PART 7

Surgical and Medical Interventions

CHAPTER 46

Improvised Medicine in the Wilderness

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At the heart of wilderness medicine is improvisation, a creative blend of medicine and commonsense problem solving in a resource-limited setting. Medical emergencies can arise at any time in wilderness settings; clinicians in these environments often encounter situations requiring interventions when little or no medical equipment is at hand. Even when they have supplies, clinicians venturing into a wilderness environment often find that some improvisation may still be necessary. When improvising medical equipment, they must consider (1) whether it will accomplish the intended purpose, (2) if it is practical, and (3) if it could worsen the situation.[33] Improvisation encompasses many variations, is governed by few absolute rights and wrongs, and is limited more often by imagination than by personnel or equipment.

This chapter is not exhaustive, but rather exemplifies the types of improvisation that may be possible in wilderness or remote settings. We have either tested the featured methods, often in the field, or drawn them from the peer-reviewed literature. We have tested methods that are more anecdotal or historical, noting their usefulness. We highly recommend that clinicians test these procedures in a safe setting before performing them on patients. Although some of the improvisations, such as transportation methods, border on first aid, they are an integral part of remote medical care.

GENERAL ASSESSMENT AND VITAL SIGNS

Measurements may help the practitioner or rescue crew to plan improvised care or transport. Because having a measuring tape or weighing scales would not be the norm in a backcountry situation, one way to accurately estimate wound size is to measure it against the clinician's comparable body parts. Table 46-1 gives approximate lengths and areas for upper-extremity body parts to use with measurements corresponding with Caucasian men. Because body sizes vary greatly, especially between men and women, practitioners should measure these sites on their own bodies before using them to estimate patient measurements.

Another option is to mark standard measurements in centimeters or in inches on personal medical equipment, such as the metal tubes of a stethoscope (Figure 46-1), or on a knife, penlight, or scissors.

HEIGHT

To measure a non-ambulatory patient's height, one may use preplaced marks in 0.5-cm (or 0.5-inch) increments on an avalanche probe, walking stick, ski pole, or along the sides of an evacuation stretcher. Doubling the longest measurement from midsternum to the tip of the patient's third finger produces a very accurate height estimate.[81]

WEIGHT

Knowing a patient's weight may be important when administering weight-based medications or when considering aeromedical transport. A relatively accurate way to obtain an adult patient's weight is simply to ask the person for an estimate. Although paramedics' estimates may correlate to some degree with adult cardiac arrest patients' actual weights,[85] estimates by health care workers are generally not reliable. Weight estimates of children also can be difficult. Parents estimate their child's weight within 10% of the actual weight only 78% of the time. This is better than the estimate from a Broselow tape, which is within 10% of the measured weight only 61% of the time and is progressively less accurate as children age, generally underestimating weight in less-developed countries.[4,10,50,82] All other forms of guessing a child's weight, including the Argall, Advanced Pediatric Life Support, and Best Guess methods, perform poorly.[78] If the parents are not present, use the following formula:

$$(2 \times \text{age in years}) + 8 = \text{weight in kilograms}$$

PULSES IN ADULTS AND CHILDREN

Clinicians typically decide whether an adult has a pulse by palpating it. This may not be applicable in infants because of the difficulty in knowing where and how to locate it, especially if it is barely (or not) palpable. Palpating a critically ill infant's pulse can be very difficult. However, determining infant cardiac activity is quickly and easily done by simply placing one's ear against the infant's chest wall and listening for heart sounds[56] (Figure 46-2).

RADIAL PULSE AND TRAUMA PROGNOSIS

Tachycardia is a much more reliable sign of hypovolemia than is blood pressure (BP). In trauma patients age 18 to 50 years without a head injury, a weak radial pulse indicates greatly increased mortality (29%), compared with only 3% of patients with a normal-quality pulse, as well as high likelihood that the patient will require endotracheal intubation and intensive care unit admission. Patients in shock without a radial pulse are usually moribund.[90] However, a *shock index* (SI), defined as the heart rate/systolic BP, greater than 0.9 suggests significant bleeding; patients with SI greater than 1.4 have about a 60% chance of being in a condition of ongoing massive hemorrhage.[113]

BLOOD PRESSURE

Research has shown that the "ability to obtain a blood pressure measurement in an austere environment is often limited by time constraints, equipment availability, and noisy conditions."[90] This often makes perfusion signs, such as mental status, capillary refill, and urine output, more useful indicators of tissue perfusion.

Blood Pressure without a Cuff

Pulse characteristics are an unreliable sign—and "should be used only as a last resort."[90] Without scientific evidence, the advanced trauma life support (ATLS) course taught that systolic BP can be estimated from whether radial (>80 mm Hg), femoral (70 to 80 mm Hg), or carotid (60 to 70 mm Hg) pulses are palpable. This method tends to overestimate the patient's BP. Although the radial pulse always disappears before the femoral pulse, which always disappears before the carotid pulse, most patients' actual BP is lower than that predicted by these guidelines. That information by itself could be useful in a crisis.[22]

TABLE 46-1 Clinical Measurements Using Body Parts*

Approximate Length	Measurement Site(s)
1.3 cm (0.5 inch)	*Finger width:* Greatest width of the distal phalanx of the small finger
2.5 cm (1 inch)	*Phalanx:* Length of the middle phalanx of the small finger
	Thumb: Width of thumb at the interphalangeal joint
	Span: When opening the hand and spreading the fingers widely, from the tip of the thumb to the tip of the index finger
3.8 cm (1.5 inch)	*Grasp:* Greatest diameter of the circle formed with the thumb and index finger

Data from Iserson KV: *Improvised medicine: Providing care in extreme environments*, New York, 2012, McGraw-Hill.
http://faculty.marianopolis.edu/c.belanger/quebechistory/encyclopedia/MeansofMeasurementbyIndians.htm.
*Some of these measurements were suggested by White J, editor: *Handbook of Indians of Canada*, published as an appendix to the Tenth Report of the Geographic Board of Canada, Ottawa, 1913, pp 280-281.

Nonhypothermic patients with extremities that are cooler than normal may have hypoperfusion. If it is assessed in multiple extremities, especially in both an arm and a leg, checking a palpable temperature effectively diagnoses hypoperfusion. These patients have lower cardiac indexes, lower pH, lower bicarbonate levels, lower mixed venous oxygen saturation, and higher lactate levels.[74] In a neonate, a cold or dusky sole of the foot can indicate hypothermia, hypoxemia, and/or hypotension.[21]

Blood Pressure without a Stethoscope

Because it may be difficult to auscultate BP when transporting patients by air or in a noisy ambulance, one may palpate the systolic BP. If done carefully, this method can be as accurate as using a stethoscope. Alternatively, use a pulse oximeter by obtaining a good waveform on a finger, inflating the sphygmomanometer until the waveform disappears, and then slowly deflating the cuff until the waveform reappears. That cuff measurement is the systolic BP.[88]

Wrist and Calf Blood Pressure Measurements

To be accurate, a BP cuff must cover two-thirds the length of the upper arm. Too small a BP cuff causes an abnormally high BP reading; one that is too large results in an abnormally low reading. If you do not have a cuff that fits the arm properly, move it to a location where it fits.

Place a BP cuff on the patient's forearm (Figure 46-3) or ankle to obtain better access during patient transport (1) with very obese patients on whom the standard adult cuff is too small; (2) for adults when only a child's-size cuff is available; (3) to avoid periodically occluding an arteriovenous fistula (e.g., dialysis patients) or intravenous (IV) line; or (4) or to avoid compressing an area that has been injured.

Blood pressure cuffs at these sites produce the same or almost the same mean and diastolic BP readings, but the systolic pressures vary. Use the mean BP with a forearm or calf BP cuff.[16] Calculate this by multiplying the diastolic pressure by 2 and adding the systolic pressure, then dividing the sum by 3:

$$MAP = [(2 \times DBP) + SBP]/3$$

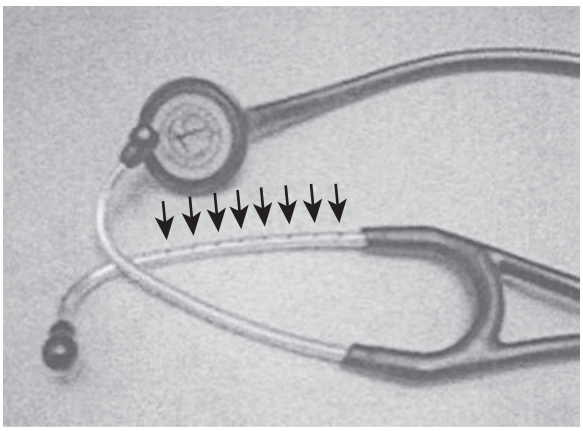


FIGURE 46-1 Stethoscope marked to measure lengths.

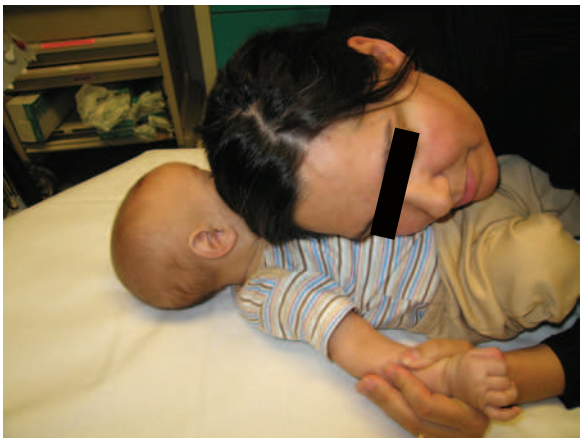


FIGURE 46-2 Direct precordial auscultation.

IMPROVISED DIAGNOSTIC EQUIPMENT AND SUPPLIES

STETHOSCOPES

Ear to Patient

If no other option is available, put one ear directly against the chest, heart, or abdomen. The sound quality is less than with a stethoscope, but adequate for cardiac rhythm, abnormal chest sounds, and bowel sounds. Putting one's ear on an infant's chest is the fastest and most accurate method for assessing the presence of cardiac activity. Whenever possible, put a plastic barrier between you and the patient's skin (universal precautions).

Improvised Standard Stethoscopes

Easily fashion a Laennec-type monaural stethoscope by using rolled paper, short lengths of garden hose, metal pipes, ivory or wood hollow tubes, or bamboo. The cardboard tube from a roll of toilet paper or paper towels works well. A slightly more sophisticated stethoscope can be fashioned from a funnel attached to two pieces of tubing (Figure 46-4). The juncture of IV tubing works well as a T-tube connector. Put the tube ends directly in your ears, or fashion earpieces as described in Stethoscope Earpiece, below.

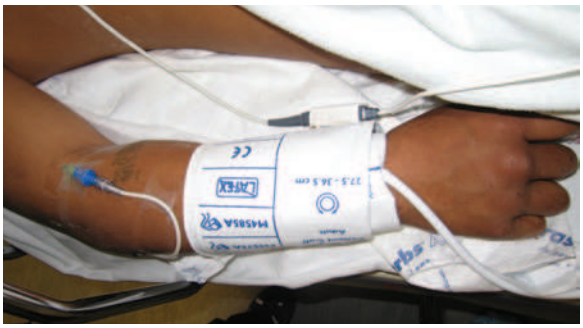


FIGURE 46-3 Blood pressure cuff on forearm.

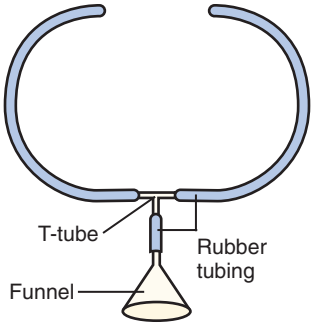


FIGURE 46-4 Improvised stethoscope.

Precordial Stethoscope

Use a precordial stethoscope to monitor patients when access to them is difficult, such as during transport or procedural sedation. Taped to the patient's chest, it provides the clinician with continuous assessment of the patient's audible heart rate and rhythm, as well as breath sounds. In adults, place the stethoscope over the midsternum, in the suprasternal notch, in the perilaryngeal area, or in the axilla. In infants, place the stethoscope on the left chest so that both the heartbeat and breath sounds can be heard. Several improvised methods will produce a workable precordial stethoscope. Only one earpiece is normally used so that the caregiver can also monitor external sounds.

The quickest way to make a precordial stethoscope is to attach a piece of rubber tubing (this goes in the clinician's ear) to the top of a screw-top plastic bottle that has a narrow, tapered opening, such as a condiment dispenser. Alternatively, cut off the tapered end of a rubber-bulb suction syringe or stethoscope head.[25,130]

A slightly more complex method to make a precordial stethoscope uses a 20-mL syringe, a three-way stopcock, IV tubing, and an earpiece. Cut the 20-mL syringe 2.5 cm (1 inch) from the infusion end (Figure 46-5A). Then, smooth the edges with a file, and place adhesive tape around the cut end of the syringe. Remove the plunger's rubber sealer (Figure 46-5B), cut a large hole in its center (Figure 46-5C), and insert the rubber piece into the small piece cut off the syringe barrel (Figure 46-5D). After connecting this to a three-way stopcock, connect the stopcock to IV tubing (or other tubing) and then to an earpiece (Figure 46-5E).[19]

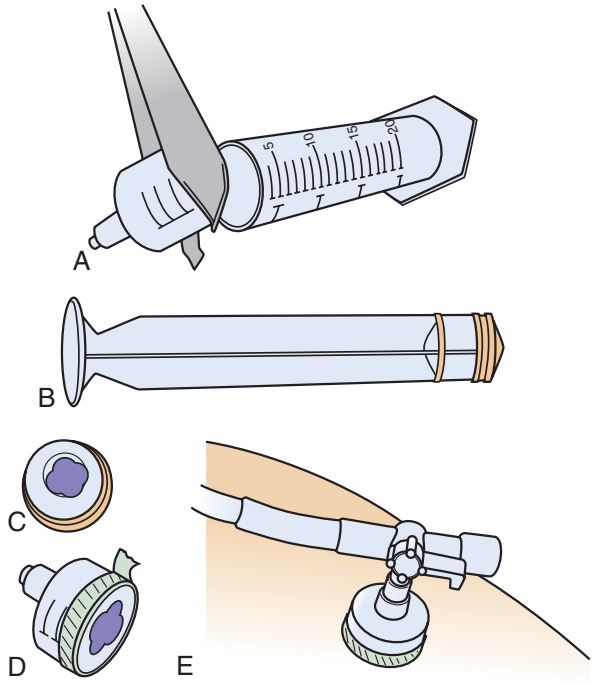


FIGURE 46-5 Improvised precordial stethoscope. See text for explanation.

Stethoscope Earpiece

To replace a stethoscope earpiece, use the nipple from a baby bottle or pacifier (Figure 46-6A) or the rubber bulb from a medicine or eye dropper. Make the normal pinhole opening in the nipple slightly larger, and tie the nipple in place on the stethoscope. Cut the nipple so that only the distal 1 to 2 cm (0.4 to 0.8 inch) of the rubber piece is used (Figure 46-6B).[59] In compact medical kits, some opt simply to bring the bell of a stethoscope without the standard rubber tubing and earpiece assembly; should a stethoscope be needed, the bell can be hooked to the open end of a Foley catheter and the balloon end inserted into the listener's ear (Figure 46-6C). In our experience, however, this does not provide adequate auscultation.

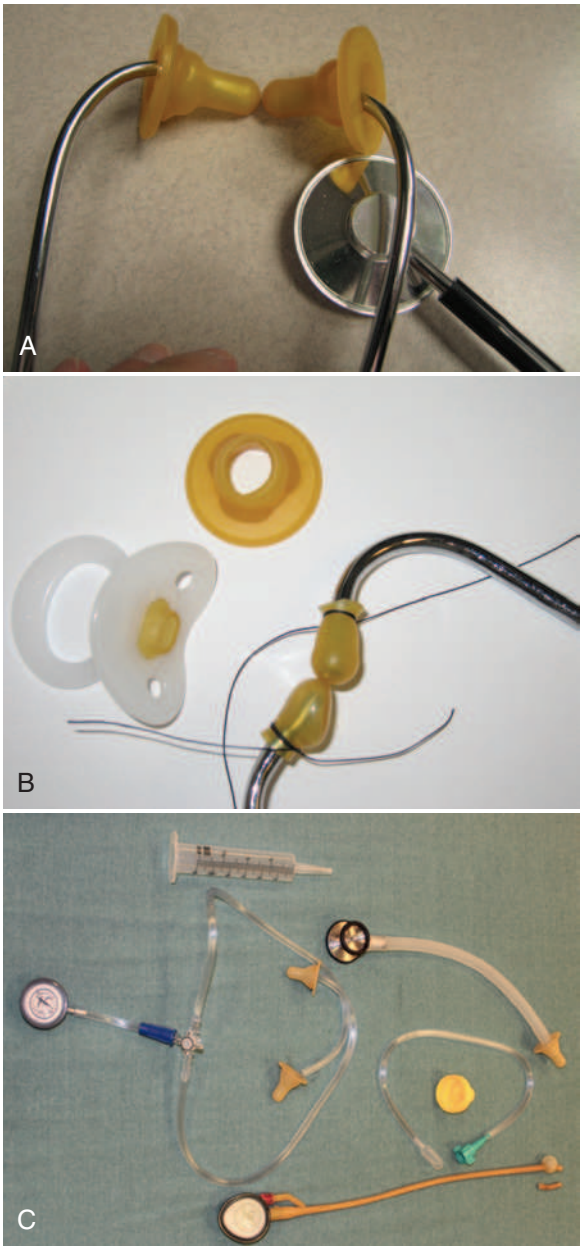


FIGURE 46-6 **A** and **B,** Earpieces for a stethoscope. **C,** Examples of improvised stethoscopes. Note the Toomey syringe on the top left is improved when a clear piece of plastic is stretched over the larger end. Also note that the tip of the end of the Foley catheter must be cut, allowing the balloon to go into the ear.

IMPROVISED TREATMENT EQUIPMENT AND SUPPLIES

GOWNS, GLOVES, MASKS, BOOTIES, AND GOGGLES

Standard Precautions

In an austere environment, the basic protective gear used for standard or universal precautions often must be improvised rather than purchased.

Gowns

A plastic garbage bag with head and arm holes cut out is the simplest waterproof gown and can be enhanced with arm pieces cut from another bag and stapled or taped in place. Although not waterproof, a sheet cut like a poncho will also work. Using a safety pin or a tie to cinch the waist can make the poncho less cumbersome.

Caps and Masks

Use masks to protect the examiner and patients against particulate matter, virulent organisms, and nauseating smells. Tincture of benzoin dabbed on one's mask covers up most bad smells. Caps protect patients' wounds from the examiner's hair. The most basic masks are towels or a cloth held to the mouth and nose, either with one's hand or tied in place. Make a basic face mask with a clean cloth or balaclava. The effectiveness depends on the cloth, with dish towels being one of the best materials. However, no improvised mask used alone is adequate for use with the most virulent organisms (e.g., Ebola virus).

Eye Protection

Put tape around the edges of standard eyeglasses to help protect eyes from splattered blood and fluids. An alternative is to wear ski or woodworking goggles, separately or over eyeglasses.

Booties, Shoes, and Vapor Barrier Liners

Fabricate waterproof booties from a plastic bag and strong tape. Attach a flat piece of wood or tire rubber with tape for traction and sole protection. A similar mountaineering trick for cold feet that rescuers can also use for a victim during transport in cold conditions is to have them don a thin wicking sock covered with a plastic bag, followed by an insulating sock and boot. Whenever using plastic bag booties for long periods, wear a moisture-wicking sock or aerate/dry the foot often to prevent maceration.

SYRINGES, NEEDLES, AND INTRAVENOUS EQUIPMENT

Saline Locks

To quickly make a saline lock, slip the rubber end of the plunger from a 2- or 3-mL disposable syringe over the end of an IV catheter (Figure 46-7). This also works on a straight needle if it needs to be used as an IV catheter. Fill the catheter with saline (or heparin) as would normally be done.[65]

Intravenous Tubing

One way to adjust IV flow rates is to wrap a small piece of malleable metal (e.g., heavy aluminum foil) around the tubing. Tighten it around the tubing to achieve the desired flow rate.

Pressure for Intravenous Fluids and Blood Bags

Hands-off ways to increase an IV or blood bag's external pressure include inflating a BP cuff around it or wrapping it with an elastic bandage, belt ("stretchy" if possible), nylon stockings, rope, or similar materials. The IV bag can also be placed under an adult patient; body pressure keeps the liquid flowing.

IMPROVISED AIRWAY MANAGEMENT

(See Chapter 19)

Because establishing or maintaining a patient's airway is the most fundamental lifesaving skill, clinicians must be able to do this despite limited equipment.

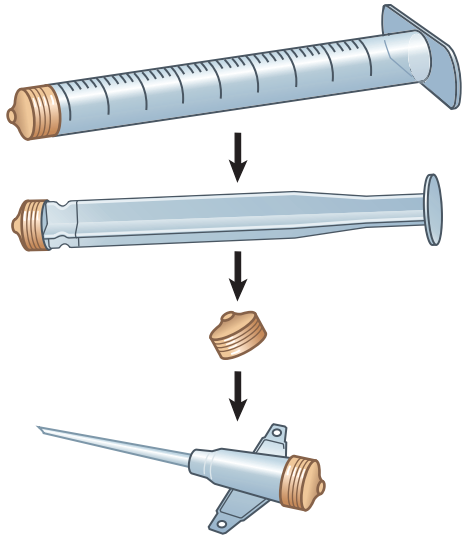


FIGURE 46-7 Improvised saline lock.

POSITIONING FOR SAFE AIRWAY

Preventing aspiration may be all that is necessary in many patients to maintain an airway, especially in the presence of copious secretions or vomiting. Use the "rescue" or "recovery" position, with patients on their side and face aimed downward (Figure 46-8). If possible, elevate the feet slightly so that the patient is in the head-down position.

OPENING THE AIRWAY

Chin Lift–Jaw Thrust

For many patients with diminished consciousness, simply elevate the chin to keep the airway patent (Figure 46-9). If a cervical spine injury is suspected, the alternative is to push the jaw forward from the mandibular angles.

Head Turn

When cervical spine injury is not a concern, turning the patient's head to one side is a simple but rarely used procedure to open an airway. In patients with a significant amount of redundant tissue in the pharynx and hypopharynx, this procedure usually improves airflow.

Positioning the Tongue

If a suspected cervical spine injury prevents head repositioning, or if the patient's tongue still blocks the airway after positioning the head, grasp the tongue with gauze and pull it forward. If this opens the airway, as it usually does, put a heavy (e.g., 2-0) suture vertically through the tip of the tongue in the midline (Figure 46-10A), or use a wire fishing line (or similar material) or safety pin (Figure 46-10B). This placement avoids significant bleeding. Initially, have someone hold the suture; if needed for long-term use, pass it through the skin of the lower lip and tie it. Do not use a clamp or forceps that "may in the excitement

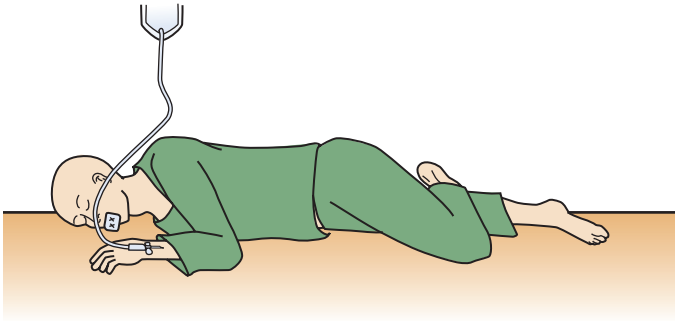


FIGURE 46-8 Rescue/recovery position.

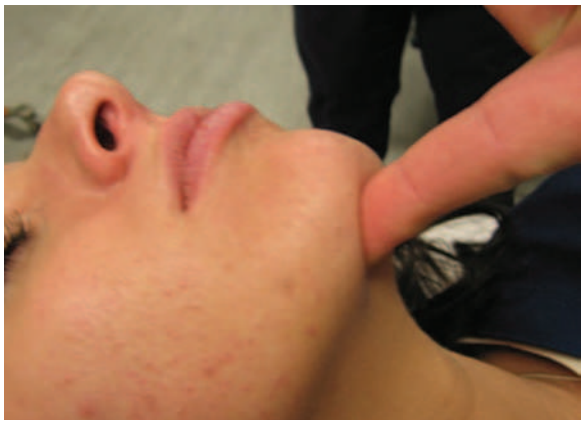


FIGURE 46-9 Chin lift.

of the moment be so firmly applied as to nip a piece out of the tongue."[40] These techniques, often used in the operating room, cause minimal complications.[60]

Nasal Airways

A nasal airway "trumpet" is effective and easy to improvise. Put a safety pin through the end of any soft piece of rubber tubing (e.g., Foley catheter, uncuffed endotracheal tube, radiator hose, solar shower hose, siphon tubing, inflation hose from kayak flotation bag or sport pouch) that is the appropriate size for the patient's nostril (Figure 46-11). In adults, the tube's length beyond the safety pin equals the length from their nare to the meatus of the ear (11 to 13 cm [4.5 to 5 inches]).[40] This "Goldman's airway" can be made to fit any nose. Consider using nasal airways bilaterally. When placing a nasal airway, lubricate it well, and pass it along the floor of the nose (straight back, not cephalad).[64]

MOUTH-TO-MOUTH RESCUE BREATHING BARRIER

A glove can be modified and used as a barrier shield for performing rescue breathing. Cut and discard the middle finger of the glove near its halfway point. Insert the cut finger part of the glove into the patient's mouth. Stretch the rest of the glove across the mouth and nose, and blow into the glove as if inflating a balloon. After each breath, remove the part of the glove covering the nose to allow the patient to exhale. The cut finger creates a one-way valve, preventing backflow of the patient's saliva (Figure 46-12).

Surgical Airway (Cricothyrotomy)

Use a cricothyrotomy to relieve life-threatening upper-airway obstruction when a victim cannot be ventilated effectively from the mouth or nose and endotracheal intubation is not feasible.

BOX 46-1 Improvised Cricothyrotomy Tubes

Intravenous (IV) *administration set drip chamber:* Cut the plastic drip chamber of a macrodrip (15 drops/mL) IV administration set at its halfway point with a knife or scissors. Remove the end protector from the piercing spike, and insert the spike through the cricothyroid membrane. The plastic drip chamber is almost the same size as a 15-mm endotracheal tube adapter and fits snugly in the valve fitting of a bag-valve-mask device (see Figure 46-17).

Syringe barrel: Cut the barrel of a 2- or 3-mL syringe with the plunger removed at a 45-degree angle at its midpoint to create an improvised cricothyroid airway device. The proximal flange of the syringe barrel helps secure the device to the neck and prevents it from being aspirated (see Figure 46-16).

Any small hollow object: Examples include a small flashlight or penlight casing, some pen casings, or small hollow tube. The key determination is whether sufficient air can pass through the tube for effective ventilation.

Modified from Iserson KV: *Improvised medicine: Providing care in extreme environments,* New York, 2012, McGraw-Hill.

This may occur in a patient with severe laryngeal edema or with trauma to the face and upper larynx. Cricothyrotomy may also be useful when a person's upper airway is obstructed by a foreign body that cannot be extracted by abdominal thrusts or direct laryngoscopy.

To perform a cricothyrotomy, cut a hole in the thin cricothyroid membrane and place an endotracheal tube (ETT) or, in austere circumstances, a suitable hollow object into the trachea to allow ventilation (Box 46-1). Locate the cricothyroid membrane by palpating the patient's neck, beginning at the top. The first and largest prominence felt is the thyroid cartilage ("Adam's apple"); the second prominence (below the thyroid cartilage) is the cricoid cartilage. The small space between these two, noted by a depression, is the cricothyroid membrane (Figure 46-13). With the victim lying on his or her back, cleanse the neck around the cricothyroid membrane with an antiseptic if one is readily available. Put on protective gloves. Make a vertical 2.5-cm (1-inch) incision with a knife through the skin over the membrane (extend slightly above and below the membrane) while using the fingers of your other hand to pry the skin edges apart. Anticipate copious bleeding from the wound. After the skin is incised and spread, puncture the exposed membrane by stabbing it with a knife or other sharp, penetrating object (Figure 46-14A). Stabilize the larynx between the fingers of one hand and insert the improvised cricothyrotomy tube through the membrane with the other hand (Figure 46-14B). Secure the object in place with tape.

If an ETT is unavailable, improvise a cricothyrotomy device (Figure 46-15). To use a 3-mL syringe, cut the barrel midway at a 45-degree angle, and insert it into the cricothyroid membrane

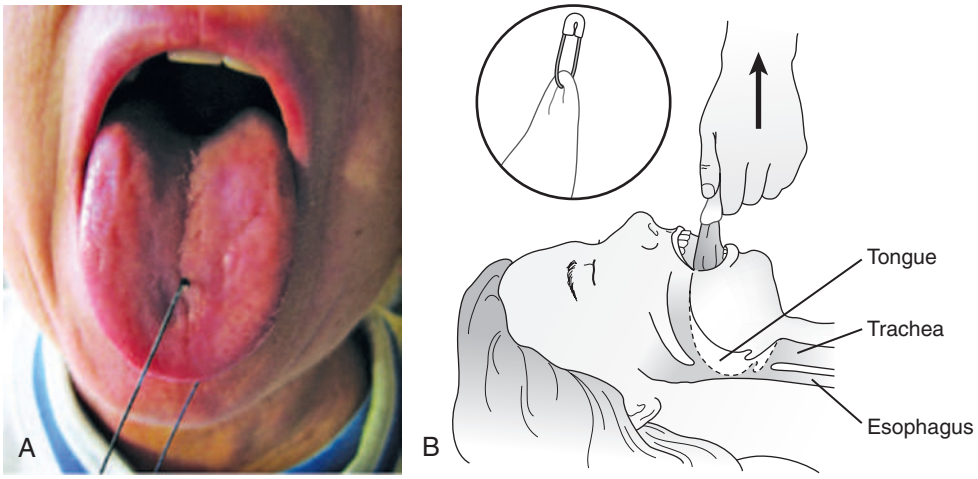


FIGURE 46-10 **A,** Suture used to hold the tongue out. **B,** Safety pin holding the tongue out. This can be pinned to the lower lip. Also shown is manual traction on the tongue using a cloth or gauze bandage. (***B** from Auerbach PS. Medicine for the outdoors: the essential guide to first aid and medical emergencies, ed 6, Philadelphia, 2016, Elsevier.)*

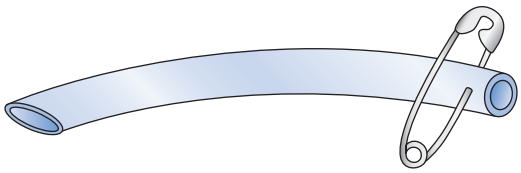
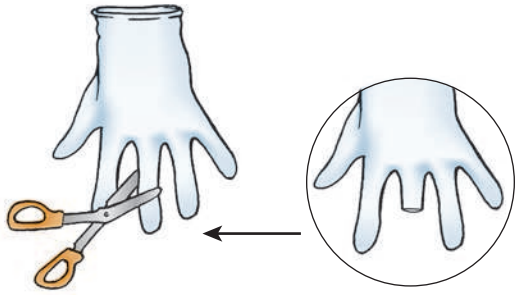


FIGURE 46-11 Goldman's nasopharyngeal airway.



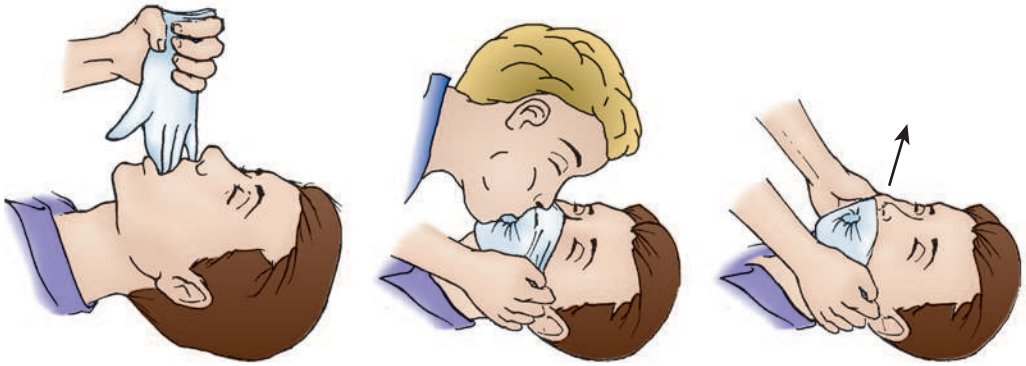


FIGURE 46-12 An improvised cardiopulmonary resuscitation (CPR) barrier is created using a latex or nitrile glove.

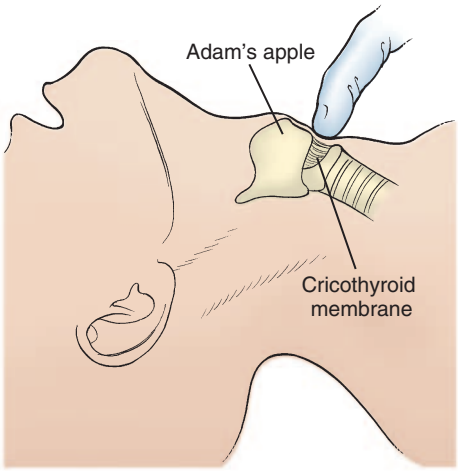
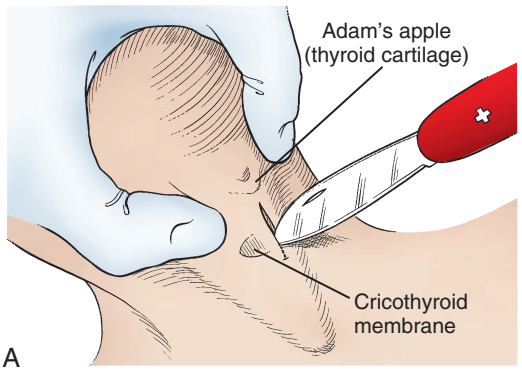


FIGURE 46-13 The cricothyroid membrane is found in the depression between the "Adam's apple" (thyroid cartilage) and the cricoid cartilage.



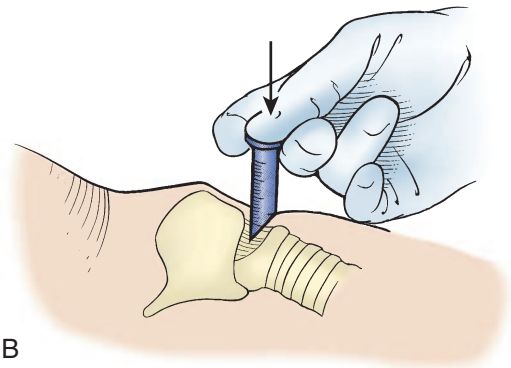


FIGURE 46-14 Cricothyrotomy. **A,** Locate the cricothyroid membrane, and make a vertical 2.5-cm (1-inch) incision through the skin. **B,** Insert the pointed end of an improvised cricothyrotomy tube through the membrane.

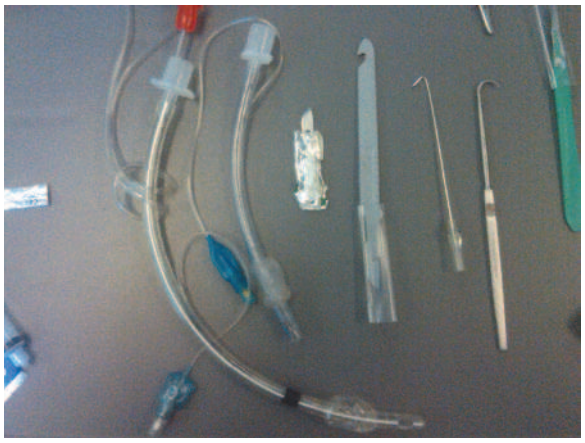


FIGURE 46-15 Improvised devices for a cricothyrotomy.

(Figure 46-16). The flange end can help secure this tube, and an adapter from an adult-sized ETT couples the bag-valve-mask (BVM) device to the syringe. Note that IV drip chambers used for cricothyrotomies do not fit well with modern BVM devices, and the recommended ballpoint pen cricothyrotomies are not ideal because they are prone to cause hypercarbia, significant airflow resistance, and the air leaks common to all uncuffed devices.[49]

Complications associated with this procedure include hemorrhage at the insertion site, subcutaneous or mediastinal emphysema resulting from positioning the tube into the subcutaneous tissues, and air leaks from an uncuffed tube. To control hemorrhage, one may pack gauze around the tube at the insertion site.

Although a cricothyrotomy can be improvised, a BVM device cannot; bellows, bicycle pumps, empty water bottles, and similar items have been attempted with little success. For rescue or expedition work, carry small, compact, commercially available BVM devices (Figure 46-17).

OTHER ALTERNATIVES TO SECURE AN AIRWAY

An effective way to improvise a laryngoscope is to perform transillumination combined with a spoon. A spoon handle or other malleable device acts as a laryngoscope blade, and the larynx is transilluminated through the cricothyroid membrane using a bright flashlight, penlight, or headlamp (Figure 46-18).

If lacking a laryngoscope, use digital tactile intubation in an unconscious patient. Stand on the patient's right side, and insert your left index and middle fingers along the curve of the tongue to the epiglottis. Simultaneously, drag down the corner of the mouth with the intubating hand to allow even short fingers to reach the epiglottis. If the patient still can bite, use an oral airway to prevent finger injury. Slightly separate the two intubating fingers around the epiglottis. Insert an ETT with a stylet bent at an acute forward angle, or use a bougie, and slide it between the fingers entering the trachea (Figure 46-19).

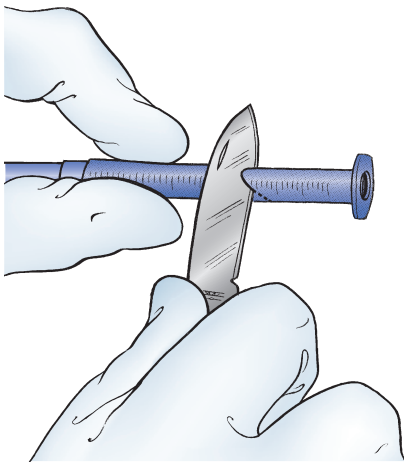


FIGURE 46-16 Improvised cricothyroid airway device can be created by cutting the barrel of a syringe at a 45-degree angle at its midway point.

FIGURE 46-17 Improvised cricothyrotomy tube with bag-valve-mask (BVM) device. A plastic drip chamber is cut at the halfway point, and the spike from the drip chamber can be inserted through the cricothyroid membrane.

IMPROVISED WOUND MANAGEMENT

The same principles that govern wound management in the emergency department apply in the wilderness setting. The main problem faced in the wilderness is access to adequate supplies. In deciding whether to close a wound or pack it open, take into account the mechanism of injury, degree of contamination, patient's age, site of the wound, and ability to cleanse the wound effectively.

WOUND HEMOSTASIS

Initial wound management requires managing blood loss. Direct pressure and pressure dressings are the first-line treatments for hemorrhage; commercially available hemostatic bandages may help.[104] Although spider webs (containing Hageman factor, responsible for hemostasis) have been used in some cultures for centuries, there have been reports of infection, allergic reactions, and tetanus associated with contaminated webs.[105,133] Vasoconstricting sprays (e.g., 1 mL of 1:1000 aqueous epinephrine in 400 mL normal saline [NS]) or gels (e.g., 1 mL of 1:1000 aqueous epinephrine mixed with newly opened tube of K-Y Jelly) stop bleeding from large superficial wounds.[95] For severe extremity bleeding that fails to stop with direct pressure, use tourniquets at least 4 cm (1.5 inches) wide with a windlass mechanism (see Chapter 18).

WOUND ANESTHESIA

Anesthetize wounds with anesthetics by infiltration or topical administration, or with nerve blocks. For direct wound infiltration or use of field blocks around a wound, lidocaine or bupivacaine is typically used. If these are unavailable, consider using injectable medications with antihistamine activity (e.g., 1 mL of 5% diphenhydramine in 4 mL NS for injection), sterile water for injection, bacteriostatic NS (alone or 0.2 mL epinephrine 1:1000 mixed into

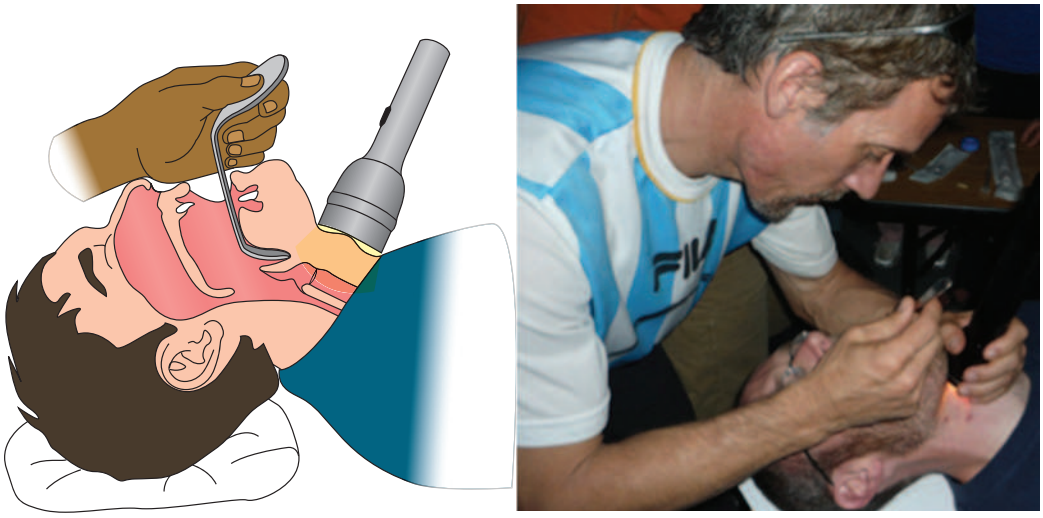
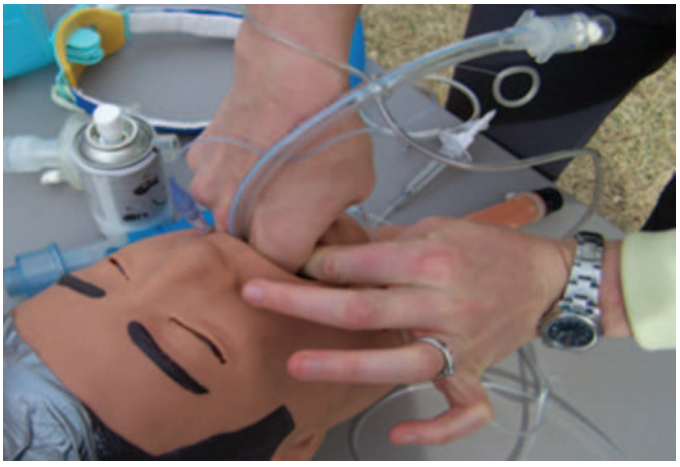


FIGURE 46-18 Intubation using flashlight transillumination.



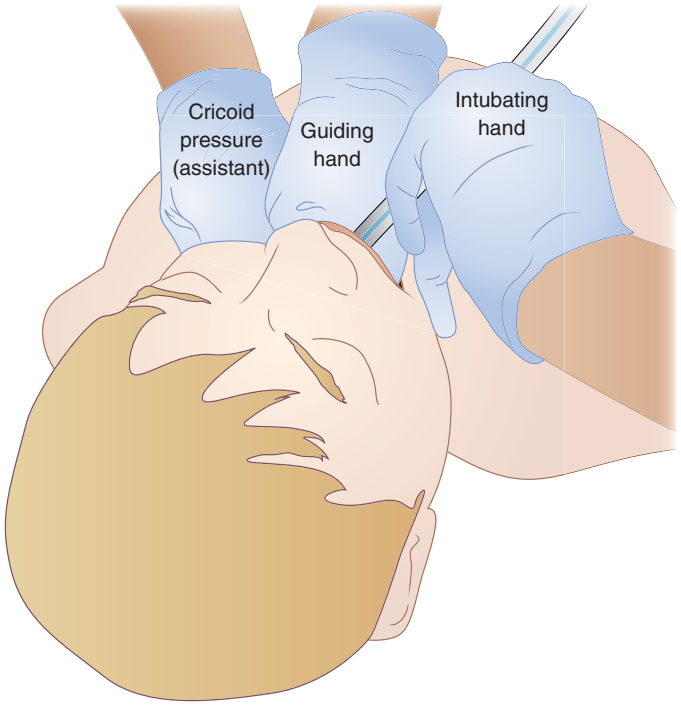


FIGURE 46-19 Digital tactile intubation.

a 20 mL vial of NS containing benzyl alcohol 0.9%), or opioids.[66] Use these alternatives only if a standard anesthetic is unavailable.

Although using ice as a topical anesthetic to decrease pain before needle injection is effective, our experience using it for laceration repair is disappointing.[79] In addition, applying ice may be detrimental to crushed tissue. Vapocoolant sprays, such as ethyl chloride, are ineffective for suturing. LET (4% lidocaine, 0.1% epinephrine, and 0.5% tetracaine) could conceivably be compounded locally and applied topically to a wound. Put it on simple lacerations for 20 minutes to achieve anesthesia.[34,135] Cocaine (maximum 4 cc or 4% solution) can also be used topically, even on mucous membranes.

WOUND IRRIGATION

The primary determinants of wound infection are bacterial counts and amount of devitalized tissue remaining in the wound.[30] Ridding a wound of bacteria and other particulate matter requires more than soaking and gentle washing with a disinfectant.[80] Irrigating the wound with a sufficiently forceful stream is the most effective method of reducing bacterial counts and removing debris and contaminants.[115] The cleansing capacity of the stream depends on the hydraulic pressure at which the fluid is delivered.[29] Irrigation is best accomplished by attaching an 18-gauge catheter to a 35-mL syringe. This creates hydraulic pressure in the range of 0.49 to 0.56 kg/cm^2 (7 to 8 psi).[106] The solution is directed into the wound from a distance of 2.5 to 5 cm (1 to 2 inches) at an angle perpendicular to the wound surface. The amount of irrigation fluid varies with the size and contamination of the wound but should average no less than 250 mL.[38]

Potable tap water (and presumably boiled/cooled water) has been found to be as effective as sterile saline for irrigating wounds. In one study, the infection rate was significantly lower after tap water irrigation.[3] However, potable tap water is often unavailable in sufficient quantities in remote settings.

WOUND CLOSURE

Before a wound is closed, remove all foreign material and obviously devitalized tissue, being careful to leave a manageable wound. Debridement can be accomplished using scissors, knife, or any sharp object. Close the wounds with sutures, staples, tape, safety pins, or glue.

Wound Glues

Tissue glue is ideal for backcountry use because it precludes the need for topical anesthesia, is easy to use, reduces the risk for needlestick injury, and requires less space than a conventional suture kit. When applied to the skin surface, tissue glue facilitates excellent wound healing and peels off in 4 to 5 days without leaving evidence of its presence.[39] It provides a faster and less painful method for closing lacerations than does suturing and

BOX 46-2 Technique for Gluing Lacerations

1. Irrigate the wound with copious amounts of disinfected water.
2. Control any bleeding with direct pressure. Place a gauze pad moistened with oxymetazoline (Afrin) nasal spray into the wound to help control bleeding.
3. Once hemostasis is obtained, approximate the wound edges using fingers or forceps.
4. Paint the tissue glue over the apposed wound edges using a very light brushing motion of the applicator tip. Avoid excess pressure of the applicator on the tissue, because this could separate the skin edges, forcing glue into the wound. Apply multiple thin layers (at least three), allowing the glue to dry between each application (~2 minutes).
5. Glue can be removed from nontissue surfaces with acetone, or loosened from skin with petroleum jelly.

has yielded similar cosmetic results in children with facial lacerations (Box 46-2).[102] Tissue glue evokes a mild acute inflammatory reaction with no tissue necrosis.[124]

Although cyanoacrylate ("superglue") is the most common tissue adhesive, clinicians can use other common glues for wound closure. These include wood glue, panel adhesive, hobby cement, and various native (e.g., plant) substances. Be aware, however, that (1) these common glues may irritate the tissues, (2) the incidence of dehiscence is higher, and (3) they may contain toxins. Tincture of benzoin is useful as an indirect tissue adhesive, securely fixing tape to skin. If benzoin is unavailable, obtain the same effect by saturating standard alcohol wipes with povidone-iodine. As the alcohol evaporates, the remaining residue is similar to tincture of benzoin.[101]

Cyanoacrylate. In austere circumstances, cyanoacrylate's advantages for wound closure are that (1) it is readily available, (2) it can be applied rapidly, (3) it requires little technical knowledge, (4) a small tube can be used for many patients, (5) it can be used on many parts of the body, and (6) it can be applied over wound tapes rather than directly to the wound.

Cyanoacrylate's primary disadvantages are more frequent wound dehiscence because of its lower tensile strength compared with sutures, and contamination of other body parts. Using petroleum-based ointments and salves, including antiseptic ointments, on wounds closed with cyanoacrylate can weaken the polymerized film and cause wound dehiscence. One can lessen the incidence of dehiscence by not using cyanoacrylate on wounds that are under tension, such as over joints, and by using it only when a 5-0 or smaller suture would be appropriate to close a wound.[32]

Availability. Cyanoacrylate, which bonds surfaces almost instantly, is available in a wide variety of commercial brands for medical and nonmedical use. Carrying medical-grade cyanoacrylate is optimal. However, nonmedical "superglues" effectively hold wounds closed in a similar manner to medical tissue adhesives, without complications. Although they are not sterile, they are unlikely to create wound infections. Two benefits of medical-grade cyanoacrylate (2-octyl) are that its bond is about four times stronger than those of nonmedical products, and its more viscous gel form is better controlled when applied to wounds.[45] Nonmedical cyanoacrylate is often used to treat painful oral aphthous ulcers and superficial painful fissures of the fingertips ("polar hands"), which frequently occur in cold climates and at high elevations.

Contamination of Other Areas. If cyanoacrylate spreads onto the eyelids or lips during wound closure on the face, it can seal them shut for hours. Avoid this by positioning the patient so that any extra cement runs away from, rather than toward, the eyes or mouth. This may require the patient to turn the head away from the wound, lie on his or her side, sit up, or, if the wound is above the eye, lie in Trendelenburg position. If cyanoacrylate sticks to an unwanted area, use a petroleum-based product, such as ophthalmic bacitracin, erythromycin ointment, or mineral oil, to remove it. One may simply wait (usually <1 day), and the lids will open without treatment.[68]

Staples

Although staples are an effective way to close wounds quickly, they cannot be improvised. The best suggestion, if you know that you will be in an austere medical situation, is to carry a staple gun and the staples with you. They are lightweight and relatively inexpensive.

Binding and Taping

Skin tapes are useful for shallow, nongaping wounds and have several advantages over suturing, including reduced need for anesthesia, ease of application, decreased incidence of wound infection, and availability. Almost any tape can be used to close a wound. There is no magical quality to medical tapes. Other than cleaning and debriding a wound, using tape requires no technical expertise and is fast, painless, and virtually cost free. Duct tape or any adhesive tape can be used to close a wound. Cut it lengthwise into strips, and allow gaps during placement if it is necessary to have part of the wound visible (Box 46-3). Puncturing holes in the tape before application helps prevent exudate from building up under the tape.

Tapes do not stick well on areas that are hairy, wet, or prone to perspiring or that are under tension, such as joints. As with medical tape, use benzoin, cyanoacrylate, or another adhesive to help the strips stick. Once wound tapes are applied, applying wound glues over the tapes also helps them adhere to the skin. Alternatively, wrap a bandage around the closed wound, or use additional tape.

Suture Needles

To make a "swaged" suture needle (i.e., suture attached to the needle) from a hypodermic needle, first pass the suture through the needle from the sharp end. When the end of the suture appears, hold it in place and break off the hub by repeatedly bending it (Figure 46-20). Then, pull the suture through the needle so that only a small amount remains within the needle. Finally, crimp the "hub" end of the needle to fix the suture in place.[41] Do this at the patient's bedside, or prepare several in advance, wrapping the sutures around a piece of cardboard and autoclaving them en masse.[126] Newer safety needles may make this process difficult.

Another way to facilitate suturing is to insert the hypodermic needle through both wound edges. Thread the suture material through the beveled end of the needle, and pull the suture material through the hub, with enough remaining on the beveled side to create a knot (Figure 46-21A). Retract the needle; then cut and tie the remaining suture. Repeat the process to place interrupted sutures. To suture more quickly, after inserting the suture through the needle, withdraw the needle and carefully puncture the opposite side of the wound where you want to place the next suture. This leaves a loop of suture on that side (Figure 46-21B). Tie all loops to close the wound edges.

BOX 46-3 Wound-Taping Technique

1. Obtain hemostasis and dry the wound edges.
2. Apply benzoin or cyanoacrylate to the skin adjacent to the wound. Benzoin should be allowed to dry long enough for it to become tacky, but tape should be applied to the glue while the glue is still wet.
3. Tape should be cut to 6- to 13-mm (0.25- to 0.5-inch) widths, depending on the size of the laceration, and to a length that allows for 2 to 2.5 cm (0.75 to 1 inch) of overlap on each side of the wound.
4. Secure one-half the tape to one side of the wound. Appose the opposite wound edge using a finger while the tape is secured to the other side.
5. Wound tapes should have gaps of 1.5 to 3 mm (0.06 to 0.12 inch) between them to allow for serous drainage.
6. Cross-stays of tape can be placed perpendicular over the tape ends to prevent them from peeling off.
7. Additional glue can be applied to the tape edges every 24 hours to reinforce adhesion.

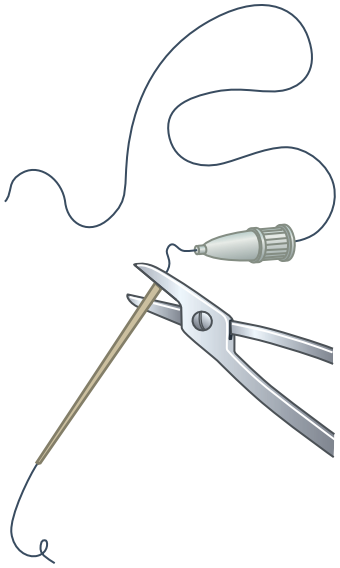


FIGURE 46-20 Swaging a needle.

FIGURE 46-22 Obtaining improvised suture material from a fibrous plant. Note the fibrils already teased out on one side while scraping a new segment of leaf with the blunt end of a knife against a hard surface.

Natural and Alternative Suture Materials

Fishing Line. Nylon fishing line is used routinely around the world as suture material. It emulates medical nylon suture material. It is easy to obtain, inexpensive, and often available in wilderness gear. Cut it to length, and disinfect it with alcohol before use.[42]

Horsehair Sutures. Hairs from the tail of a horse can also serve as effective sutures. First, wash the hairs with soap and water, followed by alcohol. Sterilize them before use in boiling water or steam. Horsehairs are not as strong as silk but are able to resist all the tension that any suture should.[38]

Silk and Yucca or Agave Fibers. Threads of natural silk or linen and flax make excellent sutures. Soak them in sterile water or wax them before use for better flexibility.[55] To obtain the fiber "threads" from yucca or agave plants, pull on the point of the leaf with your teeth, and the thread comes out. An alternative method is to use the blunt but rounded edge of a knife to tease out the fibrils (Figure 46-22).

Cotton Thread. Cotton and linen threads, long used as standard surgical sutures, are reasonable, nonirritating substitutes for standard suture materials. Disinfect the thread by boiling it. Boil any colored thread long enough to extract as much dye as possible.[39] In austere circumstances, a guideline is to use ordinary sewing cotton, No. 30, white, for heavier work on the abdominal wall, and No. 40 or No. 50, black, for finer work on intestine or delicate tissues.[100]

Dental Floss. Although dental floss is often cited as a good suture alternative, most dental floss is about the size of a 0 or 00 suture. This is far too thick for most wounds. Additionally, floss is produced from many different fibers (e.g., silk, nylon, Teflon), which may make it difficult to know how tissues will react.[27] Use floss only as a last resort.

Other Nontraditional Wound Closure Methods[5]

Hair Tying (with Glue) for Scalp Wound Closure. If a patient with a scalp laceration less than 10 cm (4 inches) long has hair over the area that is longer than 3 cm (1.2 inches), use the hair to close the wound. To do this, twist a few strands of hair together on each side of the laceration. Tie them and immediately place benzoin or a similar adhesive on the knot to hold it in place. Cut these knots out after the wound heals. Alternatively, pull several strands of hair across the laceration to the opposite sides, and twist them around each other one time. Place a drop of cyanoacrylate at the juncture (Figure 46-23). These "sutures" need not be removed; the cyanoacrylate gradually

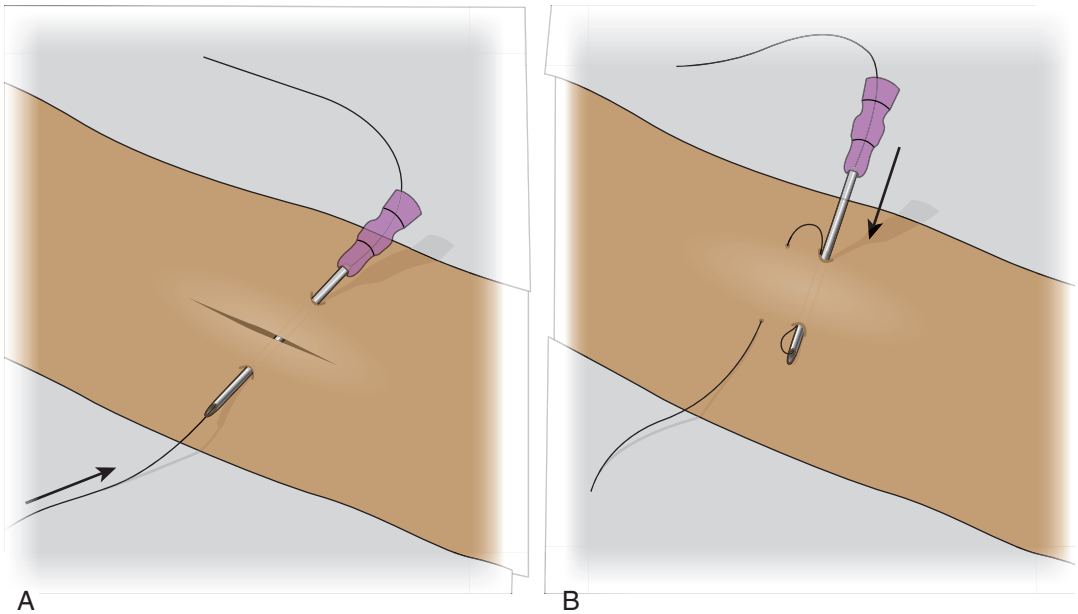


FIGURE 46-21 Suturing with thread and a hypodermic needle. **A,** Thread through the beveled end of needle, with the suture exiting out of hub; pull back on the needle partway, out of the wound, while stabilizing the suture. **B,** Puncture with the needle, and thread the assembly through the wound lateral to the first insertion site. One may withdraw the needle while retaining the loop on the inserted suture.

FIGURE 46-23 Wound closure with hair and cyanoacrylate.

wears off.[67] Tell patients not to wash their hair or put petroleum hair products near the wound for 48 hours.[96,98]

Chicken Egg Membrane. Use a fresh chicken egg membrane (the thin layer just inside the shell) to close wounds and reduce bleeding. The membrane contains types I, V, and X collagen; retains albumin; and prevents bacterial penetration. It has been used as a skin graft donor-site dressing and, in one patient, used to close a full-thickness lip laceration. It stopped the bleeding and, when removed after 3 days, revealed a well-healed wound.[134]

Disinfecting Rather Than Sterilizing Alternative Suture Materials

High-level disinfection of most alternative suture materials is more practical in austere situations than sterilization. Boil the material in water, or soak it in alcohol (medicinal, drinking), hydrogen peroxide, bleach, surgical or regular soapy water, or lemon juice. Note that some of the substances used to disinfect the suture (e.g., alcohol, bleach, lemon juice) may be painful if they enter an open wound; use local anesthetic if possible. If sterilizing the suture material is desired, wrap it in foil and put it in or near the coals from a fire. How well this works without destroying the suture material varies depending on the type of material used.

SCALPELS

Use any clean, preferably sterile, sharp object to perform surgical procedures (Figure 46-24). Pieces of metal can often be sharpened sufficiently to serve as scalpels. Make a scalpel from a common multiblade disposable razor by first carefully separating a blade from its plastic holder. Use pliers or a similar tool to bend one end; the blades pop out. Then, put a few drops of cyanoacrylate glue on both the blade and the utensil/holder; the blade can be affixed along the metal blade of a butter knife, handle of a spoon, or similar item that can be easily held and manipulated during a procedure. The blade should extend over the side of the handle and should be parallel to the other side for better control of the scalpel.[69] Wait 10 minutes for the cyanoacrylate glue to dry and set. Consider using the top of a tin can or edge of a beverage can. Scrape or burn off any paint and sharpen as needed.

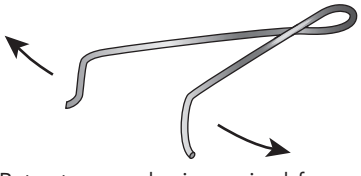


FIGURE 46-24 Retractors can be improvised from a safety pin with the end cut off and bent, or any type of wire bent over itself that retains a spring type of motion could suffice.

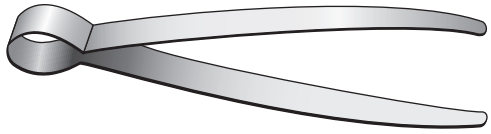


FIGURE 46-25 Surgical "pickups."

TWEEZERS

Fashion tweezers from pieces of thin metal strips, such as the metal straps that secure boxes for shipping. If folded with a slight bulge in the end, the improvised tweezers are less likely to break at the fold after repeated use (Figure 46-25). Use a file to make as fine a pointed tip as needed.

CLEANING AND REUSING MEDICAL SUPPLIES AND EQUIPMENT

Much modern medical equipment is labeled as "disposable," which is problematic in resource-limited settings. Caregivers do not want to put patients at risk by introducing infections from previously used equipment or by using malfunctioning equipment. Using appropriate cleaning, disinfection, and sterilization before reusing this equipment helps lessen any risk to patients.

CLEANING

Equipment must be cleaned before it can be properly disinfected or sterilized. Cleaning removes visible dirt and secretions, including dust, soil, large numbers of microorganisms, and organic matter (e.g., blood, vomit) on which microorganisms grow.[114] Immediately after use, clean equipment by washing it thoroughly with warm water without soap. Then, brush it thoroughly with warm water and soap, rinse with water, and dry completely. Clean surgical instruments with a small brush, such as a soft toothbrush. Leave scissors and forceps open during drying.[61]

DISINFECTION

Disinfection reduces the number of microorganisms to a level that is not harmful to health; the process does not necessarily kill or remove all microorganisms or bacterial spores.[114] The U.S. Centers for Disease Control and Prevention (CDC) recognizes three levels of disinfection: high, intermediate, and low[16]:

- High-level disinfection using a chemical germicide, such as bleach or ethyl alcohol, kills all organisms, except when there are high levels of bacterial spores.
- Intermediate-level disinfection kills mycobacteria, most viruses, and bacteria with a chemical germicide registered as a "tuberculocide" by the U.S. Environmental Protection Agency (EPA).
- Low-level disinfection kills some viruses and bacteria with a chemical germicide, such as soap.

Boiling

The best way to disinfect equipment in austere environments is to boil (100° C [212° F] for 10 to 30 minutes at sea level) it in clean water, with or without soap. Disinfection time begins when the water has come to a full rolling boil. The temperature at which water boils decreases at higher altitudes, so a longer boiling time will be required. In theory, the time should be increased by 5 minutes for each 300-m (1000-foot) rise in altitude. For example, at 4000 m (~12,000 feet) above sea level, water boils at 86° C (187° F), so boiling for 60 minutes or longer is required for disinfection. A way to increase the disinfection effect of boiling is to use "double boiling." Boil the instruments for 30 to 40 minutes. Let them cool, and then repeat the boil for another 30 to 40 minutes.[54] This process kills all organisms except bacterial spores.

Alcohols

Alcohols (e.g., methanol, ethanol, isopropanol) have good activity against bacteria and many viruses on previously cleaned equipment. Alcohol does not inactivate bacterial spores or hydrophilic viruses (i.e., poliovirus, coxsackievirus).[75] Use 70% or greater alcohol concentration, and have the equipment in contact with the alcohol for at least 10 minutes or longer; the longer the contact time, the better. To prepare a 70% ethanol solution, add 8 parts 90% ethanol to 2 parts water. To prepare a 70% isopropanol solution, add 7 parts standard isopropanol to 3 parts water.[121]

Povidone-Iodine

Povidone-iodine (PVP; Betadine) may be used for disinfecting instruments if nothing else is available. The CDC supports its use, citing manufacturer's data that it is fungicidal, virucidal, and bactericidal but may not be tuberculocidal,[108] and that it is not sporicidal. There is more active free iodine (as iodophors, the active ingredient) in disinfectants, such as 10% scrub and solution, than in antiseptics.[68] The recommendation is to add 1 part 10% povidone-iodine solution to 3 parts water and soak instruments for 15 minutes.[121]

DISINFECTING SPECIFIC ITEMS

"Sterile" Dressings

Clean and disinfect cloth for dressings by boiling it in water for 15 minutes or saturating it in alcohol. Let it dry before use.

Syringes and Needles

It is not possible to clean the interior of disposable syringes and needles well enough to use them safely on multiple patients. Although syringe reuse is a common practice throughout the world, it is so dangerous that it negates any good produced by medical treatment. Only when no other option exists should cleaning and reusing syringes be considered. If this is the case, the best option is one that the U.S. governmental agencies recommend for needles and syringes potentially contaminated with human immunodeficiency virus (HIV): they should be thoroughly cleaned and then immersed in full-strength bleach (5.25% sodium hypochlorite) for 10 minutes.[76,107] Presumably, the syringes and needles would be allowed to dry, although injecting small amounts of bleach is harmless.[43]

Single-Use Needle on Same Patient

With proper precautions, single-use needles can be used multiple times on the same patient. For example, diabetic persons routinely reuse the same syringe for up to 1 week. They do this by keeping the needle clean, storing it in a clean container, and ensuring that no one else uses it.

Surgical Instruments

Use alcohol or, if the equipment does not contain plastic or polymers, acetone to chemically sterilize sharp or delicate instruments. After this soak, rinse the items in hot, sterile water and let them dry.[112] Surgical instruments without a fine tolerance or sharp edges, such as mosquito clamps, towel clips, and large needle holders, may be disinfected in boiling water.[112] If you do not mind that sharp instruments will lose their edge, metal can be sterilized by holding it over a flame or, even better, by placing the instrument in a pot with a small amount of isopropyl alcohol (enough to create a small pool on the bottom of the pot). Ignite the alcohol and allow it to burn out, which takes about 5 to 10 minutes. The alcohol burns without leaving significant carbon residue. These techniques may be used when sterilization is really needed or when available resources or time do not allow for disinfection by boiling or soaking in alcohol.[62]

Endoscopes

Do not use iodophors, chlorine solutions, alcohols, quaternary ammonium compounds, or phenolics, because these lack proven efficacy against all microorganisms found on endoscopic instrumentation or have materials incompatibility.[108]

DRESSINGS AND BANDAGES

Dressings go directly on wounds and can be adherent or nonadherent, wet or dry, and absorbent or nonabsorbent. Dressings are often sterile. Bandages cover dressings, can hold them in place, and maintain pressure on the wound. Although most clean fabrics function well as dressings or bandages, avoid using paper products because they disintegrate when wet and leave fibers in the wound.

Dressings

Most clean, closed wounds do not need a dressing. Wounds without dressings permit easier inspection for infection or dehiscence, eliminate a moist, possibly anaerobic space over the wound, and make it much easier to keep the wound clean. Have the patient cleanse the wound with soap and water.

Dressing Material. Several types of dressings help to absorb serosanguineous drainage, keep wounds moist after small plastic closures, and keep air off open wounds to lessen pain. To soak up wound drainage, use any absorbent material, including menstrual pads and absorbent cotton fabrics. If nothing else is available, use moss, especially sphagnum (peat) moss, a traditional absorbent dressing. Make nonadherent dressings from sheer synthetic materials, such as parachute cloth or nylon stockings. Use any clean piece of plastic sheeting, such as a plastic bag, to fashion an occlusive dressing for open chest wounds. Using clean, rather than sterile, dressings will rarely affect the incidence of infection. Applying honey to burn wounds and lacerations is useful, given its known bactericidal and bacteriostatic effects.[9,94,125] Although sugar is also bacteriostatic, it can impair healing. Although honey and sugar seem to be similar, honey has a variety of antibacterial substances (inhibines) that sugar lacks.[128]

Bandages

Bandages hold dressings in place; if there is a dressing, a bandage is usually needed. The most expedient bandages are torn from expendable pieces of cloth, often clothing. If no cloth bandage is available, use duct tape to secure a dressing after first shaving the area to help the dressing adhere and to reduce pain on removal. Poke holes in most tapes (especially thick adhesives such as duct tape) to reduce moisture and allow fluids and sweat to escape. Clean the skin with soap and water, or alcohol, to remove oils. To make an adhesive dressing, cover a small square of dressing with a piece of tape. If you need to see the wound but still want it covered, make a transparent bandage from clear plastic wrap or a piece of clear plastic bag with its edges affixed to the skin with tape or cyanoacrylate.[58]

A pressure bandage over dressings, used when there is significant seepage or concern about hematoma development, is most easily constructed from an elastic bandage, bungee cord (use only over fabric stiff enough to distribute the cord's force), or the elastic from a piece of clothing or equipment.

BLISTER MANAGEMENT

To dress blisters without moleskin, use Molefoam, or improvise a blister dressing by using a piece of duct tape. Duct tape's smooth outer surface provides protection from friction, while its adhesive side adheres strongly to skin. Duct tape is ideal for preventing blisters when "hot spots" are present.

Do not apply duct tape directly to formed blister surfaces. Cover the blister with a nonadhesive dressing, such as a plastic sandwich bag, and use the duct tape or glue to hold it in place. Cut the corner of the sandwich bag and apply a lubricant between the two surfaces (Figure 46-26).

REMOVAL OF OBJECTS

RINGS

Always remove rings soon after finger and hand trauma, because progressive swelling may cause a ring to act as a tourniquet. Before trying to remove the ring, consider reducing distal finger

FIGURE 46-26 Blister dressing improvised with plastic sandwich bag.

edema by compressing it for 3 to 5 minutes with a removable tight wrap, such as a Penrose drain.[118] If you cannot remove a ring with the aid of soap or lubricating jelly, the string wrap technique can be used. Pass a 50-cm (20-inch) length of fine string, dental floss, umbilical tape, or thick suture between the ring and the finger. Pull the string so that most of it is on the distal side of the digit. Then, wrap the string distal to the ring around the swollen finger, beginning next to the ring and continuing past the proximal interphalangeal joint. Place successive loops of the wrap close enough together to prevent any swollen skin from bulging between the strands. Remove the ring by unwinding the proximal end of the string and forcing the ring over the distal string. If the string is not long enough, the technique may require repeated wraps (Figure 46-27).

EMBEDDED FOREIGN BODIES

Cactus Spines

Smaller cactus spines are more difficult to remove than larger ones. Larger spines can be removed with forceps. Smaller, hair-like spines (glochids) tend to be too difficult to remove individually. To remove them, apply several thin coats of household glue (e.g., Elmer's Glue-All; Borden, Columbus, Ohio). Allow each coat to dry before subsequent reapplications. Commercial facial gel and warmed depilatory wax left to cool on the affected area have also been effective.[86,111] Remove the entire coat en masse; use forceps to remove any remaining glochids (Figure 46-28). Although duct tape is sometimes recommended, our experience suggests that it is not effective for glochid removal.

Stingray Spines

Treatment for embedded stingray spines is discussed in Chapter 75.

TRAUMA

TENSION PNEUMOTHORAX

In dyspneic patients, signs and symptoms that suggest tension pneumothorax include distended neck veins, tracheal deviation away from the side of the pneumothorax, unilateral absent breath sounds, hyperresonant hemithorax to percussion, subcutaneous emphysema, cyanosis, and cardiovascular collapse. In the wilderness, one may save lives by treating these patients with rapid pleural decompression.

Improvised Pleural Decompression Technique

Swab the entire chest with povidone-iodine or another antiseptic. If sterile gloves are available, put them on after washing your hands. If local anesthesia is available, infiltrate the puncture site down to the rib and over its upper border.

Insert a large-bore IV catheter, needle, or other pointed, hollow, sharp object into the chest just above the third rib in the mid-clavicular line (midway between the top of the shoulder and the nipple in line with the nipple) or between the fourth and fifth intercostal space in the midaxillary line (Box 46-4). If you contact the rib, move the needle or knife upward slightly until it passes over the top of the rib, thus avoiding the intercostal blood vessels that course along the lower edge of every rib. The chest wall is 4 to 6 cm (1.6 to 2.4 inches) thick, depending on the individual's habitus; the needle should be long enough to penetrate the pleural space. A gush of air signals that the needle has entered the pleural space; do not push the penetrating object any further. In a noisy environment, a syringe partially filled with sterile water or saline can indicate the presence of air by visible

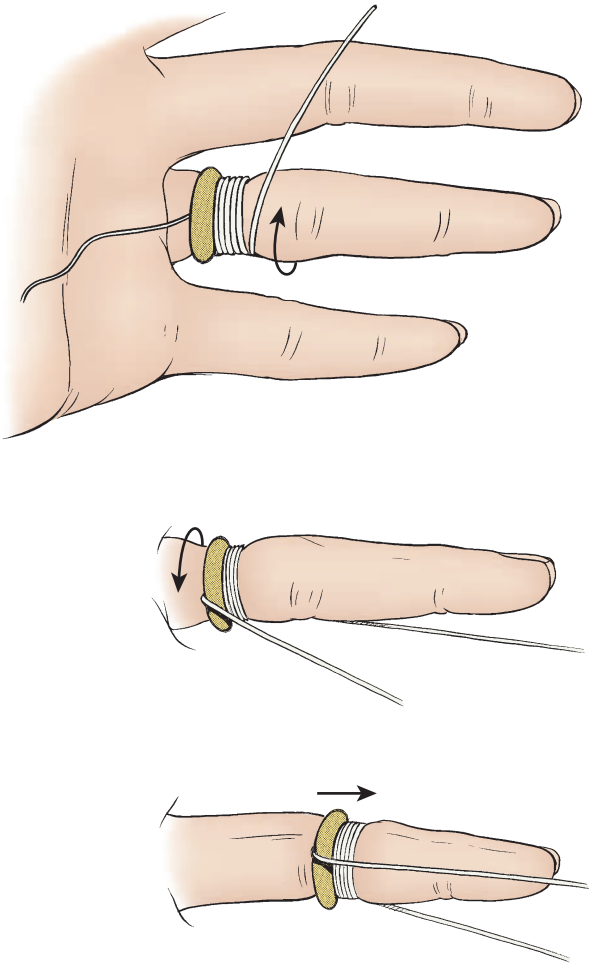


FIGURE 46-27 String technique for removing a ring from a swollen finger.

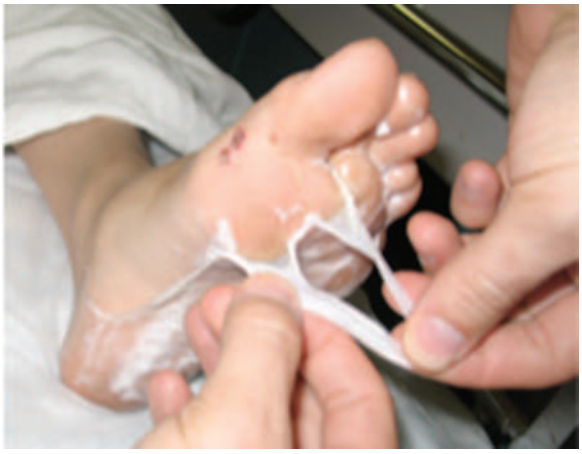


FIGURE 46-28 Technique for removing small cactus spines (glochids).

bubbling in solution or by easy retraction when the plunger is withdrawn as the needle is gently advanced (Figure 46-29). As of this writing, there are persons who advocate inserting the needle between the fourth and fifth intercostal space, in the midaxillary line.[16a]

Leave the needle or catheter in place (it often comes out of the pleural space with respiration, so continue monitoring it), and use a rubber glove to make a flutter valve (Figure 46-30). Ideally, the flutter valve (and syringe, if applicable) is assembled before initial decompression. Use the entire glove, and push a needle through one finger (best) or cut the finger from a rubber glove, making a tiny slit cut at the tip. Place this over the external opening to create a unidirectional flutter valve that allows continuous egress of air from the pleural space. To create a one-way flutter valve, cut one finger portion of a latex glove off at the proximal end of the finger, and insert the needle or catheter into the open end of the glove finger, through the tip as shown (see Figure 46-30A). The cut-out finger portion of the glove creates a unidirectional flutter valve that allows egress of air from the pleural space during expiration, but it collapses to prevent air entry on inspiration (see Figure 46-30B,C).

At best, this is a tenuous device that can easily slip out of place. If symptoms redevelop, insert another decompression needle. Keep in mind that an ETT and other semirigid devices work well as makeshift chest tubes. Foley catheters only work well in infants, however, and collapse in older children and adults because of pressure from the chest wall.[70]

OPEN ("SUCKING") CHEST WOUND

Penetrating trauma to the chest can produce a chest wound that allows air to be sucked into the pleura on inspiration. Place a piece of plastic food wrap or aluminum foil or one side of a plastic sandwich bag on top of the wound, and tape it on three sides. The untaped fourth side serves as a relief valve to prevent formation of a tension pneumothorax. In more urgent situations, simply seal all four sides and intermittently undo the dressing, or perform needle decompression as previously described.[14]

DRAINAGE SYSTEM (BOTTLES/BAGS)

Chest tube drainage systems allow air to leave the thorax (chest) without any air being able to reenter the chest through the system. A physician working in a remote area constructed a two-bottle chest tube drainage system from an empty 1-L plastic sterile water bottle, a 100-mL IV bag, two IV tubing sets, and a 24-French

BOX 46-4 Improvised Pleural Decompression Devices

Large-bore (12- or 14-gauge) intravenous catheter or needle
Endotracheal tube (instead of a chest tube)
Section of a tent pole
Hose from a hydration pouch

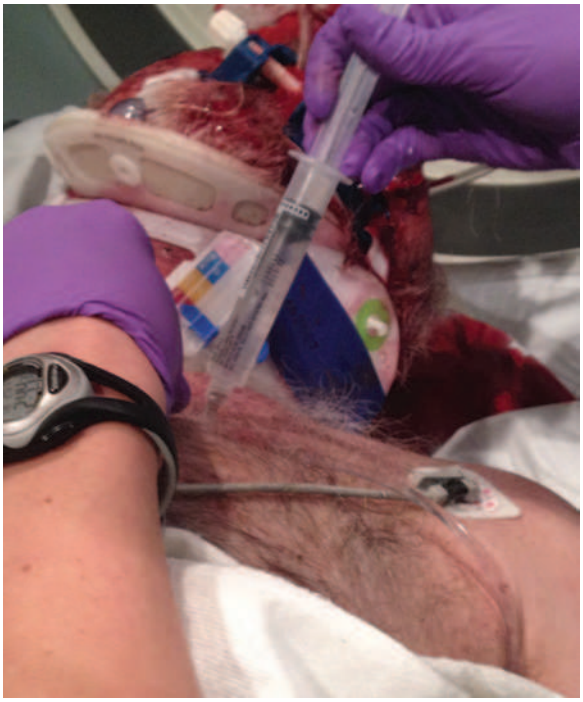


FIGURE 46-29 Fluid-filled syringe helps to identify bubbles on aspiration in the presence of a tension pneumothorax in noisy environments.

(24F) chest tube or ETT.[127] This system collects and measures fluid that drains out of the tube. To reproduce this system, empty a plastic sterile water bottle and put two holes in the bottle, one in the cap and one in the neck, each of which allows just enough room for passage of the IV tubing (Figure 46-31). Cut the patient end (IV catheter attachment) off one (the "first") IV tubing set, and insert this cut end into the hole in the top of the empty saline bottle. The other end (IV bag-spike) should fit into the end of the 24F chest tube. Take the other (the "second") IV

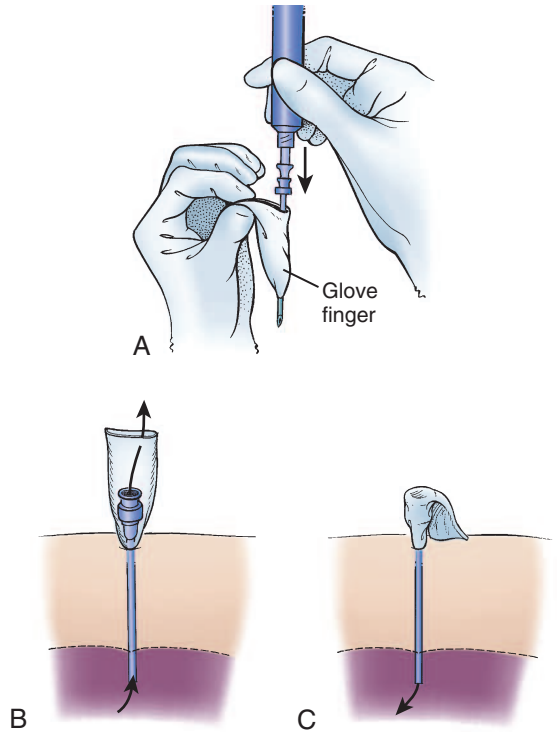


FIGURE 46-30 **A,** Finger of (or, even better, the entire) glove is attached to needle or catheter to create a flutter valve. **B,** Flutter valve allows air to escape. **C,** Flutter valve collapses to prevent air entry.

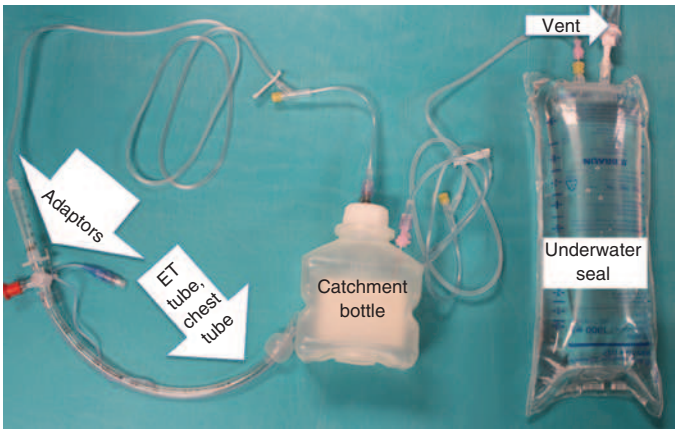


FIGURE 46-31 Sterile water bottle, IV tubing, and IV bag can serve as a chest tube drain. *(Modified from Vinson ED: Improvised chest tube drain for decompression of an acute tension pneumothorax,* Mil Med *169:403, 2004.)*

tubing set and cut off the spike end between the filter and the catchment bottle, or reservoir. Position the fluid-filled IV bag with ports up and the saline bag down, and insert the cut-off spike from the second IV tubing set into one port in the IV bag. This is now the system's air vent. Open the other port (cut or remove the rubber cap) and insert the cut end of the second IV tubing so that it is well beneath the level of the saline in the bag. Place the other end of the second IV line through the hole in the neck of the empty saline bottle. Use duct tape, cyanoacrylate, or any other cement to seal the tubes in position. Keep the IV bag inverted to provide a water seal for the system. To dispose of any accumulated fluid, clamp the chest tube, unscrew the cap from the water bottle, and empty it out. Then, screw the cap back on and unclamp the chest tube.[79]

IMPROVISED TREATMENT FOR ORTHOPEDIC INJURIES

GENERAL GUIDELINES

If time allows, test any improvised system on a noninjured person ("work out the bugs") before applying it to a patient, and continually reevaluate it while in use. Creativity is needed when searching for improvisational materials, although it helps to carry materials that lend themselves to improvisation, as described later. The patient's gear can also provide needed items, such as foam pads and the straps and internal stays from backpacks. Especially in patients with diminished consciousness, observe for pressure points and friction potential, because these devices are not ergonomically efficient. When in doubt, add extra padding.

