugs, and liver disease can adversely affect drug metabolism. Liver disease also influences mechanical ventilation because of the respiratory compromise it produces through hepatopulmonary syndrome, pleural effusions, and pulmonary hypertension. Liver disease also predisposes patients to excessive bleeding via the impaired synthesis of the vitamin K–dependent factors II, VII, IX, and X. Surgery is contraindicated in patients with the following:

- Acute or fulminant hepatitis
- Alcoholic hepatitis
- Severe chronic hepatitis

Patients with cirrhosis have typically been risk-stratified according to the Child classification, but the model for end-stage liver disease (MELD)
system may be superior. According to the MELD system, patients with a score less than 10 can undergo elective surgery, those with a score of 10 to 15 may undergo elective surgery with caution, and those with a score greater than 15 should not undergo elective surgery.

Liver function is easily assessed with simple blood tests:
- Liver enzymes (alanine transaminase [ALT], aspartate transaminase [AST], γ-glutamyltransferase [GGT])
- Bilirubin
- Prothrombin time (PT), INR, partial thromboplastin time (PTT)
- Serologic markers for hepatitis (A, B, or C) in the setting of acute hepatitis or when establishing exposure or carrier status; otherwise of limited value

The perioperative goals for the patient with liver disease are:
- Correct elevated PT with vitamin K, fresh frozen plasma (FFP), or activated factor VII
- Correct impaired platelet function with desmopressin acetate (DDAVP) to increase von Willebrand factor (vWF)
- Manage ascites with diuretics or peritoneal tap
- Limit IV fluid administration and total sodium
- Correct electrolyte abnormalities such as hypokalemia
- Treat elevated plasma ammonia levels with dietary modification and lactulose
- Reduce the risk of infection with antibiotic prophylaxis
- Monitor blood volume, cardiac output, and urine production
- Consider H₂ receptor antagonists to minimize potential gastric bleeding

**Blood Disorders**

**Anemia**

Anemia is an absolute or relative reduction in the hemoglobin concentration or hematocrit. It may be due to a decrease in RBC production or an increase in RBC destruction. The net result is hypoxemia due to a lack of oxygen-carrying capacity. Decreased RBC production can be divided into microcytic, normocytic, and macrocytic anemia.
Decreased RBC production (decreased reticulocyte count < 2%)
- Microcytic anemia (mean corpuscular volume [MCV] < 80 fL)
  - Iron deficiency (decreased Fe$^{2+}$, decreased ferritin, increased total iron binding capacity [TIBC])
  - Thalassemia (gel electrophoresis)
  - Sideroblastic anemia (increased Fe$^{2+}$, increased ferritin, bone marrow biopsy)
  - Sickle cell trait/disease (blood smear)

- Normocytic anemia (MCV 80 to 100 fL)
  - Renal failure
  - Anemia of chronic disease (decreased Fe$^{2+}$, decreased TIBC)
  - Aplastic anemia

- Macrocytic anemia (MCV > 100 fL)
  - Vitamin B$_{12}$ deficiency (decreased B$_{12}$ level, increased serum methylmalonic acid, increased serum homocysteine)
  - Folic acid deficiency (decreased folic acid, normal methylmalonic acid, increased serum homocysteine)

Increased RBC destruction (increased reticulocyte count > 2%)
- Hereditary spherocytosis (spherocytes on blood smear)
- Autoimmune hemolysis (direct Coombs test)
- Cold agglutinin disease (indirect Coombs test)
- Mechanical destruction (schistocytes on blood smear)
  - Disseminated intravascular coagulation (DIC)
  - Prosthetic heart valve
  - Thrombotic thrombocytopenic purpura (TTP)
  - Hemolytic uremic syndrome (HUS)

The general workup of the anemic patient should include a complete blood cell (CBC) count, peripheral smear, MCV, and reticulocyte count. Additional tests should be requested based on the initial results and may include Fe$^{2+}$, ferritin, TIBC, B$_{12}$, folate, Coombs test (direct and indirect), methylmalonic acid, homocysteine, and bone marrow biopsy.
Iron deficiency anemia

Iron deficiency anemia, when identified, mandates an active search for blood loss. This may be due to heavy menses in female patients, but the possibility of gastroesophageal sources of blood loss must be considered. In male patients, this mandates screening for occult blood loss in the stool or by colonoscopy/esophagoscopy. Iron deficiency anemia should be treated with iron supplements only after a reason for blood loss has been identified.

Megaloblastic anemias

Megaloblastic anemias should be treated with vitamin B$_{12}$ or folate, depending on the reason for the deficiency. Dietary change should also be initiated in all cases except for pernicious anemia, in which a lack of intrinsic factor renders dietary B$_{12}$ ineffective. Megaloblastic anemia can be seen in vegetarians and those who avoid leafy green vegetables. Dietary inadequacy may also be seen in patients with chronic alcoholism.

Sickle cell anemia

Sickle cell anemia is seen frequently in African-American patients. Sickle cell trait is heterozygous and of minor clinical significance. Sickle cell disease is homozygous and clinically important. Deoxygenated RBCs polymerize and sickle. This leads to vaso-occlusive crises causing pain; cardiomyopathy; and infarcts of bone, lungs, and kidneys. Most patients undergo autosplenectomy due to splenic infarction. Intravascular hemolysis also leads to gallstones in young patients. The treatment of patients with sickle cell anemia requires:

- Hydration (IV or oral)
- Oxygen (supplemental)
- Hydroxyurea (decreases incidence and severity of pain crises)
- Narcotic pain medication during crises
- Aggressive treatment of infection
- Vaccination for the autosplenectomy

Myeloproliferative Disease

Myeloproliferative disease encompasses a group of disorders characterized by a clonal proliferation of myeloid cells. All of these entities have the potential to transform into acute leukemias, although this process occurs
slowly. Myeloproliferative diseases can be classified according to the cell line responsible. Although bone marrow biopsy is the most sensitive diagnostic test, a CBC count and smear is often suggestive.

Polycythemia vera (increased RBC count)
- Hyperviscosity syndrome
- Headache, cerebrovascular accident (CVA), angina, pruritus, amaurosis, claudication, splenomegaly
- Treat with phlebotomy, aspirin, and hydroxyurea

Essential thrombocytosis (increased platelets)
- Symptoms as per polycythemia vera
- Treat with aspirin, platelet exchange, hydroxyurea

Myelofibrosis (increased fibrous tissue)
- Fatigue, weight loss, extramedullary hemopoesis, massive splenomegaly
- Treat with human stem cell transplant

Chronic myelogenous leukemia (see leukemia section)

Leukemia
Leukemia includes a group of malignant diseases of lymphocytes or myeloid cells that are classified according to cell type.

Acute lymphoblastic leukemia
- Affects children
- Fever, anemia, thrombocytopenia, leukocytosis
- Treat with chemotherapy and radiotherapy for central nervous system (CNS) involvement

Acute myelogenous leukemia
- Affects adults
- Fever, thrombocytopenia, leukocytosis
- Treat with chemotherapy and all-trans retinoic acid (ATRA)

Chronic myelogenous leukemia
- A myeloproliferative disease
- Philadelphia chromosome, long course with chronic, accelerated, and blast phases
- Treat with tyrosine kinase inhibitors and bone marrow transplant

Chronic lymphocytic leukemia
- Fever, anemia, thrombocytopenia, leukocytosis
- Treat with chemotherapy

All leukemias tend to present with similar perioperative concerns. Anemia, thrombocytopenia, and ineffective leukocytosis present multiple surgical challenges. Infections are more common and should be treated aggressively. The medical management of leukemia causes further immunocompromise, increasing patient morbidity.

Lymphoma and Multiple Myeloma

Lymphoma is a malignancy of lymphoid tissue that resides predominantly within lymphoid tissue. It can be classified as Hodgkin or non-Hodgkin lymphoma depending on the presence of Reed-Sternberg cells in the former. Waldenström macroglobulinemia is a low-grade form of non-Hodgkin lymphoma in which the cells secrete immunoglobulin leading to a hyperviscosity syndrome, peripheral neuropathy, and Raynaud phenomenon.

Multiple myeloma is a malignant proliferation of plasma cells that produce a monoclonal antibody (M component). The disease progresses slowly but can lead to:
- Anemia
- Bone pain and hypercalcemia
- Renal disease and nephrotic syndrome
- Hyperviscosity syndrome

The treatment of lymphoma and multiple myeloma includes chemotherapy and occasionally radiotherapy. The additional use of bisphosphonates in multiple myeloma to reduce lytic lesions and bone pain has greatly improved patients’ quality of life but has resulted in the development of bisphosphonate-related osteonecrosis of the jaw (BRONJ) in a number of patients (see chapter 18).
Thrombocytopenia

Thrombocytopenia results from decreased platelet production, increased platelet destruction, or splenic sequestration. The net result is bleeding episodes that occur spontaneously or with minimal trauma when the platelet count falls below 50,000 cells/mm$^3$. The causes of thrombocytopenia include:

- **Idiopathic thrombocytopenic purpura (ITP)**
  — Autoimmune mechanism that can follow an upper respiratory infection
  — Treat with steroids or splenectomy

- **Thrombotic thrombocytopenic purpura (TTP)**
  — Hemolytic anemia, renal failure, fever, thrombocytopenia, and neurologic disease, including seizure
  — Treat with plasmapheresis or intravenous immunoglobulin (IVIG), steroids

- **Hemolytic uremic syndrome (HUS)**
  — Hemolytic anemia, renal failure, and thrombocytopenia
  — Treat with plasmapheresis and steroids

- **Disseminated intravascular coagulation (DIC)**
  — Secondary to trauma, sepsis, malignancy
  — Consumptive coagulopathy resulting in anemia and thrombocytopenia

Surgical patients are at risk for significant perioperative bleeding. The condition of all patients should be optimized prior to any surgical procedure. This entails managing the underlying condition and increasing the platelet count. Patients with DIC should not be considered surgical candidates.

Coagulopathy

Abnormalities of the platelets, the intrinsic clotting pathway, and the extrinsic clotting pathway are all causes of coagulopathy. The propensity for bleeding is best assessed with a CBC (platelet) count, platelet aggregation tests, PT,
INR, and PTT. Abnormalities of the platelets include inherited and acquired defects.

1. Inherited defects
   - Bernard-Soulier syndrome and Glanzmann thrombasthenia
   - von Willebrand disease
     — Common
     — Multiple types (autosomal dominant)
     — Decreased vWF and factor VIII
     — Treat with DDAVP, factor VIII concentrate, recombinant vWF, cryoprecipitate

2. Acquired defects
   — Aspirin
   — Clopidogrel or ticlopidine
   — Factor IIb/IIIa inhibitors
   — Uremia

The aim with surgical patients is to achieve a platelet count above 100,000 cells/mm$^3$. Additionally, platelet function may be abnormal and should be assessed and corrected if possible. The use of adenosine diphosphate (ADP) antagonists, clopidogrel, and ticlopidine is not usually problematic for minor surgery, but the potential for bleeding exists, which needs to be considered prior to any major surgery. Platelet transfusion may be required for patients with thrombocytopenia and thrombasthenia. It should be remembered that the development of autoantibodies to platelets may result in an unpredictable response to platelet transfusion.

Hemophilia A

Hemophilia A is an X-linked recessive inherited disorder characterized by a deficiency in factor VIII. Males are affected, although females may be carriers and can have hemophilia if they are homozygous. Factor VIII is the key coagulation factor in the extrinsic cascade, and any deficiency can be measured with the PTT. The factor VIII level can also be measured to quantitate disease severity. Hemophilia A can be classified based on the factor VIII level.

- Mild hemophilia (factor VIII level 5% to 25%)
- Moderate hemophilia (factor VIII level 1% to 5%)
- Severe hemophilia (factor VIII level < 1%)

The treatment of hemophilia A depends on the factor VIII level and the nature of the proposed surgical procedure. Minor surgical procedures may only require modest elevations in factor VIII level to 50% of the normal value. Major surgical procedures usually dictate that the level be restored to near 100%. The most common treatment involves replacement with recombinant factor VIII. The vasopressin analog DDAVP (0.3 mg/kg/d) can be used in mild cases and results in a small increase in factor VIII level through endothelial cell release. Cryoprecipitate can also be used but carries the risk of disease transmission because it is obtained from pooled human donors. If antibodies have developed to factor VIII (ie, factor VIII concentrate from pooled human donors) then activated factor VII may be required. With a half-life of 12 hours, factor VIII replacement needs to be given twice a day. Despite the correction of the PTT with DDAVP or factor VIII replacement, local measures at the time of surgery are also of paramount importance in helping to reduce the risk of postoperative bleeding. Antifibrinolytics should also be considered in this patient population. Tranexamic acid and $\varepsilon$-aminocaproic acid (EACA) can be administered systemically or applied topically as a mouthwash/gargle and have been shown to reduce the number of postoperative bleeding episodes.

Hemophilia B

Hemophilia B (Christmas disease) is less common than hemophilia A and is autosomal recessive in the mode of inheritance. It is due to a deficiency in factor IX. The half-life of factor IX is 18 hours; accordingly, replacement is required every 18 hours. Distinguishing between hemophilia A and B can be difficult initially because they both present with an elevated PTT. A factor IX assay is required to make the diagnosis of hemophilia B. Treatment is with factor IX replacement.

Warfarin therapy

Many patients who present for treatment are anticoagulated with warfarin. They may have a history of atrial fibrillation, a prosthetic heart valve, peripheral vascular disease, deep venous thrombosis, or pulmonary embolism. The adequacy of treatment is measured by evaluating the PT or the INR. The actual therapeutic level required is determined primarily by the
medical condition necessitating the anticoagulation. Warfarin is a potent inhibitor of the vitamin K–dependent proteins II, VII, IX, X, C, and S. The latter two proteins (C and S) are involved in the fibrinolytic pathway, and as such a deficiency of these proteins tends to promote coagulation. The type of surgery planned, the INR, and the underlying medical need for warfarin will dictate whether to discontinue it prior to surgery.

**Minor surgery options (eg, simple extractions)**
- Maintain warfarin if the INR is less than 3.0 (controversial).
- Reduce warfarin dose to obtain an INR less than 3.0 if initially higher (controversial).
- Local hemostatic measures during surgery.
- Tranexamic acid or EACA systemically or topically.

**Minor surgery options (eg, impacted teeth)**
- Maintain warfarin if the INR is 2.0 or less.
- Reduce warfarin dose to obtain an INR 2.0 or less if initially higher.
- Stop warfarin 4 days prior to the procedure and resume after surgery (if medically appropriate to discontinue).
- Stop warfarin 4 days prior to the procedure. Bridge with enoxaparin 1 mg/kg SC twice a day, and resume warfarin after surgery. Do not give enoxaparin the night before or morning of the procedure; resume the evening of the procedure.
- Local hemostatic measures during surgery.
- Tranexamic acid or EACA systemically or topically.

**Major surgery options**
- Stop warfarin 4 days prior to the procedure and resume after surgery (if medically appropriate to discontinue).
- Stop warfarin 4 days prior to the procedure. Bridge with enoxaparin 1 mg/kg SC twice a day, and resume warfarin after surgery. Do not give enoxaparin the night before or morning of the procedure; resume the evening of the procedure.
• Stop warfarin 4 days prior to the procedure. Bridge with unfractionated heparin infusion, and resume warfarin dose after surgery. Do not give heparin 6 hours prior to the procedure; resume 6 hours after surgery.
• Local hemostatic measures during surgery.
• Tranexamic acid or EACA systemically or topically.

Active bleeding/emergent need for surgery

Warfarin can be reversed with the administration of vitamin K. It has a short half-life and may need to be redosed every 6 hours. Acute bleeding necessitates immediate replacement of clotting factors with FFP.
• No bleeding: vitamin K 1 to 5 mg orally (also may be given SC or IV)
• Bleeding: vitamin K 2.5 to 10 mg IV; FFP (2 to 4 units)

Hypercoagulation diseases

There are multiple inherited and acquired conditions that predispose patients to thrombosis. The acquired states include antithrombin III deficiency, protein C and S deficiency, factor V Leiden deficiency, and factor II mutation. Causes of acquired states include prolonged immobilization, pregnancy, oral contraceptives, malignancy, smoking, nephritic syndrome, and systemic lupus erythematosus. Acquired causes should be eliminated whenever possible. Patients may be receiving warfarin, and this may influence the perioperative management. Additional perioperative steps that can reduce the risk for deep venous thrombosis and pulmonary embolism should be instituted:
• Encourage ambulation as soon as possible
• Use sequential calf compressors
• Use thromboembolic deterrent stockings (ie, TED hose)
• Use unfractionated heparin at 5,000 units SC every 8 hours or enoxaparin 30 mg SC every 12 hours

Deep venous thrombosis should be suspected if leg pain, leg swelling, lower extremity pitting edema, chest pain, shortness of breath, unexplained
sinus tachycardia, or an alveolar-arterial \( \text{O}_2 \) gradient is noted. Appropriate workup should include a duplex ultrasound of the lower extremity, D-dimer assay, and spiral CT of the chest if pulmonary embolus is suspected. Appropriate management requires heparin anticoagulation and long-term warfarin treatment (> 6 months).

**Endocrine Diseases**

**Diabetes Mellitus**

Diabetes affects a significant proportion of the population and is characterized by autoimmune destruction of the pancreatic islet cells or the development of insulin resistance. Elevated blood glucose levels and the by-products of glucose metabolism lead to cardiovascular disease, cerebrovascular disease, nephropathy, neuropathy, and retinopathy. Elevated or decreased blood glucose levels may also lead to altered mental status and metabolic encephalopathy. Signs and symptoms of diabetes include polyphagia, polydipsia, polyuria, acanthosis nigricans, peripheral skin pigmentation/ulcers, peripheral neuropathy, decreased visual acuity, nonketotic hyperosmolar coma, diabetic ketoacidosis, altered mental status, and hyponatremia.

The diagnosis of diabetes can be established using the following algorithm:

1. Random blood glucose above 200 mg/dL is suggestive
2. Fasting blood glucose (8 hours) between 100 and 125 mg/dL indicates prediabetes
3. Fasting blood glucose above 126 mg/dL is diagnostic
4. When random or fasting blood glucose levels are inconclusive, the oral glucose tolerance test (8-hour fast then 75 mg oral glucose followed by 2-hour postprandial glucose measurement) can be used.
   - Blood glucose 140 to 199 mg/dL indicates prediabetes
   - Blood glucose > 200 mg/dL is diagnostic

A significant number of patients with diabetes will require surgical procedures. For diabetic patients undergoing procedures under local anesthesia, no change in their medication regimen is warranted provided that they will resume a normal diet postoperatively. Diabetic patients receiving a general anesthetic or intravenous sedation will be fasting and require a change in their normal regimen. Management of the diabetic patient can be
divided into the preoperative and postoperative periods. Furthermore, management depends on whether the patient is treated with insulin or oral hypoglycemic agents.

Preoperative management

The general goal is to avoid excessive hyperglycemia (blood glucose > 180 mg/dL) or hypoglycemia (blood glucose < 80 mg/dL). The following guidelines are considered ideal, but may not be appropriate for all patients.

**Surgery in diabetic patients treated with oral hypoglycemic**

**Fasting (general anesthesia or intravenous sedation)**

- Minor and major surgery
  - Hold all oral agents on the day of surgery.
  - For patients with “fair” metabolic control (fasting blood glucose < 180 mg/dL), cover with regular or rapid-acting insulin (lispro, aspart, glulisine) as needed using the sliding scale (see page 149).
  - For patients with “poor” metabolic control (fasting blood glucose > 180 mg/dL), start continuous insulin infusion (CII); use of insulin on a sliding scale is unlikely to obtain adequate blood glucose control.
  - Restart oral antidiabetic medications postoperatively when patient starts eating.

**Not fasting (local anesthesia)**

- No change in medication is required if the patient will be able to eat postoperatively.

**Surgery in diabetic patients treated with insulin**

**Fasting (general anesthesia or intravenous sedation)**

- Minor surgery
  - Withhold all oral agents (if treated with combination therapy) on the day of surgery.
  - Patients with “fair” metabolic control (fasting blood glucose < 180 mg/dL): Withhold the short-acting
insulin (regular) and give half of the dose of intermediate-acting insulin (ie, NPH) the morning of the surgery.
—While the patient is taking nothing by mouth, infuse D5NS plus KCl (10 to 20 mEq/L) at 100 mL/h.
—Check blood glucose every 4 to 6 hours while the patient is taking nothing by mouth and supplement with short-acting insulin (regular) using the sliding scale (see page 149).
—Patients treated with basal (glargine) insulin should receive their usual basal insulin dose. Similarly, patients treated with continuous insulin infusion therapy (insulin pump) should receive their usual basal infusion rate.
—Restart preadmission insulin therapy once food intake is tolerated.

• Major surgery
  —Withhold all oral agents the day of surgery if patient on combination therapy.
  —Start insulin infusion prior to surgery and continue during perioperative period; the use of insulin on a sliding scale is unlikely to obtain adequate blood glucose control.

Not fasting (local anesthesia)
  —No change in medication is required if the patient will be able to eat postoperatively.

Perioperative sliding scale for use of insulin
• Blood glucose measured every 6 hours
• Administer subcutaneous regular insulin according to formula:

\[
\text{No units regular insulin} = \frac{\text{Blood sugar level} - 140}{30}
\]
Diabetic ketoacidosis (DKA)

This can occur in patients with insulin-dependent diabetes, usually as a result of inadequate insulin, infection, or drug use. It is associated with severe hyperglycemia and ketosis. There is a 1% mortality rate even when treated appropriately. The clinical manifestations include polyuria, polydipsia, dehydration, hypotension, nausea, emesis, abdominal pain, altered mental status, and Kussmaul respiration (deep respirations). The diagnosis is supported by the presence of anion gap metabolic acidosis, hyperglycemia, pseudohyponatremia, and ketoacidosis (acetoacetate and β-hydroxybutyrate) in serum and urine. The treatment of DKA requires:

- Aggressive hydration (normal saline 10 mL/kg/h)
- Insulin (regular) 10 units IV push
- Insulin (regular) 0.1 units/kg/h infusion
- Potassium 20 to 40 mEq/L in IV fluids
- Bicarbonate if pH < 7 or cardiac instability
- Change fluids to D$_5$½NS when the blood sugar level reaches 250 mg/dL

Nonketotic hyperosmolar hyperglycemia

This tends to occur in older patients with type 2 diabetes. Precipitating factors are the same as for DKA. The mortality rate is higher than with DKA due in part to the patients’ age. The key clinical features are severe dehydration and altered mental status. Significant hyperglycemia and an increase in serum osmolality (> 350 mOsm/L) are typical. The treatment includes aggressive rehydration with normal saline (up to 10 L). This should be accompanied by regular insulin IV 10 units push and then 0.05 to 0.1 units/kg/h infusion.

Thyroid Disease

Hypothyroidism

Hypothyroidism is typically characterized by the progressive destruction of thyroid tissue. It occurs in Hashimoto thyroiditis, in which an autoimmune lymphocytic infiltrate develops along with antithyroid peroxidase antibodies. Subacute thyroiditis is also an autoimmune disease and often follows a flulike illness. The condition is usually self-limiting and resolves in a few months. Hypothyroidism may also be iatrogenic, resulting from the medical treatment of hyperthyroidism. The signs and symptoms of hypothyroidism include
fatigue, weight gain, cold intolerance, constipation, facial edema, delayed deep tendon reflexes, and altered mental status. The diagnosis is usually established by observing a decreased free T4 and elevated thyroid-stimulating hormone (TSH) level. The treatment of hypothyroidism requires levothyroxine. The adequacy of treatment can be measured through TSH levels, which should be normal. Myxedema is the only emergent hypothyroid condition that can develop as a result of infection, surgery, medications, or a stressful event. The diagnosis of acute myxedema is based on a history of hypothyroidism and the development of altered mental status, seizures, coma, or hypotension. Treatment typically requires intravenous levothyroxine and corticosteroid.

Hyperthyroidism

Hyperthyroidism is most often due to Graves disease, an autoimmune disease characterized by the presence of thyroid-stimulating antibodies. There is the potential for thyroid storm with fever, cardiac arrhythmias, high output cardiac failure, coma, and death. The signs and symptoms of hyperthyroidism include tachycardia, anxiety, tremors, heat intolerance, weight loss, atrial fibrillation, diarrhea, elevated systolic blood pressure, exophthalmos, and pretibial myxedema. The diagnosis can be established by the presence of an increased free T4, decreased TSH (in most patients), and thyroid-stimulating antibodies. The treatment of hyperthyroidism may require any or all of the following, although the development of a thyroid crisis mandates a rapid and aggressive approach:

- Beta blockers (eg, metoprolol 50 to 100 mg orally twice a day)
- Propylthiouracil (PTU) or methimazole
- Sodium iodide (1 hour after PTU if thyroid crisis)
- Radioactive iodine

Adrenal Disease

Patients with adrenal disease may present with excessive adrenal function or adrenal insufficiency. Cushing syndrome is characterized by excessive plasma cortisol levels. It can be secondary to adrenal hyperplasia, pituitary adenoma, ectopic adrenocorticotropic hormone (ACTH) production, or exogenous corticosteroid administration. Patients present with truncal obesity, moon facies, abdominal striae, hirsutism, hyperglycemia, hypertension, and purpura. The diagnosis is confirmed with a 24-hour urine-
free cortisol level, a high dexamethasone suppression test, and an abnormal ACTH level. Conn syndrome is characterized by excessive aldosterone levels. This is usually secondary to adrenal hyperplasia or an adenoma of the glomerular zone, but can occur as a result of renal disease and increased serum rennin. Patients present with hypertension, hypernatremia, hypokalemia, hyperchloremia, and alkalosis. The diagnosis is confirmed by elevated serum aldosterone levels.

Adrenal insufficiency (Addison disease) can be primary as a result of destruction of the adrenal cortex due to autoimmune disease, infection, or infarction or secondary due to pituitary failure. Primary and secondary adrenal insufficiency can be distinguished based on the presence of hyperpigmentation, the ACTH (cosyntropin) level, and the cosyntropin stimulation test. The inadequate levels of adrenal cortisol place the patient at risk for an acute adrenal crisis when stressed with surgery or any infection. The acute adrenal crisis is characterized by severe hypotension and electrolyte abnormalities. Patients with adrenal insufficiency are usually managed with prednisolone replacement therapy. Patients should also receive mineralocorticoid replacement (fludro-cortisone) if the adrenal insufficiency has resulted in decreased aldosterone production. The perioperative management of patients is as follows:

- For minor procedures (extractions) the patient should double the usual corticosteroid dose on the morning of the procedure. Blood pressure should be monitored during and after the procedure. Hypotension should be treated with hydrocortisone 100 mg intramuscularly (IM) or IV.
- For major procedures the patient should have an IV line placed and receive IV corticosteroids during the perioperative period.
  — Hydrocortisone 100 mg IV every 6 hours (increase dose or frequency if needed to maintain blood pressure) OR
  — Dexamethasone 4 mg IV every 6 hours

Pituitary Disease

Diabetes insipidus (DI) can occur as a result of a decrease in pituitary antidiuretic hormone (ADH) (central DI) or as a result of renal insensitivity to ADH (nephrogenic DI). Patients present with severe polyuria, polydipsia, increased serum osmolality (> 300 mmol), and mild hypernatremia. The diagnosis of DI can be made with the water deprivation test. Central DI is
treated with DDAVP nasal spray, whereas nephrogenic DI is treated with sodium restriction and liberal water intake.

**Immunocompromised Patients**

**Autoimmune Disease**

There are a multitude of autoimmune diseases that are likely to affect surgical patients. These include rheumatoid arthritis, systemic lupus erythematosus, progressive systemic sclerosis, scleroderma, ankylosing spondylitis, mixed connective tissue disease, Sjögren syndrome, polyarteritis nodosa, and polymyositis. The autoimmune disease as well as the medical treatment of the disease results in suppression of the immune system and increases the likelihood of perioperative complications. In addition, organ systems may be the target of the disease, resulting in airway, pulmonary, cardiovascular, neurologic, musculoskeletal, renal, hematologic, and neurologic difficulties. Wound healing may also be impaired in many of these patients, and surgical infections are more common.

**Immunodeficiencies**

Deficiencies in cell-mediated immunity predispose patients to infection from bacteria, mycobacteria, viruses, fungi, and parasites. Cell-mediated immunity can be assessed by observation of the prototypical cell-mediated response of the delayed hypersensitivity reaction. This is classically seen with the Mantoux test, in which an intradermal injection of tuberculin is given. A more predictable method of evaluating cell-mediated immunity is by measuring the ratio of CD4 helper T cells to the CD8 suppressor cells. This provides quantitative information and might not necessarily correlate with normal function.

Deficiencies in humoral immunity also predispose patients to infection. Humoral immunity is best assessed by measuring the serum level of the five classes of immunoglobulin. Prior vaccination may also lead to the development of circulating antibodies, which can be readily measured to determine adequacy of the humoral system.

Deficiencies in the nonspecific immune pathway are often manifested by abnormalities of neutrophil function. This predisposes patients to bacterial and fungal infections. Although the absolute neutrophil count can be
measured, neutrophil function should also be evaluated. An absolute neutrophil count below 500 cells/mL defines neutropenia and requires patient isolation and prophylactic antibiotic and antifungal treatment. Deficiencies in complement also predispose patients to bacterial infections. Individual complement factors, as well as function of the entire complement cascade, can be measured through the CH50 assay.

Patient Management

Immunocompromised patients should have a thorough history to ascertain whether there is a history of recurrent infections, opportunistic infections, generalized lymphadenopathy, or weight loss. A thorough understanding of any identified autoimmune disease or immunodeficiency is necessary to further appreciate the effect on organ systems. Laboratory investigations may include:

- CBC with differential cell count
- Chest radiograph
- CD4/CD4 ratio
- Immunoglobulins
- Functional assays for neutrophil and complement function

Immunocompromised patients need to be aggressively treated when infections occur. The use of perioperative and postoperative antibiotics should be considered in all but the simplest surgical procedures.

Patients who have lost their spleen through surgery or sickle cell disease are a special group of patients who are at risk for bacterial infections, particularly from bacteria with polysaccharide capsules such as pneumococcus, meningo-coccus, and hemophilus. These patients should be vaccinated against these organisms. Early and aggressive treatment of infections and the use of antibiotics are appropriate.

A significant number of patients take immunosuppressive agents to modulate autoimmune disease, treat malignancy, or suppress organ transplant rejection. A multitude of drugs are used, and these can result in neutropenia and lymphopenia. This places patients at risk for poor wound healing, infection, and overwhelming sepsis. Treatment of the underlying autoimmune disease should be optimized prior to any elective surgery. Perioperative
antibiotics should be considered in most patients, and any infections that do develop should be treated aggressively.

The human immunodeficiency virus (HIV) results in progressive destruction of CD4 helper T cells. This may lead to AIDS with opportunistic infections. HIV should be considered in any patient with signs and symptoms of such infections. The diagnosis of HIV infection requires a screening enzyme-linked immunosorbent assay (ELISA) and a confirmatory Western blot test. The CD4 count is used to assess disease progression and is often combined with the polymerase chain reaction (PCR) test to evaluate viral load and the response to highly active antiretroviral therapy (HAART). The CD4 count should be known prior to all surgical procedures. This allows for risk stratification and appropriate prophylaxis against opportunistic infections. Every organ system can be affected by HIV/AIDS, and management of these patients can be challenging.

For a CD4 count below 200 cells/mL, pneumocystis pneumonia (PCP) prophylaxis with trimethoprin/sulfamethoxazole, dapsone, atovaquone, or penta-madine is needed. As the CD4 count declines below 100 cells/mL, prophylaxis against toxoplasmosis becomes important. When the CD4 count falls below 50 cells/mL, the potential for *Mycobacterium avium-intracellularare* complex (MAC) necessitates prophylaxis with a macrolide antibiotic.

**Neurologic Disease**
Seizures can occur during the perioperative period in healthy patients as well as in those with a known seizure disorder. Seizures can be classified as partial or generalized. Unremitting generalized seizure activity is termed status epilepticus. Potential causes include:

- Anesthesia (local anesthetic overdose, meperidine)
- Metabolic derangements (Na$^+$, Ca$^{2+}$, Mg$^{2+}$, glucose, urea, NH$_4^+$)
- Drug and alcohol withdrawal (benzodiazepines and barbiturates)
- Epilepsy, degenerative CNS disease
- Intracranial trauma or surgery, cerebrovascular accident (CVA)

Patients already taking antiseizure medication should be maintained on their usual regimen. New-onset seizures require treatment as well as a search for the cause. Signs and symptoms of seizure include altered mental status,
confusion, abnormal movement, and loss of consciousness. The diagnosis of a seizure may require an electroencephalogram (EEG) as well as serum electrolyte measurements and a toxicology screening. The initial treatment of perioperative seizures requires phenytoin 20 mg/kg IV at a rate of 50 mg/min. Benzodiazepines may also be used to terminate a seizure but play no role in the long-term management of recurrent seizure activity.

Cerebrovascular Accident

Atherosclerosis of the carotid blood vessels places patients at risk for transient ischemic attacks or a CVA during the perioperative period. Atrial fibrillation also increases the risk for a CVA, and patients with atrial fibrillation, who are typically on warfarin, should continue the medication during the perioperative period whenever possible. Aspirin and ticlopidine have both been shown to reduce the frequency of transient ischemic attacks and should also be continued whenever possible.

Myasthenia Gravis

Myasthenia gravis (MG) is an autoimmune disease characterized by the development of antibodies against the acetylcholine receptor. It is often the result of thymus hyperplasia or thymoma. Stressors, including surgery, infections, and certain medications (eg, aminoglycosides, phenytoin, and meperidine), may predispose patients to a myasthenic crisis. The signs and symptoms of a myasthenia crisis include weakness, fatigue, ptosis, diplopia, dysarthria, dysphonia, dysphagia, and respiratory distress. The diagnosis of MG requires:

- A history of repetitive muscle activity with progressive weakness
- Tensilon (edrophonium chloride) test (2-mg IV test dose, then 8 mg IV).

This produces rapid transient improvement in symptoms.

- Electromyogram (EMG) with decreased amplitude on repetitive nerve stimulation
- Presence of anti-acetylcholine receptor (anti-AChR) antibody
- CT or magnetic resonance imaging (MRI) for chest thymoma
The treatment of MG may involve:

- Thymectomy
- Anticholinesterase medication (pyridostigmine)
- Intravenous immunoglobulin therapy, plasmapheresis (MG crisis)
chapter 11

Managing Medical Emergencies

Because of their involvement in the care of the severely traumatized patient and their frequent use of anesthetic agents, it is imperative for oral and maxillofacial surgeons to be well versed in the management of medical emergencies. This chapter discusses the most common medical emergencies, as well as the current protocols for basic and advanced cardiac life support.

Basic Cardiac Life Support (BCLS)
Cardiopulmonary Resuscitation (CPR)

Indications
- Initial evaluation for all medical emergencies
- Respiratory insufficiency or arrest
- Cardiac arrest

Technique (ABCs): Adult

Airway: Should be evaluated and managed for patency and obstruction (see following section on advanced cardiac life support [ACLS], page 157).
- Chin lift
- Jaw thrust
- Head tilt

Breathing: Evaluate and manage for adequate rate and volume (look, listen, and feel).
• Give two full breaths (2.0 seconds per breath), allowing lungs to deflate between breaths.
• If just applying rescue breathing, then continue at one breath every 5 to 6 seconds.

Circulation: Establish pulselessness (5 to 10 seconds) by carotid artery palpation.
• Single rescuer:
  — Begin chest compressions (1.5 to 2 inches of sternal depression).
  — Continue with cycles of 30 compressions and two breaths at a rate of 100 compressions/min. Push hard and fast.
  — Check pulse every five cycles.

• Two rescuers:
  — Cycle remains 30 compressions for each two breaths unless the airway is protected with an advanced airway (eg, with intubation; then use 10 to 12 breaths per minute with continuous compressions at 100/min).
  — Rate remains at 100 compressions/min even when using an advanced airway and two-rescuer CPR.
  — Rescue breather may assess adequacy of chest compressions by feeling for a pulse during CPR.
  — Reevaluate pulselessness for 5 to 10 seconds at each change of rescuer position. This should occur approximately every 2 minutes or 5 cycles of CPR.

Obstructed Airway

Causes
• Partial or complete occlusion of the airway by foreign body, dentures, vomitus, blood, food (ie, “cafe coronary”)
• Incorrect or inadequate positioning of head

Technique

Unconscious patient: First examine for foreign body; then open airway and attempt to ventilate. If unable to do so, perform the
Heimlich maneuver (subdiaphragmatic abdominal thrusts) until foreign body is expelled. Chest compressions may be used in the unconscious patient.

Conscious patient (who gives the universal sign of choking or indicates an airway obstruction by an inability to speak): Attempt the Heimlich maneuver as above.

Obese or pregnant patients: Chest compressions may be done more easily than abdominal thrusts.

Note: Finger sweeps should only be used when solid material can be visualized in the airway.

Pediatric BCLS

Indications: Respiratory insufficiency, respiratory arrest, or cardiac arrest in an infant or child.

Technique

Child (1 to 8 years)
- Can use one hand or two hands for chest compressions.
- Depress sternum ½ to ⅓ the depth of the chest at a rate of 100 times/min.
- Give two breaths for every 30 compressions as for an adult in one-rescuer CPR. For two-rescuer CPR, revert to 15:2.
- With an obstructed airway do not perform blind finger sweeps. Perform abdominal thrusts (Heimlich maneuver). If abdominal thrusts fail, chest compressions may be used.
- If performing rescue breathing only, give one breath every 3 to 5 seconds.

Infant (birth to 1 year)
- Use brachial pulse rather than carotid.
- Use only two fingers for chest compressions, which are given on the sternum just below a line between the two nipples.
Depress the sternum ½ to ⅓ the depth of the chest at a rate of at least 100 times/min.
Give one breath for each five compressions.
With an obstructed airway do not perform blind finger sweeps. Perform back blows and chest compressions. Do not perform abdominal thrusts.
If performing rescue breathing only, give one breath every 3 to 5 seconds.

Advanced Cardiac Life Support (ACLS)

General Indications

- **Myocardial infarction:** Any patient with chest pain of unknown cause who is unresponsive to nitroglycerin should be assumed to be having a myocardial infarction and should be entered into the emergency medical system.
- **Arrhythmias**
- **Cardiac arrest**

Adjuncts for Airway Management

Patient positioning

*Indications:* Patients with spontaneous respiration but with upper airway compromise will often benefit from changes in head, body, or jaw position.

*Upper airway obstruction:* May be seen in the unconscious patient due to lack of tonicity of the tongue and suprahypoid musculature, with resultant relaxation of the tongue against the posterior pharyngeal wall.

*Traumatic injuries:* Rule out cervical spine injuries before making significant positional changes.

*Technique:* If a cervical spine injury has been ruled out, the patient may be turned onto one side or placed in the supine position.
position and the neck hyperextended to open the airway. Alternatively, or in the patient with a possible cervical spine injury, the chin may be pulled anteriorly (“chin lift”) or the angles of the jaw pushed forward (“jaw thrust”) without significant neck flexion or extension.

Oropharyngeal airway

*Indications:* Designed for use in the unconscious but spontaneously breathing patient who has airway compromise due to tongue position problems. This device may also be used to assist in airway maintenance during controlled bag-valve-mask ventilation.

*Mode of action:* Physically separates tongue from posterior pharyngeal wall.

*Tolerance:* Not tolerated by conscious patients as it activates the gag reflex.

*Length:* When the tip of the airway is at the corner of the mouth, the flange should be at the angle of the mandible.

*Technique:* Device is inserted over the tongue with the assistance of a tongue blade or by inverting the airway and rotating it over the tongue during insertion.

Nasopharyngeal airway

*Indications:* May be used in the conscious or unconscious patient who has spontaneous ventilation but demonstrates airway compromise due to tongue position problems. May also be used to assist in airway maintenance for bag-valve-mask ventilation.

*Mode of action:* Physically separates tongue from posterior pharyngeal wall.

*Tolerance:* Unlike the oropharyngeal airway, this is usually well
tolerated by conscious patients.

*Internal diameter*
- Large adult: 8 to 9 mm
- Small adult: 6 to 8 mm

*Length:* The length should equal the distance from the tip of the nose to the earlobe.

*Technique:* Inserted, after lubrication, directly through the nostril into pharynx.

Bag-valve-mask

*Indications:* Primarily used to initially and rapidly provide controlled or assisted ventilation in the unconscious patient with respiratory insufficiency or arrest. May also be used as a supplemental oxygen source in the spontaneously breathing patient.

*Description:* The device consists of a self-filling reservoir bag, a one-way valve, and a mask attachment. Ideally, the mask and bag should be transparent to allow early visualization and treatment of vomiting. It is often combined with an oropharyngeal or nasopharyngeal airway to allow controlled ventilation.

*Advantages*
- Can be used directly with an endotracheal tube for controlled ventilation
- Can be used with supplemental O₂ for high oxygen concentrations
- Allows for spontaneous ventilation

*Disadvantages*
- Proper use requires special training and practice.
- Adequate ventilation depends on good mask fit, which is often difficult to maintain.
• Use of this device does not ensure an adequate airway.

Technique
• Attach mask of appropriate size to bag.
• Place mask over mouth and nose and ensure good seal. Make sure airway is open (see page 155).
• Hold mask in place with one hand while the other hand intermittently squeezes the bag to accomplish controlled ventilation.

Cricothyrotomy

Indications
• Severe, multiple facial trauma with airway compromise
• Upper airway trauma (eg, trauma to pharynx or tongue)
• Inability to intubate patient

Hazards
• Hemorrhage
• Perforation of the esophagus by going too deep with the initial incision
• Passage of the airway device into the surrounding tissues during insertion
• Subcutaneous or mediastinal emphysema

Technique
• Cleanse area with povidone-iodine.
• Palpate thyroid cartilage (Adam’s apple) and cricoid cartilage directly below it.
• Make 2-cm horizontal incision through skin and cricothyroid membrane into trachea.
• Insert scalpel handle into incision and rotate 90 degrees to open airway.
• Place an appropriately sized endotracheal tube or
tracheotomy tube into opening.
- Ventilate with a bag-valve-mask or other positive-pressure ventilation device.

Tracheotomy: Because of the possible complications, this procedure should only be performed in an appropriate operating room setting after securing the airway emergently with another modality (endotracheal tube, cricothyrotomy). Because this is not an emergency procedure, it is not discussed in this chapter (see chapter 9).

Laryngeal mask airway (LMA)

*Indications:* The LMA is an excellent advanced rescue airway. It may be used in place of an endotracheal tube for both assisted and controlled ventilation. It should be used only for the unconscious patient because it is not tolerated by the conscious patient.

*Advantages*
- Provides an advanced, maintainable, and secure airway
- Easy placement
- Does not require special placement equipment (eg, laryngoscope)
- Can be, and usually is, placed blindly
- Provides for 100% oxygen supplementation
- Compatible with a bag-valve-mask or other standard 15-mm connector

*Hazards*
- Does not provide a seal against aspiration
- May induce vomiting and aspiration in obtunded but not unconscious patient
- May not always provide an adequate seal for controlled ventilation

*Equipment*
- Adult: size 3 to 4
• Child: size 1 to 2

**Technique**

• With patient in the supine position, open mouth with thumb and forefinger.
• Place LMA inverted (using the reverse curve just as with an oropharyngeal airway) until the soft palate is reached; then rotate it and place it down the airway until seated in the hypopharynx.
• The LMA should feel solidly seated in the hypopharynx. If not, remove and place again.
• A laryngoscope can be used to assist with correct placement, if desired.
• Inflate balloon with approximately 15 to 20 mL of air or until no leak occurs with ventilation.
• Connect end of tube to any 15-mm adaptor (eg, bag-valve-mask).

**Endotracheal intubation**

*Indications:* May be used in any patient when there is a need for airway maintenance, airway protection, or the ability to provide mechanical or assisted ventilation. Although it is most commonly used in the unconscious patient, it may also be used for the conscious patient under appropriate circumstances.

**Advantages**

• Provides a secure airway that is easy to maintain once obtained.
• Provides ability for positive-pressure ventilation and 100% O₂ supplementation.
• Compatible for use with either a bag-valve-mask or an anesthesia machine using a standard 15-mm connector.
• A cuffed endotracheal tube helps prevent aspiration.
• Provides ability to directly suction airway.
• Provides possible route of administration for certain emergency medications when intravenous (IV) access is not available, including NAVEL:

  Naloxone
  Atropine
  Valium
  Epinephrine
  Lidocaine

_Hazards_
• Endobronchial intubation (usually into right main bronchus)
• Esophageal intubation
• Damage to vocal cords
• Passage of tube into surrounding tissue planes
• Damage to teeth from injudicious use of laryngoscope

_Equipment_
• Endotracheal tubes
  1. Size
     —Woman: approximately 7 to 8 mm
     —Man: approximately 8 to 9 mm
     —Child: use formula (age + 16)/4 mm

  2. Cuff
     —Low volume/high pressure (5 to 10 mL).
     —High volume/low pressure (20 to 35 mL); this has the advantage of decreased tracheal pressure if tube is needed for prolonged period of time.
     —Cuffless tubes may be used for children younger than 8 years because their narrower subglottic area will act as an effective cuff.
Laryngoscope blades

1. Straight blades: Used by lifting tongue and epiglottis simultaneously; sizes are:
   — Adult: size 3 to 4
   — Child: size 2 to 3
   — Infant: size 1 to 2

2. Curved blades: Used by placing tip of blade in the vallecula (recess between base of tongue and epiglottis) and lifting tongue only. The epiglottis will be indirectly lifted out of the way to allow direct viewing of the vocal cords; sizes are:
   — Adult: size 3 to 4
   — Child: size 2 to 3
   — Infant: size 1 to 2

Technique
- With patient in supine position, elevate and extend head slightly into “sniffing” position.
- Open mouth and carefully slide laryngoscope blade down the tongue until epiglottis is visualized.
- When using a straight blade, pick up the epiglottis with the end of the blade and elevate the tongue and epiglottis vertically until the vocal cords are seen.
- When using a curved blade, place the top of the blade into the vallecula and elevate the base of the tongue vertically until vocal cords are seen.
- Pass endotracheal tube through vocal cords until cuff is just beyond cords.
- Inflate cuff until there is no air leakage around tube.
- Verify bilateral breath sounds by auscultation.

Adjuncts for Supplemental Oxygenation
Because oxygen saturation and delivery may be compromised during
emergency situations, an increased inspired \( \text{O}_2 \) concentration should be considered.

Nasal cannula

\textit{Indications:} Spontaneously breathing patient who requires minimal oxygen supplementation (e.g., stable post–cardiac infarction patient).

\textit{Oxygen concentration:} Ranges from 24\% to 40\%, depending on inspired flow rate of 1 to 6 L/min.

\textit{Flow:} Each liter increase in flow increases the inspired \( \text{O}_2 \) by about 4\%.

Facemask

\textit{Indications:} Spontaneously breathing patient who requires moderate oxygen supplementation.

\textit{Inspired oxygen concentrations:} Range from 40\% to 60\%, depending on liter flow.

\textit{Oxygen flow rate:} Should be greater than 6 L/min to prevent rebreathing of exhalant. By modifying the facemask with an oxygen reservoir, a greater percentage of the total inspired volume can be provided with each breath. At 6 L/min the inspired \( \text{O}_2 \) concentration is approximately 60\%; at 10 L/min it can approach 100\% if a tight-fitting mask is used.

Venturi mask

\textit{Indications:} Spontaneously breathing patients who require carefully controlled or limited inspired oxygen concentrations. Provides a range of fixed \( \text{O}_2 \) concentrations. Most commonly used in patients with chronic obstructive pulmonary disease.
(COPD) who are on oxygen-based respiratory drive rather than CO\textsubscript{2}-induced drive. High O\textsubscript{2} concentrations can block the stimulation of the respiratory centers and lead to respiratory depression.

Concentrations: May be fixed at 24% or 28% at 4 L/min flow; 35% or 40% at 8 L/min flow.

Defibrillation and Cardioversion

Indications
- Ventricular fibrillation
- Ventricular tachycardia
- Certain atrial tachycardias

General considerations
- Most effective treatment for ventricular fibrillation.
- Effectiveness of shock is related to time from onset of defibrillation.
- Preferred defibrillators are powered by direct current.
- Power ranges from 0 to 360 J.
- Defibrillator may be synchronized to the R wave for nonfibrillating arrhythmias (synchronized cardioversion).
- Automatic external defibrillators: These devices automatically sense an inappropriate rhythm and either advise a shock or, in some cases, automatically defibrillate the patient.

Technique
- Apply conductive medium to paddles.
- Standard anterolateral placement is performed by placing the paddles over the right anterior chest just below the clavicle and at the left midaxillary line adjacent to the left nipple.
- Anterior-posterior electrodes may also be used by placing them directly over the precordium near the left sternal border and on the back beneath the anterior electrode.
- Always place paddles at least 5 inches from a cardiac pacemaker.
- Clear the area of personnel touching the patient.
- Apply pressure to paddles and press buttons.
• Automatic external defibrillator: after applying the electrocardiogram (ECG) pads, depress the “analyze” button. The machine will either provide a shock, if needed, or instruct you to depress the “shock” button.

Power settings and usage: See individual protocols for each type of arrhythmia (next section).

Automatic External Defibrillator

Indications: Allows prompt defibrillation by personnel not trained in dysrhythmia recognition

General considerations
• Automatically recognizes fatal arrhythmias and advises operator when shock is indicated
• Automatically performs appropriate shock when activated
• Relatively inexpensive
• Can be operated with little training

Technique
• First, power on the defibrillator.
• Attach adhesive pads according to package directions (generally right shoulder and left lower side).
• Activate “analyze” button for automatic rhythm recognition.
• If shock indicated, notify all personnel to “stand clear.”
• Activate “shock” button.
• Follow audible commands of defibrillator until “no shock indicated” is directed.
• Check pulse. Begin sequence of CPR, if indicated.

Specific Protocols

Advanced cardiac life support protocols are only guidelines, and resuscitative measures should be performed as guided by the patient’s condition. For ECG dysrhythmia recognition, see chapter 5. The various drugs used in ACLS are listed in Table 11-1.

Pulseless arrest (Fig 11-1): Composed of several conditions:
• Ventricular fibrillation: Uncoordinated fibrillation of the myocardium
with an ineffective cardiac output.

- **Pulseless ventricular tachycardia**: A tachyarrhythmia of the ventricles that does not produce a cardiac output.
- **Asystole**: A “flat line” rhythm with no electrical complexes. When occasional complexes are seen it is termed an *agonal rhythm*.

**Table 11-1 Emergency cart drugs**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indication</th>
<th>Dosage</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine</td>
<td>Tachycardia</td>
<td>6–12 mg</td>
<td>Rapid IV push</td>
</tr>
<tr>
<td>Albuterol</td>
<td>Bronchoconstriction</td>
<td>1–2 inhalations</td>
<td>Inhaled</td>
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<tr>
<td>Aminophylline</td>
<td>Asthma</td>
<td>5–7 mg/kg</td>
<td>IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>loading</td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Arrhythmias</td>
<td>150 mg/10 min</td>
<td>IV</td>
</tr>
<tr>
<td>Ammonia aromatic</td>
<td>Syncope</td>
<td>1 carpule</td>
<td>Inhaled</td>
</tr>
<tr>
<td>Atropine</td>
<td>Bradycardia</td>
<td>0.5–1.0 mg</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Dextrose 50% (D50)</td>
<td>Hypoglycemia</td>
<td>50 mL (25 g)</td>
<td>IV, oral</td>
</tr>
<tr>
<td>Diazepam</td>
<td>Seizures, anxiety</td>
<td>2.5–10 mg</td>
<td>IV</td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>Allergy,</td>
<td>25–50 mg</td>
<td>IV, IM, oral</td>
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<td></td>
<td>hypersensitivity</td>
<td></td>
<td></td>
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<tr>
<td>Dopamine</td>
<td>Hypotension</td>
<td>3–20 μg/kg/min</td>
<td>IV</td>
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<td>Epinephrine</td>
<td>Bronchoconstriction,</td>
<td>0.2–0.5 mg</td>
<td>SC, IM</td>
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<tr>
<td></td>
<td>hypersensitivity</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Cardiac arrest</td>
<td>0.5–1.0 mg</td>
<td>IV</td>
</tr>
<tr>
<td>Ephedrine</td>
<td>Vasopressor</td>
<td>2.5–25 mg</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>Adrenal insufficiency</td>
<td>100 mg</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Ventricular ectopy</td>
<td>1 mg/kg</td>
<td>IV</td>
</tr>
<tr>
<td>Morphine</td>
<td>Myocardial infarction</td>
<td>2–8 mg</td>
<td>IV, IM</td>
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<td>Naloxone</td>
<td>Narcotic overdose</td>
<td>0.4 mg</td>
<td>IV, IM</td>
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<td>Nitroglycerin</td>
<td>Angina</td>
<td>1/50–1/200 g</td>
<td>SL</td>
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<td>Phenytoin</td>
<td>Seizures</td>
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<tr>
<td>Verapamil</td>
<td>Supraventricular tachycardias</td>
<td>5–10 mg</td>
<td>IV</td>
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</tbody>
</table>

*IV = intravenous; IM = intramuscular; SC = subcutaneous; SL = sublingual.*

- **Pulseless electrical activity (PEA)**: The uncoupling of the electrical complexes and the physical contraction of the myocardium; although an electrical rhythm is present, there is no effective cardiac output.
Tachycardia with a pulse (Fig 11-2): A tachyarrhythmia of atrial or ventricular origin; the patient may or may not be symptomatic. Includes paroxysmal supraventricular tachycardia and ventricular tachycardia.

Bradycardia (Fig 11-3): Slowing of the heart rate below 50 to 60 beats/min (BPM) with inadequacy of clinical perfusion.
Fig 11-1 Pulseless arrest algorithm. (Reproduced with permission. Advanced Cardiovascular Life Support Provider Manual, 2006. Copyright American Heart Association.)
Fig 11-2 Tachycardia algorithm. (Reproduced with permission. Advanced Cardiovascular Life Support Provider Manual, 2006. Copyright American Heart Association.)
Other Medical Emergencies
Loss of Consciousness (No Specific Diagnosis)

Etiology: May be caused by a variety of disorders, including syncope, hypotension, drug reaction, seizure, insulin reaction, cerebrovascular accident, hyperventilation, and acute adrenal insufficiency. In general, the mechanisms involved are:

- Psychogenic
- Decreased cerebral perfusion
- Metabolic changes
- Drug reaction
Recognition: Unconsciousness is a state of diminished responsiveness and arousability of the patient; protective reflexes may also be obtunded.

- Recognition of unconsciousness
  — Lack of response to auditory and physical stimuli
  — Loss of protective reflexes
  — Lack of ability to maintain an airway

Management

- Activate office or local emergency system.
- Place patient in supine or “astronaut” position (torso horizontal and legs elevated).
- Begin CPR (see BCLS section, page 155).
- Assess response to treatment and treat for specific etiology (eg, insulin shock) as soon as recognized.
- If reason for unconsciousness is not apparent, consider administering 0.4 mg of naloxone IV to rule out narcotic overdose and 25 g of dextrose (50 mL of 50% dextrose solution [D$_{50}$]) to rule out insulin shock.

Prevention

- Adequate medical evaluation to elicit history of predisposing medical conditions.
- Decrease physical and psychologic stress of procedure; use appropriate measures for pain control to prevent psychic mechanisms.
- Use the supine position whenever possible to increase venous return and minimize the risk of syncope.

Syncope (Fainting, Vasovagal Reaction)

Etiology: Transient cerebral ischemia leading to loss of consciousness; usually caused by peripheral pooling of blood

- Psychogenic factors: Anxiety, stress, pain
- Nonpsychogenic factors: Sitting or standing position during surgery, hypoglycemia, exhaustion

Recognition
**Presyncopal**
- Loss of facial color (pallor)
- Diaphoresis
- Nausea
- Yawning and hyperpnea
- Tachycardia followed by hypotension and bradycardia

**Syncopal**
- Transient, sudden loss of consciousness
- Irregular, diminished, or absent ventilation
- Convulsive movements
- Hypotension and bradycardia

**Management**
- Place patient in supine or astronaut position; this increases venous return, which increases cerebral blood flow.
- Establish a patent airway (see “Adjuncts for Airway Management,” page 157).
- Loosen restrictive clothing.
- Monitor vital signs.
- Administer supplemental oxygen (see “Adjuncts for Supplemental Oxygenation,” page 161).
- Administer reflex stimulants such as ammonia inhalants and cold compresses.
- If recovery does not begin within a few minutes, continue basic life support and consider other causes of unconsciousness.

**Prevention**
- Minimize patient’s anxiety and stress; use appropriate sedation and pain control methods.
- Advise appropriate dietary intake before appointment.
- Put patient in the supine or semisupine position whenever possible.

**Hyperventilation Syndrome**
Etiology: A state of rapid breathing manifested by an increased depth and/or frequency of ventilation. The usual cause is anxiety, but it may also be produced by metabolic acidosis, hypercarbia, drug reaction, organic central nervous system disturbance, or pain.

Recognition
- Tightness in chest
- Dyspnea
- Apprehension
- Palpitation
- Chest or abdominal discomfort
- Hyperventilation (25 to 30 breaths/min)
- Paresthesia of mouth, hands, or feet
- Carpopedal spasm
- Eventual loss of consciousness

Management
- Position patient in a comfortable position; usually this will be sitting rather than supine.
- Calm patient in a reassuring manner.
- Have patient attempt slow breathing at a reasonable rate; instructing the patient on intermittent breath-holding or “cadence” (measured) breathing is often useful.
- If necessary, allow patient to correct respiratory alkalosis by rebreathing his or her own exhaled air; this can be done with a paper bag or a full facemask on an anesthesia machine (with no O₂ flow).
- If the syndrome continues, consider sedation to reduce anxiety (eg, diazepam 5 to 10 mg or midazolam 2.5 to 5.0 mg).

Prevention
- Ascertain previous medical history.
- Use appropriate pain control and sedation technique to reduce anxiety.

Asthmatic Reaction

Etiology: A paroxysmal state of hyperactivity of the tracheobronchial tree
**Extrinsic asthma:** Bronchospasm as a result of an extrinsic allergen (food, pollen); this is immunoglobulin E (IgE) mediated and usually occurs in children.

**Intrinsic asthma:** Bronchoconstriction caused by nonallergic factors such as infections, inhaling irritating fumes, smoking, and emotional stress; more likely in adults and usually more severe and chronic in such persons.

**Recognition**
- May occur suddenly or have a slow onset
- Sense of tightness in chest
- Coughing (productive or nonproductive)
- Wheezing (with expiratory phase longer than inspiratory phase)
- Dyspnea
- Anxiety
- Tachycardia

**In more severe cases**
- Cyanosis
- Use of accessory muscles of respiration
- Nasal flaring
- Supraclavicular retraction
- Agitation
- Hypoxia

**In status asthmaticus**
- Prolonged asthma attack lasting hours to days
- Fatigue
- Hypoxia
- Shock
- Tendency to airway obstruction and occasional fatality

**Management**
- Position patient in most comfortable position.
- Administer inhalant therapy with aerosol bronchodilator (albuterol,
epinephrine, isoproterenol, metaproterenol); use patient’s own medication if available; if not, use what is in emergency kit.

