22 - Injuries; Poisoning

Chapter 321. Approach to the Trauma Patient

Injury is the number one cause of death for people aged 1 to 44. In the US, there were 175,000 trauma deaths in 2006, about two thirds being accidental. Of intentional injury deaths, about 60% were due to self-harm. In addition to deaths, injury results in about 40 million emergency department visits annually.

Patients whose injuries are serious but not immediately fatal benefit the most from treatment in designated trauma centers, hospitals that have special staffing and protocols to provide immediate care to critically injured patients. Criteria for such designation (and for the necessity of transport to them) vary by state but usually follow guidelines of the American College of Surgeons' Committee on Trauma.

Many traumatic injuries are discussed elsewhere in THE MANUAL; for bone and joint injuries, see p. 3201; for spinal cord injury, see p. 3227; for head injury, see p. 3218; for facial injury, see p. 3231; for eye injury, see p. 3235; for genitourinary injury, see p. 3238; for lacerations, see p. 3193.

Etiology

Of the myriad ways people are injured, most can be categorized as blunt or penetrating. Blunt injury involves a forceful impact (eg, blow, kick, strike with object, fall, motor vehicle crash, blast). Penetrating injury involves breach of the skin by an object (eg, knife, broken glass) or projectile (eg, bullet, shrapnel from an explosion).

Other injury types include thermal and chemical burns, toxic inhalations or ingestions, and radiation injury.

Pathophysiology

All injuries, by definition, cause *direct* tissue damage, the nature and extent depending on the anatomic site, mechanism, and intensity of trauma. Severe direct tissue damage to critical organs (eg, to the heart, brain, spinal cord) is responsible for most immediate trauma deaths.

Additionally, patients surviving the initial insult may develop *indirect* injury effects. Disruption of blood vessels causes hemorrhage, which may be external (and hence visible) or internal, either confined within an organ as a contusion or hematoma, or as free hemorrhage into a body compartment (eg, peritoneal cavity, thorax). Small amounts of hemorrhage (ie, < 10% of blood volume) are tolerated well by most patients. Larger amounts cause progressive declines in BP and organ perfusion (shock—see p. 2292), leading to cellular dysfunction, organ failure, and eventually death. Hemorrhagic shock causes most short-term (ie, within hours) deaths, and multiple organ failure from prolonged shock causes many of the near-term (ie, first 14 days) deaths. Additional near-term deaths result from infection because of disruption of normal anatomic barriers and immune system dysfunction.

Evaluation and Treatment

- A, B, C, D, E evaluation and stabilization of *A*irway, *B*reathing, *C*irculation, *D*isability (neurologic status), and *E*xposure/*e*nvironmental control

- After stabilization, head-to-toe examination

- Liberal use of CT and other imaging studies

Care in the emergency department rather than emergency care delivered at the accident site is discussed here. Evaluation and treatment are done simultaneously, beginning with systems that pose the most immediate threat to life if damaged. *Attending to dramatic but not deadly injuries (eg, open lower-extremity fracture, finger amputations) before evaluating immediate life threats can be a fatal mistake.* A helpful mnemonic is A, B, C, D, E. Systems are rapidly examined for serious abnormalities (primary survey); a more detailed examination (secondary survey) is done after the patient is stable.

Airway: Airway patency is threatened by blood clots, teeth, or foreign bodies in the oropharynx; soft-tissue laxity and posterior retraction of the tongue caused by obtundation (eg, from head injury, shock, intoxication); and edema or hematoma due to direct neck trauma. These obstructions are readily visible on direct inspection of the mouth or neck; having the patient speak can rapidly confirm that the airway is not likely in immediate danger.

Blood and foreign material are removed by suction or manually. Obtunded patients whose airway patency is in doubt and patients with significant oropharyngeal injury require endotracheal intubation; usually drugs are given for paralysis and sedation before intubation is done (see p. 2273). If patients require an artificial airway and endotracheal intubation is not possible (eg, due to edema of the airway caused by a thermal burn) or contraindicated (eg, due to severe maxillofacial injury), surgical cricothyrotomy is indicated (see p. 2277). NOTE: When evaluating or manipulating a patient's airway, cervical spine immobilization should be maintained (eg, by rigid collar, inline immobilization techniques) until cervical spine injury has been excluded by examination, imaging, or both.

Breathing: Adequate ventilation is threatened by decreased central respiratory drive (usually from head injury, intoxication, or nearly fatal shock) or by chest injury (eg, hemothorax or pneumothorax, multiple rib fractures, pulmonary contusion).

Adequacy of air exchange is usually apparent on auscultation. Tension pneumothorax (see p. 2002) may cause the trachea to deviate to the side opposite the injury, as well as decreased breath sounds and sometimes distended neck veins. The chest wall is fully exposed to look for ample chest wall expansion and is palpated for obvious rib fractures and presence of subcutaneous air (sometimes the only finding in pneumothorax).

Pneumothorax is decompressed by chest tube (see p. 2003) and must be excluded before initiating positive-pressure ventilation (which may markedly enlarge a pneumothorax and convert it to a tension pneumothorax). Suspected tension pneumothorax can be decompressed with needle thoracostomy (eg, a 14-gauge needle inserted in the midclavicular line, 2nd intercostal space) to stabilize the patient if a chest tube cannot be inserted immediately. Inadequate ventilation is treated with endotracheal intubation and mechanical ventilation.

Circulation: Significant external hemorrhage can occur from any major vessel but is always apparent. Life-threatening internal hemorrhage is often less obvious. However, this volume of hemorrhage can occur in only a few body compartments: the chest, abdomen, and soft tissues of the pelvis or thigh (eg, from a pelvic or femoral fracture).

Pulse and BP are assessed, and signs of shock are noted (eg, tachypnea, dusky color, diaphoresis, altered mental status). Abdominal distention and tenderness, pelvis instability, and thigh deformity and instability are often present when internal hemorrhage in those areas is large enough to be life threatening.

External hemorrhage is controlled by direct pressure. Two large-bore (eg, 14- or 16-gauge) IVs are started with 0.9% saline or Ringer's lactate; rapid infusion of 1 to 2 L (20 mL/kg for children) is given for signs of shock and hypovolemia. Subsequently, additional fluids and, if necessary, blood component therapy is given as indicated (see p. 2296). Patients in whom there is strong clinical suspicion of serious intra-abdominal hemorrhage may require immediate laparotomy. Patients with massive intrathoracic hemorrhage may require immediate thoracotomy and possibly autotransfusion of blood recovered via tube thoracostomy.

Disability: Neurologic function is evaluated for serious deficits involving the brain and spinal cord. The Glasgow Coma Scale (GCS—see Table 324-2 on p. 3221 and, for infants and children, see Table 324-3 on p. 3222) and pupillary response to light are used to screen for serious intracranial injury. Gross motor movement and sensation in each extremity are evaluated to screen for serious spinal cord injury. The cervical spine is palpated for tenderness and deformity and stabilized in a rigid collar until cervical spine injury is excluded. With careful manual stabilization of the head and neck, the patient is

logrolled onto a side to allow palpation of the thoracic and lumbar spine, inspection of the back, and rectal examination to check tone (decreased tone indicates possible spinal cord injury), the prostate (a high-riding prostate suggests urethral injury), and presence of blood. In the US, most patients arriving by ambulance are immobilized on a long, rigid board for ease of transport and to stabilize possible spinal fractures. If examination reveals no sign of spinal injury, patients are taken off the board because it is quite uncomfortable and pressure ulcers may occur within a few hours.

Patients with severe traumatic brain injury (GCS < 9) require endotracheal intubation, neurosurgical evaluation, and therapy to prevent secondary brain injury (eg, osmotic diuresis, sometimes hyperventilation for patients with signs of impending brain herniation—see Fig. 174-1 on p. 1657).

Exposure/environmental control: To ensure injuries are not missed, patients are completely undressed (by cutting off garments) and the entire body surface is examined for signs of occult trauma. The patient is kept warm (eg, with heated blankets and by using only warmed IV fluids) to prevent hypothermia.

Secondary survey: After immediate life threats are assessed and the patient is stable, a more thorough evaluation is done, and a focused history is obtained. If only limited conversation is possible, an "AMPLE" history covers essential information:

• *A*llergies

• *M*edications

• *P*ast medical history

• *L*ast meal

• *E*vents of the injury

After the patient is completely undressed, the examination generally proceeds from head to toe; it includes all orifices and a more detailed look at areas examined in the initial survey. All soft tissues are inspected for lesions and swelling, all bones are palpated for tenderness, and range of motion is assessed in joints (unless there is obvious fracture or deformity).

A urinary catheter is usually placed in seriously injured and obtunded patients provided there is no evidence of urethral injury (eg, blood at the meatus, ecchymosis of the perineum, high-riding prostate). Seriously injured patients often also have a nasogastric tube placed, provided there is no serious midface trauma (rare reports exist of intracranial tube insertion through a cribriform plate fracture).

Open wounds are covered with sterile dressings, but cleansing and repair are deferred until completion of evaluation and treatment of more serious injuries. Serious clinically apparent dislocations with marked deformity or neurovascular compromise are imaged and reduced as soon as immediate life threats have been addressed. Obvious or suspected fractures are splinted pending full assessment of serious injuries and appropriate imaging studies.

Testing: Imaging tests are the cornerstone; laboratory tests are generally ancillary. Patients with penetrating trauma typically have focal injuries that can limit necessary imaging to the obviously involved region or regions. Blunt trauma, particularly when significant deceleration is involved (eg, serious fall, motor vehicle crash), can affect any part of the body, and imaging is used more liberally. Such patients traditionally have x-rays of the chest, cervical spine, and pelvis unless they are awake and alert, completely lacking in symptoms or findings suggesting injury to those areas, and have no distracting injuries (eg, femur fracture) that might keep them from complaining about injuries elsewhere. These imaging tests are directed at life threats that may not be clinically obvious.

Chest x-ray can identify airway disruption, lung injury, and pneumothorax and can suggest thoracic aorta tears (eg, by mediastinal widening).

CT of the chest, abdomen and pelvis, spine, head, or, particularly, combinations of these is increasingly being used instead of plain x-rays for patients who require imaging after severe multiple blunt trauma.

Identification of intra-abdominal injury is essential. Bedside ultrasonography (FAST examination: *f*ocused *a*ssessment with *s*onography in *t*rauma) is being used increasingly, particularly for unstable patients; it is sensitive for significant volumes of intraperitoneal blood and thus the need for immediate laparotomy. If patients are stable, CT has the advantages of high accuracy, imaging of the retroperitoneal structures and bones, and showing the volume and sometimes the origin of hemorrhage. For unstable patients in whom bedside ultrasonography is not feasible, diagnostic peritoneal aspiration can be used, in which a peritoneal dialysis catheter is inserted through the abdominal wall into the peritoneal cavity. If > 10 mL of blood is aspirated, immediate laparotomy is indicated.

Head CT is typically done in patients with altered mental status or focal neurologic abnormalities and in patients who sustained loss of consciousness (some clinicians feel that patients with a brief loss of consciousness who are completely alert and neurologically intact do not require CT). Imaging is obtained more liberally in children < 2 yr with scalp hematoma, the elderly, patients taking anticoagulants, and patients who are alcoholics.

Aortic injury should be considered in patients with severe deceleration chest injury or suggestive signs (eg, pulse deficits or asymmetric BP measurements, end-organ ischemia, suggestive findings on chest x-ray); these patients may require CT angiography or other aortic imaging. All patients suspected of having significant blunt chest injury have an ECG to diagnose myocardial injury and cardiac monitoring for subsequent arrhythmias. Patients with abnormalities on ECG usually have blood levels of cardiac markers measured and sometimes echocardiography (see p. 2053).

Plain x-rays are obtained of any suspected fractures and dislocations. Other imaging tests are obtained for specific indications (eg, angiography to diagnose and sometimes embolize vascular injury; CT to better delineate spinal, pelvic, or complex joint fractures).

Laboratory tests that may be useful include ABGs for PO_2, PCO_2, and base deficit; urine examination for blood; CBC to establish a baseline to monitor ongoing hemorrhage; glucose to evaluate for hypoglycemia; and type and cross-match for possible blood transfusion. Measures of perfusion (serum lactate, base deficit on ABG measurement, and, in patients with a catheterized central vein, central venous O_2 saturation) may help identify early or partially treated shock. Other reflexively obtained tests (eg, electrolytes and other chemistries, coagulation studies) are unlikely to be helpful unless suggested by relevant medical history (eg, renal insufficiency, diuretic use). Toxicology screening (eg, blood alcohol, urine drug screen) is often done; results of this testing rarely change immediate management but can help identify substance abuse causative of injury, allowing intervention to prevent subsequent trauma.

Chapter 322. Lacerations

Introduction

Care of lacerations enables prompt healing, minimizes risk of infection, and optimizes cosmetic result.

Physiology

Healing begins immediately after injury with coagulation and introduction of WBCs; neutrophils and macrophages remove debris (including devitalized tissue) and bacteria. Macrophages also encourage fibroblast replication and neovascularization. Fibroblasts deposit collagen, typically beginning within 48 h and reaching a maximum in about 7 days. Collagen deposition is essentially complete in 1 mo, but collagen fiber strength builds more slowly as fibers undergo crosslinking. Wound tensile strength is only about 20% of ultimate by 3 wk, 60% by 4 mo, and maximum at 1 yr; strength never becomes equivalent to the undamaged state.

Epithelial cells from the wound edge migrate across the wound shortly after injury. In a surgically repaired wound (healing by primary intention), they form an effective protective barrier to water and bacteria in 12 to 24 h and resemble normal epidermis within 5 days. In a wound that is not repaired (ie, heals by secondary intention), epithelialization is prolonged proportionally to the defect size.

There are static forces on the skin because of its natural elasticity and the underlying muscles (see Fig. 322-1). Because scar tissue is not as strong as adjacent undamaged skin, these forces tend to widen scars, sometimes resulting in a cosmetically unacceptable appearance after apparently adequate wound closure. Scar widening is particularly likely when the forces are perpendicular to the wound edge. This tendency (and resultant wound stress) is readily observed in the fresh wound; gaping edges indicate perpendicular tension, and relatively well approximated edges indicate parallel forces.

Scars tend to be red and prominent for about 8 wk. As collagen remodeling occurs, the scar becomes thinner and loses its erythema. In some patients, however, the scar hypertrophies, becoming unsightly and raised. Keloids are exuberant scars that extend beyond the limits of the original wound (see p. 743).

The most common factors that interfere with wound healing involve tissue ischemia, infection, or both (see Table 322-1); tissue ischemia predisposes to infection.

[**Fig. 322-1.** Representative minimal skin tension lines.]

Lower extremities are usually at greatest risk of poor healing due to impaired circulation. The scalp and face are at lowest risk. Certain drugs and disorders can also interfere with wound healing.

Bite wounds (see p. 3306) are usually heavily contaminated.

Evaluation

Sequential steps in evaluation include the following:

• Finding and treating serious injuries

• Obtaining hemostasis

• Looking for damage to underlying structures

Clinicians must find and treat serious injuries (see p. 3190) before focusing on skin lacerations, however dramatic. Actively bleeding wounds require hemostasis before evaluation. Hemostasis is best obtained by direct pressure and, when possible, elevation; clamping bleeding vessels with instruments is generally avoided because of the possibility of damaging adjacent nerves. Use of topical anesthetics containing epinephrine may also help reduce bleeding. Wound evaluation also requires good lighting. Magnification

(eg, with magnifying glasses) can help, particularly for examiners with imperfect near-vision.

[Table 322-1. Factors that Interfere with Wound Healing]

Full wound evaluation may require probing or manipulation, and thus local anesthesia, but sensory examination should precede administration of a local anesthetic.

The wound is evaluated for damage to underlying structures, including nerves, tendons, vessels, joints, and bones, as well as the presence of foreign bodies or body cavity penetration (eg, peritoneum, thorax). Failure to recognize these complications is one of the most significant errors in wound management.

Nerve injury is suggested by sensory abnormality distal to the wound; suspicion is increased for lacerations near the course of significant nerves. Examination should test light touch and motor function. Two-point discrimination is useful for hand and finger injuries; the clinician touches the skin with 2 ends of a bent paper clip simultaneously to determine the minimum separation that allows perception of 2 points (usually 2 to 3 mm). Normal varies among patients and by location on the hand; the identical site on the uninjured side is the best control.

Tendon injury is suspected in any laceration over the course of a tendon. Complete tendon laceration usually causes a resting deformity (eg, foot drop from Achilles tendon laceration, loss of normal resting finger flexion with digital flexor laceration) because forces from antagonist muscles are unopposed. Resting deformity does not occur with partial tendon laceration, which may manifest with only pain or relative weakness on strength testing or be discovered only on exploration of the wound. The injured area should be examined through the full range of motion; the injured tendon may sometimes retract and not be visible on inspection or wound exploration when the injured area is in the resting position.

Vascular injury is suggested by signs of ischemia, such as pallor, decreased pulses, or perhaps delayed capillary refill distal to the laceration (all compared with the uninjured side). Vascular injury is occasionally suspected in the absence of ischemia when a laceration traverses the territory of a major artery and is deep or complex or results from penetrating trauma. Other signs of vascular injury can include a rapidly expanding or pulsatile mass or a bruit.

Bone injury is possible, particularly after penetrating trauma or when injury occurs over a bony prominence. If the mechanism or location of injury is concerning, plain x-rays are taken to rule out fracture.

Foreign bodies are sometimes present in wounds, depending on the mechanism. Wounds involving glass are likely to have foreign bodies, lacerations from sharp metal rarely do, and wounds involving other substances are of intermediate risk. Although not very sensitive, a patient's complaint of feeling a foreign body is fairly specific and should not be ignored. Localized pain or tenderness in a high-risk wound also is suggestive, particularly if pain worsens with active or passive motion. Wound examination and exploration are not sensitive for small foreign bodies unless the wound is superficial and its full depth is visible. Imaging studies are recommended for all wounds involving glass and for other wounds if a foreign body is suspected because of the mechanism, the symptoms, or an inability to examine the wound's full depth. If glass or inorganic material (eg, stones, metal fragments) is involved, plain x-rays are taken; glass bits as small as 1 mm are usually visible. Organic materials (eg, wood splinters, plastic) are rarely detected with plain x-rays (although the outline of larger objects may be visible because of their displacement of normal tissue); various other modalities have been used, including xerography, ultrasonography, CT, and MRI. None of these is 100% sensitive, but CT may offer the best balance between accuracy and practicality. A high index of suspicion and careful exploration of all wounds are always appropriate.

Joint penetration should be suspected when wounds near a joint are deep or involve penetrating trauma.

Penetration of the abdominal or thoracic cavity should be considered in any wound over those locations in which the bottom of the laceration is not clearly visible. Wounds should not be blindly probed; blind probing is unreliable and may cause further injury. Patients with suspected thoracic lacerations require a chest x-ray initially, with a repeat film after 4 to 6 h of observation; any slowly developing

pneumothorax should be visible by that time. In patients with abdominal lacerations, local anesthesia facilitates exploration (lacerations can be extended horizontally if necessary). Patients with wounds penetrating the fascia should be observed in the hospital; sometimes abdominal CT is used to identify hemoperitoneum.

Treatment

Treatment involves

• Cleansing and local anesthesia (sequence can vary)

• Exploration

• Debridement

• Closure

Tissue should be handled as gently as possible.

Cleansing: Both the wound and the surrounding skin are cleaned. Subepidermal tissue in the wound is relatively delicate and should not be exposed to harsh substances (eg, full-strength povidone iodine, chlorhexidine, hydrogen peroxide) and vigorous scrubbing.

Removing hair from laceration edges is not necessary for wound hygiene but can make markedly hairy areas (eg, scalp) easier to work on. If necessary, hair is removed by clipping with scissors, not shaving; razors create microtrauma, allowing skin pathogens to enter and increasing risk of infection. Hair is clipped before wound irrigation so that any clipped hair entering the wound is removed. Eyebrows are never trimmed because the hair-skin border is needed for proper alignment of wound edges. Furthermore, eyebrows may grow back abnormally or not at all.

Although wound cleansing is not particularly painful, local anesthesia is usually administered first, except for heavily contaminated wounds; these wounds are best initially cleansed with running tap water and mild soap before a local anesthetic is administered. Tap water is clean and free of typical wound pathogens, and, used in this manner, does not seem to increase risk of infection. Wounds are then cleansed by a high-velocity stream of liquid and sometimes scrubbed with a fine-pore sponge; brushes and rough materials are avoided. An appropriate irrigation stream can be created using a 20-, 35-, or 50-mL syringe with a 20-gauge needle or IV catheter; commercially available devices incorporating a splash guard help limit splatter. Sterile 0.9% saline is an effective irrigant; specialized surfactant irrigants are costly and of doubtful additional benefit. If bacterial contamination is of particular concern (eg, bites, old wounds, organic debris), povidone iodine solution diluted 1:10 in 0.9% saline may be beneficial and is not harmful to tissues at this concentration. The volume necessary varies. Irrigation continues until visible contamination is removed and at least 100 to 300 mL has been applied (more for large wounds).

Painting the skin with povidone iodine before suturing may reduce skin flora, but the substance should not be introduced into the wound.

Local anesthesia: Generally, injectable local anesthetics are used. Topical anesthetics are beneficial in certain cases, especially for wounds of the face and scalp and when topical skin adhesives are used to close wounds.

Common injectable agents are lidocaine 0.5%, 1%, and 2%, and bupivacaine 0.25% and 0.5%, both from the amide group of local anesthetics; the ester group includes procaine, tetracaine, and benzocaine. Lidocaine is most commonly used. Bupivacaine has a slightly slower onset (several minutes vs almost immediate) and a significantly longer duration (2 to 4 h vs 30 to 60 min). Duration of action of both can be prolonged by adding epinephrine 1:100,000, a vasoconstrictor. Because vasoconstriction may impair wound vascularity (and thus defenses), epinephrine is mostly used for wounds in highly vascular areas (eg, face, scalp). Although traditional teaching has been to avoid using epinephrine in distal parts (eg, nose, ears, fingers, penis) to prevent tissue ischemia, complications from use on distal parts are rare, and

such use is now considered safe. Epinephrine can be particularly helpful in achieving hemostasis in wounds that are bleeding heavily.

The maximum dose of lidocaine is 3 to 5 mg/kg (1% solution = 1 g/100 mL = 10 mg/mL), and that of bupivacaine is 2.5 mg/kg. Addition of epinephrine increases the allowable dose of lidocaine to 7 mg/kg, and of bupivacaine to 3.5 mg/kg.

Adverse reactions to local anesthetics include allergic reactions (hives and, occasionally, anaphylaxis —see p. 1120) and sympathomimetic effects from epinephrine (eg, palpitations, tachycardia). True allergic reaction is rare, particularly to amide anesthetics; many patient-reported events represent anxiety or vagal reactions. Furthermore, allergic reactions are often due to methylparaben, the preservative used in multidose vials of anesthetic. If the offending agent can be identified, a drug from another class (eg, ester instead of amide) can be used. Otherwise, a test dose of 0.1 mL preservative-free (single-dose vial) lidocaine can be given intradermally; if there is no reaction within 30 min, that anesthetic can be used.

Techniques recommended to minimize the pain of injection include the following:

- Using a small needle (a 27-gauge needle is best, and a 25-gauge is acceptable; a 30-gauge may be too flimsy)
- Giving the injection slowly
- Giving the injection into the subcutaneous plane instead of intradermally
- Buffering lidocaine with 1 mL of $NaHCO_3$ (concentration from 4.2 to 7.4%) for every 9 to 10 mL of lidocaine solution (NOTE: Buffering decreases the shelf life of multidose lidocaine vials, and buffering is ineffective for bupivacaine.)
- Warming the anesthetic solution to body temperature

Local or occasionally regional nerve blocks are sometimes preferred to wound injection. Nerve blocks cause less distortion of wound edges by injected anesthetic; this decreased distortion is important when alignment of wound edges must be particularly precise (eg, infraorbital nerve block for lacerations through the vermilion border of the lip) or when wound injection would be difficult because the space for injection is small (eg, digital nerve block for finger lacerations). Also, large areas can be anesthetized without using toxic doses of anesthetic. Slight disadvantages of nerve blocks are slower onset of anesthesia and sometimes < 100% effectiveness with the first injection.

Use of **topical anesthesia** makes injection unnecessary and is completely painless—factors particularly desirable in children and fearful adults. The most common solution is LET, which consists of lidocaine 2 to 4%, epinephrine 1:2000, and tetracaine 0.5 to 2%. A cotton dental pledget (or cotton ball) the length of the wound soaked in several milliliters of the solution and placed within the wound for 30 min usually provides adequate anesthesia; sometimes supplemental injectable anesthetic is required. If anesthesia is incomplete after application of a topical anesthetic, supplementary local anesthetic can be injected, usually with minimal pain.

Exploration: The full extent of the wound is explored to look for foreign material and possible tendon injury. Foreign material may also often be discerned by palpating gently with the tip of a blunt forceps, feeling for a discrete object and listening for a click characteristic of glass or metal foreign bodies. Occasionally, contaminated puncture wounds (eg, human bite wounds near the metacarpophalangeal joint) must be extended so that they can be adequately explored and cleansed. Deep wounds near a major artery should be explored in the operating room by a surgeon.

Debridement: Debridement uses a scalpel, scissors, or both to remove dead tissue, devitalized tissue (eg, tissue with a narrow base and no viable blood supply), and sometimes firmly adherent wound contaminants (eg, grease, paint). Macerated or ragged wound edges are excised; usually 1 to 2 mm is sufficient. Otherwise, debridement is not used to convert irregular wounds into straight lines. Sharply

beveled wound edges are sometimes trimmed so that they are perpendicular.

Closure: Decision to close a wound depends on the wound's location, age, cause, and degree of contamination and on patient risk factors.

Most wounds can be closed immediately (primary closure). Primary closure is usually appropriate for uninfected and relatively un-contaminated wounds < 6 to 8 h old (< 12 to 24 h for face and scalp wounds).

Many other wounds can be closed after several days (delayed primary closure). Delayed primary closure is appropriate for wounds too old for primary closure, particularly if signs of infection have begun to appear, and for wounds of any age with significant contamination, particularly if organic debris is involved. The threshold for using delayed primary closure is lowered for patients with risk factors for poor healing. At initial presentation, anesthesia, exploration, and debridement are done at least as thoroughly as for other wounds, but the wound is loosely packed with moist gauze. The dressing is changed at least daily and evaluated for closure after 3 to 5 days. If there are no signs of infection, the laceration is closed by standard techniques. Loosely closing such wounds initially is ineffective and inappropriate because the wound edges nonetheless seal shut within 12 to 24 h.

Some wounds should not be closed. These wounds include the following:

• Cat bites

• Small bites to hands or feet (see also p. 3307)

• Puncture wounds

• High-velocity missile wounds

Materials and methods: Traditionally, sutures have been used for laceration repair, but metal staples, adhesive strips, and liquid topical skin adhesives are now used for certain wounds, mainly linear lacerations subject to only small amounts of tension. Whatever the material used, preliminary wound care is the same; a common error is to do cursory exploration and no debridement because a noninvasive closure not requiring local anesthesia is planned.

Staples are quick and easy to apply and, because there is minimal foreign material in the skin, are less likely to cause infection than sutures. However, they are suited mainly for straight, smooth cuts with perpendicular edges in areas of low skin tension. Improper wound edge apposition (sometimes causing wound edges to overlap) is the most common error.

Topical skin adhesives usually contain octylcyanoacrylate, butylcyanoacrylate, or both. They harden within a minute; are strong, nontoxic, and waterproof; form a microbial barrier; and have some antibacterial properties. However, adhesive should not be allowed into the wound. Infections are very unlikely, and cosmetic results are generally good. Adhesive is best for simple, regular lacerations; it should not be used for wounds under tension unless tension is relieved with deep dermal sutures, immobilization, or both. In wounds requiring debridement, deep dermal suturing, or exploration under local anesthesia, the advantages of decreased pain and time are minimized. However, patients do not require follow up for suture or staple removal. With long lacerations, skin edges can be held together by a 2nd person or with skin tapes while the adhesive is applied. One or 2 layers are applied as recommended by the manufacturer. The adhesive sloughs spontaneously in about a week. Excess or inadvertently applied adhesive can be removed with any petrolatum-based ointment or, in areas away from the eyes or open wounds, acetone.

Adhesive strips are probably the quickest repair method and have a very low infection rate. They are useful for wounds not subject to tension. Use on lax tissue (eg, dorsum of hand) is difficult because edges tend to invert. Adhesive strips cannot be used on hairy areas. Adhesive strips are particularly advantageous for lacerations in an extremity that is to be casted (thus blocking appropriate suture removal). Adhesive strips can also be used to reinforce wounds after suture or staple removal. Skin must

be dry before application. Many clinicians apply tincture of benzoin to boost adhesion. Improper application may result in blister formation. Adhesive strips may be removed by the patient.

Sutures are the best choice for irregular or complex lacerations, areas of loose skin, areas under tension, and other wounds requiring deep dermal closure. Because sutures can serve as an entry site for bacteria and there is a significant amount of foreign material under the skin, they have the highest rate of infection. Suture materials can be monofilament or braided and absorbable or nonabsorbable. Characteristics and uses vary (see Table 322-2); generally, absorbable material is used for deep dermal sutures, and nonabsorbable material is used for cutaneous ones. Braided material generally has higher tissue reactivity and thus poses a slightly higher risk of infection than does monofilament but is soft and easy to handle and has good knot security.

Suture technique: General goals include the following:

• Closely approximating skin margins

• Everting wound edges

• Eliminating dead space

• Minimizing tension in the wound and of individual sutures

• Minimizing the amount of subcutaneous material

The relative importance of minimizing wound tension and minimizing the amount of material buried under the skin (eg, deep dermal sutures) vary by wound location. For example, in facial wounds, cosmetic result is very important and, because of the excellent vascular supply, infection risk is low. Thus, for gaping wounds, deep dermal sutures, which decrease wound tension and improve cosmetic result, are desired; infection risk is low even if they are used. In areas where vascular supply or cosmetic result is less important, deep dermal sutures are less desirable.

Sutures may be placed and tied individually (interrupted sutures) or be continuous

[**Table 322-2.** Suture Materials]

(running suture). They may be completely buried under the skin (subcuticular or deep dermal sutures) or enter and exit the skin to be tied externally (percutaneous sutures).

If the wound is gaping, deep dermal suturing tends to be used initially (see Fig. 322-2); the resultant narrow epidermal gap is then closed by percutaneous sutures. For wounds on the face, any gaping > 5 to 10 mm may benefit from deep dermal suturing (not used on nose and eyelids); in other body areas, a wider gap is acceptable. Interrupted sutures using size 4-0 or 5-0 (smaller numbers indicate thicker material) braided absorbable material (eg, polyglactic acid) are most common. They are placed with the knot at the bottom of the wound to avoid a palpable lump and must not be too tight. A running subcuticular suture is sometimes used, especially for cosmetic repairs.

[**Fig. 322-2.** Simple deep dermal suture.]

Epidermal closure is typically with simple, interrupted sutures (see Fig. 322-3) of nonabsorbable monofilament (eg, nylon, polypropylene). In areas over large joints and the scalp, size 3-0 or 4-0 sutures are used; size 6-0 sutures are used for repairing face wounds; in most other areas, size 4-0 or 5-0 sutures are used. Suture size can vary slightly depending on how much static and dynamic tension is predicted (eg, for facial lacerations subject to frequent movement or high tension, size 5-0 sutures may be used). Sutures are placed about as deep as they are wide and are spaced as far apart as the distance from the needle entry point to wound edge (see Fig. 322-4). Small bites (suture typically inserted 1 to 3 mm from the wound edge) are used for repairs in areas where cosmetic results are of particular concern and when tissues are thin. For other repairs, wider

bites are used, varying with the tissue thickness. Wound edges can be everted by making the width of the bite greater at the deepest part of the wound than at the surface. Eversion is more easily obtained when the skin is entered with the needle at a 90° angle and angled slightly away from the skin edge.

A vertical mattress suture (see Fig. 322-5) is sometimes used instead of a layered closure, provided skin tension is not marked; it also helps ensure proper edge eversion in loose tissue. A running suture (see Fig. 322-6) is quicker to place than interrupted sutures and can be used when wound edges are well aligned.

[**Fig. 322-3.** Simple cutaneous suture.]

[**Fig. 322-4.** Suture spacing.]

In all cases, epidermal closure must precisely realign edges horizontally using natural skin landmarks (eg, folds, creases, lip margins) when available. Vertical alignment is equally important to avoid a step-off deformity. Excess tension after closure is evidenced by indenting of the skin or a sausage link appearance. Such a repair should be redone, adding deep dermal sutures, additional percutaneous sutures, or both as needed. Adjustments to suture technique are needed to achieve optimal alignment when wound edges are beveled. For example, edges may be debrided or suture bite size may differ from one side of the wound to the other.

Aftercare: Tetanus immunization is given if necessary (see Table 140-1 on p. 1299).

[**Fig. 322-5.** Vertical mattress suture.]

[**Fig. 322-6.** Running suture.]

Topical antibiotic ointment is applied daily; it can reduce risk of infection and help maintain a moist wound environment that optimizes healing. However, ointment is not used over tissue adhesives or adhesive strips.

Prophylactic systemic antibiotics are not indicated except for the following cases:

• Bite wounds on the extremities (see p. 3307)

• Human bites

• Wounds involving tendons, bones, or joints

• Possibly intraoral lacerations

• Some heavily contaminated wounds

If deemed necessary, antibiotics are given as early as possible; the first dose may be given parenterally.

Wounds are immobilized because excess movement of the affected area may interfere with healing. Wounds near joints should be immobilized with splints. Bulky dressings are used to immobilize fingers and hands. Wounds should be elevated, above heart level when feasible, for the first 48 h after suturing. A sling may help maintain some degree of elevation of an upper extremity wound. Patients with distal lower extremity lacerations (other than minor) should probably stay off their feet for several days (eg, by using crutches); restrictions on walking probably result in better healing.

Wound care is meticulous. The wound is kept clean and dry; dressings that are nonadherent and impermeable to bacteria are usually applied. Antibiotic ointment is applied daily until the wound closure device is removed. A reliable patient may inspect minor, clean lacerations, but early physician examination is preferable for higher risk wounds and wounds in unreliable patients. After 12 h, well-healing wounds

can be cleansed gently of residual secretions with water, half-strength hydrogen peroxide, or soap and water. Brief wetting in the shower is safe, but prolonged soaking should be avoided.

Wound infection occurs in 2 to 5% of lacerations; steadily increasing pain ≥ 12 h after closure is often the earliest manifestation, and initial signs are redness more than about 0.5 cm from the wound edge, swelling, tenderness, and warmth. Later signs may include fever, purulent drainage, and ascending lymphangitis. Systemic antibiotics effective against skin flora are begun; a 1st-generation cephalosporin (eg, cephalexin 500 mg po qid) or, for intraoral infection, penicillin 500 mg po qid, is typically used. Infection beginning > 5 to 7 days after injury suggests retained foreign body.

Closure material (except for tissue adhesive) is removed after various intervals depending on location. For facial lacerations, sutures are removed in 3 to 5 days to prevent cross-hatching and visible needle entrance marks; some clinicians apply adhesive strips to bolster the wound for a few more days. Sutures and staples on the torso and upper extremities are removed in 7 to 10 days. Sutures and staples on the extensor surface of the elbow, knee, and anywhere below the knee should remain for 10 to 12 days.

Abrasions

Abrasions are skin scrapes that may involve epidermis or part or all of the dermis.

Abrasions are evaluated, cleansed, and debrided similarly to lacerations. They are harder to anesthetize, however, which is particularly problematic when large amounts of dirt, stones, or glass are embedded as is frequently the case, particularly with deep, scraping wounds; a regional nerve block or IV sedation may be needed. After thoroughly removing all debris (vigorous scrubbing may be needed), antibiotic ointment (eg, bacitracin) and a nonadherent gauze dressing that is impermeable to bacteria can be applied. Other commercial wound dressings may be used; the goals are to keep the wound from drying out, because drying interferes with re-epithelialization, and to keep the dressing from adhering.

Chapter 323. Fractures, Dislocations, and Sprains

Introduction

Fractures, joint dislocations, ligament sprains, muscle strains, and tendon injuries are common injuries that vary greatly in severity and treatment. Limbs are most often affected, although any part of the body can be. Injuries may be open (in communication with a skin wound) or closed.

Complications may be serious. Some are potentially life threatening:

- **Rapid blood loss:** Bleeding can be external or internal. Sometimes transfusion is required.

- **Fat embolism (**see Sidebar 194-1 on p. 1910**):** This rare, possibly preventable, complication may occur when a long bone is fractured.

Complications may also threaten limb viability or cause permanent limb dysfunction. Such complications occur in only a small percentage of limb injuries. The greatest threats come from open injuries that predispose to infection and injuries that disrupt the vascular supply (causing ischemia), primarily by directly injuring arteries or occasionally veins. However, some closed injuries (eg, posterior knee dislocations, hip dislocations, displaced supracondylar humeral fractures) can also disrupt the vascular supply, causing ischemia. The following can threaten a limb:

- **Compartment syndrome:** Tissue pressure increases in a closed fascial space, disrupting the vascular supply and reducing tissue perfusion. Crush injuries or markedly comminuted fractures are a common cause. Compartment syndrome can lead to rhabdomyolysis and thus infection, which threatens limb viability and, if untreated, survival.

- **Nerve or spinal cord injuries:** A penetrating injury may sever a peripheral nerve (see p. 3227). A blunt, closed injury may result in neuropraxia (bruised peripheral nerve) or axonotmesis (crushed nerve), which is more severe.

- **Dislocations:** The bones in a joint are completely separated, sometimes disrupting the vascular supply and injuring nerves. Vascular and nerve injuries are more likely when reduction (realignment of fracture fragments or dislocated joints) is delayed. Partial dislocation, termed subluxation, can also result in significant sequelae.

- **Infection:** Open injuries can become infected, potentially leading to osteomyelitis, which can be difficult to cure.

Closed injuries that do not involve blood vessels or nerves, including fractures, sprains, strains, and tendon tears, are least likely to result in serious complications.

Evaluation

In the emergency department, if the mechanism suggests potentially severe or multiple injuries (as in a high-speed motor vehicle crash or fall from a height), patients are first evaluated from head to toe for serious injuries to all organ systems and are resuscitated (see p. 3190). Patients, especially those with pelvic or femoral fractures, are evaluated for hemorrhagic shock due to occult blood loss. If the limb is injured, patients are immediately evaluated for symptoms or signs of ischemia (eg, absent pulses, marked pallor, coolness distal to the injury, severe pain).

History: The mechanism (eg, the direction of force, or torque, applied to a bone or joint) often suggests the type of injury. However, many patients do not remember, or cannot describe, the exact mechanism. Fractures and serious ligamentous injuries usually cause immediate pain; pain that begins hours to days after the injury suggests minor injury. Pain out of proportion to the apparent severity of the injury or pain that steadily worsens in the first hours to days immediately after injury suggests compartment syndrome or ischemia; compartmental pressure is then measured (see p. 3213). If a patient reports a deformity that has resolved before the patient is medically evaluated, the deformity should be assumed to be a true

deformity that spontaneously reduced. A perceived snap or pop at the time of injury may signal a fracture or a ligament or tendon injury.

Physical examination: Examination includes vascular and neurologic assessment, inspection for deformity, swelling, ecchymoses, and decreased or abnormal motion and palpation for tenderness, crepitation, and gross instability. Motor or sensory deficits suggest neurologic injury. Paresthesias or sensory deficits alone suggest neuropraxia; motor plus sensory deficits suggest axonotmesis. Deformity suggests dislocation, subluxation (partial separation of bones in a joint), or fracture. Swelling commonly indicates a significant musculoskeletal injury but may require several hours to develop. If no swelling occurs within this time, fracture or severe ligament disruption is unlikely. With some fractures (eg, buckle fractures, small fractures without displacement), swelling may be subtle but is rarely absent.

Nearly all injuries are tender, and for many patients, palpation anywhere around the injured area causes discomfort. However, a noticeable increase in tenderness in one localized area (point tenderness) suggests a fracture or sprain. Localized ligamentous tenderness and pain with stressing the joint are consistent with sprain.

Crepitation (a characteristic cracking or popping sound) may be a sign of fracture. Gross joint instability suggests dislocation or severe ligamentous disruption. Stability of an injured joint is evaluated by stress testing (see p. 3215); however, if fracture is suspected, stress testing is deferred until x-rays exclude fracture.

Some partial tendon injuries escape initial clinical detection since function appears intact. Tendon tenderness, dysfunction, weakness, or palpable defects suggest partial tendon tears. Partial tendon tears may be impossible to detect initially; they may progress to complete tears with continued use. If the mechanism or examination suggests partial tendon injury, or if the examination is inconclusive, a splint that limits further injury is applied. Subsequent examination, occasionally supplemented with MRI, may further delineate the extent of injury. A partial tendon tear generally heals well if the joint is immobilized for 3 wk to prevent progression,

Attention to certain areas during examination can help detect commonly missed injuries (see Table 323-1).

If muscle spasm and pain limit physical examination (particularly stress testing), examination is sometimes easier after the patient is given a systemic or local anesthetic with or without sedation. Alternatively, the injury can be immobilized for a few days, and then the patient can be reexamined.

Imaging: Evaluation of suspected vascular injury, typically by arteriography, takes precedence over bone imaging.

Not all limb injuries require imaging. Some fractures are minor and are treated similarly to soft-tissue injuries. For example, most injuries of toes 2 through 5 are treated symptomatically whether a fracture is present or not. Many ankle sprains do not require x-rays during the initial evaluation because the probability of finding a fracture that would require a change in treatment is low. There are explicit, generally accepted criteria for obtaining certain kinds of x-rays; eg, if ankle sprain is suspected, x-rays are unnecessary unless specific criteria suggest fracture.

Plain x-rays show primarily bone (also joint effusion secondary to bleeding or occult fracture) and thus are useful for diagnosing most fractures as well as dislocations and subluxations that have not spontaneously reduced. X-rays are usually indicated for a suspected fracture or dislocation that requires treatment. Plain x-rays and other imaging studies should include at least 2 views taken in different planes (usually 1 anteroposterior and 1 lateral view). Additional views (such as oblique) may be obtained when the evaluation suggests fracture and 2 projections are negative.

CT or MRI can be used to better delineate fractures (eg, complex pelvic fractures) identified on plain x-rays and to check for fractures that require treatment and that are suspected even though plain x-rays do not show them (common with scaphoid fractures and impacted subcapital hip fractures). MRI can also be done to diagnose complex sprains (including complete rupture of a ligament) and other soft-tissue injuries

(eg, meniscal tears, cartilaginous injuries). Arteriography may be necessary for suspected arterial injuries (eg, some popliteal artery injuries). Nerve conduction studies may be indicated for nerve injuries.

Treatment

• Treatment of life- or limb-threatening injuries

• Splinting

• Definitive treatment (eg, reduction) for certain injuries

• Rest, ice, compression, and elevation (RICE)

• Usually immobilization

In the emergency department, hemorrhagic shock is treated. Injuries to arteries are repaired surgically unless they affect only small arteries with good collateral circulation. Severed nerves are surgically repaired; for neuropraxia and axonotmesis, initial treatment is usually observation, supportive measures, and sometimes physical therapy.

Most injuries, particularly grossly unstable ones, are immobilized immediately by splinting (immobilization with a nonrigid or noncircumferential device) to prevent further injury to soft tissues by unstable injuries and to decrease pain. In patients with long-bone fractures, splinting may prevent fat embolism. Pain is treated, typically with opioids (see p. 1623). Definitive treatment often involves reduction, which usually requires analgesia or sedation. Closed reduction (without skin incision) is done when possible; if not, open reduction (with skin incision) is done. Closed reduction

[**Table 323-1.** Examination for Some Commonly Missed Injuries]

of fractures is usually maintained by casting; some dislocations require only a splint or sling. Open reduction is usually maintained by various surgical hardware (eg, pins, screws, plates, external fixators).

RICE: Patients who have soft-tissue injuries, with or without musculoskeletal injuries, benefit from RICE (rest, ice, compression, elevation). Rest prevents further injury and may speed healing. Ice and compression minimize swelling and pain. Ice is enclosed in a plastic bag or towel and applied intermittently during the first 24 to 48 h (for 15 to 20 min, as often as possible). Injuries can be compressed by a splint, an elastic bandage, or, for certain injuries likely to cause severe swelling, a Jones compression dressing. The Jones dressing is 4 layers; layers 1 (the innermost) and 3 are cotton batting, and layers 2 and 4 are elastic bandages. The injured limb is elevated above the heart for the first 2 days in a position that allows gravity to help drain edema fluid and thus minimize swelling. After 48 h, periodic application of warmth (eg, a heating pad) for 15 to 20 min may relieve pain and speed healing.

Immobilization: Immobilization decreases pain and facilitates healing by preventing further injury and is helpful except for very rapidly healing injuries. Joints proximal and distal to the injury should be immobilized.

A cast is usually used for fractures or other injuries that require weeks of immobilization. Rarely, swelling under a cast is severe enough to contribute to compartment syndrome (see p. 3213). Sometimes, if severe swelling is likely, a cast (and all padding) is cut open from end to end medially and laterally (bivalved). Patients with casts should be given written instructions:

• To keep the cast dry

• Never to put an object inside the cast

• To inspect the cast's edges and skin around the cast every day and apply lotion to any red or sore areas

• To pad any rough edges with soft adhesive tape, cloth, or other soft material to prevent the cast's edges

from injuring the skin

- To seek medical care at once if an odor emanates from within the cast or if a fever, which may indicate infection, develops

Good hygiene is important.

A splint (see Fig. 323-1) can be used to immobilize some stable injuries, including some suspected but unproven fractures, sprains, and other injuries that require immobilization for several days or less. A splint allows patients to apply ice and to move more and does not contribute to compartment syndrome.

Immobilization with bed rest, which is occasionally required for fractures (eg, some vertebral or pelvic fractures), can cause problems (eg, deep venous thrombosis, UTI).

Prolonged immobilization (more than 3 to 4 wk) of a joint can cause stiffness, contractures, and muscle atrophy. These complications may develop rapidly and may be permanent, particularly in the elderly. Some rapidly healing injuries are best treated with resumption of active motion within the first few days or weeks (early mobilization); this approach may minimize contractures and muscle atrophy, thus accelerating functional recovery.

Fractures

(For vertebral compression fractures, see p. 356; for dental fractures, see p. 524;

[**Fig. 323-1.** Joint immobilization as acute treatment: some commonly used techniques.]

for spinal fractures, see p. 3227; for fractures of the temporal bone, jaw and contiguous structures, and nose, see pp. 3232-3334; for metatarsal stress fractures, see p. 3304; for orbital fractures, see p. 3238, and for fractures that occur during birth, see p. 2774.)

Fractures are cracks in bones. Symptoms include pain, swelling, ecchymosis, crepitation, deformity, and abnormal motion. Occasional complications include fat embolism, arterial injury, compartment syndrome, nerve injuries, and infection. Diagnosis is by clinical criteria and usually plain x-rays. Treatment involves analgesics, immobilization, and sometimes surgery.

Most fractures result from a single application of significant force to otherwise normal bone. Pathologic fractures result from application of mild or minimal force to a bone weakened by a disorder such as cancer, cysts, or osteoporosis. Stress fractures (eg, metatarsal stress fracture—see p. 3304) result from repetitive application of force.

Pathophysiology

If Ca and vitamin D levels are adequate and bone tissue is healthy and the fracture edges are kept reasonably close to each other and with little or no relative motion, most fractures heal within weeks or months via remodeling. New tissue (callus) is produced within weeks, and bone reshapes at variable rates during the first weeks or months. Ultimately, optimal remodeling requires gradual resumption of normal motion and load-bearing stress. However, remodeling can be disrupted and refracture can occur if force is applied or the joint moves prematurely; thus, immobilization is usually needed.

Serious complications are unusual. Arteries are injured occasionally in closed supracondylar fractures of the humerus and femur but rarely in other closed fractures. Compartment syndrome or nerve injury may occur. Open fractures predispose to bone infection (see p. 370), which can be intractable. Fractures of long bones may release fat (and other marrow contents) that embolizes to the lungs and causes respiratory complications (see Sidebar 194-1 on p. 1910). Fractures that extend into joints usually disrupt articular cartilage; misaligned articular cartilage tends to scar, causing osteoarthritis and impairing joint motion. Occasionally, fractures do not heal (called nonunion); rarely, nonunion occurs even when treatment is expeditious and correct. If the vascular supply is injured by the initial injury (such as a

scaphoid fracture), aseptic necrosis may ensue even if the fracture was properly immobilized.

Symptoms and Signs

Pain is usually immediate. Swelling increases for several hours. Children may not exhibit significant soft-tissue swelling in the presence of a fracture (buckle [torus] fracture or greenstick fracture). Pain and swelling usually begin to resolve after 12 to 24 h; worsening pain after this period suggests compartment syndrome. Other symptoms and signs may include bone tenderness, ecchymosis, decreased or abnormal motion, deformity, and crepitation. With some fractures (eg, rib fractures), motion can be sensed by the patient and is described as a popping or cracking sensation.

Diagnosis

• Clinical assessment for complicating injuries

• Plain x-rays

• Sometimes CT or MRI

Patients with findings that suggest fracture are examined for ischemia, compartment syndrome, and nerve injury. If a wound is close to a fracture, open fracture is assumed. Fractures are diagnosed by imaging, beginning with plain x-rays. If no fracture line is obvious, bone density, trabecular pattern, and cortical margins are examined for subtle clues to fracture. If a fracture is not visible on plain x-rays but is strongly suspected or if more detail is needed to guide treatment, MRI or CT is done. Some experts recommend imaging the joints proximal and distal to the fracture.

A fracture's appearance on x-rays can be described precisely using 5 terms:

• Type of fracture line (see Fig. 323-2)

• Location of fracture line

• Angulation

• Displacement (see Fig. 323-3)

• Open or closed

Location may be the bone's head (sometimes involving the articular surface), neck, or shaft (proximal, middle, or distal third).

Treatment

• Analgesia, splinting, and reduction as indicated

• Treatment of complicating injuries

• Immobilization

• Sometimes surgery

Immediate treatment includes analgesics and, for suspected unstable fractures or fractures of long bones, splinting. Suspected open fractures require sterile wound dressings, tetanus prophylaxis, and broad-spectrum antibiotics (eg, a 2nd-generation cephalosporin plus an aminoglycoside).

[**Fig. 323-2.** Common types of fracture lines.]

Rotational malalignment or significant angulation or displacement is corrected with reduction (realignment of bone fragments by manipulation). Exceptions include some diaphyseal fractures in children. In these fractures, remodeling gradually corrects some types of significant angulation, and end-to-end realignment of fractured bone fragments can stimulate bone growth, which may then be excessive.

Closed reduction (without skin incision) is done when possible; if not, open reduction (with skin incision) is done.

In open reduction and internal fixation (ORIF), fracture fragments are aligned and held in place using hardware. ORIF is usually indicated for the following:

• When an intra-articular fracture is displaced (to precisely align the joint cartilage)

• When ORIF has been shown to have better results for a particular type of fracture

• When closed reduction was ineffective

• When the fracture traverses a cancerous lesion (because normal bone healing does not occur)

• When prolonged immobility (required for callus formation and remodeling) is undesirable (eg, for hip fractures), because ORIF provides early structural stability, which facilitates mobilization

Surgery is required when injury to a major vessel is suspected (for vessel repair) or when the fracture is open (for irrigation and debridement to prevent infection). Open reduction may be done without using hardware when closed reduction is ineffective.

Fractures, whether they require reduction, surgery, or neither, are typically immobilized, as are the proximal and distal joints. Usually, a cast is applied for weeks or months, but a splint may be used instead, particularly for fractures that heal faster when mobilized early. Home care for fractures includes supportive measures such as RICE (rest, ice, compression, elevation—see p. 3203).

[**Fig. 323-3.** Spatial relationship between fracture fragments.]

Patients are told to seek care immediately if symptoms of compartment syndrome occur (see p. 3213).

Geriatrics Essentials

The elderly are predisposed to fractures because of osteoporosis, a tendency to fall frequently, drug adverse effects, and impaired protective reflexes during falls. Age-related fractures tend to affect the metaphysis (the flared area between the end and shaft). They include fractures of the distal radius, proximal humerus, proximal tibia, proximal femur, pubic ramus, and vertebrae.

The goal of treatment is rapid return to activities of daily living rather than restoration of perfect limb alignment and length. Because immobilization (joint immobilization or bed rest) is more likely to cause adverse effects in the elderly, use of ORIF is increasing. Early mobilization and physical therapy are essential to recovery of function. Coexisting disorders (eg, arthritis) can interfere with recovery.

Specific Fractures

Stress fractures: Stress fractures are small and result from repetitive force (eg, from overuse); they occur most often in the metatarsals (usually in runners—see p. 3304), followed by the fibula and tibia. Symptoms include gradual onset of intermittent pain that worsens with weight bearing and eventually becomes constant. Sometimes swelling occurs.

Examination detects localized bone tenderness. Plain x-rays are done but may not show the fracture at first. Thus, many such fractures are treated presumptively, and plain x-ray is repeated 2 to 3 wk later when callus may be visible. Treatment is rest, elevation, analgesics, and sometimes immobilization. CT or

MRI is rarely needed.

Growth plate fractures: Bone grows as tissue is added proximally by the epiphyseal disk (growth plate), which is bordered by the metaphysis proximally and the epiphysis distally (see Fig. 323-4). The age at which the growth plate closes and bone growth stops varies by bone, but the growth plate is closed in all bones by the end of puberty. If there is question about a growth plate injury or if a fracture is suspected, opposite side comparison x-rays may be helpful.

The growth plate is the most fragile part of the bone and thus is usually the first structure disrupted when force is applied. Growth plate fractures are classified by the Salter-Harris system (see Fig. 323-5). Disruption of future bone growth is common with types III, IV, and V but uncommon with types I and II.

Growth plate fractures are suspected in children with tenderness localized over the growth plate. These fractures cause circumferential tenderness and thus can be clinically

[**Fig. 323-4.** Epiphyseal disks (growth plates).]

[**Fig. 323-5.** Salter-Harris classification of epiphyseal disk (growth plate) fractures.]

differentiated from contusions. In fracture types I and V, x-rays may appear normal. If so, these fractures can sometimes be differentiated from each other by injury mechanism—eg, distraction (separation in longitudinal axis) vs compression.

Closed treatment is usually sufficient for types I and II; ORIF is often required for types III and IV. Patients with type V injuries should be referred to a pediatric orthopedist because such injuries almost always lead to growth abnormalities.

Rib fractures: Typically, rib fractures result from blunt injury to the chest wall, usually involving a strong force (eg, from high-speed deceleration, a baseball bat, a major fall); however, sometimes in the elderly, only mild or moderate force (eg, in a minor fall) is required. Concomitant injuries may include

- Aortic, subclavian, or cardiac injuries (uncommon but can occur with high-speed deceleration, particularly if rib 1 or 2 is fractured)
- Splenic or abdominal injuries (with fractures of any of ribs 7 through 12)
- Pulmonary laceration or contusion
- Pneumothorax
- Other tracheobronchial injuries (uncommon)

Pain is severe, is aggravated by movement of the trunk (including coughing or deep breathing), and lasts for several weeks. Inspiratory splinting (incomplete inspiration due to pain) can cause atelectasis and pneumonia, especially in the elderly or those with multiple fractures. Young, healthy patients and those with 1 or 2 rib fractures rarely develop these complications.

Palpation of the chest wall may identify some fractures, and sometimes the patient and the examining clinician can feel the broken ribs move when the lungs are expanded. A chest x-ray is taken routinely to check for concomitant injuries (eg, pneumothorax, pulmonary contusion). Many rib fractures are not visible on a chest x-ray; specific rib views may be needed, but identifying all rib fractures by x-rays is not always necessary. Other tests are done to check for concomitant injuries that are clinically suspected.

Treatment requires opioid analgesics, which can depress respiration and worsen atelectasis. To minimize pulmonary complications, patients should consciously and frequently (eg, hourly) breathe deeply or cough while awake. Holding (essentially splinting) the affected area with the flat palm of the hand or a pillow can help minimize the pain during deep breathing or coughing. Patients are hospitalized if they have ≥ 3

fractures or underlying cardiopulmonary insufficiency. Immobilization (eg, by strapping or taping) should usually be avoided; it constricts respiration and may predispose to atelectasis and pneumonia.

Clavicle fractures: The usual injury mechanism is a fall on an outstretched arm or a direct blow. About 80% involve the middle one third of the bone and are immobilized with a sling. Previously used figure-of-eight braces are no more helpful (and are more uncomfortable) than a simple sling. Reduction is not necessary even for greatly angulated fractures. Clavicle fractures that significantly tent the skin or that involve areas other than the middle one third of the bone may require additional intervention.

Proximal humeral fractures: The usual injury mechanism is direct force or a fall on an outstretched arm. Usually, displacement and angulation are minimal. Contractures may develop after only a few days of immobilization, particularly in the elderly. Minimally displaced or angulated fractures are treated with immobilization in a sling and swathe (see Fig. 323-1) and early range-of-motion exercises. More severe fracture may require ORIF or surgery to insert a prosthetic joint (shoulder replacement).

Distal humeral fractures: The usual injury mechanism is direct force or a fall on an outstretched arm. The brachial artery or radial nerve may be damaged. Angulation, if present, must be corrected. Casting with closed reduction may be tried, but ORIF may be necessary.

Radial head fractures: The usual injury mechanism is a fall on an outstretched arm. The radial head is palpated on the lateral elbow as a structure that rotates during pronation and supination. Routine anteroposterior and lateral x-rays usually show a joint effusion or a displaced anterior fat pad (sail sign) but often do not show the fracture. Patients with localized radial head tenderness and effusion require oblique views (which are more sensitive for fracture) or presumptive treatment of a fracture. For fractures with only minimal angulation and displacement, treatment is a splint with the elbow flexed 90° or a sling. Arthrocentesis to remove blood from the joint often helps relieve pain and facilitate recovery. Starting range-of-motion exercises 10 days after the injury maximizes joint flexibility.

Distal radial fractures: The usual injury mechanism is wrist hyperextension, usually during a fall. Dorsally displaced or angulated fractures (sometimes called Colles' fractures) are common. Treatment is reduction and immobilization at 15 to 30° of wrist extension. ORIF may be necessary if the joint is disrupted or if there is excessive impaction or shortening.

Metacarpal neck fractures (except thumb): The usual injury mechanism is an axial load (eg, from punching with a clenched fist). If wounds are near the metacarpophalangeal joint, contamination with human oral flora should be considered, and measures to prevent infection are often required (see p. 3307). Reduction is necessary for fractures of the 2nd and 3rd metacarpals but is unnecessary for dorsal or volar angulation of < 35° for the 4th metacarpal or of 45° for the 5th metacarpal. Treatment is a splint (eg, an ulnar gutter splint for fractures of the 4th or 5th metacarpal—see Fig. 323-1).

Scaphoid (navicular) fractures: The usual injury mechanism is wrist hyperextension, usually during a fall on the outstretched hand. Avascular necrosis is a common complication, even when initial care is ideal, and can cause disabling, degenerative arthritis of the wrist. Fracture signs include pain with axial compression of the thumb, pain with wrist supination against resistance, and, particularly, tenderness in the anatomic snuffbox with ulnar wrist deviation. The anatomic snuffbox is palpated just distal to the radius between the extensor pollicis longus, extensor pollicis brevis, and abductor pollicis longus tendons. The initial plain x-ray is often normal. If a fracture is still suspected, MRI, which is more sensitive than x-rays, is done, or fracture is presumed and treated with a thumb spica splint (see Fig. 323-1), with a follow-up plain x-ray taken in 1 to 2 wk. Rarely, this subsequent x-ray is falsely normal.

Fingertip (tuft of the distal phalanx) fractures: The usual mechanism is a crush injury. Subungual (beneath the nail) hematoma usually occurs and produces a blue-black, tender bruise, which may elevate the nail; hematoma indicates a nail bed laceration. Most fingertip fractures are treated symptomatically with a protective covering (eg, commercially available aluminum and foam splint material) wrapped around the fingertip. Subungual hematomas can be drained to relieve pain by puncturing the nail (trephination), usually with an 18-gauge needle in a rotatory motion or, if no nail polish is on the nail, with an electrocautery device. If trephination is done gently and rapidly, anesthesia is often unnecessary. Large displaced fractures are rarely repaired surgically. Markedly disrupted nail beds are repaired with sutures

but are best left alone if the nail is closely adherent to the nail bed. Hyperesthesia frequently persists long after a large fracture has healed and requires desensitization therapy.

Pelvic fractures: Pelvic fractures may be stable or unstable. Compression of the pubic symphysis or simultaneous compression of both anterior superior iliac spines is often painful, particularly in severe fractures. For pelvic fractures, CT is more sensitive than plain x-rays.

Stable fractures do not disrupt the pelvic ring. Some (eg, symphyseal or pubic ramus fractures) result from minor injuries (eg, falls at home), especially in patients with osteoporosis. Treatment is often symptomatic, particularly if patients can walk unaided.

Unstable fractures disrupt the pelvic ring in ≥ 2 places; disruptions can be fractures within bones or separations between the fibrous joints (syndesmoses) between bones. Unstable fractures usually result from substantial forces (eg, high-speed motor vehicle crashes). Intestinal injuries may occur. Concomitant GU injuries (eg, urethral or bladder tears) are common, particularly with anterior pelvic fractures. Vascular injuries may occur and cause hemorrhagic shock, especially with posterior pelvic fractures. Mortality rate is high. Initial evaluation and treatment are directed at associated injuries. The fracture often requires surgical repair.

Hip fractures: Hip fractures are most common among the elderly, particularly those with osteoporosis (mostly women—see p. 356). Most fractures result from falls, but in the elderly, seemingly minimal force (eg, rolling over in bed, getting up from a chair, walking) can result in hip fracture, usually because osteoporosis has weakened bone. Subcapital femoral neck and intertrochanteric fractures are the most common types. Hip fractures often cause referred pain in the knee and thus may be misinterpreted as a knee abnormality. Pubic ramus fractures can cause hip pain.

Subcapital fractures may result from a single injury but often result from repeated stress or minimal force, resulting in a small or large stress fracture. A fall after the initial fracture may worsen or displace the fracture. Patients with small fractures may be ambulatory and have only mild pain. However, such patients may be unable to flex the entire lower limb against resistance with the knee extended. Passive hip rotation with the knee flexed aggravates the pain, helping to differentiate hip fracture from extra-articular disorders such as trochanteric bursitis. Large or displaced fractures tend to limit hip motion more, shorten the leg, and cause the leg to rotate externally. Displacement predisposes to osteonecrosis of the femoral head and fracture nonunion.