SPINAL TRAUMA

Because of its mobility, the cervical spine is the area of the spinal column most often injured in trauma. Previously, many textbooks addressing the issue of spinal trauma stated that any obvious or suspected cervical spine injury demands full spine immobilization using both a rigid (or semirigid) cervical collar (C-collar) and long-board immobilization. Current protocols for prehospital complete spine immobilization have a strong historical or medicolegal, rather than scientific, underpinning, based on fear of imminent neurologic deterioration. Liberal immobilization policies do not stand up to current evidence showing that most injuries will have already occurred by the time rescuers arrive.[91] In addition, the time taken to do spine immobilization, as well as the added difficulties with patient extraction in an austere environment, may put rescuers at risk and cause patient injury.[83,103] Patient complications may include airway compromise, hypoxemia, and increased intracranial pressure.[47,77,87,90,109] Therefore, the priority should be safe and timely evacuation rather than fabricating or using an "optimal" spine immobilization system.

In many situations, however, spinal immobilization may be appropriate. When standard equipment is unavailable, consider using one of the following methods.

Improvised Cervical Collars

Patients self-extricating from vehicles with a C-collar in place have less cervical spine movement than when with full immobilization and professional assistance.[24,31] This suggests that in some instances, clinicians may have to fashion C-collars. The decision to immobilize the cervical spine is usually extrapolated from recognized decision guidelines, including the NEXUS[51] and Canadian Cervical Spine Rules that identify patients at greatest risk, at least when tested in urban prehospital emergency medical services (EMS) systems.[116,26,13]

Rigid "hospital-style" C-collars do not guarantee "perfect" cervical spine immobilization.[53] Therefore, the goal for an improvised collar is to protect the spine against axial loading, particularly during evacuations that involves tilting the patient's body uphill or downhill. An improvised C-collar works effectively if it includes the following features:

- Is rigid or semirigid
- Fits properly (many improvised designs are too small)
- Does not constrict or choke the victim
- Allows the victim's mouth to remain open if vomiting occurs

Closed-Cell Foam System. The best closed-cell foam systems incorporate a full-size or three-quarter-length pad folded longitudinally into thirds that is centered over the back of the patient's neck and wrapped forward. The pad is crossed under the chin, contoured underneath opposite axillae, and secured. Tape or tie on extensions if the pad is not long enough. This system also works well with blankets, beach towels, or even a rolled plastic tarp. Avoid small, flexible C-collars that fail to extend the chin-to-chest distance.

Padded Hip Belt. A padded hip belt or fanny pack removed from a large internal- or external-frame backpack can sometimes be modified to serve as a C-collar. Wider is usually better. Take up excess circumference by overlapping the belt, and secure the excess material with duct tape (Figure 46-32).

Clothing. Bulky clothing, such as a fiber pile or fleece jacket, can be rolled and then wrapped around the patient's neck to make a C-collar. Use the extended sleeves to secure the collar. Prewrapping a wide, elasticized (e.g., Ace) wrap around the jacket compresses the material and makes it more rigid and supportive.

Malleable Aluminum Splint. Well-padded aluminum splints (e.g., structural aluminum malleable [SAM] splint) can be molded into various configurations. With a thin aluminum core sandwiched between two layers of closed-cell foam, the splints can be adjusted to fit almost any size of neck. When properly applied, this splint is as effective as a standard Philadelphia collar.[89] This does not mean simply wrapping the splint around the neck, increasing chin-to-chest distance, and resulting in

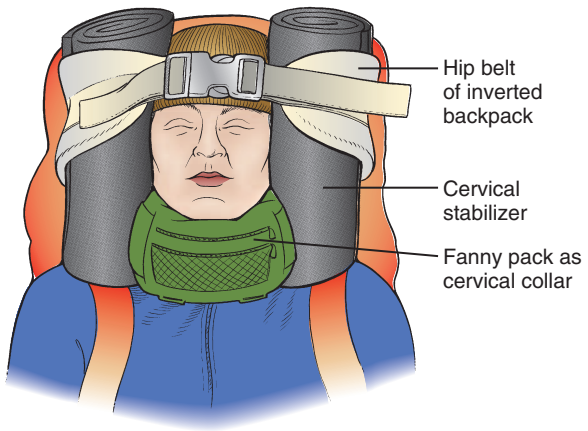


FIGURE 46-32 Inverted pack used as spine board. The backpack waist belt can be seen encircling the head.

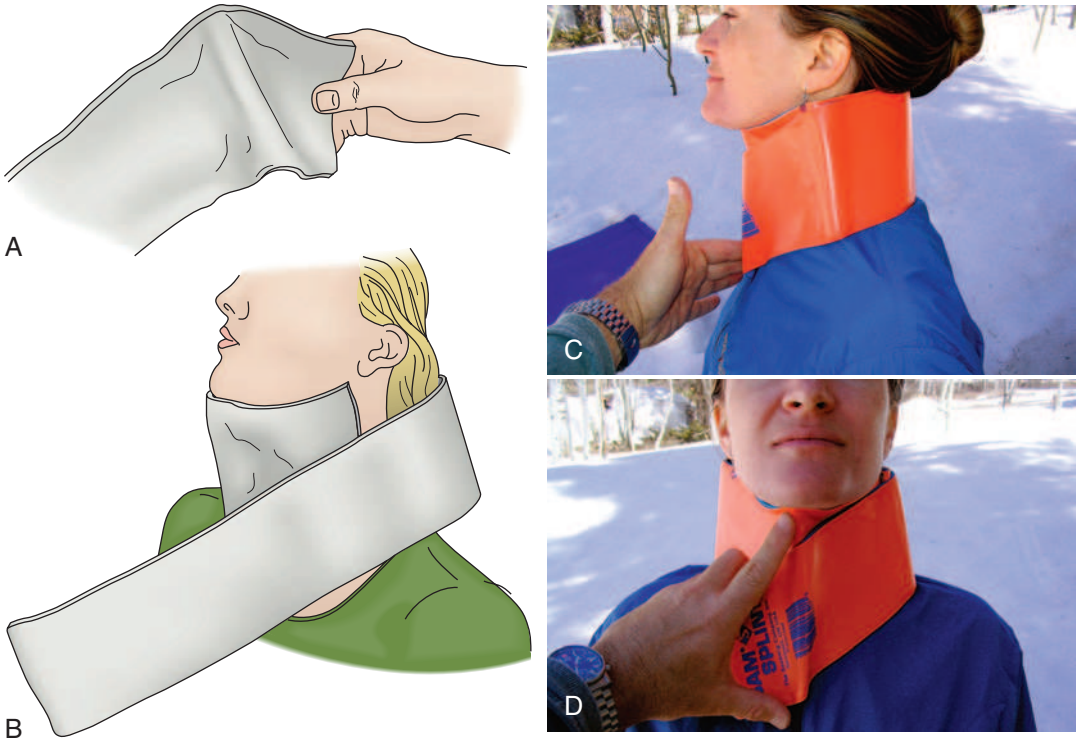


FIGURE 46-33 Padded aluminum-splint cervical collar (C-collar). **A,** Place a vertical bend in the malleable aluminum splint approximately 15 cm (6 inches) from one end to form a vertical pillar. Then, add bilateral flares to make the splint comfortable for the patient where it rests against the lower mandible. **B,** Place the anterior pillar securely beneath the patient's chin, and wrap the remaining length of the splint around the patient's neck. **C,** Side view of C-collar fashioned from a SAM splint. Note the formation of lateral, and if possible, posterior pillars. **D,** Frontal view. The end is angled inferiorly to provide an adequate chin-to-chest distance. Note formation of the anterior pillar.

excessive neck extension and patient discomfort. Rather, overlap the splint inferiorly, providing adequate chin-to-chest distance (Figure 46-33).

Improvised Spinal Protection Systems

As noted, an improvised C-collar may be used alone or with spine protection. Long- or short-board spine protection may still be indicated for suspected thoracolumbar trauma.[132] Two acceptable immobilization systems are (1) short-board immobilization, used for difficult or short-duration transport (i.e., removing patient from immediate danger) or used with a long board, and (2) long-board immobilization, used for definitive immobilization during extensive transport. The use of improvised spine protection devices have not been validated in the literature.

Several methods exist to improvise cervical spine protection with spine boards. These include lateral "towel rolls" constructed from small sections of closed-cell foam sleeping pads. Alternatively, make a U-shaped head support from any rolled garment, blanket, tarp, tent fly, or padded aluminum splint (Figure 46-34); secure it in place over the patient's head in an inverted U. Secured to the board, hiking socks or stuff bags filled with dirt, sand, or gravel also work well for this purpose. The patient's boots, filled with rocks, sand, or other heavy materials, can provide extra stabilization. If the patient requires rapid immobilization, quickly wrap duct tape around both the head and the spine board. Although snow-filled stuff bags can act as temporary support while more definitive systems are being constructed, never use them for long-term head support, because excessive head or neck motion occurs when the snow melts. If used as a temporizing measure, place insulation between the stuff sack and patient's skin to reduce cold-induced skin injury.

Short-Board "Immobilization"

Internal-Frame Pack and Snow Shovel System. Convert some internal-frame backpacks into a short spine board by inserting a snow shovel through the centerline attachment points. The shovel handgrip may need to be removed first. The patient's head is supported by the lightly padded shovel to which it is taped (Figure 46-35). Then, use this system's shoulder and sternum straps with hip belt to secure the patient. This method works

FIGURE 46-34 Improvised headrest.

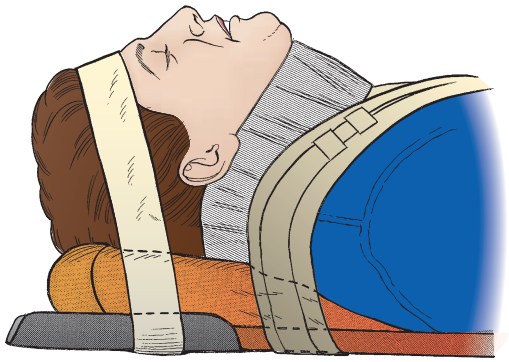


FIGURE 46-35 Head immobilized on a padded shovel.

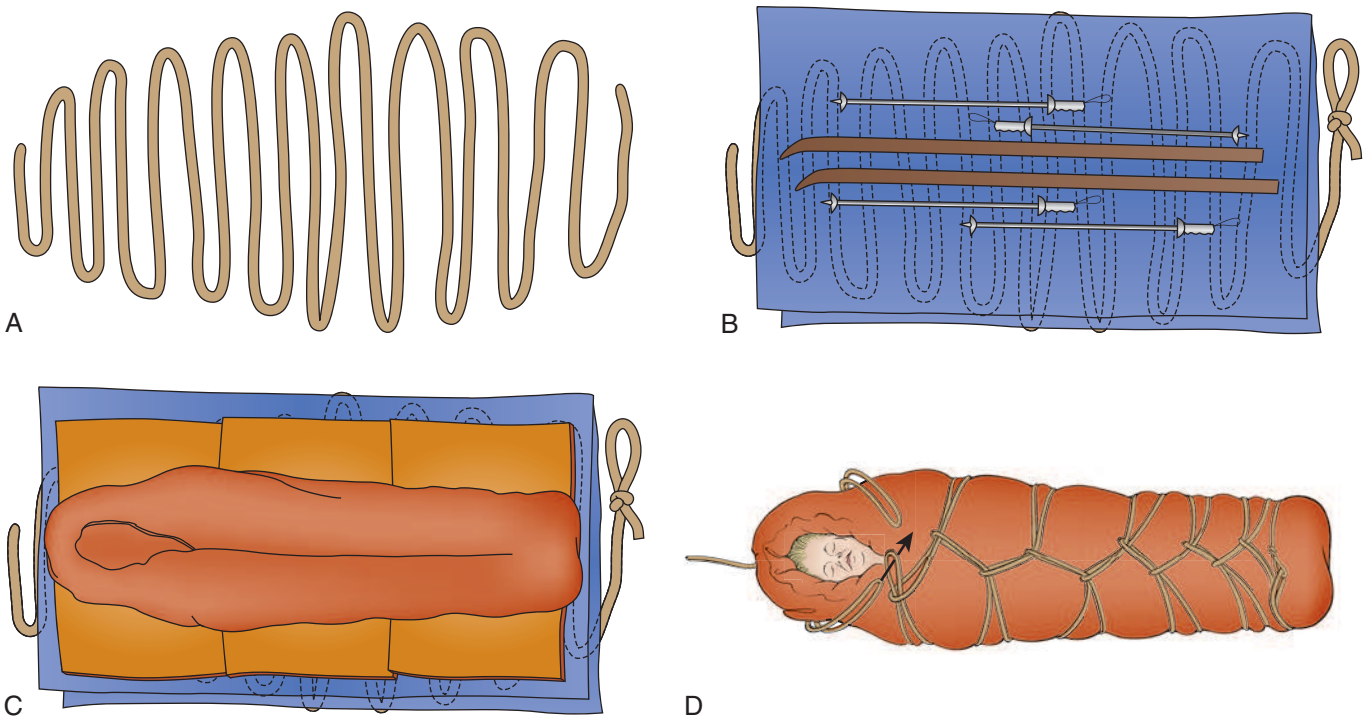


FIGURE 46-36 Continuous loop, or "mummy," litter made with a climbing rope. **A,** Rope is laid out with even U-shaped loops. **B,** Stiffeners, such as skis and poles, are placed underneath the patient to add structural rigidity. It is important to pad between the stiffeners and the patient. **C,** Sleeping bag may be used in addition to the foam pads. **D,** Loop of rope is brought over each shoulder and tied off.

well with other long-board designs, such as the continuous loop system (see later). Once in place, modify the system so that there is minimal cervical spine flexion or extension.

Inverted-Pack System. Make a short spine board using an inverted internal- or external-frame backpack. The padded hip belt provides head support while the frame becomes the spine board. Use this with a rigid or semirigid C-collar.

Turn the pack upside down, and lash the patient's shoulders and torso to the pack. Fasten the waist belt around the patient's head, as in the top section of a Kendrick extrication device. Because the hip belt is typically too long, reduce the excess with bilateral closed-cell foam rolls tucked under the belt and by wrapping the overlapping belt with duct tape. Unlike the snow shovel system, this system requires that the person be lashed to the pack. Reconfigure the pack's straps and buckles to further secure the patient (e.g., pack straps can be crisscrossed or simply tied with knots).

Snowshoe/Snowboard System. A snowshoe or inverted snowboard can be made into a fairly reliable short spine board. Pad the device, and attach it to the patient with available tape or straps.[38]

Long Spinal Protection Devices

Continuous Loop System. Also known as *daisy chain, cocoon wrap,* or *mummy litter*, this quick and efficient improvised system appears to provide some spine protection. It is useful for both short- and long-distance transports when preexisting planks, tables, or boards are not available. To construct this system, the following items are needed:

- Long climbing or rescue rope
- Large tarp (or tent fly)
- Sleeping pads
- Stiffeners (e.g., skis, poles, snowshoes, canoe paddles, tree branches)

To fashion the device, lay the rope out in even, U-shaped loops (Figure 46-36A). Make the midsection wide enough to conform to the patient's width. After tying a small loop at the foot end of the rope, place a tarp over the rope loops. If a tarp is unavailable, use a large poncho, tent fly, space blanket, or similar item. When using a short tarp, angle it so that the patient is positioned over its maximum length. In cold or wet conditions, place the tarp so that extra material can be folded up over the person's feet (i.e., hypothermia or "burrito wrap"). Lay foam pads over the full length of the tarp, overlapping pads to add length. Next, lay stiffeners on top of the pads in the same axis as the patient (Figure 46-36B). Add additional foam pads on top of the stiffeners followed, if available, with a sleeping bag (Figure 46-36C). Place the patient on the pads. To form the daisy chain, bring a single loop through the pretied loop, pulling the loops to the center, and feeding them through the loops brought up from the opposite side. It is important to continuously take up the rope slack. When the patient's armpits are reached, bring a loop over each shoulder and tie it off or clip it off with a carabiner (Figure 46-36D).

Backpack Frame Litters. Construct functional litters from external-frame backpacks. Traditionally, two frames are used, but combining three or four frames provides a larger, more stable litter (Figure 46-37). Use cable ties or fiberglass strapping tape

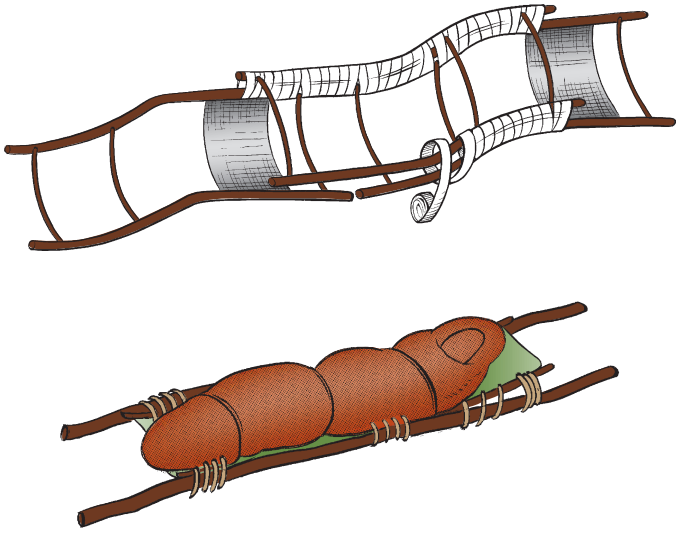


FIGURE 46-37 Backpack frame litter.

to secure them together, reinforcing the system with ice axes or ski poles.

Kayak/Canoe System. Properly modified, a kayak makes an ideal rigid long-board litter. First, remove the seat along with sections of the upper deck as needed; a serrated river knife or camp saw facilitates this improvisation. Alternatively, open-deck canoes can be used with minimal modification except for removing the flotation material. The canoe can also be used to (very carefully) transport patients along the railroad tracks that run parallel to many rivers. Place the canoe perpendicular to the tracks and slide it along by pulling on both bow and stern lines. Be certain to have an escape contingency plan for an approaching train, and post a lookout person for oncoming trains whenever possible.

EXTREMITY FRACTURES AND DISLOCATIONS

Physical Examination and Diagnosis

Lacking radiographs or other imaging capability, clinicians need to rely solely on physical signs and symptoms to diagnose fractures and dislocations. Table 46-2 lists the common signs and symptoms of fractures, ranking them according to their diagnostic usefulness.

Fracture vs. Dislocation. When using clinical findings, it may sometimes be difficult to differentiate between fractures and dislocations. In general, fractures, once reduced, do not remain in place without fixation, whereas the reverse may be true of dislocations: "As a rule, after reduction in the latter case, displacement will not again occur."[46]

Tuning Fork/Percussion Diagnoses. A myth perpetuated in the medical literature is that fractures can be diagnosed using a tuning fork and a stethoscope.[15,20,99,122] This has long been shown to be untrue. Stimson, for example, wrote in 1910 that even under the best circumstances, it added nothing to the diagnosis: "Auscultatory percussion, the stethoscope being moved from one fragment to the other while percussion is made upon the first, will sometimes give a marked change in the sound as the line of fracture is crossed; but it is rarely significant, except in cases in which the diagnosis can be made by other means."[117]

Shoulder Dislocation Diagnosis without Radiographs. To diagnose a shoulder dislocation, feel for the "hole" made by the now empty joint at the glenoid (Figure 46-38). If there is any

TABLE 46-2 Usefulness of Clinical Information for Diagnosing Fractures

Clinical Factor*	Diagnostic Information
Open fracture with observable bone fragments (10)	When bone fragments or fat that contains blood extrude from a wound, the question is not whether a fracture exists, but rather its extent and how best to treat it.
False point of motion (10)	This is definitive proof of a disruption in the bone and, after acute trauma, indicates a fracture. Its absence does not indicate anything. Testing for this is detailed below.†
Palpable discontinuity in bone (9)	This is an excellent indicator of bony discontinuity, especially in the patella, long bones, and diastasis of the symphysis pubis. Soft tissue defects, edema, and hematomas may limit palpation of or simulate this defect.
Deformity (8)	Presume diagnosis of fracture or dislocation if deformity can be discerned. This is more likely seen if the patient presents before there is significant edema or several days later when edema has resolved, and if the fracture is away from the joints, is angular, is not a partial (e.g., stress, torus) fracture, and is not a compression fracture. Old fractures and some chronic bone deformities, such as rickets, may appear to be a fracture but generally will not have the acute pain and skin tenting often seen after trauma.
Crepitus (8)	Crepitus or a grating sound, caused by contact of the broken surfaces with each other, is highly suggestive of a fracture. Crepitus is generally a rough, grating sensation. In fractures with many spicules, the sensation is often that of a click. Fractures that are badly comminuted, impacted, and days or weeks old may have no crepitus. Occasionally, chronic joint disease or blood clots near a suspected fracture may produce crepitus, so the sign is not perfect. Test for crepitus in the same way as for false motion.*
Shortening (7)	This is a very convincing sign of a long-bone fracture. Both extremities should be measured using easily identified landmarks (e.g., anterior iliac spine, medial malleolus). The difference may be 2.5 cm (1 inch) or more. Dislocations or prior injuries on either side may also cause shortening, but other signs can often differentiate between these. Measurements can also be used to determine if adequate traction has been applied. Note that the measurements must be taken accurately.
Loss of function (4)	Many injuries can limit the ability or willingness to use the extremity. There is often inability to lift the limb because of pain or separation of the bony lever on which the muscles act. However, this inability does not extend to all fractures, and especially does not exist in all partial or impacted fractures. Mild, nonsedating analgesics may help make the diagnosis by allowing a patient to better test the use of an extremity.
Pain and tenderness (1)	Unreliable sign. Often present, even severe, with only soft tissue injury; may be minimal or absent with a fracture.
Swelling (1)	Unreliable acutely. Swelling is such a common sign that it loses its diagnostic significance. If the swelling is out of proportion to the soft tissue injury or persists longer than such apparent damage would warrant, it may indicate a fracture. If deep swelling persists after the skin edema resolves, it often indicates a displaced bone or callus.
Ecchymosis (1)	Although ecchymosis is more common in fractures than in dislocations, even if it occurs acutely, it has minimal diagnostic value. If it persists over several days, it probably indicates ongoing bleeding from a deep (probably bony) vessel.
Altered percussion (1)	Although highly touted, listening for changes of tone across fractures produced by percussion or a tuning fork is grossly unreliable. False-positive and false-negative results are the rule rather than the exception.

Modified from Iserson KV: *Improvised medicine: Providing care in extreme environments,* New York, 2012, McGraw-Hill.
*10 = most useful; 1 = least useful.
†Testing for abnormal ("false point of") motion in an extremity, a pathognomonic sign of a fracture. To test for a false point of motion, grasp the bones firmly above and below the suspected fracture site. Gently manipulate the area to see if it moves (1) in an anteroposterior direction, (2) laterally, (3) so that one broken end slides over the other, (4) laterally, and (5) rotationally. The keys are to grasp the bones firmly and to use gentle manipulation.

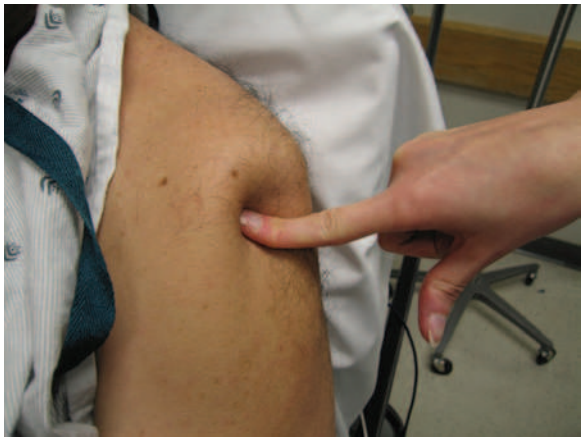


FIGURE 46-38 Digital diagnosis of shoulder dislocation.

doubt, feel the other side. Observe for flattening of the shoulder, inability to bring the elbow to the side of the body, and the head of the humerus in an abnormal position, usually below the coracoid process. Finally, if the arm is measured from the tip of the acromion to the external condyle, the dislocated arm will be shorter than the other, especially when the arm is abducted.[36]

Treatment

Reduction Treatment without Radiographs. There are three goals of fracture treatment:

1. Reposition bone fragments to eliminate any bone angulation and to restore limb length. This is particularly important if pulses are absent below the site of the injury or when the limb's position will interfere with patient transport. Whenever possible, undertake a dislocation reduction as soon as possible after the injury, that is, when muscle spasm and resistance are minimal. Try to minimize pain during this process by using local or regional anesthesia, hypnosis, sedation, or distraction.[79] Music, short videos, and vibration have all been successfully used as distractions (using a cell phone) for reductions in austere settings.
2. Immobilize the fragments by using splints and, if necessary, traction to keep the limb at length.
3. Restore function when possible. Evaluate the limb's neurovascular status before and after any manipulation.

Remeasure. Measure the affected limb and compare it to the opposite extremity before any manipulation. Repeat the process after manipulating any fracture where there has been shortening of the extremity. If the limb is still shorter than the contralateral limb, the reduction was unsuccessful. With successful shoulder relocation, a patient is usually able to touch the contralateral (opposite) shoulder with the ipsilateral hand. If the shoulder remains dislocated, attempt another reduction; if that fails, splint the limb.[37]

Shoulder Dislocation Reduction (see Chapter 22). Clinicians need to know several methods to reduce shoulder dislocations because these occur so often, and no single method has proved uniformly successful. In wilderness settings, it may be best to use nonsedating methods so that the patient can continue to function, or self-extricate. When sedation is needed, consider using an intraarticular injection with lidocaine (or similar agent).[92] Hypnosis works well for joint reductions.[57,72]

One easy method is the "Snowbird" traction technique. Have the patient sit facing the back of a chair (or on a rock, bicycle seat, or stool), with the affected arm draped over the chair's back, or over a low-lying tree limb. After placing padding under the axilla, flex the elbow to 90 degrees and drape a stockinet, sling, or other loop over the proximal forearm, letting the draped loop hang about 30 cm (1 foot) off the ground. Using the chair back or tree limb as countertraction, the clinician then places his or her foot in the loop and applies firm, steady downward pressure. In this way, the clinician's hands are free to apply pressure or to rotate the patient's arm (Figure 46-39). The success rate

FIGURE 46-39 "Snowbird" traction for anterior shoulder dislocation.

with this technique is reported as 97%; more than 90% of these procedures can be successful without sedation or narcotic analgesia.[131]

Another method is the Boss-Holzach-Matter method of autoreduction of an anterior shoulder dislocation.[73,25] The patient sits on a hard surface, clasping the hands together around the knee ipsilateral to the shoulder injury. Tying a band to both wrists to help keep the hands together may assist the patient to maintain this position. The patient then leans back with the neck in extension while the clinician instructs the person to perform an anterior shoulder shrug, facilitating relocation (Figure 46-40).

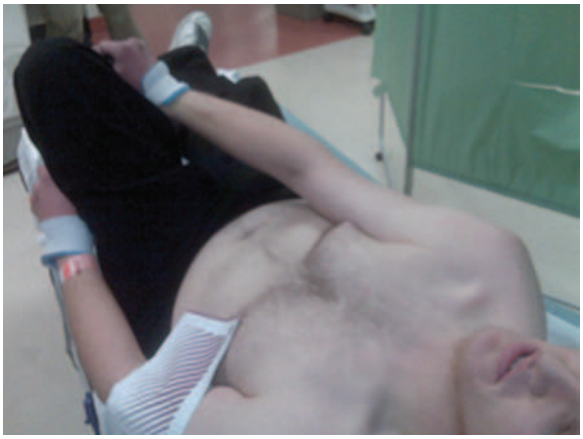


FIGURE 46-40 Patient performs autoreduction of an anterior shoulder dislocation.

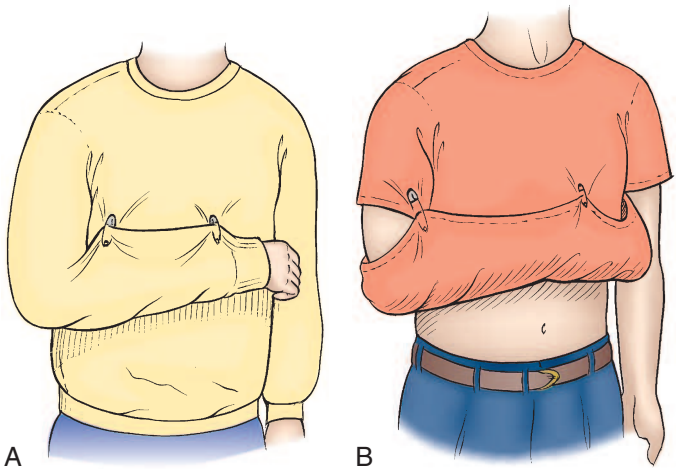


FIGURE 46-41 Techniques for pinning the arm to the shirt as an improvised sling. **A,** With a long-sleeved shirt or jacket, the sleeved arm is simply pinned to the chest portion of the garment. **B,** With a short-sleeved shirt, the bottom of the shirt is folded up over the injured arm and secured to the sleeve and upper shirt.

SPLINTING AND TRACTION METHODS

NONRIGID SPLINTS

Sling (Arm)

A sling is the most basic upper-extremity support. It can be improvised by putting the patient's wrist inside his or her shirt and buttoning around it. For more support, use a long-sleeved shirt and pin the sleeve containing the affected arm to the shirt's opposite shoulder (Figure 46-41A). Alternatively, fold the bottom of a short-sleeved shirt or a T-shirt up over the forearm and pin it to both shoulder areas (Figure 46-41B). Fashion slings from any available material (Figure 46-42). Looping the ends of a strip of material around the wrist and neck produces a "collar-cuff" sling, which allows movement of the shoulder and is ideal to avoid a "frozen" shoulder.

Although most splints are designed to immobilize an injured extremity completely, in the backcountry a splint may need to allow for limited range of motion so that patients can facilitate their own rescue. Many functional splints can be improvised quickly using nothing more than a closed-cell foam sleeping pad and tape or elastic wrap. Because foam pads are not as ubiquitous as they once were in the backcountry, splints also can be made using a partially inflated sleeping pad. Once applied, these pads can be inflated to provide the necessary support, fit, and comfort (Figure 46-43).

FIGURE 46-42 Webbing sling. A 2.5-m (8-foot) length of 2.5-cm (1-inch) tubular or flat webbing is used to form a functional arm sling. A Crazy Creek Chair can be used to improvise both upper-extremity and lower-extremity splints. Its inherent integral strapping system precludes the need for additional straps or tape.

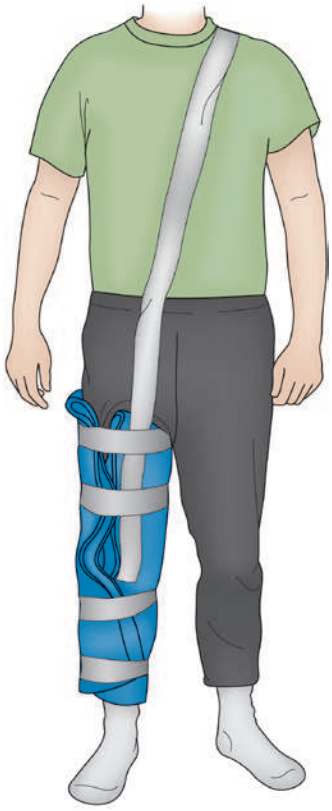


FIGURE 46-43 Functional knee and lower-leg immobilizer. Wrap a sleeping pad around the lower leg from the midthigh to the foot. Fold the pad so that the top of the leg is not included in the splint. This provides better visualization of the extremity and leaves room for swelling. A full-length pad forms a very bulky splint and may need to be trimmed before rolling. Because of the conical shape of the lower extremity and the effects of gravity, foam-pad lower-extremity splints tend to work their way inferiorly when the patient ambulates. A simple solution is to use duct tape "suspenders" to keep the splint from migrating downward.

Shoulder Immobilizer (Shoulder Spica Wrap)

After reducing a dislocated shoulder, immobilize it with a functional system made with a 15-cm (6-inch) elastic wrap around the torso. This method allows patients limited function to facilitate their own evacuation (e.g., ski poling, kayak paddling) but prevents complete abduction of the arm (Figure 46-44).

"Buddy" Taping

One of the simplest effective methods for splinting an injured finger or toe is to tape it to the adjacent digit. The keys to success are to put absorbent cloth or gauze between the digits before taping and to change the material at least three times a day, or any time it becomes moist, to avoid macerating the tissues. Keep the "buddy tape" in place until the injured digit feels better without the tape than with it. A field-expedient method of stabilizing long-bone fractures in a lower extremity is to buddy-tape the legs together, preferably with padding between them (Figure 46-45). This may be more comfortable than a rigid splint. Buddy taping can also be used for humeral fractures in infants by bandaging the arm to the torso.

"Pillow" Splints

Make pillow splints for joint fractures, dislocations, or other injuries. Fashion them from pillows, blankets, sleeping bags,

FIGURE 46-44 Shoulder spica wrap.

sleeping pads, or similar items (Figure 46-46). Wrap a pillow or multiple layers of other materials around the injured part. Secure it tightly with bandaging material. It is often a more comfortable method of splinting than using traditional materials.

Sandbag Splints

Use a garbage bag, fanny pack, or similar soft-sided container filled with sand or dirt to effectively splint extremities. Mold the bag to the extremity, and tape or bandage it in place. Because these splints are heavy, the patient tends to move the extremity less than with a pillow splint.

RIGID SPLINTS

Splint all fractures, either if left unreduced or after reduction. In general, the splint should incorporate the joints above and below the fracture. Ideally, a splint should immobilize the fractured bone in a functional position. In general, "functional position" means that the legs should be straight or slightly bent at the knees, the ankle and elbow bent at 90 degrees, the wrists straight, and the fingers flexed in a curve as if the person were attempting to hold a beverage can or baseball. If possible, conform the splint to the uninjured extremity and then transfer to the injured extremity.

Numerous items can be used as splints, including sticks or tree limbs, rolled-up magazines, books or newspapers, ice axes, tent poles, dirt-filled garbage bags, or fanny packs. Materials often available for splints include the flexible aluminum stays from internal-frame packs, skis and ski poles, and canoe/kayak paddles. Kayak and canoe flotation airbags can be converted into pneumatic splints for arm and ankle injuries. The Minicel pillars found in most kayaks can be removed and fashioned to provide upper- and lower-extremity splints. A personal flotation device (PFD, life jacket) can be molded into a cylinder splint for knee immobilization or into a pillow splint for the ankle.

Secure splints in place with strips of clothing, belts, pieces of rope or webbing, pack straps, gauze bandages, or elastic bandage wraps. When molded into any of several "structural curves," SAM Splints become much more rigid and can be used to immobilize most fractured or injured extremities. If prolonged use is anticipated (more than a few hours), place absorbent material, such as cotton cloth, between the splint and the skin to prevent skin irritation. Also, to prevent uncomfortable pressure points during prolonged use, place soft padding, such as gauze pads, around all bony prominences (Figures 46-47 to 46-52).

PELVIC FRACTURES

Compressing the iliac crests reduces morbidity and mortality in patients with unstable pelvic fractures by effectively controlling venous hemorrhage.[11] Clothes, sheets, a sleeping bag, pads, an air mattress, tent, or tent fly can be used to improvise a very

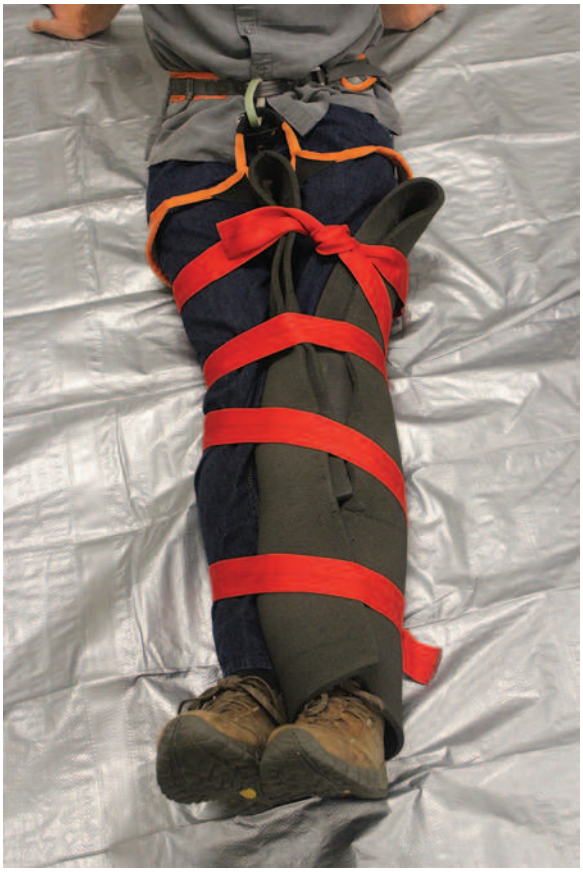


FIGURE 46-45 "Buddy tape" expedient splint.

effective pelvic binder in the backcountry. The object should be wide enough so that it does not cut into the patient when tightened.

Applying an Improvised Pelvic Binder

1. Ensure that objects have been removed from the patient's pockets and that any belt has been removed so that the pressure of the sheet or object does not cause additional pain by pressing items against the pelvis.
2. Gently slide the improvised sling under the patient's buttocks, and center it under the bony prominences at the outer part of the upper thigh or hips (greater trochanters, symphysis pubis). Cross the object over the front of the pelvis, and tighten the sling by pulling both ends; secure it with a knot, clamp, or duct tape. Another tightening technique is to wrap the sling snugly around the pelvis, and tie an overhand knot. Place tent poles, a stick, or similar object on the knot, and tie another overhand knot. Twist the poles or stick until the sling becomes tight (Figure 46-53). The sling should be tightened so that it is snug, and the pelvis is returned to its normal anatomic position.
3. Theoretically, if a Therm-a-Rest pad or other inflatable sleeping pad is available, it could be folded in half so that it approximates the size of the pelvis. Gently slide the pad under the patient's buttocks, and center it under the greater trochanters and symphysis pubis. Secure the pad with duct tape, then inflate the pad as you would normally until it produces a snug fit and the pelvis is reduced (Figure 46-54). As of this writing, there is no known report that this method has actually been used in a patient with a pelvic fracture.
4. Place padding between the legs, and gently tie the legs together to further stabilize the fracture in a position that is most comfortable for the patient.

FEMORAL FRACTURES

In the backcountry environment, traction for femoral fractures may limit blood loss in the thigh and sometimes reduce pain, although no conclusive research to date validates this. However, one small study demonstrated that mean traction force, stability, and comfort of these techniques were equivalent to standard commercial devices.[129] However, any traction device might be so bulky and awkward that wilderness extrication by litter, technical rescue, or helicopter becomes impossible.

General Principles of Femoral Traction

The potential variety of femoral traction designs is unlimited, but five key design principles should be considered when evaluating any system:

1. Does the splint provide inline traction, or does it incorrectly pull the leg off to the side or needlessly plantar-flex the foot?
2. Is the splint comfortable? Ask the patient.

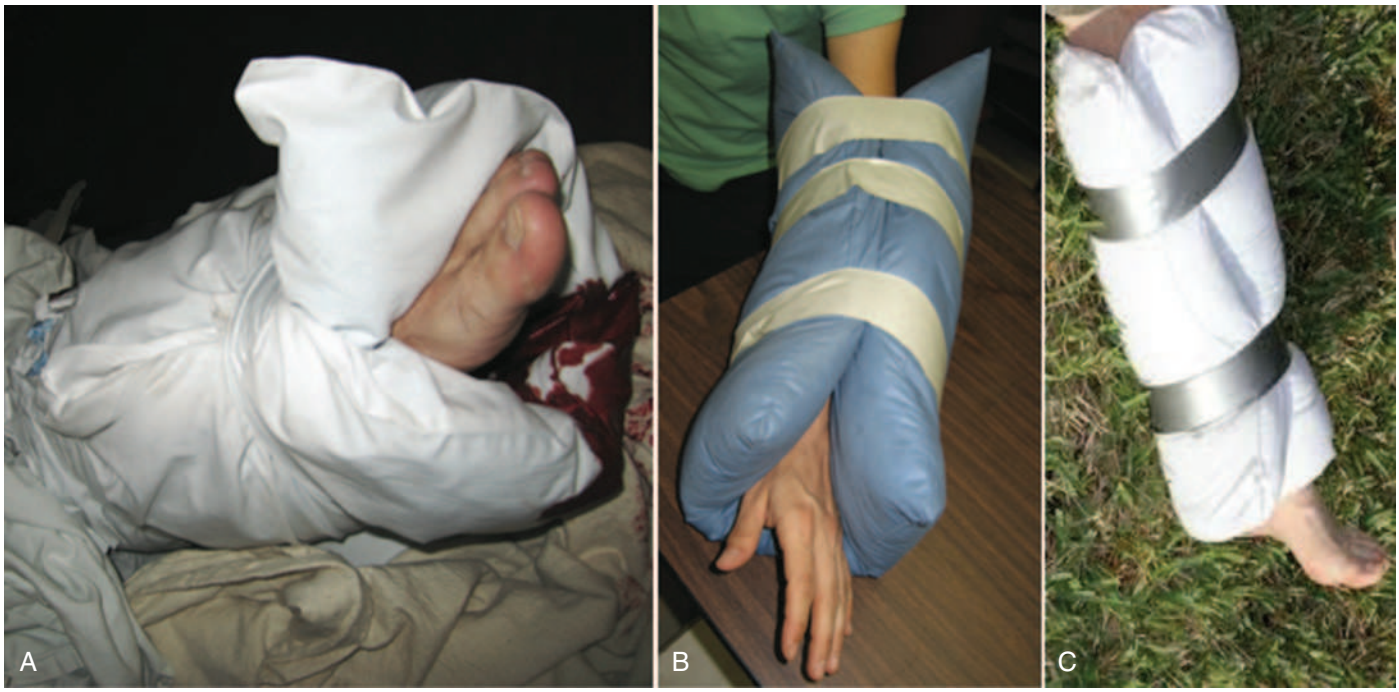


FIGURE 46-46 "Pillow" splints on **A,** ankle; **B,** elbow; and **C,** knee.

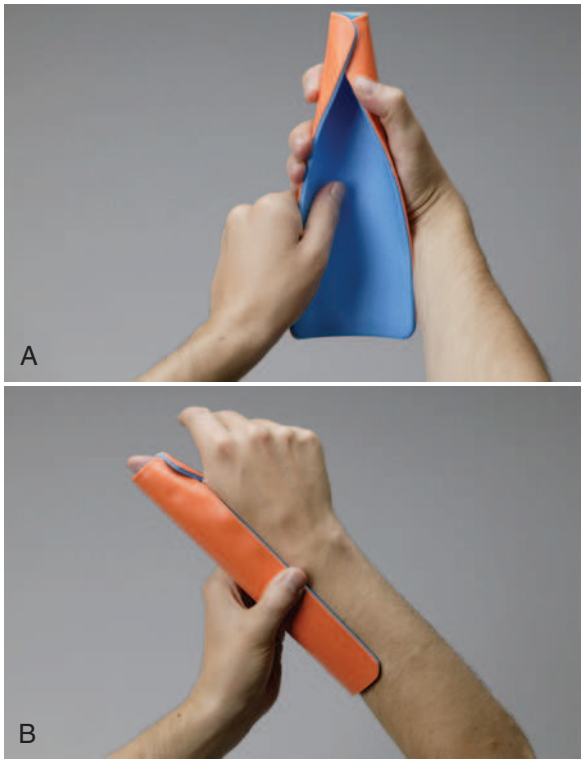


FIGURE 46-47 Padded aluminum thumb spica splint.

3. Does the splint compromise neurologic or vascular function? Constantly check distal neurovascular function.
4. Is the splint durable, or will it break when subjected to back-country stresses? Try improvised traction devices on an uninjured person, stressing it to determine its strength.
5. Is the splint so cumbersome that it will hinder evacuation?

Femoral Traction System

Every femoral traction system has six components:

1. Ankle hitch
2. Rigid support that is longer than the leg
3. Traction mechanism
4. Proximal anchor
5. Method for securing the splint to the leg
6. Additional padding

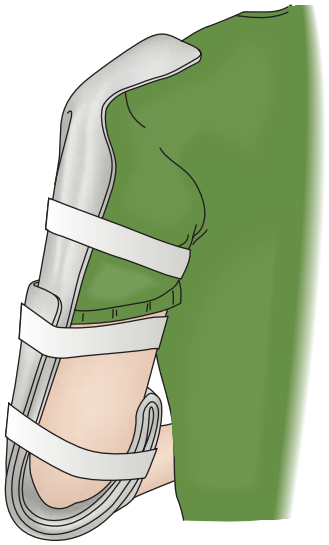


FIGURE 46-48 Humerus splint. Used in conjunction with a sling and swath, this splint adds extra support and protection for a fractured humerus.

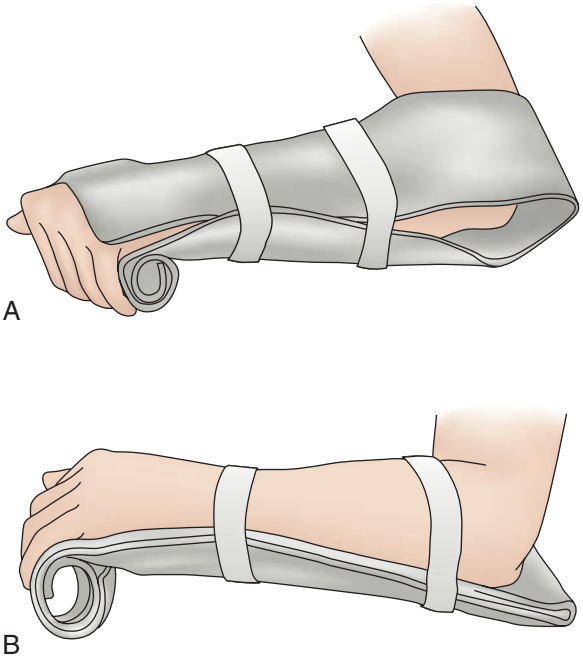


FIGURE 46-49 Forearm splint. These splints are used for treatment of wrist or forearm fractures. **A,** The sugar-tong splint prevents pronation and supination and has the advantage of greater security and protection than **B,** the volar splint, because of its anterior-posterior construction. Tape is shown for purposes of illustration; elastic wrap or self-adherent wrap (Coban) is more comfortable and efficient.

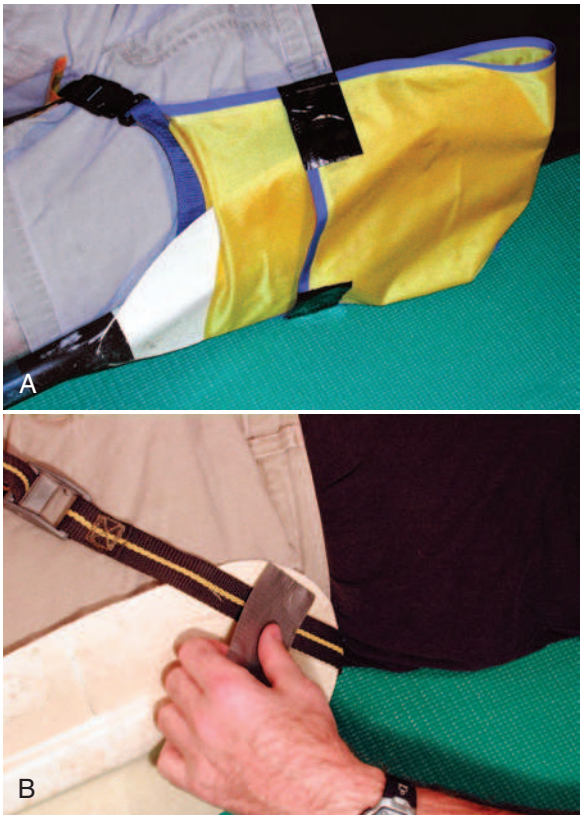


FIGURE 46-50 **A,** River dry bag used to create a simple proximal support with a kayak paddle. **B,** A paddle is pushed up against the proximal support strap. It is taped for security, but the forces are pushing against the strap, not the tape.

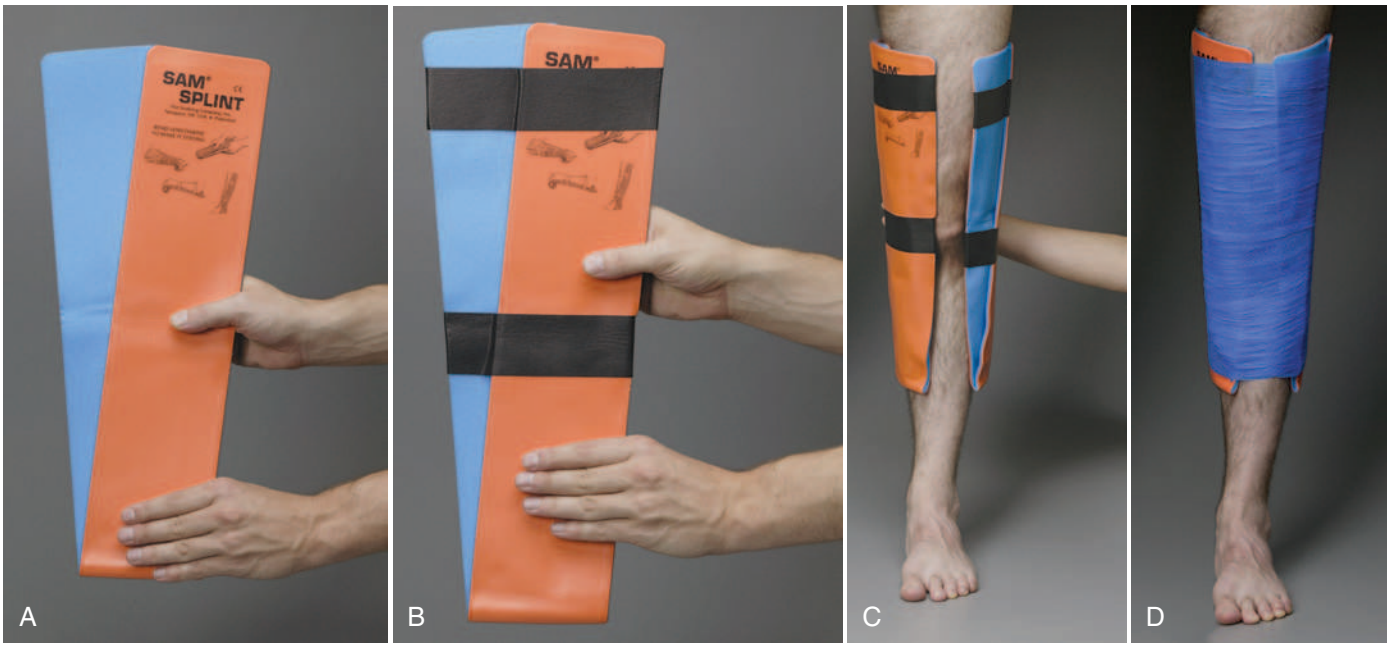


FIGURE 46-51 Knee immobilizer using two padded aluminum splints. **A,** The splints are folded in half, then fanned out (wider at the top for the thigh). **B,** Tape is applied to maintain the fan shape. **C** and **D,** Splint is then applied bilaterally and secured.

Ankle Hitch. Various techniques are used to anchor the distal extremity to the splint. Many work well but may be impossible to recall in an emergency. Choose an easy-to-remember technique, and practice it. It is best to leave the shoe on the patient's foot, and apply the hitch over it. Cut out the toe section of the shoe in order to be able to periodically check circulation. Ensure that the patient does not have a concomitant distal tibia-fibula or ankle injury.

Single-Runner System. Loop a long piece of webbing, shoelace, belt, or rope over itself, bringing one end through the middle to create a stirrup. After rotating it away from the patient

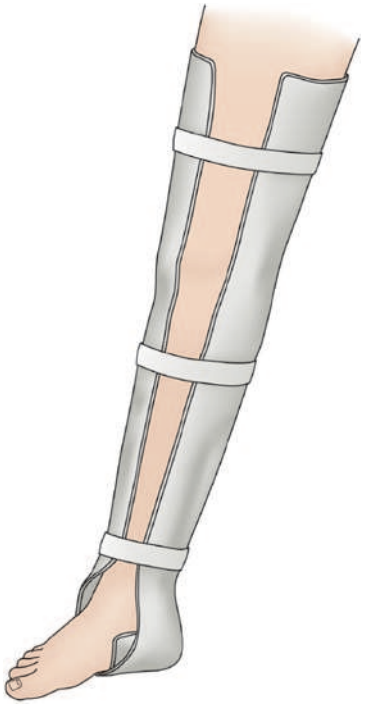


FIGURE 46-52 Lower-leg or ankle splint. A sugar-tong splint can be used to immobilize fractures of the tibia, fibula, or ankle. When used on an adult, two splints should be used. A third splint may be placed posteriorly for additional support.

FIGURE 46-53 Pelvic binder improvised with jacket.

FIGURE 46-54 Pelvic binder improvised with inflatable sleeping pad and duct tape.

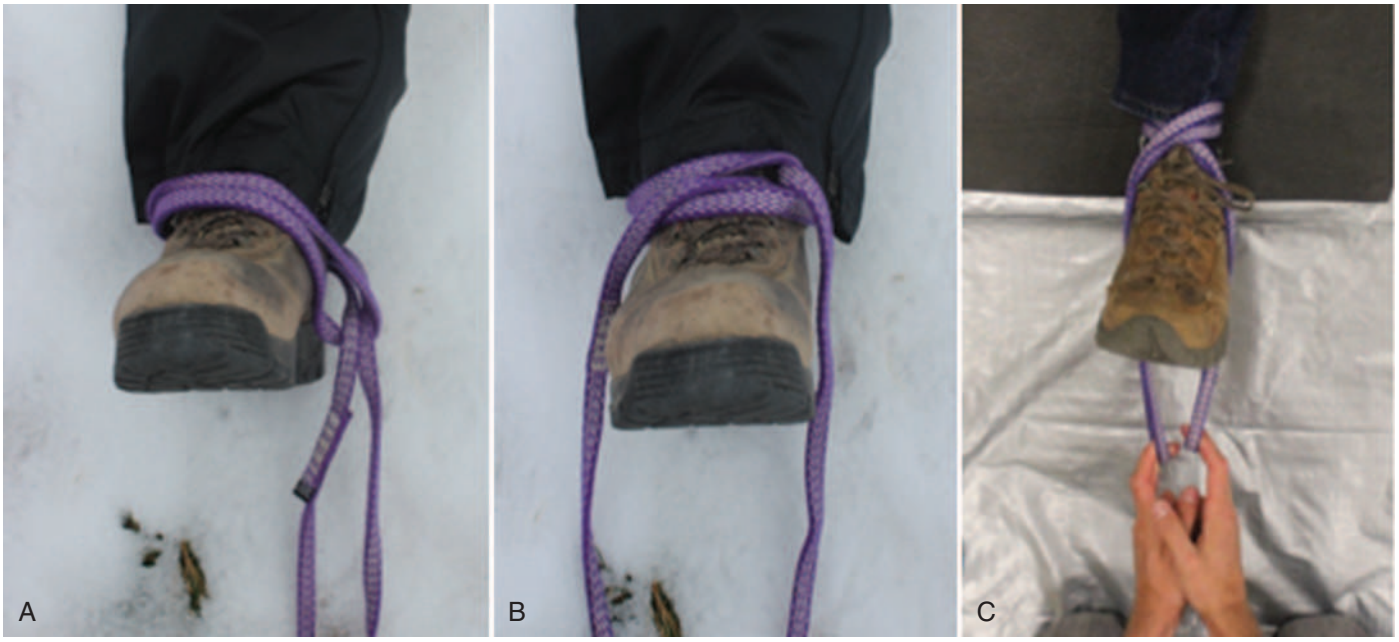


FIGURE 46-55 Single-runner ankle hitch. Looped piece of webbing is looped into a stirrup **(A),** and the ends are equally placed on the sides of the foot for even traction **(B),** or crossed over and secured to traction **(C).** A knot or tape at the crossover prevents slippage.

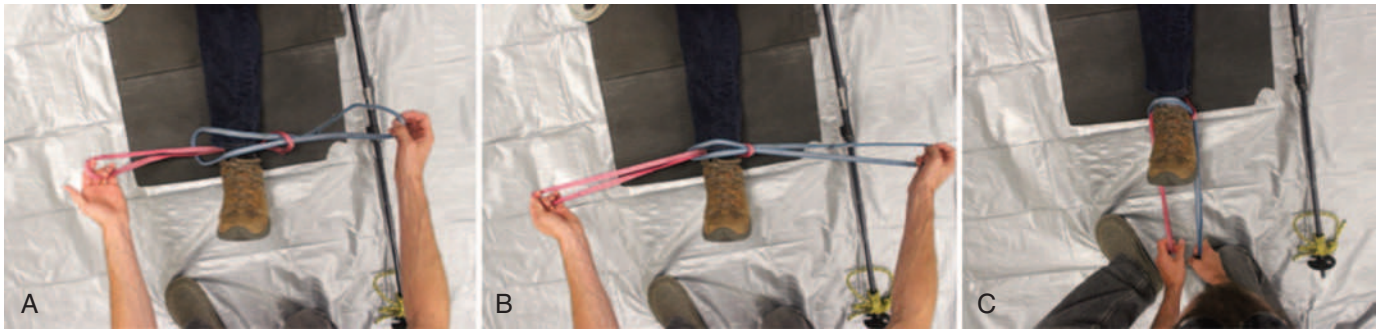


FIGURE 46-56 Double-runner ankle hitch. **A** and **B,** Two webbing loops (runners) are laid over and under the ankle. **C,** Completed double-runner ankle hitch. The traction can be easily centered from any angle, ensuring in-line traction.

180 degrees, slip the hitch over the shoe and ankle (Figure 46-55A and B). Alternatively, crisscross the long loop (Figure 46-55C), where a knot or duct tape can be placed, precluding the need to unravel the system should less tension or more padding (not shown) become necessary.

Double-Runner System. In this straightforward technique, lay two short webbing loops ("runners") over and under the ankle (Figure 46-56A). Pass the long loop sides through the short loop on both sides (Figure 46-56B) and adjust as needed (Figure 46-56C). One advantage of this system is that it is infinitely adjustable, enabling the rescuer to center the pull from any direction. As always, proper padding is essential, especially for long transports. The patient's boot can distribute pressure over the foot and ankle but will obscure visualization and palpation of the foot. A reasonable compromise is to leave the boot on and cut out the toe section for observation.

Patient's Boot System. Another efficient system uses the patient's own boot as the hitch. Cut two holes into the side walls of the boot just above the midsole and in line with the ankle joint. Thread a piece of nylon webbing or cravat through the holes to complete the ankle hitch (Figure 46-57). Cutting away the toe may be necessary for neurovascular assessment.

Buck's Traction. For extended transport, Buck's traction can be improvised using a closed-cell foam pad (Figure 46-58). Wrap the pad around the lower leg as shown, and loop a stirrup below the foot from the medial to the lateral calf. Fasten this assembly with a second cravat wrapped circumferentially around

FIGURE 46-57 Traction using cut boot and cravat.

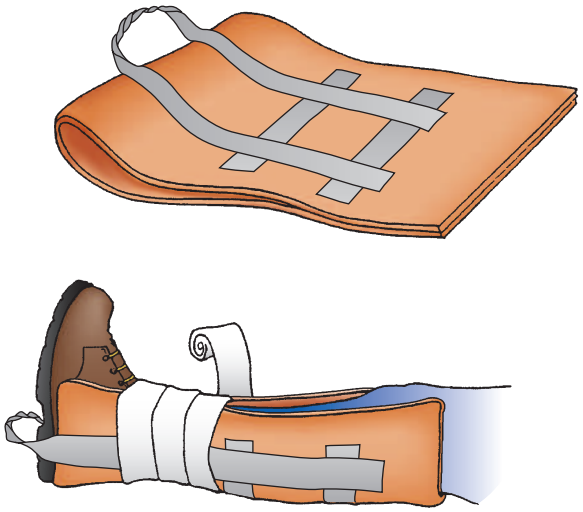


FIGURE 46-58 Buck's traction. Duct tape stirrups are added to a small foam pad that is wrapped around the leg. The entire unit is wrapped with an Ace bandage. This system helps distribute the force of the traction over a large surface area.

the calf over the closed-cell foam. Duct tape or nylon webbing can be used instead of cravats. This system increases the surface area over which the stirrup is applied and thus decreases the potential for neurovascular complications and dermal ischemia. In addition, improvised Buck's traction has been used to manage backcountry hip fractures. However, it has been suggested that this technique may have little benefit.[2] If Buck's traction is used for a hip injury, use smaller amounts of traction (~2.5 kg [5.5 lb]). Consider this method for distal tibia-fibula or ankle injuries.

Rigid Support. The rigid support can be fabricated as a unilateral support, similar to the Sager traction splint or Kendrick Traction Device, or as a bilateral support, such as the Thomas half-ring or Hare traction splint. Unilateral supports tend to be easier to apply than are bilateral supports. The following are some ideas for rigid support.

Double–Ski Pole or Canoe Paddle System. This is fashioned similar to a Thomas half-ring splint, with the interlocked pole straps slipped under the proximal thigh to form the ischial support. Some mountain guides carry a prefabricated, drilled ski pole section, an aluminum bar, or an ice pick that can be used to stabilize the distal end of this system. One can adjust tension easily using a clove hitch or a ratcheting device (Figure 46-59).

Single Ski Pole or Canoe/Kayak Paddle. Use a single ski pole or paddle either between the legs, which is ideal for bilateral femoral fractures, or lateral to the injured leg. The ultimate rigid support is to use an adjustable telescoping ski pole laterally. Adjust the pole to the appropriate length for each patient, making the splint compact for litter work or helicopter evacuation (Figure 46-60).

Tent Poles. This system uses conventional sectioned tent poles. Fit the poles together to create the ideal length for rigid support. Because of their flexibility, tent poles must be well secured to the leg to prevent them from flexing out of position. Place a blanket pin or a bent tent stake in the end of the pole to provide an anchor for the traction system. Alternatively, use a Prusik knot to secure the system to the end of the tent pole (Figure 46-61).

Miscellaneous Objects. Any suitable object, such as one or two ice axes taped together at the handles or a straight branch, can be used to make a rigid support (Figure 46-62). Although skis immediately come to mind as suitable rigid components, they are too cumbersome to work effectively. Because of their length, skis may extend far beyond the patient's feet or require placement into the axillae, which is unnecessary and inhibits mobility (e.g., sitting up during transport). The best way to employ skis for leg immobilization is to use the prefabricated canvas pockets with tip and tail attachment grommet, available from the National Ski Patrol System.

Traction Mechanism. The amount of traction required may be difficult to estimate. A general rule is to use 10% of the

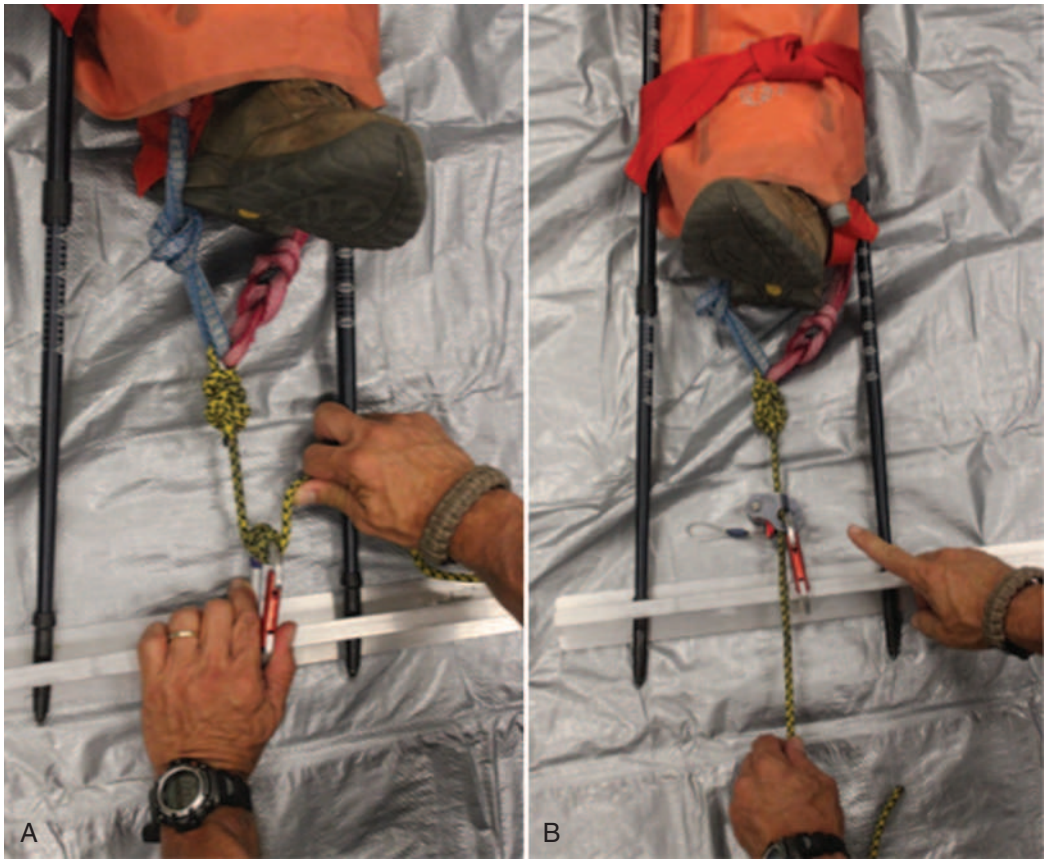


FIGURE 46-59 Two poles secure the traction device, consisting of a carabiner and a sliding clove hitch, which can lock on itself. **A,** Horizontal stay, an ice picket, stabilizes the system. **B,** Autolocking rope ascender is used to easily adjust tension. This camming device prevents the yellow cord from retracting, which can be released by disengaging the red cam by pulling on the swaged wire.

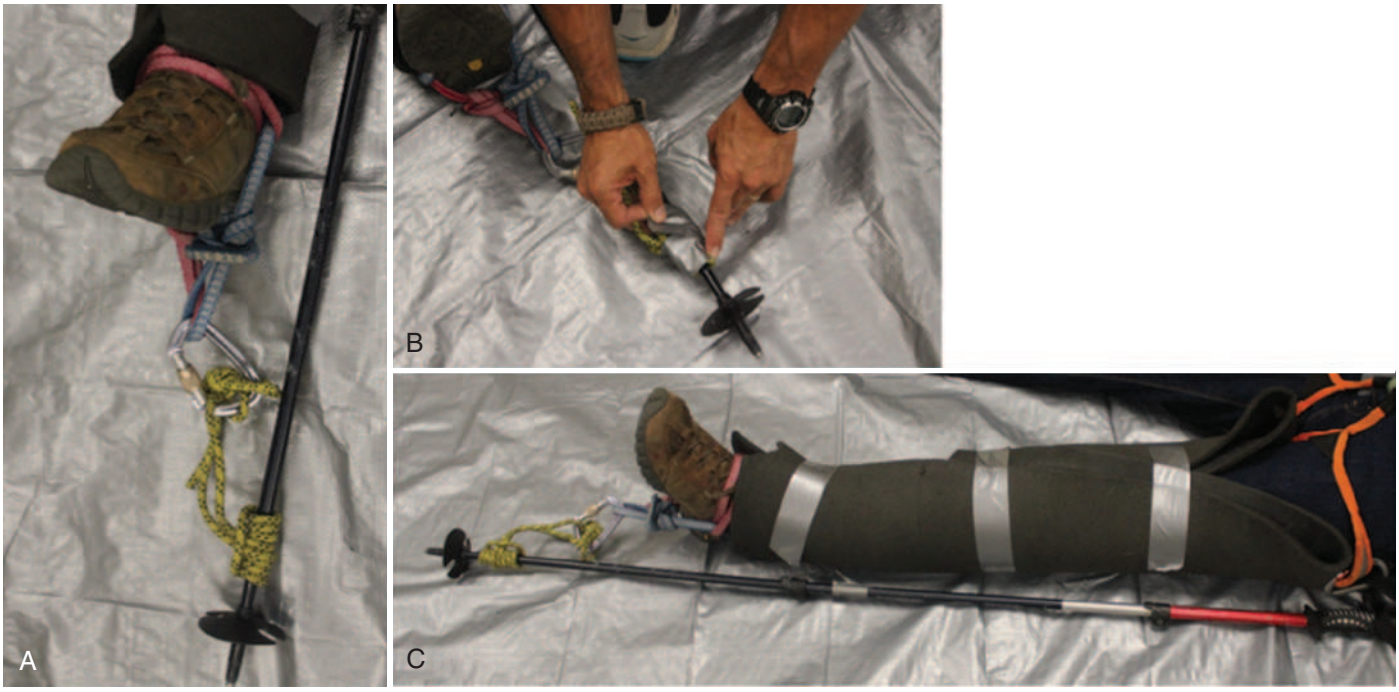


FIGURE 46-60 Single femoral traction device. **A,** Prusik knot made from a small-diameter cord is used as an adjustable distal traction anchor. Alternatively, the stirrup can be attached to a carabiner placed on the other end of the basket, where the pole tip is located. **B,** Several wraps or duct tape can prevent knot sliding on a smooth pole. **C,** Telescoping pole can add tension. Note the liberal use of padding. For more security, the pole can be secured to the leg after desired traction reached (not shown).

patient's body weight or about 4.5 kg (10 lb) to a maximum of 7 kg (15 lb) of traction force.[1] Consider practicing in advance with a commercial system, such as a Sager traction device (Minto Research and Development, Redding, California), to become familiar with the process and magnitude of forces. Check the opposite extremity to compare lengths. After the traction is applied, recheck distal neurovascular function (circulation, sensation, and movement). Some sources advise applying traction to maximize patient comfort.[6] However, patients with these injuries are often in extremis, and relative "comfort" is difficult to assess. Lacking other indicators, applying traction to approximate the opposite leg's length is a reasonable goal.

Cam Lock or Fastex-Like Slider. This simple, effective system uses straps that have a Fastex-like slider (ITW Fastex, Des Plaines, Illinois). Such straps are often used as waist belts or to attach items to packs. Alternatively, use a cam lock with nylon webbing. Attach the belt to the distal portion of the rigid support and then to the ankle hitch. Traction is easily applied by cinching the nylon webbing (Figure 46-63).

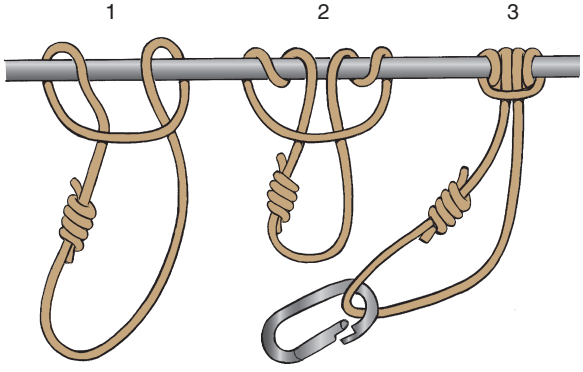


FIGURE 46-61 Prusik knot made from a small-diameter cord is used as an adjustable distal traction anchor. Although two wraps are shown for simplicity, an additional one to two wraps are usually necessary to add further security when applied to a smooth surface, such as a kayak paddle.

Trucker's Hitch. A windlass can be easily fashioned using small-diameter line (e.g., parachute cord) and a standard trucker's hitch for additional mechanical advantage.

Prusik Knot. Almost any system can be rigged with a Prusik knot (see Figure 46-61). Prusiks are ideal for providing traction from rigid supports with few tie-on points (e.g., canoe paddle shaft, tent pole). The Prusik knot can be used to apply the traction (by sliding the knot distally) or simply as an attachment point for one of the traction mechanisms already mentioned.

Spanish Windlass. The first popularized modern improvised traction mechanism was the Spanish windlass. These systems can be awkward to apply and are often not durable. The windlass can unwind if inadvertently jarred and can apply rotational forces to the leg. If using this system, firmly secure the windlass to the rigid supports with duct tape or ties.

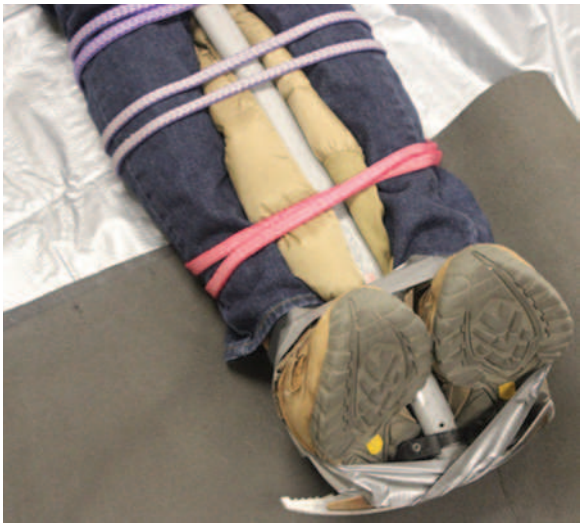


FIGURE 46-62 An ice axe is used as a splint and may be secured by any means. Ensure that all sharp points are covered.

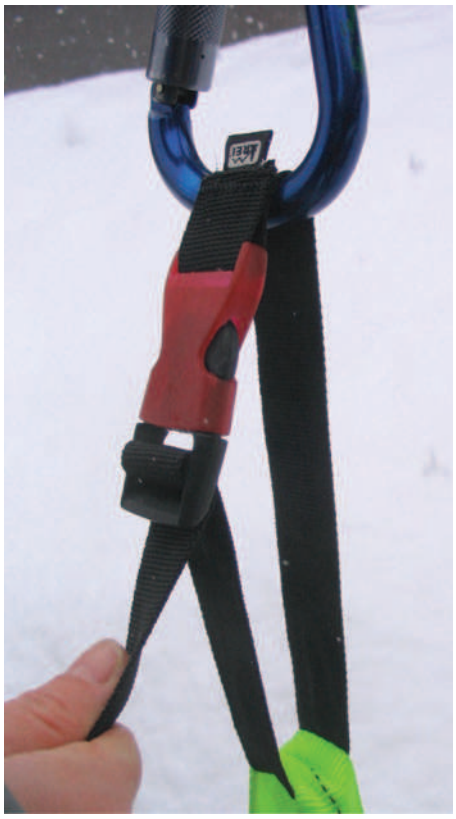


FIGURE 46-63 Fastex-type or cam-lock utility strap makes a very efficient traction device and can be adapted for most systems.

Litter Traction. If no rigid support is available and a rigid litter (e.g., Stokes) is being used, apply traction from the rigid bar at the foot end of the litter. If this system is used, ensure that the patient is immobilized on the litter with adequate countertraction, such as inguinal straps, so that traction does not vary when the patient is in the head-down position.

Proximal Anchor. The simplest proximal anchor uses a single proximal thigh strap, which can be made from a piece of climbing webbing or a prefabricated strap, belt, or cam lock (Figure 46-64). A cloth cravat can be used if necessary. On the river, a PFD (life jacket) can be used. When climbing, a climbing harness is ideal.

Securing and Padding. Check all potential pressure points to ensure that they are adequately padded. An excellent padding system can be made by first covering the upper and lower leg with a folded length of Ensolite (closed-cell foam) or an inflatable mattress (Figure 46-65A). This can be advantageous over a circumferential wrap because the folded system allows one to see the extremity. The patient is more comfortable if femoral traction is applied with the knee in slight flexion (padding placed beneath knee during transport). Secure the splint firmly to the leg. Almost any strap-like object will work, but a 10- to 15-cm (4- to 6-inch) elasticized bandage wrapped circumferentially provides a comfortable and secure union. Finally, strap or tie the ankles or feet together with adequate padding between the legs to give the system additional stability. Tying the ankles together also prevents the injured leg from excessive external rotation during transport, which greatly improves patient comfort. A SAM splint applied to a lower leg also provides added torsional stability (Figure 46-65B).

PATELLAR DISLOCATION

Common mechanisms producing a patellar dislocation include a forceful quadriceps contraction, along with valgus and external

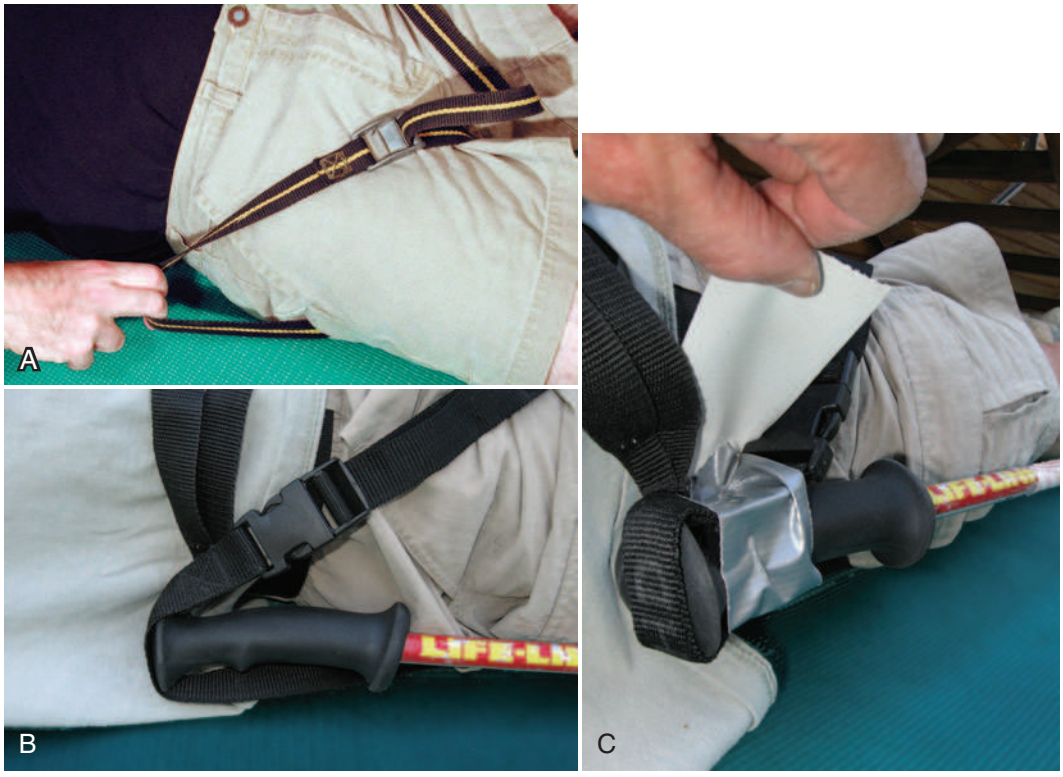


FIGURE 46-64 **A,** Proximal anchor using cam-lock belt. The belt is applied as shown. The strap is adjusted loosely to allow the belt to ride up to the point of the hip. If improperly tightened, the strap can create pressure over the fracture, and it moves the traction point to a less optimal distal position. Padding is helpful, but not always necessary, if the patient is wearing pants and the strap properly adjusted. **B** and **C,** End of the ski pole is pushed into the strap, then taped in place. Note that forces are applied to the strap, not the duct tape.

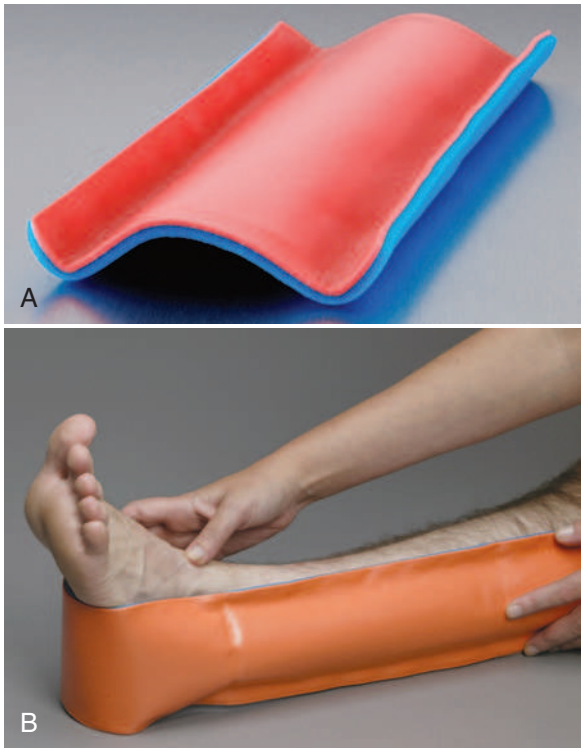


FIGURE 46-65 Lower-extremity structural aluminum malleable (SAM) splint. **A,** Note the reverse bends at the edge to add structural rigidity. **B,** Lower-leg/ankle splint with reverse bends.

rotation strain, or with a direct blow to the flexed knee.[48] The most common dislocation is laterally. The knee is in flexion, with the patella laterally displaced. Simply straightening the leg usually relocates the patella; gentle medial pressure applied to the patella while elevating the most lateral edge of the patella over the femoral condyle will enhance success.[52] The knee can then be placed in an improvised immobilizer or splint (see Figures 46-51 and 46-52).

ANKLE INJURIES

Ankle dislocations may need to be emergently reduced in the backcountry if there is neurovascular compromise or tenting of the skin threatening to produce an open fracture. An initial step to restore neurovascular function may simply be to remove the shoe or boot, and keep the affected extremity warm thereafter. The goal is to restore neurovascular function, as opposed to "perfect" anatomic alignment. Reduction may then be necessary if this maneuver fails to improve neurovascular status. Reduction without parenteral analgesia may be difficult; however, a hematoma block or hypnosis may be sufficient to mitigate pain. Experience has demonstrated that the most effective method to reduce the injury is to have an assistant firmly stabilize the lower extremity while you firmly grasp the heel with one hand and the forefoot with the other hand. Keeping your arms straight, lean backward. This exerts a constant pull with sufficient force to rapidly reduce the fracture or dislocation. Immobilize the reduced ankle, and transport the patient in a non–weight-bearing manner. Do not use a femoral traction device on these patients.