- Administer supplemental oxygen (see “Adjuncts for Supplemental Oxygenation,” page 161).
- If attack continues, administer epinephrine 0.2 to 0.4 mg subcutaneously.
- An infusion of aminophylline 5 to 7 mg/kg can be started and given slowly if the attack continues after epinephrine has been given.

Prevention

- Minimize anxiety with appropriate pain control and sedation measures.
- Confirm proper serum levels of medications patient is taking for asthma.
- Have patient’s own medication available for use if an attack occurs.

**Table 11-2 Clinical characteristics of hypoglycemia and hyperglycemia**

<table>
<thead>
<tr>
<th></th>
<th>Hypoglycemia</th>
<th>Hyperglycemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset</td>
<td>Rapid</td>
<td>Slow</td>
</tr>
<tr>
<td>Skin</td>
<td>Pale, clammy</td>
<td>Dry, flushed</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Rare</td>
<td>Often</td>
</tr>
<tr>
<td>Breath</td>
<td>Normal</td>
<td>Acetone or fruity</td>
</tr>
<tr>
<td>Respiration</td>
<td>Normal</td>
<td>Kussmaul</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal</td>
<td>Hypotension</td>
</tr>
<tr>
<td>Pulse</td>
<td>Full, bounding</td>
<td>Weak, rapid</td>
</tr>
<tr>
<td>Seizures</td>
<td>Occasional</td>
<td>None</td>
</tr>
<tr>
<td>Insulin level</td>
<td>Excessive</td>
<td>Deficient</td>
</tr>
</tbody>
</table>
Avoid large doses of barbiturates or narcotics if sedation is being used; these may increase inspissation of secretions.

Attempt to remove or eliminate possible allergens in the office; avoid unnecessarily prescribing aspirin or penicillin products, as these are common allergens.

Acute Hypoglycemia (Insulin Reaction)

Etiology: Sudden decrease in serum blood sugar, usually seen in patients with diabetes, caused by an overdose of insulin or the lack of normal dietary intake following normal insulin dosage (eg, postoperatively). The onset is usually rapid when the patient is taking injectable insulin, and much slower when taking oral antihyperglycemics.

Recognition (Table 11-2)

- Blood glucose of less than 80 mg/dL (normal = 80 to 110 mg/dL)
- Suddenly decreased cerebral function as manifested by mental confusion, lethargy, an inability to think clearly, or diminished verbal skills
- Hunger
- Nausea and increased gastric motility
- Diaphoresis with cold, clammy extremities
- Tachycardia
- Peculiar behavioral patterns manifested by belligerence and lack of cooperation
- Eventual unconsciousness
- Seizures
- Hypotension, shock, and eventual death

Management

- Early recognition by history and physical examination.
- If patient is conscious, administer oral carbohydrate such as orange juice or a sugar-containing soft drink; provide this slowly, especially in severe cases.
- If patient is unconscious, start an IV infusion and administer 25 g of dextrose (50 mL of D$_{50}$) over 2 minutes; recovery should be rapid.
An alternative to be considered if IV access is not available is glucagon 1 mg intramuscularly (IM).

Continual observation of the patient for residual or repeated complications.

Prevention

- Obtain history of diabetes, insulin dosages and time taken, and dietary habits.
- Provide specific insulin and dietary guidelines to be followed during the perioperative period (eg, patients who should have nothing by mouth may be given half their normal insulin dose and an infusion with a solution of 5% dextrose in water (D\textsubscript{5}W) begun at the time of surgery).
- Monitor blood or urine glucose levels during the preoperative, perioperative, and postoperative phases.

Acute Hyperglycemia (Diabetic Coma)

Etiology: A state of elevated blood glucose, usually a manifestation of diabetes mellitus. This may be caused by pancreatic disease or other endocrine abnormality, trauma, or drugs. Other specific precipitating factors include pregnancy, exercise, hyperthyroidism, overdose of thyroid replacement drugs, epinephrine, steroid therapy, or infections.

Recognition (see Table 11-2): The symptoms of hyperglycemia, unlike the rapid onset of the symptoms of hypoglycemia, are usually slow to develop, often taking days. The emergency situation is usually caused by the final phase of hyperglycemia: ketoacidosis and coma.

- History or signs and symptoms of diabetes, including polydipsia, polyphagia, polyuria, and weight loss
- Fatigue
- Headache
- Abdominal pain, nausea, vomiting
- Dyspnea (respiratory compensation for metabolic acidosis)
- Warm, dry skin
- Acetone smell on breath (also described as sweet or fruity)
- Weak, rapid pulse
- Loss of mental acuity
- Coma and death
**Management:** It is of the utmost importance to remember that a patient with a history of diabetes who suddenly manifests abnormal or bizarre behavior, or who lapses into unconsciousness, should be treated for hypoglycemia until proven otherwise. A small dose (dextrose, 25 g) will do little to affect hyperglycemia but will rapidly treat hypoglycemia. If hyperglycemia has been proved:

- Assess airway, breathing, and circulation (ABCs) and manage as necessary (see CPR section, page 155).
- Begin an IV infusion with normal saline to facilitate definitive treatment.
- In an office setting, the patient should be immediately transported to an emergency facility for management, with constant serum glucose measurements.
- In the hospital setting, the patient can be treated with regular insulin with the dosage dependent on the level of hyperglycemia.

**Prevention**

- Obtain diabetic history, including dietary intake and insulin usage; provide appropriate instructions before surgery.
- Avoid stress through appropriate pain control and sedation techniques.
- Treat infections aggressively.
- Avoid the use of steroids in diabetic patients.

**Seizures**

**Etiology:** A condition of paroxysmal neuronal discharge in the brain characterized by an alteration in consciousness and uncoordinated muscle activity or abnormal sensory phenomena, behavior, or perception. May be focal or generalized. Seizures may be idiopathic or caused by fever, cerebrovascular accidents, central nervous system infection, head injury, toxic and metabolic disorders (eg, hypoxia, hypercapnia, hypocalcemia, hypoglycemia), or drug overdose. Must differentiate epileptic seizures from syncope, cerebrovascular accident, or hypoglycemia.
Recognition: Several types of seizures must be recognized.

*Generalized (grand mal) convulsive seizures (tonic-clonic)*: May be divided into three distinct phases.

- **Prodromal phase**
  - May occur several minutes to several hours before motor activity.
  - May see minor or overt changes in personality, with anxiety or depression.
  - Patient may have an aura immediately before motor activity; this is characterized by visual, auditory, or olfactory sensations.

- **Ictal (convulsive) phase**
  - Loss of consciousness
  - Tonic extensor rigidity of the extremities
  - May see dyspnea or stertorous breathing
  - May see cyanosis
  - Generalized clonic movements
  - Frothing at the mouth
  - Usually lasts 2 to 5 minutes
  - Urinary or fecal incontinence

- **Postictal phase**
  - Gradual return of consciousness
  - Generalized relaxation and deep sleep
  - Disorientation and confusion
  - Amnesia

*Petit mal (absence) seizures*

- Primarily in children.
- Patient appears distracted or confused.
- May see intermittent blinking.
- May see blank stare.
- No prodromal or postictal phase.
**Status epilepticus**
- Recurrence of seizures without any recovery period.
- Seizure activity is the same as in generalized seizure.
- May last hours to days.
- Most commonly seen with metabolic disturbances or drug or alcohol withdrawal.
- Hyperthermia (temperature higher than 105°F), tachycardia, and hypertension.
- May lead to death from cardiac arrest or brain damage from cerebral hypoxia or decreased cerebral blood flow.

**Management**

*Generalized convulsive seizures*: Management is centered on protecting the patient from injury.
- Place patient supine on floor with head turned to side.
- Place soft object under head.
- Consider placing a soft object (gauze-wrapped tongue blades or handkerchief) between the teeth to prevent tongue biting; avoid hard objects or placement in such a way as to cause airway obstruction.
- Remove nearby objects that may cause harm.
- Gently aspirate secretions in the buccal sulcus, if possible.
- Consider supplemental oxygen if necessary (eg, cyanosis).
- Check ABCs and provide support as needed.
- Monitor vital signs during recovery.

Petit mal (absence) seizures: No emergency treatment needed.

*Status epilepticus*: Consider terminating any seizure activity lasting longer than 5 minutes.
- Treat initially as for generalized convulsive seizure.
• Begin an intravenous infusion and administer diazepam 2 mg/min until seizures terminate or a dose of 10 to 15 mg is reached (divide dose in half for children).
• If diazepam is not available, consider a short-acting barbiturate (pentobarbital 25 mg/min or methohexital 10 to 20 mg/min); these are more likely to increase postictal cerebral and respiratory depression.

Prevention
• Ascertain medical history and medication compliance.
• Avoid toxic doses of local anesthetics because this is the most likely cause of a seizure in the office setting.
• Avoid factors that may cause physical or psychologic stress or fatigue.
• Stress preoperative dietary intake to prevent hypoglycemia.

Angina Pectoris

Etiology: Acute onset of chest pain associated with ischemic coronary artery disease resulting from insufficient coronary blood flow to meet myocardial oxygen demands. Precipitating factors include emotional or physical stress, smoking, fever, and hypoxia.

Recognition
• Chest pain; usually a crushing, substernal tightening, pressing, or aching pain.
• Radiation of pain to left shoulder, arm, neck, mandible, and face; occasional radiation to right shoulder and arm may also occur.
• Episodes are usually characteristic and consistent for each patient; variation may indicate an impending infarction.
• Tachycardia.
• Hypertension.
• Diaphoresis.
• Occasional dyspnea.
• May see ECG dysrhythmias.

Management: Treatment, in general, is aimed at increasing coronary blood flow and decreasing myocardial oxygen demand.
• Position patient in most comfortable position (usually sitting upright).
• Administer supplemental oxygen (see “Adjuncts for Supplemental Oxygenation,” page 161).
• Administer a coronary vasodilator. Nitroglycerin is the initial drug of choice. Because nitroglycerin comes in three dosages, the patient should take one of his or her own tablets sublingually, if available. If the patient does not have nitroglycerin, it should be provided from the emergency cart (1/150 mg tablets should be used).
• If the first tablet fails to produce relief, a second and third tablet may be administered at 5-minute intervals.
• If the patient fails to respond to three doses of nitroglycerin or amyl nitrate in a 10-minute period, myocardial infarction must be assumed and the patient transported to an acute care facility for treatment and monitoring.

Prevention
• Ascertain previous medical history of angina or myocardial infarction.
• Eliminate stress and anxiety by using appropriate pain management and sedation techniques.
• Consider administering prophylactic nitroglycerin before stressful procedure.

Myocardial Infarction
Etiology: Infarction and necrosis of heart muscle caused by the imbalance of myocardial oxygen demand and myocardial oxygen supply (ie, prolonged tissue hypoxia or anoxia).

Recognition
• Chest pain; usually a crushing, substernal tightening, pressing, or aching pain.
• Variation of the chest pain from the patient’s typical angina may occur.
• Tachycardia or bradycardia.
• Hypertension or hypotension.
• Diaphoresis.
• Dyspnea.
• ECG dysrhythmias.
• May lead to cardiac arrest.
Management

- Initial treatment is the same as for angina pectoris.
- After failure of the third dose of nitroglycerin to relieve pain, a myocardial infarction must be assumed.
- Continue oxygen supplementation.
- Monitor ECG for specific dysrhythmias and treat as per specific ACLS protocols (see “Specific Protocols,” page 163).
- Consider morphine, 2 to 8 mg, to relieve pain and anxiety.
- Give aspirin, 325 mg orally.
- Transport to an acute care facility for monitoring and definitive treatment.

Prevention: Same as for angina pectoris

Allergy/Anaphylaxis

Etiology: A state of hypersensitivity caused by exposure or re-exposure to a particular antigen. Symptoms range from mild urticaria to fatal anaphylaxis. Possible causative agents include antibiotics, analgesics, barbiturates, local anesthetics, acrylic monomer, and preservatives. Reactions may be immediate or delayed.

Recognition

**Allergy**

- Itching
- Rash
- Redness
- Angioedema
- Dyspnea
- Wheezing
- Tachycardia

**Anaphylaxis**

- Skin
  - Intense pruritus
  - Conjunctivitis
—Rhinitis
—Piloerection

- Gastrointestinal and genitourinary
  —Diarrhea
  —Nausea and vomiting
  —Incontinence

- Respiratory
  —Dyspnea
  —Cyanosis
  —Wheezeing
  —Substernal tightness

- Cardiac
  —Palpitations
  —Tachycardia
  —Hypotension
  —Dysrhythmias
  —Cardiac arrest

Management

**Allergy**
- Assess ABCs.
- Administer diphenhydramine 50 mg IM.
- Continue diphenhydramine 50 mg orally every 4 hours for 2 days.

**Anaphylaxis/Immediate allergic reactions**
- Assess ABCs. Perform BCLS as indicated (see CPR section, page 155).
- Administer epinephrine 0.3 mg (0.3 mL of 1:1,000 solution) subcutaneously or IM; may repeat this in 3 to 5 minutes, if
necessary.

- Administer 100% oxygen; intubate patient if necessary.
- When clinical improvement is noted, consider diphenhydramine 25 to 50 mg IV or IM and hydrocortisone 100 mg IV or IM to help prevent a recurrence of symptoms.
- In case of cardiac arrest, define specific rhythm and treat using appropriate ACLS protocol (see “Specific Protocols,” page 163).
- Transport patient to an appropriate acute care facility.

Prevention

- Ascertain medical history and family history of allergy.
- Consider skin testing, as necessary, before using questionable drugs.

Narcotic Overdose

Etiology: A dose of a narcotic resulting in an absolute or relative blood level that produces adverse clinical reactions. This dose will vary depending on a multitude of factors, including body weight, age, sex, genetic predisposition, presence of pathology, and route and rate of administration. The main effect is based on the direct depressant effect of narcotics on the medullary respiratory center.

Recognition

- Oversedation or unconsciousness
- Bradypnea
- Decreased tidal volume
- Hypercarbia
- Hypoxia
- Hypotension
- Respiratory arrest
- Cardiac arrest

Management

- Place patient in supine or astronaut position.
- Assess ABCs; administer assisted ventilation, controlled ventilation, or
CPR as indicated.
- Maintain patent airway; intubate if necessary.
- Administer supplemental oxygen.
- Administer naloxone 0.2 to 0.4 IV. If IV route is not possible, IM is acceptable (although slower).
- If naloxone is given IV, consider an additional slower-acting 0.4-mg IM dose to prevent renarcotization (especially with longer-acting narcotics, eg, morphine).
- Monitor vital signs and observe for renarcotization.

Prevention
- Obtain history of adverse reaction, drug intolerance, or addiction.
- Titrate narcotics slowly.
- Carefully monitor respiratory parameters when using narcotics.

Acute Adrenal Insufficiency
Etiology: A depressed physiologic state caused by an inadequate serum level of adrenocortical steroids. When caused by primary adrenal disease (Addison disease), the onset is usually slow and progressive. When secondary to adrenal suppression by exogenous steroid administration, stressful situations may increase the relative need and cause an acute insufficiency syndrome. Stress may be physiologic or psychologic.

Recognition
- Progressive confusion
- Fatigue
- Muscle weakness
- Nausea and vomiting
- Extracellular fluid depletion
- Hyperkalemia
- Syncope
- Hypotension and tachycardia
- Coma and death

Management
- Assess ABCs and administer BCLS as needed.
- Position patient in supine or astronaut position.
- Administer supplemental oxygen.
- Begin an IV infusion with D$_5$W and replace extracellular volume aggressively.
- Administer hydrocortisone sodium succinate 100 mg IV (or IM if IV is not possible).
- Administer additional steroid doses as needed.
- Examine electrolytes and correct accordingly (especially sodium and potassium).
- Administer a vasopressor as needed (eg, dopamine 1 to 4 µg/kg/min).

Prevention
- Check for medical history of Addison disease or exogenous steroid use.
- If a patient undergoing surgery has been on 20 mg of cortisol (or its equivalent) for a continuous period within the past 2 years, supplemental steroids should be administered.
- This will help prevent an Addisonian crisis due to lack of intrinsic cortisol production.
- Use appropriate sedation and pain control methods to decrease stress associated with the procedure.

Cerebrovascular Accident (CVA)

Etiology: A neurologic disease state caused by infarction and necrosis of brain tissue. May be caused by thrombosis, embolism, vascular spasm or vascular insufficiency, atherosclerosis, or intracranial hemorrhage. Patients with hypertension and diabetes are at an especially increased risk. Transient ischemic attacks (TIAs) are short episodes (seconds to minutes) of CVA symptoms, but no actual loss of brain tissue occurs.

Recognition: Signs and symptoms are associated with the specific area of the brain involved in the CVA and may have either a gradual or abrupt onset. These include:
- Headache (mild or severe)
- Nausea and vomiting
- Dizziness
- Sweating
- Chills
- Focal neurologic signs, including hemiparesis or paralysis, difficulty with speech (aphasia), incontinence, and unequal pupillary size (anisocoria)
- Hypertension
- Loss of consciousness

Management: Treatment for TIA and CVA is essentially symptomatic.
- Assess ABCs and initiate BCLS as warranted.
- Position patient supine or, if hypertension is present, with head slightly elevated (as long as a patent airway can be maintained).
- Administer supplemental oxygen if unconsciousness or respiratory distress is present. (High oxygen concentrations may, however, cause unwanted cerebral vasoconstriction.)
- If symptoms remain more than a few minutes, the patient should be transported to an acute care facility.

Prevention
- Obtain any previous history of TIA, CVA, or cerebral ischemia.
- Avoid central nervous system depressants in high-risk patients because these may lead to hypoxia.
- Avoid hypotension or hypertension.
- Minimize stress by using appropriate pain control and sedation techniques.
chapter 12 Management of Postoperative Medical Problems

There are numerous problems that may be a consequence of an operative intervention. The key is to identify the issue in an expedient manner and treat it accordingly, if necessary. This chapter addresses common postoperative problems, including: fever, hemorrhage, cardiac and renal complications, nausea and vomiting, wound care, and pain.

Fever
The normal human body temperature is 98.6°F (37.0°C). A low-grade fever is a common sequel to most surgical procedures as a result of an inflammatory stimulus. However, it is usually not significant if under 100.5°F (38.2°C).

Fevers greater than 101.5°F (38.6°C) usually demand evaluation.

Broadly speaking, fevers within the first 24 hours after surgery are commonly due to:
- Pulmonary atelectasis
- Aspiration pneumonitis
- Response to surgery itself, likely due to cytokine release in response to stressful stimuli (interleukin 1, tumor necrosis factor α, interferon-γ)
- Anesthetic medications used perioperatively
- Immune-mediated response to transfused blood products

Fevers occurring between 24 and 72 hours postoperatively are most likely caused by atelectasis (secondary to a poor respiratory effort), urinary tract infection (especially in the elderly and the female population), bacterial pneumonia or evolution of aspiration pneumonitis, or thrombophlebitis. Deep venous thrombosis (DVT) must always be considered, especially in the population with malignancies of any type.
After 72 hours, the principal causes include:
- Pneumonia
- Pulmonary embolism
- Intravenous (IV) catheter infection
- Wound infection
- Urinary tract infection

Other fevers during this period can be due to drugs or, occasionally, malignancies.

Principal Causes of Fever
A simple mnemonic to remind one of the principal causes of postoperative fever is the 5 Ws:
- Wound (surgical site or intravenous catheter site)
- Wind (pneumonia)
- Water (urinary tract)
- Walking (DVT and pulmonary embolism)
- Wonder drugs (variety of medications used postoperatively)

Wound
Tissue that has been traumatized in any manner surgically can be at risk for infection and resultant fever. The skin provides a barrier to bacterial penetration, but once it has been incised the patient is at risk for contamination from the environment. Preoperative preparation is paramount to reducing the risk of infection, using careful surgical debridement and copious lavage. In a previously clean or clean-contaminated wound it often takes at least 72 hours for a rising temperature to be attributable to an infection. Traditional signs of infection (swelling, erythema, pain, purulence) can variably evolve based on wound type and location.

IV site
- Peripheral and central catheters each have their own risk for becoming infected and causing a fever. Any indwelling catheter of greater than 24-hour duration is at risk for becoming infected and, as such, sterile technique for
placement is imperative (especially in the case of central catheters of any type). The peripherally inserted central catheter (PICC) is becoming more commonly used at many hospitals because of its central access through the periphery and lower rate of infection, but it must be suspected for colonization or infection at any time there is persistent postoperative fever. Although most central catheters can remain in place for a substantial period, all peripheral IV lines should be moved to a new site every 72 hours. Peripheral IV lines have a significantly increased risk of infection.

- Pain, tenderness, swelling, erythema, and streaking on the limb should alert the observer to the possibility of an IV site infection or phlebitis.
- Treatment generally consists of removing the offending IV line, elevating the limb, and applying warm, moist packs.
- Antibiotic therapy for line infections is initially empiric and depends on the severity of the clinical disease, patient risk factors, and likely pathogens. Vancomycin tends to be the first choice in hospitals with methicillin-resistant staphylococci due to its coverage of both coagulase-negative staphylococci and *Staphylococcus aureus*. In cases where methicillin-resistant *Staphylococcus* is not a general issue, nafcillin or oxacillin should be used. The use of additional agents for coverage of *Pseudomonas aeruginosa* (piperacillin/tazobactam, cefepime, levofloxacin) and *Candida* (fluconazole) may be indicated for the severely ill or immunocompromised patient who has a suspected catheter-related bloodstream infection.
- Antibiotic therapy should be continued pending the results of blood cultures; if they are positive, consultation with the infectious disease specialist should be prompt due to the potentially serious complications (such as infective endocarditis) that can follow an infection from an IV site.

**Breakdown in aseptic technique:**

- Most wound infections become apparent between
postoperative days 3 and 7 and are frequently ascribed to a breakdown in aseptic technique.

- Erythema, tenderness, crepitation, or discharge all point to wound infection, with a need for Gram staining and cultures in a sterile fashion and potential opening of the operative incision.

- Until the results of the cultures and antibiotic sensitivity tests are known, empiric broad spectrum coverage is warranted, most often with β-lactam antibiotics with or without a β-lactamase inhibitor. Penicillin, 1 to 2 million U IV twice a day, or ampicillin/sulbactam, 3 g IV every 6 hours, are common treatments of choice. Penicillins are effective against most oral flora, have extraordinarily low morbidity, and will not harm pregnant patients. Clindamycin, 600 mg IV every 6 hours, and vancomycin dosed based on renal function are frequently used for patients with allergies to β-lactam antibiotics. In the immunologically compromised patient, initial therapy usually includes broader spectrum antibiotics such as piperacillin/tazobactam or imipenem or double coverage based on hospital practices. This should be continued until laboratory reports make it possible to narrow the antibiotic coverage to one that is more specific.

**Identifying fluid accumulations:**

- The presence of pus mandates opening the wound.
- Computed tomography (CT) scans with IV contrast may be particularly helpful in identifying fluid or pus accumulations that may threaten the airway, yet are not visible or palpable.
- At times, the antibiotics and antipyretic compounds prescribed may mask the fulminating nature of an infection. If the patient has swelling in the head and neck region, has a modest rise in temperature, and is becoming either dyspneic or dysphagic, the CT scan is of inestimable aid in determining whether there are undrained accumulations of pus. However, it is important to remember that due to the normal postoperative edema and swelling, the CT findings may at times mimic an infectious process. Nevertheless, in
the patient with a fever, a high index of suspicion for infection and early operative intervention is warranted.

- The role of fever in combating infection has only recently been recognized, and prescribing of antipyretic drugs for fevers of less than 101.5°F is likely not advantageous.

Respiratory complications (Wind)

Respiratory complications cause 25% of all postoperative deaths. The most frequent respiratory complications following oral and maxillofacial surgery are pulmonary atelectasis, aspiration pneumonitis, pneumonia, and pulmonary embolus. Each of these can give rise to fever.

*Pulmonary atelectasis*: Imperfect expansion of the lung can occur in a small area of alveoli, or it can involve larger portions of lung segments especially at the bases. It may even entail complete collapse of a lobe of the lung. Patients with a history of smoking are at a higher risk of atelectasis and have more severe complications from atelectasis.

- Fever associated with atelectasis usually begins within the first 48 hours of surgery.
- Atelectasis following both deep sedation and general anesthesia may be attributed to various factors, including:
  - Use of cuffed endotracheal tubes
  - Depressed mucosalivary clearance due to the drying effect of the inhaled gases
  - Long periods of preoperative fasting, which can lead to dehydration
  - Prolonged anesthesia
  - Depression of respiratory effort and cough reflex by pain and narcotic analgesics

- As a result of these various factors, mucus is retained in the airways, obstruction occurs in the distal bronchial segments, and the affected segments collapse when the air is absorbed.
The invasion of these mucus plugs by bacteria that normally reside in the upper airway can lead to lung infection.

- The early clinical features of atelectasis vary according to the extent of the lung affected and are made more difficult to detect because radiography will reveal only large areas of hypoaerated lung. Clinical features may be masked until an infection occurs.
- Signs usually appear 1 to 3 days postoperatively and include fever, tachypnea, and dyspnea, as well as leukocytosis.
- Auscultatory findings are usually minimal.
- If atelectasis is suspected, and the symptoms are not severe, physiotherapy may be all that is necessary. Currently, deep breathing exercises and ambulation are thought to be the best additional ways of preventing and treating this condition. The use of mist, tents, ultrasonic nebulizers, incentive spirometers, and intermittent positive pressure breathing have been variably documented to decrease complications, but have not been studied in rigorous randomized prospective trials.
- More serious symptoms, including spiking fever and dyspnea, should be evaluated with a chest radiograph to exclude pneumonia or major segmental collapse. Pneumonia requires antibiotic therapy, and major segmental collapse may require bronchoscopic evaluation.

**Aspiration pneumonitis:** Depression of the cough reflex following general anesthesia or during sedation increases the risk of aspiration pneumonitis.

- Inhalation of foreign material into the lungs is a particular problem in patients who are in maxillomandibular fixation.
- Postoperative pneumonitis frequently manifests itself in the right lower lobe and can sometimes progress to fulminant pneumonia. An indolent fever can occur as early as 2 to 5 days or as late as 2 to 3 weeks after surgery.
- The patient presents with malaise, cough, production of sputum, and pleuritic pain. If aspiration pneumonitis is suspected, a chest radiograph should be ordered. Findings
can range from nothing to a very focal infiltrate in the right lower lobe. No treatment is necessary initially because pneumonitis is an inflammatory process; the patient should be observed. If progression occurs, then antibiotics should be started, especially in cases of a developing productive cough and leukocytosis. The patient’s white blood cell counts can be followed and appropriate coverage for hospital-acquired pneumonia should ensue.

_Pulmonary embolus:_ This refers to a blood clot lodged in the pulmonary artery or any segmental branch. Commonly the clots are formed peripherally, break free from the formation site, and become trapped in the pulmonary vascular circulation.

- Although most pulmonary emboli originate from thrombi in the deep venous systems of the legs, only 30% to 50% of general surgery patients with documented DVTs have clinical signs.
- In patients older than 40 years, 1% to 2% will suffer from a postoperative pulmonary embolus without necessarily exhibiting clinical features. In oral and maxillofacial surgery, this is generally not a concern because the patients are usually ambulating early in the postoperative period.
- Generally, 3 to 10 days of bed rest may precede the development of DVT. The chief causes, sometimes referred to as the Virchow triad, are:
  - Damage to the endothelial lining of the vessel
  - Stasis or diminution in the rate of flow in the vein
  - A change in the blood constituents attributable to a postoperative increase in the number and adhesiveness of the platelets

- At one time it was thought that patients on oral contraceptives had a higher incidence of DVT; with the lower levels of progesterone now used in such drugs, it appears that this slightly higher incidence has diminished.
- Preoperative use of compression hose, sequential compression devices, and subcutaneous heparin with
continued use postoperatively may help prevent DVT and subsequent pulmonary embolism.

- Clinical evidence of DVT, such as lower extremity swelling and tenderness, may not be found on examination, even after embolism is documented.
- Homans sign (pain in the calf with passive dorsiflexion of the foot), once regarded as pathognomonic for the presence of DVT, is no longer advised because there are concerns such maneuvers may increase the risk of embolization of a venous clot.
- The clinical features of pulmonary embolism include:
  - Fever
  - Chest pain
  - Sudden dyspnea
  - Tachypnea

- Hemoptysis is uncommon and is indicative of possible pulmonary infarction.
- If a pulmonary embolus is a possibility, duplex scans of the legs can increase suspicion. Confirmation can be sought by ventilation/perfusion scans, spiral CT, and the gold standard — angiography. However, with improved scanning techniques, angiograms are now less likely to be performed.
- Treatment usually consists of limb elevation and systemic anticoagulation with heparin. Subsequently, chronic therapy may include low-molecular-weight heparin, but more commonly warfarin is used because of the lower cost and convenience of oral therapy. Thrombolytic therapy should be avoided in the postoperative period, as severe bleeding may occur. Continuous anticoagulation, however, can usually be undertaken safely, even in patients with recent surgery.

Urinary tract infection (Water)

In hospitalized patients, urinary tract infection is frequently caused by an indwelling catheter or through intermittent catheterization when urinary
retention is present. Infection is uncommon in the absence of indwelling catheters or instrumentation of the genitourinary tract. Catheters should be avoided unless absolutely necessary.

- Women are at greater risk than men because of the short urethra. In the general population, about 10% to 15% of women are said to have asymptomatic urinary tract infections, but only half of these women ever seek medical attention. Many have asymptomatic bacteriuria in which there are more than $10^5$ colony forming units per cubic milliliter of urine.
- The stress of surgery may unmask an asymptomatic bacteriuria and allow a symptomatic urinary tract infection to develop.
- Fever from urinary tract infection often occurs 72 hours or more postoperatively, but can occur sooner if a subclinical infection had been present prior to surgery. Other symptoms include:
  - Dysuria (painful or difficult urination. This includes burning on urination)
  - Increased urinary frequency
  - Cloudy urine
- Urinanalysis may reveal elevated leukocyte esterase, positive nitrites, and even white blood cells. Gram stain may reveal the organisms of concern. Based on the results of these studies, antibiotics may be indicated and are often administered orally.

DVT (Walking)

The word walking serves as a reminder that a lower limb can be the source of the fever due to thrombosis occurring there (see the discussion of pulmonary embolus in the section on respiratory complications, page 185).

Drugs and transfusions (Wonder drugs)

Many drugs (eg, antibiotics, quinidine, methyldopa) have been implicated in febrile reactions. However, bacterial etiology should be considered before the fever is attributed to medication.

- The presence of an eosinophilia, the absence of a prominent leukocytosis, and a general lack of other systemic symptoms may suggest the diagnosis.
• It has been said that a fever secondary to a drug reaction is not accompanied by an increase in heart rate. Usually, for every 1°C rise in body temperature, there is an 8 to 10 beats-per-minute (BPM) increase in the heart rate.
• Treatment consists of discontinuing the offending drug.

Transfusions: Transfusions are a common source of fever (see chapter 8). Occasionally, the febrile reaction is mild and does not require treatment. If, however, the fever occurs in conjunction with tachycardia, chills, back pain, dyspnea, or microvascular bleeding, a major transfusion reaction should be assumed. The transfusion should be stopped and the patient’s blood crossmatched again. Should significant hemolysis occur, the patient will also require a forced diuresis and alkalization of the urine to prevent renal toxicity.

Unusual Causes of Fever
Unusual causes of fever include alcohol withdrawal, hot beverage consumption, hyperthermia from a warm environment and surroundings, and factitious causes wherein the patient wishes to mislead the physician.

Hemorrhage
Postoperative bleeding can occur as a rebound effect of hypotensive anesthesia, in which case there may be some local swelling. Usually there is no cause for concern. Hemorrhage can also result from incompletely ligated or cauterized vessels. If the size of the vessel is large enough, this kind of postoperative bleeding can cause a very rapid swelling.

Treatment: Sometimes the bleeding site is obvious (eg, on an alveolar ridge or an extraction site), and it may be necessary only to insert additional sutures under local anesthesia. Hemostatic agents, such as microfibrillar collagen, oxidized cellulose, or topically sprayed thrombin (alone or mixed in a matrix or with fibrin), may be used to correct a troublesome local ooze.

There is no time for delays in the management of significant hemorrhage. Postoperative bleeding can lead to aspiration and airway compromise.
Despite the seeming inconvenience and disruption occasioned by a return to the operating room, this is often the wisest course of action. In a dental office setting, with the patient under local anesthesia, but conscious, and the lighting and suction less than ideal, it is often difficult for the surgeon to gain optimal access to the bleeding site. In the operating room, the surgeon can calmly and systematically explore the wound without the patient’s anxiety and other distractions interfering. These circumstances aid immeasurably in the discovery and ligation or cauterization of the bleeding vessel(s).

Infection: If there is postoperative bleeding several days after surgery, one should suspect wound infection. Normal clot lysis occurs at 7 to 10 days postoperatively, often after the patient has been discharged, and bleeding may also occur at that time.

Coagulopathy: The possibility that the hemorrhage is caused by a coagulopathy should be remote if a full history was taken and the patient is not on anticoagulants. However, unusual and rare coagulopathies are possible, and this must always be borne in mind.

**Cardiac Complications**

**Hypertension**

It is not unusual for patients to experience bouts of hypertension 30 to 90 minutes after surgery, while they are still in the recovery area.

**Causes**

- Increase in pain and anxiety as the anesthetic agents wane in their efficacy
- Overdistention of the bladder (this can cause a 30- to 40-mm Hg rise in systolic pressure)
- Hypoxia or hypercapnia

**Treatment**

If the bladder is emptied by catheterization or by the patient, a reduction in blood pressure should follow shortly. To determine whether the patient needs to urinate, simply palpate the bladder. An overdistended bladder can sometimes be difficult to empty, and catheterization may be required if the patient is unable to void.
Pain and anxiety can be relieved by suitable analgesics, but if the systolic blood pressure is persistently greater than 160 to 170 mm Hg, drugs such as nifedipine, labetalol, esmolol, or hydralazine may be used to lower the blood pressure. Patients managed this way require careful observation and may need to be evaluated for myocardial infarction and monitored more closely or even in an intensive care unit.

In a previously healthy patient, excess fluid therapy is unlikely to elevate the blood pressure more than a few millimeters. Redistribution of the fluid volume and suppression of antidiuretic hormone bring about urinary discharge fairly promptly. However, in the elderly patient who may have some degree of cardiac dysfunction, the administration of excess fluids and sodium may lead to pulmonary edema and congestive heart failure. Therefore, the fluid volume administered must be monitored very carefully, particularly in the elderly or in those with known cardiopulmonary dysfunction.

Hypotension

A hypotensive episode is one in which the blood pressure falls by 10% to 20% of the preoperative level.

Causes

- In the postoperative period, the most likely cause is an intravascular hypovolemia. The anesthetic record should be studied to determine whether fluids were adequately replaced during the procedure. It is not often appreciated that about 60 mL of fluid are routinely lost per hour:
  — Obligatory urine loss: 0.33 mL/min
  — Respiratory loss: 0.33 mL/min
  — Perspiration: 0.25 mL/min
  — Stool loss: 0.125 mL/min
  — Total: 1.035 mL/min, or 60 to 65 mL fluid loss/h

Thus, 8 to 12 hours of fluid abstinence before surgery can produce a very significant fluid deficit after surgery. This, combined with inadequate replacement and/or more extensive blood loss than was measured (possibly due to not weighing sponges or a postoperative ooze), can result in a postoperative hypotensive episode.
• In oral and maxillofacial surgery, extensive blood loss can occur in many surgical procedures. Replacement of blood may be delayed because of the desire to avoid transfusion, leading to hypovolemia. It should be noted that tachycardia is an early sign of hypovolemia and that hypotension is a late sign of hypovolemia resulting from more extensive intravascular losses. The hematocrit can help assess blood loss, but it is not absolutely reliable because there may be a large dilutional effect if there has been a considerable amount of crystalloid replacement. Moreover, because the hematocrit is stated as a percentage, acute blood loss without fluid replacement can result in a normal hematocrit in a hypovolemic state.
• If excessive blood loss is ruled out as a cause in the recovery room, consideration can be given to a rewarming vasodilatation. Myocardial depression from the effects of the anesthetic agents also must be suspected.
• Hypothyroidism can also be a cause; however, this is unusual.

Treatment
• Early treatment of a hypotensive episode can begin by simply elevating the legs. If this restores the blood pressure adequately, the patient should be monitored clinically over the next 24 hours, with assurance of proper fluid management. It may well be that the hypotensive episode can be attributed to the anesthetic agents. After 24 hours, the hematocrit will help determine actual blood loss, as most of the dilutional effects will have waned by that time.
• If elevating the legs is not helpful and the fall in blood pressure is 20% or more, aggressive fluid resuscitation should be considered based on the hematocrit and overall fluid status of the patient. Blood transfusion guidelines continue to evolve based on controlled studies from intensive care unit settings delineating previously underappreciated risks of transfusion, including postoperative infection and cardiac events. Triggers for transfusion should take into account the patient’s previous cardiac history, degree of symptoms associated with the acute anemic state, and the anticipated additional blood loss. Currently, new guidelines set the trigger for transfusion in an otherwise healthy patient without symptoms at hemoglobin of 7.0 g/dL or hematocrit of 21%.
Fluid resuscitation, including the use of blood products in elderly patients, should be done with caution; it is contraindicated to administer large volumes of fluid, particularly if there is a degree of cardiac impairment, because pulmonary edema may ensue. Similarly, patients who have suffered extensive trauma or have large areas of septic tissue may suffer from “leaking capillaries,” allowing large volumes of fluid to escape into the “third space” without improving the blood pressure.

- For acute episodes of hypotension, a small dose (eg, 5 to 10 mg in a 70-kg person) of intravenous phenylephrine may be administered.
- If, despite the above measures, the patient remains hypotensive, more extensive and invasive procedures must be undertaken to determine whether a “pump failure” has occurred. If there is cardiac depression, intensive monitoring and therapy are obviously necessary.

**Arrhythmias**

- Ventricular premature beats may be precipitated by hypoxia, pain, or fluid overload.
- In the recovery area, where patients are carefully monitored, the administration of oxygen or analgesics and the correction of fluid and electrolyte abnormalities may be all that is needed to manage these arrhythmias.
- Lidocaine is the drug of choice for the treatment of frequent or worrisome premature ventricular contractions.
- Supraventricular arrhythmias are uncommon in patients who have not had heart disease prior to surgery. Should they occur, oxygen should be administered, an electrocardiogram should be performed, and both the electrolytes and cardiac enzymes (troponin I and creatine kinase MB [CK-MB]) should be checked.
- Measurement of arterial blood gases may be useful.
- Specific drug therapy may be required to treat the arrhythmia if hydration and adequate oxygenation fail to correct the situation or if the arrhythmia is causing any persistent instability.

**Myocardial Infarction**

- Myocardial infarction is rare in individuals younger than 40 years who lack a history of heart disease.
- The presenting symptoms that are so typical in nonsurgical patients may
be masked by sedatives and analgesic drugs in those who have undergone anesthesia and surgery.

- Chest pain remains the most important feature. It is usually described as a heavy, oppressive sensation. The pain may or may not radiate down the arm. It is frequently accompanied by nausea, vomiting, weakness, and, at times, a desire to defecate.
- A strong suspicion of the diagnosis can be based on the history and elevated cardiac-specific blood enzyme levels (CK-MB or troponin I). The electrocardiogram (ECG) may not be diagnostic, but in patients who have undergone oral and maxillofacial surgery any elevation of the cardiac enzyme levels can be attributed to a myocardial infarction.
- If the patient is suspected of having had a myocardial infarction, appropriate studies should be ordered and consultation with a primary care physician or cardiologist is indicated. Patients should then undergo standard treatment to reduce cardiac injury due to cardiac ischemia prior to undergoing more comprehensive therapy.
- A common mnemonic for the initial treatment of a patient with suspected myocardial infarction is MONA:
  — Morphine: Offered as a final agent to decrease chest pain and reduce anxiety, thereby minimizing oxygen demand and decreasing preload.
  — Oxygen (nasal cannula, facemask, or non-rebreather): To provide maximal oxygen delivery to minimize ischemia.
  — Nitrates: To allow cardiac vasodilation and thereby minimize pain and discomfort. Chest pain alleviation with nitrates portends a higher likelihood of cardiac origin.
  — Aspirin: To prevent further propagation of blood clot within cardiac vessels to minimize the zone of cardiac injury.

Renal Complications
Normal bladder capacity: 350 to 500 mL. If the bladder is greatly distended (eg, more than 650 mL), this can inhibit the ability to micturate.

Low urine output: Low urine output is quite common postoperatively and needs to be evaluated due to significant risk of morbidity if ignored. A minimum of 0.5 mL/kg/h for adults and 1 mL/kg/h for children is produced.
with properly functioning kidneys. The three major classifications of low urine output causes are prerenal, renal, and postrenal.

**Prerenal causes:** By far the most common causes of low urine output postoperatively are prerenal. Hypovolemia related to large blood loss intraoperatively can cause shocklike symptoms such as tachycardia, hypotension, and low urine output. Appropriate fluid replacement is required to minimize the possibility of permanent renal damage. In addition, preoperative abstinence from fluids must be considered because patients frequently take nothing by mouth for more than 8 hours and are dehydrated even prior to starting the operation. Rarely, low cardiac output, systemic vasodilation, and renal vasoconstriction (nonsteroidal anti-inflammatory drugs [NSAIDs], IV contrast dye, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, and immunomodulators [cyclosporine or tacrolimus]) can be causative factors.

- **Evaluation:** Urine output may be low for a variety of reasons, but invariably urine is not being produced because of prerenal or renal causes. A fractional excretion of sodium (FENa) analysis can be helpful in distinguishing between prerenal and renal causes:

  ![Urine sodium × Plasma creatinine](image)
  ![Urine creatinine × Plasma sodium](image)

  A FENa below 0.01 (1%) is indicative of a prerenal cause. In addition, the patient’s blood urea nitrogen (BUN) may be significantly elevated compared to the creatinine, with a ratio of greater than 20:1.

- **Treatment:** Preoperative and intraoperative fluid replacement should reflect both maintenance IV fluid requirements (4:2:1 rule: 4 mL/kg for first 10 kg, 2mL/kg for second 10 kg, 1 mL/kg for every kg above 20 kg) and blood loss estimates from the surgery (3 mL crystalloid for every 1 mL lost, 1 mL colloid or blood product for every 1 mL lost).
Renal causes: Acute tubular necrosis (ATN), which is characterized by renal tubular cell death and sloughing, may have a variety of causes. Medications such as the aminoglycosides, amphotericin, and IV contrast dye provided perioperatively (toxic ATN), as well as intrinsic damage to the kidneys from severe progressive prerenal disease (ischemic ATN), can cause low postoperative urine output.

- Evaluation: FENa analysis is used to distinguish renal from prerenal causes (value greater than 0.03 [3%] indicates renal cause). Urinalysis demonstrates pigmented, granular “muddy brown” casts.
- Treatment: Removal of the offending agent with toxic ATN or ensuring adequate rehydration for ischemic ATN are the primary treatments. Avoiding further renal injury is the key, with use of plenty of maintenance IV fluids and bolus fluids as necessary, until the patient is tolerating adequate oral intake.

Postrenal causes: Any portion of the genitourinary system outside of the kidneys can be responsible for low urine output. The general anesthetic can depress the micturitic reflex, and a bladder can distend significantly without allowing for appropriate bladder neck relaxation. Many patients find themselves unable to pass urine while lying in bed; this “positional inhibition” is surprisingly common and is difficult to overcome. Men older than 55 years suffer a higher risk of postrenal low urine output due to a high likelihood of prostate hypertrophy or prostate cancer. Baseline neurogenic bladder is common in multiparous women. Use of anticholinergic medications perioperatively can also decrease bladder contractility and function, causing an inability to urinate postoperatively.

- Evaluation: Noninvasive bladder imaging will often distinguish postrenal causes from prerenal/renal causes by identifying a full and possibly distended bladder. If any question exists, straight catheterization or Foley catheter placement will identify this as a source and effectively treat it.
Treatment: If the patient complains of suprapubic abdominal pain, or if on palpation there is an obviously full bladder, the patient is conscious, and 4 to 6 hours have passed since surgery, the patient can be assisted to micturate. Simple standing or attempted ambulation can provide the patient with a sense to void. If this fails, intermittent straight catheterization every 6 hours, or more frequently if indicated, can bypass the cause, thereby minimizing potential complications. On occasion, an enlarged prostate can be difficult to bypass reliably and Foley catheter placement will be necessary to avoid the swelling caused by frequent catheterization. Tamsulosin is the most commonly used postoperative medication for treating benign prostatic hypertrophy due to the quick onset of action and low side effects. In any scenario, a Foley catheter may need to be maintained throughout the early postoperative period. If the patient is unable to successfully void once the catheter is removed, replacement may be considered, although straight catheterization is usually all that is required. Rare circumstances may warrant urologic consultation and evaluation.

Nausea and Vomiting (see also page 219)

Causes

- Hypotension
- Hypoxia
- Drugs: Medications that excite the chemoreceptor trigger zone (CTZ), the emesis center in the medulla. Narcotics are the principal drugs stimulating the CTZ, and they may cause further problems by decreasing gastrointestinal motility.

Occurrence: More frequent in children than adults, and more common in women than men. Obese individuals are also more likely to become nauseated and vomit, as are those who suffer from motion sickness. For all
patients, the longer the duration of the surgery, the greater likelihood that there will be postoperative nausea and vomiting.

Treatment:

- To control postanesthetic nausea, anesthesiologists use droperidol or metoclopramide. More recently, promethazine and ondansetron, frequently combined with dexamethasone, have been used for preoperative prevention with promising results. Generally these drugs are administered about 20 minutes before the end of surgery or very soon after the patient is brought to the recovery room. The drugs act to depress the CTZ (eg, droperidol), relax the gastric sphincter and increase gastric mobility (eg, metoclopramide), or acts as a serotonin antagonist centrally and peripherally in the gastrointestinal tract (ondansetron). Subsequent doses may be necessary to cover the period of nausea, which in some patients can continue for up to 2 days.
- Compazine (prochlorperazine) and similar drugs, though they no longer enjoy the popularity they once had, may still be used to treat patients with mild postanesthetic nausea.

Wound Care

Intraoral wounds: Either warm water or saline rinses should be used after meals to help remove food debris clinging to the sutures.

Extraoral wounds

- Pressure dressings help minimize swelling and reduce capillary oozing.
- Impregnating the dressing with an antiseptic or antibiotic is a strategy frequently used to minimize bacterial growth on the skin.
- Many clinicians recommend that the wound not be exposed to sunlight during the very early postoperative period to prevent hyperpigmentation of the scar. Patients should always be encouraged to wash the wound with gentle soap and water to minimize bacterial load and subsequent infection.

Vacuum drains: If these have been inserted, the quantity of accumulated blood should be measured and, as soon as the volume is less than 10 to 15 mL for an 8-hour period and less than 30 mL for over 24 hours, the drains should be removed. Drainage usually decreases with each passing day.
Penrose drains used for infections can slowly be advanced and shortened at times when the infection is appearing to resolve. Advancement can be carried out 1 cm to 1 inch every 24 to 48 hours based on the resolution of the infection and length of the drain.

**Pain**
Some postoperative discomfort generally can be expected; one should endeavor to determine the cause and site of the pain.

**Causes**
- Sore throat from a cuffed tube
- Head pain from dressings applied too tightly over the ears
- Improperly positioned wires that impale the lips or oral mucosa
- Patient’s low pain threshold and tolerance to pain medications, especially with those on chronic pain management.

**Treatment:** If the pain is severe, morphine or hydromorphone IV is often effective; however, it should not have to be repeated more than two or three times for most outpatient or 23-hour observation oral and maxillofacial procedures. Some surgeons inject bupivacaine into the surgical site postoperatively to aid in minimizing pain. Transition to oral pain medications helps the patient tolerate the pain better over time because of their absorption rate and metabolism profiles.
Although complications from dentoalveolar surgery and the associated administration of local anesthesia are not common, when such complications do occur they can cause considerable morbidity. Most complications can be avoided by careful surgical planning, proper instrumentation, and attention to detail and surgical technique. However, when complications do occur they must be recognized and treated early.

Complications Associated with Local Anesthesia

Toxic (Overdose) Reactions

- Toxic reactions can arise from either the local anesthetic or the vasoconstrictor in the solution. Toxicity results when too-high levels of these agents are introduced rapidly into the systemic circulation. Toxic blood levels are most often caused by an intravascular injection or by administering too large a dose of the anesthetic in a susceptible patient. Although local anesthetics possess a wide margin of safety and tolerance, debilitated and pediatric patients remain vulnerable to overdose. The dose should always be predicated on the patient’s weight and physical status.

*Suggested maximum doses for healthy adults*

- Articaine 4% (with vasoconstrictor): 3.2 mg/lb; 500 mg maximum
- Lidocaine 2% (no vasoconstrictor): 2 mg/lb; 300 mg
- Lidocaine 2% (with vasoconstrictor): 3.2 mg/lb; 500 mg maximum
- Mepivacaine 3% (no vasoconstrictor): 3 mg/lb; 400 mg maximum
- Mepivacaine 2% (with vasoconstrictor): 3 mg/lb; 400 mg maximum
- Prilocaine 4% (with or without vasoconstrictor): 3.6 mg/lb; 600 mg maximum
- Bupivacaine 0.5% (with vasoconstrictor): 0.9 mg/lb; 90 mg maximum
- Etidocaine 1.5% (with vasoconstrictor): 3.6 mg/lb; 400 mg maximum

Note: Clark’s Rule can be used to calculate dosage for children:

Child dose = child’s wt/150 lb × adult dose

- Intravascular injection is most often associated with the inferior alveolar nerve block, especially when the Gow-Gates technique is used. However, intravascular injection into the internal maxillary artery and pterygoid plexus of veins is also possible.
- A retrograde flow of the local anesthetic into the cerebral circulation has been postulated as occurring after an intravascular injection during inferior alveolar nerve block. Aspiration in at least two planes is recommended to minimize the possibility of intravascular injection.

Signs and symptoms of local anesthetic overdose (toxicity)

- **Neurologic signs and symptoms**: Mental confusion, headache, drowsiness, dizziness, visual and auditory disturbances, disorientation, anxiety, talkativeness, muscular twitch, tremors, nystagmus, loss of consciousness, and tonic-clonic seizures.
- **Cardiovascular signs and symptoms**: There is an initial stimulation followed by depression of the central nervous system (CNS). There may
be a transient rise in blood pressure, pulse rate, and respiratory rate followed by a fall in these parameters.

- Local anesthetic agents depress the myocardium, but only at levels higher than those required to cause CNS symptoms. Caution is advised, however, when administering the more potent longer-acting anesthetic agents such as bupivacaine, which have been shown to be potentially more cardiotoxic than the other local anesthetics.

Signs and symptoms of toxic reaction to vasoconstrictor (epinephrine)

- Fear, anxiety, restlessness, throbbing headache, weakness, palpitations, respiratory difficulty, marked increase in pulse rate and blood pressure.
- Because of the short half-life of epinephrine (1 to 3 minutes), systemic reactions are short-lived and self-limiting. Some patients, however, are sensitive even to the effects of nontoxic doses of exogenous epinephrine and will subsequently experience a more heightened response to toxic levels. In addition, patients with cardiovascular disease may be particularly vulnerable to the effects of increased blood pressure and heart rate.

Treatment of overdose reactions

- It may be difficult to determine if a reaction is due to the local anesthetic or the vasoconstrictor.
- In general, the management of overdose reactions to either the local anesthetic or vasoconstrictor is the same. Supportive treatment in the form of reassurance to the patient, administration of oxygen, and monitoring of the vital signs is indicated.
- Persistent seizure activity may require the administration of parenteral diazepam.
- Close observation and monitoring of the patient is required in the event that basic and advanced life support become necessary (see chapter 11).

Allergic Reactions

- True immunoglobulin E (IgE) antibody–mediated allergic reactions to amide-type local anesthetic agents have been reported, but are exceedingly rare. Careful questioning of the patient with alleged allergy to a local anesthetic about the signs and symptoms that occur is necessary.
Many patients will equate fainting or symptoms of anxiety with allergy. Any report of rash, urticaria, pruritus, or respiratory symptoms indicates a possible true allergic response. Allergy testing is then indicated. Testing should be done in a controlled environment by those capable of managing possible reactions. Only those local anesthetic agents intended for clinical use should be tested.

Patients may be allergic to preservatives such as sodium bisulfite and methylparaben used in certain local anesthetic preparations. Methylparaben is still used as a preservative in multiple-dose vials of local anesthetics. Sodium bisulfite is used as a preservative for epinephrine.

Treatment of allergic reactions
- Immediate reactions (occurring within 1 hour of local anesthetic administration) should be managed aggressively. Epinephrine 0.3 mL (0.15 mL child) 1:1,000 intramuscularly (IM) or 1 mL 1:10,000 intravenously (IV) should be administered. An antihistamine (diphenhydramine 50 mg IM) is also indicated, along with the administration of supplemental oxygen. The patient should be observed closely. An anaphylactic reaction is possible and should be managed accordingly (see chapter 11).
- Delayed reactions are treated with oral diphenhydramine 50 mg every 6 hours for at least 3 to 4 days.

Local Complications Resulting from Needle Insertion

Hematoma
- Needle injury to a blood vessel will cause leakage of blood into the tissues with resultant swelling. Rapid swelling usually results from arterial leakage, whereas a slower onset indicates venous involvement.
- Hematoma formation is most often associated with the posterior-superior alveolar and inferior alveolar nerve block techniques.
- Hematomas are usually self-limiting and treated conservatively; however, some hematomas can be quite large and frightening to the patient. Patient reassurance, along with the immediate application of cold (to minimize the swelling) and heat (after 24 hours, to help resolve the condition), is the indicated treatment. Patients with hematomas in the pterygomandibular space should be observed to ensure that there is no
airway compromise. Antibiotics should be prescribed to prevent secondary infection of the hematoma. Complete resolution may take 1 week or longer.

Prolonged anesthesia
- Mechanical injury at the time of needle insertion can cause an altered nerve sensation for varying lengths of time.
- Direct injury to the neural sheath, or hematoma formation in the neural sheath resulting in nerve ischemia, can cause prolonged nerve anesthesia.
- Injection of contaminated anesthetic solutions will result in nerve injury. Cartridges stored in alcohol are at risk of leakage of the alcohol into the cartridge. Cartridges should not be stored in any solution.
- It has been postulated that certain patients can enzymatically hydrolyze both ester and amide local anesthetics into an alcohol. If this should occur, the alcohol concentrated near the nerve could have the same effect as an alcohol block in terms of nerve injury.
- Treatment of prolonged anesthesia is based on its severity and duration. Most cases will resolve spontaneously (see chapter 22). Careful documentation of symptoms, area of distribution, and degree of resolution over time is recommended. Surgical intervention should be considered only in cases where symptoms persist longer than 6 months.

Trismus
- Postinjection trismus will result from trauma to muscles and blood vessels in the pterygomandibular space and infratemporal fossa. Muscle trauma and inflammation can result either directly from needle injury or indirectly from hematoma formation.
- High concentrations of vasoconstrictor resulting from multiple injections can also cause tissue necrosis and trismus.
- Postinjection trismus is treated with nonsteroidal anti-inflammatory drugs (NSAIDs), moist heat applications, and muscle relaxants, if necessary. Physiotherapy, including mouth opening and lateral excursive movement exercises, is also recommended.

Broken needles
- Although rarely encountered, needles can break under certain
conditions.

- Multiple bending of the needle can cause fatigue at the hub and breakage under pressure from the injection or sudden patient movement.
- Rarely, a manufacturing defect will result in needle breakage during injection.
- When giving deep injections, the needle should never be inserted to the hub. Always leave at least 5 mm of the shaft exposed during the injection. In the event of breakage, the segment can then be removed with a hemostat.
- Locating a broken needle in tissue can be very difficult and should be performed only after a careful three-dimensional radiographic analysis of its location. The incision and dissection to locate a needle should be in a plane perpendicular to the long axis of the needle.

Postinjection infection

- Sites of needle insertion can become infected, usually following hematoma formation or the introduction of bacteria into the tissues on the tip of the needle (ie, needle tract infection).
- Needles generally become contaminated from contact with the oral mucosa, although it is with the patient’s own oral flora. However, other organisms can also be introduced by inadvertent contamination from exogenous sources.
- Often postinjection infections are not preventable. The oral cavity cannot be sterilized, and swabbing the injection site with an antiseptic does not appear to be clinically effective and has not gained wide acceptance.
- When a diagnosis of postinjection infection is made, anaerobic bacteria should be considered as the causative organisms and appropriate antibiotics should be used.

Unanticipated nerve block

- Cases involving anesthesia of nerves far removed from the site of needle insertion have been reported.
- A needle placed too high during an inferior alveolar nerve block can cause the anesthetic to pass through the sigmoid notch into the parotid gland and anesthetize the facial nerve. The resulting facial muscle dysfunction will mimic Bell palsy.
• Injection of an anesthetic into the pterygoid fossa during a block of the maxillary division of the trigeminal nerve (V₂), or retrograde blood flow into the cavernous sinus from either arterial or venous penetration after a high inferior alveolar nerve block, can cause a clinical result similar to a superior orbital fissure syndrome. There will be paralysis of the extraocular muscles innervated by cranial nerves III, IV, and VI, along with proptosis of the eye. A temporary loss of vision (amaurosis) will also result should the optic nerve be involved. Permanent amaurosis following inferior alveolar nerve block has been reported.

• Cervical sympathetic nerve block following an intraoral local anesthetic injection for an inferior alveolar nerve block has been reported. It has been determined that the anesthetic solution enters the prevertebral space to ultimately affect the stellate ganglion. Clinically, Horner syndrome ensues, which includes ptosis of the upper lid (Müller muscle) and pupillary constriction (miosis) caused by the parasympathetic fibers of the oculomotor nerve (III) acting unopposed due to paralysis of the sympathetically innervated dilator muscle.

• No treatment is indicated for unanticipated nerve blocks because the symptoms resolve with metabolism of the anesthetic. In cases where the lid reflex is compromised, however, the patient should be given an eye patch to wear until normal function returns.

Soft Tissue Reactions

Ulceration and sloughing

• Ulceration and tissue sloughing can result from the injection of excessive amounts of anesthetic solution under a firmly adherent mucosa, such as that found on the palate, or from the use of an excessively high concentration of vasoconstrictor, especially noradrenaline.

• The resulting ischemia causes necrosis and sloughing of the overlying tissue.

• High concentrations of anesthetic often found in topically administered anesthetics, especially benzocaine, also can cause soft tissue irritation and sloughing.

Aphthous ulcers and herpetic lesions
• Occasionally aphthous ulcers or herpetic lesions will occur in the vicinity of the injection site. Herpetic lesions occur when a dormant herpetic virus in the nerve ganglion is activated after exposure of the nerve to an anesthetic injection. This is seen especially along the track of the greater palatine nerve.
• Palliative treatment of these lesions is recommended. Topical agents containing steroid should not be used when herpetic lesions are suspected because they may cause the lesions to spread.