Plain x-rays are occasionally normal when fractures are small or impacted or when osteoporosis is severe. If a fracture is still suspected, MRI is done; if MRI is unavailable or contraindicated, CT is done. If patients are expected to resume walking and have no contraindication to surgery, treatment is usually surgical repair (typically ORIF—see Fig. 323-6) and early ambulation.

If patients are elderly, are not active, and have displaced fractures, treatment is often prosthetic replacement of the femoral head, typically with a Moore prosthesis, or total hip replacement. Occasionally, the femoral head must be replaced when the fracture is displaced in younger adults, particularly those who are inactive. Usually, prolonged bed rest should be avoided in elderly patients. Bed rest increases the risk of deep venous thrombosis, a common complication of hip fractures. Prophylactic anticoagulation may reduce the incidence of post-hip fracture venous thrombosis.

[**Fig. 323-6.** Open reduction with internal fixation (ORIF).]

Intertrochanteric fractures usually result from falls or direct blows. Patients have tenderness, ecchymosis, and swelling over the hip; usually, the leg is shortened and rotates externally. Plain x-rays are usually diagnostic. Treatment is usually ORIF and early mobilization.

Femoral shaft fractures: The usual injury mechanism is severe direct force or an axial load to the flexed knee. Fracture due to trauma causes obvious swelling, deformity, and instability. Up to 1.5 L of blood for each fracture may be lost. Treatment is immediate splinting, then ORIF.

Ankle fractures: The ankle bones and ligaments form a ring that connects the tibia and fibula to the talus and calcaneus. Within the ring, stability is provided by 2 bones (the medial malleolus of the tibia and lateral malleolus of the fibula) and 2 ligament complexes (medially, the deltoid ligament; laterally, mainly the anterior and posterior talofibular ligaments and calcaneofibular ligament—see Fig. 323-7). Ankle fractures are common and can result from multiple injury mechanisms. Fractures that disrupt the ring in one place often disrupt it in another (eg, if only one bone is fractured, a ligament is often simultaneously and severely torn). If fractures disrupt ≥ 2 of the structures stabilizing the ankle ring, the ankle is unstable. Disruption of the medial deltoid ligament also causes instability. For unstable injuries, surgery may be required, and prognosis is guarded. Most stable ankle fractures without other indications for surgery can be treated with a cast for 6 wk; prognosis is good.

Fractures of the 2nd metatarsal bone base with dislocation (Lisfranc's fracture-dislocation): The usual mechanism is a fall on a foot in plantar flexion. Usually there is significant soft-tissue swelling. These rare fractures are difficult to appreciate on plain x-rays and are often misdiagnosed, leading to sometimes serious complications, such as osteoarthritis and rarely compartment syndrome. A plain x-ray can show a fracture at the base of the 2nd metatarsal

[**Fig. 323-7.** Ligaments of the ankle.]

or chip fractures of the cuneiform but may not show disruption of the tarsometatarsal joint, which should be suspected even if it is not visible on plain x-rays. Dislocations often spontaneously reduce, but immediate referral, usually for closed reduction, which requires general anesthesia, may be warranted.

Fractures of the 5th metatarsal bone base (dancer's fracture): The usual injury mechanism is a twist (typically, inversion) or crush injury. These fractures usually heal relatively quickly; nonunion is uncommon. Treatment is a protective walking shoe.

Fractures of the 5th metatarsal bone diaphysis (Jones fracture): The usual injury mechanism is a crush injury. These fractures are less common than those of the metatarsal bone base, and delayed union or nonunion occurs more commonly. Treatment is a cast that immobilizes the ankle. Avulsion fractures of the base of the fifth metatarsal can occur with inversion ankle injuries and are less significant than a true Jones fracture, the latter being predisposed to nonunion.

Toe fractures: The usual injury mechanism is a crush injury. Unless rotational deformity or joint involvement is suspected or the proximal phalanx of the great toe is injured, x-rays are usually unnecessary. Treatment is taping the injured toe to an adjacent toe (dynamic splinting or buddy taping). Markedly displaced toe fractures should be reduced to restore alignment.

Compartment Syndrome

Compartment syndrome is increased tissue pressure within a closed fascial space, resulting in tissue ischemia. The earliest symptom is pain out of proportion to the severity of injury. Diagnosis is by measuring compartmental pressure. Treatment is fasciotomy.

Compartment syndrome is a self-perpetuating cascade of events. It begins with the tissue edema that normally occurs after injury (eg, because of soft-tissue swelling or a hematoma). If edema develops within a closed fascial compartment, typically in the anterior or posterior compartments of the leg, there is little room for tissue expansion, so interstitial (compartmental) pressure increases. As compartmental pressure exceeds about 20 mm Hg, cellular perfusion slows and may ultimately stop. (NOTE: Because 20 mm Hg is much lower than arterial pressure, cellular perfusion can stop long before pulses disappear.) Resultant tissue ischemia further worsens edema in a vicious circle. As ischemia progresses, muscles necrose, sometimes leading to rhabdomyolysis and infections; these complications can cause loss of limb and, if untreated, death. If arteries are injured, arterial pressure can drop below even mildly elevated compartmental pressures, causing or worsening compartment syndrome.

Etiology

Common causes include fractures and severe contusions. Rare causes include snakebites, severe

exertion, drug overdose (heroin, cocaine), casts, tight bandages, and other rigid circumferential devices that limit swelling and thus increase compartmental pressure. Prolonged pressure on a muscle during coma may cause rhabdomyolysis.

Symptoms and Signs

Compartment syndrome usually occurs in the anterior lower leg. The earliest symptom is worsening pain. It is typically out of proportion to the severity of the apparent injury and is exacerbated by passive stretching of the muscles within the compartment (eg, for the anterior leg compartment, by passive toe flexion, which stretches the toe extensor muscles). Pain, one of the 5 P's of tissue ischemia, is followed by the other 4: paresthesias, paralysis, pallor, and pulselessness. Compartments may feel tense when palpated.

Diagnosis

• Measurement of compartmental pressure

Diagnosis must be made and treatment started before pallor or pulselessness develop, indicating necrosis. Diagnosis is by measuring compartmental pressure (normal ≤ 20 mm Hg), usually with a commercially available wick catheter.

Treatment

• Often fasciotomy

Pressures of 20 to 40 mm Hg can sometimes be treated conservatively with analgesics, elevation, and splinting; casts, if present, are removed or bivalved. Pressures > 40 mm Hg usually require immediate fasciotomy to relieve pressure. If necrosis occurs, amputation may be needed.

Dislocations

(For spinal dislocations, see p. 384; for atlantoaxial subluxation, see p. 385; and for mandibular dislocation, see p. 524.)

Dislocation is a complete separation of the bone ends that normally articulate to form a joint; subluxation is a partial separation.

The most commonly dislocated limb joint is the glenohumeral (shoulder). Arterial and nerve injuries, although uncommon, are a risk with dislocations (eg, of the knee, elbow, or hip), particularly those that are not rapidly reduced.

Symptoms and signs include pain, swelling, deformity, and inability to move. Diagnosis is by plain x-rays. Treatment is usually closed reduction as soon as possible; it requires sedation and analgesia or, occasionally, general anesthesia. Neurovascular assessment is done before and after reduction. If closed reduction is ineffective, open reduction is necessary.

Specific Dislocations

Glenohumeral (shoulder) dislocations: Glenohumeral dislocations are anterior in ≥ 95% of patients; the cause is abduction and external rotation of the humerus. Occasionally, the axillary nerve is injured, or the greater tuberosity is fractured, particularly in patients > 45. The acromion is prominent; the humeral head is displaced anteriorly and inferiorly and cannot be palpated in its usual position. Sensation over the lateral deltoid is tested to check for axillary nerve injury. Treatment is usually closed reduction with using conscious sedation. The traction-countertraction technique is one of many commonly used methods of reduction (see Fig. 323-8). After reduction, the joint is immobilized immediately with a sling and swathe (see Fig. 323-1).

Occasionally, dislocation is posterior—a commonly missed injury—or inferior (luxatio erecta). In patients

with luxatio erecta, the brachial artery or brachial plexus is often also injured.

Elbow dislocations: Most elbow dislocations result from a fall on an extended, abducted arm. They are common and usually posterior. Associated injuries may include fractures, injuries to the ulnar or median nerve, and possibly injury to the brachial artery. The joint is usually flexed about 45°, and the olecranon is prominent and posterior to the humeral epicondyles; however, these anatomic relationships may be difficult to determine because of swelling. Reduction is usually with sustained, gentle traction after sedation and analgesia.

Radial head subluxations (nursemaid's elbow): In adults, the radial head is wider than the radial neck; consequently, the head cannot fit through ligaments that tightly surround the neck. However, in toddlers (about 2 to 3 yr old), the radial head is no wider than the radial neck and can easily slip through these ligaments (radial head subluxation). Subluxation results from traction on the forearm as when a caregiver pulls a reluctant toddler forward or catches the toddler by the wrist during a fall—actions many caregivers do not remember. Symptoms may include pain and tenderness; however, many toddlers cannot describe their symptoms and simply avoid moving the affected elbow (pseudoparalysis).

[Fig. 323-8. Traction-countertraction technique for reducing anterior shoulder dislocations.]

Plain x-rays are normal and considered unnecessary by some experts unless an alternate diagnosis is clinically suspected. Reduction may be diagnostic and therapeutic. The elbow is completely extended and supinated, then flexed, usually without sedation or analgesia. Reduction is often marked by a subtle palpable pop or click as the radial head resumes normal position. Children may start to move the elbow after about 20 min. Immobilization is unnecessary. If pain or dysfunction lasts longer than 24 h, incomplete reduction or an occult fracture should be suspected.

Proximal interphalangeal (PIP) joint dislocations: PIP joint dislocations are common. Dorsal dislocations, which are more common than volar, are usually due to hyperextension, sometimes displacing the volar joint structures intra-articularly. Volar dislocations can rupture the central slip of the extensor tendon, causing boutonniere deformity (see p. 387). Dislocations usually cause obvious deformities. A lateral x-ray should be taken with the affected digit visibly separated from the others.

For most dislocations, closed reduction using digital block anesthesia is done. Axial traction and volar force are used for dorsal dislocations, and dorsal force is used for volar dislocations. Dorsal dislocations are splinted at 15° of flexion for 3 wk. Volar dislocations are splinted at extension for 1 to 2 wk. Some dorsal dislocations require open reduction.

Hip dislocations: Most hip dislocations are posterior and result from a severe posteriorly directed force to the knee with the knee and hip flexed (eg, against a car dashboard). Complications may include arterial injury (particularly if the dislocation is anterior) with subsequent avascular necrosis of the femoral head and sciatic nerve injury. Treatment is reduction as soon as possible, followed by bed rest or joint immobilization.

Knee (tibiofemoral) dislocations: Most anterior dislocations result from hyperextension; most posterior dislocations result from a posteriorly directed force to the proximal tibia with the knee slightly flexed. Most knee dislocations result from severe trauma (eg, high-speed motor vehicle collisions), but seemingly slight trauma, such as stepping in a hole, coupled with a twisting movement, can dislocate the knee. Some dislocations spontaneously reduce before medical evaluation, resulting in a large hemarthrosis and gross instability of the knee. Concomitant arterial injury may be seen with a spontaneously reduced knee dislocation.

Popliteal artery injury is a serious complication, but it is often a subtle injury in the early stages. The popliteal artery may be injured, even if ischemia is not initially evident. When the intima is torn, occlusion of the artery may be delayed. Thus, some experts believe that serial clinical evaluations of the distal pulse can rule out a popliteal artery injury if the pulse is normal over a period of time. However, some experts believe that angiography is indicated for every patient with a knee dislocation or with gross instability.

Treatment is immediate reduction and surgical repair.

Lateral patellar dislocations: The usual injury mechanism is quadriceps contraction plus flexion and external tibial rotation. Most patients have an underlying chronic patellofemoral abnormality. Many dislocations spontaneously reduce before medical evaluation. Treatment is reduction; with the hip flexed, the patella is gently moved medially while the knee is extended. Reduction is followed by a cylindrical leg cast or surgical repair.

Sprains, Strains, and Tendon Tears

Tears may occur in ligaments (sprains), in muscles (strains), or in tendons. Tears may be graded as minimal (1st degree), moderate to severe (2nd degree), or complete (3rd degree). Third-degree sprains may result in joint instability and are differentiated from 2nd-degree sprains by stress testing. Third-degree tendon tears disrupt muscle function. Treatment of all tears includes analgesics, immobilization, and, for some 3rd-degree sprains and tendon tears, surgical repair.

Sprains commonly involve the acromioclavicular joint, proximal interphalangeal joint, knee (see p. 3217), or ankle. Tendon tears commonly involve the knee extensor mechanism or Achilles tendon. Various muscles are commonly strained. Sprains, strains, and tendon tears cause pain, tenderness, and usually swelling. Second-degree sprains are very painful when stretched. Third-degree sprains often cause joint instability because ligaments that stabilize joints may be disrupted. In 3rd-degree tendon tears, the muscle cannot move the bone normally attached to it by the tendon; a tendon defect may be palpable.

Diagnosis

• Bedside stress testing

Bedside stress testing involves passively opening the joint in a direction other than the normal range of motion (stressing) to check for joint instability; this test helps differentiate between 2nd- and 3rd-degree sprains. Because muscle spasm during acutely painful injuries may mask joint instability, the surrounding muscles are relaxed as much as possible, and examinations are begun gently, then repeated, with slightly more force each time. Findings are compared with those for the opposite, normal side. With 2nd-degree sprains, stress is painful, and joint opening is limited. With 3rd-degree sprains, stress is less painful because the ligament is completely torn and is not being stretched, and joint opening is less limited. If muscle spasm is severe, the examination should be repeated after injection of a local anesthetic, after use of systemic analgesia or sedation, or after a few days, when the spasm has subsided.

Treatment

• Rest, ice, compression, and elevation (RICE)

• Immobilization and repair as indicated based on injury location and severity

Treatment of all tears includes RICE (see p. 3203) and, if necessary, analgesics. For 1st-degree tears, early mobilization is usually best. Mild 2nd-degree tears are often immobilized with a sling or splint for a few days. Severe 2nd-degree and some 3rd-degree sprains and tendon tears are immobilized for days or weeks, sometimes with a cast. Many 3rd-degree sprains and tendon tears require surgical repair.

Specific Sprains and Tendon Tears

Acromioclavicular joint sprains (separation): The usual injury mechanism is a fall on the point of a shoulder or on an outstretched arm. Severe sprains tear the coracoclavicular ligament, displacing the clavicle upward from the acromion. Treatment is immobilization (eg, with a sling) and early mobility exercises. Some severe sprains are surgically repaired. This injury is often termed a shoulder separation.

Ulnar collateral ligament sprains (gamekeeper's thumb): The ulnar collateral ligament connects the

base of the thumb's proximal phalanx to the thumb's metacarpal bone on the ulnar aspect of the joint. The usual injury mechanism is lateral deviation of the thumb. Falling on the hand while holding a ski pole is a common mechanism. Diagnosis is by stress testing to check for radial deviation of the thumb; digital nerve block anesthesia is required. Treatment is immobilization with a thumb spica splint; if maximum possible radial deviation is > 20° more than that in the opposite thumb, surgical repair is necessary.

Quadriceps tendon injuries: The quadriceps tendon can be partially or completely disrupted. The elderly and people who have osteoarthritis or who are taking corticosteroids are especially at risk. The mechanism can seem minor, but includes forceful flexion of the knee, often while descending stairs. Patients with complete tears cannot stand, do a straight leg raise while lying on their back, or extend the knee while seated. An examiner can sometimes feel a rent in the tendon. X-rays may be normal or show a high-riding patella. Swelling in the area is diffuse and may be misinterpreted as a ligamentous knee joint injury with hemarthrosis. MRI confirms the diagnosis. Treatment is surgical repair as soon as possible, but long-term complications (eg, loss of motion and weakness) are common.

Ankle sprains: The most important ankle ligaments are the deltoid (the strong, medial ligament) and the anterior and posterior talofibular and calcaneofibular (lateral—see Fig. 323-7). Ankle sprains are very common, typically resulting from turning the foot inward (inversion); inversion tears the lateral ligaments, usually beginning with the anterior talofibular ligament. Severe 2nd- and 3rd-degree sprains often cause chronic joint buckling and instability and predispose to additional sprains. Ankle sprains cause pain and swelling, which are usually maximal at the anterolateral ankle. Third-degree sprains often cause more diffuse pain and swelling (sometimes egg-shaped). Forcefully turning the foot outward (eversion) may tear the syndesmotic ligaments between the tibia and fibula proximal to the ankle (high ankle sprain). Occasionally, the deltoid ligament is sprained during eversion, often with simultaneous fracture of the fibular head.

Diagnosis is primarily clinical. The ankle anterior drawer test assesses stability of the anterior talofibular ligament, helping differentiate between 2nd- and 3rd-degree lateral sprains. For this test, patients sit or lie supine with the knee at least slightly flexed; one of the examiner's hands prevents forward movement of the anterior distal tibia while the other hand cups the heel, pulling it anteriorly. Avulsion fractures of the base of the 5th metatarsal and Achilles tendon injuries may be misdiagnosed as ankle sprains. High ankle sprains are considered when eversion is the mechanism and when eversion reproduces pain. Routine ankle x-rays are done to exclude significant fractures in patients with any of the following:

• Age > 55

• Inability to bear weight immediately after the injury and inability to take 4 steps when first examined

• Bone tenderness at the posterior edge or tip of either malleolus

Complex ligamentous injuries to the ankle may require additional testing, such as MRI.

Most ankle sprains do well with minimal intervention and early immobilization. Splinting alleviates pain but does not appear to affect final outcome. First-degree sprains are treated with RICE and early weight bearing. Second-degree sprains are treated with RICE and immobilization of the ankle in a neutral position with a posterior splint, followed by mobilization; for mild sprains, mobilization can occur within a few days. Third-degree sprains may require casting or surgical repair. If clinical evaluation or determination of the extent of injury is impossible (eg, due to muscle spasm or pain), the injury may be immobilized and reexamined after a few days. Rarely MRI is used. High ankle sprains usually require a cast for several weeks.

Achilles tendon tears: The usual injury mechanism is ankle dorsiflexion, particularly if the tendon is taut. The calf is squeezed while the patient is prone (Thompson test); decreased passive ankle plantar flexion indicates a tear. Most tears are complete. Partial tears are often missed. Treatment of incomplete tears and some complete tears is a posterior ankle splint with the ankle in plantar flexion for 4 wk. Treatment of complete tears is usually immediate surgical repair. Spontaneous Achilles tendon rupture has been associated with the use of fluoroquinolone antibiotics.

Knee Sprains and Meniscal Injuries

Sprains of the external (medial and lateral collateral) or internal (anterior and posterior cruciate) ligaments or injuries of the menisci commonly result from knee trauma. Symptoms include pain, effusion, instability (with severe sprains), and locking (with some meniscal injuries). Diagnosis is by physical examination and sometimes MRI or arthroscopy. Treatment is RICE (rest, ice, compression, elevation) and, for severe injuries, casting or surgical repair.

Many structures that help stabilize the knee are located mainly outside the joint muscles (eg, quadriceps, semimembranosus), their insertions (eg, pes anserinus), and extracapsular ligaments. The lateral collateral ligament is extracapsular; the medial (tibial) collateral ligament has a superficial extracapsular portion and a deep portion that is part of the joint capsule.

Inside the knee, the joint capsule and the posterior and highly vascular anterior cruciate ligaments help stabilize the joint. The medial and lateral menisci are intra-articular cartilaginous structures that act mainly as shock absorbers but provide some stabilization (
Fig. 323-9).

The most commonly injured knee structures are the medial collateral and anterior cruciate ligaments. The most common mechanism for ligamentous knee injuries is an inward, medial force usually accompanied by some external rotation and flexion (as when being tackled in football). In such cases, the medial collateral ligament is usually injured first, followed by the anterior cruciate ligament, then the medial meniscus. The next most common mechanism is an outward force, often injuring the lateral collateral ligament, anterior cruciate ligament, or both. Anterior or posterior forces and hyperextension typically injure the cruciate ligaments. Weight bearing and rotation at the time of injury tend to cause meniscal injuries.

[**Fig. 323-9.** Ligaments of the knee.]

Symptoms and Signs

Swelling and muscle spasm progress over the first few hours. With 2nd-degree sprains, pain is typically moderate or severe. With 3rd-degree sprains, pain may be mild, and surprisingly, some patients can walk unaided. An audible pop suggests an anterior cruciate tear but is uncommon. An effusion suggests injury to the anterior cruciate and possibly other intra-articular structures. However, with severe 3rd-degree tears of the medial collateral ligament or anterior cruciate, no effusion may be apparent because these tears can result in an open joint capsule, allowing blood to exit the joint. Tenderness is often maximal over the injured structure: Medial meniscal injuries cause tenderness in the joint plane (joint line tenderness) medially, and lateral meniscal injuries cause tenderness in the joint plane laterally. These injuries also cause swelling and sometimes restrict passive motion (called locking).

Diagnosis

• Bedside testing for specific injuries

• Imaging (eg, MRI) or arthroscopy as indicated

A spontaneously reduced knee dislocation should be suspected in patients with a large hemarthrosis, gross instability, or both; serial evaluation of distal pulses or immediate angiography should be considered. Otherwise, the knee is fully examined first. Knee extension is assessed to check for disruption of the extensor mechanism (eg, tears of the quadriceps muscle or patellar tendon, which can be missed on x-rays; fracture of the patella or tibial tubercle). Knee pain and effusion may indicate disruption of the extensor mechanism.

Other bedside testing is done to check for specific injuries. For the Apley compression test, the patient is prone, and the examiner stabilizes the patient's thigh. The examiner flexes the patient's knee 90° and rotates the lower leg while pressing the lower leg downward toward the knee (compression), then rotates the lower leg while pulling it away from the knee (distraction). Pain during compression and knee rotation

suggests a meniscal injury; pain during distraction and knee rotation suggests a ligamentous or joint capsule injury.

For assessment of the medial and lateral collateral ligaments, the patient is examined supine, with the knee flexed about 20° and the hamstring muscles relaxed. The examiner puts one hand over the side of the knee opposite the ligament being tested. With the other hand, the examiner cups the heel and pulls the lower leg outward to test the medial collateral ligament or inward to test the lateral collateral ligament. Moderate instability after acute injury suggests that a meniscus or cruciate ligament is torn as well as the collateral ligament.

Lachman's test is the most sensitive physical test for acute anterior cruciate ligament tears. With the patient supine, the examiner supports the patient's thigh and calf, and the patient's knee is flexed 20°. The lower leg is moved anteriorly. Excessive passive anterior motion of the lower leg from the femur suggests a significant tear.

If patients cannot tolerate stress testing (eg, because of pain or muscle spasm), they should be reexamined after injection of a local anesthetic, after use of systemic analgesia and sedation, or at a follow-up examination 2 to 3 days later (after swelling and spasm have subsided); alternatively, MRI or arthroscopy can be done. MRI or arthroscopy is also done when severe injury cannot be excluded clinically.

Treatment

• Rest, ice, compression, and elevation (RICE)

• Immobilization and repair as indicated based on injury location and severity

Draining large effusions (see
Fig. 32-3 on p.
288) may decrease pain and spasm. Most 1st-degree and mild or moderate 2nd-degree injuries can be treated initially with RICE and immobilization of the knee at 20° of flexion with a commercially available knee immobilizer or splint. Severe 2nd-degree and most 3rd-degree sprains and most meniscal injuries require casting for ≥ 6 wk. However, some 3rd-degree injuries of the medial collateral ligament, anterior cruciate ligament, and menisci require arthroscopic surgical repair.

Chapter 324. Traumatic Brain Injury

Introduction

Traumatic brain injury (TBI) is physical injury to brain tissue that temporarily or permanently impairs brain function. Diagnosis is suspected clinically and confirmed by imaging (primarily CT). Initial treatment consists of ensuring a reliable airway and maintaining adequate ventilation, oxygenation, and blood pressure. Surgery is often needed in patients with more severe injury to place monitors to track and treat intracranial pressure, decompress the brain if intracranial pressure is increased, or remove intracranial hematomas. In the first few days after the injury, maintaining adequate brain perfusion and oxygenation and preventing complications of altered sensorium are important. Subsequently, many patients require rehabilitation.

In the US, as in much of the world, TBI is a common cause of death and disability. Causes include motor vehicle crashes and other transportation-related causes (eg, bicycle crashes, collisions with pedestrians), falls (especially in older adults and young children), assaults, and sports activities.

Pathology

Structural changes from head injury may be gross or microscopic, depending on the mechanism and forces involved. Patients with less severe injuries may have no gross structural damage. Clinical manifestations vary markedly in severity and consequences. Injuries are commonly categorized as open or closed.

Open injuries involve penetration of the scalp and skull (and usually the meninges and underlying brain tissue). They typically involve bullets or sharp objects, but a skull fracture with overlying laceration due to severe blunt force is also considered an open injury.

Closed injuries typically occur when the head is struck, strikes an object, or is shaken violently, causing rapid brain acceleration and deceleration. Acceleration or deceleration can injure tissue at the point of impact (coup), at its opposite pole (contrecoup), or diffusely; the frontal and temporal lobes are particularly vulnerable. Axons, blood vessels, or both can be sheared or torn. Disrupted blood vessels leak, causing contusions, intracerebral or subarachnoid hemorrhage, and epidural or subdural hematomas (see
Table 324-1).

Concussion: Concussion is defined as a transient and reversible posttraumatic alteration in mental status (eg, loss of consciousness or memory) lasting from seconds to minutes and, by arbitrary definition, < 6 h. Gross structural brain lesions and serious neurologic residua are not part of concussion, although temporary disability can occur due to symptoms, such as nausea, headache, dizziness, and memory disturbance (postconcussion syndrome).

Brain contusions: Contusions (bruises of the brain) can occur with open or closed injuries and can impair a wide range of brain functions, depending on contusion size and location. Larger contusions may cause brain edema and

[**Table 324-1.** Common Types of Traumatic Brain Injury]

increased intracranial pressure (ICP). Contusions may enlarge in the hours and days following the initial injury and cause neurologic deterioration.

Diffuse axonal injury: Diffuse axonal injury (DAI) occurs when deceleration causes shear-type forces that result in generalized, widespread disruption of axonal fibers and myelin sheaths. A few DAI lesions may also result from minor head injury. Gross structural lesions are not part of DAI, but small petechial hemorrhages in the white matter are often observed on CT and on histopathologic examination. DAI is sometimes defined clinically as a loss of consciousness lasting > 6 h in the absence of a specific focal lesion. Edema from the injury often increases ICP, leading to various manifestations (see below). DAI is typically the underlying injury in shaken baby syndrome.

Hematomas: Hematomas (collections of blood in or around the brain) can occur with open or closed injuries and may be epidural, subdural, or intracerebral. Subarachnoid hemorrhage (SAH—bleeding into the subarachnoid space—see p. 1654) is common in TBI, although the appearance on CT is not usually the same as aneurysmal SAH.

Subdural hematomas are collections of blood between the dura mater and the piaarachnoid mater. Acute subdural hematomas arise from laceration of cortical veins or avulsion of bridging veins between the cortex and dural sinuses. They often occur with head trauma from falls and motor vehicle crashes. Compression of the brain by the hematoma and swelling of the brain due to edema or hyperemia (increased blood flow due to engorged blood vessels) can increase ICP. When these processes both occur, mortality and morbidity can be high. A chronic subdural hematoma may appear and cause symptoms gradually over several weeks after trauma. These hematomas occur more often in elderly patients (especially in those taking antiplatelet or anticoagulant drugs, or in those with brain atrophy). Elderly patients may consider the head injury relatively trivial or may have even forgotten it. In contrast to acute subdural hematomas, edema and increased ICP are unusual.

Epidural hematomas are collections of blood between the skull and dura mater and are less common than subdural hematomas. Epidural hematomas that are large or rapidly expanding are usually caused by arterial bleeding, classically due to damage to the middle meningeal artery by a temporal bone fracture. Without intervention, patients with arterial epidural hematomas may rapidly deteriorate and die. Small, venous epidural hematomas are rarely lethal.

Intracerebral hematomas are collections of blood within the brain itself. In the traumatic setting, they result from coalescence of contusions. Exactly when one or more contusions become a hematoma is not well defined. Increased ICP, herniation, and brain stem failure can subsequently develop, particularly with contusions in the temporal lobes.

Skull fractures: Penetrating injuries by definition involve fractures. Closed injuries may also cause skull fractures, which may be linear, depressed, or comminuted. The presence of a fracture suggests that significant force was involved in the injury. However, most patients with simple linear fractures and no neurologic impairment are not at high risk of brain injuries. Fractures that involve special risks include

- Fractures in patients with neurologic impairment: Such patients are at increased risk of intracranial hematomas.

- Depressed fractures: These fractures have the highest risk of tearing the dura, damaging the underlying brain, or both.

- Temporal bone fractures that cross the area of the middle meningeal artery: In these fractures, an epidural hematoma is a risk.

- Fractures that cross one of the major dural sinuses: These fractures may cause significant hemorrhage and venous epidural or venous subdural hematoma. Injured venous sinuses can later thrombose and cause cerebral infarction.

- Fractures that involve the carotid canal: These fractures can result in carotid artery dissection.

- Fractures of the occipital bone and base of the skull (basilar bones): These bones are thick and strong, so fractures in these areas indicate a high-intensity impact and meaningfully increase risk of brain injury. Basilar skull fractures that extend into the petrous part of the temporal bone often damage middle and inner ear structures and can impair facial, acoustic, and vestibular nerve function.

- Fractures in infants: The meninges may become trapped in a linear skull fracture with subsequent development of a leptomeningeal cyst and expansion of the original fracture (growing fracture).

Pathophysiology

Brain function may be immediately impaired by direct damage (eg, crush, laceration) of brain tissue. Further damage may occur shortly thereafter from the cascade of events triggered by the initial injury.

TBI of any sort can cause cerebral edema and decrease brain blood flow. The cranial vault is fixed in size (constrained by the skull) and filled by noncompressible CSF and minimally compressible brain tissue; consequently, any swelling from edema or an intracranial hematoma has nowhere to expand and thus increases ICP. Cerebral blood flow is proportional to the cerebral perfusion pressure (CPP), which is the difference between mean arterial pressure (MAP) and mean ICP. Thus, as ICP increases (or MAP decreases), CPP decreases. When CCP falls below 50 mm Hg, the brain may become ischemic. Ischemia and edema may trigger various secondary mechanisms of injury (eg, release of excitatory neurotransmitters, intracellular Ca, free radicals, and cytokines), causing further cell damage, further edema, and further increases in ICP. Systemic complications from trauma (eg, hypotension, hypoxia) can also contribute to cerebral ischemia and are often called secondary brain insults.

Excessive ICP initially causes global cerebral dysfunction. If excessive ICP is unrelieved, it can push brain tissue across the tentorium or through the foramen magnum, causing herniation (see Fig. 174-1 on p. 1657) and increased morbidity and mortality. If ICP increases to equal MAP, CPP becomes zero, resulting in complete brain ischemia and brain death; absent cranial blood flow is objective evidence of brain death (see p. 1667).

Hyperemia and increased brain blood flow may result from concussive injury in adolescents or children. Second impact syndrome is a rare and debated entity defined by sudden increased ICP and death after a second traumatic insult that follows a minor head injury. It is attributed to loss of autoregulation of cerebral blood flow that leads to vascular engorgement, increased ICP, and herniation.

Symptoms and Signs

Initially, most patients with moderate or severe TBI lose consciousness (usually for seconds or minutes), although some patients with minor injuries have only confusion or amnesia (amnesia is usually retrograde and lasts for seconds to a few hours). Young children may simply become irritable. Some patients have seizures, often within the first hour or day. After these initial symptoms, patients may be fully awake and alert, or consciousness and function may be altered to some degree, from mild confusion to stupor to coma. Duration of unconsciousness and severity of obtundation are roughly proportional to injury severity but are not specific.

The **Glasgow Coma Scale** (GCS—see Table 324-2) is a quick, reproducible scoring system to be used during the initial examination to estimate severity of TBI. It is based on eye opening, verbal response, and the best motor response. The lowest total score (3) indicates

[**Table 324-2.** Glasgow Coma Scale*]

likely fatal damage, especially if both pupils fail to respond to light and oculovestibular responses are absent. Higher initial scores tend to predict better recovery. By convention, the severity of head injury is initially defined by the GCS:

• 14 or 15 is mild TBI

• 9 to 13 is moderate TBI

• 3 to 8 is severe TBI

However, the severity and prognosis are predicted more accurately by also considering CT findings and other factors. Some patients with initially moderate TBI and a few patients with initially mild TBI deteriorate. For infants and young children, the Modified Glasgow Coma Scale for Infants and Children is used (see Table 324-3). Because hypoxia and hypotension can decrease the GCS, GCS values after resuscitation from cardiopulmonary insults are more specific for brain dysfunction than values determined before

resuscitation. Similarly, sedating drugs can decrease GCS values and should be avoided before full neurologic evaluation.

Symptoms of various types of TBI may overlap considerably. Symptoms of epidural hematoma usually develop within minutes to several hours after the injury (the period without symptoms is the so-called lucid interval) and consist of increasing headache, decreased level of consciousness, and focal neurologic deficits (eg, hemiparesis). Pupillary dilation with loss of light reactivity usually indicates herniation. Some patients lose consciousness, then have a transient lucid interval, and then gradual neurologic deterioration. Most patients with subdural hematomas have immediate loss of consciousness. Intracerebral hematoma and subdural hematoma can cause focal neurologic deficits such as hemiparesis, progressive decrease in consciousness, or both. Progressive decrease in consciousness may result from anything that increases ICP (eg, hematoma, edema, hyperemia).

[**Table 324-3.** Modified Glasgow Coma Scale for Infants and Children]

Vomiting may indicate increased ICP but is nonspecific. Markedly increased ICP classically manifests as a combination of hypertension (usually with increased pulse pressure), bradycardia, and respiratory depression (Cushing's triad); respirations are usually slow and irregular. Severe diffuse brain injury or markedly increased ICP may cause decorticate or decerebrate posturing. Both are poor prognostic signs.

Transtentorial herniation (see Fig. 174-1 on p. 1657) may result in coma, unilaterally or bilaterally dilated and unreactive pupils, hemiplegia (usually on the side opposite a unilaterally dilated pupil), and Cushing's triad.

Basilar skull fracture may result in leakage of CSF from the nose (CSF rhinorrhea) or ear (CSF otorrhea), blood behind the tympanic membrane (hemotympanum) or in the external ear canal if the tympanic membrane has ruptured, and ecchymosis behind the ear (Battle's sign) or in the periorbital area (raccoon eyes). Loss of smell and hearing is usually immediate, although these losses may not be noticed until the patient regains consciousness. Facial nerve function may be impaired immediately or after a delay. Other fractures of the cranial vault are sometimes palpable, particularly through a scalp laceration, as a depression or step-off deformity. However, blood under the galea aponeurotica may mimic such a step-off deformity.

Patients with chronic subdural hematomas may present with increasing daily headache, fluctuating drowsiness or confusion (which may mimic early dementia), and mild-to-moderate hemiparesis or other focal neurologic deficits.

Long-term symptoms: Amnesia may persist and be both retrograde and anterograde. Post-concussion syndrome, which commonly follows a moderate or severe concussion, includes headache, dizziness, fatigue, difficulty concentrating, variable amnesia, depression, apathy, and anxiety. Commonly smell (and thus taste), sometimes hearing, or rarely vision is altered or lost. Symptoms usually resolve spontaneously over weeks to months.

A range of cognitive and neuropsychiatric deficits can persist after severe and even moderate TBI, particularly if structural damage was significant. Common problems include amnesia, behavioral changes (eg, agitation, impulsivity, disinhibition, lack of motivation), emotional lability, sleep disturbances, and decreased intellectual function.

Late seizures (> 7 days after the injury) develop in a small percentage of patients, often weeks, months, or even years later. Spastic motor impairment, gait and balance disturbances, ataxia, and sensory losses may occur.

A persistent vegetative state (see p. 1665) can result from a TBI that destroys forebrain cognitive functions but spares the brain stem. The capacity for self-awareness and other mental activity is absent; however, autonomic and motor reflexes are preserved, and sleep-wake cycles are normal. Few patients recover normal neurologic function when a persistent vegetative state lasts for 3 mo after injury, and almost none recover after 6 mo.

Neurologic function may continue to improve for a few years after TBI, most rapidly during the initial 6 mo.

Diagnosis

• Initial rapid trauma assessment

• Glasgow coma scale and neurologic examination

• CT

Initial measures: An initial overall assessment of injuries should be done (see p. 3190). Diagnosis and treatment occur simultaneously in seriously injured patients.

A rapid, focused neurologic evaluation is part of the initial assessment, including assessment of the components of the GCS, adequacy of the airway and breathing, and pupillary light response. Patients are ideally assessed before paralytics and sedatives are given. Patients are reassessed at frequent intervals (eg, q 15 to 30 min initially, then q 1 h after stabilization). Subsequent improvement or deterioration helps estimate injury severity and prognosis.

Complete clinical evaluation: Complete neurologic examination is done as soon as the patient is sufficiently stable. Infants and children should be examined carefully for retinal hemorrhages, which may indicate shaken baby syndrome. Funduscopic examination in adults may disclose traumatic retinal detachment and absence of retinal venous pulsations due to elevated ICP, but examination may be normal despite brain injury. Concussion is diagnosed when loss of consciousness or memory lasts < 6 h and symptoms are not explained by brain injury seen on neuroimaging. DAI is suspected when loss of consciousness exceeds 6 h and microhemorrhages are seen on CT. Diagnosis of other types of TBI is made by CT or MRI.

Neuroimaging: Imaging should always be done in patients with more than transiently impaired consciousness, GCS score < 15, focal neurologic findings, persistent vomiting, seizures, a history of loss of consciousness, or clinically suspected fractures. However, a case can be made for obtaining a CT scan of the head in all patients with more than a trivial head injury, because the clinical and medicolegal consequences of missing a hematoma are severe.

Although plain x-rays can detect some skull fractures, they cannot help assess the brain and they delay more definitive brain imaging; thus, plain x-rays are usually not done. CT is the best choice for initial imaging, because it can detect hematomas, contusions, skull fractures (thin cuts are obtained to reveal clinically suspected basilar skull fractures, which may otherwise not be visible), and sometimes DAI. On CT, contusions and acute bleeding appear opaque (dense) compared with brain tissue. Arterial epidural hematomas classically appear as lenticular-shaped opacities over brain tissue, often in the territory of the middle meningeal artery. Subdural hematomas classically appear as crescent-shaped opacities overlying brain tissue. A chronic subdural hematoma appears hypodense compared with brain tissue, whereas a subacute subdural hematoma may have a similar radiopacity as brain tissue (isodense). Isodense subdural hematoma, particularly if bilateral and symmetric, may appear only subtly abnormal. In patients with severe anemia, an acute subdural hematoma may appear isodense with brain tissue. Among individual patients, findings may differ from these classic appearances. Signs of mass effect include sulcal effacement, ventricular and cisternal compression, and midline shift. Absence of these findings does not exclude increased ICP, and mass effect may be present with normal ICP. A shift of > 5 mm from the midline is generally considered to be an indication for surgical evacuation of the hematoma.

MRI may be useful later in the clinical course to detect more subtle contusions and DAI. It is usually more sensitive than CT for the diagnosis of very small acute or isodense subacute and isodense chronic subdural hematomas. Preliminary, unconfirmed evidence suggests that certain MRI findings predict prognosis. Angiography, CT angiography, and magnetic resonance angiography are all useful for the evaluation of vascular injury. For example, vascular injury is suspected when CT findings are inconsistent with the physical examination findings (eg, hemiparesis with a normal or nondiagnostic CT due to suspected evolving ischemia secondary to vascular thrombosis or embolism from a carotid artery dissection).

Prognosis

In the US, adults with severe TBI who are treated have a mortality rate of about 25 to 33%. Mortality is lower with higher GCS scores. Mortality rates are lower in children ≥ 5 yr (≤ 10% with a GCS score of 5 to 7). Children overall do better than adults with a comparable injury.

The vast majority of patients with mild TBI retain good neurologic function. With moderate or severe TBI, the prognosis is not as good but is much better than is generally believed. The most commonly used scale to assess outcome in TBI patients is the Glasgow Outcome Scale. On this scale the possible outcomes are:

• Good recovery (return to previous level of function)

• Moderate disability (capable of self-care)

• Severe disability (incapable of self-care)

• Vegetative (no cognitive function)

• Death

Over 50% of adults with severe TBI have a good recovery or moderate disability. Occurrence and duration of coma after a TBI are strong predictors of disability. Of patients whose coma exceeds 24 h, 50% have major persistent neurologic sequelae, and 2 to 6% remain in a persistent vegetative state at 6 mo. In adults with severe TBI, recovery occurs most rapidly within the initial 6 mo. Smaller improvements continue for perhaps as long as several years. Children have a better immediate recovery from TBI regardless of severity and continue to improve for a longer period of time.

Cognitive deficits, with impaired concentration, attention, and memory, and various personality changes are a more common cause of disability in social relations and employment than are focal motor or sensory impairments. Posttraumatic anosmia and acute traumatic blindness seldom resolve after 3 to 4 mo. Hemiparesis and aphasia usually resolve at least partially, except in the elderly.

Treatment

• For mild injuries discharge and observation

• For moderate and severe injuries optimization of ventilation, oxygenation, and brain perfusion; treatment of complications (eg, increased ICP, seizures, hematomas); and rehabilitation

Multiple noncranial injuries, which are likely with motor vehicle crashes and falls, often require simultaneous treatment. Initial resuscitation of trauma patients is discussed elsewhere (see p. 3190).

At the injury scene, a clear airway is secured and external bleeding is controlled before the patient is moved. Particular care is taken to avoid displacement of the spine or other bones to protect the spinal cord and blood vessels. Proper immobilization should be maintained with a cervical collar and long spine board until stability of the entire spine has been established by appropriate examination and imaging (see p. 3229). After the initial rapid neurologic assessment, pain should be relieved with a short-acting opioid (eg, fentanyl).

In the hospital, after quick initial evaluation, neurologic findings (GCS and pupillary reaction), BP, pulse, and temperature should be recorded frequently for several hours because any deterioration demands prompt attention. Serial GCS and CT results stratify injury severity, which helps guide treatment (see Table 324-4).

The cornerstone of management for all patients is maintenance of adequate ventilation, oxygenation, and brain perfusion to avoid secondary brain insult. Aggressive early management of hypoxia, hypercapnia,

hypotension, and increased ICP helps avoid secondary complications. Bleeding from injuries (external and internal) is rapidly controlled, and intravascular volume is promptly replaced with crystalloid (eg, 0.9% saline) or sometimes blood transfusion to maintain cerebral perfusion. Hypotonic fluids (especially 5% D/W) are contraindicated because they contain excess free water, which can increase brain edema and ICP.

Other complications to check for and to prevent include hyperthermia, hyponatremia, hyperglycemia, and fluid imbalance.

Mild injury: Injury is mild (by GCS score) in 80% of patients who have TBI and present to an emergency department. If there is brief or no loss of consciousness and if patients have stable vital signs, a normal head CT scan, and normal mental and neurologic function, they may be discharged home provided family members or friends can observe them closely for an additional 24 h. These observers are instructed to return patients to the hospital if any of the following develop: decreased level of consciousness, focal neurologic deficits, worsening headache, vomiting, or deterioration of mental function.

[**Table 324-4.** Management of Traumatic Brain Injury Based on Severity of Injury]

Patients who have had loss of consciousness or have any abnormalities in mental or neurologic function and cannot be observed closely after discharge are generally observed in the emergency department or overnight in the hospital, and follow-up CT is done in 4 to 8 h. Patients who have no neurologic changes but minor abnormalities on head CT (eg, small contusions, small subdural hematomas with no mass effect, or punctuate or small traumatic subarachnoid hemorrhage) may need only a follow-up CT within 24 h. With a stable CT and normal neurologic examination results, these patients may be discharged home.

Moderate and severe injury: Injury is moderate in 10% of patients who have TBI and present to an emergency department. They often do not require intubation and mechanical ventilation (unless other injuries are present) or ICP monitoring. However, because deterioration is possible, these patients should be admitted and observed even if head CT is normal.

Injury is severe in 10% of patients who have TBI and present to an emergency department. They are admitted to a critical care unit. Because airway protective reflexes are usually impaired and ICP may be increased, patients are intubated endotracheally while measures are taken to avoid increasing ICP. Close monitoring using the GCS and pupillary response should continue, and CT is repeated, particularly if there is an unexplained ICP rise.

Increased intracranial pressure: Treatment principles for patients with increased ICP include

- Rapid sequence orotracheal intubation
- Mechanical ventilation
- Monitoring of ICP and CCP
- Ongoing sedation as needed
- Maintaining euvolemia and serum osmolality of 295 to 320 mOsm/kg
- For intractable increased ICP, possibly CSF drainage, temporary hyperventilation, decompressive craniotomy, or pentobarbital coma

Patients with TBI who require airway support or mechanical ventilation undergo rapid sequence oral intubation (using paralysis) rather than awake nasotracheal intubation (see Ch. 225), which can cause coughing and gagging and thereby raise the ICP. Drugs are used to minimize the ICP increase when the airway is manipulated—eg, lidocaine 1.5 mg/kg IV 1 to 2 min before giving the paralytic. Etomidate is an excellent induction agent because it has minimal effects on BP; IV dose in adults is 0.3 mg/kg (or 20 mg for an average-sized adult) and in children is 0.2 to 0.3 mg/kg. An alternative, if hypotension is absent and unlikely, is propofol 0.2 to 1.5 mg/kg IV. Succinylcholine 1.5 mg/kg IV is typically used as a paralytic.

Pulse oximetry and ABGs (if possible, end-tidal CO_2) should be used to assess adequacy of oxygenation and ventilation. The goal is a normal $PaCO_2$ level (38 to 42 mm Hg). Prophylactic hyperventilation ($PaCO_2$ 25 to 35 mm Hg) is no longer recommended. The lower $PaCO_2$ reduces ICP by causing cerebral vasoconstriction, but this vasoconstriction also decreases cerebral perfusion, thus potentiating ischemia. Therefore, hyperventilation (target $PaCO_2$ of 30 to 35 mm Hg) is used only during the first several hours and if ICP is unresponsive to other measures.

In patients with severe TBI who cannot follow simple commands, especially those with an abnormal head CT scan, ICP and CPP monitoring (see p. 2246) and control are recommended. The goal is to maintain ICP at < 20 mm Hg and CPP as close as possible to 60 mm Hg. Cerebral venous drainage can be enhanced (thus lowering ICP) by elevating the head of the bed to 30° and by keeping the patient's head in a midline position. If needed, a ventricular catheter can be inserted for CSF drainage to lower the ICP.

Preventing agitation, excessive muscular activity (eg, from delirium), and pain can also help prevent increases in ICP. For sedation, propofol is often used in adults (contraindicated in children) because it has quick onset and very brief duration of action; dose is 0.3 mg/kg/h continuous IV infusion, titrated gradually upward as needed (up to 3 mg/kg/h). An initial bolus is not used. The most common adverse effect is hypotension. Prolonged use at high doses can cause pancreatitis. Benzodiazepines (eg, midazolam, lorazepam) can also be used for sedation, but they are not as rapidly acting as propofol and individual dose-response can be hard to predict. Antipsychotics can delay recovery and should be avoided if possible. Rarely, paralytics may be needed; if so, adequate sedation must be ensured. Opioids are often needed for adequate pain control.

Patients should be kept euvolemic and normosmolar or slightly hyperosmolar (target serum osmolality 295 to 320 mOsm/kg). Osmotic diuretics (eg, mannitol) may be given IV to lower ICP and maintain serum osmolality. However, they should be reserved for patients whose condition is deteriorating or used preoperatively for patients with hematomas. Mannitol 20% solution is given 0.5 to 1 g/kg IV (2.5 to 5 mL/kg) over 15 to 30 min and repeated in a dose ranging from 0.25 to 0.5 g/kg (1.25 to 2.5 mL/kg) given as often as needed (usually q 6 to 8 h); it lowers ICP for a few hours. Mannitol must be used cautiously in patients with severe coronary artery disease, heart failure, renal insufficiency, or pulmonary vascular congestion because mannitol rapidly expands intravascular volume. Because osmotic diuretics increase renal excretion of water relative to Na, prolonged use of mannitol may also result in water depletion and hypernatremia. Furosemide 1 mg/kg IV is also helpful to decrease total body water, particularly when the transient hypervolemia associated with mannitol is to be avoided. Fluid and electrolyte balance should be monitored closely while osmotic diuretics are used. A hypertonic saline solution (usually 2% to 3%) is being studied as another potential osmotic agent to control ICP.

When increased ICP is refractory to other interventions, decompressive craniotomy can be considered. For this procedure, a bone flap is removed (to be replaced later), and duraplasty is done to allow outward brain swelling.

A more involved and currently less popular option for intractable increased ICP is pentobarbital coma. Coma is induced by giving pentobarbital 10 mg/kg over 30 min, 5 mg/kg/h for 3 h, then 1 mg/kg/h maintenance infusion. The dose may be adjusted to suppress bursts of EEG activity, which is continuously monitored. Hypotension is common and managed by giving fluids and, if necessary, vasopressors.

Therapeutic systemic hypothermia has not proved helpful. Corticosteroids are not useful to control ICP and are not recommended; they were associated with a worse outcome in a recent multinational study. A variety of neuroprotective agents are being studied, but thus far, none has demonstrated efficacy in clinical trials.

Seizures: Seizures can worsen brain damage and increase ICP and therefore should be treated promptly. In patients with significant structural injury (eg, larger contusions or hematomas, brain laceration, depressed skull fracture) or a GCS score < 10, a prophylactic anticonvulsant should be considered. If phenytoin is used, a loading dose of 20 mg/kg IV is given (at a maximum rate of 50 mg/min

to prevent cardiovascular adverse effects such as hypotension and bradycardia). The starting maintenance IV dose for adults is 2 to 2.7 mg/kg tid; children require higher doses (up to 5 mg/kg bid for children < 4 yr). Serum levels should be measured to adjust the dose. Duration of treatment depends on the type of injury and EEG results. If no seizures develop within 1 wk, anticonvulsants should be stopped because their value in preventing future seizures is not established. Newer anticonvulsants are under study. Fosphenytoin, a form of phenytoin that has better water solubility, is being used in some patients without central venous access because it decreases the risk of thrombophlebitis when given through a peripheral IV. Dosing is the same as for phenytoin.

Skull fractures: Aligned closed fractures require no specific treatment. Depressed fractures sometimes require surgery to elevate fragments, manage lacerated cortical vessels, repair dura mater, and debride injured brain. Open fractures require debridement. Use of antibiotic prophylaxis is controversial because of limited data on its efficacy and the concern that it promotes drug-resistant strains.

Surgery: Intracranial hematomas may require urgent surgical evacuation to prevent or treat brain shift, compression, and herniation; hence, early neurosurgical consultation is mandatory. However, not all hematomas require surgical removal. Small intracerebral hematomas rarely require surgery. Patients with small subdural hematomas can often be treated without surgery. Factors that suggest a need for surgery include a midline brain shift of > 5 mm, compression of the basal cisterns, and worsening neurologic examination findings. Chronic subdural hematomas may require surgical drainage but much less urgently than acute subdural hematomas. Large or arterial epidural hematomas are treated surgically, but small epidural hematomas that are thought to be venous in origin can be followed with serial CT.

Rehabilitation: When neurologic deficits persist, rehabilitation is needed. Rehabilitation is best provided through a team approach that combines physical, occupational, and speech therapy, skill-building activities, and counseling to meet the patient's social and emotional needs (see also p. 3467). Brain injury support groups may provide assistance to the families of brain-injured patients.

For patients whose coma exceeds 24 h, 50% of whom have major persistent neurologic sequelae, a prolonged period of rehabilitation, particularly in cognitive and emotional areas, is often required. Rehabilitation services should be planned early.

Chapter 325. Spinal Trauma

Introduction

Trauma to the spine may cause injuries involving the spinal cord, vertebrae, or both. Occasionally, the spinal nerves are affected (see p. 1791). The anatomy of the spinal column is reviewed in Ch. 186 (see p. 1800).

Etiology

Cord injury: During a typical year, there are about 11,000 spinal cord injuries in the US. Nearly 48% occur in motor vehicle crashes, and 23% result from falls. The remainder are attributed to violence (14%), sports (9%), and work-related accidents. About 80% of patients are male.

Spinal cord injuries occur when blunt physical force damages the vertebrae, ligaments, or disks of the spinal column, causing bruising, crushing, or tearing of spinal cord tissue, and when the spinal cord is penetrated (eg, by a gunshot or a knife wound). Such injuries can also cause vascular injury with resultant ischemia or hematoma (typically extradural), leading to further damage. All forms of injury can cause spinal cord edema, further decreasing blood flow and oxygenation. Damage may be mediated by excessive release of neurotransmitters from damaged cells, an inflammatory immune response with release of cytokines, accumulation of free radicals, and apoptosis.

Vertebral injury: Fractures may involve the vertebral body, lamina, and pedicles as well as the spinous, articular, articular, and transverse processes. Dislocations typically involve the facets. Subluxation involves ligament rupture without bony injury. In the neck, fractures of the posterior elements and dislocations can damage the vertebral arteries, causing a stroke-like syndrome.

Unstable vertebral injuries are those in which bony and ligamentous integrity is disrupted sufficiently that free movement can occur, potentially compressing the spinal cord or its vascular supply and resulting in marked worsening of neurologic function or pain. Such vertebral movement may occur even with a shift in patient position (eg, for ambulance transport, during initial evaluation). Stable fractures are able to resist such movement.

Specific injuries typically vary with mechanism of trauma. Flexion injuries can cause wedge fractures of the vertebral body or spinous process fractures. Greater flexion force may cause bilateral cervical cervical facet dislocation, or if the force occurs at the level of C1 or C2, odontoid fracture, atlantooccipital or atlantoaxial subluxation, or both fracture and subluxation. Rotational injury can cause unilateral facet dislocation. Extension injury most often causes posterior neural arch fracture. Compression injuries can cause burst fractures of vertebral bodies.

Cauda equina injury: The lower tip of the spinal cord (conus medullaris) is usually at the level of the L1 vertebra. Spinal nerves below this level comprise the cauda equina. Findings in spinal injuries below this level may mimic those of spinal cord injury, particularly conus medullaris syndrome.

Symptoms and Signs

The cardinal sign of cord injury is a discrete injury level in which neurologic function above the injury is intact, and function below the injury is absent or markedly diminished. Specific manifestations depend on the exact level (see
Table 325-1) and whether cord injury is complete or partial. Priapism may occur in the acute phase of spinal cord injury.

Vertebral injury, as with other fractures and dislocations, typically is painful, but patients who are distracted by other painful injuries (eg, long bone fractures) or whose level of consciousness is altered by intoxicants or head injury may not complain of pain.

Complete cord injury: Transection leads to immediate, complete, flaccid paralysis (including loss of anal sphincter tone), loss of all sensation and reflex activity, and autonomic dysfunction below the level of

the injury.

High cervical injury (at or above C5) affects the muscles controlling respiration, causing respiratory insufficiency; ventilator dependence may occur, especially in patients with injuries at or above C3. Autonomic dysfunction from cervical injury can result in bradycardia and hypotension, termed neurogenic shock; unlike in other forms of shock, the skin remains warm and dry. Arrhythmias and BP instability may develop. Pneumonia is a frequent cause of death in people with a high spinal cord injury, especially in those who are ventilator dependent.

Flaccid paralysis gradually changes over hours or days to spastic paralysis with increased deep tendon reflexes due to loss of descending inhibition. Later, if the lumbosacral cord is intact, flexor muscle spasms appear and autonomic reflexes return.

Partial cord injury: Partial motor and sensory loss occurs, and deep tendon reflexes may be exuberant. Motor and sensory loss may be permanent or temporary depending on the etiology; function may be lost briefly due to concussion or more lastingly due to a contusion

[Table 325-1. Effects of Spinal Cord Injury by Location]

or laceration. Sometimes, however, rapid swelling of the cord results in total neurologic dysfunction resembling complete cord injury. Termed spinal shock (not to be confused with neurogenic shock), symptoms resolve over one to several days; residual disability often remains.

Manifestations depend on which portion of the cord is involved; several discrete syndromes are recognized.

Brown-Sequard syndrome results from unilateral hemisection of the cord. Patients have ipsilateral spastic paralysis and loss of postural sense below the lesion, and contralateral loss of pain and temperature sensation.

Anterior cord syndrome results from direct injury to the anterior spinal cord or to the anterior spinal artery. Patients lose motor and pain sensation bilaterally below the lesion. Posterior cord function (vibration, proprioception) is intact.

Central cord syndrome usually occurs in patients with a narrowed spinal canal (congenital or degenerative) after a hyperextension injury. Motor function in the arms is impaired to a greater extent than that in the legs. If the posterior columns are affected, posture, vibration, and light touch are lost. If the spinothalamic tracts are affected, pain, temperature, and, often, light or deep touch are lost. Hemorrhage in the spinal cord from trauma (hematomyelia) is usually confined to the cervical central gray matter, resulting in signs of lower motor neuron damage (muscle weakness and wasting, fasciculations, and diminished tendon reflexes in the arms), which is usually permanent. Motor weakness is often proximal and accompanied by selective impairment of pain and temperature sensation.

Cauda equina lesions: Motor or sensory loss, or both, usually partial, occurs in the distal legs. Sensation is usually diminished in the perineal region (saddle anesthesia). Bowel and bladder dysfunction, either incontinence or retention, may occur. Men may have erectile dysfunction, and women diminished sexual response. Anal sphincter tone is lax, and bulbocavernosus and anal wink reflexes are abnormal. These findings may be similar to those of conus medullaris syndrome, a spinal cord injury.

Complications: Sequelae depend on the severity and level of the injury. Breathing may be impaired if the injury is at or above the C5 segment. Reduced mobility increases the risk of blood clots, UTIs, contractures, atelectasis and pneumonia, and pressure ulcers. Disabling spasticity may develop. Autonomic dysreflexia may occur in response to triggering events such as pain or pressure on the body. Chronic neurogenic pain may manifest as burning or stinging.

Diagnosis

• Consideration of injury in high-risk patients, even those without symptoms

• CT

Spinal cord injuries with trauma are not always obvious. Injury to the spine and spinal cord must be considered in patients with injuries that involve the head, pelvic fractures, penetrating injuries in the area of the spine, in most motor vehicle crashes, in any kind of major blunt injury, and in any injuries related to falling from heights or diving into water.

Injury should also be considered in patients with altered sensorium, localized spinal tenderness, painful distracting injuries, or compatible neurologic deficits. Motor function is tested in all extremities. Sensation testing should involve both light touch (posterior column function), pinprick (anterior spinothalamic tract), and position sense. Identification of the sensory level is best done by testing from distal to proximal and by testing thoracic roots on the back to avoid being misled by the cervical cape. Priapism indicates spinal cord damage. Rectal tone may be decreased, and deep tendon reflexes may be exuberant or absent.

When spinal cord injury is suspected, the spine is immediately immobilized. Traditionally, plain x-rays are taken of any possibly injured areas. CT is done of areas that appear abnormal on x-rays and areas at risk of injury based on clinical findings. However, CT is being used increasingly as the primary imaging study for spinal trauma because it has better diagnostic accuracy and, at many trauma centers, can be obtained rapidly. MRI helps identify the type and location of cord injury; it is the most accurate study for imaging the spinal cord and other soft tissues but may not be immediately available. Manifestations of injury may be characterized using the ASIA (American Spinal Injury Association) Impairment Scale or a similar instrument (see
Table 325-2).

Prognosis

Transected or degenerated nerves in the cord do not recover, and functional damage is permanent. Compressed nerve tissue can recover its function. Return of a movement or sensation during the first week after injury heralds a favorable recovery. Dysfunction remaining after 6 mo is likely to be permanent.

[**Table 325-2.** Spinal Injury Impairment Scale*]

Treatment

• Immobilization

• Maintenance of oxygenation and perfusion

• Supportive care

• Sometimes surgical stabilization

• Possibly methylprednisolone for blunt injuries

• Long-term symptomatic care and rehabilitation

Immediate care: An important goal is to prevent secondary injury to the spine or spinal cord. In unstable injuries, flexion or extension of the spine can contuse or transect the cord. Thus, when injured people are moved, inappropriate handling can precipitate paraplegia, quadriplegia, or even death from spinal injury. Patients who may have a spinal injury should have the spine immobilized immediately; the neck is held straight manually (in line stabilization) during endotracheal intubation. As soon as possible, the spine is fully immobilized on a firm, flat, padded backboard or similar surface to stabilize the position without excessive pressure. A rigid collar should be used to immobilize the cervical spine. Patients with thoracic or lumbar spine injuries can be carried prone or supine. Those with cervical cord damage that could induce respiratory difficulties should be carried supine, with attention to maintaining a patent airway and avoiding chest constriction. Transfer to a trauma center is desirable.

Medical care should be directed at avoiding hypoxia and hypotension, both of which can further stress the injured cord. In cervical injuries higher than C5, intubation and respiratory support are usually needed.

Large doses of corticosteroids, started within 8 h after spinal cord injury, may improve the outcome in blunt injuries, but this finding has not been firmly established. Methylprednisolone 30 mg/kg IV over 1 h, followed by 5.4 mg/kg/h for the next 23 h is the recommended regimen. Injuries are treated with rest, analgesics, and muscle-relaxing drugs with or without surgery until swelling and local pain have subsided. Additional general treatment for trauma patients is discussed elsewhere (see p. 3190).

Unstable injuries are immobilized until bone and soft tissues have healed to ensure proper alignment; surgery with fusion and internal fixation is sometimes needed. Patients with incomplete cord injuries can have significant neurologic improvement after decompression. In contrast, in complete injury, return of useful neurologic function below the level of the injury is unlikely. Thus, surgery aims to stabilize the spine to allow early mobilization. The timing of surgery in incomplete lesions is debatable. Early surgery (eg, within 24 h) may have a better neurologic outcome and allows for earlier mobilization and rehabilitation. For complete injuries, surgery is sometimes done in the first few days, but it is not clear that this is necessary.

Nursing care includes preventing urinary and pulmonary infections and pressure ulcers—eg, by turning the immobile patient every 2 h (on a Stryker frame when necessary). Deep venous thrombosis prophylaxis is required. An inferior vena cava filter could be considered in immobile patients.

Long-term care: Drugs effectively control spasticity in some patients. Oral agents such as baclofen 5 mg po tid (maximum, 80 mg during a 24-h period) and tizanidine 4 mg po tid (maximum, 36 mg during a 24-h period) are typically used for spasticity occurring after spinal cord injury. Intrathecal baclofen 50 to 100 µg once/day may be considered in patients in whom oral drugs are ineffective.

Rehabilitation is needed to help people recover as fully as possible (see p. 3467). Rehabilitation, best provided through a team approach, combines physical therapies, skill-building activities, and counseling to meet social and emotional needs. The rehabilitation team is best directed by a physician with training and expertise in rehabilitation (physiatrist); it usually includes nurses, social workers, nutritionists, psychologists, physical and occupational therapists, recreational therapists, and vocational counselors (see also Ch. 350).