TRAUMA AND HYPOTHERMIA

While treating any patient in a wilderness setting, remember to avoid conductive heat loss. An immediate priority is to place insulation material between patient and ground. Rescuers can also provide warmth by using a fire and/or shelter, and by covering the patient as much as possible (Figure 46-66).

FIGURE 46-66 Managing hypothermia should be done expeditiously while fabricating an orthopedic device or awaiting transport.

EYE, EAR, NOSE, MOUTH, AND THROAT IMPROVISATION

EPISTAXIS

Epistaxis is a common problem among travelers. Reduced humidity in airplanes, cold climates, and high-altitude environments can produce drying and erosion of the nasal mucosa. Other etiologic factors include facial trauma, infections, and inflammatory rhinitis. Although most cases of epistaxis are minor, some become life-threatening emergencies.[118]

Anterior epistaxis from one side of the nasal cavity occurs in 90% of cases. If pinching the nostrils against the septum for a full 10 minutes does not control the bleeding, nasal packing may be needed. Soak a piece of cotton or gauze with a vasoconstrictor such as oxymetazoline nasal spray, insert it into the nose, and leave it in place for 5 to 10 minutes. Petroleum jelly–impregnated gauze or strips of a nonadherent dressing can then be packed into the nose so that both ends of the gauze remain outside the nasal cavity (Figure 46-67). This prevents the patient from inadvertently aspirating the nasal packing.[118]

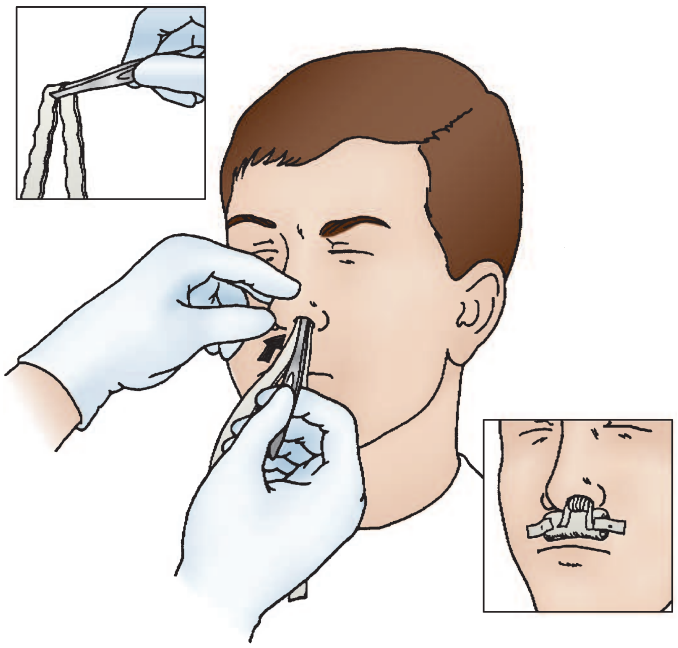


FIGURE 46-67 Anterior epistaxis from one side of the nasal cavity can be treated using nasal packing soaked in a vasoconstrictor. Petroleum jelly–impregnated gauze or strips of nonadherent dressing can be packed in the nose so that both ends of the gauze remain outside the nasal cavity.

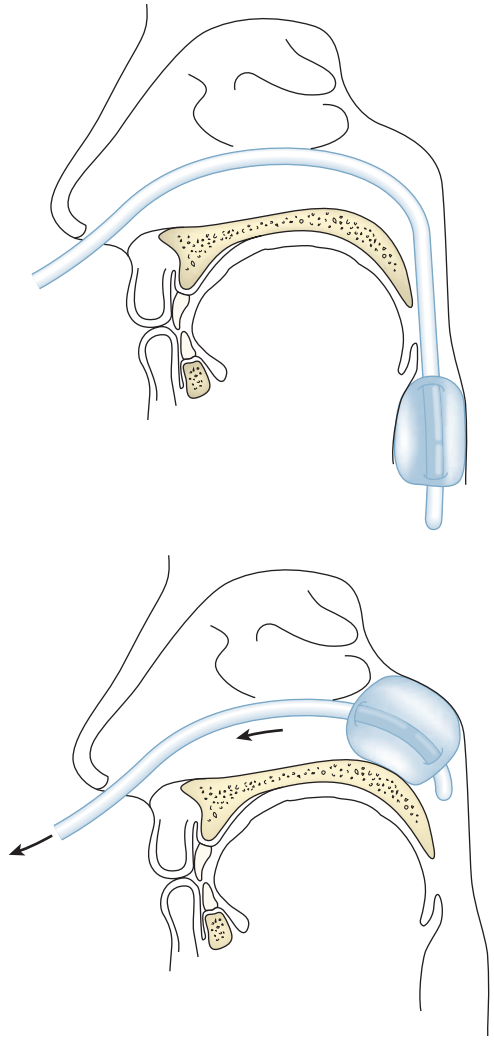


FIGURE 46-68 Packing the back of the nose. Insert a Foley catheter into the nose, and gently pass it back until it enters the back of the throat. After the tip of the catheter is in the patient's throat, carefully inflate the balloon with 10 to 12 mL of air or water from a syringe. Inflation should be done slowly and should be stopped if painful. After the balloon is inflated, gently pull the catheter back out until resistance is met.

Complete packing of the nasal cavity of an adult patient requires a minimum of 1 m (3.3 feet) of packing to fill the nasal cavity and tamponade the bleeding site. Although commercial packing is available, a tampon or the balloon tip from a Foley catheter is more likely to be available, and both work well as improvised packing.[118]

Anterior nasal packing blocks sinus drainage and predisposes to sinusitis. Evidence suggests that it is reasonable to withhold prophylactic antibiotics (generally amoxicillin-clavulanate or clarithromycin) in an otherwise healthy patient if the pack is removed within 48 hours.[123]

If the bleeding site is located posteriorly, use a 14F to 16F Foley catheter with a 30-mL balloon to tamponade the site (Figure 46-68). Prelubricate the catheter with either petroleum jelly (Vaseline) or a water-based lubricant, and then insert it through the nasal cavity into the posterior pharynx. Inflate the balloon with 10 to 15 mL of water, and gently withdraw it back into the posterior nasopharynx until resistance is met. Secure the catheter firmly to the patient's forehead with several strips of tape. Pack the anterior nose in front of the catheter balloon as described earlier.

DENTAL TRAUMA

There is a paucity of literature on backcountry dentistry, even though dental issues can threaten an expedition. Any dental trauma can produce pulpitis, diagnosed by (1) temperature sensitivity, (2) percussion sensitivity, (3) palpation sensitivity, and (4) acute and nonlingering pain. Trauma to the enamel tends to be uncomplicated; involvement of the pulp or root is much more complex and may necessitate evacuation. Fractures to the dentin create sensitivity to cold, air, and liquids and require temporary coverage if pain is severe. Pulp or root injuries have similar findings, with the additional complication of bleeding, and ultimately require endodontic therapy to restore function, alleviate pain, and prevent future infection.[119] Fractured teeth can generally be treated with pain management and anesthetic infiltration, local application of an anesthetic (e.g., benzocaine gel, eugenol, oil of cloves), with a temporary filling placed over the defect. One may use soft wax, tape, cyanoacrylate, "old gum," Cavit, or intermediate restorative material (IRM).[44] We have used a mixture of eugenol and zinc oxide as an improvised paste.[18] Cavit or IRM is packed into the dental defect using a flat stick, knife, or similar tool, plugging the defect. The person can lightly bite on a flat, covered defect to remove excess material. Note that these products may not work well when there is significant bleeding, which can be stopped with the patient biting on gauze impregnated with a topical anesthetic enhanced by a vasoconstrictor.

Subluxed or avulsed teeth require a different approach. A search for the tooth and removal of tooth fragments (if the tooth is not intact or attached to the root) is imperative; if not found, the tooth may have been swallowed or aspirated, or it may have become intruded (impacted) into the tooth socket, mandating evacuation. An avulsed, intact tooth should not be scrubbed, wiped, or scraped in any way; debris should be removed by gentle rinsing with sterile saline or water. Hold the tooth only by the crown, not by the root surface. Avoid desiccation of the tooth, and replace the clean tooth within 10 minutes back in the socket, or in the solutions described next. Avulsed teeth can be stored in sterile saline, the patient's saliva, milk, or commercially based products that contain Hank's Balanced Salt Solution. Milk keeps the periodontal ligament viable for 4 to 8 hours; commercially available products can preserve the ligament for 12 to 24 hours.[84,97] The best treatment is to replace an intact tooth in the original socket. Do not replace a fractured tooth. Gentle evacuation of socket clots with sterile saline irrigation is best; avoid scraping, scrubbing, or otherwise applying trauma to the socket, which could destroy the periodontal ligament. After anesthetizing the socket, gently but firmly replace the tooth, held only by the crown, into the defect.[8] Having the patient bite down on gauze sets the tooth further. The tooth can then be splinted with a commercially available product, such as Coe-Pak, wax, or a small piece of SAM Splint. The unstable tooth is attached to stable teeth on either side to make this technique effective.

A tooth can also be splinted with a suture (Figure 46-69) or with the use of a hypodermic needle, paper clip, or similar item, with the sides filed down smoothly and covered, so as not to damage adjacent structures. The needle can then be bent to conform to the curvature of the adjacent teeth. Thin ligature wire,

FIGURE 46-69 Figure-of-eight suture or wire (*green*) can hold an avulsed tooth in place. A curved paper clip or syringe can do likewise when secured to solid teeth (an improvised eugenol/zinc oxide mixture was used); wax (*blue*) can also hold teeth in place, or cover defects and cavities. Teeth must be thoroughly dry before applying wax, glue, or other dental pastes.

26-gauge snare/hobby wire, suture, or even dental floss is twisted or tied around each tooth to stabilize the entire bridge assembly.[23] The ends of the wires can be tucked and covered with wax, tape, or a similar product to avoid puncturing the adjacent mucosa.

A soft or liquid diet would be prudent until more definitive care can be obtained. In truly remote areas, tooth extraction may be necessary because of persistent pain from decay, periodontal disease, or trauma. If the tooth is not loose, the procedure can be difficult and requires prior training. Improvised dental tools have been successfully used, but this is not recommended.[71]

ESOPHAGEAL FOREIGN BODIES

Esophageal foreign bodies can cause significant morbidity. Respiratory compromise caused by tracheal compression or by aspiration of secretions may occur. Mediastinitis, pleural effusion, pneumothorax, and abscess may be seen with perforations of the esophagus from sharp objects or pressure necrosis caused by large objects.

Foley balloon-tipped catheters can be used safely to remove blunt esophageal foreign bodies, either by using them to push or pull the object.[28] Success rates of 98% have been cited.[7] Associated complications include laryngospasm, epistaxis, pain, esophageal perforation, and tracheal aspiration of the dislodged foreign body.[93] Sharp, ragged foreign bodies or an uncooperative patient preclude use of this technique.[120]

Lubricate a 12F to 16F Foley catheter, and place it orally into the esophagus while the patient is seated. After placing the patient in Trendelenburg position, pass the catheter beyond the foreign body, and inflate the balloon with water. Withdraw the catheter with steady traction until the foreign body can be removed from the hypopharynx or is expelled by coughing. Take care to avoid lodging the foreign body in the nasopharynx. Terminate the attempt if there is any significant impedance to withdrawal.

IMPROVISED EYEGLASSES

Exposure of unprotected eyes to ultraviolet radiation at high altitudes may produce photokeratitis (snowblindness). Symptoms are delayed, and the patient is often unaware that an eye injury is developing. When sunglasses are lost at 4267 m (14,000 feet) in the snow, photokeratitis can develop in 20 minutes. One can improvise sunglasses from duct tape, cardboard, or any light-impermeable material that can be cut. Cardboard "glasses" with narrow eye slits can be taped over the eyes for protection.

Slits can also be cut into a piece of duct tape that has been folded over on itself with the sticky sides opposing. After a triangular wedge is removed for the nose, apply another piece of tape to secure the glasses to the head.

If a sunglass lens is broken or lost, the previous technique can be used over the existing frame (Figure 46-70).

Pinhole tape glasses can improve vision in a myopic person whose corrective lenses have been lost. With myopia, parallel light rays from distant objects focus in front of the retina. The pinhole directs entering light to the center of the cornea, where refraction (bending of the light) is unnecessary. Light remains in focus regardless of the refractive error of the eye (Figure 46-71). Pinhole glasses decrease both illumination and field of vision, so puncture a piece of duct tape or cardboard repeatedly with a safety pin, needle, fork, or other sharp object until enough light enters to focus on distant objects. Secure the device to the face (Figure 46-72).

USEFUL TOOLS AND EQUIPMENT TO CARRY

Many of the items listed next should usually be a part of a good survival kit that the astute outdoor adventurer carries; thus the items should not be necessarily burdensome to pack.

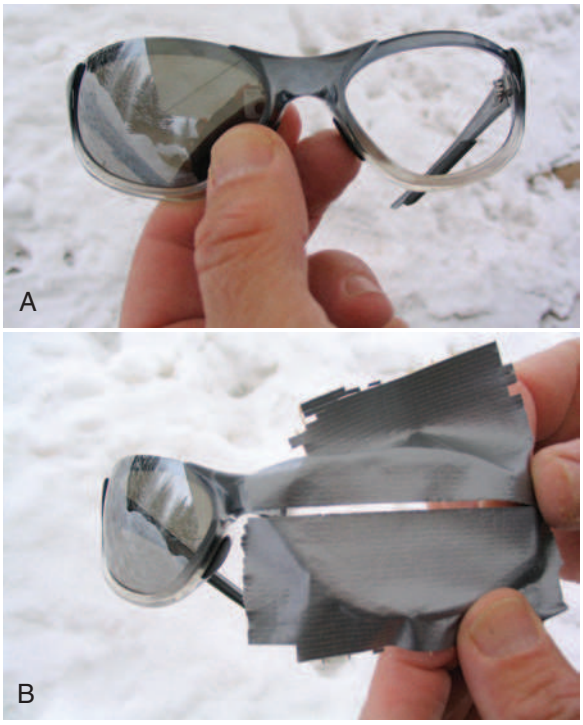


FIGURE 46-70 Improvised lens for sunglasses.

MULTITOOL

The multitool (multiple-use tool) can be a fairly simple model, but it should have an awl for drilling holes into skis, poles, sticks, and so on. This allows creating well-fitted components during improvisation (e.g., drilled crossbar attached to ski tips for improvised rescue toboggan). Filing the awl may be necessary before sojourning into the backcountry. If possible, any needed additional material should be extracted from the patient's belongings. The pliers often can be used to hold, crimp, or unscrew items while fashioning equipment.

TAPE AND GLUE

Carry some form of strong, sticky tape. All duct tape is not created equal. Look for an exterior-grade, weatherproof product.

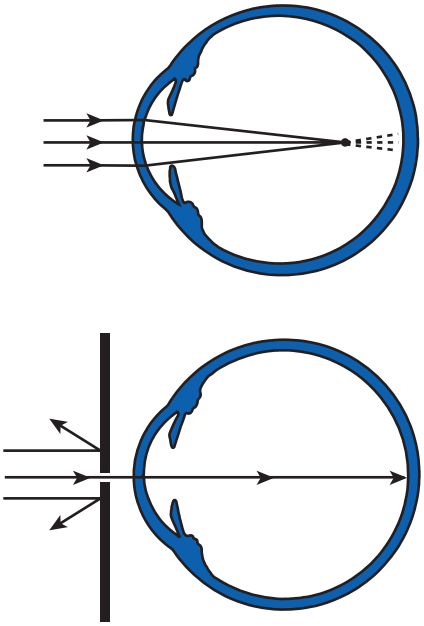


FIGURE 46-71 Pinholes improve vision in person with myopia.

FIGURE 46-72 Pinholes in duct tape to improve vision in person with myopia.

This item cannot be improvised. Use either cloth adhesive tape (already in the medical kit) or duct tape. Duct tape is ideal for nearly all tasks, even on skin when needed (e.g., close wounds, treat blisters, tape ankle). Note, however, that some persons may be sensitive to the adhesive. Fiberglass strapping tape has greater tensile strength and is ideal for joining rigid components, such as taping two ice axes together. However, it is less sticky than duct tape and not as useful for patching torn items. Extra tape can be carried by wrapping lengths of it around pieces of gear or ski poles.

Cyanoacrylate is good for medical use, or to repair holes in down-filled fabrics. Boiled animal tendon can be mixed with boiling water to form a glue, which solidifies well when cooled.

SPECIFIC EQUIPMENT

Plastic Cable Ties

Lightweight cable ties can be used to bind almost anything together (e.g., bind pack frames together for improvised litters or ski poles together for improvised carriers). Ties are also perfect for repairing many items in the backcountry.

Parachute Cord

Parachute cord has hundreds of uses in the backcountry. It can be used for trucker's hitch traction and for tying together complex splints. Parachute cord is light; carry a good supply.

Safety Pins

Box 46-5 lists various uses for safety pins.

Wire

Braided picture-hanging wire works well because it is supple and ties like line. Its strength makes it superior for repairing and improvising components under an extreme load (e.g., fabricating improvised rescue sleds, repairing broken or detached ski bindings). One of the authors (DJM) carries nonbraided floral craft or snare wire for the same purpose, as part of a survival kit.

Bolts and Wing Nuts

Bolts and wing nuts make the job of constructing an improvised rescue sled much easier (see Improvised Rescue Sled or Toboggan, later). Bolts are useful only if holes can be created to put them through. Therefore, a knife with an awl is needed for drilling holes through skis, poles, or other improvised items.

Prefabricated Crossbar

A prefabricated crossbar can be used to construct double–ski pole traction splint systems. A crossbar is easily fabricated from a branch or short section of a ski pole, but carrying a prefabricated device, such as a 15-cm (6-inch) predrilled ski pole section, saves time.

Closed-Cell Foam Pads

Since the introduction of Therm-a-Rest types of inflatable pads, closed-cell foam has become increasingly scarce; however, closed-cell foam is still the ultimate padding for almost any improvised splint or rescue device. The uses for closed-cell foam are virtually unlimited. Even Therm-a-Rest advocates should carry a small amount of closed-cell foam, which is lightweight and doubles as a comfortable seat cushion. Furthermore, unlike inflatable pads, closed-cell foam will not puncture and deflate.

Inflatable pads also have their place and are useful padding for long-bone splints and immobilizers (e.g., improvised universal knee immobilizer). An inflatable pad can also be used to cushion pelvic fractures. First, wrap the deflated pad around the pelvis, then secure the pad with tape and inflate it. It helps to wrap duct tape circumferentially around the pad at the level of the pelvis.

Fluorescent Surveyor's Tape

Surveyor's tape can be used to help locate a route into or out of a rescue scene. It is also ideal for marking shelters in deep snow and can serve as a wind sock during helicopter operations on improvised landing zones. Surveyor's tape is not biodegradable, so it should always be removed from the site after the rescue is completed.

Space Blanket or Lightweight Tarp

For improvising hasty shelters in times of emergency, some form of tarp is essential. In the snow, a slit-trench shelter can be built in a matter of minutes using a tarp. Otherwise, the complex and time-consuming construction of improvised structures, such as snow caves, igloos, or tree branch shelters, might be necessary. Typically, little time or help is available for this task during emergencies. In addition, tarps are essential for "hypothermia wraps" when managing injured persons in cold or wet conditions. The only advantage of a space blanket over other tarps is its small size, which means there is a good chance it is packed for the trip.

IMPROVISED TRANSPORT

CARRIES

Three-Person Wheelbarrow Carry

The three-person system is extremely efficient and can be used for prolonged periods on relatively rough terrain. The patient places his or her arms over the shoulders of two rescuers

BOX 46-5 How to Use a Safety Pin: 22 Improvised Uses

1. To hold the tongue out to maintain an airway, place a pin vertically through the anterior midline of the tongue (see Figure 46-10B).
2. To replace the lost screw in a pair of eyeglasses, which prevents the lens from falling out.
3. To make pinhole eyeglasses.
4. To perform neurosensory skin testing.
5. To puncture plastic bags for irrigation of wounds.
6. To remove embedded foreign bodies from the skin.
7. To drain an abscess or blister.
8. To relieve a subungual hematoma.
9. As a fishhook.
10. As a finger splint (for mallet finger).
11. As a sewing needle, using dental floss as thread.
12. To hold gaping wounds together.
13. To replace a broken zipper on clothing.
14. To hold gloves or mittens to a coat sleeve.
15. To unclog jets in a camping stove.
16. To pin triage notes to patients' clothing.
17. To remove a corneal foreign body (with ophthalmic anesthetic).
18. To secure a sling and swath for shoulder or arm injuries.
19. To fix a ski binding.
20. To extract the clot from a thrombosed hemorrhoid.
21. To pin a strap or shirt tightly around the chest for rib fracture support.
22. To remove ticks.

FIGURE 46-73 Three-person wheelbarrow carry. The patient places his or her arms over two rescuers' shoulders (the rescuers stand side by side). The patient's legs are then placed over a third rescuer's shoulders.

standing side by side. The patient's legs are then placed over a third rescuer's shoulders. This system equalizes the weight of the patient very efficiently (Figure 46-73).

Two-Hand Seat

Two carriers stand side by side. Each carrier grasps the other carrier's wrists with his or her opposite hand (e.g., right hand to left). The patient sits on the rescuers' joined forearms. The carriers might maintain one free hand to place behind the back of the patient for support (support hands can be joined). This system places great stress on the carriers' forearms and wrists.

Four-Hand Seat

Two carriers stand side by side. Each carrier grasps his or her own right forearm with the left hand, palms facing down. Each carrier then grasps the forearm of the other with his or her free hand to form a square "forearm" seat. With the forearm seat, the patient must support himself or herself with a hand around the rescuers' backs (Figure 46-74).

Ski Pole or Ice Ax Carry

Two carriers with backpacks stand side by side with two to four ski poles or joined ice ax shafts resting between them and the base of the pack straps (Figure 46-75). The ski poles or ice ax shafts can be joined with cable ties, adhesive tape, duct tape, wire, or cord. Because the rescuers must walk side by side, this technique requires wide-open, gentle terrain. The patient sits on the padded poles or shaft with his or her arms over the carriers' shoulders.

Split-Coil Seat

The split-coil seat ("Tragsitz") transport method uses a coiled climbing rope to join the rescuer and patient together in a piggyback fashion (Figure 46-76). The patient must be able to support himself or herself to avoid falling back, or must be tied in.

Two-Rescuer Split-Coil Seat

The two-rescuer split-coil seat is essentially the same as the split-coil transport, except that two rescuers split the coil over their shoulders. The patient sits on the low point of the rope between the rescuers (Figure 46-77). Each rescuer maintains a free hand to help support the patient.

Backpack Carry

A large backpack could be modified by cutting leg holes at the base, although this destroys the pack. The patient sits in it as a child would in a baby carrier. Some large internal-frame packs incorporate a sleeping bag compartment in the lower portion of the pack that includes a compression panel. With this style of pack, the patient can sit on the suspended panel and place the legs through the unzipped lower section without damaging the pack, or the patient can simply sit on the internal sleeping bag compression panel, without the need to cut holes. The rescuer can also place the upright patient's legs into the straps of the pack, then wear the pack as usual (Figure 46-78).

Nylon Webbing Carry

Nylon webbing can be used to attach the patient to the rescuer like a backpack (Figure 46-79). At least 4.5 to 6 m (15 to 20 feet) of nylon webbing is needed to construct this transport. The center of the webbing is placed on the patient like a climbing harness (Figures 46-79A and B). The webbing ends are then crossed and brought over the rescuer's shoulders, then down around the back of the patient's thighs. Finally, the webbing ends are brought forward and tied around the rescuer's waist. Additional padding is needed for this system, especially around the posterior thighs of the patient.

NONRIGID LITTERS

Many nonrigid litter systems have been developed over the years. These systems are best suited for transporting noncritically injured patients over moderate terrain.

Blanket Litter

A simple nonrigid litter can be fabricated from two rigid poles, branches, or skis and a large blanket or tarp. The blanket or tarp is wrapped around the skis or poles as many times as possible, and the poles are carried. The blanket or tarp should not be simply draped over the poles, but securely fastened. For easier carrying, the poles can be rigged to the bases of backpacks. Large external-frame packs work best, but internal-frame packs can be rigged to do the job. Alternatively, a padded harness to support

FIGURE 46-74 Four-hand seat carry.

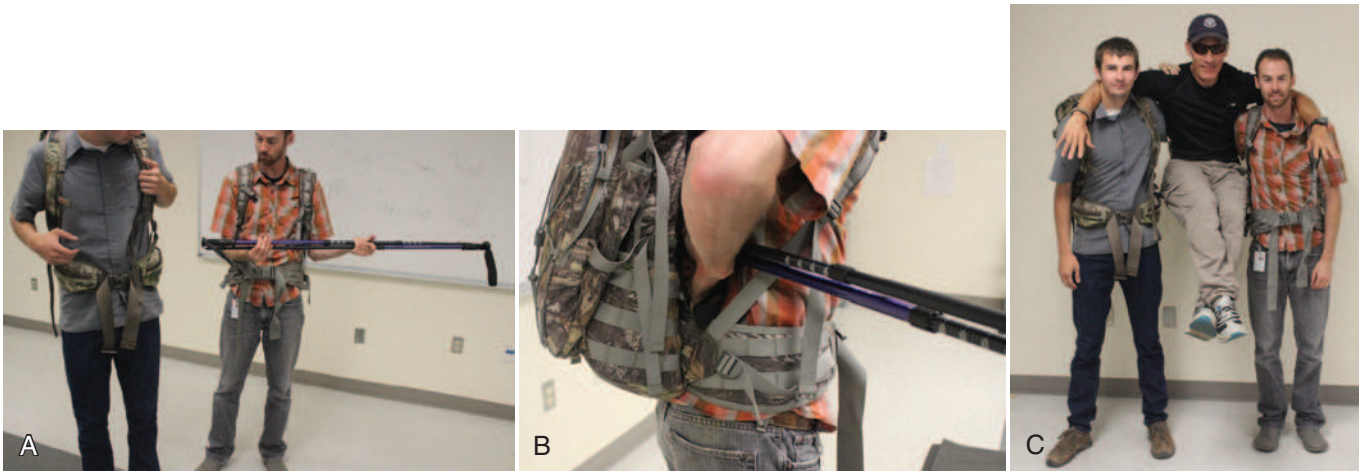


FIGURE 46-75 Ski pole seat. **A** and **B,** Ski poles are anchored by the packs. **C,** The patient is supported by the rescuers.

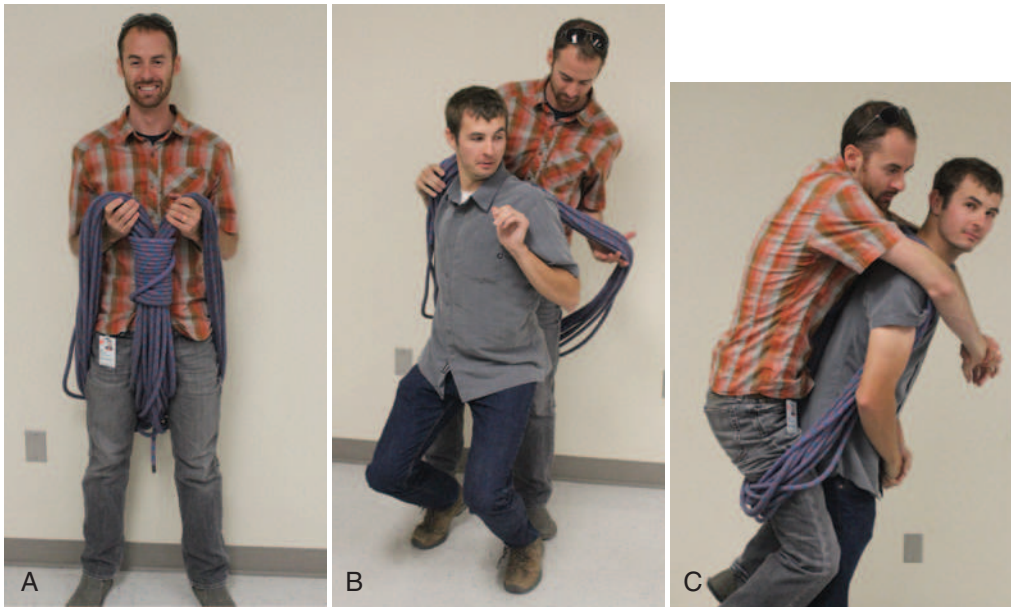


FIGURE 46-76 Split-coil seat. **A,** Rope coil is split. **B,** Patient climbs through rope. **C,** Rescuer hoists the sitting patient.

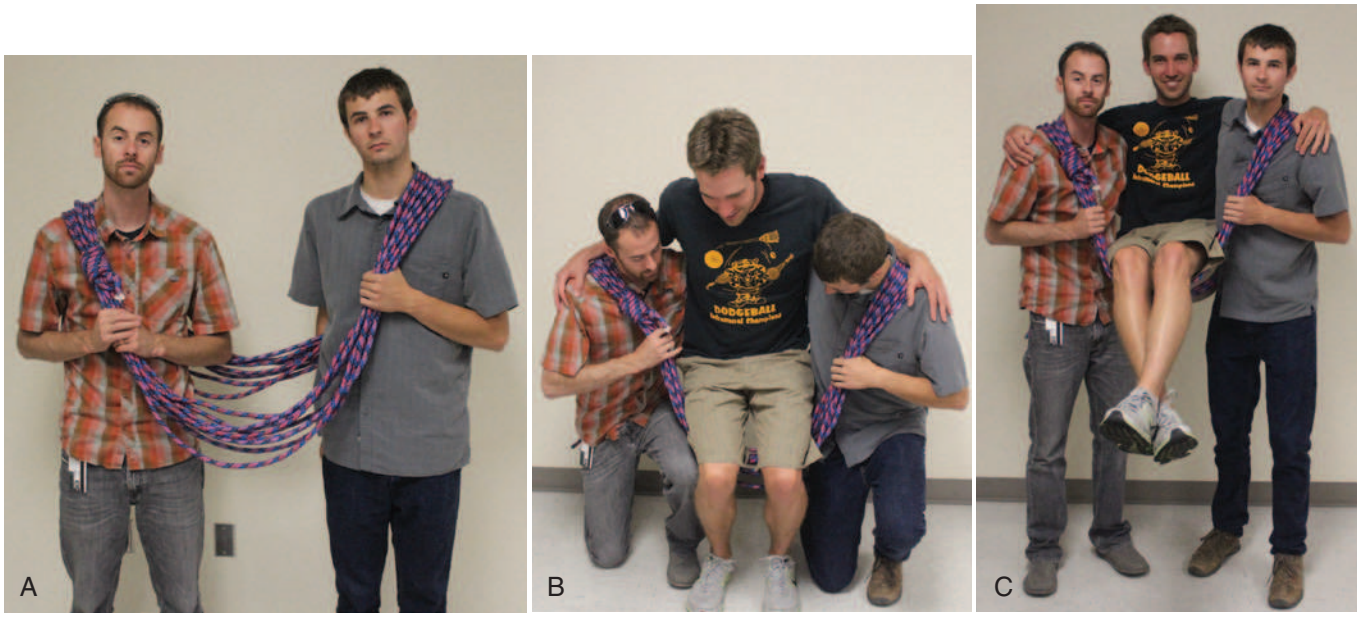


FIGURE 46-77 Two-rescuer split-coil seat. Balance could be improved by using a longer coil to carry the patient lower.

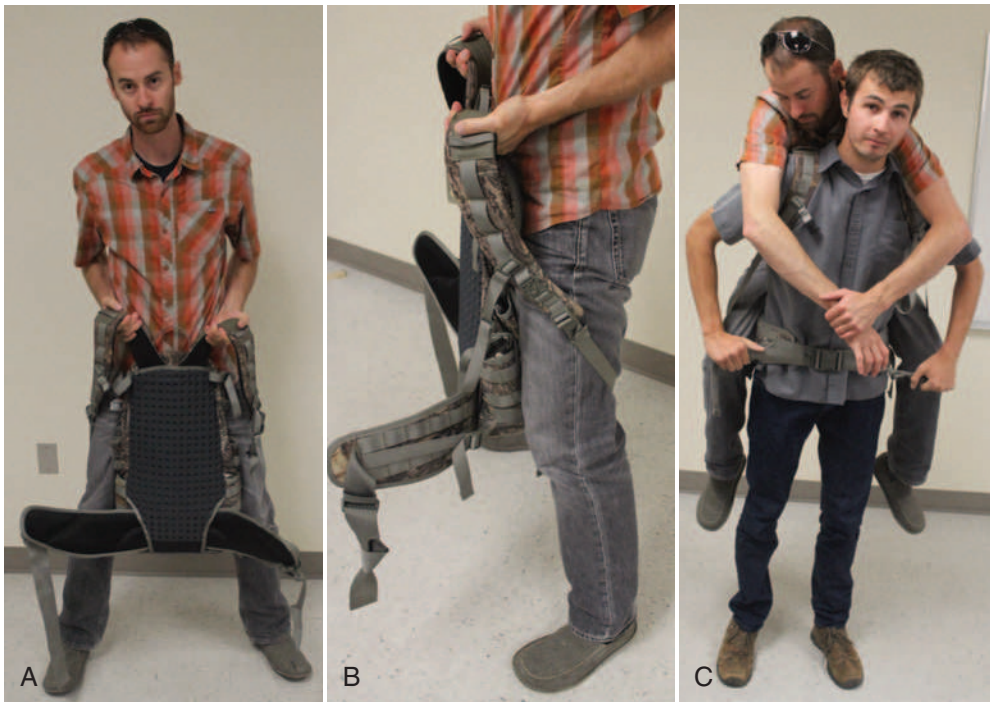


FIGURE 46-78 Backpack carry. **A** and **B,** Place patient's legs through the shoulder straps from behind the backpack. **C,** Rescuer places and secures the backpack in usual fashion.

the litter can be made from a single piece of webbing, in a design similar to a nylon webbing carry.

Tree Pole Litter

The tree pole litter is similar to the blanket litter. In the tree pole litter, however, instead of a blanket or a tarp, the side poles are laced together with webbing or rope and then padded. Again, the poles may be fitted through pack frames to aid carrying. To give this litter more stability and to add tension to the lacing, the rescuer should fabricate a rectangle with rigid crossbars at both ends of the poles before lacing.

Parka Litter

Two or more parkas can be used to form a litter (Figure 46-80). Skis or branches are slipped through the sleeves of heavy parkas, and the parkas are zipped shut with the sleeves inside. Ski edges should be taped first to prevent them from tearing through the parkas.

Internal-Frame Pack Litter

The internal-frame pack litter is constructed from two to three full-size internal-frame backpacks that must have lateral

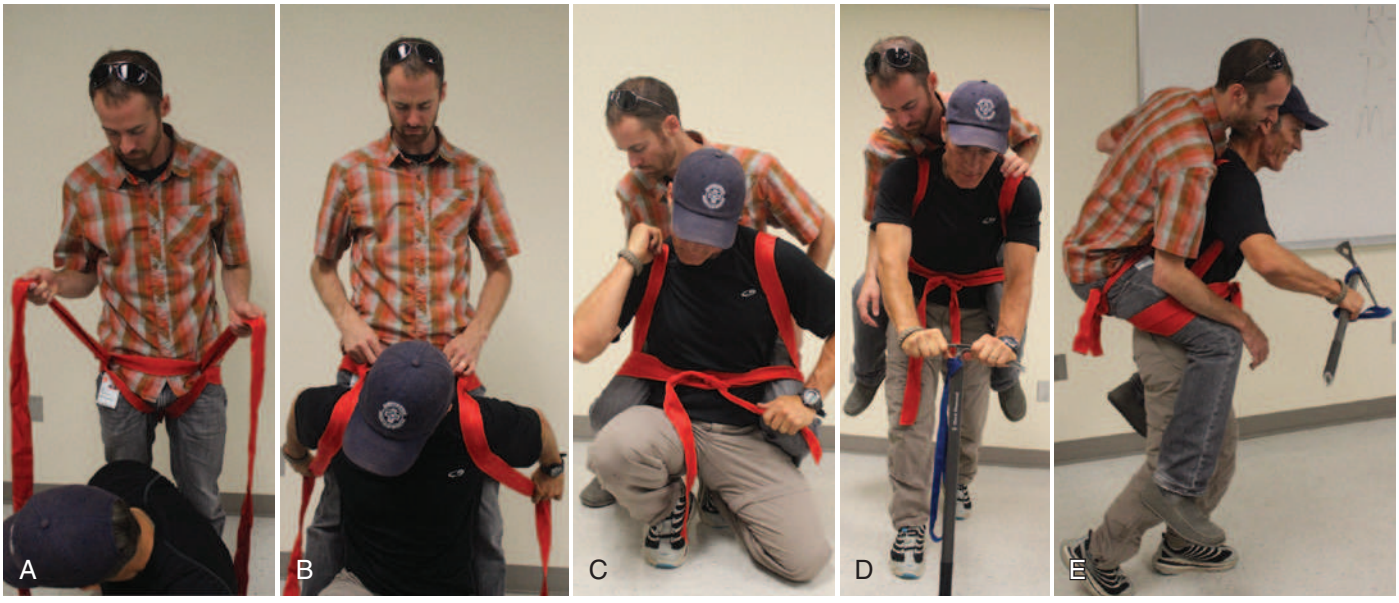


FIGURE 46-79 Webbing carry. Webbing crisscrosses in front of the patient, using the central part of the webbing like a climbing harness, before passing the ends over the rescuer's shoulders, around the patient's thighs, and finally around the rescuer's waist. A pole, staff, or ice axe assists the rescuer to stand properly.

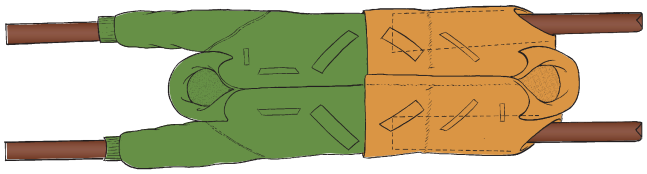


FIGURE 46-80 Parka litter. On the right, the sleeves are zipped inside to reinforce the litter.

compression straps (day packs are suboptimal). Slide poles or skis through the compression straps; the packs then act as a supportive surface for the patient.

Personal Floatation Device (Life Jacket) Litter

Person flotation devices (PFDs) can be placed over paddles or oars to create a makeshift nonrigid litter.

Rope Litter

On mountaineering trips, the classic rope litter can be used, but this system offers little back support and should not be used for patients with suspected spine injuries. The rope is uncoiled and staked onto the ground with sixteen 180-degree bends (eight on each side of the rope's center). The rope bends should approximate the size of the finished litter. The free rope ends are then used to create a clove-hitch to tie off each bend (leaving 5 cm [2 inches] of bend to the outside of each clove hitch). The leftover rope is threaded through the loops at the outside of each clove hitch. This gives the rescuers a continuous handhold and protects the bends from slipping through the clove hitches. The rope ends are then tied off. The litter is padded with packs, Therm-a-Rest pads, or foam pads. This improvised litter is somewhat ungainly and requires six or more rescuers for an evacuation of any distance. A rope litter can be tied to poles or skis to add lateral stability if needed.

IMPROVISED RESCUE SLED OR TOBOGGAN

A sled or toboggan can be constructed from one or more pairs of skis and poles that are lashed, wired, or screwed together. Many designs are possible. Improvised rescue sleds may be clumsy and often bog down hopelessly in deep snow. Nonetheless, they can be useful for transporting a patient over short distances to a more sheltered camp or to a more appropriate landing zone. They have sometimes been used for more extensive transports, but do not perform as well as easy-to-carry commercial rescue sleds, such as the Brooks-Range Rescue Sled (Brooks-Range Mountaineering, Fremont, California).

To build an improvised rescue sled or toboggan, the rescuer needs a pair of skis (preferably the patient's) and two pairs of ski poles; three 61-cm (2-foot)-long sticks (or ski pole sections); 24 m (80 feet) of nylon cord; and extra lengths of rope for sled hauling.

The skis are placed 0.6 m (2 feet) apart. The first stick is used as the front crossbar and is lashed to the ski tips. Alternatively, holes can be drilled into the stick and ski tips with an awl, and bolts can be used to fasten them together. The middle stick is lashed to the bindings. One pair of ski poles is placed over the crossbars (baskets over the ski tips) and lashed down. The second set of poles is lashed to the middle stick with baskets facing back toward the tails. A third rear stick is placed on the tails of the skis and lashed to the poles. The lashings are not wrapped around the skis; the crossbar simply sits on the tails of the skis under the weight of the patient. Nylon cord is then woven back and forth across the horizontal ski poles. The hauling ropes are passed through the baskets on the front of the sled. The ropes are then brought around the middle crossbar and back to the front crossbar. This rigging system reverses the direction of pull on the front crossbar, making it less likely to slip off the ski tips.[123]

Another sled design uses a predrilled snow shovel incorporated into the front of the sled. A rigid backpack frame can also be used to reinforce the sled. This requires drilling holes into the ski tips and carrying a predrilled shovel. This system holds the skis in a wedge position and may offer slightly greater durability.[110]

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Complete references used in this text are available online at expertconsult.inkling.com.

CHAPTER 47 Principles of Pain Management

ANDREW A. HERRING, MICHAEL L. KENT, AND MEGANN YOUNG

Wilderness travel can involve pain and discomfort ranging from sore feet to a major traumatic injury. Effective and tailored management of pain is crucial to the overall success of an expedition.[25,45,86,108] Maximizing comfort and analgesia reduces both the physiologic and the psychological stress of acute injury, which has obvious benefits in a wilderness setting, where prolonged care, harsh environmental conditions, and complex evacuation can occur.[30] Conversely, undertreated pain—all too common in the prehospital setting—has significant adverse consequences, including preventable suffering, delirium, aggravation of the systemic stress response to injury, and distraction from essential tasks (Table 47-1). Undertreatment of pain frequently results from failure to prioritize pain treatment, lack of necessary supplies, and deficient provider training in advanced acute pain management.[2,11,31,85]

Optimal wilderness pain management includes integrated pain assessment and treatment at the point of contact,[45] a synergistic combination of complementary analgesic agents and modalities,[108] use of regional anesthesia whenever appropriate, and attention to associated problems, such as nausea and anxiety[25,60,86] (Figure 47-1). Each intervention should be tailored to work synergistically to reduce pain while also minimizing the unwanted adverse effects of any one medication, as may occur with large doses of opioids. This strategy, which employs a broad combination of pharmaceutical, psychological, interventional, and complementary strategies, is referred to as *multimodal analgesia*[15,39,60,63,113] (Figure 47-2 and Table 47-2).

This chapter provides a guide to the practical application of state-of-the-art multimodal analgesia in austere and wilderness settings (Table 47-3). With a multimodal approach, providers

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TABLE 47-1 Adverse Consequences of Inadequate Analgesia

Category	Effects
Patient experience	Decreased patient satisfaction Needless suffering
Physiologic	Metabolic stress response Hyperglycemia Protein catabolism Increased free fatty acids (decreased myocardial contractility, increased myocardial oxygen demand, impaired vasodilation) Hypercoagulability Thromboembolic events Pulmonary complications Immunosuppression Delirium Development of chronic pain states Complex regional pain syndrome
Long-term psychological	Posttraumatic stress disorder Insomnia
Logistic	Increased duration of intensive care unit and hospital stay Decreased participation in rehabilitation

integrate many diverse interventions, including cognitive behavioral therapy, topical preparations, opioids, ketamine, nonsteroidal antiinflammatory drugs (NSAIDs), anxiolytics, acupuncture, and regional anesthesia, into a creative and adaptive analgesic strategy to meet the challenge of effective pain management in the uniquely challenging wilderness environment.

FIRST CONTACT

SCENE STABILIZATION

Fundamental actions to stabilize and de-escalate an acute injury or pain event are as follows:

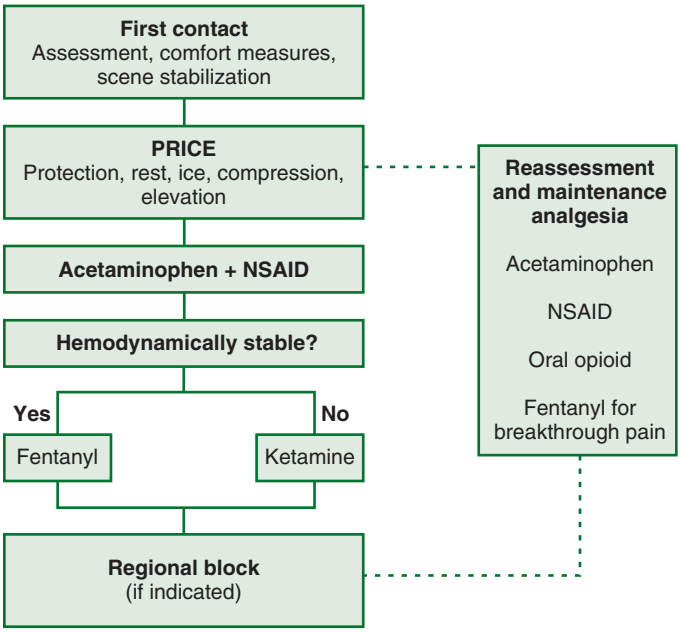


FIGURE 47-1 Summary approach for pain management in the wilderness.

1. Create a safe and calm environment with clear leadership for pain treatment. Chaotic and stressful situational factors, such as obvious threats, environmental exposure, noise, and the presence of nonessential personnel, should be addressed as soon as possible.[101]
2. Clearly communicate reassurance, allay fears, and encourage calm and confidence. Recruiting persons of trust to provide companionship and calming words can be helpful.
3. After completing a primary survey to assess and manage life-threatening injuries, objectively assess the nociceptive pain generator (injury), functional status of the injured party, and expedition situation. For example, a minor animal bite is typically accompanied by a degree of surprise and fear but can be expected to result in moderate pain, whereas a patient with a long-bone fracture may present quietly in a partially dissociated or "stunned" state but will have severe ongoing pain and functional limitation.
4. A basic history, including medication allergies, should be obtained if possible.[11,25,86]

COMFORT MEASURES

Fear, anxiety, and confusion are potent stimulators of pain.[30] From the point of contact throughout the course of any wilderness pain situation, utilization of psychosocial strategies should not be overlooked (Box 47-1). For some patients, fear and associated anxiety may be primary drivers of their suffering.[8] When possible, the best care is provided in a location protected from environmental exposure with a clearly designated and trusted caregiver who provides reassurance and displays organization and competency. In this setting, pain can be best assessed and a comprehensive analgesic plan developed.[25,86]

Tactical Breathing

Coaching a patient through a simple technique of controlled breathing, referred to as "combat tactical breathing" by the U.S. Armed Forces, can effectively produce calm and reduce fear in acutely injured patients.[92] Encourage patients to breathe from their diaphragm, allowing the stomach to expand as they breathe in and to contract as they breathe out. Have the patient take three to five breaths, visualizing each number as they count, as follows:

- Breathe in while counting "1-2-3-4."
- Stop and hold breath while counting "1-2-3-4."
- Exhale while counting "1-2-3-4."
- Repeat the breathing for three to five cycles.

PAIN ASSESSMENT

Analgesic interventions should be balanced against the physical and cognitive demands to be expected of the injured person. Thorough analysis of the global situation should shape an analgesic plan that maximizes comfort while preserving necessary functional capacity. Fundamentally, the essential goal is to promote performance of essential duties by maximizing tolerability of acute injury through analgesia, while minimizing potentially disabling cognitive impairment and sedation.

Accurate wilderness pain assessment is important because both undertreatment and overtreatment of pain have potentially serious consequences.[60] Undertreated pain potentially results in needless suffering, reduced functional capacity, and worsening injury.[11,45,85] Overtreatment of pain may waste limited resources and reduce functional capacity, as with oversedation using an opioid or loss of limb function from a nerve block.[52]

Pain assessment can be facilitated by consideration of the three primary domains of the pain experience—the nociceptive pain generator, patient self-appraisal of the pain, and patient expressions of the pain—as part of an overall assessment of the individual and the wilderness scenario. The outward expression of pain is the external manifestation of the interaction between the actual tissue injury (the nociceptive pain generator), how the team member is interpreting the injury (self-appraisal; confident in safety vs. the perception of threat), and how the team member expresses pain (stoic vs. agitated) (Figure 47-3). Pain

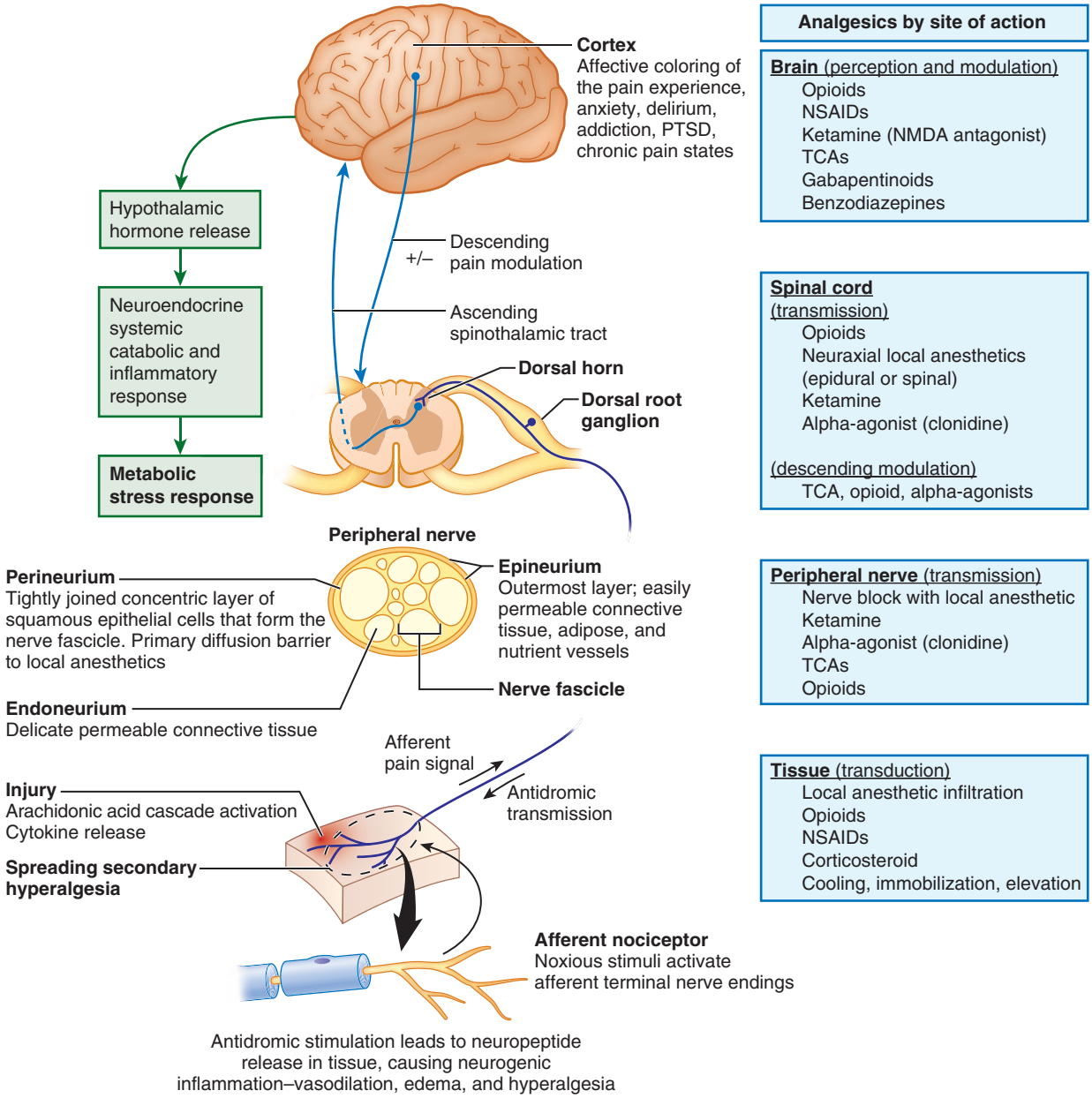


FIGURE 47-2 Pathophysiology and anatomy of pain and common multimodal analgesics. NMDA, *N*-methyl-D-aspartate; NSAIDs, nonsteroidal antiinflammatory drugs; TCAs, tricyclic antidepressants.

assessment is ultimately a subjective and iterative process linked in real time to analgesic interventions and ongoing situational analysis.[16,108]

In situations of prolonged analgesic titration, such as a lengthy evacuation, the Defense and Veterans Pain Rating Scale provides a useful method for ongoing trending of the efficacy of analgesic interventions that combines pain self-report of pain severity, emotional response, and behavior[12] (Figure 47-4).

PRICE: Protection, Rest, Ice, Compression, and Elevation

After first contact, significant pain relief can be achieved through commonsense physical *protection* and stabilization of an injured extremity, removal of constricting garments, and movement to a position of maximum comfort. *Rest* minimizes additional pain and inflammation.

Ice or another cold application (e.g., immersing a sprained ankle in a cold stream) can reduce inflammation and pain from an acute injury. Ice should be applied intermittently by alternating ice with no ice in 20-minute intervals to avoid the risks of decreased blood flow to the region, which could result in decreased oxygen delivery, decreased clearance of cellular byproducts, or even frostbite. A towel or a similar material should be placed between the ice and skin, and tissue status closely monitored.[26,86]

Compression with a close-fitting elastic bandage can reduce swelling and associated pain after an acute extremity injury. The bandage should provide support, protection, and light compression that does not constrict blood flow. As an injury evolves and swelling increases, regular checks of the bandage and distal tissues should be done to prevent potentially harmful constriction.[7] Care should be taken when using a nonelastic bandage that could result in overcompression with reduction of sufficient blood flow and potentially ischemia.

Distended tissues increase pain. *Elevation* reduces swelling by increasing venous return of blood and alleviating tissue edema. Left in the dependent position, extremity swelling increases after injury, potentially causing compression of the veins, which in turn leads to worsening bleeding, swelling, and pain. Further swelling may compress nerves and arteries (i.e., compartment syndrome), which will result in ischemia distal to the injury. If possible, the injured site should be maintained above the level of the heart.[7,68,86]

TABLE 47-2 Multimodal Analgesia Terms

Term	Definition and Description
Transduction	Chemical, thermal, or mechanical noxious stimulus is converted into an electrochemical signal by pain receptors in the skin, the periosteum, and joint surfaces referred to as *nociceptors.* Thinly myelinated Aδ and unmyelinated C fibers transmit nociception to the spinal cord. Fast-conducting Aδ fibers respond to mechanical stimuli and transmit sharp sensations. Slow-conducting C fibers respond to chemical, thermal, and mechanical stimuli and transmit burning sensations. Acute injury of the skin results in simultaneous transmission through Aδ and C fibers. Cell bodies of the Aδ and C fibers are located in the dorsal root ganglion, with projections to the posterior horn of the spinal cord. Aδ and C fibers synapse with second-order neurons in the dorsal horn that cross to the contralateral side of the spinal cord and ascend in the anterolateral spinal tracts to the thalamic nuclei, brainstem, and midbrain, where they synapse with third-order neurons and project to the cerebral cortex. At the site of tissue damage, proinflammatory pronociceptive substances are released, including substance P, bradykinin, and glutamate, initiating pain transmission and sensitizing surrounding nociceptors. Antidromic collateral nerve pathways produce hyperalgesia, vasodilation, and edema in tissue surrounding the noxious stimulus. An opposing system of pain-inhibiting substances, such as endogenous opioids, β-endorphins, acetylcholine, γ-aminobutyric acid, and somatostatin, impedes nociceptive transmission.
Transmission	The signal is conducted to the central nervous system (CNS) via Aδ and C fibers. At the level of the spinal cord, nociception and the autonomic nervous system (ANS) are under descending inhibitory or facilitatory modulation from supraspinal levels.
Modulation	The brain is able to independently modulate pain, either suppressing or enhancing pain transmission. Serotonin and norepinephrine appear to be the primary neurotransmitters that regulate descending inhibitory control; however, others (e.g., α_2-agonists, cannabinoids) may be involved. The most well-described facilitatory or pronociceptive pathways involve *N*-methyl-D-aspartate (NMDA) receptor transmission.
Perception	Lastly, the signal is consciously appreciated as pain as the end result of a complex system initiated by stimulating peripheral nociceptors in the presence of a milieu of local mediators, modulated and transmitted via the spinal cord and finally interpreted and perceived at the cerebral level.

PHARMACOLOGIC TREATMENT OF ACUTE PAIN IN THE WILDERNESS

SUMMARY OF INTEGRATED MULTIMODAL ANALGESIA FOR AN INJURED PATIENT IN THE WILDERNESS

Broadly, persons injured in the wilderness can be treated according a scheme developed by the U.S. military for soldiers injured on the battlefield: Tactical Combat Casualty Care battlefield analgesia recommendations[14] (see Table 47-3). These guidelines have emerged from recent large-scale experiences managing severe pain in austere conditions during military operations and represent a state-of-the-art approach consistent with the available evidence at this time.[6,60,88]

Injured patients in the wilderness with mild to moderate pain able to take oral medication should be given a base of acetaminophen plus an NSAID. Any extremity injury should be evaluated for a regional nerve block as soon as this is practically feasible, if such equipment is available.[61,81,96]

Persons with moderate to severe pain not controlled with acetaminophen plus an NSAID should be considered for escalation of analgesia with a rapid-acting opioid. If opioids are used, both naloxone for reversal of respiratory depression and an antiemetic such as diphenhydramine, ondansetron, or promethazine should be immediately available. An oral transmucosal fentanyl citrate lozenge is an ideal option that can

TABLE 47-3 Multimodal Wilderness Analgesia

Step	Goals	Strategies
First contact	Establish therapeutic alliance; defuse anxiety and fear.	Designate a caregiver who can communicate calm, concern and reassurance; display focused attention, organization, and competence.
	Make situational pain assessment to guide triage.	Assess initial pain severity in the context of anticipated physical challenges ahead.
Stabilization	Remove any provoking physical situation or garment.	PRICE: Protect and splint injured extremities; provide rest, ice/cryotherapy, compression wrappings, and elevation.
	Extricate patient from injurious situation or garments; move to calm, quiet, and secure area.	
Pharmacologic treatment	Analgesia for mild to moderate pain Patient able to take oral medicines	Acetaminophen, 1000 mg, plus NSAID (ibuprofen, 400-800 mg) orally
	Analgesia for moderate to severe pain Patient able to take oral medicines without signs of shock	Acetaminophen, 1000 mg, plus NSAID (ibuprofen, 400-800 mg) orally Transmucosal fentanyl, 800 mcg every 15 minutes, *or* fentanyl, 100 mcg IN, *or* fentanyl, 50 mcg IV
	Analgesia for severe pain with concern for shock	Ketamine, 50 mg IM or 20 mg IV, repeated every 20 minutes IV or 30 minutes IM
Regional anesthesia	Place block as close to time and place of injury as feasible and safe.	Anatomic and situational assessment for regional anesthesia

IM, Intramuscularly; *IN,* intranasally, *IV,* intravenously; *NSAID,* nonsteroidal antiinflammatory drug.

BOX 47-1 Wilderness Pain Assessment: Principle and Special Issues Basic Principle

Is the pain tolerable?
What can be done to make the pain tolerable while maximizing functional capacity required for the wilderness situation?

Guided Imagery or Self-Hypnosis

This is often overlooked as a modality for pain management. The ability to transport the sensorium out of the painful experience into a different context is the aim of this technique. Although it is seemingly in the realm of a vaudeville show, self-hypnosis has been demonstrated to lessen the pain experience of patients with breast cancer, and it is putatively thought to activate descending inhibitory pathways. Although these techniques are often viewed in the context of pain relief, their use to heighten the state of concentration has other uses in human performance, so learning the techniques before traveling is worth the investment of time.

The mind is capable of extraordinary feats, as illustrated by the experience of Aron Ralston, whose arm was trapped between a rock and a canyon wall while canyoneering alone in a remote area of Utah. Aron's initial attempts to free himself produced excruciating pain. After reassessing his situation, relaxing, and making a plan, he touched his crushed hand and realized that it had lost sensation. This produced a sense that his arm was isolated from his body. He noted the disfigurement of this arm and observed that is was strange that he was not suffering pain. Unable to free himself and stranded alone with a limited water supply, he concluded that he needed to amputate his own arm and to focus all his attention on this task. Not able to cut the bone, he levered the trapped arm, breaking the ulna and radius bones and thus freeing the arm. Although he felt a "blaze of pain" with the maneuver, he refocused on survival and was able to make his way to rescue. This is a story of an extraordinary person that illustrates the power of the human mind.[82]

Combination Acetaminophen and Opioid Products Are *Not* Recommended for Wilderness Analgesia

In the United States, combination products containing acetaminophen and an opioid, such as oxycodone and hydrocodone, are often prescribed for pain. In general, these products contain a very low dose of opioid (e.g., 5 mg of hydrocodone) and are weak analgesics, but they have sufficient opioid to produce psychomotor impairment, difficulty concentrating, and subjective feelings of dizziness or "dreaminess" in most opioid-naive individuals. Thus, the combination products tend to promote a worst-case scenario: a small dose of opioid that produces only mild analgesia with potentially significant functional impairment. A superior strategy is conscientiously to apply maximal nonopioid analgesia with selective and closely titrated opioid as needed to achieve analgesic and functional capacity goals.[105,114]

be repeated every 15 minutes as needed.[109] Alternatively, intranasal[53] or intravenous[91] fentanyl can be used.

Severely injured patients in severe pain at risk for cardiopulmonary or hemorrhagic instability are preferentially treated with ketamine because of the powerful analgesia with reduced respiratory or cardiovascular suppression.[36,47,49,79,88] Additional doses can be repeated every 15 minutes intravenously (IV) or 30 minutes intramuscularly (IM). It is now widely accepted that ketamine does not need to be withheld in patients with eye injuries or traumatic brain injury because its effect on intraocular and intracranial pressure is not likely to be clinically significant. Ketamine may be used after patients have received opioids.[14,67]

ACETAMINOPHEN

Acetaminophen is generally a weak analgesic when used alone but has been shown to provide surprisingly strong enhancement of other analgesics (e.g., NSAIDs, opioids).[60,113] The exact mechanism of action of acetaminophen is not well understood, and it has no known endogenous binding sites. Acetaminophen is not an antiinflammatory agent and lacks known peripheral cyclooxygenase (COX)-1 or COX-2 activity. There may be some, as yet poorly defined, central nervous system (CNS) COX and serotonin (5-HT) neurotransmission activity. Acetaminophen can be given orally (PO) or IV. Intravenous administration more rapidly achieves a higher CNS concentration than when the drug is given PO. Acetaminophen is generally well tolerated without the gastrointestinal (GI) adverse effects of the COX inhibitors. Patients should not take more than 4 g of acetaminophen in 24 hours (2 g in patients with preexisting liver disease) because hepatic toxicity may occur above these levels.[35]

NONSTEROIDAL ANTIINFLAMMATORY DRUGS

The NSAIDs are very effective analgesics for a wide range of conditions, from acute fractures to migraine headaches. Prostaglandins are released by the COX-1 and COX-2 enzymes in response to tissue damage, promoting a nociceptive response (pain) and local inflammation.[21,30] COX inhibition is also responsible for many of the adverse effects of NSAIDs, including platelet inhibition, GI complications, impaired bone and tendon healing, and kidney injury. Selective COX-2 inhibitors (e.g., celecoxib, 200 mg PO, or meloxicam, 15 mg PO) have similar analgesic efficacy but lack the platelet inhibition and associated bleeding risks of nonselective COX inhibitors (e.g., ibuprofen).[60,63] There is likely no advantage to intramuscular (IM) or intravenous (IV) NSAIDs (e.g., ketorolac) if the patient is able to take medications by mouth.[4]

In wilderness settings, when treating a significant injury that may have hemorrhagic complications, a selective COX-2 inhibitor is preferred.[14] GI injury and bleeding, as well as acute kidney injury, are well-known risks associated with NSAIDs.[21] In particular, kidney injury may be more likely in the volume-depleted wilderness patient, so aggressive hydration should accompany NSAID use.

OPIOIDS

Opioids have been used to ease pain and suffering for thousands of years. Understanding how best to use opioids as part of a multimodal approach is an essential component of wilderness pain management (Table 47-4). Most opioids can be given by a variety of routes: IM and IV are the most common, but recently, intranasal and transmucosal administration are becoming more

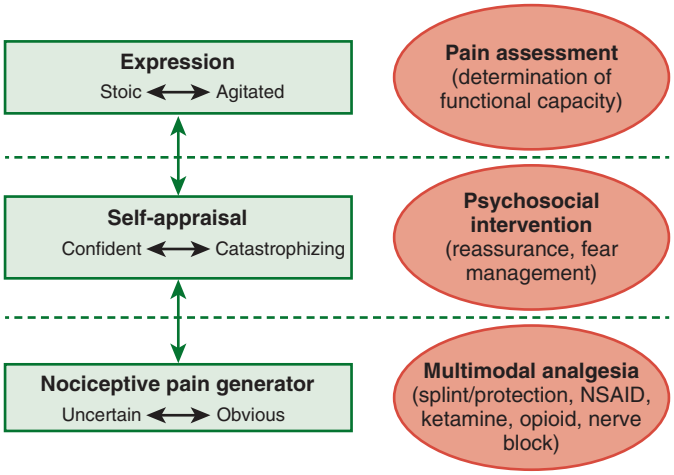


FIGURE 47-3 Conceptual map of the psychology and subjectivity of pain expression and assessment. When encountering a patient in pain, the observed state is the culmination of a complex state of internal processes. How patients express their pain can vary by personal and cultural norms between stoicism and agitation. Outward expression of pain severity may not relate directly to the severity of injury, which complicates assessment. Internal appraisal of the injury significantly influences the pain experience. Patients who believe their pain is a sign of serious illness may have increased fear that in turn increases pain-related suffering; reassurance helps promote confidence. Finally, assessing the actual tissue injury as independent from the subjective response is crucial to planning appropriate analgesia.

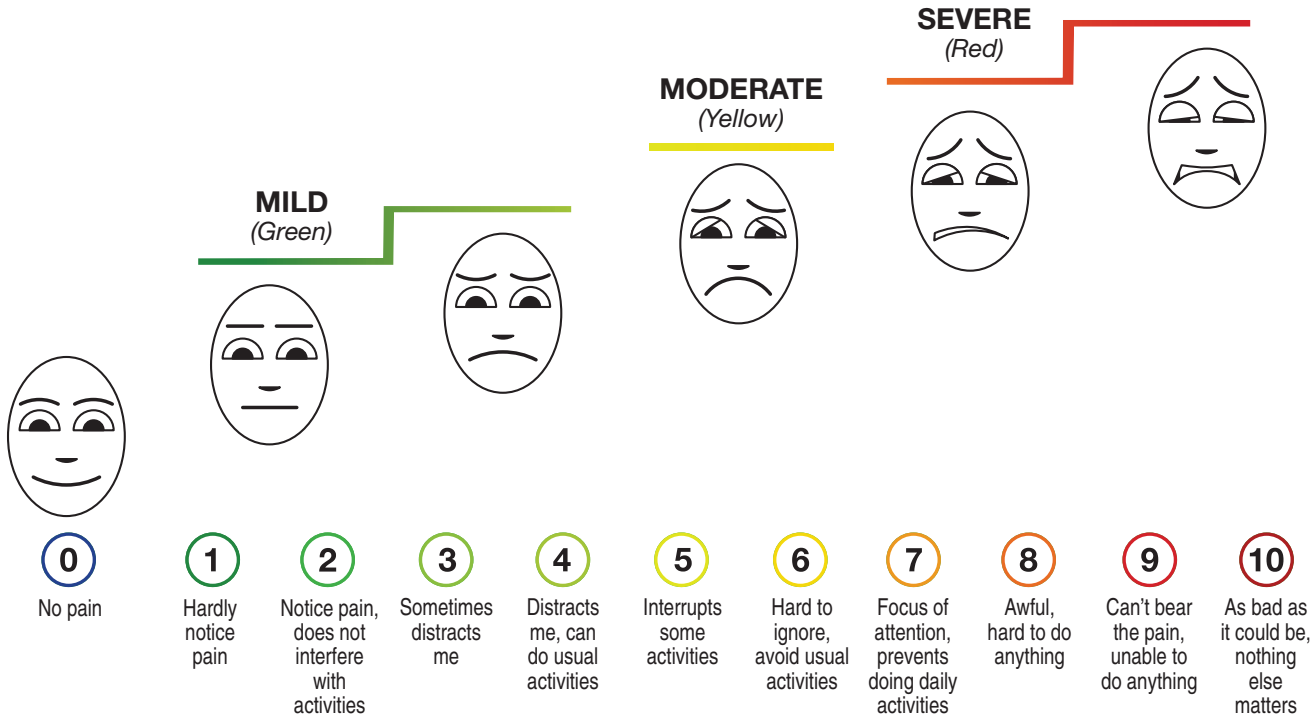


FIGURE 47-4 Defense and Veterans Pain Rating Scale. *(From Buckenmaier CC III, Galloway KT, Polomano RC, et al: Preliminary validation of the Defense and Veterans Pain Rating Scale (DVPRS) in a military population,* Pain Med *14(1):110-123, 2013.)*

TABLE 47-4 Common Opioids Used in Acute Analgesia

	Fentanyl	Hydromorphone	Morphine
Comments	100% synthetic that is strongly lipophilic with rapid onset and minimal histamine release	Semisynthetic morphine derivative that is moderately lipophilic with moderate histamine release	Natural alkaloid derived from opium that is lipophobic with delayed CNS accumulation; potent histamine release
Onset after (IV bolus)	<1 minute	3-5 minutes	5-10 minutes
Peak effect (IV bolus)	2-5 minutes	8-10 minutes	10-15 minutes
Duration (IV bolus)	30-45 minutes	1-4 hours	1-4 hours
Equianalgesic dosing (IV bolus)	100 mcg	1.5 mg	10 mg

CNS, Central nervous system; *IV,* intravenous.

widely used. Most opioids used in wilderness analgesia are primarily selective mu (μ) receptor agonists; μ stimulation produces analgesia as well as euphoria, respiratory depression, constipation, and miosis. Opioids have agonist activity at multiple additional classes of receptors, including kappa (κ), delta (δ), and sigma (σ), each with their own subtypes.[21] Because potentially fatal respiratory depression can occur with any opioid, naloxone for IV, IM, or intranasal reversal should be readily available whenever opioids are used.

The complexity of opioid interaction with these receptors underlies both the subtle and the dramatic differences in clinical effects between opioids and among patients. The most important of the mediators of unwanted side effects is the κ receptor, which promotes GI dysmotility and dysphoria. In general, μ selectivity decreases as the opioid dose increases, resulting in reduced incremental additive analgesia with a relatively greater increase in severity of largely undesirable side effects (Table 47-5). The aim of opioid-sparing multimodal regimens is to allow use of the lowest possible opioid dose, thereby achieving maximal analgesia with minimal side effects.[15,39,80]

Opioids Require Close Titration To Be Effective

The effective clinical dose at which side effects are minimized and analgesia is maximized is determined through individualized rapid drug titration using short intervals[5,17] (Table 47-6). The clinical potency of an opioid for a given individual is notoriously difficult to determine simply by weight-based calculations. Numerous studies have suggested that both overdosing and underdosing are common in the emergency and prehospital settings, likely in part because of empirical dosing with insufficient

TABLE 47-5 Limitations of Opioid Analgesics

Category	Limitations
Acute side effects	Respiratory depression, nausea and vomiting, pruritus, vasodilation and hypotension, immunosuppression
Logistic	Most common source of dosing errors Prolonged recovery and increased duration of stay Resource-intensive monitoring at higher doses
Pharmacologic	Opioid-induced hyperalgesia and tolerance that limit effectiveness Risk for abuse and dependence that affects both patients and providers

TABLE 47-6 Opioid Titration Dosing: Fentanyl and Morphine

Route	Initial Dose for Average Adult (Per-Kilogram Dosing; Maximum)	Subsequent Titration Dose
Fentanyl		
Intravenous	50-100 mcg (1-3 mcg/kg; max 100 mcg)	25 mcg
Intranasal	180 mcg (1.5 mcg/kg)	60 mcg
Transmucosal	OTFC, 800 mcg (10-15 mcg/kg)	
Morphine		
Intravenous	5-10 mg (100 mcg/kg; max 10 mg)	5 mg
Intramuscular	10-20 mg (200 mcg/kg; max 10 mg)	10 mg
Intraosseous	5-10 mg (100 mcg/kg; max 10 mg)	5 mg

OTFC, Oral transmucosal fentanyl citrate.

reassessment and adjusted redosing.[2,5,17] Synthetic opioids, such as IV fentanyl, can be titrated every 5 minutes, whereas IM and oral (PO) routes are, as expected, more difficult to titrate.[21,63] Importantly, in states of hemodynamic shock, absorption from the IM compartment may be inconsistent and unpredictable because of peripheral vasoconstriction, leading to both acute underdosing and delayed overdosing when peripheral blood flow is restored during resuscitation and IM narcotic drug is recruited to the central circulation.[52,91]

MORPHINE

Although morphine has long been considered the "gold standard" analgesic, it is now believed that the synthetic opioids such as fentanyl are better suited for acute pain control in austere settings. The main disadvantages of morphine are its potent stimulation of histamine release and associated vasodilation, decreased systemic vascular resistance, and decreased arterial pressure. Additionally, absorption of morphine from the plasma across the blood-brain barrier into the CNS is relatively slow compared to that of the synthetic or semisynthetic opioids, potentially resulting in delayed sedation and respiratory depression (Figure 47-5). The analgesic potency of a 10-mg IM dose of morphine sulfate has been shown to be less than that of a typical NSAID PO dose, such as 400 mg of ibuprofen, and is no longer recommended as a first-line opioid in the wilderness setting.[60]

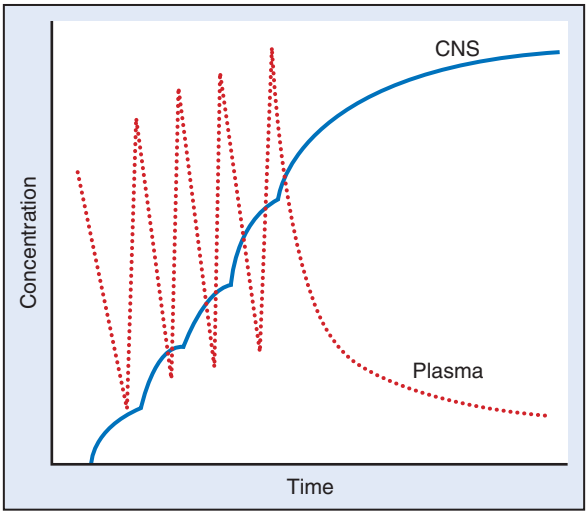


FIGURE 47-5 Morphine pharmacokinetics. Morphine has delayed entry into the central nervous system (CNS) from the plasma after intravenous (IV) injection, and delayed sedation and respiratory depression can occur up to 60 minutes after the last dose.

FENTANYL

Recently, synthetic opioids such as fentanyl have emerged as the most predictable, easily titratable, and safest option for opioid analgesia in austere settings. Fentanyl can be delivered as an oral lozenge with combined mucosal and gastric absorption, as an atomized intranasal spray, and as an IV push.[52,109] Fentanyl citrate is a synthetic opioid with strong analgesic properties—80 to 100 times more potent than morphine—and without the side effect of histamine-related hypotension.[14,25,53,68,86,91,101] Fentanyl quickly crosses the blood-brain barrier, producing rapid onset (<30 seconds after IV push) of pain relief that can be rapidly titrated. Given its potency and rapid CNS absorption, respiratory depression and apnea can occur with large IV boluses (>2 mcg/kg IV). Vomiting and nausea are common side effects. Opioid-induced chest wall rigidity is a rare complication associated with synthetic opioids such as fentanyl at high doses (>10 mcg/kg). It is treated with naloxone reversal and paralysis with a nondepolarizing neuromuscular blocking agent.[52]

Transmucosal and Intranasal Fentanyl

Oral transmucosal fentanyl citrate (OTFC) and intranasal fentanyl provide alternative opioid analgesics for severe pain that does not rely on venous access.[84,109] Both routes provide rapid delivery of drug to the central circulation, allowing rapid treatment comparable with IV morphine without the difficulty of establishing venous access.[84] OTFC has many advantages as a first-line opioid in the wilderness setting (Figure 47-6). It is a rapid-onset analgesic prepared as a lozenge on a stick. One-quarter the oral dose is rapidly absorbed directly through the oral mucosa, resulting in onset of analgesia within 5 to 10 minutes. Prolonged analgesia results from absorption of the remaining drug through the GI tract. Between the mucosal and GI tract absorption, approximately 50% of the administered dose reaches the systemic circulation. After OTFC is swallowed, it undergoes substantial first-pass metabolism in the liver and intestinal mucosa, with only one-third the swallowed drug actually reaching the systemic circulation. Dosage strengths range from 200 to 1600 mcg. A typical starting transmucosal dose for a hemodynamically stable 70-kg (154-lb) adult is 800 mcg repeated every 15 minutes as needed[52,109] (see Table 47-6).

Intranasal fentanyl (INF) via a mucosal atomizer device is a simple, practical, and effective modality for administration in settings where IV access is difficult or not possible. Approximately 70% of INF is absorbed into the systemic circulation. Initial

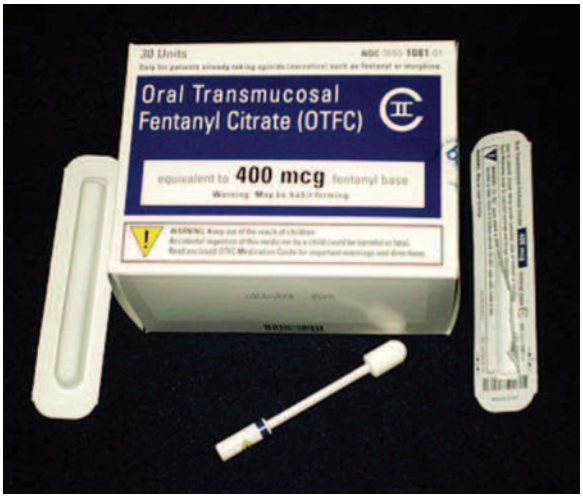


FIGURE 47-6 Oral transmucosal fentanyl citrate (OFTC). *(Open-source image from Wikipedia.)*

dosing is 1.5 mcg/kg, or approximately 100 mcg for a typical adult. Highly concentrated formulations of INF, such as 300 mcg/mL, are ideal. When a highly concentrated formulation is not available, using both nostrils will help maximize mucosal absorption.[53,76,84] Intranasal sufentanil (0.5 mcg/kg) likely is similarly effective.[93,95]

KETAMINE

In one form or another, COX inhibitors and opioids have been the primary analgesics in medicine for centuries, and in the case of opioids, millennia. Ketamine was only first described in 1965, with much of the early work focused on its role in sedation and general anesthesia at high doses.[67,78] More recently, ketamine at low doses of 0.1 to 0.3 mg/kg IV has gained widespread acceptance as a primary analgesic or adjuvant to opioid analgesia for acute pain.[1,89,100] In particular, ketamine has emerged as a first-line agent for severely injured patients at risk of hemorrhagic shock or respiratory depression in emergency and austere settings.[1,47,49,70,79] The popularity of ketamine in prehospital and wilderness medicine stems from its ability to provide consistent analgesia while not only maintaining respiratory drive and reflexes, but also preserving cardiovascular stability in all but the most critically ill patients.[58,67] Importantly, recent evidence suggests that ketamine does not increase intraocular or intracranial pressure in a clinically significant manner. Its cardiovascular and respiratory stability make it an ideal agent for situations such as intubation for head-injured patients[29] (Table 47-7).

Unique Considerations

Ketamine is a pharmacologically complex drug with an analgesic pathway totally unique from opioids or NSAIDs.[78] As such, pain scores do not adequately provide a measure for comparing ketamine analgesia with that of opioids. Importantly, ketamine subjectively provides a local anesthetic quality to the analgesia, making it ideal for severely painful injuries or procedures (e.g., burn injury, fracture reduction). The effects of a ketamine bolus come on quickly (<60 seconds) and are relatively brief (peak within 10 minutes); anesthesia can be prolonged with an infusion.[1]

As with opioids, ketamine analgesia is coupled with sedation. However, the sedation, even at a very low dose, has a dissociative quality tending toward hallucinations and complex emotional states. If unexpected, this effect can be unsettling and even frightening. Conversely, with clear coaching beforehand, patient satisfaction is high and unpleasant experiences unusual.[1] Recent experience suggests that the psychomimetic effects of ketamine noted at lower doses are rarely bothersome, and true agitation is uncommon. Indeed, ketamine is an excellent intervention for the agitated and confused trauma patient because it induces rapid onset of sedation and analgesia with preservation of cardiopulmonary status. After the acute effects have worn off, an elevated mood may be clinically noticeable; this potential antidepressant effect is currently the subject of intense study.[71]

Pharmacology

The unique qualities of ketamine analgesia are thought to result primarily result from *N*-methyl-D-aspartate receptor antagonism, through which ketamine provides potent analgesia without the respiratory and cardiovascular depression associated with opioids. The clinical analgesic potency of ketamine is clearly strongest in opioid-tolerant patients and patients with chronic pain associated with central sensitization. This has led some authors to conjecture that ketamine is primarily an "antihyperalgesic" versus an analgesic per se.[43] However, recent studies of acute pain suggest an independent, potent, and primary analgesic effect.[78]

Ketamine is highly lipid soluble with rapid onset of analgesia within 1 minute when given IV[47] or 5 minutes when given IM or intranasally.[49] Ketamine is a mild sympathomimetic that directly stimulates the brainstem, promoting catecholamine release as well as inhibition of norepinephrine reuptake; this typically results in a moderate increase in heart rate, stroke volume, and blood pressure.[29,58] Additionally, ketamine is a potent bronchodilator and does not affect respiratory drive or airway reflexes, making it ideal for patients in respiratory distress.

In summary, ketamine's potent analgesia, synergistic potentiation of opioids, and wide safety margin make it an ideal agent for wilderness pain control.

TABLE 47-7 Subdissociative Dosing for Ketamine Analgesia

Route	Typical Starting Dose for 70-kg (154-lb) Adult*	Range
Intravenous	10-20 mg	0.1-1 mg/kg
Intraosseous	10-20 mg	0.1-1 mg/kg
Intramuscular	30-50 mg	0.25-5 mg/kg
Intranasal	30-50 mg	0.25-4 mg/kg

*Dose should be titrated to clinical effect. The dose should be lowered by one-half in hypotensive critically ill patients.