Tissue blanching
• Blanching of the skin over an intraoral injection site or at distant sites can occur after the injection of a local anesthetic.
• Blanching of the skin over the injection site can be due to either the reflex contraction of blood vessels from needle stimulation or direct stimulation of sympathetic vasoconstrictor fibers that supply the skin.
• Blanching of the skin at sites distant from the injection is usually due to an intra-arterial injection causing reflex vasospasm.
• Blanching of the skin under the eye and of the posterior palatal mucosa has been reported after administering a local anesthetic with vasoconstrictor for an inferior alveolar nerve block using the Gow-Gates technique. It is suspected that the anesthetic enters the inferior alveolar artery, passes retrograde to the internal maxillary artery, and is then distributed to its terminal ophthalmic and greater palatine branches.
• No treatment is necessary for tissue blanching because it resolves when the vasoconstrictor is metabolized.

Complications of Dentoalveolar Surgery
Root Fracture
• The incidence of root fracture can be minimized by adequately elevating the tooth before attempting forceps delivery.
• Teeth with curved, dilacerated, or hypercementosed roots; endodontically treated teeth; teeth exposed to heavy occlusal forces; or teeth with total root diameters greater than the mesiodistal crown width are prone to root fracture.
• It is not necessary to regard all root fragments as foreign bodies. If large amounts of healthy bone must be sacrificed to gain access to the root
fragment, or it is close to areas such as the maxillary sinus or the inferior alveolar canal, root tips measuring 5 mm or less can very often be left in place provided there is no associated pathosis; use clinical judgment. Usually healing will be uneventful. The patient must receive a full explanation of your decision, and complete documentation in the patient’s record is advised.

Displaced Roots and Root Fragments

Submandibular space

- When mandibular molar root displacement into the submandibular space is suspected, immediately apply upward external pressure in the submandibular region medial to the mandible. This pressure will help prevent further displacement of the root. If the root cannot be visualized through the socket, reflect a lingual gingival flap that is carried as far anterior as the premolar region. The mylohyoid muscle should then be sharply detached from its insertion on the mandible. Careful dissection in this region should reveal the root or root fragment.
- Postoperative antibiotic therapy is indicated.

Mandibular canal

- Root tips thought to be displaced into the mandibular canal should be verified with a periapical and an occlusal radiograph; the root may actually be in a large marrow space or beneath the buccal mucosa.
- If the root can be visualized clinically, carefully remove the surrounding bone in order to grasp it with either a small hemostat or a Winter splinter forceps.
- Often the root tip is not clinically visible because of either its location or accompanying brisk bleeding. In either case, recovery of the root fragment is best performed as a secondary procedure.

Maxillary sinus

- Verify location of the root or root tip by a periapical radiograph.
- The palatal root of the first molar is the root most often displaced into the antrum.
- Frequently, the buccal root of a molar is actually lodged between the buccal plate and the periosteum. The root also may be between the intact antral membrane and alveolar bone.
• There may be an actual perforation of the sinus membrane, but the root is still attached to the alveolus by the apical periodontal fibers. Conservative enlargement of the socket may reveal the root fragment.
• Several local measures are useful to retrieve root tips from the sinus:
  —Have the patient blow through the nose with the nostrils closed. Carefully observe the perforation for the root to appear.
  —Use a fine-suction tip to bring the root back into the extraction defect.
  —Perform antral lavage with sterile isotonic saline in an effort to flush the root out through the defect.
  —If the opening is already large, pack iodoform gauze into the antrum and remove it in one stroke; often the root tip will adhere to the gauze.
• If local measures do not result in root retrieval, direct entry into the sinus is necessary. The Caldwell-Luc approach affords the best visualization.

Oral-Antral Communication
• Suspected small openings into the antrum need not be confirmed. Probing, irrigation, or having the patient blow with the nostrils occluded may enlarge an existing opening or create one where it did not previously exist. Such procedures may also cause infection.
• Gingival tissues should be approximated as closely as possible and sutured over the socket; removal of a small amount of buccal alveolar plate may facilitate the closure. Closure of large openings (5 mm or more) may require mobilization of tissue to create a buccal flap. Incising the periosteum on the underside of the flap is helpful in increasing the amount of such tissue available for closure.
• A piece of absorbable gelatin sponge may be placed in the occlusal third of the extraction site if further support of the underlying clot is necessary. This is not necessary in most cases, however, especially if primary or near primary closure is achieved.
• The patient should be warned not to forcefully blow the nose for at least 1 week and to keep the mouth open if sneezing becomes necessary.
• If there is an existing sinusitis, prescribe antibiotics and sinus decongestants.
Extraction of the Wrong Tooth

- This complication can be minimized by careful preoperative assessment, planning, and communication with the patient.
- Occurs most frequently when a patient is referred for extraction without specific instructions or a written order from the referring dentist.
- Patients in the mixed dentition stage often present a challenge in terms of identifying the tooth indicated for removal.
- Prior to applying an elevator or forceps, a “time-out,” as now practiced in hospital operating rooms, should be called to identify the tooth to be removed.
- If the error is immediately realized, the tooth should be reimplanted into the socket and stabilized.
- If the tooth was to be removed for orthodontic reasons, and the orthodontic treatment cannot be altered to accommodate for the wrong tooth removal, extraction of the correct tooth should be deferred until the outcome of the reimplantation can be determined, usually in 4 to 5 weeks.
- The patient, or the parent in the case of minors, and the referring dentist must be informed of the error.

Bleeding

Intraoperative (primary)

- The usual sources of troublesome intraoperative bleeding are granulation tissue, vessels in the periosteum, or nutrient arteries in bone, especially in the anterior mandible.
- Bleeding vessels in the periosteum are best identified under good lighting by adequate flap retraction and careful suctioning. Once identified, the bleeding vessel should be clamped with a small hemostat. Discreet use of cautery may be necessary.
- Burnishing or crushing the surrounding bone will generally control bleeding from nutrient vessels. If this fails, bone wax can be applied.
- Bleeding from granulation tissue will usually cause welling up of blood in the surgical field when the pressure packs are removed. Adequate curettage and debridement of the granulation tissue are necessary to control the bleeding.

Postoperative (secondary)
When the bleeding patient is initially seen, a physical evaluation should first be performed with close attention to signs of hypovolemic shock such as hypotension, pallor, diaphoresis, or a weak and rapid pulse. These conditions must be addressed because they indicate significant blood loss and a possible need for fluid replacement therapy.

- Identify the source of the postoperative bleeding before administering a local anesthetic with a vasoconstrictor.
- If the wound has been sutured initially, all previously placed sutures should be removed. Occasionally the bleeding will be coming from one of the suture points.
- Absorbable gelatin sponge, oxidized cellulose, and oxidized regenerated cellulose can all be used as hemostatic agents.
  - The gelatin sponge promotes platelet disruption and forms a framework for fibrin strands. It can be moistened with a thrombin solution. Gelatin sponge is absorbed in 4 to 6 weeks. It is useful in controlling capillary-type bleeding.
  - The hemostatic action of oxidized cellulose results from the formation of an artificial clot. Absorption of this material occurs between the second and seventh day, or may take longer, depending on the amount used, rate of degradation, and blood supply to the area.
  - The clinical uses for oxidized cellulose and oxidized regenerated cellulose are basically the same. The regenerated form is less friable and has less of a tendency to stick to instruments.
  - Because these agents have the potential to be toxic when in direct contact with nerves, they should be used with caution in cases where exposure of the inferior alveolar nerve is suspected.

Active sites of bleeding can occur in the bone or mucoperiosteal flap. Often these sites were not evident during initial surgery because a local anesthetic with a vasoconstricor was used. Remember that there can be rebound vasodilation following the use of a vasoconstrictor. For this reason, use of epinephrine-soaked packs to control bleeding is not recommended.

Ecchymosis/Hematoma
- Ecchymosis occurs with such frequency that it should probably not be
referred to as a complication. It is most commonly seen in elderly patients who have a naturally increased capillary fragility and diminished tissue elasticity.

- Extensive hematoma formation and ecchymosis usually result from poor hemostasis during or immediately following surgery. Intermittent ice packs applied during the first 24 hours, subsequently followed by intermittent moist heat, will help resolve the condition. Large hematomas can be evacuated with a large-bore needle. When this is done, it is advisable to administer antibiotics.

Localized Alveolar Osteitis (Dry Socket)

- Dry socket most often develops on the second to the fifth postoperative day.
- The chief clinical finding is pain. The patient may also complain of a fetid odor or a bad taste.
- Treatment is conservative. Under no circumstances should the extraction site be curetted or aggressively manipulated in an attempt to stimulate bleeding.
- Management should include gentle irrigation with warm saline followed by placement of a sedative dressing. Eugenol impregnated on a ¼-inch gauze strip can be used. Dressings should be changed every 24 to 48 hours, depending on the patient’s degree of discomfort. Unless there is overt suppuration or associated lymphadenopathy, antibiotics are not indicated.

A localized osteitis can occasionally progress to an osteomyelitis. If a patient returns after a second dressing change because of continued pain, a panoramic radiograph should be made to assess the extraction site for a root tip, foreign body, or sequestrum. It should be remembered that from 30% to 50% bone demineralization must take place before radiographic changes to identify osteomyelitis occur.

Wound Dehiscence

- Wound dehiscence occurs as a result of compromised blood supply to the mucosa.
- Tearing or overretraction of tissues, or too tightly suturing surgical flaps, can lead to wound dehiscence.
• Wound dehiscence should be allowed to heal by secondary intention. Do not attempt to resuture the wound.
• Minimally and carefully reduce sharp or rough edges of exposed underlying bone using a rongeur or bone file. If there is a large area of nonvital bone exposed, it is often advisable to wait until it completely separates from its base and can be lifted out with cotton pliers rather than to attempt to remove it surgically.

Trismus
• Postoperative trismus can be caused by infection, muscle spasm, or from injection of a local anesthetic (see discussion of trismus as a local reaction resulting from needle insertion, page 198). Trismus caused by infection is usually of slow onset and accompanied by other clinical signs and symptoms of infection such as swelling, fluctuance, and palatal draping in cases involving the pterygomandibular space.
• Trismus due to muscle spasm or an anesthetic injection is generally managed by range of motion exercises and assisted opening with tongue blades or the fingers. However, such exercises should not be used in patients with trismus caused by an infection until the infection has been resolved for fixation.

Tuberosity Fracture
• The maxillary tuberosity can fracture during removal of single, isolated maxillary molars. To avoid this when extracting several molars, remove the most distal tooth first.
• If the roots are bulbous, divergent, or dilacerated, consider prophylactic sectioning of the tooth. Expansion of the buccal plate using an elevator or osteotome can also be beneficial.
• Should the tuberosity fracture, terminate the procedure and, if possible, stabilize the tooth and/or completely take it out of occlusion. This approach is desirable when bone retention is necessary for preparing the arch prior to fabrication of a denture. Allow 4 to 6 weeks of healing before reattempting extraction. At that time, sectioning of the tooth is generally advised.
• If immediate removal of the tooth is necessary (ie, because of pain or infection), grasp the tooth with a forceps and attempt to separate it from the surrounding bone with an elevator. If extraction is successful,
stabilize the bone fragment by suturing the overlying mucosa or by first using intraosseous wires for fixation.

Puncture Wounds

- Puncture wounds most commonly involve the palate or tongue.
- Injury occurs due to uncontrolled force applied with a straight elevator or slippage of a periosteal elevator.
- Applied force with elevators is best controlled by firm finger rests, support of the mandible with the opposite hand, and placement of a protective retractor.
- Puncture wounds generally should not be sutured, but it may be advisable to prescribe an antibiotic.

Mandible Fracture

- Most often results from excessive force used in removing third molars.
- If resistance is met during elevation of an impacted tooth, the procedure should be stopped and determination made of the cause of the resistance. Often the distobuccal region at the crown height of contour has not been sufficiently exposed.
- Other causes include: weakening of the mandible due to bone loss from an associated cyst or tumor, a deeply impacted tooth, ie, roots reaching to the inferior border of the mandible, decreased mandibular height, mandibular atrophy, and excessive bone removal.
- Often there will be a distinctive “popping” sound when the mandible fractures.
- A panoramic radiograph should be made to verify if a fracture occurred.
- Once diagnosed, the fracture must be treated. Depending on the nature of the fracture and degree of displacement, the reduction and fixation can be performed immediately or as a secondary procedure, applying the same principles used to treat any other mandibular fracture.
- Fractured mandibles associated with third molar removal can also occur during the postoperative period in older, especially male, patients with an intact dentition. Because of the increased bone density and lack of bone elasticity in such patients, more bone has to be removed to accomplish removal of the impacted tooth. In the second or third week following otherwise uneventful surgery, when the patient resumes a regular diet, the weakened mandible can fracture under the stress of
chewing. Patients should be warned of this potential complication and the record documented accordingly.

Aspiration of Foreign Objects

- If a foreign body obstructs the patient’s airway, the procedures recommended in the basic cardiac life support protocol should be followed (see chapter 11).
- Cricothyrotomy is necessary if the object cannot be cleared and the patient develops acute distress and becomes cyanotic.
- If the case is not acute, the patient should be referred to the emergency room for evaluation. A chest radiograph will be done to confirm the presence of the foreign body in the lung. Bronchoscopy may be necessary to retrieve the object.
- Erupted maxillary third molars are particularly vulnerable to being swallowed or aspirated. A gauze screen placed lightly in the oropharynx should be used during all dentoalveolar surgical procedures to minimize the risk of swallowing or aspirating a foreign body.

Swallowed Foreign Objects

- A tooth or a tooth fragment are the most frequently swallowed foreign bodies. Erupted maxillary third molars are particularly vulnerable to being swallowed or aspirated.
- A gauze screen lightly placed in the oropharynx should be used during all dentoalveolar surgical procedures to minimize the risk of swallowing foreign bodies.
- If any foreign body is lost during a surgical procedure, documentation of its exact location is necessary. If the situation is not acute, the patient should be referred for a chest radiograph as well as abdominal views to locate the foreign object.
- If the foreign body is in the stomach, the patient is prescribed a bulky, high-roughage diet. If practical, radiographs should be made to follow the progress of the object through the gastrointestinal tract. Stools should be examined by the patient to confirm passage of the object. It will usually take 3 days to pass a foreign body.

Broken Instruments

- Stop the procedure as soon as it is recognized that an instrument has
broken. Without irrigating, carefully suction the operative site to determine the location of the fragment.

- Instrument fragments can migrate under a mucoperiosteal flap, be lodged in the cortical bone or cancellous bone spaces, be displaced into the maxillary sinus or mandibular canal, or be suctioned away during the procedure.
- If the foreign body is not visualized, a radiograph that includes the entire surgical site must be made. If possible, the radiograph should be obtained immediately.
- A reasonable attempt to remove the instrument fragment should be made. This is best done at the time of initial surgery.
- If removal is delayed, a separate informed consent must be obtained for the second procedure.
- Not all instrument fragments must be removed. The risks of surgery must be weighed against the benefit of removal. The decision to leave the fragment must be disclosed, discussed with the patient, and documented in the record.
- The fact that an instrument broke during the procedure does not mean that the surgeon was negligent.

**Temporomandibular Joint (TMJ) Pain**

- A thorough examination of the TMJ should be performed and any positive findings documented prior to any procedure involving removal of teeth.
- Postoperative TMJ pain is often caused by prolonged mouth opening, distraction of the joint on the side where mandibular surgery is performed, or pressure on the joint contralateral to the surgical side. Stabilizing the joint with a mouth prop and providing intraoperative mandibular support will help minimize postoperative joint discomfort.
- Postoperative joint pain is managed with moist heat applied to the affected side, a soft diet, resting the joint, and NSAIDs.
- If symptoms do not resolve, consider magnetic resonance imaging (MRI) to rule out intrinsic joint disease.

**Interstitial Emphysema**

- Air from a handpiece forced under pressure into connective tissue or fascial planes is referred to as interstitial emphysema.
- It generally presents as a sudden facial swelling, although a late onset can occur. Crepitation on palpation will distinguish emphysema from a hematoma. Emphysema in the superficial tissues is usually self-limiting and is treated conservatively with an antibiotic such as a cephalosporin to avoid secondary infection. Complete resolution may take several days.
- In some cases, however, air forced into the tissues can lead to serious morbidity and even mortality. Air forced into the venous system during dentoalveolar surgery (ie, through the pterygoid or intraosseous venous plexus of veins) can form an embolus that eventually reaches the heart, possibly resulting in cardiac arrest. Air forced into the tissue can continue to expand and dissect through the fascial planes and eventually track to the mediastinum. In such complicated cases, the patient must first be stabilized and then transferred to the emergency department for definitive and supportive treatment.
Diagnosis and Management of Sedative and Anesthetic Emergencies

The use of minimal, moderate, and deep sedation as well as the administration of general anesthesia is an integral part of the contemporary practice of oral and maxillofacial surgery. Any depression of the central nervous system with possible attendant respiratory and cardiovascular compromise can result in morbidity and mortality unless a meticulous preoperative evaluation, including a detailed health history and focused physical examination, is conducted (as described in chapter 1). Any medical emergency (eg, myocardial infarction, hypertension, glycemic issues, or allergic reaction) can manifest itself during the perioperative period either directly or indirectly because of the delivery of a sedative or anesthetic. This chapter focuses on those directly attributable to sedation/anesthesia for the oral surgery patient.

Patient Evaluation

A decision regarding the type of sedative or anesthetic and whether it is appropriate to perform treatment in the office environment versus the hospital depends upon:

- The patient’s coexisting medical conditions
- The extent of the surgery
- Anatomical and airway considerations

History

Elements of the history of special importance in sedative/anesthetic care include:

- Coexisting medical illnesses that may complicate the anesthetic course.
Medications. Of special importance are antihypertensive, antianginal, anticoagulant, anticonvulsant, antidysrhythmic as well as specific endocrine (eg, insulin) medications. As a general rule, drug therapy can be continued up to the time of surgery.

Allergies and other drug reactions.

Patient and family anesthetic history
  —Malignant hyperthermia
  —Atypical pseudocholinesterase
  —Past anesthetic experiences

Social history
  —Smoking habits
  —Drug and alcohol habits

A review of systems should be performed with particular attention to those associated with an increased risk of perioperative morbidity and mortality (eg, acute and chronic pulmonary disease, ischemic heart disease, diabetes, hypertension). Due to the teratogenicity and effects on uterine blood flow of many anesthetic agents, all women of childbearing age should be questioned as to the likelihood of current pregnancy. Routine laboratory screening for ambulatory procedures are rarely useful and, if indicated, should be selected based upon the patient’s medical condition. If necessary, appropriate consultations regarding specific issues of the planned procedures should be obtained.

Airway Evaluation

Physical examination is directed toward the cardiovascular and pulmonary systems and a thorough evaluation of the airway. Some factors in the history that may be associated with potential difficulty in airway management are:

- Previous problems during sedation or anesthesia
- Stridor, snoring, or sleep apnea
- Advanced rheumatoid arthritis
- Chromosomal abnormality (eg, trisomy 21) or other genetic disorders

Physical examination of the airway centers around potential airway issues and includes:
• Body habitus: Significant obesity (especially involving the neck and facial structures)
• Head and neck: Short neck, limited neck extension and flexion, decreased thyromental distance (< 3 cm in adult), neck masses, cervical arthritis or spine disease, trauma, tracheal deviation, dysmorphic facial features, infection
• Mouth: Small opening (< 3 cm in adult), protruding maxillary incisors, high arched palate, macroglossia, tonsilar hypertrophy
• Mandible: Micrognathia, retrognathia, trismus, temporomandibular joint (TMJ) pathology

Airway classification

Airway classification based upon the Mallampati scoring system has become an accepted method of attempting to determine the difficulty of airway management during sedation/anesthesia. The airway is assessed with the patient in an upright sitting position with the mouth wide open and tongue protruded maximally.

• Class I: The tonsillar faucial pillars, soft palate, and uvula are visible.
• Class II: Tonsillar faucial pillars and soft palate are visible, but the uvula is hidden by base of the tongue.
• Class III: Only the soft and hard palate are visible.
• Class IV: Only the hard palate is visible. Class III and IV may indicate potential difficult airway management.

Determining Fasting Status

A determination of the patient’s fasting status to reduce morbidity and mortality from intraoperative aspiration of gastric contents is mandatory. Guidelines regarding fasting status for patients about to undergo sedation/anesthesia have been in a state of flux, with many still advocating that adults have nothing by mouth after midnight the day before surgery. For healthy patients undergoing ambulatory surgery for elective procedures, the following guidelines are suggested with the caveat that following them does not guarantee complete gastric emptying.

Minimal
### Ingested material

<table>
<thead>
<tr>
<th>Ingested material</th>
<th>Fasting period</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clear liquids (eg, water, black coffee, clear tea, fruit juices without pulp,</td>
<td>2 h</td>
</tr>
<tr>
<td>carbonated beverages)</td>
<td></td>
</tr>
<tr>
<td>Breast milk</td>
<td>4 h</td>
</tr>
<tr>
<td>Infant formula</td>
<td>6 h</td>
</tr>
<tr>
<td>Nonhuman milk</td>
<td>6 h</td>
</tr>
<tr>
<td>Light meal (eg, toast and clear liquids)</td>
<td>6 h</td>
</tr>
<tr>
<td>Heavy meal</td>
<td>8 h</td>
</tr>
</tbody>
</table>

**Physical Status Classification**

The complexity of patient’s coexisting medical conditions is documented prior to the selection of technique. Anesthesia care providers have adopted the American Society of Anesthesiologists (ASA) physical status classification for this purpose (Table 14-1). ASA I and II patients, as well as selected ASA III patients, are routinely sedated and/or anesthetized in the oral and maxillofacial surgery office.

**Determining Level of Sedation/Anesthesia**

Depression of the central nervous system can range from minimal to moderate to deep sedation and general anesthesia (Table 14-2).

The sedative/anesthetic plan is dictated by:

- The physical status of the patient
- Planned procedure and duration
- Airway considerations

Techniques can include:

- Premedication to reduce anxiety
- Minimal or moderate sedation using oral, intravenous (IV),
intramuscular (IM), inhalation (nitrous oxide–oxygen), or intranasal routes of administration along with local anesthesia

- Deep sedation and general anesthesia using IV, IM, or inhalation routes with or without local anesthesia

**Table 14-1 ASA physical status classification**

<table>
<thead>
<tr>
<th>ASA Class</th>
<th>Description</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>A normal, healthy patient, without organic, physiologic, or psychiatric disturbances</td>
<td>Healthy with good exercise tolerance</td>
</tr>
<tr>
<td>II</td>
<td>A patient with controlled medical conditions without significant systemic effects</td>
<td>Controlled hypertension, controlled diabetes mellitus, cigarette smoking without evidence of COPD, anemia, mild obesity, age younger than 1 year or older than 70 years, pregnancy</td>
</tr>
<tr>
<td>III</td>
<td>A patient having medical conditions and significant systemic effects intermittently associated with significant functional compromise</td>
<td>Controlled CHF, stable angina, poorly controlled hypertension, morbid obesity, bronchospastic disease with intermittent symptoms, chronic renal failure</td>
</tr>
<tr>
<td>IV</td>
<td>A patient with a medical condition that is poorly controlled, associated with significant dysfunction, and a</td>
<td>Unstable angina, symptomatic COPD, symptomatic CHF, hepatorenal failure</td>
</tr>
</tbody>
</table>
A patient with a critical medical condition that is associated with little chance of survival with or without the surgical procedure

Multiorgan failure, sepsis syndrome with hemodynamic instability, hypothermia, poorly controlled coagulation

COPD, chronic obstructive pulmonary disease; CHF, congestive heart failure.

### Table 14-2 Levels of sedation/anesthesia

<table>
<thead>
<tr>
<th></th>
<th>Minimal sedation (anxiolysis)</th>
<th>Moderate sedation (conscious sedation)</th>
<th>Deep sedation</th>
<th>General anesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Responsiveness</td>
<td>Normal response to verbal and physical stimulation</td>
<td>Purposeful response to verbal or physical stimulation</td>
<td>Purposeful response following repeated or painful stimuli</td>
<td>Unarousable, even with painful stimuli</td>
</tr>
<tr>
<td>Airway</td>
<td>Unaffected</td>
<td>Unaffected</td>
<td>Intervention may be required</td>
<td>Intervention often required</td>
</tr>
<tr>
<td>Spontaneous ventilation</td>
<td>Unaffected</td>
<td>Adequate</td>
<td>May be inadequate</td>
<td>Frequently inadequate</td>
</tr>
<tr>
<td>Cardiovascular function</td>
<td>Unaffected</td>
<td>Usually maintained</td>
<td>Usually maintained</td>
<td>May be impaired</td>
</tr>
</tbody>
</table>

**Sedative/Anesthetic Complications**

**Monitoring**

The type of monitoring during sedation/anesthesia depends upon the depth of sedation/anesthesia, technique used, and medical condition of the patient. The purpose of monitoring is to warn the oral surgeon of changes in oxygenation,
ventilation, circulation, and level of consciousness that signal an impending dangerous trend. Visual observation of the patient as well as a subjective evaluation of the cardiovascular system can detect airway obstruction and the character of respiration (eg, rate, depth, and rhythm).

Oxygenation
To ensure adequate oxygenation of the blood, a pulse oximeter is mandatory for all sedated patients. An inspired oxygen analyzer will ensure adequate inspired oxygen concentration.

Ventilation
To ensure adequate ventilation, direct methods such as chest excursion and observation of the reservoir bag are useful. Continual auscultation of breath sounds via a pretracheal stethoscope is a very useful ventilation monitor that noninvasively provides instantaneous and continuous information.

Capnography or capnometry, providing continual monitoring for the presence of expired (end-tidal) carbon dioxide, is mandatory whenever an endotracheal tube or laryngeal mask airway is inserted. It can also be a useful ventilatory monitor in nonendotracheal techniques.

Circulation
Cardiovascular function is monitored by:

- Precordial stethoscope (heart rate and rhythm)
- Sphygmomanometer and stethoscope (or automated vital signs monitor)
- Blood pressure monitor
- Electrocardiogram (ECG) (cardiac rate and rhythm)
- Pulse oximetry (peripheral pulse)

Level of consciousness
The level of consciousness for minimal and moderate sedation techniques is measured via patient response to verbal command.

Local Complications
Intravenous: Localized vascular redness and itching may be seen following injection of opioids due to histamine release.
Treatment

- Self-limiting
- Lidocaine (10 to 20 mg) and diphenhydramine (25 mg) IV

Phlebitis: Inflammatory process of veins following needle or catheter insertion accompanied by pain, bruiselike discoloration of vessel, increased local temperature, and leading to induration and decannulation of the vessel.

Treatment: Most cases of phlebitis are not caused by infection; treatment is palliative with the application of warm compresses, exercise limitation, and prescription of an nonsteroidal anti-inflammatory drug (NSAID). If there is a noticeable upward progression of redness and induration, a vascular consultation is indicated.

Infiltration or extravasation: Caused by infusion of IV fluids or medications in soft tissue rather than the lumen of a vein

Treatment

- Firm pressure applied over the injection site
- Application of warm, moist compresses
- Explanation to patient that the fluid will be absorbed from tissues over time

Intra-arterial injection: Inadvertent intra-arterial injection of fluid/medications

Prevention

- Knowledge of venous antecubital fossa anatomy.
- If detected prior to drug administration, immediate withdrawal of needle and firm pressure to injection site for 5 minutes.

Diagnosis: Patient will experience intense pain distal to injection
Sequelae

- Intense arterial spasm
- Pain
- Ischemia

Morbidity: Loss of digits or even the entire hand

Treatment

- Injection of 2 to 3 mL of preservative-free local anesthetic without epinephrine
- Immediate transfer to hospital
- Sympathetic nerve block
- Possible heparinization

Airway Complications

Airway evaluation, management, and technique are the hallmarks of safe sedation/anesthesia. Decisions regarding anesthetizing location (office versus hospital) and technique (nonendotracheal versus laryngeal mask versus endotracheal intubation) depend upon the extent of the procedure and anticipated patient-specific airway issues.

The ASA difficult airway algorithm (Fig 14-1) provides an excellent basic framework for treating the difficult airway.

Laryngospasm: A laryngospasm is a reflex closure of the vocal cords resulting in partial or complete glottic obstruction.

Etiology: Irritating stimulus to the airway during light anesthesia:

- Secretions
- Blood
- Vomitus
- Irritating, pungent, volatile anesthetics
- Painful stimuli
**Diagnosis**
- “Crowing” respirations or stridor, but if total spasm can be silent
- “Rocking boat” respiration

**Treatment**
- Stopping the painful stimulation
- Application of continuous, gentle positive pressure on the airway
- Suctioning the airway
- Possibly increasing the anesthetic depth
- Administration of small dose of succinylcholine (eg, 10 to 20 mg)
- Ventilation with 100% oxygen

Bronchospasm: Intraoperative reflex bronchiolar constriction may be centrally mediated or due to a local response to airway stimulation.

**Causes**
- Acute asthma
- Histamine release related to drugs
- Anaphylaxis
- Cigarette smoking with chronic bronchitis
- Noxious stimuli
- Pain
- Secretions
**Diagnosis**

- Conscious patient
  - Wheezing
  - Tachypnea
  - Dyspnea

- Anesthetized patient
  - Difficulty in ventilation due to increased airway resistance
  - Increase in intrathoracic pressure impedes venous return and results in decreased cardiac output and hypotension

**Treatment**

- Inhalation administration of $\beta_2$-adrenergic agonists
- For severe life-threatening bronchospasm unresponsive to $\beta_2$-adrenergic agonists, IV epinephrine, with the usual caveats, may be considered.

Chest wall rigidity: Fentanyl (and its derivatives) can produce rigidity of the chest wall in doses as small as 50 µg, but it is more often encountered with the rapid infusion of large doses of opioids and the addition of nitrous oxide. A patient experiencing chest wall rigidity cannot breathe and cannot be ventilated via positive pressure.

**Treatment**

- Small doses of succinylcholine (10 to 20 mg IV)
- Positive pressure ventilation

Pulmonary edema: Pulmonary edema may be due to:

- Acute congestive heart failure
- Myocardial infarction
• Increased capillary permeability
• Airway obstruction
• Naloxone administration

**Diagnosis**

- Rales on auscultation
- Pink, frothy sputum

**Treatment**

- Symptomatic
- Oxygen
- Diuretics

Aspiration: With depression of airway reflexes, aspiration of gastric contents is always a danger during deep sedation and general anesthesia. The severity depends upon the volume and pH of gastric material aspirated. Evidence of massive aspiration includes bronchospasm, decreased lung compliance, and hypoxemia. Predisposing factors include recent food intake, gastro-esophageal reflux, pregnancy, obesity, and hiatal hernia.

**Treatment**

- Position patient in Trendelenburg position to decrease aspiration into trachea
- Turn head to side
- Airway suctioning
- Endotracheal intubation and suctioning prior to positive pressure ventilation
- Antibiotic therapy, lavage with saline, and use of steroids are controversial
- Bronchoscopy may clear large airways
- Chest radiograph should be obtained, although evidence of infiltrates may be delayed
Cardiovascular Complications

Hypotension: Clinically significant decrease of arterial blood pressure (15% to 20%) from baselines in the perioperative period may be due to:

- Decrease in cardiac function (contractility)
  - Volatile liquids, barbiturates, and large doses of benzodiazepines can cause a direct dose-dependent depression of the myocardium
  - Myocardial ischemia/infarction

- Decrease in systemic vascular resistance (SVR)
  - Some inhalation anesthetics
  - Opioids
  - Drugs that release histamine
  - Sepsis
  - Allergic reactions
  - Inadequate venous return
  - Hypovolemia
  - Dysrhythmias
  - Tachycardia
  - Atrial fibrillation, atrial flutter
  - Bradydysrhythmias

Treatment

- Symptomatic
- Place patient in semi-Fowler position
- Decrease anesthetic depth
- Increase intravascular fluid volume
- Drugs

Hypertension: Causes of hypertension include release of catecholamines due to:

- Light anesthesia (pain response)
- Hypoxia
- Hypercarbia
- Preexisting essential hypertension
• Drug interactions (eg, epinephrine with monamine oxidase inhibitors)

_Treatment_
  • Symptomatic depending upon cause
  • Improving oxygenation and ventilation
  • Deepening anesthesia
  • Drug therapy
    —β-adrenergic blocking agents (labetalol, 5 to 10 mg IV; esmolol, 5 to 10 mg IV)
    —Vasodilators (hydralazine, 2.5 to 20 mg IV)

Arrhythmias: Arrhythmias occurring during sedation/anesthesia may be seen due to intrinsic cardiac disease, but sedation/anesthesia–specific etiologies must always be considered. These include:
  • Oxygenation and/or ventilation issues
    —Hypoxia
    —Hypercarbia
  • Medications (eg, succinylcholine)
  • Pain
  • Oculocardiac reflex
  • Hypotension
  • Hypertension
  • Fever
  • Hypovolemia

_Treatment_
  • Symptomatic and directed at correction of any oxygenation/ventilation problems and any other underlying causes.
  • Sinus bradycardia rate of less than 60 BPM with accompanying hypotension is treated with atropine (0.4 to 0.8 mg IV)
• Sinus tachycardia rate greater than 100 beats/minute (BPM) may be treated by increasing the depth of anesthesia and addressing hypovolemia.

Myocardial infarction

**Clinical signs**

• Severe crushing chest pain
• Nausea/vomiting, pallor, feeling of impending doom
• Cardiac arrhythmias and changes in ECG morphology
• Acute congestive heart failure
• Pulmonary edema

**Causes**

• Hypoxia/hypercarbia
• Hypo-/hypertension
• Anesthetic overdose
• Hypovolemia
• Pain and/or anxiety

**Treatment**

• Administer 100% oxygen
• Nitroglycerin (sublingual or IV)
• Analgesics, if in pain
• Aspirin
• Treat symptomatically with ACLS MI protocol (see page 175)

Malignant hyperthermia: Hypermetabolic syndrome seen in genetically susceptible individuals when exposed to anesthetic triggering agents. Triggering agents include all of the volatile liquids (halothane, enflurane, isoflurane, desflurane, sevoflurane) and succinylcholine.
Clinical signs
- Unexplained tachycardia
- Hypercarbia or tachypnea in spontaneously breathing patient
- Masseter spasm or muscle rigidity after administration of succinylcholine
- Dysrhythmias
- Hypoxemia
- Hyperkalemia
- Fever
- Myoglobinuria

Treatment
- Discontinue anesthetic agents
- Hyperventilate with 100% oxygen
- Dantrolene, 2.5 mg/kg IV; repeat to total dose of 10 mg/kg
- Call malignant hyperthermia hotline (800) 644-9737 (within US and Canada) or 011 (315) 464-7079 (outside US and Canada)

Reversal Agents
In cases of oversedation, prolonged recovery, and/or respiratory depression from benzodiazapines, opioids, or a combination of both, the use of reversal agents may be contemplated.
- Reversal of systemic opioid effect by antagonism via competitive inhibition
  — Naloxone; adult dose: 0.04 to 0.4 mg IV every 2 to 3 minutes
  — Important to note that reversal of analgesia may be accompanied by hypertension, tachycardia, dysrhythmias, and, rarely, pulmonary edema. Renarcotization may occur due to its short duration.

- Reversal of benzodiazepine sedation by competitive antagonism at central nervous system benzodiazepine receptors
  — Flumazenil; adult dose: 0.2 to 1.0 mg IV every 20 minutes at 0.2
mg/min
—May induce central nervous system (CNS) excitation, including seizures. Duration of action dependent on dose and type of benzodiazepine and dose of flumazenil. Resedation may occur due to its short duration.

Postoperative Nausea and Vomiting
Predisposing Factors

- History of nausea/vomiting after anesthesia
- History of severe motion sickness
- Obesity
- Female sex
- Administration of narcotics or inhaled anesthetics
- Pain

Prophylaxis and Treatment

The 5-HT₃ serotonin antagonists (eg, ondansetron, 4 to 8 mg IV) appear to be somewhat effective in reducing the incidence of nausea and vomiting during the perioperative period. Other traditional preoperative and postoperative antiemetic therapy includes:

- Phenothiazines
  —Prochlorperazine; 5 to 10 mg IM, oral, or rectal suppositories

- Antihistamines (H₁ receptor antagonists)
  —Promethazine; 25 to 50 mg oral or IV, 12.5 to 50 mg rectal suppositories
  —Hydroxyzine; 25 to 50 mg IV or IM

- Bezamides
  —Metoclopramide; 0.15 mg/kg or 10 mg IV or IM

- Anticholinergics
  —Scopolamine patch; 1.5 mg transdermal
Recovery/Discharge
Recovery from anesthesia can be divided into the following stages:

- Early recovery
  — The immediate postoperative period during which awakening and return of protective reflexes occur
  — Patient must be continually observed and monitored

- Intermediate recovery
  — When the patient has met discharge criteria

- Late recovery
  — When full recovery has occurred

Discharge Criteria
Discharge criteria may include:

- Return of vital signs to preoperative levels
- Ability to move voluntarily
- Minimal nausea and vomiting
- No excessive pain or bleeding from surgical site
- Discharge by the surgeon to a vested escort
- Written postoperative instructions and contact information
Differential Diagnosis and Treatment of Cysts and Tumors

Odontogenic cysts and tumors are the most common intrabony noninflammatory lesions found in the maxilla and mandible. However, nonodontogenic benign neoplasms, primary and metastatic malignancies, and diffuse bony pathology such as fibro-osseous lesions, developmental anomalies, and reactive or dysplastic processes must also be considered in the differential diagnosis of jaw lesions (see chapter 24).

Previously, some developmental cysts were considered “fissural” cysts. However, the concept of developmental epithelium being trapped along embryonal lines of fusion has been questioned in recent years. Irrespective of their origin, developmental and odontogenic cysts tend to increase slowly in size, theoretically in response to elevated hydrostatic luminal pressures.

Odontogenic tumors are characterized by diverse histopathologic, radiographic, and clinical behavior. Some of these lesions are true neoplasms that rarely exhibit malignant behavior. Others may represent tumorlike malformations.

Think before you cut! Establish an initial differential diagnosis by obtaining a complete history and performing a physical examination (see chapter 1). These preliminary data will influence the diagnostic tests to be ordered and direct the choice of incisional or excisional biopsy.

Establishing a Differential Diagnosis

History

Pain: Usually not a prominent feature of cysts and tumors unless the lesion is secondarily infected or the tumor is invasive or neural in origin.
Swelling: Persistent, slow growth suggests an expanding lesion; rapid growth suggests concurrent infection or an aggressive lesion.

Sensory changes: Altered or lost sensation along the distribution of the trigeminal nerve portends long-standing infection, an invasive malignancy, an aggressive benign tumor, metastatic disease, or a primary neural tumor of the jaws.

Systemic findings: A history of malignancy of the breast, prostate, kidney, lung, thyroid, gastrointestinal tract, or lymph nodes should lead one to consider metastatic jaw tumors. A family history of a syndrome with well-established jaw involvement, such as basal cell nevus syndrome and Gardner syndrome, should also make one consider such a possibility as the cause of jaw pathology.

Physical Examination

A focused examination strategy involving inspection, palpation, auscultation, and olfaction, supported by an in-depth understanding of oral pathology, will facilitate a thorough physical evaluation. The primary diseased tissue should be determined and its boundaries established, as well as the extent of any spread into local, regional, or distant tissues.

Surface changes: Are the surface changes related to chemical, traumatic, vesicular-bullous, neoplastic, metabolic, or inflammatory conditions?

Swelling: Location, tissue of origin, time of onset, rate of enlargement, variability of size, and changes in size related to eating or jaw function are factors that contribute to the differential diagnosis.

Loss of function: Changes in jaw mobility may be related to intracapsular or extracapsular temporomandibular joint (TMJ) disease. Tumors of the glenoid fossa and condyle are rare. Most disturbances of jaw function are caused by benign abnormalities of the TMJ or muscles of mastication. However, malignant or aggressive benign tumors of the jaws that extend into the surrounding soft tissues and interfere with the elevator and depressor muscles of the jaw may also disrupt the mobility of the mandible. Pathologic fracture from extensive destruction of the mandible causes an acute change in occlusion, jaw mobility, and facial form. Tumors or expanding cysts of the maxilla can cause nasal stuffiness, deviation of the nasal septum, nasoantral openings, oroantral openings, signs of orbital involvement, and ear stuffiness.
Auscultatory findings: Radiolucencies of the jaws, particularly multilocular expanding lesions, must be considered vascular in origin until proven otherwise. When a vascular lesion is suspected, the area overlying the lesion should be auscultated to identify bruits and pulsations. Aspiration should also be done prior to any invasive procedure.

Olfactory findings: Necrotic lesions of the jaws and surrounding tissues usually portend advanced disease. Necrotic tissue has a characteristic odor that is readily identifiable. Anaerobic infections associated with cysts or tumors of the jaws also often have a foul smell. Sebaceous material from cysts is easily identified by its appearance and strong odor.

Diagnostic Imaging

The merits and limitations of radiographic and other forms of imaging are described in Table 15-1. Plain radiographs may be useful in detecting advanced changes in the jaws. However, early and even moderately extensive destruction of the mandible may not be apparent on plain films when the cortical plates are still intact. Such lesions of the jaws require more sensitive diagnostic imaging techniques to detect destruction of the medullary and cortical bone. For this purpose, tomography, a computed tomography (CT) scan, or radionuclide scanning is useful. Magnetic resonance imaging (MRI) may also be useful when tumors of the soft tissues of the oral cavity and contiguous structures are suspected.

Table 15-1 Diagnostic imaging techniques
### Classification of odontogenic cysts

**Developmental**
- Dentigerous cyst

**Inflammatory**
- Radicular cyst

<table>
<thead>
<tr>
<th>Procedures</th>
<th>Uses</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plain radiographs</td>
<td>• Screening</td>
<td>• Available • Inexpensive • Simple • Eliminates overlying structures</td>
<td>• Low discrimination • Two-dimensional image</td>
</tr>
<tr>
<td>Tomography</td>
<td>• Producing positional information</td>
<td>• Structures seen in a preselected plane • Allows accurate measurements</td>
<td>• Potentially a high-radiation-dose examination • Expensive • Limited availability</td>
</tr>
<tr>
<td>Computed tomography scan</td>
<td>• Locating and staging primary tumors • Following results of treated tumors</td>
<td>• Produces cross-sectional images • Smaller densities are visible</td>
<td>• Requires highly complicated equipment • Examination is time-consuming • Expensive</td>
</tr>
<tr>
<td>Magnetic resonance imaging</td>
<td>• Primary soft tissue tumors • Metastatic tumors • Extension of jaw tumors to adjacent soft tissues</td>
<td>• No ionizing radiation • Noninvasive • Tissue contrast • Tissue discrimination</td>
<td>• High cost • Poor bone detail • Examination is time-consuming</td>
</tr>
<tr>
<td>Radionuclide imaging</td>
<td>• Detecting metastasis • Investigating arthritis • Detecting skeletal infections</td>
<td>• Detects widespread disease • Shows early bone changes</td>
<td>• Studies take several hours • Organs other than those being examined are exposed • Size not shown accurately</td>
</tr>
</tbody>
</table>
1. Eruption cyst
2. Odontogenic keratocyst
3. Gingival (alveolar) cyst of the newborn
4. Gingival cyst of the adult
5. Lateral periodontal cyst
6. Calcifying odontogenic cyst
7. Glandular odontogenic cyst

Box 15-2 Classification of developmental cysts

**Oral cavity**
- Palatal cysts of the newborn (Epstein pearls; Bohn nodules)
- Nasolabial cyst
- Globulomaxillary cyst
- Nasopalatine duct cyst
- Median palatal cyst
- Median mandibular cyst
- Oral lymphoepithelial cyst

**Facial skin and neck**
- Epidermoid cyst
- Dermoid cyst
- Thyroglossal duct cyst
- Cervical lymphoepithelial cyst (branchial cleft cyst)
Differential Diagnosis

Cysts: The majority of jaw cysts are odontogenic and are subclassified as developmental or inflammatory in origin. Box 15-1 presents the 2005 World Health Organization (WHO) classification of odontogenic cysts. Classes of nonodontogenic developmental cysts of the head and neck are presented in Box 15-2.

Tumors: Odontogenic tumors are classified according to the inductive interactions between the odontogenic epithelium and the odontogenic mesodermal elements. Some odontogenic tumors are composed only of odontogenic epithelium without any influence of odontogenic ectomesenchyme. Box 15-3 presents the categories of odontogenic tumors based on the 2005 WHO classification.

Primary and metastatic bone tumors of nonodontogenic origin should be considered when more common jaw diseases have been excluded or when the history, clinical examination findings, and laboratory tests suggest such a diagnosis (Box 15-4).

**Box 15-3 Classification of odontogenic tumors**

<table>
<thead>
<tr>
<th>Tumors of odonto- genic epithelium without odontogenic ectomesenchyme</th>
<th>Tumors of odonto- genic epithelium with odontogenic ectomesenchyme*</th>
<th>Tumors of odontogenic ectomesenchyme with or without included odontogenic epithelium</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Ameloblastoma</td>
<td>• Ameloblastic fibroma</td>
<td>• Odontogenic fibroma</td>
</tr>
<tr>
<td>• Calcifying epithelial</td>
<td>• Ameloblastic fibroodontoma</td>
<td></td>
</tr>
</tbody>
</table>
Box 15-4 Nonodontogenic tumors of the jaws

**Benign**
- Osteoid osteoma
- Osteoblastoma
- Chondroma
- Chondromyxoid fibroma
- Schwannoma
- Desmoplastic fibroma

**Malignant**
- Fibrosarcoma
- Osteosarcoma
- Chondrosarcoma
- Ewing sarcoma

**Metastatic**
- Breast
- Prostate
- Kidney
- Colon
- Thyroid
- Lung

*With or without dental hard tissue formation.*
Bone pathology of hereditary origin, unusual nonpathologic radiographic changes, and nonodontogenic or nondevelopmental lesions of the jaws must be considered when the more common odontogenic cysts and tumors of the jaws are ruled out (Box 15-5). The radiographic pathology and special features of selected periapical and pericoronal jaw lesions are presented in Table 15-2. Unilocular and multilocular lesions not related to the periapical or pericoronal regions are presented in Table 15-3.

**Box 15-5 Nonodontogenic and nondevelopmental lesions of the jaws**

**Hereditary**
- Osteogenesis imperfecta
- Osteopetrosis (Albers-Schönberg disease)
- Cleidocranial dysplasia
- Gardner syndrome

**Fibro-osseous lesions of the jaws**
- Fibrous dysplasia
- Cemento-osseous lesions of the jaws
- Periapical cemento-osseous dysplasia
- Focal cemento-osseous dysplasia
- Florid cemento-osseous dysplasia
<table>
<thead>
<tr>
<th>Lesion Type</th>
<th>Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic osteolytic and osteosclerotic lesions</td>
<td>- Ossifying (cemento-ossifying) fibroma</td>
</tr>
<tr>
<td></td>
<td>- Focal osteoporotic marrow defect</td>
</tr>
<tr>
<td></td>
<td>- Idiopathic osteosclerosis</td>
</tr>
<tr>
<td></td>
<td>- Paget disease of bone</td>
</tr>
<tr>
<td></td>
<td>- Langerhans cell disease</td>
</tr>
<tr>
<td></td>
<td>- Central giant cell granuloma</td>
</tr>
<tr>
<td></td>
<td>- Cherubism</td>
</tr>
<tr>
<td>Lesions with bleeding tendencies</td>
<td>- Aneurysmal bone cyst</td>
</tr>
<tr>
<td></td>
<td>- Central hemangioma</td>
</tr>
<tr>
<td></td>
<td>- Arteriovenous malformation</td>
</tr>
</tbody>
</table>

*Table 15-2* Radiographic and other features of pericoronal and periapical jaw lesions
Table 15-3 Radiographic and other features of nonapical and noncoronal jaw lesions

<table>
<thead>
<tr>
<th>Condition</th>
<th>Radiolucency</th>
<th>Special features</th>
<th>Occurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral radicular cyst</td>
<td>Unilocular</td>
<td>Nonvital tooth—lateral to or between teeth</td>
<td></td>
</tr>
<tr>
<td>Lateral periodontal cyst</td>
<td>Unilocular</td>
<td>Usually in mandibular canine-premolar region</td>
<td></td>
</tr>
<tr>
<td>Residual cyst</td>
<td>Unilocular</td>
<td>Edentulous area</td>
<td></td>
</tr>
<tr>
<td>Central giant cell granuloma</td>
<td>Unilocular/</td>
<td>Usually in anterior mandible</td>
<td></td>
</tr>
<tr>
<td></td>
<td>multilocular</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 15-3 Radiographic and other features of nonapical and noncoronal jaw lesions
<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Location</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stafne bone defect</td>
<td>Unilocal</td>
<td>Posterior mandible below mandibular canal</td>
</tr>
<tr>
<td>Fibro-osseous lesions</td>
<td>Unilocal</td>
<td>Early-stage lesions</td>
</tr>
<tr>
<td>Langerhans cell disease</td>
<td>Unilocal</td>
<td>Histiocytosis X—usually children and young adults</td>
</tr>
<tr>
<td>Melanotic neuroectodermal tumor of infancy</td>
<td>Unilocal</td>
<td>Anterior maxilla</td>
</tr>
<tr>
<td>Median palatal cyst</td>
<td>Unilocal</td>
<td>Midline swelling of hard palate</td>
</tr>
</tbody>
</table>

Jaw lesions presenting as multilocular radiolucencies include odontogenic keratocyst, ameloblastoma (posterior mandible), central giant cell granuloma (anterior mandible), ameloblastic fibroma, odontogenic myxoma (posterior mandible), calcifying epithelial odontogenic tumor, vascular malformation, aneurysmal bone cyst (young patients), cherubism (onset in childhood), and hyperparathyroidism (brown tumor). Radiolucencies with poorly defined or ragged borders are suggestive of malignant tumors, osteomyelitis, traumatic bone cyst, and hematopoietic bone marrow defects. Multifocal or generalized radiolucencies are suggestive of cemento-osseous dysplasia, basal cell nevus syndrome, multiple myeloma, hyperparathyroidism, and Langerhans cell disease.

Radiopacities that present with well or poorly demarcated borders include retained root tips, condensing osteitis, odontoma, cemento-osseous dysplasia (late stage), sclerosing osteomyelitis, Paget disease, osteosarcoma (may have “sunburst” cortical change), and chondrosarcoma.
Indications for Biopsy

Aspiration: Radiolucent lesions should be routinely aspirated before incisional or excisional biopsy to rule out the presence of a vascular lesion. Inadvertent access to a hemangioma or arteriovenous malformation may result in lethal hemorrhage.

Incisional biopsy: Used when a malignancy or an aggressive benign tumor is suspected. Because such lesions may require a surgical procedure that is disfiguring or functionally impairing, it is essential to establish a definitive diagnosis before initiating final therapy.

Excisional biopsy: Used for small, benign lesions; suspected small, malignant lesions when satisfactory margins can be removed at the time of biopsy; and for clinically obvious benign lesions that are large (eg, odontogenic cyst or epulis fissuratum).

Indications for Sialography

Sialography is useful to distinguish swellings of the submandibular gland from lymphadenitis and the extensions of mandibular cysts or tumors into the submandibular triangle. Injecting dye into a major salivary gland can be painful and difficult when cannulation of the duct is problematic. MRI, positron emission tomography (PET) scan, and CT imaging with or without contrast offer very effective alternatives to the sialogram.

Intraoperative and Postoperative Management of Cysts

Treatment Options

- Marsupialization
- Marsupialization and secondary enucleation
- Enucleation
- Enucleation and treatment of adjacent bone
  - Cryotherapy
  - Electrocautery
  - Chemical cautery
- En bloc resection: jaw continuity maintained
• Partial resection: jaw continuity lost

Wound Healing and Care of the Wound
• Obliterate dead space.
• Use layered soft tissue closure.
• Use drains when serosanguinous products may collect in deep tissues.
• Close the wound primarily whenever the center of a bony defect is in close approximation to well-vascularized tissue.
• Consider immediate management of the bony defect with an allograft material.
• Place pressure packs over the wound for 2 hours.
• Instruct patient to:
  — Avoid rinsing and spitting for 24 hours.
  — Use saltwater rinses after meals.
  — Maintain a soft diet.
  — Restrict activity.

Antimicrobial Agents
Noninfected lesion: Antibiotics are generally not indicated for such patients.
Infected lesion: Do a culture and antibiotic sensitivity test of the infected surgical site. Initiate an antibiotic regimen empirically, but change if indicated by these tests or poor clinical response.

Rehabilitation
• Establish a level of physical activity, diet, and social and employment restrictions.
• Restore interincisal opening and lateral excursive movements early during the postoperative period. Begin jaw stretching exercises within pain-free limits. The patient should be cautioned to avoid trauma to the surgical site.
• Speech therapy should be initiated when speech patterns have been altered by surgical therapy.

Intraoperative and Postoperative Management
of Tumors
Treatment Options
- Enucleation or curettage
- Enucleation or curettage and treatment of adjacent bone
  — Cryotherapy
  — Electrocautery
  — Chemical cautery
- En bloc resection: jaw continuity maintained
- Partial resection: jaw continuity lost
- Subtotal resection: extensive resection of the jaw
- Radiation therapy
- Chemotherapy

Wound Healing and Care of the Wound
(same considerations as for cysts; see page 228).

Complications and Their Management
Wound Dehiscence
- Irrigate with sterile saline to cleanse the wound of necrotic tissue and food debris.
- Excise any bony sequestra and necrotic tissue to the edge of bleeding tissue.
- Generally, attempts at resutting the wound will be unsuccessful.
- Instruct patient in home wound care.
- Packing should be placed when soft tissue dehiscence exposes an underlying bony cavity. Use iodoform gauze to obliterate dead space and promote organization of the wound. The pack should be advanced slowly over a period of several days.

Infection
- Remove the cause (dead bone and granulation tissue).
- Perform incision and drainage when indicated.
- Do a culture and antibiotic sensitivity test.
- Initiate an antibiotic regimen.
- Provide supportive care.
Trismus

- Establish routine postoperative TMJ physiotherapy.
- Restore mouth opening and excursive movements within pain-free limits.
- Release any scar tissue attached to the ramus; revise anterior temporals shortening with Z-plasties of the muscle at the anterior border of the ramus.

Pathologic Fracture (see chapter 16)

- Reduce the fracture.
- Fix the fracture.
- Stabilize the jaw.
- Prevent infection.
Management of Maxillofacial Trauma

The primary objective in managing an acutely injured patient is to provide an accurate and rapid assessment in a standardized and sequential fashion. Two phases of assessment and management are recognized. In the primary phase, life-threatening injuries are identified and managed simultaneously. Once the patient is initially stabilized and properly monitored, the secondary phase of assessment is begun in a methodical, yet rapid, fashion.

Initial Evaluation
Primary Assessment and Management

Identify and treat life-threatening problems (ABCs, see chapter 11, page 155)

- Manage airway obstruction/provide cervical spine stabilization (the A for advanced trauma life support [ATLS] is airway/cervical spine stabilization).
- Evaluate and support the cardiopulmonary system.
- Control external hemorrhage.
- Assess and manage shock.
- Monitor appropriate indicators of function.
  - Establish access to the vascular system.
  - Draw blood for necessary studies.
  - Consider a central venous pressure line or Swan-Ganz catheter.
  - Insert a nasogastric tube and Foley catheter.

Secondary Assessment and Management

- Obtain history of the injury, past medical history, present medications, and known allergies, if possible.
- Complete a rapid yet accurate physical examination, paying particular attention to the already identified life-threatening injuries.
Splint fractures.
Obtain necessary radiographs.
Perform peritoneal lavage if abdominal trauma is suspected.

Specific Management Considerations
Airway Obstruction

Listen for either complete or partial obstruction. Complete obstruction is characterized by lack of breath sounds, and partial obstruction is characterized by sonorous breath sounds. Inspect the neck and chest wall for signs of accessory respiratory muscle involvement that may be present in either a complete or partial obstruction.

Causes: Altered level of consciousness, facial or neck trauma, foreign bodies, blood, vomitus, or edema of the oropharyngeal or laryngeal areas.

Maneuvers: Management of an upper airway obstruction demands immediate attention. The first maneuver in airway management is either the chin lift (preferred) or the jaw thrust, which will effectively move the tongue anteriorly. Take great care not to hyperextend the neck in cases of suspected cervical spine injury.

A cervical collar should be applied whenever a cervical spine injury is suspected, if the patient is unconscious, or if multiple injuries are noted. The collar should not be removed until cervical spine injuries are ruled out with radiographs. If an obstruction is still present following the chin-lift or jaw-thrust maneuver, suction the oral cavity, pharynx, and hypopharynx, and examine for foreign bodies.

Removal of foreign bodies can be done with a suction catheter, the index finger, or Magill forceps. To maintain an established airway, use an oropharyngeal airway in an unconscious patient and a nasopharyngeal airway in a patient with an intact gag reflex.

Protection: Once an upper airway has been established, it must be protected from hemorrhage, vomitus, or aspiration. This is especially critical if the patient is obtunded or unconscious and positive pressure ventilation is necessary. Endotracheal intubation is thus indicated in these situations. In an apneic patient, or if major midfacial trauma is present, the oral route is
preferable. Nasotracheal intubation is the preferred route if there are known or suspected cervical spine fractures.

Surgical access: Indicated if the endotracheal route is inaccessible secondary to edema, laryngeal fractures, or severe hemorrhage.

- Transtracheal needle insufflation is performed with a 14-gauge angiocatheter inserted through the cricothyroid membrane. High-flow oxygen is intermittently insufflated through the catheter (30 to 40 times/min). Ventilation can be supported up to 45 minutes; hypercapnia results quickly and limits the prolonged use of this method.
- In adults a cricothyroidotomy is preferred over a tracheotomy. An endotracheal tube is placed through this access so as not to limit application of a cervical collar. In children a tracheotomy is preferred due to the small diameter of the trachea.
- A tracheotomy is reserved for the controlled environment of an operating room and can be performed electively after gaining access via an endotracheal route, the needle insufflation route, or the cricothyroidotomy route. (see chapter 9, page 126)

Breathing

Assessment: Once the upper airway is established and maintained, a rapid assessment of respiration is performed. The assessment must include inspection of the rate and depth of respiration, the continuity and symmetry of movement of the chest wall, and the skin for evidence of hypoxemia. Palpation of the chest wall will aid in identifying fractures and flail segments. Percussion and auscultation are useful in noting hyperresonance of a tension pneumothorax or the dullness of a significant hemothorax. Respiratory injuries may involve abnormalities of either ventilation, exchange of gases across the alveolocapillary membrane, circulation of blood to and from the tissue beds, or any combination of these.

Injuries: Injuries to the chest can produce hypoxia, hypocarbia, hypercarbia, and significant acid-base imbalances. Complete cardiovascular collapse may result from lesions that shift the mediastinum, rupture major vessels within the chest cavity, or cause cardiac dysfunction (cardiac tamponade). The following injuries need to be considered:
Thoracic cage injuries
- Contusions
- Rib and sternal fractures
- Flail segments
- Rupture of diaphragm

Space-occupying pleural injuries
- Closed and open pneumothorax
- Tension pneumothorax
- Hemothorax

Parenchymal injuries
- Contusions
- Lacerations and punctures

Cardiovascular injuries
- Myocardial contusion
- Pericardial tamponade
- Disruption of great vessels

Management considerations
- Provide supplemental oxygen via a mask or nasal cannula at a rate of 8 L/min if the patient is not intubated. The objective is to provide a fraction of inspired oxygen (FIO₂) of 40%, which will effectively increase hemoglobin saturation and improve oxygen delivery to the tissues.
- Assist or control ventilation via established upper airway route when the rate, effort, or blood gases are abnormal. If continuous positive pressure is necessary, the airway is best protected by intubating the trachea. Remember to continuously examine the chest for signs of a tension pneumothorax when using positive pressure ventilation.
- Cover sucking chest wall injuries with a flutter valve dressing (a plastic patch over the wound taped on three sides) and consider placement of a chest tube remote from the site if ventilation does not improve. (see
• Treat flail segments initially with a compressive dressing. If ventilation continues to be unsatisfactory, assist or control ventilation with positive pressure. If ventilatory pressures exceed 50 cm of H₂O, chest tubes should be placed.