Physical therapy focuses on exercises for muscle strengthening, passive stretch exercises to prevent contractures, and appropriate use of assistive devices such as braces, a walker, or a wheelchair that may be needed to improve mobility. Strategies for controlling spasticity, autonomic dysreflexia, and neurogenic pain are taught. Occupational therapy focuses on redeveloping fine motor skills. Bladder and bowel management programs teach toileting techniques, which may require intermittent catheterization. A bowel regimen, involving timed stimulation with laxatives, is often needed.

Vocational rehabilitation involves assessing both fine and gross motor skills, as well as cognitive capabilities, to determine the likelihood for meaningful employment. The vocational specialist then helps identify possible work sites and determines need for assistive equipment and workplace modifications. Recreation therapists use a similar approach in identifying and facilitating participation in hobbies, athletics, and other activities.

Emotional care aims to combat the depersonalization and the almost unavoidable depression that occur after losing control of the body. Emotional care is fundamental to the success of all other components of rehabilitation and must be accompanied by efforts to educate the patient and encourage active involvement of family and friends.

Treatments to promote nerve regeneration are under study. Such treatments include injections of autologous, incubated macrophages; epidural administration of BA-210, an experimental drug that may be neuroprotective and may stimulate nerve growth; and oral administration of HP-184 for treatment of chronic spinal cord injury. Also, one trial aims to determine optimal timing of surgery. Stem cell research is in its infancy; some early animal studies have shown promising results.

Spinal Cord Injury in Children

Although children < 10 yr have the lowest rate of spinal cord injuries, such injuries are not rare. In children < 8 yr, cervical spine injuries occur most commonly above C4; in those > 8 yr, injuries at C5 to C8 are more common. Of increasing importance has been the recognition of spinal cord injury without evidence of radiologic abnormality (SCIWORA). This type of injury occurs almost exclusively in children and is related to direct spinal cord traction, spinal cord concussion, and vascular injury.

Diagnosis

Spinal cord injury should be suspected in any child that has been in a motor vehicle crash, has fallen from a height ≥ 3 m, or has had a submersion injury. SCIWORA is suspected in children who have even transient symptoms of neurologic dysfunction or lancinating pains down the spine or extremities and a mechanism of injury compatible with spinal cord injury.

Treatment

Treatment is similar to that in adults, with immobilization and attention to the adequacy of oxygenation, ventilation, and circulation. Treatment may also include high-dose corticosteroids (same weight-based dose as for adults). Children with significant spinal cord trauma should be transferred to a pediatric trauma center as soon as possible.

Chapter 326. Facial Trauma

Introduction

Patients with trauma to the face and head require evaluation of all structures; injuries often occur in combination (for dental trauma, see p. 524; for tympanic membrane trauma, see p. 453; and for eye trauma, see p. 3235).

External Ear Trauma

Trauma to the external ear may result in hematoma, laceration, avulsion, or fracture.

Subperichondral hematomas: Blunt trauma to the pinna may cause a subperichondrial hematoma; the accumulation of blood between the perichondrium and cartilage renders all or part of the pinna a shapeless, reddish purple mass. Because the perichondrium supplies blood to the cartilage, infection, abscess formation, or avascular necrosis of the cartilage may follow. The resultant destruction causes the cauliflower ear characteristic of wrestlers and boxers.

Treatment consists of evacuating the clot through an incision and preventing reaccumulation of the hematoma with through-and-through ear sutures over dental gauze rolls or insertion of a Penrose drain plus a pressure dressing. Because these injuries are prone to infection, an oral antibiotic effective against staphylococci (eg, cephalexin 500 mg tid) is given for 5 days.

Lacerations: If lacerations of the pinna penetrate the cartilage and skin on both sides, the skin margins are sutured; then the cartilage is splinted externally with benzoin-impregnated cotton, and a protective dressing is applied. If there is sufficient skin to fully cover the cartilage, the cartilage should be repaired. Otherwise, external splinting suffices. Oral antibiotics are given as for a hematoma.

Avulsions: Complete or partial avulsions are repaired by an otolaryngologist, facial plastic surgeon, or plastic surgeon.

Trauma secondary to mandibular fractures: Forceful blows to the mandible may be transmitted to the anterior wall of the ear canal (posterior wall of the glenoid fossa). Displaced fragments from a fractured anterior wall may cause stenosis of the canal and must be reduced or removed surgically after a general anesthetic is given.

Fractures of the Jaw and Contiguous Structures

Blunt facial trauma can fracture the jaw and other bones of the midface. Symptoms depend on the location of the fracture. A dental x-ray or CT scan is diagnostic. Treatment may include surgery and/or external fixation.

Fractures of the lower jaw (mandible) are suspected in patients with post-traumatic malocclusion or focal swelling and tenderness over a segment of the mandible. Other clues include defects (stepoff) of the dental occlusal surface, alveolar ridge disruptions, and anesthesia in the distribution of the inferior alveolar or mental nerve. Some fractures result in palpable instability. Fractures of the mandibular condyle usually cause preauricular pain, swelling, and limited opening of the mouth (trismus). With a unilateral condylar fracture, the jaw deviates to the affected side when the mouth is opened.

Fractures of the midface, which includes the area from the superior orbital rim to the maxillary teeth, can cause irregularity in the smooth contour of the cheeks, malar eminences, zygomatic arch, or orbital rims. Infraorbital nerve anesthesia, enophthalmos, or diplopia suggests an orbital floor fracture. An injury near the orbit requires an eye examination, including, at least, assessment of visual acuity, pupils, and extraocular movements. Trismus and a defect on palpation of the zygomatic arch suggest zygomatic arch fracture. The Le Fort classification (see Fig. 326-1) can be used to describe midface fractures. Traumatic malocclusion and upper alveolar ridge fractures may suggest a maxillary fracture that involves the occlusal surface. Brain injury and fractured cervical vertebrae are possible when trauma has been severe enough to fracture facial bones. In major

impact injuries, hemorrhage and edema due to a facial fracture may compromise the airway.

A panoramic dental x-ray is preferred for an isolated mandibular fracture. Fine-cut CT (1-mm slices) is done in axial and coronal planes to diagnose facial fractures.

Treatment

An oral endotracheal airway may be required to maintain airway patency in patients with hemorrhage, edema, or significant tissue

[Fig. 326-1. Le Fort classification of midface fractures.]

disruption. Definitive facial fracture management is complex and may include internal fixation.

Tooth socket fractures: Fractures through a tooth socket are open fractures. They require antibiotic prophylaxis (typically with a broad-spectrum antibiotic that is particularly effective against anaerobes, such as penicillin) given orally as a liquid or parenterally.

Mandible fractures: For a fractured mandible, treatment ranges from soft diet alone to maxillomandibular fixation (wiring the jaw shut), rigid open fixation, or both. If fixation is available within the first few hours after injury, closure of any lip or oral lacerations should be delayed until the fracture has been reduced. For maxillomandibular fixation, metal bars (arch bars) are attached to the buccal surface of the upper and lower teeth and then wired to each other after correct occlusion has been established. Patients with maxillomandibular fixation should always carry wire cutters in case of vomiting. Fixation may need to last several weeks. Eating is restricted to liquids, pureed foods, and supplements. Because only part of the teeth surfaces can be brushed, control of plaque formation, infection, and halitosis is accomplished using a 60-sec rinse with 30 mL of chlorhexidine 0.12% every morning and evening. Jaw-opening exercises usually help restore function after fixation is discontinued.

Condylar fractures may require only 2 to 3 wk of maxillomandibular fixation, followed by a soft diet. However, severely displaced, bilaterally fractured condyles may require open reduction and fixation. Condylar fractures in children should not be rigidly immobilized because ankylosis and abnormal facial development may result. Flexible (elastic) fixation for 5 to 10 days is usually sufficient.

Midface fractures: Fractures of the midface are treated surgically if they cause malocclusion, enophthalmos, diplopia, infraorbital nerve anesthesia, or unacceptable cosmetic deformity. Surgical treatment usually consists of internal stabilization using fine screws and plates. Surgery can often be delayed until swelling subsides, particularly if the indication for surgery is not clear.

Fractures of the Nose

Fractures of the nasal bones or cartilaginous injury may result in swelling, point tenderness, hypermobility, crepitus, epistaxis, and periorbital bruising. Diagnosis is usually clinical. Treatment may include reduction, stabilization through internal packing, and splinting. A septal hematoma is drained without delay.

The nasal bones are the most frequently fractured facial bones because of their central location and protrusion. Depending on the mechanism of injury, fractures of the maxilla, orbit, or cribriform plate and injury to the nasolacrimal ducts may also occur.

Complications include cosmetic deformity and functional obstruction. Septal hematoma may lead to avascular or septic necrosis of the cartilage with resultant deformity. Cribriform plate fracture may cause a CSF leak, with increased risk of meningitis or brain abscess.

Symptoms and Signs

Facial trauma resulting in epistaxis may indicate a nasal fracture. Other symptoms and signs include obvious or subtle nasal deformity, swelling, point tenderness, crepitus, and instability. Lacerations,

ecchymosis (nasal and periorbital), septal deviation, and nasal obstruction may be present. Septal hematoma appears as a purplish bulge on the septum. CSF rhinorrhea appears as clear drainage but may be mixed with blood, making it difficult to identify.

Diagnosis

• Examination

Diagnosis is based on physical examination. Plain x-rays of an uncomplicated nasal fracture are not helpful because their sensitivity and specificity are poor. If other facial fractures or complications are suspected, CT of facial bones is done.

Treatment

• Symptomatic care

• For deformities, delayed reduction

• For septal hematomas, immediate drainage

Immediate treatment includes symptomatic control with ice and analgesics. Reduction is needed only for fractures causing clinically visible deformity or nasal airway obstruction. The end-point of reduction is determined by clinical appearance or improved airway. Reduction is usually deferred for 3 to 5 days after injury to allow swelling to subside but should take place within 2 to 3 wk of the injury, before bony callous formation. Nasal fractures in adults may be reduced after a local anesthetic is given; children require general anesthesia. A blunt elevator is passed through the nares and placed under the depressed nasal bone, which is lifted anteriorly and laterally while pressure is applied to the other side of the nose to bring the nasal dorsum to the midline. The nose may be stabilized with internal packing (consisting of antibiotic-impregnated strip gauze) placed high within the nasal vestibule, as well as with external splinting. Internal packing is left in place for 4 to 7 days; external splinting is left for 7 to 14 days. Antibiotic prophylaxis effective against staphylococci is required for the duration of nasal packing, to decrease the risk of toxic shock syndrome.

Cartilaginous injuries often do not require reduction. In the rare circumstance that a deformity persists after swelling subsides, reduction and splinting after a local anesthetic is given are usually sufficient.

Septal hematomas must be immediately incised and drained to prevent infection and cartilage necrosis. Septal fractures are difficult to hold in position and often require septal surgery later. Patients with cribriform plate fractures and CSF leak require hospital admission with bedrest and placement of a lumbar drain for 5 days. If the CSF leak does not resolve, surgical repair of the skull base may be required.

Temporal Bone Fractures

Temporal bone fractures can occur after severe blunt trauma to the head and sometimes involve structures of the ear, causing hearing loss, vertigo, balance disturbance, or facial paralysis.

Temporal bone fractures are suggested by Battle's sign (postauricular ecchymosis) and bleeding from the ear. Bleeding may come from the middle ear (hemotympanum) through a ruptured tympanic membrane or from a fracture line in the ear canal. A hemotympanum makes the tympanic membrane appear blue-black. CSF otorrhea indicates a communication between the middle ear and the subarachnoid space.

Temporal bone fractures have been classified by orientation with respect to the long axis of the petrous portion of the temporal bone. Longitudinal fractures make up 70 to 90% of temporal bone fractures, and transverse fractures make up 10 to 30%. Some fractures may have characteristics of both patterns. Longitudinal fractures can extend through the middle ear and rupture the tympanic membrane; they cause facial paralysis in 20% of cases and may cause hearing loss (usually conductive). Transverse fractures cross the fallopian canal and otic capsule, causing facial paralysis in about 40% of patients and

sometimes hearing loss (usually sensorineural) and vestibular dysfunction (eg, vertigo, balance disturbance). Rarely, fluctuating sensorineural hearing loss and vestibular dysfunction occur with temporal bone fracture and may be due to a perilymph fistula. Immediate complete facial paralysis may indicate a severed or crushed facial nerve, whereas delayed-onset complete facial paralysis usually indicates edema within an intact nerve.

Diagnosis

• CT

• Assessment of hearing and facial nerve function

If a temporal bone fracture is suspected, immediate CT of the head with special attention to the temporal bone is recommended. The Weber and Rinne tuning fork tests can be done during the initial physical examination in conscious patients to help differentiate between conductive and sensorineural hearing loss. However, formal audiometric examination is required for all patients with temporal bone fractures. If facial paralysis is present, electrical testing of the facial nerve is warranted.

Treatment

• Management of facial nerve injury, hearing loss, vestibular dysfunction, and CSF leakage

If immediate facial nerve paralysis occurs with loss of electrical response, surgical exploration may be warranted. Delayed-onset or incomplete facial paralysis almost always resolves with conservative management, including use of corticosteroids, which are gradually tapered.

Conductive hearing loss requires ossicular chain reconstruction several weeks to months after the injury. Good results can be expected. When sensorineural hearing loss occurs, it is typically permanent, and there are no medical or surgical therapies available to improve hearing. However, in the rare case of fluctuating sensorineural hearing loss, an exploratory tympanotomy to search for a perilymph fistula may be indicated.

When vestibular dysfunction results from perilymph fistula, repair may reduce severity and frequency of vertiginous episodes. When dysfunction results from injury to the vestibular nerve or vestibular labyrinth, few interventions can improve outcome. Symptoms may subside when benzodiazepines are used. More lasting improvement may occur with vestibular rehabilitation.

Patients who have a temporal bone fracture and CSF otorrhea should be hospitalized because meningitis is a risk. The leak usually stops spontaneously within a few days, although a lumbar drain or surgical closure of the defect is occasionally required.

Chapter 327. Eye Injuries

Introduction

(See also Retinal Detachment on p. 617.)

Common causes of eye injury include domestic accidents (eg, during hammering or exposure to household chemicals or cleaners), assault, car battery explosions, sporting injuries (including air- or paint pellet-gun injuries), and motor vehicle crashes (including air-bag injuries). Injury may be to the eyeball (globe), surrounding soft tissues (including muscles, nerves, and tendons), or bones of the orbit.

General evaluation should include the following:

- Tests of visual acuity
- Range of extraocular motion
- Pupillary appearance and responses
- Intraocular pressure determination
- Visual fields to confrontation
- Depth of anterior chamber
- Location and depth of lid and conjunctival lacerations and of foreign bodies
- Presence of anterior chamber or vitreous hemorrhage, cataract, or red reflex
- Retinal examination

Detailed examination of the anterior segment, lens, and anterior vitreous is best done with a slit lamp. Detailed examination of the posterior vitreous and the retina is best done with indirect ophthalmoscopy, usually by an ophthalmologist. Indications include clinical suspicion of traumatic cataracts, vitreous abnormalities (eg, hemorrhage, foreign body), and retinal abnormalities; clinical suspicion may be based on injury mechanism, absence of the red reflex, or retinal abnormalities visible with direct ophthalmoscopy. About 15 to 30 min before this examination, the pupil is usually dilated with 1 drop of cyclopentolate 1% and 1 drop of phenylephrine 2.5%. If an intraocular or orbital foreign body or an orbital fracture is suspected, CT is done.

Use of eye guards, goggles, or special eyeglasses, such as those constructed of polycarbonate lenses in a wrap-around polyamide frame, is a simple precaution that greatly reduces the risk of injury.

When eye drops are prescribed, each dose includes only one drop.

Ocular Burns

Thermal burns: The blink reflex usually causes the eye to close in response to a thermal stimulus. Thus, thermal burns tend to affect the eyelid rather than the conjunctiva or cornea. Eyelid burns should be cleansed thoroughly with sterile isotonic saline solution followed by application of an antimicrobial ointment (eg, bacitracin bid). Most thermal burns affecting the conjunctiva or cornea are mild and heal without significant sequellae. They are treated with topical antibiotics and corticosteroids.

Chemical burns: Burns of the cornea and conjunctiva can be serious, particularly when strong acid or alkali is involved. They are a true emergency; treatment must begin immediately.

Burns should be irrigated with copious amounts of water or with 0.9% saline if available. The eye may be anesthetized with one drop of proparacaine 0.5%, but irrigation should not be delayed and should last for

at least 30 min. In acid and alkali burns, some experts suggest 1 to 2 h of irrigation; others recommend that the pH of the conjunctiva be measured with expanded pH paper (which measures over a wide range of pH) and irrigation continued until pH is normal.

After irrigation, the conjunctival fornices should be examined for chemical embedded in the tissue and swept with a swab to remove trapped particles. The superior fornices are exposed by using double eyelid eversion (ie, pushing the fornix downward until its mucosal surface is visible using a swab inserted under the everted eyelid).

Chemical iritis is suspected in a patient with photophobia (deep eye pain with exposure to light) that develops hours or days after a chemical burn and is diagnosed by finding flare and WBCs in the anterior chamber on slit-lamp examination. Chemical iritis is treated by instilling a long-acting cycloplegic (eg, a single dose of homatropine 2% or 5% or scopolamine 0.25% solution). Corticosteroid drops (eg, prednisolone 1% qid) may be given by an ophthalmologist. Used inappropriately, topical corticosteroids can result in corneal perforation after chemical burns and should be used only by an ophthalmologist. Corneal epithelial defects are treated by applying an antibiotic ointment (eg, erythromycin 0.5%). Topical anesthetics should be avoided after initial irrigation; significant pain may be treated with acetaminophen with or without oxycodone.

Severe chemical burns require treatment by an ophthalmologist to save vision and prevent complications such as uveitis, perforation of the globe, and lid deformities. Patients with severe conjunctival hyperemia, ciliary flush (prominent conjunctival injection around the limbus), true photophobia (ie, not just sensitivity to light), avascular areas of conjunctiva, or loss of conjunctival or corneal epithelium as demonstrated by fluorescein staining should be examined by an ophthalmologist within 24 h.

Corneal Abrasions and Foreign Bodies

Corneal abrasions are self-limited, superficial epithelial defects.

The most common conjunctival and corneal injuries are foreign bodies and abrasions. Improper use of contact lenses can damage the cornea. Superficial foreign bodies often spontaneously exit the cornea in the tear film, occasionally leaving a residual abrasion, but other foreign bodies remain on or within the cornea. Sometimes, a foreign body trapped under the upper lid causes a vertical corneal abrasion that worsens with blinking. Intraocular penetration can occur with seemingly minor trauma, particularly with foreign bodies resulting from high-speed machines (eg, drills, saws, anything with a metal-on-metal mechanism), hammering, or explosions. Infection generally does not develop from a corneal injury. However, if intraocular penetration is not recognized, infection within the eye (endophthalmitis), although somewhat rare, may develop.

Symptoms and Signs

Symptoms and signs of abrasion or foreign body include foreign body sensation, tearing, redness, and occasionally discharge. Vision is rarely affected (other than by tearing).

Diagnosis

• Slit-lamp examination, usually with fluorescein staining

After an anesthetic (eg, 2 drops of proparacaine 0.5%) is instilled into the conjunctiva, each lid is everted, and the entire conjunctiva and cornea are inspected with a binocular lens (loupe) or a slit lamp. Fluorescein staining (see p. 538) with cobalt light illumination renders abrasions and nonmetallic foreign bodies more apparent. Patients with a high-risk intraocular injury or (more rarely) visible globe perforation undergo CT to rule out intraocular foreign body and complete examination by an ophthalmologist, including slit-lamp examination and indirect ophthalmoscopy with eye dilation.

Treatment

• For surface foreign bodies, irrigation or removal with a small needle

• For corneal abrasions, antibiotic ointment and pupillary dilation

• For intraocular foreign bodies, surgical removal

After an anesthetic is instilled into the conjunctiva, clinicians can remove conjunctival foreign bodies by irrigation or lift them out with a moist sterile cotton applicator. A corneal foreign body that cannot be dislodged by irrigation may be lifted out carefully on the point of a sterile spud or of a 25- or 27-gauge hypodermic needle under loupe or, preferably, slit-lamp magnification; the patient must be able to stare without moving the eye during removal. Steel or iron foreign bodies remaining on the cornea for more than a few hours may leave a rust ring on the cornea that also requires removal under slit-lamp magnification by scraping or using a low-speed rotary burr; removal is usually done by an ophthalmologist.

Abrasions: For all abrasions, an antibiotic ointment (eg, bacitracin/polymyxin B or ciprofloxacin 0.3% qid for 3 to 5 days) is used. Contact lens wearers with corneal abrasions require an antibiotic with optimal antipseudomonal coverage (eg, ciprofloxacin 0.3% ointment qid). For symptomatic relief of larger abrasions (eg, area > 10 mm^2), the pupil is also dilated with a short-acting cycloplegic (eg, one drop cyclopentolate 1% or homatropine 5%). Eye patches may increase risk of infection and are usually not used, particularly for an abrasion caused by a contact lens or an object that may be contaminated with soil or vegetation. Ophthalmic corticosteroids tend to promote the growth of fungi and reactivation of herpes simplex virus and are contraindicated. Continued use of topical anesthetics can impair healing and is thus contraindicated.

The corneal epithelium regenerates rapidly; even large abrasions heal within 1 to 3 days. A contact lens should not be worn for 5 to 7 days. Follow-up examination by an ophthalmologist 1 or 2 days after injury is wise, especially if a foreign body was removed with a needle or spud.

Intraocular foreign bodies: Intraocular foreign bodies require immediate surgical removal by an ophthalmologist. Systemic and topical antimicrobials (effective against *Bacillus cereus* if the injury involved contamination with soil or vegetation) are indicated; they include ceftazidime 1 g IV q 12 h, in combination with vancomycin 15 mg/kg IV q 12 h and moxifloxacin 0.5% ophthalmic solution q 1 to 2 h. Ointment should be avoided if the globe is lacerated. A protective shield (such as a Fox shield or the bottom third of a paper cup) is placed and taped over the eye to avoid inadvertent pressure that could extrude ocular contents through the penetration site. Tetanus prophylaxis is indicated after open globe injuries. As with any laceration of the globe, vomiting, which can increase intraocular pressure, should be prevented. If nausea occurs, an antiemetic is given.

Eye Contusions and Lacerations

Consequences of blunt trauma to the eye range from eyelid to orbital injury.

Eyelids: Eyelid contusions (which result in black eyes) are more cosmetically than clinically significant, although more serious injuries may sometimes accompany them and should not be overlooked. Uncomplicated contusions are treated with ice packs to inhibit swelling during the first 24 to 48 h, followed by hot compresses to aid absorption of the hematoma.

Minor lid lacerations not involving the lid margin or tarsal plate may be repaired with nylon (or, in children, plain gut) 6-0 or 7-0 sutures. Lid-margin lacerations are best repaired by an ophthalmic surgeon to ensure accurate apposition and to avoid a notch in the contour. Major lid lacerations, which include those of the medial portion of the lower or upper eyelid (possibly involving the lacrimal canaliculus), through-and-through lacerations, those in which the patient has ptosis, and those that expose orbital fat or involve the tarsal plate, should also be repaired by an ophthalmic surgeon.

Globe: Trauma may cause the following:

• Conjunctival, anterior chamber, and vitreous hemorrhage

• Retinal hemorrhage, edema, or detachment

• Laceration of the iris

• Cataract

• Dislocated lens

• Glaucoma

• Globe rupture (laceration)

Evaluation can be difficult when massive lid edema or laceration is present. Even so, unless the need for immediate eye surgery is obvious (necessitating evaluation by an ophthalmologist as soon as possible), the lid is opened, taking care not to exert inward pressure, and as complete an examination as possible is conducted. At a minimum, the following are noted:

• Visual acuity

• Pupillary responses

• Extraocular movements

• Anterior chamber depth or hemorrhage

• Presence of red reflex

An analgesic or, after obtaining any surgical consent, an anxiolytic may be given to facilitate examination. Gentle and careful use of eyelid retractors or an eyelid speculum makes it possible to open the lids. If a commercial instrument is not available, the eyelids can be separated with makeshift retractors fashioned by bending the ends of paper clips 180°. Globe laceration should be suspected with any of the following:

• A corneal or scleral laceration is visible.

• Aqueous humor is leaking.

• The anterior chamber is very shallow (eg, making the cornea appear to have folds) or very deep (due to rupture posterior to the lens).

• The pupil is irregular.

If globe laceration is suspected, measures that can be taken before an ophthalmologist is available consist of applying a protective shield (see above) and combating possible infection with systemic antimicrobials as for intraocular foreign bodies (see p. 3236). Topical antibiotics are avoided. Vomiting, which can increase intraocular pressure (IOP) and contribute to extravasation of ocular contents, is suppressed using antiemetics as needed. Because fungal contamination of open wounds is dangerous, corticosteroids are contraindicated until after wounds are closed surgically. Tetanus prophylaxis is indicated after open globe injuries. Very rarely, after laceration of the globe, the uninjured, contralateral eye becomes inflamed (sympathetic ophthalmia—see p. 612) and may lose vision to the point of blindness unless treated. The mechanism is an autoimmune reaction; corticosteroid drops can prevent the process.

Anterior chamber hemorrhage (hyphema): This injury may be followed by recurrent bleeding, glaucoma, and blood staining of the cornea, any of which may result in permanent vision loss. Symptoms are of associated injuries unless the hyphema is large enough to obstruct vision. Direct inspection typically reveals layering of blood or the presence of clot or both in the anterior chamber. Layering is seen as a meniscus-like blood level in the lower part of the anterior chamber. Microhyphema, a less severe

form, may be detectable by direct inspection as haziness in the anterior chamber or by slit-lamp examination as suspended RBCs.

An ophthalmologist should attend to the patient as soon as possible. The patient is placed on bed rest with the head elevated 30° and is given an eye shield to protect the eye from further trauma (see p. 3237). Patients who are at high risk of recurrent bleeding (eg, those with large hyphemas, bleeding diatheses, anticoagulant use, or sickle cell disease), who have IOP that is difficult to control, or who are not likely to adhere to recommended treatment may be hospitalized. Oral and topical NSAIDs are contraindicated because they may contribute to recurrent bleeding.

IOP can rise acutely (within hours, usually in patients with sickle cell disease or trait) or months to years later. Thus, IOP is monitored daily for several days and then regularly over subsequent weeks and months and if symptoms develop (eg, eye ache, decreased vision, nausea—similar to symptoms of acute angle-closure glaucoma). If pressure rises, timolol 0.5% bid, brimonidine 0.2% or 0.15% bid, or both are given. Response to treatment is determined by pressure, often checked every 1 or 2 h until controlled or until a significant rate of reduction is demonstrated; thereafter, it is usually checked once or twice daily. Mydriatic drops (eg, scopolamine 0.25% tid or atropine 1% tid for 5 days) and topical corticosteroids (eg, prednisolone acetate 1% 4 to 8 times/day for 2 to 3 wk are often given. Administration of aminocaproic acid 50 to 100 mg/kg po q 4 h (not exceeding 30 g/day) for 5 days may reduce recurrent bleeding. In these cases, miotic or mydriatic drugs must be given by an ophthalmologist. Rarely, recurrent bleeding with secondary glaucoma requires surgical evacuation of the blood.

Blowout fracture: Blowout fracture occurs when blunt trauma forces the orbital contents through the most fragile portion of the orbital wall, typically the floor. Medial and roof fractures also can occur. Symptoms include diplopia, enophthalmos, inferiorly displaced globe, hypesthesia of the cheek and upper lip (from infraorbital nerve injury), and subcutaneous emphysema. Epistaxis, lid edema, and ecchymosis may occur. Diagnosis is best made using CT. If diplopia or cosmetically unacceptable enophthalmos persists beyond 2 wk, surgical repair is indicated. Patients should be told to avoid blowing the nose. Using a topical vasoconstrictor for 2 to 3 days may alleviate epistaxis.

Posttraumatic Iridocyclitis

(Traumatic Anterior Uveitis; Traumatic Iritis)

Posttraumatic iridocyclitis is an inflammatory reaction of the uvea and iris, typically developing within 3 days of blunt eye trauma.

Symptoms of posttraumatic iridocyclitis include tearing, throbbing ache and redness of the eye, photophobia, and blurred vision. Diagnosis is by history, symptoms, and slit-lamp examination, which typically reveals flare (due to an increase in protein content of the aqueous humor from the inflammatory exudate) and WBCs in the anterior chamber. Treatment involves a cycloplegic (usually scopolamine 0.25% tid, or homatropine 5% tid). Topical corticosteroids (eg, prednisolone acetate 1% 4 to 8 times/day) are often used to shorten symptom duration.

Chapter 328. Genitourinary Tract Trauma

Introduction

The GU tract can be injured by blunt trauma (eg, motor vehicle crashes, falls) or penetrating trauma (eg, gunshot or stab wounds). Some injuries are caused during surgical procedures. Symptoms and signs are often subtle or nonspecific; therefore, diagnosis requires a high level of suspicion. Whenever urinalysis shows any hematuria in a patient with trauma, GU injury should be presumed until proved otherwise. Depending on the suspected site of injury, imaging studies, most often contrast-enhanced CT, are typically used to make a diagnosis. General evaluation of the trauma patient is discussed elsewhere (see p. 3190).

Bladder Trauma

Bladder injuries are caused by either blunt or penetrating trauma to the lower abdomen, pelvis, or perineum. Blunt trauma is the more common mechanism, usually by a sudden deceleration, such as in a high-speed motor vehicle crash or fall, or from an external blow to the lower abdomen. The most frequently accompanying injury is a pelvic fracture, occurring in > 95% of bladder ruptures caused by blunt trauma. Other concomitant injuries include long bone fractures and CNS and chest injuries. Penetrating injuries, most often gunshot wounds, account for about 25% of bladder injuries.

The bladder is the most frequently injured organ during pelvic surgery. Such injuries can occur during transurethral surgery, colon resection, or gynecologic procedures (most commonly abdominal hysterectomy, cesarean section, pelvic mass excision). Predisposing factors include scarring from prior surgery or radiation therapy, inflammation, and extensive tumor burden.

Bladder injuries are classified as contusions or ruptures based on the extent of injury seen radiographically. Bladder ruptures can be extraperitoneal, intraperitoneal, or both.

Complications of bladder injuries include infection, fistula, incontinence, and bladder instability. Mortality with bladder rupture approaches 20% from the concomitant organ injuries rather than the bladder injury.

Symptoms and Signs

Symptoms may include suprapubic pain and inability to void; signs may include suprapubic tenderness, distention, and, in the case of intraperitoneal rupture, peritoneal signs.

Diagnosis

• Retrograde cystography

Diagnosis is suspected on the basis of history and physical examination findings and hematuria (gross or microscopic). Confirmation is by retrograde cystography using plain film x-rays or CT. Plain film x-rays are accurate, but CT also helps delineate concomitant intra-abdominal injuries. If urethral disruption is suspected in a male, retrograde catheter placement is avoided, pending results of urethrography.

Treatment

• Catheter drainage

• Sometimes surgical repair

All penetrating trauma and intraperitoneal ruptures from blunt trauma require surgical exploration and repair. Contusions require only catheter drainage until gross hematuria resolves. Extraperitoneal ruptures require only catheter drainage if urine is draining freely and the bladder neck is spared. If the bladder neck is involved, surgical exploration and repair are required.

Genital Trauma

Most genital trauma occurs in men and includes injury to the testes, scrotum, and penis. Genital mutilation of women by removing the clitoris, which is done in some cultures, is a form of genital trauma and child abuse (see p. 3067).

Most **testicular injuries** result from blunt trauma; penetrating testicular injuries are less common. Testicular injuries are classified as contusions or, if the tunica albuginea is disrupted, as ruptures.

Scrotal injury may be caused by penetrating trauma, burns, and avulsions.

Penile injuries have diverse mechanisms. Zipper injuries are common. Penile fractures, which are ruptures of the corpus cavernosum, occur most often when the penis is forcibly bent during sexual activity; urethral injury may also be present. Amputations (usually self-inflicted or from clothing trapped by heavy machinery) and strangulations (usually from constricting penile rings used to enhance erections) are additional mechanisms. Penetrating injuries, including animal bites and gunshot wounds, are less common and may also involve the urethra.

Complications of genital injuries include erectile dysfunction, hypogonadism, infection, tissue loss, and urethral scarring.

Symptoms and Signs

Symptoms after a direct scrotal blow are usually scrotal pain and swelling. Signs may include scrotal discoloration and a tender, firm scrotal mass that fails to transilluminate, suggesting a hematocele. Scrotal penetration suggests the possibility of testicular involvement. Often the examination is limited by patient discomfort. Penile fracture typically results in a cracking sound, immediate pain, marked swelling and ecchymosis, and usually visible deformity.

Diagnosis

• Clinical evaluation

• Sometimes ultrasonography or retrograde urethrography

Diagnosis of external scrotal and penile injury is made clinically. Clinical diagnosis of testicular contusion and rupture can be difficult because the degree of injury may be out of proportion to the physical findings, so patients with blunt testicular injury typically require scrotal ultrasonography. Most penile injuries are evident on physical examination. An x-ray with urethral contrast (retrograde urethrogram)

[
Fig. 328-1. Zipper removal from penile skin.]

should be done for any patient with penile fracture or penetrating penile injury because of the high incidence of coexisting urethral injury.

Treatment

• Sometimes surgical repair

Patients with penetrating testicular injuries or clinical or sonographic characteristics that suggest testicular rupture require surgical exploration and repair. Similarly, all penile fractures and penetrating injuries should be surgically explored and the defects repaired. Penile amputations should be repaired by microsurgical reimplantation if the amputated segment is viable. Strangulation injuries can usually be managed simply by removing the constricting agent. Zippers should be removed (see Fig. 328-1).

Renal Trauma

The kidney is injured in up to 10% of patients who sustain significant abdominal trauma. About 65% of

GU injuries involve the kidney.

Most renal injuries (85 to 90% of cases) occur from blunt trauma, typically due to motor vehicle crashes, falls, or assaults. Most injuries are low grade. The most common accompanying injuries are to the head, CNS, spleen, and liver. Penetrating injuries usually result from gunshot wounds. Such patients usually have multiple intra-abdominal injuries, most commonly to the liver, intestine, and spleen.

Renal injuries are classified according to severity into 5 grades (see Table 328-1).

Diagnosis

• Urinalysis and Hct

• If moderate or severe injury is suspected, contrast-enhanced CT

Diagnosis should be suspected in any patient with the following:

• Penetrating injury between the mid chest and lower abdomen

• Significant deceleration injury

• Direct blow to the flank

In such patients, hematuria strongly suggests renal injury; other indicators include the following:

• Seat belt marks

• Diffuse abdominal tenderness

• Flank contusions

• Lower rib fractures

Patients who develop hematuria after relatively minor trauma may have a previously undiagnosed congenital renal anomaly.

Laboratory testing should include Hct and urinalysis. When imaging is indicated, contrast-enhanced CT is usually used to determine the grade of renal injury and identify accompanying intra-abdominal trauma and complications, including retroperitoneal hemorrhage and urinary extravasation. Patients with blunt trauma and microscopic hematuria usually have minor renal injuries that almost never require surgical repair; thus, CT is usually unnecessary. CT is indicated in blunt trauma with any of the following:

• The mechanism involves a fall from a significant height or a high-speed motor vehicle crash

• Gross hematuria

• Microscopic hematuria with hypotension (systolic pressure < 90 mm Hg)

• Clinical signs potentially suggesting severe renal injury (eg, flank contusion, seat belt marks, lower rib or vertebral transverse process fractures)

For **penetrating trauma**, CT is indicated for all patients with microscopic or gross hematuria. Rarely, angiography is indicated to assess persistent or delayed bleeding and may be combined with selective arterial embolization.

Pediatric renal injuries are evaluated similarly, except that all children with blunt trauma in whom urinalysis shows > 50 RBCs/high-power field require imaging.

[Table 328-1. Grades of Renal Injury]

Treatment

• Strict bed rest

• Surgical repair for moderate or severe injuries and some penetrating injuries

Most blunt renal injuries, including all grade 1 and 2 and most grade 3 and 4 injuries, can be safely treated without surgery. Patients require strict bed rest until gross hematuria has resolved. Surgical repair is required for patients with the following:

• Persistent bleeding (ie, enough to necessitate treatment for hypovolemia)

• Expanding perinephric hematoma

• Renal pedicle avulsion

Penetrating trauma usually requires surgical exploration, although observation may be appropriate for patients in whom the renal injury has been accurately staged by CT, BP is stable, and no associated intra-abdominal injuries require surgery.

Ureteral Trauma

Most ureteral injuries are iatrogenic. Procedures that most often injure a ureter include ureteroscopy, hysterectomy, low anterior colon resection, and abdominal aneurysm repair. Noniatrogenic ureteral injury accounts for only about 1 to 3% of all GU trauma. It usually results from gunshot wounds and rarely from stab wounds. In children, avulsion injuries are more common. Complications include ureteral stricture, obstruction, or both; peritoneal or retroperitoneal urinary leakage; and fistula (eg, ureterovaginal, ureterocutaneous) formation.

Diagnosis

• Imaging, exploratory surgery, or both

Diagnosis is suspected on the basis of history and requires a high index of suspicion, because symptoms are nonspecific and hematuria is absent in > 30% of patients. Diagnosis is confirmed by imaging (eg, CT with contrast, IVU), exploratory surgery, or both. Flank pain and fever are the main symptoms of otherwise occult injuries.

Treatment

• For minor, nephrostomy tube or ureteral stent

• For major, surgical repair

All injuries require intervention. A diverting percutaneous nephrostomy tube or cystoscopic placement of a ureteral stent is often sufficient for minor injuries (eg, contusions or partial transections). Complete transection or avulsion injuries typically require reconstructive techniques, including ureteral reimplantation, primary ureteral anastomosis, anterior bladder flap, ileal interposition, and, as a last resort, autotransplantation.

Urethral Trauma

Urethral injury usually occurs in men. Most major urethral injury is due to blunt trauma. Penetrating urethral trauma is less common, occurring mainly from gunshot wounds, or, alternatively, from inserting objects into the urethra during sexual activity or because of psychiatric illness.

Urethral injuries are classified as contusions, partial disruptions, or complete disruptions, and they may involve the posterior or anterior urethral segments. Posterior urethral injuries occur almost exclusively with pelvic fractures. Anterior urethral injuries are often consequences of a perineal straddle injury from a fall, perineal blow, or motor vehicle crash.

Complications include stricture formation, infection, erectile dysfunction, and incontinence.

Symptoms and Signs

Symptoms include pain with voiding and inability to void. Blood at the urethral meatus is the most important sign of a urethral injury. Additional signs include perineal, scrotal, and penile ecchymosis, edema, or both, and a high-riding prostate on rectal examination.

Diagnosis

• Retrograde urethrography

In any patient with suggestive symptoms or signs, the diagnosis is confirmed by retrograde urethrography, which should be done before catheterization. Urethral catheterization in a patient with an undetected significant urethral injury may potentiate urethral disruption (eg, convert a partial disruption to a complete disruption).

Treatment

• Usually urethral catheterization or suprapubic cystostomy

Contusions can be safely treated with an indwelling transurethral catheter for 10 days. Partial disruptions are best treated with bladder drainage via suprapubic cystostomy. In selected cases of posterior partial disruptions, primary urethral realignment using catheterization may be attempted; if successful, this approach limits subsequent urethral strictures.

Complete disruptions are treated with bladder drainage via suprapubic cystostomy. This option is simplest and can be used safely in all patients. Definitive surgery is deferred for about 8 to 12 wk until the urethral scar tissue has stabilized and the patient has recovered from any accompanying injuries.

Selected penetrating urethral injuries and blunt urethral injuries that occur with penile fractures may be sutured primarily.

Chapter 329. Burns

Introduction

(See also Ocular Burns on p. 3235 and Caustic Ingestion on p. 3335.)

Burns are injuries of skin or other tissue caused by thermal, radiation, chemical, or electrical contact. Burns are classified by depth (1st-degree, superficial and deep partial-thickness, and full-thickness) and percentage of total body surface area (BSA) involved. Complications and associated problems include hypovolemic shock, inhalation injury, infection, scarring, and contractures. Patients with large burns (> 20% BSA) require fluid resuscitation. Treatment for burn wounds includes topical antibacterials, regular cleaning, elevation, and sometimes skin grafting. Intensive rehabilitation, consisting of range-of-motion exercises and splinting, is often necessary.

Burns cause between 3000 and 4000 deaths/yr in the US and about 2 million physician visits.

Etiology

Thermal burns may result from any external heat source (flame, hot liquids, hot solid objects, or, occasionally, steam). Fires may also result in toxic smoke inhalation (see Sidebar 329-1 and p. 3334).

Radiation burns most commonly result from prolonged exposure to solar ultraviolet radiation (sunburn —see p. 676) but may result from prolonged or intense exposure to other sources of ultraviolet radiation (eg, tanning beds) or from exposure to sources of x-ray or other nonsolar radiation (see also p. 3252).

Chemical burns may result from strong acids, strong alkalis (eg, lye, cement), phenols, cresols, mustard gas, phosphorus, and certain petroleum products (eg, gasoline, paint thinner). Skin and deeper tissue necrosis caused by these agents may progress over several hours.

Electrical burns (see also p. 3248) result from heat generation and electroporation of cell membranes associated with massive current of electrons. Electrical burns may cause extensive deep tissue damage to electrically conductive tissues, such as muscles and nerves, despite minimal apparent cutaneous injury.

Events associated with a burn (eg, jumping from a burning building, being struck by debris, motor vehicle crash) may cause other injuries. Abuse should be considered in young children and elderly patients with burns.

Pathophysiology

Burns cause protein denaturation and thus coagulative necrosis. Around the coagulated tissue, platelets aggregate, vessels constrict, and marginally perfused tissue (known as the zone of stasis) can extend the injury. Around the zone of stasis, tissue is hyperemic and inflamed.

Damage to the normal epidermal barrier allows bacterial invasion and external fluid loss; damaged tissues often become edematous, further enhancing volume loss. Heat loss can be significant because thermoregulation of the damaged dermis is absent, particularly in wounds that are exposed.

Sidebar 329-1 Smoke Inhalation

Burns and smoke inhalation often occur together but may occur separately. When smoke is inhaled, toxic products of combustion injure airway tissues. Hot smoke usually burns only the pharynx because the incoming gas cools quickly. A common exception is steam, which carries much more heat energy than smoke and thus can also burn the lower airways (below the glottis). Many toxic chemicals produced in routine house fires (eg, hydrogen chloride, phosgene, sulfur dioxide, toxic aldehydes, ammonia) injure lower airways chemically. Some toxic products of combustion, such as carbon monoxide (see p. 3334) or

cyanide, impair cellular respiration systemically.

Upper airway injury usually causes symptoms within minutes but occasionally over several hours; upper airway edema may cause stridor. Lower airway injury may also occur with upper airway injury and usually causes symptoms (eg, oxygenation problems highlighted by increasing O_2 requirements or decreases in lung compliance) 24 h or later.

Smoke inhalation is suspected in patients with respiratory symptoms, a history of confinement in a burning environment, or carbonaceous sputum. Perioral burns and singed nasal hair may also be clues.

Diagnosis of upper airway injury is by endoscopy (laryngoscopy or bronchoscopy) that is adequate to see the upper airways and trachea fully and that shows edema or soot in the airways; however, injury occasionally develops after an early normal study. Endoscopy is done as soon as possible, usually with a flexible fiberoptic scope typically followed by endotracheal intubation in patients with significant findings. Diagnosis of lower airway injury is by chest x-ray and oximetry or ABGs, but abnormalities may develop only days later. Cyanide and carbon monoxide toxicity should be considered; carboxyhemoglobin levels are measured in patients with significant smoke inhalation.

All patients at risk of smoke inhalation injury are given 100% O_2 by face mask initially. Patients with airway obstruction or respiratory distress require endotracheal intubation or another artificial airway and mechanical ventilation (see p. 2279). Patients with edema or significant soot in the upper airways require intubation as soon as possible because intubation becomes more difficult as edema increases. Bronchoscopy is usually done at the same time as intubation. Patients with lower airway injury may require supplemental O_2, bronchodilators, and other supportive measures.

Burn depth: First-degree burns are limited to the epidermis.

Partial-thickness (also called 2nd-degree) burns involve part of the dermis (see Plate 76) and can be superficial or deep.

Superficial partial-thickness burns involve the papillary (more superficial) dermis. These burns heal within 1 to 2 wk and rarely scar. Healing occurs from epidermal cells lining sweat gland ducts and hair follicles; these cells grow to the surface, then migrate across the surface to meet cells from neighboring glands and follicles.

Deep partial-thickness burns involve the deeper dermis and take ≥ 2 wk to heal. Healing occurs only from hair follicles, and scarring is common and may be severe.

Full-thickness (3rd-degree) burns extend through the entire dermis and into the underlying fat (see Plate 75). Healing occurs only from the periphery; these burns, unless small, require excision and skin grafting.

Complications

Burns cause both systemic and local complications. The major factors contributing to systemic complications are breakdown of skin integrity and fluid loss. Local complications include eschars and contractures and scarring.

Systemic: The greater the percentage of BSA involved, the greater the risk of developing systemic complications. Risk factors for severe systemic complications and mortality include all of the following:

• Burns of > 40% of BSA

• Age > 60 yr or < 2 yr

• Presence of simultaneous major trauma or smoke inhalation

The most common systemic complications are hypovolemia and infection.

Hypovolemia, causing hypoperfusion of burned tissue and sometimes shock, can result from fluid losses due to burns that are deep or that involve large parts of the body surface; whole-body edema from escape of intravascular volume into the interstitium and cells also develops. Hypoperfusion of burned tissue also may result from direct damage to blood vessels or from vasoconstriction secondary to hypovolemia.

Infection, even in small burns, is a common cause of sepsis and mortality, as well as local complications. Impaired host defenses and devitalized tissue enhance bacterial invasion and growth. The most common pathogens are streptococci and staphylococci during the first few days and gram-negative bacteria after 5 to 7 days; however, flora are almost always mixed.

Metabolic abnormalities may include hypoalbuminemia that is partly due to hemodilution (secondary to replacement fluids) and partly due to protein loss into the extravascular space through damaged capillaries. Dilutional electrolyte deficiencies can develop; they include hypomagnesemia, hypophosphatemia, and hypokalemia. Metabolic acidosis may result from shock. Rhabdomyolysis or hemolysis can result from deep thermal or electrical burns of muscle or from muscle ischemia due to constricting eschars. Rhabdomyolysis causing myoglobinuria or hemolysis causing hemoglobinuria can lead to acute tubular necrosis and renal failure.

Hypothermia may result from large volumes of cool IV fluids and extensive exposure of body surfaces to a cool emergency department environment, particularly in patients with extensive burns.

Ileus is common after extensive burns.

Local: Eschar is stiff, dead tissue caused by deep burns. A circumferential eschar, which completely encircles a limb (or sometimes the torso), is constricting. A constricting eschar limits tissue expansion in response to edema; instead, tissue pressure increases, eventually causing local ischemia. The ischemia threatens viability of limbs and digits, and an eschar around the thorax can compromise respiration.

Scarring and contractures result from spontaneous healing of deep burns; if the burn is located near joints or in the hands, feet, or perineum, function can be severely impaired. Infection can increase scarring. Keloids form in some patients with burns, especially in patients with darker skin.

Symptoms and Signs

Wound symptoms and signs depend on burn depth:

- **First-degree burns:** These burns are red, blanch markedly and widely with light pressure, and are painful and tender. Vesicles or bullae do not develop.

- **Superficial partial-thickness burns:** These burns blanch with pressure and are painful and tender. Vesicles or bullae develop within 24 h. The bases of vesicles and bullae are pink and subsequently develop a fibrinous exudate.

- **Deep partial-thickness burns:** These burns may be white, red, or mottled red and white. They do not blanch and are less painful and tender than more superficial burns. A pinprick is often interpreted as pressure rather than sharp. Vesicles or bullae may develop; these burns are usually dry.

- **Full-thickness burns:** These burns may be white and pliable, black and charred, brown and leathery, or bright red because of fixed Hb in the subdermal region. Pale full-thickness burns may simulate normal skin except the skin does not blanch to pressure. Full-thickness burns are usually anesthetic or hypoesthetic. Hairs can be pulled easily from their follicles. Vesicles and bullae usually do not develop. Sometimes features that differentiate full-thickness from deep partial-thickness burns take a few days to develop.

Diagnosis

• Clinical assessment of burn extent and depth

• Laboratory testing and chest x-ray in admitted patients

Location and depth of burned areas are recorded on a burn diagram. Burns with an appearance compatible with either deep partial-thickness or full-thickness are presumed to be full-thickness until differentiation is possible.

The percentage of BSA involved is calculated; only partial-thickness and full-thickness burns are included in this calculation. For adults, the percentage BSA for parts of the body is estimated by the rule of nines (see Fig. 329-1); for smaller scattered burns, estimates can be based on the size of the patient's entire hand (not the palm only), which is about 1% of BSA. Children have proportionally larger heads and smaller lower extremities, so the percentage BSA is more accurately estimated using the Lund-Browder chart (see Fig. 329-1).

In patients who require hospitalization, Hb and Hct, serum electrolytes, BUN, creatinine, albumin, protein, phosphate, and ionized Ca should be measured. ECG, urinalysis for myoglobin, and a chest x-ray are also required. Myoglobinuria (suggesting hemolysis or

[**Fig. 329-1.** (A) Rule of nines (for adults) and (B) Lund-Browder chart (for children) for estimating extent of burns.]

rhabdomyolysis) is suggested by urine that is grossly dark or that tests positive for blood on dipstick in the absence of microscopic RBCs. These tests are repeated as needed. Muscle compartments are evaluated in patients with myoglobinuria.

Infection is suggested by wound exudate, impaired wound healing, or systemic evidence of infection (eg, feeding intolerance, decrease in platelet count, increase in serum glucose level). Fever and WBC count elevation are common in burns without infection, and therefore are unreliable signs of developing sepsis. If the diagnosis is unclear, infection can be confirmed by biopsy; cultures from the wound surface or exudate are unreliable.

Treatment

• IV fluids for burns > 10% BSA

• Wound cleaning, dressing, and serial assessment

• Supportive measures

• Transfer or referral of selected patients to burn centers

• Surgery and physical therapy for deep partial-thickness and full-thickness burns

Initial treatment: Treatment begins in the prehospital setting. The first priorities are the same as for any injured patient: ABC (airway, breathing, and circulation). An airway is provided, ventilation is supported, and possible associated smoke inhalation is treated with 100% O_2 (see Sidebar 329-1). Ongoing burning is extinguished, and smoldering and hot material is removed. All clothing is removed. Chemicals, except powders, are flushed with water; powders should be brushed off before wetting. Burns caused by acids, alkalis, or organic compounds (eg, phenols, cresols, petrochemicals) are flushed with copious amounts of water continuing for at least 20 min after nothing of the original solution seems to remain.

Intravenous fluids: IV fluids are given to patients in shock or with burns > 10% BSA. A 14- to 16-gauge venous cannula is placed in 1 or 2 peripheral veins through unburned skin if possible. Venous cutdown, which has a high risk of infection, is avoided.

Initial fluid volume is guided by treatment of clinically evident shock (see p. 2296). If shock is absent, fluid administration aims to replace the predicted deficit and supply maintenance fluids. The Brooke formula (2 mL/kg/%BSA burned) or Parkland formula (4 mL/kg/%BSA burned) is used to estimate the fluid volume needs in the first 24 h after the burn (not after presentation to the hospital). Both formulas use lactated Ringers' solution.

For example, in a 100-kg man with a 50% total BSA burn, fluid volume by the Brooke formula would be 2 × 100 × 50 = 10,000 mL. Half of the volume, 5 L, is given in the first 8 h after injury as a constant infusion, and the remaining 5 L is given over the following 16 h. In practice, these formulas are only a starting point, and infusion rates are adjusted based on clinical response. Urine output, typically measured with an indwelling catheter, is the usual indicator of clinical response; the goal is to maintain output at ≥ 30 mL/h in adults and between 0.5 and 1.0 mL/kg/h in children. When giving typical large volumes of fluid, it is also important to avoid fluid overload and consequent heart failure. Clinical parameters, including urine output and signs of shock or heart failure, are recorded at least hourly on a flow chart.

Some clinicians give colloid after 12 h to patients who have larger burns, are very young or very old, or have heart disease and require large fluid volumes.

If urine output is inadequate despite administration of a large volume of crystalloid, consultation with a burn center is necessary. Such patients may respond to an infusion of colloid or other measures. Patients with inadequate urine output despite administration of a large volume of crystalloid are at risk of resuscitation complications including compartment syndromes.

For patients of any age with rhabdomyolysis, fluid should be given to maintain urine output between 0.5 and 1 mL/kg/h. Some authorities recommend alkalinizing the urine by adding 50 mEq $NaHCO_3$ (one 50-mL ampule of 8.4% solution) to a liter of IV fluid.

Initial wound care: After analgesia, the wound is cleaned with soap and water, and all loose debris is removed. Water should be room temperature or warmer to avoid inducing hypothermia. Blisters, except for small ones on palms, fingers, and soles, are debrided. In patients who are to be transferred to a burn center, clean dry dressings can be applied (burn creams interfere with burn wound assessment at the receiving facility), and patients are kept warm and relatively comfortable with IV opioids.

After the wound is cleaned and is assessed by the final treatment provider, burns can be treated topically. For shallow partial-thickness burns, topical treatment alone is usually adequate. All deep partial-thickness burns and full-thickness burns should ultimately be treated with excision and grafting, but in the interim, topical treatments are appropriate.

Topical treatment may be with antimicrobial salves (eg, 1% silver sulfadiazine), commercial dressings incorporating silver (eg, sustained-release nanocrystalline silver dressings), or biosynthetic wound dressings (also called artificial skin products). Topical salves must be changed daily, and silver sulfadiazine may induce transient leukopenia. Silver-impregnated dressings must be kept moist but can be changed only every 3 days. Artificial skin products are not changed routinely but can result in underlying purulence necessitating removal, particularly with deeper wounds. Burned extremities should be elevated.

A tetanus toxoid booster (0.5 mL sc or IM) is given to patients with all but minor burns who have been previously fully vaccinated and who have not received a booster within the past 5 yr. Patients whose booster was more remote or who had not received a full vaccine series are given tetanus immune globulin 250 units IM and concomitant active vaccination (see p. 1299).

Escharotomy (incision of the eschar) of constricting eschars may be necessary to allow adequate expansion of the thorax or perfusion of an extremity. However, constricting eschars rarely threaten extremity viability during the first few hours, so if transfer to a burn center can occur within that time, escharotomy can typically be deferred until then.

Supportive measures: Hypothermia is treated (see p. 3274), and pain is relieved. Opioids (eg,

morphine, fentanyl) are always given IV. Treatment of electrolyte deficits may require supplemental Ca, Mg, K, or phosphate (PO_4).

Nutritional support (see p. 20) is indicated for patients with burns > 20% BSA or preexisting undernutrition. Support with a feeding tube begins as soon as possible. Parenteral support is rarely necessary.

Hospitalization and referral: After initial treatment and stabilization, the need for hospitalization is assessed. Inpatient treatment, optimally at a burn center, is required for

- Full-thickness burns > 1% BSA
- Partial-thickness burns > 5% BSA
- Burns of the hands, face, feet, or perineum (partial-thickness or deeper)

In addition, hospitalization may be necessary if

- Patients are < 2 yr or > 60 yr.
- Adherence to home care measures is likely to be poor or difficult (eg, if continuous elevation of the hands or feet, usually difficult at home, is required).

Many experts recommend that all burns, except for 1st-degree burns < 1% BSA, be treated by experienced physicians and that brief inpatient care be strongly considered for all burns > 2% BSA. Maintaining adequate analgesia and exercise can be difficult for many patients and caregivers.

Infection: Prophylactic antibiotics are not given.

Initial empiric antibiotic treatment for apparent infection during the first 5 days should target staphylococci and streptococci (eg, with vancomycin for inpatients). Infections that develop after 5 days are treated with broad-spectrum antibiotics that are effective against gram-positive and gram-negative bacteria. Antibiotic selection is subsequently adjusted based on culture and sensitivity results.

Surgery: Surgery is indicated for burns that are not expected to heal within 2 wk, which includes most deep partial-thickness burns and all full-thickness burns. Eschars are removed as soon as possible, ideally within 3 days to prevent sepsis and facilitate early wound grafting, which shortens hospitalization and improves the functional result. If burns are extensive and life threatening, the largest eschars are removed first to close as much burn area as early as possible.

After excision, grafting proceeds ideally using partial-thickness autografts (the patient's skin), which are permanent. Autografts can be transplanted as sheets (solid pieces of skin) or meshed grafts (sheets of donor skin that are stretched to cover a larger area by making multiple, regularly spaced, small incisions). Meshed grafts are used in areas where appearance is less of a concern when burns are > 20% BSA and donor skin is scarce. Meshed grafts heal with an uneven gridlike appearance, sometimes with excessive hypertrophic scarring.

When burns are > 40% BSA and the supply of autograft material appears insufficient, an artificial dermal regeneration template can be used as temporary coverage. Allografts (viable skin usually from cadaver donors) or xenografts (eg, pig skin) can also be used temporarily; they are rejected, sometimes within 10 to 14 days. Both types of temporary coverage must ultimately be replaced with autografts.

Fasciotomy is done when edema within a muscle compartment elevates compartment pressure > 30 mm Hg.

Physical and occupational therapy: Physical and occupational therapy are begun at admission to help minimize scarring and contractures, particularly for body surfaces with high skin tension and frequent movement (eg, face, hands), and to optimize function. Active and passive range-of-motion exercises

become easier as the initial edema subsides; they are done once or twice daily. After grafting, exercises are usually suspended for 3 days, then resumed. Extremities affected by deep partial-thickness burns or full-thickness burns are splinted in functional positions as soon as possible and kept splinted continuously (except during exercise) until the graft has been placed, healing has occurred, or both.

Outpatient treatment: Outpatient treatment includes keeping burns clean and, to the extent possible, keeping the affected body part elevated. Dressings should be changed daily for burns treated with topical salves. The salve is applied and then covered with a dry nonadherent gauze dressing and compression wraps. Silver dressings should be changed every 3 to 7 days. Dressing change simply involves removing the older dressing and replacing it with new one. Biosynthetic wound dressings should not be changed in the absence of purulence. Biosynthetic dressings should simply be covered with dry gauze, which is changed daily.

Outpatient follow-up visits are scheduled as needed depending on burn severity (eg, for very minor burns, initial visit within 24 h, then subsequent visits every 5 to 7 days). Visits include debridement if indicated, reassessment of burn depth, and evaluation of the need for physical therapy and grafting. Patients should return earlier if they note signs of infection, such as increasing redness extending from the wound edges, increasing purulence and pain, or a change in the appearance of the wound with development of black or red spots. Should these signs occur, medical evaluation should ensue urgently. Outpatient treatment is acceptable for minor burn-wound cellulitis in healthy patients aged 2 to 60 yr; hospitalization is indicated for other infections.

Chapter 330. Electrical and Lightning Injuries

Introduction

Electricity may be from generated sources (eg, from high- or low-voltage power lines) or atmospheric (lightning).

Electrical Injuries

Electrical injury is damage caused by generated electrical current passing through the body. Symptoms may include skin burns, damage to internal organs and other soft tissues, cardiac arrhythmias, and respiratory arrest. Diagnosis is by history, clinical criteria and selective laboratory testing. Treatment is supportive, with aggressive care for severe injuries.

Although accidental electrical injuries encountered in the home (eg, touching an electrical outlet or getting shocked by a small appliance) rarely result in significant injury or sequelae, accidental exposure to high voltage causes about 400 deaths annually in the US.

Pathophysiology

Traditional teaching is that the severity of electrical injury depends on Kouwenhoven's factors:

• Type of current (direct [DC] or alternating [AC])

• Voltage and amperage (measures of current strength)

• Duration of exposure (longer exposure increases injury severity)

• Body resistance

• Pathway of current (which determines the specific tissue damaged)

However, electrical field strength, a newer concept, seems to predict injury severity more accurately.

Kouwenhoven's factors: AC changes direction frequently; it is the current usually supplied by household electrical outlets in the US and Europe. DC flows in the same direction constantly; it is the current supplied by batteries. Defibrillators and cardioverters usually deliver DC current. How AC affects the body depends largely on frequency. Low-frequency (50- to 60-Hz) AC is used in US (60 Hz) and European (50 Hz) households; it can be more dangerous than high-frequency AC and is 3 to 5 times more dangerous than DC of the same voltage and amperage. Low-frequency AC causes extended muscle contraction (tetany), which may freeze the hand to the current's source, prolonging exposure. DC is most likely to cause a single convulsive contraction, which often forces the person away from the current's source.

Usually, for both AC and DC, the higher the voltage (V) and amperage, the greater the ensuing electrical injury (for the same duration of exposure). Household current in the US is 110 V (standard electrical outlet) to 220 V (large appliance, such as a dryer). High-voltage (> 500 V) currents tend to cause deep burns, and low-voltage (110 to 220 V) currents tend to cause muscle tetany and freezing to the current's source. The threshold for perceiving DC current entering the hand is about 5 to 10 milliamperes (mA); for AC at 60 Hz, the threshold is about 1 to 10 mA. The maximum amperage that can cause flexors of the arm to contract but that allows release of the hand from the current's source is called the let-go current. Let-go current varies with weight and muscle mass. For an average 70-kg man, let-go current is about 75 mA for DC and about 15 mA for AC.

Low-voltage 60-Hz AC traveling through the chest for a fraction of a second can cause ventricular fibrillation at amperage as low as 60 to 100 mA; for DC, about 300 to 500 mA are required. If current has a direct pathway to the heart (eg, via a cardiac catheter or pacemaker electrodes), < 1 mA (AC or DC) can cause ventricular fibrillation.

Amount of dissipated heat energy equals amperage^2 × resistance × time; thus, for any given current and duration, tissue with the highest resistance tends to suffer the most damage. Body resistance (measured in ohms/cm^2) is provided primarily by the skin. Skin thickness and dryness increase resistance; dry, well-keratinized, intact skin averages 20,000 to 30,000 ohms/cm^2. For a thickly calloused palm or sole, resistance may be 2 to 3 million ohms/cm^2; for moist, thin skin, resistance is about 500 ohms/cm^2. Resistance for punctured skin (eg, cut, abrasion, needle puncture) or moist mucous membranes (eg, mouth, rectum, vagina) may be as low as 200 to 300 ohms/cm^2. If skin resistance is high, much electrical energy may be dissipated at the skin, resulting in large skin burns where the energy contacts the skin but less internal damage. If skin resistance is low, skin burns are less extensive or absent, with more electrical energy transmitted to internal structures. Thus, the absence of external burns does not predict the absence of electrical injury, and the severity of external burns does not predict the severity of electrical injury.