MUSCLE RELAXANTS

Muscle relaxants may help relieve acute muscle spasms related to injury. Baclofen is a relatively potent muscle relaxer with γ-aminobutyric acid B receptor activity. However, sedation is a common side effect that may limit its usefulness. Carisoprodol (Soma), metaxalone (Skelaxin), and cyclobenzaprine (Flexeril) are generally less effective alternatives.[111]

PHARMACOLOGIC TREATMENT OF SPECIFIC PATIENT POPULATIONS IN THE WILDERNESS

STABLE PATIENTS WITH MILD TO MODERATE PAIN ABLE TO TAKE MEDICINES BY MOUTH

The base of any multimodal intervention is acetaminophen (usually 1000 mg) in combination with a NSAID (e.g., ibuprofen, 400 to 800 mg).[6,20,60,86] Use of acetaminophen combined with a NSAID reduces the possibility of respiratory depression, oversedation, and reduced functional capacity seen with field use of opioids.[14] Acetaminophen and NSAIDs are easy to transport and have a long shelf life and low abuse potential, making them ideal for wilderness travel.[27] Pain will be successfully treated in many circumstances with simultaneous administration of acetaminophen and a NSAID without need for escalation to an opioid or ketamine.[86]

STABLE PATIENT WITH SEVERE PAIN

Escalation of analgesia for a patient with severe or intolerable pain not sufficiently managed by initial nonpharmacologic intervention and administration of a combination of acetaminophen and a NSAID can be achieved with the addition of an opioid, ketamine, or a combination of both.[14,25,47,78,79,86] If opioids are to be used, preparation for respiratory depression is mandatory by having naloxone readily available. Additionally, the psychomotor effects of opioids are idiosyncratic and difficult to predict. Some patients may experience significant impairment that could endanger them in the wilderness setting if proper precautions are not taken. Naloxone is available in easy-to-use preparations for intranasal (1 to 2 mg/dose), IM (1 to 2 mg/dose), or IV (0.4 to 2 mg/dose) use. Ondansetron, 4- to 8-mg dissolving tablet, and diphenhydramine, 25- to 50-mg tablet, are useful antiemetic agents.

As discussed earlier, OFTC is an ideal opioid for austere settings and frequently used by combat medics for analgesic treatment of battlefield injuries.[52,109] One strategy in situations where close observation is not possible is to tape the lozenge to the patient's finger so that if excessive sedation occurs, the finger falls from the mouth and dosing ceases.[14] Fentanyl can also be given intranasally with similar efficacy to IV morphine.[84] If IV access is established, fentanyl (25 mcg IV every 10 minutes) or

morphine (5 mg IV every 10 minutes) can also be used.[14,20,86] IM morphine has been found to have unpredictable onset and efficacy; transmucosal or intranasal routes are now considered superior, with IV administration remaining the gold standard.[20,45,60,88] With all opioids, observation is required to titrate properly to the desired level of analgesia and sedation, because individuals can have greatly different responses that are not easily predicted.[17,60,63]

POTENTIALLY UNSTABLE PATIENT WITH SEVERE PAIN

For the severely injured, potentially unstable patient at risk of hemorrhagic or cardiopulmonary demise, ketamine, 50 mg intranasally via mucosal atomizer, 50 mg IM, or 20 mg IV, is the agent of choice, to be repeated every 20 minutes (IV) or every 30 minutes (IM), using analgesia or nystagmus as end points.[1,14] Ketamine does not need to be withheld in patients with eye injuries or traumatic brain injury because the risk for any additional injury is low, and again, its effect on intraocular and intracranial pressure is likely not clinically significant.[67] In the critically ill patient near death from hemorrhagic shock, the negative inotropic properties of ketamine can unexpectedly predominate because of a hypothesized catecholamine depletion, resulting in worsening cardiovascular shock.[58,99] To avoid this rare but well described occurrence, using one-half doses (e.g., 10 mg slow IV push titrated every 10 minutes) should be considered.

INTRODUCTION TO REGIONAL ANESTHESIA FOR WILDERNESS PAIN MANAGEMENT

Regional techniques provide a safe and effective approach to managing pain in the wilderness.[16,61,86,96,108] In contrast to systemic therapy, where a drug is taken into general circulation to exert both peripheral and central effects throughout the body, regional anesthesia anatomically positions a nerve blocking agent, such as a local anesthetic, at a specific site (Table 47-8). Recent development of small, portable ultrasound machines is allowing wilderness medicine providers to use ultrasound in the out-of-hospital setting to target nerves with real-time guidance, greatly increasing the accuracy, efficacy, and safety of regional anesthesia in austere settings (Figure 47-7).

The goal of regional anesthesia is to inject a local anesthetic into a joint space, around wounded tissue, or perineurally to prevent or diminish transmission of the pain signal from peripheral afferent nerves to the CNS. By blocking the nociceptive barrage to the CNS, the potentially harmful systemic stress reaction is reduced, and the need for systemic analgesics is eliminated or greatly reduced.[21,32] With successful regional anesthesia, patients can be kept mentally alert, neither distracted by pain nor dulled by systemic medications. This anatomically targeted approach to pain treatment avoids or lessens the inevitable complications of systemic therapies. The depth of pain control with a block generally is far beyond what can be achieved with systemic medications in a conscious patient, allowing potentially very painful procedures to be completed in relative comfort. For example, a patient with significant lower-extremity fracture can have the broken bone manipulated and splinted and can be transported after a regional block without the need for sedation or large doses of systemic opioids or ketamine (Box 47-2).

For these reasons, regional anesthesia is becoming an essential skill for wilderness pain management. Although nerve blocks can be technically complex, with adequate training and ongoing practice, paramedics, nurses, and physicians can successfully and safely perform them.[32,69,90,96] Nerve blocks are discussed later in this chapter.

FIGURE 47-7 Regional anesthesia can be transformative in austere settings. Pain in austere settings, where IV opioids are often scarce, and respiratory depression, sedation, and delirium associated with opioids are particularly difficult to manage, often leads to undertreatment of pain. Introduction of regional anesthesia for limb trauma and procedural pain can bring greatly improved pain management to these settings. *(Courtesy Margaret Salmon.)*

TABLE 47-8 Types of Emergency Regional Anesthesia

Regional Anesthesia Technique	Comments
Tissue infiltration	Simple and easy; however, infiltration is painful, and field of anesthesia is limited.
Field blocks	Can be very effective in select anatomic locations, such as the superficial cervical plexus and the antebrachial cutaneous nerves of the arm.
Intraarticular injection	Analgesia is limited to joint surface in contact with injected local anesthetic; relatively short-lived.
Fracture hematoma injection	Can be rapidly placed once fracture is identified; limited duration and scope of analgesia.
Regional nerve block	"Gold standard" analgesia for extremity trauma.
Neuraxial and intravenous (IV) techniques	Wilderness applications are currently limited; higher risk profile and unclear benefit compared with regional nerve blocks.

AURICULAR ACUPUNCTURE IN AUSTERE ENVIRONMENTS

Acute pain in austere environments may range from that caused by minor trauma (e.g., sprains, myofascial disturbances) to that associated with more severe trauma (e.g., fractures, burns). In each condition, optimization of acute pain through nonpharmacologic techniques is a reasonable approach in order to avoid

BOX 47-2 Benefits of Regional Anesthesia

- Targets profound analgesia to a specific anatomic area and nowhere else
- Reduces need for opioids and related side effects
- Reduces need for sedative medications and related side effects
- In hypovolemic patients, may reduce incidence of hypotension compared to traditional sedation and analgesia
- May reduce the stress response to injury and contribute to improved recovery
- Improved quality of pain control and greater patient comfort
- Awake and alert patients with well-controlled pain have decreased need for close medical supervision.

side effects as well as the depletion of limited medications. Use of auricular acupuncture can be a helpful adjunct.

The history of auricular acupuncture begins in ancient China. Modern use stems from the work of Paul Nogier, who characterized specific points on the ear that represent specific parts of the body ("somatotopic map").[22,55] This characterization mirrors the concept of the sensory "homunculus" within the somatosensory cortex, whereby the ear is a "microsystem" that represents the entire body.[77] Although some discrepancies exist between various mapping sources (Chinese vs. European vs. French), Nogier's original mapping scheme has been the source of numerous auricular acupuncture protocols. Such protocols often involve treating the part of the ear that corresponds to the source of pain, mixed with various "master points" that are theorized to affect various functions, such as autonomic signaling, or that also possess sedating properties.

MECHANISM

The predominant body of literature regarding whole-body acupuncture suggests a mechanism based on transmission of signaling to the spinal cord, resulting in local release of endogenous opioid-like substances, in addition to enhancing signal inhibition by promoting release of antinociceptive mediators (serotonin, norepinephrine, and dopamine).[107] Furthermore, central signaling may involve activation of transmission through the notable "pain matrix" within the cerebral cortex, as seen on functional magnetic resonance imaging (fMRI).[46] However, mechanisms of auricular acupuncture are less clear. With its innervation from the greater auricular nerve, lesser occipital nerve, auriculotemporal nerve, and auricular branch of the vagus nerve, some hypothesize that activation of parasympathetic centers may be related to its analgesic effect.[40]

BATTLEFIELD AURICULAR ACUPUNCTURE

Developed by Col (ret) Richard Niemtzow, battlefield auricular acupuncture (BFA) is a specific type of auricular acupuncture that utilizes five ear points in sequential order[75] (Figure 47-8). The U.S. Department of Defense has invested millions of dollar to train and disseminate the BFA protocol among medics and lower-level providers in forward-deployed settings. The aim of this program is to equip these first responders with a nonopioid pain option that has the potential to diminish the amount of narcotic administration while simultaneously promoting a multimodal analgesic practice. Of note, in one military complementary and alternative medicine wellness clinic, ear acupuncture was performed on 58.1% of 2756 clinic visits.[24]

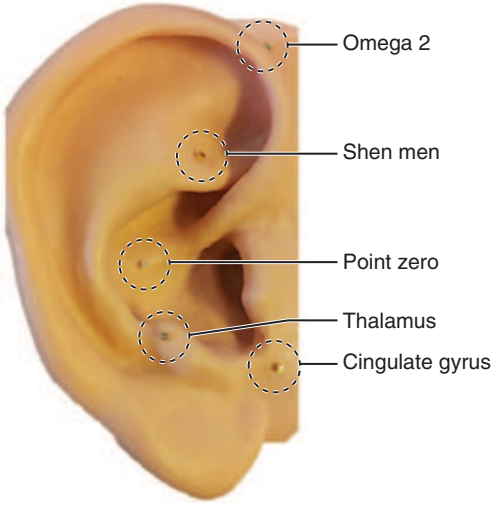


FIGURE 47-8 Five points for Battlefield Auricular Acupuncture (BFA). The U.S. military now routinely uses auricular acupuncture for acute pain management in the field setting. The simple technique does not require advanced training and involves self-injecting needles inserted and left in place at five easily identified needling points on the ear. *(Copyright Richard Niemtzow, 2013.)*

TABLE 47-9 Wilderness Topical Anesthesia and Analgesia*

Procedural Anesthetics	Comments
Intact Skin	
Eutectic 1:1 mixture of 2.5% lidocaine and 2.5% prilocaine (EMLA)	Onset of action is 60 minutes; occlusive dressing is required; methemoglobinemia is a rare complication of prilocaine.
4% Lidocaine cream in a liposomal matrix (LMX)	Onset of action is 30 minutes, and no occlusive dressing is required.
Vapocoolant spray	Ethyl chloride is the most commonly used agent. Spray until skin blanches for up to 10 seconds 7.5 to 22.5 cm (3 to 9 inches) from skin surface; duration of action is approximately 1 minute after blanching.
Nonintact Skin	
Lidocaine 4%, epinephrine 0.1%, and tetracaine 0.5% (LET)	Onset of action is 30 minutes after direct application to open wound.
Analgesia	
Capsaicin ointment 8% Lidocaine ointment, 5% Lidocaine patches, 5% Diclofenac, 1% gel Diclofenac, 1.3% patch	For wilderness travelers with chronic musculoskeletal pain such as from osteoarthritis or neuropathy, topicals may help ease trail soreness and flare-up pain.
Corneal Analgesia	
Proparacaine hydrochloride 0.5% ophthalmic drops, 10-mL vial	

*Agents containing cocaine and benzocaine are less commonly used because of concerns about systemic toxicity (cocaine) and methemoglobinemia (benzocaine).

The performance of BFA involves sequential placement of five needles located at various auricular acupuncture points, to include cingulate gyrus, thalamus point, omega 2, point zero, and shen men. Usually, this technique is performed bilaterally but can also be performed on the side of the injury if supplies or time are limited. Auricular semipermanent (ASP) needles are often used and can remain in the ear for 3 to 4 days, after which they will dislodge automatically. For patients that cannot tolerate ASP needles, smaller needles can be placed and left in place for 20 to 30 minutes and then removed.[75]

Evidence for the specific use of BFA is limited, but it represents a reasonable auricular protocol based on foundational principles of auricular acupuncture. With the benefits of ease of training, speed of application, extreme unlikelihood of adverse effects, and low equipment burden, BFA is a definite option for acute pain management in austere environments.

TOPICAL THERAPIES

Flare-up of chronic musculoskeletal pain on the trail is common and well suited to topical medications, including NSAID gels and patches, lidocaine ointments and patches, and capsaicin creams and lotions (Table 47-9). Additionally, topicals can be a good option for bothersome pain from minor strain or contusion. Topical analgesic medication should only be used on intact skin.[3]

The most commonly available topical NSAID in the United States is diclofenac, either as a 1% gel or a 1.3% patch. Outside the United States, other topical NSAIDs may be available. Topical 1% diclofenac gel is applied four times daily over the site of pain and is indicated for relief of osteoarthritis pain in joints amenable

to topical therapy. The diclofenac patch (Flector Patch) is applied twice daily to the site of pain and is indicated for acute pain that results from minor strains, sprains, or contusions. Topical lidocaine is available as a 5% ointment, 4% cream, and 5% patch. Ointment and cream are applied up to three times daily. Up to three patches at a time may be applied, and the patches are to remain on the skin for no more than 12 hours during any 24-hour period. Lidocaine patches are indicated for postherpetic neuralgia; however, they may be effective for any localized pain condition.

Capsaicin cream is available in preparations that range from 0.025% to 8%. Capsaicin analgesia results from activation of the transient receptor potential vanilloid subfamily member 1 (TRPV1) in cutaneous afferent nociceptive terminals. Brief activation (experienced as a burning sensation) is followed by a prolonged refractory period or "defunctionalization" of the nociceptive neuron, rendering it unresponsive to noxious stimuli and preventing pain signaling.[3]

Begin with the least concentrated formulation of capsaicin, and apply it with lidocaine 5% ointment three or four times daily. Initially, use three parts of lidocaine ointment to one part of capsaicin. As the application becomes more tolerable, increase to a one-to-one concentration; this will eventually be followed by full-strength capsaicin.[3]

ANALGESIA FOR CORNEAL ABRASIONS

Corneal abrasions are common and very painful in the first 24 hours after injury. In the wilderness setting, this may lead to poor sleep in addition to functional impairment if negotiation of challenging terrain is required. Topical application of local anesthetic, such as 0.5% proparacaine, brings near-complete relief within moments after application. For more extended pain relief, a diluted mixture can be prepared by premixing 9 mL of normal saline with 1 mL of 0.5% proparacaine. Diluted proparacaine is then used as needed for comfort.

There are valid concerns for impairment of corneal epithelial healing, as well as the loss of protective corneal sensation to touch. Prolonged use of proparacaine beyond 24 to 48 hours should be avoided, and patients should be cautious to protect the eye from additional injury when the corneal epithelium is anesthetized.[106]

Standard care also includes antibiotic eye drops, typically a fourth-generation fluoroquinolone plus polymyxin B and trimethoprim (Polytrim) to add gram-positive coverage. A bandage soft-contact lens, cycloplegic agent (e.g., homatropine) to counter ciliary spasm, and topical NSAID (e.g., diclofenac 0.1%) may also be used. An eye patch is no longer recommended for corneal abrasion.[110]

PRETRAVEL PREPARATION

Optimally, a pretravel evaluation should assess the individual's suitability for the wilderness experience and should include determination of chronic medical conditions. A thorough history and physical examination should include the individual's use of medications as well as any allergies and intolerances. Determining the use of chronic pain medications is important, because there are common and potential side effects to these medications. Some medications may cause cognitive impairment and psychomotor changes that may increase the individual's risk for injury. Use of opioids may be associated with side effects, including cognitive changes, constipation, and tolerance, as well as withdrawal if the medication dose is significantly reduced or terminated abruptly. Termination of adjuvant medications, including antidepressants (e.g., desipramine, duloxetine), benzodiazepines (e.g., lorazepam, alprazolam), and anticonvulsants (e.g., carbamazepine, gabapentin), can also cause withdrawal-type syndromes.

Individuals who are receiving chronic opioids or adjuvants for chronic pain may not have a predictable response to promptly administered analgesic medications for acute injuries. Because management of chronic pain has become more complex, and as more sophisticated implanted devices have become available, it is more likely that individuals with chronic pain will be traveling in the wilderness. These devices include spinal cord stimulators, peripheral nerve stimulators, cortical and deep brain stimulators, and implanted intrathecal infusion devices. If an individual has one of these devices, it will be important for the person to understand how to operate the device and to have contingency plans in case the device fails. For example, individuals with implanted pumps that deliver intrathecal medication require periodic refills of the reservoir, typically every 1 to 3 months.

Persons who use these devices and persons with allergies should wear identification bracelets. Device manufacturers have a worldwide network of support that is accessible by telephone or e-mail, but this may not be practical in the wilderness, so written instructions should be available. Having the device information (name of manufacturer, model number) and contact information for local health care providers and manufacturers will facilitate resolving difficulties when civilization is reached or if remote communication is possible.

When medications are transported internationally and specifically by air carrier, the Travel Security Administration in the United States may require specific handling of medications. Because regulations change periodically, one should check for updates before traveling. Medications should be properly labeled with appropriate prescription and patient identification information.

Box 47-3 lists the contents of a pain management first-aid kit.

BOX 47-3 Pain Management First-Aid Kit

Basics
Materials for splinting, protection, and compression wrapping of an injured extremity

Oral Medications
NSAIDs: acetaminophen, 500-mg tablets; ibuprofen, 200-mg tablets
Opioid: hydrocodone, 5-mg tablets, or other oral opioid
Antiemetic: ondansetron, 8-mg sublingual tablets; diphenhydramine, 25 mg
Anxiolytic: lorazepam, 1-mg tablets, or other benzodiazepine

Injectable Medications
Opioid: fentanyl, 0.05 mg/mL, 5-mL vial; morphine, 5 mg/mL, 5-mL vial
Opioid reversal: naloxone, two 0.4-mg ampules
Ketamine, 50 mg/mL, 10-mL vial
Benzodiazepine: lorazepam, 2 mg/mL, 4-mL vial

Topical Therapies
Capsaicin ointment, 0.1%, 2-g tube
Lidocaine ointment, 5%, 50 g or lidocaine patch, 5%
Diclofenac, 1% gel, 100 g, or 1.3% patch (Flector Patch)
Proparacaine hydrochloride 0.5% ophthalmic solution, 10-mL vial

Regional Anesthetics and Equipment
Extension tubing set and syringes, 10 mL, 20 mL, 30 mL
Block needles: 22-gauge blunt tip, 50 mm; 18- to 22-gauge Tuohy tip, 80 mm
1% and 2% Lidocaine with epinephrine, 20-mL vials
3% 2-Chloroprocaine, 30-mL vials
1% Ropivacaine, 30-mL vials
0.9% Normal saline, 10-mL vials

Acupuncture Materials
Auricular semipermanent (ASP) needles, stainless steel; box of 80 needles with injectors

PRACTICAL GUIDE TO REGIONAL ANESTHESIA FOR EMERGENCY AND WILDERNESS PAIN MANAGEMENT

BENEFITS OF REGIONAL ANESTHESIA

Typically, regional anesthesia involves either injecting a local anesthetic around a target nerve (nerve block) or injecting the

local anesthetic so that it comes into direct contact with the injured tissue (intraarticular injection, wound infiltration, and hematoma block). Regional anesthesia can be administered either using landmark-based anatomic techniques or with ultrasound guidance[21] (see Table 47-8).

Integration of nerve blocks placed as close to the time of injury as logistically feasible is one of the most important recent advances in emergency and austere environment pain management.[6,14,86,96] Widespread adoption of early regional anesthesia has proved revolutionary in the contemporary military trauma setting.[13] Nerve blocks have multiple important advantages. First, they reduce or eliminate the need for opioids. Moreover, by halting the nociceptive barrage from an acute injury, nerve blocks may help reduce chronic pain after injury by reducing pain-induced CNS sensitization, or "wind-up."[32]

Traumatic injury results in a catabolic stress response that is directly proportional to the degree of tissue injury and may be attenuated by regional anesthesia that blocks peripheral afferent noxious stimuli from reaching the CNS.[19,62,112] The barrage of noxious stimuli to the CNS after traumatic injury triggers a cascade of metabolic, neurohormonal, and inflammatory events leading to detrimental physiologic changes such as hyperglycemia, hypercoagulability, and immunosuppression. Normally, the stress response is an adaptive "fight or flight" mechanism to promote survival, yet in the medical context, it may be associated with delayed recovery, delayed healing, and increased morbidity.[21,32,39]

PHARMACOLOGY OF LOCAL ANESTHETICS

Local anesthetics produce clinical nerve blockade through reversible inhibition of sodium ion (Na^+) channels at the neuronal cell membrane. Apart from local anesthetics, perineural infiltration of other agents, such as opioids, ketamine, corticosteroids, and clonidine, can cause some degree of nerve conduction blockade. However, these agents are rarely used except as adjuncts to local anesthetics. Typically, local anesthetics are classified as either an amino ester or an amino amide. Familiar amino ester local anesthetics are procaine, chloroprocaine, and tetracaine. Amino amides include lidocaine, prilocaine, mepivacaine, bupivacaine, and ropivacaine. Local anesthetics vary substantially in their safety profile, potency, and duration of action. Analgesia can be tailored to the scenario by selecting the appropriate local anesthetic and adjusting the concentration and volume to achieve the desired block profile[21,28,74] (Table 47-10).

Amide ester local anesthetics undergo hydrolysis; this results in production of paraaminobenzoic acid, which is more likely to precipitate an allergic reaction than are amino amides, which undergo hepatic metabolism. A local anesthetic may contain preservatives that can cause rare allergic reactions. An allergic reaction to a local anesthetic of one class does not increase the risk of an allergic reaction to the other class of local anesthetic, unless the reaction is to the preservative.[21]

LOCAL ANESTHETIC TOXICITY

Systemic toxicity with local anesthetics is related to peak plasma concentration of the drug. Local anesthetic may enter the systemic circulation by direct injection at the time the block is placed or as a result of ongoing systemic absorption of drug from tissues after placement of the block. The most common scenarios of local anesthetic toxicity (LAST) are (1) inadvertent injection of a local anesthetic directly into an artery or vein during placement of a nerve block and (2) systemic absorption after a large volume of local anesthetic has been injected diffusely into tissue, such as with multiple large wounds. Absorption of local anesthetic depends on the dose administered, vascularity of the site of injection, use of added epinephrine, and chemical properties of the anesthetic.[28]

Systemic toxicity with local anesthetics may affect both the CNS and the cardiovascular system. The typical pattern of LAST occurs as plasma levels of local anesthetics increase. Patients may experience perioral and tongue numbness, followed by vertigo, tinnitus, and blurred vision, which then progresses to seizures. If plasma levels continue to rise, cardiovascular toxicity results from relaxation of vascular smooth muscle and direct myocardial depression. Effects on vascular smooth muscle may cause hypotension. Cardiac depression is thought to be related to sodium channel blockade. Slowed conduction can result in prolonged PR and QRS intervals and reentry ventricular dysrhythmias.[72]

Prevention and Management

Managing LAST in the wilderness setting is a challenge best avoided. To avoid LAST during wound infiltration, it is best to use a local anesthetic with a low toxicity profile, such as lidocaine with epinephrine; the maximum nontoxic dosage of lidocaine with epinephrine is 7 mg/kg, up to a total 500 mg or 50 mL of the 1% solution. Addition of epinephrine limits systemic absorption. Lidocaine toxicity typically involves seizures that are easily extinguished with a benzodiazepine. At the 1% concentration, lidocaine is effective for wound infiltration, fracture periosteal infiltration, and intraarticular injection. It is relatively weak as a nerve blocker when used for anything but smaller peripheral nerves. When used for a major, proximal nerve block, such as a femoral nerve block or brachial plexus block, a 2% concentration of lidocaine is generally preferred.

For short procedural anesthesia, such as a shoulder or ankle dislocation reduction, 3% 2-chloroprocaine is an excellent option. This is an ester local anesthetic that is very unlikely to result in LAST because it is rapidly metabolized in plasma by hydrolysis of the ester linkage by pseudocholinesterase, resulting in a half-life in the bloodstream of less than 30 seconds. The low toxicity allows use of concentration that promotes rapid onset of a short block (60 to 90 minutes). 2-Chloroprocaine is typically not effective for infiltration anesthesia.[54]

For a proximal block when a long duration of anesthesia and analgesia is desired, such as a nerve femoral block, a long-acting

TABLE 47-10 Local Anesthetics for Peripheral Nerve Blocks

Indication	Agent	Duration	Toxicity potential	Comments
Minor procedure	2-Chloroprocaine 3%	Very fast onset with 60-90 minutes of anesthesia	Minimal	Very little toxic potential
	Lidocaine 2%	Fast onset with 2-3 hours of anesthesia	Moderate	Seizures are possible with systemic toxicity.
Major procedure	Mepivacaine 1.5%	Fast onset with 2-3 hours of surgical anesthesia	Moderate	Favored by many because it produces a dense block with prolonged analgesia.
	Lidocaine 2%	Similar to Mepivicaine	Moderate	
Prolonged analgesia	Ropivacaine 0.5%	Slow onset, dense block, with 4-6 hours of anesthesia, potentially much longer	Severe	Developed as potentially less cardiotoxic alternative to bupivacaine.
	Bupivacaine 0.25%	Similar to ropivacaine	Severe	Very potent and long-acting Intravascular injection can precipitate cardiovascular collapse.

local anesthetic is preferred. All long-acting local anesthetics can cause catastrophic cardiovascular toxicity. Because some detoxification may occur as venous blood transits the lungs, the greatest risk for significant cardiovascular events is likely to occur with a proximal arterial injection, such as injection into a vertebral artery during a brachial plexus block. The most common long-acting local anesthetic is bupivacaine. Ropivacaine (0.5%) is a safer option with clinically similar block characteristics, so it should be preferentially used whenever possible.[19,32,54]

Monitoring is not practical in most austere settings, placing more burden on the provider to use meticulous technique when placing a nerve block. Specifically, this entails (1) use of larger-bore, blunt-tipped needles less likely to puncture a vessel inadvertently, (2) aspiration before any injection, to check for blood indicating inadvertent intravascular needle placement, (3) slow injection of small (3-mL) aliquots of anesthetic, (4) use of ultrasound guidance or nerve stimulation whenever possible, and (5) use of the lowest volume and concentration of anesthetic needed to produce a sufficient block. Extra care and dose reduction should be used with older patients and those with significant renal impairment.

Treatment

If a LAST reaction is suspected, lipid 20% emulsion (intralipid) should be administered IV as part of the resuscitation.[72] Although data are exclusively from laboratory animals or case reports, there is widespread endorsement of intralipid for LAST. The precise mechanism of action for intralipid resuscitation of severe LAST is poorly understood, but likely involves creating a "lipid sink" capable of extracting lipophilic molecules, such as potent local anesthetics, from free circulation (Box 47-4).

FRACTURE INFILTRATION ("HEMATOMA BLOCKS")

The so-called hematoma block is often used to provide acute analgesia for manipulation of a fractured bone. The moniker derives from the history of using the presence of aspirated blood as indicator of physical proximity to the fracture. While the concept of a fracture hematoma has value, it is important to understand that analgesia develops from the local anesthetic physically contacting the terminal afferent nerves in the fracture periosteum and surrounding injured tissue. Practically, this means that when performing periosteal infiltration for a fracture, there may be multiple hematomas that do not completely communicate. Entry into a hematoma with aspiration of blood should be considered a helpful sign, but not a conclusive guide to 360-degree infiltration of the injured periosteum. Periosteal infiltration for a fracture can be done using anatomic landmarks and palpation or with ultrasound guidance (Figure 47-9).

BOX 47-4 Treatment of Local Anesthetic Toxicity

First Response

Follow basic life support and advanced cardiac life support (BLS/ACLS) guidelines.

Suppress seizures with benzodiazepines.

Initiate lipid rescue with 20% lipid emulsion:

- Intravenous bolus, 1.5 mL/kg over 1 minute (~100 mL for 70-kg [154-lb] patient)
- Continuous infusion at 0.25 mL/kg/min (~20 mL/min for 70-kg patient)
- Repeat bolus one or two times for persistent cardiovascular collapse.
- Double the infusion rate to 0.5 mL/kg (~40 mL/min for 70-kg patient) per minute if blood pressure remains low.
- Continue infusion for at least 10 minutes after attaining circulatory stability.
- Recommended maximum dose: approximately 12 mL/kg (~840 mL for 70-kg patient) of lipid emulsion over the first 30 minutes

Cautions

Avoid propofol, vasopressin, calcium channel blockers, β-blockers, and local anesthetic.

Avoid high-dose epinephrine greater than 1 mcg/kg.

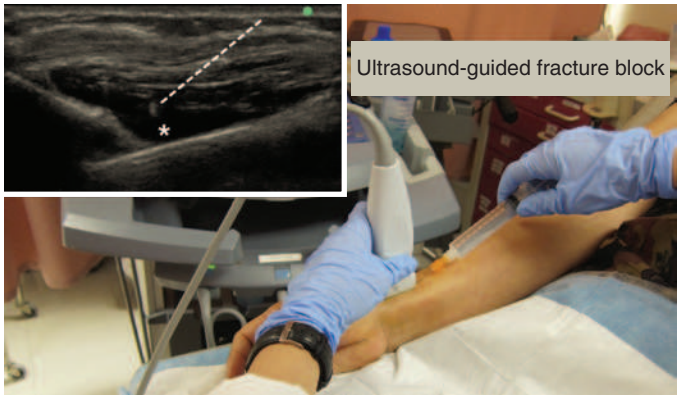


FIGURE 47-9 Ultrasound-guided fracture block for a distal radius fracture. Ultrasound can be used to identify most long-bone fractures quickly and definitively as a discontinuity in the brightly echoic cortical surface. At the fracture site, the needle (*dashed line*) is guided with ultrasound visualization to the disrupted periosteal surface, where local anesthetic is injected Any long-bone fracture can be managed with a fracture block. Care should be taken to maintain sterility and not to exceed recommended maximum local anesthetic dosing.

The advantage of periosteal infiltration in fracture pain management is that the procedure can be rapidly accomplished with minimal technical difficulty. Acute fractures are fairly easy to identify based on tissue deformity, providing an obvious target area for injection. The main disadvantage is that inconsistent analgesia occurs because of the somewhat unpredictable flow of local anesthetic after injection. Additionally, the duration of analgesia tends to be relatively brief because local anesthetic is cleared by the prominent vascular bed exposed by the fracture site. An associated risk is that rare and unpredictable local anesthetic toxicity can occur when local anesthetic is rapidly absorbed into the systemic circulation at the fracture site. Aside from rapid absorption from the vascular bed of the injured bone, large injuries with significant tissue damage often require too much local infiltration to be practical or safe. However, closed fractures of even a large long bone, such as a femoral shaft fracture, typically respond well to direct fracture periosteal infiltration. Although there is a hypothetical risk of introducing infection into a fracture site, this is likely very rare if standard antiseptic precautions are taken.[23,34]

Intraarticular Injections

Intraarticular injections are useful for acute fracture and dislocation reductions and exacerbation of chronic inflammatory disease. For an acute dislocation, such as a glenohumeral shoulder dislocation, a larger volume of local anesthetic, such as 20 mL of 2% lidocaine or 20 mL of 0.5% bupivacaine with or without epinephrine, is injected into the joint space for anesthesia and analgesia to allow reduction[48] (Figure 47-10). Elbow, knee, and ankle dislocations can be similarly managed. Hip dislocations involve such a large tissue area that it may be difficult to produce more than partial analgesia.

For acute on chronic exacerbations of chronic pain, smaller volumes of bupivacaine combined with a corticosteroid such as triamcinolone acetonide are a potentially effective intervention. An example is an intraarticular knee injection of 8 mL of 0.5% bupivacaine and 2 mL of triamcinolone (40 mg/mL) for a patient with a flare-up of osteoarthritis. It is particularly important to maintain sterility when performing such injections in austere settings to prevent infectious complications.[33]

PERIPHERAL NERVE BLOCKS

A peripheral nerve is composed of efferent and afferent axons of various types. Each axon is suspended in a delicate connective matrix termed the *endoneurium*. Bundles of axons

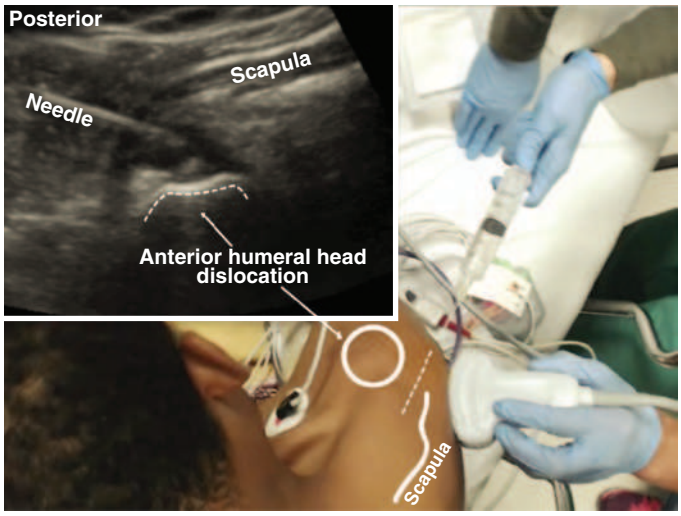


FIGURE 47-10 Ultrasound-guided intraarticular glenohumeral injection for shoulder dislocation reduction. Dislocated joints are readily anesthetized with intraarticular local anesthetic injection. For shoulder dislocation, the ultrasound-guided posterior approach is reliable and effective. The needle (*dashed line*) is advanced anterior and lateral to the scapula into the glenohumeral space, where 20 mL of lidocaine is then slowly injected.

and endoneurium are organized into fascicles by an encasing squamous epithelial sheath termed the *perineurium*. The macroscopic peripheral nerve is formed by the epineurium's connective tissue that bundles fascicles together into a distinct, cord-like structure.

Peripheral nerves typically travel within a fascial compartment or "gliding sheath" that allows musculoskeletal freedom of movement while minimizing mechanical stress on the nerve itself. A regional nerve block is accomplished by perineural injection of local anesthetic inside this sheath, which then spreads along the length of the nerve and diffuses inward from the epineurium toward the axonal membrane, where its direct action takes place (Figure 47-11).

ANATOMY FOR REGIONAL ANESTHESIA: MATCHING THE PERIPHERAL BLOCK TO THE INJURY

The most challenging aspect of administering emergency regional anesthesia is matching the correct peripheral nerve block to the clinical injury or procedure in the most site-specific and effective manner. Given the unpredictability of injuries encountered in the wilderness, a well-established knowledge of peripheral innervation patterns and a broad working armamentarium of peripheral blocks are essential.

ULTRASOUND GUIDANCE

As ultrasound technology becomes more compact and portable, ultrasound guidance is increasingly available to providers in austere, resource-limited settings. Ultrasound guidance allows direct visualization of the target nerve and relevant tissue structures, the needle, and real-time flow of local anesthetic during injection. Direct visualization assists the provider to be more accurate with needle placement and to avoid inadvertent intravascular puncture or other potentially injurious complications. As a result, use of ultrasound guidance to assist with nerve blocks is rapidly becoming the standard of care.[37,65,74]

In general, the target nerve is imaged in a short-axis, cross-sectional plane perpendicular to the long axis of the nerve as it travels from proximal to distal. The two basic techniques to visualize the needle with ultrasound are in-plane and out-of-plane. *In-plane* refers to the technique where the needle is aligned parallel to the long axis of the ultrasound transducer to obtain an image of the entire shaft and tip of the advancing needle. This is generally considered the safest and most precise approach (Figure 47-12). The *out-of-plane* technique involves inserting the needle perpendicular to the long axis of the transducer, as is common for the vascular axis. This technique is generally less precise because only the needle tip is visualized, and the operator requires interpretation of the pattern of deformation in the surrounding tissue as an additional guide to determine the needle tip location.

In both techniques, three fingers should be on the transducer, with the base of the palm and the fourth and fifth digits on the patient's skin to provide maximal stability and control. Subtle, controlled movements of pressure, alignment, rotation, and tilting are used to maximize target imaging (Figure 47-12). An additional strategy to locate the needle tip, known as *hydrodissection*, involves tiny (<1 mL) injections of local anesthetic or normal saline as the target is approached. The fluid injection is visualized knowing that a small amount of local anesthetic is unlikely to cause harm, and that local spread within the tissue helps locate the needle tip.

PERIPHERAL NERVE INJURY

Nerve injury after a regional nerve block is so rare that obtaining reliable estimates is difficult. The American Society of Regional Anesthesia and Pain Medicine (ASRA) recommends advising patients that nerve injury is estimated to occur anywhere from 1 in 4000 blocks to 1 in 200,000 blocks, depending on the block type and patient risk factors.[73] The injury itself may result from direct needle trauma of the nerve or secondary complications such as infection or hematoma formation. Most injuries are mild

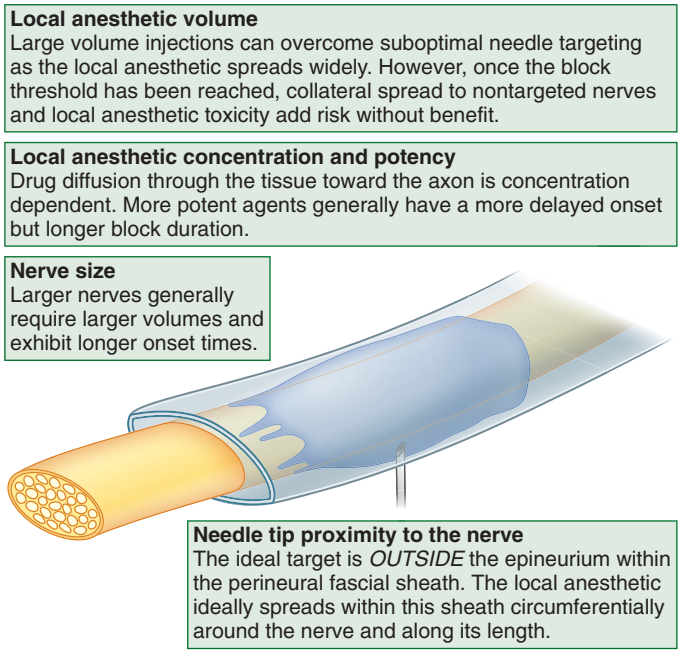


FIGURE 47-11 Nerve block fundamentals. A nerve block is the act of placing local anesthetic solution around a peripheral nerve. The target is usually not the nerve itself, but more often the fascial compartment in which the nerve travels, sometimes referred to as a "gliding sheath." The ideal is to target the fascial space at the anatomic point that results in maximal flow of local anesthetic around the nerve while minimizing the risk for needle-to-nerve contact. In general, small nerves are more easily blocked than are large nerves and require less local anesthetic. The wide variety of local anesthetics vary in potency and duration. Typically, long-acting local anesthetics have slow onset of action, and short-acting anesthetics have more rapid onset of action. Magnitude and duration of a block with any given local anesthetic depend on the total amount of drug delivered to the perineural space; a greater volume of more concentrated local anesthetic results denser and more prolonged blockade.

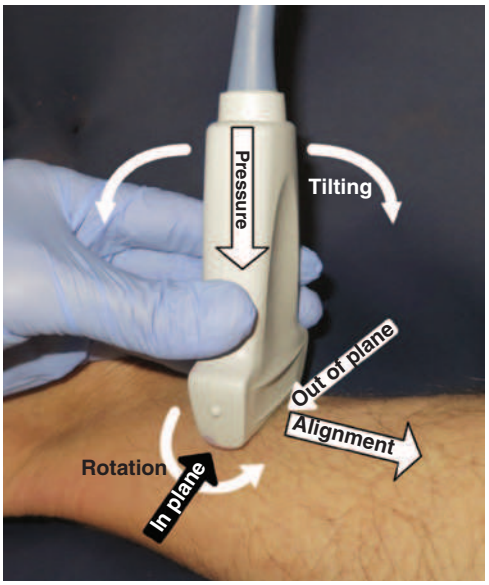


FIGURE 47-12 Ultrasound transducer handling terminology. An ultrasound-guided nerve block can be performed either in-plane, so that the entire shaft of the needle is visualized in its longitudinal axis, or out-of-plane (perpendicular to the scanning plane). Standard ultrasound probe maneuvers to improve imaging are pressure, alignment, rotation, and tilting.

or subclinical neuropathies that resolve over weeks to months. Permanent, significant injury is extremely rare. As many as 10% of patients experience paresthesias in the immediate days after a block. All patients should have neurologic follow-up until all deficits have resolved or stabilized.[32]

Prevention of Nerve Injury

Patient Selection. Preexisting neurologic disease increases the incidence of injury after a nerve block. Practically, patients with an existing nerve deficit should not receive a block under normal circumstances. Untangling the contributions of the underlying nerve deficit, the acute injury, subsequent operative interventions, and the nerve block in the case of a worsening nerve deficit is virtually impossible. Most wilderness and emergency providers would be best advised to avoid being caught up in this complex scenario.[32]

Uncontrolled delirium, intoxication, and psychiatric disease are relative contraindications to nerve block. For a block to be safely placed, the patient must be able cooperate, remain still, and then care for the anesthetized limb for the duration of the block. A large body habitus in general results in degraded imaging quality and deeper nerve targets with decreased accuracy of needle tip positioning. Although hypothetical, risk of inadvertent intraneural injection is likely increased in patients with a large body habitus.

Needle Selection. The expert consensus currently favors short-beveled needles to reduce risk of nerve injury. The evidence is weak, somewhat contradictory, and largely based on animal models.[83] Unfortunately, definitive evidence is unlikely in the foreseeable future, so there is ongoing controversy.[44,73] Proponents of short-bevel needles believe the dull bevel allows superior perception of tissue planes, thereby reducing the chance of inadvertently passing the needle tip into a nerve fascicle, where nerve injury is thought to occur. Conversely, the long-bevel needle tips traverse the anatomic planes with minimal tactile resistance.[66] Needle type may be most important for proximal approaches to the brachial plexus, where the nerve roots are thought to be particularly vulnerable to nerve contact injury. We generally favor use of dedicated, short-bevel, 22-gauge block needles or blunt Touhy needles for proximal blocks, such as the brachial plexus, and for femoral and sciatic nerve blocks. In more distal blocks, such as in the forearm, the advantage of a blunt needle is not clear.

PAINLESS, ULTRASOUND-GUIDED "STAY-AWAY" TECHNIQUE

Regional anesthesia has evolved significantly from the "paresthesia technique," where pain or paresthesia was actively sought, toward a general stance of cautious avoidance of any needle-to-nerve contact.[21] In particular, pain or paresthesia during needling should be considered an indication that the needle tip is too close to the nerve, prompting cessation of injection and redirection of the needle away from the nerve. This is obviously more challenging with landmark-based techniques.

Unlike vascular access, where the goal is to place the needle tip into the target vessel, in regional anesthesia the goal is place the needle tip in an anatomic space that results in local anesthetic being able to spread to the target nerve. This small point is crucial to promoting safety in regional anesthesia. By using anatomic knowledge and expertise in ultrasound imaging to identify the fascial plane, needle tip placement in contact with the nerve itself is unnecessary for most nerve blocks. The principle of a "stay-away" technique is to position the needle as far away from the actual nerve as possible, using the minimum number of repositionings to achieve the clinical block.[9] For example, a femoral nerve block is easily achieved with needle positioning deep to the fascia iliaca, even when it is several centimeters lateral to the nerve itself. After injection, the local anesthetic passively flows through the confined fascial space surrounding the nerve, without the need to place the needle tip directly adjacent to the nerve. Using a hand-on-needle technique with extension tubing may assist with needle control, versus using a needle directly mounted onto a syringe.[51]

PRESSURE MONITORING

The underlying principle to monitoring the pressure of fluid being injected during regional anesthesia is that if the needle tip is in direct contact with the nerve or actually intrafascicular, the opening pressure of the needle-tubing-syringe system will be elevated above 15 to 20 psi. Because using tactile feel alone is subjective and unreliable, methods for objective measurement of injection pressure have been developed. One method, the *compressed-air injection technique,* involves simply drawing up an extra column of air in the syringe and using the compressibility of that column to detect high-pressure injection (Figure 47-13). In-line pressure monitors also are commercially available. Pressure monitoring is of greatest importance during brachial plexus, sciatic nerve, and femoral nerve blocks.[73]

LOCAL ANESTHETIC NEUROTOXICITY

Under normal clinical scenarios, local anesthetics injected perineurally are rapidly diluted and cleared without evidence of direct neural toxicity. However, when using high concentration or after interneural injection, all common local anesthetics are potentially neurotoxic. Epinephrine-containing solutions may increase neurotoxic effects. Similarly, myotoxicity has been observed in experimental models. The concerns, although hypothetical, underscore the importance of avoiding intramural injection and using the lowest clinically effective dose of local anesthetic, as well as avoiding regional anesthesia in nerves with existing disease or injury.[44,73]

WILDERNESS REGIONAL ANESTHESIA BY AREA OF INJURY (Box 47-5)

Regional Anesthesia for the Neck and Clavicle

The neck and clavicle region is primarily innervated by the terminal nerves of the superficial cervical plexus (Figure 47-14). These include the lesser occipital, greater auricular, transverse cervical, and supraclavicular nerves. Anesthesia for superficial wound irrigation and repair or abscess incision and drainage can be achieved with a superficial cervical plexus block. Clavicle fractures can be quite painful. Relief can be achieved either through direct infiltration of local anesthetic into the clavicle fracture hematoma or direct infiltration combined with a superficial cervical plexus block.[42]

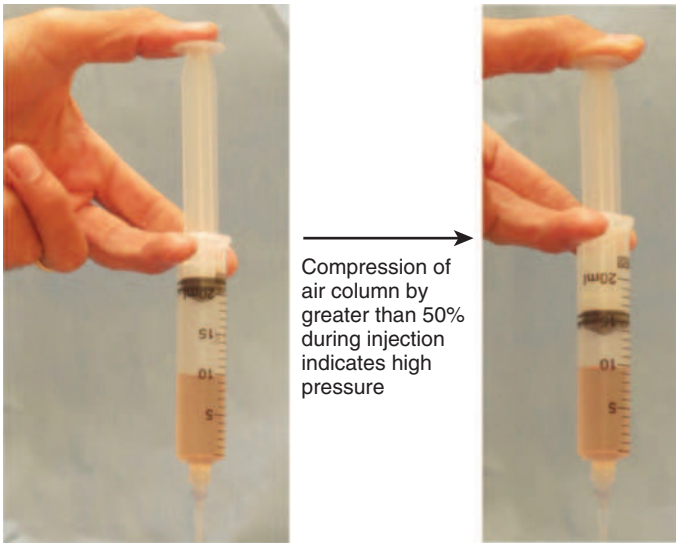


FIGURE 47-13 The compressed-air technique for pressure monitoring during regional anesthetic injection is a simple method to detect a high-pressure injection. When a column of air is drawn into the injection syringe, it acts as a dampener. If the injection resistance requires compression of the air column of 50% or more, this indicates a high-pressure injection, potentially resulting from unwanted and possibly injurious intraneural needle tip placement. *(Based on Tsui BC, Li LX, Pillay JJ: Compressed air injection technique to standardize block injection pressures, Can J Anesth 53(11):1098-1102, 2006.)*

BOX 47-5 Overview of Peripheral Nerve Blocks

Head and Face
Infraorbital nerve block
Mental nerve block
Supraorbital, supratrochlear, and infratrochlear nerve blocks
Dorsal nasal nerve block
Zygomaticotemporal nerve block
Zygomaticofacial nerve block
Mandibular nerve block

Neck and Upper Extremity
Superficial cervical plexus block
Suprascapular nerve block
Brachial plexus blocks
Cutaneous blocks of the forearm
Peripheral radial, median, and ulnar nerve blocks

Trunk
Intercostal and paravertebral blocks
Transabdominis plane and other abdominal wall blocks

Lower Extremity
Femoral nerve block
Lateral femoral cutaneous nerve block
Saphenous nerve block
Popliteal sciatic nerve block
Common peroneal nerve block
Posterior tibial nerve block

Regional Anesthesia for the Shoulder

Shoulder dislocations are common and respond well to a low-volume interscalene brachial plexus block. The key component of a successful reduction is muscle relaxation, which is generally achieved consistently with an interscalene brachial plexus block. A short-acting local anesthetic, such as 3% 2-chloroprocaine, is ideal. Alternatively, an intraarticular injection can provide effective analgesia, although the degree of muscle relaxation is generally less than that achieved with an interscalene block.[9]

Emergency Regional Anesthesia for the Arm

Injuries below the mid-upper arm, such as elbow dislocation, supracondylar fracture, and distal radius fracture, can be well managed with a brachial plexus block placed above (interscalene, or supraclavicular) or below (infraclavicular, axillary) the clavicle. In most wilderness circumstances, the axillary approach will be the most practical landmark-based approach to blockade

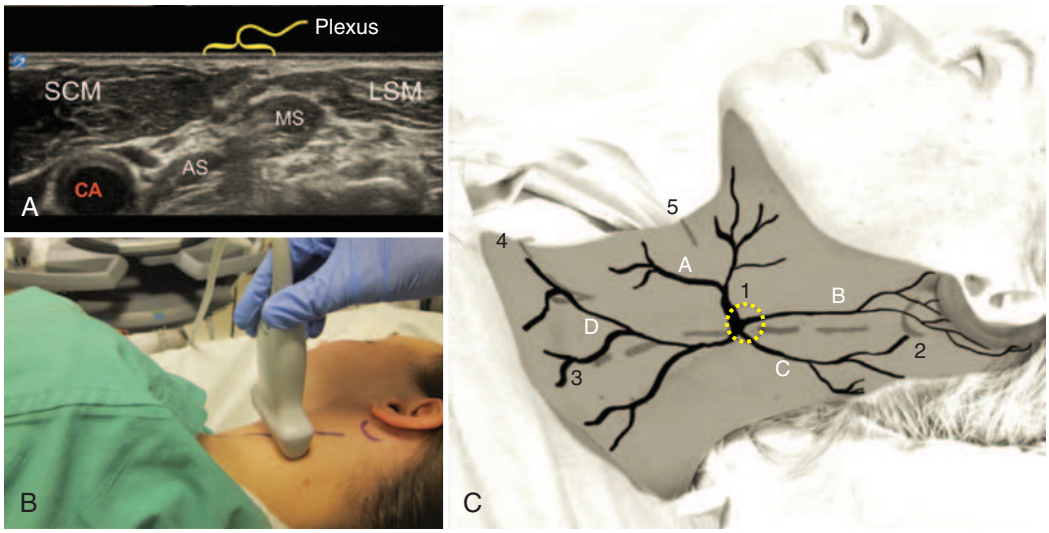


FIGURE 47-14 Ultrasound-guided superficial cervical plexus block. **A,** Sonographic anatomy: the superficial cervical plexus exits to the skin at the posterolateral border of the sternocleidomastoid muscle (*SCM*), passing in the intramuscular plane between the SCM and the levator scapulae muscle (*LSM*). The middle (*MS*) and anterior (*AS*) muscles are seen deep and medial adjacent to the carotid artery (*CA*). **B,** Patient and ultrasound transducer positioning along the posterolateral border of the SCM, approximately halfway between mastoid process and clavicle. **C,** Cutaneous innervation provided by the superficial cervical plexus is shown in gray shading. The four components of the superficial cervical plexus are *A,* transverse cervical nerves; *B,* greater auricular nerve; *C,* lesser occipital nerve; and *D,* supraclavicular nerves. These are targeted (*dashed circle 1*) where they exit to the skin along the posterolateral border of the SCM. Additional landmarks are *2,* mastoid process; *3,* SCM clavicular insertion; *4,* sternal notch; and *5,* superior pole of thyroid cartilage.

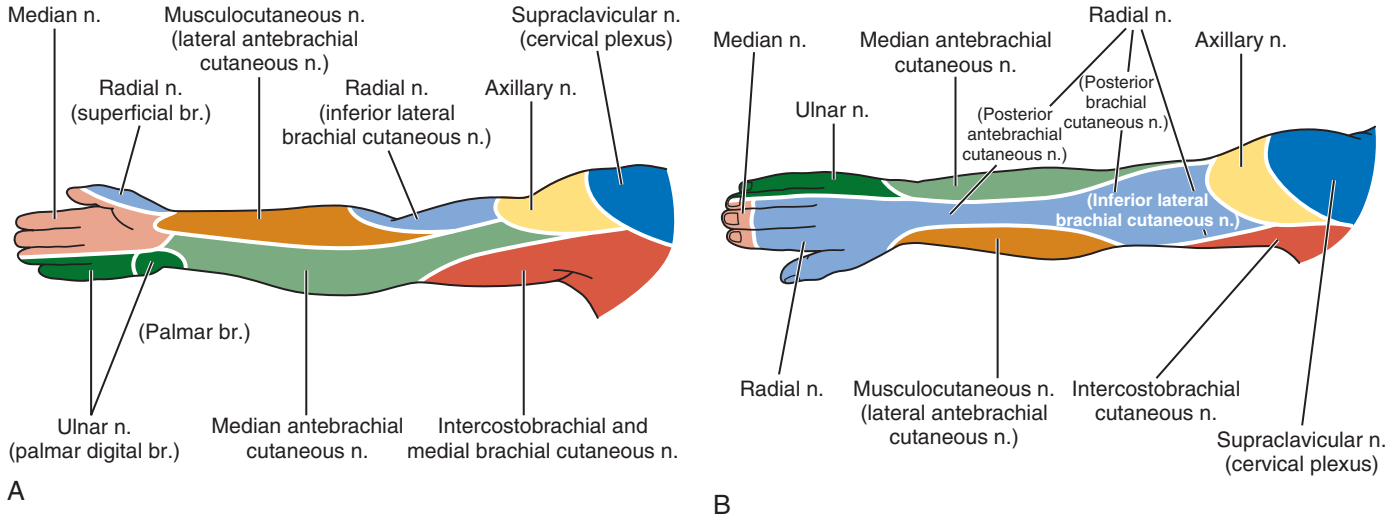


FIGURE 47-15 Cutaneous innervation of the upper extremity. **A,** Ventral nerve distribution of the upper extremity. **B,** Dorsal nerve distribution of the arm. br, Branch. *(From Brown D:* Atlas of regional anesthesia, *Philadelphia, 1999, Saunders. Illustrations by Jo Ann Clifford.)*

of the brachial plexus (Figure 47-15). To support the nerve block, many patients receive excellent anesthesia from a fracture hematoma block.[98]

Emergency Regional Anesthesia for the Hand

The hand is well anesthetized with a wrist block or distal blocks of the radial, median, and ulnar nerves, in combination or selectively, depending on the injury. These are beginner-level, superficial blocks that are generally well visualized with ultrasound or performed using a landmark technique.[57]

Emergency Regional Anesthesia for the Thorax

The pain from rib fractures can be ameliorated with intercostal nerve blocks (Figure 47-16). Even for a single rib fracture, multiple blocks are required because of nerve crossover between thoracic levels. Local anesthetic absorption is high with this block given the tissue vascularity. Particular caution should be taken to avoid use of excessively large volumes of local anesthetic or intravascular injection. Local anesthetic containing epinephrine is recommended. Use of ultrasound guidance helps avoid pleural puncture and pneumothorax.[97]

Emergency Regional Anesthesia for the Lower Extremity

The leg is innervated by branches of the lumbar plexus (L1 to L5; lateral femoral cutaneous, femoral, and obturator nerves) and the sacral plexus (L4 to S4; sciatic nerve and posterior cutaneous nerve of thigh) (Figure 47-17). The hip, femur, and knee are primarily innervated by the lumbar plexus, with smaller contributions from the sciatic nerve, and are optimally blocked with either a fascia iliaca compartment or a femoral nerve block.[10,21,38] The

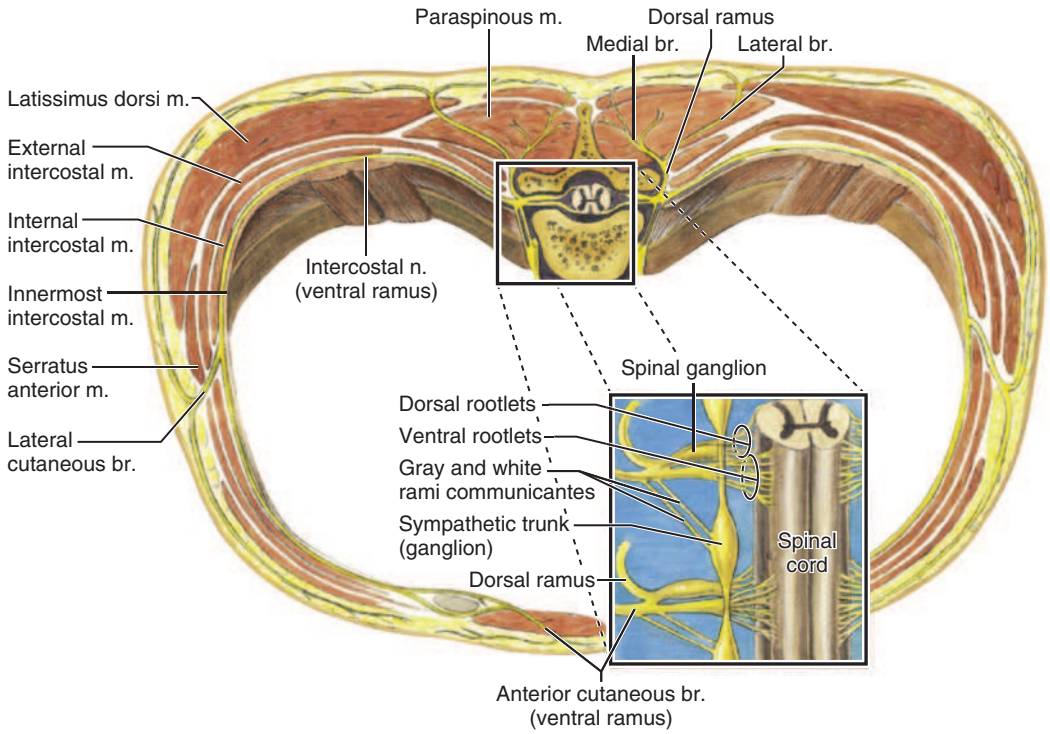


FIGURE 47-16 Cutaneous innervation of the trunk and intercostals. br, Branch. *(From Brown D:* Atlas of regional anesthesia, *Philadelphia, 1999, Saunders. Illustrations by Jo Ann Clifford.)*

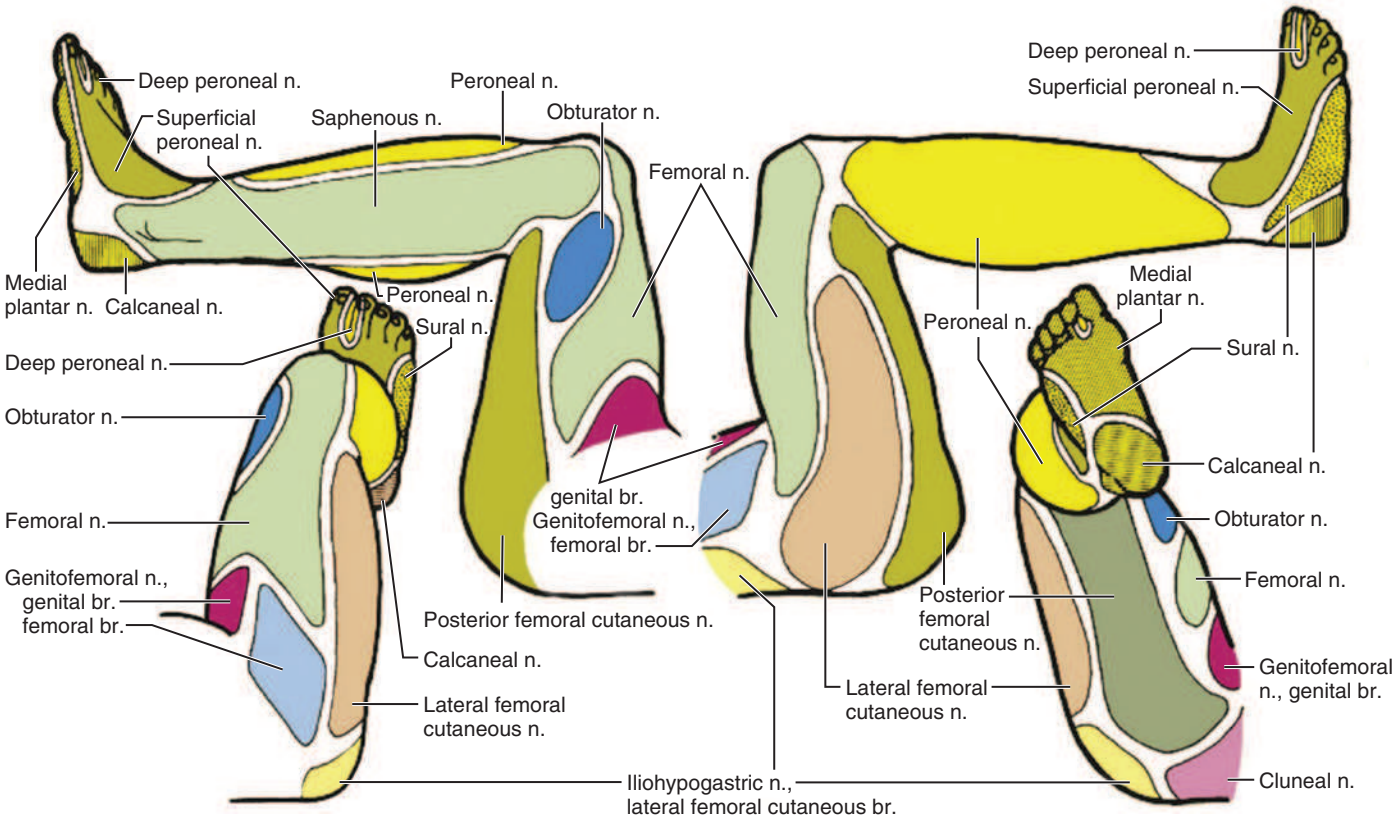


FIGURE 47-17 Lower-extremity innervation. br, Branch. *(From Brown D:* Atlas of regional anesthesia, *Philadelphia, 1999, Saunders. Illustrations by Jo Ann Clifford.)*

anterior thigh, proximal medial thigh, and quadriceps muscles are primarily innervated by the femoral nerve, which is easily blocked in the inguinal region. The lateral thigh is innervated by the lateral femoral cutaneous nerve, which runs in the fascia iliaca compartment lateral to the main femoral nerve. The posterior thigh and inferior portion of the buttock are innervated by the posterior cutaneous nerve of the thigh (branch of sacral plexus) that can be blocked with a subcutaneous skin wheal across the gluteal cleft, A small area of the medial distal thigh is innervated by the obturator nerve.[10,21,38,94,102]

Below the knee, the primary innervation is from the sciatic nerve. However, the saphenous branch of the femoral nerve provides cutaneous innervation to the medial aspect of the calf, as well as partial innervation of the ankle.[10,21,38,94] Fractures of the tibia and fibula are numbed with a block of the distal sciatic nerve at its bifurcation into the tibial and common peroneal nerves. Isolated fibula fractures can be blocked with a selective common peroneal nerve block. For complete anesthesia of the ankle, both the sciatic nerve (popliteal block) and the saphenous nerve must be blocked. Most of the innervation is sciatic, but in some patients, even a small, unblocked area of saphenous nerve innervation can allow significant pain and discomfort. The saphenous nerve is easily blocked in the midthigh, where it courses in the adductor canal deep to the sartorius muscle.[87]

The foot, apart from the medial ankle (saphenous nerve), is innervated by branches of the sciatic nerve that can be blocked at the popliteal fossa or individually more distally. The heel and calcaneus are well anesthetized with a posterior tibial nerve block. This is particularly useful for calcaneal fracture pain control. The posterior tibial nerve also innervates the sole of the foot and deeper structures, making a block an invaluable tool for foreign body exploration. The top of the foot is innervated primarily by the peroneal nerve, with a small contribution along the lateral aspect of the foot from the sural nerve (branch of the tibial nerve).[10,21,38,59,94,104]

SPECIFIC BLOCK TECHNIQUES

SUPERFICIAL CERVICAL PLEXUS BLOCK

Anatomy

The skin and superficial tissue overlying the submandibular region, neck, and clavicle, extending to include the "cape" of the shoulder and upper chest to the second thoracic vertebra (T2) level, are innervated by the superficial cervical plexus (Figure 47-18; see also Figure 47-14). This large anatomic territory makes the superficial cervical plexus block particularly useful in wilderness settings. The superior cervical plexus is composed of four nerves that exit to the skin at approximately the C4 level along the posterolateral border of the sternocleidomastoid muscle (SCM). The greater auricular nerve and supraclavicular nerves can be blocked individually, but most often, the cervical plexus is blocked altogether. The anterior cutaneous nerve of the neck (transverse cervical nerve) and greater auricular nerves supply the submandibular region and anterior neck, where abscesses in the male shaving area can occur. The supraclavicular nerves supply a large territory across the anterolateral neck and superior portion of the shoulder. The most dorsal areas overlying the scapular and medial neck are supplied by the cutaneous branches of the spinal nerves.[42]

Indications

Blockade of the superficial cervical plexus is done for clavicle fracture analgesia, central venous catheter placement, and abscess drainage or laceration repair in the innervated territory.

Landmark-Based Procedure

The posterolateral border of the SCM is palpated at the C4 level, approximately halfway between its clavicular insertion and the mastoid process. This is typically the level of the superior pole of the thyroid cartilage. The external jugular vein often crosses

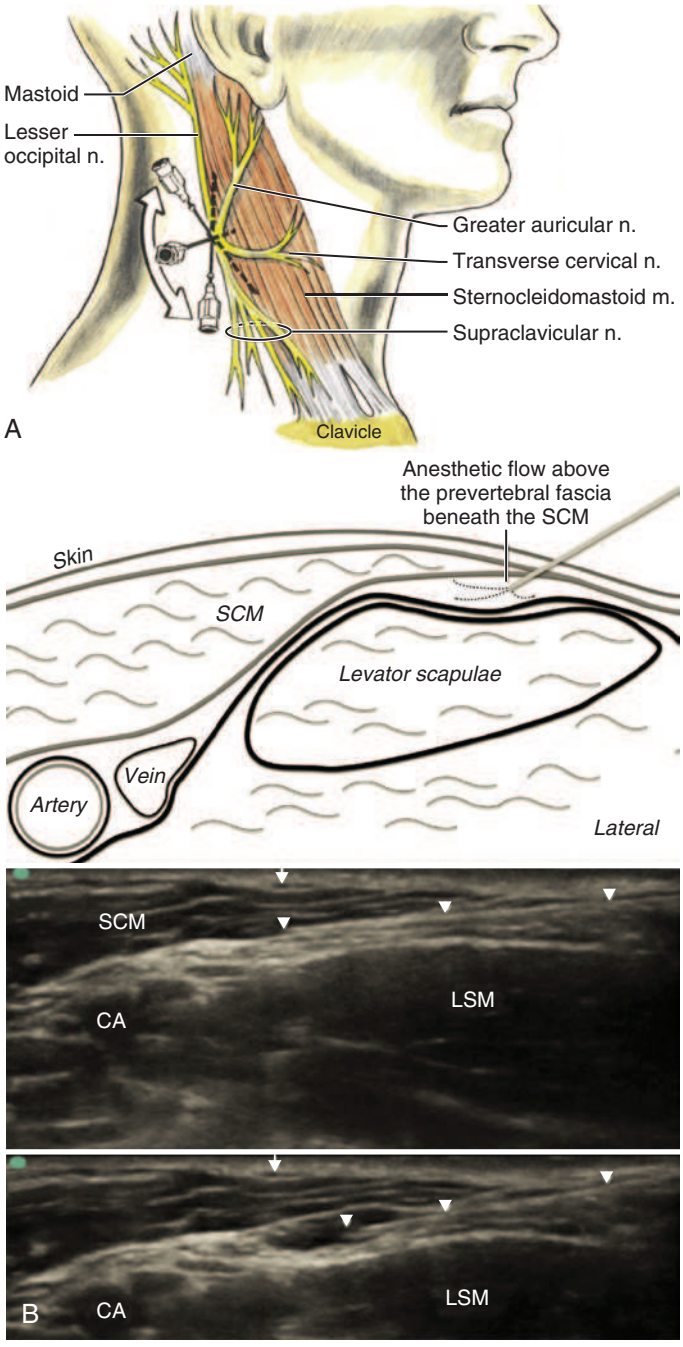


FIGURE 47-18 **A,** Landmark-based technique for superficial cervical plexus block. The needle injection point is along the posterior, lateral edge of the sternocleidomastoid muscle (*SCM*), approximately halfway between the SCM insertion on the clavicle and the mastoid process, as shown. **B,** Ultrasound-guided superficial cervical plexus block. As shown in the drawing, the ultrasound transducer is placed in the transverse plane at the anatomic location as described in **A.** *Top image,* At this scanning position, the tapering border or "beak" of the SCM is seen with the levator scapulae muscle (*LSM*) just deep and lateral; the carotid artery (*CA*) is seen medially. The greater auricular nerve (*arrow*) can often be found on the superficial aspect of the SCM. The needle (*arrowheads*) is advanced in a lateral-to-medial direction just underneath the SCM muscle belly. *Bottom image,* During injection, the local anesthetic should spread easily under the SCM superficial to the deeper fascia. (***A** from Brown D:* Atlas of regional anesthesia, *Philadelphia, 1999, Saunders. Illustrations by Jo Ann Clifford.)*

at this level, and care should be taken to avoid intravascular puncture. Approximately 5 mL of local anesthetic is injected just underneath the border of the SCM. Then, 2 to 3 mL is injected subcutaneously along the SCM border, 1 to 2 cm (each injection) above and below the initial injection point (see Figure 47-18).

Ultrasound-Guided Procedure

Positioning. The patient should be seated or in lateral decubitus position with enough space behind the head to allow maneuvering the syringe as needed, with a direct line of sight to the ultrasound display.

Needle Approach. For the posterior, in-plane approach, the transducer is placed in a transverse orientation across the neck with the probe marker facing medially (toward the thyroid cartilage) at the posterior border of the SCM at the level of the superior pole of the thyroid cartilage (C4). Here, the superficial cervical plexus is surrounded by hyperechoic fascia posterior to the SCM, and it can be difficult to identify the individual nerves. The greater auricular nerve is a useful landmark reliably identified as a small hypoechoic round structure just superficial to the SCM. The levator scapulae muscle (LSM) and carotid artery should be identified. Apply color Doppler to assess for other vessels. The needle is then inserted at the posterolateral border of the SCM and positioned to inject local anesthetic just deep to the SCM but superficial to the prevertebral fascia. Deeper injection should be avoided because it can result in a deep cervical plexus block.

Local Anesthetic

For brief procedures, use 5 to 10 mL of short-acting local anesthetic (3% 2-chloroprocaine). When prolonged anesthesia and analgesia are desired, a similar volume of long-acting local anesthetic (bupivacaine 0.25% to 0.5%) is used.

Potential Complications

Intravascular injection is a particular concern given the presence of the external jugular vein near the injection site. Frequent aspiration, slow injection of small aliquots, and steady, firm probe pressure to occlude veins are necessary precautions. Deeper injection can lead to a partial brachial plexus block or Horner's syndrome.[42]

THE ARM: BLOCKS OF TERMINAL BRANCHES OF BRACHIAL PLEXUS

The forearm block is useful for injuries and procedures to the hand, and the axillary block for the same in the forearm, wrist, and hand. With ultrasound guidance, it is possible to block the nerves at any point along their course. The five most common positions are as follows (Figure 47-19; see also Figure 47-15).

1. *Wrist blocks.* The radial, medial, and ulnar nerves are blocked either by landmark or with ultrasound guidance.

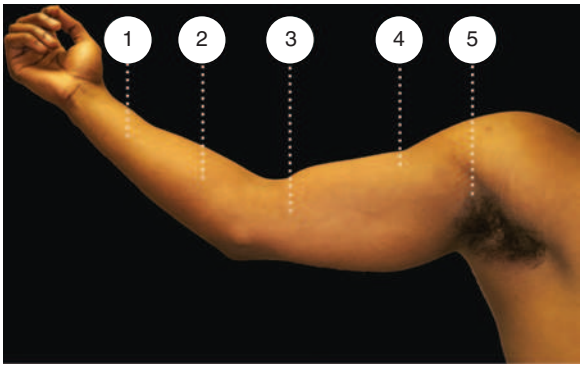


FIGURE 47-19 Locations for upper-extremity regional anesthesia. Distal to the neck and clavicular region, there are five regions where components of the brachial plexus can be targeted for regional anesthesia: *1,* wrist; *2,* forearm; *3,* supracondylar area; *4,* midhumeral area; and *5,* axilla.

2. *Forearm blocks.* The radial, medial, and ulnar nerves are blocked individually under direct ultrasound guidance.
3. *Elbow blocks.* Using ultrasound guidance, the radial nerve is most easily identified in the belly of brachialis muscle on the lateral aspect of the arm just above the elbow. The nerve can be blocked before the take-off of the superficial branch, resulting in greater anesthesia to the wrist as well as clinical wrist-drop from extensor motor paralysis. The median nerve can be blocked here as well, although the nerve at this location is closely associated with vascular structures, adding a level of complexity. The ulnar nerve can blocked at the elbow; however, caution due to the tight anatomic space and potential for nerve injury is warranted.[10]
4. *Midhumeral blocks.* Using ultrasound guidance, the median nerve and ulnar nerve remain adjacent to the brachial artery, and are easily blocked together. At this level, the radial nerve moves into the spiral grove and must be blocked separately. The musculocutaneous nerve travels separately underneath the biceps brachii on top of the brachialis muscle.
5. *Axillary block.* The radial, median, and ulnar nerves are closely clustered around the axillary artery within a tight fascial sheath. The nerves are generally quite superficial and easily blocked. The musculocutaneous nerve travels closely in a separate compartment within the coracobrachialis muscle. The block can be performed using landmarks or with ultrasound guidance.

Axillary Block

Indications. The axillary brachial plexus block can be used for any injury to the upper extremity from the midhumerus and distally, including elbow, wrist, and hand fractures (Figure 47-20).

Landmark-Based Procedure. The axillary block entails placing local anesthetic near the musculocutaneous, median, radial, and ulnar nerves at the distal axilla, guided by the axillary artery pulse (Figure 47-21). This block is performed by having the patient supine with shoulder abducted to 90 degrees and externally rotated, with the elbow flexed. Stand or sit at the patient's side caudal to the arm, and identify the axillary pulse over the proximal humerus. The site of entry is sterilized and local anesthesia injected locally before a blunt needle is placed. A 22-gauge blunt needle is then advanced superior to the pulse. The musculocutaneous nerve lies deep to the artery, and this is where 5 to 10 mL of local anesthetic is deposited. Superficial to the musculocutaneous nerve and the pulse of the axillary artery is the median nerve, where an additional 5 to 10 mL is deposited. The needle is withdrawn to the skin and then redirected inferior (medial) and deep to the artery, where the radial nerve is located; another 5 to 10 mL is deposited. Lastly, the needle is withdrawn to the depth of the axillary artery, where the ulnar nerve is located, and the remaining 5 to 10 mL of local anesthetic is deposited. During this procedure, the patient may experience paresthesias, which can be used to verify the location of the

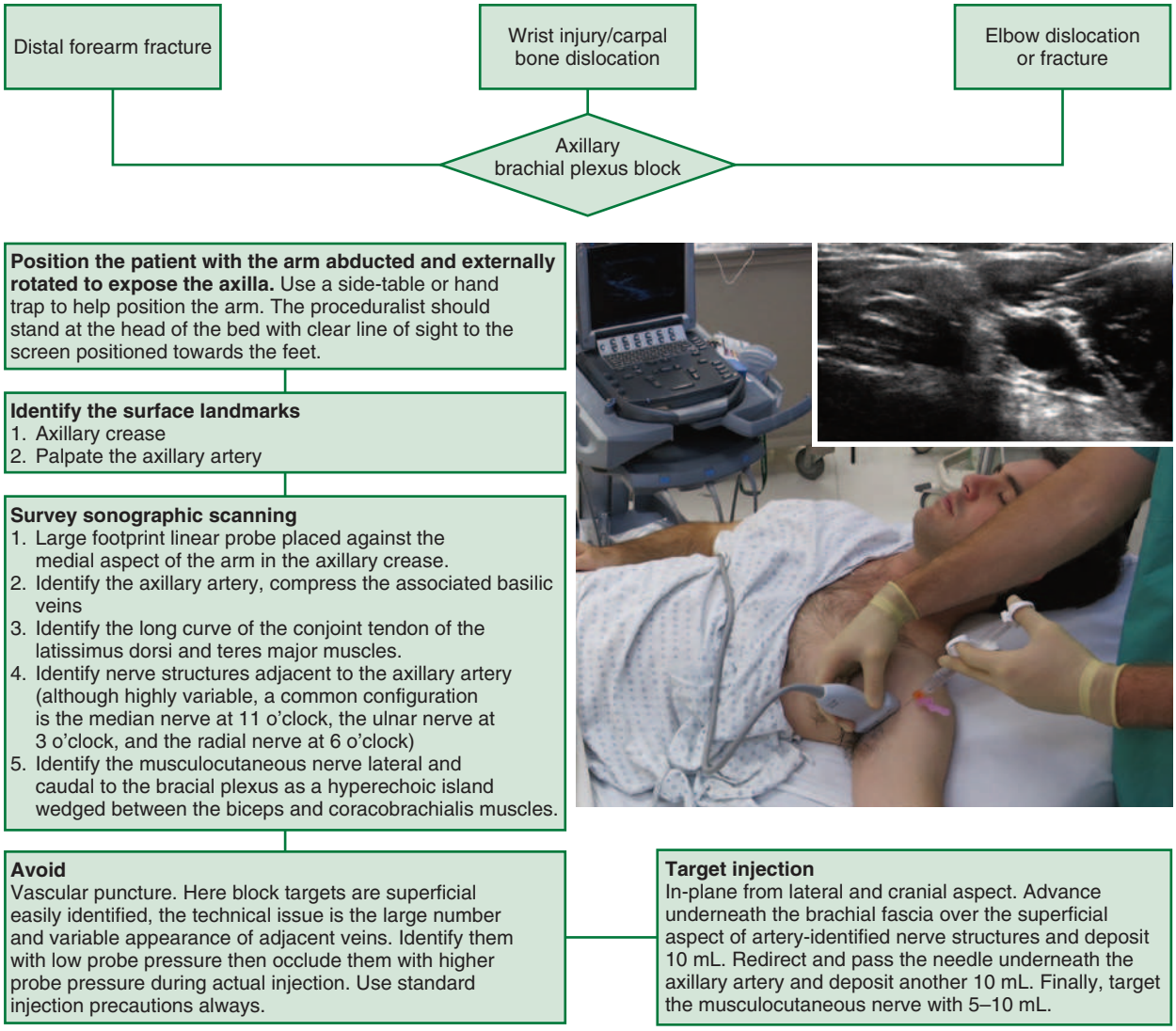


FIGURE 47-20 Summary guide for the ultrasound-guided axillary brachial plexus block.

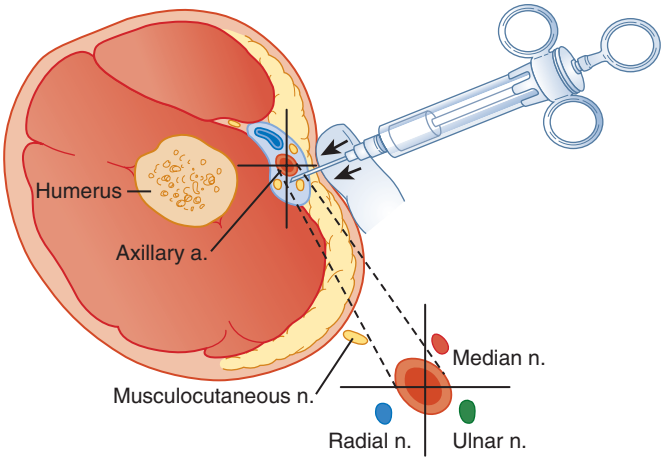


FIGURE 47-21 Landmark-based axillary brachial plexus block. *(From Brown D: Atlas of regional anesthesia, Philadelphia, 1999, Saunders. Illustrations by Jo Ann Clifford.)*

needle, although this is not necessary. If the axillary artery is entered, continue to advance the needle through the artery, and then aspirate to make sure that the needle is deep to the artery (not intravascular) before depositing the local anesthetic. If the artery is entered, direct pressure should be maintained over the site for at least 5 minutes to limit bleeding.[10]

Ultrasound-Guided Procedure

Anatomy. The radial, median, and ulnar nerves have a variable position around the axillary artery, and specific identification is not required. The radial nerve tends to lie deep (posterior) to the axillary artery and is the most likely to be missed if the posterior aspect of the compartment is not targeted. The musculocutaneous nerve travels in its own compartment and is blocked separately[38,74] (Figure 47-22).

Positioning. The patient can be supine or reclined with the arm abducted and externally rotated on a Mayo stand. The clinician stands by the patient's head with the ultrasound screen positioned to allow a direct line of sight at the patient's waist (Figure 47-23).

Needle Approach. The needle approach is in-plane from the lateral (cephalad) aspect toward the posterior aspect of the axillary artery targeting the radial nerve. A second injection is

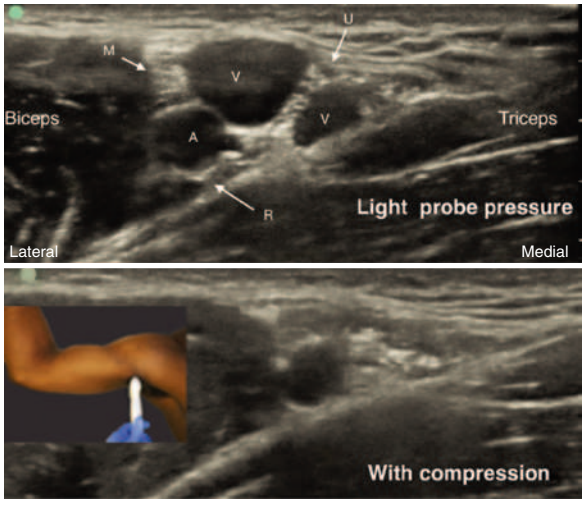


FIGURE 47-22 Ultrasound scanning of axillary brachial plexus and effect of probe pressure on visualization of axillary veins. The radial (*R*), median (*M*), and ulnar (*U*) nerves are seen close to the axillary artery (*A*) and veins (*V*). To identify all the vascular structures and avoid intravascular injection, light pressure scanning is used to visualize veins that can be easily compressed with probe pressure during needle advancement.

then made along the anterior (superficial) aspect of the artery to target the median and ulnar nerves. Lastly, the musculocutaneous nerve is blocked separately. The out-of-plane approach can also be used (Figure 47-23).

Local Anesthetic. For brief procedures, use 20 to 30 mL of short-acting local anesthetic (3% 2-chloroprocaine). When prolonged anesthesia and analgesia are desired, a long-acting local anesthetic (bupivacaine 0.25% to 0.5%) is used.