• Evacuate air or fluid-filled pleural spaces. A closed pneumothorax with less than 20% collapse, without progression and a normal contralateral lung, usually requires no treatment. A collapse of more than 20%, with unstable vital signs or signs of progression, requires treatment, usually with a chest tube in the fourth to sixth intercostal space in the midaxillary line. A tension pneumothorax requires immediate treatment. An 18-gauge needle is placed in the second interspace in the midclavicular line until a chest tube can be inserted. A major hemothorax is treated with a chest tube usually inserted through the fourth to sixth interspace in the midaxillary line.

Circulation

Shock is defined as a clinical syndrome in which cellular hypoxia is the result or cause of a general state of severe circulatory inadequacy that significantly alters cellular metabolism.

Classification

*Hypovolemic shock*: Secondary to acute loss of blood volume. It is the usual type of shock in a traumatized patient. Volume loss to the external environment is obvious after a thorough inspection of the patient. Additionally, the examiner must examine the rectum, vagina, vomitus, and urine for any other visible blood. Occult blood loss can be significant; therefore, the examiner must take great care in assessing the abdomen and thorax. Pelvic fractures can cause, on the average, 1,500 mL volume loss and long bone fractures can cause 500 to 1,000 mL of loss.

*Cardiogenic shock*: May be secondary to a direct myocardial injury, a contusion, or a penetrating injury. Other possible causes of cardiogenic shock are mediastinal shifting secondary to a tension pneumothorax, cardiac tamponade, or rupture of the
diaphragm.

*Endotoxic shock:* Secondary to septicemia, which causes ineffective blood volume because of peripheral pooling of blood in the venous tree.

*Neurogenic shock:* Secondary to the loss of sympathetic control, which causes dilatation of arterioles and venules and thus a decrease in effective blood volume.

Indicators or parameters to determine the degree of shock in the primary phase of assessment

*Blood pressure:* This is the most commonly used parameter, but it is inaccurate because blood pressure response to volume depletion is nonlinear. Compensatory mechanisms can offset the initial 20% loss in healthy individuals. A volume loss of greater than 20% usually is reflected as a precipitous decline in blood pressure.

*Pulse rate:* A commonly used parameter; however, it is limited because of its lack of specificity. Pain, emotional state, and local environment can result in tachycardia. Nevertheless, a pulse rate of greater than 120 beats/min (BPM) should be considered an indicator of hypovolemia until proven otherwise.

*Skin perfusion:* Accurate indicator of acute volume loss. The first physiologic compensation for volume loss is vasoconstriction of vessels to the skin and muscles. Cool, pale extremities thus indicate a significant volume loss.

*Urine output:* A sensitive indicator of acute volume loss and one of the best indicators in judging adequacy of volume resuscitation. A decrease in renal blood flow secondary to acute volume loss immediately decreases urine output. The normal minimum urine output is 0.5 mL/kg/h.

*Mental status alteration:* A poor indicator of hypovolemia. Compensatory mechanisms maintain cerebral blood flow quite
well until the systolic pressure reaches 30 to 60 mm Hg. Altered mental states are also produced by alcohol, numerous drugs, hypoxia, and hypoglycemia.

Resuscitation: Morbidity and mortality of hypovolemic shock are significantly lessened the sooner treatment is started. The goals of resuscitation are to improve blood flow and tissue perfusion, prevent renal failure, and shift the oxygen dissociation curve to the right.

*Direct pressure:* Control external bleeding by direct pressure dressings. The use of tourniquets and direct clamping of blood vessels are not indicated. Fractures and major lacerations of the extremities can be managed with an air splint over a dressing. When there are major injuries to the abdomen, pelvis, or lower extremities, medical antishock trousers can effectively decrease hemorrhage and increase venous return from the lower extremities. Before the garment is removed, make sure that blood volume has been adequately restored to prevent sudden shock due to pooling of blood in the capacitant vasculature.

*Supplemental oxygen:* Used via the established airway at a flow rate of 8 L/min. The objective is to provide an FIO₂ of 40%.

*Establish vascular access:* Use at least two large-bore percutaneous catheters (16 gauge) in accessible arm veins. Alternative routes if percutaneous routes are inaccessible are cutdowns on the antecubital veins or the saphenous veins (see chapter 9, page 117). Subclavian and internal jugular venous catheterization may also be used.

*Fluid replacement:* The ideal fluid for resuscitation of hypovolemic shock should restore extracellular fluid volume, exchange oxygen for metabolic wastes at the cellular level, restore loss of substrates, and be free of adverse effects. Such a solution is not yet available and, therefore, fluid resuscitation uses products that approximate the ideal solution. Crystalloid solutions are preferred over colloid solutions in the initial resuscitative
phases because they are inexpensive, do not require compatibility testing, do not transmit infectious diseases, restore lost ions, and effectively expand the extracellular space. Normal saline and lactated Ringer solution are the crystalloids of choice. Lactated Ringer solution is generally preferred because it contains isotonic quantities of anions and cations. The ratio of fluid replacement to estimated blood loss is 3:1 because of the large space distribution that occurs.

**Blood and colloid replacement:** Although crystalloid solutions are the preferred initial resuscitation fluid, they are not a replacement for blood. Proper resuscitation should proceed in a stepwise fashion. If it is necessary to administer blood, type-specific, crossmatched, whole blood is preferred. If time is critical, or if type-specific, crossmatched blood is unavailable, uncrossmatched, type-specific whole blood should be used. Incompatibilities resulting from uncrossmatched, type-specific blood are usually minor. Uncrossmatched, type O-negative blood should be used only in treating massive, exsanguinating injuries. Colloids such as plasma, albumin, and hetastarch effectively replace volume losses. However, early use of these products can cause transudation of fluid across the alveolar membrane, resulting in adult respiratory distress syndrome. Additionally, these products are costly, and transmission of infectious diseases is a concern.

Monitoring: Objectives during the primary assessment and management phase are twofold. First, monitoring of vital functions provides a baseline that aids in identifying the deficits that are initially present. Second, monitoring provides the responses or the lack of responses to a given therapy.

**Pulse rate, respiratory rate, and blood pressure:** Provides information regarding cardiac and pulmonary function and indicates responses to volume replacement therapy.

**Urine quantity and quality:** Provides an assessment of fluid status, cardiac output, and injuries to the kidneys or outflow
Oximetry and arterial blood gases: Provides rapid information relative to oxygen saturation, adequacy of ventilation, and acid-base deficits.

Electrocardiogram: Identifies arrhythmias and cardiac irritability.

Central venous pressure or Swan-Ganz catheters: Not necessary in all patients. Such monitoring will give necessary information regarding cardiac function and the status of fluid replacement in patients requiring massive volume replacement, in patients requiring ventilatory support, and in patients with preexisting cardiac or pulmonary dysfunction. Central venous pressure (CVP) reflects the function of the right heart and provides a relative evaluation of the volume status (ie, changes of the initial value during fluid replacement rather than the initial value alone are important). The Swan-Ganz catheter provides accurate evaluation of left heart function and can be used, along with the CVP, to make an accurate assessment of volume changes and cardiac function.

Classification of hemorrhage: The treatment of acute hemorrhage is based on volume loss, which is often very difficult to estimate in an acute situation. Therefore, the following guidelines, developed by the Committee on Trauma of the American College of Surgeons, should be used to assist in management of a traumatized patient.

Class I: Acute loss of 15% of the total blood volume. Blood pressure, pulse, and respiratory rates are minimally affected. Blanching of the nail capillary bed by pressure may be increased, and the “tilt test” (ie, having the patient sit up for 90 seconds without vertigo or pulse drop) is usually tolerated. Examples of conditions that can cause class I blood loss include pronounced epistaxis, most fractures, and minor injuries to the intra-abdominal organs. Treatment consists of replacing the volume loss with lactated Ringer solution in a 3:1 ratio.
Class II: Acute loss of 15% to 30% of the total blood volume. Clinical symptoms include tachycardia (> 100 BPM), tachypnea (> 24 breaths/min), and hypotension. There is a fall in cardiac output, an increase in peripheral resistance, and a narrowing of the pulse pressure. The tilt test and the capillary blanch test are positive. Class II hemorrhage commonly occurs in pelvic and long bone fractures, contained vascular injuries, and splenic and liver trauma. Appropriate fluid treatment is with lactated Ringer solution in a 3:1 ratio until clinical symptoms improve. Blood replacement is usually unnecessary if hemorrhage is controlled. However, in many injuries it is unclear if hemorrhage is controlled, and one should anticipate the need for blood replacement over the next 48 hours.

Class III: Acute loss of 30% to 40% of the total blood volume. All clinical signs and symptoms of volume loss previously described are present, plus a diminished urine output. Common injuries associated with this classification include spleen and liver rupture, vascular and thoracic injuries, and multiple traumas with fractures. Resuscitation begins with lactated Ringer solution in a 3:1 ratio but also includes use of whole blood or red blood cell concentrates.

Class IV: Blood loss exceeds 40% to 50% of total volume. All signs and symptoms of hemorrhagic shock are accentuated. Vital signs are depressed, and the patient is obtunded. Resuscitation proceeds as above, but one must also consider use of platelet concentrates and fresh frozen plasma if more than 10 units of whole blood are administered. Hyperkalemia and hypocalcemia can cause significant cardiac arrhythmias in massive transfusions; monitoring with an electrocardiogram (ECG) is mandatory.

Soft Tissue Injuries
Soft tissue injuries to the facial region vary from simple abrasions and linear lacerations to complex lacerations and tissue avulsions that may involve vital underlying tissues. Every effort should be made to definitively treat soft
tissue injuries of the face within the first 24 hours. If other injuries take precedence over such treatment, the soft tissue injuries should be grossly debrided, irrigated, and dressed with saline-moistened dressings until definitive treatment can be rendered. General principles of wound care that must be adhered to involve knowledge of the regional anatomy and physiology, thorough inspection and palpation of the wound, debridement of foreign bodies and necrotic tissue, meticulous hemostasis, gentle tissue handling, debridement of skin margins, and closure of the wound in layers with minimal tension.

Classification

Epidermal injuries
- Superficial abrasion
- First-degree burn

Intradermal injuries
- Deep abrasion
- Second-degree burn

Subdermal injuries
- Contusion
- Third-degree burn
- Puncture
- Laceration
  — Simple
  — Complex

General Management Considerations

Inspection: All soft tissue injuries should be thoroughly inspected to the depth of the wound. Observe the extent and area of the anatomy injured (nerve, duct, muscle, cartilage, or vessel injuries) and note any avulsion of tissue or underlying structures. Pay particular attention to foreign bodies and debris, which are common in most injuries. All soft tissue injuries should be palpated to check for underlying bony fractures or foreign bodies not visualized.
Timing of definitive care: This varies depending on the complexity of the facial injuries (both soft and hard tissue) and the extent of other injuries. Ideally, definitive treatment should proceed within 24 hours. However, treatment of simple lacerations can be delayed up to 4 days and complex lacerations up to 2 days, if necessary, with little overall compromise. If definitive treatment must be delayed, all wounds should be thoroughly debrided and irrigated, and hemostasis achieved. If necessary, several subcutaneous sutures to stabilize flaps can be placed in the emergency room or operating room while other injuries are being treated. The wounds should be dressed with bulky, saline-moistened dressings that are changed at least daily.

Anesthetic requirements: For early and definitive treatment, these vary with the clinical setting. Local anesthesia can be used if a patient is cooperative and the repairs are simple. Children, patients with chemically altered states, and the mentally deranged are generally uncooperative and will require a general anesthetic. Management of complex wounds, no matter how compliant the patient, requires skilled assistance, appropriate instrumentation, good lighting, and time to perform the proper definitive care. These requirements are best met in an operating room with the patient under general anesthesia.

Antibiotic coverage and antitetanus therapy: These are prudent in most oral and facial soft tissue injuries because of contamination. Penicillin is still a good empirical choice for oral cavity lacerations. For cutaneous lacerations, especially those secondary to human or animal bites, consider a broad-spectrum antibiotic such as amoxicillin-clavulanate or moxifloxacin. Antitetanus therapy is as follows:

- Previously immunized more than 5 years ago but within 10 years, give 0.5 mL of fluid tetanus toxoid booster
- Previously immunized within 5 years, no booster needed
- Previously immunized more than 10 years ago
  — Uncontaminated wound: 0.5 mL of fluid tetanus toxoid booster
  — Grossly contaminated wound: 0.5 mL of tetanus toxoid booster and 25 U of tetanus human immune globulin
- No previous immunization
  — Clean, minor wound: Immunize with alum-precipitated tetanus
toxoid; give 0.5 mL of toxoid at once and repeat at 2, 6, and 20 months
—Contaminated wound: Immunize with 0.5 mL of toxoid and repeat at 1, 2, and 6 months. In addition, give 250 U of human immune globulin

Specific Management Considerations

Debridement: This is the most critical aspect in wound care and must be performed meticulously. The first step is irrigating the wound with copious amounts of saline. A water-jet lavage is particularly useful in grossly contaminated wounds. Next, the wounds should be carefully scrubbed with a surgical soap and again irrigated with saline solution. Debridement follows and involves removing any remaining foreign bodies and devitalized soft and hard tissues. Skin margins are inspected and, if irregular, should be excised sharply and judiciously to provide an even, perpendicular edge.

Hemostasis: This must be achieved to prevent subsequent hematoma formation and possible infection and wound breakdown. Large arterial and venous bleeders can be clamped and tied. Smaller vessels can be electrocoagulated. At times, packing the wound may be necessary to control hemorrhage if bleeding is profuse (eg, intranasal bleeding).

Abrasions: The wound should be meticulously irrigated and scrubbed with a surgical soap to remove any debris that may cause a permanent tattoo. After application of an antiseptic solution, the area should be covered with a dressing to allow for eschar formation. Contusions generally require no treatment. Occasionally, a subcutaneous hematoma liquefies and may need draining. Contusions over the neck must be carefully examined and followed because of possible injury to the carotid system, larynx, or trachea.

Primary repair of lacerations: This provides the most satisfactory esthetic and functional result in most cases. The repair begins in the deepest extent of the wound and continues in layers to the dermis, using an absorbable 3-0 or 4-0 suture material. If the deepest portion involves the oral mucosa (ie, a through-and-through wound), the oral mucosa is generally closed first. The dermis is then accurately aligned and repaired with buried 4-0 or 5-0 absorbable sutures, which should produce close approximation of the skin margins. Any tension should be relieved by this layered closure. Skin sutures should be
Management of Specialized Structures

Auricular Trauma

Contusions, lacerations, and segmental losses of the auricle are frequent because of its exposed position away from the temporal bone. In all cases of auricular trauma the external auditory meatus and the tympanic membrane should be thoroughly evaluated. The blood supply to the auricle is abundant; therefore, debridement of soft tissue should be very conservative. Small tissue fragments and/or skin margins that appear to be partially compromised should be included in the repair if they are structurally important.

Simple lacerations: Accurate placement of sutures is essential, especially if the helix is involved. The helix should be sutured first to restore proper anatomy and contour. The posterior skin is closed using 4-0 or 5-0 monofilament suture material if the laceration involves this layer. If the laceration is small, there usually is no need to suture cartilage. However, lacerations involving the cartilage in which there is lack of support of the cartilage need to be sutured with 5-0 absorbable material in a simple or figure-eight fashion. The lateral or anterior skin is closed with interrupted 5-0 or 6-0 monofilament sutures.

Segmental avulsion: An avulsed segment that is no larger than 3 cm should be treated as a composite graft. Dermal contact between the vascularized and nonvascularized segment is critical, and it is often necessary to resect a portion of the cartilage to ensure good dermal opposition. Use of antibiotics, heparinization, supportive dressings, and ice to the replanted segment are important to the success of the graft. With larger avulsed segments, the skin of the avulsed segment should be dermabraded, the cartilage and retroauricular skin of the segment coapted to the retroauricular skin and cartilage of the intact auricle, and then the segment buried in a posterior auricular skin pocket. Several weeks later the flap is separated, and the avulsed segment is elevated from the bed. The postauricular defect is skin grafted.

Abrasions: If the perichondrium is not exposed, the abrasion should be treated like any other abrasion and allowed to reepithelialize by secondary
intention. If the perichondrium is exposed or missing, the site should be skin grafted using retroauricular skin from the contralateral side.

Hematoma: Should be treated promptly by aspiration or incision and drainage. After evacuating the hematoma, a carefully adapted pressure dressing must be applied.

Eyelid Injuries

The presence of any eyelid or periorbital contusion, laceration, or hematoma should prompt a thorough examination of the globe. Even small lacerations can be associated with a penetrating injury to the globe, especially if orbital fat is visualized.

Superficial lacerations of the skin and muscle: Close in layers in the usual fashion using 5-0 absorbable material for the muscle and 6-0 or 7-0 nylon for the skin.

Marginal lid lacerations or tarsal lacerations: Best managed with a conjunctivotarsal repair using inverted absorbable suture material and then an orbicularis-skin repair as described previously. Proper alignment of the lid margin and the global surface of the laceration are critical for cosmesis and function of the lid against the cornea. Use of 7-0 silk sutures, one at the ciliary line, one at the gray line, and one at the orifices of the meibomian glands, will ensure proper alignment. These sutures are cut long and tied over the skin closure sutures to prevent abrasion of the cornea.

Lacerations to the medial canthal region: Such injuries may lacerate the lacrimal canaliculus. To assess patency, fluorescein is carefully injected through one punctum while carefully obstructing the other with finger pressure. A patent lacrimal canaliculus is indicated by the appearance of the fluorescein in the nose. Reconstruction of a canaliculus laceration is performed over a fine silicone tube (Crawford tube) under the operating microscope using 9-0 or 10-0 nylon sutures.

Facial Nerve Trauma (see also chapter 22)

Anatomy: After exiting the stylomastoid foramen, the facial nerve passes in front of the posterior belly of the digastric muscle and lateral to the styloïd process and external carotid artery. The nerve reaches the posterior border of
the mandible 2.5 to 3.5 cm superior to the mandibular angle and bifurcates in the parotid gland into the temporofacial and cervicofacial trunks. Further subdivisions occur to form five terminal branches: temporal, zygomatic, buccal, marginal mandibular, and cervical. Anastomoses between these branches occur with varying frequencies. The buccal and zygomatic branches often exhibit interconnections, whereas the temporal and marginal mandibular branches have little interconnection. Thus, the temporal or marginal mandibular branches are more prone to irreversible injury. Anterior to the parotid gland, the temporal, zygomatic, and buccal branches lie deep to the superficial masseteric fascia. The mandibular and cervical branches lie deep to the platysma muscle. As these branches proceed anteriorly, they perforate the respective muscle fascia and innervate the appropriate facial muscles.

General considerations

- The most vulnerable site of injury secondary to either blunt or penetrating trauma is anterior to the parotid gland.
- Assessment of facial nerve function is important in all facial trauma patients. This assessment must be made prior to use of a local anesthetic.
- Nerve deficits anterior to a vertical line drawn from the lateral angle of the eye will usually resolve spontaneously.
- Ideally, nerve repairs should be performed within 72 hours after injury. If multiple traumas preclude early repair, the lacerations should be explored and, using a nerve stimulator, the distal severed ends should be identified and tagged with a suture to facilitate retrieval at a later time. The ideal time for delayed repair is 3 to 4 weeks postinjury.
- If a parotid duct injury is suspected, and a facial nerve deficit is noted or suspected, do not inject a dye such as methylene blue or brilliant green into the duct. Spillage of such dyes will make identification and repair of severed nerves difficult under the operating microscope. Use a lacrimal probe or inject saline or infant formula into the duct orifice to aid in identification of a duct laceration. Infant formula is water soluble and will not stain nerve fibers or surrounding structures.

Specific treatment considerations

- The primary goal of nerve repair is to accurately restore anatomy without tension, preferably with an epineural anastomosis. Tension
results in fibrosis, which prevents nerve regeneration.

- Resection of the proximal and distal stumps to remove damaged or fibrotic fibers is critical to the success of a repair.
- An epineural repair with 9-0 or 10-0 nylon is recommended.
- If repair cannot be achieved without tension, a sural or greater auricular nerve graft is indicated. When a graft is used, an epineural repair is done.

Parotid Duct Injury

Anatomy: The parotid duct is 6 to 7 cm in length and 2 to 4 mm in diameter. It leaves the gland, often accompanied with an accessory lobe, at the anterosuperior border and crosses the masseter muscle. It turns medially at the anterosuperior border of the masseter and passes through the buccal fat pad and buccinator. The duct empties into the oral cavity at the tip of the papilla. The buccal branch of the facial nerve and transverse facial artery are superior to the duct.

Diagnosis: A complete transection secondary to a laceration can be identified by cannulating the distal end of the duct with a lacrimal probe. Small ductal lacerations can be identified by retrograde injection of methylene blue if there is no facial nerve injury, or using sterile saline or infant formula if a facial nerve deficit is noted.

Repair of a transected duct in the proximal one-third: If access is good and the proximal end can be well isolated, a repair with 8-0 or 9-0 nylon over a silicone catheter is indicated (see chapter 20, page 309). The catheter should remain in place 10 to 14 days. However, if the repair is difficult, the injury is usually treated by ligation of both the proximal and distal ends. Atrophy of the gland will usually ensue.

Injury to the duct as it passes through the buccal fat pad and buccinator muscle: The injury is either primarily repaired, as above, or the proximal severed end is rerouted into the oral cavity posterior to the papilla. When the latter is done, care should be taken to reroute the proximal end obliquely through the buccinator so as to restore the sphincter mechanism. The cut margin of the proximal end is sutured to the oral mucosa and a silicone catheter is inserted and left in place for 7 to 10 days.

Dressing: After repair of the facial laceration at the site of ductal injury, a
pressure dressing is placed for 72 hours. Prophylactic antibiotic coverage is advisable.

Fractures of the Facial Skeleton
Frontal Sinus Fractures

Diagnosis: Primarily made by inspection and neurologic evaluation. The following findings should lead you to suspect such a fracture:

- Epistaxis
- Anterior cranial contusion, hematoma, laceration
- Depression of the glabellar area
- Periorbital emphysema
- Cerebrospinal fluid leak
- Pneumocephalus
- Presence of nasal, orbital, and midface injury
- Neurologic deficits indicative of frontal lobe injury

Radiographic evaluation

- Plain films (lateral and anteroposterior skull, Waters, and Caldwell views) should be assessed for sinus clouding, air-fluid levels, fractures, and asymmetries. Posterior sinus wall and floor fractures are difficult to assess.
- Computed tomography (CT) is the study of choice to thoroughly evaluate frontal sinus fractures.

Treatment considerations

- Nondisplaced anterior wall fractures require no treatment. Displaced anterior wall fractures require reduction and often fixation. Exposure is made either through lacerations, an open-sky incision, or a coronal flap.
- Management of nasofrontal duct injuries is controversial. The Lynch fron-toethmoidectomy procedure and attempts at splinting the duct with a drainage tube have been accompanied by significant complications. Contemporary literature supports not treating the duct injuries and treating any complications endoscopically.
- Nondisplaced posterior wall fractures generally require observation for signs of a cerebrospinal fluid leak. Displaced and/or comminuted fractures should be managed with the neurosurgical service. A coronal
approach is the choice for exposure. Management of posterior wall fractures is also controversial and varies from repair of the dura mater and reduction of the wall fractures to complete cranialization and obliteration of the ducts.

Complications
- Sinusitis
- Persistent cerebrospinal fluid rhinorrhea
- Pneumocephalus
- Mucoceles or mucopyoceles

Supraorbital Rim Fractures

Diagnosis
- Ecchymosis
- Laceration
- Deformity on inspection or palpation
- Paresthesia in distribution of supraorbital nerve
- Displacement of globe
- Upper lid ptosis
- Superior orbital fissure or apex syndrome
- Extraocular muscles restricted in upward gaze

Radiographic evaluation
- Examine Waters, Caldwell, and lateral skull views for irregularities of rim and roof of orbit.
- CT is the study of choice. Coronal and sagittal views are very helpful.

Treatment considerations
- Assessment of any globe injury is critical.
- Reduction of displaced fragments can be achieved through a laceration at the site, a brow incision, or a coronal flap.
- Fixation is accomplished either by direct wiring or bone plating.

Nasal Fractures

Diagnosis
- Edema
• Ecchymosis
• Epistaxis
• Lacerations
• Obstruction to airflow
• Deviation or depression of nasal pyramid
• Pain, mobility, crepitation on palpation
• Septal hematoma, lacerations, deviation, or dislocation

Radiographic evaluation
• Waters, axial, and lateral soft tissue views of nasal pyramid may reveal a fracture.
• In 10% to 40% of patients with nasal fractures, radiographs appear normal. The physical examination is usually more valuable in diagnosing nasal fractures.

Treatment considerations
• Displaced fractures are best treated within 2 to 3 hours of the incident. Significant edema or hematoma over the nasal pyramid often complicates the diagnosis. Additionally, proper reduction is often difficult in the presence of significant edema or hematoma formation. Therefore, if not done early, it is better to wait 7 to 10 days to reevaluate form and function. Delayed treatment usually does not affect the result.
• Closed reduction of both the nasal pyramid and the septum is adequate in most cases. Topical and local anesthetics can be used effectively when performing these procedures. Use of a blunt-end elevator placed under the depressed or laterally displaced bone, while palpating and directing pressure with the opposite forefinger and thumb, is the most sensitive method of reduction. Often, a laterally displaced fracture is of the green-stick type, and the fracture must be completed to reduce it. Septal deviations or dislocations can be reduced effectively with an Asch forceps.
• After successful reduction of the fractures, apply an external cast or splint, which aids in maintaining reduction of fragments and reduces edema and hematoma formation. Maintain the cast or splint for 7 days. Intranasal packing is usually not necessary if reduction is successful and if epistaxis is minimal.
• Indications for open reduction include impacted bony fragments that
cannot be reduced; fractured and dislocated septal elements that cannot be reduced; or compound fracture of the dorsum, the tip, or the supratip region.

- Access to the nasal bones can be achieved through lacerations, an open-sky incision, an incision directly over the bridge, Lynch incisions, or a coronal flap.
- Access to the bony and cartilaginous septum is best afforded by a unilateral hemitransfixion incision. Overriding septal cartilage is managed by a minimal resection at the site of fracture or dislocation and direct suturing with an absorbable suture material. Fragments of the vomer or perpendicular plate of the ethmoid that are either compounded into the nasal passage or restricting reduction of the cartilage should be resected.
- Dislocated upper lateral cartilages are usually reduced when the septal cartilage and nasal bones are adequately reduced. However, compound fractures must be thoroughly evaluated for dislocated or lacerated upper or lower lateral cartilages. Following reduction of supporting structures, such dislocations or lacerations should be reduced and fixed directly with absorbable sutures.

Complications

**Early**
- Epistaxis
- Septal hematoma

**Late**
- Synechiae
- Webbing of the valve area
- Bony and cartilaginous deformities secondary to scarring

Naso-orbital-ethmoidal Fractures

Naso-orbital-ethmoidal fractures most often consist of posteriorly impacted and laterally displaced nasal bones, frontonasal processes of the maxilla, and
lacrical bones. This type of injury often is accompanied by frontal sinus, frontobasilar, and high Le Fort fractures.

Diagnosis

- Periorbital hematoma and/or edema
- Lacerations
- Epistaxis
- Cerebrospinal fluid leak
- Pneumocephalus
- Subconjunctival hemorrhage
- Depressed interorbital space and/or orbital hypertelorism
- Crepitation and mobility of the naso-orbital complex on palpation
- Displaced nasal septum
- Neurologic deficits indicative of frontal lobe injury

Radiographic evaluation

- Review Waters, Caldwell, and lateral skull views for ethmoidal sinus clouding and obvious lateral or posterior displacement of the complex.
- CT is the study of choice. Direct or good-quality reconstructed coronal views are invaluable in assessing the extent of the injury. If time permits, three-dimensional studies can be obtained, which greatly assist in formulating a treatment plan.

Treatment considerations

- Ideally, reconstruction of naso-orbital-ethmoidal fractures is performed in one initial, well-planned procedure. Open reduction and fixation techniques best afford restoration of anatomy and function.
- Adequate exposure to the area is essential. Surgical access is afforded via lacerations, an open-sky incision, Lynch incisions, a coronal incision, and subciliary or transconjunctival incisions if the orbital floor must be exposed.
- Reconstruction starts with stabilization of the orbital rims and frontal bones and proceeds with reduction and fixation of the nasal bones and frontal processes of the maxilla to these stabilized bones. When there is no comminution, rigid fixation is used. When there is comminution, a combination of direct wiring and plating affords the best fixation. Bone grafts are often necessary to reconstruct excessively fragmented bones.
• Next, attention is directed to the orbital floor and medial wall fractures. Autologous and homologous grafts and alloplastic implants are used to reconstruct any defects.
• The lacrimal sac and canaliculi are examined by inspection, catheterization, and dye injection. Any bone fragments should be carefully debrided, and lacerations of the sac and canaliculi should be repaired. Silicone catheters should be used if extensive damage is noted and should be retained for 3 months.
• Attention is now directed to the medial canthal ligaments. Often, the ligaments remain attached to the frontal processes of the maxilla and lacrimal bones and are adequately repositioned with reduction of these bones. At times, the ligaments are avulsed or the attachment area is excessively comminuted and a canthoplasty is necessary. The ligaments are anchored by transnasal horizontal 30-gauge stainless steel wire mattress sutures. A bone graft may be incorporated in the transnasal wiring for support of a comminuted medial rim and wall.
• Juxtaposition of the skin and subcutaneous tissue to the lateral nasal region is necessary and is best afforded by passing a second set of transnasal wires in a horizontal mattress fashion just anterior to the anterior lacrimal crest. Acrylic resin buttons or padded lead plates are contoured and fixed with the wires to coapt the skin over the bony repair. The wires should be twisted snugly to prevent lateral traction of the skin and hematoma formation. The plates and wires are removed in 10 days.
• Last, attention is directed to the nasal septum. Hematomas should be drained, and fractures and dislocations of the septum should be treated as previously outlined.

Complications
• Injuries to regional structures (eg, dura mater, brain, sinuses, globe and adnexa, lacrimal system, nasal system)
• Cosmetic deformities secondary to inadequate primary reconstruction

Orbital Floor and Wall Fractures
Fractures of intraorbital bones may occur independently but usually are associated with other fractures (eg, zygoma, naso-orbital-ethmoidal, Le Fort II and III fractures). In pure blowout fractures the medial wall or orbital floor
are involved because of the thinness of the bone.

**Diagnosis**

- Periorbital edema or hematoma.
- Subconjunctival edema or hemorrhage.
- Infraorbital nerve paresthesia or anesthesia.
- Diplopia in primary or peripheral fields of gaze.
- Enophthalmos when edema has resolved.
- Dystopia.
- Orbital emphysema.
- Ipsilateral epistaxis.
- Positive forced duction test. This maneuver is often subjective, and injury to the globe contents can occur.
- Radiographic evidence.
- Globe injuries are common; the visual screening examination should consist of:
  - Visual acuity check
  - Confrontation fields
  - Extraocular motion
  - Pupillary reactions
  - Examination of anterior and posterior chambers
  - Measurement of intraocular pressure

**Radiographic evaluation**

- Waters, Towne, and Caldwell views are usually obtained and should be reviewed for sinus clouding, orbital emphysema, and possible fractures.
- Axial, coronal, sagittal, and/or three-dimensional CT studies are essential in evaluating orbital floor and wall fractures.

**Treatment considerations**

- Operative versus nonoperative treatment of such fractures is controversial. The trend is to delay operative intervention up to 14 days and intervene if the following signs and symptoms persist or worsen:
  - Enophthalmos
  - Restriction of ocular mobility
  - Vertical malposition of globe (dystopia)
• Significant dehiscence (> 2 cm²) of the orbital floor or medial wall should be explored and reconstructed without delay.
• Surgical approaches to the orbital floor are via subciliary, lid crease, or inferior fornix incisions. A Caldwell-Luc approach also should be considered.
• Surgical approaches to the medial wall are via a coronal flap, a lower lid subciliary incision, and/or a medial upper lid incision. The medial canthal ligaments may have to be mobilized.
• Surgical objectives are to elevate incarcerated tissues and either reduce the fractures or reconstruct the defect with an alloplast or autograft.
• Antibiotics should be used in both nonoperative and operative cases, and nose blowing should be restricted.

Complications
• Hemorrhage and expanding hematomas; blindness can result
• Infection
• Extrusion of alloplasts
• Persistent diplopia
• Persistent enophthalmos
• Ectropion
• Infraorbital nerve paresthesia, anesthesia, or neuralgia

Zygoma Fractures
Fractures of the zygoma are common because of its prominent position and inherent weak buttresses. Many of these fractures are complex, and successful anatomical and functional reduction and fixation can only be gained by correlating the clinical and radiographic findings in a three-dimensional plane.

Diagnosis
• Periorbital edema, ecchymosis, or lacerations
• Subconjunctival hemorrhage and chemosis
• Hematoma in the maxillary buccal sulcus
• Unilateral epistaxis
• Depression of the malar prominence
• Depression of the lateral canthus
• Restricted extraocular movements
- Palpable step deformities
- Dystopia or enophthalmos following resolution of edema
- Infraorbital nerve paresthesia or anesthesia
- Limited range of motion of mandible
- At times, a complaint of malocclusion or limited mouth opening

Radiographic evaluation
- Caldwell, Waters, and submentovertex views should be reviewed for maxillary sinus clouding and separation at the infraorbital rim, zygomaticofrontal suture, and zygomaticotemporal suture areas. The extent of orbital floor involvement is difficult to ascertain.
- Axial and coronal CT has essentially replaced the standard plain films for diagnosis and provides excellent visualization of the orbital floor.

Treatment considerations

*Nonoperative therapy*
- Nondisplaced fractures without signs of orbital floor involvement.
- Patients with minimally displaced fractures should be recalled in 7 to 10 days and examined for cosmetic deformities.

*Operative therapy*
- Contour deformities
- Significant displacement of fractured segments
- Enophthalmos
- Diplopia
- Dystopia
- Restricted range of mandibular motion

*Treatment procedures*
- Closed reduction (towel clip) or open reduction without fixation (Gillies or Keen technique) can be used when orbital floor involvement is minimal. However, reduction is often compromised and recurrent displacement can occur.
• Displaced fractures are best managed by open approaches that allow for adequate fixation techniques and visualization of the orbital floor when involvement necessitates exploration.
  —Zygomaticofrontal suture: brow, upper lid blepharoplasty, hemicoronal, or a subciliary incision with or without a lateral canthoplasty extension
  —Inferior orbital rim and floor: inferior fornix, lid crease, or a subciliary incision
  —Zygomaticomaxillary buttress: buccal sulcus incision

• A minimum of two-point fixation with either transosseous wires or bone plates is recommended.
• The goals of management of orbital floor fractures are to free incarcerated tissues and reduce the fractures. Reconstruction of the floor with either an alloplast or autograft may be indicated.
• Lateral orbital wall fractures are usually reduced when the body of the zygoma is reduced and fixed. However, significant comminution that results in volume loss of the orbit or fragments that restrict globe motion need to be treated. Approaches to the lateral wall are either via an upper lid blepharoplasty incision with a lateral extension or a subciliary incision in the lower lid with a lateral extension. Mobilization of the lateral canthal ligament may be necessary for access.

Complications
• Globe injuries
• Hemorrhage and hematoma formation
• Infection
• Relapse
• Malunion
  —Enophthalmos
  —Dystopia
Isolated Zygomatic Arch Fractures

Diagnosis

- Edema or ecchymosis
- Restricted range of mandibular motion
- Palpable deformity
- Cosmetic deformity after resolution of edema

Radiographic evaluation

- Submentovertex view
- CT

Treatment considerations

**Nonoperative therapy:** Nondisplaced fractures and displaced fractures without coronoid impingement or cosmetic deformity.

**Operative therapy:** Displaced fracture causing coronoid impingement or cosmetic deformity.

**Surgical approaches:** To reduce a displaced arch fracture, Gillies approach or a transoral approach (Keen or Carmody-Batson).

**Fixation:** Need for fixation of reduced fractures is rare. The temporal and masseteric fasciae provide adequate fixation in most cases. In unstable cases, gauze can be packed medial to the fractures, or transcutaneous wire sutures can be placed around the fractured segments and fixed to an acrylic resin or aluminum splint. A hemicoronal incision or extended preauricular incision can also be used for direct wiring or plating of an unstable arch fracture.

Complications

- Infection
- Inadequate reduction
Maxillary Fractures

Maxillary fractures are classified according to the Le Fort classification. Clinically, combinations and/or comminution of the different types of Le Fort fractures are the rule. Nevertheless, the classification system aids in providing a systematic approach to these complex injuries.

Le Fort I fracture: Extends horizontally above the apices of the dentition, across the maxillary sinuses and nasal septum. Posteriorly, the fracture involves the pyramidal process of the palatine bone and the pterygoid process of the sphenoid bone.

Le Fort II fracture: Known as a pyramidal fracture, it extends across the nasal bones and septum, down and posterior through the medial orbital rim or the medial wall, across the inferior orbital rim and then curves posteriorly and inferiorly across the infraorbital foramen and passes under the zygomatic buttress. Posteriorly, it involves the same bones as a Le Fort I fracture.

Le Fort III fracture: Represents a craniofacial disjunction. The fracture lines extend across the orbital floor and then are redirected superiorly and posteriorly through the lateral orbital wall and across the frontozygomatic suture area. The fracture involves the anterior aspect of the zygomatic arch and continues through the pterygoid process of the sphenoid, usually at a more superior level than a Le Fort I fracture.

Diagnosis

- Midfacial edema, ecchymosis, lacerations.
- Signs and symptoms of zygomatic, orbital, nasal, or naso-orbital-ethmoidal fractures (Le Fort II and III).
- Malocclusion secondary to a posteriorly and inferiorly displaced maxilla.
- Mobility of maxilla and/or midface. However, fractures with significant impaction may show little or no mobility.
- Elongated or depressed midface following resolution of edema.
- Cerebrospinal fluid leak, pneumocephalus, or orbital emphysema (Le Fort II and III).

Radiographic evaluation
Waters, Caldwell, and lateral skull views.
CT is favored when complex fractures are evident. Direct or reconstructed coronal studies are especially helpful, as are three-dimensional studies.

Treatment considerations

Le Fort I fracture: The key in treating Le Fort I fractures is an intact mandible. Mandibular or alveolar fractures are usually reduced and fixed first.

- Disimpaction and mobilization of the maxilla is necessary when the maxilla is displaced. Reduction is aided by reestablishing the patient’s pretrauma occlusion. Maxillomandibular fixation is used for 4 to 6 weeks.
- To afford extra stabilization, use of suspension wires should be considered. Wires from the zygomatic arch to the arch bar have the disadvantage of exerting a posterior force and can retract the maxilla. Infraorbital or piriform aperture wires to the arch bar are more efficacious. All suspension wire techniques should be used with caution in comminuted fractures because of the possibility of causing loss of vertical dimension.
- Open fixation techniques (ie, direct wiring and/or miniplate fixation) reduce the time of, or obviate the need for, maxillomandibular fixation. Additionally, vertical stability of comminuted fractures is better afforded by the use of miniplates.
- Sagittal fractures of the maxilla are not uncommon. An acrylic resin palatal splint and/or open fixation technique should be used to prevent rotation of the segments.
- Edentulous or partially edentulous maxillary fractures are treated with the same principles as previously mentioned. However, the patient’s full or partial dentures, if available, are used to reduce and fix the fractures. Full maxillary dentures may be stabilized by suspension wires, palatal screws, or nonthreaded pins. Mandibular dentures are
secured by circummandibular wires.

- Prophylactic antibiotic treatment is indicated.

**Le Fort II fracture**

- Treatment involves disimpaction, mobilization, and reduction by establishing the pretrauma occlusion and using maxillomandibular fixation at the Le Fort I level.
- Craniomaxillary suspension wires are commonly used but have the same disadvantages mentioned for Le Fort I fractures. Open fixation techniques are preferred if additional stabilization is necessary (e.g., through lacerations or open-sky, lower lid, labiobuccal sulcus, and coronal incisions).
- Comminuted Le Fort II fractures are not uncommon. Treatment involves reducing and fixing the maxilla first, followed by reduction and fixation of the midfacial components as outlined under nasal and naso-orbital-ethmoidal fractures. If frontobasilar fractures are present, reduction and fixation of these fractures are performed first to provide a stable base for reduction and fixation of the naso-orbital fractures.
- Prophylactic antibiotic treatment is indicated.

**Le Fort III fracture**

- The same principles are involved as for reduction and fixation of the other types of maxillary fractures.
- Reduction and fixation of the midfacial fractures should proceed in an orderly fashion to stabilize the maxilla and frontal bone structures. Open reduction and fixation techniques are preferred. Primary bone grafting is indicated to reconstruct areas of avulsed or comminuted bone. Prophylactic antibiotic treatment is indicated.

Complications
Le Fort I
- Early: Bleeding, airway obstruction, aspiration of teeth and denture fragments, infection
- Late: Nonunion, malunion, malocclusion, and sinusitis

Le Fort II and III
- Early: Bleeding, airway obstruction, aspiration of foreign bodies, infection, and complications in specific areas already described
- Late: Nonunion, malunion, malocclusion, infection, and complications in specific areas already outlined

Mandibular Fractures
Mandibular fractures are extremely common because of the prominent position of the mandible in the lower third of the face. The mandible is able to withstand considerable compressive force, but when this force exceeds the limits, a fracture may occur in a variety of predictable locations.

Diagnosis
- Pain
- Malocclusion
- Trismus
- Paresthesia
- Hemorrhage
- Ecchymosis/edema
- Lacerations
- Step deformity
- Mobility on palpation
- Fractured or avulsed teeth
- Deviation on opening

Radiographic evaluation
- Mandible series (ie, posteroanterior view of mandible, Towne view, right and left lateral oblique views of mandible)
- Panoramic radiograph
- Occlusal views
- CT is useful in evaluating intracapsular and condylar neck fractures

Treatment considerations: The general principles of treatment are the same as for any other fracture, that is, reduction of the fragments, provision for adequate fixation for a sufficient time to allow for bone repair, and general supportive care and rehabilitation. A plethora of treatment modalities are available, and the selection depends on many interrelated factors, such as the patient’s age and condition, the surgeon’s experience and expertise, the location of the fracture(s), the type of fracture(s), the general state of periodontal health, the presence or absence of teeth, and the availability of equipment.

Closed reduction: Appropriate for most mandibular fractures. Success is dependent on reestablishing the correct alignment of the natural or artificial dentition as it existed prior to the fracture. Ligation of Erich arch bars to the dentition is the most commonly used technique. Maxillomandibular fixation is accomplished using stainless steel wire loops or elastics. Immobilization is maintained for a minimum of 4 weeks. Other treatment modalities are direct interdental wiring (ie, Ivy loops, Stout wires, Risdon wires); lingual, labial, or occlusal acrylic resin splints; circummandibular wires; and transcutaneous skeletal pin fixation devices; eg, Joe Hall Morris device.

Indications for open reduction and fixation
- When closed techniques cannot provide for anatomical reduction
- When closed techniques cannot provide for adequate immobilization
- In the presence of midfacial fractures to establish a foundation to reconstruct the midface (eg, dislocated condylar fractures, displaced fractures distal to the dentition)

Relative contraindications to open reduction and fixation
- Multiple comminuted fragments
The patient is a poor risk secondary to injuries or debilitating diseases.

**Advantages of open reduction:** Provides exact bone alignment and, combined with realignment and fixation of the occlusion, ensures stable immobilization of the fractures and hastens bone repair. Fixation techniques include transosseous wiring, lag screws, and bone plating (compression and noncompression). A relative indication for open reduction and fixation is to lessen the time of, or obviate the need for, maxillomandibular fixation.

**Indications for the removal of teeth in the line of fracture**
- Fracture through a root or bifurcation.
- Excessive mobility of a tooth.
- Periapical pathology and advanced periodontal disease. Considerable controversy exists relative to management of impacted teeth in the line of fracture. Serious consideration should be given to removing impacted teeth that communicate with the oral cavity because local conditions predispose the site to a high incidence of infection. Deep impactions without communication with the oral cavity generally are not removed.

**Multiple fractures of the mandible:** Often require open reduction and fixation techniques at least at one site, usually the most displaced fracture. Condylar and mandibular neck fractures associated with other mandibular fractures usually require open techniques for the body and/or symphysis fractures to provide for early mobilization of the condylar fracture(s).

**Edentulous fractures:** Can be treated in a number of ways if there is sufficient alveolar ridge and basal bone height. Dentures or Gunning splints can be used to reduce and immobilize fractures if the prosthesis overlays the fracture site. A minimum of three circummandibular wires are necessary to provide stabilization. External rigid fixation techniques and open reduction and fixation techniques can also be used. Fractures of the severely atrophic mandible may require an autogenous bone graft.
Pediatric mandibular fractures: Usually treated with closed reduction and fixation techniques. Reduction and immobilization using the primary and mixed dentition is often a technical challenge. In addition to standard arch bar techniques, eyelet loops, circummandibular wires, piriform wires, and acrylic resin overlay, occlusal splints should be a part of the armamentarium. Immobilization of most fractures is for 14 days. If open reduction and fixation are necessary, the developing tooth buds must be avoided. Monocortical wires should be used. Condylar fractures in children are most often treated by closed reduction. Nondisplaced fractures without a change in occlusion are treated with active function. Displaced or dislocated intracapsular and high mandibular neck fractures with occlusal changes are immobilized for 7 to 14 days, followed by the use of training elastics to maintain the occlusion. Dislocated, low mandibular neck fractures may require open reduction and fixation. Close, long-term follow-up is necessary in all condylar fractures in children to observe for possible mandibular growth disturbances.

Antibiotic treatment: Warranted for all compound fractures and dentoalveolar fractures. Penicillin remains a good empiric choice.

Considerations in specific regions

Coronoid process fractures: Rarely require open reduction and fixation. The temporalis tendon and masseteric fascia usually limit displacement, and bony union will occur in most cases.

Ramus fractures: Unusual and, if present, generally show little displacement. Closed reduction and fixation are the usual treatments of choice.

Angle fractures with no or minimal displacement: Treated by closed reduction and immobilization. Displaced fractures usually require open reduction and fixation because of their position posterior to the dentition. Open reduction and fixation can be performed either transorally or transcutaneously.
Body fractures: Can usually be treated by closed reduction and fixation techniques if the dentition anterior and posterior to the fracture is sound. Lack of dentition, significant displacement, and the necessity to mobilize the mandible secondary to condylar fractures may require open reduction and fixation.

Symphysis and parasymphysis fractures: Often require special attention. Open reduction and fixation, transorally or transcutaneously, are often indicated if displacement is present because of the muscle forces exerted in this area. Additionally, condylar fractures frequently occur in conjunction with these fractures. If early mobilization is necessary, additional transosseous fixation besides an arch bar is necessary to prevent mobility and displacement of the segments during function. A lingual acrylic resin splint can be of great aid in either closed or open reduction and fixation techniques.

Condylar and mandibular neck fractures: Common. Treatment modalities are controversial. The treatment philosophy of closed reduction for all types of condylar and mandibular neck fractures is not always achieved (ie, anatomical reduction of the fragments, sufficient time of immobilization, and rehabilitation). Thus, results are often compromised. Modern surgical instrumentation and operative techniques justify open reduction and fixation of significantly displaced or dislocated condylar or mandibular neck fractures. The degree of displacement, the location of the fracture(s), the condition of the dentition, the age of the patient, and the presence of other facial fractures dictate the treatment modality to be chosen. Suggested guidelines are as follows:

- Displaced or dislocated fractures of the condyle within the capsule, most of which exhibit comminution, are usually treated by closed reduction, maxillomandibular immobilization for a brief time, and then active mobilization to prevent fibrous and bony ankylosis. If there is obstruction to function due to displaced fragment(s), the fragment(s) must either be reduced and fixed, if possible, or removed.
- Nondisplaced or slightly displaced mandibular neck or subcondylar fractures require closed reduction and
maxillomandibular immobilization for 4 to 6 weeks.

- Moderately displaced mandibular neck or subcondylar fractures can be treated by closed reduction, but a loss of the vertical dimension on the ipsilateral side is often the result. It is recommended to immobilize the mandible for a brief period and then use maxillomandibular training elastics in an attempt to maintain pretrauma occlusion and function. Open reduction and intrafragment fixation with a miniplate or a lag screw ensure maintenance of the vertical dimension.

- Dislocated mandibular neck or subcondylar fractures have a greater propensity for posttreatment dysfunction than displaced fractures. Closed reduction can be used, but early mobilization and training elastics are necessary in an attempt to control the position of the mandible. Open reduction and intrafragment fixation afford anatomical repositioning and allow bony union to occur.

- Open reduction and fixation of displaced and dislocated mandibular neck and subcondylar fractures are usually necessary when concomitant midfacial fractures exist that require an anatomically positioned mandible to reconstruct the midface.

- The surgical approach to an intracapsular fracture, when necessary, is through a preauricular incision. Approach to a mandibular neck fracture can be via either a preauricular, retromandibular, or submandibular incision. The preauricular approach may predispose to a limited range of motion secondary to fibrosis of the capsule and ligaments. The retromandibular incision provides the most direct approach. The submandibular approach to a mandibular neck or subcondylar fracture is made with an incision approximately 2 to 3 cm long inferior and posterior to the angle of the mandible. It is often necessary to place the screws percutaneously.

Complications
Principles of Managing Panfacial and Craniofacial Trauma

The development and refinement of level I regional trauma centers has enabled maxillofacial trauma surgeons to treat a greater number of patients with severe, complex facial and craniofacial trauma than were treated prior to the mid-1960s. As a result, new treatment protocols and modalities, such as craniofacial techniques, primary bone grafting, and open reduction and rigid fixation, have been developed to better manage these cases. The principles of fracture management in specific areas have already been outlined. Management of panfacial and craniofacial fractures requires adherence to these principles. In addition, it requires a well-considered treatment plan implemented in a stepwise, orderly fashion.

Caudad to cephalad reconstruction: This is the usual method of treatment for panfacial trauma without cranial involvement or with cranial trauma and avulsed bone. Treatment starts intraorally, when necessary, by debridement, reducing and fixing dentoalveolar fractures, and closing oral lacerations. Mandibular fractures are then reduced and fixed to restore the correct three-dimensional anatomy that existed pretrauma. When indicated, open reduction and fixation techniques, especially for condylar fractures, should be used to restore anatomy. Next, maxillary fractures are reduced and fixed to the intact mandible, which establishes the correct anteroposterior dimension. Midfacial fractures are treated by working from lateral to medial. The vertical dimension is then addressed. Suspension wires are satisfactory if sufficient
bone contact exists to establish the vertical dimension. Often, however, comminuted fractures are present, and open reduction and rigid fixation best afford establishment of the vertical dimension. In addition, primary bone grafting is often necessary.

Cephalad to caudad reconstruction: Indicated in the presence of frontobasilar and/or supraorbital rim fractures without avulsed segments and at times when midfacial trauma coexists with severe, comminuted mandibular fractures. First, any neurosurgical procedures are completed and the cranial base is reduced and fixed. Midfacial fractures are then reduced and fixed to the established cranial base, usually in a lateral to medial fashion. From this point, treatment can proceed in one of two ways. If the mandibular fractures can be reduced and fixed, the mandible is treated first, followed by reduction and fixation of the maxilla to the mandible. Vertical dimension is established as previously outlined. If the mandibular fractures are severely comminuted, the maxilla is reduced and fixed directly to stable zygomatic and piriform buttresses. The mandible is then reduced and fixed to the reconstructed craniomaxillary complex.

Surgical approaches to the cranial base and midface: The coronal flap is versatile and provides access to the frontal region of the cranium, naso-orbital region, zygomatic arches, and zygomaticofrontal regions. Other approaches that are commonly used are the subciliary, lid crease, inferior fornix, open-sky, lateral brow, and buccal vestibular incisions.
Infections should be treated promptly and aggressively to avoid the following complications:

- Spread to potential fascial spaces and airway compromise
- Orbital and intracranial spread by direct extension or via the facial and nasal veins
- Spread into the neck, with large-vessel complications (carotid erosion or venous thrombosis)
- Septic shock from gram-negative organisms
- Loss of tissue, including bone and teeth
- Scarring and sinus tracts or fistulae with facial disfigurement

Initial Evaluation (Quick Survey)
Clinical Considerations

Airway

*Neck (deep cervical spaces)*: External swelling, neck rigidity, pain, dysphagia, dysphonia, tracheal deviation

*Tongue*: Elevated, edematous, protruded, and immobile

*Floor of mouth*: Elevated with full, tense plicae

*Palate*: Unilateral involvement with palatal draping, uvular deviation and erythema; bilateral involvement with uvula extended posteroinferiorly

*Parapharyngeal space*: Asymmetric bulging of pharyngeal wall;
dysphagia; stridor, if severe involvement

*Mediastinal spread:* Distant heart sounds, murmur, or pericardial friction rubs; heart (pump) failure

Neurologic manifestations

**Symptoms**
- Headache, chills or fever, pain, and/or rigidity in neck or back, nausea, visual disturbances, or photophobia.
- Infants, the elderly, and debilitated patients may not be able to provide adequate history; therefore, be vigilant for signs of agitation or changing (depressed) levels of consciousness.

*Signs and symptoms:* Intracranial spread of infection, brain abscess, and edema will produce a mass effect with the following:
- Paresis, ataxia
- Decreased visual acuity, papilledema, nerve palsy, ophthalmoplegia
- Focal seizure activity
- Nausea and vomiting
- Progressively depressed sensorium leading to coma

Possible Need for Hospitalization

*Immunocompromised patient:* Uncontrolled diabetic, alcoholic, malnourished, or otherwise immunodeficient patient

*Systemic involvement:* Fever (> 101.5°F), malaise, fatigue, dehydration, inability to take fluids, and signs of organ failure

*Patient compliance:* Patient unreliable or incapable of properly caring for self

*Rapid spread:* Skin color changes or sloughing suggestive of
necrosis, trismus, paresthesia

Need for parenteral antibiotics

Special features: For example, resistant organisms, osteomyelitis, actinomycosis

Diagnostic Workup

Patient Assessment

History

- Duration of infectious process
- Sequence of events and changes in symptoms or signs
- Antibiotics prescribed, dosage, and response
- Review of systems with emphasis on neuro-ophthalmologic, cardiopulmonary, and immune systems
- Social history: Exposure, travel (fungal or parasitic infections), chemical dependency

Physical examination

Head and neck examination

- Swelling, asymmetry, and loss of normal anatomical borders or contours
- Abscess (fluctuance) versus cellulitis
- Lymphadenopathy
- Trismus, alteration of facial animation
- Nasoendoscopy
  —Discharge from ostia or ethmoid bullae (acute sinusitis)
  —Mucopurulent exudate
  —Pharyngeal fullness or asymmetry, soft palatal fullness or deviation of uvula

- Integument
  —Draining fistulae or sinus tracts
  —Tissue slough or dusky appearance (necrotizing
—Change in sensation—decreased in necrotizing fasciitis; increased in early cellulitis or viral (herpes) infection
—Crepitation (gas production)
—Rash, erythema migrans (Lyme disease)

**Neuro-ophthalmologic examination**

- Altered mental status
- Neck rigidity; positive Kernig sign (with the hip and knee flexed 90 degrees, extending the knee causes pain) is evidence of meningitis
- Focal neurologic features
  - Motor and sensory deficits
  - Seizures
  - Nausea and vomiting

- Ophthalmologic features
  - Proptosis, chemosis, dacryocystitis
  - Visual field defects
  - Photophobia
  - Ophthalmoplegia (cranial nerves III, IV, and VI) with sluggish or fixed, dilated pupils
  - Diplopia (from mass effect of a postseptal or orbital abscess)

**Mediastinal examination:** Spread of infection from the head to the mediastinum involves contiguous routes through fascial spaces. Mediastinitis is characterized by:

- Dyspnea, pleuritic or substernal chest pain
- Friction rubs and distant heart sounds
- Distended neck veins
- Septicemia and high fever
- Mediastinal widening radiographically
Laboratory Studies
Serum chemistry

Fever and dehydration
- Decreased Na\(^+\) and Cl\(^-\) if excessive sweating
- Increased Na\(^+\) and Cl\(^-\) if volume depleted
- K\(^+\) and HCO\(_3^-\) remain unchanged
- Blood urea nitrogen (BUN) may be elevated

Septic shock
- Exaggeration of above findings
- Evidence of acute renal failure
  — K\(^+\), Cl\(^-\), and volume retention
  — Renal (metabolic) acidosis with compensatory HCO\(_3^-\) reduction

Serum albumin: Levels may decrease in osteomyelitis and necrotizing infections (streptococcal and anaerobic), particularly in compromised patient.

Hematology

Leukocytosis (> 12,000/mm\(^3\))
- Acute infection is characterized by high white blood cell count with shift to the left (band and segmented forms of neutrophils present).
- Chronic infection is associated with normal or mildly increased white blood cell numbers.
- Overwhelming acute bacterial infections and some viral illnesses may result in leukopenia (< 5,000/mm\(^3\)).

Normocytic, normochromic anemia: Particularly in chronic
infections, osteomyelitis, and during convalescence from acute infection

*Thrombocytosis* (platelet count > 500,000/mm³): In early stages of infection

*Erythrocyte sedimentation rate (ESR)*
- Nonspecific increase with most bacterial and fungal infections
- No increase with most viral infections

Urinalysis
- Proteinuria with extensive infections
- Slight increase in specific gravity (> 1.025)
- Oliguria and anemia in septic shock or early phase of acute tubular necrosis due to volume depletion and renal hypoperfusion

Imaging

Plain films
- Examination of dental structures.
- Osseous and soft tissue densities. Bone changes are evident only after 5 to 14 days of infection and 35% to 50% demineralization.
- Lateral and frontal cervical soft tissue films to demonstrate encroachment on the airway.
- Sinus views.