Damage to internal tissues depends also on their resistance and additionally on current density (current per unit area; energy is concentrated when the same current flows through a smaller area). For example, as electrical energy flows in an arm (primarily through lower-resistance tissues, eg, muscle, vessels, nerves), current density increases at joints because a significant proportion of the joint's cross-sectional area consists of higher-resistance tissues (eg, bone, tendon), which decreases the area of lower-resistance tissue; thus, damage to the lower-resistance tissues tends to be most severe at joints.

The current's pathway through the body determines which structures are injured. Because AC current continually reverses direction, the commonly used terms "entry" and "exit" are inappropriate; "source" and "ground" are more precise. The hand is the most common source point, followed by the head. The foot is the most common ground point. Current traveling between arm and arm or between arm and foot is likely to traverse the heart, possibly causing arrhythmia. This current tends to be more dangerous than current traveling from one foot to the other. Current to the head may damage the CNS.

Electrical field strength: Electrical field strength determines the degree of tissue injury. For instance, 20,000 volts (20 kV) distributed across the body of a man who is about 2 m (6 ft) tall result in a field strength of about 10 kV/m. Similarly, 110 volts, if applied only to 1 cm (eg, across a young child's lip), result in a similar field strength of 11 kV/m; this relationship is why such a low-voltage injury can cause the same severity of tissue injury as some high-voltage injuries applied to a larger area. Conversely, when considering voltage rather than electrical field strength, minor or trivial electrical injuries technically could be classified as high voltage. For example, the shock received from shuffling across a carpet in the winter involves thousands of volts but produces inconsequential injury.

The electrical field effect can cause cell membrane damage (electroporation) even when the energy is insufficient to cause any thermal damage.

Pathology: Application of low electrical field strength causes an immediate, unpleasant feeling (being "shocked") but seldom results in serious or permanent injury. Application of high electrical field strength may cause thermal or electrochemical damage to internal tissues. Damage may include hemolysis, protein coagulation, coagulation necrosis of muscle and other tissues, vascular thrombosis, dehydration, and muscle and tendon avulsion. High electrical field strength injuries may result in massive edema, which, as veins coagulate and muscles swell, results in compartment syndromes. Massive edema may also cause hypovolemia and hypotension. Muscle destruction may result in rhabdomyolysis and myoglobinuria. Myoglobinuria, hypovolemia, and hypotension increase risk of acute renal failure. Electrolyte disturbances can also occur. The consequences of organ dysfunction do not always correlate with the amount of tissue destroyed (eg, ventricular fibrillation may occur with relatively little tissue destruction).

Symptoms and Signs

Burns may be sharply demarcated on the skin even when current penetrates irregularly into deeper tissues. Severe involuntary muscular contractions, seizures, ventricular fibrillation, or respiratory arrest

due to CNS damage or muscle paralysis may occur. Brain, spinal cord, and peripheral nerve damage may result in various neurologic deficits. Cardiac arrest may occur without burns in bathtub accidents (when a wet [grounded] person contacts a 110-V circuit—eg, from a hair dryer or radio).

Young children who bite or suck on extension cords can burn their mouth and lips. Such burns may cause cosmetic deformities and impair growth of the teeth, mandible, and maxilla. Labial artery hemorrhage, which results when the eschar separates 5 to 10 days after injury, occurs in up to 10% of these young children.

An electrical shock can cause powerful muscle contractions or falls (eg, from a ladder or roof), resulting in dislocations (electrical shock is one of the few causes of posterior shoulder dislocation), vertebral or other fractures, injuries to internal organs, and other blunt force injuries.

Diagnosis

• Head to toe examination

• Sometimes ECG, cardiac enzyme measurement, and urinalysis

The person, once away from current, is assessed for cardiac arrest (see p. 2255) and respiratory arrest (see p. 2269). Necessary resuscitation is done. After initial resuscitation, patients are examined from head to toe.

Asymptomatic patients who are not pregnant, have no known heart disorders, and who have had only brief exposure to household current usually have no significant acute internal or external injuries and do not require testing or monitoring. For other patients, ECG, CBC, measurement of cardiac enzymes, and urinalysis (especially to check for myoglobin) should be considered. Patients with impaired consciousness may require CT or MRI.

Treatment

• Shutting off current

• Resuscitation

• Analgesia

• Sometimes cardiac monitoring for 6 to 12 h

• Wound care

Prehospital care: The first priority is to break contact between the person and the current source by shutting off the current (eg, by throwing a circuit breaker or switch, by disconnecting the device from its electrical outlet). High- and low-voltage power lines are not always easily differentiated, particularly outdoors. CAUTION: *If power lines could be high voltage, no attempts to disengage the person should be made until the power is shut off*.

Resuscitation: Patients are resuscitated while being assessed. Shock, which may result from trauma or massive burns, is treated (see p. 2295). Standard burn fluid resuscitation formulas, which are based on the extent of skin burns, may underestimate the fluid requirement in electrical burns; thus, such formulas are not used. Instead, fluids are titrated to maintain adequate urine output (about 100 mL/h in adults and 1.5 mL/kg/h in children). For myoglobinuria, alkalinizing the urine and maintaining adequate urine output decrease the risk of renal failure. Surgical debridement of large amounts of muscle tissue may also help to decrease myoglobinuric renal failure.

Severe pain due to an electrical burn is treated by the judicious titration of IV opioids.

Other measures: Asymptomatic patients who are not pregnant, have no known heart disorders, and who have had only brief exposure to household current usually have no significant acute internal or external injuries that can be mitigated by admission and can be discharged. Cardiac monitoring for 6 to 12 h is indicated for patients with the following conditions:

• Arrhythmias

• Chest pain

• Any suggestion of cardiac damage

• Pregnancy (possibly)

• Known heart disorders (possibly)

Appropriate tetanus prophylaxis (see p. 1299) and topical burn wound care (see p. 3246) are required. Pain is treated with NSAIDs or other analgesics.

All patients with significant electrical burns should be referred to a specialized burn unit. Young children with lip burns should be referred to a pediatric orthodontist or oral surgeon familiar with such injuries.

Prevention

Electrical devices that touch or may be touched by the body should be properly insulated, grounded, and incorporated into circuits containing protective circuit-breaking equipment. Ground-fault circuit breakers, which trip when as little as 5 mA of current leaks to ground, are effective and readily available. Outlet guards reduce risk in homes with infants or young children.

Lightning Injuries

Lightning injuries include cardiac arrest, loss of consciousness, and temporary or permanent neurologic deficits; serious burns and internal tissue injury are rare. Diagnosis is clinical; evaluation requires ECG and cardiac monitoring. Treatment is supportive.

Lightning strikes cause about 30 to 50 deaths and several times more injuries annually in the US. Lightning tends to strike tall or isolated objects, including trees, towers, shelters, flagpoles, bleachers, and fences. A person may be the tallest object in an open field. Metal objects and water do not attract lightning but easily transmit electricity once they are hit. Lightning can strike a person directly, or the current can be transferred to the person through the ground or a nearby object. Lightning can also travel from outdoor power or electrical lines to indoor electrical equipment or telephone lines. The force may throw the person several yards.

Because the physics of lightning injury is different from that of generated electrical energy, knowledge of the effects of exposure to household current or high voltage cannot be extrapolated to lightning injuries. For example, damage from lightning injury is not determined by voltage or amperage. Although lightning current contains a large amount of energy, it flows for an extremely brief period (1/10,000 to 1/1000 sec). It rarely, if ever, causes serious skin wounds and seldom causes rhabdomyolysis or serious internal tissue damage, unlike high-voltage and high-current electrical injury from generated sources. Patients may have intracranial hemorrhage resulting from secondary injury or, rarely, from lightning itself.

Lightning can affect the heart but primarily affects the nervous system, damaging the brain, autonomic nervous system, and peripheral nerves.

Symptoms and Signs

The electrical charge can cause asystole or other arrhythmias or cause symptoms of brain dysfunction, such as loss of consciousness, confusion, or amnesia.

Keraunoparalysis is paralysis and mottling, coldness, and pulselessness of the lower and sometimes upper extremities with sensory deficits; the cause is probably sympathetic nervous system injury. Keraunoparalysis is common and usually resolves within several hours, although some degree of permanent paresis occasionally results. Other manifestations of lightning injury may include minor skin burns in a punctate or feathered, branched pattern, tympanic membrane perforation, and, within days, cataracts. Neurologic problems may include confusion, cognitive deficits, and peripheral neuropathy. Neuropsychologic problems (eg, sleep disturbances, attention deficit, memory problems) may occur. Cardiopulmonary arrest at the time of the strike is the most common cause of death. Cognitive deficits, pain syndromes, and sympathetic nervous system damage are the most common long-term sequelae.

Diagnosis

• Recognition of cardiac and brain complications

Lightning injuries may be witnessed or unwitnessed. Unwitnessed injuries should be suspected when people found outside during or after storms have amnesia or are unconscious.

ECG may be done if injury is severe. Cardiac enzymes are measured for patients with the following:

• Chest pain

• Abnormal ECG

• Altered mental status

Patients with initially abnormal or deteriorating mental status or focal neurologic deficits compatible with a brain lesion require a head CT or MRI.

Treatment

• Supportive care

CPR is initiated for cardiac or respiratory arrest or both. If an automated external defibrillator is available, it should be used.

Supportive care is provided. Fluids are usually restricted to minimize potential brain edema. Most people who have been injured by lightning can be safely discharged unless cardiac effects or brain lesions are suspected.

Prevention

Most lightning injuries can be prevented by following lightning safety guidelines. People should know the weather forecast and have backup plans if a storm is predicted. They should have an escape plan involving evacuation to a safer area (ideally a large habitable building) and pay attention to the weather while outdoors so they can implement the escape plan if an unexpected storm comes up. By the time thunder is heard, people are already in danger and should seek shelter (eg, in a building or fully enclosed metal vehicle). Small, open structures, such as gazebos, are not safe. People should not go outdoors until 30 min after the last lightning is seen or thunder is heard. When indoors during an electrical storm, people should avoid plumbing and electrical appliances, stay away from windows and doors, and not use hard-wired telephones, video game consoles, or computers. Cellular phones, personal digital assistants (PDAs), and MP3 players are safe because they do not attract lightning.

Chapter 331. Radiation Exposure and Contamination

Introduction

Ionizing radiation injures tissues variably, depending on factors such as radiation dose, rate of exposure, type of radiation, and part of the body exposed. Symptoms may be local (eg, burns) or systemic (eg, acute radiation sickness). Diagnosis is by history of exposure, symptoms and signs, and sometimes use of radiation detection equipment to localize and identify radionuclide contamination. Management focuses on associated traumatic injuries, decontamination, supportive measures, and minimizing exposure of health care workers. Patients with severe acute radiation sickness receive reverse isolation and bone marrow support. Patients internally contaminated with certain specific radionuclides may receive uptake inhibitors or chelating agents. Prognosis is initially estimated by the time between exposure and symptoms, the severity of those symptoms, and by the lymphocyte count during the initial 24 to 72 h.

Ionizing radiation is emitted by radioactive elements and by equipment such as x-ray and radiation therapy machines.

Types of radiation: Radiation includes

• High-energy electromagnetic waves (x-rays, gamma rays)

• Particles (alpha particles, beta particles, neutrons)

Alpha particles are energetic helium nuclei emitted by some radionuclides with high atomic numbers (eg, plutonium, radium, uranium); they cannot penetrate skin beyond a shallow depth (< 0.1 mm).

Beta particles are high-energy electrons that are emitted from the nuclei of unstable atoms (eg, cesium-137, iodine-131). These particles can penetrate more deeply into skin (1 to 2 cm) and cause both epithelial and subepithelial damage.

Neutrons are electrically neutral particles emitted by a few radionuclides (eg, californium-252) and produced in nuclear fission reactions (eg, in nuclear reactors); they can penetrate deeply into tissues (> 2 cm), where they collide with the nuclei of stable atoms, resulting in emission of energetic protons, alpha and beta particles, and gamma radiation.

Gamma radiation and x-rays are electromagnetic radiation (ie, photons) of very short wavelength that can penetrate deeply into tissue (many centimeters). While some photons deposit all their energy in the body, other photons of the same energy may only deposit a fraction of their energy and others may pass completely through the body without interacting.

Because of these characteristics, alpha and beta particles cause the most damage when the radioactive atoms that emit them are *within* the body (internal contamination) or, in the case of beta-emitters, directly *on* the body; only tissue in close proximity to the radionuclide is affected. Gamma rays and x-rays can cause damage distant from their source and are typically responsible for acute radiation syndromes (ARS —see p. 3255).

Measurement of radiation: Conventional units of measurement include the roentgen, rad, and rem. The roentgen (R) is a unit of exposure measuring the ionizing ability of x- or gamma radiation in air. The radiation absorbed dose (rad) is the amount of that radiation energy absorbed per unit of mass. Because biologic damage per rad varies with radiation type (eg, it is higher for neutrons than for x- or gamma radiation), the dose in rad is corrected by a quality factor; the resulting effective dose unit is the roentgen equivalent in man (rem). Outside the US and in the scientific literature, SI units are used, in which the rad is replaced by the gray (Gy) and the rem by the sievert (Sv); 1 Gy = 100 rad and 1 Sv = 100 rem. The rad and rem (and hence Gy and Sv) are essentially equal (ie, the quality factor equals 1) when describing gamma or beta radiation.

Types of exposure: Radiation exposure may involve

• Contamination

• Irradiation

Radioactive contamination is the unintended contact with and retention of radioactive material, usually as a dust or liquid. Contamination may be

• External

• Internal

External contamination is that on skin or clothing, from which some can fall or be rubbed off, contaminating other people and objects. Internal contamination is unintended radioactive material within the body, which it may enter by ingestion, inhalation, or through breaks in the skin. Once in the body, radioactive material may be transported to various sites (eg, bone marrow), where it continues to emit radiation until it is removed or decays. Internal contamination is more difficult to remove. Although internal contamination with any radionuclide is possible, historically, most cases in which contamination posed a significant risk to the patient involved a relatively small number of radionuclides: hydrogen-3, cobalt-60, strontium-90, cesium-137, iodine-131, radium-226, uranium-235, uranium-238, plutonium-238, plutonium-239, polonium-210, and americium-241.

Irradiation is exposure to radiation but not radioactive material (ie, no contamination is involved). Radiation exposure can occur without the source of radiation (eg radioactive material, x-ray machine) being in contact with the person. When the source of the radiation is removed or turned off, exposure ends. Irradiation can involve the whole body, which, if the dose is high enough, can result in systemic symptoms and radiation syndromes (see p. 3255), or a small part of the body (eg, from radiation therapy), which can result in local effects. People do not emit radiation (ie, become radioactive) following irradiation.

Sources of exposure: Sources may be naturally occurring or man-made (see Table 331-1).

People are constantly exposed to low levels of naturally occurring radiation called background radiation. Background radiation comes from cosmic radiation and from radioactive elements in the air, water, and earth. Cosmic radiation is concentrated at the poles by the earth's magnetic field and attenuated by the atmosphere. Thus, exposure is greater for people living at high latitudes, at high altitudes, or both and during airplane flights. Radioactive elements, particularly uranium and its radioactive progeny and potassium-40, are present in many rocks and minerals. These elements end up in various substances, including food, water, and construction materials. Radon, a radioactive gas resulting from the decay of uranium, typically accounts for about two thirds of naturally occurring radiation dose to the US population. In the US, people receive an average effective dose of about 3 millisieverts (mSv)/yr from natural sources. However, in some parts of the world, people receive between 5 and 10 mSv/yr. The doses from natural background radiation are far too low to cause radiation injuries, although they may increase the risk of cancer.

In the US, people receive on the average about 3 mSv/yr from man-made sources, the vast majority of which involve medical imaging. Imaging exposure tends to be highest from CT and nuclear cardiology procedures. However, medical diagnostic procedures rarely impart doses sufficient to cause radiation injury, although they may increase the risk of cancer. Exceptions may include certain prolonged fluoroscopically guided interventional procedures (eg, endovascular reconstruction, vascular embolization, cardiac radiofrequency ablation); these procedures have caused injuries to skin and underlying tissues. Radiation therapy commonly causes injury to some normal tissues near the target tissue.

[**Table 331-1.** Average Annual Radiation Exposure in the Us]

A small portion of public exposure results from radiation accidents and fallout from nuclear weapons testing. Accidents may involve industrial irradiators, industrial radiography sources, and nuclear reactors. These accidents commonly result from failure to follow safety procedures (eg, interlocks being bypassed). Radiation injuries have also been caused by lost or stolen medical or industrial sources containing radionuclides. People seeking medical care for these injuries may be unaware that they were exposed to radiation.

Radioactive material has escaped from nuclear power plants, including the Three Mile Island plant in Pennsylvania in 1979 and at Chernobyl in Ukraine in 1986. Exposure from Three Mile Island was minimal; people living within 1.6 km of the plant received only about 0.08 mSv. However, people living in 2 villages near the Chernobyl plant received an average dose of about 300 mSv, and people at the Chernobyl plant itself received significantly higher doses. More than 30 workers and emergency responders died, and many more were injured. Low-level contamination from that accident was detected as far away as Europe, Asia, and even the US. The average cumulative exposure for the general population in various affected regions of Belarus, Russia, and Ukraine over a 20-yr period after the accident is estimated to be between 10 and 30 mSv.

Another significant radiation event was the detonation of 2 atomic bombs over Japan in August 1945, which caused about 110,000 deaths from the immediate trauma of the blast and heat. A much smaller number of deaths resulted later from radiation-induced illnesses.

While several criminal cases of intentional contamination of individuals have been reported, radiation exposure to a population through terrorist activities has not occurred but is a concern. A possible scenario involves the use of a device to contaminate an area by dispersing radioactive material (a radiation dispersal device that uses conventional explosives is referred to as a dirty bomb). Other terrorist scenarios include using a hidden radiation source to expose unsuspecting people to large doses of radiation, attacking a nuclear reactor or radioactive material storage facility, and detonating a nuclear weapon.

Pathophysiology

Ionizing radiation can damage DNA, RNA, and proteins directly, but more often the damage to these molecules is indirect, caused by highly reactive free radicals generated by radiation's interaction with intracellular water molecules. Large doses of radiation can cause cell death, and lower doses may interfere with cellular proliferation. Damage to other cellular components can result in progressive tissue hypoplasia, atrophy, and eventually fibrosis.

Factors affecting response: Biologic response to radiation varies with

• Tissue radiosensitivity

• Dose

• Duration of exposure

Cells and tissues differ in their radiosensitivity. In general, cells that are undifferentiated and those that have high mitotic rates (eg, stem cells) are particularly vulnerable to radiation. Because radiation preferentially depletes rapidly dividing stem cells over the more resistant mature cells, there is typically a latent period between radiation exposure and overt radiation injury. Injury does not manifest until a significant fraction of the mature cells die of natural senescence and, due to loss of stem cells, are not replaced.

Cellular sensitivities in approximate descending order from most to least sensitive are

• Lymphoid cells

• Germ cells

- Proliferating bone marrow cells
- Intestinal epithelial cells
- Epidermal stem cells
- Hepatic cells
- Epithelium of lung alveoli and biliary passages
- Kidney epithelial cells
- Endothelial cells (pleura and peritoneum)
- Nerve cells
- Bone cells
- Muscle and connective tissue cells

The severity of radiation injury depends on the dose and the length of time over which it is delivered. A single rapid dose is more damaging than the same dose given over weeks or months. Dose response also depends on the fraction of the body exposed. Significant illness is certain, and death is possible, after a whole-body dose > 4.5 Gy delivered over a short time interval; however, 10s of Gy can be well tolerated when delivered over a long period to a small area of tissue (eg, for cancer therapy).

Other factors can increase the sensitivity to radiation injury. Children are more susceptible to radiation injury because they have a higher rate of cellular proliferation. People who are homozygous for the ataxia-telangiectasia gene exhibit greatly increased sensitivity to radiation injury. Disorders, such as connective tissue disorders and diabetes, may increase the sensitivity to radiation injury. Chemotherapeutic agents also increase the sensitivity to radiation injury.

Cancer and teratogenicity: Genetic damage to somatic cells may result in malignant transformation, and damage to germ cells raises the possibility of transmissible genetic defects.

Ionizing radiation can cause cancer; whole-body exposure to 1 Gy increases the average adult's lifetime risk of cancer death from 25% to about 30%, a 20% relative risk increase but only a 5% absolute risk increase. The cancer risk due to commonly encountered doses (ie, from background radiation and typical imaging tests—see
Table 343-1 on p. 3403) is much less. Children are more susceptible because they have a higher number of future cell divisions and a longer life span during which cancer may manifest; CT of the abdomen done in a 1-yr-old child is estimated to increase the estimated lifetime absolute risk of developing cancer by 0.18%. Radionuclides that are incorporated into specific tissues are potentially carcinogenic at those sites (eg, radioactive iodine increases risk of thyroid cancer).

The fetus is exceptionally susceptible to high-dose radiation injury. However, at doses < 100 mGy, teratogenic effects are unlikely; the fetal risk from radiation at doses from imaging tests that pregnant women might typically undergo is small compared with the overall risk of birth defects and the potential diagnostic benefit of the examination.

Damage to reproductive cells has been shown to cause birth defects in progeny of severely irradiated animals. However, hereditary effects have not been found in children of radiation-exposed humans, including survivors of the atomic bomb attacks in Japan.

Symptoms and Signs

Clinical manifestations depend on whether radiation exposure involves the whole body (acute radiation syndrome) or is limited to a small portion of the body (focal radiation injury).

Acute radiation syndromes (ARS): After the whole body, or a large portion of the body, receives a high dose of radiation, several distinct syndromes may occur:

• Cerebrovascular syndrome

• GI syndrome

• Hematopoietic syndrome

These syndromes have 3 different phases:

• Prodromal phase (0 to 2 days after exposure): Lethargy and GI symptoms (nausea, anorexia, vomiting, diarrhea) are possible.

• Latent asymptomatic phase (0 to 31 days after exposure)

• Overt systemic illness phase: Illness is classified by the main organ system affected.

Which syndrome develops, how severe it is, and how quickly it progresses depend on radiation dose (see Table 331-2). The symptoms and time course are fairly consistent for a given dose of radiation and thus can help estimate radiation exposure.

The **cerebrovascular syndrome**, the dominant manifestation of extremely high whole-body doses of radiation (> 30 Gy), is always fatal. The prodrome develops within minutes to 1 h after exposure. There is little or no latent phase. Patients develop tremors, seizures, ataxia, and cerebral edema and die within hours to 1 or 2 days.

The **GI syndrome** is the dominant manifestation after whole-body doses of about 6 to 30 Gy. Prodromal symptoms, often marked, develop within about 1 h and resolve within 2 days. During the latent period of 4 to 5 days, GI mucosal cells die. Cell death is followed by intractable nausea, vomiting, and diarrhea, which lead to severe dehydration and electrolyte imbalances, diminished plasma volume, and vascular collapse. Necrosis of intestine may also occur, predisposing to bacteremia and sepsis. Death is common. Patients receiving > 10 Gy may have cerebrovascular symptoms (suggesting a lethal dose). Survivors also have the hematopoietic syndrome.

The **hematopoietic syndrome** is the dominant manifestation after whole-body doses of about 1 to 6 Gy and consists of a generalized pancytopenia. A mild prodrome may begin after 1 to 6 h, lasting 24 to 48 h. Bone marrow stem cells are significantly depleted, but mature blood cells in circulation are largely unaffected (circulating lymphocytes are an exception, and lymphopenia may be evident within hours to days after exposure). As the cells in circulation die by senescence, they are not replaced in sufficient numbers, resulting in pancytopenia. Thus, patients remain asymptomatic during a latent period of up to 4 1/2 wk after a 1-Gy dose as marrow production falls. Risk of various infections is increased as a result of the neutropenia (most prominent at 2 to 4 wk) and decreased antibody production. Petechiae and mucosal bleeding result from thrombocytopenia, which develops within 3 to 4 wk and may persist for months. Anemia develops slowly, because preexisting RBCs have a longer life span than WBCs and platelets. Survivors have an increased incidence of radiation-induced cancer, including leukemia.

Cutaneous radiation injury (CRI) is injury to the skin and underlying tissues from acute radiation doses as low as 3 Gy (see Table 331-3). CRI can occur with ARS or with focal radiation exposure and ranges from mild transient erythema to necrosis. Delayed effects (> 6 mo after exposure) include hyperpigmentation and hypopigmentation, progressive fibrosis,

[**Table 331-2.** Effects of Whole-Body Irradiation from External Radiation or Internal Absorption]

[**Table 331-3.** Focal Radiation Injury*]

and diffuse telangiectasia. Thin atrophic skin can be easily damaged by mild mechanical trauma. Exposed skin is at increased risk of squamous cell carcinoma. In particular, the possibility of radiation exposure should be considered when patients present with a painful nonhealing skin burn without a history of thermal injury.

Focal injury: Radiation to almost any organ can have both acute and chronic adverse effects (see Table 331-3). In most patients, these adverse effects result from radiation therapy (see p. 1064). Other common sources of exposure include inadvertent contact with unsecured food irradiators, radiotherapy equipment, x-ray diffraction equipment, and other industrial or medical radiation sources capable of producing high dose rates. Also, overexposure to x-rays during medical fluoroscopy is a source of exposure and of CRI in particular. Radiation-induced sores or ulcers may take months or years to fully develop. Patients with these injuries often have severe pain.

Diagnosis

• Symptoms, severity, and symptom latency

• Serial absolute lymphocyte counts

Diagnosis is by history of exposure, symptoms and signs, and laboratory testing. The onset, time course, and severity of symptoms can help determine radiation dose and thus also help triage patients relative to their likely consequences. However, some prodromal symptoms (eg, nausea, vomiting, diarrhea, tremors) are nonspecific, and causes other than radiation should be considered. Many patients *without* enough exposure to cause acute radiation sickness may present with similar, nonspecific symptoms, particularly after a terrorist attack or reactor accident, when anxiety is high.

After acute radiation exposure, CBC with differential and calculation of absolute lymphocyte count is done and repeated 24, 48, and 72 h after exposure to estimate the initial radiation dose and prognosis (see Table 331-4). The relationship between dose and lymphocyte counts can be altered by physical trauma, which can shift lymphocytes from the interstitial spaces into the vasculature, raising the lymphocyte count. This stress-related increase is transient and typically resolves within 24 to 48 h after the physical insult. CBC is repeated weekly to monitor marrow activity and as needed based on the clinical course.

Contamination: When contamination is suspected, the entire body should be surveyed with a thin window Geiger-Muller probe

[**Table 331-4.** Relationship Between Absolute Lymphocyte Count in Adults at 48 H, Radiation Dose,* and Prognosis]

attached to a survey meter (Geiger counter) to identify the location and extent of external contamination. Additionally, to detect possible internal contamination, the nares, ears, mouth, and wounds are wiped with moistened swabs that are then tested with the counter. Urine, feces, and emesis should also be tested for radioactivity if internal contamination is suspected.

Prognosis

Without medical care, the $LD_{50/60}$ (dose expected to be fatal to 50% of patients within 60 days) for whole-body radiation is about 3 Gy; 6 Gy exposure is nearly always fatal. When exposure is < 6 Gy, survival is possible and is inversely related to total dose. Time to death decreases as the dose increases. Death may occur within hours to a few days in patients with the cerebral syndrome and usually within 2 days to several weeks in patients with the GI syndrome. In patients with the hematopoietic syndrome, death may occur within 4 to 8 wk because of a supervening infection or massive hemorrhage. Patients exposed to whole-body doses < 2 Gy should fully recover within 1 mo, although long-term sequelae (eg, cancer) may occur.

With medical care, the $LD_{50/60}$ is 6 Gy and occasional patients have survived exposures of up to 10 Gy. Significant comorbidities, injuries, and burns worsen prognosis.

Treatment

• Treatment of severe traumatic injuries or life-threatening medical conditions first

• Minimization of health care worker radiation exposure and contamination

• Treatment of external and internal contamination

• Sometimes specific measures for particular radionuclides

• Supportive care

Radiation exposure may be accompanied by physical injuries (eg, from burn, blast, fall); *associated trauma is more immediately life threatening than radiation exposure and must be treated expeditiously* (see p. 3190). Trauma resuscitation of the seriously injured takes priority over decontamination efforts and must not be delayed awaiting special radiation management equipment and personnel. Standard universal precautions, as routinely used in trauma care, adequately protect the critical care team.

Extensive, reliable information about principles of radiation injuries, including management, is available at the US Department of Health and Human Services Radiation Event Medical Management web site (http://remm.nlm.gov). This information can be downloaded to a personal computer or personal digital assistant (PDA) in case Internet connectivity is lost during a radiation incident.

Preparation: The Joint Commission mandates that all hospitals have protocols and that personnel have training to deal with patients contaminated with hazardous material, including radioactive material. Identification of radioactive contamination on patients should prompt their isolation in a designated area (if practical), decontamination, and notification of the hospital radiation safety officer, public health officials, hazardous material teams, and law enforcement agencies as appropriate to investigate the source of radioactivity.

Treatment area surfaces may be covered with plastic sheeting to aid in facility decontamination; this preparation should never take precedence over provision of medical stabilization. Waste receptacles (labeled "Caution, Radioactive Material"), sample containers, and Geiger counters should be readily available. All equipment that has come into contact with the room or with the patient (including ambulance equipment) should remain isolated until lack of contamination has been verified. An exception is a mass casualty situation, during which lightly contaminated critical equipment such as helicopters, ambulances, trauma rooms, and x-ray, CT, and surgical facilities, should be quickly decontaminated to the extent possible and returned to service.

Personnel involved in treating or transporting the patient should follow standard precautions, wearing caps, masks, gowns, gloves, and shoe covers. Used gear should be placed in specially marked bags or containers. Dosimeter badges should be worn to monitor radiation exposure. Personnel may be rotated to minimize exposure, and pregnant personnel should be excluded from the treatment area.

Due to the low exposure rates anticipated from most contaminated patients, medical staff members caring for typical patients are unlikely to receive doses in excess of the occupational limit of 0.05 Gy/yr. Even in the extreme case of radiation casualties from the Chernobyl nuclear reactor accident, medical personnel who treated patients in the hospital received < 0.01 Sv. Several authoritative sources suggest that a dose of up to at least 0.5 Gy may be considered an acceptable risk for lifesaving activity.

External decontamination: Typical sequence and priorities are

• Removing clothing and external debris

• Decontaminating wounds before intact skin

• Cleaning the most contaminated areas first

- Using radiation survey meter to monitor progress of decontamination

- Continuing decontamination until areas are at < 2 to 3 times background radiation levels or there is no significant reduction between decontamination efforts

Clothes are removed carefully to minimize the spread of contamination and placed in labeled containers. Clothing removal eliminates about 90% of external contamination. Foreign objects should be considered contaminated until cleared by a radiation survey meter.

Contaminated wounds are decontaminated before intact skin; they are irrigated with saline and gently scrubbed with a surgical sponge. Minimal debridement of wound edges may be done if there is residual contamination after multiple attempts at cleaning. Debridement beyond the wound margin is not required, although embedded radioactive shrapnel should be removed and placed in a lead container.

If necessary, consultation is available 24 h/day from the Department of Energy Radiation Emergency Assistance Center/Training Site (REAC/TS) at (865) 576-1005 and www.orau.gov/reacts or the Centers for Disease Control and Prevention (CDC) at (888) 246-2675 and www.bt.cdc.gov/radiation/.

Contaminated skin and hair are washed with lukewarm water and mild detergent until radiation survey meter measurements indicate < 2 to 3 times normal background radiation levels or until successive washings do not significantly reduce contamination levels. All wounds are covered during washing to prevent the introduction of radioactive material. Scrubbing may be firm but should not abrade the skin. Special attention is usually required for fingernails and skinfolds. Hair that remains contaminated is removed with scissors or electric clippers; shaving is avoided. Inducing sweating (eg, placing a rubber glove over a contaminated hand) may help remove residual skin contamination.

Burns are rinsed gently because scrubbing may increase injury severity; subsequent dressing changes help remove residual contamination.

Decontamination is not necessary for patients who have been irradiated by an external source and are not contaminated.

Internal decontamination: Ingested radioactive material should be removed promptly by induced vomiting or lavage if exposure is recent. Frequent mouth rinsing with saline or dilute hydrogen peroxide is indicated for oral contamination. Exposed eyes should be decontaminated by directing a stream of water or saline laterally to avoid contaminating the nasolacrimal duct.

The urgency and importance of using more specific treatment measures depend on the type and amount of the radionuclide, its chemical form and metabolic characteristics (eg, solubility, affinity for specific target organs), the route of contamination (eg, inhalation, ingestion, contaminated wounds), and the efficacy of the therapeutic method. The decision to treat internal contamination requires knowledge of the potential risks; consultation with a specialist (eg, CDC or REAC/TS) is recommended.

Current methods to remove radioactive contaminants from the body (decorporation) include

- Saturation of the target organ (eg, potassium iodide [KI] for iodine isotopes)

- Chelation at the site of entry or in body fluids followed by rapid excretion (eg, Ca or zinc diethylenetriamine penta-acetate [DTPA] for americium, californium, plutonium, and yttrium)

- Acceleration of the metabolic cycle of the radionuclide by isotope dilution, (eg, water for hydrogen-3)

- Precipitation of the radionuclide in the intestinal lumen followed by fecal excretion (eg, oral Ca or aluminum phosphate solutions for strontium-90)

- Ion exchange in the GI tract, (eg, Prussian blue for cesium-137, rubidium-82, thallium-201)

Because a serious nuclear power reactor accident that released fission products into the environment could expose large groups of people to radioiodine, decorporation using oral KI has been studied in great detail. KI is > 95% effective when given at the optimal time (shortly before or immediately after exposure) and dose. However, effectiveness diminishes significantly within several hours after exposure. KI can be given either in tablet form or as a supersaturated solution (dosage: adult, 130 mg; age 3 to 18 yr, 65 mg; age 1 to 36 mo, 32 mg; age < 1 mo, 16 mg). KI is effective only for internal contamination with radioactive iodides and has no benefit in internal contamination with other radioactive elements. Most other drugs used for decorporation are much less effective than KI and reduce the dose to the patient only by 25 to 75%.

Specific management: Symptomatic treatment is given as needed and includes managing shock and hypoxia, relieving pain and anxiety, and giving sedatives (lorazepam 1 to 2 mg IV prn) to control seizures, antiemetics (metoclopramide 10 to 20 mg IV q 4 to 6 h; prochlorperazine 5 to 10 mg IV q 4 to 6 h; ondansetron 4 to 8 mg IV q 8 to 12 h) to control vomiting, and antidiarrheal agents (kaolin/pectin 30 to 60 mL po with each loose stool; loperamide 4 mg po initially, then 2 mg po with each loose stool) for diarrhea.

There is no specific treatment for the cerebrovascular syndrome. It is universally fatal; care should address patient comfort.

The GI syndrome is treated with aggressive fluid resuscitation and electrolyte replacement. Parenteral nutrition should be initiated to promote bowel rest. In febrile patients, broad-spectrum antibiotics (eg, imipenem 500 mg IV q 6 h) should be initiated immediately. Septic shock from overwhelming infection remains the most likely cause of death.

Management of the hematopoietic syndrome is similar to that of bone marrow hypoplasia and pancytopenia of any cause. Blood products should be transfused to treat anemia and thrombocytopenia, and hematopoietic growth factors (granulocyte colony-stimulating factor and granulocyte macrophage colony-stimulating factor) and broad-spectrum antibiotics should be given to treat neutropenia and neutropenic fever, respectively (see p. 951). Patients with neutropenia should also be placed in reverse isolation. With a whole-body radiation dose > 4 Gy, the probability of bone marrow recovery is poor, and hematopoietic growth factors should be given as soon as possible. Stem cell transplantation has had limited success but should be considered for exposure > 7 to 10 Gy (see p. 1132).

Radiation-induced sores or ulcers that fail to heal satisfactorily may be repaired by skin grafting or other surgical procedures.

Aside from regular monitoring for signs of certain disorders (eg, ophthalmic examination for cataracts, thyroid function studies for thyroid disorders), there is no specific monitoring, screening, or treatment for specific organ injury or cancer.

Prevention

Protection from radiation exposure is accomplished by avoiding contamination with radioactive material and minimizing the duration of exposure, maximizing the distance from the source of radiation, and shielding the source. During imaging procedures that involve ionizing radiation and especially during radiation therapy for cancer, the most susceptible parts of the body (eg, female breasts, gonads, thyroid) that are not being treated or imaged are shielded by lead aprons or blocks.

Although shielding of personnel with lead aprons or commercially available transparent shields effectively reduces exposure to low-energy scattered x-rays from diagnostic imaging studies, these aprons and shields are almost useless in reducing exposure to the high-energy gamma rays produced by radionuclides that would likely be used in a terrorist incident or be released in a nuclear power plant accident. In such cases, measures that can minimize exposure include using standard precautions, undergoing decontamination efforts, and maintaining distance from contaminated patients when not actively providing care. All personnel working around radiation sources should wear dosimeter badges if they are at risk for exposures > 10% of the maximum permissible occupational dose (0.05 Sv).

Public response: After widespread high-level environmental contamination from a nuclear power plant

accident or intentional release of radioactive material, exposure can be reduced either by

- Sheltering in place
- Evacuating the contaminated area

The better approach depends on many event-specific variables, including the elapsed time since initial release, whether release has stopped or is ongoing, weather conditions, availability and type of shelter, and evacuation conditions (eg, traffic, transportation availability). The public should follow the advice of local public health officials as broadcast on TV or radio as to which response option is best. If sheltering is recommended, a concrete or metal structure, particularly one below grade (eg, in a basement) is best.

Consistent and concise messages from public health officials can help reduce unnecessary panic and reduce the number of emergency department visits from people at low risk, thus keeping the emergency department from being overwhelmed. Such a communication plan should be developed prior to any event. A plan to counsel distressed people is also recommended.

People living within 16 km (10 miles) of a nuclear power plant should have ready access to KI tablets. These tablets can be obtained from local pharmacies and some public health agencies.

Preventive drugs: Radioprotective drugs, such as thiol compounds with radical scavenging properties, have been shown to reduce mortality when given before or at the time of irradiation. Amifostine is a powerful injectable radioprotective agent in this category; it prevents xerostomia in patients undergoing radiation therapy. Although thiol compounds have good efficacy in radioprotection, these compounds cause adverse effects, such as hypotension, nausea, vomiting, and allergic reactions. Other experimental drugs and chemicals have also been shown to increase survival rates in animals if given before or during irradiation. However, these drugs can be very toxic at doses necessary to provide substantial protection, and none currently are recommended.

Chapter 332. Heat Illness

Introduction

Exposure to warm environments affects many physiologic functions and may cause dehydration. Most people experience mild but uncomfortable symptoms; however, effects may range from cramps and edema to syncope, heat exhaustion, and heatstroke. Core temperature is elevated in some types of heat illness. People with dehydration (see pp. 822 and 2806) may have tachycardia, tachypnea, and orthostatic hypotension. CNS dysfunction suggests heatstroke, the most serious disorder; confusion and lethargy may further impair the ability to escape the heat and rehydrate.

Pathophysiology

Heat input comes from

• The environment

• Metabolism

Heat output occurs through the skin via the following:

• Radiation

• Evaporation (eg, via sweat)

• Convection

The contribution of each of these mechanisms varies with environmental temperature and humidity. Radiation predominates at room temperature, but as environmental temperature approaches body temperature, evaporation becomes more important, providing essentially 100% of cooling at > 35° C. However, high humidity greatly limits evaporative cooling (see Heatstroke on p. 3265).

Heat output is modulated by changes in cutaneous blood flow and sweat production. Cutaneous blood flow is 200 to 250 mL/min at normal temperatures but increases to 7 to 8 L/min with heat stress, requiring a marked increase in cardiac output. Also, heat stress increases sweat production from negligible to > 2 L/h; thus, significant dehydration can occur rapidly. Because sweat contains electrolytes, electrolyte loss may be substantial. However, prolonged exposure triggers physiologic changes to accommodate heat load (acclimatization); eg, sweat Na levels are 40 to 100 mEq/L in people who are not acclimatized but decrease to 10 to 70 mEq/L in acclimatized people.

The body can compensate for large variations in heat load, but significant or prolonged exposure to heat increases core temperature. Modest, transient core temperature elevations are tolerable, but severe elevations (typically > 41° C) lead to protein denaturation and, especially during hard work in the heat, release of inflammatory cytokines (eg, tumor necrosis factor-α, IL-1b). As a result, cellular dysfunction occurs and the inflammatory cascade is activated, leading to dysfunction of most organs and activation of the coagulation cascade. These pathophysiologic processes are similar to those of multiple organ dysfunction syndrome (see p. 2293), which follows prolonged shock.

Compensatory mechanisms include an acute-phase response by other cytokines that moderate the inflammatory response (eg, by stimulating production of proteins that decrease production of free radicals and inhibit release of proteolytic enzymes). Also, increased core temperature triggers expression of heat-shock proteins. These proteins transiently enhance heat tolerance by poorly understood mechanisms (eg, possibly by preventing protein denaturation) and by regulation of cardiovascular responses. With prolonged or extreme temperature elevation, compensatory mechanisms are overwhelmed or malfunction, allowing inflammation and multiple organ dysfunction syndrome to occur.

Etiology

Heat disorders are caused by some combination of increased heat input and decreased output (see Table 332-1).

Excess heat input typically results from strenuous exertion, high environmental temperatures, or both. Medical disorders and use of stimulant drugs can increase heat production.

Impaired cooling can result from obesity, high humidity, wearing heavy clothing, and anything that impairs sweating or evaporation of sweat.

Clinical effects of heat illnesses are exacerbated by the following:

- Inability to tolerate increased cardiovascular demands (eg, due to aging, heart failure, chronic kidney disease, respiratory disorders, or liver failure)
- Dehydration
- Electrolyte disturbance
- Use of certain drugs (see Table 332-1)

The elderly and the very young and people with cardiovascular disorders or electrolyte depletion (eg, due to diuretic use) are at highest risk.

Prevention

Common sense is the best prevention. Physicians should recommend the following measures:

- During excessively hot weather, the elderly and the young should not remain in unventilated residences without air-conditioning.
- Children should not be left in automobiles in the hot sun.

[**Table 332-1.** Common Factors Contributing to Heat Disorders]

- If possible, strenuous exertion in a very hot environment or an inadequately ventilated space should be avoided, and heavy, insulating clothing should not be worn.
- Weight loss after exercise or work can be used to monitor dehydration; people who lose 2 to 3% of their body weight should be reminded to drink extra fluids and should be within 1 kg of starting weight before the next day's exposure. If people lose > 4%, activity should be limited for 1 day.
- If exertion in the heat is unavoidable, fluid (often lost imperceptibly in very hot, very dry air) should be replaced by drinking water frequently, and evaporation should be facilitated by wearing open-mesh clothing or by using fans.

Thirst is a poor indicator of dehydration during exertion; fluids should be drunk every few hours regardless of thirst. However, over-hydration must be avoided; significant hyponatremia (see p. 823) has occurred in endurance athletes who drink very frequently during exercise. Plain water is adequate for hydration during most activity; cool water is absorbed more readily. Special hydrating solutions (eg, sports drinks) are not required, but their flavoring enhances consumption, and their modest salt content is helpful if fluid requirements are high.

Drinking fluids and consuming generously salted foods should be encouraged. Laborers or others who sweat heavily can lose ≥ 20 g of salt/day, making heat cramps more likely; such people need to replace the Na loss with drink and food. A palatable drink providing about 20 mmol of salt/L may be prepared by adding about 5 g (a rounded teaspoon) of table salt to 20 L (about 5 gallons) of any sweetened beverage prepared from a powdered mix. People on low-salt diets should increase salt intake.

Successively and incrementally increasing the level and amount of work done in the heat eventually results in acclimatization, which enables people to work safely at temperatures that were previously intolerable or life threatening. Progressing from 15 min/day of moderate activity (enough to stimulate sweating) during a hot time of day to 90 min of vigorous activity over 10 to 14 days is typically adequate. Acclimatization markedly increases the amount of sweat (and hence cooling) produced at a given level of exertion and markedly decreases the electrolyte content of sweat. Acclimatization significantly decreases risk of a heat illness.

Heat Cramps

Heat cramps are exertion-induced muscle contractions that occur during or after exertion in the heat.

Although exertion may induce cramps during cool weather, such cramps are not heat related and probably reflect lack of fitness. In contrast, heat cramps can occur in physically fit people who sweat profusely and replace lost water but not salt, thereby causing hyponatremia. Heat cramps are common among the following:

• Manual laborers (eg, engine room personnel, steel workers, roofers, miners)

• Military trainees

• Athletes

Cramping is abrupt, usually occurring in muscles of the extremities. Severe pain and carpopedal spasm may incapacitate the hands and feet. Temperature is normal, and other findings are unremarkable. The cramp usually lasts minutes to hours. Diagnosis is by history and clinical evaluation.

Treatment

Cramps may be relieved immediately by firm passive stretching of the involved muscle (eg, plantar dorsiflexion for a calf cramp). Fluids and electrolytes should be replenished orally (1 to 2 L water containing 10 g [2 level tsp] salt or sufficient amounts of a commercial sports drink) or IV (1 to 2 L 0.9% saline solution). Adequate conditioning, acclimatization, and appropriate management of salt balance help prevent cramps.

Heat Exhaustion

Heat exhaustion is a non-life-threatening clinical syndrome of weakness, malaise, nausea, syncope, and other nonspecific symptoms caused by heat exposure. Thermoregulation is not impaired. IV fluids and electrolyte replacement are needed.

Heat exhaustion is caused by water and electrolyte imbalance due to heat exposure, with or without exertion.

Rarely, severe heat exhaustion after hard work may be complicated by rhabdomyolysis, myoglobinuria, acute renal failure, and disseminated intravascular coagulation.

Symptoms and Signs

Symptoms are often vague, and patients may not realize that heat is the cause. Symptoms may include weakness, dizziness, headache, nausea, and sometimes vomiting. Syncope due to standing for long periods in the heat (heat syncope) is common and may mimic cardiovascular disorders. On examination, patients appear tired and are usually sweaty and tachycardic. Mental status is typically normal, unlike in heatstroke. Temperature is usually normal and, when elevated, usually does not exceed 40° C.

Diagnosis

• Clinical evaluation

Diagnosis is clinical and requires exclusion of other possible causes (eg, hypoglycemia, acute coronary syndrome, various infections). Laboratory testing is required only if needed to rule out such disorders.

Treatment

• IV fluid and electrolyte replacement

Treatment involves removing patients to a cool environment, having them lie flat, and giving IV fluid and electrolyte replacement therapy, typically using 0.9% saline solution; oral rehydration does not provide sufficient electrolytes. Rate and volume of rehydration are guided by age, underlying disorders, and clinical response. Replacement of 1 to 2 L at 500 mL/h is often adequate. Elderly patients and patients with heart disorders may require only slightly lower rates; patients with suspected hypovolemia may require higher rates initially. External cooling measures (see p. 3266) are usually not required. However, if patients with heat exhaustion have a core temperature of ≥ 40° C, measures may be taken to reduce it.

Heatstroke

Heatstroke is hyperthermia accompanied by a systemic inflammatory response causing multiple organ dysfunction and often death. Symptoms include temperature > 40° C and altered mental status; sweating is often absent. Diagnosis is clinical. Treatment is rapid external cooling, IV fluid resuscitation, and support as needed for organ dysfunction.

Heatstroke occurs when thermoregulatory mechanisms do not function and core temperature increases substantially. Inflammatory cytokines are activated, and multiple organ dysfunction may develop. Endotoxin from GI flora may also play a role. Organ dysfunction may occur in the CNS, skeletal muscle (rhabdomyolysis), liver, kidneys, lungs (acute respiratory distress syndrome), and heart. The coagulation cascade is activated, sometimes causing disseminated intravascular coagulation. Hyperkalemia and hypoglycemia may occur.

There are 2 variants (see Table 332-2):

• Classic

• Exertional

Classic heatstroke takes 2 to 3 days of exposure to develop. It occurs during summer heat waves, typically in elderly, sedentary people with no air-conditioning and often with limited access to fluids.

Exertional heatstroke occurs abruptly in healthy active people (eg, athletes, military recruits, factory workers). Intense exertion in a hot environment causes a sudden massive heat load that the body cannot modulate. Rhabdomyolysis is common; renal failure and coagulopathy are somewhat more likely and severe.

A syndrome similar to heatstroke may occur after using certain drugs (eg, cocaine, phencyclidine [PCP], amphetamines, monoamine oxide inhibitors). Usually, an overdose is required, but exertion and environmental conditions can be additive.

Malignant hyperthermia (see p. 3266) can result from exposure to some anesthetics in genetically predisposed patients. Neuroleptic malignant syndrome (see p. 3268) can develop in patients taking antipsychotics. These disorders are life threatening; malignant hyperthermia has a high mortality rate.

Symptoms and Signs

Global CNS dysfunction, ranging from confusion to delirium, seizures, and coma, is the hallmark. Tachycardia, even when the patient is supine, and tachypnea are common. In classic

[Table 332-2. Some Differences Between Classic and Exertional Heatstroke]

heatstroke, the skin is hot and dry. In exertional heatstroke, sweating is relatively common. In both, temperature is > 40° C and may be > 46° C.

Diagnosis

• Clinical evaluation, including core temperature measurement

• Laboratory testing for organ dysfunction

Diagnosis is usually clear from a history of exertion and environmental heat. Heatstroke is differentiated from heat exhaustion by presence of the following:

• CNS dysfunction

• Temperature > 40° C

When the diagnosis of heatstroke is not obvious, other disorders that can cause CNS dysfunction and hyperthermia should be considered. These disorders include the following:

• Acute infection (eg, sepsis, malaria, meningitis, toxic shock syndrome)

• Drugs

• Neuroleptic malignant syndrome

• Serotonin syndrome

• Status epilepticus (interictal)

• Stroke

• Thyroid storm

Laboratory testing includes CBC, PT, PTT, electrolytes, BUN, creatinine, Ca, CK, and hepatic profile to evaluate organ function. A urethral catheter is placed to obtain urine, which is checked for occult blood by dipstick, and to monitor output. Tests to detect myoglobin are unnecessary. If a urine sample contains no RBCs but has a positive reaction for blood and if serum CK is elevated, myoglobinuria is likely. A urine drug screen may be helpful. Continual monitoring of core temperature, usually by rectal or esophageal probe, is desired.

Prognosis

Mortality rate is significant but varies markedly with age, underlying disorders, maximum temperature, and, most importantly, duration of hyperthermia and promptness of cooling. About 20% of survivors have residual brain damage, regardless of intervention. In some patients, renal insufficiency persists. Temperature may be labile for weeks.

Treatment

• Aggressive cooling

• IV cooled normal saline

The importance of rapid recognition and effective, aggressive cooling cannot be overemphasized. Cooling methods that do not cause shivering or cutaneous vasoconstriction are preferred, although ice-

soaked towels and ice water immersion are effective.

Cooling techniques: Evaporative cooling is comfortable and convenient and considered the most rapid method by some experts. During the process, patients are continually wetted with water, and the skin is fanned and vigorously massaged to promote blood flow. A spray hose and larger fans are best and may be used for large groups of people in the field. Comfortable, tepid (eg, 30° C) water is adequate because evaporation causes cooling; cold or ice water is not needed. Cool water immersion in a pond or stream can also be used in the field.

Ice packs applied to the axillae and groin can be used but not as the sole cooling method. In life-threatening cases, packing a patient in ice, with close monitoring, has been advocated to rapidly reduce core temperature.

Other measures: The patient is admitted to an ICU, and IV hydration with 0.9% saline solution is begun as in heat exhaustion (see p. 3265). Theoretically, 1 to 2 L of IV 0.9% saline cooled to 4° C, as used in protocols to induce hypothermia after cardiac arrest, may also help cooling. Organ dysfunction and rhabdomyolysis are treated (see elsewhere in THE MANUAL). An injectable benzodiazepine (eg, lorazepam, diazepam) may be used aggressively to prevent agitation and seizures (which increase heat production); seizures may occur during cooling. Because vomiting and aspiration of gastric contents are possible, measures to protect the airway may be required. Severely agitated patients may require paralysis and mechanical ventilation.

Platelets and fresh frozen plasma may be required for severe disseminated intravascular coagulation. IV $NaHCO_3$ to alkalinize the urine may help prevent nephrotoxicity if myoglobinuria is present. IV Ca salts may be necessary to treat hyperkalemic cardiotoxicity. Vasoconstrictors used to treat hypotension may reduce cutaneous blood flow and decrease heat loss. Hemodialysis may be required. Antipyretics (eg, acetaminophen) are of no value. Dantrolene is used to treat anesthetic-induced malignant hyperthermia but has no proven benefit for other causes of severe hyperthermia.

Malignant Hyperthermia

Malignant hyperthermia is a life-threatening elevation in body temperature usually resulting from a hypermetabolic response to concurrent use of a depolarizing muscle relaxant and a potent, volatile inhalational general anesthetic. Manifestations can include muscle rigidity, hyperthermia, tachycardia, tachypnea, rhabdomyolysis, and respiratory and metabolic acidosis. Diagnosis is clinical; patients at risk can be tested for their susceptibility. The highest priority treatments are rapid cooling and aggressive supportive measures.

The muscle relaxant involved is usually succinylcholine; the inhalational anesthetic is most often halothane, but other anesthetics (eg, isoflurane, sevoflurane, desflurane) may also be involved. This drug combination causes a similar reaction in some patients with muscular dystrophy and myotonia.

Pathophysiology

Malignant hyperthermia affects about 1/20,000 people. Susceptibility is inherited, with autosomal dominant inheritance and variable penetrance. Most often, the causative mutation affects the ryanodine receptor of skeletal muscle; however, > 22 other causative mutations have been identified.

The mechanism may involve anesthetic-induced potentiation of Ca exit from the sarcoplasmic reticulum of skeletal muscle in susceptible patients. As a result, Ca-induced biochemical reactions are accelerated, causing severe muscle contractions and elevation of the metabolic rate.

Complications: Hyperkalemia, respiratory and metabolic acidosis, hypocalcemia, and rhabdomyolysis with CK elevation and myoglobinemia may occur, as may coagulation abnormalities (particularly disseminated intravascular coagulation [DIC]). In older patients and patients with comorbidities, DIC may increase the risk of death.

Symptoms and Signs

Malignant hyperthermia may develop during anesthesia or the early postoperative period. Clinical presentation varies, depending on the drugs used and the patient's susceptibility. Muscular rigidity, especially in the jaw, is often the first sign, followed by tachycardia, other arrhythmias, tachypnea, acidosis, shock, and hyperthermia. Temperature is usually ≥ 40° C and may be extremely high (ie, > 43° C). Urine may appear brown or bloody if rhabdomyolysis and myoglobinuria have occurred.

Diagnosis

• Clinical evaluation

• Testing for complications

• Susceptibility testing for people at risk

The diagnosis is suspected by the appearance of typical symptoms and signs within 10 min to, occasionally, several hours after inhalational anesthesia is begun. Early diagnosis can be facilitated by prompt recognition of jaw rigidity, tachypnea, tachycardia, and increased end-tidal CO_2.

There are no immediately confirmatory tests, but patients should have testing for complications, including ECG, blood tests (CBC with platelets, electrolytes, BUN, creatinine, CK, Ca, PT, PTT, fibrinogen, D-dimer), and urine testing for myoglobinuria.

Other diagnoses must be excluded. Peri-operative sepsis may cause hyperthermia but rarely as soon after induction. Inadequate anesthesia can cause increased muscle tone and tachycardia but not elevated temperature. Thyroid storm and pheochromocytoma rarely manifest immediately after anesthetic induction.

Susceptibility testing: Testing for susceptibility to malignant hyperthermia is recommended for people at risk based on a family history of the disorder or a personal history of a severe or incompletely characterized previous adverse reaction to general anesthesia. The caffeine halothane contracture test (CHCT) is the most accurate. It measures the response of a muscle tissue sample to caffeine and halothane. This test can be done only at certain referral centers and requires excision of about 2 g of muscle tissue. Genetic testing has limited sensitivity (about 30%) but is quite specific; patients in whom a mutation is identified do not require the CHCT.

Treatment

• Rapid cooling and supportive measures

• Dantrolene

It is critical to cool patients as quickly and effectively as possible (see p. 3266) to prevent damage to the CNS and also to give patients supportive treatment to correct metabolic abnormalities. Outcome is best when treatment begins before muscular rigidity becomes generalized and before development of rhabdomyolysis, severe hyperthermia, and DIC. Dantrolene (2.5 mg/kg IV q 5 min as needed, up to a total dose of 10 mg/kg) should be given in addition to the usual physical cooling measures. In some patients, tracheal intubation, paralysis, and induced coma are required to control symptoms and provide support. Benzodiazepines given IV, often in high doses, can be used to control agitation. Malignant hyperthermia has a high mortality and may not respond to even early and aggressive therapy.

Prevention

Local or regional anesthesia is preferred to general anesthesia when possible. Potent inhalational anesthetics and depolarizing muscular relaxants should be avoided in patients who are susceptible. Nondepolarizing muscular blockers are the preferred preanesthetic drugs. Preferred anesthetics include barbiturates (eg, thiopental), etomidate, and propofol. Dantrolene should be available at the bedside.

Neuroleptic Malignant Syndrome

Neuroleptic malignant syndrome is characterized by altered mental status, muscle rigidity, hyperthermia, and autonomic hyperactivity that occur when certain neuroleptic drugs are used. Clinically, neuroleptic malignant syndrome resembles malignant hyperthermia. Diagnosis is clinical. Treatment is aggressive supportive care.

Among patients taking neuroleptic drugs, about 0.02 to 3% develop neuroleptic malignant syndrome. Patients of all ages can be affected.

Etiology

Many antipsychotics and antiemetics can be causative (see Table 332-3). The factor common to all drug causes is a decrease in dopaminergic transmission; however, the reaction is not allergic but rather idiosyncratic. Etiology and mechanism are unknown. Risk factors appear to include high drug doses, rapid dose increases, parenteral administration, and switching from one potentially causative drug to another.

Neuroleptic malignant syndrome can also occur in patients withdrawing from levodopa or dopamine agonists.

Symptoms and Signs

Symptoms begin most often during the first 2 wk of treatment but may occur earlier or after many years.

The 4 characteristic symptoms usually develop over a few days and often in the following order:

- Altered mental status: Usually the earliest manifestation is a change in mental status, often an agitated delirium, and may progress to lethargy or unresponsiveness (reflecting encephalopathy).

- Motor abnormalities: Patients may have generalized, severe muscle rigidity (sometimes with simultaneous tremor, leading to cog-wheel rigidity), or, less often, dystonias, chorea, or other abnormalities. Reflex responses tend to be decreased.

[**Table 332-3.** Drugs that Can Cause Neuroleptic Malignant Syndrome]

- Hyperthermia: Temperature is usually > 38° C and often > 40° C.

- Autonomic hyperactivity: Autonomic activity is increased, tending to cause tachycardia, arrhythmias, tachypnea, and labile hypertension.

Diagnosis

- Clinical evaluation

- Exclusion of other disorders and complications

The diagnosis should be suspected based on clinical findings. Early manifestations can be missed because mental status changes may be overlooked or dismissed in patients with psychosis.

Other disorders can cause similar findings. For example:

- Serotonin syndrome (see p. 3269) tends to cause rigidity, hyperthermia, and autonomic hyperactivity, but it is usually caused by SSRIs, and patients typically have hyperreflexia. Also, temperature elevations and muscle rigidity are usually less severe than in neuroleptic malignant syndrome, and nausea and diarrhea may precede serotonin syndrome.

- Malignant hyperthermia (see p. 3266) and withdrawal of intrathecal baclofen can cause findings similar

to those of neuroleptic malignant syndrome, but they are usually easily differentiated by history.

- Systemic infections, including sepsis (see p. 2299), pneumonia, and CNS infection, can cause altered mental status, hyperthermia, and tachypnea and tachycardia, but generalized motor abnormalities are not expected. Also, in neuroleptic malignant syndrome, unlike most infections, altered mental status and motor abnormalities tend to precede hyperthermia.

There are no confirmatory tests, but patients should have testing for complications, including serum electrolytes, BUN, creatinine, glucose, Ca, Mg, and CK, urine myoglobin, and usually neuroimaging and CSF analysis. EEG may be done to exclude nonconvulsive status epilepticus.

Treatment

- Rapid cooling, control of agitation, and other aggressive supportive measures

The causative drug is stopped and complications are treated supportively, usually in an ICU. Severe hyperthermia is treated very aggressively, mainly with physical cooling (see p. 3266). Some patients may require tracheal intubation and induced coma. Benzodiazepines, given IV in high doses, can be used to control agitation. Adjunctive drug therapy can be used, although efficacy has not been shown in clinical trials. Dantrolene (0.25 to 2 mg/kg IV q 6 to 12 h; maximum of 10 mg/kg/24 h) can be given for hyperthermia. Bromocriptine (2.5 mg q 6 to 8 h) or, alternatively, amantadine (100 to 200 mg q 12 h) can be given po or via NGT to help restore some dopaminergic activity. This condition may not respond to even rapid and aggressive therapy, and mortality in treated cases is about 10 to 20%.

Serotonin Syndrome

Serotonin syndrome is a potentially life-threatening condition resulting from increased CNS serotonergic activity that is usually drug related. Symptoms may include mental status changes, hyperthermia, and autonomic and neuromuscular hyperactivity. Diagnosis is clinical. Treatment is supportive.

Serotonin syndrome can occur with therapeutic drug use, self-poisoning, or, most commonly, unintended drug interactions when 2 serotonergic drugs are used (see Table 332-4). It can occur in all age groups.

Complications in severe serotonin syndrome can include metabolic acidosis, rhabdomyolysis, seizures, acute renal failure, and disseminated intravascular coagulation. Causes probably include severe hyperthermia and muscle activity.

Symptoms and Signs

Manifestations can range widely in severity. They can be grouped into the following categories:

- Mental status alterations: Anxiety, agitation and restlessness, easy startling, delirium

- Autonomic hyperactivity: Tachycardia, hypertension, hyperthermia, diaphoresis, shivering, vomiting, diarrhea

- Neuromuscular hyperactivity: Tremor, muscle hypertonia or rigidity, myoclonus, hyperreflexia, clonus (including ocular clonus), extensor plantar responses

Neuromuscular hyperactivity may be more pronounced in the lower than the upper extremities.

Symptoms usually resolve in 24 h, but symptoms may last longer after use of drugs that have a long half-life or active metabolites (eg, monoamine oxidase inhibitors, SSRIs).

Diagnosis

• Clinical criteria

Diagnosis is clinical. Various explicit criteria have been proposed.