Potential Complications. The multitude of large vessels in the axillary brachial plexus sheath make intravascular injection a significant concern. Frequent aspiration, slow injection with small aliquots, and steady, firm probe pressure to occlude veins are necessary precautions.[74]

THE HAND

The hand is innervated by the distal branches of the radian, median, and ulnar nerves. Even massive hand injuries can be completely blocked with distal blocks below the elbow, or at the forearm or the wrist. Individual fingers are blocked with digital blocks. The common fifth metacarpal or "boxer's fracture" is well managed with an ulnar nerve block. For larger injuries or injuries in border territories, it is usually most efficient to block all three contributory nerves (ulnar, median, and radial) either as a wrist block or with ultrasound-guided forearm blocks (Figure 47-24; see also Figure 47-15).

Landmark-Based Blocks for the Hand and Fingers

Wrist Block

Positioning. To accomplish a wrist block, it may be necessary to block one or two of the three nerves that innervate the hand. To perform this nerve block, the shoulder is abducted with the arm supported and the hand in a supine position (Figure 47-25).

The ulnar nerve is located medial to the ulnar pulse at the level of the ulnar styloid. The needle is advanced at a steep angle just medial to the ulnar pulse. If a paresthesia is obtained, 3 to 5 mL of local anesthetic is injected. If no paresthesia is obtained, the needle can be moved in a fan-like distribution while injecting local anesthetic to ensure nerve block.

The median nerve is located between the ulnar styloid process and the distal radial prominence, between the flexor carpi radialis and palmaris longus tendons. With the wrist and fingers flexed, these tendons are easily identified. A needle is advanced deep to the tendons, and 3 to 5 mL of local anesthetic is injected.

The radial nerve is blocked by injecting 5 mL of local anesthetic subcutaneously as a field block over the radial aspect of the wrist proximal to the "anatomic snuffbox" (Figure 47-26).

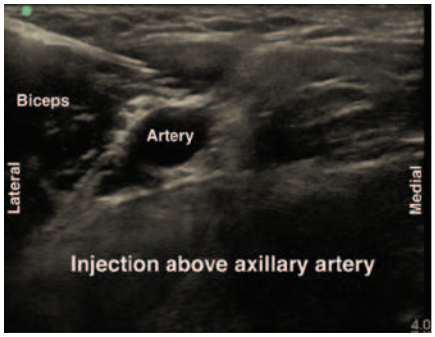
Local Anesthetic. For brief procedures, 2 to 3 mL of short-acting local anesthetic (2% lidocaine) per block is normally sufficient. When prolonged anesthesia and analgesia are desired, a long-acting local anesthetic (bupivacaine 0.25% to 0.5%) is used.

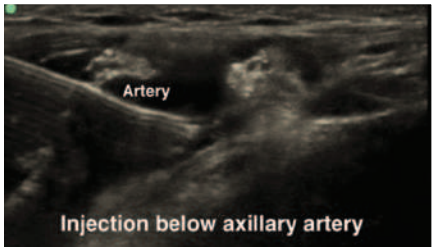
Digital Block. The dorsal digital and proper palmar digital nerves are located over the medial and lateral aspects of the digits. To perform this block, the hand is pronated and supported. The skin is entered on the dorsal aspect of the digit. A needle is inserted at the medial and lateral aspect of the proximal phalanx (Figure 47-27), and 1 to 2 mL of local anesthetic is injected on each side of the digit. Alternatively, the tumescent technique involves simply injecting underneath the volar skin into the subcutaneous tissue, above the tendon sheath. The injection is continued until swelling or tumescence is noted circumferentially, indicating local spread to both the volar and the dorsal digital nerves.[103]

ULTRASOUND-GUIDED FOREARM BLOCKS: RADIAL, MEDIAN, AND ULNAR NERVES

Positioning

Position the patient with the affected extremity held palm-up and resting comfortably. Place the ultrasound machine in-line with the practitioner's line of sight to allow an unobstructed view of the ultrasound screen.





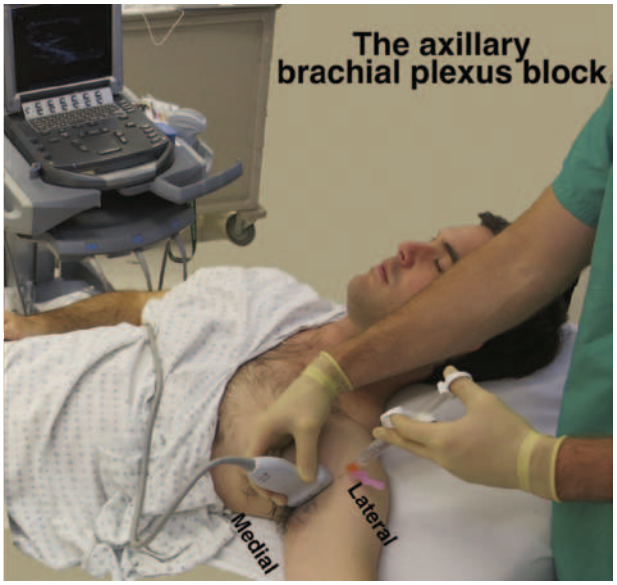
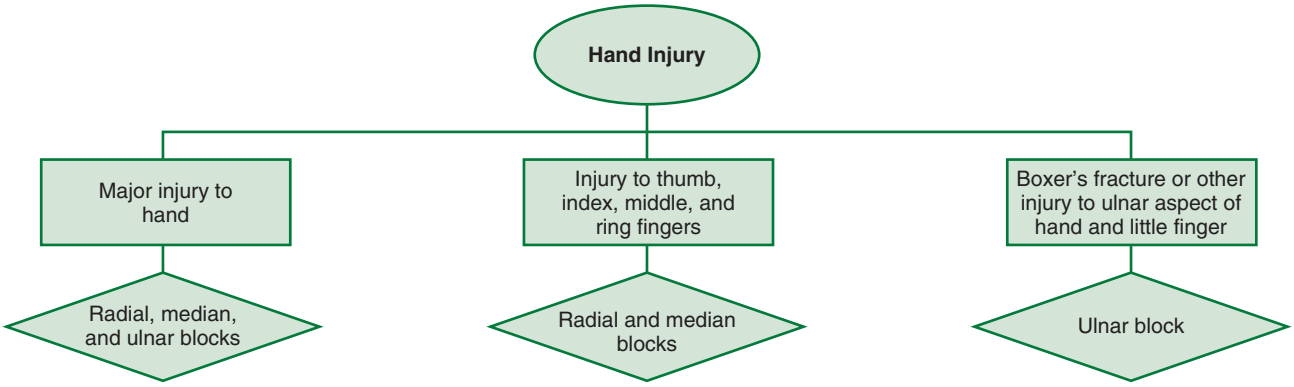


FIGURE 47-23 Ultrasound-guided axillary brachial plexus injection. *Left,* The ultrasound-guided axillary brachial plexus block involves injecting both superficial and deep to the axillary artery. *Right,* Positioning is most convenient with the patient supine and the arm abducted as shown.



Position the patient to expose the volar forearm
Sitting with the arm externally rotated and the forearm resting on a Mayo stand with the ultrasound machine at the head of the bed and the operator at the patient's waist, internally rotate the arm for the elbow radial block.

Survey sonographic scanning

Midforearm
Linear probe in the midforearm in transverse orientation

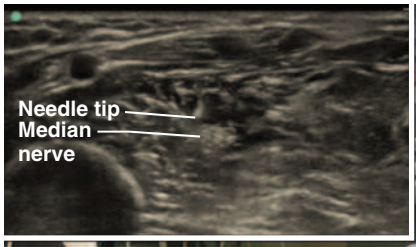
1. **Ulnar nerve** runs closely adjacent to the ulnar aspect of its artery and separates in the mid to proximal forearm
2. **Median nerve** has typical honeycomb appearance in the midforearm between the flexor digitorum superficialis and the flexor digitorum profundus at the midline of the forearm.
3. **Radial nerve;** superficial branch runs laterally, adjacent to its artery, but can be difficult to locate in some patients.

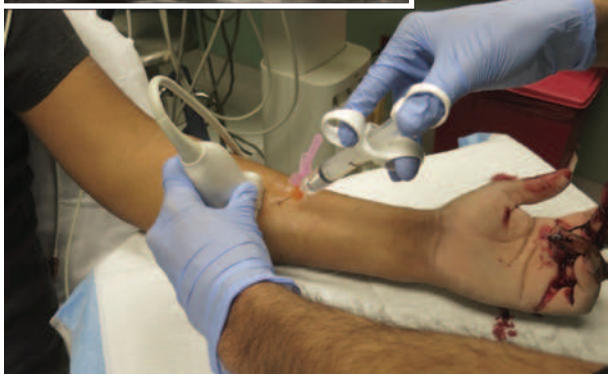
Above the elbow

4. **Radial nerve.** Transverse scan at the lateral aspect of the arm just above the elbow. Here the radial nerve is easily visible between the brachioradialis and brachialis muscles.

"Stay away" Injection
Target the fascial plane adjacent to the nerve itself. Deposit local anesthetic in a crescent around the nerve; full circumferential spread is not needed.

Avoid
Forearm blocks are very effective without requiring excessive needle manipulations that may increase the risk for accidential nerve contact.





Target injection
2–5 mL per nerve in the fascial sheath

FIGURE 47-24 Summary guide for regional anesthesia for hand injuries.

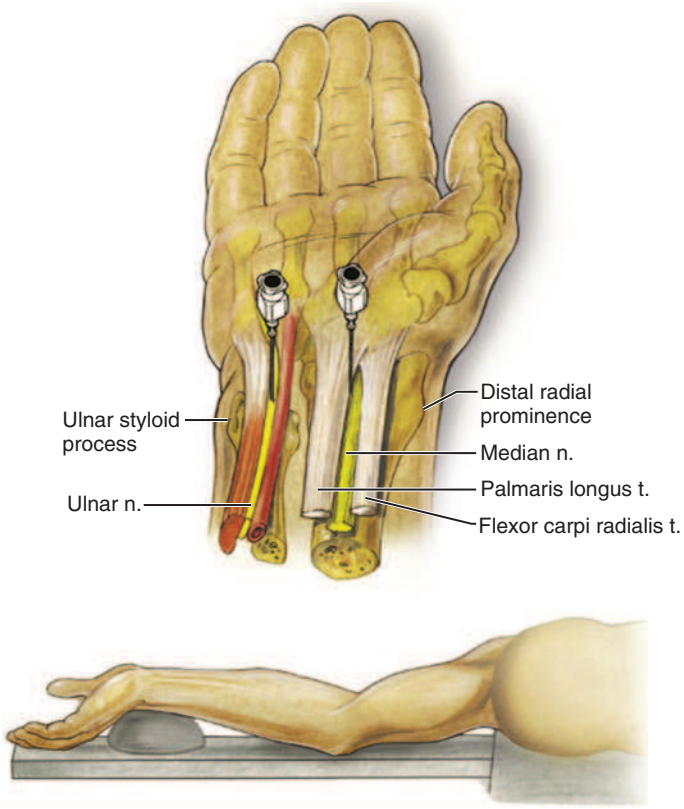


FIGURE 47-25 Landmark approach to the wrist block. t, Tendon. *(From Brown D:* Atlas of regional anesthesia, *Philadelphia, 1999, Saunders. Illustrations by Jo Ann Clifford.)*

Radial Nerve

For the supracondylar approach to the radial nerve, the transducer is placed on the lateral aspect of the arm just above the elbow. At this location, the radial nerve is most easily found coursing in the intramuscular plane between the triceps and biceps muscles; a block here will ensure that both the superficial and the deep branches are anesthetized (Figure 47-28). In the forearm, the superficial radial nerve is located radial to the radial artery. Starting at the wrist, locate the pulsatile radial artery. The radial nerve runs in the fascial plane just radial to the radial artery. Often, the radial nerve is small and difficult to identify as a separate structure at the wrist because of its proximity to the artery, so tracing it proximally can aid identification.

Median Nerve

To locate the median nerve, begin at the wrist with the midpoint of the transducer over the middle of the wrist crease. Move the probe proximally, and look for the classic "honeycomb" appearance of the median nerve in the fascial plane between the flexor digitorum superficialis and profundus at the midforearm. As the probe moves more proximal, tendon structures will disappear, where the median nerve will persist (Figure 47-29).

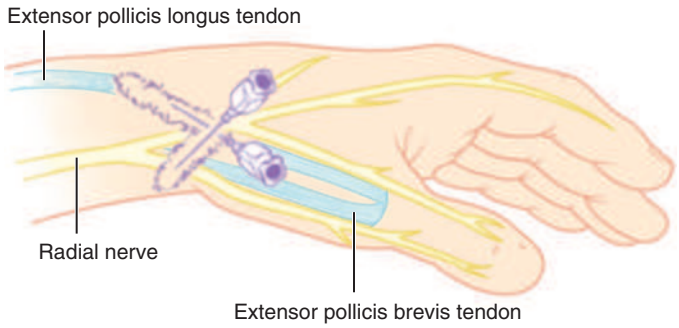


FIGURE 47-26 Anatomic landmarks and method of needle insertion for a radial nerve block at the wrist. *(Modified from Miller RD. Miller's anesthesia. 8th ed. Philadelphia: Elsevier; 2014.)*

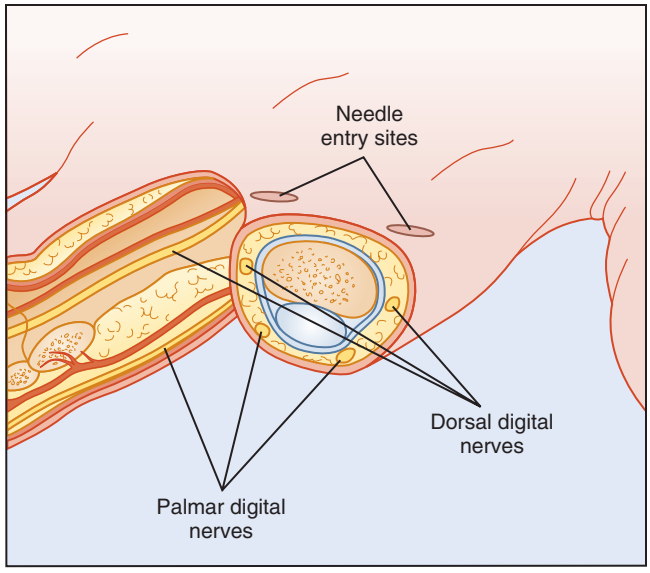


FIGURE 47-27 Anatomy and needle entry sites for digital nerve block. *(Courtesy Bryan L. Frank.)*

Ulnar Nerve

The ulnar nerve is located at the ulnar aspect of the ulnar artery. Starting at the wrist, locate the pulsatile ulnar artery. Immediately ulnar to the ulnar artery, a small nerve usually is easily visualized. Scanning proximally, the ulnar nerve will separate from the artery in characteristic fashion (Figure 47-30).

Needle Insertion and Injection

The radial, median, and ulnar nerves can be approached either with an in-plane or out-of-plane approach, depending on provider preference and ergonomics of the situation. From 3 to 5 mL of local anesthetic is normally sufficient for a complete block.[10,38,57]

FEMORAL AND FASCIA ILIACA BLOCKS

Anatomy

The femoral nerve block for hip and femoral shaft fractures is the most widely used nerve block in emergency and prehospital medicine.[50] The femoral nerve contributes significant innervation

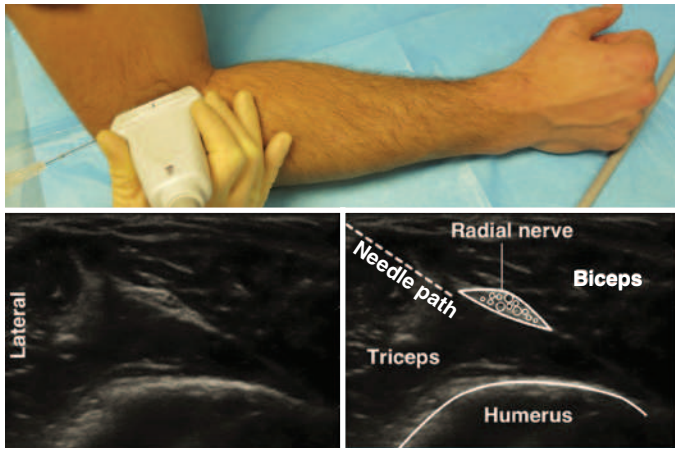


FIGURE 47-28 Ultrasound-guided supracondylar radial nerve block. The radial nerve can be targeted just above the elbow on the lateral aspect of the arm, where it is seen as an isolated, hyperechoic, honeycomb structure traveling between the biceps and triceps muscles.

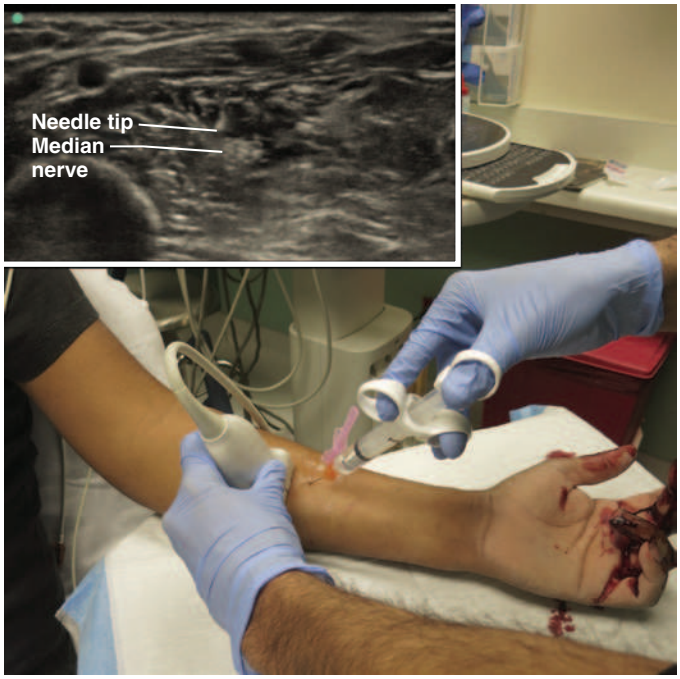


FIGURE 47-29 Ultrasound-guided median nerve block. The median nerve is easily identified on the volar surface of the midforearm as an isolated, hyperechoic, honeycomb structure traveling just deep to the superficial flexor muscles. In this example, it is being targeted using an out-of-plane approach.

FIGURE 47-31 Distribution of the femoral nerve. *(From Obrien MD: Aids to the examination of the peripheral nervous system, ed 4, United Kingdom, 2008, Saunders.)*

to the hip joint, femoral shaft, and knee. The saphenous nerve is a terminal branch of the femoral nerve that provides cutaneous innervation to the medial leg below the knee and to the medial aspect of the ankle joint (Figure 47-31). The femoral nerve travels in the fascia iliaca plane along with the lateral femoral cutaneous nerve and obturator nerve. These nerves are sometimes blocked with proximal spread of local anesthetic, creating a "3-in-1" block.[38,50,94]

Indications

A femoral nerve block can be used for proximal femoral fracture, middle and distal femoral shaft fracture, patellar fracture, knee injuries, and large soft tissue injuries to the anterior thigh or medial lower leg. For ankle fractures with significant medial pain, a femoral block can be used in addition to a sciatic nerve block[18,32] (Figure 47-32).

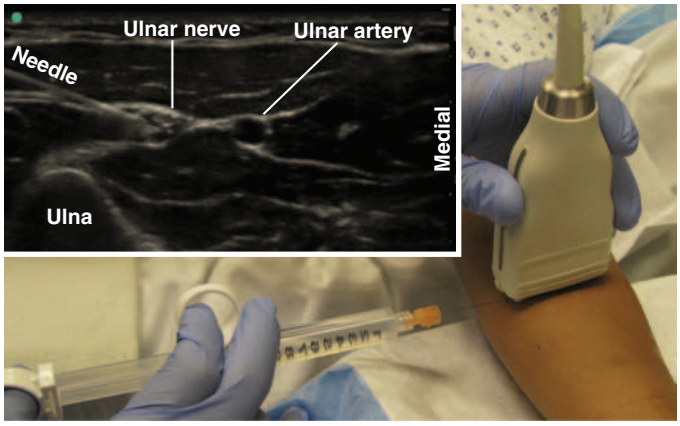


FIGURE 47-30 Ultrasound-guided ulnar nerve block. The ulnar nerve is identified on the ulnar aspect of the volar surface of the midforearm as an hyperechoic, honeycomb structure traveling just lateral to the ulnar artery and just deep to the superficial flexor muscles. In this example, it is being targeted using an in-plane approach.

Landmark-Based Procedure

With the patient supine, the inguinal ligament, anterior-superior iliac spine, pubic tubercle, and femoral pulse at the level of the inguinal ligament are identified. A 10-cm (4-inch), 22-gauge, blunt-bevel needle helps the operator feel the "pop" passing first through the fascia lata, then through the fascia iliaca, entering the skin approximately 1 cm (0.4 inch) lateral to the femoral pulse and advanced in the anteroposterior plane. As the needle is advanced, a paresthesia may be elicited, although this is variable. A large volume (20 to 50 mL) of relatively dilute local anesthetic (e.g., 0.25% bupivacaine or 0.5% ropivacaine) is injected. The femoral artery is close to the injection site, so

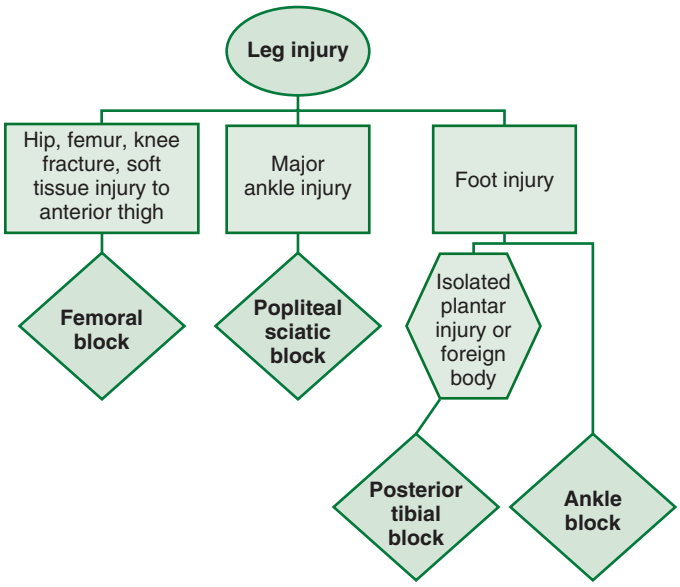


FIGURE 47-32 Summary guide for regional anesthesia for lower-extremity injuries.

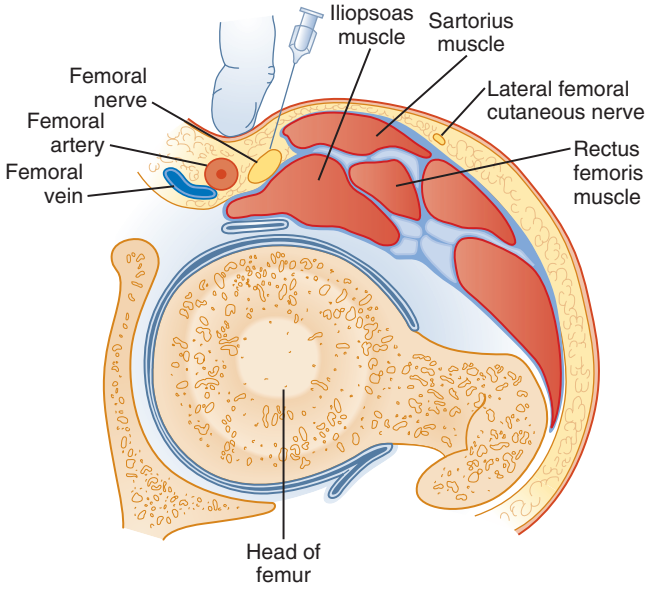


FIGURE 47-33 Landmark femoral nerve block.

arterial puncture of the artery and intravascular injection are possible and should be avoided (Figure 47-33).

Ultrasound-Guided Procedure

Anatomy and Indications. The femoral nerve travels with the iliacus muscle just underneath its fascial covering. The point of entry into this compartment can be chosen just lateral to the nerve itself (for the femoral nerve block) or 3 to 4 cm (1.2 to 1.6 inches) lateral to the femoral artery along the inguinal crease, creating the "fascia iliaca" block. Ultimately, with a large injection, the local anesthetic spread within the sub–fascia iliaca compartment is unlikely to be substantially different between the two approaches. In some patients, spread of local anesthetic will proceed proximally, resulting in blockade of the lateral femoral cutaneous and obturator nerves in addition to the femoral nerve, the 3-in-1 block.[10,21,38,94] Any injection above the fascia iliaca typically will not produce a successful block.

Positioning. With the patient supine, the inguinal ligament, anterior-superior iliac spine, pubic tubercle, and femoral pulse at the level of the inguinal ligament are identified with the ultrasound screen positioned with a direct line of sight.

Needle Approach. The needle approach is in-plane from the lateral to medial aspect toward the lateral edge of the femoral nerve. The femoral nerve is typically visualized as a hyperechoic, ovoid structure just lateral to the femoral artery (Figure 47-34). Local anesthetic should be seen to displace the nerve downward or clearly flow underneath the nerve. Local tracking superficial to the artery is a sign that the needle tip is too shallow and has not entered the fascia iliaca compartment.

Local Anesthetic. For prolonged fracture analgesia, 20 to 50 mL of dilute long-acting local anesthetic (e.g., 0.25% bupivacaine or 0.5% ropivacaine) is used.

Potential Complications. The proximity of the femoral nerve to its artery introduces risk of vascular puncture if the needle is advanced medially beyond the nerve. If puncture of the artery occurs, the procedure should be stopped and firm pressure applied to limit hematoma formation. Pain with needle advancement may indicate needle-to-nerve contact or entry into the iliopsoas muscle. Standard precautions should be taken to avoid intravascular injection and nerve injury. After successful femoral nerve block, the patient will be unable to bear weight, so appropriate fall precautions should be taken.

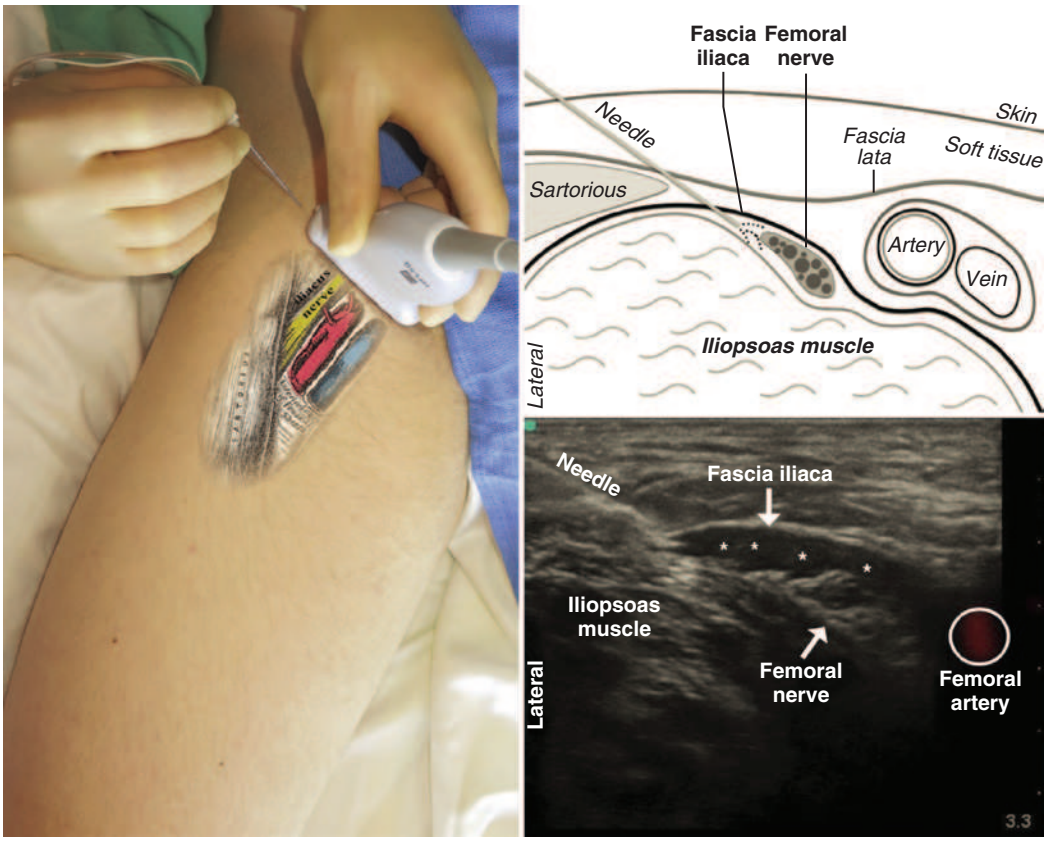


FIGURE 47-34 Ultrasound-guided femoral nerve block. The femoral nerve is most conveniently targeted with a lateral-to-medial, in-plane approach just above the inguinal crease, proximal to the femoral artery bifurcation. Here, the femoral nerve runs just underneath the fascial covering (fascia iliaca) of the iliopsoas muscle and lateral to the femoral artery. Local injection should flow underneath the fascia iliaca and surround the nerve.

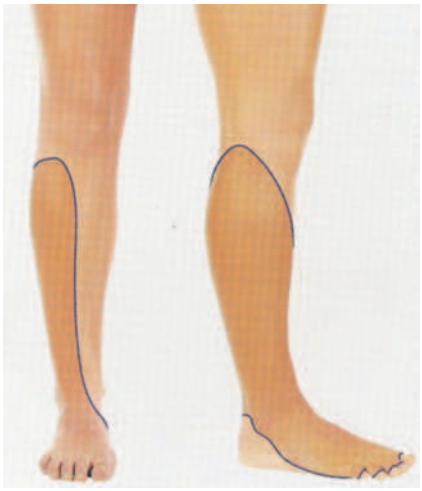


FIGURE 47-35 Distribution of the common peroneal nerve. *(From Obrien MD:* Aids to the examination of the peripheral nervous system, *ed 4, United Kingdom, 2008, Saunders.)*

POPLITEAL SCIATIC BLOCK

Anatomy and Indications

The peroneal and tibial branches of the sciatic nerve can be blocked together for major injuries distal to the knee, such as combined tibia and fibula fractures, or ankle fracture or dislocation. For complete ankle anesthesia, both components of the sciatic nerve and the saphenous nerve should be blocked. For localized injuries, the tibial nerve and peroneal nerve can be selectively blocked, depending on injury location.[18,21,32]

Landmark-Based Procedure

The landmark-based popliteal sciatic nerve block, although effective in the hands of a well-practiced anesthetist, is infrequently performed because of the risk for complications and difficulty of the procedure. The ultrasound-guided approach is discussed below.

Common Peroneal Nerve Block

The landmark-based common peroneal nerve block is a technically simple and reliable block for lateral ankle trauma, such as fibula fracture or soft tissue injury (Figure 47-35). With the patient in a lateral position, the fibular head is identified. A blunt-bevel needle is introduced just below the fibular head. A paresthesia can be elicited at a depth of 0.5 to 1 cm (0.2 to 0.4 inch), and 5 mL of local anesthetic is deposited (Figure 47-36).

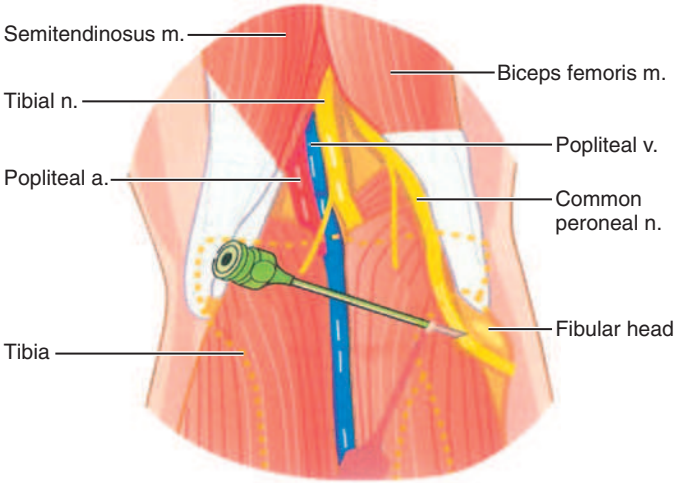


FIGURE 47-36 Common peroneal nerve block. *(From Waldman SD:* Atlas of interventional pain management, *Philadelphia, 2009, Saunders.)*

Ultrasound-Guided Popliteal Sciatic Nerve Block

Anatomy and Indications. The sciatic nerve innervates the posterior thigh and most of the leg below the knee. The sciatic nerve (L4-L5, S1-S3) arises from the lumbosacral plexus in the posterior aspect of the pelvis and travels down the posterior thigh to the popliteal fossa, where it divides into the common peroneal (lateral) and tibial (medial) nerves (Figures 47-37 and 47-38; see also Figure 47-17). Sensory innervation to the medial calf and somewhat to the ankle joint is provided separately by the saphenous branch of the femoral nerve.[10,21,38,41,94]

Positioning. In many injured patients, the most convenient approach is a partial lateral decubitus position with the injured leg superior and rotated to expose the lateral portion of the popliteal fossa. The block can also be performed with the patient supine with hip and knee flexed and while the distal leg is supported or prone.

Needle Approach. With a linear probe in transverse orientation at the popliteal crease, the tibial component of the sciatic nerve is typically found just superficial to the popliteal artery. Scanning proximally along the tibial nerve, the peroneal and tibial components of the nerve come together as the common sciatic nerve. The optimal needle tip target is just at this point, where the common sciatic nerve bifurcates into its tibial and peroneal branches (Figure 47-39). Use of color Doppler with light transducer pressure should be used to help avoid vascular puncture. A caudal transducer tilt may improve visualization. Spread of hypoechoic local anesthetic should be visualized in real time, spreading around the nerve. Local anesthetic should travel along the common sheath and distally beyond the bifurcation into the individual sheaths covering the common peroneal and tibial nerves.

Local Anesthetic. For prolonged fracture analgesia, 20 to 40 mL of long-acting local anesthetic (e.g., 0.25% bupivacaine or 0.5% ropivacaine) is used.

Potential Complications. Compartment syndrome is a major consideration for any high-velocity or crush injury below the knee.[64] In particular, tibial fractures must be monitored closely. It is not clear how a nerve block changes examination of a limb for potential compartment syndrome. Ischemic pain breaking through a block helps to identify compartment syndrome; the counterpoint is that the block may mask symptoms

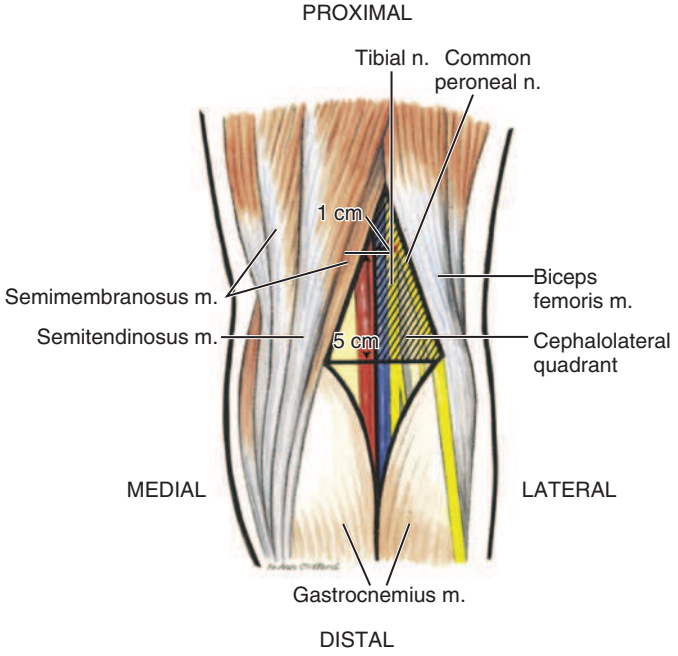


FIGURE 47-37 Anatomy of the distal sciatic nerve at the popliteal fossa. *(From Brown D:* Atlas of regional anesthesia, *Philadelphia, 1999, Saunders. Illustrations by Jo Ann Clifford.)*

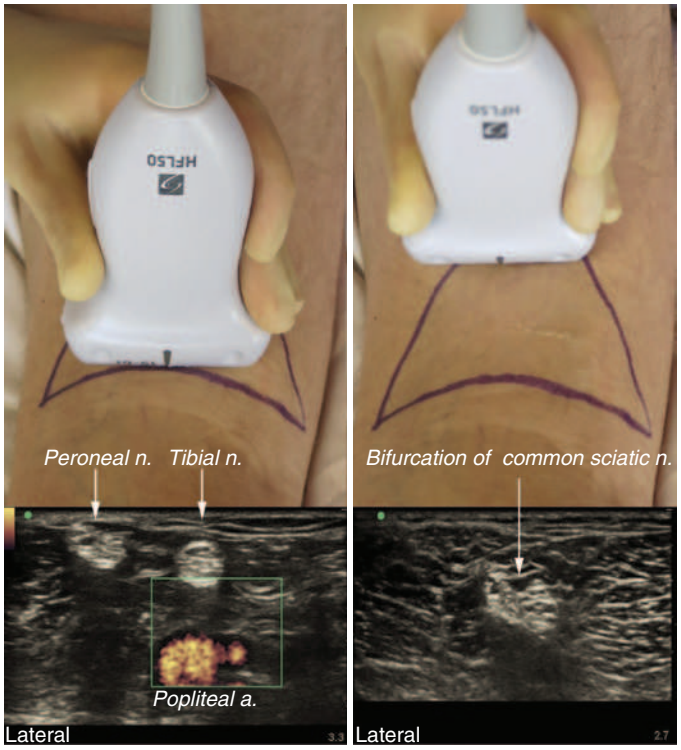


FIGURE 47-38 Ultrasound visualization of the distal sciatic nerve bifurcation at the popliteal fossa. The sciatic nerve travels down the posterior leg, then bifurcates into the peroneal and tibial branches in the popliteal fossa. This distinctive bifurcation is a useful ultrasonographic landmark. After bifurcation, the tibial nerve courses medially and the common peroneal nerve laterally.

and delay diagnosis.[56] Therefore, close attention to a potentially evolving compartment syndrome is mandatory with any block of the lower extremity. Additionally, the proximity of large vessels in the popliteal fossa make intravascular injection a significant concern. Frequent aspiration, slow injection with small aliquots, and steady, firm probe pressure to occlude veins are necessary precautions.

Landmark-Based Ankle Block

To achieve analgesia of the foot, five nerves can be blocked at the level of the ankle (Figure 47-40). Depending on the injury, one or more of the nerves may be selectively blocked. To block

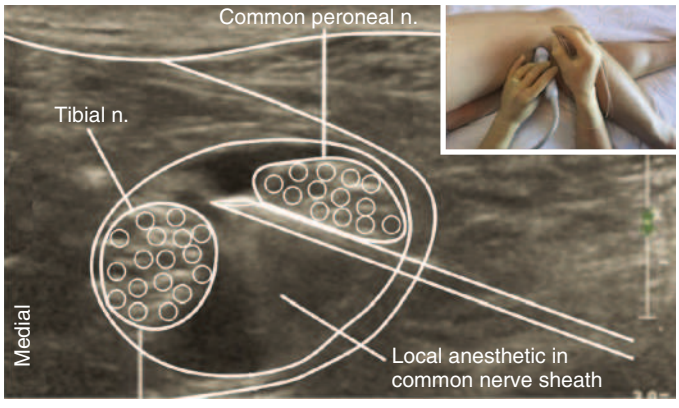


FIGURE 47-39 Ultrasound-guided popliteal nerve block. The most convenient positioning in the acutely injured patient is often lateral decubitus with the injured leg superior, as shown here. The bifurcation of the sciatic nerve is targeted using an in-plane, lateral-to-medial approach. Local anesthetic should be seen to flow around both the tibial and the peroneal component of the sciatic nerve.

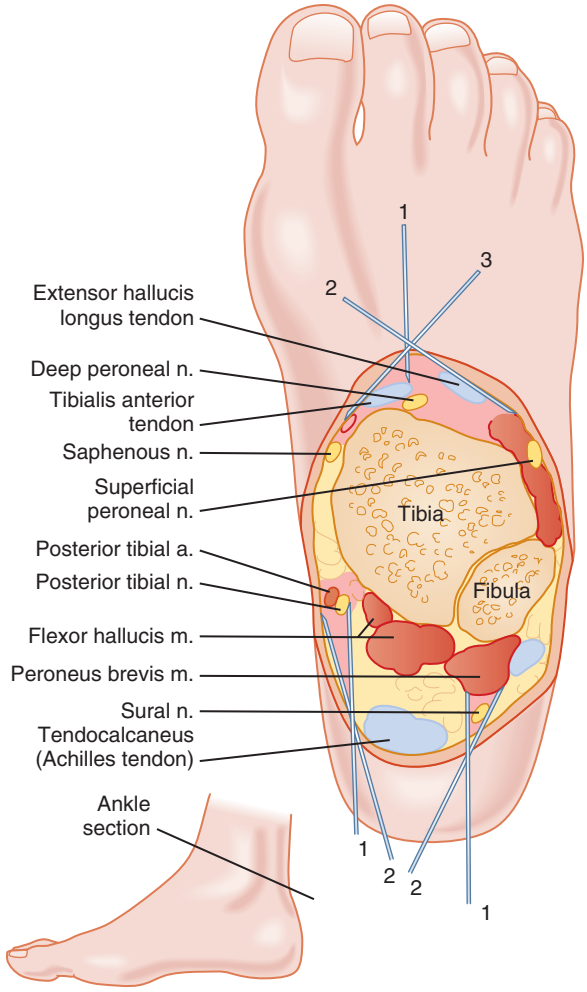


FIGURE 47-40 Landmark-based ankle block.

the posterior tibial nerve, a 22-gauge blunt-bevel needle is advanced at the level of the superior aspect of the medial malleolus from posterior to anterior, beginning just medial to the Achilles tendon. If a paresthesia is obtained, 5 mL of local anesthetic is injected. If a paresthesia is not elicited, the needle is advanced to the malleolus and local anesthetic injected. The sural nerve is blocked by entering the skin lateral to the Achilles tendon at the level of the superior aspect of the lateral malleolus. The needle is advanced from posterior to anterior until a paresthesia is obtained or the needle contacts the lateral malleolus, at which point 5 mL of local anesthetic is injected. The deep peroneal nerve is blocked by entering the skin lateral to the anterior tibial pulse, and then 5 mL of local anesthetic is injected. Field block of the superficial peroneal and saphenous nerves is performed by injecting 5 mL of local anesthetic medially and laterally from the point of entry for the deep peroneal nerve block down to the malleoli.[10,21]

Ultrasound-Guided Posterior Tibial Nerve Block

Anatomy and Indications. The posterior tibial nerve block provides excellent anesthesia for plantar foot injury or foreign body removal. The block also provides effective analgesia for calcaneal fracture. The posterior tibial nerve is a division of the sciatic nerve (L4-S3). Just above the ankle, the posterior tibial nerve passes posterior to the medial malleolus, typically just posterior or deep to the tibial artery (see Figure 47-17). The tibial nerve continues on to supply the skin on the sole of the foot and the majority of internal structures within the foot.

It is important to remember that the ankle joint has multiple innervations, necessitating both popliteal sciatic and saphenous nerve blocks for complete anesthesia; the posterior tibial nerve block alone will not suffice.[10,21,38]

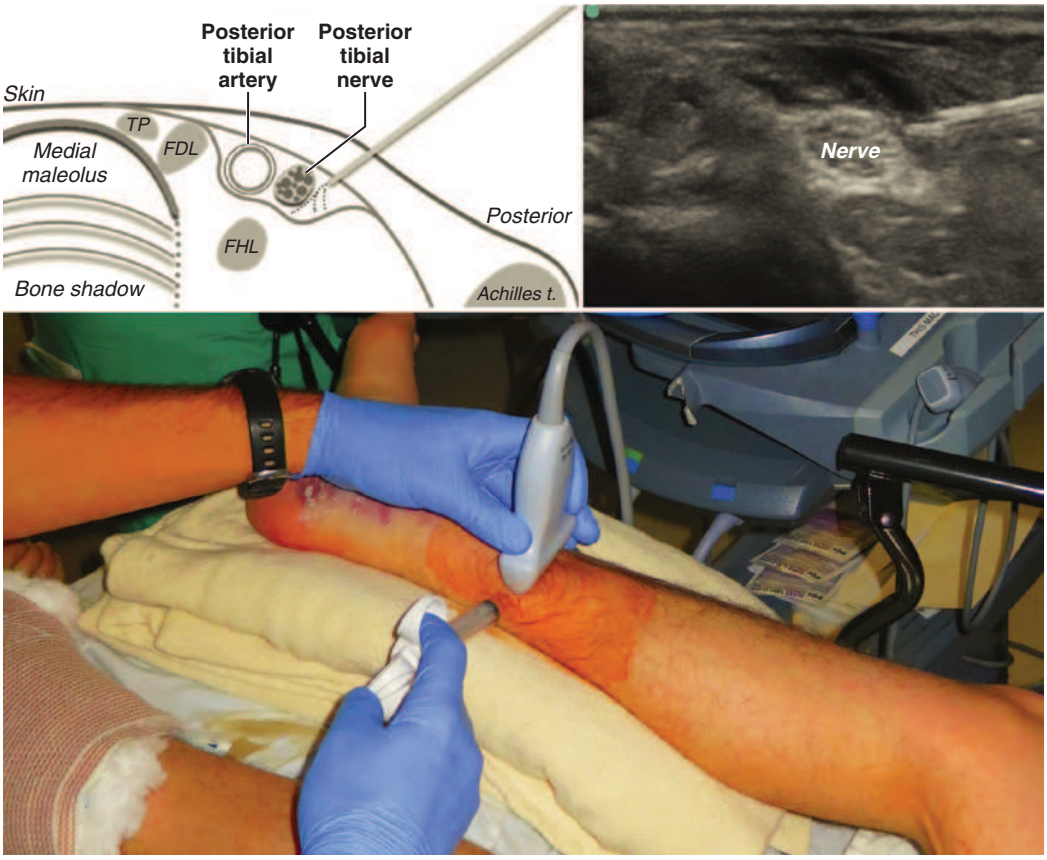


FIGURE 47-41 Ultrasound-guided posterior tibial nerve block. The posterior tibial nerve is easily blocked at the medial aspect of the ankle, where the nerve can be found as a hyperechoic, honeycomb structure just adjacent (most often posterior) to the posterior tibial artery. The flexor digitorum longus (*FDL*), flexor hallucis longus (*FHL*), and tibialis posterior (*TP*) tendons are adjacent structures that can have a nerve-like appearance on ultrasound. Tracing the structure in question proximally will clarify its identity; the tibial nerve can be followed proximally in the leg, while tendons quickly arrive at their muscle origins.

Positioning. The patient should be in a position of comfort that allows access to the medial and posterior aspect of the ankle.

Needle Approach. With the ultrasound system placed contralateral to the affected extremity, allowing an unobstructed line of sight, a linear transducer is used to scan just proximal and posterior to the medial malleolus in a transverse orientation. The tibial nerve will be seen as a hyperechoic structure adjacent to the pulsatile artery. This block can be performed using either an in-plane or an out-of-plane (perpendicular to the transducer axis) approach. The goal is to position the needle tip just adjacent to the tibial nerve without passing the needle into the nerve itself or puncturing the tibial artery (Figure 47-41).

Local Anesthetic. Depending on the clinical situation, 5 to 10 mL of a short- or long-acting local anesthetic is used. The local anesthetic should be observed to spread in real time around the nerve.

Potential Complications. The posterior tibial nerve block is relatively safe, with few potential complications. The proximity of the tibial artery mandates commonsense care when needling and injecting. Always aspirate before injection, visualize spread of local anesthetic in real time, and inject anesthetic in small aliquots of 3 to 5 mL.

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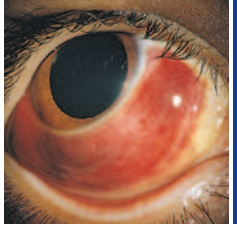

CHAPTER 48

The Eye in the Wilderness

FRANK K. BUTLER JR AND STEVEN CHALFIN

This chapter considers several commonly encountered types of eye disorders: periocular trauma; chemical injury to the eye; sudden vision loss in a white, quiet eye; acute orbital and periorbital inflammation; and the acute red eye. A diagnostic and therapeutic approach to these disorders that is suitable for the wilderness environment is presented. Additionally, eye problems that are encountered during diving and altitude exposures are discussed.

PRELIMINARY PLANNING

Pertinent ocular history items in a preliminary medical survey include contact lens wear; previous episodes of nontraumatic iritis; previous episodes of herpetic keratitis; and a history of corneal transplantation, retinal detachment, refractive surgery, or other ocular surgery. A positive response to these questions may alert the health care professional to specific ocular problems that may be encountered on the proposed trip. Congenital color vision deficiencies are common in the male population (8% in whites)[33] and typically pose no functional problems in the wilderness.

Ocular health should be reviewed as part of the medical survey, and a basic wilderness eye emergency kit should be assembled and taken on the trip.

THE WILDERNESS EYE EMERGENCY KIT

Box 48-1 lists the suggested items for a basic wilderness ocular emergency kit. A topical fluoroquinolone (e.g., moxifloxacin) is the antibiotic eye drop of choice. These medications are preferred for the treatment of bacterial keratitis in a wilderness setting. Topical tetracaine and fluorescein strips are important for diagnosis. Topical prednisolone is an excellent ocular antiinflammatory medication.

The choice of an oral antibiotic is based on the efficacy of the proposed antibiotic for the treatment of preseptal cellulitis, orbital cellulitis, and penetrating trauma to the globe. The fluoroquinolone family of antibiotics offers several good choices for these indications. The fourth-generation fluoroquinolone moxifloxacin provides good coverage against gram-positive, gram-negative, and anaerobic organisms, with a relatively mild side effect profile, excellent ocular penetration,[71] and once-daily dosing; it is the recommended first-line oral antibiotic.[100] Trovafloxacin (500-mg tablets) is a systemic fluoroquinolone with excellent coverage against gram-positive, gram-negative, and anaerobic bacteria.[62,138] It offers the convenience of once-daily dosing, but there have been a number of unpublished reports of hepatotoxicity with trovafloxacin that limit its usefulness.[106] Levofloxacin (500-mg tablets) is a systemic fluoroquinolone with very good activity against a wide variety of gram-positive and gram-negative organisms but less anaerobic coverage than with trovafloxacin.[106] Ciprofloxacin has been shown to have excellent ocular penetration when given orally,[82] but it is less efficacious against gram-positive organisms than is levofloxacin. Bacitracin is an antibiotic ointment that is suitable for use for patching corneal abrasions. Ophthalmic ointments are best applied with the use of downward pressure on the lower lid to pull it away from the eye, followed by the application of a 1-cm (0.4-inch) ribbon of ointment to the conjunctiva of the lower lid. When released, the lid returns to its normal position, and normal blinking distributes the ointment over the corneal surface. The use of ophthalmic ointments or eye drops is contraindicated in cases of ocular trauma where a ruptured globe is likely present.

Oral prednisone has at least three possible eye-related treatment uses in the wilderness: (1) severe uveitis; (2) giant cell arteritis; and (3) orbital pseudotumor. Topical scopolamine 0.25% is used to reduce ciliary muscle spasm, which causes much of the discomfort associated with iritis and corneal abrasion. However, scopolamine has the disadvantages of dilating the pupil, thus making the eye very sensitive to bright light, and preventing accommodation, thus making reading very difficult, for 5 to 7 days. Artificial tears are used to treat ocular surface drying and to flush conjunctival foreign bodies from the eye. Artificial tears are a better choice for dry eye symptoms than are ophthalmic preparations that contain vasoconstrictors, because the latter medications have been shown to cause both acute and chronic conjunctivitis.[130]

Diclofenac 0.1% drops have been shown to decrease corneal sensitivity, especially when multiple drops are used.[120,136,137] These drops have been found to be helpful for reducing the discomfort associated with traumatic corneal abrasion[68] and excimer laser refractive surgery.[47,139,142] In the unlikely event of angle-closure glaucoma in a wilderness setting, 2% pilocarpine may be used. The medications in Box 48-1 are listed in recommended priority order. In the spirit of "making do" in the wilderness, all the disorders mentioned in this chapter can be managed with only the aforementioned medications, but alternative therapies are also discussed.

Many individuals suffer from seasonal allergies, including allergic conjunctivitis, and these may be exacerbated in the wilderness. Adding olopatadine 0.1% or 0.2%, lodoxamide 0.1%, nedocromil 2%, or ketotifen 0.025% to the wilderness eye kit provides treatment options for allergic conjunctivitis.[77] Because allergic conjunctivitis is frequently chronic, individuals should carry their own medications on trips.

VISUAL ACUITY MEASUREMENT IN THE WILDERNESS

Evaluation of visual acuity is an essential element of the eye examination. Serial measurements of visual acuity are used to monitor an individual's progress while being treated for infectious, inflammatory, or traumatic eye disorders. Lack of an eye chart does not preclude the ability to obtain a quantitative measurement of visual acuity; rather, a near-vision card can be used for this purpose. Such a card should be held the prescribed distance from the eye; this is usually 35 cm (14 inches). If a near-vision card is not available, the ability to read print in a book is a useful alternative measure. If the individual's glasses have been lost, use a piece of paper with a pinhole that has been created with the tip of a pen or pencil to help to compensate for the lost refractive correction. Individuals age 40 and older may need a pinhole or reading correction to help them to focus on a near target. Although a marked decrease in visual acuity can be an important warning of a significant ocular disorder, visual acuity cannot always be considered a reliable indicator of the severity of disease. A person with corneal abrasion may initially have worse visual acuity than a person with a retinal detachment or corneal ulcer, despite the latter two entities being much more serious disorders.

BOX 48-1 The Wilderness Eye Emergency Kit

Medications	Miscellaneous
Moxifloxacin 0.5% drops	Penlight with blue filter
Tetracaine 0.5% drops	Fluorescein strips
Prednisolone 1% drops	Cotton-tipped applicators
Moxifloxacin 400-mg tabs	Metal eye shield
Levofloxacin 500-mg tabs	Tape (1 inch wide, plastic or nylon)
Bacitracin ointment	Near-vision card
Prednisone 20-mg tabs	Wound closure strips (¼ inch wide)
Artificial tears	Magnifying glass
Scopolamine 0.25% drops	Fine forceps
Diclofenac 0.1% drops	
Pilocarpine 2% drops	

GENERAL THERAPEUTIC APPROACH

The recommendations made in this chapter are not necessarily the preferred management of the disorders mentioned when one is not in the wilderness setting. Of special interest is the recommendation for an individual who is not an ophthalmologist to use a topical corticosteroid for the management of several of the disorders discussed. The use of topical ocular corticosteroids is generally best undertaken by ophthalmologists for two reasons. First, corticosteroids are usually indicated only for relatively serious ocular disorders, which should be followed by an ophthalmologist when possible. Second, topical steroid use may result in elevated intraocular pressure (IOP), cataracts, and exacerbation of certain eye infections. All the disorders for which corticosteroids are recommended in this chapter should be referred to an ophthalmologist for follow-up as soon as possible after the patient returns from the wilderness. Caution should be exercised when prescribing a topical corticosteroid for more than 3 days of use. Although cataracts are typically associated with long-term corticosteroid use, a significant rise in IOP may occur within just a few days after initiation of topical steroid therapy.[79] The use of topical corticosteroids in herpetic keratitis is strongly contraindicated, and may lead to corneal perforation.

The requirement for expedited evacuation is one of the questions that must be answered when treating an eye disease in the wilderness. In the following sections, the need for evacuation may be considered nonurgent unless an emergency (i.e., as soon as possible) or expedited (i.e., as soon as is deemed reasonable given the resources required to accomplish the task) evacuation is specified in the recommendations for treatment.

ACUTE PERIOCULAR INFLAMMATION

Box 48-2 lists causes of acute periocular inflammation. The term *preseptal cellulitis* means that the infectious process is confined to the tissues anterior to the orbital septum. Therefore, preseptal cellulitis manifests as erythema and edema of the eyelids without restricted ocular motility, proptosis, pupillary change, or decrease in visual acuity. However, some of these findings may be difficult to appreciate in the presence of marked lid edema. Historical clues include antecedent periocular trauma and insect bite or sting. *Staphylococcus aureus* and *Streptococcus* species are the most common causative organisms in adults.[40] In the past, preseptal cellulitis has been treated very aggressively because of the high incidence of *Haemophilus influenzae* infection, especially in pediatric patients, with subsequent septicemia and meningitis.

BOX 48-2 Differential Diagnosis of Acute Periocular Inflammation

Preseptal cellulitis
Orbital cellulitis
Dacryocystitis
Orbital pseudotumor
Insect envenomation

The advent of *H. influenzae* type B vaccine has changed the microbiology of this disorder and may dictate changes in treatment strategies in the future.[37] Persons who present with preseptal cellulitis may be treated with 400 mg of moxifloxacin once daily and should have an expedited evacuation. Alternative antibiotic choices include 500 mg of levofloxacin once a day, 750 mg of ciprofloxacin twice a day, 500 mg of dicloxacillin every 6 hours, or 500 mg of cephalexin four times a day.

Dacryocystitis, which is infection of the lacrimal sac, may mimic the findings of preseptal cellulitis; however, erythema, edema, and tenderness are localized to the area that is inferior to the medial aspect of the eye and over the nasolacrimal sac and duct. The presence of dacryocystitis usually indicates an obstruction in the opening between the lacrimal sac and the nasal cavity. Surgical intervention to restore the patency of this opening is usually undertaken after the acute infection is treated. The most common pathogens that cause acute dacryocystitis are *S. aureus*, *Streptococcus* species, and, in children, *H. influenzae*.[99] Treatment should be initiated with 400 mg of moxifloxacin once daily and warm compresses. Alternative antibiotic choices include 500 mg of levofloxacin once daily, 750 mg of ciprofloxacin twice daily, or 875 mg of amoxicillin and 125 mg of clavulanate every 8 hours. Worsening of the condition after 24 to 48 hours should be managed with an expedited evacuation.

Periocular insect envenomation is a preseptal cellulitis lookalike. Although secondary infection may follow envenomation, the envenomation itself may produce significant erythema and edema. Diagnostic clues include a history of insect bite or a periocular papular lesion at the site of the envenomation. Ice or cool compresses may be used to treat the envenomation, with 400 mg of moxifloxacin once daily, 500 mg of levofloxacin once daily, 500 mg of dicloxacillin every 6 hours, or 500 mg of cephalexin four times daily added if secondary infection is suspected.

The term *orbital cellulitis* means that infection has spread to or originated in the tissues posterior to the orbital septum. This may manifest as diplopia or restriction in ocular motility as the extraocular muscles are affected, proptosis as edema in the orbit pushes the globe forward, decreased vision as the optic nerve is affected, or pupillary change if innervation of the pupil is affected. Fever suggests orbital cellulitis as part of the differential diagnosis of periocular inflammation. Orbital cellulitis is more often associated with sinusitis than with periocular trauma as an antecedent disorder.[84] Most series report a 50% to 75% incidence of sinusitis or other upper respiratory infection in association with orbital cellulitis.[144] The bacteria that most frequently cause orbital cellulitis are *S. aureus*, *Streptococcus pyogenes*, and *Streptococcus pneumoniae*. Anaerobes are frequently present in patients with chronic sinusitis and should be suspected in those with orbital cellulitis that is associated with long-standing sinus disease. If not treated aggressively, orbital cellulitis may be associated with a life-threatening infection of the central nervous system (CNS). Before antibiotics became available, approximately 19% of persons with orbital cellulitis died of intracranial complications, and 20% of survivors became blind in the involved eye.[99] This disorder requires hospitalization and intravenous (IV) antibiotic therapy. Interim therapy should include 400 mg of moxifloxacin once daily. Alternative antibiotic choices are 500 mg of levofloxacin twice daily, 750 mg of ciprofloxacin twice daily, and 875 mg of amoxicillin and 125 mg of clavulanate every 8 hours. The combination of ciprofloxacin and clindamycin as an empirical treatment may be as effective as IV therapy.[27] A decongestant should be added if sinusitis is present, and emergent evacuation should be undertaken.

Orbital pseudotumor is an inflammatory disease of the orbit that may manifest very similar to orbital cellulitis. The differentiation between these two entities may be difficult.[84] The patient with orbital pseudotumor would typically not have a history of preceding sinusitis. A reasonable approach in the wilderness is to begin therapy with 400 mg of moxifloxacin once daily and then to arrange for emergent evacuation. Prednisone (1 mg/kg/day) should be added if there is no response to antibiotic therapy, if there is no fever or sign of CNS involvement, or if evacuation has not been possible by 24 to 48 hours after presentation. If

prednisone therapy is initiated, its efficacy should be evaluated after 48 hours. If there has been a decrease in pain, erythema, edema, or proptosis, therapy should be continued until evacuation is accomplished. If the patient has shown no response after 48 hours, the prednisone may be discontinued without tapering.

PERIOCULAR TRAUMA

EYELID LACERATION

The most important aspect of managing an eyelid laceration is carefully to exclude the presence of penetrating injury to the globe. Clues to the presence of an open globe are noted later (see Obvious Open Globe and Occult Ruptured Globe).

A lid laceration that is horizontally oriented on the eyelid, that does not penetrate the full thickness of the lid, and that does not involve the lid margin is relatively easily managed. In the absence of an ability to irrigate, disinfect, and suture the laceration properly, it should be managed by irrigation with the cleanest disinfected water available, application of topical antibiotic drops (e.g., ciprofloxacin, ofloxacin, tobramycin) to the laceration, drying the surrounding skin surface, and closing the laceration with tape strips. The wound may then be treated with antibiotic ointment (e.g., bacitracin, erythromycin) four times daily for 3 to 4 days. Alternatively, the laceration may be left open, treated with antibiotic ointment four times daily, and repaired 1 or 2 days later.[84] The laceration should be observed frequently while it is healing. If redness or discharge develops, the patient should be started on 400 mg of moxifloxacin once daily, 500 mg of levofloxacin once daily, or 500 mg of dicloxacillin every 6 hours. The wound closure tape should be removed from the laceration, and evacuation should be expedited, especially if the response to oral antibiotics is poor.

Complicated lid lacerations, which are defined as *stellate* or *complex,* and those that involve the lid margin or the canthi (i.e., medial and lateral ends of palpebral fissure) are more difficult to manage. These lacerations may result in secondary functional difficulties if ocular lubrication or lacrimal drainage becomes impaired. In addition, cosmesis may be poor if a meticulous repair is not done. These wounds should be managed by irrigation with the cleanest disinfected water available, application of antibiotic ointment, and coverage with a sterile dressing. Evacuation for definitive plastic surgery repair should be expedited.

CORNEAL FROSTBITE

Corneal frostbite is an uncommon disease, but it has been reported during such activities as snowmobiling without protective eye goggles when participants keep their eyes open in conditions of high windchill factor.[6] Symptoms and treatment are the same as those described under Corneal Abrasion, later.

INSTILLATION OF ADHESIVE DROPS INTO THE EYE

Inadvertent instillation of a "superglue" compound (i.e., a cyanoacrylate-type adhesive) into the eye may bind the lids tightly together. Overnight application of a pressure patch with eye pads presoaked with water has been reported to allow manual separation of the lids and to eliminate the need for general anesthetic and surgical separation of the lids.[110] Ophthalmic ointment inserted through any small opening in the adherent lids has also been reported to facilitate resolution.[94] Acetone, an ingredient in nail polish remover, is a solvent for cyanoacrylate and if available, may be used to soften the adhesive. After the lids are separated, the eye should be checked for corneal abrasion.[124]

RETROBULBAR HEMORRHAGE

Blunt or penetrating periocular trauma may result in orbital bleeding. If the bleeding is contained by the orbital septum, an orbital compartment syndrome may ensue. Because the pressure in the orbital compartment is progressively elevated, IOP will also rise. If IOP rises to a sufficiently high level, either central retinal artery occlusion or damage to the optic nerve may ensue, and vision may be permanently lost in the eye. Signs and symptoms of retrobulbar hemorrhage include pain, periorbital ecchymosis, progressive proptosis (i.e., bulging forward of the eye), decreased vision, diffuse subconjunctival hemorrhage, and an afferent pupillary defect. Definitive management of this disorder is a minor surgical procedure called *lateral canthotomy,* in which an incision is made at the lateral aspect of the eyelid to relieve pressure in the orbit (Figure 48-1).

CHEMICAL INJURY OF THE EYE

The mainstay of management of any chemical eye injury is immediate and copious irrigation of the ocular surface with water from whatever source is most readily available. Lactated Ringer's solution or normal saline is the preferred irrigation fluid, when available;[40] in the wilderness, however, bottled water may be the best option. If none of these fluids is available, treated (i.e., filtered and disinfected) water from a drinking container is the next best option, with untreated water being the last resort. Instillation of several drops of tetracaine will make the procedure much less uncomfortable for the patient. Irrigation should be continued for a minimum of 30 minutes.[84] The two most damaging chemicals are strong acids and alkalis.[84] Sulfuric acid from an exploding car battery is a typical acid, whereas cleaning products (e.g., drain cleaners) are typical alkalis. Pyrotechnic residues are frequently strongly alkaline. Caustic alkalis are more likely to damage the eye than are acids because of their profound and rapid ocular penetration. Do not attempt to neutralize the corneal surface with acidic or alkaline solutions. Chemicals other than acids and alkalis may be uncomfortable when encountered, but they are less likely to produce significant long-term damage.

After a minimum of 30 minutes of flushing has been completed, the eye should be examined for retained particles; these should be removed with a moistened cotton-tipped applicator.[84] Treatment for an acid- or alkali-induced injury includes moxifloxacin 0.5% drops (1 drop four times daily) or bacitracin ointment every 1 to 2 hours while awake until fluorescein staining confirms that the corneal epithelial defect that typically accompanies these injuries has resolved. Topical prednisolone 1% should be added if there is significant inflammation. Prednisolone drops should be used every hour while awake for 3 days. Eye pain is managed with scopolamine drops four times daily for 3 days and oral pain medications.[122] Frequent use of artificial tears may also help to relieve discomfort. Evacuation should be expedited if the cornea is found to be opaque, if a large epithelial defect is found on fluorescein staining, or if significant pain persists after 3 days. Another sign of serious injury is blanching of the conjunctiva in the limbal area.[84]

COBRA VENOM

A special type of chemical injury to the eye is ocular envenomation from a spitting cobra.[52,57,141] Ocular injury from cobra venom may be severe and result in blindness. Cobras can project this venom out to a range of 3 m (10 feet), and they typically aim directly at the victim's eyes. Ocular surface envenomation causes severe conjunctival and corneal inflammation, edema, and erosions. Corneal erosions may progress to corneal ulceration, perforation, hypopyon, and endophthalmitis. Central corneal opacification may result despite aggressive therapy. One report described nine individuals who had sustained ocular surface cobra envenomation.[141] Five had only severe conjunctivitis; corneal erosions developed in the other four. Two individuals were permanently blinded as a result.

Therapy includes immediate flushing of the eyes as described previously. After this is done, check for a corneal abrasion, and treat it with moxifloxacin 0.5% drops if an erosion is present (1 drop four times daily until erosion healed). Note that healing may be prolonged in toxin-induced corneal surface injuries. Immediate consultation with a toxicology consult service and a corneal or external disease subspecialist should be obtained, if possible, and

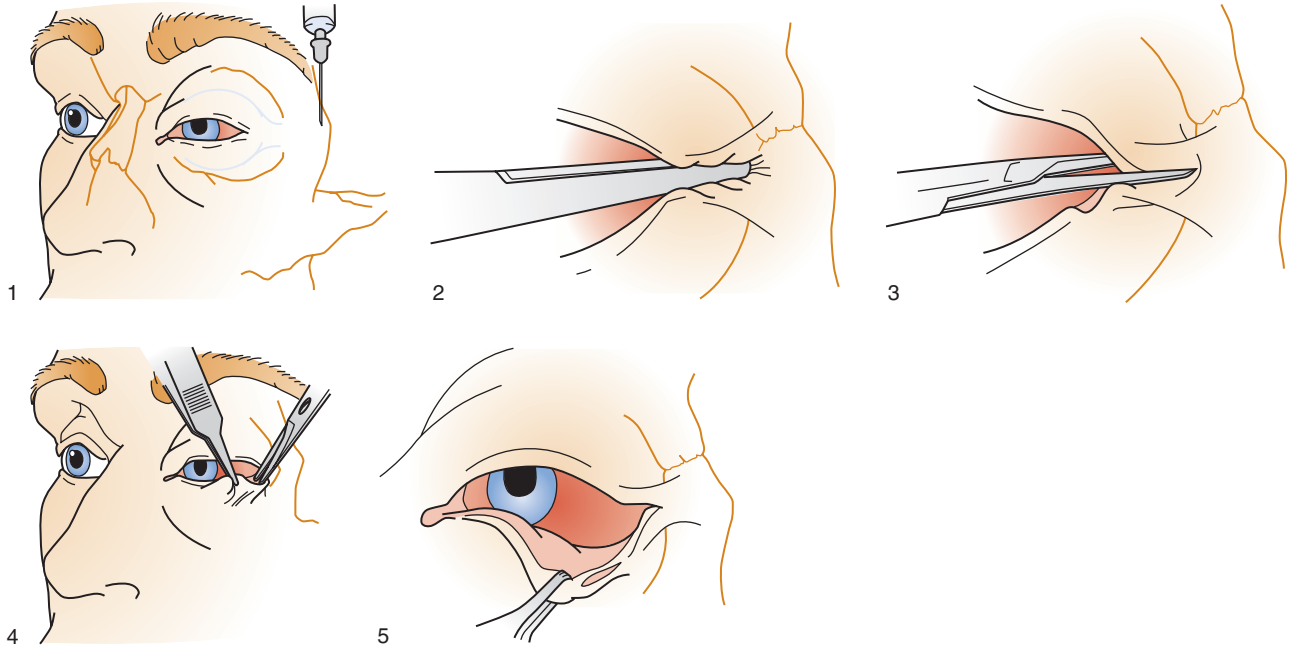

FIGURE 48-1 Lateral canthotomy and inferior cantholysis are indicated for casualties that manifest with orbital hemorrhage and evolving orbital compartment syndrome. *Step 1,* Infiltrate the lateral canthal area with a local anesthetic (e.g., 2% lidocaine with epinephrine) and use tetracaine drops in the eye for topical anesthesia. *Step 2,* Place a mosquito clamp on the lateral canthus that extends horizontally toward the orbital rim for a distance of 1 cm (0.4 inch). Leave it in place for 30 seconds to assist with hemostasis. *Step 3,* Remove the clamp, and use a pair of fine (e.g., Stevens) scissors to divide the lateral canthal tissues along the line created by the clamp. This completes the lateral canthotomy. *Step 4,* Next, use the scissors to cut the inferior crus of the lateral canthal ligament. Position the scissors perpendicular to the canthotomy incision that was made in Step 3 (i.e., not along the lid margin), and cut the ligament. This completes the inferior lateral cantholysis. *Step 5,* The completed lateral canthotomy and cantholysis are shown. These procedures allow the orbital tissues to move forward slightly, and help to relieve the pressure in the orbital compartment.

an expedited evacuation should be arranged. Topical corticosteroid therapy should be considered (1 drop of prednisolone every hour while awake) and discussed with the consultant. The decision to treat is based on the severity of symptoms and the presence or absence of a corneal erosion or ulcer. The value of the topical application of specific cobra antivenom has not been documented, so this therapy is not recommended. Systemic heparin therapy has been shown to be useful for prevention of corneal opacification in animal models of this injury.[65,66]

SKUNK MUSK

Another type of chemical injury of the eye may occur when the eyes are sprayed with skunk musk. Such an episode should be managed with immediate ocular irrigation as described previously. If discomfort persists beyond several hours, check for a corneal abrasion, and treat this with moxifloxacin 0.5% drops (1 drop four times daily until abrasion is healed). If fluorescein staining does not show an abrasion but significant discomfort persists, a 3-day course of prednisolone 1% drops (1 drop four times daily for 3 days or until symptoms have resolved) may be of benefit. If pain is severe and unrelieved by prednisolone drops, or if it persists after the 3-day course has been completed, consider an expedited evacuation and an ophthalmologic consultation.

ACUTE LOSS OF VISION IN A WHITE, QUIET EYE

Vision may be variably decreased with many of the disease entities discussed later (see Acute Red Eye). This section addresses sudden loss of vision that occurs in a white, quiet eye. Box 48-3 provides a differential diagnosis. Disorders that cause this symptom are often difficult to diagnose without ophthalmic instruments, none of which is included in the kit described in Box 48-1. There are few treatments for most of these disorders that are likely to be effective in a wilderness setting. Although an afferent pupillary defect (e.g., Marcus Gunn pupil) may be present, this is a nonspecific finding that would be expected with most of the disorders listed in Box 48-3, except for vitreous hemorrhage and high-altitude retinal hemorrhage.

An important question must be asked with acute loss of vision in a white, quiet eye: Does this person have giant cell arteritis?" Also called *temporal arteritis*, giant cell arteritis (GCA) can cause devastating anterior ischemic optic neuropathy that is often first noted on waking and that usually becomes permanent. Subsequent involvement of the second eye is common if GCA in the first-stricken eye is not promptly treated.[50] Although visual loss has been reported to occur in both eyes simultaneously, there is typically a delay of 1 to 14 days before the second eye is affected.[1] Loss of vision in the second eye can be prevented in most cases by prompt initiation of high-dose corticosteroid therapy.[1,50,60,105]

Arteritic anterior ischemic optic neuropathy is typically a disease of older individuals, with one large study reporting a mean age of onset of 70 and age of the youngest patient as 53.[1] Clues to diagnosis are temporal headache, jaw claudication, fever, weight loss, transient visual obscurations, and polymyalgia rheumatica (i.e., generalized myalgias).[105] The visual obscurations seen with GCA usually last for 2 to 3 minutes.[105] The person

BOX 48-3 Differential Diagnosis of Acute Loss of Vision in a White, Quiet Eye

Retinal detachment
Central retinal artery occlusion (CRAO)
Nonarteritic anterior ischemic optic neuropathy
Optic neuritis
Central retinal vein occlusion
Arteritic anterior ischemic optic neuropathy
Vitreous hemorrhage
High-altitude retinal hemorrhage (HARH)

thought to have GCA should be started on 80 mg of prednisone once daily, and evacuation should be expedited. If prednisone or corticosteroids of equal or greater systemic potency are not available, the evacuation should be considered an emergency.

If one suspects that loss of vision is the result of *retinal detachment* on the basis of a history of high myopia (i.e., extreme nearsightedness), floaters, or photopsias (i.e., flashing lights), expedited evacuation should be undertaken because of the need for surgical repair. Loss of central vision caused by a retinal detachment usually means that the macula is involved and that surgical repair is urgent rather than emergent. Ross and Kozy[117] found that a delay to surgery of up to 1 week in macula-off rhegmatogenous retinal detachments did not affect final visual acuity. Expedited evacuation to a facility that has retinal surgery capability allows for more precise determination of the urgency for surgical repair. The presence of "floaters" in the field of vision is a very common occurrence among individuals who are middle aged or older. An acute awareness of floaters may be caused by certain activities, such as looking at a visually homogeneous background (e.g., the sky). If the floaters are new in onset or suddenly more numerous than before, or if they are accompanied by a sensation of flashing lights (i.e., photopsias), this suggests the possibility of *posterior vitreous detachment* (PVD), which is detachment of the vitreous face from the retina. PVDs are common and usually not a problem, except for the annoying floaters; however, the suspicion of PVD should prompt a visit to the ophthalmologist as soon as practical to ensure that the PVD has not caused a retinal tear or detachment. Expedited evacuation from the wilderness environment is not required for floaters alone.

If the patient is at a high altitude (>3048 m [10,000 feet]), high-altitude retinal hemorrhage, discussed later, should be suspected; further ascent should be avoided. Descent of at least 915 m (3000 feet) should be undertaken as soon as feasible.[18]

Another potentially treatable cause of sudden loss of vision in a white, quiet eye is *central retinal artery occlusion* (CRAO). Previous conventional therapy for CRAO of ocular massage, pentoxifylline, and anterior chamber paracentesis has been reported to be unsuccessful for restoring the vision in 40 of 41 patients with CRAO, even though 11 patients presented within 6 hours of visual loss and 17 patients presented within 12 hours.[118] Primate retinas can tolerate no more than 100 minutes of ischemia caused by complete blockage of retinal blood flow.[59] However, fluorescein angiography has shown that in humans, CRAO is seldom complete, and therapy begun up to 6 hours after visual loss may be successful for restoring vision.[118]

Ocular massage and oxygen may be the best options for therapy in the wilderness setting. Ocular massage lowers IOP, increasing perfusion pressure to the retina. Additionally, massage may contribute to mechanical breakup of emboli, allowing them to move downstream in the retinal arterial circulation.

Hyperbaric oxygen was reported to be successful for restoring vision on two separate occasions in one patient with recurrent branch retinal artery occlusions that were associated with Susac syndrome.[83] Oxygen is supplied to the retina from both the retinal and the choroidal circulation. Under normoxic conditions, approximately 60% of the retina's oxygen is supplied by the choroidal circulation. Under hyperoxic conditions, the choroid is capable of supplying 100% of the oxygen required by the retina.[83] When retinal arterial flow is interrupted, the retinal tissue undergoes a period of ischemia. Blood flow may be spontaneously reestablished, which frequently happens with arterial obstruction, or ischemia may continue until cell death and necrosis occur.[88] There is period during which the tissue is still ischemic yet capable of recovery. Hyperbaric oxygen is not always required for reversal of retinal ischemia. One of the authors (FKB) has treated a monocular patient who suffered CRAO in his only seeing eye and presented to the emergency department within an hour of visual loss. The patient's vision improved from 20/400 to 20/25 within minutes after he received supplemental oxygen by reservoir mask. Unfortunately, however, his vision decreased rapidly to 20/400 whenever the supplemental oxygen was removed. The patient was heparinized and maintained on supplemental oxygen for approximately 10 hours, at which time the removal of oxygen no longer caused a decrease in vision. If oxygen is being carried for an extreme-altitude summit attempt or for other purposes, a person with sudden painless loss of vision should be given a trial of oxygen administered in as high a concentration as possible to see if this therapy results in visual improvement. CRAO has been approved by the Undersea and Hyperbaric Medical Society as an indication for hyperbaric oxygen therapy (HBOT).[95] Other ocular indications for HBOT have been summarized in a review article[23] and textbook chapter.[25] Ocular contraindications to and complications of HBOT have also been recently summarized.[22]

Care should be taken when monocular visual loss occurs. Although central vision may still be normal in the fellow eye, depth perception may be impaired, and the person may therefore be at increased risk of a fall during self-evacuation.

ACUTE RED EYE

Box 48-4 provides a partial list of disorders that can result in an acute red eye. In the absence of a slit lamp, the diagnosis must rely on the basic techniques of history, penlight inspection, fluorescein staining, response to administration of topical anesthesia, and pupillary status. The following discussion of the differential diagnosis of the acute red eye uses these clinical findings to establish the diagnosis. Figure 48-2 shows an algorithmic representation. The term *fluorescein positive* is used to denote an eye with a discrete area of staining noted with cobalt blue light after instillation of fluorescein dye. Some conditions, such as blepharitis, viral keratoconjunctivitis, and ultraviolet (UV) keratitis, may cause a pattern of punctate staining that is referred to as *superficial punctate keratitis* (SPK).

TRAUMATIC OCULAR DISORDERS

Obvious Open Globe

If there is a history of trauma and if penlight inspection of the eye reveals an obvious open globe (e.g., the eye in Figure 48-3), the examination should be discontinued and a protective rigid shield placed over the eye. Do *not* apply a pressure patch or instill any topical medication. There are two primary goals during management of this condition. The first is to minimize manipulation or additional trauma to the eye that might raise IOP and result in expulsion of intraocular contents through the corneal or scleral defect. The second is to prevent development of posttraumatic endophthalmitis, which is an infection of the aqueous and vitreous humors of the eye. This typically has devastating visual results, with only 30% of patients in one study retaining visual acuity of 20/400 or greater.[78] *Staphylococcus epidermidis* is the most common pathogen implicated in obvious open globe, but *Bacillus cereus* is an extremely aggressive pathogen that also is often isolated. After the shield is placed, the patient should receive 400 mg of moxifloxacin or 500 mg of levofloxacin once daily; 750 mg of ciprofloxacin twice daily is another option. Penetration of antibiotic into the vitreous cavity is a major concern with penetrating eye trauma, so other antibiotics should not be substituted unless known to have adequate vitreal penetration.

A patient with an obvious open globe requires surgical repair as soon as possible and should be evacuated as an emergency. Because air may have been introduced into the eye, barometric pressure changes during evacuation should be minimized, if

BOX 48-4 Differential Diagnosis of the Acute Red Eye

- Obvious open globe
- Corneal abrasion
- Corneal ulcer
- Subconjunctival hemorrhage
- Traumatic iritis
- Hyphema
- Occult open globe
- Herpes simplex virus (HSV) keratitis
- Corneal erosion
- Acute angle-closure glaucoma
- Iritis
- Scleritis
- Conjunctivitis
- Blepharitis
- Ultraviolet (UV) keratitis
- Episcleritis
- Conjunctival foreign body
- Dry eye
- Contact lens overwear syndrome

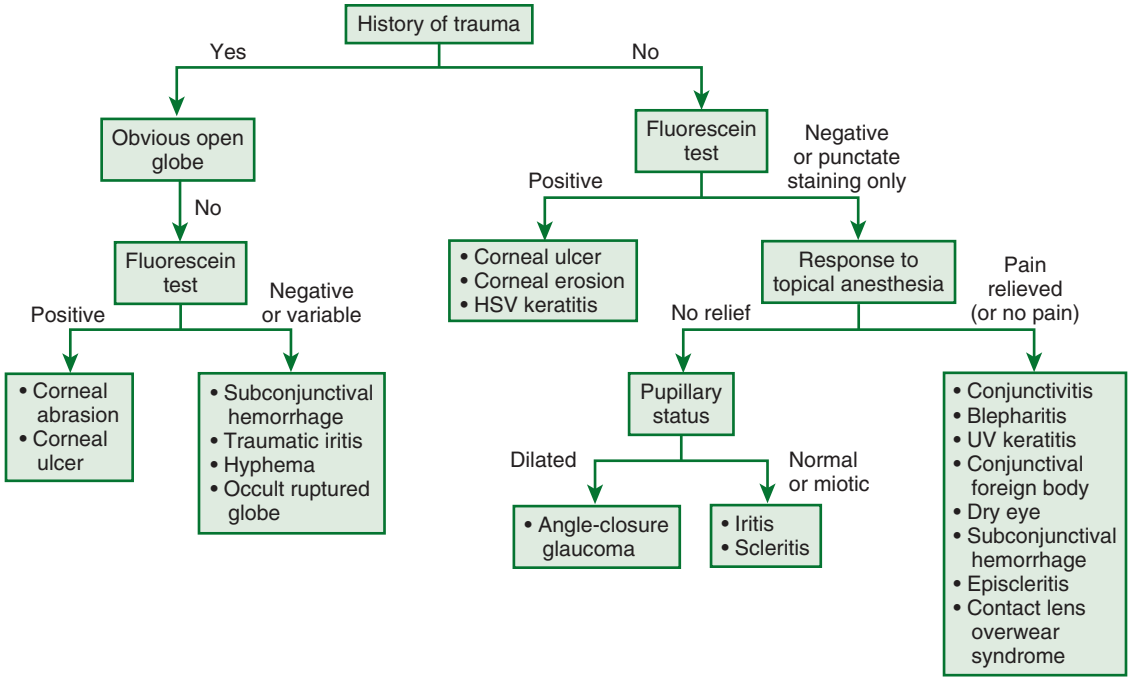

FIGURE 48-2 Algorithm that shows the wilderness diagnostic procedure for the acute red eye. *HSV,* Herpes simplex virus; *UV,* ultraviolet.

possible. However, this consideration is secondary to the need for expeditious transport to a facility where surgical repair can be performed. Emesis should be treated or prevented if antiemetic medications are available. The need for surgical intervention shortly after arrival at the hospital should be anticipated. The patient should not be allowed to become dehydrated as a result of a prolonged evacuation time from the wilderness setting, so plain water by mouth may be allowed up to several hours before arrival at the hospital.