Computed tomography (CT) scanning (usually with contrast)
- Good for determining extent of space infections
- Demonstrates airway constriction or compromise
- Determines spread of infection to contiguous structures: sinuses, orbits, neck, intracranial structures

Nuclear bone scans

*Technetium (Tc 99m)*: Labeled phosphate anion
- Half-life of 6 hours with 0.3 rad total body radiation
- Images bone as an organ system
- Triphasic scan images
  - Soft tissue flow and blood pool
  - Bone (delayed phase)
- High sensitivity, low specificity
- Normally shows uptake in sinuses, thyroid, spine and joint articulations, active cranial sutures, bladder
- Good for imaging acute osteolytic, chronic osteoblastic, and healing responses in osteomyelitis

**Gallium (Ga 67)**
- Half-life of 78 hours with 1.5 rad total body radiation
- Binds to albumin and rapidly dividing cells (tumor, white blood cells)
- High sensitivity, low specificity
- Localizes infective focus within 2 to 3 days of onset
- Diagnosis of biologic activity; particularly useful in determining efficacy of treatment

Magnetic resonance imaging (MRI)
- Noninvasive; no radiation exposure
- High soft tissue resolution
- High sensitivity and specificity
- Good for imaging extent of space infections, presence of pus, cavitation
- Gadolinium enhancement for vascularity, particularly in osteomyelitis

Ultrasonography
- Images soft tissue cavities and fluid-filled spaces
- Good for identifying infected cysts and salivary glands
- Demonstrates early infection
- Healing responses can be quantified

Positron emission tomography (PET) scan
Brain abscess with central enhancement
Excellent sensitivity in chronic osteomyelitis when other tests not helpful

Aspiration
- Dark, malodorous pus is indicative of anaerobic infections.
- White-yellow pus implicates aerobic gram-positive cocci (streptococci, staphylococci).
- Dark-stained fluid (watery or thick) is often produced by gram-negative enteric bacteria.
- “Sulfur” granules in yellowish exudate implicates Actinomyces organisms.
- Gas, with or without pus, suggests clostridial or anaerobic infections.

Culture and Antibiotic Sensitivity Testing
Indications
- Rapidly spreading or extensive infection
- Infection in compromised patient
- Infection not responding to antibiotics
- Recurrent or recalcitrant infections
- Osteomyelitis
- Postoperative infection
- Infections with unusual features
  — Tissue necrosis
  — Gas production
  — Chronic or multiple fistulae or sinus tracts
  — Hospital-acquired infections
  — Associated sensorimotor loss

Sampling Techniques
Aspiration
- Prepare site by scrubbing with germicidal soap, povidone-iodine, or chlorhexidine solution, and then cleanse with sterile saline-impregnated gauze.
- Insert an 18- to 20-gauge needle using gentle negative pressure once it is
subcutaneous.

- Remove needle, expel air from syringe, and place aspirate into culture medium or cap syringe and transport immediately to laboratory.
- Fistulae and sinus tracts should be superficially cleansed and a catheter introduced for aspiration in a syringe.

Swabbing

- Indicated for direct sampling of infected tissue or exudate; however, it is the least reliable method for obtaining a specimen for culture because of possibility of local contaminants.
- There are commercially available swabs containing transport medium.
- Surface infections are sampled by first cleansing with repeated sterile saline irrigation, then swabbing the surface once or twice; only aerobic cultures are performed for surface lesions.

Excised tissue: Tissue is the optimal specimen for culture and antibiotic sensitivity testing. It contains established colonies, yields significant numbers for culture, and serves as its own reduced culture medium for anaerobic infections.

- Excision and submission to laboratory should be performed within several minutes.
- Transport tissue in saline-moistened gauze, normal saline solution or, optimally, transport medium.
- Submit for microbiologic and histologic evaluation.
- Chronically infected bone should be placed in anaerobic vials or culture medium that will support anaerobes.

Microbiologic Evaluation

Direct Gram stain: Useful information may be obtained from a Gram-stained direct smear. Confirmation of a good specimen is based on the presence of neutrophils, high density of organisms, and absence of epithelial (surface contaminant) cells. See chapter 3 for technique.

Other useful stains to be ordered from laboratory

- *Periodic acid–Schiff stain*: Used for fungal organisms
- *Acid-fast stain*: Used for mycobacteria and several *Nocardia* species
- *Wet preparations*: Good for *Actinomyces* organisms and fungal colonies
Antimicrobial Susceptibility Testing
- Based on minimum inhibitory concentration (MIC).
- Therapeutic antibiotic dose is three to four times the MIC; in compromised host it is eight times the MIC.
- Higher therapeutic doses are necessary for protein-bound antibiotics (eg, aminoglycosides).
- Minimum bactericidal concentration (MBC) is the antimicrobial concentration that kills 99.9% of bacteria.
- Bacteriostatic agents (eg, tetracycline) may exhibit no MBC.
- Organisms are considered antibiotic resistant if MBC > MIC by 32-fold.

Blood Cultures
- Indicated for all serious head and neck infections when sepsis is present.
- Best performed at the beginning of chill or spike in fever. Done at two extremities at 5-minute intervals. Two culture bottles should be used for each site.
- Sterilize venipuncture site; draw 10 mL blood. Place the first 5 mL in the anaerobic bottle, then place the second 5 mL in the aerobic bottle.

Polymerase Chain Reaction (PCR)
- Early and rapid identification of some offending organisms
- Aids in early initiation of treatment
- Useful for specific infections, eg, mycobacteria, human immunodeficiency virus (HIV), cat scratch, syphilis
- Sensitivity, 90% to 100%; specificity, 100%

Principles of Infection Management
Management of orofacial infections generally combines medical and surgical therapy.

Medical Therapy
Nutritional support (see also chapter 7)
- Adult man, 20 to 30 kcal/kg/d.
- Younger patients require slightly more calorie intake; elderly patients require less.
- Sepsis increases caloric requirements (13% for every degree centigrade rise in temperature).
- Protein requirement for a young adult is 0.8 g/kg/d.
- Iron, magnesium, and trace elements must be carefully monitored in septic patients.

Antibiotic therapy

**Indications**
- Extensive or unusual infections
- Systemic spread or sepsis
- Chronic infections and those unresponsive to surgical therapy
- Infections in the debilitated patient
- Infections in an operative site or in the hospitalized patient

**Empiric therapy** (use parenteral rather than oral route if infection is severe)
- Most orofacial infections are odontogenic and therefore have predictable organisms.
- Penicillin is the antibiotic of choice. It is effective against most gram-positive cocci and anaerobes; 5% to 7% of the population is allergic to penicillin.
- For penicillin-allergic patients (noncompromised), alternatives are clindamycin, cephalosporins (some reported cross-sensitivity in patients with severe allergy to penicillin), macrolides, quinolones, and tetracyclines.
- For compromised hosts, and previously hospitalized patients, use:
  - Oral: clindamycin, β-lactamase inhibitor combination (amoxicillin/clavulanate), or metronidazole (+ clindamycin or penicillin)
  - Intravenous (IV): cefoxitin, ceftriaxone, clindamycin, vancomycin, β-lactamase inhibitor combination (ampicillin/subbactam; piperacillin/- tazobactam; ticarcillin/clavulanate), quinolones (ciprofloxacin), and
carbapenems

- Cephalosporins have limited anaerobic action and there is 8% to 10% cross-reactivity in patients who have accelerated reactions (anaphylaxis) to penicillin.
- Erythromycin is a broad-spectrum agent, but is less effective than penicillin.
- Metronidazole is effective against anaerobes only.
- Vancomycin for methicillin-resistant staphylococci and susceptible enterococci.
- Quinolones for established or chronic osteomyelitis.
- Spectrum of antibiotic activity is shown in Table 17-1.

**Dosage (Table 17-2)**

- Three to four times the MIC is usually sufficient; eight times in compromised patients.
- Toxicity may ensue if greater doses are used.
- Higher levels may be justified if the infected site is isolated (eg, chronic nonsuppurative osteomyelitis).

**Route**

- Oral
  - Variable absorption
  - Consistent dosing important
  - Penicillin reaches peak levels at 2 g
  - Most antibiotics should be taken in the fasting state

- Parenteral
  - Serious and established infections
  - Continue parenteral therapy for at least 5 days to ensure complete bacterial killing (acute infections)

**Table 17-1 Spectrum of antibiotic activity**
<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Organism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillin</td>
<td>Most gram-positive cocci, oral anaerobes, <em>Actinomyces</em> spp, <em>Neisseria</em> species</td>
</tr>
<tr>
<td>Penicillinase-resistant penicillins (dicloxacillin, methicillin, nafcillin, azlocillin, mezlocillin)</td>
<td>Some penicillinase-producing staphylococci (not MRSA); <em>Neisseria</em> species, some gram-negative rods</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>Gram-positive (not staph), <em>Haemophilus influenzae</em>, some <em>Bacteroides</em> species*</td>
</tr>
<tr>
<td>Amoxicillin and clavulanic acid</td>
<td>Most gram-positive cocci, <em>Haemophilus</em> species, <em>Moraxella</em> enteric flora, some staphylococci (not MRSA), anaerobes (same as penicillin)</td>
</tr>
<tr>
<td>Antipseudomonal penicillins</td>
<td>Most <em>Pseudomonae</em> and <em>Bacteroides</em>; combine with β-lactamase inhibitor for resistant forms</td>
</tr>
<tr>
<td>(ticarcillin, piperacillin, mezlocillin, imipenem)</td>
<td></td>
</tr>
<tr>
<td>Cephalothin and other cephalosporins (see inserts for specific spectra)</td>
<td>Broad action against most grampositive (not resistant staphylococci) and gram-negative (not <em>Serratia</em>) organisms, <em>Pseudomonas</em>, and resistant <em>Bacteroides</em></td>
</tr>
<tr>
<td>Antibiotic</td>
<td>Activity</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>--------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>Most gram-positive organisms, <em>Actinomyces</em>, anaerobes, some enterococci</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>Gram-positive cocci (most staph resistant), <em>Haemophilus</em>, oral anaerobes, some resistant streptococci and enterococci</td>
</tr>
<tr>
<td>Clarithromycin</td>
<td>Same spectrum of activity as erythromycin; <em>Moraxella</em></td>
</tr>
<tr>
<td>Fluoroquinolones (ciprofloxacin, norfloxacin, ofloxacin)</td>
<td>Some gram-positive cocci, most gram negative cocci; not MRSA</td>
</tr>
<tr>
<td>Tetracyclines</td>
<td>Weak broad spectrum; gram-positive cocci, <em>Haemophilus</em> and <em>Bacteroides</em> organisms, some enteric forms</td>
</tr>
<tr>
<td>Aminoglycosides (eg, gentamicin)</td>
<td>Some staphylococci and most gram-negative organisms; not active against streptococci or anaerobes</td>
</tr>
<tr>
<td>Sulfonamides</td>
<td>Most streptococci (not enterococci), coliforms, <em>Haemophilus</em>; many adverse effects</td>
</tr>
<tr>
<td>Trimethoprim and sulfamethoxazole</td>
<td>Most gram-positive cocci and gram-negative organisms (not <em>Pseudomonas</em> organisms)</td>
</tr>
<tr>
<td>Nystatin, clotrimazole, ketoconazole, fluconazole</td>
<td>Superficial candidal infections, coccidioidomycosis, histoplasmosis</td>
</tr>
</tbody>
</table>
Amphotericin B  Systemic fungal infections

MRSA = methicillin-resistant *Staphylococcus aureus*.

*Bacteroides* species = Oral (*Porphyromonas* and *Prevotella*).

**Table 17-2** Peak antibiotic serum concentrations (mg/mL) with routine dosages

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Dosage</th>
<th>Peak</th>
<th>MIC (susceptible organisms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillin G</td>
<td>1 million U IV</td>
<td>12.0</td>
<td>0.1</td>
</tr>
<tr>
<td>Penicillin V</td>
<td>500 mg oral</td>
<td>3.4</td>
<td>0.1–3.0</td>
</tr>
<tr>
<td>Dicloxacillin</td>
<td>500 mg oral</td>
<td>14.6</td>
<td>0.1–0.2</td>
</tr>
<tr>
<td>Cephalexin</td>
<td>500 mg oral</td>
<td>17.0</td>
<td>2.0–8.0</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>300 mg oral</td>
<td>4.2</td>
<td>0.1–3.1</td>
</tr>
<tr>
<td></td>
<td>600 mg IV</td>
<td>23.0</td>
<td></td>
</tr>
<tr>
<td>Erythromycin</td>
<td>500 mg oral</td>
<td>1.0</td>
<td>0.1–1.0</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>60 mg IM</td>
<td>4.7</td>
<td>0.1–3.0</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>500 mg oral</td>
<td>11.5</td>
<td>0.1–3.0</td>
</tr>
<tr>
<td>Trimethoprim/</td>
<td>80/400 mg oral</td>
<td>2.0</td>
<td>0.1–28.0</td>
</tr>
<tr>
<td>Sulfamethoxazole</td>
<td>500 mg oral</td>
<td>60.0</td>
<td></td>
</tr>
</tbody>
</table>

*IV = intravenous; IM = intramuscular.*

**Combination therapy**

- Life-threatening infections
- Necrotizing fasciitis
- Chronic osteomyelitis (quinolones have good bone penetration)
- Prevention of resistant organisms (eg, *Bacteroides*, staphylococci, mycobacteria)
- Combination therapy in orofacial infections usually involves a broad-spectrum penicillin (or cephalosporin) and an antibiotic active against gram-negative organisms (aminoglycoside or one of the β-lactamase inhibitor combinations)

**Factors requiring adjustment of antibiotic therapy**

- Nonresponsive or superinfections
• Culture and antibiotic sensitivity test results
• Toxicity
  —Aminoglycosides (nephrotoxicity and ototoxicity)
  —Clindamycin, cephalosporins (pseudomembranous colitis)
  —Erythromycin (hepatotoxicity in high doses)
  —Penicillins and cephalosporins (hypersensitivity reactions)
  —Quinolones (tendon, joint inflammation; exacerbation of myasthenia gravis)

**Adjuncts to antibiotic administration**
• Probenecid to increase blood levels in penicillin therapy, 2 g/d in divided doses
• Nystatin for fungal superinfection (*Candida*)
• Antiretroviral and antiviral agents in immunocompromised patients

Hyperbaric oxygen therapy
• Increases vascularity in hypovascular tissues (osteoradionecrosis)

---

**Table 17-3 Presentation and localization of dental abscesses**

<table>
<thead>
<tr>
<th>Teeth</th>
<th>Common location</th>
<th>Less common location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mandibular central incisors</td>
<td>Labial</td>
<td>Lingual</td>
</tr>
<tr>
<td>Mandibular lateral incisors</td>
<td>Lingual</td>
<td>Labial</td>
</tr>
<tr>
<td>Mandibular canines</td>
<td>Labial</td>
<td>Lingual</td>
</tr>
<tr>
<td>Tooth Type</td>
<td>Buccal</td>
<td>Palatal</td>
</tr>
<tr>
<td>-------------------------</td>
<td>-------------------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td>Mandibular premolars</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mandibular molars</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maxillary central incisors</td>
<td>Labial</td>
<td>Palatal</td>
</tr>
<tr>
<td>Maxillary lateral incisors</td>
<td>Labial</td>
<td>Palatal</td>
</tr>
<tr>
<td>Maxillary canines</td>
<td>Labial</td>
<td>Palatal</td>
</tr>
<tr>
<td>Maxillary first premolars</td>
<td>Labial (buccal</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>root)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Palatal (palatal root)</td>
<td></td>
</tr>
<tr>
<td>Maxillary second premolars</td>
<td>Buccal</td>
<td>Palatal</td>
</tr>
<tr>
<td>Maxillary molars</td>
<td>Buccal (buccal root)</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Palatal (palatal root)</td>
<td></td>
</tr>
</tbody>
</table>


- Aids in bone healing
- Increases antibiotic delivery to tissues

**Surgical Management**

Remove source of infection by endodontic therapy; tooth extraction; removal
of foreign bodies, including necrotic or nonviable tissue; and incision and drainage of pus.

Consider sources of infection
- Odontogenic: pulp, periodontium
- Postoperative wounds
- Trauma: fractures, lacerations, necrotic bone fragments, foreign bodies
- Paranasal sinuses
- Salivary glands: acute (mixed oral flora) versus chronic (staphylococci)

Pathways of infection
- Dental (Table 17-3)
- Sinonasal-related infections
- Orbital abscess
- Brain abscess
- Fungal infection and tissue destruction
- “Potential” fascial space infections
  —“Potential” spaces exist between fascial layers in the cervical and pharyngeal regions. These compartments may become involved through contiguous spread of infection from the jaws.

- Contiguous fascial space infections
  —Pterygomandibular region: buccal, pterygoid, masseteric, deep and superficial temporal
  —Suprahyoid area: submandibular (submental), sublingual, lateral pharyngeal, peritonsillar
  —Cervical spaces: retropharyngeal, retroesophageal (danger space 4), carotid sheath, pretracheal

Principles of incision and drainage
- Ideally, place incisions in healthy skin or mucosa.
- Place incisions in esthetically acceptable area.
- Obtain dependent drainage.
- Use two small skin incisions as drain entrance and exit points rather than one large incision. This provides an extensive subcutaneous drainage area while maintaining facial esthetics.
- Use blunt dissection to avoid damage to nerves and vessels. Advance
closed hemostat into all involved areas, spread beaks, and then remove completely before reinserting.

- Secure drains (Penrose drain or red rubber catheter) with sutures; avoid gauze drains. Advance drain as suppuration decreases.

**Infectious Clinical Syndromes**

**Cutaneous Infections**

**Impetigo**

*Diagnosis*

- Superficial papulovesicular lesions surrounded by erythema, which progress into larger yellow-crusted weeping lesions
- Caused by group A β-hemolytic streptococci and *Staphylococcus aureus*
- Bullous form, more severe blistering staphylococcus infection

*Management*

- Cleanse with warm, soapy water twice a day; no occlusive dressings
- Mupirocin topically to lesions three times a day
- Erythromycin, cefaclor, or cefadroxil, if infection is severe

**Erysipelas**

*Diagnosis*

- Acute cellulitic, lymphangitic infection of skin, with rapid progression; firm, raised, erythematous lesions
- Usually caused by β-hemolytic streptococci
- Most often involves the face bilaterally
- Patient may become toxic; usually causes fever, general malaise, and irritability in younger patients
Management
- Penicillin V or erythromycin
- Cloxacillin or cephaloxin can be used in resistant cases

Methicillin-resistant *Staphylococcus aureus* (MRSA) infections
MRSA infections usually occur in hospitalized patients or those in other health care settings. However, there is now a community-based form. There is generally a carrier involved who has the organisms on the skin or in the nasal cavity.

Diagnosis
- May present as a pimple or small boil or as furunculosis, a carbuncle, or a deep wound abscess
- Characterized by indolent, slow-spreading soft tissue infection with central tissue necrosis and slough

Management
- Sulfamethoxazole/trimethoprim, linezolid, vancomycin
- Incision and drainage of any abscess formation and frequent irrigation using antibiotic solution
- Consider elimination of carrier state with sulfamethoxazole/trimethoprim and mupirocin ointment to nasal cavity

Osteomyelitis
Acute (suppurative)

Diagnosis
- Antecedent procedure, trauma, or febrile illness (especially in children)
- Tender, but firm, swelling over affected area
• Associated neurosensory changes in area of intrabony abscess
• Radiographic studies to determine extent, suppuration, vascularity, chronicity
  —Bone scintigraphy for diagnosing early infections
  —CT scan with contrast (high sensitivity, moderate specificity)
  —Magnetic resonance imaging (MRI) + gadolinium (high sensitivity and specificity)

Management
• Debride any necrotic bone (sequestra).
• Place drains as indicated.
• Obtain aspirate, swab, or tissue specimen for culture.
• Empirically provide antibiotic coverage for gram-positive cocci, Haemophilus, and anaerobes until antibiotic sensitivity test results available.
• Provide antibiotic coverage for Salmonella and Pseudomonas in sickle-cell anemia patients.

Chronic

Diagnosis
• Unresponsive acute process with little suppuration
• Firm swelling, usually not painful
• Draining sinus tracts
• Exposed necrotic bone; pathologic fracture
• Radiographic evidence of sequestra, involucrum, and avascular zone at periphery

Management
• Debridement; obtain specimen for culture
• Drains for irrigation (antibiotic solutions)
• Consider antibiotic-impregnated bead implantation
• Sensitivity-directed antibiotic therapy (long term); infection usually polymicrobial, consider resistant staphylococcus, anaerobes (*Actinomyces*)
• Check immune status of patient
• Consider hyperbaric oxygen
• PET scans to follow course of infection

Orbital Infections
Periorbital (preseptal) cellulitis

*Diagnosis*
• Swelling, erythema of eyelids
• Often associated with sinusitis
• History of local trauma, conjunctivitis
• Typically no change in ocular function
• *Haemophilus*, gram-positive cocci

*Management*
• Elevation of head
• Warm, moist compresses
• Trimethoprim + sulfamethoxazole, cefaclor, clarithromycin, amoxicillin + clavulanate

Orbital cellulitis (or abscess)

*Diagnosis*
• Infection internal to orbital septum
• Periorbital swelling, chemosis, proptosis, ophthalmoplegia, changes in vision
• Antecedent sinusitis, particularly in children
• Patient usually more ill than with periorbital cellulitis
• CT scan to determine abscess formation or contiguous
spread from sinuses or intracranial involvement

**Management**

- Hospitalize; obtain appropriate consults
- If abscess, perform drainage (ie, sinusotomy), orbital drain placement
- IV antibiotics: ceftriaxone, ampicillin + sulbactam, clindamycin, vancomycin

**Sinusitis**

Acute (less than 2 weeks’ duration)

**Diagnosis**

- 10% odontogenic
- Often associated with upper respiratory infection (URI)
- Purulent nasal discharge
- Fever, periorbital swelling, infraorbital tenderness, maxillary molar toothache
- Radiographic evidence of fluid/pus; usually in maxillary or ethmoid sinuses
- Nasoendoscopy to evaluate ostia, structural problems
- *Streptococcus* species, *Haemophilus, Moraxella* in children
- More anaerobes in adults

**Management**

- Amoxicillin, azithromycin, combination therapy if recurrent or severe
- Antihistamines, saline nasal irrigation to improve spontaneous drainage
- Consider surgical drainage if no resolution (probable subacute bacterial sinusitis, but also with structural drainage problems)
Chronic (greater than 3 months’ duration)

**Diagnosis**
- Bacteriology changes to more anaerobes, untypable *Haemophilus, Staphylococcus, Pseudomonas*
- Episodic URI, allergic rhinitis, otitis media, asthma
- Dull ache in cheek and maxilla
- Usually have intranasal obstruction, ie, deviated septum, ostial stenosis, polyps, turbinate adenoid hypertrophy
- Rule out immunocompromised state

**Management**
- Antibiotics directed at anaerobes
- Long period of antibiotic use
- Control allergies: use steroid inhalant, mast cell stabilizer, decongestant, nasal toilet with irrigation
- Surgery for nasal obstruction, adenoidectomy, anterior ethmoidectomy, ostial enlargement

Ludwig Angina

**Diagnosis**
- Bilateral submandibular and sublingual space cellulitis
- Brawny edema with elevated tongue and floor of mouth
- Airway compromise, stridor, fever
- Usually streptococcal in origin

**Management**
- Airway control via intubation or tracheotomy if patient is unable to maintain airway
- Wide incision and drainage of involved spaces
- High-dose intravenous antibiotics
- Rehydration and assisted nourishment (nasogastric feedings) if inadequate oral intake
Necrotizing Fasciitis

Diagnosis
- Aggressive, rapidly spreading infection of muscle and fascia, often after trauma or surgery in compromised patients
- Skin dusky, with mottled appearance, progressing to dermal slough
- Skin anesthesia
- Patient toxic, with hemolysis, volume depletion, and organ failure
- Gram-positive cocci (especially group A streptococci), gram-negative anaerobes; usually polymicrobial

Management
- Wide debridement of necrotic tissue and surgical drainage; repeat as necessary
- Pulsed pressure irrigation with antibiotic solutions (bacitracin, neomycin, polymyxin combination, or kanamycin)
- Rehydration, assisted nourishment, and electrolyte stabilization
- Empiric high-dose antibiotics with coverage for gram-positive cocci and gram-negative and anaerobic organisms
- Hyperbaric oxygen
- Use of vacuum-assisted closure device to decrease bacterial load and promote tissue vascularity

Cavernous Sinus Thrombosis

Diagnosis
- Infection from maxillary, nasal, or orbital region via anterior and posterior facial veins
- Proptosis, orbital swelling, high fever, altered sensorium, severe headache, partial or total ophthalmoplegia, decreased visual acuity

Management
- Remove source of infection
- Neurosurgical consultation
- Obtain external drainage and decompression of orbit via medial canthotomy incision, if orbit involved
- High-dose antibiotics with β-lactamase inhibitor combined with third-generation cephalosporin or an aminoglycoside initially until culture and
antibiotic sensitivity test results are available
• Consider anticoagulation

Mediastinitis

Diagnosis
• Extension (descending infection) from deep cervical spaces
• High fever, chest pain, dyspnea, distant heart sounds, and radiographic
demonstration of mediastinal widening

Management
• Remove source of infection with incision and drainage of involved
cervical spaces
• Mediastinal drainage and insertion of drains for irrigation
• High-dose, long-term antibiotics
• Vigilance for airway compromise, large vessel erosion, and myocardial
failure

Special Orofacial Infections

Actinomycosis
• Does not follow tissue planes; direct contiguous spread
• Penicillin G intravenously for 4 to 6 weeks, then orally for 3 to 6 months
• Oral alternatives: tetracycline, metronidazole, clindamycin
• Surgical management of necrotic tissue

Mycotic Infections

Candidiasis (mucocutaneous)
• Nystatin suspension or troches 7 to 10 days for oral lesions
• Clotrimazole, ketoconazole 5 to 7 days
• Fluconazole for refractory or immunodeficiency states

Mucormycosis, aspergillosis
• Generally associated with compromised immune status
• Control immune status, nutrition, diabetes
• Amphotericin B (monitor renal function, hemoglobin, K⁺)
• Surgery for tissue invasion, not colonization
Histoplasmosis
- Usually disseminated from pulmonary source
- Ketoconazole if not life-threatening
- Amphotericin B (monitor renal function, hemoglobin, K⁺)

Lyme Disease (Borrelia burgdorferi)

Findings
- Multisystem immune-mediated inflammatory disorder caused by the spirochete B burgdorferi and transmitted by tick bite
- History of tick exposure, skin rash (erythema migrans), flulike illness, fever, myalgia/arthralgia
- Three stages:
  —Early: skin manifestations, fever, joint pain/stiffness, malaise
  —Early disseminated: occurs several weeks or months later, with multiple skin lesions and nervous system involvement (Bell palsy, meningitis, radiculopathy)
  —Late: more disseminated and severe or recurrent, especially arthralgias; lasts for years
- Diagnosis by history, clinical findings and positive enzyme-linked immunoabsorbent assay (ELISA), Western blot, Lyme titer

Management
- Amoxicillin or erythromycin for patients younger than 10 years
- Doxycycline for patients older than 10 years
- Ceftriaxone for late or early disseminated disease

Tuberculosis (Mycobacterium species)

Many forms classified as “atypical” or nontuberculous.

Findings
- Pulmonary form most common
- Oral form rare; secondary infection via sputum or hematogenous spread
  —Tongue most common oral site, with deep, painful, ragged ulcer
  —Bony lesions occasionally occur in angle of mandible
• Histologic findings
  — Acid-fast staining capsule on organisms
  — Caseation, with Langhans giant cells

Management
• Positive culture and microscopic evidence to confirm diagnosis (grows slowly in culture medium)
• Isoniazid, rifampin, pyrazinamide
• Administration of two of the previous three agents results in greater than 95% efficacy
• Add streptomycin or ethambutol for resistant strains
• Monthly sputum/wound cultures until negative

Cervical Lymphadenopathy (Atypical Mycobacteria), Tuberculous Cervical Lymphadenitis (scrofula)
• Usually caused by Mycobacterium scrofulaceum.
• More common in children.
• Single or multiple enlarged submandibular nodes.
• Surgical excision plus conventional treatment for tuberculosis.

Syphilis (Treponema pallidum)
Findings (three clinical stages)

Primary (chancre)
• Ulcerated nonpainful lesion occurring 2 to 4 weeks after exposure and persisting for 2 to 6 weeks
• Spirochetes present in lesion

Secondary (mucous patch)
• Generalized eruption of painless, grayish plaques 1 to 6 months after initial stage
• Followed by a latent stage with no clinical signs or symptoms
• Spirochetes present in lesions

Tertiary (gumma)
• Neurosyphilis, aortic aneurysms
• Granulomatous, noncaseating skin nodules
• No spirochetes in lesion

Diagnosis
• Clinical appearance of chancres, maculopapular rashes, mucous patches, etc
• Darkfield microscopy in primary disease to detect treponemes
• Serologic flocculation tests performed in sequence
• Venereal Disease Research Laboratory (VDRL) screening test (many falsely positive results in lupus, respiratory infections)

Fluorescent treponemal antibody absorption test to confirm diagnosis

Management
• Early syphilis
  —Benzathine penicillin G 2.4 million U intramuscularly (IM), single dose
  —Oral alternative: doxycycline 100 mg twice daily × 14 days
  —Watch for Jarisch-Herxheimer reaction, a self-limiting systemic response to massive treponemal kill; characterized by fever, malaise, myalgia

• Late syphilis
  —Benzathine penicillin G 2.4 million U IM/wk for 3 weeks
  —Alternative: ceftriaxone

Herpes Virus Infections
Herpes simplex (type 1 = oral; type 2 = genital)

Findings
• Primary herpetic gingivostomatitis, usually in young children
• Fever, malaise, dysphagia, lymphadenopathy
Painful oral mucosal lesions persisting 7 to 14 days
Secondary ("cold sore") lesions
Stress, trauma, fatigue may induce viral reactivation

Management
- Hydration, oral analgesics.
- Cauterize or laser single or small lesions.
- Apply topical 5% idoxuridine.
- Penciclovir cream applied every 4 hours for 5 days or acyclovir 200 mg orally 5 times per day for 1 week (for prevention, 400 mg twice daily); valacyclovir 2 g orally twice a day for 1 day.
- For severe, painful ulcers, elixir of bismuth subsalicylate, diphenhydramine hydrochloride, or tetracycline oral rinse.
- Chlorhexidine rinse followed by sucralfate 1 g twice daily, held in mouth until dissolved.

Herpes zoster ("shingles")

Cause: Varicella-zoster virus reactivation in nerve ganglia, usually due to trauma or illness

Findings
- Painful vesicular eruptions over nerve distribution (dermatome).
- In adults, will persist for 2 to 3 weeks.
- Postherpetic neuralgia is common.
- Ramsay Hunt syndrome can occur due to geniculate ganglion involvement, with facial nerve palsy and oropharyngeal vesiculation.

Management
- Valacyclovir 1 g three times daily for 5 to 7 days, famciclovir 500 mg three times daily for 7 days, or acyclovir 800 mg every 4 hours for 7 to 10 days.
• For lesions, drying agents such as calamine may be helpful; diphenhydramine 25 to 50 mg orally every 8 hours for itching.
• For neuralgia, prednisone 30 to 40 mg/d for 7 to 10 days, then taper dose gradually; gabapentin 300 mg daily and slowly increase to effect; or carbamazepine 200 mg three times daily if severe (watch for leukopenia).
• Admit to hospital and consult with ophthalmologist if ophthalmic division involved.

Considerations in the Pregnant or Lactating Patient with Infections
Factors that determine degree of placental transfer of antibiotics
  • Lipid solubility, ionization of compound, protein binding, and placental-fetal blood flow
  • In late pregnancy, maternal serum concentrations of most antibiotics decrease due to increased blood volume distribution

Fetal serum concentrations
  • Approximately same as maternal: penicillins, amoxicillin, ampicillin, methicillin, sulfonamides, chloramphenicol, tetracyclines
  • 20% to 50% of maternal: aminoglycosides
  • 10% to 15% of maternal: cephalosporins, clindamycin, erythromycin

Breast milk concentrations
  • Equal in maternal serum and milk: metronidazole, sulfonamides, isoniazid
  • 50% to 70% of maternal: erythromycin, chloramphenicol
  • Less than 25% of maternal: penicillin, aminoglycosides, cephalosporins

Drugs contraindicated in pregnancy: Quinolones, tetracyclines
Safe antibiotics: Penicillins, cephalosporins, nystatin, erythromycin base
Bisphosphonate therapy has been considered standard treatment in the management of cancer patients with metastatic bone disease and patients with osteoporosis. The efficacy of these drugs is due to their ability to inhibit osteoclast-mediated bone resorption. However, the postmarketing experience with intravenous and, to a much lesser extent, oral bisphosphonates has raised concerns about potential side effects related to profound bone remodeling inhibition and osteonecrosis isolated to the jaws. This chapter reviews the risk factors, incidence, pathogenesis, prevention strategies, and management of this new complication.

**Indications for Bisphosphonate Use**

**Malignancy**

Based on clinical practice guidelines established by the American Society of Clinical Oncology, the use of bisphosphonates is considered the standard of care for treatment of (1) moderate to severe hypercalcemia associated with malignancy and (2) metastatic osteolytic lesions associated with breast cancer and multiple myeloma, in conjunction with antineoplastic chemotherapeutic agents. Recently, the US Food and Drug Administration (FDA) has broadened the indications for intravenous bisphosphonates to include bone metastases from any solid tumor.

**Osteoporosis**

As a potent suppressor of osteoclast activity, bisphosphonates slow the remodeling process and increase bone mineral density, thereby reducing the risk of fracture in women with osteopenia and osteoporosis. The World
Health Organization has established criteria for bisphosphonate therapy that are based on bone density values. Patients with scores between –1.5 and –2.5 (osteopenia) or scores less than –2.5 (osteoporosis) are candidates for antiresorptive therapy.

Paget Disease

Paget disease is characterized by osteoclast hyperplasia coupled with compensatory osteoblast hyperactivity. This results in exuberant abnormal bone formation and skeletal deformities. Suppression of osteoclast function with bisphosphonates has emerged as an effective FDA-approved treatment strategy for patients with Paget disease.

Clinical Presentation of Bisphosphonate-Related Osteonecrosis of the Jaw

According to the American Association of Oral and Maxillofacial Surgeons (AAOMS) guidelines, patients may be considered to have bisphosphonate-related osteonecrosis of the jaw (BRONJ) if all of the following clinical characteristics are present:

- Current or previous treatment with a bisphosphonate
- Exposed bone in the maxillofacial region that has persisted for more than 8 weeks
- No history of radiation therapy to the jaws

The patient’s history and the findings on clinical examination are the most sensitive diagnostic tools for this condition. Areas of exposed and necrotic bone may remain asymptomatic for weeks, months, or even years. These lesions only become symptomatic when the surrounding tissues become inflamed or there is clinical evidence of infection. Signs and symptoms that may occur before the development of clinically detectable osteonecrosis include pain, tooth mobility, mucosal swelling, erythema, and ulceration. Most case series have described this complication occurring in regions of previous dentoalveolar surgery (eg, extraction sites). However, exposed bone can also develop in patients at risk with no history of trauma or in edentulous regions of the jaw. Intraoral and extraoral fistulas may develop when necrotic jawbone becomes secondarily infected. Some patients may also present with complaints of altered sensation in the affected area of the mandible as the
neurovascular bundle becomes compressed by the inflammatory process in the surrounding bone. Chronic maxillary sinusitis secondary to osteonecrosis, with or without an oroantral fistula, can be the presenting symptom in patients with maxillary bone involvement.

BRONJ occurs more commonly in the mandible than the maxilla. It is also more prevalent in areas with thin mucosa overlying bone prominences such as tori, exostoses, and the mylohyoid ridges. The size of the affected area can be variable and range from a nonhealing extraction site to exposure and necrosis of large sections of the jawbone. The area of exposed bone is typically surrounded by inflamed erythematous soft tissue. Purulent discharge at the site of the exposed bone will be present when these sites become secondarily infected. Microbial cultures from areas of exposed bone will usually reveal normal oral microbes and therefore are not always helpful. However, in cases where there is extensive soft tissue involvement, microbial culture data may help define comorbid oral infections and facilitate the selection of an appropriate antibiotic regimen.

The microscopic examination of debrided specimens will typically show bone necrosis associated with bacterial debris and granulation tissue. The necrotic bone specimens have not demonstrated any microscopic features that are unique or diagnostic for BRONJ.

*Table 18-1* Indications, route of administration, and relative potency of commonly used bisphosphonates

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indications</th>
<th>Route/Schedule</th>
<th>Relative potency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etidronate</td>
<td>Paget disease</td>
<td>Oral/Daily</td>
<td>1</td>
</tr>
<tr>
<td>Alendronate</td>
<td>Osteoporosis</td>
<td>Oral/Weekly</td>
<td>1,000</td>
</tr>
<tr>
<td>Risidronate</td>
<td>Osteoporosis</td>
<td>Oral/Weekly</td>
<td>1,000</td>
</tr>
<tr>
<td>Ibandronate</td>
<td>Osteoporosis</td>
<td>Oral/Weekly</td>
<td>1,000</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IV/Monthly</td>
<td></td>
</tr>
<tr>
<td>Pamidronate</td>
<td>Bone metastasis</td>
<td>IV/Monthly</td>
<td>1,000–5,000</td>
</tr>
<tr>
<td>Zoledronate</td>
<td>Bone metastasis</td>
<td>IV/Monthly</td>
<td>&gt; 10,000</td>
</tr>
<tr>
<td></td>
<td>Osteoporosis</td>
<td>IV/Yearly</td>
<td></td>
</tr>
</tbody>
</table>

IV, intravenous.
Risk Factors for BRONJ

Bisphosphonate Potency

The various bisphosphonates have differing degrees of potency (Table 18-1). Zoledronate is more potent than pamidronate, and pamidronate is more potent than the oral bisphosphonates. The intravenous (IV) route of administration results in a greater drug exposure than the oral route.

Duration of Therapy

Longer treatment with either the oral or IV preparations appears to be associated with increased risk.

Dentoalveolar Surgery

Patients exposed to bisphosphonates are at risk following any type of surgical procedure that involves bone injury and repair. These include, but are not limited to, extractions, dental implant placement, periapical surgery, and osseous periodontal surgery. Patients treated with IV bisphosphonates who undergo dentoalveolar procedures have a 5- to 21-fold increased risk for BRONJ than cancer patients treated with IV bisphosphonates who do not undergo dentoalveolar procedures.

Location

As previously noted, BRONJ occurs more commonly in the mandible than the maxilla (2:1 ratio) and more frequently in areas with thin mucosa overlying bony prominences (tori, exostoses, and mylohyoid ridges).

Medications

Chronic steroid therapy and certain chemotherapeutic agents may increase risk.

Oral Disease

The presence of inflammatory dental disease appears to be a risk factor for developing BRONJ.
Genetics

Single nucleotide polymorphisms (SNPs) in the cytochrome P450-2C8 gene (CYP2C8) were associated with an increased risk of BRONJ in multiple myeloma patients treated with IV bisphosphonates.

Management and Treatment of BRONJ

Goals of Treatment

- Patient education and reassurance
- Control of pain
- Control of secondary infection
- Prevention of extension of lesion and development of new areas of necrosis
- Support of continued oncologic treatment in patients receiving IV bisphosphonates

Discontinuation of Bisphosphonate Therapy

- Short-term discontinuation of IV bisphosphonates offers no benefit.
- Long-term discontinuation of IV bisphosphonates may be beneficial in stabilizing established sites of BRONJ, decreasing the risk of new site development, and reducing clinical symptoms.
- Discontinuation of oral bisphosphonate therapy in patients with BRONJ has been associated with gradual improvement in clinical disease.
- Discontinuation of oral bisphosphonates for 6 to 12 months may result in either spontaneous sequestration or resolution following debridement surgery.
- The decision to continue bisphosphonate therapy should be made only by the treating oncologist in consultation with the oral and maxillofacial surgeon and the patient.

Management of Patients Scheduled for IV Bisphosphonate Treatment

- The treatment objective for this group of patients is to minimize the risk of developing BRONJ.
- If possible, initiation of monthly IV bisphosphonate therapy should be delayed until dental health is optimized.
- Nonrestorable teeth and those with a poor prognosis should be extracted.
Bisphosphonate therapy should be delayed, if possible, until the extraction site has mucosalized (14 to 21 days) or until there is adequate osseous healing.

- Dental prophylaxis, caries control, and conservative restorative dentistry are critical to maintaining functionally sound teeth. This level of care must be continued indefinitely.
- Patients with complete or partial dentures should be examined for areas of potential mucosal trauma, especially along the mylohyoid ridge and adjacent to tori.
- Patients should be educated regarding the importance of good dental hygiene and regular dental evaluations, as well as the signs and symptoms of BRONJ.

Management of Asymptomatic Patients Receiving IV Bisphosphonates
- Procedures that involve direct osseous injury should be avoided.
- Nonrestorable teeth may be treated by removal of the crown and endodontic treatment of the remaining roots.
- Placement of dental implants should be avoided in patients exposed to the more potent IV bisphosphonates (ie, zoledronate and pamidronate) on a frequent dosing schedule (4 to 12 times per year).
- The risk of BRONJ in those patients receiving a once-per-year dose of zoledronate for osteoporosis appears low but requires further study.

Management of Asymptomatic Patients Receiving Oral Bisphosphonates
- The risk of BRONJ appears to be associated with increased duration of treatment with oral bisphosphonates (≥ 3 years).
- For individuals who have taken an oral bisphosphonate for less than 3 years and do not have any clinical risk factors, no alteration or delay in the planned surgery is necessary.
- There has been no information to suggest that monthly dosing of oral bisphosphonates (ie, ibandronate, risidronate) is associated with either an elevated or reduced risk of BRONJ when compared with weekly dosing regimens.
- For those patients who have taken an oral bisphosphonate for more than 3 years, with or without any concomitant prednisone or other steroid medication, the oral bisphosphonate should be discontinued for 3 months prior to oral surgery.
The efficacy of using a systemic marker of bone turnover, such as C-terminal telopeptide, to assess the risk of developing jaw necrosis requires further research before it can be considered a valid risk assessment tool.

For those patients who have taken an oral bisphosphonate for less than 3 years and have also received daily corticosteroids concomitantly, consider discontinuation of the oral bisphosphonate (ie, a drug holiday) for at least 3 months prior to oral surgery.

Management of Patients with BRONJ

- Treatment objectives for patients with an established diagnosis of BRONJ are to eliminate pain, control infection of the soft and hard tissue, and minimize the progression or occurrence of bone necrosis.
- Surgical treatment should be delayed if possible and reserved for those patients with stage 3 disease (see next section on the BRONJ staging system) or in those cases with a well-defined sequestrum.
- Areas of necrotic bone that are a constant source of soft tissue irritation should be removed or recontoured without exposure of additional bone.
- Loose segments of bony sequestrum should be removed without exposing uninvolved bone.
- The extraction of symptomatic teeth within exposed, necrotic bone should be considered, because it is unlikely that the extraction will exacerbate the established necrotic process.
- Avoid elective dentoalveolar surgical procedures, because these surgical sites may result in additional areas of exposed necrotic bone.

BRONJ Staging System

- Patients at risk: No apparent necrotic bone in asymptomatic patients who have been treated with IV or oral bisphosphonates
- Stage 0: No clinical evidence of necrotic bone in patients who have nonspecific symptoms or clinical and radiographic findings such as:
  —Odontalgia not explained by an odontogenic cause
  —Dull, aching bone pain in the body of the mandible, which may radiate to the temporomandibular joint region
  —Sinus pain, which may be associated with inflammation and thickening of the maxillary sinus wall
  —Altered neurosensory function
—Loosening of teeth not explained by ongoing periodontal disease
—Periapical/periodontal sinus tract that is not associated with pulpal necrosis due to caries
—Alveolar bone loss or resorption not attributable to ongoing periodontal disease
—Changes to trabecular bone pattern—dense woven bone and persistence of unremodeled bone in extraction sockets
—Thickening/obscurig of periodontal ligament (thickening of the lamina dura and decreased width of the periodontal ligament space)
—Inferior alveolar canal narrowing

- Stage 1: Exposed and necrotic bone in patients who are asymptomatic and have no evidence of infection
- Stage 2: Exposed and necrotic bone associated with infection as evidenced by pain and erythema in the region, with or without purulent drainage
- Stage 3: Exposed and necrotic bone in patients with pain, infection, and one or more of the following: exposed and necrotic bone extending beyond the region of the alveolar process (ie, inferior border and ramus of the mandible, maxillary sinus and zygoma) resulting in pathologic fracture, extraoral fistulas, oroantral/oronasal communication, or osteolysis extending to the inferior border of the mandible or sinus floor

**Box 18-1 Treatment of patients with BRONJ**

At-risk patient
- No treatment
- Patient education regarding the risks of developing BRONJ, as well as the signs and symptoms of this disease process

Stage 0
- Symptomatic treatment
- Conservative management of other local factors, such as caries and periodontal disease
• Systemic management, including the use of medication for chronic pain and control of infection with antibiotics when indicated

Stage 1
• Antibacterial mouthrinse
• Clinical follow-up on a quarterly basis
• Patient education and review of indications for continued bisphosphonate therapy

Stage 2
• Symptomatic treatment with oral antibiotics
• Oral antibacterial mouthrinse
• Pain control
• Superficial bony debridement to relieve soft tissue irritation

Stage 3
• Antibacterial mouthrinse
• Antibiotic therapy and pain control
• Surgical debridement/resection for longer-term palliation of infection and pain

BRONJ Treatment Strategies
For patients with established BRONJ, treatment is basically focused on preserving the quality of life by controlling the pain and secondary infection while supporting the ongoing oncologic or osteoporotic treatment (Box 18-1). It is also important to prevent the extension of existing areas of necrosis and the development of new lesions. For the most part, surgical treatment is reserved for those patients with extensive disease (stage 3) or with areas of exposed, loose sequestra. Aggressive surgical therapy for stages 1 or 2 may result in a larger nonhealing bone wound and should be avoided. Antibiotic oral rinses and systemic antibiotic therapy are the mainstays of treatment for patients with stage 1 or 2 disease.
Surgical correction of dentofacial and craniofacial abnormalities should be undertaken only after diagnosis and comprehensive treatment planning have occurred and the presurgical phase of orthodontic preparation is completed. In general, surgery for conditions characterized by excessive growth (mandibular prognathism, vertical maxillary excess, hemimandibular elongation, and hemimandibular hypertrophy) should be delayed until growth has slowed to its adult rate. Removal of the actively growing region of the mandible is another alternative, and this also requires reconstruction of the excised part. In conditions of deficient growth (mandibular deficiency, mandibular ankylosis, maxillary deficiency), surgery may be performed earlier with good results, but there is always a risk that postsurgical facial growth will adversely affect the outcome. In general, there is less risk that postsurgical facial growth will adversely affect the result in growth-deficient conditions than in growth-excess conditions.

Parents and patients should be informed of the potential risks of early surgery, and they must participate in the decision regarding the timing of surgery. It may not be in the best biologic interest for young patients to undergo early surgical correction, but it may be in their best psychosocial interest, and this should govern the decision about timing of surgery. The patient and family must be informed of the possibility of requiring additional surgery at complete maturation if undesirable growth occurs following initial surgery.

Dentofacial Deformities
Vertical Maxillary Excess

Vertical maxillary excess is characterized by excessive tooth display at lip repose and gingival exposure on smiling. Lip incompetence is always present. Occasionally, different segments of the maxilla are more vertically excessive, and steps in the maxillary occlusal plane are present. When this occurs, an open bite is almost always present. The facial height is always long, and the chin is rotated downward and posteriorly. This condition is exaggerated by a short upper lip and/or maxillary protrusion. Vertical maxillary excess can be seen with Class I, II, and III occlusal patterns. Cephalometric analysis normally confirms a long lower face and excessive vertical descent of the maxillary occlusal plane.

Orthodontic intervention early in life (8 to 12 years) with high-pull headgear and/or open bite bionator may control additional vertical growth and, if successful, may resolve the condition as the soft tissues and other facial structures grow.

Vertical growth of the maxilla is the last vector to cease. Often patients with excessive vertical development continue growing later in life. Relapse can occur if postsurgical vertical growth is significant, especially when an open bite is present. If the condition is severe, early surgery may be justified on the basis of psychosocial benefits. As with most deformities characterized by excessive growth, delaying surgery until growth has slowed is recommended. This condition usually responds favorably to superior repositioning of the maxilla via Le Fort I downfracture with or without segmentation.

Posterior Maxillary Vertical Excess

This condition is usually seen when opposing mandibular teeth have been removed and passive maxillary tooth eruption occurs. Inadequate interarch space results, and this condition poses a serious prosthetic challenge. When this condition occurs in the dentate state, excessive posterior maxillary vertical growth results in an open bite. The consequences of these two conditions are different, and the etiologies are different (passive eruption versus active growth).

When the condition occurs because of passive eruption secondary to
missing teeth, there is no apparent facial change because the passive eruption ceases when the teeth contact the mandibular ridge. When the condition occurs in the dentate state, the face height is excessive, the mandible rotates downward and backward, and lip incompetency is generally present. The maxillary incisor–lip relationship is normal, but during animation the gingiva in the posterior region may be visualized more than anteriorly.

Radiographic changes in both conditions include an excessive distance from the palatal plane to the first molar cusp. The maxillary sinus may become overly pneumatized, especially in the partially edentulous state. In both conditions, a distinct step in the maxillary occlusal plane is present, and it is usually at this site that an interdental osteotomy is done. If inadequate space exists between the teeth, orthodontic movement or extraction of a tooth is necessary to safely conduct this osteotomy. The space may then be closed by advancing the segment.

In the partially edentulous state, the anterior occlusion should not change if isolated posterior maxillary osteotomies are performed. In the dentate state, superior repositioning of the posterior maxilla results in closure of the open bite, shortening of the face height, and improved ability to seal the lips. The chin also projects further forward as a result of the mandibular rotation. The concerns about operating early, and the potential to grow following surgery, are similar to those with vertical maxillary excess.

Maxillary Vertical Deficiency

This condition commonly coexists with other skeletal abnormalities, such as maxillary anteroposterior and/or transverse deficiency or mandibular prognathism. The lower face height is always reduced and the freeway space is excessive. Commonly, the maxillary incisors are completely covered by the upper lip at rest, with only a portion of the crowns exposed when smiling. A proper-sized mandible will appear prognathic because of the overclosed position.

Cephalometrically, the palatal plane–first molar dimension is always reduced. The occlusion is typically Class III and centric relation–centric occlusion differences are usual.

Treatment usually involves Le Fort I osteotomy with downgrafting of the
anterior and posterior maxilla. Simultaneous mandibular advancement is commonly done to improve stability of the corrected face height.

Maxillary Anteroposterior Deficiency

Paranasal deficiencies are the typical expression of this condition, with the upper lip behind the lower lip. These findings are especially apparent in profile. The occlusion is Class III, and the maxillary anterior teeth may be flared. This condition is the most common skeletal abnormality seen in patients with facial clefts.

Cephalometric analysis may not confirm the diagnosis. Maxillary unit length may be less than normal, but not always. The maxillary incisors are usually flared in the true condition and overly retracted if premolars have been removed previously to orthodontically compensate for the mandibular deficiency.

Transverse Dental Arch Problems

The maxilla can be too narrow or too wide to accommodate the mandibular arch. Or the problem can also be that the mandible is too narrow or too wide. To facilitate the diagnosis of transverse arch discrepancies, dental study casts are necessary. Particular attention to the dental arch shape as well as the alveolar arch shape should help identify the problem. Intercanine and intermolar width measurements can also be helpful. When the problem is transverse deficiency of the maxilla, it can be addressed orthodontically, orthopedically, or surgically. When the problem is minimal, orthodontics often can resolve the issue by tipping and aligning the teeth. When the problem involves more than 2 to 3 mm of dental movement, orthopedic and/or surgical means should be considered. When the patient is younger than 14 years, orthopedic correction with expanding devices is possible in some cases. However, once the palatal suture has fused, these devices will only move teeth and not the bone. To move teeth and bone of the maxilla most predictably past age 14 years, either surgically-assisted palatal expansion or segmental maxillary surgery can be performed. When surgically-assisted expansion is used, the expansion appliance can be tooth-borne or bone-borne. When segmental surgery is used, paramidline osteotomies and bone grafts should be used. Surgical treatment is normally performed at the Le Fort I level, and bone grafts are typically used to stabilize
the position of the maxillary segments.

When the problem is maxillary transverse excess, orthodontic narrowing is a possibility. However, segmental surgery is simpler, more predictable, and more efficacious to achieve the desired result.

When the mandibular arch is narrow, orthodontic expansion is possible, but the limitation is the dentoalveolar bone. When there is significant anterior crowding or missing incisors, surgically-assisted expansion or surgical expansion can help resolve the issue. Careful planning with the orthodontist is advised.

When the problem is transverse excess of the mandible, investigation of the size of the tongue should be undertaken. Although it is possible to narrow the mandibular arch surgically, if the tongue is large this maneuver will only encroach on the tongue space, and it will likely be an unstable movement. In these situations, consideration should be given to tongue reduction as well as mandibular narrowing. An alternative is maxillary widening, which may be more physiologically prudent.

Mandibular Deficiency

Two forms of this condition exist, and they have different morphologic and occlusal presentations. In both conditions, the mandible is small. In the low mandibular plane angle variety, the face height is short, the lower lip is curled over, and the labiomental fold is deep. The maxilla may be vertically deficient as well. The angles of the mandible and the masseters are usually well developed and defined. The maxillary teeth may be upright or even palatally inclined. The curve of Spee is generally excessive in both arches, and the mandibular anterior teeth may contact the palate during occlusion. The depth of the bite is excessive. The maxillary teeth may also be contacting the mandibular anterior gingiva.

Radiographically, the ramus height is usually normal, and the mandibular angles are well developed. Angular and linear cephalometric values are usually smaller than normal.

In the high mandibular plane angle variety, the face height is normal or excessive. The condition is most commonly seen in combination with vertical maxillary excess. When it presents in this manner, all of the features of vertical maxillary excess are present, and the features of mandibular
deficiency exaggerate the appearance. The labiomental fold is flattened by excessive activity of the mentalis muscles. The chin is also small and retropositioned. The mandibular ramus is short, the condyles are usually small, and the angles of the mandible are hypoplastic, forming an excessively obtuse angle with the body of the mandible. The maxillary teeth are usually protrusive, and the arch form is narrow. The mandibular arch may be constricted as well. Class II canine and molar relationships are typical. The bite may also be open and, when it is, vertical maxillary excess is highly suspect. This is especially true in the absence of recognized conditions such as rheumatoid arthritis and temporomandibular joint (MTMJ) ankylosis.

Successful treatment of isolated mandibular deficiency usually involves mandibular advancement. In general, stability is better with smaller amounts of advancement than with large advancements. Sagittal osteotomy with screw fixation is the most frequently performed procedure for this condition. For mandibular advancements greater than 1 cm, inverted L osteotomies with rigid fixation and bone grafting should be considered. Augmentation genioplasty is a common procedure accompanying mandibular advancement, but it is not a good procedure when used alone to disguise significant mandibular deficiency. Mandibular subapical osteotomies are occasionally performed to assist in leveling the mandibular arch in high- or low-angle cases.

Postsurgical lytic condylar changes have been reported following mandibular advancement in a minority of patients. When this occurs, it is more typical in young women with a high mandibular plane angle. The exact etiology of this condition and its potential association with surgery remain obscure.

Mandibular Prognathism

Previously thought to be one of the most common dentofacial deformities in North America, it is now recognized as relatively rare. Improved recognition of maxillary deficiency is the reason for this change. Mandibular prognathism frequently coexists with maxillary deficiency, and when it does the appearance is exaggerated. Overclosure of the vertical dimension and centric relation–centric occlusion slides may coexist and exaggerate the appearance. The prominent chin is often the dominant facial feature, and the lower lip is
forward relative to the upper lip. The mandibular body is well defined, as is the angle. Occasionally, the angle of the mandible is obtuse. The occlusion is Class III, but typically the skeletal discrepancy is greater than the occlusal discrepancy. Maxillary anterior teeth are generally flared, and the mandibular anterior teeth are upright.

Surgery should be undertaken only after dental compensations are eliminated. Sagittal osteotomy, with rigid fixation, is the most frequently performed procedure for correction of this problem. Although the transoral vertical ramus osteotomy is advocated by some, problems with control of the proximal segment and adverse postsurgical occlusal changes have been reported. The extraoral vertical ramus osteotomy is also an alternative, especially when the amount of movement approaches or exceeds 1 cm.

Delaying surgery until completion of growth is best. However, when psychosocial issues are of significant concern, they should influence the timing of surgery.

Condylar Hyperplasia (Hemimandibular Elongation)

Postpubertal and sometimes prepubertal onset, with gradual development of asymmetry, is typical of this condition. The presenting characteristics include asymmetry affecting the lower facial third, deviation of the mandible away from the affected side, and secondary involvement of the maxilla with compensatory vertical growth on the affected side. Mandibular prognathism and/or maxillary deficiency may coexist. The mandibular dental midline is shifted away from the affected side, and lateral crossbite is typical, with asymmetric canine and molar relationships. The canine and molars are always further forward on the affected side.

The panoramic radiograph usually demonstrates a longer condylar neck on the affected side. The condylar head may be normal in morphology when the growth is slow. However, it may be enlarged when the growth is rapid. The cephalometric radiograph always demonstrates asymmetry of the mandibular ramus and angle. Varying degrees of dental compensation are observed. If prognathism and/or maxillary deficiency coexist, these discrepancies accentuate the problem. The anteroposterior cephalogram confirms the mandibular asymmetry, with varying degrees of dental compensation and enlargement of the affected ramus and condyle.
Eliciting a careful history of the asymmetric growth is the critical step in planning treatment. A history of recent change suggests active growth, whereas a history of lengthy presence without change indicates inactivity. Scintigraphic studies are used to confirm active growth, but false-positive interpretations are possible. The patient must participate in the decision to perform a condylotomy or condylectomy, or to delay surgical correction until growth has ceased, understanding that the longer the condition exists, the greater the deformity and the more difficult it will be to achieve facial symmetry. When condylectomy is performed, reconstruction with the remaining ramus should be done, as well as simultaneous maxillary and mandibular osteotomies to correct the facial asymmetry and malocclusion, when necessary.

Hemimandibular Hypertrophy

Like hemimandibular elongation (condylar hyperplasia), hemimandibular hypertrophy also causes facial asymmetry. Recognition of the condition usually occurs earlier than with hemimandibular elongation, sometimes even during childhood. The major distinguishing feature of this condition is elongation of the affected side of the face. The soft tissues are also affected, and this limits the amount of correction that can be achieved. The maxilla can be secondarily affected, with a downward cant on the affected side. Malocclusion and shifting of the mandibular dental midline may not always be present. Although the condition is known as hemihypertrophy, the unaffected side of the face is commonly small, and therefore, the exact pathology is obscure.

The radiographs always reveal an enlarged condyle, ramus, and body of the mandible on the affected side. The condition may terminate short of the facial midline or may cross the midline and gradually taper. An abrupt step in the inferior mandibular border may also be present, which represents the transition zone from normal to excessive growth. Commonly, the inferior alveolar neurovascular bundle is displaced close to the inferior border in the mandibular molar region; this is not typical with mandibular elongation. When hemimandibular hypertrophy commences in early childhood, the teeth on the affected side may also be enlarged.

As with condylar hyperplasia, a careful history of the growth pattern is critical for planning treatment. Scintigraphic studies may be helpful in
identifying growth activity, but falsely positive results can occur. Generally, both maxillary and mandibular osteotomies are required for maximum improvement, with elongation of the short side and shortening of the long side necessary, as well as inferior border osteotomies and bone grafting. Condylotomy or condylectomy may also be indicated if the condyle is identified as still actively growing. Reconstruction with local tissue (ramus) is necessary if condylectomy is performed.