The **Hunter criteria** are currently preferred because of ease of use and high accuracy (almost 85% sensitivity and > 95% specificity compared with diagnosis by a toxicologist). These criteria require that patients have taken a serotonergic drug and have one of the following:

• Muscle hypertonia

• Spontaneous clonus

• Tremor plus hyperreflexia

• Ocular or inducible clonus, plus either agitation, diaphoresis, or temperature > 38° C

Systemic infections, drug or alcohol withdrawal syndromes, and toxicity caused by sympathomimetic or anticholinergic drugs should also be considered in the differential diagnosis. Differentiation of serotonin syndrome from neuroleptic malignant syndrome

[**Table 332-4.** Drugs that Can Cause Serotonin Syndrome]

(see p. 3268) may be difficult because symptoms (eg, muscle rigidity, hyperthermia, autonomic hyperactivity, altered mental status) overlap. Clues to serotonin syndrome include use of serotonergic drugs, rapid onset (eg, within 24 h), and hyperreflexia, in contrast to the often decreased reflex responses in neuroleptic malignant syndrome.

There are no confirmatory tests, but patients should have testing to exclude other disorders (eg, CSF analysis for possible CNS infection, urine testing for drugs of abuse). Also, some tests (eg, serum electrolytes, platelet count, renal function tests, CK, PT, testing for urine myoglobin) may be necessary to identify complications in severe serotonin syndrome.

Treatment

• Supportive measures

• Sometimes cyproheptadine

All serotonergic drugs should be stopped. Mild symptoms are often relieved with sedation using a benzodiazepine. If symptoms rapidly resolve, patients should be observed for at least several hours. Most will require hospitalization for further testing, treatment, and monitoring.

In severe cases, admission to an ICU is required. Hyperthermia is treated by cooling (see p. 3266). Neuromuscular blockade with appropriate sedation, muscle paralysis, and other supportive measures may be necessary. Drug treatment of autonomic abnormalities (eg, hypertension, tachycardia) should be with shorter-acting drugs (eg, nitroprusside, esmolol) because autonomic effects can change rapidly.

If symptoms persist despite supportive measures, the serotonin antagonist cyproheptadine can be given orally or, after crushing, via NGT (12 mg, then 2 mg q 2 h until response occurs). Dantrolene is not recommended.

Consultation with a toxicologist is encouraged and can be accomplished by calling the United States Poison Control Network (1-800-222-1222) or accessing the WHO's list of international poison centers (www.who.int/ipcs/poisons/centre/directory/en).

Chapter 333. Cold Injury

Introduction

Exposure to cold may cause decreased body temperature (hypothermia) and focal soft-tissue injury. Tissue injury with freezing is frostbite. Tissue injury without freezing includes frostnip, immersion foot, and chilblains. Treatment is rewarming and selective, usually delayed, surgical treatment for injured tissues.

Susceptibility to all cold injury is increased by exhaustion, undernutrition, dehydration, hypoxia, impaired cardiovascular function, and contact with moisture or metal.

Prevention

Prevention is crucial. Several layers of warm clothing and protection against moisture and wind are important even when the weather does not seem to threaten cold injury. Clothing that remains insulating when wet (eg, made of wool or polypropylene) should be worn. Gloves and socks should be kept as dry as possible; insulated boots that do not impede circulation should be worn in very cold weather. A warm head covering is also important. Consuming ample fluids and food helps sustain metabolic heat production. Paying attention to when body parts become cold or numb and immediately warming them may prevent cold injury.

Nonfreezing Tissue Injuries

Acute or chronic injuries without freezing of tissue may result from cold exposure.

Frostnip: The mildest cold injury is frostnip. Affected areas are numb, swollen, and red. Treatment is rewarming, which causes pain and itching. Rarely, mild hypersensitivity to cold persists for months to years.

Immersion (trench) foot: Prolonged exposure to wet cold can cause immersion foot. Peripheral nerves and the vasculature are usually injured; muscle and skin tissue may be injured in severe cases.

Initially, the foot is pale, edematous, clammy, cold, and numb. Tissue maceration may occur if patients walk extensively. Rewarming causes hyperemia, pain, and often hypersensitivity to light touch, which persist for 6 to 10 wk. Skin may ulcerate, or a black eschar may develop. Autonomic dysfunction is common, with increased or decreased sweating, vasomotor changes, and local hypersensitivity to temperature change. Muscle atrophy and dysesthesia or anesthesia may occur and become chronic.

Immersion foot can be prevented by not wearing tight-fitting boots, keeping feet and boots dry, and changing socks frequently. Immediate treatment is rewarming by immersing the affected area in warm (40 to 42° C) water, followed by sterile dressings. Chronic neuropathic symptoms are difficult to treat; amitriptyline may be tried (see p. 1632).

Chilblains (pernio): Localized areas of erythema, swelling, pain, and pruritus result from repeated exposure to dry cold; the mechanism is unclear. Blistering or ulceration may occur. Chilblains most commonly affects the fingers and pretibial area and is self-limited. Occasionally, symptoms recur.

Pernio is often used to refer to a vasculitic disorder most common among young females with a history of Raynaud's syndrome. Endothelial and neuronal damage results in vasospasm and exaggerated sympathetic response when exposed to cold. Nifedipine 20 mg po tid may be effective for refractory pernio. Sympatholytic drugs may also help.

Frostbite

Frostbite is injury due to freezing of tissue. Initial presentation may be deceptively benign. Skin may appear white or blistered and is numb; rewarming causes substantial pain. Gangrene may develop. Severely damaged tissue may autoamputate. Treatment is rewarming in warm (40 to 42° C) water and local care. Surgical amputation is occasionally necessary, but a decision, often

guided by imaging results, should usually be delayed until after definitive demarcation of necrotic tissue.

Frostbite usually occurs in extreme cold, especially at high altitude, and is aggravated by hypothermia. Distal extremities and exposed skin are affected most often.

Ice crystals form within or between tissue cells, essentially freezing the tissue and causing cell death. Adjacent unfrozen areas are at risk because local vasoconstriction and thrombosis can cause endothelial and ischemic damage. With reperfusion during rewarming, inflammatory cytokines (eg, thromboxanes, prostaglandins) are released, exacerbating tissue injury.

Symptoms and Signs

The affected area is cold, hard, white, and numb. When warmed, the area becomes blotchy red, swollen, and painful. Blisters form within 4 to 6 h, but the full extent of injury may not be apparent for several days. Blisters filled with clear serum indicate superficial damage; blood-filled, proximal blisters indicate deep damage and likely tissue loss. Superficial damage heals without residual tissue loss. Freezing of deep tissue causes dry gangrene with a hard black carapace over healthy tissue. Wet gangrene, which is gray, edematous, and soft, is less common. Wet gangrene is characterized by infection, but dry gangrene is less likely to become infected. Depth of tissue loss depends on duration and depth of freezing. Severely damaged tissue may autoamputate. Compartment syndrome may develop. All degrees of frostbite may cause long-term neuropathic symptoms: sensitivity to cold, excessive sweating, faulty nail growth, and numbness (symptoms resembling those of complex regional pain syndrome—see p. 1633—although any relationship is speculative).

Diagnosis

• Clinical evaluation

Diagnosis is based on clinical findings. However, because many of the early characteristics of frostbite (eg, coldness, numbness, white or red color, blisters) are also characteristic of nonfreezing cold injuries, differentiation of frostbite may require repeated observation until more specific characteristics (eg, black carapace, gangrene) develop.

Treatment

• Rewarming in warm (40 to 42° C) water

• Supportive measures

• Local wound care

• Sometimes delayed surgery

Prehospital care: In the field, frostbitten extremities should be rewarmed rapidly by totally immersing the affected area in water that is tolerably warm to the touch (40 to 42° C, ideally about 40.5° C). Because the area is numb, rewarming with an uncontrolled dry heat source (eg, fire, heating pad) risks burns. Rubbing may further damage tissue and is avoided. The longer an area remains frozen, the greater the ultimate damage may be. However, thawing the feet is inadvisable if a patient must walk any distance to receive care because thawed tissue is particularly sensitive to the trauma of walking and, if refrozen, will be more severely damaged than if left frozen. If thawing must be delayed, the frozen area is gently cleaned, dried, and protected in sterile compresses. Patients are given analgesics, if available, and the whole body is kept warm.

Acute care: Once the patient is in the hospital, core temperature is stabilized and extremities are rapidly rewarmed in large containers of circulating water kept at about 40.5° C; 15 to 30 min is usually adequate. Thawing is often mistakenly ended prematurely because pain may be severe during rewarming. Parenteral analgesics, including opioids, may be used. Patients are encouraged to move the affected part

gently during thawing. Large, clear blisters are left intact or aspirated using sterile technique. Hemorrhagic blisters are left intact to avoid secondary desiccation of deep dermal layers. Broken vesicles are debrided. If there is no perfusion after thawing, thrombolytic (fibrinolytic) therapy is considered.

Anti-inflammatory measures (eg, topical aloe vera q 6 h, ibuprofen 400 mg po q 8 h, ketorolac 30 to 60 mg IV) probably help. Affected areas are left open to warm air, and extremities are elevated to decrease edema. Anticoagulants, IV low molecular weight dextran, and intra-arterial vasodilators (eg, reserpine, tolazoline) have no proven clinical benefit. Phenoxybenzamine (10 to 60 mg po once/day), a long-acting α -blocker, may theoretically decrease vasospasm and improve blood flow.

Preventing infection is fundamental; streptococcal prophylaxis (eg, with penicillin) is sometimes provided. If wet gangrene is present, broad-spectrum antibiotics are used. Tetanus toxoid is given if vaccination is not up to date. If tissue damage is severe, tissue pressure is monitored.

Ongoing care: Adequate nutrition is important to sustain metabolic heat production.

Imaging tests (eg, radionuclide scanning, MRI, microwave thermography, laser-Doppler flowmetry) can help assess circulation, determine tissue viability, and thus guide treatment. MRI and particularly magnetic resonance angiography may establish the line of demarcation before clinical demarcation and thus make earlier surgical debridement or amputation possible. However, whether earlier surgery improves long-term outcome is unclear. Usually, surgery is delayed as long as possible because the black carapace is often shed, leaving viable tissue. Patients with severe frostbite are warned that many weeks of observation may be required before demarcation and the extent of tissue loss become apparent.

Whirlpool baths at 37° C 3 times/day followed by gentle drying, rest, and time are the best long-term management. No totally effective treatment for the long-lasting symptoms of frostbite (eg, numbness, hypersensitivity to cold) is known, although chemical or surgical sympathectomy may be useful for late neuropathic symptoms.

Hypothermia

Hypothermia is a core body temperature < 35°C. Symptoms progress from shivering and lethargy to confusion, coma, and death. Mild hypothermia requires a warm environment and insulating blankets (passive rewarming). Severe hypothermia requires active rewarming of the body surface (eg, with forced-air warming systems, radiant sources) or core (eg, inhalation, heated infusion and lavage, extracorporeal blood rewarming).

Primary hypothermia causes about 600 deaths each year in the US. Hypothermia also has a significant and underrecognized effect on mortality risk in cardiovascular and neurologic disorders.

Etiology

Hypothermia results when body heat loss exceeds body heat production. Hypothermia is most common during cold weather or immersion in cold water, but it may occur in warm climates when people lie immobile on a cool surface (eg, when they are intoxicated) or after very prolonged immersion in swimming-temperature water (eg, 20 to 24° C). Wet clothing and wind chill increase risk of hypothermia.

Conditions that cause loss of consciousness, immobility, or both (eg, trauma, hypoglycemia, seizure disorders, stroke, drug or alcohol intoxication) are common predisposing factors. The elderly and the very young also are at high risk. The elderly often have diminished temperature sensation and impaired mobility and communication, resulting in a tendency to remain in an overly cool environment. These impairments, combined with diminished subcutaneous fat, contribute to hypothermia in the elderly —sometimes even indoors in cool rooms. The very young have similarly diminished mobility and communication and have an increased surface area/mass ratio, which enhances heat loss. Intoxicated people who lose consciousness in a cold environment are likely to become hypothermic.

Pathophysiology

Hypothermia slows all physiologic functions, including cardiovascular and respiratory systems, nerve conduction, mental acuity, neuromuscular reaction time, and metabolic rate. Thermoregulation ceases below about 30° C; the body must then depend on an external heat source for rewarming. Renal cell dysfunction and decreased levels of ADH lead to production of a large volume of dilute urine (cold diuresis). Diuresis plus fluid leakage into the interstitial tissues causes hypovolemia. Vasoconstriction, which occurs with hypothermia, may mask hypovolemia, which then manifests as sudden shock or cardiac arrest during rewarming (rewarming collapse) when peripheral vasculature dilates.

Immersion in cold water can trigger the diving reflex, which involves reflex vasoconstriction in visceral muscles; blood is shunted to essential organs (eg, heart, brain). The reflex is most pronounced in small children and may help protect them. Also, hypothermia due to total immersion in near-freezing water may protect the brain from hypoxia by decreasing metabolic demands. The decreased demand probably accounts for the occasional survival after prolonged cardiac arrest due to extreme hypothermia.

Symptoms and Signs

Intense shivering occurs initially, but it ceases below about 31° C, allowing body temperature to drop more precipitously. CNS dysfunction progresses as body temperature decreases; people do not sense the cold. Lethargy and clumsiness are followed by confusion, irritability, sometimes hallucinations, and eventually coma. Pupils may become unreactive. Respirations and heartbeat slow and ultimately cease. Initially, sinus bradycardia is followed by slow atrial fibrillation; the terminal rhythm is ventricular fibrillation or asystole. However, these rhythms are potentially less ominous than in normothermia.

Diagnosis

• Core temperature measurement

• Consideration of intoxication, myxedema, sepsis, and trauma

Diagnosis is by core temperature, not oral temperature. Electronic thermometers are preferred; many standard mercury thermometers have a lower limit of 34° C. Rectal and esophageal probes are most accurate.

Laboratory tests include CBC, glucose, electrolytes, BUN, creatinine, and ABGs. ABGs are not corrected for low temperature. ECG typically shows J (Osborn) waves (see Fig. 333-1) and interval prolongation (PR, QRS, QT), although these findings are not always present. Causes are sought. If the cause is unclear, alcohol level is measured, and drug screening and thyroid function tests are done. Sepsis and occult head or skeletal trauma must be considered.

Prognosis

Patients who have been immersed in icy water for 1 h or (rarely) longer have sometimes been successfully rewarmed without permanent brain damage (see p. 3281), even when core temperature was 13.7° C or when pupils were unreactive. Outcome is difficult to predict and cannot be based on the Glasgow Coma Scale. Grave prognostic markers include evidence of cell lysis (hyperkalemia > 10 mEq/L), intravascular thrombosis (fibrinogen < 50 mg/dL), and presence of a nonperfusing cardiac rhythm (ventricular fibrillation or asystole). For a given degree and duration of hypothermia, children are more likely to recover than adults.

Treatment

• Drying and insulation

• Fluid resuscitation

• Active rewarming unless hypothermia is mild, accidental, and uncomplicated

The first priority is to prevent further heat loss by removing wet clothing and insulating the patient.

Subsequent measures depend on how severe hypothermia is and whether cardiovascular instability or cardiac arrest is present. Returning patients to a normal temperature is less urgent in hypothermia than in severe hyperthermia. For stable patients, elevation of core temperature by 1° C/h is acceptable.

If hypothermia is mild and thermoregulation is present (indicated by shivering and temperature typically 31 to 35° C), insulation with heated blankets and warm fluids to drink are adequate.

Fluid resuscitation is essential for hypovolemia. Patients are given 1 to 2 L of 0.9% saline solution (20 mL/kg for children) IV; the solution is heated if possible to 45° C. More is given as needed to maintain perfusion.

Active rewarming is required if patients have cardiovascular instability, temperature < 32.2° C, hormone insufficiency (such as hypoadrenalism or hypothyroidism), or hypothermia secondary to trauma, toxins, or predisposing disorders. If body temperature is at the warmer end of the range, external rewarming with forced hot air enclosures may be used. Patients with lower temperatures, particularly those with low BP or cardiac arrest, require core rewarming.

[**Fig. 333-1.** Abnormal ECG showing J (Osborn) waves (V4).]

Core rewarming options include

• Inhalation

• IV infusion

• Lavage

• Extracorporeal core rewarming (ECR)

Inhalation of heated (40 to 45° C), humidified O_2 via mask or endotracheal tube eliminates respiratory heat loss and can add 1 to 2° C/h to the rewarming rate.

IV crystalloids or blood should be heated to 40 to 42° C, especially with massive volume resuscitations.

Heated lavage of the bladder or GI tract transfers minimal heat, although closed thoracic lavage through 2 thoracostomy tubes is very efficient in severe cases. Peritoneal lavage with 40 to 45° C dialysate requires 2 catheters with outflow suction and is especially useful with severely hypothermic patients who have rhabdomyolysis, toxin ingestions, or electrolyte abnormalities.

There are 4 types of ECR: hemodialysis, venovenous, arteriovenous, and cardiopulmonary bypass. ECR measures require a prearranged protocol with appropriate specialists. Although they are intuitively attractive and heroic, these measures are not routinely available, and they are not commonly used in most hospitals.

CPR is not done if patients have a perfusing rhythm, even if pulses are not palpable. Fluids are given, and active rewarming is done. Hypotension and bradycardia are expected when core temperature is low and, if due solely to hypothermia, need not be aggressively treated. Patients with a nonperfusing rhythm require cardiopulmonary resuscitation. Chest compressions and endotracheal intubation are done. Defibrillation is difficult if body temperature is low; one attempt may be made, but if ineffective, further attempts are deferred until temperature reaches > 28° C. Advanced life support should be continued until temperature reaches 32° C unless obviously lethal injuries or disorders are present. However, advanced cardiac life-support drugs (eg, antiarrhythmics, vasopressors, inotropes) are usually not given. Low-dose dopamine (1 to 5 µg/kg/min) or other catecholamine infusions are typically reserved for patients who have disproportionately severe hypotension and who do not respond to fluid resuscitation and rewarming. Severe hyperkalemia (> 10 mEq/L) during resuscitation typically indicates a fatal outcome and can guide resuscitation efforts.

Chapter 334. Altitude Sickness

Altitude sickness (AS) includes several related syndromes caused by decreased O_2 availability at high altitudes. Acute mountain sickness (AMS), the mildest form, is headache plus one or more systemic manifestations. High-altitude cerebral edema (HACE) is encephalopathy in people with AMS. High-altitude pulmonary edema (HAPE) is a form of noncardiogenic pulmonary edema causing severe dyspnea and hypoxemia. AMS may occur in recreational hikers and skiers in mountains. Diagnosis is clinical. Treatment of mild AMS is with analgesics and acetazolamide. Severe syndromes require descent and supplemental O_2 if available. In addition, dexamethasone may be useful for HACE, and nifedipine may be useful for HAPE.

As altitude increases, atmospheric pressure decreases while the percentage of O_2 in air remains constant; thus, the partial pressure of O_2 decreases with altitude and, at 5800 m (19,000 ft), is about one half that at sea level.

Most people can ascend to 1500 to 2000 m (5000 to 6500 ft) in one day without problems, but about 20% of those who ascend to 2500 m (8000 ft) and 40% of those who ascend to 3000 m (10,000 ft) develop some form of AS. Rate of ascent, maximum altitude reached, and sleeping altitude influence the likelihood of developing the disorder.

Risk factors: Effects of high altitude vary greatly among individuals. But generally, risk is increased by

• Going too high too fast

• Exertion

Risk is greater in people who have had previous AS and in those who live at low altitude (< 900 m [< 3000 ft]). Young children and young adults are probably more susceptible. Disorders such as diabetes, coronary artery disease, and mild COPD are not risk factors for AS, but hypoxia may adversely affect these disorders. Physical fitness is not protective.

Pathophysiology

Acute hypoxia (eg, as occurs during rapid ascent to high altitude in an unpressurized aircraft) alters CNS function within minutes. However, AS results from the body's neurohumoral and hemodynamic responses to hypoxia and develops over hours to days.

The CNS and lungs are primarily affected. In both, elevated capillary pressure, capillary leakage, and consequent edema formation probably occur.

In the lungs, hypoxia-induced elevation of pulmonary artery pressure causes interstitial and alveolar pulmonary edema, resulting in impaired oxygenation. Small-vessel hypoxic vasoconstriction is patchy, causing overperfusion with elevated pressure, capillary wall damage, and capillary leakage in less constricted areas. Various additional mechanisms have been proposed; they include sympathetic overactivity, endothelial dysfunction, decreased alveolar nitric oxide (perhaps due to decreased nitric oxide synthase), and a defect in the amiloride-sensitive Na channel. Some of these factors may have a genetic component.

Pathophysiology in the CNS is less clear but may involve a combination of hypoxia-induced cerebral vasodilation, alteration of the blood-brain barrier, and Na and water retention causing cerebral edema. One hypothesis is that patients with a low ratio of CSF to brain volume are less able to tolerate swelling (ie, by displacement of CSF) and thus are more likely to develop AS. The roles of atrial natriuretic peptide, aldosterone, renin, and angiotensin are unclear.

Acclimatization: Acclimatization is an integrated series of responses that gradually restores tissue oxygenation toward normal in people exposed to altitude. However, in spite of acclimatization, all people at high altitude have tissue hypoxia. Most people acclimatize to altitudes of up to 3000 m (10,000 ft) in a

few days. The higher the altitude, the longer full acclimatization takes. However, no one can fully acclimatize to long-term residence at altitudes > 5100 m (> 17,000 ft).

Features of acclimatization include sustained hyperventilation, which increases tissue oxygenation but also causes respiratory alkalosis. Blood pH tends to normalize within days as HCO_3 is excreted in urine; as pH normalizes, ventilation can increase further. Cardiac output increases initially; RBC mass and tolerance for aerobic work also increase. After many generations at altitude, some ethnic groups have adapted in slightly different ways.

Symptoms and Signs

The clinical forms of AS are not separate entities but parts of a spectrum in which one or more forms may be present in different degrees.

Acute mountain sickness (AMS): This form is by far the most common and may develop at altitudes as low as 2000 m (6500 ft). It may be due to mild cerebral edema and is characterized by headache plus at least one of the following: fatigue, GI symptoms (anorexia, nausea, vomiting), dizziness, and sleep disturbance. Exertion aggravates the symptoms. Symptoms typically develop 6 to 10 h after ascent and subside in 24 to 48 h, but they occasionally evolve into HAPE, HACE, or both. AMS is common at ski resorts, and some people affected by it mistakenly attribute it to excessive alcohol intake (hangover) or a viral illness.

High-altitude cerebral edema (HACE): Marked cerebral edema manifests as headache and diffuse encephalopathy with confusion, drowsiness, stupor, and coma. Gait ataxia is a reliable early warning sign. Seizures and focal deficits (eg, cranial nerve palsy, hemiplegia) are less common. Papilledema and retinal hemorrhage may be present but are not necessary for diagnosis. Coma and death may occur within a few hours.

High-altitude pulmonary edema (HAPE): HAPE usually develops 24 to 96 h after rapid ascent to > 2500 m (> 8000 ft) and is responsible for most deaths due to AS. Respiratory infections, even minor ones, appear to increase risk. HAPE is more common among men (unlike other forms of AS). Long-time high-altitude residents can develop HAPE when they return after a brief stay at low altitude.

Initially, patients have dyspnea, decreased exertion tolerance, and dry cough. Pink or bloody sputum and respiratory distress are later findings. On examination, cyanosis, tachycardia, tachypnea, and low-grade fever (< 38.5° C) are common. Focal or diffuse rales (sometimes audible without a stethoscope) are usually present. HAPE may worsen rapidly; coma and death may occur within hours.

Other disorders: Peripheral and facial edema is common at high altitude.

Headache, without other symptoms of AMS, is also common.

Retinal hemorrhages may develop at altitudes as low as 2700 m (9000 ft) and are common at > 5000 m (> 16,000 ft). They are usually asymptomatic unless they occur in the macular region; they resolve rapidly without sequelae.

People who have had radial keratotomy may have significant visual disturbances at altitudes > 5000 m (> 16,000 ft) or even as low as 3000 m (10,000 ft). These alarming symptoms disappear rapidly after descent.

Chronic mountain sickness (Monge's disease) is a disorder that affects long-time high-altitude residents; it is characterized by fatigue, dyspnea, aches and pains, cyanosis, excessive polycythemia, and occasionally thromboembolism. The disorder often involves alveolar hypoventilation. Patients should descend to low altitude; recovery is slow, and return to high altitude may cause recurrence. Repeated phlebotomy can reduce polycythemia, but polycythemia may recur.

Diagnosis

• Clinical evaluation

Diagnosis of most forms of AS is clinical; laboratory tests are nonspecific and usually unnecessary. HACE can usually be differentiated from other causes of coma (eg, infection, ketoacidosis) by the history and by absence of fever and nuchal rigidity. If done, blood and CSF studies are normal. In HAPE, hypoxemia is often severe, with pulse oximetry showing 40 to 70% saturation. If obtained, chest x-ray shows a normal-sized heart and patchy lung edema (often middle or lower lobes), unlike what is seen in heart failure.

Treatment

• For mild or moderate AMS, halting of ascent and treatment with fluids, analgesics, and sometimes acetazolamide

• For severe symptoms, immediate descent and treatment with O_2, drugs, and pressurization

AMS: Patients should halt ascent and reduce exertion until symptoms resolve. Other treatment includes fluids and analgesics for headache. For severe symptoms, descent of 500 to 1000 m (1650 to 3200 ft) is usually rapidly effective. Acetazolamide 250 mg po bid may relieve symptoms and improve sleep.

HAPE and HACE: Patients should descend to low altitude immediately. If descent is delayed, patients should rest and be given O_2. If descent is impossible, O_2, drugs, and pressurization in a portable hyperbaric bag help buy time but are not substitutes for descent.

For HAPE, nifedipine 10 mg sublingually followed by a 30-mg slow-release tablet lowers pulmonary artery pressure and is beneficial. Diuretics (eg, furosemide) are contraindicated. The heart is normal in HAPE, and digitalis is of no value. When promptly treated by descent, patients usually recover from HAPE within 24 to 48 h. People who have had one episode of HAPE are likely to have another and should be so warned.

For HACE (and severe AMS), dexamethasone 4 to 8 mg initially, followed by 4 mg q 6 h, may help. It may be given po, sc, IM, or IV. Acetazolamide 250 mg po bid may be added.

Prevention

The most important measure is a slow ascent. Drinking extra water is important because breathing large volumes of dry air at altitude greatly increases water loss, and dehydration with some degree of hypovolemia aggravates symptoms. Alcohol seems to worsen AMS and reduces nocturnal ventilation, thus accentuating sleep disturbance. Although physical fitness enables greater exertion at altitude, it does not protect against any form of AS.

Ascent: Graded ascent is essential for activity at > 2500 m (> 8000 ft). Sleeping on the first night should be at < 2500 to 3000 m (8,000 to 10,000 ft), and climbers should sleep at that altitude for 2 to 3 nights if subsequent higher sleeping altitudes are planned. Each day thereafter, sleeping altitude can be increased by about 300 m (1000 ft), although higher day hikes are acceptable with return to the lower level for sleep. Climbers vary in ability to ascend without developing symptoms; a climbing party should be paced for its slowest member.

Acclimatization reverses quickly. After descent to low levels for more than a few days, acclimatized climbers should once more follow a graded ascent.

Drugs: Acetazolamide 125 to 250 mg po q 12 h reduces the incidence of AMS. Sustained-release capsules (500 mg once/day) are also available. Acetazolamide can be started on the day of the ascent; it acts by inhibiting carbonic anhydrase and thus increasing ventilation. Acetazolamide 125 mg po at bedtime reduces the amount of periodic breathing (almost universal during sleep at high altitude), thus limiting sharp falls in blood O_2. Acetazolamide should not be given to patients allergic to sulfa drugs. Analogs of acetazolamide offer no advantage. Acetazolamide may cause numbness and paresthesias of the fingers; these symptoms are benign but can be annoying. Carbonated drinks taste flat to people

taking acetazolamide. Dexamethasone 2 mg po q 6 h is an alternative to acetazolamide.

Low-flow O_2 during sleep at altitude is effective but inconvenient and may pose logistic difficulties.

Patients who have had a previous episode of HAPE should consider prophylaxis with sustained-release nifedipine 20 to 30 mg po bid. Inhaled β-agonists may also be effective.

Analgesics may prevent high-altitude headache.

Chapter 335. Motion Sickness

Motion sickness is a symptom complex that usually includes nausea, often accompanied by vague abdominal discomfort, vomiting, dizziness, and related symptoms; it is caused by repetitive angular and linear acceleration and deceleration. Behavioral change and drug therapy can help prevent or control symptoms.

Individual susceptibility to motion sickness varies greatly. However, motion sickness is more common among women, and incidence ranges from < 1% on airplanes to nearly 100% on ships in rough seas and upon becoming weightless during space travel.

Etiology

Excessive stimulation of the vestibular apparatus by motion is the primary cause. Afferent pathways from the labyrinth to the vomiting center in the medulla are undefined, but motion sickness occurs only when the 8th cranial nerve and cerebellar vestibular tracts are intact. Movement via any form of transportation, including ship, motor vehicle, train, plane, spacecraft, and playground or amusement park ride, can cause excessive vestibular stimulation.

The trigger may involve conflicting vestibular, visual, and proprioceptive inputs. For example, visual input that indicates being stationary may conflict with the sensation of movement (eg, looking at an apparently unmoving ship cabin wall while sensing the ship rolling). Alternatively, moving visual input may conflict with lack of perception of movement (eg, viewing a rapidly moving slide with a microscope or watching a virtual reality game while sitting still). Another possible trigger is a pattern of motion that differs from the expected pattern (eg, in a zero gravity environment, floating instead of falling).

Risk factors: Factors that may increase the risk of developing motion sickness or increase the severity of symptoms include the following:

• Poor ventilation (eg, with fumes, smoke, or carbon monoxide)

• Emotional factors (eg, fear, anxiety)

• Migraine headaches

• Labyrynthitis

• Hormonal factors (eg, pregnancy, use of hormonal contraceptives)

In **space adaptation syndrome** (motion sickness during space travel), weightlessness (zero gravity) is an etiologic factor. This syndrome reduces the efficiency of astronauts during the first few days of space flight, but adaptation occurs over several days.

Symptoms and Signs

Nausea and vague abdominal discomfort are characteristic. Vomiting may also occur. These symptoms may be preceded by yawning, hyperventilation, salivation, pallor, profuse cold sweating, and somnolence. Other symptoms include aerophagia, dizziness, headache, fatigue, weakness, and inability to concentrate. Pain, shortness of breath, and visual and speech disturbances are absent. With prolonged exposure to motion, patients often adapt. However, symptoms may recur if motion increases or if motion resumes after a short respite from the inciting trigger.

Prolonged vomiting due to motion sickness may rarely lead to dehydration with hypotension, inanition, and depression.

Diagnosis

• Clinical evaluation

The diagnosis is suspected in patients with compatible symptoms who have been exposed to typical triggers. Diagnosis is clinical and usually straightforward. However, the possibility of another diagnosis (eg, CNS hemorrhage or infarction) should be considered in some people, particularly the elderly and patients with no prior history of motion sickness or those with risk factors for CNS hemorrhage or infarction, who develop acute dizziness and vomiting during travel. Patients with focal neurologic symptoms or signs, significant headache, or other findings atypical of motion sickness should be further evaluated.

Treatment

• Scopolamine, antihistamines, or antidopaminergic drugs

• Positioning

• Avoidance of alcoholic beverages and over-eating

People prone to motion sickness should take prophylactic drugs and use other preventive measures before symptoms start; interventions are less effective after symptoms develop. If vomiting occurs, an antiemetic, given rectally or parenterally, can be effective. If vomiting is prolonged, IV fluids and electrolytes may be required for replacement and maintenance.

Scopolamine: Scopolamine, an anticholinergic drug, is effective for prevention, but efficacy in treatment is uncertain. Scopolamine is available as a prescription transdermal patch or in oral form. The patch is a good choice for longer trips because after being applied behind the ear at least 4 h before travel (optimally 8 to 12 h), it is effective for up to 72 h as it releases about 1 mg. The oral form of scopolamine is given as 0.4 mg to 0.8 mg 1 h before travel and then q 8 h as needed.

Adverse effects, which include drowsiness, blurred vision, dry mouth, and bradycardia, occur less commonly with patches. Inadvertent contamination of the eye with patch residue may cause a fixed and widely dilated pupil. Additional adverse effects of scopolamine in the elderly can include confusion, hallucinations, and urinary retention. Scopolamine is contraindicated in people who are at risk of angle-closure glaucoma.

Scopolamine can be used by children > 12 yr in the same dosages as for adults. Use in children ≤ 12 yr may be safe but is not recommended due to the higher risk of adverse effects.

Antihistamines: The mechanism of action for antihistamines is probably anticholinergic. These drugs can be effective for prevention and possibly treatment. Beginning 1 h before departure, susceptible people may be given nonprescription dimenhydrinate, diphenhydramine, or meclizine 25 to 50 mg po qid (dimenhydrinate for children 2 to 6 yr, 12.5 to 25 mg po q 6 to 8 h, maximum 75 mg/day; for children 6 to 12 yr, 25 to 50 mg po q 6 to 8 h, maximum 150 mg/day), or cyclizine 50 mg po qid (for children 6 to 12 yr, 25 mg tid) to minimize vagally mediated GI symptoms. However, anticholinergic adverse effects may be troublesome, especially in the elderly. Nonsedating antihistamines do not appear to be effective.

Antidopaminergic drugs: Promethazine (25 to 50 mg po 1 h before departure and then bid; for children < 12 yr, 0.5 mg/kg po 1 h before departure and then bid) appears to be effective for prevention and treatment; adding caffeine may increase efficacy. Metoclopramide may also be effective, but evidence suggests it is less so than promethazine.

Nondrug measures: Susceptible people should minimize exposure by positioning themselves where motion is the least (eg, in the middle of a ship close to water level, over the wings in an airplane). Also, they should try to minimize the discrepancy between visual and vestibular stimuli. If traveling in a motor vehicle, driving or riding in the front passenger seat, where vehicle motion is most evident, is best. When traveling on a ship, viewing the horizon or land masses is usually better than viewing a cabin wall. Whatever the form of transportation, reading and rear-facing seats should be avoided. A supine or semirecumbent position with the head supported is best.

Adequate ventilation helps prevent symptoms. Consuming alcoholic beverages and overeating before or during travel increase the likelihood of motion sickness. Small amounts of fluids and bland food consumed frequently are preferred to large meals during extended travel; some people find that dry crackers and carbonated beverages, especially ginger ale, are best. If travel time is short, food and fluids should be avoided.

In space adaptation syndrome, movement, which aggravates the symptoms, should be avoided.

Alternative therapies: Some alternative therapies are unproved but may be helpful. These alternative therapies include wrist-bands that apply acupressure and wristbands that apply electrical stimulation. Both can be safely used by people of all ages. Ginger (0.5- to 1-g dose, which can be repeated but should be limited to 4 g/day) may help prevent motion sickness.

Chapter 336. Drowning

Drowning is respiratory impairment resulting from submersion in a liquid medium. It can be nonfatal (previously called near drowning) or fatal. Drowning results in hypoxia, which can damage multiple organs, including the lungs and brain. Treatment is supportive, including reversal of respiratory and cardiac arrest, hypoxia, hypoventilation, and hypothermia.

Drowning is one of the leading causes of accidental death in the US. It is the 2nd most common cause of death in children ages 1 to 14 yr. Rates are higher for the following:

- Children < 4 yr
- Children from African American, immigrant, or impoverished families
- Males
- People who have used alcohol or sedatives
- People with conditions that cause temporary incapacitation (eg, seizure, hypoglycemia, stroke, MI, cardiac arrhythmia)
- People with a long QT syndrome (swimming can trigger arrhythmias that cause unexplained drowning in people with a long QT syndrome, particularly LQT1)

Drowning is common in pools, hot tubs, natural water settings, and, among infants and toddlers, in toilets, bathtubs, buckets of water, and cleaning fluids. About 4 times as many people are hospitalized for nonfatal drowning as for fatal drowning.

Pathophysiology

Hypoxia: Hypoxia is the major insult in drowning, affecting the brain, heart, and other tissues; respiratory arrest followed by cardiac arrest may occur. Brain hypoxia may cause cerebral edema and, occasionally, permanent neurologic sequelae. Generalized tissue hypoxia may cause metabolic acidosis. Immediate hypoxia results from aspiration of fluid or gastric contents, acute reflex laryngospasm, or both. Lung injury due to aspiration or hypoxia itself may cause delayed hypoxia. Aspiration, particularly with particulate matter or chemicals, may cause chemical pneumonitis or secondary bacterial pneumonia and may impair alveolar secretion of surfactant, resulting in patchy atelectasis. Extensive atelectasis may make the affected areas of the lungs stiff, noncompliant, and poorly ventilated, potentially causing respiratory failure (see p. 2284) with hypercapnia and respiratory acidosis. Perfusion of poorly ventilated areas of the lungs (V/Q mismatch) worsens hypoxia. Alveolar hypoxia may cause noncardiogenic pulmonary edema.

Hypothermia: Exposure to cold water induces systemic hypothermia (see p. 3273), which can be a significant problem. However, hypothermia can be protective by stimulating the mammalian diving reflex, slowing the heartbeat, and constricting the peripheral arteries, shunting oxygenated blood away from the extremities and the gut to the heart and brain. Also, hypothermia decreases the O_2 needs of tissues, possibly prolonging survival and delaying the onset of hypoxic tissue damage. The diving reflex and overall clinically protective effects of cold water are usually greatest in young children.

Fluid aspiration: Laryngospasm often limits the volume of fluid aspirated; however, large volumes of water are occasionally aspirated, rarely enough to change electrolyte concentrations and blood volume. Seawater may increase Na and Cl slightly. In contrast, large quantities of fresh water can decrease electrolyte concentration significantly, increase blood volume, and cause hemolysis.

Associated injuries: Skeletal, soft-tissue, head, and internal injuries may occur. People who dive into shallow water may sustain cervical and other spine injuries (which may be the cause of drowning).

Rarely, drowning occurs when people develop carbon monoxide poisoning when they are swimming near

an exhaust port of a boat. Only a few breaths may cause unconsciousness.

Symptoms and Signs

Panic and air hunger occur. Children who are unable to swim may become submerged in < 1 min, more rapidly than adults. After rescue, anxiety, vomiting, wheezing, and altered consciousness are common. Patients may have respiratory failure with tachypnea, retractions, or cyanosis. Sometimes respiratory symptoms are delayed until several hours after submersion.

Diagnosis

- For concomitant injuries, clinical evaluation and sometimes imaging studies
- Pulse oximetry and, if results are abnormal or if respiratory symptoms and signs are present, ABG and chest x-ray
- Core temperature measurement to rule out hypothermia
- Possibly evaluation for causative disorders (eg, hypoglycemia, MI)
- Ongoing monitoring as indicated for delayed respiratory complications

Most people are found in or near water, making the diagnosis obvious clinically. Resuscitation may need to precede completion of the diagnostic assessment. Cervical spine injury is assumed, and the spine is immobilized in patients who have altered consciousness or whose mechanism of injury involves diving. Procedures to remove water from the lungs are generally not helpful. Secondary head injury and conditions that may have contributed to drowning (eg, hypoglycemia, stroke, MI) are considered.

All patients undergo assessment of oxygenation by oximetry or, if results are abnormal or if there are respiratory symptoms or signs, ABG and chest x-ray. Because respiratory symptoms may be delayed, even asymptomatic patients are transported to the hospital and observed for several hours.

In patients with symptoms or a history of prolonged submersion, core body temperature is measured, ECG and serum electrolytes are obtained, and continuous oximetry and cardiac monitoring are done. Patients with possible cervical spine injury undergo cervical spine imaging (see p. 3229).

Patients with altered consciousness undergo head CT. Any other suspected predisposing or secondary conditions are evaluated with appropriate testing (eg, fingerstick glucose for hypoglycemia, ECG for MI). Patients who drown without apparent risk factors are evaluated for long QT syndrome. In patients with pulmonary infiltrates, bacterial pneumonia is differentiated from chemical pneumonitis using blood cultures and sputum Gram stain and culture.

Prognosis

Factors that increase the chance of surviving submersion without permanent injury include the following:

- Brief duration of submersion
- Cold water temperature
- Young age
- Absence of underlying medical conditions, secondary trauma, and aspiration of particulate matter or chemicals
- Rapid institution of resuscitation (most important)

Survival may be possible in cold water submersion that lasts > 1 h, especially among children; thus, even

patients with prolonged submersion are vigorously resuscitated.

Treatment

• Resuscitation

• Correction of physiologic abnormalities

• Intensive respiratory support

Treatment aims to correct cardiac arrest, hypoxia, hypoventilation, hypothermia, and other physiologic insults.

Resuscitation: In apneic patients, rescue breathing is started immediately—in the water, if necessary. If spinal immobilization is necessary, it is done in a neutral position, and rescue breathing is done using a jaw thrust without head tilt or chin lift. Emergency medical services are called. If necessary, cardiac compression is started, followed by advanced cardiac life support (see p. 2256). Oxygenation, endotracheal intubation, or both are done as soon as possible. Hypothermic patients are warmed as soon as possible (see p. 3274).

Hospital care: All hypoxic or moderately symptomatic patients are hospitalized. In the hospital, supportive treatment continues, aimed primarily at achieving acceptable arterial O_2 and CO_2 levels. Mechanical ventilation may be necessary. Patients are given 100% O_2; the concentration is titrated lower based on ABG results. Positive end-expiratory pressure (see p. 2283) or intermittent positive pressure ventilation may be necessary to help expand or maintain patency of alveoli to maintain adequate oxygenation; pulmonary support may be necessary for hours or days. Nebulized β_2-agonists may help reduce bronchospasm and wheezing. Patients with bacterial pneumonia are treated with antibiotics directed at organisms identified or suspected based on results of sputum analysis or blood cultures. Corticosteroids are not used.

Fluids or electrolytes are rarely required to correct significant electrolyte imbalances. Fluid restriction is rarely indicated, even if pulmonary or cerebral edema occurs. For prolonged brain hypoxia, treatment is similar to that for brain hypoxia after cardiac arrest (see p. 2266). Concomitant disorders (eg, head or cervical injury, carbon monoxide poisoning) require treatment.

Discharge: Patients with mild symptoms and normal oxygenation can be observed in the emergency department for several hours. If symptoms resolve and oxygenation remains normal, they can be discharged with instructions to return if symptoms recur.

Prevention

Use of alcohol or drugs, a major risk factor, should be avoided before and during swimming and boating and when supervising children around water.

Swimmers should be accompanied by an experienced swimmer or swim only in guarded areas. Swimming should stop if the swimmer looks or feels very cold, because hypothermia may impair judgment. Ocean swimmers should learn to escape rip currents by swimming parallel to the beach rather than toward the beach. Swimmers should avoid swimming near a boat exhaust port, which can cause carbon monoxide poisoning.

Children must wear flotation devices when in or near water. They must be supervised by an adult when around water, including beaches, pools, and ponds. Infants and toddlers should also be supervised, ideally within arm's length, when near toilets and bathtubs. Swimming lessons are not recommended for children < 4 yr. Young children who have taken swim lessons or infant water safety classes still require supervision because these classes have not been proved to reduce drowning. Adults should remove water from containers such as pails and buckets immediately after use. Swimming pools should be surrounded with a locked fence ≥ 1.5 m in height.

Boaters are encouraged to wear flotation devices. Nonswimmers and small children are required to wear these devices.

People who are debilitated or elderly or have seizure disorders or other medical conditions that can alter consciousness require particular care when they are boating or swimming.

People with a personal and family history of unexplained drowning not attributable to alcohol, drug use, or a seizure disorder merit evaluation for long QT syndrome.

Community swimming areas should be supervised by trained lifeguards. Comprehensive community prevention programs should target high-risk groups, teach children to swim as early as possible, and teach CPR to as many adolescents and adults as possible.

Chapter 337. Injury During Diving or Work in Compressed Air

Introduction

More than 1000 diving-related injuries occur annually in the US; > 10% are fatal. Similar injuries can befall workers in tunnels or caissons (watertight retaining structures used for construction), in which pressurized air is used to exclude water from work sites. Many injuries are related to high pressure, which, at depth or in a caisson, results from the water weight above plus the atmospheric pressure at the surface. At a depth of 10 m (33 ft), seawater exerts a pressure equivalent to standard sea level atmospheric pressure, which is 0.1 kg/cm^2 (14.7 lb/sq in), 760 mm Hg, or 1 atmosphere absolute (atm abs); thus, the total pressure at that depth is 2 atm abs. Every additional 10 m of descent adds 1 atm.

The volume of gases in body compartments is inversely related to external pressure; an increase or a decrease in gas volume due to pressure change exerts direct physical forces that can disrupt various body tissues (barotrauma). The amount of gas dissolved in the bloodstream increases as ambient pressure increases. Increased gas content can cause injury directly (eg, N_2 narcosis, O_2 toxicity) or indirectly during ascent when decompression of the super-saturated blood or tissues releases N_2 bubbles (decompression sickness). Arterial gas embolism can result from barotrauma or decompression. For other diving-related injuries (eg, drowning, hypothermia, trauma), see elsewhere in THE MANUAL.

Physicians caring for patients with diving or compressed air injuries may contact the Divers Alert Network (919-684-8111; www.diversalertnetwork.org).

Barotrauma

Barotrauma is tissue injury caused by a pressure-related change in body compartment gas volume; it affects air-containing areas, including lungs, ears, sinuses, GI tract, air spaces in tooth fillings, and space contained by the diving face mask. Symptoms may include ear pain, vertigo, hearing loss, sinus pain, epistaxis, and abdominal pain. Dyspnea and loss of consciousness are life threatening and may result from alveolar rupture and pneumothorax. Diagnosis is clinical but sometimes requires imaging tests. Treatment generally is supportive but may include decongestants and analgesics for ear and sinus barotrauma or O_2 and chest tube placement for pneumothorax. If arterial gas embolism accompanies lung barotrauma, recompression therapy (in a hyperbaric chamber) is needed. Proper diving safety techniques and prophylactic use of decongestants may reduce incidence of barotrauma.

Risk of barotrauma (often called squeeze by divers) is greatest from the surface to 10 m (33 ft); risk is increased by any condition that can interfere with equilibration of pressure (eg, sinus congestion, eustachian tube blockage, structural anomaly, infection) in the air-containing spaces of the body. Ear barotrauma constitutes about two thirds of all diving injuries. In divers who inspire even a single breath of air or other gas at depth and do not let it escape freely during ascent, or when ascent is rapid, the expanding gas may overinflate the lungs. Lung overinflation occurs mostly in divers breathing compressed air but can occur even in swimming pools when compressed air is inspired at the bottom of the pool (eg, when scuba gear is used there) and, rarely, from an inverted bucket.

Manifestations depend on the affected area; all occur almost immediately when pressure changes. Some nonfatal disorders, if they occur at depth, may be disabling or disorienting and thus lead to drowning. Secondary infection is sometimes a late complication.

Diagnosis is primarily clinical; imaging tests can sometimes confirm barotrauma. Sometimes patients are evaluated for other problems or organ dysfunction.

Treatment

• Symptomatic treatment

• Other treatment depending on specific injury

Most barotrauma injuries resolve spontaneously and require only symptomatic treatment and outpatient follow-up; however, some injuries are life-threatening. Potentially life-threatening barotrauma emergencies are those involving alveolar or GI rupture, particularly in patients who present with any of the following:

• Neurologic symptoms

• Pneumothorax

• Peritoneal signs

• Abnormal vital signs

Initial stabilizing treatment includes high-flow 100% O_2 and, if respiratory failure appears imminent, endotracheal intubation. Positive pressure ventilation may cause or exacerbate pneumothorax.

Patients with suspected pneumothorax who are hemodynamically unstable or have signs of tension pneumothorax require immediate chest decompression (see p. 2004) with a large-bore (eg, 14-gauge) needle placed into the 2nd inter-costal space in the midclavicular line, followed by tube thoracostomy. Patients with neurologic symptoms or other evidence of arterial gas embolism are transported to a recompression chamber (see p. 3289) for treatment as soon as transportation can be arranged.

When stable, patients are treated for the specific type of barotrauma sustained.

Patients treated for severe or recurrent diving-related injuries should not return to diving until they have consulted with a diving medicine specialist.

Prevention of other diving injuries is discussed elsewhere (see p. 3290).

Pulmonary Barotrauma

During very deep breath-hold diving, compression of the lungs during descent may rarely lead to a decrease in volume below residual volume, causing mucosal edema, vascular engorgement, and hemorrhage, which manifest clinically as dyspnea and hemoptysis on ascent.

Overexpansion and alveolar rupture can occur when breathing compressed air during ascent, particularly rapid ascent. The result can be pneumothorax (causing dyspnea, chest pain, and unilateral decrease in breath sounds) or pneumomediastinum (causing sensation of fullness in the chest, neck pain, pleuritic chest pain that may radiate to the shoulders, dyspnea, coughing, hoarseness, and dysphagia). Pneumomediastinum may cause crepitation in the neck, due to associated subcutaneous emphysema, and a crackling sound may rarely be heard over the heart during systole (Hamman's sign). Tension pneumothorax, although rare with barotrauma, can cause hypotension, distended neck veins, hyperresonance to percussion, and tracheal deviation. Alveolar rupture often allows air into the pulmonary venous circulation with subsequent arterial gas embolism (see p. 3285).

Diagnosis

• Chest x-ray

Patients require a neurologic examination for signs of brain dysfunction due to arterial gas embolism. Chest x-ray is done to look for signs of pneumothorax or pneumomediastinum (radiolucent band along the cardiac border). If chest x-ray is negative but there is strong clinical suspicion, then helical CT, which may be more sensitive than plain film x-rays, may be diagnostic.

Treatment

• 100% O_2

• Sometimes tube thoracostomy

Suspected tension pneumothorax is treated with needle decompression followed by tube thoracostomy (see p. 1866). If a smaller (eg, 10 to 20%) pneumothorax is present and there is no sign of hemodynamic or respiratory instability, the pneumothorax may resolve when high-flow 100% O_2 is given for 24 to 48 h. If this treatment is ineffective or if a larger pneumothorax is present, tube thoracostomy is done.

No specific treatment is required for pneumomediastinum; symptoms usually resolve spontaneously within hours to days. After a few hours of observation, most patients can be treated as outpatients; high-flow 100% O_2 is recommended to hasten resorption of extra-alveolar gas in these patients. Rarely, mediastinotomy is required to relieve tension pneumomediastinum.

Prevention

Prevention of pulmonary barotrauma is usually the top priority. Proper ascent timing and techniques are essential. Patients with pulmonary blebs, Marfan syndrome, or COPD are at very high risk of pneumothorax and should not dive or work in areas of compressed air. Patients with asthma may be at risk of pulmonary barotrauma, although many people with asthma can dive safely after they are evaluated and treated appropriately.

Gastrointestinal Barotrauma

Breathing improperly from a regulator or using ear and sinus pressure-equalization techniques may cause divers to swallow small amounts of air during a dive. This air expands during ascent, causing abdominal fullness, cramps, pain, belching, and flatulence; these symptoms are self-limited. GI rupture rarely occurs, manifesting with severe abdominal pain and tenderness with rebound and guarding.

If signs of GI rupture are present, immediate upright chest x-ray or CT is done to detect free air. Milder symptoms require no testing.

Patients with GI rupture require aggressive fluid resuscitation, broad-spectrum antibiotic therapy (eg, imipenem 500 mg IV q 6 h), and immediate surgical consultation for possible exploratory laparotomy.

Ear and Sinus Barotrauma

Diving can affect the external, middle, and inner ear. Typically, divers experience ear fullness and pain during descent; if pressure is not quickly equilibrated, middle ear hemorrhage or tympanic membrane rupture may occur. Inflow of cold water to the inner ear may result in vertigo, nausea, and disorientation while submerged. On examination of the ear canal, the tympanic membrane may show congestion, hemotympanum, and lack of mobility during air insufflation with a pneumatic otoscope; conduction hearing loss is usually present.

Inner ear barotrauma often involves rupture of the round or oval window, which causes tinnitus, sensorineural hearing loss, vertigo, nausea, and vomiting. The resulting labyrinthine fistula and perilymph leakage can permanently damage the inner ear.

Sinus barotrauma most often affects the frontal sinuses, followed by the ethmoid and maxillary sinuses. Divers experience mild pressure to severe pain, with a feeling of congestion in the involved sinus compartments during ascent or descent and sometimes epistaxis. Pain can be severe, sometimes accompanied by facial tenderness on palpation. Rarely, the sinus may rupture and cause pneumocephalus with facial or oral pain, nausea, vertigo, or headache. Physical examination may detect tenderness in the sinuses or nasal hemorrhage.

Diagnosis

• Audiometry and vestibular testing

Patients with symptoms of inner ear trauma should be examined for signs of vestibular dysfunction and

referred for formal audiometry and vestibular testing (see p. 428).

Imaging (eg, plain x-rays, CT) is not necessary for diagnosis of uncomplicated sinus barotrauma, but CT is useful if sinus rupture is suspected.

Treatment

• Decongestants and analgesics

• Sometimes oral corticosteroids, surgical repair, or both

Most ear and sinus barotrauma injuries resolve spontaneously and require only symptomatic treatment and outpatient follow-up.

Drug treatment for sinus and middle ear barotrauma is identical. Decongestants (oxymetazoline 0.05%, 2 sprays each nostril bid for 3 to 5 days; pseudoephedrine 60 to 120 mg po bid to qid up to a maximum of 240 mg/day for 3 to 5 days) can help open occluded chambers. Severe cases can be treated with nasal corticosteroids. Doing the Valsalva maneuver immediately after nasal spray therapy may help distribute the decongestant into the occluded chamber. Pain can be controlled with NSAIDs or opioids. If bleeding or evidence of effusion is present, antibiotics are given (eg, amoxicillin 500 mg po q 12 h for 10 days, trimethoprim/sulfamethoxazole 1 double-strength tablet po bid for 10 days). For middle ear barotrauma, some physicians also advocate a short course of oral corticosteroids (eg, prednisone 60 mg po once/day for 6 days, then tapered over 7 to 10 days).

Referral to an otorhinolaryngologist is indicated for severe or persistent symptoms. Surgery (eg, tympanotomy for direct repair of a ruptured round or oval window, myringotomy to drain fluid from the middle ear, sinus decompression) may be necessary for serious inner or middle ear or sinus injuries.

Prevention

Ear barotrauma may be avoided by frequently swallowing or exhaling against pinched nostrils to open the eustachian tubes and equalize pressure between the middle ear and the environment. Pressure behind ear plugs cannot be equalized, so they should not be used for diving. Prophylaxis with pseudoephedrine (60 to 120 mg po bid or qid up to a maximum of 240 mg/day), beginning 12 to 24 h before a dive, can reduce the incidence of ear and sinus barotrauma. Diving should not be done if congestion does not resolve or if a URI or uncontrolled allergic rhinitis is present.

Other Types of Barotrauma

Dental barotrauma can occur during descent or ascent, when pressure in the air spaces at the roots of infected teeth or adjacent to fillings changes rapidly and causes pain or tooth damage. The affected tooth may be tender when percussed with a tongue blade.

Mask barotrauma occurs when the pressure in the space behind the face mask is not equalized during descent. The resulting relative vacuum can lead to local pain, conjunctival hemorrhage, and ecchymosis of the skin enclosed by the mask. Retro-orbital hemorrhage is possible but rare. If retro-orbital hemorrhage is suspected, head CT is done. Mask barotrauma may be avoided when pressures are equalized within the face mask by exhaling from the nose into the mask.

Eye barotrauma occurs when small air bubbles are trapped behind hard contact lenses. The air bubbles can damage the eye and cause soreness, decreased visual acuity, and halos around lights. A screening ophthalmic examination should be done to rule out other causes. Pressure behind goggles cannot be equalized, so they should not be used for diving.

Arterial Gas Embolism

(Air Embolism)

Arterial gas embolism is a potentially catastrophic event that occurs when gas bubbles enter or form in the arterial vasculature and occlude blood flow, causing organ ischemia. Arterial gas embolism can cause CNS ischemia with rapid loss of consciousness, other CNS manifestations, or both; it also may affect other organs. Diagnosis is clinical and may be corroborated by imaging tests. Treatment is immediate recompression.

Gas emboli may enter the arterial circulation in any of the following ways:

• From ruptured alveoli after lung barotrauma

• From within the arterial circulation itself in severe decompression sickness

• Via migration from the venous circulation (venous gas embolism) either via a right-to-left shunt (patent foramen ovale, atrial septal defect) or by overwhelming the filtering capacity of the lungs

Even asymptomatic venous gas embolism can cause serious manifestations (eg, stroke) in the presence of a right-to-left shunt. Venous gas embolism that does not enter the arterial circulation is less serious.

Although cerebral embolism is considered the most serious manifestation, arterial gas embolism can cause significant ischemia in other organs (eg, spinal cord, heart, skin, kidneys, spleen, GI tract).

Symptoms and Signs

Symptoms occur within a few minutes of surfacing and may include altered mental status, hemiparesis, focal motor or sensory deficits, seizures, loss of consciousness, apnea, and shock; death may follow. Signs of pulmonary barotrauma (see p. 3283) or type II decompression sickness (see p. 3287) may also be present.

Other symptoms may result from arterial gas embolism in any of the following:

• Coronary arteries (eg, arrhythmias, MI, cardiac arrest)

• Skin (eg, cyanotic marbling of the skin, focal pallor of the tongue)

• Kidneys (eg, hematuria, proteinuria, renal failure)

Diagnosis

• Clinical evaluation

• Sometimes confirmation by imaging

Diagnosis is primarily clinical. A high level of suspicion is necessary when divers lose consciousness during or immediately after ascent. Confirming the diagnosis is difficult because air may be reabsorbed from the affected artery before testing. However, imaging techniques that may support the diagnosis (each with limited sensitivity) include the following:

• Echocardiography (showing air in the cardiac chambers)

• Ventilation-perfusion scan (showing results consistent with pulmonary emboli)

• Chest CT (showing local lung injury or hemorrhage)

• Head CT (showing intravascular gas and diffuse edema)

Sometimes decompression sickness can cause similar symptoms and signs (for a comparison of features, see Table 337-1).

Treatment

• Immediate 100% O_2

• Recompression therapy

[**Table 337-1.** Comparison of Gas Embolism and Decompression Sickness]

Divers thought to have gas embolism should be recompressed promptly (see p. 3289). Transport to a recompression chamber takes precedence over nonessential procedures. Transport by air may be justified if it saves significant time, but exposure to reduced pressure at altitude must be minimized (see p. 3288).

Before transport, high-flow 100% O_2 enhances N_2 washout by widening the N_2 pressure gradient between the lungs and the circulation, thus accelerating reabsorption of embolic bubbles. Patients should remain in a supine position to decrease the risk of brain embolism. Mechanical ventilation, vasopressors, and volume resuscitation are used as needed. Placing patients in the left lateral decubitus position (Durant's maneuver) or Trendelenburg position is no longer recommended.

Immersion Pulmonary Edema

Immersion pulmonary edema is sudden-onset noncardiogenic pulmonary edema that typically occurs early during a dive while at depth.

Immersion pulmonary edema has become more common over the past 2 decades. This disorder is similar to negative pressure pulmonary edema encountered during induction of anesthesia when a patient with laryngo-spasm attempts to take deep breaths against a closed larynx, thereby causing negative intra-alveolar pressure. Immersion pulmonary edema is not related to lung barotrauma or decompression sickness. Cold water and a history of hypertension are risk factors.

Severe dyspnea develops. Divers usually ascend rapidly and have cough, frothy sputum, scattered crackles throughout both lung fields, and sometimes cyanosis. Hypoxia is present. Chest x-ray shows typical pulmonary edema. Cardiac evaluation usually shows normal right and left ventricular function and normal coronary arteries. Diuretic therapy and O_2 by positive pressure mask are usually sufficient therapy. Mechanical ventilation may be necessary. Recompression therapy is not indicated.

Decompression Sickness

(Caisson Disease; The Bends)

Decompression sickness occurs when rapid pressure reduction (eg, during ascent from a dive, exit from a caisson or hyperbaric chamber, or ascent to altitude) causes gas previously dissolved in blood or tissues to form bubbles in blood vessels. Symptoms typically include pain, neurologic symptoms, or both. Severe cases can be fatal. Diagnosis is clinical. Definitive treatment is recompression therapy. Proper diving techniques are essential for prevention.

Henry's law states that the solubility of a gas in a liquid is directly proportional to the pressure exerted on the gas and liquid. Thus, the amount of inert gases (eg, N_2, helium) dissolved in the blood and tissues increases at higher pressure. During ascent, when the surrounding pressure decreases, bubbles may form. The liberated gas bubbles can arise in any tissue and cause local symptoms, or they can travel via the blood to distant organs. Bubbles cause symptoms by blocking vessels, rupturing or compressing tissue, or activating clotting and inflammatory cascades. Because N_2 dissolves readily in fat, tissues with a high lipid content (eg, in the CNS) are particularly susceptible.

Risk factors: Decompression sickness occurs in about 2 to 4/10,000 dives. Risk factors include all of the following:

- Cold-temperature dives
- Dehydration
- Exercise after diving
- Fatigue
- Flying after diving
- Obesity
- Older age
- Prolonged or deep dives
- Rapid ascents
- Right-to-left cardiac shunts

Because excess N_2 remains dissolved in body tissues for at least 12 h after each dive, repeated dives within 1 day are most likely to cause decompression sickness. Decompression sickness can also develop if pressure suddenly decreases after recompression therapy (eg, after exposure to altitude).

Classification: Generally, there are 2 types of decompression sickness. Type I, which involves muscles, skin, and lymphatics, is milder and not typically life threatening. Type II is serious, is sometimes life threatening, and affects various organ systems. The spinal cord is especially vulnerable; other vulnerable areas include the brain, respiratory system (eg, pulmonary emboli), and circulatory system (eg, heart failure, cardiogenic shock). "The bends" refers to local joint or muscle pain due to decompression sickness but is often used as a synonym for any component of the disorder.

Symptoms and Signs

Severe symptoms may manifest within minutes of surfacing, but in most patients, symptoms begin gradually, sometimes with a prodrome of malaise, fatigue, anorexia, and headache. Symptoms occur within 1 h of surfacing in about 50% of patients and by 6 h in 90%. Rarely, symptoms can manifest 24 to 48 h after surfacing, particularly after exposure to altitude after diving.

Type I decompression sickness typically causes progressively worsening pain in the joints (typically elbows and shoulders), back, and muscles; the pain intensifies during movement and is described as "deep" and "boring." Other manifestations include lymphadenopathy, skin mottling, itching, and rash.