Occult Ruptured Globe

A penetrating injury to the eye or a ruptured globe may not always be obvious. Clues to occult rupture include large subconjunctival hemorrhage with chemosis; dark uveal tissue present at the limbus; distorted pupil (Figure 48-4); aqueous humor seen as a clear-flowing motion in the fluorescein-stained tear film from a linear or punctate corneal epithelial defect; mechanism of injury (e.g., hammering metal on metal, impaling injury); and a decrease in vision. If an occult globe rupture is suspected, the patient should be treated as just described for an obvious open globe. The relatively less severe appearance of the injury does not eliminate the threat of endophthalmitis, so systemic antibiotic therapy as noted previously should be initiated.

Snakebite injuries to the eye are fortunately uncommon but may result in occult open globe injury as a result of penetration by teeth or fangs.[5] If the snake is venomous, direct injection of toxin into the globe has been reported to cause necrosis, uveitis, and keratomalacia.

Another type of penetrating ocular injury particular to the wilderness environment is injury to the eye from a fishhook that has become embedded in the globe or in the periocular tissues. Principles of management include protecting the eye from further injury by covering it with a shield, avoiding manipulation of the hook that may worsen known or suspected injury to the globe, beginning systemic antibiotics as soon as possible, and expediting evacuation of the patient to the care of an ophthalmologist.

Corneal Abrasion

A corneal abrasion is disruption of the protective epithelial covering of the cornea (Figure 48-5). This results in intense pain, tearing, light sensitivity (i.e., photophobia), and increased susceptibility to infection until the defect has healed, usually in 2 to 3 days. There is typically a history of antecedent trauma or contact lens wear. The sine qua non for this diagnosis is an epithelial defect on fluorescein staining.

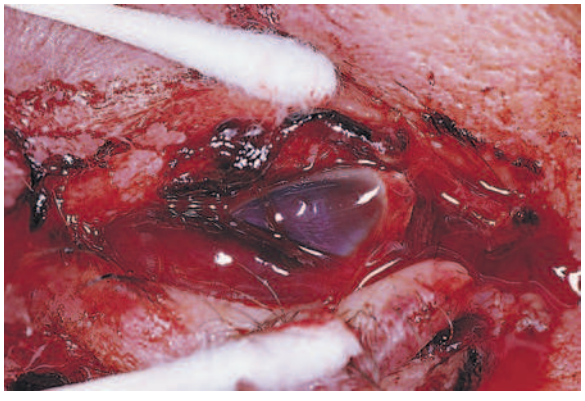

FIGURE 48-3 Obvious open globe (i.e., corneoscleral laceration). *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

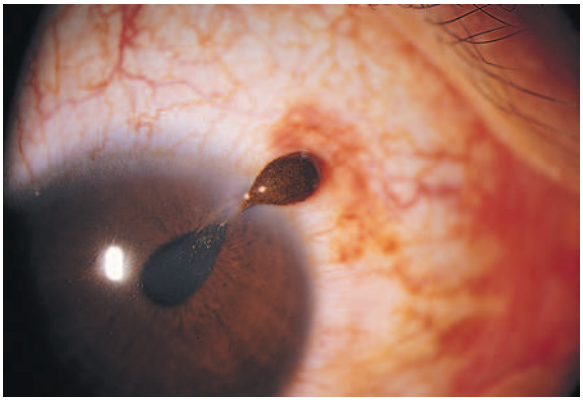

FIGURE 48-4 Occult open globe with uveal pigment at the limbus and a peaked pupil. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

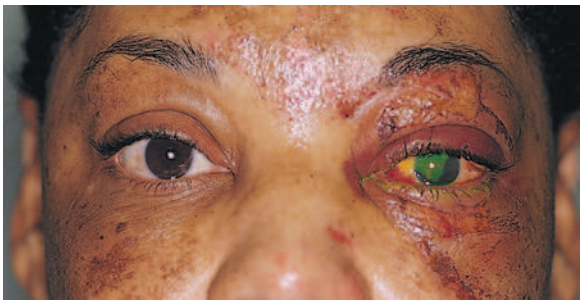

FIGURE 48-5 Corneal abrasion. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

Standard treatment of corneal abrasion in the recent past consisted of antibiotic ointment followed by application of a pressure patch. A study showed that small (<10 mm^2) and noninfected abrasions that were not related to contact lens wear healed significantly faster with less discomfort when they were not patched.[70] Standard texts now note that patching is rarely used.[40] In a wilderness setting, the nonpatching option has the additional advantages of not rendering the patient completely monocular and not adversely affecting visual field and depth perception. If the nonpatching option is chosen, the patient should be treated with moxifloxacin 0.5% drops (1 drop four times daily) or bacitracin ointment four times daily until the corneal epithelium is healed. Diclofenac drops four times daily may be helpful in reducing discomfort. Sunglasses may help to alleviate photophobia. Repeated use of a topical anesthetic for pain control is contraindicated. If the abrasion is contact-lens related, the eye should not be patched because of the increased risk of corneal ulcer that is present with a contact-lens–related abrasion.[112] Contact-lens–related corneal abrasions should be treated with moxifloxacin 0.5% drops (1 drop every 2 hours while awake) until the epithelial defect has resolved. An abrasion associated with vegetable matter also should not be patched.[112] If the abrasion is large or if the patient's discomfort is severe, scopolamine drops once or twice daily may be added to the antibiotic (wait 5 minutes between each drop). Much of the pain associated with corneal abrasion and ulcer is the result of ciliary muscle spasm, which is relieved by scopolamine. The rationale for using scopolamine only with a very painful abrasion in the wilderness is that this medication will cause the pupil to dilate (and the eye to become very sensitive to light) and lose accommodation (with a resultant decrease in near visual acuity) for approximately 5 to 7 days. Contact lenses should not be worn until the abrasion has resolved.[40]

An oral analgesic may be required for pain control. The patient should be monitored daily for development of a corneal ulcer, which is noted on penlight examination as a white or gray infiltrate on the cornea, and for progress in healing of the epithelium, as measured by the resolution of fluorescein staining.

Corneal Ulcer

The term *corneal ulcer,* as used in this chapter, denotes acute bacterial, fungal, or protozoal infection of the cornea (Figure 48-6). Chronic corneal epithelial defects caused by a variety of autoimmune or inflammatory processes are also sometimes called a corneal ulcer, but these disorders are beyond the scope of this chapter.

Although a corneal ulcer is an infectious process, it is often preceded by a traumatic corneal abrasion. The other predisposing condition for a corneal ulcer is contact lens wear, which results in microtrauma to the corneal epithelium and may allow bacteria or other microorganisms to infect the cornea. Corneal ulcers typically are significantly painful. A small, white or gray infiltrate on the cornea can be appreciated by a careful penlight examination. If it is not treated aggressively, the small initial lesion may progress to a much larger infiltrate (Figure 48-6), with a correspondingly more severe impact on visual acuity. Fluorescein staining reveals an epithelial defect overlying the infiltrate. The associated pain is usually significantly decreased by applying a topical anesthetic, but ciliary spasm may cause pain relief to be incomplete.

An inadequately treated corneal ulcer may result in visual loss from dense corneal scarring or ocular perforation with subsequent endophthalmitis. Management of this disorder in the past included hospital admission for treatment with concentrated and frequently administered aminoglycoside and cephalosporin topical drops. Outpatient therapy with fourth-generation fluoroquinolone eye drops has been shown to be comparable in efficacy to fortified antibiotic preparations.[64,101] Thus, treatment for corneal ulcer should be with moxifloxacin 0.5% drops, 1 drop every 5 minutes for five doses initially, then 1 drop every 30 minutes for 6 hours, and then 1 drop every hour thereafter around the clock.[112] One drop of scopolamine given two to four times daily may help to relieve the discomfort caused by ciliary spasm. Repeated use of topical anesthetics for pain control is contraindicated. Moxifloxacin (400 mg by mouth once daily) may be added if evacuation is delayed and either the infiltrate or the patient's pain is becoming worse.[40] Systemic analgesia may be required if pain is severe. Expedited evacuation and immediate evaluation by an ophthalmologist are recommended.

Traumatic Iritis

The term *iritis* refers to inflammation of the iris or, more accurately, of the anterior uveal tract of the eye; it may also be called *anterior uveitis* or *iridocyclitis*. The defining feature of this condition is inflammatory cells in the anterior chamber of the eye, which must be visualized with a slit lamp. In the wilderness, this diagnosis must be presumptive. Iritis may accompany a corneal abrasion, may result from blunt trauma, or may be idiopathic The diagnosis rests primarily on significant posttraumatic pain, without a corneal abrasion or ulcer noted on fluorescein staining, or pain that persists after the abrasion is healed. Initial treatment is with 1 drop of prednisolone four times daily for 3 days. One drop of scopolamine twice daily may be added if pain is severe enough to justify the blurred vision that results from this therapy. Expedited evacuation should be undertaken if severe pain persists.

Subconjunctival Hemorrhage

Subconjunctival hemorrhage is a bright-red area over the sclera of the eye that results from bleeding between the conjunctiva and the sclera (Figure 48-7). It is easily visible without the use of a slit lamp. There is often an antecedent history of minor trauma to the eye, coughing, sneezing, or Valsalva event. This injury is innocuous and resolves over several days to several weeks without treatment. In the presence of antecedent trauma, one should be alert for another, more serious injury. In particular, if hemorrhage results in massive swelling of the conjunctiva (i.e., chemosis), an occult globe rupture should be suspected.

Hyphema

The term *hyphema* is defined as blood in the anterior chamber. Although this is usually seen in the setting of acute trauma,

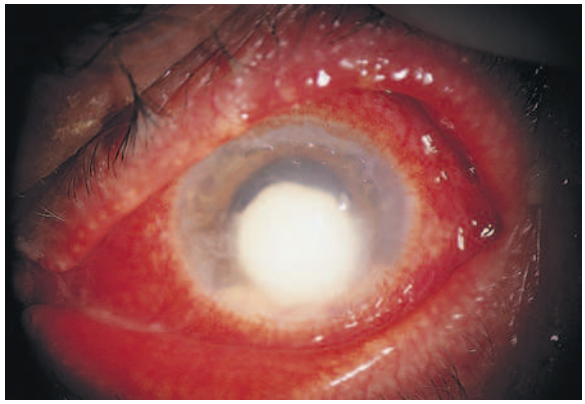

FIGURE 48-6 Corneal ulcer. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

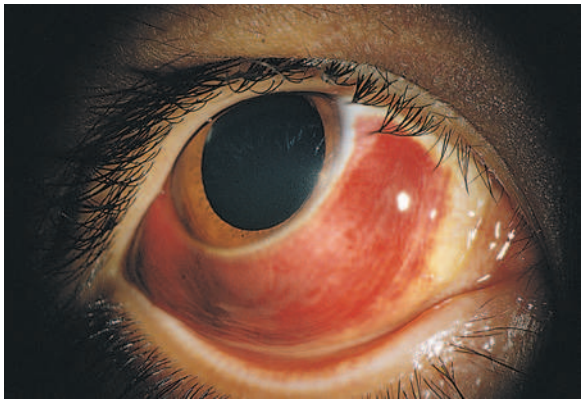

FIGURE 48-7 Subconjunctival hemorrhage. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

it may also be caused by other conditions, such as iris neovascularization. The eye should be examined with the patient sitting upright. If enough blood is present, it collects at the bottom of the anterior chamber and is visible as a layered hyphema (Figure 48-8). This may not be appreciated if the patient is examined while in a supine position or if the amount of blood is minimal.

Although most hyphemas resolve without sequelae, this disorder may be complicated by an acute rise in IOP or corneal blood staining. Treatment in the wilderness consists of activity restriction (i.e., walking only), prednisolone drops four times daily, avoidance of aspirin or nonsteroidal antiinflammatory drugs (NSAIDs), and use of a rigid eye shield until the hyphema has resolved. Do *not* use a pressure patch. Diamox (250 mg four times daily by mouth) should be added, if available, to treat potentially increased IOP. The patient should be maintained in a head-up or head-elevated posture, if possible, to promote settling of the hyphema and clearing of vision. Pain should be managed with acetaminophen or another analgesic medication that does not impair platelet function.

Retinal injury or an occult ruptured globe may accompany traumatic hyphema. A hyphema patient requires ophthalmologic evaluation, so expedited evacuation should be undertaken. If the patient experiences improvement of vision followed by a sudden worsening of vision, this may indicate that rebleeding or retinal detachment has occurred, and emergency evacuation should be considered.

NONTRAUMATIC FLUORESCEIN-POSITIVE ACUTE RED EYE

Herpes Simplex Virus Keratitis

The essential element in the diagnosis of herpes simplex virus (HSV) keratitis is the characteristic dendritic epithelial pattern on fluorescein staining (Figure 48-9). The patient will often have a history of previous episodes of HSV keratitis. Treatment is with trifluridine 1% drops nine times daily.[112] Treatment is continued until the corneal staining has resolved, at which time the frequency of dosing is reduced to four times daily for 1 week. Trifluorothymidine was not included in the list of eye medications to be taken on the expedition because trifluorothymidine drops require refrigeration. If a significant delay is anticipated before evacuation, HSV keratitis may be treated by giving 3 to 5 drops of tetracaine 1 minute apart to anesthetize the cornea and then performing gentle, cotton-tipped applicator debridement of the epithelial lesion.[112] The resulting epithelial defect should then be treated as described previously in the Corneal Abrasion section.

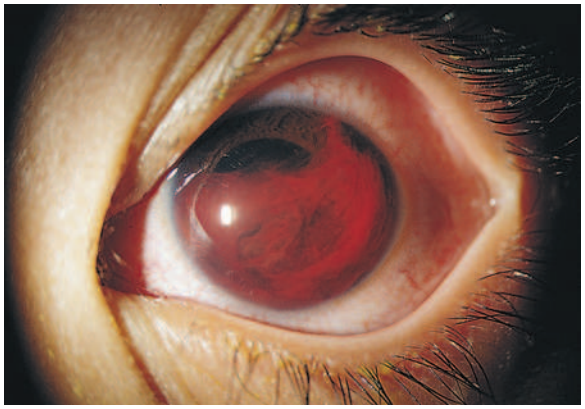

FIGURE 48-8 Hyphema. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

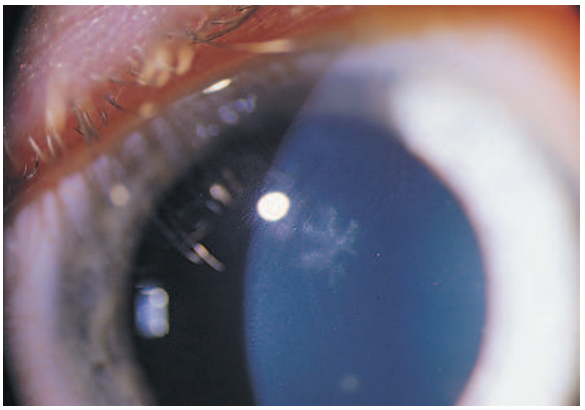

FIGURE 48-9 Herpes simplex virus epithelial keratitis. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

Corneal Erosion

A corneal erosion is an epithelial defect that is caused by nontraumatic disruption of the corneal epithelium. The fluorescein staining pattern seen with corneal erosion may be identical to that seen with corneal abrasion; pain and photophobia are present with both disorders. The diagnosis is made when the patient with the apparent corneal abrasion has no history of trauma to explain its presence. There is often a history of prior similar episodes. The two primary causes of corneal erosion are corneal dystrophies and previous ocular trauma.[105] Recurrent corneal erosions are believed to be caused by a defect in healing between the hemidesmosomes of the corneal epithelium and the underlying basement membrane.[84] At night, the epithelium may become adherent to the closed eyelid during sleep. When the individual awakens and opens the eyes, the corneal epithelium is pulled away from the basement membrane by movement of the lid; this accounts for the typical history of acute onset of pain on awakening. Signs and symptoms include red eye, pain, tearing, foreign body sensation, and usually an epithelial defect, although this may be a variable sign, depending on when the patient is evaluated.[84]

Treatment of these lesions may be difficult. The cornea should be inspected for a loose sheet of epithelium that remains partially attached to the corneal surface. If this is present, try to debride it with a cotton-tipped applicator after topical anesthesia with tetracaine. The lesion is then managed initially in the same manner as a corneal abrasion. There is a high rate of recurrence if follow-up treatment with 5% sodium chloride ointment each evening or anterior stromal puncture is not undertaken.

Corneal Abrasion and Corneal Ulcer with Contact Lens Wear

Both corneal abrasions and corneal ulcers may occur as complications of contact lens wear; management of these conditions has been described earlier. Contact-lens–related corneal abrasions have a relatively high incidence of progressing to corneal ulcers.[70] Therefore, they should not be patched, and they should

receive topical antibiotic therapy with moxifloxacin 0.5% drops every 2 hours while awake until the epithelial defect has resolved. Contact lens wear in both eyes should be discontinued immediately. If an ulcer is related to contaminated lens solutions, infection in the first eye may be followed rapidly by a similar occurrence in the other eye.

NONTRAUMATIC FLUORESCEIN-NEGATIVE ACUTE RED EYE WITH PAIN THAT IS NOT SIGNIFICANTLY IMPROVED BY TOPICAL ANESTHESIA

Acute Angle-Closure Glaucoma

This diagnosis is easily made when the intraocular pressure can be measured with a tonometer. IOP is normally between 10 and 21 mm Hg. With acute angle-closure glaucoma, IOP may increase to 50 or 60 mm Hg or more. Although handheld tonometers are available, they will not often be present outside of hospitals or clinics. The most important clues to the diagnosis of angle-closure glaucoma in the wilderness setting are the characteristics of the pain and the status of the pupil. Angle-closure glaucoma victims do not have the mild burning or foreign body–type pain that is typically seen with conjunctivitis, blepharitis, or other external eye diseases. Angle-closure glaucoma produces pain that is usually deep and severe. Although corneal abrasions and corneal ulcers may also be accompanied by severe pain, the pain associated with these two disorders is usually significantly relieved by topical anesthetics. This is not true of the pain that is seen with acute glaucoma, scleritis, or iritis. With these disorders, the pain is rarely, if ever, significantly relieved by topical anesthetics. With acute angle-closure glaucoma, the pupil is usually found to be midway dilated (i.e., 6 to 7 mm), and vision is usually decreased. The cornea may appear hazy. This disorder generally affects persons older than 50,[84] and it is more common among persons of Asian ethnic origins. There is often a history of previous transient episodes of eye pain. Nausea and vomiting may be present. Figure 48-10 shows an eye with acute angle-closure glaucoma. Some wilderness expeditions now carry portable instruments such as a Tono-Pen to measure IOP. Updated operating instructions for the Tono-Pen can be found at http://www.reichert.com/products.cfm?pcId=474.

The usual treatment is with topical ocular antihypertensive medications and laser iridotomy, which allows the aqueous humor to bypass the pupillary block. This relieves the angle closure, with a resultant decrease in IOP.[84] In the wilderness setting, treatment should be with 2% pilocarpine, with 1 drop given every 15 minutes for four doses,[105] then 1 drop given four times daily in both eyes, because of a high rate of subsequent angle-closure glaucoma in the second eye. Pilocarpine alone may be successful for relieving the angle closure and lowering IOP, but this is not a reliably effective treatment, because ischemia of the pupillary sphincter muscle may prevent pilocarpine from exerting its miotic effect. Diamox (250 mg four times daily by mouth) should be added, if available. Emergency evacuation should occur if pain is not relieved, because even a single day of very high IOP may result in permanent damage to the optic nerve and loss of vision.

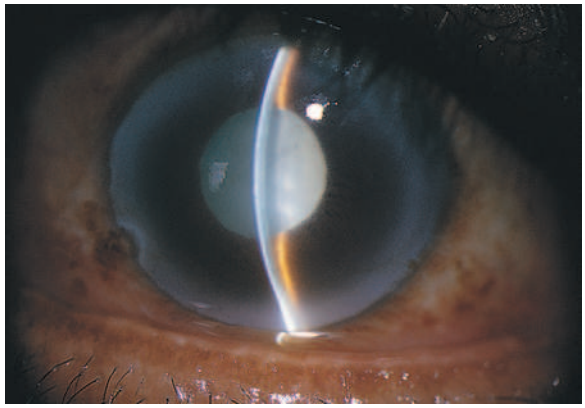

FIGURE 48-11 Angle-closure glaucoma. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

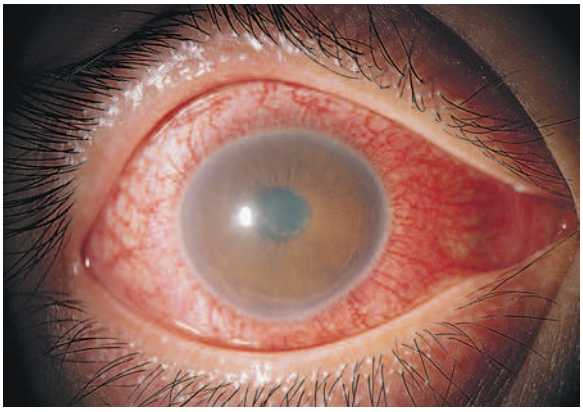

FIGURE 48-11 Nontraumatic iritis. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

Nontraumatic Iritis

Signs and symptoms of nontraumatic iritis that may be appreciated without a slit lamp include pain, redness, photophobia, perilimbal flush, and decreased vision.[105] There is often a history of previous episodes. Figure 48-11 shows an eye with nontraumatic iritis (the pupil is iatrogenically dilated). Iritis not associated with corneal trauma is typically more severe than that of the traumatic variety, but it may range from mild to very severe, and typically is not significantly relieved by topical anesthesia. Fluorescein staining is negative, and the pupil is usually miotic, thereby helping to differentiate iritis from angle-closure glaucoma. Iritis is much more common than angle-closure glaucoma, and it may be associated with a number of systemic infectious and inflammatory diseases. In the wilderness setting, the emphasis should be on immediate treatment with prednisolone 1%, with 1 drop given every hour while awake. Scopolamine 0.25% (1 drop one to four times daily) may be added for pain control and to prevent posterior synechiae (i.e., pupillary scarring) if inflammation is severe. If pain is not significantly decreased after 24 to 48 hours, 80 mg of oral prednisone per day should be added to the treatment regimen and maintained until evacuation. Evacuation should be expedited, because posterior synechiae and elevated IOP may develop.

Scleritis

It may be difficult to differentiate between nontraumatic iritis and scleritis without a slit lamp, but scleritis is much less common than iritis. The characteristics of the pain are similar, with photophobia, scleral injection, and tearing also present in scleritis.[103] A hallmark of scleritis is ocular tenderness to gentle palpation. Scleritis is often associated with rheumatologic disease, and it may be either nodular or diffuse.[105] Figure 48-12 shows the diffuse form of scleritis. The initial treatment of scleritis in the wilderness setting involves topical prednisolone (1 drop every hour while awake) and a NSAID. Prednisone (80 mg/day) should be added if there is no improvement after 24 to 48 hours, and it should be maintained until an expedited evacuation is accomplished.

NONTRAUMATIC FLUORESCEIN-NEGATIVE ACUTE RED EYE WITH NO DISCOMFORT OR WITH DISCOMFORT THAT IS IMPROVED BY TOPICAL ANESTHESIA

Conjunctivitis

One of the most common disorders in this diagnostic category is conjunctivitis. Etiologic agents of infectious conjunctivitis include bacteria, viruses, chlamydiae, fungi, and parasites.[84] Acute allergic conjunctivitis may also be encountered, especially

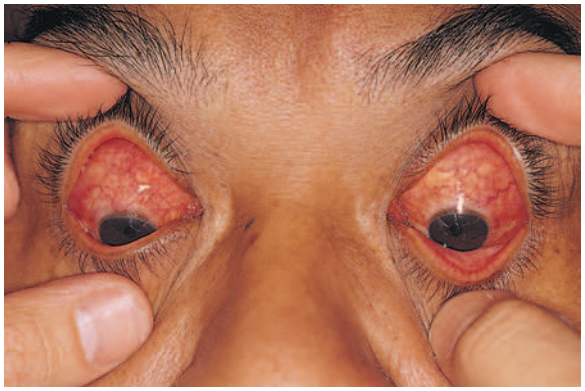

FIGURE 48-12 Diffuse scleritis. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

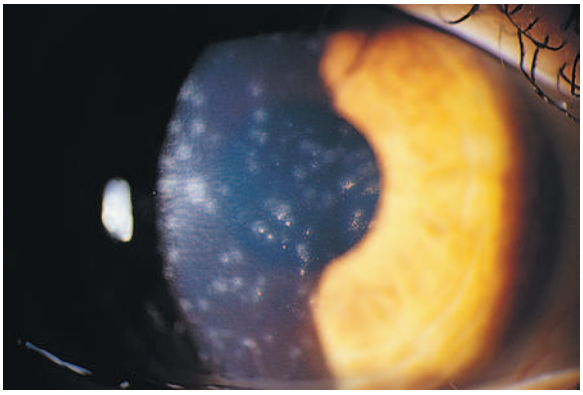

FIGURE 48-13 Epidemic keratoconjunctivitis. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

in a wilderness setting. The keys to diagnosis are the presence of discharge or tearing, relatively mild burning or foreign body–type discomfort that is relieved by topical anesthesia, and a negative or SPK fluorescein staining pattern. The diagnosis of conjunctivitis should be questioned in the absence of discharge or tearing. The primary exception to this is allergic conjunctivitis, which may not generate a significant discharge. In this case, bilaterality, significant ocular itching, and a history of ocular or systemic allergies help with the diagnosis. With infectious conjunctivitis, there are often signs or symptoms of an accompanying upper respiratory infection (URI) or a history of contact with other persons who recently had conjunctivitis.

Treatment of conjunctivitis in the wilderness setting is with moxifloxacin 0.5% drops (1 drop four times daily for 5 days) if there is a yellowish discharge. Ciprofloxacin 0.3% drops, ofloxacin 0.3% drops, tobramycin 0.3% drops, or trimethoprim–polymyxin B drops are acceptable alternatives.[84] This should be an adequate treatment duration if the conjunctivitis is bacterial. Symptoms that persist for more than 5 days suggest a viral etiology. These symptoms may take several weeks to clear, just as some viral URIs take several weeks to resolve. If there is only tearing or a watery discharge, or if there is an antecedent or associated URI that suggests a viral etiology, treatment with artificial tears and cool compresses may be substituted for antibiotic therapy. The patient should be instructed regarding infection precautions, because viral conjunctivitis can spread rapidly through a group. This is especially true if the discomfort caused by conjunctivitis is severe and accompanied by significant photophobia. These symptoms suggest epidemic keratoconjunctivitis, which, in addition to having a prolonged course, is very uncomfortable because of the corneal involvement. Figure 48-13 shows the cornea of an eye with epidemic keratoconjunctivitis. Sunglasses should be part of the treatment. One drop of scopolamine two to four times a day may be required to reduce discomfort.

As noted previously, if the predominant ocular symptom is itching, allergic conjunctivitis should be suspected. Allergic conjunctivitis may be treated with cool compresses or systemic antihistamines. Be cognizant of the sedative effects of systemic antihistamines. Severe ocular itching may be treated with a 3-day course of prednisolone (1 drop four times daily).

Hyperacute conjunctivitis with marked lid edema, conjunctival hyperemia, chemosis, and copious purulent discharge should alert the health care provider to the possibility of a gonococcal etiology.[84] Gonococcal conjunctivitis may progress to vision-threatening keratitis and should be treated aggressively with moxifloxacin (400 mg orally once daily), moxifloxacin drops four times daily, and expedited evacuation. If the conjunctivitis is chronic and associated with a preauricular node and conjunctival follicles, the individual will require additional therapy with doxycycline (100 mg twice daily for 14 days) as treatment for a possible chlamydial infection.[81]

Sexual partners should also be treated for chlamydial infections; follow up with an ophthalmologist, when feasible, is also recommended. Both the bacterial and the viral type of conjunctivitis are highly contagious. Individuals with infectious conjunctivitis should be so informed and the importance of infection precautions emphasized.

Blepharitis

Blepharitis is probably the most common external eye disease seen in the general ophthalmologist's office.[38] It is often misdiagnosed as conjunctivitis. Differentiation may often be made with the use of the patient's history. Blepharitis tends to be a bilateral and chronic disease with recurrences and exacerbations. There is chronic flaking and irritation of the skin at the base of the eyelashes that may occasionally be complicated by a bacterial superinfection. Figure 48-14 shows an eye with blepharitis. There may be an association with skin disorders, such as seborrheic dermatitis or acne rosacea.[38] The patient often has a history of chronic ocular itching and burning. Fluctuating vision may be present as well.[38] There is often a history of excessive mucus discharge in the eyes on waking.

Treatment for blepharitis should focus on the lid margins. Bacitracin or erythromycin ophthalmic ointment should be applied thinly to the lid margins at bedtime for 4 weeks. Lid hygiene is directed at reducing the amount of debris on the lid margins and consists of warm, moist compresses applied to the eyelids one to four times daily for 5 to 10 minutes, followed by gently wiping away the moistened lash debris. Artificial tears may relieve the sensation of "dry eye" that often accompanies blepharitis.[38]

Ultraviolet Keratitis

The diagnosis of UV keratitis is easy to make in the presence of a severely sunburned face and bilateral red, painful eyes. A history of sun exposure at altitude or in the setting of snow-covered ground, or extended time on the water or at the beach, is almost always present. The discomfort typically does not start until 6 to 10 hours after UV exposure, and it may awaken the person. Symptoms range from mild irritation and foreign body

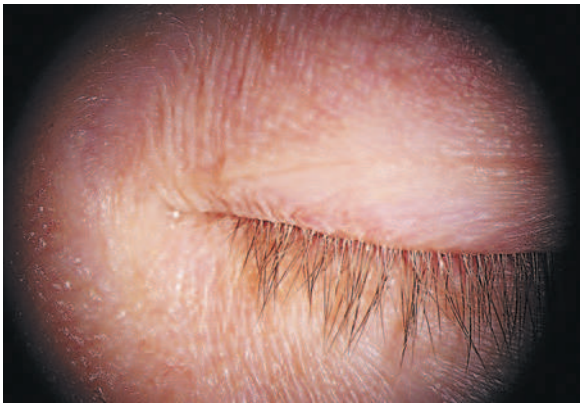

FIGURE 48-14 Blepharitis. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

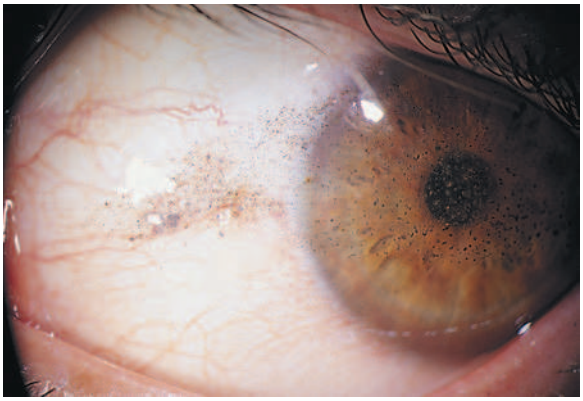

FIGURE 48-15 Conjunctival foreign body. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

sensation to severe pain, photophobia, and lid spasm.[105] Fluorescein staining usually reveals a punctate staining pattern.

Treatment consists of moxifloxacin drops four times daily. Bacitracin or erythromycin ophthalmic ointment applied to the eye four times a day is an acceptable alternative. Sunglasses help with the severe light sensitivity that accompanies UV keratitis. In a severe case, one or both eyes may be patched to help with pain control, although it may be better to patch only the more severely affected eye so that the patient is not deprived of vision in both eyes.[18] Scopolamine (1 drop twice daily) may be added for control of ciliary spasm. Systemic analgesia may also be required. The patient should be reexamined every day until there is no longer an SPK pattern present on fluorescein staining, at which time antibiotic therapy may be discontinued. The duration of discomfort from UV keratitis is typically 24 to 48 hours.[105]

Conjunctival or Corneal Foreign Body

The symptom of ocular foreign body sensation does not necessarily mean that a conjunctival or corneal foreign body is present. Although the presence of a conjunctival foreign body may be strongly suspected on the basis of the abrupt onset of discomfort after a gust of wind or other mechanism for depositing foreign material in the eye, definitive diagnosis requires visualization of the offending material, which may be quite difficult. Figure 48-15 shows an eye with a conjunctival foreign body. The patient is often able to help with foreign body localization before the instillation of topical anesthetic drops.

Treatment consists of a careful search for the foreign body with the use of adequate lighting. Topical anesthesia makes the patient much more agreeable during the search and removal efforts. A handheld magnifying lens or a pair of reading glasses provides magnification to help with visualizing the foreign body. Eyelid eversion with a cotton-tipped applicator helps the examiner to identify foreign bodies located on the upper tarsal plate. After it has been located, the foreign body should be removed with fine forceps or a cotton-tipped applicator when the eye has been anesthetized and the applicator moistened with tetracaine. The eye is then stained with fluorescein to check for a corneal abrasion. If no foreign body is visualized but the index of suspicion is high, vigorous irrigation with artificial tears or sweeps of the conjunctival fornices with a moistened cotton-tipped applicator after topical anesthesia may be successful for removing the foreign body.

Several types of foreign bodies merit special mention. If the foreign body identified is one that may have penetrated into the eye (e.g., large thorn), the patient should be managed as described previously (see Obvious Open Globe). Hot ashes or cinders from a campfire may strike the eye and result in both a foreign body and a thermal burn. Immediate instillation of topical anesthesia provides temporary pain relief to facilitate foreign-body removal and also stops ongoing thermal injury. Multiple or loose conjunctival foreign bodies may be best managed with saline irrigation.[40] After the foreign body has been removed, the corneal abrasion that typically results from a thermal injury to the cornea should be managed as outlined previously (see Corneal Abrasion).

Dry Eye

Symptomatic dry eye is often encountered in the wilderness, especially in mountainous areas where the air is very dry and where significant wind is often present.[18] Dry eye is usually bilateral and may result in secondary tearing.[112] There may be a history of previous episodes of symptomatic dry eye. Individuals with chronic dry eye are usually middle aged or older, and they may have a history of autoimmune disorders.

Treatment is with artificial tears that are used as often as needed to relieve symptoms. Dehydration may contribute to this condition, and adequate fluid intake should be maintained. The use of sunglasses may provide protection from the wind and may be of significant benefit for management of dry eye. High-viscosity drops (e.g., Celluvisc) and lubricating ointments used at bedtime may also help to relieve symptoms.[86] Where ophthalmic care is available for more severe cases, reversible punctal occlusion may be considered as well.[86]

Contact Lens Overwear Syndrome

The considerations here are much as just described for dry eye, except that the symptoms are magnified by the presence of contact lenses.

Contact lens rewetting drops and sunglasses are the first line of management. If these measures are ineffective for relief of symptoms, the contact lenses should be removed. If significant SPK is present on fluorescein staining, moxifloxacin drops (1 drop four times daily) should be used until SPK has resolved.[18] Contact lenses should not be replaced in the eye until the eye is symptom free. An individual who wears contact lenses in the wilderness should always carry a pair of glasses that can be used if contact lens problems arise. Contact lens wear in the wilderness setting should generally be discouraged because of the difficulty maintaining adequate contact lens hygiene.

Episcleritis

Episcleritis is a benign, self-limited, inflammatory condition of the lining of the eye between the conjunctiva and the sclera.[84] There is usually sectoral redness without discharge (Figure 48-16), although the redness may be diffuse in some cases.[40] There is often a history of previous episodes, and discomfort is typically mild or absent. Presence of severe pain, photophobia, or decreased vision suggests another diagnosis. Episcleritis is often misdiagnosed as conjunctivitis, but the lack of a discharge and the sectoral redness usually seen with episcleritis help to differentiate between the two disorders.

Episcleritis is usually self-limited and resolves without treatment in 3 to 4 weeks.[84] If symptoms are troublesome, the condition may be treated with prednisolone drops four times daily for 3 days.

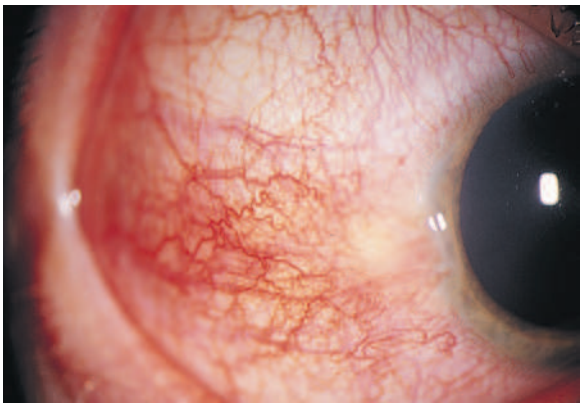

FIGURE 48-16 Episcleritis. *(Courtesy Steven Chalfin, MD, University of Texas Health Science Center, San Antonio.)*

SOLAR RETINOPATHY

The retina is protected from UV radiation damage, because this high-energy radiation is absorbed by the cornea and the lens of the eye. However, visible and near-infrared light of sufficient intensity may reach the retina and cause photochemical damage.[84] In the wilderness setting, this would most likely result from staring at the sun or a solar eclipse. Shortly after such an exposure, the individual may experience blurred or distorted vision, a central scotoma, or a headache. Visual acuity is often reduced to the 20/40 to 20/70 range. There is no effective therapy for this injury, but visual acuity may return to normal over several months in mild cases.

LOCATING A DISPLACED CONTACT LENS

Wearers of soft contact lenses may occasionally have one of their lenses become displaced, which results in blurred vision and a foreign body sensation. When the lens is displaced, it may be difficult to locate. The conjunctival fornix of the lower lid is easily examined by distracting the eyelid from the globe with gentle downward finger pressure applied to the lower lid. If the contact lens has been displaced into the superior conjunctival fornix, which is usually the case, it may be more difficult to locate. If visual inspection with a penlight and a handheld magnifying lens is not successful for finding the lens, gentle digital massage over the closed upper lid that is directed toward the medial canthus often results in the contact lens emerging at that location. Several minutes of massage may be required. A few drops of artificial tears often facilitate the process. If this maneuver is unproductive, the eye may be anesthetized with 1 drop of tetracaine, the upper lid distracted from the globe with upward finger pressure, and the fornix swept with a moistened cotton-tipped applicator.

IMPROVISATION

If you encounter a person with known or suspected globe rupture, it is of paramount importance to ensure that subsequent inadvertent trauma does not cause extrusion of the ocular contents. If an eye shield is not available, one may be fashioned with duct or rigger's tape and any available rigid flat or concave object that can be placed over the eye. Examples include small cups or bowls to provide a good standoff distance between the eye and improvised shield. Improvised wound closure strips can be made by tearing rigger's or duct tape into ¼-inch widths. Spectacles and sunglasses can be improvised by making a pinhole in a piece of paper or cardboard. (Make the pinholes before placing the paper or cardboard in front of the eyes.) Take note of the restriction in peripheral vision that is caused by use of pinhole glasses. If the purpose is to improvise sunglasses rather than to achieve a refractive effect, the pinhole can be larger or fashioned into a horizontal slit to improve peripheral vision. Improvised magnifying glasses for examining the eye or other small areas may be made by simply using a pair of reading or hyperopic glasses. Another refractive improvisation technique involves a handheld magnifying lens. This convex lens, with its plus refracting power, can be used to provide a variety of hyperopic refractive corrections by moving the lens to various distances in front of the eye.

THE EYE AT ALTITUDE

Ocular disorders that are associated with altitude exposures have been addressed in several review articles.[18,72,86]

ALTITUDE EXPOSURES AND OCULAR PHYSIOLOGY

A number of significant effects on visual function that result from the hypoxia of altitude were described by Wilmer and Berens[151] in their classic article, as well as later by Sharma.[121] The decrease in ambient pressure at altitude causes hypobaric hypoxia despite a constant oxygen fraction of 0.21, as noted in Table 48-1. Retinal blood flow has been shown to increase by 128% after 4 days at 5300 m (17,384 feet).[49] This increase in blood flow results in clinically observable changes, such as an increase in the diameter and tortuosity of retinal vessels and optic disc hyperemia, which are seen in most unacclimatized persons at altitudes of 4573 m (15,000 feet) and higher.[49,74,89,111]

Because of its avascularity, the cornea receives most of its oxygen supply from the surrounding atmosphere,[128] so it may sustain hypoxic dysfunction, even if the inspired gas mix is not hypoxic, as noted later (see Refractive Changes at Altitude after Refractive Surgery). This is an important factor when considering topics such as the suitability of contact lenses and refractive surgical procedures for mountaineers and aviators.

TABLE 48-1 Pressures and Equivalent Oxygen Fractions at Altitude

Feet	Meters	Atmospheric Pressure (mm Hg)	Equivalent of Oxygen (%)	Partial Pressure of Oxygen (mm Hg)
Sea level	Sea level	760	20.9	159
4000	1219	656	18.0	137
7000	2134	586	16.1	122
10,000	3048	523	14.4	109
15,000	4572	429	11.8	89
20,000	6096	347	9.5	73
25,000	7620	282	7.8	60
29,028	8848	253	7.0	53

HIGH-ALTITUDE RETINAL HEMORRHAGE

There are many reports of retinal hemorrhages in mountain climbers (Figure 48-17). These have been described as high-altitude retinal hemorrhages (HARHs) or as part of the more inclusive term *altitude retinopathy*.[24] A classification for HARH has been developed by Wiedman.[148,149] Butler and colleagues[24] reported a 29% incidence of HARH among climbers on a Mt Everest expedition at altitudes that ranged from 5300 to 8200 m (17,385 to 26,896 feet). McFadden and co-workers[89] found that 56% of their participants had HARH at an altitude of 5360 m (17,581 feet) and that one had a retinal nerve fiber layer infarct (i.e., cotton wool spot) (Figure 48-18). They also reported that exercise at altitude was associated with both an increased

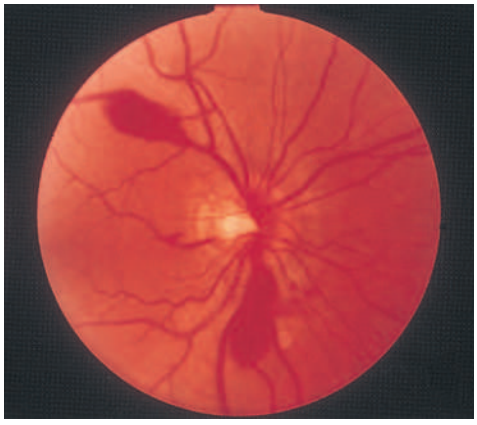

FIGURE 48-17 High-altitude retinal hemorrhages. *(Courtesy Dr. M. McFadden, University of British Columbia, Vancouver, British Columbia. In association with Dr. C. Houston, Burlington, Vermont; Dr. G. Gray, Canadian Defense and Civil Institute of Environmental Medicine, Toronto, Ontario; and Drs. J. Sutton and P. Powells, McMaster University, Hamilton, Ontario, Canada.)*

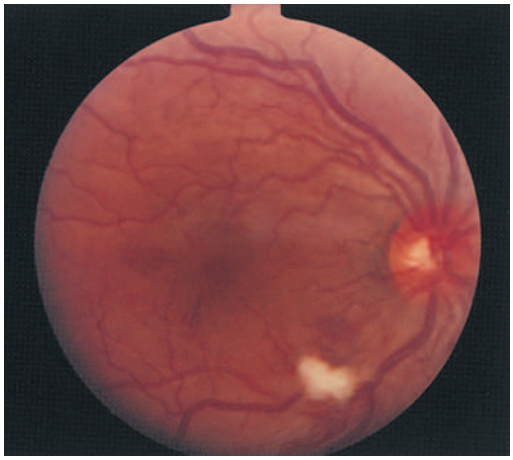

FIGURE 48-18 Cotton wool spots seen at 5400 m (17,712 feet). This occurred in a climber after the Valsalva maneuver was performed and represents the most severe form of retinopathy. *(Courtesy Dr. M. McFadden, University of British Columbia, Vancouver, British Columbia. In association with Dr. C. Houston, Burlington, Vermont; Dr. G. Gray, Canadian Defense and Civil Institute of Environmental Medicine, Toronto, Ontario; and Drs. J. Sutton and P. Powells, McMaster University, Hamilton, Ontario, Canada.)*

incidence of HARH and fluorescein leakage from the retinal vessels. Hackett and Rennie[54] described HARH in 4% of 140 trekkers examined at 4243 m (13,917 feet) at Pheriche in the Himalayas; they also found a significant correlation of retinal hemorrhages with symptoms of acute mountain sickness. Kobrick and Appleton[74] found no retinal hemorrhages in eight individuals examined after 48 hours at 4573 m (15,000 feet) in a hypobaric chamber, although all participants displayed the marked vascular engorgement and tortuosity that is typical of the eye at altitude (Figure 48-19). HARH developed in 19 of 21 climbers who climbed above 7600 m (25,000 feet). Fourteen of 19 climbers who climbed to between 4880 and 7600 m (16,000 and 25,000 feet) were found to have retinopathy.[150] Differences in the incidence of HARH for exposures at similar altitudes may be the result of differences in time at altitude before examination, acclimatization schedule, exercise levels, examination techniques, and the presence of concurrent conditions that may predispose an individual to HARH.

High-altitude retinal hemorrhage has been reported to be associated with both altitude headache and a history of vascular headache at sea level.[119] Rimsza and colleagues[113] noted that HARH may occur at lower altitudes among individuals with chronic lung disorders that interfere with oxygenation. Their case report described a woman with cystic fibrosis who had climbed as high as 3049 m (10,000 feet) but was at 1677 m (5500 feet) when she noted a sudden decrease in vision associated with preretinal hemorrhage of the right eye. These authors also noted that the ocular findings of cystic fibrosis are similar to those seen with altitude retinopathy.[113]

Although HARH is often not associated with acute visual symptoms,[24,111,147,155] affected individuals may experience a loss of visual acuity or paracentral scotomas.[48,80,123,147] Lang and Kuba[80] reported on a person who experienced decreased visual acuity and central scotoma from HARH as a result of a 7000-m (22,960-foot) altitude exposure. Permanent deficits in visual function are uncommon but have been reported.[123,147] Shults and Swan[123] reported that four survivors of an ill-fated Aconcagua expedition in Argentina in 1973 were found to have severe HARH after an altitude exposure of 6860 m (22,500 feet). Two of the survivors apparently sustained permanent paracentral scotomas. No reports were found of progressive decrease in visual acuity or progressive enlargement of paracentral scotomas as a result of remaining at altitude after development of HARH.[18] Wiedman[147] reported a patient in whom further ascent after development of HARH resulted in additional lesions. HARH that results in decreased visual acuity should be a contraindication to further ascent.[18] Butler and colleagues[24] recommended that evacuation of individuals with decreases in visual function as a result of HARH (in the absence of high-altitude cerebral edema or high-altitude pulmonary edema) be considered nonemergent unless reexamination indicates progressive deterioration of vision or increasingly severe retinopathy. HARH resolves over 2 to 8 weeks after the altitude exposure is terminated.[147] Recognizing advancing severity of HARH may allow physicians to recommend initiating treatment with oxygen, corticosteroids, diuretics, and immediate descent to prevent HARH progression, macular involvement, or potentially fatal high-altitude cerebral edema. High-altitude retinopathy is both a significant component of and a predictor of progressive altitude illness.[150]

INTRAOCULAR GAS BUBBLES AT ALTITUDE

The presence of an intraocular gas bubble presents a hazard for any person exposed to a change in ambient pressure. Intraocular gases are frequently used to provide internal tamponade during and after vitreoretinal surgery and some types of corneal surgery. Three patients described by Hart and associates[58] lost vision in their eyes with intraocular gas after subsequent nitrous oxide

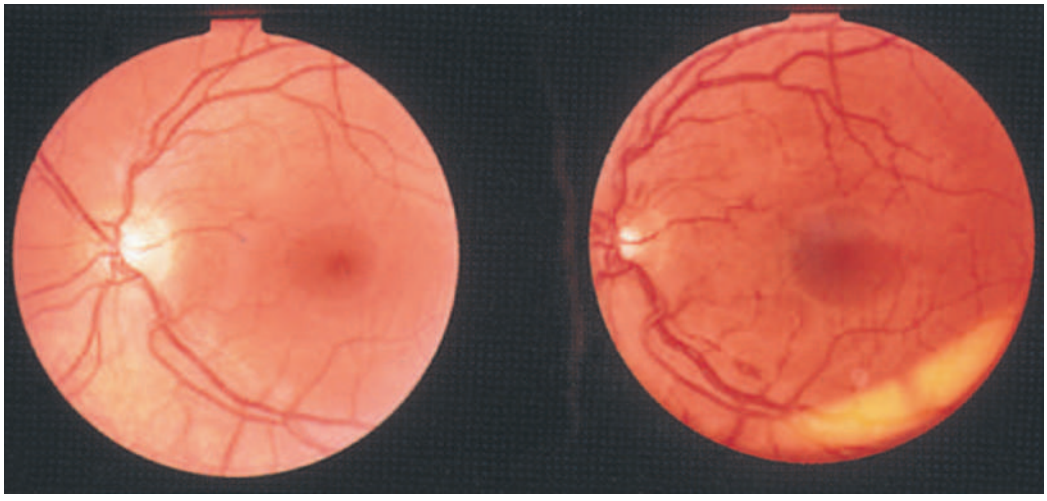

FIGURE 48-19 The normal fundus at sea level (*left*). The same fundus at 5400 m (17,712 feet) (*right*). Note the vascular engorgement and tortuosity at altitude. *(Courtesy Dr. M. McFadden, University of British Columbia, Vancouver, British Columbia. In association with Dr. C. Houston, Burlington, Vermont; Dr. G. Gray, Canadian Defense and Civil Institute of Environmental Medicine, Toronto, Ontario; and Drs. J. Sutton and P. Powells, McMaster University, Hamilton, Ontario, Canada.)*

(N_2O) general anesthesia. All three patients had visual loss from presumed retinal artery occlusion caused by expansion of the intraocular gas by N_2O administration during general anesthesia.[58] N_2O is a highly soluble inhalational anesthetic agent that diffuses rapidly down its concentration gradient into an intraocular gas bubble. N_2O is known to increase intraocular gas bubble volume during general anesthesia.[153] Resulting expansion of the bubble can lead to a rapid rise in IOP.[129]

Growth of intraocular gas bubbles can also be caused by exposure to reduced atmospheric pressure during air travel.[75,92] Even small bubbles can cause symptomatic increases in IOP. The patient with residual intraocular gas who experiences pain or dimness of vision in an ascending airplane may obtain relief if the cabin altitude is decreased.[82] A patient who had a scleral buckle and pneumatic retinopexy with a residual gas bubble 2 weeks before a commercial airline flight experienced sudden blindness, presumably from gas bubble expansion with resultant CRAO when the aircraft ascended to a cabin pressure of 2440 m (8000 feet).[108] The mechanism of this injury is presumed to be an IOP rise that exceeded central artery pressure, thereby collapsing the artery. The patient's symptoms were relieved when an onboard flight surgeon recognized the problem and cabin pressure was reset to 610 m (2000 feet).[108]

The same problem can occur if an individual with an intraocular gas bubble makes an ascent of sufficient altitude during mountaineering.[56] Fang and Huang[42] reported a case of a 46-year-old man who ascended to an altitude of 1893 m (6210 feet) after vitreoretinal surgery with a residual perfluoropropane bubble in his right eye. He noted a sensation of fullness in his right eye that was accompanied by loss of vision. When he returned to sea level, his IOP was found to be 54 mm Hg. The cause of this vision loss was believed to be CRAO caused by expansion of intraocular gas during mountain travel.[42]

CORTICAL BLINDNESS AT HIGH ALTITUDE

Hackett[53] reported six cases of cortical blindness at high altitude. These patients were found to have intact pupillary reflexes. Descent of at least 923 m (3000 feet), Gamow (hyperbaric) bag recompression, or supplemental oxygen breathing should be used in persons with such neurologic dysfunction at altitude.

OCULAR MOTILITY

Kramar and colleagues[77] reported that convergence insufficiency was found in women with altitude illness. Basnyat[7] noted that lateral gaze palsy and other focal neurologic deficits, often in the absence of other symptoms of acute mountain sickness or high-altitude cerebral edema, are seen frequently by physicians working for the Himalayan Rescue Association. Rennie and Morrissey[111] reported on a person with nystagmus at altitude that was associated with ataxia and intention tremor. Ocular motility abnormalities should be managed in the same way as cortical blindness at high altitude, as described in the preceding section.

CONTACT LENSES IN MOUNTAINEERING

Contact lenses have been used successfully at high altitude.[29,155] Clarke[29] noted that contact lenses were used successfully by five members of the British Everest Expedition in 1975 up to altitudes of 7317 m (24,000 feet). The use of contact lenses at altitude during trekking or mountaineering entails several considerations beyond those encountered with normal use. In general, overnight use of extended-wear contact lenses is not recommended because of the associated increased rate of microbial keratitis. Even soft contact lenses with high oxygen permeability decrease the oxygen available to the cornea. Lid closure during sleep further accentuates corneal hypoxia. However, removing contact lenses at night presents logistical problems in the mountaineering setting. Practicing acceptable lens hygiene during an expedition is difficult. The mountaineer who leaves contact lenses in a case that is filled with liquid solution in the tent outside of the sleeping bag at night may awaken to find the solution and lenses frozen solid. In addition, wearing contact lenses can make eyes more sensitive to glare.[45]

Guidelines for military personnel using contact lenses in austere environments have been developed in the past. Although contact lenses are no longer recommended for use in deployed field settings in the military, the following guidelines may be applied with caution to the expedition setting[18]:

1. Disposable extended-wear lenses may be left in the eye for up to 1 week. If the wearer is still in the field at the end of this period, the lenses should be removed and discarded. After an overnight period without lenses, new lenses may be inserted, with strict attention paid to contact lens hygiene.
2. Contact lens wearers should always have backup glasses available for use in the wilderness in case a lens is lost or becomes painful.
3. Individuals who wear contact lenses on expeditions should carry both fluoroquinolone eye drops and contact lens rewetting solution. Both types of drops may freeze if they are not protected from the cold.
4. Contact lens wearers often note that their eyes become dry. This discomfort may be alleviated with contact lens rewetting drops.
5. Contact lens wearers often note increased sensitivity to sunlight. Individuals who wear contact lenses in the field during daylight hours should carry sunglasses.

Continuous wearing of disposable contact lenses for 1 week, followed by discarding the lenses and insertion of fresh lenses after an overnight period without a lens, is a controversial approach to contact lens wear in an expedition setting. Whether the reduction in lens handling offsets the increased risk of microbial keratitis that results from overnight wear is unknown. The decision to wear contact lenses while mountaineering should be made carefully. Microbial keratitis (corneal ulcers) can pose a significant threat to vision under the best of circumstances. Should this disorder occur with a 7- to 10-day delay to definitive ophthalmologic care, the danger of a permanent loss of vision is great. Any eye pain that occurs among contacts lens wearers in the wilderness should be managed as described previously (see Acute Red Eye). Contact lenses that block out harmful UV rays are available (e.g., Acuvue, Precision UV), but sunglasses are still recommended to help protect the eyes from drying wind effects and the eyelids from UV exposure, even if contact lenses are worn. Considering all the potential problems, a good pair of prescription glacier glasses or laser refractive surgery might be a more reasonable alternative than contact lenses as a long-term solution to the refractive needs of mountaineers. The excellent military experience with photorefractive keratectomy (PRK) and laser-assisted in situ keratomileusis (LASIK) has led to a vision enhancement program that has largely obviated the need for contact lens use in deployed military settings.[55,132] The effects of refractive surgery on the vision of individuals at altitude are discussed in the following section.

REFRACTIVE CHANGES AT ALTITUDE AFTER REFRACTIVE SURGERY

An acute hyperopic shift in persons who have had radial keratotomy (RK) and who then experience altitude exposure was reported by Snyder and colleagues[130] in 1988 and by White and Mader[145] in 1993. This effect has been observed at altitudes of as low as 2744 m (9000 feet).[130] A dramatic example of this phenomenon was that experienced by Dr. Beck Weathers in the Mt Everest tragedy of May 1996, during which eight climbers lost their lives. Dr. Weathers had undergone bilateral RK years before the expedition. He noted a decrease in vision, which started early during his ascent.[76] Author Jon Krakauer[76] recalls that, "as he was ascending from Camp Three to Camp Four, Beck later confessed to me, 'my vision had gotten so bad that I couldn't see more than a few feet.'" This decrease in vision forced Weathers to abandon his quest for the summit shortly after leaving Camp Four and nearly resulted in his death. Another report describes two expert climbers who experienced hyperopic shifts of 3 diopters (D) or more during altitude exposures of 5000 m (16,400 feet) or higher on Denali and Mt Everest.[30] Mader and White[87]

found that the magnitude of the hyperopic shift was 1.03 ±0.16 D after 24 hours at 3659 m (12,000 feet) and 1.94 ±0.26 D at 5183 m (17,000 feet). Ng and colleagues[98] reported no refractive change after 6 hours in post-RK eyes at a simulated altitude of 3659 m (12,197 feet), which suggests that the hyperopic shift requires more than 6 hours to develop.

Further studies by Mader and colleagues[85] at 4299 m (14,100 feet) on Pike's Peak revealed that (1) a climber who had undergone RK demonstrated a progressive hyperopic shift associated with flattened keratometry findings during a 72-hour exposure; (2) control eyes and eyes that had undergone laser refractive surgery (i.e., PRK) experienced no change in their refractive state; (3) peripheral corneal thickening was seen on pachymetry in all three groups; and (4) refraction, keratometry, and pachymetry all returned to baseline after a return to sea level. Winkle and associates[152] demonstrated that exposing post-RK corneas to 100% nitrogen through goggles at 1 atm for 2 hours caused a significant hyperopic shift of 1.24 D and corneal flattening of 1.19 D. Corneal thickness increased in both post-RK and control eyes but was not associated with a hyperopic shift in control eyes. This is strong evidence that the effect of altitude exposures on post-RK eyes is caused by hypoxia rather than by hypobarism, and it further illustrates that breathing a normoxic inspired gas mix will not protect against development of hypoxic corneal changes in post-RK eyes.

The effect of the post-RK hyperopic shift seen at altitude depends on the postoperative refractive state (i.e., undercorrected patients may actually have their vision improve) and the accommodative abilities of the individual.[87] The work of Mader and colleagues provided compelling evidence that, for myopic mountaineers, PRK rather than RK should be the refractive surgical procedure of choice. Individuals who have undergone RK and who plan to undertake an altitude exposure of 2744 m (9000 feet) or higher while mountaineering should bring multiple spectacles with increasing plus lens power.[87] To our knowledge, a post-RK hyperopic shift has not been reported among airline passengers or flight crew. This may be because the latent period required for this phenomenon to develop exceeds the duration of most commercial flights, or because the approximate 2440-m (8000-foot) cabin pressure on most commercial flights does not produce sufficient corneal hypoxia for a hyperopic shift to occur.

The altitude at which RK was performed also affects the surgical outcome. RK that is performed at 1753 m (5750 feet) produces more of a hyperopic shift than does the same procedure performed at sea level.[28,35,127] The average changes in spherical equivalent cycloplegic refraction were 5.09 ±1.29 D and 6.50 ±2.24 D among patients who had undergone the same procedure by the same surgeon at sea level and at 1753 m (5750 feet), respectively.[127] In another study, the mean spherical equivalent cycloplegic refraction changes were 4.40 ±0.92 D and 6.03 ±1.13 D in patients who had had RK at sea level in Istanbul, Turkey, and at 1720 m (5641 feet) in Van, Turkey, respectively (p <0.001). The procedures at both altitudes were performed by the same surgeon and using the same technique, to demonstrate the potential need to adjust surgical nomograms for altitude.[28] Because RK surgery is almost never performed now, the findings related to that procedure are primarily of historical interest.

The most common laser refractive surgery at present is LASIK.[86] White and Mader[146] reported that a climber who had undergone LASIK developed blurred vision after reaching the summit of Alpamyo (5945 m [19,500 feet]). He noticed the blurred vision after spending two nights at 5488 m (18,000 feet), and his vision cleared 3 days after returning to 3048 m (10,000 feet).[146] Recent studies have examined the effects of altitude exposure on individuals who have undergone this procedure. Six climbers who had bilateral LASIK subsequently undertook an ascent of Mt Everest.[35] Visual acuities were measured at base camp (5365 m [17,600 feet]); subjective symptoms (but not measured acuities) were obtained at higher altitudes. All climbers ascended to at least 7927 m (26,000 feet), and four of the six reached the summit at 8852 m (29,035 feet). Five of the six climbers reported no visual changes at altitudes of up to 7927 m (26,000 feet). One climber reported mild blurring with ascent above 4878 m (16,000 feet) that improved with descent or prolonged stay at altitude. Two additional climbers reported visual blurring at altitudes of 8231 m (27,000 feet) and 8689 m (28,500 feet). The authors' conclusion was that LASIK may be a good choice for individuals involved in high-altitude activities, but those who plan to achieve extreme altitudes of 7927 m (26,000 feet) and above should be aware of possible fluctuations of vision.[35]

In another study, two physicians who had undergone myopic LASIK 8 and 14 weeks previously subsequently undertook an ascent of Aconcagua (6964 m [22,841 feet]). They prospectively assessed their vision at 610-m (2000-foot) intervals during ascent. Both climbers described moderate loss of distance acuity with preserved near and pinhole acuity. These findings are suggestive of a myopic shift at altitude. Blurred vision began to occur at 5488 m (18,000 feet) in the left eye and at the summit (6964 m [22,841 feet]) in the right eye in one climber; blurring was first noticed at 5488 m (18,000 feet) in the right eye in the second climber. These myopic shifts resolved after a return to sea level.[13] These data suggest that a small refractive shift in the myopic direction may be present at extreme altitudes. Climbers who do not ascend beyond moderate altitudes should not experience a post-LASIK refractive shift.

GLAUCOMA AT ALTITUDE

Mader and Tabin[86] discuss the hazards of going to altitude with severe glaucoma. Even individuals who have mild, well-controlled glaucoma may suffer adverse effects at altitude if they are being treated with topical β-blockers. These drugs may produce systemic side effects of lethargy and exercise intolerance that may be exacerbated at altitude.[86] Consideration should be given to other choices for topical therapy for persons who will be traveling to significant altitude. The hypoxia of altitude may also worsen glaucomatous optic neuropathy. One person with moderately severe glaucoma who was well controlled on two topical medications ascended to 6707 m (22,000 feet) on Cho Oyu in the Himalayas. He noticed a decrease in vision and descended. His IOP was normal when measured in Kathmandu, but a marked increase in optic nerve damage was later confirmed by visual field and optic nerve imaging studies.[86]

ULTRAVIOLET RADIATION DAMAGE

Ultraviolet radiation is divided into UVA (320 to 400 nm), UVB (290 to 320 nm), and UVC (100 to 290 nm). Almost all UVC radiation is absorbed by the Earth's ozone layer.[102,134] The cornea absorbs all radiation with wavelengths of less than approximately 300 nm, and the lens absorbs almost all the remaining UVB radiation that reaches it.[134] UV radiation is increased with high-altitude, low-latitude, and highly reflective environments.[102] Altitude exposures associated with mountaineering entail exposures to increases in the amount of both incident and reflected UV light (see Chapter 16).

Acute exposure to high levels of UV radiation may result in UV photokeratitis,[36,134] whereas chronic exposures may be associated with cortical lens opacities,[31] posterior subcapsular lens opacities,[12] pterygia,[10] and squamous cell carcinoma of the conjunctiva.[97] The Salisbury Eye Evaluation project quantified ocular UVB exposure with use of a UVB pyranometer.[143] This study found that cataract incidence increased (odds ratio, 1.1; 95% confidence interval, 1.02 to 1.20) with the degree of UVB sun exposure from the lowest to the highest quartile.

Diagnosis and management of UV keratitis are discussed earlier (see Ultraviolet Keratitis). The best strategy for dealing with UV radiation–induced disorders is prevention. Most experienced mountaineers and trekkers are well aware of the need for sunglasses with high ratings for UV absorption. UV attenuation in sunglasses depends on the size, shape, and wearing position as well as the absorption properties of the optical material used.[115] Wearing a brimmed hat is another effective method of decreasing UV exposure to the eye.[45,102,116] In conjunction with the use of topical sun-blocking agents, a hat may also help to prevent cutaneous neoplasms from being a lasting reminder of previous mountaineering expeditions.

SUNGLASSES SELECTION IN MOUNTAINEERING

When selecting sunglasses for use in mountaineering, absorption of essentially all UV radiation is a key consideration, because radiation in this portion of the electromagnetic spectrum is not visible and serves only to produce adverse effects in the eye. Another critical consideration is the amount of visible light that is transmitted. Sunglasses that suffice for everyday use may not be adequate for use in the mountains, especially while on snow or glaciers. The comfort zone for luminance is approximately 350 to 2000 candelas/m^2.[134] An outdoor environment that consists of sunlit fields and foliage may have a luminance of 3000 to 7000 candelas per square meter (cd/m^2); a bright beach may have a luminance of 6000 to 15,000 cd/m^2. Standard sunglasses that transmit 15% to 25% of visible light reduce the luminance in these situations to within the comfort range. In contrast, bright sun reflected off snow or clouds may result in luminances of 15,000 to 30,000 cd/m^2. Sunglasses with visible light transmittance in the 5% to 10% range are needed to reduce luminance to a comfortable range in these circumstances. (The 1986 American National Standards Institute standards for nonprescription sunglasses recommend that tinted lenses with visible light transmittances of less than 8% not be used for driving.[134]) Side shields or deeply wrapped lens designs should be used.[134] Infrared absorption is important in certain industrial occupations (e.g., for those who work with glass, iron, and steel),[134] but this is less important in protective eyewear that is to be used outdoors.

Table 48-2 provides desirable characteristics to consider when selecting sunglasses for mountaineering or other environments with high levels of luminance and UV radiation.[18] Individuals who are involved in mountaineering expeditions should always carry spare sunglasses in case the primary pair is lost or broken.

PHOTOCHROMIC LENSES

Photochromic lenses change transmittance or color when they are exposed to light or UV radiation.[134] They are designed to transmit a greater percentage of incident light when indoors or in conditions of reduced illumination and a reduced amount of light when exposed to higher levels of illumination. This is accomplished in one example of glass lenses by incorporating inorganic silver halide into the lens. When the lens is exposed to sunlight, this compound decomposes into its component silver and halide ions, and the lens turns dark gray.[134] When the lens is removed from sunlight, the process reverses. When selecting photochromic lenses, the adequacy of indoor light transmittance can be judged while trying them on. Outdoor transmittance should be approximately 5% to 10%, as noted in Table 48-2, if the glasses are to be used for mountaineering. Most photochromic lenses have outdoor transmittances of approximately 20%, which should suffice for less highly reflective outdoor environments.[134]

Several plastic photochromic lenses are now available. The darkening process in plastic lenses is accomplished with organic light-sensitive compounds that are suspended in a thin layer near the front of the lens. Plastic photochromic lenses generally do not darken well enough to be used as sunglasses in bright environments. In addition, unlike glass photochromic lenses, the photochromic reaction typically used in plastic lenses usually fades in 1 to 2 years.[134]

TABLE 48-2 Sunglasses: Selection Criteria for Mountaineering

Criterion	Description/Characteristics
Ultraviolet light absorption	99% to 100%
Visible light transmittance	5% to 10%*
Lens material	Polycarbonate or CR-39†
Optical quality	Clear image without distortion‡
Frame design features	Large lenses; side shields or wraparound design; fit close to face; good stability on face during movement; lightweight; durable
Color	Gray§

*Glasses with less than 8% transmittance of visible light should not be worn while driving. Sunglasses or any tinted lenses with a visible light transmittance of less than 80% should not be worn while driving at night.[134]
†Glass lenses typically have very good optical clarity and scratch resistance, but are heavier and more expensive. They also do not provide the same degree of impact resistance of polycarbonate lenses.
‡Hold the sunglasses at arm's length, and then move them back and forth. If the objects are distorted or move erratically, the optical quality is probably less than desirable. In addition, compare the image quality of several different pairs of sunglasses to have a basis for comparison.
§Colored lens tints can alter color perception and possibly compromise the visibility of traffic signals. Neutral gray absorbs light relatively constantly across the visible spectrum and avoids these problems.[134]

THE EYE AND DIVING

The ocular aspects of scuba diving and other hyperbaric exposures have been well reviewed.[17] This section discusses the hyperbaric environment, ocular barotrauma, the ocular manifestations of decompression sickness and arterial gas embolism, ophthalmic considerations in fitness-to-dive evaluations, and the differential diagnosis of decreased vision after diving. An expanded discussion of these issues, as well as additional material about diving after eye surgery, the effect of common eye medications on fitness to dive, and the use of hyperbaric oxygen to treat ocular disorders, is available in the review article.[17]

THE HYPERBARIC ENVIRONMENT

At sea level, the body is exposed to 1 atmosphere (atm) of pressure. This magnitude of pressure may also be expressed as 760 millimeters of mercury (mm Hg), 33 feet of seawater (fsw), and 14.7 pounds per square inch (psi).

The normal atmospheric pressure of 1 atm is often used as a reference point from which other pressures are measured. When one states that the intraocular pressure is 15 mm Hg, what is meant is that IOP is 15 mm Hg greater than the surrounding environment. In fact, the absolute pressure inside the eye at sea level is 775 (760 + 15) mm Hg. The IOP that is measured with a tonometer is therefore a "gauge" pressure, which means that the pressure displayed is the actual pressure minus the atmospheric pressure.

OPHTHALMIC CONSIDERATIONS IN THE FITNESS-TO-DIVE EVALUATION

A diver should have adequate visual acuity to be able to read gauges and to function safely underwater. Possession of a driver's license is a convenient indication that a potential diver has sufficient visual acuity to meet this standard.[17] A person who has recently undergone ophthalmic surgery should refrain from diving until the recommended convalescence interval has passed. Individuals with glaucoma may dive safely unless they have had glaucoma filtering surgery performed. Systemic carbonic anhydrase inhibitors, rarely used in the current therapy of glaucoma, are best avoided by patients who want to dive because of possible confusion between medication-induced paresthesias and decompression sickness. Any individual with an acute ocular disorder that causes significant pain, decreased visual acuity, or other disabling symptoms should refrain from diving until these symptoms have resolved.[17]

Individuals who have undergone surgical repair of retinal detachments may occasionally have gas bubbles placed in the eye to stent the retina against the retinal pigment epithelium. Gas bubbles may also be placed in the eye after certain anterior segment ophthalmic surgery procedures. Intraocular gas bubbles are an absolute contraindication to both diving and hyperbaric chamber exposures. In one study, rabbit eyes were injected with perfluoropropane to a bubble size of 60% of the vitreous cavity. When the eyes that contained the bubbles were exposed to increased atmospheric pressure, IOP dropped compared with the surrounding environment, then rose to more than 50 mm Hg when the atmospheric pressure was returned to normal.[67] The

individual who dives with a gas bubble may experience severe pain on descent as a result of compression of the globe as the gas phase is compressed,[107] or on ascent after extra gas has diffused into the bubble and the absolute pressure is reduced on surfacing, thereby causing the bubble to grow and IOP to rise.

UNDERWATER REFRACTIVE CORRECTION

If contact lenses are to be used for diving, soft contact lenses are preferred.[32,39,69,90] Hard (polymethylmethacrylate) contact lenses have been associated with corneal edema during decompression and after dives.[126,125] These changes are caused by formation of nitrogen bubbles in the precorneal tear film during decompression, which interferes with normal tear film physiology and results in epithelial edema. Bubble formation under contact lenses would be expected to be more common during dives with significant decompression stress.

Although the increased gaseous diffusion properties of rigid gas-permeable contact lenses theoretically decrease the chance of bubble formation in the tear film, use of these lenses while diving has been demonstrated to cause bubble formation under the lens and lead to secondary corneal epithelial disruption.[131] One of the authors (FKB) has personally treated a diver with foreign body sensation and blurred visual acuity that occurred during ascent while wearing gas-permeable contact lenses. Symptoms resolved on removal of the lens at the surface.

Corneal edema was not observed in one series that studied soft contact lenses.[126] The most frequent complication of soft contact lens use in diving is loss of the lens.[69,73] The risk of lens loss can be minimized by ensuring a good seal on the face mask and by minimizing the amount of water that leaks into the air space of the mask. Should the mask become displaced during the dive, resulting in direct contact of the eyes with water, narrowing of the palpebral fissures helps to decrease the chance of the contact lens floating off the surface of the eye.[69]

Betts[11] reported bubbles forming under "micro-corneal" contact lenses. When leaving the hyperbaric chamber after a simulated dive, the diver complained of blurred vision in the right eye, which appeared to have a white film over it. The left eye was similarly affected, but to a lesser extent. During ophthalmoscopic examination, a haze of fine bubbles could be seen under the contact lens; these bubbles persisted on the surface of the cornea after the lens was removed. The bubbles did not move with blinking and were thought to be in the superficial layers of the cornea; they slowly resolved over 30 minutes. The author speculates that the observed difference in bubble formation between the two eyes may have been the result of a difference in lens fit.[11]

A prescription ground face mask is another refractive alternative, as is a face mask with a lens that is bonded onto the surface of the mask. However, masks and lenses may be lost in high swells or rough surf, thereby leaving the diver without refractive correction. When contemplating purchase of an expensive prescription face mask, one needs to be mindful of the corollary of Murphy's Law that applies to diving: "Weight belts always fall on the face masks with prescription lenses."

Refractive corrections for presbyopia present special challenges underwater. Presbyopic divers with contact lens–corrected myopia may require greater near-vision correction underwater than when viewing the same objects in air because of the increased percentage of shorter-wavelength light rays underwater. Divers with presbyopia should consider monovision correction to facilitate underwater visual tasks.[14] Stick-on bifocal lens segments, which may be applied to the inner mask surface, are also available.

REFRACTIVE SURGERY AND DIVING

Laser refractive surgery is a safe and effective means of correcting refractive errors in divers.[17] PRK has been allowed in U.S. Navy divers since 1996. LASIK is now a more common procedure. Although this procedure presents the potential for both inflammation and infection under the corneal flap, as well as traumatic dislocation of the flap, these conditions have not to date been reported as complications of diving. Visual acuity appears to be maintained in the hyperbaric environment. Huang and colleagues[63] reported three patients who had undergone bilateral myopic RK, two who had undergone bilateral myopic LASIK, and three controls. Eye examinations were performed at baseline, at 4 atm in a dry chamber, and on return to the surface. Acute hyperbaric stress did not appear to alter refractive power significantly after corneal surgery.[63]

We recommend a 2-week wait after PRK and 1 month after LASIK before the individual returns to diving.[19]

OCULAR AND PERIOCULAR BAROTRAUMA

The eye is normally filled with noncompressible fluid and solid tissues and therefore protected from barotrauma. However, when a mask is placed over the face, a different circumstance exists. The face mask is an air-filled space that is bounded on one side by the eyes and the ocular adnexa. As a diver descends, if the person does not expel gas through the nose into the air space of the face mask, a relative negative pressure develops in this space. If this negative pressure becomes great enough, the eyes and ocular adnexa are drawn toward the space. Marked lid edema with ecchymosis and subconjunctival hemorrhage may develop as tissues and blood vessels are disrupted by this distention. These signs may be alarming to the diver, but they typically resolve without sequelae. In a more severe case, as may occur when an unconscious diver sinks a significant distance in the water column, more serious injury (e.g., hyphema) may occur.[46] Figure 48-20 shows a diver with face mask barotrauma.

In some settings, facial barotrauma may be quite severe. The first reported "squeeze" that occurred during diving took place on October 4, 1841, as part of the Royal George salvage work off Portsmouth Harbor, England. Ten months later, on the same project, another diver suffered a severe squeeze when his air hose burst. Colonel Pasley, who was the director of salvage operations, told of removing the diver's helmet: ". . . his face and neck were much swollen, and blood was issuing from his mouth and ears profusely, his eye closed and protruding; he vomited a great deal of blood on being laid on the deck." Severe squeezes during the early days of hard-hat diving resulted from a combined failure of both nonreturn valves and air supply hoses.[104]

Barotrauma is also possible in persons with gas bubbles in the anterior chamber or vitreous cavity. Pressure-induced changes

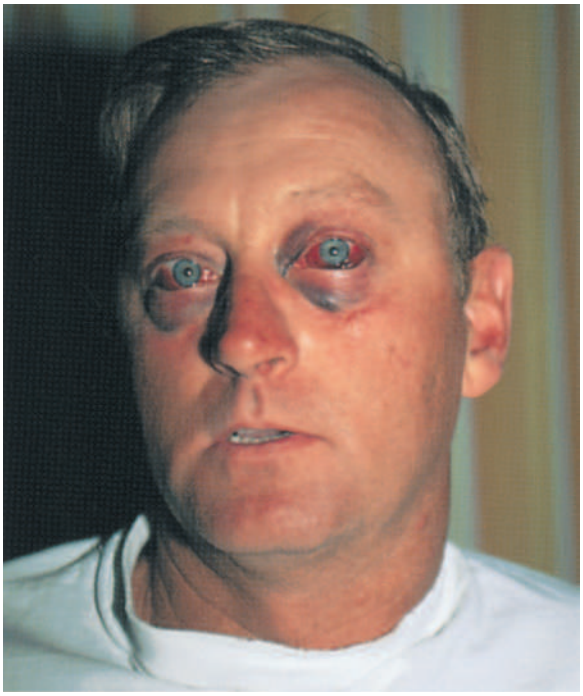

FIGURE 48-20 Mask squeeze in a diver who descended to 45 fsw without exhaling into his mask. *(Courtesy Kenneth W. Kizer, MD.)*

in the volume of this bubble may result in retinal, uveal, or vitreous hemorrhage as well as in partial collapse of the globe. Permanent loss of vision may ensue. One person who attempted to dive while an iatrogenic bubble was present in the vitreous cavity noted immediate onset of very severe eye pain on descent and quickly aborted his dive.[107] Persons with intraocular gas should not be allowed to dive as long as the bubble remains in the eye. The need to add extra gas to the face mask during descent makes it obvious that swim goggles, which cover only the eyes and not the nose, should never be used for diving.

Face mask barotrauma may also result in damage to the periocular tissues. In one report, a diver noted numbness and paresthesias in the distribution of the infraorbital nerve that resulted from maxillary sinus barotrauma.[20] Another diver, who noted the onset of pain during a dive, later experienced a bulging sensation of the eye and diplopia after the dive.[21] Ophthalmologic examination revealed a tropia, and subperiosteal orbital hemorrhage was found with magnetic resonance imaging.

DECOMPRESSION SICKNESS

Ocular involvement in decompression sickness (DCS) was first reported by Sir Robert Boyle, who observed gas bubbles in the anterior chamber of the eye of a viper that had been experimentally exposed to increased pressure.[15] Ocular manifestations of DCS are infrequently reported in the ophthalmic literature,[17] but there are a number of reports of ocular involvement with DCS in the dive medicine literature.[16,17,41,114] Reported manifestations include nystagmus, diplopia, scotomas, homonymous hemianopias, orbicularis oculi pain, cortical blindness, convergence insufficiency, optic neuropathy, and CRAO.[17] The incidence of visual symptoms in patients with DCS was found to be 7% in one large series.[114]

Physicians should also be aware of the possibility of DCS occurring after altitude exposure.[16,133] Altitude DCS that presents as optic neuropathy has been reported.[16] The risk of DCS may be increased if an altitude exposure occurs after diving without allowing for sufficient time for excess nitrogen taken up during the dive to leave the body.[17] Fitzpatrick[43] reviewed 143 cases of altitude-induced type II neurologic DCS. Visual disturbances were identified in 38 cases, for a prevalence of 26.6%. The most common visual symptoms reported were blurred vision (61%), loss of vision (34%), and scintillation or scotoma (32%). Of the cases, 45% occurred at altitude, 24% within the first hour after exposure, and 21% at 1 to 6 hours after exposure. No cases were reported to have begun more than 24 hours after the exposure.[43]

Special attention should be paid to the possibility that DCS after a hypobaric exposure may manifest with symptoms that subsequently resolve, only to recur days to even weeks later. Steigleman and associates[133] reported a 20-year-old aircrew student who had pain on ascent during altitude exposure. Pain resolved on descent but recurred 3 weeks later, when he experienced severe retro-orbital pain and moderate vision loss in the left eye. His signs and symptoms resolved completely in the first U.S. Navy Treatment Table 6, but they recurred several days later. He eventually underwent a total of four treatment tables and experienced complete resolution of his signs and symptoms.[133]

Decompression sickness is treated with oxygen breathing and recompression on an emergency basis. Ophthalmologists seldom encounter this disease in an acute setting, because most divers know to seek recompression therapy for signs or symptoms of DCS. Because treatment with a hyperbaric oxygen treatment table usually results in resolution of all symptoms, most persons with visual symptoms before treatment are asymptomatic after recompression treatment and are therefore not referred to ophthalmologists.[15] If an ophthalmologist encounters a person with acute ocular disturbances that are consistent with DCS after a hyperbaric or hypobaric exposure, the patient should be referred as an emergency to the nearest available recompression chamber and diving medicine specialist, because DCS may worsen rapidly if it is not treated. Physicians who are unsure about the location of the nearest diving medicine specialist or recompression chamber can call the Divers Alert Network at Duke University in Durham, North Carolina, at 919-684-9111.