**Facial Clefting**

By definition, facial clefts represent an interruption in normal development, and so they affect areas where tissues are normally in contact. The surrounding tissues are almost always hypoplastic. Facial clefts involve all levels of tissue development, and so the osseous as well as the soft tissues (skin, subcutaneous tissue, fat, muscle, and periosteum) are affected.

Tessier has described 14 types of facial clefting patterns, which occur with the orbit as the center of the description. Six of these facial clefts have intercranial communication, whereas eight involve the middle and/or lower face. The most common type of facial cleft affects the upper lip and/or palate.

**Cleft Lip and Palate**

Clefts involving the upper lip and/or palate can occur unilaterally or bilaterally and may affect tissue layers disproportionately. Clefts may also have incomplete expressions, such as a submucous cleft palate or an incomplete cleft lip.

During the immediate postnatal period, supportive treatment is usually necessary to facilitate feeding and respiration of an infant with a cleft. The use of long nipples with enlarged openings, as well as prone positioning, are usually all the measures that are necessary. Pigeon nipples and the Haberman feeder are other devices to assist feeding.

When the Robin sequence is present (see following section), respiration can be significantly impaired. If this is the case, glossopexy (tongue-lip adhesion), mandibular distraction, and/or tracheotomy may be necessary. When glossopexy or tracheotomy is performed, palatal repair should be completed before releasing the tongue or decannulating the trachea. Outlining treatment phases for parents during the early postnatal period provides
encouragement and hope for the family members, who may be feeling guilty and distraught.

Lip closure should be performed following the “guidelines of 10.” The child’s hemoglobin should reach a level of 10 g/100 mL, the weight should be at least 10 lb, and the infant should be at least 10 weeks of age prior to surgery. Palatal repair is undertaken when the patient begins to speak; usually this occurs between the 8th and 15th months of life.

For bilateral clefts, columella lengthening may be performed prior to commencing school. Bone grafting to the cleft maxilla and palate is usually delayed until the mixed dentition, and the timing of this procedure is dependent on development of the dentition adjacent to the cleft. It is best to place the graft prior to eruption of the permanent teeth in question, but late enough so that maxillary growth is minimally disturbed. Usually this is coincident with ages 5 to 8 years. When the bone graft is placed, residual buccal and palatal fistulas can be simultaneously closed.

Involution of the adenoids and tonsils begins after 9 years in many patients, and this may result in hypernasal speech. If this occurs, pharyngoplasty and/or a posterior pharyngeal flap may be helpful at this time. Orthognathic surgery and/or osseous contouring may benefit many patients with facial clefts. Usually these procedures take place when the patient is between 14 to 18 years of age. Hypernasality sometimes occurs following maxillary advancement in cleft patients, and when it does a pharyngeal flap or pharyngoplasty is sometimes necessary to improve it. Final lip and nasal revision should be delayed until all skeletal surgery is completed.

Because of the complexity of facial clefting, especially cleft lip and palate, affected patients should be managed by a comprehensive team. Speech and hearing therapists, oral and maxillofacial surgeons, otolaryngologists, psychologists, social workers, dentists, and orthodontists are the critical participants in caring for these patients. It is obvious that no one individual has enough expertise to manage such complex situations and, therefore, comprehensive craniofacial/cleft palate teams have been established to meet these challenges.

Robin Sequence
The findings in this condition include a U-shaped cleft palate (not affecting the maxillary alveolus), mandibular hypoplasia, and glossoptosis. Airway problems are frequent, as is gastroesophageal reflux.

Prone positioning and cleft palate feeding routines should be used initially. For severe conditions, glossopexy, mandibular distraction, and/or tracheotomy will be necessary within days or weeks of birth. If mandibular deficiency is the cause of airway compromise, early advancement by surgery or distraction osteogenesis should be considered.

In the majority of patients, “catch-up” growth of the mandible occurs within the first years of life to the extent that no additional surgery is necessary. The residual deformities seen in the minority of cases can be addressed with routine orthognathic surgery in conjunction with orthodontic treatment in late adolescence.

**Craniofacial Abnormalities**

**Craniofacial Microsomia**
This condition is associated with aplastic or hypoplastic development of portions of the face. The usual expression is unilateral, but bilateral expressions can occur. The condition has been known under various names, such as hemifacial microsomia, first and second brachial arch syndrome, and Goldenhar syndrome.

Goldenhar syndrome (oculoauriculo-vertebral dysplasia) is a subcategory that has the typical features of craniofacial microsomia; however, there are also epibulbar dermoid cysts as well as cervical spine abnormalities. Additionally, cardiac and renal problems can occur.

Craniofacial microsomia affects all tissue layers, so there are both soft tissue and skeletal deficiencies. Soft tissue abnormalities, such as dermatologic pits, ear tags, coloboma, interruption of eyebrow pattern, and microtia, should raise suspicion that the underlying bone is also affected. Additionally, many ear abnormalities are commonly present, as are hearing difficulties. Cleft lip and palate may also be present.

Facial morphology is extremely variable, but is always characterized by deficiency. The vertical vector of growth, as well as the sagittal and transverse components, is affected. The muscles of mastication are
hypoplastic or completely absent. Mouth opening usually is not affected. If the muscles of mastication are missing, lateral excursive movements are limited, and deviation on mouth opening is present.

Treatment

**Perinatal period:** Airway and feeding support are occasionally necessary. These patients rarely require tracheotomy because they usually respond to prone positioning and typical cleft palate care. The parents should be informed and the outline of treatment discussed with them. If cleft lip and palate are present, they should be repaired at the usual 3- and 9-month intervals, respectively.

When supraglottic obstruction is present, accurate diagnosis is possible using fiberoptic bronchoscopy. Mandibular advancement by distraction osteogenesis should be considered within the first month of life if the small mandible is the cause of the airway problem. Tracheotomy and/or glossopexy are other options for improving upper airway obstruction.

**Preschool period:** Ear abnormalities may become the focus of parents, and they should be cautioned about the difficulty with ear reconstruction and the need for multiple staged procedures. In view of the child’s other future surgical needs, it may be wisest to accept the deformity because it is usually readily concealed with the hair, or the ear can easily be replaced with osseointegrated implants and a prosthesis.

If a tracheotomy has been done, every effort should be made to close the stoma prior to the child going to school. Precise diagnosis of the obstruction is critical, and mandibular advancement may be possible by either surgical advancement and/or bone grafting (inverted L osteotomy) or by means of distraction osteogenesis.

**Ages 6 to 10 years:** The first stage of facial bone reconstruction should be delayed until there is an adequate source of donor bone. Unless the condyle and ramus are totally absent, there is no need
to graft with a growth center replacement. If such a graft is placed at this age, it is highly likely that further surgery to deal with the problems of lack of control of the vector and magnitude of growth or with possible ankylosis will be necessary.

Distraction osteogenesis may also be used to elongate and advance the mandible at this stage. Advantages of this technique are sparing a bone donor site and minimal length of surgery. Disadvantages include the application of an appliance, which must be worn for several months, scarring, and lack of precise control of the amount and direction of distraction. As better intraoral appliances are developed to facilitate this procedure, the technique will gain greater acceptance.

The intention of distraction osteogenesis is to elongate the ramus on the affected side and to allow the maxilla to express its vertical growth. Additionally, the soft tissues on the affected side are stretched as a result of the skeletal displacement, and it is thought that this enhances the overall result. Stabilization with an occlusal splint is necessary for at least 6 months, with gradual reduction in the size of the splint after that to permit the maxilla to descend. Monitoring the vertical position of the maxilla throughout this period will determine if another procedure will be necessary.

Ages 10 to 14 years: Active orthodontic treatment to eliminate dental compensations is undertaken at this stage. If significant asymmetry persists once the permanent canines have erupted, a genioplasty often improves the social acceptability of the patient’s appearance.

Ages 14 to 17 years: Orthognathic surgery, which may involve maxillary and/or mandibular osteotomies and always a genioplasty, may be helpful. Onlay grafts with cranial bone help contour areas of orbital and zygomatic deficiency, as well as the maxillary and mandibular deficiencies.

Age 17 to maturation: Additional contour bone grafting, soft tissue (fat) grafting by injection, and other esthetic procedures may be performed as needed.
Mandibulofacial Dysostosis (Treacher Collins Syndrome, Nager Syndrome, Miller Syndrome)

These conditions are distinct from craniofacial microsomia and have extremely variable expressions. Although autosomal dominant, the malformations can be so mild that the condition may be overlooked in a generation, only to appear in the next with severe expression. Although the malformations are characterized by missing or absent tissues, similar to craniofacial microsomia, they are always expressed bilaterally. Although the conditions have been described as symmetric, they are not, and the extent of soft tissue and bone tissue hypoplasia varies from side to side.

Soft tissue abnormalities

Coloboma may be present. The soft tissues of the cheek and orbital region are usually very thin and hypoplastic. Occasionally, tufts of hair are noted to be anterior to their normal preauricular position. There may be additional hair on the cheek. The external ear may be microtic, the canal absent or misplaced, and hearing loss is always present. Some muscles of mastication may be absent or hypoplastic and mandibular movement may be limited because of the lack of muscle and other soft tissues. Tracheoesophageal clefts may also be present, as well as tracheomalacia. Cardiac defects, renal abnormalities, and other organ system malformations may also be present.

Skeletal abnormalities

The bones forming the lateral and inferior orbital rims as well as the zygomas, including the arches, may be missing or hypoplastic. Similarly, the mandibular condyles and ramus may be missing or hypoplastic. The mandible is usually deficient in projection, and retrogenia is always present. Typically, antegonial notching and an increased mandibular plane angle are present. The maxilla is also deficient and the pterygoid plates may be completely absent. Choanal atresia may be present, as well as cleft palate. The nose appears long and large on the face of affected adults. The dorsum typically has a hump and the tip is depressed. The length of the anterior cranial base is diminished.

Treatment
**Perinatal:** Airway and swallowing difficulties occasionally lead to the need for tracheotomy and gastrostomy. Conservative management by prone positioning and the usual cleft palate feeding routines should be used prior to considering tracheotomy or gastrostomy. Perinatal distraction osteogenesis is another option, provided that there is adequate bone in the condyle and ramus. Apnea and oxygenation monitors are commonly used during this period. Because the eyelids are typically affected, adequate lid closure should be ensured. Use of ophthalmic ointments and occasionally tarsorrhaphies are necessary. Tracheomalacia and/or bronchomalacia are common in this population and should not be overlooked if airway problems develop. If mandibular hypoplasia is the cause of airway obstruction, advancement by means of surgery or distraction osteogenesis should be considered.

**Preschool:** Cleft palate repair is undertaken at the usual time. Frequent hearing tests and early use of hearing aids are recommended. Surgery to improve hearing should be considered, but issues regarding reconstruction of the external ear are similar to those with craniofacial microsomia.

It should be a goal to have the child completely tube-free prior to commencement of school. Occasionally early mandibular advancement is indicated to improve the airway. It is tempting to initiate reconstruction of the orbital region at this time but, because massive amounts of bone are required, it may be best to delay these procedures until adequate development of appropriate donor sources has occurred.

**Ages 6 to 10 years:** Initial attempts to reconstruct the orbital and zygomatic defects are often undertaken at this age. Because large quantities of bone are required, it is best to allow the patient’s statural growth and psychosocial concerns to dictate the timing of this initial effort. Traditionally, rib and ilium were used for this reconstruction. More recently, full-thickness calvarial grafts (either free or pedicled to the temporalis muscle) have been used with improved success.
Ages 10 to 14 years: During this period, the patient will undergo orthodontic preparation for jaw surgery. The occlusion is variable and may even be Class III. Open bite is typical. Mandibular retropositioning should not be undertaken because of the small hypopharynx and tongue size. Typically, the patient will benefit from counterclockwise rotation of the lower and middle third of the face, as well as increase in ramus height and augmentation of the angle region. In addition, chin surgery is always helpful.

Ages 14 to 17 years: Similar to craniofacial microsomia (see previous section).

Age 17 to maturation: Esthetic revisions are undertaken at this stage. It is important not to attempt soft tissue transfer, especially to the eyelids, until the skeleton is completely reconstructed.

Orbital Hypertelorism
This is always an expression of an underlying osseous malformation or cleft. Surgical correction before school age should be undertaken in only the severest cases because it may have profound effects on dental, nasal, and maxillary development. Orbital hypertelorism is always observed in patients with craniofron-tonasal dysplasia, frontonasal dysplasia, and Apert syndrome. It is occasionally observed in patients with midline clefts such as bilateral cleft lip and palate. Surgery before the dentition has erupted is usually performed as a transcranial frontofacial bipartite operation. In the adult, it is typically performed transcranially as well, but at the infraorbital level, because dental development is not a concern. The transcranial approach is usually the safest because the frontal lobes are visualized and protected. Prolapse of the frontal lobes into the interorbital space is common. Meningoceles are occasionally present.

Craniosynostosis
Craniosynostosis is characterized by premature fusion of cranial sutures. Commonly, it presents as an isolated suture, but multiple sutures may also be involved. More than 52 syndromes with craniosynostosis as a component have been identified. With some of the syndromes, craniosynostosis is severe and affects multiple sutures.
Growth of the head and the face is dependent on expansion of the cranium and, in the presence of craniosynostosis, craniostenosis can occur. Because growth occurs perpendicular to the cranial sutures, when a suture prematurely fuses, the head commonly develops an abnormal shape. This abnormal shape is consistent and depends on which suture fuses prematurely.

Sagittal synostosis → Scaphocephaly
Coronal synostosis → Brachycephaly
Unilateral coronal synostosis → Plagiocephaly
Lambdoid synostosis → Plagiocephaly
Metopic synostosis → Trigonocephaly

Isolated craniosynostosis usually is an esthetic problem, but functional problems can also occur. Increased intracranial pressure has been found in approximately 15% of cases with isolated unilateral coronal synostosis. Visual disturbances, cranial nerve deficits, seizures, and even death can result, depending on the severity of the increased intracranial pressure. Cerebrospinal fluid dynamics may also be affected. Fundoscopic examination by a pediatric ophthalmologist is highly recommended to rule out increased intracranial pressure. Worsening head asymmetry or abnormal head shape and radiographic confirmation of premature fusion of cranial sutures is diagnostic of the condition. CT scans with and without contrast confirm the diagnosis of craniosynostosis and permit the visualization of the bone and the ventricles.

For many children with craniosynostosis, correction is undertaken to improve the head and face shape. In others, it is done for functional and preventive measures. It is critical to distinguish craniosynostosis from head molding, because the latter usually improves on its own or with helmet therapy.

The frontal lobes quadruple in weight during the first year. Coincident with this brain growth is growth of the cranium. If maximum benefit is to be
derived from surgery to correct the synostosis, it should be undertaken during the first year. This allows subsequent growth of the head to follow growth of the brain. This is especially true for those patients with isolated suture fusion. Patients with multiple sutures involved, or with syndromes, respond quite differently and may require multiple procedures throughout early childhood and adolescence. Patients with secondary craniosynostosis (synostosis occurring because of lack of brain growth) are not good candidates to undergo surgical release procedures.

It is predictable that patients with craniofacial dysostosis (Crouzon, Apert, Pfeiffer syndromes) will benefit from surgery on at least three occasions:

Within the first year: Release of involved sutures along with forehead-supraorbital rim advancement and contouring. Frontofacial advancement is rarely, if ever, justified. Extreme exorbitism and nasopharyngeal hypoplasia would dictate this procedure during infancy.

Ages 4 to 7 years: Frontal advancement can be performed. This will finalize the forehead and supraorbital rim position. If it is successful, it will preclude the need for further intracranial surgery. It is predictable that additional midfacial surgery will be necessary, so when planning such surgery the forehead and supraorbital projection, and not the occlusion, are used as a guide to the amount of advancement.

Ages 12 to 18 years: Midface advancement at the subcranial level should be undertaken. Genioplasty commonly is performed, as is contour bone grafting of the middle face and nose. Soft tissue procedures, such as medial or lateral canthopexy, may also be performed.
Diagnosis and Treatment of Salivary Gland Diseases

Clinical History
The history is one of the most important tools in establishing a diagnosis for patients presenting with suspected salivary gland disorders. Pertinent questions should aid in the characterization of any mass (extent, duration, pain, rate of growth) and provide information about consistency of the saliva, taste characteristics, and the possible presence of other areas of involvement such as joints, eyes, and organs such as the pancreas, liver, or lungs.

Swelling: The most frequent sign causing a patient to seek care. It can be caused by nonneoplastic as well as neoplastic processes. Pertinent information about the location and occurrence of the swelling is critical in determining the differential diagnosis.

*Intermittent swelling:* If associated with eating (mealtime syndrome), it suggests an obstruction. The swelling will subside between meals if the gland is not infected. If the swelling is unrelated to mealtime, an infectious process should be considered.

*Persistent swelling:* Frequently caused by tumors or a generalized process such as Sjögren syndrome, diabetes, or alcoholism.

*Unilateral swelling:* Results from localized processes such as infections, tumors, or mechanical obstruction.

*Bilateral swelling:* Seen in association with a systemic condition
such as mumps or an endocrine dysfunction.

Pain: Pain and fullness of the gland, related only to eating, suggest obstruction. Infection and inflammation produce a more persistent pain that is not related to eating.

Salivary flow: Salivary flow can be decreased or increased. Normal unstimulated whole salivary flow is approximately 0.3 mL/min, whereas stimulated whole salivary secretion is 1 to 2 mL/min.

Xerostomia: Drugs are one of the most common causes of xerostomia. This can often be determined from the patient’s history. It can also be associated with Sjögren syndrome (see “Sjögren syndrome,” page 308) or occur secondary to radiation therapy.

Sialorrhea: This can either be a result of an increase in flow rate or, more likely, secondary to an inability to swallow normal secretions. Hypersecretion can be indicative of emotional or psychogenic factors or of a chronic neurologic disorder (eg, Parkinson disease, cerebral palsy, mental retardation, motor neuron disease, or amyotrophic lateral sclerosis).

**Physical Examination**

Size of the gland: A very large gland is more likely due to tumor than infection.

Consistency of swelling: Firm or hard (tumor), soft or rubbery (possible enlarged lymph node), movable or fixed.

Palpation for stones: Location, number, and size.

Localized or diffuse enlargement: Diffuse enlargement of a single gland suggests a chronic inflammatory disorder, whereas involvement of multiple glands is suggestive of Sjögren syndrome, an endocrine disorder (diabetes mellitus), a metabolic disorder (alcoholic cirrhosis), or a nutritional
deficiency (malnutrition).

Location of mass: In the preauricular and submandibular regions, parenchymal glandular involvement must be distinguished from regional lymph node involvement (granulomatous or hematopoietic disease).

Neurologic findings: Malignant parotid tumors can affect the facial, glossopharyngeal, vagus, spinal accessory, and hypoglossal nerves, especially when there is involvement of the deep lobe of the gland. Submandibular tumors can involve the lingual and hypoglossal nerves.

Amount and character of saliva: If involvement is unilateral, it is helpful to compare salivary flow from the gland with the one on the contralateral side. Flecks of pus or turbidity indicate an infectious process.

**Preoperative Workup**

**Plain Radiographs**

Plain radiographs can be helpful in detecting the presence of sialoliths.

Occlusal view: Used to detect calculi in the floor of the mouth. It is important to decrease the normal dose of radiation so as not to “burn out” poorly calcified stones. Two projections are needed—true occlusal and oblique. The true occlusal view can detect stones up to the first molar; the oblique projection shows stones in the hilar region and the submandibular gland.

Panoramic view: Valuable in detecting stones in the submandibular gland.

Anteroposterior (AP) view: Used for visualizing parotid stones.

**Sialography**

Sialography is based on insertion of an iodine-containing contrast medium into the ductal system via the duct orifice.

Indications: Inflammatory diseases, sialolithiasis, suspected strictures, demonstration of glandular function, systemic diseases such as Sjögren syndrome, cheek trauma

Contraindications: Suspected iodine allergy and during the acute phase of sialadenitis

**Ultrasound**
Ultrasound presents the advantage of avoiding radiation exposure.

Indications
- Detect space-occupying lesions. It can differentiate a cystic lesion from a solid mass.
- Differentiate an intraglandular neoplasm from a superficially located lesion extrinsic to the gland.
- In conjunction with the Doppler technique, it is useful to assess for vascular lesions, gland vascularity, and tumors.

Contraindications
- The deep lobe of the parotid gland cannot be visualized.
- Low operator experience level.

Interpretation
- *Pleomorphic adenoma*: Hypoechoic, slightly heterogeneous, solid lesion with smooth outline
- *Warthin tumor*: Hypoechoic
- *Carcinoma*: Hypoechoic, heterogeneous with irregular, ill-defined borders

Computed Tomography (CT)

Indications
- Masses in or adjacent to the salivary glands
- Diffuse noninflammatory enlargement
- Almost diagnostic for lipoma (rare) based on densitometric values

Contraindications
- Tumors in the submandibular glands (ultrasound or magnetic resonance imaging (MRI) preferred)
- Dental restorations (may cause artifacts that can mask tumors or stones)

Magnetic Resonance Imaging (MRI)

MRI is excellent for imaging soft tissue involvement without radiation exposure. It is associated with minimal artifact degradation caused by dental restorations. Indications
- Detection of a lesion or mass (better than CT for determining lesion margins, architecture, regional extension)
- Suspected vascular tumors

Sialo-MRI
Technique to investigate the ductal system of the salivary glands using the saliva as the contrast medium. It is therefore noninvasive and painless.

Indications/advantages
- Inflammatory diseases, sialolithiasis, swelling of unknown etiology, suspected strictures
- Demonstrates all four major glands

Disadvantage
- Relies on salivary secretion, does not demonstrate stones, expensive

Radionuclide Scan
Indications
- Evaluation of glandular parenchyma.
- Warthin tumor and oncocytoma usually show increased uptake.

Disadvantage
- Nonspecific method

Sialendoscopy
This is a simple, minimally invasive technique in which the ductal system is visualized via a miniature endoscope (0.9- to 2-mm diameter). Endoscopy is a good diagnostic tool as well as a treatment modality for inflammatory and obstructive diseases.

Indications
- Diagnosis and treatment of stones, strictures, and inflammatory conditions
- Diagnosis of swelling of unknown etiology of the major salivary glands

Contraindications
- Should not be used when there is an acute infection
Biopsy

Intraoral minor salivary gland: A labial salivary gland biopsy is done initially in suspected cases of Sjögren syndrome or sarcoidosis. If the report is negative or equivocal and contradicts other available information, a parotid gland biopsy should be considered. The labial biopsy is performed in the lower lip between the midline and commissure. The lip is everted, and digital pressure is applied on both sides of the biopsy site to facilitate the procedure. A single 1.5- to 2-cm incision is made only through the mucosa. Careful dissection is done to expose the underlying minor salivary glands and prevent injury to any small mental nerve branches. At least five minor salivary glands are removed and placed in formalin. The mucosa is approximated with 4-0 poly-glactin sutures.

Parotid gland: The incision is placed in the retromandibular crease below the inferior aspect of the ear lobule. The tail of the parotid gland is identified after dissection through the overlying superficial and deep cervical fascia and the parotideomasseteric fascia inferior and posterior to the main trunk and branches of the facial nerve, and a small portion is removed.

Fine needle aspiration (FNA): Used in the diagnosis of masses in the salivary glands. There are few false positives, but a fair number of false negatives.

**Indications**

- Determine if mass is benign or malignant.
- Differentiate enlarged parotid lymph nodes from salivary gland tumors.
- Determine tissue of origin of a mass.
- Determine mode of treatment; may require further investigation.

**Technique:** Guided ultrasound FNA procedure is the preferred method.

Sialochemistry

Saliva can be collected from the parotid and submandibular glands by cannulation of their ducts. The saliva can be analyzed for electrolytes
(sodium, potassium, chloride, phosphate), flow rate, total salivary protein (amylase, glycoproteins, albumin), and immunoglobulins.

**Salivary Gland Diseases**

Salivary gland pathology can be divided into:

- Inflammatory conditions (sialadenitis, sialolithiasis)
- Neoplasms
- Mucoceles
- Ranulas
- Rarities
- Traumatic injuries
- Pediatric salivary gland diseases

**Sialadenitis**

Sialadenitis of the major salivary glands can take many forms. The parotid glands are most prone to sialadenitis. The etiology of sialadenitis is not clear, but decreased salivary production or obstructive salivary stagnation that encourages ascending salivary duct infection is a prominent theory. Other etiologic factors that facilitate sialadenitis are anticholinergic drugs (antihistamines, psychotherapeutics), metabolic disorders, liver cirrhosis, vitamin B$_{12}$ deficiency, Sjögren syndrome, granulomatous diseases, and radioactive iodine treatment for thyroid malignancies. Juvenile recurrent parotitis, which continues to adulthood, is a form of chronic sialadenitis.

**Clinical presentation**

*Parotid gland:* Most of the parotid inflammations present as chronic recurrent parotitis (CRP). The patient develops unilateral or bilateral mildly tender swelling of the parotid area that may last from a few days to months. During this time, intermittent remissions and exacerbations may occur. Sometimes there may be abscess formation, which is characterized by a red, painful, indurated swelling. Fever and malaise may accompany the transient recurrences. Intraorally, there is swelling and redness of the Stensen duct orifice, with viscous and plaquelike saliva formation; absent or decreased salivary secretion can be observed
in 20% to 50% of patients.

**Submandibular gland:** Sialadenitis is characterized by swelling of
the submandibular region and the floor of the mouth, especially in
the area of the Wharton duct papilla. Pressure on the affected
gland may produce plaques or pus, or there may be absence of
secretion from the orifice. The patient will report having
mealtime syndrome (ie, swelling and pain in the affected gland
during eating).

Differential diagnosis

The differential diagnosis of patients with suspected glandular swelling
includes sialolithiasis, salivary gland tumors, odontogenic infections,
lymphadenitis, sialosis, and Sjögren syndrome. Rarities to be considered are
mumps, hemangioma, lymphangioma, pneumoparotid, atypical
mycobacterial infection, tuberculosis, actinomycosis, AIDS, and vascular
malformation.

Imaging methods

Imaging methods include plain radiographs, sialography, ultrasound, CT,
MRI, and sialendoscopy. The most sensitive imaging method is the
sialogram, followed by sialo-MRI, MRI, ultrasound, and CT scanning with
contrast medium. Typically, the sialogram and sialo-MRI of patients
suffering from CRP will show punctate sialectasis, strictures, and dilatation
of the ductal system (ie, a “sausagelike” appearance). Ultrasound and CT
scanning will show dilatation of the main duct and large areas of sialectasis,
but not strictures. Although ultrasound is considered a sensitive method for
imaging soft tissues, only 18% of the patients with CRP show any pathologic
findings on ultrasound.

A new and effective method of visualizing, evaluating, and treating the
ductal system is sialendoscopy. Sialendoscopy in patients with CRP will
reveal plaques, strictures, dilatations, a matted appearance of the duct,
absence of blood vessels, and ecchymosis in the lining mucosa of the duct. In
cases of submandibular sialadenitis, the diagnosis is based on the clinical
findings when imaging does not demonstrate any obstructions, but the patient
has swelling. Sialendoscopy will demonstrate plaques and sometimes
adhesions.

Treatment—Acute phase

*Parotid gland:* Antibiotic treatment directed to the main bacterial causes (*Staphylococcus aureus* and anaerobic bacteria), hydration, dilation of Stensen duct, encouraging the patient to massage the affected gland, and surgical drainage if there is an abscess.

*Submandibular gland:* Same as parotid gland, except antibiotic treatment is directed against the streptococcal flora.

Treatment—Chronic phase

*Parotid gland:* In recurrent cases (more than one episode of swelling) or if any pathology is noted on imaging (strictures, dilatations, sialectasis), proceed to the endoscopic approach to investigate and treat the affected gland. The endoscope is used to dilate strictures and to thoroughly lavage the ductal system. The dilation is performed mainly with hydrostatic pressure, sialoballons, and forced manipulation.

*Submandibular gland:* Treat in the same manner as the parotid gland with endoscopic lavage and dilation of any strictures.

The last line of treatment for both glands if endoscopic management is unsuccessful is superficial, subtotal, or total parotidectomy or submandibular sialoadenectomy.

Radioiodine-induced sialadenitis

Salivary gland dysfunction is a well-recognized side effect in patients undergoing high-dose radioiodine therapy for thyroid problems because of the selective ability of the glands to concentrate iodine. The most prevalent findings are bilateral radiation-induced parotid sialadenitis, followed by impairment of taste and smell and complete xerostomia. The main pathology
in the affected glands is strictures in the ductal system. Sialography is a simple yet reliable method of diagnosis of radioactive sialadenitis. Sialogogue administration may lessen parenchymal damage through promotion of iodine discharge from the gland. Sialendoscopy is indicated for treatment of ductal system constrictions.

Sialolithiasis

Sialolithiasis is a common finding, accounting for 50% of major salivary gland disease. The submandibular gland is most prone to sialolithiasis.

Clinical presentation

The symptoms and signs of sialolithiasis are similar to sialadenitis (see Sialadenitis section, page 302) and can range from intermittent pain and swelling during mealtime to severe infections of the gland leading to airway obstruction in the submandibular cases.

Differential diagnosis

See Sialadenitis section.

Imaging methods

Imaging of the gland for possible sialoliths involves plain radiographs (occlusal, occlusal oblique, panoramic), sialography, ultrasound, and CT (see Sialadenitis section). It is important to remember that 32% of the calculi in the submandibular gland and 63% of those in the parotid gland are radiolucent and will not be seen on radiographic imaging. Sialendoscopy can also be used to search for sialoliths in the glands and is helpful when they are suspected and not seen on radiographs.

First line of treatment

**Submandibular gland:** Treatment is based on the location, diameter, and mobility of the stones. Stones located in the anterior and middle part of the Wharton duct are extracted via intraoral sialolithotomy, and the duct and the gland are explored endoscopically. Stones in the posterior part of the duct and the hilum of the gland up to 5 mm in diameter are retrieved intra-
ductally using an endoscope. Larger posterior and hilum stones are removed through an endoscope-assisted technique (ie, first exploring and exposing the duct and the stone via an intraoral approach). If midsize or large stones are attached firmly to the ductal tissue or the intraglandular tissue in the posterior part of the duct, extracorporeal shock wave lithotripsy (ESWL) is the first treatment. It is followed by sialendoscopy or, in cases of large stones, the endoscope-assisted procedure.

**Parotid gland:** Stones located in the anterior part of the Stensen duct are removed via intraoral sialolithotomy followed by endoscopic exploration of the gland. For all other stones in the parotid region the first line of treatment is ESWL in conjunction with sialendoscopy.

**Second line of treatment**

If submandibular stones up to 5 mm in diameter in the posterior duct or hilum are not successfully removed endoscopically, the endoscope-assisted procedure is used. If the endoscope-assisted technique fails, ESWL, followed by endoscopy or the endoscope-assisted procedure, is done. For parotid stones that cannot be removed by ESWL, an endoscope-assisted extraoral approach is the next step.

**Third line of treatment**

The third line of treatment, when the other procedures fail, is sialoadenectomy.

**Neoplasms**

**Epidemiology**

- 80% of salivary gland tumors occur in the parotid gland. 80% are benign; 20% are malignant.
- 10% to 15% of the tumors occur in the submandibular gland. Up to 50% of these tumors are malignant.
- Approximately 1% of salivary gland tumors occur in the sublingual gland. Up to 90% are malignant.
- 5% to 10% of salivary gland tumors occur in minor salivary glands. Up
to 50% are malignant.

Benign tumors

Pleomorphic adenoma: This is the most common salivary gland tumor. The majority arise in the parotid gland. Clinically, the tumor appears as a slow-growing, painless, freely movable nodule usually found in persons between 40 and 70 years of age. The tumor rarely ulcerates. Intraorally, the most common sites are the hard palate and upper lip. With a tumor in the submandibular gland, complete excision of the gland is indicated, whereas for a tumor in the parotid gland extracapsular excision, partial superficial excision, or superficial or total parotidectomy is done, depending on the location and extent of the lesion. On the hard palate, the excision includes the overlying mucosa and extends down to and includes the periosteum. Pleomorphic adenomas recur in 2% to 3% of patients following parotidectomy. Malignant transformation has been reported in 3% of all pleomorphic adenomas.

Adenolymphoma (Warthin tumor, papillary cystadenoma lymphomatosum): Mostly found in the tail of the parotid gland. Most prevalent in men between 55 and 65 years of age. The tumor is usually soft and cystic in consistency and grows slowly. Growth is limited and superficial. Usually appears as a “hot” nodule on scintigraphy. Treatment consists of extracapsular excision or partial superficial or superficial parotidectomy. About 7% of adenolymphomas are bilateral.

Other rare benign salivary gland tumors: Lymphoepithelial lesion, oncocytoma, monomorphic adenoma.

Malignant tumors

Mucoepidermoid carcinoma: Occurs mainly in the parotid and minor salivary glands. Most occur in persons 40 to 60 years of
age. Clinically it can be mistaken for a mucocele and histologically with necrotizing sialometaplasia. Histologic differentiation is based on the predominant type of cell. If more than 50% of the specimen has mucus-forming cells, the tumor is classified as well differentiated or low grade, with an overall good prognosis. If the predominant cell type consists of epidermoid cells with less than 10% mucus-forming cells, the tumor is considered high grade or poorly differentiated, with a low survival rate. The low-grade tumor is treated by wide local soft tissue excision with a 1-cm margin of normal tissue. The high-grade tumor should be treated by wide resection, with strong consideration for a neck dissection and postsurgical radiotherapy.

**Adenoid cystic carcinoma:** Occurs most frequently in the palate and is the most common malignant tumor of the submandibular gland. It is commonly seen in persons 50 to 70 years of age. Perineural spread is one of the characteristics of this tumor, as well as infiltration into adjacent vessels and bone. Treatment is radical excision of the primary site with special attention to deep margins and involved nerves. The risks and benefits of postsurgical radiotherapy or chemotherapy should be carefully evaluated, especially considering the poor long-term survival rate.

**Polymorphous low-grade adenocarcinoma:** Occurs almost exclusively in minor salivary glands. Presents as a painless mass, localized in 60% of cases to the hard or soft palate. Histologically, at low power, it appears well encapsulated; however, the peripheral cells may infiltrate into adjacent bone and nerves. This tumor has a very good prognosis, with a cure rate of about 80% after wide surgical excision. It is important to differentiate this tumor from adenoid cystic carcinoma because of the difference in outcome. Even if the histologic cribriform appearance and the infiltration patterns are similar to adenoid cystic carcinoma, the presence of mitotic figures is uncommon in the polymorphous low-grade adenocarcinoma.

**Carcinoma ex pleomorphic adenoma:** In most cases it occurs in the major salivary glands. If the patient reports a long-standing lesion with recent sudden and rapid growth, it may represent a
change in biologic behavior, with a pleomorphic adenoma undergoing malignant change. Treatment involves wide surgical excision. The location of the malignant transformation in the outer surface of the tumor is a crucial point in determining treatment.

**Acinic cell carcinoma:** The majority of these low-grade malignancies occur in the parotid gland, with a peak incidence in persons 50 to 70 years of age. Treatment is wide local excision without neck dissection.

**Mucocele (Mucus Retention Phenomenon)**

A mucocele represents a leakage of saliva into the submucosal tissues from a minor salivary gland due to trauma to its duct. Mucoceles are seen mainly in children and young adults in any location in the oral region, but the lower lip is the most frequent site because it is readily traumatized during oral function. They are a painless, soft, oval, fluctuant, superficially positioned, fluid-containing bluish mass. Spontaneous rupture followed by recurrence is a characteristic finding. Infrequently, a true cyst (retention cyst) with an epithelial lining can occur; however, most mucoceles represent an extravasation phenomenon, with a connective tissue rather than an epithelial lining. A variant of a mucocele, known as a *superficial mucocele*, can be located on the palate or in the retromolar region and appears as a small vesicle.

**Differential diagnosis**

The mucocele needs to be differentiated from the hemangioma, lymphangioma, and low-grade mucoepidermoid carcinoma. Digital pressure on a hemangioma changes the color of the lesion from blue to white and reduces its volume, which does not occur with a mucocele. Mucoepidermoid carcinoma and lymphangioma can only be diagnosed microscopically.

**Treatment**

There are two surgical approaches for mucoceles: excision or marsupialization (unroofing). Excision is the treatment of choice for small- or medium-sized mucoceles. This involves an incision through the dome of the lesion and exposure of the underlying minor salivary glands, which are
excised. Marsupialization is the treatment of choice for large mucoceles when excision can result in injury to anatomic structures such as branches of the mental nerve.

Ranulas

A ranula develops from extravasation of mucus in the floor of the mouth following trauma to the sublingual gland or obstruction of its ducts. The peak frequency of ranulas is at the age of 20 to 30 years; there is a slight predilection for females. Ranulas are classified as intraoral or plunging according to whether the accumulation of mucus is located above or below the mylohyoid muscle.

Intraoral ranula: This presents as a soft, bluish, fluctuant, generally unilateral swelling in the floor of the mouth. Large ranulas can cause deviation of the tongue and interfere with tongue movement.

Plunging ranula: This occurs when there is a sufficiently large defect in the mylohyoid muscle that allows saliva from the damaged sublingual gland to penetrate the submandibular space (42% of the population has such a mylohyoid defect). The swelling is both intraoral and extraoral in the submandibular and submental spaces.

Diagnosis

Diagnosis is based on the location and clinical appearance for the intraoral ranula; for plunging ranula, a CT scan or MRI demonstrates a fluid-filled lesion inferior to the mylohyoid muscle. If there is doubt about the diagnosis, aspiration of the lesion and a laboratory test for salivary amylase should confirm the diagnosis.

Differential Diagnosis

Intraoral and plunging ranulas need to be differentiated from hemangioma, lymphangioma, and low-grade mucoepidermoid carcinoma. Plunging ranulas also must be distinguished from cystic hygroma, branchial cleft cysts, and dermoid and epidermoid cysts.

Treatment
Intraoral ranula: The first line of treatment is marsupialization (unroofing) and suturing of the wound margins to the oral mucosa with resorbable sutures, and then packing the cavity with iodoform gauze. The packing should remain for at least 10 days. Marsupialization without packing is not recommended because the failure rate can be between 61% and 89%. Removal of the sublingual gland can also be done to treat a ranula primarily or if marsupialization fails.

Plunging ranula: The treatment for this type of ranula is intraoral sublingual sialoadenectomy.

A new treatment for both intraoral and plunging ranula is the intralesional injection of OK-432 (sclerotherapy). In a recent study, 77% showed a complete response.

Rarities
Sjögren syndrome

Clinical features: Sjögren syndrome is a chronic systemic autoimmune disease characterized by the triad of xerostomia (mouth), keratoconjunctivitis sicca (eyes), and a connective tissue disease (usually rheumatoid arthritis). It can be primary, involving glandular elements, or secondary, with connective tissue being the primary target. It occurs predominantly in women (9:1) age 40 years or older. A genetic predisposition to the disease is seen in individuals with the HLA-MT2 histocompatibility antigen. Salivary, mucus, and lacrimal gland replacement by a lymphocytic infiltrate causes the classic symptoms of dry eyes, dry mouth, and parotid swelling.

Diagnosis: Diagnostic tests should include the Schirmer test for lacrimal gland activity when there are dry eyes; sialograms, sialometry, scintigraphy, and sialochemistry when dry mouth is the main pathology. When connective tissue disease is in the
differential diagnosis, tests for rheumatoid arthritis, antinuclear antibodies, and anti–SS-A (anti-Ro) and anti–SS-B (anti-La) antibodies; labial and/or parotid biopsy; red blood cell, white blood cell, and differential count; and urinalysis should be done.

*Treatment:* This involves supportive care with artificial tears and saliva for the dry eyes and mouth, along with treatment of the autoimmune connective tissue disease. Pilocarpine has been used successfully to induce muscarinic cholinergic stimulation of salivary secretion. Oral plaque control and fluoride therapy are important because of the caries susceptibility. A small percentage of patients (5% to 8%) develop lymphoma; therefore, close follow-up is necessary.

**Tuberculosis**

This is most often seen in the parotid gland. It is usually unilateral and may be primary but usually arises from the tonsil or periglandular lymph nodes. In generalized tuberculosis, the submandibular and sublingual glands are most often involved. The clinical picture is one of a firm, nontender swelling similar to a tumor. Draining sinus tracts may be present.

**Actinomycosis**

The parotid gland is affected most frequently. Infection occurs either as an acute situation with pain, swelling, abscess formation, and eventual ulceration and breakdown with sinus tract formation, or in a chronic form similar to tuberculosis, appearing as an asymptomatic mass present for several months. Actinomycosis must be differentiated from tumors and mycobacterial infection. The diagnosis is based on culture and a biopsy of the lesion showing the presence of gram-positive, microaerophilic, nonspore-forming, nonacid-fast bacteria. Frequently, biopsies of the wall of the abscess or sinus tract will aid in the diagnosis. Treatment involves antibiotics (penicillin) for weeks to months.

**Sarcoidosis**

Sarcoidosis is generally associated with pulmonary problems and bilateral hilar adenopathy. The parotid gland is involved in 6% of cases, with enlargement, xerostomia, and fever (Heerfordt syndrome; uveoparotid fever).
A skin test (Kveim) can be diagnostic, but a biopsy of the lesion will confirm the disease. Laboratory tests show elevation of serum calcium, alkaline phosphatase, gamma globulins, and amylase.

Nutritional problems

Starvation, malnutrition, and eating disorders (eg, anorexia, bulimia) predispose individuals to swelling of the parotid gland (called nutritional mumps). Swelling is usually bilateral and diffuse and results from an increase in size of the acini.

Alcoholic cirrhosis

This condition is characterized by parotid swelling that decreases or increases with improvement or decompensation of the cirrhotic process. Sialograms show a “leafless winter tree” appearance because of the lack of normal glandular arborization.

Traumatic Injuries (see also chapter 16, page 242)

Stensen duct: Whenever there is a wound in the cheek, the duct should be probed from the mouth with a lacrimal probe to ascertain if it is cut, lacerated, or intact. Injection of saline into the ductal orifice will aid in this assessment. Other possibilities include a CT scan with contrast medium injected into the duct or examination with a sialendoscope. If the duct is intact, it is sometimes advisable to insert a catheter to prevent obstruction secondary to scarring during the healing process. If transected, the duct should be repaired as soon as possible by threading a catheter (polyethylene tubing) from the distal (intraoral) to the proximal (intraglandular) stump and suturing the duct together with 8-0 or 9-0 nylon. If the wound involves damage to the gland itself, the insertion of a positive pressure drain directed from the wound to the oral cavity is essential to avoid a sialocele. It is also important to carefully suture the parenchyma and to close the glandular capsule as well as to repair the subcutaneous tissues and skin in layers. A tight pressure dressing is applied for 24 to 48 hours. The drain is maintained with vacuum for 7 days and then removed. The intraluminal catheter, which is sutured to the oral mucosa with 4-0 silk to prevent dislodgment, is removed after 10 to 14 days. If the orifice of the duct is damaged, a “fish-mouth” technique (suturing the remaining duct to the oral mucosa) is used to create a
new orifice, and a catheter is left in place for 10 to 14 days. If the duct is irreparable, the proximal end should be ligated to produce atrophy of the gland.

Wharton duct: When lacerated, the proximal end should be externalized into the mouth at the site of injury. The submandibular parenchyma is treated the same as in the parotid gland.

Pediatric Salivary Gland Disease

Inflammatory disorders in childhood are far more common than neoplastic salivary gland pathology. The most common inflammatory condition is mumps, followed by juvenile recurrent parotitis, and obstructive sialadenitis (see “Sialadenitis” page 302). Acute suppurative parotitis and submandibular sialadenitis (without obstruction) are very rare conditions in children. Infection of the salivary glands in children, without sialolithiasis, occurs mostly in the parotid gland; calculi are far more common in the submandibular glands.

Mumps

This is a nonsuppurative, acute sialadenitis of viral origin. It is a contagious disease that presents as a painful bilateral enlargement of the salivary glands, especially the parotid glands. Incubation is 2 to 3 weeks. It affects mostly children between the ages of 6 to 8 years. Laboratory tests are nonspecific except for leukopenia and an increase in serum amylase. Symptoms subside in 3 to 7 days, and complete recovery occurs within 2 to 3 weeks. Treatment involves relief of pain and fever and prevention of dehydration. Persistence of swelling indicates a secondary bacterial infection.

Juvenile recurrent parotitis

Juvenile recurrent parotitis (JRP) is defined as recurrent, nonobstructive, nonsuppurative parotid inflammation. The condition is characterized by swelling of the parotid gland, usually accompanied by pain and systemic symptoms such as fever and malaise. It is most commonly unilateral, but bilateral involvement can occur. When the symptoms are bilateral they are usually more prominent on one side. Swelling appears suddenly over a period of a few hours and may be accompanied by xerostomia.

The disease can start at any age between 3 months and 16 years, with a
peak at 5 to 7 years. Exacerbations last for several days, and the episodes may occur over many years with variable frequency.

A variety of etiologic factors have been considered for JRP: congenital ductal malformations, hereditary-genetic factors, viral or bacterial infection, allergy, and local manifestation of an autoimmune disease. The self-limiting nature of the disease, the reports of a male predilection, and the absence of immunoglobulin abnormalities indicate that JRP is probably not an autoimmune disease. However, recently the possibility of ductal malformation has been suggested as the main cause. Treatment involves sialendoscopy with dilatation and irrigation procedures.

Benign neoplasms

Neoplasms are uncommon in children. The highest incidence is in the parotid gland. The most common types are vasoformative, with a solid tumor more likely to be malignant than in the adult.

**Hemangioma**: This is the most common neoplasm. It is generally found in the parotid gland. The lesion has a soft, doughy consistency and a bluish color. It increases in size with age and during crying or straining. It can cause elevation of the lower portion of the ear, distortion of the cheek, and obliteration of the auditory canal. Treatment consists of excision if there is rapid growth, functional impairment, infection, hemorrhage, or ulceration. Surgery requires subtotal parotidectomy with facial nerve preservation.

**Lymphangioma (cystic hygroma)**: This lesion is seen shortly after birth (50% of cases before 1 year of age; 90% before 2 years of age). It occurs primarily in the neck, but can occur in the parotid gland. The lesion is cystic in nature, with a spongy consistency. It enlarges gradually, slower than a hemangioma, and can cause a cosmetic problem. Indications for removal are gradual enlargement with local extension and infection. Treatment consists of complete surgical excision with preservation of the facial nerve.

**Pleomorphic adenoma**: This is the most common benign
nonvascular tumor in children. It presents as a firm to hard, nontender, mobile mass, varying in size from 2 to 10 cm. It occurs most often in the tail of the parotid gland. A sialogram is used to differentiate it from sialadenitis (ductal ectasia). In addition, CT or fine needle aspiration biopsy can be used. Treatment is similar to that in adults (see Pleomorphic adenoma, page 305).

Malignant neoplasms

One-third of all salivary gland tumors in children are malignant. They present as a firm, painful mass, associated with rapid growth and facial nerve weakness. They are usually larger in size than benign tumors and are attached to underlying structures. Diagnosis is made by fine needle aspiration biopsy, CT, and sialography. Forty percent of the malignant tumors are mucoepidermoid carcinomas. Treatment is excision with sacrifice of the facial nerve if it is involved with the tumor.
Diagnosis and Treatment of Temporomandibular Disorders

Temporomandibular disorders (TMDs) is a collective term that includes both those pathologic conditions that involve the temporomandibular joint (TMJ) and the myofascial pain and jaw dysfunction (MPD) that can arise from the muscles of mastication. Although these two groups of conditions have differing etiologies and involve distinct anatomical regions, they have been grouped together because they frequently produce very similar signs and symptoms. Moreover, patients with chronic MPD can develop secondary TMJ involvement. Because of these factors, differential diagnosis can be a difficult problem. In the following sections, the diagnosis and treatment of the various TMDs is discussed.

Pathologic Conditions Involving the TMJ

Congenital Anomalies

Condylar agenesis (see also chapter 19)

- Hemifacial microsomia
- Goldenhar syndrome

Clinical manifestations

- Present at birth
- Usually unilateral, but can be bilateral
- Articular fossa, condylar and coronoid processes, ramus, and parts of mandibular body rudimentary or absent
- Mandibular deviation to affected side
- Macrostomia
Abnormalities of external and internal ear
Epibulbar dermoids and vertebral anomalies in Goldenhar syndrome

**Treatment**
- Reconstruction of mandible and redirection of growth by distraction osteogenesis or costochondral graft
- Ear reconstruction or prosthesis
- Orthodontics
- Orthognathic surgery

Developmental Anomalies
Condylar hypoplasia

*Clinical manifestations*
- On affected side when unilateral
  - Condylar deformity
  - Short, wide ramus
  - Antegonial notching
  - Fullness of face
- On unaffected side when unilateral
  - Elongated mandibular body
  - Deviation of the chin
  - Flatness of face
- Bilateral involvement
  - Condylar deformity
  - Inferior bending of mandible (“bird face”)
  - Severe antegonial notching
  - Severe Class II malocclusion

*Treatment*
- Reconstruction with costochondral graft
• Distraction osteogenesis
• Orthodontics and orthognathic surgery

Condylar hyperplasia

Clinical manifestations
• Facial asymmetry
• Prognathic profile
• Symmetric condylar enlargement (long condylar neck and deep sigmoid notch)
• No antegonial notching
• Bowing of inferior mandibular border

Treatment
• Determine growth status of condyle by bone scan or serial cephalometric tracings.
• If still growing, perform condylotomy; if not growing, perform orthognathic surgery.
• Recontour lower border of mandible, if necessary.

Traumatic Injuries

Traumatic arthritis (see Arthritis section, page 315)
Condylar fractures (see chapter 16)
Acute mandibular dislocation

Clinical manifestations
• Mandible locked in open mouth position

Treatment
• Attempt reduction by pushing downward on posterior mandible and then moving the jaw backward toward the
If unable to reduce or patient uncooperative, inject a local anesthetic into the lateral pterygoid muscle either intraorally or extraorally. May also need sedation or general anesthesia.

Chronic, recurrent mandibular dislocation

*Clinical manifestations*
- Patient has mandible locked in open mouth position
- History of repeated episodes of dislocation

*Treatment*
- Reduce dislocation
- Injection of sclerosing solution into TMJs (sodium morrhuate, sodium so-tradecol)
- Capsulorrhaphy
- In patients with uncontrollable etiology (eg, seizures, dyskinesia), perform lateral pterygoid myotomy

Chronic, persistent dislocation

*Clinical manifestations*
- Mandible locked in open mouth position
- Condition present for several months or longer

*Treatment*
- Attempt manual reduction under general anesthesia
- Can use traction wires placed in the mandibular angles for added downward traction
- If still unable to reduce, perform bilateral intraoral temporals myotomy
Neoplasms

Benign lesions (most common: chondroma, osteoma, osteochondroma)

Clinical manifestations
- Condylar deformity
- Elongation of condylar neck
- Mandibular deviation to unaffected side
- Crossbite malocclusion
- Ipsilateral open bite

Treatment
- Condylectomy
- Possible reconstruction if condylar neck cannot be contoured to form a new condyle
- Possible orthognathic surgery to correct mandibular deformity and reestablish the occlusion
- Possible genioplasty

Malignant lesions (most common: chondrosarcoma and osteosarcoma)

Clinical manifestations
- Pain and swelling
- Limited mouth opening
- Destruction of condylar architecture
- Mandibular deviation to affected side, depending on amount of bony resorption

Treatment
- Wide surgical excision
- Usually not radiosensitive
- Reconstruction after adequate tumor-free period
Metastatic lesions (usually from primary lesion in lung, thyroid gland, prostate, kidney, breast, adrenal gland, or gastrointestinal tract)

Clinical manifestations
- Pain
- Radiolucent area in the condyle

Treatment
- Determination of primary site, resectability of the lesion, and the extent of other metastases
- Resection of tumor, if indicated by ability to control the primary site and lack of other metastases

Arthritis
Infectious arthritis

Clinical manifestations
- Preauricular pain, swelling, and erythema
- Possible fluctuation
- Fever and malaise
- Possible presence of infection in adjacent mandible, ear, parotid gland, or throat
- Possible history of tuberculosis, syphilis, or gonorrhea

Treatment
- Aspiration or incision and drainage
- Culture and antibiotic sensitivity testing
- Antibiotic therapy
- Treatment of associated systemic disease, if present
- Possible debridement of infected bone
Traumatic arthritis

Clinical manifestations

- History of trauma to mandible without evidence of a condylar fracture
- TMJ pain and tenderness
- Limitation of mouth opening

Treatment

- Nonsteroidal anti-inflammatory drugs (NSAIDs)
- Intermittent, moist heat to joint
- Soft, nonchewy diet
- Limitation of jaw movement
- Possible arthrocentesis
- Physical therapy to prevent ankylosis once acute symptoms subside

Rheumatoid arthritis

Clinical manifestations

- Intermittent bilateral TMJ swelling and tenderness
- Dull, aching pain
- Limited mouth opening
- TMJ stiffness, worse in morning
- Condylar flattening and erosion
- Anterior open bite and/or retrognathia in advanced cases
- Evidence of other symmetric small joint involvement (hands, wrists, elbows, shoulders, ankles, toes, neck)
- Anemia, fever, malaise, anorexia
- Positive rheumatoid factor test, antinuclear antibody test, erythrocyte sedimentation rate

Treatment

- NSAIDs
• Short-term steroids
• Soft, nonchewy diet
• Mild range of motion exercises
• Immunosuppressive drugs (managed by rheumatologist)
• Surgical correction of ankylosis, apertognathia, and/or retrognathia when disease is inactive

Osteoarthritis (degenerative joint disease)

Clinical manifestations
• History of trauma or chronic parafunction
• Constant unilateral aching pain in TMJ that increases with function
• Decreased mouth opening
• TMJ tenderness
• Radiographic evidence of condylar flattening and lipping, erosion, and osteophyte formation in late stages
• Crepitant sounds in joint in late stages

Treatment
• NSAIDs
• Soft, nonchewy diet
• Limited jaw function
• Bite appliance to control parafunction
• Establishment of a stable occlusion
• Do arthroplasty only if 3 to 6 months of medical management fails and there is radiographic evidence of condylar alteration

Idiopathic Condylar Resorption (Condylar Osteolysis)

Clinical manifestations
• Frequent history of orthodontics or orthognathic surgery
• Predilection for females between 15 and 35 years of age
• Presence of Class II malocclusion and high mandibular plane angle
• Anterior open bite

Treatment
• Perform bone scan to determine activity. Do not treat if scan is positive.
• If scan is negative, perform maxillary orthognathic surgery.
• If orthognathic surgery is unsuccessful, perform condylectomy and costochondral graft or an alloplastic total joint replacement.

Ankylosis

Clinical manifestations
• History of trauma, infection, or rheumatoid arthritis
• Can be false ankylosis due to extra-articular causes such as elongated coronoid process, depressed zygomatic arch, masticatory muscle scarring, postirradiation fibrosis, paramandibular neoplasia, myositis ossificans, osteochondroma of coronoid process, or neurologic disorder
• Painless restriction of mouth opening
• Mandibular deformity if intra-articular damage to the condyle occurred during the patient’s growth period (see Condylar hypoplasia, page 313)

Treatment
• True ankylosis
  — Mechanical dilation of jaws
  — Condylectomy
  — Gap arthroplasty
  — Alloplastic total joint
  — Orthognathic surgery to correct any maxillofacial deformity

• False ankylosis
  — Site-specific treatment

Internal Derangements

Anterior disc displacement with reduction
Clinical manifestations

- TMJ clicking or popping
- Joint pain and tenderness
- History of trauma or chronic parafunction

Treatment

- Analgesic for pain
- Soft, nonchewy diet
- Limited jaw function
- Bite appliance if parafunction is diagnosed
- Do not treat further if pain is eliminated. If pain persists, perform discoplasty.

Anterior disc displacement without reduction

Clinical manifestations

- Painful, limitation of mouth opening
- No joint noise
- Prior history of TMJ clicking or popping

Treatment

- Arthrocentesis
- Arthroscopic surgery
- Discoplasty. Only perform discectomy if disc is not preservable. Patient can usually function with displaced disc as long as a normal range of mandibular movement is reestablished.

Disc adhesion

Clinical manifestations
• Painful limitation of mouth opening
• No prior history of clicking or popping
• Magnetic resonance imaging (MRI) shows disc in normal position

Treatment
• Arthrocentesis

Pathologic Conditions Involving the Muscles of Mastication
Myofascial Pain Dysfunction (MPD), Masticatory Myalgia

Clinical manifestations
• Intermittent, dull, aching facial pain
• Usually unilateral
• Limited mouth opening
• Tenderness in one or more masticatory muscles
• Pain and tenderness in neck and shoulders
• History of frequent headache and earache
• Frequent history of tooth clenching and grinding
• Pain increases with function and stress

Treatment
• Home therapy (soft, nonchewy diet; intermittent moist heat and massage of painful muscles; limited jaw function)
• Medication for pain (NSAIDs)
• Medication for muscle relaxation and stress reduction (eg, diazepam or cyclobenzaprine)
• Bite appliance to be used at night if history of parafunction
• Medication for sleep if poor sleep habits (diazepam or amitriptyline)
• Stress management for treatment of recalcitrant patients
Diagnosis and Treatment of Nerve Injuries

Injuries to branches of the trigeminal nerve as a result of accidental trauma and iatrogenic surgical damage are common in the practice of oral and maxillofacial surgery. Although most resulting neuropathies resolve spontaneously within days to weeks, some persist as permanent impairments. A methodical approach to diagnosis of traumatic neuropathies and their timely treatment are essential to the achievement of maximum recovery.

Epidemiology

Nerve injuries accompany:

- A high percentage of facial fractures.
- Most mandibular orthognathic surgeries.
- Mandibular third molar surgery. Approximately 5% of patients will sustain some degree of intraoperative neuropathy, some of which may become permanent. This represents the highest number of trigeminal neuropathies.
- Endosseous implant surgery.
- Local anesthetic injections and endodontic treatments.

The primary nerves affected are the:

- Intrabony aspect of the inferior alveolar nerve
- Vestibular aspect of the infraorbital nerve
- Lingual and chorda tympani nerves

Mechanisms of Injury
Trigeminal injuries most commonly result from:

- Mechanical trauma (stretch/ischemia, compression/crush, puncture, partial or full laceration, tissue avulsion)
- Thermal injuries (commonly from overheated surgical burs)
- Chemical neurotoxicity (local anesthetics and endodontic chemicals)

**Classification**

Nerve injuries are classified by Sunderlund and Seddon according to the extent of neuroanatomic disruption (*Table 22-1*):

- First degree (neuropraxia): Extraneural conduction block with expected good recovery
- Second to fourth degree (axon tomesis): Intraneural neuroma-in-continuity scarring with unpredictable recovery patterns, possible dysfunction, and neuropathic pain; possible need for surgical intervention

*Table 22-1 Nerve injury classification*
Fifth degree (neurotomesis): Amputation neuroma formation with need for resection, followed by neurorrhaphy, microsuture repair, and possible interpositional nerve grafting. This spectrum of neuropathology is strongly correlated with permanent sensory loss and neuropathic pain.