Type II decompression sickness tends to cause neurologic and sometimes respiratory symptoms. It typically manifests with paresis, numbness and tingling, difficulty urinating, and loss of bowel or bladder control. Headache and fatigue may be present but are non-specific. Dizziness, tinnitus, and hearing loss may result if the inner ear is affected. Severe symptoms include seizures, slurred speech, vision loss, confusion, and coma. Death can occur. The chokes (respiratory decompression sickness) is a rare but grave manifestation; symptoms include shortness of breath, chest pain, and cough. Massive bubble embolization of the pulmonary vascular tree can result in rapid circulatory collapse and death.

Dysbaric osteonecrosis is a late manifestation of decompression sickness. It is an insidious form of aseptic bone necrosis caused by prolonged or closely repeated exposures to pressurized areas (typically in people working in compressed air and in deep commercial rather than recreational divers). Deterioration of shoulder and hip articular surfaces can cause chronic pain and severe disability.

Diagnosis

• Clinical evaluation

Diagnosis is clinical. CT and MRI may be helpful to rule out other disorders that cause similar symptoms (eg, herniated intervertebral disk, ischemic stroke, CNS hemorrhage). Although these studies may show brain or spinal cord abnormalities, they are not sensitive for decompression sickness, and treatment should usually begin based on clinical suspicion. Sometimes arterial gas embolism is similar (for a comparison of features, see Table 337-1).

For dysbaric osteonecrosis, plain x-rays may show joint degeneration, which cannot be distinguished from that caused by other joint disorders; MRI is usually diagnostic.

Treatment

• 100% O_2

• Recompression therapy

About 80% of patients recover completely.

Initially, high-flow 100% O_2 enhances N_2 washout by widening the N_2 pressure gradient between the lungs and the circulation, thus accelerating reabsorption of embolic bubbles.

Recompression therapy (see p. 3289) is indicated for all patients except perhaps those whose symptoms are limited to itching, skin mottling, and fatigue; they should be observed for deterioration. Other patients are transported to a suitable recompression facility. Because time to treatment is a main determinant of outcome, transport should not be delayed even in cases that appear mild or for performance of nonessential procedures. If air evacuation is required, an aircraft capable of 1 atmosphere internal pressure is preferred. In unpressurized aircraft, low altitude (< 609 m [< 2000 ft]) must be maintained. Commercial aircraft, although pressurized, typically have a cabin pressure equivalent to 2438 m (8000 ft) at normal cruise altitude, which may exacerbate symptoms. Flying in commercial aircraft shortly after a dive can precipitate symptoms.

Prevention

Significant bubble formation can usually be avoided by limiting the depth and duration of dives to a range that does not need decompression stops during ascent (called no-stop limits) or by ascending with decompression stops as specified in published guidelines (eg, the decompression table in the US Navy Diving Manual). Many divers wear a portable dive computer that continually tracks depth and time at depth and calculates a decompression schedule. In addition to following published and computer-generated guidelines, many divers make a safety stop for a few minutes at about 4.6 m (15 ft) below the surface. However, a few cases develop after appropriately identified no-stop dives, and the incidence of decompression sickness has not decreased despite widespread use of dive computers. The reason may be that published tables and computer programs do not completely account for the variation in risk factors among divers or that people do not obey the recommendations precisely.

Dives < 24 h apart (repetitive dives) require special techniques to determine proper decompression procedures.

Gas Toxicity

Various physiologic (eg, O_2, N_2, CO_2) and nonphysiologic (eg, carbon monoxide) gases can cause symptoms during diving.

Oxygen toxicity: O_2 toxicity typically occurs when the partial pressure of O_2 exceeds 1.4 atmospheres (atm), equivalent to about 57 m (187 ft) depth when air is breathed. Symptoms include paresthesias, focal seizures, vertigo, nausea, vomiting, and constricted vision. About 10% of patients have generalized seizures or syncope, which typically results in drowning. Risk is increased when divers breathe mixtures

of O_2 and N_2 (nitrox) that have an increased percentage of O_2.

Nitrogen narcosis: When compressed air is breathed at depths of > 30 m (> 100 ft), the elevated partial pressure of N_2 can exert an anesthetic-like effect similar to that of nitrous oxide. N_2 narcosis (rapture of the deep) causes symptoms and signs similar to those of alcohol intoxication (eg, impaired intellectual and neuromuscular performance, changes in behavior and personality). Impairment of judgment can lead to drowning. Hallucinations and loss of consciousness can occur at depths of > 91 m (> 300 ft).

Because divers recover rapidly during ascent, diagnosis is clinical. Treatment entails immediate but controlled ascent. N_2 narcosis can be prevented by using helium to dilute O_2 for deep diving because helium lacks the anesthetic properties of N_2. However, using pure helium/O_2 mixtures in very deep dives (> 180 m [> 600 ft]) increases the risk of developing high-pressure neurologic syndrome (see below).

Carbon dioxide poisoning: CO_2 poisoning may be caused by any of the following:

- Inadequate respiratory effort (hypoventilation)
- A tight wetsuit
- Overexertion
- Regulator malfunction
- Deep diving
- Air supply contamination by exhaled gases

Hypoventilation can increase blood CO_2 levels and cause shortness of breath and sedation. Severe CO_2 poisoning can cause nausea, vomiting, dizziness, headache, rapid breathing, flushing, confusion, seizures, and loss of consciousness. Mild CO_2 poisoning is suspected if divers frequently have dive-related headaches or low air-use rates.

CO_2 intoxication usually resolves during ascent; thus, ABG testing after a dive typically does not detect any increase in CO_2 levels.

Treatment is gradual ascent and termination of the diving exercise or correction of the precipitating cause.

Carbon monoxide poisoning: Carbon monoxide can enter a diver's air if the air compressor intake valve is placed too close to engine exhaust or if the lubricating oil in a malfunctioning compressor becomes hot enough to partially combust (flashing), producing carbon monoxide.

Symptoms include nausea, headache, weakness, clumsiness, and mental changes. Severe carbon monoxide poisoning can cause seizures, syncope, or coma. Diagnosis is by detecting an elevated carboxyhemoglobin (COHb) level in blood; pulse oximetry readings are nondiagnostic and usually normal because pulse oximeters cannot distinguish between oxyhemoglobin and COHb. The diver's air supply can be tested for carbon monoxide.

Treatment is with high-flow 100% O_2, best given via a nonrebreather mask, which decreases the half-life of COHb from 4 to 8 h in room air to 40 to 80 min. For severe cases, hyperbaric O_2 therapy may be considered if readily available. COHb levels will drop quickly in the hyperbaric chamber (half-life 15 to 30 min); however, the benefit of hyperbaric O_2 therapy is controversial. Some studies indicate that hyperbaric O_2 therapy lessens neurologic sequelae, but others do not support this finding.

High-pressure neurologic syndrome: A poorly understood syndrome of neuromuscular and cerebral abnormalities can develop at ≥ 180 m (≥ 600 ft), particularly when divers are compressed rapidly while

breathing helium/O_2 mixtures. Symptoms include nausea, vomiting, fine tremors, incoordination, dizziness, fatigue, somnolence, myoclonic jerking, stomach cramps, and decrements in intellectual and psychomotor performance. Diagnosis is clinical. Prevention is usually accomplished by slowing the rate of compression.

Recompression Therapy

(Hyperbaric O_2 Therapy)

Recompression therapy is administration of 100% O_2 for several hours in a sealed chamber pressurized to > 1 atmosphere, gradually lowered to atmospheric pressure. In divers, this therapy is used primarily for decompression sickness and arterial gas embolism. A shorter time to start of therapy is associated with a better patient outcome. Untreated pneumothorax requires chest tube placement before or during recompression therapy.

The goals of recompression therapy in diving injuries include all of the following:

• Increasing O_2 solubility and delivery

• Increasing N_2 washout

• Decreasing gas bubble size

For carbon monoxide poisoning, mechanisms include decreasing the half-life of carboxyhemoglobin, reducing ischemia, and possibly improving mitochondrial function.

Hyperbaric O_2 therapy is also used for several disorders unrelated to diving (see Table 337-2).

Because recompression is relatively well tolerated, it should be started if there is any likelihood that it would promote recovery; recompression may help even if started up to 48 h after surfacing.

Recompression chambers are either multi-place, with space for one or more patients on a gurney and for a medical attendant, or mono-place, with space for only one patient. Although monoplace chambers are less expensive, because patients cannot be accessed during recompression, their use for critically ill patients, who may require intervention, can be risky.

[**Table 337-2.** Hyperbaric O_2 Therapy*]

Information regarding the location of the nearest recompression chamber, the most rapid means of reaching it, and the most appropriate source to consult by telephone should be known by most divers, medical staff members, and rescue and police personnel in popular diving areas. Such information is also available from the Divers Alert Network (919-684-8111; www.diversalertnetwork.org) 24 h/day; the Undersea and Hyperbaric Medical Society (www.uhms.org) is another invaluable source of general information about recompression.

Recompression protocols: Pressure and duration of treatment are usually decided by a hyperbaric medicine specialist at the recompression facility. Treatments are given once or twice/day for 45 to 300 min until symptoms abate; 5- to 10-min air breaks are added to reduce risk of O_2 toxicity. Chamber pressure is usually maintained between 2.5 and 3.0 atmospheres (atm), but patients with life-threatening neurologic symptoms due to gas embolism may begin with an excursion to 6 atm to rapidly compress cerebral gas bubbles.

Although recompression therapy is usually done with 100% O_2 or compressed air, special gas mixtures (eg, helium/O_2 or N_2/O_2 in nonatmospheric proportions) may be indicated if the diver used an unusual gas mixture or if depth or duration of the dive was extraordinary. Specific protocol tables for treatment are

included in the US Navy Diving Manual.

Patients with residual neurologic deficits should be given repetitive, intermittent hyperbaric treatments and may require several days to reach maximum improvement.

Complications and contraindications: Recompression therapy can cause problems similar to those that occur with barotrauma (see p. 3282), including ear and sinus barotrauma. O_2 toxicity can cause reversible myopia. Rarely, pulmonary barotrauma, pulmonary O_2 toxicity, hypoglycemia, or seizures result. Sedatives and opioids may obscure symptoms and cause respiratory insufficiency; they should be avoided or used only in the lowest effective doses.

Relative contraindications include

• Obstructive lung disorders

• Upper respiratory or sinus infections

• Severe heart failure

• Recent ear surgery or injury

• Fever

• Claustrophobia

• Seizure disorder

• Chest surgery

• Pneumothorax

Patients with pneumothorax require tube thoracostomy before recompression therapy.

Diving Precautions and Prevention of Diving Injuries

Diving is a relatively safe recreational activity for healthy people who have been appropriately trained and educated. Diving safety courses offered by national diving organizations are widely available.

Diving safety: Incidence of barotrauma can be decreased through active equalization of various air spaces, including the face mask (by blowing out air from the nose into the mask)

[
Table 337-3. Specific Medical Contraindications to Diving]

and the middle ear (by yawning, swallowing, or performing a Valsalva maneuver). Divers should avoid holding their breath and breathe normally during ascent, which should be no faster than 0.15 to 0.3 m/sec (0.5 to 1 ft/sec), a rate that allows for gradual offloading of N_2 and emptying of air-filled spaces (eg, lungs, sinuses). Divers should ascend with decompression stops as specified in published guidelines (eg, the decompression table in the US Navy Diving Manual). Current recommendations also include a 3- to 5-min safety stop at 4.6 m (15 ft) for further equilibration. Also, divers should not fly for 15 to 18 h after diving.

Divers should be aware of and avoid certain diving conditions (eg, poor visibility, currents requiring excessive effort). Cold temperatures are a particular hazard because hypothermia can develop rapidly and affect judgment and dexterity or induce fatal cardiac arrhythmias in susceptible people. Diving alone is not recommended.

Recreational or sedative drugs and alcohol in any amount may have unpredictable or unanticipated effects at depth and should be strictly avoided. Otherwise, prescription drugs rarely interfere with recreational diving, but if the disorder being treated is a contraindication to diving, the dive should not be pursued.

Contraindications to diving: Because diving can involve heavy exertion, divers should not have a functionally significant cardiovascular or pulmonary disorder and should have above-average aerobic capacity. Disorders that can impair consciousness, alertness, or judgment generally prohibit diving. If there is any doubt as to whether diving is contraindicated by a specific disorder, a recognized expert should be consulted. For specific diving contraindications, see Table 337-3.

Chapter 338. Exercise and Sports Injury

Introduction

Regular exercise enhances health and a sense of well-being. However, injury, particularly over-use injury, is a risk for people who exercise regularly. (For musculoskeletal injuries not particularly associated with sports, see p. 3201.)

Exercise

Exercise stimulates tissue change and adaptation, whereas rest and recovery allow such change and adaptation to occur. Recovery from exercise is as important as the exercise stimulus. Regular physical activity decreases the incidence of the major causes of death, improves functional status for sports and activities of daily living, and protects against injury. Specific exercise programs are also commonly prescribed to rehabilitate patients after MI, major surgery, and musculoskeletal injury. Regardless of indication, recommendations for exercise should be based on 2 principles:

- Goals for activity should be specific to the patient, accounting for motivation, needs, physical ability, and psychology, to maximize the likelihood of patient participation and desired outcome.

- Activity should be prescribed in a proper dose to achieve a desired effect. An exercise stimulus should be sufficient for the body to adapt to a higher state of function but not so great that it causes injury. More activity is not always better; too little or too much activity may prevent achievement of desired outcomes.

A prescription for exercise should specify intensity (level of exertion), volume (amount of activity in a session), frequency (number of exercise sessions), and progressive overload (either the amount of increase in one or more of these elements over time, or the actual load). The balance of these elements depends on individual tolerance and physiologic principles (ie, as intensity increases, volume and frequency may need to decrease, whereas as volume increases, intensity may need to decrease). Intensity, volume, and frequency can be increased concurrently but only to a point because human tolerance to strain is finite. The objective is to discover the appropriate amount of exercise for optimal benefit in the context of the patient's goals. Fixed and traditional recommendations (eg, 3 sets of 10 to 12 repetitions, running 30 min 3 times/wk) may be suboptimal because they do not address a person's specific requirements.

Exercise programs should encompass multiple dimensions of fitness:

- Stretching and flexibility

- Aerobic capacity

- Strength

- Balance

Stretching and flexibility: Flexibility is important for safe, comfortable performance of physical activities. Stretching may be beneficial in strength training to improve range of motion and help relax muscles. Specific flexibility exercises involve slowly and steadily stretching muscle groups without jerking or bouncing. These exercises can be done before or after other forms of training or as a regimen itself, as occurs in yoga and Pilates sessions. Although stretching before exercise enhances mental preparedness, there is no evidence that stretching decreases risk of injury. However, there is no need to discourage preactivity stretching if patients enjoy it. General warming-up (eg, with low-intensity simulation of the exercise to be done, jogging on the spot, calisthenics, or other light activities that increase core temperature) seems to be more effective than stretching for facilitating safe exercise. Stretching after exercise may be preferred because tissues stretch more effectively when warmed.

Aerobic exercise: Aerobic exercise is continuous, rhythmic physical activity. Exertion occurs at a level

that can be supported by aerobic metabolism (although brief periods of more intense exertion triggering anaerobic metabolism may be interspersed) continuously for at least 5 min as a starting point and increased slowly over time. Aerobic conditioning increases maximal O_2 uptake and cardiac output (mainly an increase in stroke volume), decreases resting heart rate, and reduces cardiac and all-cause mortality; however, too much activity causes excessive wear on the body and increases cellular oxidation. Examples of aerobic exercise include running, jogging, fast walking, swimming, bicycling, rowing, kayaking, skating, cross-country skiing, and using aerobic exercise machines (eg, tread-mill, stair-climbing, or elliptical machines).

Aerobic metabolism starts within 2 min of beginning activity, but more sustained effort is needed to achieve health benefits. The usual recommendation is to exercise ≥ 30 min/day at least 3 times/wk with a 5-min warm-up and a 5-min cool-down period, but this recommendation is based on convenience as much as evidence. Optimal aerobic conditioning can occur with as little as 10 to 15 min of activity per session 2 to 3 times/wk if interval cycling is used. In interval cycling, short periods of moderate activity are alternated with intense exertion. In one regimen, about 90 sec of moderate activity (60 to 80% maximum heart rate [HR_{max}]) is alternated with about 20 to 30 sec of all-out sprint-type work (85 to 95% HR_{max} or as hard as the person can exert for that time). This regimen is more stressful on joints and tissues and so should be done infrequently or alternated with more conventional low- to moderate-intensity training.

Resistance training machines or free weights can be used for aerobic exercise as long as a sufficient number of repetitions are done per set, rest between sets is minimal (20 to 60 sec), and intensity of effort is relatively high. In circuit training, the large muscles (of the legs, hips, back, and chest) are exercised followed by the smaller muscles (of the shoulders, arms, abdomen, and neck). Circuit training for only 15 to 20 min can benefit the cardiovascular system more than jogging or using aerobic exercise machines for the same amount of time because the workout is often more intense and heart rate increases more as a result.

Volume of aerobic exercise is graded simply by duration. Intensity is guided by heart rate. Target heart rate for appropriate intensity is 60 to 85% of a person's HR_{max} (the heart rate at peak O_2 consumption [VO_{2peak}], or the rate beyond which aerobic metabolism can no longer be sustained because O_2 is lacking and anaerobic metabolism begins). HR_{max} can be directly measured, or calculated as

$$HR_{max} = 220 - age$$

Alternatively, the Karvonen formula can be used to calculate target heart rate:

$$\text{Target heart rate} = [(0.50 \text{ to } 0.85) \times (HR_{max} - HR_{resting})] + HR_{resting}$$

However, the more athletic or deconditioned the person is compared to average, the less accurate these formulas are, thus making metabolic or VO_2 testing more valuable.

Chronologic age should be distinguished from biologic age. People of any age who are less accustomed to aerobic exercise (less conditioned) reach the target heart rate much sooner and with less effort, necessitating briefer exercise periods, at least initially. Obese people may be deconditioned and must move a larger body weight, causing heart rate to increase much faster and to a greater extent with less vigorous activity than in thinner people. Disorders and some drugs (eg, β-blockers) also modify the relationship between age and heart rate. For people who have a disorder or take certain drugs, a target of 50 to 60% of HR_{max} is probably sufficient, at least initially.

Strength training: Strength (resistance) training involves forceful muscular contraction against a load —typically provided by free or machine weights or sometimes body weight (eg, push-ups, abdominal crunches, chin-ups). Such training increases muscle strength, endurance, and size and improves functional ability and aerobic performance. Cardiovascular endurance and flexibility increase concurrently.

Volume typically is categorized in terms of amount of weight lifted and number of sets and number of repetitions per set. However, an equally important parameter is tension time, which is the total duration of

lifting and lowering the weight in one set. Appropriate tension time may be about 60 sec for moderate conditioning (a good balance in developing muscle mass and strength) and 90 to 120 sec for injury rehabilitation and muscular endurance. For increasing strength, tension time is more important than number of repetitions, which can vary within tension time by technique and set duration. When a person can achieve at least a 60-sec tension time with good technique, resistance (weight) can be increased so that a tension time of at least 60 sec is tolerable at the next weight level. Number of sets is determined by intensity of the training.

Intensity is generally a subjective measure of perceived effort and how close a person comes to muscular fatigue in a given set (or exhaustion in a workout). Intensity may be characterized objectively by the amount of weight lifted expressed as a percentage of the person's maximum for one repetition (1 RM) of a given exercise; ie, for a person who can deadlift at most 100 kg one time, 75 kg is 75% RM. A general guideline is to exercise with a load at 70 to 85% RM. Heavier loads increase risk of injury and are appropriate mainly for competitive strength athletes. Lifting < 30 to 40% RM provides minimal strength gain, although aerobic conditioning and muscular endurance may occur with sufficient tension time and effort.

Intensity is limited by motivation and tolerance; for many people undergoing rehabilitation, discomfort, pain, and exercise inexperience result in less effort than may be possible or tolerated, so that more sets are required to derive equal benefit. Intensity should vary on a regular basis to provide both a mental and physical hiatus. If exercise is done at the highest intensity level, it should occur no more often than in about half of the sets and workouts should be avoided for 1 to 2 wk every 3 mo. Continual high-intensity training is counterproductive, even for trained athletes. Symptoms such as fatigue or muscle heaviness when not exercising, lack of motivation to exercise, reduced exercise performance, joint and tendon pains, and increased resting heart rate suggest that exercise has been too intense; exercise should be avoided for 1.5 to 2 wk.

Good technique is important for safety and involves avoidance of jerking or dropping weights, which can cause minor tissue injury due to sudden force, and controlled breathing, which prevents dizziness (and in extreme cases, fainting) that can occur with the Valsalva maneuver. People should exhale while lifting a weight and inhale while lowering a weight. If a movement is slow, such as lowering a weight for ≥ 5 sec, people may need to breathe in and out more than once, but breathing should still be coordinated so that a final breath is taken in just before the lifting phase and released during lifting. BP increases during resistance training and tends to be highest when gripping excessively (common with the leg press exercise when working the large lower body muscles and clenching the machines hand grips very tightly). However, BP returns to normal quickly after exercise; the increase is minimal when breathing technique is correct, no matter how hard a person exerts.

Balance training: Balance training involves challenging the center of gravity by undertaking exercises in unstable environments, such as standing on one leg or using balance or wobble boards. Balance training can help some people with impaired proprioception and is often used in an attempt to prevent falls in the elderly (see p. 3295).

Hydration: Proper hydration is important, particularly when exertion is prolonged or occurs in a hot environment. People should be well-hydrated before activity, drink fluids regularly during extended exertion, and replace any deficit remaining after activity. During exertion, about 120 to 240 mL (1/2 to 1 cup) every 15 to 20 min is reasonable depending on heat and exertion level; however, *overhydration, which can cause hyponatremia and consequent seizures, is to be avoided*.

Fluid deficit after exertion is calculated by comparing preexercise and postexercise body weight and is replaced on a one-for-one basis (ie, 1 L for each kg lost, or 2 cups/lb). In most cases, plain water is acceptable. Electrolyte-containing sports drinks may be preferred. However, fluids with a carbohydrate content of > 8% (8 g/100 mL, or 20 g in a typical 250-mL serving) decrease gastric emptying and slow fluid absorption. Often it is best to mix plain water with sports drinks at a 50:50 ratio to allow faster absorption of the glucose and electrolytes. People with findings suggesting heat illness (see p. 3262) or dehydration may require oral or IV electrolyte replacement immediately.

Exercise in the Elderly

At least 75% of people age > 65 yr do not exercise at recommended levels, despite the known health benefits:

• Longer survival

• Improved quality of life (eg, endurance, strength, mood, flexibility, possibly cognitive function)

Exercise has proven benefits when begun as old as 75 yr. Because of the effects of aging and age-related disorders, the relative benefits of exercise may even be greater in the elderly (see also Sidebar 307-1 on p. 3073). In addition, exercise is one of the safest ways to improve health.

The largest health benefits occur, particularly with aerobic exercise, when changing from being sedentary to exercising. Progressively less benefit occurs as the intensity of exercise increases.

Strength decreases with age and can compromise function. For example, almost half of women > 65 and more than half of women > 75 cannot lift 4.5 kg. Strength training can increase muscle mass by 25 to 100% or more, meaningfully improving function. The same degree of muscle work demands less cardiovascular exertion; increasing leg muscle strength improves walking speed and stair climbing. Also, institutionalized elderly with more muscle mass have better nitrogen balance and a better prognosis and less deconditioning during critical illness.

Contraindications: Absolute contraindications to exercise include

• Suspected acute coronary syndrome

• 3rd-degree heart block

• Uncontrolled hypertension

• Acute heart failure

• Uncontrolled diabetes mellitus

Relative contraindications include

• Cardiomyopathy

• Valvular heart disease

• Complex ventricular ectopy

Most patients with relative contraindications can exercise, although at lower levels of intensity than other patients (see p.
3461). Other factors mandate modification of the exercise program (eg, arthritic disorders, particularly those involving major weight-bearing joints, such as the knees, ankles, and hips).

Patients should be told to stop exercising and seek medical attention if they develop chest pain, light-headedness, or palpitations.

Screening: Before beginning an exercise program, elderly patients should undergo clinical evaluation aimed at detecting cardiac disorders and physical limitations to exercise. Routine ECG is unnecessary. Exercise stress testing is usually unnecessary for elderly patients who plan to begin exercising slowly and increase intensity only gradually. For sedentary patients who plan to begin intense exercise, stress testing is indicated if they have any of the following:

• Known coronary artery disease

- Symptoms of coronary artery disease
- ≥ 2 cardiac risk factors (eg, hypercholesterolemia, hypertension, obesity, sedentary lifestyle, smoking, family history of early coronary artery disease)
- Suspected lung disease
- Suspected diabetes

Exercise program: Exercise should ideally include

- Aerobic activity
- Strength training
- Flexibility and balance

Time spent doing **aerobic activity** is similar to that for younger adults, but exercise should be less intense. Usually during exercise, the person should be able to comfortably converse, and intensity should be ≤ 6/10 on a perceived scale of exertion. Elderly people who have no contraindications can gradually increase their target heart rate to the one calculated by use of age-based formulas. Some deconditioned elderly people need to improve their functional abilities (eg, by strength training) before they are capable of aerobic exercise.

Strength training is done according to the same principles and techniques as in younger adults. When beginning, forces may need to be small (eg, using bands or weights as light as 1 kg or arising from a chair).

To help increase **flexibility**, major muscle groups should be stretched once daily, ideally after exercise when muscles are most compliant.

Balance training involves challenging the center of gravity by undertaking exercises in unstable environments, such as standing on one leg or using balance or wobble boards. Balance training can help some people with impaired proprioception and is often used in an attempt to prevent falls in the elderly. However, it is often ineffective because any balance activity is skill specific (eg, good balance while standing on a balance board does not improve balance in dissimilar activities). For most elderly people, flexibility and strengthening exercises prevent falls more effectively. Such a program develops strength around the joints and helps people hold body positions more effectively while standing and walking. In people who have difficulty standing and walking because of poor balance, more challenging balance tasks (eg, standing on a wobble board) are simply likely to facilitate injury and are contraindicated.

Screening for Sports Participation

(See also Ch. 220.)

Cardiovascular screening: Screening for all children and adults should include a thorough cardiovascular history, with questions about

- Known hypertension or heart murmur
- Chest pain
- Exercise-induced syncope, near-syncope, chest pain, or palpitations
- Family history of sudden cardiac death at age < 50 yr, arrhythmias, dilated or hypertrophic cardiomyopathy, long QT syndrome, or Marfan syndrome
- Risk factors for coronary artery disease in adults

Physical examination should routinely include BP, supine and standing cardiac auscultation, and inspection for features of Marfan syndrome. These measures aim to identify adults as well as rare, apparently healthy young people at high risk of life-threatening cardiac events (eg, people with arrhythmias, hypertrophic cardiomyopathy, or other structural heart disorders). Testing is directed at clinically suspected disorders (eg, exercise stress testing for coronary artery disease, echocardiography for structural heart disease, ECG for long QT syndrome).

Other screening measures: Noncardiovascular risk factors are more common than cardiovascular risk factors. Adults are asked about arthritic disorders, particularly those involving major weight-bearing joints (eg, knees, ankles, hips).

Two at-risk populations are commonly overlooked:

- Boys who physically mature late or are short are at greater risk of injury in contact sports with larger and stronger children.

- Overweight or obese people who participate in activities that require high agility are at greater risk of injury due to sudden stops and starts because of excess body weight and associated forces on the joints and tissues.

Adolescents and young adults should be asked about use of illicit and performance-enhancing drugs. In girls and young women, screening should detect delayed onset of menarche. Girls and young women should be screened for the presence of the female athlete triad (eating disorders, amenorrhea or other menstrual dysfunction, and diminished bone mineral density), which is becoming more common as more adolescent and young women engage in overly intensive physical activity and overly zealous loss of body fat.

Contraindications: There are almost no absolute contraindications to sports participation. Exceptions in children include

- Myocarditis, which increases the risk of sudden cardiac death

- Acute splenic enlargement because splenic rupture is a risk

- Fever, which decreases exercise tolerance, increases risk of heat-related disorders, and may be a sign of serious illness

- Possibly diarrhea and recent vomiting because dehydration is a risk

Exceptions in adults include angina pectoris and recent (within 6 wk) MI. Contraindications are more commonly relative and lead to recommendations for precautions or for participation in some sports rather than others. For example, people with a history of multiple concussions should participate in noncollision sports; males with a single testis should wear a protective cup for most contact sports; people at risk of heat intolerance and dehydration (eg, those with diabetes or cystic fibrosis) should hydrate frequently during activity; and people with suboptimal seizure control should avoid swimming, weight lifting, and sports such as archery and riflery because of risk to others.

Approach to Sports Injuries

Sports participation always has a risk of injury.

Generally, sports injury can be divided into

- Overuse injuries

- Blunt trauma

• Fractures and dislocations (see p. 3201)

• Acute soft-tissue sprains and strains

Many injuries (eg, fractures, dislocations, soft-tissue contusions, blunt trauma, sprains and strains) are not unique to sports participation and can result from activities that are not athletic or from accidents (see also p. 3201). However, athletes may need to learn how to modify faulty techniques that predispose to injuries or may resist taking an adequate period of rest to recover from a sports injury (working through the pain).

Overuse: Overuse is one of the most common causes of athletic injury and is the cumulative effect of excessive, repetitive stress on anatomic structures. It results in trauma to muscles, tendons, cartilage, ligaments, bursae, fascia, and bone in any combination. Risk of overuse injury depends on complex interactions between individual and extrinsic factors. Individual factors include muscle weakness and inflexibility, joint laxity, previous injury, bone malalignment, and limb asymmetries. Extrinsic factors include training errors (eg, exercise without sufficient recovery time, excess load, building one group of muscles without training the opposing group, and extensive use of the same movement patterns), environmental conditions (eg, excessive running on banked tracks or crowned roads—which stresses the limbs asymmetrically), and training equipment characteristics (eg, unusual or unaccustomed motions, such as those made while on an elliptical trainer). Runners most often sustain injury after too rapidly increasing their intensity or length of workouts. Swimmers may be least prone to overuse injuries because buoyancy has protective effects, although they still are at risk, particularly in the shoulders, from which most movement occurs.

Blunt trauma: Blunt athletic trauma can result in injuries such as soft-tissue contusions, concussions, and fractures. The mechanism of injury usually involves high-impact collisions with other athletes or objects (eg, being tackled in football or checked into the sideboards in hockey), falls, and direct blows (eg, in boxing or the martial arts).

Sprains and strains: Sprains are injuries to ligaments, and strains are injuries to muscles. They typically occur with sudden, forceful exertion, most commonly during running, particularly with sudden changes of direction (eg, dodging and avoiding competitors in football). Such injuries also are common in strength training, when a person quickly drops or yanks at the load rather than moving slowly and smoothly with constant controlled tension.

Symptoms and Signs

Injury always results in pain, which ranges from mild to severe. Physical signs may be absent or may include any combination of soft-tissue edema, erythema, warmth, point tenderness, ecchymosis, instability, and loss of mobility.

Diagnosis

Diagnosis should include a thorough history and physical examination. History should focus on the mechanism of injury, physical stresses of the activity, past injuries, timing of pain onset, and extent and duration of pain before, during, and after activity. Diagnostic testing (eg, x-rays, ultrasonography, CT, MRI, bone scans, electromyography) and referral to a specialist may be required.

Treatment

• Rest, ice, compression, elevation (RICE)

• Analgesics

• Cross training

• Gradual return to activity

RICE: Immediate treatment of most acute sports injuries is RICE.

Rest prevents further injury and helps to reduce swelling.

Ice (or a commercial cold pack) causes vasoconstriction and reduces soft-tissue swelling, inflammation, and pain. Ice and cold packs should not be applied directly to the skin. They should be enclosed in plastic or a towel. They should be left in place for no more than 20 min at a time. An elastic bandage can be wrapped around a tightly closed plastic bag containing ice to keep it in place.

Wrapping an injured extremity with an elastic bandage for compression reduces edema and pain. The bandage should not be wrapped too firmly because doing so may cause swelling in the distal extremity.

The injured area should be elevated above heart level so that gravity can facilitate drainage of fluid, which reduces swelling and thus pain. Ideally, fluid should drain on an entirely downhill path from the injured area to the heart (eg, for a hand injury, the elbow, as well as the hand, should be elevated). Ice and elevation should be used periodically throughout the initial 24 h after an acute injury.

Pain control: Pain control usually involves use of analgesics, typically acetaminophen or NSAIDs. However, if pain persists for > 72 h after a seemingly minor injury, referral to a specialist is recommended. For persistent pain, evaluation for additional or more severe injuries is indicated. These injuries are treated as appropriate (eg, with immobilization, sometimes with oral or injectable corticosteroids). Corticosteroids should be given only by a specialist and when necessary because corticosteroids can delay soft-tissue healing and sometimes weaken injured tendons and muscles. The frequency of corticosteroid injections should be monitored by a specialist because too-frequent injections may increase the risk of tissue degeneration and ligament or tendon rupture.

Activity: In general, injured athletes should avoid the specific activity that caused the injury until after healing occurs. To minimize deconditioning, athletes can cross-train (ie, do different or related exercises that do not cause reinjury or pain). Injury may also necessitate reducing exercise range-of-motion if there is intolerable pain at certain points of movement. Initially, exercise of previously injured areas should be low in intensity to gradually strengthen weak muscles, tendons, and ligaments without risking reinjury. It is more important to maintain a good range-of-motion, which helps direct blood to the injured area to accelerate healing, than to rapidly resume full intensity training for fear of losing conditioning. Resumption of full activity should be gradual once pain subsides. Competitive athletes should consider consultation with a professional (eg, physical therapist).

Athletes should be placed in a graduated program of exercises and physical therapy to restore flexibility, strength, and endurance. They also need to feel psychologically ready before re-engaging in an activity at full capacity. Competitive athletes may benefit from motivational counseling.

Prevention

Exercise itself helps prevent injuries because tissues become more resilient and tolerant of the forces they experience during vigorous activities.

General warming up raises muscle temperature and makes muscles more pliable, stronger, and more resistant to injury; it also improves workout performance by enhancing mental and physical preparedness. Cooling down is sometimes thought to prevent dizziness and syncope after aerobic exercise and helps remove metabolic byproducts of exercise, such as lactic acid, from muscles and the bloodstream. Removing lactic acid may help decrease muscle soreness. Cooling down also helps decrease heart rate slowly and gradually to near-resting levels—an important effect for patients with heart disorders.

Injury due to excessive pronation (turning in or inversion of the foot during weight bearing) can be prevented with use of shoe inserts or orthotics (flexible or semirigid).

Rotator Cuff Injury

Rotator cuff injury includes tendinitis and partial or complete tears. Symptoms are shoulder

pain and, with severe tears, weakness. Diagnosis is by examination and, sometimes, diagnostic testing. Treatment includes NSAIDs, maintenance of range of motion, and rotator cuff strengthening exercises.

The rotator cuff, consisting of the supraspinatus, infraspinatus, teres minor, and subscapularis (SITS) muscles, helps stabilize the humeral head in the glenoid fossa of the scapula during overhead arm motions (eg, pitching, swimming, weightlifting, serving in racket sports).

Etiology

Rotator cuff injury can be a sports injury, but it commonly occurs for reasons unrelated to sports activities and in people with no history of overuse.

A strain of the rotator cuff is a single acute, traumatic injury to the muscles. Tendinitis typically results from chronic impingement of the supraspinatus tendon between the humeral head and coracoacromial arch (the acromion, acromioclavicular joint, coracoid process, and coracoacromial ligament). Activities that require the arm to be moved over the head repeatedly, such as pitching in baseball, lifting heavy weights over the shoulder, serving the ball in racket sports, and swimming freestyle, butterfly, or backstroke, increase the risk. The supraspinatus tendon is thought to be particularly susceptible because it has an under-vascularized region near its insertion on the greater tuberosity. The resultant inflammatory reaction and edema further narrow the subacromial space, accelerating tendon irritation or damage. If the process is not interrupted, the resulting inflammation can lead to partial or complete tear of the rotator cuff. Degenerative rotator cuff tendinitis is common among older (> 40 yr) people who are not athletes for the same reason. Subacromial bursitis (inflammation, swelling, and fibrosis of the bursal area above the rotator cuff) commonly results from tendinitis of the cuff.

Symptoms and Signs

Subacromial bursitis, rotator cuff tendinitis, and partial rotator cuff tears cause shoulder pain, especially when the arm is moved overhead. The pain usually is worse between 60° and 120° (painful arc of motion) of shoulder abduction or flexion and is usually minimal or absent at < 60° or > 120°. The pain may be described as a dull ache that is poorly localized. Complete rotator cuff tears result in acute pain and weakness of the shoulder. In larger tears of the rotator cuff, weakness of external rotation is particularly apparent.

Diagnosis

• Physical examination

• Sometimes MRI or arthroscopy

Diagnosis is by history and physical examination, including provocative maneuvers. The rotator cuff cannot be palpated directly, but it can be assessed indirectly by provocative maneuvers that test its individual components; significant pain or weakness is considered a positive result.

The **supraspinatus** is assessed by having the patient resist downward pressure on the arms held in forward flexion with the thumbs pointing downward (empty can, or Jobe's test).

The **infraspinatus** and **teres minor** are assessed by having the patient resist external rotation pressure with the arms held at the sides with elbows flexed to 90°; this position isolates rotator cuff muscle function from that of other muscles such as the deltoid. Weakness during this test suggests significant rotator cuff dysfunction (eg, a complete tear).

The **subscapularis** is assessed by having the patient place the hand behind the back with the back of the hand resting on the lower back. The examiner lifts the hand off the lower back. The patient should be able to keep the hand off the skin of the back (Gerber lift-off test).

The **Neer test** checks for impingement of the rotator cuff tendons under the coracoacromial arch. It is

done by placing the arm in forced forward flexion (arm lifted overhead) with the arm fully pronated.

The **Hawkins test** also checks for impingement. It is done by elevating the arm to 90°, flexing the elbow 90°, and then forcibly rotating the shoulder internally.

The **Apley scratch test** assesses combined shoulder range of motion by having the patient attempt to touch the opposite scapula: Reaching overhead, behind the neck, and to the opposite scapula with the tips of the fingers tests abduction and external rotation; reaching under, behind the back, and across to the opposite scapula with the back of the hand tests adduction and internal rotation.

Other areas that may be the source of shoulder pain include the acromioclavicular and sternoclavicular joints, cervical spine, biceps tendon, and scapula. These areas should be assessed for any tenderness or deformity indicating a problem in those areas.

The **neck** is examined as part of any shoulder evaluation because pain can be referred to the shoulder from the cervical spine (particularly with C5 radiculopathy).

Suspected rotator cuff injury can be further evaluated with MRI, arthroscopy, or both.

Treatment

• NSAIDs

• Exercises

• Sometimes surgery

In most cases of tendinitis and bursitis, rest, NSAIDs, and rotator cuff strengthening exercises (see Sidebar 338-1) are sufficient. Injections of corticosteroids into the subacromial bursa are occasionally indicated (eg, when symptoms are acute and severe or when prior treatment has been ineffective). Surgery may be necessary in chronic bursitis that is resistant to conservative management to remove excess bone and decrease impingement. Surgical repair may be recommended if a rotator cuff injury is severe (eg, a complete tear).

Glenoid Labral Tear

The glenoid labrum usually tears as a result of a specific trauma, such as a fall onto an outstretched arm. Tears can also result from chronic overhead movement, as occurs in pitching. A glenoid labral tear causes pain during motion. Treatment is with physical therapy and sometimes surgery.

The shoulder (unlike the hip or elbow) is an inherently unstable joint; it has been likened to a golf ball sitting on a tee. To enhance structural stability, the glenoid (anatomically, a very shallow socket) is deepened by the labrum, which is a rubbery, fibrocartilaginous material attached around the lip of the glenoid. This structure can tear during athletics, especially during throwing sports, or as a result of blunt trauma when falling and landing on an outstretched upper extremity.

Sidebar 338-1 Exercises to Strengthen the Shoulders

Rotator Cuff Exercises

External Rotation

• Lying on the left side, grasp a light dumbbell in the right hand with the elbow bent 90°. Maintain this position throughout the set.

• Using the right elbow as a pivot point against the side of the waist, externally rotate the arm upward until

it is as vertical as possible.

- Do 3 sets of 10 repetitions with 1 min of rest between each set.
- Repeat with the opposite arm.
- As strength improves, increase the weight.

Internal Rotation

- Lying on the right side, grasp a light dumbbell in the right hand with the right elbow bent at 90°. Maintain this position throughout the set.
- Using the right elbow as a pivot point against the side of the waist, internally rotate the arm upward (inward) until it is vertical against the abdomen.
- Do 3 sets of 10 repetitions with 1 min of rest between each set.
- Repeat with the opposite arm.
- As strength improves, increase the weight.

Deltoid Exercises

Anterior Deltoid

- In the standing position, grasp a light dumbbell with the right hand and with the palm facing down, raise the hand and arm away and in front of the body to shoulder level, keeping the elbow straight.
- Do 3 sets of 10 repetitions with 1 min of rest between each set.
- Repeat with the opposite arm.

Middle Deltoid

- In the standing position, grasp a light dumbbell with the right hand and with the palm facing down, raise the hand and arm away and out to the side from the body to shoulder level, keeping the elbow straight.
- Do 3 sets of 10 repetitions with 1 min of rest between each set.
- Repeat with the opposite arm.

Symptoms and Signs

A glenoid labral tear results in deep shoulder pain during motion, especially when pitching a baseball. This discomfort may be accompanied by a painful clicking or clunking sensation and a feeling of catching in the shoulder.

Diagnosis

A thorough shoulder and neck physical examination should be done initially, but referral to a specialist is frequently needed because more sophisticated diagnostic tests (eg, contrast-enhanced MRI) are often the only way to definitively identify the pathology.

Treatment

Physical therapy is the initial treatment. If symptoms do not subside with physical therapy, and the

diagnosis has been confirmed by MRI, surgical debridement or repair is the treatment of choice. Surgery is usually done arthroscopically.

Lateral Epicondylitis

(Tennis Elbow)

Lateral epicondylitis results from inflammation and microtearing of fibers in the extensor tendons of the forearm. Symptoms include pain at the lateral epicondyle of the elbow, which can radiate into the forearm. Diagnosis is by examination and provocative testing. Treatment is with rest, NSAIDs, and physical therapy.

Theories about the pathophysiology of lateral epicondylitis include nonathletic and occupational activities that require repetitive and forceful forearm supination and pronation, as well as overuse or weakness (or both) of the extensor carpi radialis brevis and longus muscles of the forearm, which originate from the lateral epicondyle of the elbow. For example, during a backhand return in racket sports such as tennis, the elbow and wrist are extended, and the extensor tendons, particularly the extensor carpi radialis brevis, can be damaged when they roll over the lateral epicondyle and radial head. Contributing factors include weak shoulder and wrist muscles, a racket strung too tightly, an undersized grip, hitting heavy wet balls, and hitting off-center on the racket.

In resistance trainees, injuries often are caused by overuse (too much activity or doing the same movements too often) or by muscle imbalance between the forearm extensors and flexors. Nonathletic activities that can cause or contribute to lateral epicondylitis include those involving grasping and twisting the elbow (eg, turning a screwdriver).

With time, subperiosteal hemorrhage, calcification, spur formation on the lateral epicondyle, and, most importantly, tendon degeneration can occur.

Symptoms and Signs

Pain initially occurs in the extensor tendons of the forearm and around the lateral elbow when the wrist is extended against resistance (eg, as in using a manual screw driver or hitting a backhand shot with a racket). In resistance trainees, lateral epicondylitis is most noticeable during various rowing and chin-up exercises for the back muscles, particularly when the hands are pronated. Pain can extend from the lateral epicondyle to the mid forearm.

Diagnosis

• Provocative testing

Pain along the common extensor tendon when the fingers are extended against resistance and the elbow is held straight is diagnostic. Alternatively, the diagnosis is confirmed if the same pain occurs during the following maneuver: The patient sits on a chair with the forearm on the examination table and the elbow held flexed (bent) and the hand held palm downward; the examiner places a hand firmly on top of that of the patient, who tries to raise the hand by extending the wrist.

Treatment

• Rest, ice, NSAIDs, extensor muscle stretches

• Modification of activity

• Later, resistive exercises

Treatment involves a 2-phased approach. Initially, rest, ice, NSAIDs, and stretching of the extensor muscles are used. Occasionally a corticosteroid injection into the painful area around the tendon is needed. When the pain subsides, gentle resistive exercises of the extensor (see Sidebar 338-2) and

flexor (see Sidebar 338-3) muscles in the forearm are done followed by eccentric and concentric resistive exercises. Activity that hurts when the wrist is extended or pronated should be avoided. Use of a tennis elbow brace is often advised. Adjusting the fit and type of racket used can also help prevent further injury.

Sidebar 338-2 Exercises to Strengthen the Wrist Extensors

These exercises should be done only after the pain has subsided.

- Sit on a chair next to a table.
- Place the forearm on the table, palm facing down and elbow bent, with the wrist and hand hanging over the edge.
- Hold a light weight in the hand.
- Slowly raise and lower the hand by bending and straightening the wrist, keeping the forearm planted firmly on the table.
- Do 3 sets of 10 repetitions with 1 min of rest between repetitions.
- As the exercise becomes easier, increase the weight.

Next,

- While standing and with both arms held out in front of the body palms downward, wind up a 450-g (1-lb) weight that is attached by a rope to a piece of wood with the diameter of a broomstick (the rope will be attached to the center of the device with the hands grasping on either side). The weight should almost touch the ground when the rope is unrolled.
- Roll the weight up and down 5 or 6 times by using the strength of the forearm extensor muscles (ie, the rotation of the stick is upward and toward the body); stop if pain is felt. Repeat the exercise every other day.
- Gradually increase the weight. Do not increase the number of times that the weight is rolled up. As heavier weights are used and as tissues become stronger, frequency should decrease to once every 7 to 10 days.

Adapted from Mirkin G, Shangold M: *The Complete Sports Medicine Book for Women* . New York, Simon & Schuster, 1985, p. 109; used by permission of The Miller Press.

Although surgery is not usually needed, surgical techniques to treat lateral epicondylitis involve removing scar and degenerative tissue from the involved extensor tendons at the elbow. Surgery is usually considered only after at least 9 to 12 mo of unsuccessful conservative treatment.

Medial Epicondylitis

(Golfer's Elbow)

Medial epicondylitis is inflammation of the flexor pronator muscle mass originating at the medial epicondyle of the elbow. Diagnosis is with provocative testing. Treatment is rest and ice and then exercises and gradual return to activity.

Medial epicondylitis is caused by any activity that places a valgus force on the elbow or that involves forcefully flexing the volar forearm muscles, as occurs during pitching, golfing with improper technique, serving a tennis ball (particularly with top spin, with a racket that is too heavy or too tightly strung or has an undersized grip, or with heavy balls), and throwing a javelin. Nonathletic activities that may cause

medial epicondylitis include bricklaying, hammering, and typing.

Symptoms and Signs

Pain occurs in the flexor pronator tendons (attached to the medial epicondyle) and in the medial epicondyle when the wrist is flexed or pronated against resistance.

Diagnosis

• Provocative testing

To confirm the diagnosis, the examiner has the patient sit in a chair with the forearm resting on a table and the hand supinated. The patient tries to raise the fist by bending the wrist while the examiner holds it down. Pain around the medial epicondyle and in the flexor tendon origin confirms the diagnosis.

Treatment

• Rest, ice, and muscle stretches

• Modification of activity

• Later, resistive exercises

Treatment is symptomatic and similar to that of lateral epicondylitis (see p. 3300). Patients should avoid any activity that causes pain. Initially, rest, ice, NSAIDs, and stretching are used, occasionally with a corticosteroid injection into the painful area around the tendon. When pain subsides, gentle resistive exercises of the extensor and flexor muscles of the forearm are done, followed by eccentric and concentric resistive exercises. In general, surgery is considered only after at least 9 to 12 mo of failed conservative management. Surgical techniques to treat medial epicondylitis involve removing scar tissue and reattaching damaged tissues.

Sidebar 338-3 Exercises to Strengthen the Wrist Flexors

• Sit on a chair next to a table.

• Place the forearm on the table, palm facing up, with the wrist and hand hanging over the edge.

• Hold a light weight in the hand.

• Slowly raise and lower the hand by bending and straightening the wrist.

• The set should last about 90 to 120 sec for rehabilitation and about 50 to 70 sec for general strength and conditioning. Rest 1 min, then do additional sets until the forearms feel fatigued and worked. Repeat every 2 days while rehabilitating but only once every 7to10 days with normal or strong forearms and when using heavier weights and greater intensity of effort. If pain is felt, stop the exercise immediately, and try it again the next day.

• As the exercise becomes easier, increase the weight. For rehabilitation, frequency should decrease as the muscles become stronger and heavier weights are used.

Piriformis Syndrome

Piriformis syndrome is compression of the sciatic nerve by the piriformis muscle in the posterior pelvis, causing pain in the buttocks and occasionally sciatica. Diagnosis is by examination. Treatment is symptomatic.

The piriformis muscle extends from the pelvic surface of the sacrum to the upper border of the greater trochanter of the femur. During running or sitting, this muscle can compress the sciatic nerve at the site where it emerges from under the piriformis to pass over the hip rotator muscles. Piriformis syndrome is uncommon.

Symptoms and Signs

A chronic nagging ache, pain, tingling, or numbness starts in the buttocks and can extend along the course of the sciatic nerve, down the entire back of the thigh and calf, and sometimes into the foot. Pain worsens when the piriformis is pressed against the sciatic nerve (eg, while sitting on a toilet, a car seat, or a narrow bicycle seat or while running).

Diagnosis

• Physical examination and provocative testing

Diagnosis is by physical examination. Pain with forceful internal rotation of the flexed thigh (Freiberg's maneuver), abduction of the affected leg while sitting (Pace's maneuver), raising of the knee several centimeters off the table while lying on a table on the side of the unaffected leg (Beatty's maneuver), or pressure into the buttocks where the sciatic nerve crosses the piriformis muscle while the patient slowly bends to the floor (Mirkin test) is diagnostic. Imaging is not useful except to rule out other causes of sciatic compression. Unlike piriformis pain, lumbar disk compression of the sciatic nerve (sciatica—see p. 383) usually causes low back pain in addition to sciatic pain down the lower extremity. However, differentiation from a lumbar disk disorder is sometimes difficult, and referral to a specialist may be needed.

Treatment

• Modification of activity

• Stretches

Patients should temporarily stop running, bicycling, or doing any activity that elicits pain. Patients whose pain is aggravated by sitting should stand or, if unable to do so, change positions to remove the source of pressure around the buttock. Specific stretching exercises for the posterior hip and piriformis can be beneficial. Surgery is rarely warranted. A carefully directed corticosteroid injection near the site where the piriformis muscle crosses the sciatic nerve often helps temporarily. NSAIDs can also provide temporary pain relief.

Knee Pain

Etiology

There are many causes of pain in or around the knee in athletes, particularly runners, including

• Subluxation of the patella when bending the knee

• Chondromalacia of the undersurface of the patella (runner's knee, which is softening of the knee cap cartilage—see p. 2912)

• Intra-articular pathology, such as meniscal tears and plicae (infolding of the normal synovial lining of the knee)

• Fat pad inflammation

• Stress fractures of the tibia

• Malalignment of the lower extremities

• Patellar (or infrapatellar) tendinitis (jumper's knee, which is an overuse injury to the patellar tendon at the attachment to the lower pole of the patella—see p. 2913)

Knee pain may be referred from the lumbar spine or hip or result from foot problems (eg, excessive pronation or rolling inward of the foot during walking or running).

Evaluation

Diagnosis requires a thorough review of the injured athlete's training program, including a history of symptom onset and aggravating factors, and a complete lower-extremity examination (for knee examination, see pp. 285 and 3217).

Mechanical symptoms, such as locking or catching, suggest an internal derangement of the knee such as a meniscal tear. Instability symptoms, such as giving way and loss of confidence in the extremity when twisting or turning on the knee, suggest ligamentous injury or subluxation of the patella.

Chondromalacia is suggested by anterior knee pain after running, especially on hills, as well as pain and stiffness after sitting for any length of time (positive movie sign). On examination, pain is typically reproduced by compression of the patella against the femur.

Pain that becomes worse with weight-bearing suggests a stress fracture.

Treatment

Treatment is tailored to the specific cause of the pain.

Treatment of chondromalacia includes quad-riceps strengthening exercises with balanced strengthening exercises for the hamstrings, use of arch supports if excessive pronation is a possible contributor, and use of NSAIDs.

For patellar subluxation, use of patella-stabilizing pads or braces may be necessary, especially in sports that require rapid, agile movements in various planes (eg, basketball, tennis).

If there is excessive pronation of the foot, and all other possible causes of knee pain have been excluded, use of an orthotic insert is sometimes useful.

Stress fractures require rest and cessation of weight-bearing activity.

Intra-articular pathology often requires surgery.

Shin Splints

The term shin splints refers to nonspecific pain that occurs in the lower legs during running sports.

Repetitive impact forces during jogging, running, or vigorous walking (eg, hiking) can overload the musculotendinous unit and cause shin pain. Such pain sometimes results from a specific injury (eg, tibial stress fracture, exercise-induced compartment syndrome, tibial periostitis, excessive foot pronation), but often an exact cause cannot be identified. In such cases, the term shin splints is used.

Symptoms and Signs

Shin pain can occur in the anterior or posterior aspect of the leg and typically begins at the start of activity but then lessens as activity continues. Pain that persists during rest suggests another cause, such as stress fracture of the tibia.

Diagnosis

• Usually clinical

On examination, severe localized tenderness is usually present over the anterior compartment muscles, and sometimes there is palpable bone pain.

X-ray findings are usually unremarkable, regardless of the cause. If a stress fracture is suspected, a bone scan may be necessary.

Exercise-induced compartment syndrome is diagnosed by using a specialized manometer to document increased intra-compartmental pressure during exercise.

Treatment

• Modification of activity

• Stretches, NSAIDs

Running must be stopped until it causes no pain. Early treatment is ice, NSAIDs, and stretching of the anterior and posterior calf muscles (see Sidebar 338-4). During the rest phase of treatment, deconditioning can be minimized by encouraging cross-training techniques that do not require repetitive weight-bearing activity, such as swimming.

Once symptoms have resolved, it is advised that a return to running be gradual. Wearing supportive shoes with rigid heel counters and arch supports helps support the foot and ankle during running and can aid recovery and prevent further symptoms. Avoiding running on hard surfaces (eg, cement roads) can also help. Exercising the front of the calves by dorsiflexing the ankle against resistance (eg, rubber bands or a dorsiflexion machine) increases leg muscle strength and can help prevent shin pain.

Sidebar 338-4 Strengthening the Calf and Shin Muscles

Calf

Toe raises

• Stand up. Slowly rise up on the toes, then slowly lower the heels to the floor.

• Do 3 sets of 10 repetitions with 1 min of rest between repetitions.

• When this exercise becomes easy, do it while holding progressively heavier weights.

Shin

Heel raises

• Stand on the heels and walk 3 to 4.5 m (10 to 15 ft).

• Do this exercise 3 times.

Achilles Tendinitis

Achilles tendon injuries include inflammation of the paratenon and partial or complete tears.

Achilles tendinitis is very common among running athletes. The calf muscles attach to the calcaneus via the Achilles tendon. During running, the calf muscles help with the lift-off phase of gait. Repetitive forces from running combined with insufficient recovery time can initially cause inflammation in the tendon

paratenon (fatty areolar tissue that surrounds the tendon). A complete tear of the Achilles tendon is a serious injury, usually resulting from sudden, forceful stress (see p. 3217). Tendon tears can occur with minimal exertion in people who have taken fluoroquinolone antibiotics.

Symptoms and Signs

The primary symptom of Achilles tendon inflammation is pain in the back of the heel, which initially increases when exercise is begun and often lessens as exercise continues. A complete tear of the Achilles tendon typically occurs with a sudden forceful change in direction when running or playing tennis and is often accompanied by a sensation of having been struck in the back of the ankle and calf with an object such as a baseball bat.

Diagnosis

• Clinical evaluation

On examination, an inflamed or partially torn Achilles tendon is tender when squeezed between the fingers. Complete tears are differentiated by

• Sudden, severe pain and inability to walk on the extremity

• A palpable defect along the course of the tendon

• A positive Thompson test (while the patient lies prone on the examination table, the examiner squeezes the calf muscle; this maneuver by the examiner does not cause the normally expected plantar flexion of the foot)

Treatment

• Ice, NSAIDs, and stretches

• Modification of activities

Tendon inflammation should initially be treated with ice, gentle calf muscle stretching, and use of NSAIDs. A heel lift can be placed in the shoes to take tension off the tendon. Athletes should be instructed to avoid uphill and downhill running until the tendon is not painful and to engage in cross-training aerobic conditioning. Complete tears of the Achilles tendon usually require surgical repair.

Stress Fractures

Stress fractures are small incomplete fractures that often involve the metatarsal shafts. They are caused by repetitive weight-bearing stress.

Stress fractures do not usually result from a discrete injury (eg, fall, blow) but occur instead following repeated stress and overuse that exceeds the ability of the supporting muscles to absorb the stress. Stress fractures can involve the proximal femur, pelvis, or lower extremity. Over 50% involve the lower leg and, in particular, the metatarsal shafts of the foot. Metatarsal stress fractures (march fractures) usually occur in

• Runners who too quickly change intensity of workouts, time of workouts, or both

• Poorly conditioned people who walk long distances carrying a load (eg, newly recruited soldiers)

They most commonly occur in the 2nd metatarsal. Other risk factors include the following:

• Cavus foot (a foot with a high arch)

• Shoes with inadequate shock-absorbing qualities

• Osteoporosis

Stress fractures also may be a sign of the female athlete triad (amenorrhea, eating disorder, and osteoporosis).

Symptoms and Signs

Forefoot pain that occurs after a long or intense workout and then disappears shortly after stopping exercise is the typical initial manifestation of a metatarsal stress fracture. With subsequent exercise, onset of pain is progressively earlier, and pain may become so severe that it prohibits exercise and persists even when patients are not bearing weight.

Patients who have groin pain with weight bearing must be evaluated for a proximal femur stress fracture. Patients with such fractures should be referred to a specialist.

Diagnosis

• X-ray or bone scan

Standard x-rays are recommended but may be normal until a callus forms 2 to 3 wk after the injury. Often, technetium diphosphonate bone scanning is necessary for early diagnosis. Women with stress fractures may have osteoporosis and should undergo dual-energy x-ray absorptiometry (see p. 357).

Treatment

• Restriction of weight-bearing activity

Treatment includes cessation of weight bearing on the involved foot (in case patients have a metatarsal stress fracture) and use of crutches. Although casting is sometimes used, a wooden shoe or other commercially available supportive shoe or boot is preferable to casting to avoid muscle atrophy. Healing can take anywhere from 6 to 12 wk.

Popliteus Tendinitis

Popliteus tendinitis is inflammation in the popliteus tendon, which extends from the outer surface of the bottom of the femur diagonally across the posterior knee to the medial superior tibia.

The popliteus tendon prevents the lower leg from twisting outward during running as well as helping to prevent forward movement of the femur on the tibia. Excessive running downhill tends to put excessive stress on this tendon.

Pain and soreness, particularly when running downhill, develop along the posterolateral knee. Diagnosis is by physical examination. The patient sits with the involved extremity in a cross-legged position (ie, the hip flexed, abducted, and externally rotated and the knee flexed with the leg crossed over the opposite extremity). The examiner then palpates the posterior lateral corner for tenderness.

Treatment includes rest, NSAIDs, ice, and occasionally physical therapy. Patients should not run until the area is free of pain and then should limit their workouts and downhill running for at least 6 wk. Bicycling is a good alternative exercise during healing.

Hamstring Strain

A hamstring strain is a partial tear of the hamstring muscles most commonly at the musculotendinous junction.

Hamstring strains are common among runners. Athletes at risk include those with poor flexibility of the hamstring muscles, inadequate pre-participation stretching and warm up, and previous injury. Older athletes are also at higher risk. As with any muscle strain, the amount of force that caused the muscle to tear determines the degree of injury.

Symptoms and Signs

Strains of the hamstring muscles can manifest as an acute painful area in the posterior thigh when sprinting or running or develop more slowly, usually because of inadequate flexibility training.

Sidebar 338-5 Strengthening the Hamstrings

Upper part of the hamstrings

- Attach a 2-kg (5-lb) weight to the foot on the injured side. Lie face down on a bed with the lower part of the body (from the waist down) off the bed and the toes touching the floor.
- Keeping the knee straight, slowly raise and lower the leg.
- Do 3 sets of 10 repetitions with 1 min of rest between repetitions.
- As strength returns, use increasingly heavier weights.
- Do this exercise every other day.

Lower part of the hamstrings

- Attach a 2-kg (5-lb) weight to the foot on the injured side. Stand on the other leg.
- Without bending the hip, slowly raise the weighted foot toward the buttocks by bending the knee, and lower it toward the floor by straightening the knee.
- Do 3 sets of 10 repetitions with 1 min of rest between repetitions.
- As strength returns, use increasingly heavier weights.
- Do this exercise every other day.

Diagnosis

- Clinical evaluation

The diagnosis is confirmed by finding ham-string pain with knee flexion against resistance as well as on palpation of the posterior thigh. In mild strains, tenderness and mild swelling are present. In more severe strains, ecchymosis, moderate to severe swelling, and poor muscle function caused by pain and weakness are present.

Treatment

- Rest, ice, and compression
- Stretching, then strengthening exercises

Ice and compression with use of a thigh sleeve should begin as soon as possible. NSAIDs and analgesics are prescribed as necessary, and crutches may be required initially if walking is painful.

Once pain begins to resolve, patients should begin gentle hamstring stretching. When the pain has completely resolved, gradual strengthening of the quadriceps and hamstrings is begun (see Sidebar 338-5).

Only when satisfactory strength has been achieved should patients resume running. Athletes must be made aware that recovery from hamstring injury can often take up to several months, depending on the severity.

Chapter 339. Bites and Stings

Introduction

More than 90,000 bites and stings are reported to poison control centers each year; many more occur but are not reported. Bites and stings result in about 100 deaths/yr in the US. All patients with bites and stings should receive appropriate tetanus prophylaxis (see
Table 140-1 on p. 1299).

Centipede and Millipede Bites

Some larger centipedes can inflict a painful bite, causing swelling and redness. Symptoms rarely persist for more than 48 h. Millipedes do not bite but may secrete a toxin that is irritating, particularly when accidentally rubbed into the eye.

An ice cube placed on a centipede bite usually relieves the pain. Toxic secretions of millipedes should be washed from the skin with large amounts of soap and water. If a skin reaction develops, a corticosteroid cream should be applied. Eye injuries should be irrigated immediately.

Human and Mammal Bites

(See also Rat-Bite Fever on p. 1272.)

Human and other mammal (mostly dog and cat, but also squirrel, gerbil, rabbit, guinea pig, and monkey) bites are common and occasionally cause significant morbidity and disability. The hands, extremities, and face are most frequently affected, although human bites can occasionally involve breasts and genitals.