An incomplete response to treatment or recurrence of symptoms after treatment may bring a patient with ocular DCS to the ophthalmologist on a nonemergency basis. The patient should be managed in conjunction with a dive medicine specialist. Recompression therapy and hyperbaric oxygen should be administered even when a significant delay has occurred between the onset of symptoms and initial patient evaluation, because treatment may be effective despite delays of up to several weeks.[16,17,140]

OCULAR TEAR FILM AS INDICATOR OF DECOMPRESSION STRESS

There has been interest in observing bubble formation in the ocular tear film and using the presence and numbers of bubbles observed after hyperbaric exposures as an indicator of the magnitude of decompression stress encountered. Nine healthy adult volunteers made a series of dives to 50 fsw and 90 fsw.[93] In the first series, all divers spent the 15-minute bottom time resting, whereas on the second series, they exercised on a bicycle ergometer. Dive profiles were accomplished in accordance with the Canadian Defense and Civil Institute of Environmental Medicine tables. On the resting profiles, the divers were found to have no bubbles on postdive eye examination, whereas on the exercising profiles, bubbles were present.[93] In another study, 11 divers completed two series of chamber dives in accordance with the Professional Association of Diving Instructors tables.[91] The first dive series was to 70 fsw for 15, 29, and 40 minutes. The second was maximum-duration no-stop diving at 40 fsw (140 minutes), 70 fsw (40 minutes), 90 fsw (25 minutes), and 120 fsw (13 minutes). Tear film bubble assessment was observed to be more sensitive for detecting decompression stress than was the conventional Doppler ultrasonic surveillance of the precordial region.[91]

Bennett and colleagues[9] observed 42 recreational divers after a single day of diving and 11 divers after repetitive multiday diving. After diving, bubble counts increased significantly ($p < 0.01$) compared with predive values. Single-day divers reached a maximum bubble count at 48 hours of 1 bubble per eye (range, 0 to 2.25 bubbles). Bubble counts were not significantly correlated with inert gas load, body mass index, age, or diving experience.[9] Another study subjected 11 volunteers to pressures of 4 bar for 15 minutes. During the predive examinations, there was only one individual with one bubble observed. Postdive examinations found a mean of 6 bubbles per eye (range, 3 to 12 bubbles; $p < 0.001$).[135] Whether these observations will eventually be useful for clinicians or decompression researchers remains to be determined.[8]

OCULAR FUNDUS LESIONS IN DIVERS

Fluorescein angiography of divers has documented retinal pigment epithelial abnormalities that are indistinguishable from those that are seen in eyes with choroidal ischemia.[109] These changes were attributed to decompression-induced intravascular gaseous microemboli. The incidence of these lesions was related to the duration of diving and a history of DCS. Other abnormalities that were noted more frequently in divers were low retinal capillary density at the fovea, microaneurysms, and small areas of capillary nonperfusion. Retinal pigment epithelium defects were seen in 1 of 23 nondivers and in one-half the divers. All observed changes were noted to be consistent with obstruction of the retinal and choroidal circulations. Although no diver was reported to have loss of visual acuity as a result of these abnormalities, the long-term effects of this phenomenon remain to be studied.

Another study reported fundus fluorescein angiography performed on 26 divers who had reportedly used safe diving practices for at least 10 years and on seven controls. There was no significant difference in the incidence of macular abnormalities between divers and nondivers in this study. The authors concluded that adherence to safe diving practice confers some protection against the macular abnormalities known to occur in divers with a history of DCS.[61]

A third study compared fluorescein angiograms of 55 British Royal Navy divers with those of 24 nondivers. No differences were found between divers and nondivers, and prevalence of abnormalities was not correlated with diving experience. Visual fields were normal in all controls and divers. Of both controls and divers, 25% had retinal pigment epithelium defects. The authors believed that their divers, as members of the Royal Navy, may have followed safer diving practices and thus avoided the lesions that were observed in other studies.[96]

ARTERIAL GAS EMBOLISM

Retrochiasmal defects such as hemianopias and cortical blindness may be seen with arterial gas embolism. CRAO may also result from gas emboli in the ophthalmic artery.[17] Management is similar to that for DCS, with emergency recompression and HBOT indicated in all cases.

HYPEROXIC MYOPIA

Individuals who undergo prolonged or repeated exposures to hyperoxic gas mixtures may experience lenticular oxygen toxicity that is initially manifested by a myopic refractive shift. This myopic shift is progressive and usually reversible if the hyperoxic exposures are discontinued.[2] Although this condition is most common among patients who are undergoing repeated hyperbaric oxygen exposures in a chamber for medical conditions,[2,4] it has also been reported in a scuba diver doing a series of prolonged dives using a gas mix with a constant partial pressure of oxygen (P_{O_2}) of 1.3 atm.[26] This condition has also been noted among caisson workers.[103]

OCULAR JELLYFISH STINGS

Divers and swimmers may occasionally be stung by jellyfish on and around the eyes. The pain from this injury may be severe, but is typically self-limited, and usually resolves within 24 to 48 hours. Glasser and colleagues[51] described a series of five cases associated with unusually severe and prolonged iritis and IOP elevation. IOP ranged from 32 to 48 mm Hg and was treated successfully with topical β-blockers and oral carbonic anhydrase inhibitors. One patient developed chronic unilateral glaucoma. The iritis persisted for up to 8 weeks. This report illustrates the potential for serious long-term sequelae as a result of ocular jellyfish stings. All patients with ocular jellyfish stings that do not resolve within 24 to 48 hours should be seen urgently by an ophthalmologist.[51]

DIFFERENTIAL DIAGNOSIS OF DECREASED VISION AFTER DIVING

Decompression sickness and arterial gas embolism should be considered whenever vision is acutely decreased after diving because of the possible emergency need for recompression therapy, especially if any other manifestation of DCS or arterial gas embolism is present. Other disorders may also affect vision after a dive. Corneal edema that results from formation of gas bubbles under polymethylmethacrylate and rigid gas-permeable contact lenses may cause decreased vision. An individual who wears soft contact lenses and who complains of blurred vision after a dive may have a lost or displaced lens.

Another possible cause of nondysbaric decreased vision after a dive is *epithelial keratopathy* induced by chemical agents used to reduce face mask fogging. The time-honored application of saliva or toothpaste to the interior surface of the mask reduces but does not eliminate fogging. This led to development of commercial antifog agents designed to be applied to the inside surfaces of face masks. These agents may contain volatile compounds that are potentially toxic to the corneal epithelium, including glycols, alcohols, and phenol derivatives. Exposure to these compounds may result in blurred vision, photophobia, tearing, and blepharospasm that may not develop until several hours after the dive.[154] Slit-lamp examination typically reveals diffuse superficial punctate keratopathy. Development of this syndrome often results from improper use of the antifog agent, such as overly generous application or the failure to rinse the mask before use.

One of the authors (FKB) has personally treated several persons with recurrent mild ocular irritation and blurring of vision after dives during which soft contact lenses were worn. The lenses were noted to be tightly adherent to the cornea, probably as a result of a decrease in water content in the lens after contact with hypertonic seawater. Symptoms were relieved with a few drops of isotonic artificial tears. Decreased movement of soft contact lenses on the cornea has been reported to occur after exposure to swimming pool water as well.[34]

Divers sometimes use a transdermal scopolamine patch placed behind the ear to prevent motion sickness. This may result in mydriasis, decreased accommodation, and blurred vision in the ipsilateral eye. If proper application technique is not used and scopolamine is inadvertently transferred to the eyes, ipsilateral, contralateral, or bilateral symptoms will be seen. Hyperoxic myopia may be a cause of vision loss if the diver has had recurrent or prolonged exposures to elevated P_{O_2}.[26]

Divers often use oxymetazoline nasal spray to assist with shrinking mucous membranes to equalize pressure in the middle ear. Vasoconstriction caused by this medication has been reported to cause anterior ischemic optic neuropathy. Fivgas and Newman[44] described a 43-year-old woman evaluated for sequential bilateral vision loss after using oxymetazoline nasal spray. Visual loss in both eyes was temporally associated with use of the medication. A thorough evaluation for other causes of optic neuropathy was negative.

Migraine-like phenomena have been reported after hyperbaric exposures. Anderson and co-workers[3] described four individuals who had migrainous signs or symptoms after hyperbaric exposure. Three of the four individuals had a history of migraine headaches and noted the sudden appearance of scintillating scotomas 10 to 90 minutes after surfacing, with no other manifestations of DCS. Three of the four patients experienced resolution of symptoms with surface oxygen only, and one patient was recompressed as well. These events may represent a migraine event that is temporally associated with diving, or may be an atypical manifestation of DCS.[3]

Lastly, loss of vision may be caused by ocular disorders that occurred during or shortly after the dive but that were not a direct result of the dive itself. Box 48-5 presents a differential diagnosis of decreased vision after diving.

BOX 48-5 Differential Diagnosis of Decreased Vision after Diving

- Decompression sickness
- Arterial gas embolism
- Bubbles under contact lenses
- Displaced contact lens
- Antifog agent keratopathy
- Contact lens adherence syndrome
- Transdermal scopolamine
- Hyperoxic myopia
- Oxymetazoline optic neuropathy
- Diving-induced migraine phenomena
- Eye disorders not related to diving

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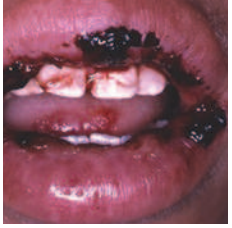

CHAPTER 49

Wilderness Dentistry

GEORGE R. DEEB AND AMBER M.H. JOHNSON

Dental emergencies are common medical ailments that cause evacuation of a patient from a wilderness expedition.[26] Dental emergencies range from minor trauma to severe dental infections that can be life threatening. Basic knowledge of dental conditions and dental analgesia, and pretrip planning are important for anyone venturing into the backcountry.

DENTAL ANATOMY

Adult (permanent) dentition is composed of 32 teeth: 16 in the maxilla (upper jaw) and 16 in the mandible (lower jaw). Permanent dentition is divided into four classes: incisors, canines, premolars, and molars. Incisors and canines are anterior teeth. Premolars and molars are posterior teeth (Figure 49-1). The entire dentition can be evaluated on a panoramic radiograph (Figure 49-2).

Pediatric (primary) dentition is composed of 20 teeth. Primary teeth begin erupting as early as 6 months of age. Complete primary dentition is typically present from 2 to 6 years of age (Figure 49-3). At 6 years of age, permanent dentition begins to erupt and primary teeth begin to exfoliate, resulting in the mixed-dentition phase. The mixed-dentition phase can be evaluated for proper tooth development and exfoliation with panoramic radiographs (Figure 49-4).

Each tooth has a crown (portion of the tooth above the gumline) and a root (portion of the tooth below the gumline) (Figure 49-5). The crown's outermost part is made of enamel, the hardest substance in the human body. The root's outermost part is made of cementum. Dentin lies below enamel and cementum, and surrounds pulpal tissue (the neurovascular bundle). If pulpal tissue becomes exposed or inflamed, it is painful. Tubules within dentin extend to pulpal tissue. Exposure of these tubules can result in pain to stimuli (e.g., hot, cold, pressure).

The following tooth-numbering systems are used:

- International Numbering System (Figure 49-6)
- Pediatric International Numbering System (Figure 49-7)
- Universal Numbering System (used in the United States) (Figure 49-8)

HISTORY AND EXAMINATION

As for any medical emergency, begin evaluation with a problem-based history and examination of the wilderness patient with dental pathology. Elicit the chief complaint and history of present illness to formulate a diagnosis and reveal complicating medical factors (e.g., presence of conditions requiring antibiotic prophylaxis, bleeding disorders, immunocompromised states, allergies, current medications) that may alter treatment.

Begin with a broad solicitation of the patient's complaint and then ask more specific questions. Are you having any pain or difficulty swallowing? Do you have a sore throat, voice change, or difficulty breathing? Does your bite feel different? Did pain begin suddenly or gradually? Was there any trauma? Do you have any numbness in your face or jaw? Is your pain localized or diffuse?

Examination should proceed in systematic manner to allow efficient collection of all relevant data. Examination begins with a general appraisal of the patient that optimally includes vital signs. First, note signs of airway compromise (e.g., significant facial swelling with possible tracheal deviation, inability to swallow saliva, voice changes, difficulty breathing). Observe the head, neck, and face for any asymmetry. Palpate for evidence of point tenderness, crepitus, foreign bodies, or swelling. Palpate the temporomandibular joints in the preauricular area, muscles, lymph nodes, and areas of suspected injury. Have the patient slowly open and close the mouth. Observe for the extent of opening, range of motion, and deviation on opening.

Intraoral examination begins with the lips, then cheeks, floor of mouth, tongue, hard palate, soft palate, and pharynx. Inspection and palpation are critical. Masses may be hard (e.g., bone) or fluctuant (e.g., abscess). Bimanual palpation is helpful when examining the lips, cheeks, and floor of the mouth. Observe the gingivae for color, firmness, recession, and swelling. Inspect all teeth for caries, fractures, presence of plaque and calculus, wear, and loose restorations. Test tooth mobility by using moderate force in a side-to-side direction. Gently percuss teeth with a metal instrument to reveal injury or disease of supporting structures. Test individual teeth with a cold object to evaluate for increased sensitivity or lingering pain that can indicate tooth necrosis. Inspect for malocclusion by retracting the lips and asking the patient to bite down. Is there pain when biting on a specific tooth, or do teeth not come together normally? If there is malocclusion, evaluate the position of individual teeth. Are they displaced? Is there a large space or step-off between adjacent teeth? Radiographs and other technology-dependent diagnostic tests are valuable, but are assumed to be unavailable in a wilderness setting. Therefore, the history and physical examination are essential.

Boxes 49-1 to 49-3 list American Heart Association recommendations for antibiotic prophylaxis (to prevent subacute bacterial endocarditis [SBE]). Premedication is no longer recommended for patients with most types of heart murmurs.[53] American Academy of Orthopaedic Surgeons and American Dental Association (ADA) Clinical Practice Guidelines state, "practitioner(s) might consider discontinuing the practice of routinely prescribing prophylactic antibiotics for patients with hip and knee prosthetic joint implants undergoing dental procedures." This recommendation had limited supporting evidence.[38] If a patient has a history of joint replacement, decide whether to premedicate for dental procedures after discussion with the orthopedic surgeon. In the field, and in the absence of such guidance, it is reasonable to administer prophylactic antibiotics.

DENTAL TRAUMA

Wilderness recreation creates many opportunities for orofacial injury. Dental injuries are the most common type of orofacial injuries and can be seen in all types of recreational athletes. The ADA and International Academy of Sports Dentistry recommend use of mouth guards for participation in 29 sports.[2] A recent survey of consecutive trauma patients admitted to an oral and maxillofacial surgery service in Innsbruck, Austria, found that 31% had sports-related injuries. Of those, 44% could be considered "backcountry" trauma, with the majority related to skiing and snowboarding.[50] A study of children (5 to 18 years of age) found the most common sports-related injury locations to be wrist/hand (28%), head/face (22%), and ankle/foot (18%).[47]

Quickly evaluate the injured patient according to current advanced trauma life support (ATLS) and advanced wilderness life support (AWLS) protocols. The primary survey identifies and corrects any inadequacy in respiration or circulation. Examine the mouth and pharynx for foreign bodies (e.g., blood clots; tooth or bone fragments; displaced dentures, crowns, or bridges). Sufficient, sustained suction to clear the oropharynx promptly is

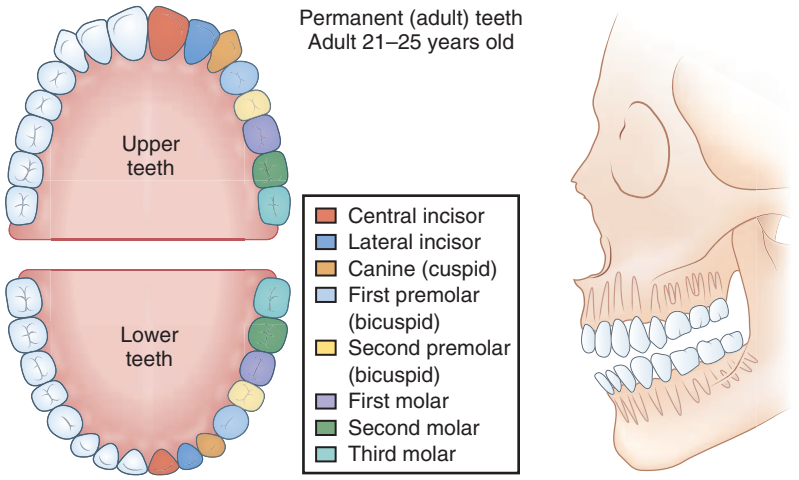

FIGURE 49-1 Adult dentition. *(Redrawn from A.D.A.M.: Dental x-rays.* http://printer-friendly.adam.com/content.aspx?productId=117&pid=1&gid=003801&c_custid=758.*)*

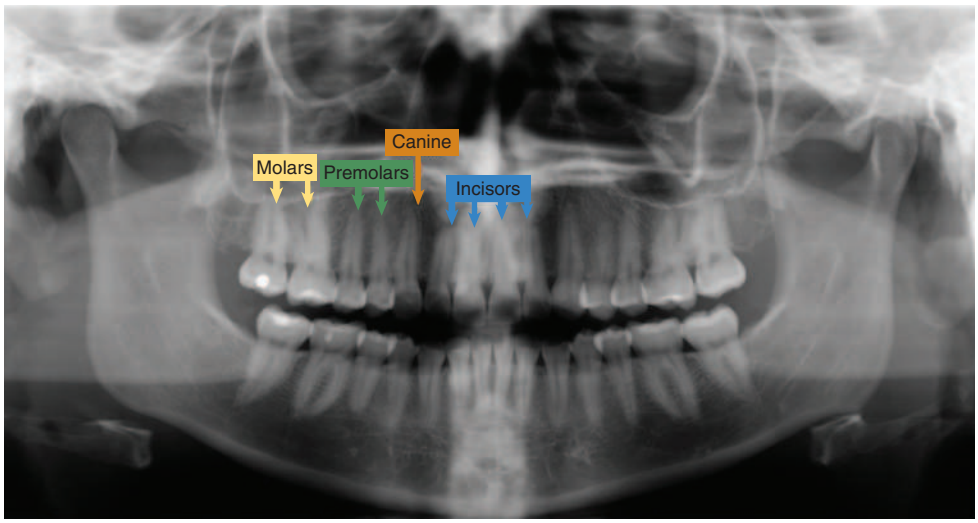

FIGURE 49-2 Panoramic radiograph of adult dentition.

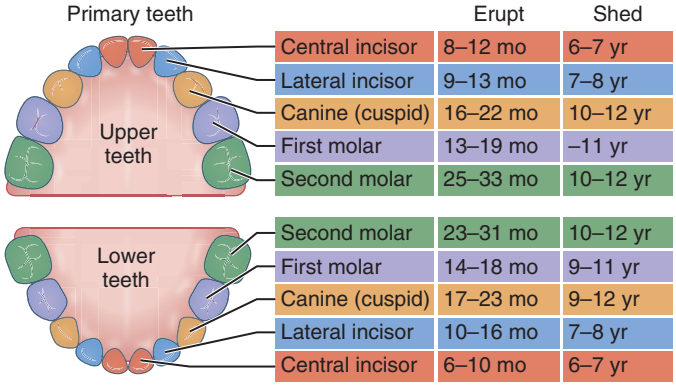

FIGURE 49-3 Pediatric dentition. *(Redrawn from* http://www.pediatricdentisthoustontexas.com/dental-topics/*.)*

rarely available in austere environments. Remove foreign bodies digitally or with any immediately available implement. If the airway is obstructed, perform a chin lift or jaw thrust to help lift the tongue from the posterior pharynx (see Chapter 19). An oropharyngeal or nasopharyngeal airway may be used to help maintain the airway. If a cervical spine fracture is suspected, limit neck movement. If not, position the patient in the lateral decubitus "recovery" position to facilitate airway patency and breathing. If these measures fail to achieve an adequate airway, advanced management may be necessary.

When the airway is secured and patient is hemodynamically stable, perform a secondary survey. Inquire about the time and nature of the accident, symptoms, loss of consciousness, nausea, vomiting, visual disturbances, neck pain, paresthesias, and headache. A baseline mental status examination and neurologic exam (including cranial nerves) should be performed. Cleanse the face, mouth, head, and neck of blood and debris to unmask soft tissue injuries and facilitate diagnosis. Gently pull back the lips with teeth closed to examine soft tissues and assess occlusion. Note malocclusion or gross displacement of tooth segments that may indicate a mandibular or alveolar fracture. Examine all lacerations carefully to rule out retained foreign material or penetration through the lip(s). Dry the teeth with gauze and examine for

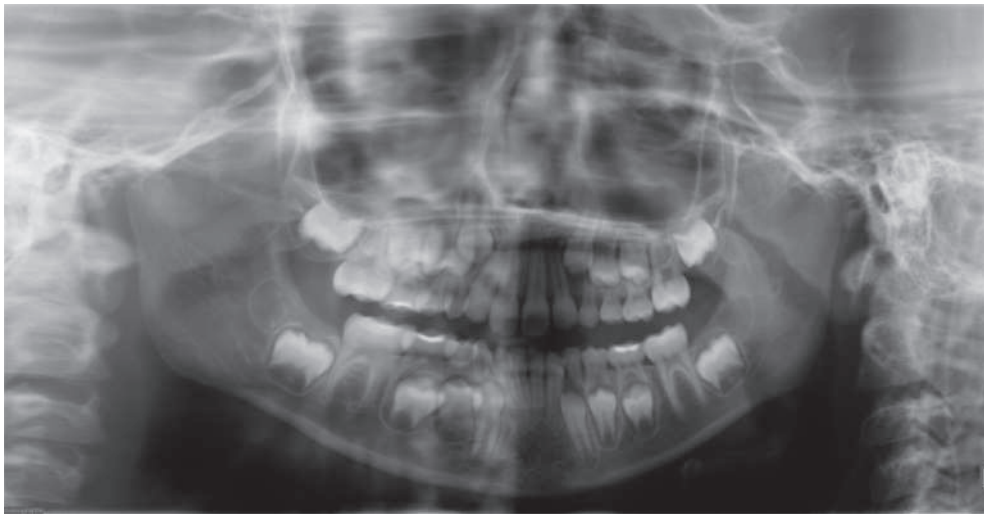

FIGURE 49-4 Panoramic radiograph of mixed dentition.

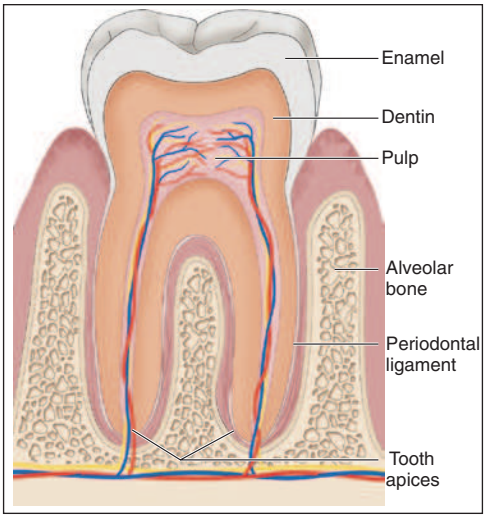

FIGURE 49-5 Anatomy of the tooth. *(From Buttaravoli P, Leffler S. Minor emergencies, 3 ed, Philadelphia, Elsevier, 2012.)*

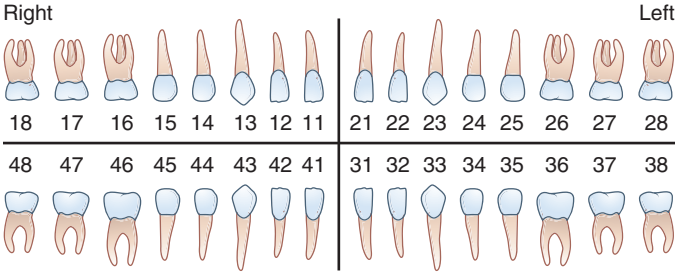

FIGURE 49-6 International Numbering System (numbered looking at the patient). *(Redrawn from* http://www.mouthandteeth.com/anatomy/teeth-names-numbers.htm.*)*

Upper Right					Upper Left				
55	54	53	52	51	61	62	63	64	65
85	84	83	82	81	71	72	73	74	75
Lower Right					Lower Left				

FIGURE 49-7 Pediatric International Numbering System. *(Redrawn from* http://what-when-how.com/dental-anatomy-physiology-and-occlusion/introduction-to-dental-anatomy-dental-anatomy-physiology-and-occlusion-part-1/.*)*

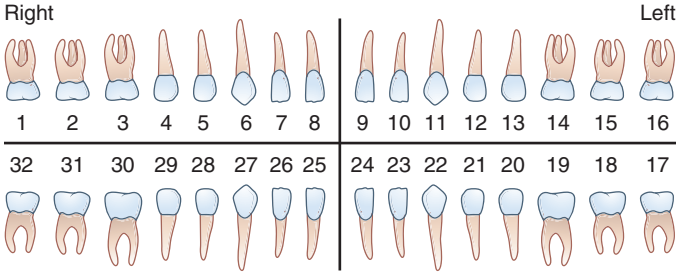

FIGURE 49-8 Universal Numbering System. *(Redrawn from* http://www.emergucate.com/2012/10/12/a-tooth-by-any-other-name-a-quick-overview-of-dental-nomenclature/.*)*

BOX 49-1 Antibiotic Prophylaxis for Dental Procedures in Patients With Cardiac Conditions

Antibiotic prophylaxis for dental procedures is reasonable only for patients with cardiac conditions associated with the highest risk for adverse outcomes from endocarditis.

Prosthetic cardiac valve or prosthetic material used in valve repair

Previous endocarditis

Congenital heart disease only in the following categories:

- Unrepaired cyanotic congenital heart disease, including those with palliative shunts and conduits
- Completely repaired congenital heart disease with prosthetic material or device, whether placed by surgery or catheter, during the first 6 months after the procedure (prophylaxis is reasonable because endothelialization of prosthetic material occurs within 6 months after the procedure)
- Repaired congenital heart disease with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization)

Cardiac transplantation recipients with cardiac valvular disease

Data from Wilson W, Taubert KA, Gewitz M, et al: Prevention of infective endocarditis: Guidelines from the American Heart Association—A guideline from the American Heart Association Rheumatic Fever, Endocarditis and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group, *J Am Dent Assoc* 139:3S, 2008.

BOX 49-2 Dental Procedures for Which Antibiotic Prophylaxis is Reasonable and is Not Recommended in Patients With Cardiac Conditions*

Antibiotic prophylaxis is reasonable in all dental procedures that involve:
- Manipulation of gingival tissue or the periapical region of teeth
- Perforation of the oral mucosa

Antibiotic prophylaxis is NOT recommended for the following dental procedures or events:
- Routine anesthetic injections through noninfected tissue
- Taking dental radiographs
- Placement or adjustment of removable prosthodontic or orthodontic appliances
- Placement of orthodontic brackets
- Shedding of deciduous teeth
- Bleeding from trauma to the lips or oral mucosa

Data from Wilson W, Taubert KA, Gewitz M, et al: Prevention of infective endocarditis: Guidelines from the American Heart Association—A guideline from the American Heart Association Rheumatic Fever, Endocarditis and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group, *J Am Dent Assoc* 139:3S, 2008.
*See Box 49-1.

BOX 49-4 Classification of Dental Trauma

Injuries to Hard Dental Tissues and Pulp
Crown infraction
Uncomplicated crown fracture
Complicated crown fracture
Uncomplicated crown-root fracture
Complicated crown-root fracture
Root fracture

Injuries to Periodontal Tissues
Concussion
Subluxation
Intrusive luxation
Lateral luxation
Extrusive luxation
Exarticulation (avulsion)

Injuries to Supporting Bone
Comminution of alveolar socket
Fracture of alveolar socket wall
Fracture of alveolar process
Fracture of jaw

Injuries to Soft Tissues

From Andreasen JO, Andreasen FM: *Textbook and color atlas of traumatic injuries to the teeth,* ed 3, St Louis, 1994, Mosby.

fractures or pulp exposure. Tap each tooth with an instrument handle to detect hypersensitivity. Palpate the bones of the maxilla and mandible for tooth root (i.e., apical) tenderness. Tenderness on percussion (tapping) and palpation indicates tooth or periodontal injury. Test each tooth for abnormal mobility. Advanced diagnostic tests, such as electrical pulp vitality testing, dental radiographs, and soft tissue radiographs, may need to be obtained after evacuation.[34] (See Chapter 20 for diagnosis and treatment of facial injuries.)

Box 49-4 provides a classification of traumatic injuries to teeth and supporting structures. Injuries often occur in combination, with one or more teeth and surrounding structures exhibiting multiple injuries. In austere environments, proper emergency care improves prognosis and makes patients more comfortable. Definitive treatment of dental injuries will often be delayed after evacuation because of limited instrumentation, but specific steps may be initiated in the field that will improve outcomes and perhaps provide some relief from discomfort.

BOX 49-3 Antibiotic Prophylactic Regimens for Dental Procedures (Single Dose 30 to 60 Minutes Before Procedure)

Oral Amoxicillin
Adults: 2 g
Children: 50 mg/kg

If Unable to Take Oral Medication
Adults: ampicillin, 2 g IM or IV, or cefazolin or ceftriaxone, 1 g IM or IV
Children: ampicillin, 50 mg/kg IM or IV, or cefazolin or ceftriaxone, 50 mg/kg IM or IV

Allergic to Penicillins or Ampicillin
Cephalexin*
Adults: 2 g PO
Children: 50 mg/kg PO
OR Clindamycin
Adults: 600 mg PO
Children: 20 mg/kg PO
OR Azithromycin or clarithromycin
Adults: 500 mg PO
Children: 15 mg/kg PO

Allergic to Penicillins or Ampicillin and Unable to Take Oral Medication
Cefazolin or ceftriaxone
Adults: 1 g IM or IV
Children: 50 mg/kg IM or IV
OR Clindamycin
Adults: 600 mg IM or IV
Children: 20 mg/kg IM or IV

Data from Wilson W, Taubert KA, Gewitz M, et al: Prevention of infective endocarditis: Guidelines from the American Heart Association—A guideline from the American Heart Association Rheumatic Fever, Endocarditis and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group, *J Am Dent Assoc* 139:3S, 2008.
IM, Intramuscularly; *IV,* intravenously; *PO,* orally.
*Or other first- or second-generation oral cephalosporin in equivalent adult or pediatric dosage.

SPECIFIC DENTAL INJURIES

Specific dental injuries are categorized by the structures involved.[11,14] Injuries may be limited to teeth or may involve supporting structures, such as periodontium. Injuries to surrounding soft tissues should be examined for debris and copiously irrigated to decrease risk of infection. The International Association of Dental Traumatology website describes diagnosis and treatment of these injuries: http://www.dentaltraumaguide.org/.

Crown Infraction

Crown infraction is an incomplete fracture or crack in the enamel with no loss of tooth structure. Tiny cracks may be visible in the tooth. Reassure the patient. Treatment is not necessary.

Uncomplicated Crown Fracture

Uncomplicated crown fracture involves enamel or enamel-dentin. The tooth is fractured, but no pulp tissue is visible. This can be an Ellis Class 1 fracture (only enamel) or an Ellis Class 2 fracture (enamel and dentin). The tooth may be sensitive to cold, but other exam findings are within normal limits. Save any broken fragment if possible. No emergency treatment is absolutely necessary, but irritating sharp edges can be smoothed with a fingernail file. If thermal sensitivity is moderate to severe, apply temporary filling material.

Complicated Crown Fracture

Complicated crown fracture describes disruption of enamel and dentin with pulp exposure. This is an Ellis Class 3 fracture. All levels of the tooth (enamel, dentin, and pulp) are involved. Small pulp exposures that are not grossly contaminated should be capped with calcium hydroxide (Dycal) or Intermediate Restorative Material (IRM). IRM is a reinforced zinc oxide–eugenol composition for intermediate restorations intended to last for up

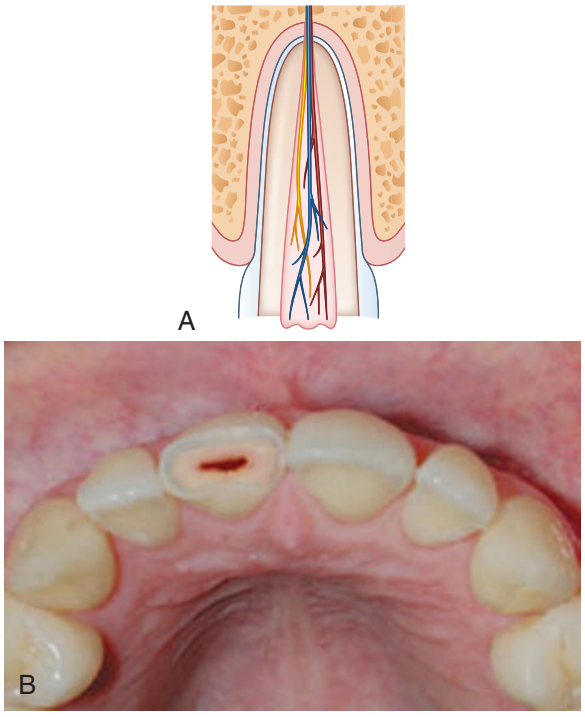

FIGURE 49-9 **A,** Complicated crown fracture. **B,** Complicated crown fracture seen from an occlusal view. *(Modified from* www.dentaltraumaguide.org/Permanent_enamel-dentin-pulp_fracture_Description.aspx.*)*

to 1 year. If the exposure is larger than 1 mm or if pulp tissue has been exposed for more than 24 hours, amputate approximately 2 mm of the pulp with a sharp, sterile instrument. If bleeding continues for more than a few minutes, use a cotton pellet soaked in local anesthetic solution, hydrogen peroxide, or Dycal to obtain hemostasis. Fill the top of the canal with Dycal or IRM. Protect the tooth as for a crown fracture (Figure 49-9).

Crown-Root Fracture

Crown-root fractures involve enamel, dentin, and cementum. Pulp may or may not be exposed. The tooth has been fractured obliquely, resulting in a mobile fragment attached to gingiva. The pulp may be exposed (Figure 49-10). Remove the mobile fragment. Treat pulp exposure as a complicated crown fracture.[14] It is possible to glue tooth fragments together temporarily with 2-octyl cyanoacrylate (Dermabond).[17]

Root Fracture

Root fractures involve dentin, cementum, and pulp and may be difficult to diagnose without radiographs. The crown may be mobile or malpositioned, although this can be caused by extrusion of the entire tooth or by root fracture with luxation of the coronal (i.e., portion of tooth exposed above gingiva) portion. Reposition the tooth as precisely as possible, and splint rigidly (see Splinting, later). Hard tissue union of the fragments usually occurs within 3 months. If the tooth's coronal portion proves impossible to stabilize and definitive treatment is days away, remove the mobile fragment. Do not attempt to extract the apical fragment.

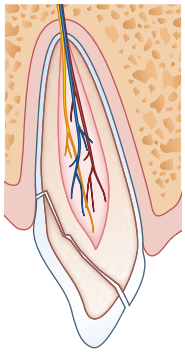

FIGURE 49-10 Crown-root fracture. The smaller, mobile fragment should be removed. The larger fragment should be left in place. *(Redrawn from* http://www.dentaltraumaguide.org/Permanent_Crown-root_fracture_without_pulp_involvement_Description.aspx.*)*

Injuries to the Periodontium

Periodontium is the dentition's support system and consists of gingiva, mucosa, periodontal ligament, and alveolar bone. Injuries to these structures can cause sensitivity to percussion, as well as mobility or even loss of teeth. Close follow-up with a dentist is recommended to monitor teeth after injury because of possible pulpal necrosis, which should be treated with root canal therapy. If mobile after injury, teeth may need to be splinted to stabilize the tooth or bony segment during healing. A flexible/nonrigid splint allows physiologic movement of the tooth and helps prevent ankylosis (tooth fusing to bone). This allows formation of a normal fibrous union between the tooth and bone. Splinting is used for avulsed or luxated teeth. A rigid or semirigid splint minimizes movement of the tooth and bone to facilitate hard tissue union. It is used when the alveolar bone or roots are fractured.

Concussion. Concussion is injury to the tooth's supporting structures (periodontal ligament, bone, and gingiva) without mobility or displacement. Treat with soft diet and analgesics as needed. Follow-up with a dentist is recommended because pulpal necrosis is possible.

Subluxation. Subluxation is injury to the periodontium resulting in abnormal tooth mobility, but no displacement. Sulcular bleeding may be present. A flexible splint may be applied for up to 2 weeks for comfort. The opposing tooth can be smoothed with a file to eliminate any occlusal interferences.

Intrusion. With intrusion, the tooth has been driven into bone by a vertical force. It typically appears shortened. For a permanent tooth, the tooth should be repositioned manually/surgically or orthodontically. After the tooth is repositioned, apply a splint. Analgesics and a soft diet are important. Endodontic treatment of permanent teeth (to prevent inflammatory root resorption and infection) should begin within 3 weeks of injury. If a primary tooth is intruded, emergency treatment is palliative only.

Extrusion. With extrusion, the tooth is partially displaced from the socket (partial avulsion) and grossly mobile. The tooth also appears elongated. Gentle, steady pressure is used to reposition the tooth. This allows time to displace blood that has collected in the apical region of the socket. After reduction, the tooth is nonrigidly splinted for up to 2 weeks.

Lateral Luxation. In lateral luxation, the tooth is displaced in a direction other than vertically. The tooth is often displaced by a horizontal blow. It is frequently not mobile, because the tooth's apex is impacted within alveolar bone. Tooth displacement is accompanied by fractures of surrounding alveolar bone. This injury and its treatment are painful. Two fingers are used to reduce the tooth (Figure 49-11). One finger guides the apex down and back, while the other repositions the crown. This requires judicious, but firm, application of force. The tooth may snap back into position and be quite stable. Check the tooth the next day. Breakdown of marginal bone and inflammation may cause the tooth to become mobile. Prolonged splinting may be needed if marginal bone breaks down.

Avulsion. In avulsion, or exarticulation, a tooth is completely displaced from its socket. Following total avulsion, prognosis after replantation depends on the length of time that the tooth has been out (i.e., "dry time"), health of periodontal ligament cells on the tooth's root, overall health of periodontium, amount of alveolar bone disruption, and amount of contamination.[34] Minimize dry time and reimplant the tooth as soon as possible. Immediate replacement is ideal. Teeth reimplanted within 20 minutes have the best prognosis. Teeth out for more than 2 hours have a poor prognosis.[34] If the avulsed tooth must

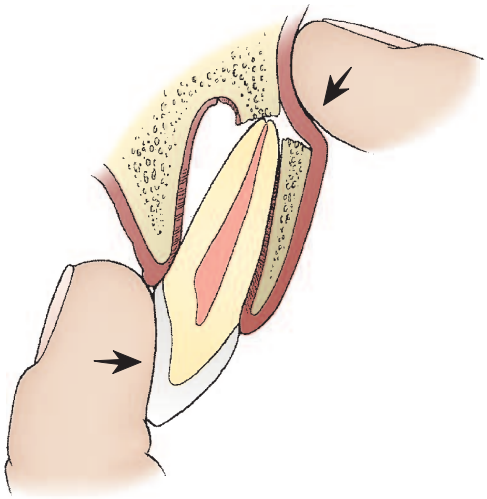

FIGURE 49-11 Two fingers are used to reduce a lateral luxation.

be stored, keep it moist. Ideal transport media include Hank's balanced salt solution (HBSS) or saline solution. Alternative transport media are whole milk, the patient's saliva, sports drinks, and water (in order of preference). The goal is to preserve the periodontal ligament cells in the most isotonic, pH-balanced solution available until the tooth can be reimplanted. If capable of doing so safely, the patient may store the tooth in the mouth (underneath tongue or in vestibule of cheek). Gently rinse with saline to remove debris. Never scrub, curette, or use disinfectant on the root surface. When removing clotted blood from the socket, use gentle irrigation and suction. Avoid scraping socket walls. Examine the socket for fracture of the alveolar wall. If fractured bone is present, reduce it with a blunt instrument. Ease the tooth into place with slow, continuous pressure (Figure 49-12). After replantation, splint the tooth nonrigidly for 7 to 10 days. After splinting, suture any gingival lacerations. Administer an antibiotic (e.g., penicillin, clindamycin) for 7 to 10 days, and give appropriate tetanus prophylaxis. The tooth will likely need endodontic therapy and should be evaluated within 2 weeks.[34]

The preferred approach to tooth avulsion or severe luxation is immediate reduction in the field, followed by evacuation for definitive treatment. The next most desirable option is to store the tooth properly and transport the patient and tooth for replantation within hours of injury. If the extraoral dry time exceeds 60 minutes, periodontal ligament cells are presumed necrotic and should be removed by curettage or acid etching. Treat the root before replantation, ideally with sodium fluoride 2% or enamel matrix proteins (Emdogain).[49] If field conditions prevent these treatments, delayed replantation procedure may be indicated. Store the tooth dry, and visit a dentist as soon as possible.

Comminution of Alveolar Socket or Fracture of Alveolar Socket Wall

The bone of the alveolar socket is fractured into one or more segments. Stabilize the associated teeth.

Dentoalveolar Fracture

Fractures of supporting alveolar bone results in displacement of two or more teeth as a unit (Figure 49-13). Teeth are not mobile with respect to one another. The segment is repositioned (this may be painful even with local anesthesia). Place rigid splinting for 4 to 6 weeks.

Soft Tissue Injuries

Wounds of oral mucosa and the face should be treated after repair of dental injuries and jaw fractures. Time is more critical when treating intraoral, hard tissue injuries. Also, lacerations are likely to be reopened if closed before intraoral manipulations. See Chapter 20 for diagnosis and treatment of facial injuries and Chapter 21 for wound care.

Patient Instructions

After any of the intraoral injuries previously described, the patient should be placed on a soft diet for 10 to 14 days, instructed to

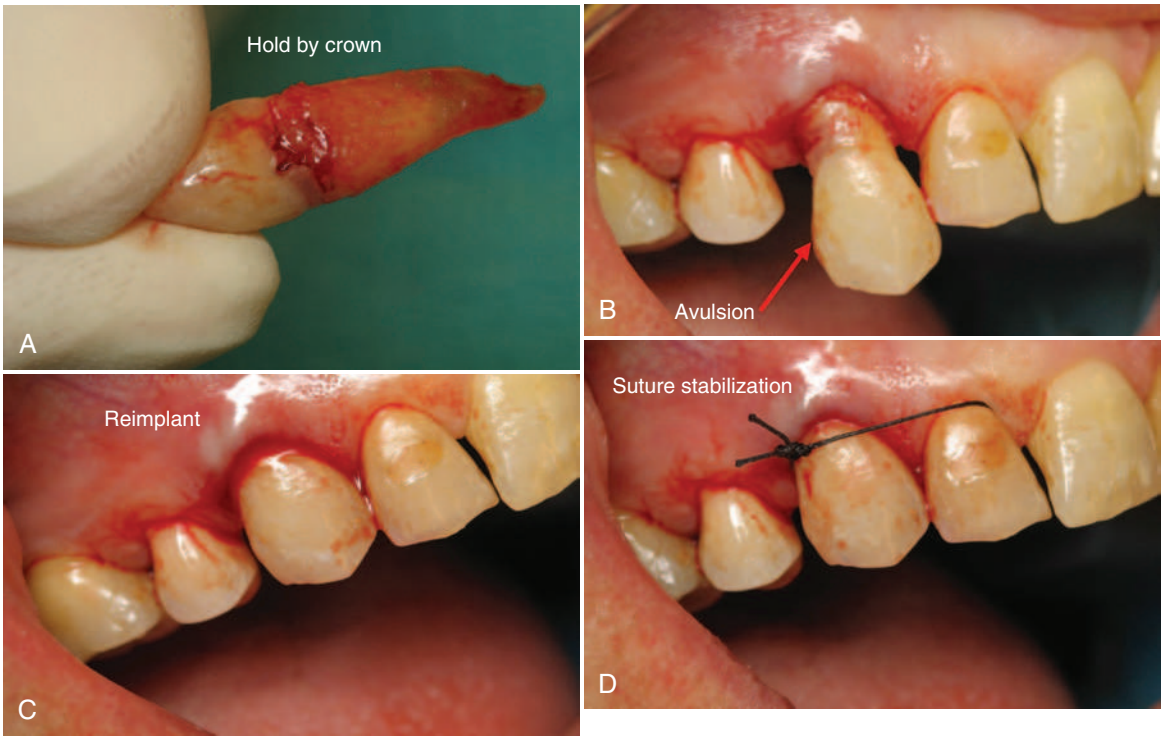

FIGURE 49-12 **A,** Avulsed tooth. **B,** Tooth being replanted. **C,** The tooth has been replanted and is in proper anatomic position. **D,** Suture is used to stabilize the replanted tooth.

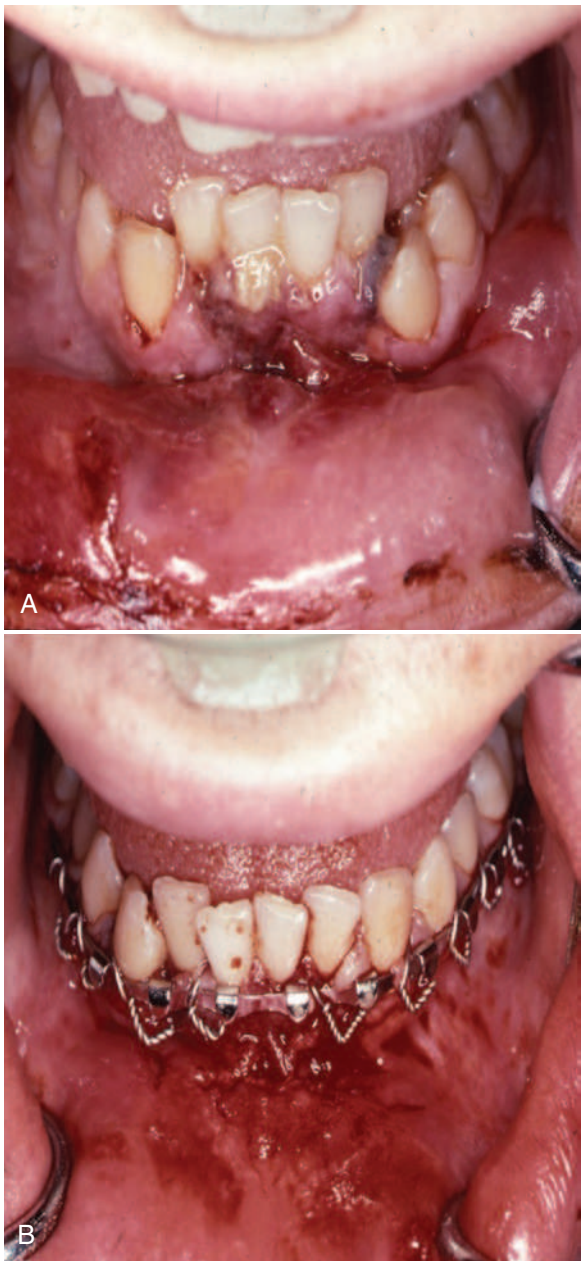

FIGURE 49-13 **A,** Dentoalveolar fracture. **B,** Rigid splinting of dentoalveolar fracture with arch bars.

brush gently, but thoroughly, with a soft toothbrush after every meal, and rinse twice each day with a chlorhexidine-containing mouthwash or warm saline.

Proper Reduction

It is sometimes difficult to know when a displaced tooth has been returned to its proper position. Typically, a tooth should be positioned similar to its contralateral mate. In some cases, asking the patient can help. It may be that one tooth has always been longer than the others or in a different position. Occlusion is always the best guide to proper position. If patient bites and contacts only the injured tooth, further positioning is necessary. In many cases, it is not possible to reposition the tooth completely because of swelling or organized clot formation. Adjustment of the opposing tooth (using an emery board or pocketknife file) may be necessary to allow proper occlusion. If the injured tooth receives additional trauma with each bite, it will be uncomfortable and healing will not occur.

Splinting

When the goal of splinting is to establish a normal fibrous union between tooth and bone, a short-term nonrigid technique is used. When hard tissue union is desired (e.g., for a root fracture or alveolar segment fracture), longer-term rigid splinting is used.[4,34] Ideally, a single avulsed or loosened tooth is bonded to an adjacent tooth or teeth with an acid etch and composite technique.[34] For jaw or dentoalveolar segment fractures, arch bars and wire splints are often used to provide rigid stabilization. Rescuers lacking adequate materials should use ingenuity and improvisation to splint teeth. During a very short evacuation (hours), the patient can hold his tooth in approximate position by closing (biting gently) on a gauze pad. Softened wax can be adapted to a loosened tooth and teeth on either side to lend support. Sport mouth guards can also be used. Use sutures to hold a tooth in place for 1 to 5 days (Figure 49-12D). For a sturdier splint, fashion a crude arch bar (e.g., cut from a SAM Splint). The splint can also be fashioned from paper clips or a soft wire obtained from copper electrical wiring or twist ties.

When replacing an avulsed tooth, isolate the area by placing a cotton roll or gauze in the vestibule of the mouth. Anesthetize the area by injecting local anesthesia at the apex of the tooth socket on both the buccal and the palatal/lingual side. Hold the tooth by the crown, and gently clean the root by irrigating with normal saline. If debris is still present, lightly brush the root with a toothbrush, but do not scrub vigorously, to avoid possibly removing the periodontal ligament tissues. Irrigate the socket gently with normal saline. Press the tooth firmly into the socket. Keep digital pressure on the tooth for a few minutes. This will help displace the clot and allow the tooth to be properly positioned. To assess proper position of the tooth, compare it to the surrounding teeth. You can also have the patient bite down to check occlusion. If using suture stabilization, the tooth can be secured in both vertical and lateral dimensions. To secure it in the vertical dimension, use a nonresorbable suture (e.g., silk). Insert the suture needle through palatal tissue, taking an anchoring "bite" of tissue. Pass the suture over the tooth, then take a bite of buccal gingival tissue. After you have determined that the suture is passing over the crown of the tooth, tie it firmly enough to hold the tooth in place. To secure the tooth in the lateral dimension, "floss" a suture between the contacts of the avulsed tooth and the adjacent tooth, to anchor it by tying the suture to the adjacent tooth. You may do this on both sides of the tooth if necessary for stabilization. (See Video 49-1.)

Injuries to Primary Teeth

Avulsed primary (i.e., children's) teeth should not be reimplanted. Severely extruded teeth, infected teeth, or those intruded into the developing permanent tooth should be extracted.[14] Most minor subluxations and luxations require only symptomatic treatment. Allow for spontaneous repositioning if there is no occlusal interference. If there is interference, gently reposition the tooth. Spontaneous repositioning often occurs over weeks. Repositioning a tooth increases risk of pulpal necrosis.[14] Do not extract fragments of primary roots if their removal may damage a permanent tooth. Normal resorption will occur.

Dislodged Fillings, Crowns, and Bridges

In the absence of other symptoms, no emergency treatment is necessary for a dislodged filling, small piece of tooth, crown, or bridge. File off any excessively sharp areas of any tooth that is irritating the patient's soft tissues. Otherwise, leave the tooth alone. If making an attempt to create a temporary filling or recement a crown, do not create an occlusal problem by leaving the restoration too high. This will increase patient discomfort. When recementing a crown, first inspect the dislodged crown. Remove debris in the crown, including old cement, with a dental pick or other available instruments. If you see a large piece of tooth in the crown, it means the tooth has fractured, and you will not be able to recement the crown. After the crown is free of debris, place it on the tooth and seat it down. Check occlusion to make sure the crown is not too high. If it is too high, the

patient will be biting on that tooth first, and the other teeth will not be occluding properly or at all.

Clean the tooth with the patient's toothbrush, then isolate the area with gauze. Use any available temporary cement. Mix the cement with the end of a cotton swab, and apply a very small amount of cement into the crown. There should be a very thin layer on all surfaces of the inside of the crown. Too much cement will interfere with seating of the crown, and it will be too high. After the cement is placed, position the crown on the tooth, and press firmly for 3 to 4 minutes. Cement may extrude from around the tooth. Remove the excess cement with a dental pick or dry toothbrush. Have the patient bite down while the cement dries. Make sure that occlusion is good, and that the teeth are not occluding only on the recemented crown. If the crown is sitting too high, try to press it down farther. If this does not work, remove the crown and repeat the entire process again. If the crown falls off after recementation, it is better to leave it off until the patient can be seen by a dentist. (See Videos 49-2 and 49-3.)

Appliance Sores

Oral appliances (e.g., complete or partial dentures, retainers, mouth guards) can cause mucosal sores. The mucosa will appear red or ulcerated. The patient can often do without the appliance until definitive treatment is available. If not, scrape away a small amount of material from the appliance where it is irritating or rubbing the mucosa. It can be a challenge to identify the precise area to modify on the appliance. Mark any sore spot with a tiny piece of adhesive tape (sticky side away from the gingiva) or a slurry of water and grape drink mix, insert the dried appliance, and then remove it. Ideally, the mark is transferred to the appliance, indicating the exact site needing adjustment. If the appliance cannot be adjusted, the patient should discontinue its use until seen by a dentist.

Orthodontic appliances can cause soft tissue irritation or ulceration. Cover the offending fixture with soft wax. Bend protruding wires until the sharp portion faces away from soft tissues. If a wire must be cut, try fingernail clippers or repeated bending until the metal fatigues and breaks. If a bracket or wire becomes excessively loose, remove it with judicious tinkering.

OROFACIAL PAIN

Orofacial pain may arise from different regions of the head and neck.[30,31,37] If the patient's symptoms do not seem consistent with dental origin, suspect other causes. Disorders sometimes confused with dental pain include myofascial pain, maxillary sinusitis, temporal arteritis, and trigeminal neuralgia (Box 49-5). Table 49-1 lists characteristics of different types of tooth-related pain.

BOX 49-5 Conditions That Cause Facial Pain

- Pulpitis (acute, chronic, hyperplastic)
- Periapical osteitis (acute, chronic)
- Cracked tooth syndrome
- Periodontal infections
  - Periodontal abscess
  - Acute necrotizing ulcerative gingivitis
  - Primary herpetic gingivostomatitis
  - Pericoronitis
- Temporomandibular disorders
- Maxillary sinusitis
- Neurologic pain
  - Trigeminal neuralgia
  - Trigeminal neuritis
  - Herpes zoster neuritis
  - Postherpetic neuralgia
- Atypical facial pain
- Psychogenic facial pain
- Dental causalgia (atypical odontalgia)
- Vascular pain
  - Migraine headache
  - Temporal arteritis
  - Cluster headache
- Referred pain
- Referred pulpitis
- Referred pain of subacute thyroiditis
- Referred pain of myocardial infarction or angina pectoris

PULPITIS

The common toothache is caused by inflammation of dental pulp. Convergence of neurons in the trigeminal spinal tract nucleus can make it difficult for the patient to identify the offending tooth.[23] The painful tooth is rarely sensitive to percussion or palpation. Obvious causes (e.g., large carious lesion) may be found, but often all teeth appear intact. Pulpitis can arise spontaneously, or from tooth decay, new restorations, or trauma. If mild, pain may only be elicited by cold temperature or sweets and disappears within seconds when the stimulus is removed. Moderate pulpitis is characterized by sensitivity to hot as well as to cold temperature, greater discomfort, and a longer interval between removal of the stimulus and resolution of pain. Severe pulpitis causes intense, continuous, and debilitating pain[7,31] (see Severe Pulpitis for emergency treatment recommendations).

Mild Pulpitis (Characterized by Transient Thermal Sensitivity)

Examine the mouth. Structures will likely appear within normal limits. If a tooth defect is found, it can be temporarily filled. Reassure the patient that although this condition is annoying, rapid progression is unlikely.

Moderate Pulpitis (Longer Episodes of Pain)

Treat as for mild pulpitis. In addition, use a non-narcotic analgesic such as ibuprofen, 600 mg orally (PO) every 6 hours (q6h) as needed, and reduce physical activity. If a large defect (cavity) is found in the offending tooth, a cotton pellet with eugenol can be applied to exposed pulp. Fill the defect with zinc oxide–eugenol cement (IRM) or Cavit.

Severe Pulpitis (Intense, Continuous Pain)

The preferred approach is pain relief using a local anesthetic, followed by evacuation of the patient.[35,48] A nerve block with bupivacaine (Marcaine) 2% with 1:200,000 epinephrine can provide up to 8 hours of pain relief without central nervous system (CNS) depression (see Local Anesthesia, later). Nonsteroidal antiinflammatory drugs (NSAIDs) are unlikely to provide significant relief. Large doses of narcotics should not be used; they will likely compromise the patient's ability to participate in evacuation. Applying cold (e.g., continuously sipping cold water) may provide relief to hyperemic pulp. A course of antibiotics can be initiated. Antibiotics may not be indicated if immediate treatment (root canal therapy or extraction) of the tooth is undertaken, but they will likely be helpful and decrease the risk of worsening infection in the remote setting. In an extraordinary circumstance, an experienced rescuer could locate the offending tooth, expose the pulp, remove inflamed pulpal tissue with a barbed broach, and then cover the opening with temporary filling material. Extraction may also be considered but is typically contraindicated (see Exodontia, later).

PERIAPICAL OSTEITIS/ACUTE APICAL PERIODONTITIS

Inflammation of supporting structures at the tooth's root is characterized by constant, often throbbing pain. Unlike pulpitis, the patient can usually point to the exact source of pain, or the examiner may gently tap individual teeth to locate tenderness. The area over the tooth's apex is usually tender, but there is no frank swelling. Tooth trauma can result in periapical inflammation, but the most common cause is egress of bacteria from necrotic pulp. This is often a precursor to an acute apical abscess. Minor swelling around the apex extrudes the tooth slightly, causing increased forces on the tooth during occlusion and worsened pain. Emergency treatment includes an analgesic (e.g., ibuprofen, 600 mg PO q6h as needed) or a local anesthetic,

TABLE 49-1 Characteristics of Dental Conditions That Cause Pain

	Erupting Teeth	Reversible or Mild Pulpitis	Irreversible (Moderate to Severe) Pulpitis	Necrotic Teeth (Periapical Osteitis)	Cracked Tooth Syndrome	Periodontal Infection	Fractured Teeth
Onset; Duration	Sudden; lasts for several days	Episodes last a few to several seconds	Episodes last a few to several minutes	Pain comes on quickly, lasts several days	Episodes last a few seconds	Gradual progression; lasts for days	Sudden onset
Previous history	Possibly	None; or may be preceded by gingival recession or lost restoration	Yes; probably preceded by symptoms of mild pulpitis	None; or possible history of pulpitis symptoms, or history of restoration months earlier	None; or possible history of sudden crack while chewing	Often recurrent, but may be the first episode	May have a previous history of sensitivity to cold
Location	Tissue in area of eruption	Patient usually points to the general area but may not know the specific tooth.	Patient usually points to the general area but may not know the specific tooth.	Patient usually points to a specific tooth.	Patient may not be able to tell if it is upper or lower tooth.	It is usually easy for patient to point to exact area of pain and swelling.	Patient can usually point to specific tooth.
Severity	Mild to moderate	Mild to moderate	Moderate to severe	Moderate to severe; mild if abscess is draining	Mild to severe	Mild to moderate	Moderate to severe
Type	Dull, throbbing pain	Sharp, quick, shock-like feeling	Sharp, followed by lingering pain	Constant unrelenting ache	Sharp, shock-like pain	Dull, throbbing pain Patient typically knows it is coming from the gingiva, not the tooth.	Sharp pain on release of pressure
Trigger	Spontaneous or chewing	Cold, including air Sweets	Cold, including air; heat Sometimes biting; sometimes spontaneous	Biting or even mild pressure as with patient's tongue	Biting hard or chewy food in just the right way	Spontaneous; touch increases pain	Pain on biting on a specific cusp

antibiotics, and a soft diet. Antibiotics may decrease the risk of worsening infection. Ideally, contacting areas of opposing teeth are reduced to relieve occlusal forces. This usually is impractical in the field. The patient can be given a strip of webbing or something similar to place between the teeth on the nonpainful side. This will keep the offending tooth out of occlusion and reduce pain.

CRACKED TOOTH SYNDROME

The patient complains of transient, recurrent sharp pain when chewing or on releasing his bite. The patient may report that the tooth feels "weak," or that "it only hurts when I bite just the right way on something hard." Symptoms occur when forces of proper magnitude and direction open the tooth's incomplete fracture.[36] Pain varies depending on the crack's depth and how close it comes to pulp. Significantly, there is no pain when chewing soft foods. The patient often has a history of large restorations or a habit of chewing ice or clenching and grinding.[19] This condition usually progresses slowly. Reducing occlusion on the cracked tooth will relieve some of the pain but is often impractical in the field. Advise the patient to avoid chewing on the affected side and to seek definitive dental treatment as soon as possible.

ODONTOGENIC REFERRED PAIN

Pain originating in the tooth's pulp can be referred to distant sites. Common referral patterns include the maxillary incisors to the forehead, the maxillary canine to the infraorbital area, the maxillary molars to the temple or ear area, the mandibular anterior teeth to the mental foramen area, the mandibular premolars to the posterior jaw, and the mandibular molars to the angle of the jaw, ear, or neck. The patient may sense that pain is coming from a tooth several teeth in front of or behind the source of pain. Pulpitis pain can be referred from an upper tooth to a lower tooth, or vice versa, but never crosses the midline. The dental literature is replete with cases where experienced practitioners have treated the wrong tooth or performed multiple dental treatments for pain that is not odontogenic.[23,28]

MAXILLARY SINUSITIS

Maxillary sinusitis pain can be referred to a posterior maxillary tooth. It is usually described as a relatively continuous throbbing ache that is intensified by postural change. A typical statement is, "My tooth really hurt when we were hiking down the hill. I could feel it pound with every step. When we got to camp, I lay down, but it got even worse." The pain is unilateral or bilateral. It is usually located in the infraorbital region and often referred to the cheek, frontal region, and maxillary premolars and molars. A complaint of multiple maxillary toothaches with no evidence of carious teeth should raise suspicion for maxillary sinusitis. Tenderness may be elicited by pressure infraorbitally or over the bony prominence above the first molar. The patient may also have elevated temperature and nasal or postnasal discharge.

Treat maxillary sinusitis with analgesia (e.g., ibuprofen, 600 mg PO q6h as needed); inhalation of steam; oxymetazoline 0.05% (Afrin), one spray in each nostril twice daily to shrink nasal

membranes and improve sinus drainage; and an antibiotic. Appropriate antibiotics include amoxicillin (875 mg) with clavulanic acid (125 mg) (Augmentin) PO twice daily for 10 days, or trimethoprim (160 mg) with sulfamethoxazole (800 mg) (Septra DS) PO twice daily for 10 days. Azithromycin (Zithromax), 500 mg PO the first day and then 250 mg PO for the next 4 days, is a convenient alternative.[1,16]

TEMPOROMANDIBULAR DISORDER

Temporomandibular disorder (TMD) is a cluster of conditions, multifactorial in origin, and with overlapping symptoms that often respond to a variety of therapies, including placebo.[29] Patients with TMD may answer "yes" to any of the following questions: Does it hurt to open the mouth widely or yawn? Do you have earaches or pain in front of your ear? Is pain worse in the morning? Have you had trauma to your face or jaw? Do you have temporal pain or headaches? Has your jaw ever gotten stuck open or closed? Does your jaw pop or click on opening or closing?

Patients with TMD may have primary masticatory muscle involvement (myofascial pain and dysfunction [MPD]) and/or internal derangements of the temporomandibular joint (TMJ). Internal derangements, traumatic injuries, and dislocation of the TMJ are covered in Chapter 20.

Myofascial Pain and Dysfunction

Muscle hyperactivity is an important etiologic factor in MPD. This may result from parafunction (e.g., gum chewing, clenching or grinding teeth). Occlusal interferences can also cause muscle hyperactivity. This occurs when a lower tooth contacts an upper tooth prematurely during mouth closure and causes reflex jaw muscle contraction that shifts the mandible to avoid premature contact. For this reason, this condition is often referred to as an "occluso-muscle disorder." Psychological stress is also an important factor in causing excessive muscle tension.[22]

High physiologic and psychological demands of many expeditions may create risk factors for MPD. Increased jaw function (e.g., from chewing granola, beef jerky, and other dried foods typically brought on wilderness expeditions) may precipitate an acute episode of MPD.

Symptoms of MPD include pain in the muscles of mastication that is typically unilateral and increases with chewing. Other features may include headache, earache, limitation of jaw movement, or a change in bite. Pain originating in muscles of mastication is often referred to teeth, resulting in a chief complaint of toothache.[23,41] Pain may be acute in onset or preceded by a long history of waxing and waxing pain with various treatments.

Objective signs include tenderness of jaw muscles to palpation, muscle spasm or "knots," and abnormal jaw movements (e.g., inability to open the mouth widely, deviation of the chin to one side on opening). Tenderness of the TMJ or joint noise/crepitus suggest internal joint derangement.[22]

Treat by resting the muscles (e.g., soft diet, control of tooth-clenching/grinding habits) and applying moist heat. Hold soft material (e.g., folded gauze) between the front teeth or use a sport mouth guard. This often provides immediate relief because it keeps teeth from touching and allows muscles to relax. An analgesic should be given on a scheduled basis (e.g., ibuprofen, 600 mg PO q6h) to break the cycle of muscle pain and spasm. A muscle relaxant (e.g., cyclobenzaprine [Flexeril], 10 mg PO three times daily) or sedative (e.g., diazepam [Valium], 2 to 10 mg PO three times daily) may be helpful if primary treatment is ineffective. These agents can cause significant CNS depression and thus should be used in the lowest effective dose and only if more conservative therapy has failed.[8,29,44]

MAXILLOFACIAL INFECTIONS

VIRAL INFECTIONS

Herpes labialis (i.e., cold sore, fever blister) is the most common oral viral infection. It is a self-limiting disease of about 7 to 14 days' duration characterized by yellow, fluid-filled vesicles that rupture to leave ragged labial ulcers (Figure 49-14). Recurrent herpetic outbreaks can also affect the palate, tongue, and buccal mucosa. The patient can be given valacyclovir (Valtrex), 2 g PO twice daily for 1 day, with doses taken 12 hours apart.[42] Begin treatment as soon as the patient becomes aware of the prodromal "tingle" (i.e., paresthesia). Although this regimen of valacyclovir may prevent outbreaks, conservative methods (e.g., use of sun-blocking preparations on lips) are preferred.[6,27,45]

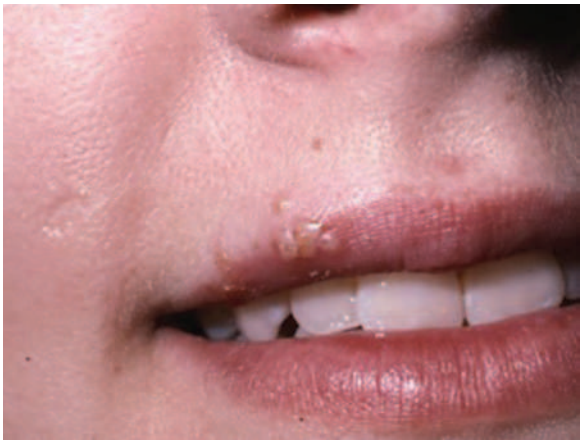

FIGURE 49-14 Herpes labialis showing vesicles. *(Courtesy Dr. James Burns.)*

Primary herpetic gingivostomatitis is characterized by numerous mucosal vesicles, fever, lymphadenopathy, and sore throat. The entire mouth and throat, as well as perioral skin, can be involved[32] (Figure 49-15). Vesicles typically affect attached gingiva surrounding the teeth and tongue. This and other viral infections of the oral cavity are self-limited. The patient should be reassured that the condition will resolve in approximately 10 days. Treat with analgesia (ibuprofen, 600 mg PO q6h as needed for pain and fever) and soothing mouth rinses (e.g., warm saline or a mixture of equal amounts of diphenhydramine [Benadryl] elixir, 12.5 mg/5 mL, with kaolin-pectin [Kaopectate] and viscous lidocaine 2%). Rinse and expectorate 5 mL every 2 hours.

Herpes zoster (shingles) is caused by reactivation of latent varicella-zoster virus (VZV) within a sensory nerve. It can occur in the head and neck region when associated with facial or trigeminal nerves (Figure 49-16). Oral lesions may occur with trigeminal nerve involvement and result in devitalization of teeth or bone necrosis. Prodromal pain precedes rash by 1 to 4 days in 90% of patients. Pain may be mistaken for tooth pain, migraine, or other types of atypical facial pain.[32] Early treatment with antiviral therapy (acyclovir, 800 mg PO every 4 hours while awake [five times daily] for 7 to 10 days; valacyclovir, 1 g PO every 8 hours for 7 days; or famciclovir, 500 mg PO four times daily for

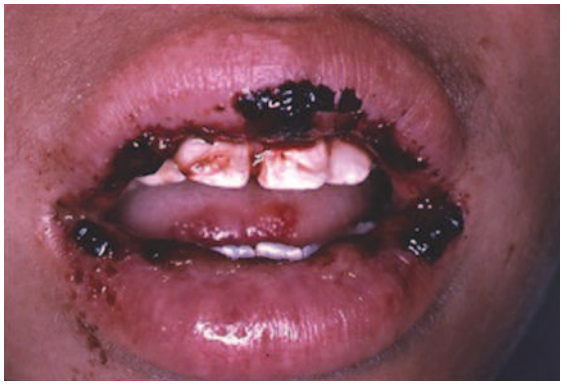

FIGURE 49-15 Primary herpetic gingivostomatitis. *(Courtesy Dr. James Burns.)*

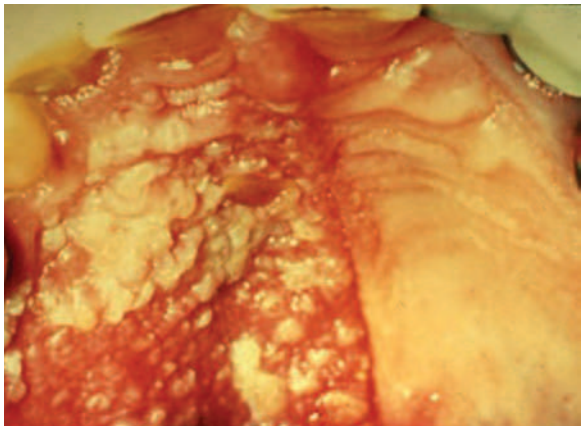

FIGURE 49-16 Intraoral herpes zoster.

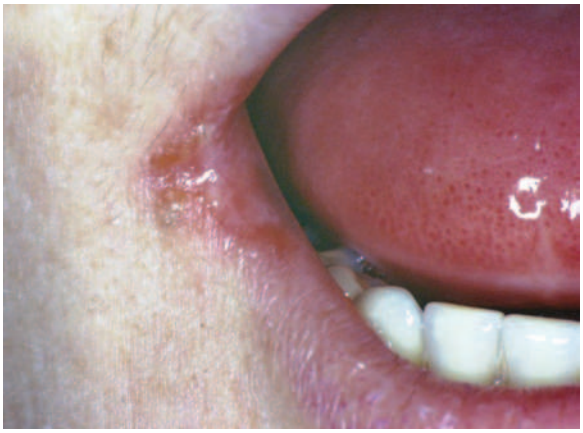

FIGURE 49-18 Close-up of angular cheilitis, a yeast infection. *(Courtesy Dr. James Burns.)*

7 days) accelerates healing and decreases risk of postherpetic neuralgia.[51]

YEAST INFECTIONS

Oral yeast infections occur most frequently in persons who are debilitated, are immunocompromised, have xerostomia, or are taking an antibiotic or corticosteroid. Classic oral candidiasis (thrush) is characterized by white patches on the mucosa that can be rubbed off, leaving a red and raw surface (Figure 49-17). Candidiasis can also manifest as erythematous mucosa, with no white patches, or as chronic angular cheilitis (Figure 49-18). Candidiasis is treated with an antimycotic mouth rinse (e.g., nystatin oral suspension, 100,000 U/mL; rinse with 5 mL for 2 minutes and swallow, four times a day for 10 days) or lozenges (clotrimazole [Mycelex], one troche four times daily; leave in the mouth for 5 minutes, and expectorate the remains). In the field, nystatin preparations meant for vaginal treatment (nystatin vaginal suppository, used as an oral lozenge three times daily for 10 days) can be used.

BACTERIAL INFECTIONS

Maxillofacial bacterial infection can become a serious health threat and should be taken seriously. Most odontogenic infections are caused by mixed aerobic and anaerobic bacteria that are typically present as normal oral flora. Within tissue, these infections can become aggressive.

Bacterial behavior (e.g., production of collagenase or hyaluronidase) determines clinical presentation. Infection may be diffuse (cellulitis) or local (abscess). Oral infections typically spread slowly, but rapid spread to deep facial spaces can occur. Regional lymphadenopathy is common. Severe systemic symptoms are rare. Although bone is often involved, osteomyelitis is uncommon.[40]

Acute Apical Abscess/Cellulitis

Acute apical infection begins with bacteria invading dental pulp. Infection spreads to surrounding bone through the apical foramen. Because the apices of most teeth are located closer to the facial (i.e., lateral) aspect of the jaw, swelling typically occurs in facial soft tissues rather than in the lingual or palatal side.

The patient presents with pain and swelling, often fluctuant and usually in the buccal vestibule (Figure 49-19). There is often a history of prior toothache, but tooth pain is often absent. The offending tooth can be localized by percussion, the site of swelling, condition of teeth, and radiographs. The affected tooth does not respond to hot or cold. The patient may be dehydrated because of decreased fluid intake.

Primary treatment for an apical abscess is drainage by incision, extraction, or endodontic therapy.[25,43] Treatment depends on equipment and personnel, advisability of retaining the offending tooth, and clinical judgment. Antibiotics are necessary only if there are complicating factors[9,25,43,46] (Box 49-6). Penicillin V potassium, 500 mg PO four times daily, is the most commonly used antibiotic in dental practice, but a cephalosporin (e.g., cephalexin [Keflex], 500 mg PO four times daily) is acceptable. Clindamycin, 150 to 300 mg PO four times daily, is a good alternative.[20,43,46] Combination antibiotic therapy is not indicated except for life-threatening sepsis, or when organisms particularly

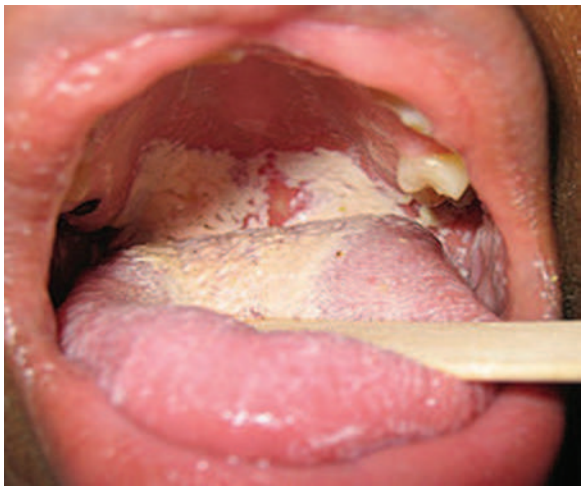

FIGURE 49-17 Thrush.

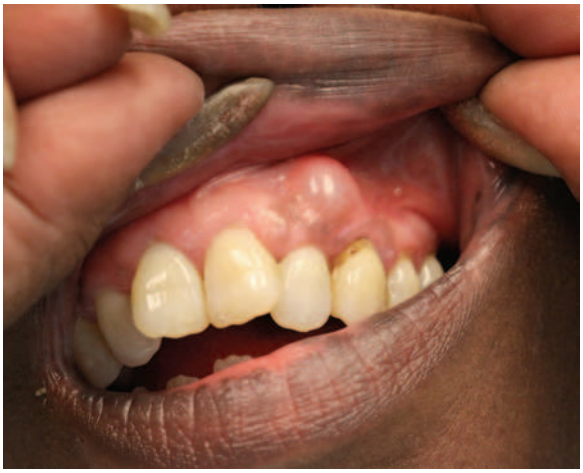

FIGURE 49-19 Apical abscess manifesting with swelling in the buccal vestibule.

BOX 49-6 Indications for Antibiotic Use in Dental Emergencies

- Prophylaxis for persons at risk for bacterial endocarditis (see Box 49-1)
- Prophylaxis for persons having prosthetic joint implants within the past 2 years
- Local infections
  - If the patient is immunocompromised
  - If drainage cannot be established
  - If there will be a long delay before definitive care
  - If infection persists after local treatment
- Disseminated infections
  - Lymphadenopathy
  - Fascial plane involvement
  - Trismus
  - Systemic symptoms (fever, chills, malaise)
- Compound maxillofacial fractures, including all fractures of tooth-supporting bone
- Exarticulation (avulsion) of teeth
- Soft tissue wounds open for 6 hours or more before closure
- Surgical procedures under nonsterile conditions

sensitive to combination therapy have been identified. Evidence exists that delayed drainage, combined with overreliance on antibiotic therapy, can lead to serious exacerbation of dental infections.[9]

Incision and Drainage

Incision and drainage (I&D) is often the treatment of choice. An I&D procedure can be performed by a nondentist using commonly available supplies and is indicated for fluctuant swelling caused by apical abscess. It may also be effective for nonfluctuant swelling associated with infection. Infiltration of a local anesthetic helps to reduce the pain of incision. If medications are not available, adequate anesthesia can often be obtained by applying cold to the area to be incised (e.g., using ice, snow, or frigid water). An incision is made down to bone in one continuous brief motion, ideally with a No. 11 or 15 surgical scalpel blade. A knife handle or beak of a hemostat is used to spread the incision. A drain may be improvised from a piece of latex glove and retained without sutures (Figure 49-20). Systemic antibiotics, hydration, a soft diet, an analgesic (e.g., ibuprofen, 600 mg PO q6h as needed), and warm saline rinses are helpful postoperative measures.

Deep Fascial Space Infections

Acute apical infections occasionally spread beyond the local region. Caregivers should be suspicious if a dental infection causes swelling or tenderness in the floor of the mouth, swelling of the tongue, dysphagia, breathing difficulty, or trismus, or if it fails to respond to appropriate therapy. The most frequently involved fascial spaces are canine, buccal, masticator, and submandibular.[10]

The canine space is located lateral to the nose. Infection originates in the maxillary canine tooth. Swelling causes the eye to close. Infection is drained through an intraoral approach over the root of the canine tooth.

Buccal space infection is characterized by rounded swelling of the cheek (Figure 49-21). Offending teeth are maxillary and mandibular molars. Drainage is obtained transcutaneously through an incision approximately 2 cm (0.8 inch) below the lower border of the mandible, to avoid the marginal mandibular branch of the facial nerve. You can aspirate with an 18-gauge needle attached to a syringe to aid in location of the abscess (Figure 49-22A). Dissect sharply through subcutaneous tissue. Next, dissect bluntly with a hemostat towards the infection (Figure 49-22B). Once the abscess is located and evacuated, a drain can be placed and secured with a suture. This will allow continued drainage until the infection improves (Figure 49-22C).

The masticator space is divided into masseteric, pterygoid, superficial temporal, and deep temporal spaces, all of which

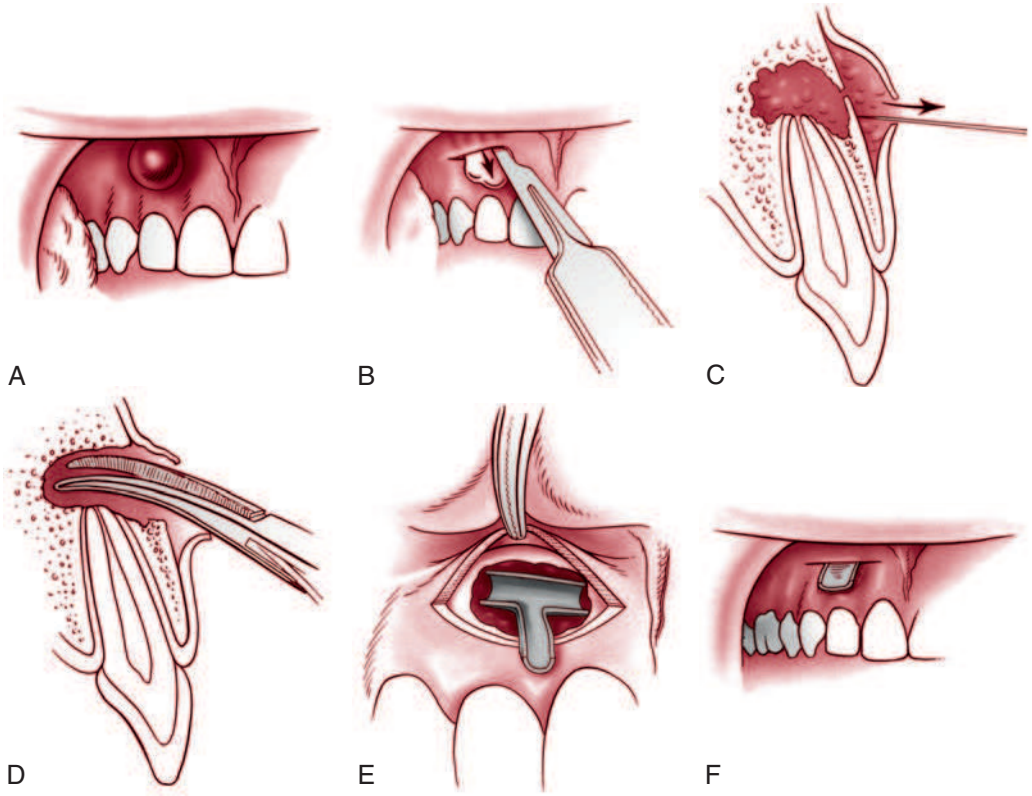

FIGURE 49-20 Incision and drainage (I&D) technique. **A,** Fluctuant abscess. **B,** Abscess incised with scalpel. Purulent drainage is removed by suction or caught in gauze sponges. **C,** Cross section showing incision carried to bone. **D,** Incision is spread with a hemostat. **E,** T-shaped drain often stays in place without sutures. **F,** Drain in place. *(Redrawn from Ingle JI, Beveridge EE:* Endodontics, *ed 2, Philadelphia, 1976, Lea & Febiger.)*

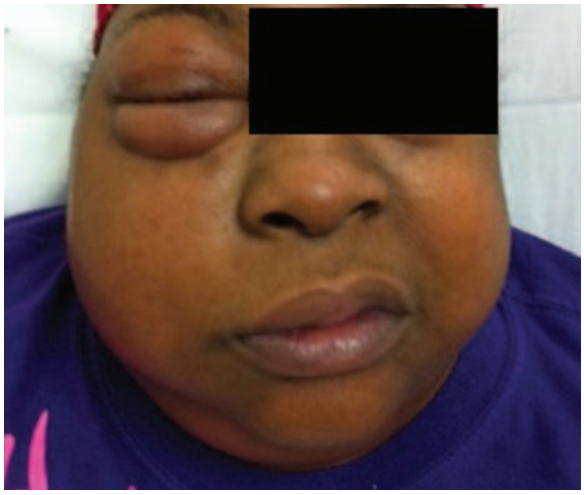

FIGURE 49-21 Buccal space infection.

communicate. Trismus is the hallmark of involvement. Swelling may be minimal because of the deep location of the abscess. The masseteric and pterygoid spaces are drained at the angle of the mandible. The temporal space may be drained from an intraoral approach or through an incision just superior to the zygomatic arch.