### Diagnosis of Nerve Injuries

**Primary Diagnostic Steps**

Patient evaluation following trigeminal nerve injury involves a series of key diagnostic measures:

- Neuropathic symptom assessment
Assessment of general and orofacial functional impairment
Maxillofacial clinical examination and imaging findings
Quantitative sensory test responses

Assessing neuropathic symptoms

Patients should be asked to describe the anatomical location(s) of their altered sensations and to estimate their current level of discomfort or pain on a scale where:

- 0 = no discomfort
- 25% = mild discomfort
- 50% = moderate discomfort/pain
- 75% = severe pain
- 100% = intolerable pain

Patients should then be prompted to characterize their altered sensations by circling terms from a list of neuropathic terms such as: constant, intermittent, rhythmic, steady, brief, triggered, spontaneous, numb, itching, dry, tickling, twitching, wet, rubbery, stretched, swollen, woody, crawling, moving, quivering, vibrating, cool, warm, cold, hot, burning, pricking, stinging, electric, tender, sore, painful, aching, excruciating, cramping, shocking, bitter, sweet, sour, salty, tasteless, other.

These baseline patient symptom responses are used for assessment of recovery at future clinic visits.

Assessing general and orofacial functional impairment

Patients are asked to estimate their current (baseline) levels of functional impairment following injury, where:

- 0 = no impairment
- 25% = mild impairment
- 50% = moderate impairment
- 75% = severe impairment
- 100% = complete impairment

Patients are then prompted to characterize their specific impairments following injury by selecting from a list of terms such as: eating, talking, swallowing, tasting, toothbrushing, dental care, face washing, smelling,
Maxillofacial imaging

Neural imaging has not yet reached the levels of accuracy that precisely define anatomical details of nerve injuries. Nevertheless, postinjury imaging using plain films, tomography, digital, and computed tomography (CT) techniques can be used to assess:

- Paraneural foreign bodies (broken instruments, implants)
- Paraneural bone or dental root impingement
- Irregularities of nerve canal or foramina

Maxillofacial clinical examination

Following nerve injury, a maxillofacial examination is carried out in order to:

- Rule out nonneural sources of noxious pathoses (temporomandibular joint pain, active odontogenic or periodontal disease, sinusitis, sialadenitis)

• Detect sources of secondary nerve injury (mobile bone fractures, osteotomy segments, infection, invasive or compressive pathology)
• Reveal signs of traumatic neuroma formation (pain and tingling responses to digital palpation of nerve trunk distribution (Tinel sign).

Quantitative sensory testing (QST)

The objectives of clinical QST are to determine:
• Loss of sensory detection (hypoesthesia) in the injured nerve distribution
• Presence of neuropathic sensitization (hyperesthesia)
• Overall level of neurosensory recovery toward normalcy

Measuring sensory loss (hypoesthesia) (Fig 22-1)

Clinical neurosensory testing involves the application of graded stimuli to an uninjured (control) nerve distribution, comparing the patient’s normal detection capacities to the detection thresholds found within the injured nerve distribution. This is done using:
• Fine touch stimuli such as thin von Frey filaments, cotton, or brush strokes; these stimuli test for integrity of large myelinated nerve fibers (level A)

- Crude touch stimuli such as thick von Frey filaments or light pin touch; these stimuli test for integrity of small myelinated fibers (level B)
- Noxious heat stimuli (45°C to 49°C), deeper pin stick, or hemostat pinch; these stimuli test the integrity of A delta and unmyelinated C fibers (level C)
The level of sensory loss (deafferentation) determined from these tests is then plotted on the hypoesthesia scale (see Fig 22-1) and is used to map the anatomical zones of sensory loss on the facial diagrams (Fig 22-2).

**Sensory recovery scaling**

The level of functioning sensory fibers measured with hypoesthesia testing is also used to rate nerve regeneration on a four-level recovery scale where 0 = anesthesia and 3 = full normal recovery (see Fig 22-1).

**Detecting and measuring neuropathic sensitization (see Fig 22-1)**

The graded QST stimuli are also used to reveal signs of neural sensitization and possible neuroma formation in the injured nerve distribution, including:

- Painful response to fine touch (level A) stimuli, indicating the presence of allodynia, a sign of large fiber and possible central sensitization
- Painful tingling response to crude stimuli (level B), indicating the presence of hyperpathia and possible neuroma-in-continuity
- Excessive painful response to noxious stimuli (level C) in hypoesthetic zones, indicating the presence of hyperalgesia and possible amputation neuroma

These neuropathic findings are rated for severity and plotted on the hyperesthesia scale (see Fig 22-1).

**Recording of Baseline Diagnostic Impressions and Injury Mapping**

At the completion of the baseline assessments, the following key diagnostic information is documented in the patient’s record (see Fig 22-2):

- Opinion of the mechanism and severity (type) of nerve injury
- Mapping of symptom distribution drawn on anatomical diagram
- Mapping and quantified results of QST
- Presence and severity of neuropathic sensitization signs (triggering)

**Patient Communication and Follow-up**

All communication with the patient and family is accurately documented in the record, including:

- The diagnosis and likely cause of injury
- Prognosis and expectations for recovery
• Plans for treatment
• Possible need for secondary opinions or referrals
• Importance of follow-up visits and recovery monitoring

**Treatment of Nerve Injuries**

The purposes of nerve injury treatment are to restore orofacial neurosensory function and reduce neuropathic pain and discomfort using behavioral, pharmacologic, physical, and surgical interventions. The key variables that determine the timing and nature of treatment are:

- Whether the nerve injury was observed or unobserved
- Injury severity type (Sunderlund first to fifth degree) and related neuroma formation
- Whether the injured nerve is located in soft tissue (lingual, mental, infraorbital) or is intraosseous (inferior alveolar-mandibular, infraorbital-orbital)
- Access and feasibility of the nerve for surgical repair
- Whether the nerve injury is acute or chronic

**Fig 22-3** Treatment algorithm for trigeminal nerve injuries. (Modified from Gregg JM. Treatment of trigeminal nerve injuries. In: Fonseca RJ, Turvey TA, Marciani RD (eds). Oral and Maxillofacial Surgery, vol 1, ed 2. St Louis:
Pharmacologic Treatment of Acute Injuries

Medical treatment in the first days after nerve injury or immediate nerve repair is directed toward counteracting the excitatory effects on the nervous system generated by the traumatized nerve; a combination of four agents is recommended:

- Adrenocorticosteroid: 5-day tapering “dose-pack” course of dexamethasone, 4 mg orally three times a day
- Peripheral neural blockade: Paraneural lidocaine blocks or 5% lidocaine patch every 12 hours on the injured nerve distribution
- Short-acting anticonvulsant-anxiolytic: Oral clonazepam, 50 to 150 mg three times daily titrated to reduced neuropathic shocking
- Analgesic: Acetaminophen orally or transdermal opioids as needed for pain

Surgical Treatment of Acute Injuries

Immediate surgical management of nerve injuries (Fig 22-3) is directed toward:

- Removal of foreign bodies (broken instrument, implants), decompression of impinging bone or tooth fragments adjacent to the nerve injury site
- Elimination of secondary sources of nerve injury (mobile fractures or osteotomy segments, odontogenic infection, pathosis, toxic chemicals)
- Providing nerve protection with an isotonic barrier such as an absorbable gelatin sponge, collagen, or porcine nerve wrap for observed in-continuity (first to fourth degree), or aligned, nonavulsed fifth degree nerve injuries

Table 22-2 Sensory reeducation

<table>
<thead>
<tr>
<th>Stimulus</th>
<th>Areas to be stimulated</th>
<th>Method</th>
</tr>
</thead>
</table>

Saunders, 2009:265.)
<table>
<thead>
<tr>
<th>Cotton swab</th>
<th>Lip, vermilion border Chin, face</th>
<th>Back and forth, up and down, circular, back and forth</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Tongue, lateral border</td>
<td>Back and forth</td>
</tr>
<tr>
<td></td>
<td>Gingiva</td>
<td>Up and down</td>
</tr>
<tr>
<td>Toothbrush</td>
<td>Tongue, dorsal surface</td>
<td>Back and forth, circular</td>
</tr>
<tr>
<td>Hairpin, paper clip</td>
<td>Lip, vermilion border</td>
<td>Two-point touches</td>
</tr>
<tr>
<td></td>
<td>Chin, face</td>
<td>Two-point touches</td>
</tr>
<tr>
<td></td>
<td>Tongue, lateral border</td>
<td>Two-point touches</td>
</tr>
</tbody>
</table>

- Carrying out baseline primary epineural microsuture or preparing patient for secondary consultation and microrepair in cases of observed fifth degree transection (lingual, mental, infraorbital) or unsupported, unaligned intrabony (inferior alveolar) nerve injuries

**Pharmacologic Treatment of Chronic Neuropathic Symptoms**

Medical interventions for chronic neuropathic symptoms are directed toward management of central nervous system sensitization and dysfunction.

A combination of four agents is recommended:

- Peripheral neural blockade: 5% lidocaine patch every 12 hours applied to area of neuropathic pain
- Long-acting anticonvulsant: Gabapentin titrated 600 to 2,400 every day or pregabalin 100 to 500 every day titrated to hyperesthesia reduction
- Tricyclic antidepressant: Amitriptyline or equivalent 50 to 150 at bedtime
- Analgesic: Nonsteroidal anti-inflammatory drug/cyclooxygenase-2 inhibitor and/or sustained-release scheduled opioid with breakthrough
Sensory Reeducation

The spontaneous recovery of central neurologic responses to nerve injury and recovery following nerve repair surgery are enhanced by engaging patients in a self-therapy program of sensory reeducation. Important factors in an individual’s recovery from nerve injury include reestablishment of anatomical continuity (ie, surgery), spontaneous regeneration or physiologic maturation (healing) of the nerve, and establishment of new connections in the central nervous system by the brain’s accommodation to disabilities.

Successful recovery of sensory function, in particular, is not a passive experience. Once a nerve has started to recover, its progress can be hastened and the final result enhanced by exercises designed to help the brain learn to receive sensory impulses carried along new nerve connections that may differ from those present before injury and surgical repair. Experience has shown that recovery of sensation in the affected area of lip, chin, gingiva, cheek, tongue, or face can be made more complete and, in some cases, normal by regularly performing these exercises. Similar exercises have proved to be helpful in patients with nerve injuries in the hand.

The exercise protocol, as demonstrated by the surgeon or nurse, is shown in Table 22-2. The patient faces the mirror and applies the stimulus using the following steps:

- Stimulate the normal side opposite injured side with eyes open
- Stimulate the abnormal, injured side with eyes open
- Stimulate the normal side with eyes closed
- Stimulate the abnormal side with eyes closed, imagining the stimulation

While applying the stimulus on the abnormal (numb) side, it is important that the patient recall and visualize the sensation felt on the normal side. This helps extinguish (blot out) the abnormal subjective feelings. If the abnormal areas are hypersensitive, the surgeon may prescribe a neurotropic medication to reduce the discomfort and allow effective stimulation.

Exercises should begin as soon as possible after injury or surgical repair, be done three times per day, and be continued for at least 12 months for maximum benefit. There will be little noticeable change from day to day.
Progress is plotted, however, by direct nerve testing by the surgeon or assistant at 2- to 3-month intervals.

Final adjustments to any residual deficit, discomfort, or abnormal sensation (eg, numb, swollen, tight, tingling) are usually overcome gradually through the phenomenon of accommodation, in which the central nervous system “tunes out” or learns to ignore the sensory distractions. This process may take 1 year to several years to reach its maximum effect. Continuing daily sensory reeducation exercises during this time can assist with this accommodation.

Surgical Options for Definitive Treatment of Nerve Injuries

A spectrum of surgical options are available for treating nerve-injured patients (see Fig 22-3):

- Surgical decompression: The first phase of all repair procedures whereby the nerve injury site is exposed and the paraneural tissues are atraumatically released to enable inspection of nerve quality or need for additional repair. For first- and second-degree and some third-degree injuries, decompression with barrier nerve protection may be the definitive treatment.
- Neuroma biopsy and resection: The resection is carried into anatomically normal proximal and distal nerve tissue in preparation for secondary microrepair.
- Neurorrhaphy: The microsurgical suture repair of a transected nerve or after neuroma resection. It is the preferred repair procedure when it can be accomplished with minimal tension on the sutures.
- Graft microrepair (using autografting [sural, great auricular, antebrachial cutaneous nerve], allogeneic, or alloplastic materials): Indicated for repair of continuity defects that cannot be managed by direct neurorrhaphy.
- Neuroablative lesioning: Redirection and implantation of nerve stump in cases of neurectomy, chemical (alcohols, glycerols), or thermal lesions (radiofrequency or cryosurgical). Reserved for special cases such as those refractory to or unsuitable for medical management or surgical repair.
- Central neurosurgical procedures: Includes procedures such as stereotactic (gamma knife) radiosurgery, microvascular posterior fossa decompression, and deep brain stimulation. Reserved for patients with
extreme, refractory posttraumatic neuropathic pain.

Indications for Surgery

Exploration and surgical repair of injured nerves are indicated when there is:

- Observed or suspected mechanical nerve injury
- Presence of paraneural foreign bodies; compression by bone or dental structure
- Lack of improvement in anesthesia or dysfunctional hypoesthesia 2 or more months after injury based on serial examinations and QST
- Medically intractable and intolerable neuropathic pain or dysfunction

Contraindications for Surgery

Surgical repair of injured nerves may be contraindicated in the presence of:

- QST signs of improving neurosensory function
- Patient acceptance of existing levels of impaired function and discomfort
- Clinical signs of neuropathic central sensitization (spreading hyperalgesia)
- Deafferentation pain unrelieved by local anesthetic nerve block
- Signs of autonomic derangement (burning sensations, erythema, swelling)
- Extremes of age or concomitant systemic or neurologic disease
- Excessive time since the original nerve injury
- Unrealistic patient expectations (restoration of immediate, normal, pain-free sensory function)

Timing and Goals of Secondary (Delayed) Surgery

Selection of optimal timing for surgical intervention following nerve injury is done on a case-specific basis using serial examinations and quantitative sensory testing after injury; certain general guidelines are helpful, although not comprehensive:

- When surgical intervention is indicated, early nerve repair is simpler and has a better prognosis than long-delayed repair.
- Soft tissue laceration nerve injuries (lingual, mental, infraorbital) have a high priority for early repair, ideally within 2 to 4 months of injury, because of their incapacity for spontaneous regeneration.
• Intraosseous lacerated nerves, such as the inferior alveolar nerve, can still be effectively repaired at longer postinjury times (more than 6 months) because of their potential for spontaneous regeneration within the mandibular canal.
• Surgical treatment of nerve injuries is considered successful when it has resulted in restoration of crude touch and protective senses and when there has been a 50% or greater reduction of preexisting pain.

**Specific Clinical Situations**

Although the diagnostic methods and principles of treatment of all nerve injuries are similar, there are variances in managing the injuries associated with facial fractures, orthognathic surgery, endosseous implants, dentoalveolar surgery, local anesthetic injections, and endodontic therapy.

**Nerve Injuries Associated with Facial Fractures**

Inferior alveolar nerve injuries accompany 80% of mandibular body and angle fractures, and infraorbital nerve injuries occur with 75% of zygomaticomaxillary fractures; one-third of nerve injuries associated with facial trauma are permanent. Key guidelines for fracture-related nerve injury treatment are:

• Early fracture immobilization to prevent secondary repetition injuries
• Closed reduction of mandibular fractures, when indicated, because of lower rates of permanent inferior alveolar nerve neuropathy than after open reduction
• Open reduction of orbital and zygomaticomaxillary fractures because of lower rates of infraorbital neuropathy than after closed treatment
• Immediate intraoperative microsurgical neurorrhaphy when anatomical access permits
• Sensory reeducation protocol begun immediately after treatment

**Nerve Injuries Associated with Orthognathic Surgery**

Inferior alveolar neuropathy occurs transiently in over 90% of mandibular bilateral sagittal split osteotomies, with over 11% persisting for more than 1 year. Lingual neuropathies are more rare, resulting most often from instrumentation during segment fixation. Neuropathies associated with Le
Fort procedures are also frequent but resolve quickly. There should be thorough presurgical patient education/consent regarding nerve injury risks for all orthognathic surgery patients. Key guidelines for prevention and treatment of such injuries are:

- Using nerve-protecting retractors during drilling, screw placement, and plate fixation of osteotomy segments
- Achieving rigid fixation to avoid secondary nerve trauma
- Doing immediate intraoperative neurorrhaphy for fifth degree full separation inferior alveolar or infraorbital nerve injuries; considering nerve wrap protection in all instances
- Beginning sensory reeducation protocol immediately after treatment

Nerve Injuries Associated with Dental Implants

The highest incidence of inferior alveolar nerve injury, approximately 5% to 15% permanent, follows endosseous implant procedures in cases of severe mandibular atrophy where implants that were too long were placed or where nerve lateralization procedures were used. It is important that all such patients are educated about the possible risks and give informed consent. Key guidelines for avoiding or limiting nerve injury are:

- Accurate presurgical imaging for delineation of neurovascular anatomy
- Providing nerve protection during surgical drilling through presurgical depth analysis
- Avoiding overheating with rotating instruments (< 55°C)
- Doing postimplant placement imaging for visualization of nerve proximity
- Performing early (72-hour) nerve decompression in cases of verified implant or displaced bone impingement; either remove implant completely or move implant to > 2 mm from nerve canal
- Immediately using corticosteroid and anticonvulsant therapy when there has been nerve impingement (see Pharmacologic Treatment of Acute Injuries, page 326)
- Performing secondary surgical decompression, neuroma resection, and microsurgical nerve repair in the event of chronic unresolving sensory loss and/or medically intractable or intolerable pain

Nerve Injuries Associated with Dentoalveolar Surgery
Injury to the inferior alveolar nerve during removal of impacted mandibular third molars is the most frequent cause of trigeminal neuropathy, occurring in approximately 5% of cases, with 15% of these resulting in permanent injury; lingual nerve injuries occur much less often, but are more functionally disabling and have much lower spontaneous recovery. Key guidelines for avoiding, limiting, and managing nerve injury are:

- Obtaining accurate preoperative imaging for identification of nerve proximity and planning nerve protection
- Considering coronectomy/root retention for extreme cases, especially in the elderly
- Locating incisions and access flaps in the lateral vestibule
- Avoiding drill overheating and blind drilling
- Avoiding lingual bone removal unless specifically exposing and retracting the lingual nerve
- Assessing the type of injury; adapting or suturing the nerve endings, using a protective barrier, if possible
- Following up with imaging and serial clinical QST
- Initiating early corticosteroid and anticonvulsant therapy and use of analgesics if necessary (see Pharmacologic Treatment of Acute Injuries, page 326)
- Doing serial examinations to plot recovery or persistence of neuropathy
- Carrying out or arranging for secondary repair in the event of failing recovery patterns

Nerve Injuries Associated with Local Anesthetic

Local anesthetic nerve injuries, either from needle contact or chemical neurotoxicity, occur after routine mandibular blocks in approximately 1 in every 100,000 cases; the lingual nerve is most commonly involved. Although most cases resolve within 10 days, those that persist beyond 3 weeks are likely permanent. Unfortunately, there are currently no known reliably effective surgical repair procedures. Key guidelines for avoiding or limiting anesthetic nerve injury and its management are:

- Minimizing multiple injections in the same nerve distribution
- Avoiding a direct injection into the mental foramen
- Avoiding bone contact with needle tips during injection, which causes burs that can then injure the nerve
• Initiating acute nerve injury medical protocol with a corticosteroid, an anticonvulsant, and analgesic agents if necessary (see Pharmacologic Treatment of Acute Injuries, page 325)
• Initiating sensory reeducation exercises
• Carrying out serial examinations; prepare patient for possible long-term neuropathy and its medical management (see Pharmacologic Treatment of Chronic Neuropathic Symptoms, page 326)

Nerve Injuries Associated with Endodontic Therapy

The incidence of nerve injuries following endodontic surgery is unknown. They occur almost exclusively in the inferior alveolar and mental nerve distributions and are caused by the neurotoxicity of phenols, aldehydes, and the high-pH calcium hydroxide medicaments or by damage from materials such as gutta percha placed under pressure and high heat. The full range of injury may be seen, from mild resolving paresthesia to extremes of permanent sensory deficit and intractable burning dysesthesia. Treatment is directed toward:

• Immediate opening of treated canals and lavage with buffered solution when due to chemical injury; consider extraction of tooth
• Early decortication, nerve decompression, and removal of any foreign materials in the mandibular canal
• Delayed decortication, neuroma resection, and microsurgical repair in the event of unresolving sensory dysfunction and intractable pain
Differential Diagnosis and Management of Oral Mucosal Lesions

Although the differential diagnosis of some oral mucosal lesions is straightforward, others pose a particular challenge to even the most astute clinician because they can represent any one of several pathogenetically diverse conditions that share similar or overlapping clinical features. To distinguish these conditions from one another requires good clinical observations, accurate description of findings, an informed and thorough history, and a list of reasonable diagnostic possibilities. The differential diagnosis holds the key to determining whether diagnostic tests are necessary and precisely which tests to obtain in order to expeditiously establish a definitive diagnosis.

The Diagnostic Maxims

Among the basic principles of diagnosis that any good clinician must recognize and respect are the simple but essential maxims that guide the diagnostic process. They are as follows:

- **Common things occur commonly.** Most oral lesions encountered represent familiar, frequently encountered benign responses to local inflammatory or factitial stimuli. Their causative agents may be readily identified in most cases by taking a good history and conducting a proper examination.

- **Lesions and diseases are characterized by a constellation of distinguishing (ie, classic) features.** Essential elements leading up to the diagnosis include recognition of the lesion type (eg, ulcer, plaque, vesicle, nodule) and its location/distribution, surface characteristics, texture, size, and borders, as well as the history, symptoms/signs, and if relevant or available, results of diagnostic tests.
• Things that look alike are not necessarily the same. Diverse pathologic conditions sometimes bear superficial resemblance to other distinct and separate disease entities that require different treatment or management. It is therefore incumbent on the clinician to analyze and synthesize diagnostic data with a disciplined, systematic, and informed approach in order to avoid diagnostic errors.

**Differential Diagnosis of White Oral Lesions**

An increase in surface epithelial thickness and/or keratinization of the oral mucosa will result in a white, nonwipable (ie, keratotic) plaque. Discussed below are conditions that present in the oral cavity as white lesions.

White, Nonwipable Plaques and Papules (Keratotic White Oral Lesions)

Keratotic white oral lesions in trauma- or friction-prone oral locations generally represent benign reactive hyperkeratoses. They are usually painless, rough-surfaced, and located directly opposite the offending source of friction or irritation and generally reflect the shape of the irritant. With removal of the presumptive source of irritation, benign reactive hyperkeratoses will often disappear within several weeks but can reappear if the irritant is reintroduced.

**Leukoplakia**

White keratotic plaques that are in relatively friction- or trauma-protected locations (floor of the mouth, lingual mandibular vestibules, ventrolateral tongue, soft palate, uvula, tonsillar pillars) or that cannot be accounted for or diagnosed on clinical grounds (ie, no obvious inciting agent, no history of trauma, and no features characteristic of other specific keratotic diseases) are termed *leukoplakias*. This term is neither a definitive diagnosis nor a synonym for premalignant or malignant change. It is merely a provisional clinical designation that is applied until a definitive diagnosis is established through a tissue biopsy. The majority of clinically leukoplakic lesions are benign hyperkeratoses. Oral leukoplakic lesions that are most likely to demonstrate a precancerous epithelial maturational disturbance (intraepithelial dysplasia) are summarized in Table 23-1.

**Lichen planus and other lichenoid oral lesions**

The classic clinical lesions of oral lichen planus are white keratotic striae
distributed bilaterally. The history and/or clinical examination are often positive for cutaneous pruritic lesions, other mucosal involvement, or both. The typical features of this benign, cell-mediated, mucocutaneous immunologic condition are compared with those of benign reactive hyperkeratoses and precancerous leukoplakias in Table 23-1.

Other oral lesions with clinically lichenoid features include: nonspecific idiopathic stomatitides, benign lichenoid responses to local stimuli, lichenoid drug reactions, mucosal lesions of chronic discoid lupus erythematosus (CDLE) or systemic lupus erythematosus (SLE), and epithelial dysplasia, a precancerous oral lesion that is unrelated to lichen planus both etiologically and pathogenetically (Table 23-2).

Other diagnostically specific benign oral leukoplakias
- Oral hairy leukoplakia (OHL)
- Candidal leukoplakia (chronic hyperplastic candidiasis)

The salient features and differences between OHL and candidal leukoplakia are shown in Table 23-3.

**Table 23-1** Comparison of benign hyperkeratosis, precancerous oral leukoplakia, and lichen planus
<table>
<thead>
<tr>
<th>Benign reactive hyperkeratosis</th>
<th>Leukoplakia</th>
<th>Lichen planus</th>
</tr>
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<tbody>
<tr>
<td><strong>History</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Any patient, no age predilection</td>
<td>• Middle-aged or older person</td>
<td>• Middle-aged or older person; more common in women</td>
</tr>
<tr>
<td>• Painless, nonwipable white lesion</td>
<td>• With or without history of past or active tobacco use (including smokeless tobacco) and/or alcohol use</td>
<td>• Chronic, persistent mucocutaneous eruption</td>
</tr>
<tr>
<td>• History of trauma or presence of obvious source of chronic, low-grade friction (eg, sharp tooth, faulty dental restoration, prosthesis or appliance, foreign object)</td>
<td>• No trauma history</td>
<td>• With or without history of onset related to new medication</td>
</tr>
<tr>
<td>• History of bruxism, clenching, chronic gum chewing</td>
<td>• No evidence of trauma</td>
<td>• With or without prior or concurrent history of pruritic, scaly skin lesions on extremities and/or trunk</td>
</tr>
<tr>
<td>• With or without history of chronic xerostomia</td>
<td>• Lesion persists after elimination of presumed irritant</td>
<td>• Genital mucosal involvement possible</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Location</th>
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</thead>
<tbody>
<tr>
<td>• Trauma-prone site (buccal and labial mucosa, gingiva, hard palate, posterior and lateral tongue surfaces)</td>
<td>• Trauma-protected anatomical site/cancer-prone oral site</td>
<td>• Bilateral distribution on mucosa gingiva; other intraoral buccal sites, lip vermilion possible</td>
</tr>
<tr>
<td>• Site of recalled chronic friction (per history)</td>
<td>• Site of placement of tobacco product</td>
<td>• With or without genital, anal mucosa, cutaneous involvement</td>
</tr>
</tbody>
</table>

Continues on next page.
<table>
<thead>
<tr>
<th>Benign reactive hyperkeratosis</th>
<th>Leukoplakia</th>
<th>Lichen planus</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical features</strong></td>
<td><strong>Least suspicious:</strong></td>
<td></td>
</tr>
<tr>
<td>• Nonwipable white, rough plaque; linear, nonerythematous; with or without superficial erosion</td>
<td>• Homogeneous white nonwipable plaque; usually asymptomatic</td>
<td></td>
</tr>
<tr>
<td>• Usually painless</td>
<td>• Persistent</td>
<td></td>
</tr>
<tr>
<td>• Directly opposed to and in contact with source of friction</td>
<td><strong>Most suspicious:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Heterogeneous, nonwipable white, rough plaque with erythematous component or more predominantly erythroplakic appearance.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Pebbly, nodular, irregular surface texture</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• With or without ulceration, erosion</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Invasive lesions are indurated, fixed to underlying tissues</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Classic lesions:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• No biopsy.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Clinical observation semiannually.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Incisional biopsy for definitive diagnostic confirmation if necessary.</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Symptomatic lesions:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• See Table 23-10.</td>
<td></td>
</tr>
<tr>
<td><strong>Management</strong></td>
<td><strong>Incisional biopsy</strong> (or multiple samplings if lesion is extensive) of representative lesional site(s).</td>
<td></td>
</tr>
<tr>
<td>• Attempt to eliminate suspected irritant.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Reevaluate in 1 month.</td>
<td>• Diagnosis of dysplasia (or carcinoma) mandates excision of all remaining clinically suspicious areas and ongoing clinical observation at regular intervals.</td>
<td></td>
</tr>
<tr>
<td>• Biopsy only if other factors create a suspicious profile.</td>
<td>• Recurrences and/or new primary lesions possible in the future. Rebiopsy indicated accordingly.</td>
<td></td>
</tr>
<tr>
<td>• If irritant cannot be identified by history and exam, incisional biopsy to rule out dysplasia.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Benign hyperkeratoses need not be rebiopsied.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Table 23-2 Clinical features of lichenoid stomatitis

<table>
<thead>
<tr>
<th>Oral lichenoid lesions</th>
<th>Defining clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lichen planus</td>
<td>• Bilateral distribution</td>
</tr>
<tr>
<td></td>
<td>• Skin and/or other mucosa involved (see Table 23-1)</td>
</tr>
<tr>
<td>Nonspecific lichenoid stomatitis</td>
<td>• Oral mucosal involvement</td>
</tr>
<tr>
<td></td>
<td>• May be focal or multifocal</td>
</tr>
<tr>
<td></td>
<td>• Lacy white with or without erythema, erosions</td>
</tr>
<tr>
<td>Lichenoid direct contact hypersensitivity reaction</td>
<td>• On mucosal surface in direct prolonged or repeated contact with sensitizing agent (eg, amalgam restoration, cinnamon product)</td>
</tr>
<tr>
<td></td>
<td>• With or without discomfort</td>
</tr>
<tr>
<td></td>
<td>• Resolves (in several weeks) with complete elimination of precipitant sensitizing agent</td>
</tr>
<tr>
<td>Lichenoid drug reactions</td>
<td>• Onset temporally related to introduction of medication</td>
</tr>
<tr>
<td></td>
<td>• May be bullous, erosive</td>
</tr>
<tr>
<td></td>
<td>• May not resolve with withdrawal of medication</td>
</tr>
</tbody>
</table>
Chronic discoid lupus erythematosus

- Predominantly cutaneous involvement: hyperpigmented, hypopigmented plaques, scarring
- Oral lesions may be unilateral or multifocal, erythematous or erosive with white radiating striae peripherally

Systemic lupus erythematosus

- Systemic autoimmune condition
- Oral lesions resemble CDLE lesions or are more nondescript and erythematous, erosive
- Palate often involved
- Gingival erythema, bleeding

Epithelial dysplasia* with lichenoid inflammatory infiltrates (histologic diagnosis)

- Isolated erythroleukoplakic lesion, with or without striated white component.
- May be erosive or ulcerated
- With or without indurated component
- Often on oral cancer-prone site

*Premalignant.

**Table 23-3** Features of oral hairy leukoplakia and candidal leukoplakia

<table>
<thead>
<tr>
<th>Oral hairy leukoplakia</th>
<th>Candidal leukoplakia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Opportunistic</td>
</tr>
<tr>
<td><strong>Etiology</strong></td>
<td>• Infection with Epstein-Barr virus</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td></td>
<td>• Associated with HIV/AIDS (or, rarely, with other causes of immune suppression)</td>
</tr>
<tr>
<td></td>
<td>• Lateral borders of tongue</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Location</strong></th>
<th>• Lateral borders of tongue</th>
<th>• Any oral mucosal site</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Rarely, other oral mucosal sites</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Painless, white, nonwipable plaques</td>
<td>• Painless, white, rough or corrugated, nonwipable plaque</td>
</tr>
<tr>
<td></td>
<td>• Filiform appearance</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Clinical features</strong></th>
<th>• Other candidal phenotypes (pseudomembranous, erythematous, angular cheilitis) may be seen concurrently</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Biopsy: characteristic benign histologic features suggestive of OHL</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Diagnostic confirmation</strong></th>
<th>• Biopsy: candidal hyphae, inflammation within hyperkeratotic surface of epithelium</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• In situ hybridization reveals EBER+ cells with immunohistochemistry</td>
</tr>
<tr>
<td></td>
<td>• With or without epithelial atypia (reactive)</td>
</tr>
<tr>
<td></td>
<td>• If patient is known to be HIV Ab+, antiviral treatment optional</td>
</tr>
<tr>
<td></td>
<td>• Topical antifungal treatment (clotrimazole troches 5 times/day for 14 days)</td>
</tr>
</tbody>
</table>
Management

- If HIV status is unknown, serum Ab testing is indicated
- Identify and address predisposing factors

HIV, human immunodeficiency virus; EBER, Epstein-Barr virus-encoded RNA; Ab, antibody.

Benign Developmental Hyperkeratoses (Nonwipable White Plaques)

- Leukoedema: Diffuse, milky-white appearance; on buccal mucosa; disappears on stretching; usually in darker-skinned individuals. Considered a variant of normal.
- White sponge nevus: Spongy-appearing contiguous white nonwipable plaques on buccal mucosa, floor of mouth, tongue. Autosomal dominant, positive family histories, early onset, diffuse.

Wipable White Plaques (Nonkeratotic White Oral Lesions)

White lesions that can be wiped away with a gauze sponge or a tongue blade consist of accumulated surface debris. Common examples include:

_Table 23-4 Oral presentation of candidal infection*

<table>
<thead>
<tr>
<th>Type</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pseudomembranous (thrush) type</td>
<td>- White, wipable, curdlike accretions on erythematous mucosal background.</td>
</tr>
<tr>
<td></td>
<td>- Cytological smear demonstrates hyphae, spores.</td>
</tr>
<tr>
<td>Erythematous (atrophic) type</td>
<td>- Diffuse erythema, atrophic surface, may be symptomatic.</td>
</tr>
</tbody>
</table>
• Often seen in chronic xerostomia.
• Smears may be unrevealing.
• Fungal cultures (special medium) positive for *Candida*.

**Angular cheilitis (perleche)**

• Crusting, fissuring, erythematous lip commissures.
• Fungal cultures (special medium) positive for *Candida*.
• Bacterial cultures positive for streptococci, staphylococci.

**Hyperplastic (candidal leukoplakia) type spores**

• White, nonwipable, rough or corrugated-appearing plaques.
• May or may not be other clinical forms of candidal presentation evident.
• Diagnosis cannot be determined clinically
• Biopsy reveals abundant hyphae, within surface keratin.

*Any or all types may be seen in combination in an individual patient.*

- Pseudomembranous candidal colonies (*Table 23-4*)
- Coagulatative necrosis of thermally or chemically burned surface epithelium (eg, aspirin burn)
- Collapsed roof of a bulla
- Fibrinopurulent pseudomembrane covering an ulcer base
- Other nonspecific surface accretions
**Differential Diagnosis of Oral Mucosal Ulcers**

Oral mucosal ulcers are encountered frequently. The pathologic processes characterized by ulcerations include:

- Reactive lesions (resulting from direct factitial injury)
- Infectious diseases
- Systemic influences (usually immune-mediated or hematologic)
- Malignant neoplasms

Ulcerations of the oral cavity can range from acute to chronic and from symptomatic to asymptomatic. Conditions that typically present with symptomatic oral ulcerations include:

<table>
<thead>
<tr>
<th><strong>Box 23-1 Salient features of traumatic ulcers</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Location</strong></td>
</tr>
<tr>
<td>• Trauma-prone oral mucosal sites</td>
</tr>
<tr>
<td>• Prolonged duration of a benign ulcer may be attributable to:</td>
</tr>
<tr>
<td>— Repeated trauma</td>
</tr>
<tr>
<td><strong>History</strong></td>
</tr>
<tr>
<td>• History of injury as related by patient</td>
</tr>
<tr>
<td>— Depth of wound</td>
</tr>
<tr>
<td>• Evidence of local source of injury or trauma revealed in clinical exam</td>
</tr>
<tr>
<td>— Vascular compromise (eg, diabetes, vascular disease)</td>
</tr>
<tr>
<td><strong>Symptoms</strong></td>
</tr>
<tr>
<td>• Early lesions are usually painful; long-</td>
</tr>
<tr>
<td><strong>Management</strong></td>
</tr>
<tr>
<td>• Attempt to</td>
</tr>
</tbody>
</table>
Standing lesions may be less symptomatic than lesions that are acute or subacute. It is important to identify and treat the presumptive source of injury. Avoid secondary trauma.

Course
- Usually resolve or show evidence of progress toward resolution within 2 to 3 weeks unless source of injury persists or is repeated or the wound is extensive (ie, wide and deep)
- Reevaluate clinically in 2 to 3 weeks if lesion persists without evidence of resolution, biopsy is indicated

Palliative measures (preferably topical) as needed

Avoid secondary trauma

≥ Advanced squamous cell carcinoma and other malignancies
≥ Immune-mediated conditions
≥ Granulomatous infections (bacterial, eg, tuberculosis; "deep" fungal infections, eg, histoplasmosis)
≥ Primary or recurrent intraoral human herpesvirus (HHV) lesions
≥ Aphthous ulcers
≥ Coxsackievirus infections

Traumatic Ulcers
Ulcers caused by direct physical injury (ie, traumatic ulcers) are the most common of all oral ulcerations. A majority of traumatic ulcers present as individual isolated lesions (Box 23-1).

Persistent or nonhealing ulcers

If an ulcer is presumed to be traumatic or otherwise benign but fails to respond to conservative management within a prescribed period of time or cannot be diagnosed clinically on the basis of local conditions and history, an incisional biopsy is indicated to rule out malignancy, a noninfectious or infectious granulomatous condition, or any other more unusual diagnostic possibilities (Box 23-2).

**Box 23-2 Characteristics of oral ulcers that require biopsy**

- Ulcer cannot be attributed to a local insult or any other specific conditions on the basis of history and/or clinical examination.
- Persistent ulcer fails to demonstrate any evidence of progress toward resolution 2 to 3 weeks following elimination of suspected injurious agent(s).
- No history of pain at onset.
- Late onset of pain and/or paresthesia.
- Surrounding mucosa is erythroleukoplakic.
- Ulcer borders are raised, rolled, and/or indurated on palpation.
- Located in an oral cancer-prone site.
- Previous history of or evident signs and symptoms suggestive of systemic influence: granulomatous process (eg, deep fungal or bacterial infection, sarcoidosis, Crohn disease), blood dyscrasia, malignancy, etc.

Recurrent Aphthous Ulcers (Recurrent Aphthous Stomatitis, Canker Sores)
Aphthous stomatitis is one of the most common recurrent oral ulcerative conditions. Although the precise etiology remains unclear, evidence favors cellular autoimmunity and genetic predisposition rather than infection or trauma. Affected patients usually report a unique trigger (or triggers) that precedes episodic outbreaks. The frequency and severity of recurrences varies widely. In some patients there may be periods when episodes are sufficiently frequent that they tend to overlap. In such cases, therapeutic measures can be taken to break the cycle of ulceration.

Typical aphthous ulcers usually range from 0.5 cm or less in diameter (minor aphthous ulcers) to 1.0 cm in diameter or larger (major aphthous ulcers, periadenitis mucosa necrotica recurrens [PMNR], Sutton aphthae). So-called herpetiform aphthae are the least common. They consist of crops or clusters of very small ulcers that often coalesce, resulting in large surface lesions.

Minor aphthae usually run their natural course within about 10 days. Major aphthae, however, can run a much more protracted course and may heal with scarring. Irrespective of their size, aphthous ulcers are extremely symptomatic. In patients who experience overlapping episodes, speech, mastication, and the ability to eat comfortably may be compromised for weeks or months.

Oral Aphthouslike Ulcers

Oral aphthouslike ulcers may be associated with certain systemic conditions (Boxes 23-3 and 23-4).

**Box 23-3 Systemic conditions associated with aphthouslike ulcerations**

**Hematologic**
- Cyclic neutropenia
- Selected anemias
Behçet syndrome (see Box 23-4)

Inflammatory bowel diseases
- Ulcerative colitis
- Crohn disease

Celiac disease* (gluten-sensitive enteropathy, celiac sprue)

Relapsing polychondritis syndromes
- MAGIC syndrome (mouth and genital ulcers with inflamed cartilage)
- Others

HIV infection

PFAPA syndrome† (periodic [ie, relapsing] fevers, aphthous stomatitis, pharyngitis, lymphadenopathy)

Others
* Avoidance of gluten products in patients with celiac disease may prevent recurrence of aphthous stomatitis.
† PFAPA is rare and favors children. However, most children with a positive history of aphthous ulcers do not have PFAPA.

Box 23-4 Behçet syndrome: A recurrent multisystemic immunologically-mediated vasculitic syndrome*

Essential diagnostic feature:
Oral aphthous ulcers (three or more recurrent episodes within a single 12-month period)

Any two of the following Other less common
Additional features accompany oral ulcers:

- **Cutaneous component**
  - Pustular or nodular lesions
  - Positive cutaneous pathergy (pustule forms at site of injection of sterile saline or other inert substance 24 to 48 hours postinjection)
- **Inflammatory bowel disease–associated symptoms**
  - Positive cutaneous pathergy
- **Neurologic disturbances**
  - Psychiatric disorders
  - Meningoencephalitis
- **Erythema, ulcers, superficial thrombophlebitis, acneiform lesions**
- **Ocular component**
  - Retinal vasculitis
  - Uveitis
- **Genital component**
  - Recurring ulcers of vulva, penis, scrotum

Recurrent (Reactivated) Herpes Simplex Lesions

Mucocutaneous lesions of both recurrent (reactivated) and primary herpes simplex virus (HSV) infections (HHV-1 and HHV-2) and varicella-zoster virus infections (HHV-3) are vesicular at the outset. Recurrent (reactivated) HSV lesions of the perioral skin and vermilion border (ie, cold sores) are common. They present themselves less often within the oral cavity. However, when they do occur, they appear as localized, vesicular, croplike, painful eruptions that rupture rapidly, leaving painful, coalescing ulcerations surrounded by edema and erythema. They affect keratinized, bound-down mucosa (ie, hard palate and attached gingiva) and are often preceded by trauma, such as a local anesthetic injection, a dental or intraoral surgical procedure, or other stressful circumstances.

It is important to recognize and differentiate intraoral recurrent HSV lesions from aphthous ulcers (Table 23-5). Unlike the ulcers associated with herpesvirus-induced lesions, aphthous ulcers are not preceded by vesicles.

Vesiculoulcerative, Erosive, and Desquamative Conditions

Vesiculoulcerative, erosive, and desquamative conditions of the oral cavity occur as acute or chronic conditions.

Acute conditions

The conditions that are characterized by an acute, often rapid onset and multifocal, painful oral mucosal ulcers or erosions that are sometimes preceded by vesicles may be difficult to distinguish from one another. They include:

- Severe recurrent aphthous stomatitis (exclusively nonvesicular ulcers; see Table 23-5)
- Primary (acute) herpetic gingivostomatitis
- Coxsackievirus infections
- Erythema multiforme (an acute immunologic reaction pattern)

The key diagnostic features of frequently confused acute oral vesiculoulcerative conditions are listed in Table 23-6.
In primary (acute) herpetic gingivostomatitis, all intraoral mucosal surfaces and the skin and vermillion borders of the lips may be involved with vesiculoulcerative lesions. A characteristic manifestation of primary HSV infection is acute, generalized, extremely painful vesiculoulcerative gingivitis.

Constitutional signs of systemic infection (malaise, low-grade fever, lymphadenopathy) are notable and precede the onset of actual oral lesions. Severe discomfort frequently impairs the ability to drink or eat. In young children (classic primary herpes patients) this may be problematic, for there is considerable risk of dehydration. Unless there is underlying immunosuppression, the primary disease resolves within about 2 weeks in otherwise healthy individuals.

Primary varicella-zoster virus (first degree HHV-3) infection (chickenpox) Primary infection with varicella-zoster virus (HHV-3; chickenpox) presents with constitutional signs of systemic infection and a generalized vesicular cutaneous exanthema that begins on the face, head, and neck and eventually involves the trunk and extremities. The oral mucosal involvement is much more attenuated than in primary herpetic gingivostomatitis and is limited to a few isolated vesicles that ulcerate.

**Table 23-5 Comparison of recurrent aphthous ulcers and recurrent intraoral HSV lesions**

<table>
<thead>
<tr>
<th></th>
<th>Recurrent aphthous ulcers</th>
<th>Recurrent intraoral herpes simplex lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Etiology</strong></td>
<td>Focal immune dysregulation; genetic predisposition likely</td>
<td>HHV-1 (usually);</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HHV-2 (uncommon)</td>
</tr>
<tr>
<td><strong>Precursor</strong></td>
<td>Usually stress or trauma; other factors as diverse and unique as the</td>
<td>Local trauma, stress, systemic</td>
</tr>
<tr>
<td>Affected Individual</td>
<td>Immune Fluctuation</td>
<td></td>
</tr>
<tr>
<td>---------------------</td>
<td>--------------------</td>
<td></td>
</tr>
<tr>
<td>Prodrome</td>
<td>Seldom</td>
<td>Typical</td>
</tr>
<tr>
<td>Sites of involvement</td>
<td>Unkeratinized (movable) oral mucosa and dorsal surface of tongue</td>
<td>Keratinized (bound-down) mucosa exclusively</td>
</tr>
<tr>
<td>Distribution</td>
<td>Focal or multifocal throughout the oral cavity</td>
<td>Unifocal exclusively (if patient is immunocompetent)</td>
</tr>
<tr>
<td>Description of lesions</td>
<td>Round, erythema-bordered ulcers with white-yellow fibronecrotic centers; vary in size and number/episode</td>
<td>Clusters of vesicles that ulcerate; erythema; swelling; unifocal</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Pain (irrespective of size of lesion)</td>
<td>Pain</td>
</tr>
<tr>
<td>Diagnostic confirmation</td>
<td>History, clinical features; cytology is negative for virally modified epithelial cells; biopsy usually unnecessary</td>
<td>History, clinical presentation; smear findings are positive for viral cytopathic changes; biopsy usually not necessary</td>
</tr>
<tr>
<td>Clinical course</td>
<td>7 to 14 days (minor, herpetiform types); major aphthae may take longer than 3 weeks with potential for scarring</td>
<td>10 to 14 days (if patient is immunocompetent)</td>
</tr>
<tr>
<td>Management</td>
<td>Palliative agents (over-the counter or prescription); depending on</td>
<td>Antiviral medications</td>
</tr>
</tbody>
</table>
severity, topical corticosteroids or combined systemic corticosteroid or other immune-modulating agent for 3 weeks and topical agents indefinitely

(systemic), eg, acyclovir, valacyclovir

Table 23-6 Acute vesiculoulcerative conditions
<table>
<thead>
<tr>
<th></th>
<th>Primary (acute) herpetic gingivostomatitis</th>
<th>Oral coxsackievirus infection</th>
<th>Erythema multiforme</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Etiology</strong></td>
<td>Usually HHV-1 (HHV-2 less commonly)</td>
<td>Coxsackievirus A groups</td>
<td>Immunologic reaction pattern to precipitant (eg, drug [medical or recreational], alcohol, recent HSV or mycoplasma infection)</td>
</tr>
<tr>
<td><strong>Transmission</strong></td>
<td>Direct contact with active first- or second-degree HSV lesions</td>
<td>Via oral-fecal route</td>
<td>Noninfectious, nontransmissible</td>
</tr>
<tr>
<td><strong>Classic patient</strong></td>
<td>Child older than 6 months of up to late adolescence; less often in adults</td>
<td>Children and adolescents; adults less often</td>
<td>Most often young men with recent exposure to sensitizing agent</td>
</tr>
<tr>
<td><strong>Types of lesions</strong></td>
<td>• Multiple crops of coalescing vesicles that rupture and ulcerate</td>
<td>• Erythema</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Multiple individual vesicles, ulcers</td>
<td></td>
</tr>
<tr>
<td><strong>Sites of involvement</strong></td>
<td>• Perioral skin</td>
<td>• Posterior oral cavity, oropharynx, tonsillar areas</td>
<td>Vermilion and skin of lips and movable oral mucosa (minimal gingival involvement)</td>
</tr>
<tr>
<td></td>
<td>• Lip vermilion</td>
<td>• Less common on more anterior oral locations</td>
<td>Skin and/or conjunctivae</td>
</tr>
<tr>
<td></td>
<td>• Keratinized and nonkeratinized oral surfaces, especially gingiva</td>
<td>• May involve feet, hands, and other cutaneous sites</td>
<td>Genital</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pain, swelling, hemorrhage, and crusting of lips, oral mucous membranes</td>
</tr>
<tr>
<td><strong>Signs, symptoms</strong></td>
<td>• General malaise, fever, lymphadenopathy</td>
<td>• Headache, anorexia</td>
<td>Concomitant lesions on the skin and/or other mucous membranes possible</td>
</tr>
<tr>
<td></td>
<td>• Oral mucosal, gingival pain</td>
<td>• Fever, malaise, lymphadenopathy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Inability to eat or drink comfortably</td>
<td>• Nausea, vomiting</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• White coating on tongue</td>
<td>• Pharyngitis, dysphagia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Variable oral pain</td>
<td></td>
</tr>
</tbody>
</table>

Continues on next page.
<table>
<thead>
<tr>
<th>Primary (acute) herpetic gingivostomatitis</th>
<th>Oral coxsackievirus infection</th>
<th>Erythema multiforme</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical course</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Acute onset</td>
<td>• Acute</td>
<td>• Explosively acute onset within 24 to 48 hours of exposure to precipitating factor</td>
</tr>
<tr>
<td>• Immunocompetent individuals: typically 10- to 14-day course</td>
<td>• 7- to 10-day course</td>
<td>• May become chronic if untreated</td>
</tr>
<tr>
<td>• May be slightly longer in adults</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Diagnostic confirmation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• History and clinical features</td>
<td>• History and clinical features</td>
<td>• History of exposure and clinical presentation</td>
</tr>
<tr>
<td>• Cytologic smears demonstrate viral cytopathic changes in epithelial cells</td>
<td>• Cytologic smears negative for viral cytopathic changes</td>
<td>• Smears negative for viral cytopathic epithelial cell changes</td>
</tr>
<tr>
<td>• Positive cultures</td>
<td>• Serum antibody titers elevated</td>
<td>• Tissue biopsy if needed to rule out other conditions</td>
</tr>
<tr>
<td><strong>Management</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Supportive care (hydration, soft diet, rest)</td>
<td>• Supportive care (hydration, soft diet, rest)</td>
<td>• Extent of involvement dictates treatment</td>
</tr>
<tr>
<td>• Palliative rinses, analgesics</td>
<td>• Palliative rinses, analgesics</td>
<td>• Eliminate precipitant</td>
</tr>
<tr>
<td>• Systemic antiviral treatment effective if initiated within 24 to 72 hours of onset; after 72 hours, curtails viral shedding only</td>
<td>• Others should avoid direct contact with infected patient’s body fluids</td>
<td>• Begin tapered course (5 to 7 days) of systemic corticosteroid</td>
</tr>
<tr>
<td>• Protect others from direct contact with infected patient’s body fluids</td>
<td>•</td>
<td>• If due to HSV infection, systemic antiviral treatment for 5 to 7 days</td>
</tr>
<tr>
<td><strong>Prognosis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Viral latency</td>
<td>• Self-limiting</td>
<td>• Resolves within 21 days following initiation of treatment</td>
</tr>
<tr>
<td>• Potential for future reactivation with attenuated, local vesicular eruptions on external lip or keratinized oral mucosa (hard palate, gingiva)</td>
<td>• No viral latency</td>
<td>• Can recur if host is exposed to same precipitant</td>
</tr>
<tr>
<td>• Reinfecition is possible</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Recurrent (reactivated) varicella-zoster virus (second-degree HHV-3) infection (herpes zoster, shingles)

In cases of reactivated HHV-3 infection (herpes zoster, shingles) involving one or more branches of the trigeminal nerve, there are typically striking unilateral intraoral and extraoral crops of coalescing vesicles and ulcers distributed linearly. The lesions are generally preceded and accompanied by edema and severe pain or paresthesia (“zoster-related neuralgia”), which may persist for long periods following resolution of the clinical vesiculoulcerative changes. An uncommon but significant maxillary or mandibular sequela of intraoral herpes zoster is osteonecrosis of the alveolar bone due to viral osteomyelitis.

Management includes the use of valacyclovir plus systemic corticosteroid.

Chronic conditions

Chronic oral mucosal conditions characterized by erythema, vesiculobullous lesions, ulcerations, and desquamation pose a diagnostic challenge because they could represent any one of several possible distinctive mucocutaneous conditions with overlapping clinical features. These include:

- Mucous membrane (cicatrical) pemphigoid (mucous membrane lesions exclusively or predominantly)
- Bullous pemphigoid (cutaneous involvement predominantly)
- Pemphigus (oral mucosal lesions often precede cutaneous involvement)
- Erosive lichen planus (cutaneous and/or other mucosal involvement)
- Benign lichenoid stomatitides
- Linear immunoglobulin A (IgA) disease (usually cutaneous involvement; rarely oral or other mucosal lesions exclusively)
- Epidermolysis bullosum (inherited types; skin and mucosal involvement)
- Epidermolysis bullosum acquisita (predominantly skin involvement; immune mediated)
- Systemic or chronic cutaneous (discoid) lupus erythematosus (cutaneous and oral involvement)

The degree of gingival involvement, the tendency for surface epithelial
sloughing, mucosal fragility, the ability to raise a bulla on the mucosa by applying slight pressure (ie, desquamation indicative of a positive Nikolsky sign), and the presence or absence of a keratotic component are helpful clinical indicators that should narrow down the differential diagnosis (Table 23-7). To establish a definitive diagnosis, it is necessary to obtain a representative tissue biopsy specimen for conventional microscopic analysis and immunofluorescence (Box 23-5 and Tables 23-8 and 23-9). Management strategies for chronic vesiculoulcerative conditions are described in Table 23-10.

**Table 23-7** Differential diagnosis for dessquarnative ulcerative stomatitis
<table>
<thead>
<tr>
<th></th>
<th>Mucous membrane (cicatrical) pemphigoid</th>
<th>Pemphigus vulgaris</th>
<th>Erosive lichenoid stomatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>History</strong></td>
<td>• Middle-aged or older patient (F &gt; M)</td>
<td>• Middle-aged or older patient (M = F)</td>
<td>• Middle aged or older patient (F &gt; M)</td>
</tr>
<tr>
<td></td>
<td>• Chronic gingival erythema, fragility, desquamative tendency</td>
<td>• Insidious onset, progressive</td>
<td>• Chronic gingival erythema, desquamative tendency</td>
</tr>
<tr>
<td></td>
<td>• With or without obvious vesiculobullous lesions</td>
<td>• Extreme mucosal fragility, tendency to ulcerate</td>
<td>• White, nonwipable, lacy lesions on gingival papillae; often other oral mucosal sites involved</td>
</tr>
<tr>
<td></td>
<td>• Discomfort upon eating spicy, acidic, abrasive foods</td>
<td>• With or without obvious vesiculobullous lesions</td>
<td>• With or without difficulty maintaining oral hygiene</td>
</tr>
<tr>
<td></td>
<td>• Difficulty maintaining oral hygiene</td>
<td>• Pain upon eating spicy, acidic, abrasive foods</td>
<td>• May wax and wane in severity</td>
</tr>
<tr>
<td></td>
<td>• May wax and wane in severity</td>
<td>• With or without pharyngeal discomfort, dysphagia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• With or without conjunctival erythema</td>
<td>• Difficulty maintaining oral hygiene</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• With or without history of cutaneous bullae (skin involvement suggests bullous pemphigoid)</td>
<td>• With or without preceding history of bullous, ulcerative cutaneous lesions</td>
<td></td>
</tr>
<tr>
<td><strong>Location</strong></td>
<td>• Classic oral site of involvement is the gingiva</td>
<td>• All intraoral mucosal locations can be involved</td>
<td>• Distribution may be gingival exclusively, widespread, or limited to one or more oral sites</td>
</tr>
<tr>
<td></td>
<td>• Other oral mucosal surfaces may be involved</td>
<td>• Gingiva, hard palate, posterior tongue involvement not as severe as other sites</td>
<td>• Lip vermilion involvement possible</td>
</tr>
<tr>
<td></td>
<td>• Other mucous membranes (conjunctivae, vulva) may be affected</td>
<td>• With or without history of skin and/or other mucosal lesions</td>
<td>• With or without skin and/or other mucosal lesions</td>
</tr>
</tbody>
</table>

F, female; M, male.
<table>
<thead>
<tr>
<th>Mucous membrane (cicatricial) pemphigoid</th>
<th>Pemphigus vulgaris</th>
<th>Erosive lichenoid stomatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical features</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Primarily erythematous, desquamative gingival lesions; other sites can be involved</td>
<td>• Painful, extensive, ragged, maplike ulcerations</td>
<td>• Gingival atrophy, erythema</td>
</tr>
<tr>
<td>• With or without bullae</td>
<td>• Especially on areas subject to trauma, friction (buccal and labial mucosa, oral floor, anterior tongue)</td>
<td>• With or without erosions, ulcerations</td>
</tr>
<tr>
<td>• With or without ulcers, erosions</td>
<td>• With or without fragile bullae that readily rupture</td>
<td>• Lacy white, nonwipable, confluent papules (subtle or obvious) on gingiva, other sites</td>
</tr>
<tr>
<td>• Positive Nikolsky sign possible</td>
<td>• Positive Nikolsky sign</td>
<td></td>
</tr>
</tbody>
</table>

| **Diagnostic confirmation**            |                    |                             |
|  • Obtain biopsies for conventional and direct immunofluorescence microscopic analysis to confirm diagnosis |  • Obtain biopsies for conventional and direct immunofluorescence microscopic analyses to confirm diagnosis |  • If involvement is predominantly gingival (desquamation, erosions, erythema), diagnosis is indeterminate clinically |
|                                        |                    |  • Obtain biopsies for conventional and direct immunofluorescence microscopic analyses to rule out pemphigoid |
Box 23-5 Diagnostic protocol for biopsy of vesiculoulcerative lesions

- Obtain representative tissue biopsy specimen
- Submit in 10% neutral buffered formalin for conventional microscopic analysis
- Obtain a sample of intact (normal-appearing) perilesional tissue
- Submit in proper transport medium* for direct immunofluorescence analysis

*Request appropriate transport medium from pathology laboratory or rush fresh specimen on saline-moistened gauze to laboratory for processing

Table 23-8 Immunofluorescence signatures

<table>
<thead>
<tr>
<th>Conditions with “clean” subepithelial separation</th>
<th>Direct immunofluorescence findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mucous membrane (cicatricial) pemphigoid</td>
<td>IgG, C3</td>
</tr>
<tr>
<td>Bullous pemphigoid</td>
<td>Anti-BP antigen</td>
</tr>
<tr>
<td>Linear IgA disease (stomatitis)</td>
<td>IgA, C3</td>
</tr>
<tr>
<td>Epidermolysis bullosum (inherited forms)</td>
<td>Negative for immune reactants</td>
</tr>
</tbody>
</table>
### Table 23-9

<table>
<thead>
<tr>
<th>Epidermolysis bullosa acquisita</th>
<th>Anticollagen type IV, VII antibodies</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgG, immunoglobulin G; C3, complement 3; BP, bullous pemphigoid; IgA, immunoglobulin A.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pemphigoid</td>
</tr>
<tr>
<td>----------------------</td>
<td>-----------------------------------</td>
</tr>
<tr>
<td>Target antigen</td>
<td>Hemidesmosome</td>
</tr>
<tr>
<td></td>
<td>Mucous membrane pemphigoid: BP 80, laminin 5</td>
</tr>
<tr>
<td></td>
<td>Bullous pemphigoid: BP 180, BP 230</td>
</tr>
</tbody>
</table>
| Conventional histopathologic findings | “Clean” epithelial (epidermal) separation from (ie, epithelium “lifts off”) underlying lamina propria at BMZ | Intraepithelial separation within stratum spinosum | **Lichen planus:**  
|                      |                                    | Acantholytic spinous cells “float” within suprabasal cleavage |  
| Direct immuno-fluorescence findings | IgG, C3 (occasionally IgA) linear fluorescence at BMZ (ie, epithelial-stromal interface) | IgG, C3 fluorescence between epithelial spinous cells  
|                      |                                    |                                    | Fibrinogen in BMZ (immunoglobulins, complement rarely) |

BP, bullous pemphigoid; BMZ, basement membrane zone; IgG, immunoglobulin G; C3, complement 3; IgA, immunoglobulin A.
**Table 23-10** Management strategies: Mucous membrane pemphigoid, pemphigus, and lichenoid stomatitis
<table>
<thead>
<tr>
<th>Mucous membrane pemphigoid</th>
<th>Pemphigus</th>
<th>Lichenoid stomatitis</th>
</tr>
</thead>
</table>
| **Consultations** | • Ophthalmology consultation to rule out, intercept, and prevent complications from conjunctival involvement  
• OB/GYN consultation if patient reports undiagnosed, symptomatic vulvar lesions to rule out vulvar involvement | • Dermatology consultation to evaluate the entire body surface, intercept and prevent complications from preexisting cutaneous lesions, and to administer and monitor potent systemic immune modulation therapy | • Dermatology and/or OB/GYN evaluation if history is suggestive of possible involvement of skin and/or genitalia |
| **Treatment** | • Severity, extent of oral involvement dictates treatment  
**Topical treatment:**  
• Topical corticosteroids: (clobetasol, fluocinonide, betamethasone ointments) delivered in thin custom trays that cover the gingiva, three to five times/day and at bedtime  
• Dexamethasone elixir rinses  
**Systemic treatment:**  
• Doxycycline hyclate 20 mg (gingival lesions)  
• Low- to moderate-dose prednisone and/or azathioprine (coupled with a topical corticosteroid) | • Systemic corticosteroids (moderate- to high-dose prednisone, etc)  
• Cyclophosphamide/azathioprine  
• Mycophenolate mofetil  
• Other powerful systemic immune modulators  
• Select monoclonal antibodies | • Asymptomatic lesions require no treatment  
• Symptomatic lesions (erosive, ulcerative); treatment depends on severity, extent  
**Topical treatment:**  
• Topical corticosteroid agents (clobetasol, fluocinonide, betamethasone ointments), three to five times/day and at bedtime; use custom trays for gingival erosive lesions  
• Dexamethasone elixir rinses  
**Systemic treatment:**  
• Doxycycline hyclate 20 mg twice daily (gingival lesions)  
• 3-week course of prednisone coupled with topical corticosteroid |
| **Oral care** | • Frequent professional cleanings (eg, four times/year) and ongoing oral health maintenance annually | • Frequent professional cleanings (eg, four times/year) and ongoing oral health maintenance annually | • Ongoing oral health maintenance (frequent cleanings for gingival involvement); reevaluate at least annually |

OB/GYN, obstetrics/gynecology.
Oral Mucosal Masses or Nodules
The differential diagnosis for oral mucosal masses or nodules (Table 23-11) includes:

- Exuberant inflammatory and reactive hyperplastic lesions
- Benign neoplasms
- Malignant neoplasms

Exuberant Reactive Hyperplasias
These mucosal nodules or masses of submucosal tissue arise in response to local inflammatory or factitial injury or stimulation. Although they are benign, exuberant reactive hyperplasias require excision because: (1) they will not resolve spontaneously; (2) they can be impediments to maintaining optimal oral hygiene; (3) if they are composed of highly vascular granulation tissue (eg, pyogenic granulomas, peripheral giant cell granulomas) they can bleed on minor provocation; and (4) they are frequently located in sites where they can be traumatized repeatedly and are therefore an annoyance to the patient.