Bites by large animals sometimes cause significant tissue trauma; about 10 to 20 people, mostly children, die from dog bites each year. However, most bites involve relatively minor wounds.

Infection: In addition to tissue trauma, infection from the biting organism's oral flora is a major concern. Human bites can theoretically transmit viral hepatitis and HIV. However, HIV transmission is unlikely because the concentration of HIV in saliva is much lower than in blood and salivary inhibitors render the virus ineffective.

Rabies is a risk with certain mammal bites (see p. 1732). Monkey bites, usually restricted in the US to animal laboratory workers, carry a small risk of herpesvirus simiae infection, which causes vesicular skin lesions at the inoculation site and can progress to encephalitis, which is often fatal.

Bites to the hand (see also p. 388) carry a higher risk of infection, specifically cellulitis, tenosynovitis, septic arthritis, and osteomyelitis, than other bite sites; this higher risk is a particular concern in human bites resulting from a clenched-fist strike to the mouth (fight bite), the most common human bite wound. In fight bites, the skin wound moves away from the underlying damaged structures when the hand is opened, trapping bacteria inside, and patients often delay seeking treatment, allowing bacteria to multiply. Human bites to sites other than the hand have not been proved to carry a greater risk of infection than bites from other mammals. Cat bites to the hand also have a high risk of infection because cats' long, slender teeth often penetrate deep structures, such as joints and tendons, and the small punctures are then sealed off.

Diagnosis

- Evaluation in the position in which the bite was inflicted
- Assessment for damage to underlying nerve, tendon, bone, and vasculature and for presence of foreign bodies

Human bites sustained in an altercation are often attributed to other causes to avoid involvement of the authorities or to ensure insurance coverage. Domestic violence is often denied.

Wounds are evaluated for damage to underlying structures (eg, nerves, vasculature, tendons, bone) and for foreign bodies (see p. 3193). Evaluation should focus on careful assessment of function and the extent of the bite. Wounds over or near joints should be examined in the position in which they were inflicted (eg, with fist clenched) and explored under sterile conditions to assess tendon, bone, and joint involvement and to detect retained foreign bodies. Wounds inflicted by chomping may appear to be minor abrasions but should be examined to rule out deep injury.

Culturing fresh wounds is not valuable for targeting antimicrobial therapy, but infected wounds should be cultured. For patients with human bites, screening for hepatitis or HIV is recommended only if the attacker is known or suspected to be seropositive.

Treatment

• Meticulous wound care

• Selective wound closure

• Selective use of prophylactic antibiotics

Hospitalization is indicated if complications mandate very close monitoring, particularly when patient characteristics predict a high risk of nonadherence with outpatient follow-up. Hospitalization should be considered in the following circumstances:

• When a human bite is infected (including clenched-fist injuries)

• When a nonhuman bite is moderately or severely infected

• When loss of function is evident

• When the wound threatens or has damaged deep structures

• When a wound is disabling or difficult to care for at home (eg, significant wounds to both hands or both feet, hand wounds that require continuous elevation)

Priorities of treatment include wound cleaning, debridement, closure, and infection prophylaxis.

Wound care: Wounds should first be cleaned with a mild antibacterial soap and water (tap water is sufficient), then pressure irrigated with copious volumes of saline solution using a syringe and IV catheter. A dilute povidoneiodine solution (10:1 with 0.9% saline) may also be used. A local anesthetic should be used as needed. Dead and devitalized tissue should be debrided.

Wound closure is done only for select wounds. Many wounds should initially be left open, including the following:

• Puncture wounds

• Wounds to the hands, feet, perineum, or genitals

• Wounds more than several hours old

• Wounds that are heavily contaminated

• Wounds that are markedly edematous

• Wounds that show signs of inflammation

• Wounds that involve deeper structures (eg, tendon, cartilage, bone)

• Wounds due to human bites

• Wounds sustained in a contaminated environment (eg, marine, field, sewers)

In addition, wound resolution in immunocompromised patients may be better with delayed closure. Other wounds (ie, fresh, cutaneous lacerations) can usually be closed after appropriate wound hygiene. Results with delayed primary closure are comparable to those with primary closure, so little is lost by leaving the wound open initially if there is any question.

Hand bites should be wrapped in sterile gauze, splinted in position of function (slight wrist extension, metacarpophalangeal and both interphalangeal joints in flexion). If wounds are moderate or severe, the hand should be continuously elevated (eg, hanging from a pole).

Facial bites may require reconstructive surgery given the cosmetic sensitivity of the area and the potential for scarring.

Infected wounds may require debridement, suture removal, soaking, splinting, elevation, and IV antibiotics, depending on the specific infection and clinical scenario. Joint infections and osteomyelitis may require prolonged IV antibiotic therapy and orthopedic consultation.

Antimicrobials: Thorough wound cleaning is the most effective and essential way to prevent infection and often suffices. There is no consensus on indications for prophylactic antibiotics. Studies have not confirmed a definite benefit, and widespread use of prophylactic antibiotics has the potential to select resistant organisms. Drugs do not prevent infection in heavily contaminated or inadequately cleaned wounds. However, many practitioners prescribe prophylactic antibiotics for bites to the hand and some other bites (eg, cat bites, monkey bites).

Infections are treated with antimicrobials initially chosen based on animal species. Culture results, when available, guide subsequent therapy.

• **Human and dog bites:** For outpatients, the preferred drug is amoxicillin/clavulanate 500 to 875 mg po bid for 3 days for prophylaxis or 5 to 7 days for treatment. Ampicillin/sulbactam 1.5 to 3.0 g IV q 6 h is a reasonable empiric choice for inpatients; it covers α-hemolytic streptococci, *Staphylococcus aureus,* and *Eikenella corrodens,* the organisms most commonly cultured from human bites, as well as *Pasteurella* species (*P. canis* and *P. multocida*) and *Capnocytophaga canimorsus*, present in dog bites. Penicillin-allergic patients with human bites can be treated with trimethoprim/sulfamethoxazole 160/800 mg IV q 12 h plus clindamycin 150 to 300 mg IV q 6 h. For penicillin-allergic patients with infected dog bites, doxycycline is an acceptable alternative, except for children > 8 yr and pregnant women. Erythromycin can be used, but the risk of treatment failure is higher because of antimicrobial resistance. Other acceptable combinations include clindamycin and a fluoroquinolone for adults or clindamycin and trimethoprim/sulfamethoxazole for children.

• **Cat bites:** A fluoroquinolone (eg, ciprofloxacin 500 mg po bid for 5 to 7 days) for prophylaxis and treatment is recommended because of the prevalence of *P. multocida*. (*Bartonella henselae*—see p. 1244—is also transmitted by cat bites.) Alternatives for penicillin-allergic patients are clarithromycin 500 mg po bid for 7 to 10 days or clindamycin 150 to 300 mg po qid for 7 to 10 days.

• **Squirrel, gerbil, rabbit, and guinea pig bites:** These bites rarely become infected, but when they do, they can be treated with the same drugs as infected cat bites.

• **Monkey bites:** Monkey bites should be treated prophylactically with IV acyclovir 800 mg 5 times/day for 14 days.

Patients with human bites should receive postexposure prophylaxis for viral hepatitis (see p. 254) and HIV (see p. 1459) as indicated by patient and attacker serostatus. If status is unknown, prophylaxis is not indicated.

Insect Stings

Stinging insects are members of the order Hymenoptera of the class Insecta. Hymenoptera venoms cause local toxic reactions in all people and allergic reactions only in those previously sensitized. Severity depends on the dose of venom and degree of previous sensitization. Patients exposed to swarm attacks and patients with high venom-specific IgE levels are most at risk of anaphylaxis; many children never outgrow the risk. The average unsensitized person can safely tolerate 22 stings/kg body weight; thus, the average adult can withstand > 1000 stings, whereas 500 stings can kill a child.

Unexpectedly large numbers of people seek medical attention for stings and their complications after hurricanes and possibly other environmental disasters.

Major Hymenoptera subgroups are

• Apids (eg, honeybees, bumblebees)

• Vespids (eg, wasps, yellow jackets, hornets)

• Formicids (eg, nonwinged fire ants)

Apids usually do not sting unless provoked; however, Africanized honeybees (killer bees), migrants from South America that reside in some southern and southwestern US states, are especially aggressive when agitated. Apids typically sting once and dislodge their barbed stinger into the wound, introducing venom and killing the insect. Melittin is thought to be the main pain-inducing component of the venom. The venom of Africanized honeybees is no more potent than that of other honeybees but causes more severe consequences because these insects attack in swarms and inflict multiple stings, increasing the dose of venom. In the US, bee stings cause 3 to 4 times more deaths than do venomous snakebites.

Vespid stingers have few barbs and do not stay in the skin, so these insects can inflict multiple stings. The venom contains phospholipase, hyaluronidases, and a protein termed antigen 5, which is the most allergenic. Although vespids also avoid stinging unless provoked, they nest close to humans, so provocative encounters are more frequent. Yellow jackets are the major cause of allergic reactions to insect stings in the US.

Fire ants are present in the southern US, particularly in the Gulf region, where in urban areas, they may sting as many as 40% of the population, causing at least 30 deaths/yr. There are several species, but *Solenopsis invicta* predominates and is responsible for an increasing number of allergic reactions. The ant bites to anchor itself to the person and stings repeatedly as it rotates its body in an arc around the bite, producing a characteristic central bite partially encircled by a reddened sting line. The venom has hemolytic, cytolytic, antimicrobial, and insecticidal properties; 3 or 4 small aqueous protein fractions are probably responsible for allergic reactions.

Symptoms and Signs

Local apid and vespid reactions are immediate burning, transient pain, and itching, with an area of erythema, swelling, and induration up to a few centimeters across. Swelling and erythema usually peak at 48 h, can persist for a week, and can involve an entire extremity. This local chemical cellulitis is often confused with secondary bacterial cellulitis, which is more painful and uncommon after envenomation. Allergic reactions may manifest with urticaria, angioedema, bronchospasm, refractory hypotension, or a combination; swelling alone is not a manifestation of allergic reaction.

Symptoms and signs of a fire ant sting are immediate pain followed by a wheal and flare lesion, which often resolves within 45 min and gives rise to a sterile pustule, which breaks down within 30 to 70 h. The lesion sometimes becomes infected and can lead to sepsis. In some cases, an edematous, erythematous, and pruritic lesion, rather than a pustule, develops. Anaphylaxis due to fire ant stings probably occurs in < 1% of patients. Mononeuritis and seizures have been reported.

Diagnosis

Diagnosis is clinical. Apid stings are checked for the stinger. Upper and lower airways are assessed for signs of allergic reaction. Secondary bacterial cellulitis is rare but is considered when erythema and swelling begin a day or two after the sting (rather than immediately) and pain is significant.

Treatment

• Parenteral epinephrine and antihistamines for systemic allergic reactions

• Removal of any apid stingers

• Analgesics and antihistamines for local reactions

Stingers, if present, should be removed as quickly as possible. Suggested methods include scraping with a thin dull edge (eg, edge of a credit card, dull side of a scalpel, thin table knife).

Pain, burning, and itching can be reduced by placing an ice cube over the sting as soon as possible and giving oral H_1 blockers, NSAIDs, or both. Other possibly effective local measures include antihistamine lotion (eg, with diphenhydramine or tripelennamine), lidocaine patches, eutectic mixture of local anesthetic cream, intradermal injection of 1% lidocaine (with or without 1:100,000 epinephrine), and mid-potency corticosteroid creams or ointments (eg, triamcinolone 0.1%). Most folk remedies (eg, application of meat tenderizer) are of limited effectiveness.

Allergic reactions are treated with IV antihistamines; anaphylaxis is treated with parenteral epinephrine and IV fluids and vasopressors if necessary (see p. 1121).

People with known hypersensitivity to stings should carry a kit containing a prefilled syringe of epinephrine. They should use it as soon as possible after a sting and seek medical care immediately. People who have a history of anaphylaxis or a known allergy to insect bites should wear identification such as an alert bracelet.

Prevention

People who have had anaphylaxis are at risk from subsequent stings. Desensitization immunotherapy can be considered. Venom immunotherapy (see also p. 1116) is highly effective, reducing the chance of recurrent anaphylaxis from 50% to about 10% after 2 yr of therapy and to about 2% after 3 to 5 yr of therapy. Children who receive venom immunotherapy have a significantly lower risk of systemic reaction to stings 10 to 20 yr after treatment. Venom immunotherapy seems to be safe for use during pregnancy. Single-venom therapy is adequate. After initial immunotherapy, maintenance doses may be needed for up to 5 yr.

Puss Moth Caterpillar (ASP) Stings

Puss moth caterpillars (*Megalopyge opercularis*), of the order Lepidoptera, are also known as asps. They are one of the most toxic caterpillars in North America. Puss moth caterpillars are endemic to the southern US and live in shade trees and shrubbery around homes and schools and in parks. They are teardrop shaped and, because they have long silky hair, resemble a tuft of cotton or fur. Their color varies from yellow or gray to reddish brown. When a puss moth caterpillar rubs or is pressed against skin, venomous hairs become embedded.

Envenomation causes intense throbbing pain, burning, and a rash with erythematous spots. More susceptible patients can experience swelling, nausea, abdominal pain, headache, lymphadenopathy, lymphadenitis, shock, and respiratory distress. Wound pain usually subsides within an hour, and the erythematous spots disappear in a day.

Treatments for local reactions can include putting tape on the site and pulling it off to remove embedded hairs. Applying a baking soda slurry or calamine lotion can be soothing, and putting an ice pack on the site can ease pain. Treatment of systemic reactions is symptomatic. Treatment of severe reactions is like

that for insect stings.

Marine Bites and Stings

(For fish poisonings [eg, scombroid, ciguatera, fugu] and paralytic shellfish poisoning, see p. 3337.)

Some marine bites and stings are toxic; all create wounds at risk of infection with marine organisms, most notably *Vibrio* sp, *Aeromonas* sp, and *Mycobacterium marinum*.

Shark bites result in jagged lacerations with near-total or total amputations and should be treated in the same way as other major traumas (see p. 3190).

Cnidaria (Coelenterates)

Cnidaria include the following:

• Corals

• Sea anemones

• Jellyfish (including sea nettles)

• Hydroids (eg, Portuguese man-of-war)

Cnidaria are responsible for more envenomations than any other marine animal. However, of the 9000 species, only about 100 are toxic to humans. The multiple, highly developed stinging units (nematocysts) on cnidaria tentacles can penetrate human skin; one tentacle may fire thousands of nematocysts into the skin on contact.

Symptoms and Signs

Lesions vary with the type of cnidaria. Usually, lesions initially appear as small, linear, papular eruptions that develop rapidly in one or several discontinuous lines, at times surrounded by a raised erythematous zone. Pain is immediate and may be severe; itching is common. The papules may vesiculate and proceed to pustulation, hemorrhage, and desquamation. Systemic manifestations include weakness, nausea, headache, muscle pain and spasms, lacrimation and nasal discharge, increased perspiration, changes in pulse rate, and pleuritic chest pain. Uncommonly, fatal injuries have been inflicted by the Portuguese man-of-war in North American waters and by members of the Cubomedusae order, particularly the box jellyfish (sea wasp, *Chironex fleckeri*), in Indo-Pacific waters.

Treatment

• Removal of tentacles

• Symptomatic treatment

• Various rinses, depending on the specific animal

Cnidaria sting treatment includes removal of adherent tentacles with a forceps (preferably) or fingers (double-gloved if possible) and liberal rinsing to remove invisible stinging cells. The type of rinse varies by the stinging animal:

• For jellyfish stings sustained in nontropical waters and for coral stings, seawater rinse can be used.

• For jellyfish stings sustained in tropical waters, vinegar rinse followed by seawater rinse can be used. Fresh water should not be used because it can activate undischarged nematocysts.

• For box jellyfish stings, vinegar inhibits nematocyst firing and is used as the initial rinse if available,

followed by seawater rinse. Fresh water should not be used because it can activate undischarged nematocysts.

- For Portuguese man-of-war stings, saltwater rinse can be used. Vinegar should not be used because it can activate undischarged nematocysts.

Any difficulty breathing or alteration in level of consciousness, no matter how mild, is a medical emergency, requiring transport to a medical center and possibly injection of epinephrine.

Symptoms are treated supportively. Pain caused by sea nettle stings, usually short-lived, can be relieved with baking soda in a 50:50 slurry applied to the skin. For other stings, hot water or cold packs, whichever feels better, can help relieve pain, as can an NSAID or other analgesic. For severe pain, opioids are preferred. Painful muscle spasms may be treated with benzodiazepines. IV fluids and epinephrine can be given if shock develops. Antivenom is available for the stings of the box jellyfish *C. fleckeri* but not for the stings of North American species.

Seabather's eruption: This stinging, pruritic, maculopapular rash affects swimmers in some Atlantic locales (eg, Florida, Caribbean, Long Island). It is caused by hypersensitivity to stings from the larvae of the sea anemone (eg, *Edwardsiella lineate*) or the thimble jellyfish (*Linuche unguiculata*). The rash appears where the bathing suit contacts the skin. People exposed to these larvae should shower after taking off their bathing suit. Cutaneous manifestations can be treated with hydrocortisone lotion and, if needed, an oral antihistamine. More severe reactions may require the addition of oral or IV prednisone.

Stingrays

Stingrays once caused about 750 stings/yr along North American coasts; the present incidence is unknown, and most cases are not reported. Venom is contained in the one or more spines on the dorsum of the animal's tail. Injuries usually occur when an unwary swimmer wading in ocean surf, bay, or backwater steps on a stingray buried in the sand and provokes it to thrust its tail upward and forward, driving the dorsal spine (or spines) into the patient's foot or leg. The integumentary sheath surrounding the spine ruptures, and the venom escapes into the patient's tissues.

Symptoms and Signs

The main symptom is immediate severe pain. Although often limited to the injured area, the pain may spread rapidly, reaching its greatest intensity in < 90 min; in most cases, pain gradually diminishes over 6 to 48 h but occasionally lasts days or weeks. Syncope, weakness, nausea, and anxiety are common and may be due, in part, to peripheral vasodilation. Lymphangitis, vomiting, diarrhea, sweating, generalized cramps, inguinal or axillary pain, respiratory distress, and death have been reported.

The wound is usually jagged, bleeds freely, and is often contaminated with parts of the integumentary sheath. The edges of the wound are often discolored, and some localized tissue destruction may occur. Generally, some swelling and edema are present. Open wounds are subject to infection.

Treatment

- Irrigation and debridement

Injuries to an extremity should be gently irrigated with salt water in an attempt to remove fragments of spine, glandular tissue, and integument. The spine should be removed in the field only if it is superficially embedded and is not penetrating the neck, thorax, or abdomen or creating a through-and-through injury of a limb. Significant bleeding should be staunched with local pressure. Warm water immersion, although recommended by some experts, has not been verified as an effective early treatment for stingray injuries.

In the emergency department, the wound should be reexamined for remnants of the sheath and debrided; a local anesthetic may be given as needed. Embedded spines are treated similarly to other foreign bodies. Patients stung on the trunk should be evaluated closely for puncture of viscera. Treatment of systemic manifestations is supportive. Tetanus prophylaxis should be given, and an injured extremity

should be elevated for several days. Use of antibiotics and surgical wound closure may be necessary.

Mollusks

Mollusks include cones (including cone snails), cephalopods (including octopi and squids), and bivalves.

Conus californicus: This type is the only known dangerous cone in North American waters. Its sting causes localized pain, swelling, redness, and numbness that rarely progresses to paralysis or shock.

Treatment is largely supportive. Local measures seem to be of little value, and reports that local injection of epinephrine and neostigmine are helpful are unproved. Severe *Conus* stings may require mechanical ventilation and measures to reverse shock.

Cone snails: These snails are a rare cause of marine envenomation among divers and shell collectors in the Indian and Pacific Oceans. When the snail is aggressively handled (eg, during shell cleaning, when placed in a pocket), it injects its venom through a harpoon-like tooth. Multiple neurotoxins in the venom block ion channels and neurotransmitter receptors, resulting in paralysis, which is usually reversible but has resulted in some deaths.

Treatment is supportive and may include local pressure immobilization (eg, by wrapping wide crepe or other fabric bandages around the limb), immersion in hot water, and tetanus prophylaxis. Severe cases may require respiratory support.

Octopi: The bites of North American octopi are rarely serious.

Bites from the blue-ringed octopus, most common in Australian waters, cause tetrodotoxin envenomation, with local anesthesia, neuromuscular paralysis, and respiratory failure; treatment is supportive.

Squid: The large (up to 1.5 m), aggressive Humboldt squid is present off the west coast of the Americas; it has reportedly bitten fishermen and divers. Other squid species are of less concern.

Sea Urchins

Sea urchins are present worldwide. Most sea urchin injuries result when spines break off in the skin and cause local tissue reactions. Without treatment, the spines can migrate into deeper tissues, causing a granulomatous nodular lesion, or they may wedge against bone or nerve. Joint and muscle pain and dermatitis may also occur. A few sea urchins (eg, *Globiferous pedicellariae*) have calcareous jaws with venom organs, enabling them to inject venom, but injuries are rare.

Diagnosis is usually obvious by history. A bluish discoloration at the entry site may help locate the spine. X-rays can help when the location is not obvious during examination.

Treatment is immediate removal. Vinegar dissolves most superficial spines; soaking the wound in vinegar several times a day or applying a wet vinegar compress may be sufficient. Hot soaks may help relieve pain. Rarely, a small incision must be made to extract the spine; care must be taken because the spine is very fragile. A spine that has migrated into deeper tissues may require surgical removal. Once spines are removed, pain may continue for days; pain beyond 5 to 7 days should trigger suspicion of infection or a retained foreign body.

G. pedicellariae stings are treated by washing the area and applying a mentholated balm.

Mite Bites

There are multiple kinds of biting mites. Chiggers are probably the most common. Chiggers are mite larvae that are ubiquitous outdoors except in arid regions; they bite, feed in the skin, then fall off. Outside the US, chiggers may carry *Rickettsia tsutsugamushi* (see p. 1285). They do not burrow into the skin, but because they are small, they are not readily seen on the skin surface.

Common mite species that bite and burrow into the skin include *Sarcoptes scabiei*, which causes scabies (see p. 713), and *Demodex* mites, which cause a scabies-like dermatitis (sometimes referred to as mange) and are a possible etiologic agent in rosacea.

Dermatitis is caused by mites that occasionally bite humans but are ordinarily ectoparasites of birds, rodents, or pets and by mites associated with plant materials or stored food or feed.

- Bird mites may bite people who handle live poultry or pet birds or who have birds' nests on their homes.
- Rodent mites from cats, dogs (especially puppies), and rabbits may bite people.
- Swine mange mites (*S. scabiei var suis*) from pig farms or pet pigs may also bite humans.
- The straw itch mite (*Pyemotes tritici*) is often associated with seeds, straw, hay, and other plant material; it is a parasite of soft-bodied insects that are or have been present in such materials. These mites often bite people who handle the infested items. Granary workers, people who handle grass seeds or grass hay, and people who make dried plant arrangements are most at risk.

Allergic dermatitis or grocer's itch is caused by several species of mites associated with stored grain products, cheese, and other foods. These mites do not bite but cause allergic dermatitis because people become sensitized to allergens on the mites or their waste products.

House dust mites do not bite but feed on sloughed skin cells in pillows and mattresses and on floors (especially on carpets). They are significant because many people develop pulmonary hypersensitivity to allergens in the exoskeletons and feces of house dust mites.

Symptoms and Signs

Most bites cause some version of pruritic dermatitis; pruritus due to chigger bites is especially intense.

Diagnosis

- Clinical evaluation

Diagnosis of nonburrowing mite bites is presumptive based on the patient's history (eg, living, working, and recreational environments) and physical examination. The mites themselves are rarely found because they fall off after biting, the skin reaction is usually delayed, and most patients seek a physician's assistance only after several days. Lesions caused by different mites are usually indistinguishable and may superficially resemble other skin conditions (eg, other insect bites, contact dermatitis, folliculitis).

Diagnosis of burrowing mites can often be made presumptively based on history and a scabies-like pattern of skin lesions. If the diagnosis is unclear or if treatment is ineffective, the diagnosis can be confirmed by skin biopsy.

Treatment

- Topical corticosteroids or oral antihistamines
- Antimicrobial therapy for burrowing mites

Treatment of nonburrowing mite bites is symptomatic. Topical corticosteroids or oral antihistamines are used as needed to control pruritus until the hypersensitivity reaction resolves. Through discussion of possible sources, the physician can help patients avoid repeated exposure to mites. For *Demodex* bites, veterinary consultation is needed. For treatment of scabies, see p. 714.

Scorpion Stings

Although all scorpions in North America sting, most are relatively harmless. The stings usually cause only

localized pain with minimal swelling, some lymphangitis with regional lymphadenopathy, increased skin temperature, and tenderness around the wound.

A significant exception in North America is the bark scorpion (*Centruroides sculpturatus*, also known as *C. exilicauda*), present in Arizona, in New Mexico, and on the California side of the Colorado River. This species is venomous and can cause more serious injury and illness. Initial symptoms are immediate pain and sometimes numbness or tingling over the involved part. Swelling is usually absent, and there are few skin changes. Serious symptoms, most common among children, include restlessness; muscle spasms; abnormal and random head, neck, and eye movements; anxiety and agitation; and sialorrhea and diaphoresis. In adults, tachycardia, hypertension, increased respirations, weakness, muscle spasms, and fasciculations may predominate. Respiratory difficulties are rare in both age groups.

C. sculpturatus stings have resulted in death in children < 6 yr and in hypersensitive people.

Diagnosis

• Clinical evaluation

Diagnosis is obvious from the history. Determining the scorpion species is usually not. Several species of scorpions kept as exotic pets in the US (known by names that falsely suggest toxicity, such as yellow death stalker and black death scorpion) are similar in appearance to foreign species with dangerously toxic venom. However, the actual species of pet scorpion is seldom known by the patient or, if provided, may be unreliable. Stings should be treated as potentially dangerous until signs or lack of signs indicates otherwise.

Treatment

• Supportive care

• Antivenom for severe cases in North America

Treatment of nonvenomous scorpion stings is based on symptoms. An ice pack over the wound and oral NSAIDs reduce pain. Treatment of venomous *Centruroides* stings consists of bedrest, benzodiazepines for muscle spasms, and IV drugs as needed to control hypertension, agitation, and pain. Patients should be kept npo for 8 to 12 h after the bite. Antivenom, available only in Arizona, should be given in an ICU setting to all patients with severe cases and to patients who are unresponsive to supportive care, particularly children. Information about availability and dosing may be obtained by contacting a regional poison control center.

Snakebites

Of about 3000 snake species throughout the world, only about 15% worldwide and 20% in the US are dangerous to humans because of venom or toxic salivary secretions (see Table 339-1). At least one species of venomous snake is native to every state in the US except Alaska, Maine, and Hawaii. Almost all are crotalines (also called pit vipers because of pitlike depressions on either side of the head, which are heat-sensing organs):

• Rattlesnakes

• Copperheads

• Cottonmouths (water moccasins)

About 45,000 snakebites (of which 7000 to 8000 are venomous) occur annually in the US. Rattlesnakes account for the majority of bites and almost all deaths. Copperheads and, to a lesser extent, cottonmouths account for most other venomous bites. Coral snakes (elapids) and imported species (in zoos, schools, snake farms, and amateur and professional collections) account for < 1% of all bites. Most patients are males between 17 and 27 yr; 50% of them are intoxicated and deliberately handled or

molested the snake. Most bites occur on the upper extremities. Five or 6 deaths occur annually in the US. Risk factors for death include age extremes, handling of captive snakes (rather than wild encounters), delay in treatment, and undertreatment.

Outside the US, fatal snakebites are much more common, accounting for > 100,000 deaths yearly.

[Table 339-1. Significant Venomous Snakes by Region]

Pathophysiology

Snake venoms are complex substances, chiefly proteins, with enzymatic activity. Although enzymes play an important role, the lethal properties of venom are caused by certain smaller polypeptides. Most venom components appear to bind to multiple physiologic receptors, and attempts to classify venom as toxic to a specific system (eg, neurotoxin, hemotoxin, cardiotoxin, myotoxin) are misleading and can lead to errors in clinical judgment.

Pit vipers: The complex venom of most North American pit vipers has local effects as well as systemic effects such as coagulopathy. Effects may include

• Local tissue damage

• Vascular defects

• Hemolysis

• A disseminated intravascular coagulation (DIC)-like (defibrination) syndrome

• Pulmonary, cardiac, renal, and neurologic defects

Venom alters capillary membrane permeability, causing extravasation of electrolytes, albumin, and RBCs through vessel walls into the envenomated site. This process may occur in the lungs, myocardium, kidneys, peritoneum, and, rarely, the CNS. Common clinical syndromes secondary to severe pit viper envenomation include the following:

• **Edema:** Initially, edema, hypoalbuminemia, and hemoconcentration occur.

• **Hypovolemia:** Later, blood and fluids pool in the microcirculation, causing hypotension, lactic acidemia, shock, and, in severe cases, multisystem organ failure. Effective circulating blood volume falls and may contribute to cardiac and renal failure.

• **Bleeding:** Clinically significant thrombocytopenia (platelet count < 20,000/μL) is common in severe rattlesnake bites and may occur alone or with other coagulopathies. Venom-induced intravascular clotting may trigger DIC-like syndrome, resulting in bleeding.

• **Renal failure:** Renal failure may result from severe hypotension, hemolysis, rhabdomyolysis, nephrotoxic venom effects, or a DIC-like syndrome. Proteinuria, hemoglobinuria, and myoglobinuria may occur in reaction to severe rattlesnake bites.

The venom of most North American pit vipers causes very minor changes in neuromuscular conduction, except for Mojave and eastern diamondback rattlesnake venom, which may cause serious neurologic deficits.

Coral snakes: Venom of these snakes contains primarily neurotoxic components, which cause a presynaptic neuromuscular blockade, potentially causing respiratory paralysis. The lack of significant proteolytic enzyme activity accounts for the paucity of symptoms and signs at the bite site.

Symptoms and Signs

A snakebite, whether from a venomous or nonvenomous snake, usually causes terror, often with autonomic manifestations (eg, nausea, vomiting, tachycardia, diarrhea, diaphoresis), which may be difficult to distinguish from systemic manifestations of envenomation.

Nonvenomous snakebites cause only local injury, usually pain and 2 to 4 rows of scratches from the snake's upper jaw at the bite site.

Symptoms and signs of envenomation may be local, systemic, or a combination, depending on degree of envenomation and species of snake. Anaphylaxis can occur, particularly in snake handlers who have been previously sensitized.

Pit vipers: About 25% of pit viper bites are dry (venom is not deposited), and no systemic symptoms or signs develop.

Local signs include ≥ 1 fang marks and scratches. If envenomation has occurred, edema and erythema at the bite site and in adjacent tissues occur, usually within 30 to 60 min. Edema can progress rapidly and may involve the entire extremity within hours. Lymphangitis and enlarged, tender regional lymph nodes may develop; temperature increases over the bite area. In moderate or severe envenomations (see Diagnosis, below), ecchymosis is common and may appear at and around the bite site within 3 to 6 h. Ecchymosis is most severe after bites by eastern and western diamondbacks, cottonmouths, and prairie, Pacific, and timber rattlesnakes. Ecchymosis is less common after copperhead and Mojave rattlesnake bites. The skin around the bite may appear tense and discolored. Bullae—serous, hemorrhagic, or both—usually appear at the bite site within 8 h. Edema resulting from North American rattlesnake envenomations is usually limited to dermal and subcutaneous tissues, although severe envenomation rarely causes edema in subfascial tissue, causing compartment syndrome (defined as compartment pressures ≥ 30 mm Hg over 1 h). Necrosis around the bite site is common after rattlesnake envenomations. Most venom effects on soft tissues peak within 2 to 4 days.

Systemic manifestations of envenomation can include nausea, vomiting, diaphoresis, anxiety, confusion, spontaneous bleeding, fever, hypotension, and shock. Some patients with rattlesnake bites develop a rubbery, minty, or metallic taste in their mouth. The venom of most North American pit vipers causes minor neuromuscular conduction changes, including generalized weakness and paresthesias and muscle fasciculations. Some patients have alterations in mental status. Venom of Mojave and eastern diamondback rattlesnakes may cause serious neurologic deficits, including respiratory depression. Rattlesnake envenomations may induce various coagulation abnormalities, including thrombocytopenia, prolongation of PT (measured by the INR) or activated PTT, hypofibrinogenemia, elevated fibrin degradation products, or a combination of these disorders, resembling a DIC-like syndrome. Thrombocytopenia is usually the first manifestation and may be asymptomatic or, in the presence of a multicomponent coagulopathy, cause spontaneous bleeding. Patients with coagulopathy typically hemorrhage from the bite site or from venipuncture sites or mucous membranes, with epistaxis, gingival bleeding, hematemesis, hematochezia, hematuria, or a combination. A rise in Hct is an early finding secondary to edema and hemoconcentration. Later, Hct may fall as a result of fluid replacement and blood loss due to DIC-like syndrome. In severe cases, hemolysis may cause a rapid fall in Hct. Anaphylaxis can cause systemic symptoms immediately.

Coral snakes: Pain and swelling may be minimal or absent and are often transitory. The absence of local symptoms and signs may erroneously suggest a dry bite, producing a false sense of security for both patient and clinician. Weakness of the bitten extremity may become evident within several hours. Systemic neuromuscular manifestations may be delayed for 12 h and include weakness and lethargy; altered sensorium (eg, euphoria, drowsiness); cranial nerve palsies causing ptosis, diplopia, blurred vision, dysarthria, and dysphagia; increased salivation; muscle flaccidity; and respiratory distress or failure. Once the neurotoxic venom effects manifest, they are difficult to reverse and may last 3 to 6 days. Untreated, respiratory muscle paralysis may be fatal.

Diagnosis

• Identification of the snake

• Grading severity of envenomation

Definitive diagnosis requires positive identification of the snake and clinical manifestations of envenomation. History should include the time of bite, description of the snake, type of field therapy, underlying medical conditions, allergy to horse or sheep products, and history of previous venomous snakebites and therapy. A complete physical examination, including baseline measurements of limb circumference proximal and distal to the bite site, should be done.

Snakebites should be assumed to be venomous until proved otherwise by clear identification of the species or by a period of observation.

Snake identification: Patients often cannot recall details of the snake's appearance; however, pit vipers differ from nonvenomous snakes (see Fig. 339-1). Consultation with a zoo, an aquarium, or a poison control center can help in the identification of snake species.

Coral snakes in the US have round pupils and black snouts but lack facial pits. They have blunt or cigar-shaped heads and alternating bands of red, yellow (cream), and black, often causing them to be mistaken for the common nonvenomous scarlet king snake, which has alternating bands of red, black, and yellow. The distinguishing feature in the coral snake is that the red bands are adjacent to only yellow bands, not black bands. ("red on yellow, kill a fellow; red on black, venom lack"). Coral snakes have short, fixed fangs and inject venom through successive chewing movements.

Fang marks are suggestive but not conclusive; rattlesnakes may leave single or double fang marks or other teeth marks, whereas bites by nonvenomous snakes usually leave multiple superficial teeth marks. However, the number of teeth marks and bite sites may vary because snakes may strike and bite multiple times.

A dry pit viper bite is diagnosed when no symptoms or signs of envenomation appear over 8 h.

Severity of envenomation: Severity of envenomation depends on the following:

• Size and species of the snake (rattlesnakes > cottonmouths > copperheads)

• Amount of venom injected per bite (cannot be determined by history)

[**Fig. 339-1.** Identifying pit vipers.]

• Number of bites

• Location and depth of the bite (eg, envenomation in bites to the head and trunk tends to be more severe than in bites to the extremities)

• Age, size, and health of the patient

• Time elapsed before treatment

• Patient's susceptibility (response) to the venom

Severity of envenomation can be graded as minimal, moderate, or severe based on local findings, systemic symptoms and signs, coagulation parameters, and laboratory results (see Table 339-2). Grading should be determined by the most severe symptom, sign, or laboratory finding.

Envenomation may progress rapidly from minimal to severe and must be continually reassessed.

If systemic symptoms begin immediately, anaphylaxis should be assumed.

Treatment

• First aid

• Supportive care

• Antivenom

• Wound care

General approach: Treatment begins immediately, before patients are moved to a medical facility.

In the field, patients should move or be moved beyond the snake's striking distance. They should avoid exertion and be reassured, kept warm, and rapidly transported to the nearest medical facility. A bitten extremity should be loosely immobilized in a functional position just below heart level, and all rings, watches, and constrictive clothing should be removed. Pressure immobilization to delay systemic absorption of venom (eg, by wrapping wide crepe or other fabric bandages around the limb) may be appropriate for coral snake bites but is not recommended in the US, where most bites are from pit vipers; pressure immobilization may cause arterial insufficiency and necrosis. First responders should support airway and breathing, administer O_2, and establish IV access in an unaffected extremity while transporting patients. All other out-of-hospital interventions (eg, tourniquets, topical preparations, wound suction by mouth or with a device with or without incision, cryotherapy, electrical shock) are of no proven benefit, may be harmful, and may delay appropriate treatment. However, tourniquets that are already placed, unless causing limb-threatening ischemia, should remain in place until patients are transported to the hospital and envenomation is excluded or definitive treatment is initiated.

Serial assessment and testing begin in the emergency department. Extremity circumference should be measured on arrival and every 15 to 20 min until local progression subsides; outlining the margins of local edema with an indelible marker can help clinicians assess progression of local envenomation. All but trivial pit viper bites require a baseline CBC (including platelets), coagulation profile (eg, PT, PTT, fibrinogen), measurement of fibrin degradation products, and urinalysis, as well as measurement of serum electrolytes, BUN, and creatinine. For moderate and severe envenomations, patients require blood typing and cross-matching, ECG, chest x-ray, and CK tests, as governed by the patient's status, often as frequently as every 4 h for the first 12 h and then daily. In coral snake bites, neurotoxic venom effects require monitoring of O_2 saturation and baseline and serial pulmonary function tests (eg, peak flow, vital capacity).

[**Table 339-2.** Severity of Pit Viper Envenomation]

Duration of close observation for all patients with pit viper bites should be > 8 h in the emergency department or ICU. Patients without evidence of envenomation after 8 h may be sent home after adequate wound care (see p. 3319). Coral snake bite patients should be monitored for at least 12 h in an intensive care setting in case respiratory paralysis develops. Envenomation initially assessed as mild may progress to severe within several hours.

Supportive care may include respiratory support, benzodiazepines for anxiety and sedation, opioids for pain, and fluid replacement and vasopressor support for shock. Transfusions (eg, packed RBCs, fresh frozen plasma, cryoprecipitate, platelets) may be required but should not be given before patients have received adequate quantities of neutralizing antivenom because most coagulopathies respond to sufficient quantities of neutralizing antivenom. Suspected anaphylaxis (eg, with immediate onset of systemic symptoms) is treated with standard measures, including epinephrine. Tracheostomy may be needed if trismus, laryngeal spasm, or excessive salivation is present.

Antivenom: Along with aggressive supportive care, antivenom is the mainstay of treatment for patients with moderate to severe envenomation.

For pit viper envenomation, equine-derived antivenom has been largely replaced by ovine-derived Crotalidae polyvalent immune FAb antivenom (purified FAb fragments of IgG harvested from pit viper venom-immunized sheep). The effectiveness of the equine-derived antivenom is time and dose related; it

is most effective within 4 h after the envenomation and less effective after 12 h, although it may reverse coagulopathies after 24 h. Case reports suggest that Crotalidae polyvalent immune FAb may not be affected by time and dose and may be effective even when started 24 h after envenomation. Crotalidae polyvalent immune FAb is also safer than equine-derived antivenom, although it can still cause acute (cutaneous or anaphylactic) reactions and delayed hypersensitivity reactions (serum sickness). Serum sickness develops in up to 16% of patients 1 to 3 wk after administration of the FAb product. A loading dose of 4 to 6 vials of reconstituted Crotalidae polyvalent immune FAb diluted in 250 mL of normal saline should be infused slowly at 20 to 50 mL/h for the first 10 min; then if no adverse reactions occur, the remainder is infused over the next hour. The same dose can be repeated 2 times as needed to control symptoms, reverse coagulopathies, and correct physiologic parameters. In children, the dose is not decreased (eg, based on weight or size). Measuring the circumference of the involved extremity at 3 points proximal to the bite and measuring the advancing border of edema every 15 to 30 min can guide decisions about the need for additional doses. Once control is achieved, a 2-vial dose in 250 mL saline is given at 6, 12, and 18 h to prevent recurrence of limb swelling and other venom effects.

Pit viper species can affect dose. Cottonmouth envenomation may require smaller doses. Antivenom is usually unnecessary for copperhead and pygmy rattlesnake bites, except in children, the elderly, and patients with other medical conditions (eg, diabetes mellitus, coronary artery disease).

For **coral snake envenomation**, equine-derived polyvalent coral snake antivenom is given at a dose of 5 vials for suspected envenomation and an additional 10 to 15 vials if symptoms develop. Dose is similar for adults and children.

Equine-derived antivenom can cause hypersensitivity reactions and serum sickness. When equine-derived antivenom is required, skin testing for sensitivity to horse serum is controversial. Skin testing has no predictive value for development of acute hypersensitivity reactions, and a negative result does not completely preclude an immediate hypersensitivity reaction. However, if the skin test result is positive and the envenomation is considered life or limb threatening, H_1 and H_2 blockers should be given before antivenom administration in a critical care setting equipped to treat anaphylaxis. Early anaphylactoid reactions to antivenom are common and usually result from too-rapid infusion; treatment is temporary discontinuation of the infusion and treatment with epinephrine, H_1 and H_2 blockers, and IV fluid depending on severity. Usually, antivenom can be resumed after diluting the antivenom further and infusing it at a slower rate. Serum sickness is common and manifests 7 to 21 days after treatment as fever, rash, malaise, urticaria, arthralgia, and lymphadenopathy (see p. 1122). Treatment is H_1 blockers and a tapering course of oral corticosteroids.

Adjunctive measures: Patients should receive tetanus prophylaxis (toxoid and sometimes Ig) as suggested by history (see Table 140-1 on p. 1299). Snakebites rarely become infected, and antibiotics are indicated only for clinical evidence of infection. If necessary, options include a 1st-generation cephalosporin (eg, oral cephalexin, IV cefazolin) or a broad-spectrum penicillin (eg, oral amoxicillin/clavulanate, IV ampicillin/sulbactam). Subsequent antibiotic choices should be based on culture and sensitivity results from wound cultures.

Wound care for bites is similar to that for other puncture wounds. The area is cleaned and dressed. For limb bites, the extremity is splinted in a functional position and elevated. Wounds should be examined and cleaned daily and covered with a sterile dressing. Blebs, bloody vesicles, or superficial necrosis should be surgically debrided between days 3 and 10, in stages if needed. Sterile whirlpool may be indicated for wound debridement and physical therapy. Fasciotomy for compartment syndrome is rarely necessary but is an option when compartment pressure rises ≥ 30 mm Hg over 1 h, causes severe vascular compromise, and is unresponsive to limb elevation, mannitol 1 to 2 g/kg IV, and antivenom. Joint motion, muscle strength, sensation, and limb girth should be evaluated within 2 days after the bite. Contractures can be prevented by interrupting immobilization with frequent periods of gentle exercise, progressing from passive to active.

Regional poison control centers and zoos are excellent resources when dealing with snakebites, including those by nonnative snakes. These facilities maintain a list of physicians trained in snake identification and snakebite care as well as the Antivenin Index, published and periodically updated by the

American Zoo and Aquarium Association and the American Association of Poison Control Centers; this index catalogs the location and number of vials of antivenom available for all native venomous snakes and most exotic species. A national help line is available at 800-222-1222.

Other Reptile Bites

Other reptile bites of significance include those of venomous lizards, alligators and crocodiles, and iguanas.

Venomous lizards: These lizards include the following:

• Gila monster (*Heloderma suspectum*), present in the southwestern US and Mexico

• Beaded lizard (*H. horridum*) of Mexico

The complex venom of these lizards contains serotonin, arginine esterase, hyaluronidase, phospholipase A_2, and ≥ 1 salivary kallikreins but lacks neurotoxic components or coagulopathic enzymes. Bites are rarely fatal. When venomous lizards bite, they clamp on firmly and chew the venom into the person.

Symptoms and signs include intense pain, swelling, ecchymosis, lymphangitis, and lymphadenopathy. Systemic manifestations, including weakness, sweating, thirst, headache, and tinnitus, may develop in moderate or severe cases. Cardiovascular collapse occurs rarely. The clinical course is similar to that of a minimal to moderate envenomation by a larger species of rattlesnake.

Treatment in the field involves removing the lizard's jaws by using pliers, by applying a flame to the lizard's chin, or by immersing the animal entirely underwater. In a hospital, treatment is supportive and similar to that for pit viper envenomation; no antivenom is available. The wound should be probed with a small needle for broken or shed teeth and then cleaned. If the wound is deep, an x-ray can be taken to rule out a retained foreign body or bone fracture. Prophylactic antibiotics are usually not recommended.

Alligators and crocodiles: Bites usually result from handling; however, rarely, native encounters occur. Bites are not venomous, are notable for a high frequency of soft-tissue infections by *Aeromonas* sp, and are generally treated as major trauma.

Wounds are irrigated and debrided; then delayed primary closure is done or the wounds are allowed to heal by secondary intention. Patients are treated preventively with clindamycin and trimethoprim/sulfamethoxazole (first choice) or tetracycline.

Iguanas: Bites and claw injuries are becoming more frequent as more are kept as pets. Wounds are superficial, and treatment is local. Soft-tissue infection is uncommon, but when infection occurs, *Salmonella* is a common cause; infection can be treated with a fluoroquinolone.

Spider Bites

Almost all of the 30,000 species of spiders are venomous. However, the fangs of most species are too short or too fragile to penetrate the skin. Serious systemic reactions most frequently occur with bites from brown spiders (eg, violin, fiddleback, brown recluse—*Loxosceles* sp) and widow spiders (black widow—*Latrodectus* sp). Brown spiders are present in the Midwest and south central US, not in the coastal and Canadian border states, except when imported through clothing or luggage. Widow spiders are present throughout the US. Several venomous species (eg, *Pamphobeteus*, *Cupiennius*, *Phoneutria*) are not native to the US but may be imported on produce or other materials or through commercial trade in spiders as novelty pets. Spider bites cause < 3 deaths/yr in the US, usually in children.

Only a few spider venoms have been studied in detail. Of greatest significance are those having necrotizing venom components (in brown and some house spiders) and neurotoxic venom components (in widow spiders). The most toxic component of widow spider venom seems to be a peptide that affects neuromuscular transmission. The specific fraction of brown spider venom that causes the characteristic

necrotic lesion has not been isolated.

Symptoms and Signs

Brown spider bites are most common in the US. Some bites are painless initially, but pain, which can be severe and involve the entire extremity, develops within 30 to 60 min in all cases. The bite area becomes erythematous and ecchymotic and may be pruritic. Generalized pruritus may also be present. A central bleb forms at the bite site, often surrounded by an irregular ecchymotic area (bull's eye lesion). The lesion may mimic pyoderma gangrenosum. The central bleb becomes larger, fills with blood, ruptures, and leaves an ulcer; a black eschar forms over the ulcer and eventually sloughs.

Most bites leave minimal residual scarring but some can leave a large tissue defect, which may involve muscle. Loxoscelism, a venom-induced systemic syndrome, may not be detected until 24 to 72 h after the bite and is uncommon. Systemic effects (eg, fever, chills, nausea and vomiting, arthralgias, myalgias, generalized rash, seizures, hypotension, disseminated intravascular coagulation, thrombocytopenia, hemolysis, renal failure) are responsible for all reported fatalities.

Widow spider bites usually cause an immediate, sharp, stinging sensation. Within 1 h after envenomation, there may be progression to persistent local pain, diaphoresis, erythema, and piloerection at the bite site. The pain may be described as dull and numbing and may be disproportionate to the clinical signs. Latrodectism, a systemic syndrome caused by neurotoxic venom components, manifests as restlessness, anxiety, sweating, headache, dizziness, nausea and vomiting, hypertension, salivation, weakness, diffuse erythematous rash, pruritus, ptosis, eyelid and extremity edema, respiratory distress, increased skin temperature over the affected area, and cramping pain and muscular rigidity in the abdomen, shoulders, chest, and back. Abdominal pain may be severe and mimic acute surgical abdomen, rabies, or tetanus. Latrodectism is very uncommon and most commonly develops in patients at age extremes and those with other medical conditions. Death is extremely rare. Symptoms lessen over 1 to 3 days, but residual spasms, paresthesias, agitation, and weakness may persist for weeks to months.

Tarantula bites are extremely rare and nonvenomous, but agitation of the spider may cause it to throw needle-like hairs. The hairs act as foreign bodies in skin or eyes and can trigger mast cell degranulation and an anaphylactoid reaction (eg, urticaria, angioedema, bronchospasm, hypotension) in sensitized people, usually pet owners who handle the spider daily.

Diagnosis

• Clinical evaluation

• Careful consideration of alternative diagnoses

Spider bites are often falsely suspected by patients. Diagnosis is typically suspected based on history and physical signs, but confirmation is rare because it requires witnessed biting, identification of the spider (the spider is rarely recovered intact), and exclusion of other causes. In nonendemic areas, a brown spider bite should not be diagnosed without identifying the spider. Many patients incorrectly attribute much more common methicillin-resistant *Staphylococcus aureus* (MRSA) skin infections to brown recluse spiders bites. Such infections should be excluded, as should other conditions that mimic spider bites (see
Table 339-3). Severe cases of latrodectism should be distinguished from acute abdomen, rabies, or tetanus.

Spiders are identified by location and markings. Widow spiders live outdoors in protected spaces (eg, rock piles, firewood cords, hay bales, outhouses) and have a red or orange hourglass marking on the ventral abdomen. Brown spiders live indoors in protected spaces (eg, in clothing, behind furniture, under baseboards) and have a fiddle- or violin-like marking on the dorsal cephalothorax, ranging from the eyes to the abdomen. This marking may be difficult to recognize even in the intact spider.

Treatment

• Routine wound care

• Delayed excision for necrotic brown spider bites

• Ca gluconate and possibly antivenom for latrodectism

Treatment common to all spider bites includes wound cleaning, ice to reduce pain, extremity elevation, tetanus prophylaxis, and

[Table 339-3. Disorders that Mimic Spider Bites]

observation. Most local reactions respond to these measures alone.

For **brown spider bites**, limiting intervention to standard wound care and measures that minimize infection risk is usually most prudent:

• Ulcerating lesions should be cleaned daily and debrided as needed; topical antibiotic ointment (eg, polymyxin/bacitracin/neomycin) may be used.

• Urticarial lesions can be treated with antihistamines, topical corticosteroids, or both.

• Necrotic lesions caused by brown recluse spider bites should be cleaned and bandaged. Surgical excision, if necessary, should be delayed until the area of necrosis is fully demarcated, a process that may take weeks.

No intervention has been proved to reduce morbidity or improve outcome after a brown spider bite. Commonly touted or poorly studied treatment options are controversial or potentially harmful. Dapsone (eg, 100 mg po once/day until inflammation subsides) is often considered for ulcers > 2 cm, but its benefit is unproved. Benefit is variable, and dose-related hemolysis almost always develops; agranulocytosis, aplastic anemia, and methemoglobinemia have been documented. Local injection of corticosteroids into necrotic lesions has no value.

Latrodectism is initially treated supportively. Myalgias and muscle spasms due to widow spider bites respond poorly to muscle relaxants and opioid analgesics. A 10% Ca gluconate bolus given slowly IV in increments of 2 to 3 mL as needed may relieve pain briefly but requires continuous cardiac monitoring. Patients < 16 yr or > 60 yr, those with hypertension, and those with symptoms of severe envenomation should be hospitalized. Equine-derived antivenom is available for patients with severe latrodectism. It may be considered early in the course if symptoms are severe but can be effective up to 36 h after the bite. Clinical response can be dramatic. The dose for children and adults is 1 vial (6000 units) IV in 10 to 50 mL of normal saline usually over 3 to 15 min. The manufacturer recommends skin testing before administering the antivenom; however, skin testing does not always predict adverse reactions such as acute anaphylaxis.

Tick Bites

(See also Lyme Disease on p. 1268. Ch. 139 on p. 1279, and Babesiosis on p. 1375.)

Most tick bites in the US are from various species of Ixodidae, which attach and feed for several days if not removed. Disease transmission becomes more likely if ticks are attached for a longer duration.

Tick bites most often occur in spring and summer and are painless. The vast majority are uncomplicated and do not transmit disease; they often cause a red papule at the bite site and may induce hypersensitivity or granulomatous foreign body reactions. The bites of *Ornithodoros coriaceus* ticks (pajaroello) cause local vesiculation, pustulation with rupture, ulceration, and eschar, with varying degrees of local swelling and pain. Similar reactions have resulted from bites of other ticks.

Diagnosis

Diagnosis is by clinical evaluation and identification of the attached tick.

Treatment

• Tick removal with blunt, curved forceps

Ticks should be removed as soon as possible to reduce the cutaneous immune response and the likelihood of disease transmission. If the patient presents with the tick still attached, the best method of extracting the tick and all of its mouth parts from the skin is by using a blunt forceps with medium-sized, curved tips. The forceps should be placed parallel to the skin to grasp the tick's mouth parts firmly as close to the skin as possible. Care should be taken to avoid puncturing the patient's skin and the tick's body. The forceps should be pulled slowly and steadily, directly away from the skin without twisting. Curved-tip forceps are best because the outer curve can be laid against the skin while the handle remains far enough from the skin to grasp easily. Tick mouth parts that remain in the skin and are readily visible should be carefully removed. However, if the presence of mouth parts is questionable, attempts at surgical removal may cause more tissue trauma than would occur if the parts are left in the skin; leaving mouth parts in the skin does not affect disease transmission and, at most, prolongs irritation. Other methods of tick removal, such as burning it with a match (which can damage the patient's tissues) or covering it with petroleum jelly (which is ineffective), are not recommended.

After tick removal, an antiseptic should be applied. If local swelling and discoloration are present, an oral antihistamine may be helpful. Although rarely practical, the tick may be saved for laboratory analysis to check for etiologic agents of tick-borne disease in the geographic area where the patient acquired the tick.

A single dose of doxycycline (200 mg for adults and 4 mg/kg to a maximum of 200 mg for children ≥ 8 yr) should be considered when all of the following criteria are met:

• The tick is an adult or nymphal *Ixodes scapularis*.

• The tick is estimated to have been attached for ≥ 36 h based on degree of engorgement or certainty about time of exposure.

• Prophylaxis can be started within 72 h after the tick was removed.

• The local rate of infection of ticks with *Borrelia burgdorferi* is ≥ 20%.

• Doxycycline is not contraindicated.

Pajaroello tick lesions should be cleaned, soaked in 1:20 Burow's solution, and debrided when necessary. Corticosteroids are helpful in severe cases. Infections are common during the ulcer stage but rarely require more than local antiseptic measures.

Tick Paralysis

Tick paralysis is a rare, ascending, flaccid paralysis that occurs when toxin-secreting Ixodidae ticks bite and remain attached for several days.

In North America, some species of *Dermacentor* and *Amblyomma* cause tick paralysis due to a neurotoxin secreted in tick saliva. The toxin is not present in tick saliva during early stages of feeding, so paralysis occurs only when a tick has fed for several days or more. A single tick can cause paralysis, especially if it is attached to the back of the skull or near the spine, but multiple ticks should be sought over the entire body surface.

Symptoms and signs include anorexia, lethargy, muscle weakness, impaired coordination, nystagmus, and ascending flaccid paralysis. Bulbar or respiratory paralysis may develop.

Tick paralysis should be considered in North American patients with acute ascending flaccid paralysis or

bulbar paralysis. Differential diagnosis includes Guillain-Barre syndrome, botulism, myasthenia gravis, hypokalemia, and spinal cord tumor.

The paralysis is rapidly reversible with removal of the tick or ticks. If breathing is impaired, O_2 therapy or respiratory assistance may be needed.

Other Arthropod Bites

The more common biting non-tick arthropods in the US include sand flies, horseflies, deerflies, blackflies, stable flies, mosquitoes, fleas, kissing bugs, lice (see p. 711), bedbugs, wheel bugs, and certain water bugs. All of these arthropods, except wheel bugs and water bugs, also suck blood, but none are venomous.

Arthropod saliva composition varies considerably, and the lesions caused by bites vary from small papules to large ulcers with swelling and acute pain. Dermatitis may also occur. Most serious consequences result from hypersensitivity reactions or infection; in sensitized people, they can be fatal. Flea allergens may trigger respiratory allergy even without a bite in some people.

The location and pattern of wheals and lesions is sometimes diagnostic of the bite source. For example, blackfly bites are usually on the neck, ears, and face; flea bites may be numerous, mostly on the feet and legs; and bedbug bites often occur in linear patterns, most commonly on the torso.

The bite should be cleaned, and an antihistamine or corticosteroid cream or ointment should be applied for itching. Severe hypersensitivity reactions should be treated (see p. 1120).

Chapter 340. Poisoning

Introduction

Accidental poisoning and intentional self-poisoning result in many emergency department visits and a few deaths. (For effects of alcohol and illicit drug use, see Ch. 160.)

General Principles of Poisoning

Poisoning is contact with a substance that results in toxicity. Symptoms vary, but certain common syndromes may suggest particular classes of poisons. Diagnosis is primarily clinical, but for some poisonings, blood and urine tests can help. Treatment is supportive for most poisonings; specific antidotes are necessary for a few. Prevention includes labeling drug containers clearly and keeping poisons out of the reach of children.

Most poisonings are dose-related. Toxicity may result from exposure to excess amounts of normally nontoxic substances. Some poisonings result from exposure to substances that are poisonous at all doses. Poisoning is distinguished from hypersensitivity and idiosyncratic reactions, which are unpredictable and not dose-related, and from intolerance, which is a toxic reaction to a usually nontoxic dose of a substance.

Poisoning is commonly due to ingestion but can result from injection, inhalation, or exposure of body surfaces (eg, skin, eye, mucous membranes). Many commonly ingested non-food substances are generally nontoxic (see Table 340-1); however, almost any substance can be toxic if ingested in excessive amounts.

Accidental poisoning is common among young children, who are curious and ingest items indiscriminately despite noxious tastes and odors; usually, only a single substance is involved. Poisoning is also common among older children, adolescents, and adults attempting suicide; multiple drugs, including alcohol, acetaminophen, and other OTC drugs, may be involved. Accidental poisoning may occur

[**Table 340-1.** Substances Usually not Dangerous when Ingested*]

in the elderly because of confusion, poor eyesight, mental impairment, or multiple prescriptions of the same drug by different physicians.

Occasionally, people are poisoned by someone who intends to kill or disable them (eg, to rape or rob them). Drugs used to disable (eg, scopolamine, benzodiazepines, γ-hydroxy-butyrate) tend to have sedative or amnestic properties or both. Rarely, parents, who may have some medical knowledge, poison their children because of unclear psychiatric reasons or a desire to cause illness and thus gain medical attention (a disorder called Munchausen syndrome by proxy—see p. 1576).

Most poisons are metabolized, pass through the GI tract, or are excreted. Occasionally, tablets (eg, aspirin, iron, enteric-coated drugs) form large concretions (bezoars) in the GI tract, where they tend to remain, continuing to be absorbed and causing toxicity.

Symptoms and Signs

Symptoms and signs vary depending on the substance (see Table 340-8 on p. 3345). Also, different patients poisoned with the same substance may present with very different symptoms. However, 6 clusters of symptoms (toxic syndromes, or toxidromes) occur commonly and may suggest particular classes of substances (see Table 340-2). Patients who ingest multiple substances are less likely to have symptoms characteristic of a single substance.

Symptoms typically begin soon after contact but, with certain poisons, are delayed. The delay may occur because only a metabolite is toxic rather than the parent substance (eg, methanol, ethylene glycol, hepatotoxins). Ingestion of hepatotoxins (eg, acetaminophen, iron, *Amanita phalloides* mushrooms) may

cause acute liver failure that occurs one to a few days later. With metals or hydrocarbon solvents, symptoms typically occur only after chronic exposure to the toxin.

Ingested toxins generally cause systemic symptoms. Caustics and corrosive liquids damage mainly the mucous membranes of the GI tract, causing stomatitis, enteritis, or perforation. Some toxins (eg, alcohol, hydrocarbons) cause characteristic breath odors. Skin contact with toxins can cause various acute cutaneous symptoms (eg, rashes, pain, blistering); chronic exposure may cause dermatitis. Inhaled toxins are likely to cause symptoms of upper airway injury if they are water-soluble and symptoms of lower airway (lung parenchyma) injury and noncardiogenic pulmonary edema if they are less water-soluble. Inhalation of carbon monoxide, cyanide, or hydrogen sulfide gas can cause organ ischemia or cardiac or respiratory arrest. Eye contact with toxins (solid, liquid, or vapor) may damage the cornea and lens, causing eye pain, redness, and loss of vision.

Some substances (eg, cocaine, phencyclidine, amphetamine) can cause severe agitation, which can result in hyperthermia, acidosis, and rhabdomyolysis.

Diagnosis

• Consideration of poisoning in patients with altered consciousness or unexplained symptoms

• History from all available sources

• Selective, directed testing

The first step of diagnosis is to assess the overall status of the patient. Severe poisoning may require rapid intervention to treat cardiopulmonary collapse.

Poisoning may be known at presentation. It should be suspected if patients have unexplained symptoms, especially altered consciousness. If purposeful self-poisoning occurs in adults, multiple substances should be suspected.

History is often the most valuable tool. Because many patients (eg, preverbal children, suicidal or psychotic adults, patients with altered consciousness) cannot provide reliable information, friends, relatives, and rescue personnel should be questioned. Even seemingly reliable patients may incorrectly report the amount or time of ingestion. When possible, the patient's living quarters should be inspected for clues (eg, partially empty pill containers, evidence of recreational drug use). Pharmacy and medical records may provide useful information. In potential workplace poisonings, coworkers and supervisors should be questioned. All industrial chemicals must have a material safety data sheet (MSDS) readily available at the workplace; the MSDS provides detailed information about toxicity and any specific treatment.

In the US, Europe, and parts of Asia and South America, information about household and industrial chemicals can be obtained from poison control centers. Consultation with the centers is encouraged because ingredients, first-aid measures, and antidotes printed on product containers are occasionally inaccurate or outdated. Also, the container may have been replaced, or the package may have been tampered with. Poison control centers may be able to help identify unknown pills based on their appearance. The centers have ready access to toxicologists. The telephone number of the nearest center is often listed with other emergency numbers in the front of the local

[**Table 340-2.** Common Toxic Syndromes (Toxidromes)]

telephone book; the number is also available from the telephone operator or, in the US, by dialing 1-800-222-1222. More information is available at the American Association of Poison Control Centers web site (www.aapcc.org).

Physical examination sometimes detects signs suggesting particular types of substances (eg, toxidromes, breath odor, needle tracks suggesting IV drug use, stigmas of chronic alcohol use).

Even if a patient is known to be poisoned, altered consciousness may have other causes (eg, CNS infection, head trauma, hypoglycemia, stroke, hepatic encephalopathy, Wernicke's encephalopathy), which should also be considered. Attempted suicide must always be considered in older children, adolescents, and adults who have ingested a drug. After such patients are stabilized, psychiatric intervention should be considered.

Testing: In most cases, laboratory testing provides limited help. Standard, readily available tests to identify common drugs of abuse (often called toxic screens) are qualitative, not quantitative. These tests may provide false results and they check for only a limited number of substances. Also, the presence of a drug of abuse does not necessarily indicate that the drug caused the patient's symptoms or signs. Urine drug screening is used most often but has limited value and usually detects classes of drugs or metabolites rather than specific drugs. For example, an opioid urine immunoassay test does not detect fentanyl or methadone but does react with very small amounts of morphine or codeine analogues. The test used to identify cocaine detects a metabolite rather than cocaine itself.

For most substances, blood levels cannot be easily determined or do not help guide treatment. For a few substances (eg, acetaminophen, aspirin, carbon monoxide, digoxin, ethylene glycol, iron, lithium, methanol, phenobarbital, phenytoin, theophylline), blood levels may help guide treatment. Many authorities recommend measuring acetaminophen levels in all patients with mixed ingestions because acetaminophen ingestion is common, is often asymptomatic during the early stages, and can cause serious delayed toxicity that can be prevented by an antidote. For some substances, other blood tests (eg, PT for warfarin overdose, methemoglobin levels for certain substances) may help guide treatment. For patients who have altered consciousness or abnormal vital signs or who have ingested certain substances, tests should include serum electrolytes, BUN, creatinine, osmolality, glucose, and ABGs. Other tests may be indicated for specific substances.

For certain poisonings (eg, due to iron, lead, arsenic, other metals, or to packets of cocaine or other illicit drugs ingested by so-called body packers), plain abdominal x-rays may show the presence and location of ingested substances.

For poisonings with drugs that have cardiovascular effects or with an unknown substance, ECG and cardiac monitoring are indicated.

If blood levels of a substance or symptoms of toxicity increase after initially decreasing or persist for an unusually long time, a bezoar, a sustained-release preparation, or reexposure (ie, repeated covert exposure to a recreationally used drug) should be suspected.

Treatment

• Supportive care

• Activated charcoal for serious oral poisonings

• Occasional use of specific antidotes or dialysis

• Only rare use of gastric emptying

Seriously poisoned patients may require assisted ventilation or treatment of cardiovascular collapse. Those with impaired consciousness may require continuous monitoring or restraints. The discussion of treatment for specific poisonings, below and in
Tables 340-3,
340-4, and 340-8, is general and does not include specific complexities and details. Consultation with a poison control center is recommended for any poisonings except the mildest and most routine.

Initial stabilization: Treatment of any systemic poisoning begins with airway, breathing, and circulatory stabilization (see p. 2256).

If patients have apnea or compromised airways (eg, foreign material in the oropharynx, decreased gag

reflex), an endotracheal tube should be inserted. If patients have respiratory depression or hypoxia, supplemental O_2 or mechanical ventilation should be provided as needed.

In patients with apnea, IV naloxone (2 mg in adults; 0.1 mg/kg in children) should be tried while airway support is maintained. In opioid addicts, naloxone may precipitate withdrawal, but withdrawal is preferable to apnea. If respiratory depression persists despite use of naloxone, endotracheal intubation and continuous mechanical ventilation are required. If naloxone relieves respiratory depression, patients are monitored; if respiratory depression recurs, patients can be treated with another bolus of IV naloxone or mechanical ventilation. Using a low-dose continuous naloxone infusion to maintain respiratory drive without precipitating withdrawal has been suggested but in reality is very difficult to accomplish.

If patients have altered consciousness, blood glucose should be measured immediately at bedside, or IV dextrose (50 mL of a 50% solution for adults; 2 to 4 mL/kg of a 25% solution for children) should be given empirically. For adults with suspected thiamin deficiency (eg, alcoholics, undernourished patients), thiamin 100 mg IV is given with or before glucose.

Hypotension is treated with IV fluids. If fluids are ineffective, invasive hemodynamic monitoring may be necessary to guide fluid and vasopressor therapy. The first-choice vasopressor for most poison-induced hypotension is norepinephrine 0.5 to 1 mg/min IV infusion, but treatment should not be delayed if another vasopressor is more immediately available.

Topical decontamination: Any body surface (including the eyes) exposed to a toxin is

[Table 340-3. Common Specific Antidotes]

flushed with large amounts of water or saline. Contaminated clothing, including shoes and socks, and jewelry should be removed.

Activated charcoal: Charcoal is usually given, particularly when multiple or unknown substances have been ingested. Use of charcoal adds little risk, unless patients are at risk of vomiting and aspiration, but has not been proved to reduce overall morbidity or mortality. When used, charcoal is given as soon as possible. Activated charcoal adsorbs most toxins because of its molecular configuration and large surface area. Multiple doses of activated charcoal may be effective for substances that undergo enterohepatic recirculation (eg, phenobarbital, theophylline) and for sustained-release preparations. Charcoal may be given at 4- to 6-h intervals for serious poisoning with such substances unless bowel sounds are hypoactive. Charcoal is ineffective for caustics, alcohols, and simple ions (eg, cyanide, iron, other metals, lithium). The recommended dose is 5 to 10 times that of the suspected toxin ingested. However, because the amount of toxin ingested is usually unknown, the usual dose is 1 to 2 g/kg, which is about 10 to 25 g for children < 5 yr and 50 to 100 g for older children and adults. Charcoal is given as a slurry in water or soft drinks. It may be unpalatable and results in vomiting in 30% of patients. Administration via a gastric tube may be considered, but caution should be used to prevent trauma caused by tube insertion or aspiration of charcoal. Activated charcoal should probably be used without sorbitol or other cathartics, which have no clear benefit and can cause dehydration and electrolyte abnormalities.

Gastric emptying: Gastric emptying, which used to be well-accepted and seems intuitively beneficial, is not routinely done. It does not clearly reduce overall morbidity or mortality and has risks. Gastric emptying is considered if it can be done within 1 h of a life-threatening ingestion. However, many poisonings manifest too late, and whether a poisoning is life threatening is not always clear. Thus, gastric emptying is seldom indicated and, if a caustic substance has been ingested, is contraindicated (see p. 3335).

If gastric emptying is used, gastric lavage is the preferred method. Gastric lavage may cause complications such as epistaxis, aspiration, or, rarely, oropharyngeal or esophageal injury. Syrup of ipecac has unpredictable effects, often causes prolonged vomiting, and may not remove substantial amounts of poison from the stomach. Syrup of ipecac may be warranted if the ingested agent is highly toxic and transport time to the emergency department is unusually long, but this is uncommon in the US.

For gastric lavage, tap water is instilled and withdrawn from the stomach with a tube. The largest tube possible (usually > 36 French for adults or 24 French for children) is used so that tablet fragments can be

retrieved. If patients

[**Table 340-4.** Guidelines for Chelation Therapy]

have altered consciousness or a weak gag reflex, endotracheal intubation should be done before lavage to prevent aspiration. Patients are placed in the left lateral decubitus position to prevent aspiration, and the tube is inserted orally. Because lavage sometimes forces substances farther into the GI tract, a 25-g dose of charcoal is instilled through the tube first. Then aliquots (about 3 mL/kg) of tap water are instilled, and the gastric contents are withdrawn by gravity or syringe. Lavage continues until the withdrawn fluids appear free of the substance; usually, 500 to 3000 mL of fluid must be instilled. After lavage, a 2nd 25-g dose of charcoal is instilled.

Whole-bowel irrigation: This procedure flushes the GI tract and theoretically decreases GI transit time for pills and tablets. Irrigation has not been proved to reduce morbidity or mortality. Irrigation is indicated for any of the following:

- Some serious poisonings due to sustained-release preparations or substances that are not adsorbed by charcoal (eg, heavy metals)
- Drug packets (eg, latex-coated packets of heroin or cocaine ingested by body packers)
- A suspected bezoar

A commercially prepared solution of polyethylene glycol (which is nonabsorbable) and electrolytes is given at a rate of 1 to 2 L/h for adults or at 25 to 40 mL/kg/h for children until the rectal effluent is clear; this process may require many hours or even days. The solution is usually given via a gastric tube, although some motivated patients can drink these large volumes.

Alkaline diuresis: Alkaline diuresis enhances elimination of weak acids (eg, salicylates, phenobarbital). A solution made by combining 1 L of 5% D/W with 3 50-mEq ampules of $NaHCO_3$ and 20 to 40 mEq of K can be given at a rate of 250 mL/h in adults and 2 to 3 mL/kg/h in children. Urine pH is kept at > 8. Hypernatremia, alkalemia, and fluid overload may occur but are usually not serious. However, alkaline diuresis is contraindicated in patients with renal insufficiency.

Dialysis: Common toxins that may require dialysis or hemoperfusion include

- Ethylene glycol
- Lithium
- Methanol
- Salicylates
- Theophylline

These therapies are less useful if the poison is a large or charged (polar) molecule, has a large volume of distribution (ie, if it is stored in fatty tissue), or is extensively bound to tissue protein (as with digoxin, phencyclidine, phenothiazines, or tricyclic antidepressants). The need for dialysis is usually determined by both laboratory values and clinical status. Methods of dialysis include hemodialysis, peritoneal dialysis, and lipid dialysis (which removes lipid-soluble substances from the blood), as well as hemoperfusion (which more rapidly and efficiently clears specific poisons—see Ch. 240).

Specific antidotes: For the most commonly used antidotes, see Table 340-3. Chelating drugs are used for poisoning with heavy metals and occasionally with other drugs (see Table 340-4). IV fat emulsions have been used in 10% and 20% concentrations to successfully treat several different cardiac toxins (eg, bupivacaine, verapamil).

Ongoing supportive measures: Most symptoms (eg, agitation, sedation, coma, cerebral edema, hypertension, arrhythmias, renal failure, hypoglycemia) are treated with the usual supportive measures (see elsewhere in THE MANUAL).

Drug-induced hypotension and arrhythmias may not respond to the usual drug treatments. For refractory hypotension, dopamine, epinephrine, other vasopressors, an intra-aortic balloon pump, or even extracorporeal circulatory support may be considered.

For refractory arrhythmias, cardiac pacing may be necessary. Often, torsades de pointes can be treated with Mg sulfate 2 to 4 g IV, overdrive pacing, or a titrated isoproterenol infusion.

Seizures are first treated with benzodiazepines. Phenobarbital or phenytoin can also be used. Severe agitation must be controlled; benzodiazepines in large doses, other potent sedatives (eg, propofol), or, in extreme cases, induction of paralysis and mechanical ventilation may be required.

Hyperthermia is treated with aggressive sedation and physical cooling measures rather than with antipyretics. Organ failure may ultimately require kidney or liver transplantation.

Hospital admission: General indications for hospital admission include altered consciousness, persistently abnormal vital signs, and predicted delayed toxicity. For example, admission is considered if patients have ingested sustained-release preparations, particularly of drugs with potentially serious effects (eg, cardiovascular drugs). If there are no other reasons for admission and if symptoms are gone after patients have been observed for 4 to 6 h, most patients can be discharged. However, if ingestion was intentional, patients require a psychiatric evaluation.

Prevention

In the US, widespread use of child-resistant containers with safety caps has greatly reduced the number of poisoning deaths in children < 5 yr. Limiting the amount of OTC analgesics in a single container reduces the severity of poisonings, particularly with acetaminophen, aspirin, or ibuprofen. Preventive measures also include clearly labeling household products and prescription drugs, storing drugs and toxic substances in cabinets that are locked and inaccessible to children, promptly disposing of expired drugs by mixing them in cat litter or some other nontempting substance and putting them in a trash container that is inaccessible to children, and using carbon monoxide detectors. Public education measures to encourage storage of substances in their original containers (eg, not placing insecticide in drink bottles) are important. Use of imprint identifications on solid drugs helps prevent confusion and errors by patients, pharmacists, and health care practitioners.

Acetaminophen Poisoning

Acetaminophen poisoning can cause gastroenteritis within hours and hepatotoxicity 1 to 3 days after ingestion. Severity of hepatotoxicity after a single acute overdose is predicted by serum acetaminophen levels. Treatment is with *N*-acetylcysteine to prevent or minimize hepatotoxicity.

Acetaminophen is contained in > 100 products sold OTC. Products include many children's preparations in liquid, tablet, and capsule form and many cough and cold preparations. Many prescription drugs also contain acetaminophen. Consequently, acetaminophen overdose is common.

Pathophysiology

The principal toxic metabolite of acetaminophen, *N*-acetyl-*p*-benzoquinone imine (NAPQI), is produced by the hepatic cytochrome P-450 enzyme system; glutathione stores in the liver detoxify this metabolite. An acute overdose depletes glutathione stores in the liver. As a result, NAPQI accumulates, causing hepatocellular necrosis and possibly damage to other organs (eg, kidneys, pancreas). Theoretically, alcoholic liver disease or undernutrition could increase risk of toxicity because hepatic enzyme preconditioning may increase formation of NAPQI and because undernutrition (also common among alcoholics) reduces hepatic glutathione stores. However, whether the risk is actually increased is unclear.

Acute alcohol ingestion may be protective because hepatic P-450 enzymes preferentially metabolize ethanol and thus cannot produce toxic NAPQI.

Acute Acetaminophen Poisoning

To cause toxicity, an acute overdose must total ≥ 150 mg/kg (about 7.5 g in adults) within 24 h.

Symptoms and Signs

Mild poisoning may not cause symptoms, and when present, symptoms are usually minor until ≥ 48 h after ingestion. Symptoms, which occur in 4 stages (see Table 340-5), include anorexia, nausea, vomiting, and right upper quadrant abdominal pain. Renal failure and pancreatitis may occur, occasionally without liver failure. After > 5 days, hepatotoxicity resolves or progresses to multiple organ failure, which can be fatal.

Diagnosis

• Diagnosis considered in all patients with nonaccidental ingestions

• Serum acetaminophen levels

• Rumack-Matthew nomogram

Acetaminophen overdose should be considered in all patients with nonaccidental ingestions that may be suicide attempts because formulations containing acetaminophen are frequently ingested in such overdoses and are not reported. Also, acetaminophen often causes minimal symptoms during the early stages and is potentially lethal but treatable.

Likelihood and severity of hepatotoxicity caused by an acute ingestion can be predicted by the amount ingested or, more accurately, by the serum acetaminophen level. If the time of ingestion is known, the Rumack-Matthew nomogram (see Fig. 340-1) is used to estimate likelihood of hepatotoxicity; if the time of ingestion is unknown, the nomogram cannot be used. For a single acute overdose of traditional acetaminophen or rapid-relief acetaminophen (which is absorbed 7 to 8 min faster), levels are measured ≥ 4 h after ingestion and plotted on the nomogram. A level ≤ 150 µg/mL (≤ 990 µmol/L) and absence of toxic symptoms indicate that hepatotoxicity is very

[**Table 340-5.** Stages of Acute Acetaminophen Poisoning]

[**Fig. 340-1.** Rumack-Matthew nomogram for single acute acetaminophen poisoning.]

unlikely. Higher levels indicate possible hepatotoxicity. For a single acute overdose with extended-relief acetaminophen (which has 2 peak serum levels about 4 h apart), acetaminophen levels are measured ≥ 4 h after ingestion and 4 h later; if either level is above the Rumack-Matthew line of toxicity, treatment is required.

If poisoning is confirmed or strongly suspected, other tests are done. Liver function tests are done and, in suspected severe poisoning, PT is measured. AST and ALT results correlate with the stage of poisoning (see Table 340-5). AST levels > 1000 IU/L are more likely to result from acetaminophen poisoning than from chronic hepatitis or alcoholic liver disease. If poisoning is severe, bilirubin and INR may be elevated.

Prognosis

With appropriate treatment, mortality is uncommon.

Poor prognostic indicators at 24 to 48 h postingestion include all of the following:

• pH < 7.3 after adequate resuscitation

• INR > 3

• Serum creatinine > 2.6

• Hepatic encephalopathy grade III (confusion and somnolence) or grade IV (stupor and coma)

• Hypoglycemia

• Thrombocytopenia

Acute acetaminophen toxicity does not predispose patients to cirrhosis.

Treatment

• Oral or IV *N*-acetylcysteine

• Possibly activated charcoal

Activated charcoal may be given if acetaminophen is likely to still remain in the GI tract.

N-Acetylcysteine is an antidote for acetaminophen poisoning. This drug is a glutathione precursor that decreases acetaminophen toxicity by increasing hepatic glutathione stores and possibly via other mechanisms. It helps prevent hepatic toxicity by inactivating the toxic acetaminophen metabolite NAPQI before it can injure liver cells. However, it does not reverse damage to liver cells that has already occurred.

For acute poisoning, *N*-acetylcysteine is given if hepatotoxicity is likely based on acetaminophen dose or serum level. The drug is most effective if given within 8 h of acetaminophen ingestion. After 24 h, the benefit of the antidote is questionable.

N-Acetylcysteine is equally effective given IV or orally. IV therapy is given as a continuous infusion. A loading dose of 150 mg/kg in 200 mL of 5% D/W given over 15 min is followed by maintenance doses of 50 mg/kg in 500 mL of 5% D/W given over 4 h, then 100 mg/kg in 1000 mL of 5% D/W given over 16 h. For children, dosing may need to be adjusted to decrease the total volume of fluid delivered; consultation with a poison control center is recommended.

The oral loading dose of *N*-acetylcysteine is 140 mg/kg. This dose is followed by 17 additional doses of 70 mg/kg q 4 h. Oral acetylcysteine is unpalatable; it is given diluted 1:4 in a carbonated beverage or fruit juice and may still cause vomiting. If vomiting occurs, an antiemetic can be used; if vomiting occurs within 1 h of a dose, the dose is repeated. However, vomiting may be protracted and may limit oral use. Allergic reactions are unusual but have occurred with oral and IV use.

Liver failure is treated supportively. Patients with fulminant liver failure may require liver transplantation.

Chronic Acetaminophen Poisoning

Chronic excessive use or repeated overdoses cause hepatotoxicity in a few patients. Usually, chronic overdose is not an attempt at self-injury but instead results from taking inappropriately high doses to treat pain. Symptoms may be absent or may include any of those that occur with acute overdose.

Diagnosis

• AST, ALT, and serum acetaminophen levels

The Rumack-Matthew nomogram cannot be used, but likelihood of clinically significant hepatotoxicity can be estimated based on AST, ALT, and serum acetaminophen levels.

- If AST and ALT levels are normal (< 50 IU/L) and the acetaminophen level is < 10 µg/mL, significant hepatotoxicity is very unlikely.

- If AST and ALT levels are normal but the acetaminophen level is ≥ 10 µg/mL, significant hepatotoxicity is possible; AST and ALT levels are remeasured after 24 h. If repeat AST and ALT levels are normal, significant hepatotoxicity is unlikely; if the levels are high, significant hepatotoxicity is assumed.

- If initial AST and ALT levels are high, regardless of the acetaminophen level, significant hepatotoxicity is assumed.

Treatment

- Sometimes *N*-acetylcysteine

The role of *N*-acetylcysteine in treatment of chronic acetaminophen toxicity or in the presence of established acute hepatotoxicity is unclear. Theoretically, the antidote may have some benefit if given > 24 h after an ingestion if residual (unmetabolized) acetaminophen is present. The following approach has not been proved effective but may be used:

- If hepatotoxicity is possible (if AST and ALT levels are normal and acetaminophen level is initially elevated), *N*-acetylcysteine is given 140 mg/kg po loading dose and 70 mg/kg po q 4 h for the first 24 h. If repeat AST and ALT levels (after 24 h) are normal, *N*-acetylcysteine is stopped; if repeat levels are high, they are remeasured daily, and *N*-acetylcysteine is continued until levels are normal.

- If hepatotoxicity is likely (especially if initial AST and ALT levels are high), a full course of *N*-acetylcysteine is given.

Prognostic factors are similar to those in acute acetaminophen poisoning.

Aspirin and Other Salicylate Poisoning

(Salicylism)

Salicylate poisoning can cause vomiting, tinnitus, confusion, hyperthermia, respiratory alkalosis, metabolic acidosis, and multiple organ failure. Diagnosis is clinical, supplemented by measurement of the anion gap, ABGs, and serum salicylate levels. Treatment is with activated charcoal and alkaline diuresis or hemodialysis.

Acute ingestion of > 150 mg/kg can cause severe toxicity. Salicylate tablets may form bezoars, prolonging absorption and toxicity. Chronic toxicity can occur after several days of high therapeutic doses; it is common, often undiagnosed, and often more serious than acute toxicity. Chronic toxicity tends to occur in elderly patients.

The most concentrated and toxic form of salicylate is oil of wintergreen (methyl salicylate, a component of some liniments and solutions used in hot vaporizers); ingestion of < 5 mL can kill a young child. Any exposure should be considered serious. Bismuth subsalicylate (8.7 mg salicylate/mL) is another potentially unexpected source of large amounts of salicylate.

Pathophysiology

Salicylates impair cellular respiration by uncoupling oxidative phosphorylation. They stimulate respiratory centers in the medulla, causing primary respiratory alkalosis, which is often unrecognized in young children. Salicylates simultaneously and independently cause primary metabolic acidosis. Eventually, as salicylates disappear from the blood, enter the cells, and poison mitochondria, metabolic acidosis becomes the primary acid-base abnormality.

Salicylate poisoning also causes ketosis, fever, and, even when systemic hypoglycemia is absent, low brain glucose levels. Renal Na, K, and water loss and increased but imperceptible respiratory water loss

due to hyperventilation lead to dehydration.

Salicylates are weak acids that cross cell membranes relatively easily; thus, they are more toxic when blood pH is low. Dehydration, hyperthermia, and chronic ingestion increase salicylate toxicity because they result in greater distribution of salicylate to tissues. Excretion of salicylates increases when urine pH increases.

Symptoms and Signs

With acute overdose, early symptoms include nausea, vomiting, tinnitus, and hyperventilation. Later symptoms include hyperactivity, fever, confusion, and seizures. Rhabdomyolysis, acute renal failure, and respiratory failure may eventually develop. Hyperactivity may quickly turn to lethargy; hyperventilation (with respiratory alkalosis) progresses to hypoventilation (with mixed respiratory and metabolic acidosis) and respiratory failure.

With chronic overdose, symptoms and signs tend to be nonspecific and vary greatly. They include subtle confusion, changes in mental status, fever, hypoxia, noncardiogenic pulmonary edema, dehydration, lactic acidosis, and hypotension.

Diagnosis

• Serum salicylate level

• ABGs

Salicylate poisoning is suspected in patients with any of the following:

• History of a single acute overdose

• Repeated ingestions of therapeutic doses (particularly in patients with fever and dehydration)

• Unexplained metabolic acidosis

• Unexplained confusion and fever (in elderly patients)

If poisoning is suspected, serum salicylate (drawn at least a few hours after ingestion), urine pH, ABGs, serum electrolytes, serum creatinine, plasma glucose, and BUN are measured. If rhabdomyolysis is suspected, serum CK and urine myoglobin are measured.

Significant salicylate toxicity is suggested by serum levels much higher than therapeutic (therapeutic range, 10 to 20 mg/dL), particularly 6 h after ingestion (when absorption is usually almost complete), and by acidemia plus ABG results compatible with salicylate poisoning. Serum levels are helpful in confirming the diagnosis and may help guide therapy, but levels may be misleading and should be clinically correlated.

Usually, ABGs suggest primary respiratory alkalosis during the first few hours after ingestion; later, they suggest compensated metabolic acidosis or mixed metabolic acidosis/respiratory alkalosis. Eventually, usually as salicylate levels decrease, poorly compensated or uncompensated metabolic acidosis is the primary finding. If respiratory failure occurs, ABGs suggest combined metabolic and respiratory acidosis, and chest x-ray shows diffuse pulmonary infiltrates. Plasma glucose levels may be normal, low, or high. Serial salicylate levels help determine whether absorption is continuing; ABGs or serum electrolytes should always be determined simultaneously. Increased serum CK and urine myoglobin levels suggest rhabdomyolysis.

Treatment

• Activated charcoal

• Alkaline diuresis with extra KCl

Activated charcoal is given as soon as possible and, if bowel sounds are present, may be repeated every 4 h until charcoal appears in the stool.

After volume and electrolyte abnormalities are corrected, alkaline diuresis can be used to increase urine pH, ideally to ≥ 8. Alkaline diuresis is indicated for patients with any symptoms of poisoning and should not be delayed until salicylate levels are determined. This intervention is safe and exponentially increases salicylate excretion. Because hypokalemia may interfere with alkaline diuresis, patients are given a solution consisting of 1 L of 5% D/W, 3 50-mEq ampules of $NaHCO_3$, and 40 mEq of KCl at 1.5 to 2 times the maintenance IV fluid rate. Serum K is monitored.

Drugs that increase urinary HCO_3 (eg, acetazolamide) should be avoided because they worsen metabolic acidosis and decrease blood pH. Drugs that decrease respiratory drive should be avoided if possible because they may impair hyperventilation and respiratory alkalosis, decreasing blood pH.

Fever can be treated with physical measures such as external cooling (see p. 3266). Seizures are treated with benzodiazepines. In patients with rhabdomyolysis, alkaline diuresis may help prevent renal failure.

Hemodialysis may be required to enhance salicylate elimination in patients with severe neurologic impairment, renal or respiratory insufficiency, acidemia despite other measures, or very high serum salicylate levels (> 100 mg/dL [> 7.25 mmol/L] with acute overdose or > 60 mg/dL [> 4.35 mmol/L] with chronic overdose).

Carbon Monoxide Poisoning

Carbon monoxide (CO) poisoning causes acute symptoms such as headache, nausea, weakness, angina, dyspnea, loss of consciousness, and coma. Neuropsychiatric symptoms may develop weeks later. Diagnosis is by carboxyhemoglobin levels and ABGs, including measured O_2 saturation. Treatment is with supplemental O_2. Prevention is often possible with household carbon monoxide detectors.

CO poisoning, one of the most common fatal poisonings, occurs by inhalation. CO is a colorless, odorless gas that results from incomplete combustion of hydrocarbons. Common sources of CO in poisonings include house fires and improperly vented automobiles, gas heaters, furnaces, hot water heaters, wood- or charcoal-burning stoves, and kerosene heaters. CO is produced when natural gas (methane or propane) burns. Inhaling tobacco smoke results in CO in the blood but not enough to cause poisoning.

Pathophysiology

The elimination half-life of CO is about 4.5 h with inhalation of room air, 1.5 h with 100% O_2, and 20 min with 3 atmospheres (pressure) of O_2 (as in a hyperbaric chamber—see p. 3289).

Mechanisms of CO toxicity are not completely understood. They appear to involve

• Displacement of O_2 from Hb (because CO has greater affinity for Hb than does O_2)

• Shifting of the O_2-Hb dissociation curve to the left (decreasing release of O_2 from Hb to tissues—see Fig. 189-4 on p. 1857)

• Inhibition of mitochondrial respiration

• Possibly direct toxic effects on brain tissue

Symptoms and Signs

Symptoms tend to correlate well with the patient's peak blood carboxyhemoglobin levels. Many symptoms

are nonspecific.

- Headache and nausea can begin when levels are 10 to 20%.
- Levels > 20% commonly cause vague dizziness, generalized weakness, difficulty concentrating, and impaired judgment.
- Levels > 30% commonly cause dyspnea during exertion, chest pain (in patients with coronary artery disease), and confusion.
- Higher levels can cause syncope, seizures, and obtundation.

Hypotension, coma, respiratory failure, and death may occur, usually when levels are > 60%.

Patients may also have many other symptoms, including visual deficits, abdominal pain, and focal neurologic deficits. If poisoning is severe, neuropsychiatric symptoms and signs (eg, dementia, psychosis, parkinsonism, chorea, amnestic syndromes) can develop days to weeks after exposure and become permanent. Because CO poisoning often results from house fires, patients may have concomitant airway injuries (see Sidebar 329-1 on p. 3243), which may increase risk of respiratory failure.

Diagnosis

- Diagnosis considered when patients at risk have nonspecific symptoms or metabolic acidosis
- Venous carboxyhemoglobin level

Because symptoms can be vague, nonspecific, and variable, the diagnosis is easily missed. Many cases of mild poisoning with nonspecific symptoms are mistaken for viral syndromes. Physicians must maintain a high level of suspicion. If people from the same dwelling, particularly a heated dwelling, experience nonspecific symptoms, CO exposure should be considered.

If CO poisoning is suspected, the carboxy-hemoglobin level is measured with a COoximeter; venous samples can be used because arteriovenous differences are trivial. ABGs are not measured routinely. ABGs and pulse oximetry, alone or combined, are inadequate for diagnosis of CO poisoning because O_2 saturation reported in ABGs represents dissolved O_2 and is thus unaffected by carboxy-hemoglobin concentration; furthermore, the pulse oximeter cannot differentiate normal Hb from carboxyhemoglobin and thus provides a falsely elevated oxyhemoglobin reading. Although elevated carboxyhemoglobin levels are clear evidence of poisoning, levels may be falsely low because they decrease rapidly after CO exposure ends, particularly in patients treated with supplemental O_2 (eg, in an ambulance). Metabolic acidosis can be a clue to the diagnosis. Other tests may help evaluate specific symptoms (eg, ECG for chest pain, CT for neurologic symptoms).

Treatment

- 100% O_2
- Possibly hyperbaric O_2

Patients should be removed from the source of CO and stabilized as necessary. They are given 100% O_2 (by nonrebreather mask) and treated supportively. Hyperbaric O_2 therapy typically should be considered for patients who have any of the following:

- Life-threatening cardiopulmonary complications
- Ongoing chest pain
- Altered consciousness

• Loss of consciousness (no matter how brief)

• A carboxyhemoglobin level > 25%

Hyperbaric O_2 therapy should also be considered for pregnant patients.

Patients are placed in a chamber at 2 to 3 atmospheres of O_2. Hyperbaric O_2 therapy may decrease the incidence of delayed neuropsychiatric symptoms. However, this therapy may cause barotrauma and, because therapy is not available at most hospitals, may require transfer of patients, who may not be stable; also, a chamber may not be available locally, and evidence for the efficacy of hyperbaric O_2 therapy is somewhat inconclusive.

Prevention

Prevention involves checking sources of indoor combustion to make sure they are correctly installed and vented to the outdoors. Exhaust pipes should be inspected periodically for leaks. CO detectors should be installed because they provide early warning that CO is free in a dwelling's atmosphere. If CO is suspected in a dwelling, windows should be opened, and the dwelling should be evacuated and evaluated for the source of CO.

Caustic Ingestion

Caustics (strong acids and alkalis), when ingested, burn upper GI tract tissues, sometimes resulting in esophageal or gastric perforation. Symptoms may include drooling, dysphagia, and pain in the mouth, chest, or stomach; strictures may develop later. Diagnostic endoscopy may be required. Treatment is supportive. Gastric emptying and activated charcoal are contraindicated. Perforation is treated surgically.

Common sources of caustics include solid and liquid drain and toilet bowl cleaners. Industrial products are usually more concentrated than household products and thus tend to be more damaging.

Pathophysiology

Acids cause coagulation necrosis; an eschar forms, limiting further damage. Acids tend to affect the stomach more than the esophagus. Alkalis cause rapid liquefaction necrosis; no eschar forms, and damage continues until the alkali is neutralized or diluted. Alkalis tend to affect the esophagus more than the stomach, but ingestion of large quantities severely affects both.

Solid products tend to leave particles that stick to and burn tissues, discouraging further ingestion and causing localized damage. Because liquid preparations do not stick, larger quantities are easily ingested, and damage may be widespread. Liquids may also be aspirated, leading to upper airway injury.

Symptoms and Signs

Initial symptoms include drooling and dysphagia. In severe cases, pain, vomiting, and sometimes bleeding develop immediately in the mouth, throat, chest, or abdomen. Airway burns may cause coughing, tachypnea, or stridor.

Swollen, erythematous tissue may be visible intraorally; however, caustic liquids may cause no intraoral burns despite serious injury farther down the GI tract. Esophageal perforation may result in mediastinitis, with severe chest pain, tachycardia, fever, tachypnea, and shock. Gastric perforation may result in peritonitis. Esophageal or gastric perforation may occur within hours, after weeks, or any time in between.

Esophageal strictures can develop over weeks, even if initial symptoms had been mild and treatment had been adequate.

Diagnosis

• Endoscopy

Because the presence or absence of intraoral burns does not reliably indicate whether the esophagus and stomach are burned, meticulous endoscopy is indicated to check for the presence and severity of esophageal and gastric burns when symptoms or history suggests more than trivial ingestion.

Treatment

• Avoidance of gastric emptying

• Oral fluids

Treatment is supportive. (CAUTION: *Gastric emptying by emesis or lavage is contraindicated because it can reexpose the upper GI tract to the caustic. Attempts to neutralize a caustic acid by correcting pH with an alkaline substance [and vice versa] are contraindicated because severe exothermic reactions may result. Activated charcoal is contraindicated because it may infiltrate burned tissue and interfere with endoscopic evaluation.*)

Oral fluids are started when they can be tolerated. Esophageal or gastric perforation is treated with antibiotics and surgery (see p. 111). IV corticosteroids and prophylactic antibiotics are not recommended. Strictures are treated with bougienage or, if they are severe or unresponsive, with esophageal bypass by colonic interposition.

Mushroom Poisoning

Numerous mushroom species cause toxicity when ingested. Symptoms vary by species. Identification of specific species is difficult, so treatment usually is guided by symptoms.

Differentiating toxic and nontoxic species in the wild is difficult, even for highly knowledgeable people. Folklore rules are unreliable, and the same species may have varying degrees of toxicity depending on where they are harvested. If patients have eaten an unidentified mushroom, identifying the species can help determine specific treatment. However, because an experienced mycologist is seldom available for immediate consultation, treatment of patients who become ill after mushroom ingestion is usually guided by symptoms. If a sample of the mushroom, uningested or from the patient's emesis, is available, it can be sent to a mycologist for analysis.

All toxic mushrooms cause vomiting and abdominal pain; other manifestations vary significantly by mushroom type. Generally, mushrooms that cause symptoms early (within 2 h) are less dangerous than those that cause symptoms later (usually after 6 h). Activated charcoal may be useful.

Early GI symptoms: Mushrooms that cause early GI symptoms (eg, *Chlorophyllum molybdites*, the little brown mushrooms that often grow in lawns) cause gastroenteritis, sometimes with headaches or myalgias. Diarrhea is occasionally bloody. Symptoms usually resolve within 24 h. Treatment is supportive.

Early neurologic symptoms: Mushrooms that cause early neurologic symptoms include hallucinogenic mushrooms, which are usually ingested recreationally because they contain psilocybin, a hallucinogen. The most common are members of the *Psilocybe* genus, but some other genera contain psilocybin.

Symptoms begin within 15 to 30 min and include euphoria, enhanced imagination, and hallucinations. Tachycardia and hypertension are common, and hyperpyrexia occurs in some children; however, serious consequences are rare.

Treatment occasionally involves sedation (eg, with benzodiazepines).

Early muscarinic symptoms: Mushrooms that cause early muscarinic symptoms include members of the *Inocybe* and *Clitocybe* genera.

Symptoms may include SLUDGE syndrome (see Table 340-2 on p. 3325), including miosis, bronchorrhea, bradycardia, diaphoresis, wheezing, and fasciculations. Symptoms are usually mild, begin within 30 min, and resolve within 12 h.

Atropine may be given to treat severe muscarinic symptoms (eg, wheezing, bradycardia).

Delayed GI symptoms: Mushrooms that cause delayed GI symptoms include members of the *Amanita*, *Gyromitra*, and *Cortinarius* genera.

The most toxic *Amanita* mushroom is *Amanita phalloides*, which causes 95% of mushroom poisoning deaths. Initial gastroenteritis, which may occur 6 to 12 h after ingestion, can be severe; hypoglycemia can occur. Initial symptoms abate for a few days; then liver failure and sometimes renal failure develop. Initial care involves close monitoring for hypoglycemia and possibly repeated doses of activated charcoal. Treatment of liver failure may require liver transplantation; other specific treatments (eg, *N*-acetylcysteine, highdose penicillin, silibinin, IV fat emulsion) are unproved.

Gyromitra mushrooms can cause hypoglycemia simultaneously with or shortly after gastroenteritis. Other manifestations may include CNS toxicity (eg, seizures) and, after a few days, hepatorenal syndrome. Initial care involves close monitoring for hypoglycemia and possibly repeated doses of activated charcoal. Neurologic symptoms are treated with pyridoxine 70 mg/kg slow IV infusion over 4 to 6 h (maximum daily dose of 5 g); liver failure is treated supportively.

Most *Cortinarius* mushrooms are endogenous to Europe. Gastroenteritis may last for 3 days. Renal failure, with symptoms of flank pain and decreased urine output, may occur 3 to 20 days after ingestion. Renal failure often resolves spontaneously.

Plant Poisoning

A few commonly grown plants are highly poisonous, and many are moderately poisonous (see Table 340-6). Few plant poisonings have specific antidotes. Most plant ingestions, including the plants listed in Table 340-6, result in minimal symptoms unless the leaves and other components are concentrated into a paste or brewed into a tea.

Highly toxic and potentially fatal plants include the following:

• Castor beans and jequirity beans

• Oleander and foxglove

• Hemlock

Castor beans and jequirity beans: Castor beans contain ricin, an extremely concentrated cellular poison. Jequirity beans contain abrin, a related and even more potent toxin. In both, the beans have a relatively impervious shell; thus, the bean must be chewed to release the toxin. However, the seed coating of the jequirity bean is often not intact, and simple bacterial digestion can release the abrin toxin.

Symptoms of either poisoning may include delayed gastroenteritis, sometimes severe and hemorrhagic, followed by delirium, seizures, coma, and death. Whole-bowel irrigation should be considered because it aims to remove all beans ingested.

Oleander and foxglove: These plants and lily of the valley (which is similar but less toxic) contain digitalis glycosides. Toxicity includes gastroenteritis, confusion, hyperkalemia, and arrhythmias. The serum digoxin level can confirm ingestion but is not useful as quantitative information.

K levels are closely monitored. Hyperkalemia may respond only to hemodialysis. Ca is not recommended for arrhythmias. Digoxin-specific fractionated antibody (Fab) fragments have been used to treat ventricular arrhythmias.

Hemlock: Hemlock poisoning (poison hemlock and water hemlock) can cause symptoms within 15 min.

Poison hemlock has nicotinic effects, beginning with dry mouth and progressing to tachycardia, tremors, diaphoresis, mydriasis, seizures, and muscle paresis. Rhabdomyolysis and bradycardia may occur.

Water hemlock seems to enhance γ-aminobutyric acid (GABA) activity. Symptoms may include gastroenteritis, delirium, refractory seizures, and coma.

Fish and Shellfish Poisoning

Fish and shellfish poisoning commonly causes GI, neurologic, or histamine-mediated manifestations.

Ciguatera poisoning: Ciguatera poisoning may result from eating any of > 400 species of fish from the tropical reefs of Florida, the West Indies, or the Pacific, where a dinoflagellate produces a toxin that accumulates in the flesh of the fish. Older fish and large fish (eg,

[Table 340-6. Moderately Poisonous Plants]

grouper, snapper, kingfish) contain more toxin. No known processing procedures, including cooking, are protective, and flavor is unaffected. A commercial product is available to test for ciguatoxin in fish.

Symptoms may begin 2 to 8 h after eating. Abdominal cramps, nausea, vomiting, and diarrhea last 6 to 17 h; then, pruritus, paresthesias, headache, myalgia, reversal of hot and cold sensation, and face pain may occur. For months afterward, unusual sensory phenomena and nervousness may cause debilitation.

IV mannitol has been suggested as a treatment, but no clear benefit has been shown.

Scombroid poisoning: Scombroid poisoning is caused by high histamine levels in fish flesh due to bacterial decomposition after the fish is caught. Commonly affected species include

- Tuna
- Mackerel
- Bonito
- Skipjack
- Mahi mahi

The fish may taste peppery or bitter. Facial flushing and possibly nausea, vomiting, epigastric pain, and urticaria occur within a few minutes of eating and resolve within 24 h. Symptoms are often mistaken for those of a seafood allergy. Unlike other fish poisonings, this poisoning can be prevented by properly storing the fish after it is caught.

Treatment may include H_1 and H_2 blockers.

Tetrodotoxin poisoning: Tetrodotoxin poisoning is most commonly due to eating the puffer fish (fugu), a sushi delicacy, but > 100 fresh and salt water species contain tetrodotoxin. Symptoms are similar to those of ciguatera poisoning; potentially fatal respiratory paralysis can also occur. Treatment is supportive care with attention to ventilatory assistance until the toxin is metabolized, which may take days.

The toxin cannot be destroyed by cooking or freezing.

Shellfish poisoning: Paralytic shellfish poisoning can occur from June to October, especially on the Pacific and New England coasts, when mussels, clams, oysters, and scallops are contaminated by the

poisonous dinoflagellate responsible for red tide. This dinoflagellate produces the neurotoxin saxitoxin, which is resistant to cooking. Circumoral paresthesias occur 5 to 30 min after eating. Nausea, vomiting, and abdominal cramps then develop, followed by muscle weakness. Untreated respiratory paralysis may be fatal; for survivors, recovery is usually complete.

Hydrocarbon Poisoning

Hydrocarbon poisoning may result from ingestion or inhalation. Ingestion, most common among children < 5 yr, can result in aspiration pneumonitis. Inhalation, most common among adolescents, can result in ventricular fibrillation, usually without warning symptoms. Diagnosis of pneumonitis is by clinical evaluation, chest x-ray, and oximetry. Gastric emptying is contraindicated because aspiration is a risk. Treatment is supportive.

Ingestion of hydrocarbons, such as petroleum distillates (eg, gasoline, kerosene, mineral oil, lamp oil, paint thinners), results in minimal systemic effects but can cause severe aspiration pneumonitis. Toxic potential mainly depends on viscosity, measured in Saybolt seconds universal (SSU). Hydrocarbon liquids with low viscosity (SSU < 60), such as gasoline and mineral oil, can spread rapidly over large surface areas and are more likely to cause aspiration pneumonitis than are hydro-carbons with SSU > 60, such as tar. Hydro-carbons, if ingested in large amounts, may be absorbed systemically and cause CNS or hepatic toxicity, which is more likely with halogenated hydrocarbons (eg, carbon tetrachloride, trichloroethylene).

Recreational inhalation of halogenated hydrocarbons (eg, glues, paint, solvents, cleaning sprays, gasoline, fluorocarbons used as refrigerants or propellants in aerosols—see p. 1531), called huffing or bagging, is common among adolescents. It can cause euphoria and mental status changes and can sensitize the heart to endogenous catecholamines. Fatal ventricular arrhythmias may result; they usually occur without premonitory palpitations or other warning, often when patients are startled or chased.

Symptoms and Signs

After ingestion of even a very small amount of liquid hydrocarbon, patients initially cough, choke, and may vomit. Young children may have cyanosis, hold their breath, and cough persistently. Older children and adults may report burning in the stomach. Aspiration pneumonitis causes hypoxia and respiratory distress. Symptoms and signs of pneumonitis may develop a few hours before infiltrates are visible on x-ray. Substantial systemic absorption, particularly of a halogenated hydrocarbon, may cause lethargy, coma, and seizures. Nonfatal pneumonitis usually resolves in about 1 wk; mineral or lamp oil ingestion usually resolves in 5 to 6 wk. Arrhythmias usually occur before presentation and are unlikely to recur after presentation unless patients have excessive agitation.

Diagnosis

• Chest x-ray and oximetry done about 6 h after ingestion

If patients are too obtunded to provide a history, hydrocarbon exposure may be suspected if their breath or clothing has an odor or if a container is found near them. Paint residue on the hands or around the mouth may suggest recent paint sniffing.

Diagnosis of aspiration pneumonitis is by symptoms and signs as well as by chest x-ray and oximetry, which are done about 6 h after ingestion or sooner if symptoms are severe. If respiratory failure is suspected, ABGs are measured.

Treatment

• Supportive care

• Avoidance of gastric emptying

Any contaminated clothing is removed, and the skin is washed. (CAUTION: *Gastric emptying, which*

increases risk of aspiration, is contraindicated.) Charcoal is not recommended. Patients who do not have aspiration pneumonitis or other symptoms after 4 to 6 h are discharged. Patients who have symptoms are admitted and treated supportively; antibiotics and corticosteroids are not indicated.

Organophosphate and Carbamate Poisoning

Organophosphates and carbamates are common insecticides that inhibit cholinesterase activity, causing acute muscarinic manifestations (eg, salivation, lacrimation, urination, diarrhea, emesis, bronchorrhea, bronchospasm, bradycardia, miosis) and some nicotinic symptoms, including muscle fasciculations and weakness. Neuropathy can develop days to weeks after exposure. Diagnosis is clinical and sometimes with a trial of atropine, measurement of RBC acetylcholinesterase level, or both. Bronchorrhea and bronchospasm are treated with titrated highdose atropine. Neuro-muscular toxicity is treated with IV pralidoxime.

Organophosphates and carbamates, although different structurally, both inhibit cholinesterase activity. Some are used medically to reverse neuromuscular blockade (eg, neostigmine, pyridostigmine, edrophonium) or to treat glaucoma, myasthenia gravis, and Alzheimer's disease (eg, echothiophate, pyridostigmine, tacrine, donepezil).

Some organophosphates were developed as nerve gases. One, sarin, has been used by terrorists. Organophosphates and carbamates are commonly used as insecticides (see Table 340-8). Those most often implicated in human poisoning include

• Carbamates: Aldicarb and methomyl

• Organophosphates: Chlorpyrifos, diazinon, dursban, fenthion, malathion, and parathion

Organophosphates and carbamates are common causes of poisoning and poison-related deaths worldwide.

Pathophysiology

Organophosphates and carbamates are absorbed through the GI tract, lungs, and skin. They inhibit plasma and RBC cholinesterase, preventing breakdown of acetylcholine, which then accumulates in synapses. Carbamates are cleared spontaneously within about 48 h after exposure. Organophosphates, however, can irreversibly bind to cholinesterase.

Symptoms and Signs

Acute: Organophosphates and carbamates cause acute muscarinic and nicotinic cholinergic toxidromes (see Table 340-2). Muscle fasciculations and weakness are typical. Most patients have bradycardia and, if poisoning is severe, hypotension. CNS toxicity is common, sometimes with seizures and excitability and often with lethargy and coma. Pancreatitis is possible, and organophosphates may cause arrhythmias such as heart block and QTc interval prolongation.

Delayed: Weakness, particularly of proximal, cranial, and respiratory muscles, may develop 1 to 3 days after exposure to organo-phosphates or rarely carbamates despite treatment (the intermediate syndrome); these symptoms resolve in 2 to 3 wk. A few organ-ophosphates (eg, chlorpyrifos, triorthocresyl phosphate) may cause an axonal neuropathy that begins 1 to 3 wk after exposure. The mechanism may be independent of RBC cholinesterase, and the risk is independent of the severity of poisoning. Long-term, persistent sequelae of organophosphate poisoning may include cognitive deficits or parkinsonism.

Diagnosis

• Muscarinic toxidrome with muscle fasciculations and weakness

• Sometimes RBC cholinesterase levels

The diagnosis is usually based on the characteristic muscarinic toxidrome in patients with neuromuscular findings, particularly in patients at risk. If findings are equivocal, reversal or abatement of muscarinic symptoms after 1 mg of atropine (0.01 to 0.02 mg/kg in children) supports the diagnosis. The specific toxin should be identified if possible. Many organophosphates have characteristic garlic-like or petroleum odors.

RBC cholinesterase activity, which can be measured by some laboratories, indicates the severity of poisoning. If it can be measured rapidly, values can be used to monitor the effectiveness of treatment.

Treatment

• Supportive therapy

• Atropine for respiratory manifestations

• Decontamination

• Pralidoxime for neuromuscular manifestations

In-hospital treatment: Supportive therapy is key. Patients should be closely monitored for respiratory failure due to weakness of respiratory muscles.

Atropine is given in amounts sufficient to relieve bronchospasm and bronchorrhea rather than to normalize pupil size or heart rate. Initial dosage is 2 to 5 mg IV (0.05 mg/kg in children); the dose can be doubled every 3 to 5 min prn. Grams of atropine may be necessary for severely poisoned patients.

Decontamination is pursued as soon as possible after stabilization. Caregivers should avoid self-contamination while providing care. For topical exposure, clothes are removed, and the body surface is flushed thoroughly. For ingestion within 1 h of presentation, activated charcoal can be used. Gastric emptying is usually avoided. If done, the trachea is intubated beforehand to prevent aspiration.

Pralidoxime (2-PAM) is given after atropine to relieve neuromuscular symptoms. Pralidoxime (1 to 2 g in adults; 20 to 40 mg/kg in children) is given over 15 to 30 min IV after exposure to an organophosphate or carbamate because, frequently, whether the poison is an organophosphate or carbamate is unknown at the time of treatment. An infusion can be used after the bolus (8 mg/kg/h in adults; 10 to 20 mg/kg/h in children).

Benzodiazepines are used for seizures. Prophylactic diazepam may help prevent neurocognitive sequelae after moderate to severe organophosphate poisoning.

Out-of-hospital exposure: People exposed to these toxins away from a hospital can give themselves low doses of atropine using commercially prepared autoinjectors (2 mg for adults and for children > 41 kg; 1 mg for children19 to 41 kg; 0.5 mg for children < 19 kg). Autoinjection of 10 mg diazepam has been recommended for people exposed to a chemical attack.

Iron Poisoning

Iron poisoning is the leading cause of poisoning deaths in children. Symptoms begin with acute gastroenteritis, followed by a quiescent period, then shock and liver failure. Diagnosis is by measuring serum iron, detecting radiopaque iron tablets in the GI tract, or detecting unexplained metabolic acidosis in patients with other findings suggesting iron poisoning. Treatment of a substantial ingestion is usually whole-bowel irrigation and chelation therapy with IV deferoxamine.

Many commonly used OTC preparations contain iron. Of the many iron compounds used in OTC and prescription preparations, the most common are

• Ferrous sulfate (20% elemental iron)

• Ferrous gluconate (12% elemental iron)

• Ferrous fumarate (33% elemental iron)

To children, iron tablets may look like candy. Prenatal multivitamins are the source of iron in most lethal ingestions among children. Children's chewable multivitamins with iron usually have such small amounts that toxicity rarely occurs.

Pathophysiology

Iron is toxic to the GI system, cardiovascular system, and CNS. Specific mechanisms are unclear, but excess free iron is inserted into enzymatic processes and interferes with oxidative phosphorylation, causing metabolic acidosis. Iron also catalyzes free radical formation, acts as an oxidizer, and, when plasma protein binding is saturated, combines with water to form iron hydroxide and free H^+ ions, compounding the metabolic acidosis. Coagulopathy may appear early because of interference with the coagulation cascade and later because of liver injury.

Toxicity depends on the amount of elemental iron that has been ingested. Up to 20 mg/kg of elemental iron is not toxic, 20 to 60 mg/kg is mildly to moderately toxic, and > 60 mg/kg can cause severe symptoms and morbidity.

Symptoms and Signs

Symptoms occur in 5 stages (see Table 340-7); however, symptoms and their progression vary significantly. The severity of

[**Table 340-7.** Stages of Iron Poisoning]

stage 1 symptoms usually reflects the overall severity of poisoning; late-stage symptoms develop only if stage 1 symptoms are moderate or severe. If no symptoms develop within the first 6 h after ingestion, risk of serious toxicity is minimal. If shock and coma develop within the first 6 h, the mortality rate is about 10%.

Diagnosis

• Abdominal x-ray

• Determination of serum iron, electrolytes, and pH 3 to 4 h after ingestion

Iron poisoning should be considered in mixed ingestions (because iron is ubiquitous) and in small children with access to iron and unexplained metabolic acidosis or severe or hemorrhagic gastroenteritis. Because children often share, siblings and playmates of small children who have ingested iron should be evaluated.

Abdominal x-ray is usually recommended to confirm ingestion; it detects intact iron tablets or iron concretions but misses chewed and dissolved tablets, liquid iron preparations, and iron in multivitamin preparations. Serum iron, electrolytes, and pH are determined 3 to 4 h after ingestion. Toxicity is assumed if suspected ingestion is accompanied by any of the following:

• Vomiting and abdominal pain

• Serum iron levels > 350 µg/dL (63 µmol/L)

• Iron visible on x-ray

• Unexplained metabolic acidosis

These iron levels may indicate toxicity; however, iron levels alone do not predict toxicity accurately. Total iron binding capacity (TIBC) is often inaccurate and not helpful in diagnosing serious poisoning and is not recommended. The most accurate approach is to serially measure levels of serum iron, HCO_3, and pH (with calculation of the anion gap); these findings are then evaluated together, and results are correlated with the patient's clinical status. For example, toxicity is suggested by increasing iron levels, metabolic acidosis, worsening symptoms, or, more typically, some combination of these findings.

Treatment

• Whole-bowel irrigation

• For severe toxicity, IV deferoxamine

If radiopaque tablets are visible on abdominal x-ray, whole-bowel irrigation with polyethylene glycol 1 to 2 L/h for adults or 25 to 40 mL/kg/h for children is done until no iron is visible on repeat abdominal x-ray. Gastric lavage is usually not helpful because vomiting tends to empty the stomach more efficiently. Activated charcoal does not adsorb iron and should be used only if other toxins also were ingested.

All patients with more than mild gastroenteritis are hospitalized. Patients with severe toxicity (metabolic acidosis, shock, severe gastroenteritis, or serum iron level > 500 µg/dL) are treated with IV deferoxamine to chelate free serum iron. Deferoxamine is infused at rates up to 15 mg/kg/h IV, titrated until hypotension occurs. Because both deferoxamine and iron poisoning can decrease BP, patients receiving deferoxamine require IV hydration.

Lead Poisoning

(Plumbism)

Lead poisoning often causes minimal symptoms at first but can cause acute encephalopathy or irreversible organ damage, commonly resulting in cognitive deficits in children. Diagnosis is by whole blood lead level. Treatment involves stopping lead exposure and sometimes using chelation therapy with succimer or edetate Ca disodium, with or without dimercaprol.

Leaded paint was commonly used until 1960, used to some degree until the early 1970s, and mostly eliminated in 1978. Thus, for a significant number of older housing units, leaded paint still poses some hazard. Lead poisoning is usually caused by direct ingestion of leaded paint chips (from cracked, peeling paint). During home remodeling, patients may be exposed to significant amounts of aerosolized lead in the form of particles scraped or sanded off during surface preparation for repainting.

Some ceramic glazes contain lead; ceramic ware (eg, pitchers, cups, plates) that is made with these glazes (common outside the US) can leach lead, particularly when in contact with acidic substances (eg, fruits, cola drinks, tomatoes, wine, cider). Lead-contaminated moonshine whiskey and folk remedies are possible sources, as are occasional lead foreign objects in the stomach or tissues (eg, bullets, curtain or fishing weights). Bullets lodged in soft tissues near synovial fluid or CSF may increase blood lead levels, but that process takes years.

Occupational exposure can occur during battery manufacture and recycling, bronzing, brass making, glass making, pipe cutting, soldering and welding, smelting, or working with pottery or pigments. Certain ethnic cosmetic products and imported herbal products and medicinal herbs contain lead and have caused cluster outbreaks of lead poisoning in immigrant communities. Fumes of leaded gasoline (in countries other than the US) recreationally inhaled for CNS effects may cause lead poisoning.

Symptoms and Signs

Lead poisoning is most often a chronic disorder and may not cause acute symptoms. With or without acute symptoms, poisoning eventually has irreversible effects (eg, cognitive deficits, peripheral neuropathy, progressive renal dysfunction).

Risk of cognitive deficits increases when the whole blood lead level (PbB) is ≥ 10 mg/dL (≥ 0.48 mmol/L) for an extended period, although the cutoff may be even lower. Other symptoms (eg, abdominal cramping, constipation, tremors, mood changes) may occur if PbB is > 50 mg/dL (> 2.4 mmol/L). Encephalopathy is likely if PbB is > 100 mg/dL (> 4.8 mmol/L).

In children: Acute lead poisoning may cause irritability, decreased attentiveness, and acute encephalopathy. Cerebral edema develops over 1 to 5 days, causing persistent and forceful vomiting, ataxic gait, seizures, altered consciousness, and, finally, intractable seizures and coma. Encephalopathy may be preceded by several weeks of irritability and decreased play activity.

Chronic lead poisoning in children may cause intellectual disability, seizure disorders, aggressive behavior disorders, developmental regression, chronic abdominal pain, and anemia.

In adults: Adults with occupational exposure characteristically develop symptoms (eg, personality changes, headache, abdominal pain, neuropathy) over several weeks or longer. Encephalopathy is unusual. Adults may develop loss of sex drive, infertility, and, in men, erectile dysfunction.

In children and adults: Anemia may develop because lead interferes with the normal formation of Hb. Children and adults who inhale tetra-ethyl or tetra-methyl lead (in leaded gasoline) may develop toxic psychosis in addition to more characteristic symptoms of lead poisoning.

Diagnosis

• Lead levels in capillary or whole blood

Lead poisoning is suspected in patients with characteristic symptoms. However, because symptoms are often nonspecific, diagnosis is often delayed. Evaluation includes CBC and measurement of serum electrolytes, BUN, serum creatinine, plasma glucose, and PbB levels. An abdominal x-ray should be taken to look for lead particles, which are radiopaque. X-rays of long bones are taken in children. Horizontal, metaphyseal lead bands representing lack of RBC remodeling and increased Ca deposition in the zones of provisional calcification in children's long bones are somewhat specific for poisoning with lead or other heavy metals but are insensitive. Normocytic or microcytic anemia suggests lead toxicity, particularly when the reticulocyte count is elevated or RBC basophilic stippling occurs; however, sensitivity and specificity are limited. Diagnosis is definitive if the PbB level is ≥ 10 µg/dL.

Because measuring PbB is not always possible and can be expensive, other preliminary or screening tests for lead poisoning can be used. Capillary blood testing for lead is accurate, inexpensive, and quick. All positive tests should be confirmed with PbB. The erythrocyte protoporphyrin (also called zinc protoporphyrin or free erythrocyte protoporphyrin) test is often inaccurate and now is seldom used.

The edetate Ca disodium ($CaNa_2$ EDTA) mobilization test, previously used for diagnosis and treatment, is considered obsolete by most toxicologists and is usually not done.

Treatment

• Source of lead eliminated (eg, whole-bowel irrigation if lead in GI tract)

• Chelation for adults with symptoms of poisoning plus PbB > 70 mg/dL

• Chelation for children with encephalopathy or PbB > 45 mg/dL (> 2.15 mmol/L)

For all patients, the source of lead is eliminated. If lead chips are visible on abdominal x-ray, whole-bowel irrigation with a polyethylene glycol electrolyte solution at 1 to 2 L/h for adults or 25 to 40 mL/kg/h for children is done until repeat x-ray shows no lead. If the cause is bullets, surgical removal should be considered. Children with PbB > 70 µg/dL (> 3.40 µmol/L) and all patients with neurologic symptoms should be hospitalized. Patients with acute encephalopathy are admitted to an ICU.

Chelating drugs (eg, succimer [meso-2,3-dimercaptosuccinic acid], $CaNa_2$EDTA, dimercaprol [British

antilewisite, or BAL]) can be given to bind lead into forms that can be excreted (see also Table 340-4). Chelation should be supervised by an experienced toxicologist. Chelation is indicated for adults with symptoms of poisoning plus PbB > 70 mg/dL and for children with encephalopathy or PbB > 45 mg/dL (> 2.15 mmol/L). Liver and kidney disorders are relative contraindications for chelating drugs. Chelating drugs should not be given to any patient with ongoing exposure to lead because chelation can increase GI absorption of lead. Chelation removes only relatively small amounts of metal. If total body burden of lead is very large, multiple chelations over many years may be required.

Regimens: Patients with encephalopathy are treated with dimercaprol 75 mg/m^2 (or 4 mg/kg) IM q 4 h and $CaNa_2EDTA$ 1000 to 1500 mg/m^2 IV (infusion) once/day. The first dose of dimercaprol should precede the first dose of $CaNa_2EDTA$ by at least 4 h to prevent redistribution of lead into the brain. Dimercaprol may be stopped after the first few doses depending on lead levels and symptom severity. Dimercaprol-$CaNa_2EDTA$ combination therapy is given for 5 days, followed by a 3-day washout period; then the need for continued chelation is reassessed.

Patients without encephalopathy are usually treated with succimer 10 mg/kg po q 8 h for 5 days, followed by 10 mg/kg po q 12 h for 14 days. If these patients have symptoms, they can alternatively be treated for 5 days with dimercaprol 50 mg/m^2 needed deep IM injection q 4 h plus $CaNa_2EDTA$ 1000 mg/m^2 IV once/day.

Drugs: Dimercaprol, which can cause vomiting, is given with parenteral or oral fluids. Dimercaprol can also cause pain at the injection site, numerous systemic symptoms, and, in patients with G6PD deficiency, moderate to severe acute intravascular hemolysis. This drug should not be given concurrently with iron supplements. Dimercaprol is formulated with peanut derivatives and thus is contraindicated in patients with known or suspected peanut allergy.

$CaNa_2EDTA$ can cause thrombophlebitis, which can be prevented by giving the drug IM, not IV, and by using an IV concentration of < 0.5%. Before beginning treatment with $CaNa_2EDTA$, adequate urine flow must be confirmed. Serious reactions to $CaNa_2EDTA$ include renal insufficiency, proteinuria, microscopic hematuria, fever, and diarrhea. Renal toxicity, which is dose-related, is usually reversible. Adverse effects of $CaNa_2EDTA$ are probably due to zinc depletion.

Succimer may cause rash, GI symptoms (eg, anorexia, nausea, vomiting, diarrhea, metallic taste), and transient elevations of liver enzymes.

Lower lead levels: Patients with PbB > 10 µg/dL should be monitored closely, and they or their parents should be taught how to reduce their exposure to lead.

Prevention

Patients at risk should be screened by measuring PbB. Measures that reduce risk of household poisoning include regular hand washing, regular washing of children's toys and pacifiers, and regular cleaning of household surfaces; drinking water, household paint (except in houses built after 1978), and ceramic ware made outside the US should be tested for lead. Adults exposed to lead dust at work should use appropriate personal protective equipment, change their clothing and shoes before going home, and shower before going to bed.

Specific Poisons

Symptoms and treatment of specific poisons vary (see Table 340-8); including all the specific complexities and details is impossible. Consultation with a poison control center is recommended for any poisonings except the mildest and most routine.

[**Table 340-8.** Symptoms and Treatment of Specific Poisons]