Benign Neoplasms
Unlike the exuberant reactive hyperplasias, benign neoplasms present themselves as asymptomatic nodules that arise in any location for no apparent reason. They feel discrete and circumscribed on palpation, grow slowly and symmetrically, and will continue to enlarge if they are not excised.

Malignant Neoplasms
Both primary and metastatic malignant neoplasms of the oral mucosa are relatively uncommon. A majority of primary oral cancers are squamous cell carcinomas that develop from precancerous leukoplakic or erythroleukoplakic changes in the stratified squamous epithelium that lines the surface of the oral cavity (see Tables 23-1 and 23-11). Other primary malignant neoplasms include tumors of minor salivary gland origin, mucosa-associated malignant lymphomas (MALTomas), and rarely, sarcomas.

Metastatic malignancies rarely present exclusively as mucosal masses.
They are more likely to present as intraosseous lesions with overlying mucosal involvement. There is usually a history of cancer and often a documented history of metastatic disease in other anatomical locations.

The following clinical features are suggestive of malignancy:

- Ulcerated mass
- Ill-defined on visualization, palpation
- Irregular surface
- Telangiectasias
- Bleeding tendency
- Pain, paresthesia
- Underlying bone involvement possible, especially when tumors are metastatic to jaws (eg, ill-defined, uncorticated lytic changes, loss of lamina dura, tooth mobility, pain, paresthesia)

*Table 23-11* Characteristics of masses of the oral mucosa
<table>
<thead>
<tr>
<th>Type of mass</th>
<th>Classic location</th>
<th>Classic history</th>
<th>Classic clinical features</th>
</tr>
</thead>
</table>
| *Exuberant reactive hyperplastic lesion* (common examples: traumatic fibroma, pyogenic granuloma, peripheral giant cell granuloma, epulis fissuratum, traumatic neuroma) | Sites of acute or chronic injury, irritation, inflammatory stimulation (eg, gingiva, hard palatal mucosa, buccal mucosa, labial mucosa, dorsal and lateral tongue surfaces, vermillion border of lip) | • Recalled or identified injury or irritant agent in the area involved  
  • Possible rapid growth of mass  
  • Painless  
  • Persists in presence of irritant  
  • May bleed if vascular (eg, pyogenic granuloma or peripheral giant cell granuloma) | • Raised tissue mass  
  • Asymptomatic  
  • Sessile or pedunculated  
  • Firm or soft; nonindurated  
  • Discrete but not encapsulated  
  • With or without surface ulceration  
  • With or without erythema, surface keratosis |
| *Benign neoplasm* (selected examples: pleomorphic adenoma, true fibroma, (angio)leiomyoma, lipoma, hemangioma, squamous papilloma) | Any intraoral site is possible; for benign minor salivary gland neoplasms: palate, upper labial mucosa, buccal mucosa | • No history of irritation  
  • Mass arises inexplicably  
  • Slow, steady, unprovoked increase in size; continues to enlarge if not excised  
  • Asymptomatic  
  • May become secondarily traumatized | *Submucosal benign neoplasms:*  
  • Asymptomatic  
  • Sessile, raised masses  
  • Discrete, nodular, round, encapsulated  
  • Smooth surface  
  • Mucosa colored  
  *Surface epithelial neoplasms:*  
  • Asymptomatic  
  • Fingerlike, exophytic papillary projections  
  • White (keratotic) or pink  
  • Pedunculated or sessile |
<table>
<thead>
<tr>
<th>Type of mass</th>
<th>Classic location</th>
<th>Classic history</th>
<th>Classic clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malignant neoplasm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Squamous cell carcinoma</td>
<td>Most commonly on intraoral cancer-prone sites</td>
<td>Adult, 45 years of age or older, Chronic past or active tobacco use, Heavy alcohol use</td>
<td>Mass, Irregular borders, nondiscrete, Associated with or arising in longstanding white, non-wipeable plaque or erythro- leukoplakic lesion, Ulceration, Induration, With or without pain, paresthesia</td>
</tr>
<tr>
<td>(classic oral cancer)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lip cancer (squamous cell</td>
<td>Lower lip vermilion border</td>
<td>Older, fair-skinned individual, Chronic sun exposure (UVA and UVB radiation)</td>
<td>Actinic cheilitis: pallor, erythroleukoplakic changes, Indistinct vermilion-skin border of lower lip, Chronic ulceration, erosion of vermilion border</td>
</tr>
<tr>
<td>carcinoma)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minor salivary gland</td>
<td>Palate, sublingual region, lower labial mucosa</td>
<td>Enlarging mass</td>
<td>Submucosal firm mass, Irregular surface, With or without ulceration, With or without telangiectasias, Adjacent teeth vital</td>
</tr>
<tr>
<td>malignancies</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type of mass</td>
<td>Classic location</td>
<td>Classic history</td>
<td>Classic clinical features</td>
</tr>
<tr>
<td>--------------------------------------------------</td>
<td>-------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Malignant melanoma</td>
<td>Gingiva, hard palate</td>
<td>• Nodule preceded by and/or accompanied by macular pigmentation</td>
<td>• Asymmetric, ill-defined mass</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Variegated pigmentation (brown, black, tan, mucosa-colored)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Extensive changes</td>
</tr>
<tr>
<td>Malignant lymphoma</td>
<td>Palate, gingiva overlying alveolar bone involvement</td>
<td>• History of malignant lymphoma</td>
<td>• With or without systemic signs: fatigue, malaise, weakness, weight loss, persistent indurated lymphadenopathy, pruritis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• No history of malignant lymphoma but mass on palate; loose teeth, fleshy mass on gingiva; pain or paresthesia</td>
<td></td>
</tr>
<tr>
<td>Other less common primary malignancies (sarcomas)</td>
<td>Any location</td>
<td>• Unexplained, submucosal mass</td>
<td>• Rapidly growing, destructive mass</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• With or without ulceration</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• With or without symptoms</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• If underlying osseous involvement, ill-defined lytic changes</td>
</tr>
<tr>
<td>Metastatic malignancy to oral cavity (eg, breast, lung, gastrointestinal system, prostate)</td>
<td>Bone (mandible &gt; maxilla); soft tissue metastatic foci are rare</td>
<td>• Usually known history of cancer</td>
<td>• Usually involves mandibular premolar-molar area or maxillary canine-premolar area.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Usually evidence of extraoral, extragnathic metastases</td>
<td>• Ill-defined lytic changes in bone</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Mass</td>
<td>• Fleshy, ulcerated mass</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• With or without pain, paresthesia</td>
<td>• Tooth mobility</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• With or without loose teeth</td>
<td>• Pain, paresthesia</td>
</tr>
</tbody>
</table>

UVA, ultraviolet A, UVB, ultraviolet B.
Differential Diagnosis of Intraosseous Lesions

Although in most cases the definitive diagnosis of an intraosseous lesion is established through microscopic review of a tissue biopsy specimen, the surgeon’s approach leading up to the biopsy is dictated by the differential diagnosis. The essential elements for the differential diagnosis of an intraosseous lesion include, first and foremost, a thorough and accurate description of the radiographic findings:

- Type of lesion (e.g., radiolucency, radiopacity, mixed lesion)
- Size of the lesion
- Type of borders (e.g., well- or ill-defined, corticated or uncorticated)
- Precise location and/or distribution of lesion(s) within the jaw(s)
- Relationship of lesion to adjacent structures (e.g., expansion, erosion of cortex, root resorption, tooth displacement)

The history, any symptoms or clinical signs of disease, and, if relevant or available, the results of diagnostic tests are also essential diagnostic data. If a radiographic or clinical condition is located adjacent to the teeth, the teeth in that region must be properly and thoroughly tested to determine pulpal vitality.

Individual intraosseous lesions can be categorized as radiolucent, radiopaque, and mixed type. Table 24-1 describes the general features of the pathologic processes that present as radiolucencies and radiopacities.

The Significance of Specific Radiographic Findings
Corticated versus Uncorticated Borders
Cortication at the border of a radiolucency or a mixed radiolucent-radiopaque lesion indicates that the lesion is slow growing, potentially expansile, and almost certainly benign. In essence, the surrounding bone has had sufficient time to respond by laying down a peripheral wall of reactive bone. Radiolucencies and mixed radiolucent-radiopaque lesions with corticated borders include:

- Cysts (odontogenic and nonodontogenic) (see chapter 15)
- Reactive lesions (e.g., condensing osteitis and focal cemento-osseous dysplasia; focal sclerosing osteitis)
- Benign neoplasms (see chapter 15)

*Table 24-1* Radiographic characteristics of pathologic processes in the jaws
<table>
<thead>
<tr>
<th>Pathologic process</th>
<th>Borders</th>
<th>Location/Distribution</th>
<th>Other factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammatory/reactive</td>
<td>• Discrete, corticated*</td>
<td>• Adjacent to teeth (periapical, in lateral periodontium)</td>
<td>• Nonvital tooth or teeth</td>
</tr>
<tr>
<td>process</td>
<td>• Diffuse, uncorticated*</td>
<td>• In sites of prior trauma, extraction</td>
<td>• Active or past history of periodontitis</td>
</tr>
<tr>
<td></td>
<td>• Demarcated, sclerotic*</td>
<td>• Mandible &gt; maxilla</td>
<td>• History of trauma or dentoalveolar injury, surgery, extraction</td>
</tr>
<tr>
<td></td>
<td>• Sclerotic, demarcated, or diffuse†</td>
<td>• May be single or multiple†</td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute osteomyelitis</td>
<td>• No radiographic changes until after 60% demineralization (5 to 7 days)*</td>
<td>• Associated with periapical abscess</td>
<td>• Nonvital tooth may be the source</td>
</tr>
<tr>
<td></td>
<td>• Radiolucency with ill-defined, uncorticated, moth-eaten borders</td>
<td>• Area of traumatic injury (accidental or iatrogenic) to dentoalveolar process or cortical bone</td>
<td>• If teeth vital, history of injury</td>
</tr>
<tr>
<td></td>
<td>• With or without* internal opacities coupled with sequestra</td>
<td>• Extraction site</td>
<td>• Severe pain</td>
</tr>
<tr>
<td>Chronic osteomyelitis</td>
<td>• Usually mixed radiolucency-opacity with sclerosis of surrounding bone</td>
<td>• Evidence of extant or prior infectious source (eg, periodontal bone loss, nonvital tooth, persistent extraction socket, fracture)</td>
<td>• Tooth mobility</td>
</tr>
<tr>
<td></td>
<td>• Diffuse or defined borders</td>
<td></td>
<td>• Fluctuant swelling, purulent exudate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Erythema</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• With or without fever, regional lymphadenopathy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Paresthesia (uncommon)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• With or without history of acute osteomyelitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• With or without intermittent symptoms/signs of activity (discomfort/pain; swelling; exudate)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• With or without history of exfoliation of necrotic bone (sequestration)</td>
</tr>
</tbody>
</table>

*Specific to pathologic processes presenting as radiolucencies.
†Specific to pathologic processes presenting as radiopacities.
<table>
<thead>
<tr>
<th>Pathologic process</th>
<th>Borders</th>
<th>Location/Distribution</th>
<th>Other factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign neoplasm</td>
<td>• Round, symmetric radiolucency* or opacity or mixed radiolucency-opacity</td>
<td>• Mandible &gt; maxilla</td>
<td>• Painless, expansile, bony, hard mass</td>
</tr>
<tr>
<td></td>
<td>• Discrete, corticated borders</td>
<td></td>
<td>• With or without facial asymmetry</td>
</tr>
<tr>
<td></td>
<td>• Scalloped, corticated borders possible*</td>
<td></td>
<td>• Overlying mucosal tissue intact, normal color</td>
</tr>
<tr>
<td></td>
<td>• May have internal loculations*</td>
<td></td>
<td>• No inflammatory stimulus, no history of trauma</td>
</tr>
<tr>
<td></td>
<td>• May displace adjacent structures†</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• With or without external root resorption†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant neoplasm</td>
<td>• Ill-defined, asymmetric lytic changes</td>
<td>• Primary malignant tumor of oral mucosa that invades underlying bone</td>
<td>• Overlying soft tissue mass with irregular borders, ulceration</td>
</tr>
<tr>
<td></td>
<td>• No discernible borders*</td>
<td>• Other primary malignancy within bone</td>
<td>• Pain, paresthesia (mandible)</td>
</tr>
<tr>
<td></td>
<td>• With or without vague opacities within lytic foci</td>
<td>• Metastatic neoplasm to jaw</td>
<td>• Progressive unexplained tooth mobility</td>
</tr>
<tr>
<td></td>
<td>• Loss of lamina dura</td>
<td>• Mandible &gt; maxilla</td>
<td>• Vital teeth</td>
</tr>
<tr>
<td></td>
<td>• With or without irregular, spiky root resorption</td>
<td></td>
<td>• Usually history of distant malignancy*</td>
</tr>
</tbody>
</table>

*Specific to pathologic processes presenting as radiolucencies.
†Specific to pathologic processes presenting as radiopacities.

Continues on next page.
<table>
<thead>
<tr>
<th>Pathologic process</th>
<th>Borders</th>
<th>Location/Distribution</th>
<th>Other factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic process</td>
<td>• Diffuse, ill-defined</td>
<td>• Usually multifocal, multiquadrant involvement (both arches)</td>
<td>• No local source of injury, irritation</td>
</tr>
<tr>
<td></td>
<td>• Discrete, uncorticated*</td>
<td>• Bilateral</td>
<td>• With or without† loosening of teeth</td>
</tr>
<tr>
<td></td>
<td>• Corticated, septated lucencies*</td>
<td>• History of other skeletal, visceral lesions</td>
<td>• Vital teeth</td>
</tr>
<tr>
<td></td>
<td>• Multiple opacities†</td>
<td>• With or without biochemical abnormalities (eg, elevated Ca**, phosphate, alkaline phosphate)</td>
<td>• With or without swelling, symptoms</td>
</tr>
<tr>
<td></td>
<td>• Associated radiolucentes†</td>
<td>• With or without hematologic abnormalities</td>
<td>• With or without history of specific systemic illness with potential for osseous involvement (eg, hyper-involvement (eg, hyper-parathyroidism, sarco...</td>
</tr>
<tr>
<td></td>
<td>• Both arches usually involved†</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Specific to pathologic processes presenting as radiolucentes.
†Specific to pathologic processes presenting as radiopacities.
Uncorticated but discrete borders may be seen in association with lesions that represent a spectrum of pathologic processes ranging from developmental anomalies to benign reactions to inflammatory stimuli or traumatic injury to multicentric systemic or neoplastic diseases. Examples include but are not limited to:

- Periapical granuloma
- Simple (traumatic) bone cyst
- Stafne defect (lingual mandibular bone concavity)
- Multiple myeloma
- Langerhans cell histiocytosis

The differential diagnosis of lytic lesions with uncorticated, ill-defined or ragged borders, with or without internal opacities, must include selected systemic conditions, malignancies, and destructive, aggressive local processes.

- Infections (osteomyelitides) (see chapter 17)
- Systemic diseases with osseous involvement (eg, sarcoidosis, hyperparathyroidism, resorptive phase of Paget disease of bone)
- Primary malignant neoplasms
- Malignancies metastatic to the jaw

Radiolucencies

Periapical region radiolucency

Radiolucency in the periapical region is among the most common finding in the tooth-bearing regions of the jaws (see chapter 15). When a radiolucency is seen in association with the root of a tooth, the possibility that it represents an inflammatory cyst; periapical granuloma; or abscess attributable to pulpal necrosis, inflammation, or infection should be ruled out through tooth vitality testing.

A reasonable differential diagnosis for a radiolucency with no internal contents that is located in the periapical region, in the lateral periodontium between two teeth, or in an edentulous space previously occupied by an extracted tooth or teeth includes:

- Inflammatory odontogenic lesion (ie, associated with a nonvital tooth)
—Periapical (radicular) cyst
—Periapical granuloma
—Inflammatory lateral periodontal cyst (either or both of the associated teeth is nonvital)
—Residual periapical cyst
—Residual inflammatory lateral periodontal cyst

• Developmental odontogenic cyst (vital adjacent tooth or teeth)
  —Developmental (ie, noninflammatory) lateral periodontal cyst (including botryoid type—often multilocular)
  —Residual lateral periodontal cyst
  —Residual follicular (dentigerous) cyst
  —Odontogenic keratocyst (also referred to as keratinizing cystic odontogenic tumor [KCOT])

• Nonodontogenic (fissural) cyst (midline, maxilla; vital teeth)
  —Nasopalatine canal cyst
  —Median palatal cyst

• Benign odontogenic or nonodontogenic neoplasm
  —Ameloblastoma (bony septations possible)
  —Odontogenic myxoma (bony septations possible)
  —Ameloblastic fibroma
  —Squamous odontogenic tumor (very uncommon)
  —Odontogenic fibroma
  —Various benign nonodontogenic neoplasms (mesenchymal origin)

Pericoronal radiolucency with a corticated border
• Dental follicle
• Dentigerous (follicular) cyst
• Odontogenic keratocyst (KCOT)
• Ameloblastoma
• Adenomatoid odontogenic tumor (usually surrounds the crown and much of the root of an unerupted tooth; typically associated with unerupted permanent canine and/or premolar teeth); may contain small calcifications in addition to unerupted tooth
• Other benign odontogenic neoplasms or hamartomas (eg, odontogenic
myxoma, ameloblastic fibroma)

Radiolucencies with internal calcifications that are not exclusively pericoronal
- Ameloblastic fibro-odontoma
- Calcifying epithelial odontogenic tumor (CEOT; Pindborg tumor)
- Odontogenic fibroma
- Keratinizing and calcifying odontogenic cyst (KCOC; Gorlin cyst)
- Central giant cell lesion (largely radiolucent but may contain wispy trabeculae) (Table 24-2)
- Osteoblastoma/osteoid osteoma
- Other benign neoplasms (with bone and/or cementum)
- Benign fibro-osseous diseases (Table 24-3)

Radiopacities
Radiopacities (radiodensities) in the jaws may be seen as isolated findings or more often in association with one or more radiolucencies (corticated or uncorticated) that are localized or widely distributed in accordance with the pathologic process represented. They may represent osseous tissue, dental hard tissue (dentin, enamel, or cementum, combined or alone) or a combination of bone and tooth elements. Occasionally a radiopacity can represent a foreign material (eg, contrast medium, gutta-percha, metallic restorative material, etc). The precise nature of a radiopacity of the maxilla or mandible is determined by its histomorphology on microscopic examination.

Radiopaque lesions with discrete, corticated borders
- Cementoblastoma (periapical/periradicular location, intimate attachment to root[s], external root resorption, premolar-molar regions)
- Ameloblastic fibro-odontoma
- KCOC (Gorlin cyst)
- CEOT (Pindborg tumor)
- Odontoma (compound, complex, composite)
- Osteoma
- Osteoblastoma
- Benign fibro-osseous neoplasms (eg, ossifying fibroma, cemento-ossifying fibroma, juvenile [active] ossifying fibroma) (see Table 24-3)
- Some lesions of cemento-osseous dysplasia (eg, periapical cemento-
osseous dysplasia, florid cemento-osseous dysplasia, focal cemento-osseous dysplasia) (see Table 24-3)

**Location/Distribution Within the Jaw: Local versus Systemic Origin**

A radiographic finding that is discrete and isolated to a single location is more likely a lesion of local rather than systemic origin. Lesions stemming from an underlying systemic condition tend to be more widely distributed. They often involve more than one quadrant and in some instances can be found in both arches. With systemic influences, the historical and clinical data may also suggest or reveal involvement of other, extragnathic skeletal sites. Some examples of intraosseous lesions of systemic origin include but are not limited to: lytic lesions of hyperparathyroidism, sarcoidosis, Langerhans cell disease, multiple myeloma, and metastatic carcinoma; mixed radiolucent-radiopaque changes seen in Paget disease of bone (osteitis deformans); and the ground-glass type of trabecular densities characteristic of fibrous dysplasia.

**Relationship of Radiographic Finding to Adjacent Structures**

In general, evidence of smooth rather than ragged or “spiky” external root resorption is indicative of a lesion that is growing slowly. Such lesions tend to push away or press on adjacent structures as they expand, whereas more rapidly moving processes (eg, aggressive infections, malignancies) tend to insinuate themselves among neighboring structures and produce a more irregular, ragged, or erosive resorptive pattern, reflective of a destructive process.

Benign, more indolent bone lesions tend to push on adjacent teeth and can divert or displace them. They can also expand and thin out the cortical bone. The overlying mucosal tissue usually remains intact unless it is factitiously disrupted.

Aggressive lesions in bone (eg, infections, malignancies) tend to erode into and through adjacent structures (eg, teeth, cortical and alveolar bone) and defy natural boundaries. Overlying mucosal tissue may be ulcerated, may
appear as a nodular or irregularly-shaped swelling, and may exhibit surface erythema and/or telangiectasia.

*Table 24-2 Characteristics of giant cell lesions*
| **Central giant cell granuloma**  
(Giant cell lesion) | **Brown tumor**  
(Hyperparathyroidism) | **Aneurysmal bone cyst** | **Cherubism** |
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Etiology</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| • Nonneoplastic (ie, nonaggressive lesion): few if any symptoms, slow growth, no cortical perforation, no root resorption | • Primary or secondary hyperparathyroidism  
• Osteoclastic bone resorption mediated by increased parathyroid hormone (PTH) secretion: Secondary > primary hyperparathyroidism  
• Genetic abnormality | • Uncertain etiology  
**Possibly:**  
• Trauma-induced  
• Vascular malformation  
• Neoplasm that disrupts normal hemodynamics of bone, resulting in hemorrhagic extravasation | • Autosomal dominant genetic trait  
• Familial  
• Males > females  
• Spontaneous mutations |
| **Clinical/historical features** | | | | |
| • Wide age range; 60% in patients younger than 30 years  
• Most are asymptomatic  
• Painless expansion  
• Few with pain, paresthesia, cortical perforation, overlying mucosal ulceration | • Usually in middle-aged or older adults  
• Increased serum Ca++, PO₄⁻⁻  
• Osteitis fibrosa cystica  
• With or without history of subperiosteal bone resorption, osteoporosis, pathologic functioning  
• With or without history of kidney stones, gastrointestinal disturbances, and neurologic and/or mental disturbances | • Intraosseous blood-filled spaces invested by fibrous connective tissue interwoven with reactive bone  
• Possibly a hemorrhagic communication with feeder vessels, leading to giant cell response  
• May be primary or secondary lesions  
• Wide age range affected  
• More common in children, young adults  
• Favors posterior segments of mandible, maxillary sinus/nasal complex | • Early onset (ages 2 to 5 years)  
• Slow progression until puberty, then stabilizes  
• Bilateral facial swellings  
• Hypertelorism  
• Eyes turned upward; sclera inferior to iris exposed (cherubic appearance)  
• Painless |
<table>
<thead>
<tr>
<th>Central giant cell granuloma (Giant cell lesion)</th>
<th>Brown tumor (Hyperparathyroidism)</th>
<th>Aneurysmal bone cyst</th>
<th>Cherubism</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Radiographic features</strong></td>
<td><strong>Histologic features</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Unilocular or multilocular radiolucency</td>
<td>• Abundant multinucleated giant cells in a cellular or loose fibrovascular tissue background</td>
<td>• Varying sizes of blood-filled spaces (not endothelially lined) outlined by cellular fibrous connective tissue containing multinucleated giant cells and trabeculae of bone and osteoid</td>
<td>• Bilateral multilocular, expansile radiolucencies involving mandibular rami and angles; occasionally body of mandible also involved</td>
</tr>
<tr>
<td>• Well demarcated</td>
<td>• With or without fibrosis</td>
<td>• May be associated with fibrous dysplasia lesions, central giant cell lesions</td>
<td>• Maxillary involvement may be less pronounced</td>
</tr>
<tr>
<td>• Margins with or without cortication</td>
<td>• Hemorrhagic foci common</td>
<td>• More fibrous and fewer giant cells with time</td>
<td></td>
</tr>
<tr>
<td>• Large expansile lesions radiographically indistinguishable from ameloblastoma, OKC (KCOT)</td>
<td>• With or without osteoid, new bone</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Histologic features**

- • Unilocular or multilocular radiolucency
- • Generalized, diffuse diminished calcification of the jawbones (osteoporotic changes)
- • Loss of lamina dura
- • Discrete, expansile radiolucencies with or without corticated borders
- • With or without root resorption
- • Unilocular or multilocular radiolucency
- • Thinning and expansion of cortical bone
- • Well-defined or diffuse borders possible
- • With or without radiopaque foci internally
Table 24-3 Characteristics of benign fibro-osseous lesions
<table>
<thead>
<tr>
<th>Fibrous dysplasia</th>
<th>Cemento-osseous dysplasias</th>
<th>Benign fibro-osseous neoplasms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Etiology</strong></td>
<td>Genetic</td>
<td>Benign neoplasms arise unprovoked</td>
</tr>
<tr>
<td></td>
<td>Prototypical hamartomatous</td>
<td>Probably result from genetic</td>
</tr>
<tr>
<td></td>
<td>benign fibro-osseous disease</td>
<td>mutation</td>
</tr>
<tr>
<td><strong>Clinical features</strong></td>
<td>Early onset</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td></td>
<td>With or without family history</td>
<td>Bony hard mass</td>
</tr>
<tr>
<td></td>
<td>Monostotic or polyostotic skeletal involvement</td>
<td>Discrete</td>
</tr>
<tr>
<td></td>
<td>Painless enlargement of affected bone (maxilla &gt; mandible)</td>
<td>Slow, symmetric growth</td>
</tr>
<tr>
<td></td>
<td>May continue to be active throughout life</td>
<td>Expansile swelling</td>
</tr>
<tr>
<td></td>
<td>May be component of a genetic syndrome (eg, Albright syndrome)</td>
<td>Mandible &gt; maxilla</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Periapical cemento-osseous dysplasia</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Significant predilection for adult African-American women</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mandibular anterior region</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Usually asymptomatic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Teeth vital</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Florid cemento-osseous dysplasia</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adult African-American women</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Early: asymptomatic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Later: painless swellings</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Both maxilla and mandible may be involved, all sextants</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bilateral, not symmetric</td>
</tr>
<tr>
<td></td>
<td></td>
<td>May develop superimposed osteomyelitis with periodic exacerbations (swelling, pain, erythema), exfoliation of sequestra, and sinus tracts</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Focal cemento-osseous dysplasia</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adults, no racial predilection</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Posterior mandible (premolar-molar area)</td>
</tr>
<tr>
<td>Fibrous dysplasia</td>
<td>Cemento-osseous dysplasias</td>
<td>Benign fibro-osseous neoplasms</td>
</tr>
<tr>
<td>-------------------</td>
<td>-----------------------------</td>
<td>-------------------------------</td>
</tr>
</tbody>
</table>
| **Clinical features (cont)** | • With or without swelling and other symptoms  
• Evidence or history of inflammatory source (e.g., odontogenic infection, periodontal inflammatory stimulus, previous extraction)  
• Adjacent teeth need to be tested for pulpal vitality | • Well-circumscribed radiolucency  
• Corticated borders  
• Expansion possible  
• With or without internal opacifications  
• May displace adjacent tooth roots  
• May cause external root resorption  
• Does not devitalize adjacent teeth |
| **Radiographic features** | • Ground-glass (fine, dense, diffuse) trabecular pattern  
• Blends into surrounding bone (indiscernible borders)  
• With or without cystic-like areas | **Periapical cemento-osseous dysplasia**  
• Mandibular incisor-canine region  
• Periapical area(s)  
• Borders discrete, with or without cortication  
• Usually multiple round to ovoid radiolucencies  
• With or without internal opacities; tend to become more sclerotic over time  
**Florid cemento-osseous dysplasia**  
• Multiple radiolucent-radiodense nodular-appearing lesions  
• Found in tooth-bearing regions of both arches; periapical, periodontal, and edentulous areas  
• With or without cystic-like lucencies |
<table>
<thead>
<tr>
<th>Fibrous dysplasia</th>
<th>Cemento-osseous dysplasias</th>
<th>Benign fibro-osseous neoplasms</th>
</tr>
</thead>
</table>
| **Radiographic features (cont)** | • Expansile (alveolar crestal bone, buccal and lingual cortices)  
  *Focal cemento-osseous dysplasia*  
  • Single lesion  
  • Periapical or in edentulous space  
  • Often adjacent to obvious inflammatory source  
  • Mixed radiolucent-radiopaque or exclusively radiolucent changes  
  • With or without expansion  
  • Borders discrete with or without cortication | |
| **Histopathology** | • Diffuse, unencapsulated benign fibrovascular tissue with varying quantities of osteoid, individual and anastomosing trabeculae of woven and lamellar bone | • All of the cemento-osseous dysplasias have identical features  
  • Combinations of osteoid, bony trabeculae, and droplet-like cementum and/or osseous tissue in a benign delicate fibrous tissue background  
  • With or without inflammatory cells—usually minimal to absent |
| | | • Encapsulated mass  
  • With or without shell of surrounding bone  
  • Benign, moderately or densely cellular fibrocollagenous tissue  
  • With or without combinations of bone and cementum-like hard tissue droplets or trabeculae, or one or the other exclusively JOFs |
<table>
<thead>
<tr>
<th></th>
<th>Fibrous dysplasia</th>
<th>Cemento-osseous dysplasias</th>
<th>Benign fibro-osseous neoplasms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Histopathology</strong></td>
<td></td>
<td>• Biopsy usually not necessary for periapical cemento-osseous dysplasias</td>
<td>• Encapsulated mass</td>
</tr>
<tr>
<td>(cont)</td>
<td></td>
<td>• Florid cemento-osseous dysplasias with superimposed osteomyelitis may demonstrate necrotic bone and/or involucrum, acute and/or chronic inflammatory infiltrates, dense bone, and/or cementumlike components</td>
<td>• Benign cellular fibrovascular stromal tissue</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• • Garlandlike trabeculae of bone, osteoid</td>
<td>• Psammomalike droplets of cementum or bone also possible</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• With or without foci of multinucleated giant cells</td>
<td></td>
</tr>
</tbody>
</table>
Differential Diagnosis and Management of Neck Masses

Diagnosis of a neck mass and its subsequent treatment require strict attention to detail in obtaining a history, performing a physical examination, and acquiring special imaging studies. Establishing a differential diagnosis is paramount in this process. Only after this point are invasive procedures such as a biopsy performed. The temptation to expediently perform a biopsy without first obtaining a history, performing a physical examination, and establishing a differential diagnosis violates the scientific approach and may be detrimental to the patient. Moreover, an incisional biopsy does not have a role in the management of neck masses, with the possible exception of an infectious process. Finally, the explicit purpose of establishing a differential diagnosis of a neck mass is to rule out a pathologic process that requires expediency in treatment, as would be the case with neoplastic disease, particularly malignant disease.

History

Age

The patient’s age represents the first important piece of information that can accurately guide the surgeon’s workup of a neck mass. For the purpose of developing a differential diagnosis, patient ages are categorized as follows:

- Pediatric patient: 12 years of age or younger
- Young adult patient: Between 13 and 40 years of age
- Adult patient: Older than 40 years of age

These age designations are important because the prevalence of congenital, inflammatory, and neoplastic diseases may vary according to age (Table 25-
In general, neck masses in patients older than 40 years should alert the clinician to the possibility of neoplastic disease. For example, a nonthyroid neck mass in an older patient has an 80% chance of being neoplastic, and 80% of these masses are malignant.

Chief Complaint
The neck mass may be apparent to the patient, possibly brought to his or her attention by the referring doctor or, in the case of a pediatric or young adult patient, noticed by the patient’s parents.

*Table 25-1* Common conditions found in the neck in various age groups
<table>
<thead>
<tr>
<th>Age 0–12 y</th>
<th>Age 13–40 y</th>
<th>Age &gt; 40 y</th>
</tr>
</thead>
</table>
| Inflammatory | • Adenitis: bacterial, viral  
   • Granulomatous lesion  
   • Sialadenitis: parotid (mumps), submandibular | • Adenitis: bacterial, viral  
   • Granulomatous lesion  
   • Sialadenitis: parotid, submandibular | • Sialadenitis: parotid, submandibular |
| Congenital/Developmental | • Lymphangioma  
   • Thyroglossal duct cyst  
   • Dermoid cyst  
   • Salivary gland (plunging ranula) | • Branchial cleft cyst  
   • Thyroglossal duct cyst  
   • Salivary gland (plunging ranula) | • Salivary gland (plunging ranula) |
| Neoplastic | • Lymphoma | • Thyroid tumor  
   • Lymphoma  
   • Carotid body tumor  
   • Salivary gland tumor: parotid, submandibular | • Thyroid tumor  
   • Lymphoma  
   • Carotid body tumor  
   • Salivary gland tumor: parotid, submandibular |
History of Present Illness

The history of present illness should provide the following information:

- The presence or absence of pain
- The presence or absence of erythema and/or drainage from the mass
- The chronicity of the neck mass
- The presence of a solitary mass versus multiple neck masses
- The presence of additional masses located in the axilla, groin, or elsewhere
- The perception of progression of the size of the neck mass
- The existence of a first-time mass or a recurrent process
- Recent animal contact and possible scratches
- Travel history

A history of a neck mass in a fetus discovered by ultrasound, noted for the first time on delivery, or identified shortly after birth, suggests a lymphangioma (cystic hygroma). The presence of a long-standing process (ie, years) not associated with pain and with minimal change in size over time in a patient of any age likely points to a benign process. In general, a mass present for 7 days may be inflammatory in its etiology, a mass present for 7 months is probably neoplastic, and a mass present for 7 years may be congenital. A history of animal contact with scratches would require the placement of cat-scratch disease (*Bartonella henselae*) on the differential diagnosis list, whereas multiple, enlarged neck masses with or without fever or associated with masses located in the axilla, groin, or elsewhere suggest a possible diagnosis of lymphoma. The presence of pain, erythema, fever, and drainage associated with the neck mass should certainly lead one to suspect an abscess.

Past Medical History

The past medical history should focus on circumstances that could be responsible for the development of a neck mass:

- Tuberculosis (scrofula)
- Recent medical care for an animal scratch
- Lymphoma
• Oral or head and neck cancer

Past Surgical History

The past surgical history may also reveal information of significance in establishing a differential diagnosis of a neck mass:

• Removal of mucosal or cutaneous malignant disease (metastatic adenopathy)
• Prior removal of a neck mass (recurrent disease)
• Excision of cancer of the breast, lung, or other organs distant from the neck (metastatic adenopathy)

Under any of these circumstances, the details of the previous surgical excision of a lesion, including its diagnosis, should be pursued.

Family History

Obtaining a patient’s family history is important because conditions such as Hodgkin lymphoma or melanoma may occur in multiple members of a family.

Social History

The patient’s social history should focus on exposure to possible carcinogens:

• Use of tobacco (cigarettes, cigars)
• Use of smokeless tobacco (chewing tobacco, dry snuff, moist snuff)
• Significant sun exposure

Review of Systems

The review of systems will disclose symptoms that can assist in establishing the differential diagnosis:

• Fever
• Night sweats
• Recent weight loss
• Anorexia
• Epistaxis
• Hoarseness
• Dysphagia
- Odynophagia
- Hemoptysis

Fever, night sweats, and weight loss are referred to as *B symptoms* and may be associated with a diagnosis of lymphoma. Night sweats, hemoptysis, anorexia, and weight loss may be symptoms in patients with tuberculosis. A history of hoarseness with or without dysphagia or odynophagia may be suggestive of thyroid cancer.

**Vital Signs**

In the examination of a patient with a neck mass, an elevated temperature is one sign suggestive of an infectious process that might explain its presence. In addition, some Hodgkin lymphomas are associated with fever (Pel-Ebstein fever).

**Physical Examination**

The following areas should be examined in a patient with a neck mass:

- **Skin:** Inspect and palpate for masses, ulcers, erythema
- **Oral cavity:** Inspect and palpate for mucosal lesions
- **Oropharynx:** Inspect for mucosal lesions as well as the size and character of palatine tonsils, if present
- **Nasal cavity:** Inspect for masses, ulcers, and signs of recent hemorrhage
- **Nasopharynx/hypopharynx:** Assess with indirect mirror examination and consider nasopharyngoscopy to evaluate these regions

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**Box 25-1 Anatomical boundaries and oncologic levels of the neck**

**Anatomical regions of the neck**

- *Anterior triangle:* Demarcated by the midline of the neck (anterior border), the inferior border of the mandible (base) and the sternocleidomastoid muscle
The anterior triangle may be subclassified into the central compartment lymph nodes along the trachea and the parotid tail region.

- **Posterior triangle**: Demarcated by the sternocleidomastoid muscle (anterior border), the trapezius muscle (posterior border), and the clavicle (inferior border).

### Oncologic levels of the neck

- **Level IA (submental)**: Lymph nodes within the triangular boundary of the anterior bellies of the digastric muscles and the hyoid bone.
- **Level IB (submandibular)**: Lymph nodes within the boundaries of the anterior belly of the digastric muscle and the stylohyoid muscle and the inferior border of the mandible.
- **Level IIA and IIB (upper jugular)**: Lymph nodes located around the upper third of the internal jugular vein and the adjacent spinal accessory nerve; level IIA lymph nodes are located anterior (medial) to the spinal accessory nerve; level IIB lymph nodes are located posterior (lateral) to the spinal accessory nerve.
- **Level III (middle jugular)**: Lymph nodes located around the middle third of the internal jugular vein; nodes are located between the lower border of the hyoid bone and the inferior border of the cricoid cartilage.
- **Level IV (lower jugular)**: Lymph nodes located around the lower third of the internal jugular vein; nodes extend from the inferior border of the cricoid cartilage to the clavicle.
- **Level V (posterior triangle)**: Lymph nodes located along the lower half of the spinal accessory nerve and the transverse cervical artery; supraclavicular lymph nodes.
- **Level VI (central compartment)**: Lymph nodes in the prelaryngeal, pretracheal, paratracheal, and tracheoesophageal groove; boundaries are the hyoid bone to the suprasternal notch and between the medial
borders of the carotid sheaths.

- Neck
  - Document the anatomical site and oncologic level of the mass (Box 25-1)
  - Note the size of the mass
  - Determine fixation to surrounding structures
  - Note the presence or absence of pain on palpation of the mass
  - Check for adenopathy in the remainder of the bilateral neck regions
  - Determine the character and consistency of the mass, eg, fluctuant, indurated, pulsatile
  - Note the presence or absence of a palpable thrill
  - Note the presence or absence of an auscultated bruit
  - Determine whether the neck mass moves during swallowing

**Box 25-2 Anatomical location of specific neck masses**

<table>
<thead>
<tr>
<th>Midline neck</th>
<th>Lateral upper neck</th>
<th>Lateral lower neck</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Thyroglossal duct cyst</td>
<td>• Submandibular gland neoplasms</td>
<td>• Lymphoma</td>
</tr>
<tr>
<td>• Dermoid cyst</td>
<td>• Inflammatory conditions</td>
<td>• Metastatic adenopathy from head and neck malignancy</td>
</tr>
<tr>
<td>• Submental lymphadenopathy</td>
<td>• Plunging ranula</td>
<td></td>
</tr>
<tr>
<td>• Thyroid cancer</td>
<td>• Branchial cleft cyst</td>
<td>• Metastatic adenopathy from chest, abdominal, pelvic malignancy (Virchow node: left supraclavicular node)</td>
</tr>
</tbody>
</table>
A general physical examination of the patient presenting with a neck mass should also be performed so as to be medically complete and to properly prepare for future surgery.

**Differential Diagnosis**

The patient’s age, assessment of possible exposure to carcinogens, the location and character of the neck mass, and other findings on the physical examination will permit the development of a differential diagnosis that should be established in the order of the likelihood of the diagnosis (Box 25-2; see also Table 25-1). The differential diagnosis of a neck mass may be subsequently refined based on the results of a variety of available diagnostic imaging studies.

**Diagnostic Imaging Studies**

In establishing a differential diagnosis, the surgeon should determine the utility of obtaining imaging studies to provide additional information about the neck mass. These imaging studies may be obtained prior to or after the performance of a fine needle aspiration biopsy (FNAB) or excision of the neck mass. The differential diagnosis will guide the proper sequence.
Plain Films (Panoramic Radiograph)
Plain films help to rule out an odontogenic etiology for the problem. They may also be of assistance in the diagnosis of a neck mass suspected of representing a specific vascular lesion. For example, the presence of phleboliths (multiple, small intravascular calcifications) in the region of the facial vessels at the inferior border of the mandible are most suggestive of a hemangioma. In addition, the identification of a calcification in level IB of the neck on a panoramic radiograph in a patient with a mass in this area would point to a likely diagnosis of submandibular sialolithiasis.

Computed Tomography (CT Scans)
CT scans provide structural (anatomical) assessment of the neck mass. In general, intravenous contrast should be used unless the patient has an iodine allergy or has been diagnosed with, or is predisposed to, renal insufficiency/failure. Pretreatment with corticosteroids may be considered if the patient has an iodine allergy. The use of intravenous contrast can provide information regarding the possible vascular nature (carotid body tumor) of a neck mass when physical examination reveals a bruit or thrill associated with it.

Magnetic Resonance Imaging (MRI) Scans
MRI scans provide superior structural imaging of soft tissue lesions.

Positron Emission Tomography (PET) and CT Scans
PET scans combined with CT scans may be particularly useful in a patient with a neck mass whose medical/surgical histories revealed a malignancy of the oral or head and neck regions, or for a patient for whom a diagnosis of lymphoma is favored. These scans provide functional imaging (PET) as well as structural imaging (CT) associated with neck masses.

Angiogram
Angiograms provide information regarding the vascular nature of a neck mass, including specific feeder vessels.
CT or Magnetic Resonance (MR) Angiogram

CT or MR angiograms may be ordered for patients in whom the history and physical examination are most suggestive of a vascular etiology of a neck mass. The CT or MR component of the study will provide structural imaging of the neck mass, whereas the angiogram component will elucidate the vascular nature.

The radiologist interpreting an imaging study should be asked to establish the radiologic differential diagnosis to which the surgeon can compare his or her clinical differential diagnosis. This exercise is most useful in planning the next step in the workup.

Fine Needle Aspiration Biopsy

Numerous clinical circumstances dictate the performance of a fine needle aspiration biopsy (FNAB) during the course of the workup of a mass in the neck. For example, the patient with an isolated mass of level II of the neck and a current or past history of exposure to identifiable carcinogens for oral or head and neck cancer should undergo FNAB of the neck mass when no apparent mucosal disease is discovered on physical examination. Such a patient might be appropriately labeled as having an unknown primary cancer. Proceeding directly to isolated lymph node excision in such a patient violates the oncologic principles of neck surgery. Performing the FNAB will, in most cases, identify squamous cell carcinoma and result in the proper performance of a comprehensive neck dissection.

Another example is the presence of a parotid tail mass extending into the neck that is identified on physical examination and confirmed by CT or MRI scans. Under these circumstances, it may be appropriate to perform an FNAB to determine whether the tumor is benign or malignant preoperatively. Such a determination assists the surgeon in deciding the need and utility of performing a neck dissection at the time of performing the parotidectomy.

On the other hand, a patient without mucosal disease and identifiable exposure to carcinogens may be subjected to excision of the neck mass without preoperative FNAB. This scenario is typical for patients with congenital /developmental masses and inflammatory diseases and includes the following:
- A neck mass present in a pediatric patient when the CT scans identify a fluid-filled lesion that suggests a lymphangioma.
- A midline mass of the neck that moves superiorly during swallowing in a young adult or adult patient is suggestive of a thyroglossal duct cyst. This empiric diagnosis requires that a complete physical examination of the oral cavity and oropharynx shows no evidence of mucosal disease.
- A plunging ranula in which a diagnosis can be made primarily on physical examination and is supported by the appearance of a fluid-filled mass in the neck on the CT scan.
- Obstructive salivary gland disease that is diagnosed clinically based on the patient’s history and physical examination. Under such circumstances, CT scans will show signs consistent with sialadenitis and possibly sialolithiasis.
- Suspicion of a carotid body tumor, which typically results in obtaining a vascular study and negates the performance of an FNAB.

When FNAB is indicated, it should be performed so as to not violate the neck harboring malignant disease, which should be managed with a neck dissection.

**Treatment**
The decision as to the form of therapy for a neck mass is based on the specific diagnosis. Surgical options include the following:

**Excision**
*Excision* is defined as the isolated surgical removal of the soft tissue mass only, leaving surrounding tissue intact. It is performed for inflammatory diseases (bacterial or viral adenitis only when a clinical diagnosis is inconclusive; special histologic stains of the tissue from the neck mass are required to make a definitive diagnosis). It is also indicated for congenital/developmental lesions such as the branchial cleft cyst, dermoid cyst, hemangioma, and lymphangioma. The plunging ranula is an example of a mass that may be treated with sublingual gland excision and mere expression of the saliva from the plunging component of the lesion without direct removal of the neck mass. Sialadenitis and sialolithiasis of the submandibular gland are often treated with submandibular gland excision as
definitive treatment. When an FNAB and sophisticated histologic workup is inconclusive in a patient suspected of having a diagnosis of lymphoma, excision of the neck mass (lymph node) may be performed to provide more tissue for a lymphoma workup.

Extended Excision

Extended excision represents surgical removal of the mass with surrounding tissue. The best example is a benign or malignant tumor of the tail of the parotid gland that is excised either by a superficial or total parotidectomy. Imaging studies will dictate which is performed, and FNAB will often dictate whether a neck dissection should be performed along with the parotid surgery. The management of a submandibular gland tumor also represents an example of an extended excision and is performed identically to excision of a parotid tumor. A suspected thyroglossal duct cyst should be surgically excised with a Sistrunk procedure that includes the tract connecting the cyst to the base of the tongue as well as the associated hyoid bone.

Neck Dissection

Neck dissection includes a number of variations performed according to the diagnosis suggested by the history, physical examination, imaging studies, and FNAB result. Neck dissections are performed for treatment of demonstrated or suspected disease in cervical lymph nodes. For example, the patient with a neck mass thought to represent metastatic squamous cell carcinoma by history, physical examination, and possibly FNAB is generally treated with a comprehensive neck dissection. The type I modified radical neck dissection is the most commonly performed comprehensive neck dissection. This is true regardless of whether the cervical adenopathy represents an unknown primary or a metastatic focus from a primary oral cavity squamous cell carcinoma. A patient with a mass in the thyroid will undergo partial or total thyroidectomy with frozen sections, after which a decision is made regarding the remaining lobe of the thyroid in the case of a partial thyroidectomy. A neck dissection may not be indicated in these patients in the absence of demonstrable cervical adenopathy by physical examination, imaging studies, or FNAB. However, when cervical adenopathy exists based on the latter parameters, the patient with thyroid cancer should
undergo a neck dissection.
Appendix

Prevention of Infective Endocarditis Guidelines from the American Heart Association

Patients who have taken prophylactic antibiotics routinely in the past but no longer need coverage include those with:

- mitral value prolapse
- rheumatic heart disease
- bicuspid valve disease
- calcified aortic stenosis
- congenital heart conditions such as ventricular septal defect, aeral septal defect, and hypertrophic cardiomyopathy.

Cardiac conditions associated with the highest risk of adverse outcome from endocarditis for which prophylaxis with dental procedures is still recommended:

- artifical heart valves
- a history of having had infectious endocarditis
- certain specific, serious congenital heart conditions, including
  —unrepaired or incompletely repaired cyanotic congenital heart disease, including those with palliative shunts and conduits;
  —a completely repaired congenital heart defect with prosthetic material or device; whether placed by surgery or by catheter intervention, during the first six months after the procedure; or
  —any repaired congenital heart defect with residual defect at the site or adjacent to the site of a prosthetic patch or prosthetic device.

- A cardiac transplant that develops a problem in a heart valve.

Patients with congenital heart disease can have complicated circumstances.
The practitioner should check with the patient’s cardiologist if there is any question as to the category that best fits the patient’s needs.

*Antibiotic prophylactic reimens*¹
<table>
<thead>
<tr>
<th>Situation</th>
<th>Antibiotic†</th>
<th>Regimen‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standard prophylaxis</td>
<td>Amoxicillin</td>
<td>Adults, 2.0 g; children, 50 mg/kg orally 1 h before procedure</td>
</tr>
<tr>
<td>Cannot use oral medications</td>
<td>Ampicillin</td>
<td>Adults, 2.0 g IM§ or IV§; children, 50 mg/kg IM or IV within 30 min before procedure</td>
</tr>
<tr>
<td>Allergic to penicillin</td>
<td>Clindamycin</td>
<td>Adults, 600 mg; children, 50 mg/kg orally 1 h before procedure</td>
</tr>
<tr>
<td></td>
<td>Cephalaxin or cefadroxil</td>
<td>Adults, 2.0 g; children, 50 mg/kg orally 1 h before procedure</td>
</tr>
<tr>
<td></td>
<td>Azithromycin or clarithromycin</td>
<td>Adults, 500 mg; children, 15 mg/kg orally 1 h before procedure</td>
</tr>
<tr>
<td>Allergic to penicillin and unable to take oral medications</td>
<td>Clindamycin</td>
<td>Adults, 600 mg; children, 15 mg/kg IV 1 h before procedure</td>
</tr>
<tr>
<td></td>
<td>Cefazolin</td>
<td>Adults, 1.0 g; children, 25 mg/kg IM or IV within 30 min before procedure</td>
</tr>
</tbody>
</table>


‡Cephalosporins should not be used in patients with immediate-type hypersensitivity reaction (urticaria, angioedema, or anaphylaxis) to penicillins.

§Total children’s dose should not exceed adult dose.

IM: Intramuscular; IV: Intravenous.
FDA pregnancy categories

A—Controlled studies in pregnant women fail to demonstrate a risk to the fetus in the first trimester with no evidence of risk in later trimesters. The possibility of fetal harm appears remote.
B—Either animal-reproduction studies have not demonstrated a fetal risk but there are no controlled studies in pregnant women, or animal-reproduction studies have shown an adverse effect (other than a decrease in fertility) that was not confirmed in controlled studies in women in the first trimester and there is no evidence of a risk in later trimesters.
C—Either studies in animals have revealed adverse effects on the fetus (teratogenic or embryocidal effects or other) and there are no controlled studies in women, or studies in women and animals are not available. Drugs should be given only if the potential benefits justify the potential risk to the fetus.
D—There is positive evidence of human fetal risk, but the benefits from use in pregnant women may be acceptable despite the risk (eg, if the drug is needed in a life-threatening situation or for a serious disease for which safer drugs cannot be used or are ineffective).
X—Studies in animals or human beings have demonstrated fetal abnormalities or there is evidence of fetal risk based on human experience, or both, and the risk of the use of the drug in pregnant women clearly outweighs any possible benefit. The drug is contraindicated in women who are or may become pregnant.

Note: The notation, eg, C/D/X indicates the FDA pregnancy categorization of the parent drug and its major metabolites.

DEA schedule of controlled substances

Schedule I (C-I)—The drugs and other substances in this schedule have no legal medical uses except research. They have a high potential for abuse. They include selected opiates such as heroin, opium derivatives, and hallucinogens.

Schedule II (C-II)—The drugs and other substances in this schedule have legal medical uses and a high abuse potential which may lead to severe
dependence. They include former “Class A” narcotics, amphetamines, barbiturates, and other drugs.

Schedule III (C-III)—The drugs and other substances in this schedule have legal medical uses and a lesser degree of abuse potential which may lead to moderate dependence. They include former “Class B” narcotics and other drugs.

Schedule IV (C-IV)—The drugs and other substances in this schedule have legal medical uses and low abuse potential which may lead to moderate dependence. They include barbiturates, benzodiazepines, propoxyphenes, and other drugs.

Schedule V (C-V)—The drugs and other substances in this schedule have legal medical uses and low abuse potential which may lead to moderate dependence. They include narcotic cough preparations, diarrhea preparations, and other drugs.

Note: These are federal classifications. Your individual state may place a substance into a more restricted category. When this occurs, the more restricted category applies. Consult your state law.

**Conversion Chart**

**Linear Measures**

millimeters × 0.04 = inches

centimeters × 0.4 = inches

meters × 3.3 = feet

**Weight**

grams × 0.035 = ounces

kilograms × 2.2 = pounds

**Volume**

milliliters × 0.03 = fluid ounces

liters × 2.1 = pints
liters × 1.06 = quarts

teaspoons × 5 = milliliters

tablespoons × 15 = milliliters

Temperature

Centigrade (C°) to Fahrenheit (F°) - \( \frac{9}{5} \) (C°) + 32

Fahrenheit (F°) to Centigrade (C°) - \( \frac{5}{9} \) (F°) - 32
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Tests. See Laboratory testing.
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Tetracyclines
Thorax
Three cell differential count
Throat culture
Thrombin test
Thrombin time
Thrombocytopenia
Thrombocytosis
Thrombotic thrombocytopenic purpura
Thyroid gland
disease of
hyperthyroidism
hypothyroidism
physical examination
Ticarcillin
Ticlopidine
TMJ. See Temporomandibular joint.
Tomography. See Computed tomography.
Tooth extraction
Total body water
Total protein levels
Tracheotomy
Transfusion. See Blood transfusions.
Transient ischemic attacks
Transverse dental arch
Trauma
maxillofacial. See Maxillofacial trauma.
mucosal ulcers caused by
temporomandibular joint
Traumatic arthritis
Traumatic fibroma
Traumatic neuroma
Treacher Collins syndrome. See Mandibulofacial dysostosis.
Treponema pallidum
Triceps skin fold
Trigeminal nerve injury
Trigeminal neuropathy
Trimethoprim/sulfamethoxazole
“Triple-rule-out” chest computed tomography
Trismus
TSF. See Triceps skin fold.
T-tube cholangiogram
Tube placement
chest
nasogastric
Tuberculosis
Tuberculous cervical lymphadenitis
Tumors. See also Cyst(s).
- biopsy of
- complications
- differential diagnosis of
- imaging of
- intraoperative care
- nonodontogenic
- odontogenic
- postoperative care

U

Ultrasound
Upper gastrointestinal series
Urethritis
Uric acid
Urinalysis
Urinary tract
- catheterization of
- general evaluation of
Urinary tract infection
- common pathogens of
- fever and
Urine
- culturing of
- electrolyte levels
- laboratory testing of. See Urinalysis.
- normal volume
Urine output, low
Urticaria

V
Valacyclovir
Valium. See Diazepam.
Valvular heart disease
Vancomycin
Varicella-zoster virus
Vascular access, intraosseous
Vascular system
Venereal Disease Research Laboratories test
Venous cannulation
  central
  peripheral
Venous cutdown
Ventilation perfusion mismatch
Ventilation/perfusion lung scan
Ventilator-associated pneumonia
Ventricles, of heart
  hypertrophy
  premature contractions
Ventricular dysrhythmias
Ventricular fibrillation
Ventricular standstill. See Asystole.
Ventricular tachycardia
Verapamil
Vesiculoulcerative lesions
Virtual colonoscopy
Visual fields
Vitamin K
Vitamins
  fat soluble
  water soluble
Voice sounds
Voiding cystourethrogram
Vomiting
von Willebrand disease

Warfarin
Warthin tumor
Water soluble vitamins
Weber test
Wenckebach phenomenon
Wharton duct
White blood cells
White oral lesions
White sponge nevus
Whole blood
  changes during storage
  transfusion of
Wounds
  care of
  from cysts and tumors
dehiscence of
  from cysts and tumors
  from dentoalveolar surgery
description of
  fever and
  postoperative
  puncture
Wright stain technique

X

Xerostomia

Z

Zoledronate
Zygoma fractures
Zygomatic arch fractures