The mylohyoid muscle divides the floor of the mouth into sublingual and submandibular spaces. These communicate posteriorly and across the midline. An infection originating in a mandibular tooth can involve these spaces. Both sublingual and submandibular space infections should be drained through an extraoral (i.e., transcutaneous) approach. All incisions in the facial area are made parallel to branches of the facial nerve.[10,21]

Fascial space infections can be life threatening, typically by compromising the airway. Mild dental infection can progress to a life-threatening emergency in as soon as 48 hours.[13] Closely monitor airway patency. *Ludwig's angina,* a bilateral submandibular space infection that elevates the tongue and obstructs breathing, is the most feared infection and is associated with high mortality rate (Figure 49-23). Other complications include widespread sepsis, cavernous sinus venous thrombosis, and mediastinitis.[11,13]

Treatment of fascial space infections includes airway management, proper hydration and electrolyte balance, aggressive I&D, intravenous antibiotics, and pain control. These objectives are best met in a controlled environment. Any person with a suspected fascial space infection should be evacuated immediately.

Chronic Apical Abscess

The hallmark of chronic apical infection is a draining fistula, or "gum boil" (Figure 49-24). Because bacteria and purulence have a route to escape, there is typically minimal pressure or pain. The tooth may be mildly sensitive when eating. This is not an emergency, except in the unlikely event of acute exacerbation. Antibiotics are not indicated. Definitive treatment (root canal therapy or tooth extraction) is necessary.

An infected deciduous (pediatric) tooth usually manifests with a fistula and with limited to no swelling. Severe pain is rarely present. Emergency care involves an antibiotic with dosage adjusted for the child's weight. Perform I&D only if the abscess is not draining. Extract the tooth only if it is very loose and the patient is weeks from comprehensive care.

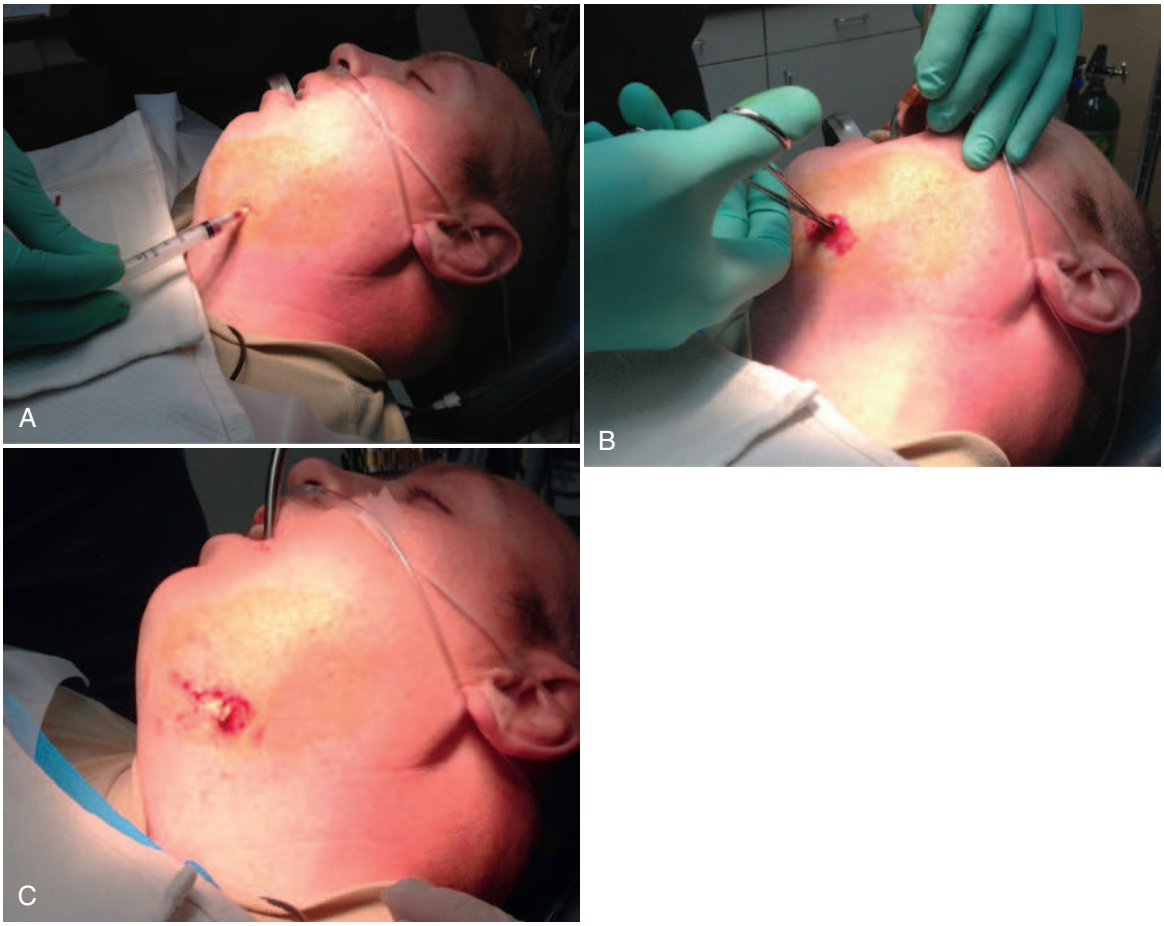

FIGURE 49-22 **A,** Aspiration of submandibular abscess with 18-gauge needle used to locate the abscess. **B,** Blunt dissection with hemostat. **C,** Penrose drain placed in the submandibular space.

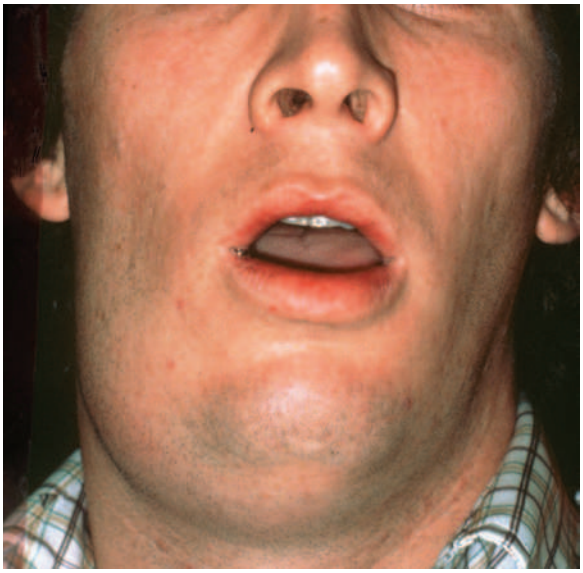

FIGURE 49-23 Ludwig's angina.

Periodontal Abscess

Periodontal abscess is an accumulation of pus between the gingiva and tooth. Swelling is near the gingival margin rather than in the vestibule (as found with periapical abscess). The tooth is sensitive to percussion, but not to hot and cold. A potential communication may exist between the abscess and mouth. Find the passage by probing the gingival margin with a small, blunt instrument, using a local anesthetic if available. Gentle probing establishes drainage. No incision is necessary. Hot saline rinses are advised. Rapid recovery is likely.

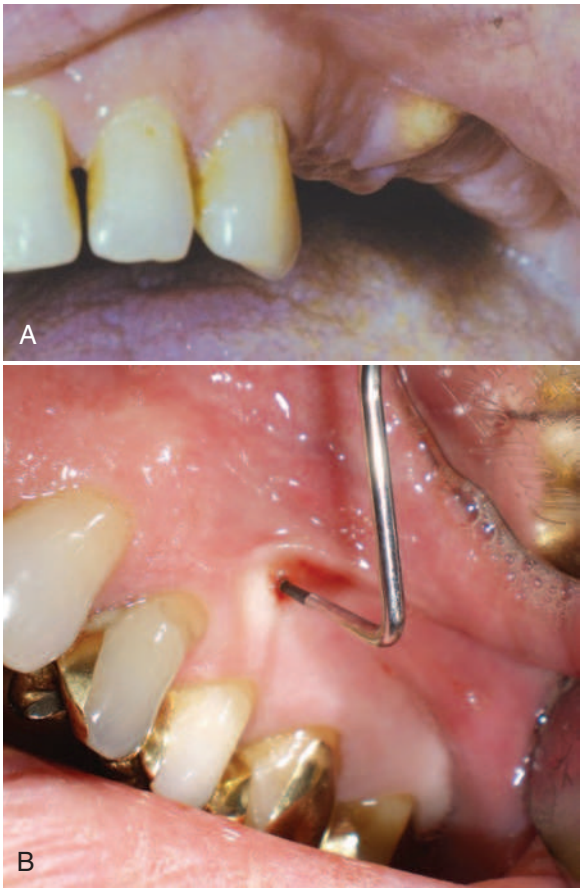

FIGURE 49-24 **A,** Fistula, indicating a chronic abscess. **B,** Periodontal probe showing depth of fistulous tract. *(Courtesy Dr. James Burns.)*

Pericoronitis

Pericoronitis is an infection of the gingival flap over a partially erupted tooth, most often of the mandibular third molar. Pericoronitis is typically caused by streptococci and seldom produces purulence. This may mimic streptococcal pharyngitis or tonsillitis. The infected site is always tender. Trismus is a common sign. Field treatment consists of saline irrigation of the space under the flap using a syringe. Place the patient on hot saline rinses every 2 hours and begin antibiotic therapy. Appropriate antibiotics include penicillin V potassium, 500 mg PO four times daily; a cephalosporin such as cephalexin (Keflex), 500 mg PO four times daily; clindamycin, 150 to 300 mg PO four times daily; or azithromycin, 500 mg PO first dose, then 250 mg PO once daily for the next 4 days.[20,43,46]

Necrotizing Ulcerative Gingivitis

Necrotizing ulcerative gingivitis (NUG, trench mouth) is characterized by crateriform ulceration and blunting of interdental papillae. The gingiva between teeth appears punched out and covered by gray-white pseudomembrane. Surrounding gingival tissue is very red (Figure 49-25). The primary complaint is pain, but the patient may report gingival bleeding, metallic taste, and foul odor. This infection is caused by fusiform bacteria and spirochetes. NUG usually occurs in young and middle-aged adults with poor oral hygiene, stress, and suboptimal nutrition (e.g., while partaking in a difficult wilderness expedition). Treat with gentle debridement of plaque, calculus, and food from around the teeth. Resolution may require several sessions of careful cleaning 1 to 2 days apart for 1 week to 10 days. Each debridement results in some healing and allows more aggressive subsequent treatment. The patient with NUG is also given an antibiotic (metronidazole [Flagyl], 250 mg PO four times daily for 10 days, or amoxicillin-clavulanate [Augmentin], 500/125 mg PO three times daily for 10 days)[52] and an analgesic (e.g., ibuprofen, 600 mg PO q6h as needed) and is instructed to keep the area clean with proper brushing, flossing, and rinsing. Use chlorhexidine mouth rinse, if available, or warm saline or diluted hydrogen peroxide as a rinse substitute.[32]

APHTHOUS ULCERS

Aphthous ulcers likely represent a local immune response with various triggers, including stress, trauma, immunosuppression, nutritional deficiencies (vitamin B_{12}, folic acid, iron), acidic foods, and food allergies. Lesions are round or ovoid with a yellowish center and red border. They typically occur on nonkeratinized, movable mucosa and can be quite painful (Figure 49-26). The patient often gives a history of similar ulcerations in the past. There are three types of aphthous ulcers. Minor aphthae are 3 to 10 mm (0.12 to 0.4 inch) in diameter and usually last 7 to 14

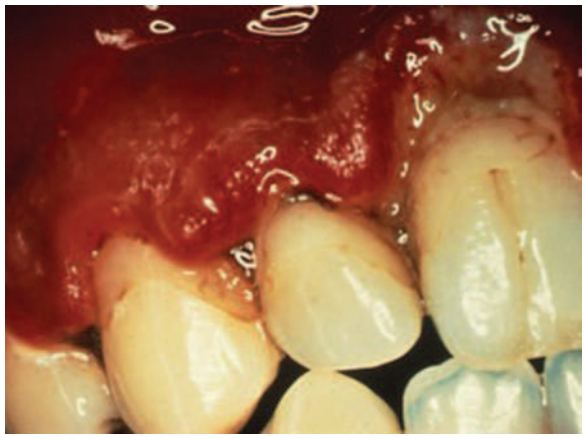

FIGURE 49-25 Necrotizing ulcerative gingivitis.

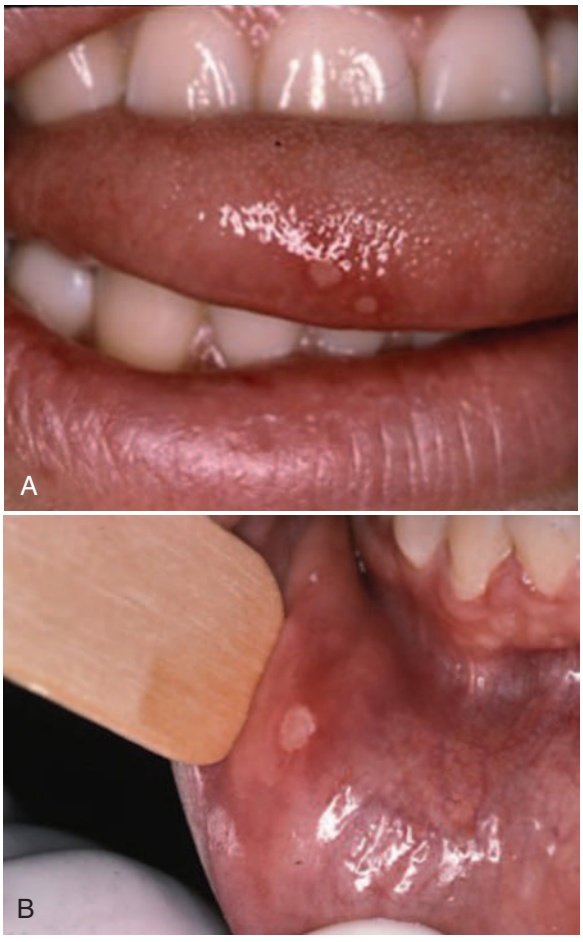

FIGURE 49-26 **A,** Aphthous ulcer on tongue. **B,** Aphthous ulcer on lip. *(Courtesy Dr. James Burns.)*

days. Major aphthae are larger than 1 cm (2.5 inches) and may last weeks to months. Herpetiform aphthae are clusters of small, shallow ulcers that resemble intraoral herpes.

Many treatments have been proposed for aphthous ulcers, but none has been found predictably effective. Apply a topical corticosteroid to reduce pain and hasten healing by 3 to 4 days. Mix fluocinonide (Lidex) 0.05% ointment with Orabase, and gently place the mixture over each ulcer six to eight times per day, especially after meals and before bedtime. Do not mix medications until you are ready to apply them. Do not rub the mixture into lesions. Premixed preparations (e.g., Kenalog in Orabase) applied to ulcers six to eight times per day are more convenient but deliver only about 10% of the agent's antiinflammatory effect. Dexamethasone (Decadron) elixir (0.5 mg/5 mL) may also be used; rinse with 5 mm for 2 minutes and expectorate four times daily. A systemic corticosteroid (e.g., prednisone, 40 mg PO once daily for 3 days) may be used for very severe cases. If these preparations are not available, apply tincture of benzoin or a topical anesthetic (e.g., viscous lidocaine 2%) to ulcers, with the surface dried as best possible prior to application, before meals and at bedtime to control pain.[12,32]

TRAUMATIC ULCERS

Traumatic ulcers are similar to aphthous ulcers but are typically more irregular in outline and lack the red border. They occur from chronic rubbing, such as from a sharp tooth against the tongue or a poorly fitting denture against the palate. Topical anesthetic (e.g., viscous lidocaine 2%) can be used for treatment, but the most important intervention is to remove the source of irritation.

MEDICATION-RELATED OSTEONECROSIS OF THE JAWS

Osteonecrosis of the jaws (ONJ) can be associated with multiple medications, including antiresorptive drugs (e.g., bisphosphonates, denosumab) and antiangiogenic therapies. Oral bisphosphonates (e.g., alendronate [Fosamax]) are widely used to treat osteoporosis, and intravenous bisphosphonates (e.g., zoledronic acid [Zometa]) are used to manage bone metastases in various cancers. RANK ligand inhibitors (e.g., denosumab) are antiresorptive medications that inhibit osteoclast function and are used for osteoporosis and skeletal involvement of various cancers. Antiangiogenic medications are used for treatment of multiple cancers. A side effect of each can be delayed healing of exposed maxillofacial bone. Bisphosphonates alter bone metabolism long after the medications have been discontinued. Long-term effects of newer medications are unknown.

Medication-related osteonecrosis of the jaws (MRONJ) may occur after bone is exposed by acute trauma, chronic trauma (e.g., from a poorly fitting dental prosthesis), dental surgery, or tooth extraction. Risk of MRONJ in cancer patients exposed to bisphosphonates and denosumab is approximately 1% (about 50 to 100 times greater than for controls). Risk for MRONJ in patients exposed to antiangiogenic agents ranges from 0.2% to 0.9%. ONJ risk among osteoporosis patients exposed to bisphosphates or denosumab ranges from 0.017% to 0.04%, similar to that of controls.[3]

Diagnosis of BRONJ is made when exposed, necrotic bone persists for at least 8 weeks in a patient with no history of mandibular radiation treatment and who currently uses or previously used antiresorptive or antiangiogenic agents. Small lesions may be asymptomatic (Figure 49-27). Exposed areas become infected, with pain, erythema of the surrounding soft tissue, and possible purulent drainage. To prevent MRONJ, avoid bone exposure in at-risk patients. Emergency management consists of treating pain and controlling infection. Asymptomatic lesions are treated with an antibacterial mouth rinse, such as chlorhexidine. Treat painful, infected MRONJ with an antibacterial mouth rinse and antibiotic (e.g., penicillin V potassium [500 mg], cephalexin [500 mg], or clindamycin [150 mg] PO four times daily), and pain control. Evacuation for surgical care is indicated for patients with MRONJ complicated by pathologic fracture, extraoral fistula, or necrosis extending to the inferior border of the mandible.[3,24,39]

MUCOCELE

Mucocele (mucous retention phenomenon) occurs when a minor salivary gland duct is blocked or traumatized. This results in accumulation of saliva beneath the mucosa. It most frequently occurs in the lower lip. A *ranula* is a mucocele in the floor of the mouth. Most mucoceles are a few millimeters in diameter, lie just under the surface, and have a translucent or bluish tint. They

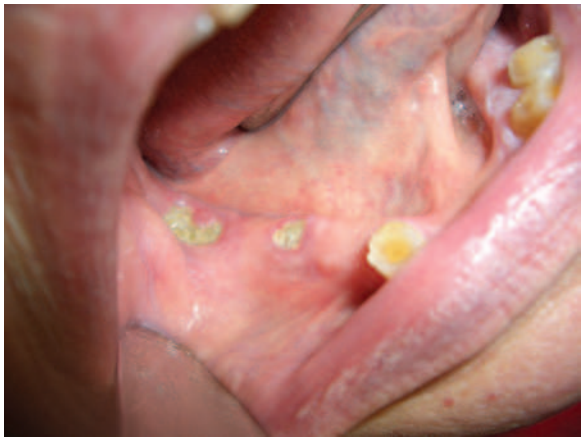

FIGURE 49-27 Two small areas of medication-related osteonecrosis of jaw (MRONJ).

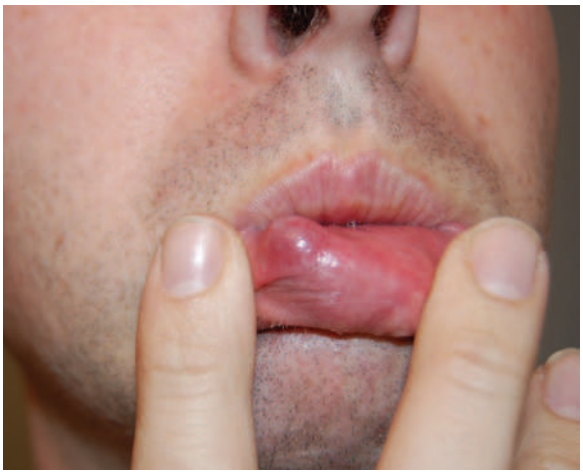

FIGURE 49-28 Mucocele (typically occurs on inside of lower lip). *(Courtesy Dr. James Burns.)*

often form, spontaneously drain, then recur repeatedly. Most eventually disappear without surgical intervention. No emergency treatment is necessary (Figure 49-28).

EXODONTIA (TOOTH EXTRACTION)

A tooth extraction is considered definitive treatment and should not be attempted in the field except under extraordinary circumstances (Box 49-7). Extraction requires training, special instruments, and profound anesthesia, all which may be difficult to obtain. Never extract a tooth without a definitive diagnosis. For example, symptoms thought to originate in a maxillary molar may have originated in a lower molar or muscular trigger point. Premedication with a sedative or narcotic may be necessary. Intraoperative and postoperative complications are common. Focus on treating pain and infection with local anesthetics, analgesics (e.g., ibuprofen, 600 mg PO q6h as needed, or hydrocodone [7.5 mg] and acetaminophen [750 mg] [Vicodin ES] PO every 4 to 6 hours as needed), I&D, and antibiotics (see Acute Apical Abscess/Cellulitis, earlier) as appropriate.[35,43] Extraction (or other definitive care) can be rendered after evacuation, because a seemingly doomed tooth can often be saved.

Before exodontia, consider the degree of mouth opening, relationship of the root to its corresponding maxillary sinus, condition of the clinical crown, alignment of the tooth in the dental arch, and history of previous endodontic treatment (after root canal treatment, a tooth is usually very brittle).[18] Weigh possible complications arising from the contemplated extraction, including fractured roots, fractured alveolus, fractured mandible or tuberosity, damage to adjacent teeth and restorations, soft tissue injury, loss of root tip in a sinus, prolonged bleeding, and infection.

BOX 49-7 Factors to Consider Before Extraction of Teeth

- Desires of patient
- Patient's medical history
- Available alternative treatments
- Difficulty/desirability of evacuation
- Certainty of diagnosis (Are you sure you have the correct tooth?)
- Possible complications if tooth is not extracted
- Factors relating to difficulty of procedure
  - Mobility or immobility of the tooth
  - Position of the tooth
  - Condition of tooth structure above gingival margin
  - Patient's mouth-opening ability
  - Available supplies and instruments
  - Experience of rescuers
- Possible complications arising from extraction
  - Fractured root(s)
  - Fractured alveolus
  - Soft tissue injury
  - Root tip lost in sinus
  - Prolonged bleeding
  - Localized osteitis ("dry socket")

If, after careful consideration, extraction is deemed the best course of treatment, proceed as follows. Review the patient's medical history. Rule out history of bleeding disorders, severe cardiovascular disease, immunocompromise, or dental implications for many frequently prescribed medications.[5] Discussion of indications for dental surgery in patients receiving anticoagulant therapy is beyond the scope of this chapter. Some patients (e.g., those with cardiac conditions or recent prosthetic implant) may require premedication with antibiotics.[53] (see Boxes 49-1 to 49-3). Consider the patient's emotional state, and decide if sedation is prudent.

Plan the procedure, and gather all necessary equipment. Secure a good light source and means to keep the operative field dry (i.e., suction and/or plenty of gauze). Support the patient's head. Place the patient in a supine position with the head slightly elevated. Obtain good local anesthesia, and test by touching soft tissues with a sharp instrument. A 10 × 10–cm (4 × 4–inch) gauze curtain placed in the rear of the patient's mouth prevents aspiration of teeth and debris.

Teeth are inflexible and brittle. Heavy forces, especially if applied quickly (high acceleration), will break teeth. Bone has more flexibility, the degree of which depends on the individual patient. Judiciously applied, moderate forces slowly expand bone. The tooth will eventually be delivered in the same direction as if it continued to erupt. Do not attempt to initially "pull" the tooth in that direction. The direction of force needed to loosen a tooth depends on the root's anatomy. A straight, conical root can be loosened by twisting forces. This technique often works well on upper front teeth. Sometimes a tooth can be removed by alternating 30 seconds of steady pressure toward the cheek with 30 seconds of steady pressure in the opposite direction, until the root gradually loosens.[55]

A variety of instruments can be used to apply force to the tooth. Elevators are firmly wedged between the tooth and bone in the interproximal area. Avoid putting pressure on adjacent teeth. Forceps should be applied as far apically as possible. Spend time working forceps well under the gingiva. Lower molars are often removed with "cowhorn" forceps, which are designed to apply force to the tooth in a coronal direction simply by squeezing the handles. An experienced exodontist using careful technique and a single forceps (e.g., a No. 150 universal extraction forceps) will have success extracting almost any nonimpacted tooth, whereas a reckless operator using dozens of specialized instruments will have difficulty.[18,55]

Despite utmost care, a tooth may break during the extraction procedure. If this occurs, stop and reevaluate. The root canal will probably now be exposed. If treating a case of infection, perhaps drainage can be obtained through a root canal procedure. If treating a case of severe pulpitis, perhaps pulpal tissue can now be removed. When rendering emergency care, it may not be necessary to remove the remaining root. Some teeth are impossible to remove without sectioning or can be removed in only one direction.

After removing a tooth, examine the root to confirm that it was entirely extracted. If necessary, remaining fragments can be removed later. Compress the expanded socket using your thumb and forefinger. If gingiva is loose, place a suture to help speed healing. Have the patient apply direct pressure to the wound by biting firmly on a gauze pack for 30 minutes while sitting in an upright position. Complete hemostasis may require several hours of steady pressure. Caution the patient to avoid rinsing, spitting, toothbrushing, and smoking for 24 hours. For the first day, apply cold packs in a cycle of 15 minutes on and 15 minutes off, and use analgesic antiinflammatory medication (e.g., ibuprofen) to reduce swelling and pain. The day after surgery, have the patient rinse with warm saline to cleanse the area.

Persistent bleeding several hours after the extraction is a common postoperative complication. This may be accompanied

by a poorly organized clot that resembles a piece of raw liver growing out of the socket. Remove this clot, and have the patient apply firm, uninterrupted pressure to the bleeding socket by biting on a gauze pack or wet tea bag (tannic acid helps promote clotting) for 20 minutes. If patient cannot do this, apply manual pressure. If bleeding continues after 20 minutes, consider packing the socket with hemostatic gauze (e.g., Gelfoam, Surgicel) or sterile gauze. If needed, consider suturing. Resume direct pressure.

Acute alveolar osteitis ("dry socket") is another postextraction complication. This typically occurs about 3 days after a dental extraction. The patient reports moderate to severe pain, foul odor, and a bad taste. Examination reveals an empty socket and exposed bone caused by loss of blood clot, but no suppuration. Treat by anesthetizing the area, then gently irrigate with warm saline, and pack with a strip of gauze dipped in eugenol. Administer an oral analgesic. Change the pack every 24 to 48 hours until the symptoms subside, which may take up to 10 days. The patient should avoid drinking alcohol or carbonated beverages and should not use tobacco products during treatment.

LOCAL ANESTHESIA

Local anesthesia is a prerequisite to many emergency dental, oral, and maxillofacial procedures. Anesthesia of any upper tooth and most lower teeth, along with associated buccal and lingual soft tissues, can be obtained by infiltration. Place approximately 2 mL of local anesthetic solution as close to the tooth's apex as possible, just above the periosteum on the buccal and lingual/palatal side of the teeth to be anesthetized (Figure 49-29). By holding the syringe parallel to the tooth's long axis, the needle tip is guided in the proper direction. Articaine (Septocaine) 4% with 1:100,000 epinephrine has become the most widely used local anesthetic in dentistry.[54] Its 4% concentration makes it better suited for infiltration anesthesia than other local anesthetics. Lidocaine 2% with 1:100,000 epinephrine is also often used; bupivacaine (Marcaine) 2% with 1:200,000 epinephrine is useful for longer-duration pain relief. Any available local anesthetic can be substituted if needed.

Mental nerve block is a simple method for obtaining anesthesia of teeth and the buccal mucosa from the second premolar forward. Proceed as for the infiltration, but the target area is the mental foramen located at the apex of the second premolar (Figure 49-30). After depositing anesthetic solution, gently massage the area for 30 seconds. To obtain additional lingual anesthesia in this area, lingual infiltration will be necessary.

The inferior alveolar nerve block is more difficult to learn but produces anesthesia of all the lower teeth up to the midline, as well as buccal soft tissues forward of the mental foramen. This injection also blocks the lingual nerve, producing numbness of the anterior two-thirds of the tongue and lingual gingiva (Figure 49-31). The target area is the pterygomandibular raphe. In vertical dimension, the raphe bisects the occlusal plane of the upper and lower teeth. Have the patient open the mouth widely. Approach

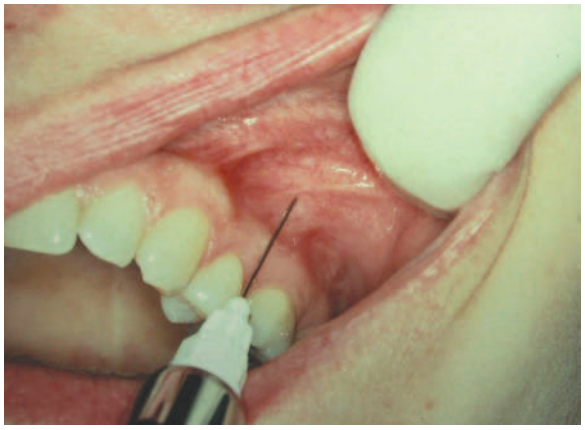

FIGURE 49-29 Technique for infiltration of local anesthetic.

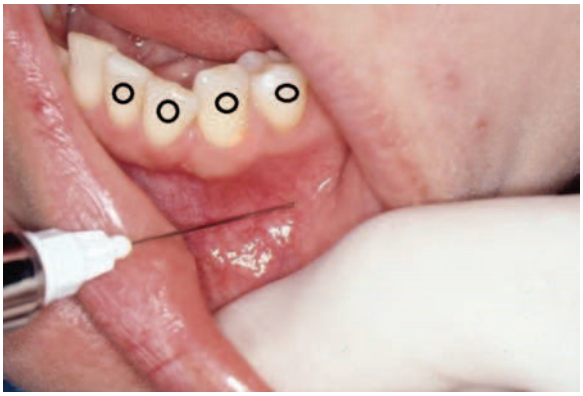

FIGURE 49-30 Technique for blocking inferior alveolar nerve near mental foramen (mental nerve block). Marked teeth will be anesthetized.

the area to be blocked from the opposite side. The syringe barrel should be located at the contralateral commissure. The needle tip will then touch the most distal end of the pterygomandibular raphe.[48] Insert the needle slowly until contact is made with bone. When bone is contacted in the target area, deposit anesthetic solution. Experienced dentists miss the target area, and thus fail to produce adequate anesthesia, in approximately 10% of injections for inferior alveolar nerve block. Repeat as necessary.

DENTAL FIRST-AID KIT

Most supplies needed to treat dental emergencies can be found in a good, general-purpose first-aid kit. These include antibiotics and analgesics, gauze, tweezers, scalpel, hemostat, and sutures. Items specific to dental emergencies can be added to a wilderness first-aid kit without significant space or weight penalties.

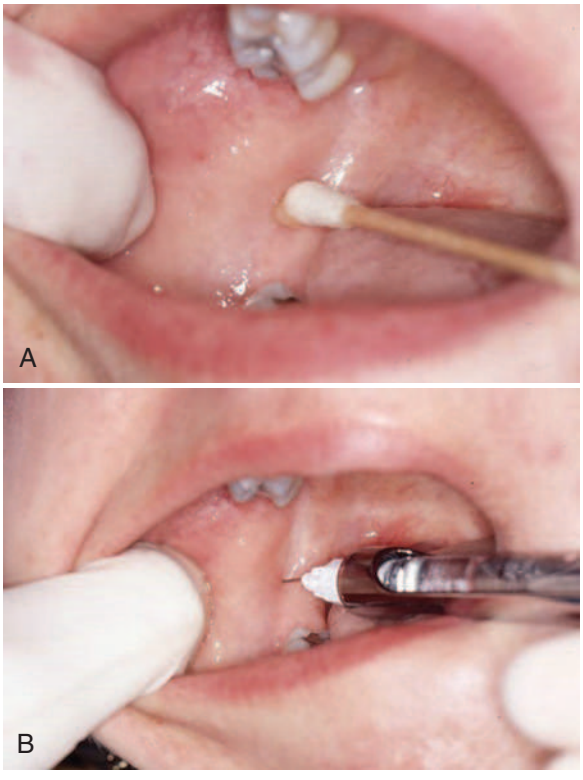

FIGURE 49-31 **A,** Location for inferior alveolar nerve block demonstrated while applying topical anesthesia. **B,** When administering inferior alveolar block, target area lies on medial surface of mandibular ramus, halfway between the rescuer's thumb and forefinger. Keep the syringe parallel to the plane of the lower teeth.

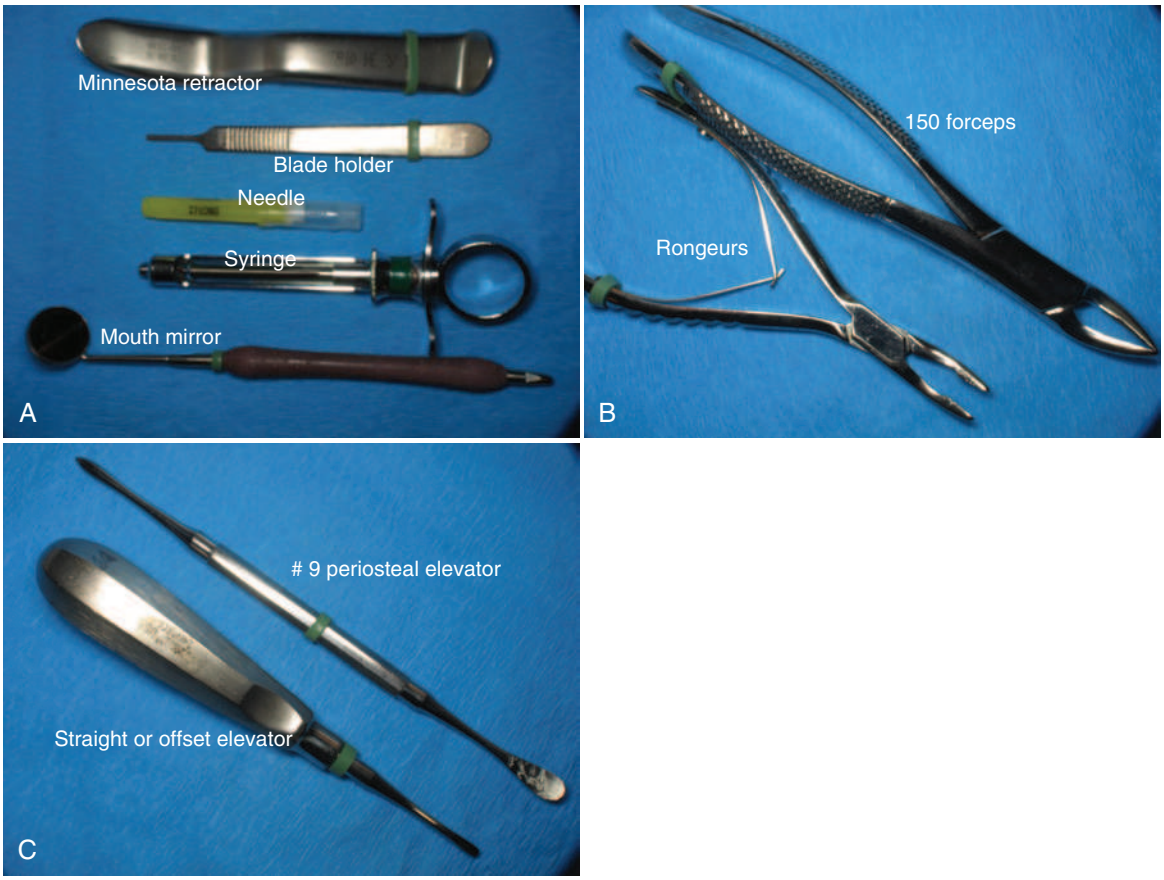

FIGURE 49-32 **A** to **C,** Instruments recommended for a dental first-aid kit.

Cavit is temporary filling material that requires no mixing and is easy to use. Squeeze a small amount of material from the tube and place it directly into the target dental cary ("cavity"). Wet a dental packing instrument (or cotton-tipped applicator or toothpick) to prevent sticking, and pack the Cavit well. Remove any excess. Have the patient bite to displace material that would interfere with occlusion. This particular filling material sets up firm a few minutes after contact with saliva (see Video 49-3).

Temp Bond is a useful two-phase material used to recement loose fillings or to replace portions of missing fillings. Open the material according to directions, and place on a nonporous surface to mix. Once mixed, apply inside the mouth (for missing fillings) or to the inside of the loose crown (when recementing). Use the plain end of a cotton-tipped applicator or a toothpick to place material into the missing filling or crown (see Video 49-2). Zinc oxide–eugenol temporary cements (e.g., Temp Bond, IRM) have advantages compared to Cavit, most importantly the soothing effect of eugenol on painful teeth (e.g., with pulpitis) or sensitive teeth (e.g., with dislodged fillings or crowns).

A more complete kit for extended expeditions should include a No. 150 universal extraction forceps, periosteal elevator, rongeurs, Minnesota retractor, and a straight elevator for extracting teeth (Figure 49-32). Complete the kit with a mouth mirror, Orabase with benzocaine, Temp Bond/IRM, Cavit, orthodontic wax, dental floss, dental syringe, 27-gauge needles, and anesthetic cartridges. These items fit in a small case to create a kit that weighs approximately 14 oz. A customized dental first-aid kit is preferred over commercial dental "travel kits," which contain unnecessary items and lack essential ones.

In austere conditions, techniques must often be adapted or improvised depending on items available. For example, Figure 49-12, *D,* shows how a suture can be used to splint an avulsed or extruded tooth. A temporary filling can be fashioned from softened candle wax, an emery board can be used to smooth a sharp tooth, and a pocketknife can be used to perform a drainage procedure.

PREVENTION

The vast majority of dental emergencies can be prevented, beginning with pretrip planning. Before any extended travel to a remote area, a thorough dental examination, radiographs, periodontal care, and treatment of potentially troublesome teeth are indicated. The Peace Corps requires certification of dental examination and treatment before members are assigned to developing countries. The National Science Foundation has similar requirements and requires extraction of impacted and unopposed third molars before assignment to Antarctica.

In the field, minimal precautions offer significant benefits. Lip balm with sunscreen can inhibit herpes labialis outbreaks. Routine personal oral hygiene will prevent many odontogenic infections and painful inflammatory conditions. Ideal care should include twice-daily toothbrushing and flossing. Toothpaste is not essential. Mechanical removal of plaque and stimulation of gingiva are the most important aspects of oral care. The fuzzy end of a hickory twig or even a finger can be used if a toothbrush is not available. In the wilderness, daily oral hygiene not only helps prevent dental emergencies but may contribute to an overall sense of well-being (e.g., when an expedition is tent-bound because of weather).

Custom-made mouth guards prevent more than 200,000 injuries every year in interscholastic sports.[33] Mouth guard use in backcountry recreation, now almost nonexistent, would likely prevent many dental injuries. There is no scientific evidence that mouth guards can prevent traumatic brain injury.[15]

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CHAPTER 50

Wilderness Cardiology

AARON L. BAGGISH AND BENJAMIN D. LEVINE

The wilderness environment places high demands on the cardiovascular system. Certain wilderness activities, including diving, long-distance trekking, and high-altitude mountaineering, pose specific challenges to the cardiovascular system by imposing additional environmental stressors, such as water immersion or hypoxia, on top of the physical demands of the specific activity. In addition, many wilderness endeavors involve prolonged exposure to high or low ambient temperatures, posing challenges to thermoregulation. As popularity of wilderness adventures continues to grow, the number of people with established cardiovascular disease or at increased risk for disease who partake in wilderness activities also is rising. Probably the most important consideration of wilderness activities for patients with known cardiovascular disease is the remote environment, which makes modern medical technology and "usual" advanced-level care partially or totally inaccessible. Any patient with cardiovascular disease who chooses to participate in wilderness activities needs to accept the risk that a cardiovascular event that occurs in a remote environment may have a more adverse outcome than a similar event occurring in a location with access to modern medical resources.

This chapter reviews basic cardiovascular physiologic adaptations to wilderness environments. Although basic cardiovascular responses to exercise are central to all wilderness activities, a detailed discussion of exercise physiology is beyond the scope of this chapter, and the reader is referred elsewhere for this information.[8,39] We also review cardiovascular conditions of relevance to the wilderness traveler and physician, with emphasis on basic management of common cardiovascular emergencies in the wilderness setting.

CARDIOVASCULAR SYSTEM RESPONSE TO SPECIFIC WILDERNESS ENVIRONMENTS

HYPERBARIA (UNDERWATER DIVING)

Diving places a unique combination of physical stressors on the cardiovascular system. The physiologic stress of diving arises from the combination of underwater immersion and sustained moderate-intensity exercise. Immersion significantly reduces the effect of gravity on the diver's body and thus on the lower-extremity venous blood pool when the diver is upright.[24] This results in central redistribution of blood, with increases in intrathoracic blood volume and right atrial and ventricular filling pressures.[25] The direction and magnitude of the fluid shift induced by water immersion depend on the position of the diver in the water. If the diver is upright, the extensively researched physiology of upright water immersion applies. However, if the diver is horizontal or head-down, entirely different hydrostatic gradients are induced. These physiologic changes may be dynamic as the diver changes position in the water. For upright divers, as dictated by the Frank-Starling phenomenon, increase in cardiac preload leads to immediate increases in stroke volume, cardiac output, and myocardial work. Patients with cardiomyopathy, with or without prior congestive heart failure (CHF), may be especially sensitive to cardiac volume overload and may become acutely symptomatic with these sudden shifts in volume status.

Physical exertion is an important contributor to overall cardiovascular stress of underwater exercise. Divers typically use large-muscle groups at low intensity to propel themselves through the water, although this depends on the experience of the diver and the nature of the dive. This action may be continuous for the entire duration of the dive. The cardiovascular system facilitates this activity by increasing heart rate, stroke volume, and cardiac output. This translates into mildly increased myocardial oxygen demand and may cause myocardial ischemia in patients with underlying coronary artery disease (CAD) who are not well compensated. Fear, excitement, and emotional stressors may compound the physical stresses of diving, especially for inexperienced divers.

The underwater environment can be particularly remote; if decompression stops are indicated, emergency ascent and access to medications (e.g., nitroglycerin) or medical care can be complicated. As with many other wilderness activities, it is not only the stress of the activity that is problematic, but also the risk of a cardiovascular event occurring in a remote setting.

The diving reflex is a unique autonomic reflex response to water immersion that has direct relevance to the safety of this popular sport. It was first described in 1870 by the French physiologist Paul Bert, who observed that the heart rate slowed dramatically during forced water submersion. The full diving reflex response involves transient slowing of heart rate, typically by 20 to 30 beats per minute, with subsequent reduction in cardiac output accompanied by peripheral vasoconstriction to maintain systemic blood pressure.[7] The reflex is mediated by increased vagus nerve activity. Heart rates lower than 10 beats/min have been measured in accomplished breath-hold divers.[22] Although typically well tolerated, such intense bradycardia may result in supraventricular and ventricular escape arrhythmias in susceptible individuals.[52]

Individuals with underlying heart disease may poorly tolerate diving, as evidenced by data that document a significant percentage of annual diving fatalities attributable to cardiac causes. Depending on the age of the population, sudden cardiac death and myocardial infarction account for 12% to 27% of annual diving fatalities.[19] This statistic does not mean that diving "caused" the fatality, but only that acute coronary events occur in middle-aged divers who may not have access to usual emergency care if an event occurs during diving. Adequate screening for underlying cardiovascular disease is essential before any diving excursion, especially in patients with known disease. Individuals with significant cardiovascular disease, including incompletely revascularized CAD and cardiomyopathy/CHF, are typically at high risk for complications during diving and should be given restrictive recommendations. In addition, patients who have undergone coronary artery bypass grafting (CABG) and had chest tubes placed occasionally have scarring and air trapping that could place them at risk for pneumothorax during compressed-air diving. At the least, a chest radiograph should document no evidence for scarring, and occasionally, other tests for air trapping, including computed tomography (CT) of the chest, should be considered. Recommendations for patients with cardiac disease who want to participate in competitive sports may be useful for diving and other wilderness activities.[47] If a patient has normal left ventricular function, has been well revascularized, and has no provocative ischemia or arrhythmias, consideration could be given to allow diving. Any recommendation to allow diving should have the corollary that divers also put their dive partners at risk if an episode of disability or incapacitation requires assistance or a rescue.

Decompression sickness (DCS) is a multisystem disease process caused by sudden drops in surrounding pressure. The underlying

pathophysiology of DCS involves formation of venous gas emboli that develop when tissue or blood that is supersaturated with nitrogen is exposed to rapid reductions in ambient pressure. This process may occur during too rapid an ascent from undersea depths. Although experienced divers routinely employ techniques to reduce the risk of DCS, including use of algorithms to determine safe ascent profiles, DCS remains somewhat unpredictable.[59] The risks and clinical implications of DCS in the presence of a patent foramen ovale (PFO) have been topics of considerable interest and controversy.[23,49] In theory, venous gas bubbles that form during DCS may migrate from the venous to the arterial circulation, from where they may further travel to end organs, including the brain, with such consequences as migraine headache and stroke. Although the true risk of PFO-related DCS outcomes remains incompletely understood, factors such as PFO size, prior documented DCS, and diving depth/time of exposure have been reported.[55] Recreational divers who typically obtain relatively shallow depths for short periods apparently are at minimal risk. Individuals who dive to deeper depths and for longer periods, a typical profile for commercial and military personnel, may be at substantial risk for PFO-related DCS complications.

We recommend that any diver with an established PFO, particularly with a history of prior DCS, who plans to engage in lengthy deep-water dives, discuss the inherent risks with a clinician well versed in dive physiology. In select cases, percutaneous PFO closure may be a reasonable consideration.[31] However, the vast majority of sport and recreational divers, particularly those who follow established safe ascent profiles, will not require any intervention. Also, it must be emphasized that currently, no data document reductions in DCS incidence or severity after PFO closure. Diving with a PFO is controversial in many ways. Just because a PFO increases risk does not mean that closing the PFO decreases risk, and the known risk of complications from closure might outweigh the protection. For those whose livelihood depends on diving, particularly if they have had an episode of DCS, it may be reasonable to accept the risk/benefit ratio of closure. For a recreational diver who dives infrequently and then only to shallow depths, a conservative dive profile may be sufficient.

HYPOXIA (HIGH-ALTITUDE ACTIVITY)

The cardiovascular system's response to hypoxia is a dynamic process that begins immediately on exposure and evolves over days, weeks, and years of prolonged exposure. Although the temporal nature of this response has inherent interindividual variability, it is useful to consider the response in two distinct phases: acute hypoxia and sustained hypoxia. Figure 50-1 summarizes important changes in cardiovascular function during acute and sustained hypoxia.

Acute hypoxia decreases alveolar and arterial oxygen content, leading to activation of the sympathetic nervous system.[34,48] This

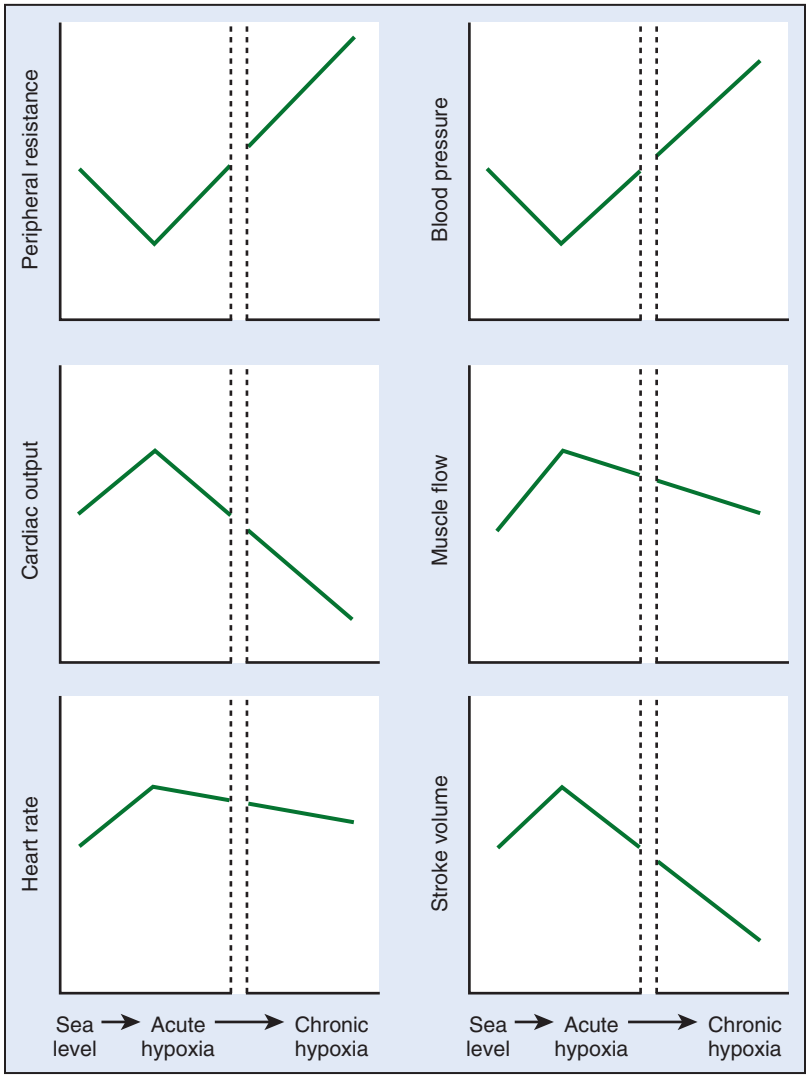

FIGURE 50-1 Changes in cardiovascular parameters during acute and sustained exposure to hypoxia. *(Modified from Baggish AL, Wolfel EE, Levine BD: Cardiovascular system: Heart and systemic circulation. In Swenson ER, Bärtsch P, editors:* High altitude: Human adaptation to hypoxia, *New York, 2014, Springer.)*

sympathetic activation is the key element of the acute hypoxic response of the cardiovascular system. Increased sympathetic activity (with or without vagal withdrawal) leads to increased heart rate and thus increased cardiac output. However, peripheral vasoconstriction, the expected vascular response to increased sympathetic tone, appears to be blunted by release of local vasodilatory substances, constituting a phenomenon equivalent to the "functional sympatholysis" that occurs during sustained endurance exercise.[30] Thus, cardiac output increases while systemic vascular resistance and blood pressure decrease transiently. Acute hypoxia also causes dilation of the coronary arteries, although in CAD patients who have endothelial dysfunction, appropriate coronary dilation may not occur. There may even occur paradoxical vasoconstriction.[6]

As hypoxia becomes sustained, arterial oxygen content increases because of hemoconcentration, ventilatory acclimatization, and increased red blood cell mass. These changes lead to some abatement of the hyperdynamic state of the circulatory system. Ventricular contractile function remains normal, but reductions in plasma volume and ventricular filling lead to decreased stoke volume, as predicted by the Frank-Starling mechanism.[32] Persistent sympathetic hyperactivity leads to down-regulation of cardiac beta receptors[61] and increased vagal activity,[12] which combine to promote reduction in resting heart rate. However, resting heart rate remains elevated during sustained hypoxia, when compared to sea-level conditions. These heart rate and stroke volume reductions lead to an overall decrease in cardiac output, especially at maximal exercise. In the systemic circulation, blood pressure gradually rises as the peripheral mechanisms responsible for local vasodilation diminish. For patients with hypertension, this sympathetic activation may result in substantial increases in blood pressure during high-altitude excursions.

High-altitude activities, such as hiking, trekking, and mountaineering, also generally include sustained low-intensity exercise for prolonged periods. The combination of reduced arterial oxygen content and perhaps paradoxical vasoconstriction during exercise (increased myocardial oxygen demand) results in provocation of myocardial ischemia with acute altitude exposure at work rates somewhat less than that provoked at sea level.[42] However, with at least 5 days of acclimatization, responses near those at sea level can be restored. Mountain walking, even at the relatively moderate altitudes in the Alps, is associated with slightly increased risk for sudden cardiac death,[13] although this risk seems greatest in those who are least fit. Even brief periods of acclimatization may reduce this risk substantially and should be considered mandatory for patients with known cardiovascular diseases in the mountains.[43] Overall, there is one sudden cardiac death per 780,000 hiking hours, one sudden cardiac death per 1,630,000 skiing hours in the Alps,[13] and one "cardiac event" per 957,000 hours of mountain activities,[53] which increases substantially after age 50 to 60 years. This risk seems somewhat higher than the reported one death per 3 million jogging hours.[56] For patients with known CAD, if they are adequately revascularized, exercise at high altitude appears reasonably safe.[21,54]

HYPERTHERMIA

The healthy human body is capable of tolerating and even successfully performing vigorous physical exercise during exposure to high ambient temperature. The hypothalamic thermoregulatory center prevents elevation of core body temperature during ambient heat exposure by causing peripheral vasodilation and increased sweat production. Humans thermoregulate predominantly by sending blood to the skin. During acute heat stress, up to 8 to 10 L/min of cardiac output may be directed to the skin (and therefore unavailable to other organs) to maintain body temperature. Heat exposure may be poorly tolerated especially by patients with compromised circulation, such as those with heart failure.[5,16] With ample oral hydration and electrolyte repletion, moderate physical exercise can be performed by those with significant thermal exposure for extended periods, although safe performance requires adequate heat acclimatization. The absolute intensity and duration of work that a given individual can perform in the heat are dictated by numerous factors, including prior heat acclimatization, physical fitness, and relative humidity. Drug use, particularly sympathomimetics such as cocaine, can greatly alter the response to thermal stress and may also be an important complicating factor.[14]

Heatstroke occurs when this system fails and core body temperature rises. Heatstroke is defined clinically as a core body temperature that rises above 42° C (107.6° F) and is accompanied by hot, dry skin and central nervous system abnormalities, such as delirium, convulsions, and coma.[11] Although heatstroke has long been recognized as a complication of wilderness travel and extreme physical activity in hot climates, recent data confirm its relevance in organized sporting activities, such as competitive long-distance running.[17,63] Cardiovascular complications of heatstroke include atrial fibrillation, various electrocardiographic (ECG) abnormalities (T-wave changes, prolongation of QT interval), pulmonary edema, pericardial effusion, right ventricular dysfunction, hypotension, and ventricular arrhythmias.[64]

HYPOTHERMIA

Classically, *hypothermia* is defined as core body temperature less than 35° C (95° F). Hypothermia may develop in numerous wilderness scenarios, including but not limited to cold-water immersion (see Chapter 8), snow burial (see Chapter 4), and exercise in cool to cold ambient temperatures (see Chapter 7). Unlike heat exposure, humans do not acclimatize to an appreciable degree to cold exposure; survival in the cold depends on behavior (e.g., clothing, shelter). The initial response to cold exposure is increased sympathetic nervous system activity, leading to peripheral vasoconstriction and tachycardia.[37] This causes a rise in systemic arterial blood pressure and a tendency toward central redistribution of blood volume. Acute exercise in the cold in CAD patients is associated with increased risk of acute coronary events and is thought to be a particularly dangerous combination.

With sustained *mild* hypothermia (core body temperature of 33° to 35° C [91.4° to 95° F]), a central volume shift causes diuresis, which can lead to dehydration. As hypothermia becomes more pronounced (*moderate* hypothermia, 30° to 33° C [86° to 91.4° F]), heart rate and cardiac output fall, and the electrocardiogram may show characteristic findings of prolonged QT interval and Osborn waves. Bradycardia, caused by direct slowing of sinoatrial function and concomitant changes in tissue pH, oxygen concentration, and electrolyte concentration, becomes more marked as core temperature continues to decrease.[44] The drop in core temperature and pulse rate typically follows a linear relationship; therefore, deviations from this pattern, particularly tachycardia in the moderately hypothermic patient, should raise suspicion of a secondary process, such as toxin ingestion or hypovolemia. Premature atrial/ventricular beats and atrial fibrillation typically occur in the moderate hypothermia range.

In *severe* hypothermia (core body temperature <30° C [86° F]), there is significant reduction in stroke volume, heart rate, and blood pressure. All types of atrial and ventricular tachyarrhythmias can occur during severe hypothermia.[18] Importantly, rough handling or jostling of the severely hypothermic patient can trigger arrhythmias, so care should be taken to avoid unnecessary physical manipulation of these patients.[37] At temperatures below 25° C (77° F), spontaneous ventricular fibrillation and asystole may occur. As discussed later, victims of hypothermia may tolerate periods of cardiac arrest longer than those who sustain cardiac arrest during normothermia. Therefore, the appropriate window for initiating and continuing resuscitation for a hypothermic patient may exceed that in other cardiac arrest situations.

SCREENING AND PREPARATION FOR WILDERNESS TRAVEL

ASYMPTOMATIC WILDERNESS ADVENTURERS

Participation in wilderness activities can place considerable stress on the cardiovascular system. Healthy individuals without known

preexisting cardiovascular disease should seek medical consultation before wilderness activity from a clinician familiar with demands of the particular wilderness environment, similar to recommendations for individuals considering beginning an intensive exercise program or participation in competitive sports. Assessment of the asymptomatic individual should focus on determining risk of an underlying, previously unrecognized cardiovascular condition. A detailed medical history focused on prior exercise experience, wilderness adventure experience, medications, allergies, and detailed family history of cardiovascular disease or premature sudden/unexplained death should be obtained. Individuals should be specifically questioned about established cardiovascular risk factors, including tobacco use, hypertension, diabetes, family history of premature coronary disease, and dyslipidemia.

A comprehensive physical examination should be performed. This should begin with basic assessment of vital signs to exclude systemic hypertension and arrhythmias. Auscultation of the heart and lungs should be performed to exclude congenital or acquired valvular heart lesions and pulmonary vascular congestion. Peripheral pulse examination should be performed to detect the presence of atherosclerotic peripheral vascular disease and aortic coarctation. Although not specifically designed for wilderness travelers, the American College of Cardiology and American Heart Association athletic preparticipation medical history and physical examination template may prove to be a useful guide for clinicians evaluating persons who intend to travel in the wilderness.[46]

Asymptomatic wilderness travelers with a normal medical history and physical examination typically tolerate wilderness activity without cardiovascular complications and should not be restricted from any specific activities based on cardiac risk. Individuals with abnormalities detected during medical history or physical examination may require further testing. Some combination of 12-lead electrocardiogram, transthoracic echocardiography, exercise stress testing, and ambulatory rhythm monitoring may be useful in such individuals. The decision to perform one or more of these diagnostic tests should be individualized.

ESTABLISHED CORONARY ARTERY DISEASE

Atherosclerotic coronary arterial disease (ACAD) is the leading cause of death in middle-aged and older individuals in the developed world. Atherosclerosis is the process of arterial lumen narrowing caused by accumulation of lipids, inflammatory cells, and calcium within arterial walls. ACAD leads to clinically relevant myocardial ischemia in one of two principal ways. ACAD may progress steadily and often slowly over months to years to the point at which arterial lumen narrowing is sufficient to cause myocardial demand–related ischemia. In this setting, ACAD manifests as exertional chest discomfort referred to as *angina pectoris*. Patients with angina pectoris are managed clinically with medications to reduce myocardial oxygen demand (e.g., β-blockers, nitrates), prescribed exercise therapy, aggressive risk factor modification, and symptom-driven revascularization.

Alternatively, ACAD may present with sudden and complete occlusion of a coronary artery. This phenomenon is triggered by ulceration or rupture of an atherosclerotic plaque, with subsequent development of a platelet-rich clot, and may occur even in blood vessels that previously were not severely narrowed, and therefore may have been clinically silent. This dramatic and often catastrophic ACAD presentation often leads to myocardial infarction and/or a malignant ventricular arrhythmia. Effective management of plaque rupture and complete coronary arterial occlusion requires pharmacologic (systemic thrombolytics) or mechanical reperfusion (percutaneous coronary angioplasty and stenting), which are typically not feasible in the wilderness environment.

Patients with clinically stable ACAD (i.e., patients fully revascularized or incompletely revascularized but without recent change in anginal threshold) who are on a well-tolerated medical regimen should not universally be excluded from wilderness activities. We recommend all individuals with established ACAD undergo some form of exercise stress testing before wilderness activity because this provides accurate assessment of exercise capacity and efficacy of medical therapy.[40] Stress testing using continuous 12-lead ECG monitoring can be done using a number of exercise modalities, including treadmill running/walking and upright or supine cycle ergometry. Figure 50-2 shows typical ECG findings of myocardial ischemia. The choice of exercise modality can be made based on patient and clinician preferences. Concomitant myocardial imaging may be necessary to achieve adequate diagnostic accuracy in patients with certain pretest features (Box 50-1). Imaging can be performed using nuclear perfusion tracers or echocardiography. Individuals with clinically stable ACAD who have normal left ventricular function, and no myocardial ischemia during exercise testing performed at least to a workload commensurate with the demands of the expected activity, should not be restricted from wilderness activity based on cardiovascular disease.

In general, we routinely work to tailor exercise testing to match the anticipated demands of the individual patient. Although this approach is feasible for athletes who engage in organized sports, laboratory-based simulation of wilderness environments is inherently limited. No laboratory exercise test is a perfect simulator of the wilderness environment. Even use of acute hypoxia or thermal stress during pretravel exercise testing may fail to

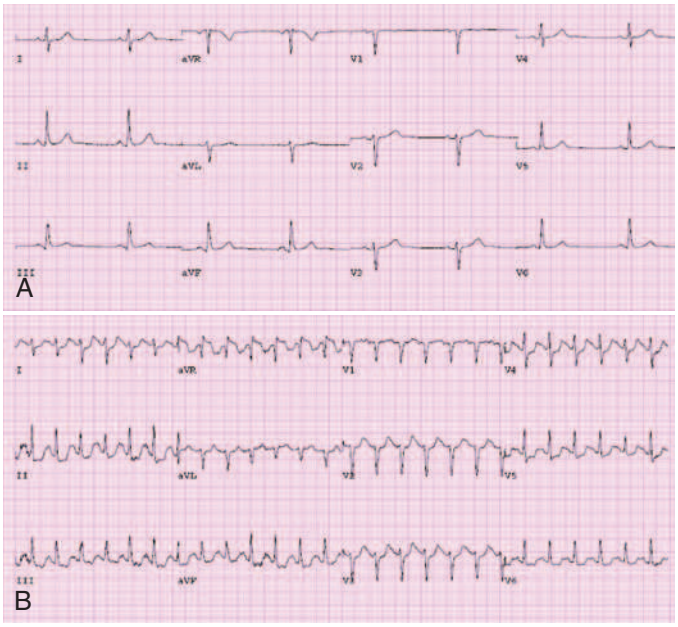

FIGURE 50-2 Representative example of 12-lead electrocardiogram at rest (**A**) and peak exercise (**B**) in a 56-year-old male patient with cardiac risk factors of diabetes and dyslipidemia who underwent screening before high-altitude travel. **B** demonstrates more than 1 mm of ST-segment depression in numerous contiguous ECG leads. This patient was found to have multivessel coronary artery disease with high-risk features necessitating coronary artery bypass grafting surgery.

BOX 50-1 Baseline Patient Characteristics Necessitating Myocardial Imaging as an Adjunct to Exercise Stress Testing for Establishing Diagnosis of Myocardial Ischemia

1. Prior percutaneous (PCI) or surgical revascularization (CABG) if ECG is abnormal
2. Preexcitation (Wolff-Parkinson-White) syndrome
3. Resting ST-segment depression ≥1 mm
4. Electronically paced rhythm
5. Left bundle branch block
6. Resting repolarization abnormalities caused by LVH or digitalis use

CABG, coronary artery bypass grafting; *ECG*, electrocardiogram; *LVH*, left ventricular hypertrophy; *PCI*, Percutaneous coronary intervention.

capture the demands of prolonged exposure. The combination of intense physical exercise and environmental extremes may precipitate myocardial ischemia that could not be triggered during stress testing. As such, we emphasize several principles to our ACAD patients who choose to engage in wilderness activity. First, we encourage all ACAD patients to be knowledgeable about the typical symptoms of this disease (e.g., exertional chest pain/pressure/tightness, inappropriate exertional dyspnea). Development of any of these symptoms during wilderness activity should prompt the patient to abort the activity and seek immediate medical attention. Second, we encourage all ACAD patients to prepare as fully as possible for the physical demands of wilderness activity. This is best accomplished by using a progressive exercise training program designed to mimic the specific challenges of the planned wilderness activity. Such patients should also be receiving optimal guideline-based medical therapy, including appropriate doses of statins. The combination of exercise training and cholesterol lowering is likely to normalize endothelial function as much as possible and limit the risk of wilderness activity.[27,29] Third, we emphasize the value of allowing ample time for wilderness environment acclimatization. Acclimatization techniques vary by activity and environment and may include gradual or staged high-altitude ascent or prolonged low-intensity exercise under hot conditions. Finally, we suggest that all ACAD patients develop a comprehensive evacuation plan before wilderness travel. This critical step requires lengthy discussions with trip planners (i.e., travel guide companies) and may be best accomplished by the purchase of individual travel evacuation insurance or services.

CARDIOMYOPATHY AND CONGESTIVE HEART FAILURE

Cardiomyopathy refers to abnormal function of the heart muscle. Cardiomyopathy may be inherited/congenital (e.g., hypertrophic cardiomyopathy, familial dilated cardiomyopathy) or acquired (e.g., ischemic cardiomyopathy, cardiomyopathy secondary to valvular pathology) and thus can affect individuals of all ages. Risk associated with wilderness activity in patients with cardiomyopathy is determined by etiology and severity. Certain forms of cardiomyopathy (e.g., hypertrophic cardiomyopathy) are associated with increased risk of sudden cardiac death in the context of vigorous physical activity. Patients with these forms of cardiomyopathy should either be excluded from wilderness activity or counseled extensively about the inherent risks associated with physical activity and wilderness isolation.

In addition to sudden death risk, cardiomyopathy may lead to heart failure (HF). HF is a syndrome defined as inability of the circulatory system to provide adequate circulation of blood to meet the demands of the body and to fill adequately at a sufficiently low pressure to avoid congestion. HF severity can be assessed based on symptom severity and results of an assessment of cardiac structure and function. Current practice guidelines utilize a HF severity classification scheme that incorporates symptom severity and cardiac structural/functional parameters (Table 50-1).[33] Cardinal symptoms of HF include dyspnea, fatigue, exercise intolerance, and systemic venous congestion, manifesting most frequently as peripheral edema. Noninvasive imaging, such as echocardiography, can provide important information that includes left ventricular size and ejection fraction.

Individuals with Class D HF are typically not capable of wilderness activity, so exposure to hypoxia, hyperbaria, or thermal extremes should be avoided. Individuals at risk for HF with (Class B) or without (Class A) documented structural or functional cardiac abnormalities should undergo assessment of functional capacity by exercise testing before participating in wilderness activity. Class A and B patients who demonstrate normal, age-appropriate exercise capacity during formal testing can be expected to tolerate wilderness activities without complications and do not require restriction. The most difficult clinical decisions arise when considering individuals with Class C disease. Class C individuals, particularly those with stable disease, can tolerate moderate-intensity exercise in controlled environments, such as cardiac rehabilitation programs, and may express interest in wilderness activities. These patients are at considerable risk for decompensation if activity intensity exceeds a certain threshold. Because this threshold differs greatly among Class C patients and may change rapidly in any given individual, we advise conservative recommendations in this patient population. In our opinion, the majority of Class C HF patients should be restricted from aggressive or extreme wilderness activities that significantly increase stress on the cardiovascular system. Clearance to perform low-intensity wilderness activity in this patient population must be considered carefully on a case-by-case basis.

TABLE 50-1 ACC/AHA Classification of Stages of Heart Failure

Heart Failure Stage	Definition
At Risk for Heart Failure	
Stage A	Patients at high risk for developing heart failure in the future but no functional or structural heart disorder
Stage B	Structural heart disorder but no symptoms prior heart failure at any stage
Heart Failure Present	
Stage C	Previous or current symptoms of heart failure in the context of an underlying structural heart disorder
Stage D	Advanced disease requiring hospital-based support, a heart transplant, or palliative care

ACC, American College of Cardiology; *AHA*, American Heart Association.

CONGENITAL HEART DISEASE

Congenital heart disease (CHD) is a term that includes a wide array of cardiac abnormalities that are present at birth. CHD includes abnormalities of the heart muscle, cardiac valves, coronary arteries, and electrical conduction system and may be confined to the heart or a component of a systemic disorder. From a functional perspective, CHD can be categorized as either cyanotic or acyanotic. *Cyanotic* CHD is characterized by reduced arterial oxygen saturation resulting from right-to-left intracardiac shunting with direct venous-to-arterial transit of blood. Cyanotic CHD is relatively rare and involves complex and severe structural cardiac malformations, such as tetralogy of Fallot. Most persons with cyanotic CHD present early in life with failure to thrive or greatly reduced exercise capacity and are treated with surgical reconstruction.

Individuals with complex cyanotic CHD are at high risk for decompensation during wilderness activity and should be restricted for this reason. Among patients with successful surgical correction of cyanotic CHD, the risks associated with wilderness activity are highly variable. For example, patients who have undergone a Fontan operation and who do not have right-to-left shunting tolerate acute exposure to high altitude surprisingly well.[26] Nevertheless, the impact of chronic exposure to high altitude in such patients has not been well defined. The decision to prohibit or restrict activity in this patient population should be individualized in consultation with a cardiovascular specialist with expertise in CHD.

Acyanotic CHD, defined by the presence of an abnormality associated with normal arterial oxygen saturation, is more common than cyanotic disease. Common causes of acyanotic CHD include atrial and ventricular septal defects. In these conditions, intracardiac shunting of blood travels in a left-to-right direction and does not produce arterial hypoxemia. Many individuals with acyanotic CHD go undetected through childhood and adulthood and can be expected to have normal exercise capacity and life expectancy.

However, normal development and previous clinical stability in a patient with acyanotic heart disease does not necessarily indicate an acceptable risk for wilderness activity. This is

particularly true of wilderness activity that involves exposure to hypoxia, such as occurs during high-altitude travel. Hypoxia facilitates pulmonary vasoconstriction, leading to increased pulmonary vascular resistance. The increased PVR and resultant increase in right ventricular and atrial pressures may lead to right-to-left intracardiac shunting and conversion to cyanotic CHD physiology. The risk associated with wilderness travel in patients with common acyanotic CHD is variable and largely determined by the specific form and severity of the intracardiac lesion. Individuals with acyanotic CHD should consult with a cardiovascular specialist with expertise in CHD before wilderness activity.

Two other conditions deserve specific comment. One rare condition is congenital absence of the right pulmonary artery. This abnormality places patients at serious risk for high-altitude pulmonary edema (HAPE), even at relatively low altitudes, and should be considered whenever a patient presents with HAPE at altitudes below 2743 m (9000 feet). It is crucial for practitioners of wilderness medicine to be aware of this condition and counsel such patients to avoid high-altitude exposure.

The second condition is patent foramen ovale (see earlier discussion of decompression sickness). PFO is quite common, present in perhaps 5% of the adult population, and has been associated with HAPE.[2,41] However, there is no evidence that closing a PFO has any effect on exercise performance at high altitude or the risk of high-altitude illness. Therefore, such procedures should be guided by other clinical indications. For individuals with a known PFO wanting to travel to high altitude, consideration should be given to HAPE prophylaxis with a pulmonary vasodilator such as nifedipine[51] or tadalafil.[45]

ARRHYTHMIAS

Arrhythmias are usually categorized by site of anatomic origin. Supraventricular arrhythmias arise from foci above the atrioventricular node, typically in the atria or adjacent vessels, such as the pulmonary veins. In contrast, ventricular arrhythmias originate below the atrioventricular node within the ventricular myocardium or His-Purkinje conduction fibers.

In general, supraventricular arrhythmias are better tolerated than ventricular arrhythmias. Atrial fibrillation, one of the most common supraventricular arrhythmias, appears to be more common in seasoned endurance athletes than in sedentary, or more typically active, age-matched controls. Clinicians who care for wilderness travelers, many of whom are lifelong exercisers, should be familiar with diagnostic and management considerations for patients with atrial fibrillation. Many individuals experience completely asymptomatic supraventricular arrhythmias. Individuals with asymptomatic supraventricular arrhythmias who have no other forms of cardiac disease may participate fully in wilderness activities. Patients with a history of symptomatic supraventricular arrhythmias, including those taking medications for maintenance of sinus rhythm or heart rate control, should undergo exercise testing to assess for rhythm stability and circulatory sufficiency during high-intensity exercise. Carrying a short-acting β-blocker or a class 1C antiarrhythmic, planned for use following the "pill in the pocket" approach,[1] may be a useful precaution for such patients. Some patients with paroxysmal or chronic supraventricular arrhythmias are managed with anticoagulation. These patients may be susceptible to bleeding complications of trauma and should be advised to take appropriate precautions in the wilderness environment.

For patients whose atrial fibrillation is paroxysmal, careful weighing of the risk of recurrent atrial fibrillation in a remote environment (particularly at high altitude) and the consequent risk of stroke, compared with the risk of bleeding from prophylactic anticoagulation, must be discussed with the patient's cardiologist before undertaking wilderness activity. Patients with a history of ventricular arrhythmia, tachycardia, or fibrillation should be considered at risk for recurrence unless the underlying cause of the rhythm has been identified and corrected. We typically recommend restriction from high-intensity exercise, including competitive sports and wilderness activities.

IMPLANTED CARDIAC DEFIBRILLATORS AND PACEMAKERS

Implantable cardiac defibrillators (ICDs) are placed for primary and secondary prevention of sudden cardiac death in patients susceptible to malignant ventricular arrhythmias. Recent expansion of ICD appropriateness criteria has led to continually increasing numbers of patients with these devices.[20] By definition, individuals with ICDs are at high risk for arrhythmia and device discharge during strenuous activity.[36] Device discharge may be painful and render the patient temporarily incapacitated. Therefore, all wilderness activities during which transient compromises in cognitive or motor function may have serious consequences for the patient or partners are contraindicated in patients with ICDs. Examples include underwater diving, rock climbing, and hang gliding. Low- to moderate-intensity exercise may be appropriate for patients with ICDs. We encourage careful assessment of heart rate and rhythm response during formal exercise testing and comprehensive, individualized risk/benefit discussions in ICD patients who seek guidance about pursuing such activities.

Cardiac pacemakers are used in patients with native conduction system disease and associated symptoms. Although individuals with a pacemaker have no specific exercise limitations because of their device, early-generation pacemakers were not designed to accommodate the physiologic demands of exercise and resulted in reduced exercise capacity for recipients. Recent advances in pacemaker technology have been substantial. With respect to exercise capacity and wilderness activity, the most important advances involve the ability of newer devices to augment heart rate and therefore cardiac output during volitional exercise. Modern devices sense the need for heart rate/cardiac output augmentation by tracking some combination of patient body motion, respiratory rate, and properties of the intact native conduction system. The presence of a pacemaker should not be grounds for restriction from wilderness activity. Patients should undergo device interrogation for battery life and sensing/pacing thresholds before participation. Clinicians who care for patients with pacemakers that feature rate-response technology should be aware that the normal increases in respiratory rate that occur during acute and subacute high-altitude exposure may lead to increases in heart rate, even during periods of relative inactivity.

HYPERTENSION

Because blood pressure rises with chronic altitude exposure, hypertension that is well controlled at sea level may become uncontrolled at high altitude. Patients with hypertension, especially those with other comorbidities or who have had complications of uncontrolled or malignant hypertension, are advised to carry a small, wrist-type blood pressure cuff to check pressure. Before travel, they should discuss with their physician(s) a strategy to increase the dose of current medications or add a new drug(s) if necessary. Drugs with combined α/β-adrenergic blocking effects, such as carvedilol or labetalol, may be especially effective at high altitude, as may be other vasodilatory β-blockers such as nebivolol.[57,60]

BASIC MANAGEMENT OF CARDIOVASCULAR EMERGENCIES IN THE WILDERNESS ENVIRONMENT

CHEST PAIN

Myocardial ischemia most often presents with chest discomfort. The quality of this discomfort varies between individuals and may be reported as heaviness, tightness, or a dull ache. The chest discomfort attributable to myocardial ischemia is often accompanied by other symptoms, such as diaphoresis, nausea, dyspnea, and pain in adjacent anatomic structures, including the neck, jaw, or left arm. Among wilderness adventurers, new-onset chest discomfort without a clear musculoskeletal cause should be considered an ominous sign. It should be assumed that nontraumatic chest discomfort is indicative of an active coronary artery process

until proved otherwise. Definitive and effective therapy for active myocardial ischemia requires access to hospital-based resources.[3,4] As such, wilderness travelers who experience chest discomfort of suspected myocardial ischemic origin should place a high emphasis on gaining access to a capable medical facility with minimal delay. In some cases, assistance from search and rescue organizations with the capability to provide rapid transport may be appropriate. In the field, administration of salicylic acid (aspirin), preferably in a chewable and thus rapidly absorbed form, should be undertaken immediately as soon as myocardial ischemia is suspected. Nitroglycerin-based agents, additional anticoagulants (including low-molecular-weight heparin), and analgesic medications may be appropriate if administered in the field by a clinician trained in their use.

ARRHYTHMIAS

A rapid but regular heart rate with normal sinoatrial node origin (sinus tachycardia) may be caused by appropriate physiologic response to the intense physical or emotional stress of the wilderness environment. In contrast, pathologic tachyarrhythmias that develop suddenly may originate in the atria, atrioventricular node, or ventricles. Sinus tachycardia is typically associated with a clear stimulus, develops gradually as the stimulus intensifies, and abates gradually once the stimulus is removed, although it may also be a sign of inadequate compensation to altitude or heat. Sinus tachycardia is generally not associated with additional symptoms unless an individual harbors significant occult or previously documented heart disease. In contrast, pathologic tachyarrhythmias may start suddenly either at rest or during exertion, and typically fail to resolve gradually with activity cessation. They are often accompanied by symptoms that include lightheadedness, palpitations, and chest discomfort. Resolution of tachyarrhythmia is typically abrupt.

Differentiation between normal sinus tachycardia and a pathologic tachyarrhythmia can be difficult in the wilderness setting. A medical history of prior confirmed pathologic arrhythmia (e.g., Wolff-Parkinson-White syndrome, atrial fibrillation/flutter) or underlying structural heart disease can be helpful. Brief physical examination may provide additional clues. The patient's pulse should be assessed for rate, regularity, and beat-to-beat variability. The jugular venous column should be inspected for the presence of atrioventricular dissociation or flutter waves. Direct auscultation of the heart using a stethoscope or simply placing an ear on the patient's bare chest can provide clues about rhythm origin, such paradoxical splitting of the second heart sound (characteristic of left bundle branch block that accompanies ventricular tachycardia, or supraventricular tachycardia with aberrancy) or presence of Rytand's murmur (a murmur of varying intensity that indicates advanced heart block).

Portable ambulatory rhythm-monitoring devices, some with smart phone telemedicine technology, are proving useful in the wilderness environment. Patients with established paroxysmal arrhythmia syndromes, working in conjunction with a cardiologist knowledgeable in wilderness medicine, may benefit from using such devices. Rhythm strips derived from portable devices in the field can be rapidly transmitted to providers, who may then assist with management and decisions regarding the need for trip revision or evacuation.

Management of the symptomatic patient with suspected symptomatic tachyarrhythmia should focus on rapid transportation to medical facilities with advanced cardiac life support (ACLS) capabilities.[10] When transportation to professional medical attention is not an immediate option, several therapeutic maneuvers can be attempted. Carotid sinus massage can be used to terminate several forms of tachyarrhythmia. External pressure on the carotid sinus stimulates baroreceptors that trigger increased vagus nerve activity and decreased sympathetic nervous system activity, thereby potentially terminating the arrhythmia. The patient with tachyarrhythmia should be placed in a supine or lateral decubitus position with the neck fully extended and turned away from the care provider. The carotid pulse should be palpated close to the angle of the jaw to approximate the common carotid artery bifurcation. Firm pressure should then be applied with a gentle rotating motion for 5 to 10 seconds. If there is no response, the procedure should be attempted on the patient's other side. The procedure should never be performed on both sides simultaneously. In patients who do not respond to carotid sinus massage, alternative vagus nerve stimulation maneuvers may be attempted. Prolonged breath holding, Valsalva maneuver, placing the face in cold water (to induce the "diving reflex"), or a strong cough against a closed glottis may also cause an adequate vagal response.

SYNCOPE

Syncope is defined as transient loss of consciousness accompanied by loss of postural tone. Syncope results from rapid reduction in cerebral blood flow that culminates in significant central nervous system hypoperfusion. Syncope may be attributed to underlying cardiovascular pathology that includes structural, valvular, and electrical heart disease. Such underlying processes need to be considered when evaluating the patient with a prior syncopal event. Most often, syncope occurs in healthy individuals without explanatory heart disease and is attributed to autonomic mechanisms, called "vasovagal," or neurally mediated, syncope. The physiology of neurally mediated syncope is beyond the scope of this text; the interested reader is referred to several excellent reviews.[35,38,50]

Neurally mediated syncope is common among physically fit individuals who may choose to engage in wilderness activity.[15] It has been reported to occur during the early phases of acclimatization to high altitude as a result of plasma volume loss, hyperventilation, and perhaps hypoxia-mediated vasodilation. Individuals predisposed to neurally mediated syncope may be at increased risk for syncope during wilderness activity that produces dehydration (prolonged strenuous exercise) or significant peripheral vascular dilation (thermal exposure). The individual with neurally mediated syncope will typically report presyncopal feelings of warmth, diaphoresis, and lightheadedness, which culminate in loss of consciousness ranging from several seconds to minutes.

Management of wilderness syncope begins with removing the patient from dangerous conditions. Individuals who succumb to syncope should be removed immediately from locations (e.g., partial water submersion, precarious heights) where transient loss of consciousness could lead to trauma or further complications. Patients should be assessed for spontaneous ventilation and presence of a regular pulse in a peripheral or central artery. If spontaneous cardiorespiratory function is confirmed, it is best to observe the unconscious individual for several minutes to allow spontaneous resumption of normal cognition and motor function. Once revived, efforts to correct or minimize the potential syncopal trigger, such as rehydration, active cooling, or activity cessation, are warranted.

A physician should assess all individuals who have syncope during wilderness activity in a proper medical facility as soon as possible to exclude underlying cardiac pathology. This assessment should include detailed history, physical examination, and 12-lead electrocardiogram, with further diagnostic evaluation on an individualized basis, unless typical "vasovagal" circumstances and symptoms are clearly present.

CARDIAC ARREST

The initial approach to the patient in a wilderness setting with presumed cardiac arrest, typically individuals with sudden collapse or those who sustain prolonged and severe hypoxia (e.g., avalanche burial, drowning) should be to confirm safety of the first responder(s) and accompanying medical team. Once the environment has been deemed safe for medical care, the patient should be assessed to confirm the absence of spontaneous circulation. This is performed by manual palpation of a large central artery (e.g., carotid, femoral) and simultaneous inspection of skin tone, motor activity, and chest wall motion. In patients with a palpable pulse but evidence of primary respiratory collapse, maneuvers to clear the upper and lower airways can be initiated.

Once the absence of spontaneous circulation is confirmed, resuscitation in the form of basic life support (BLS) measures should immediately be initiated. Historically, BLS has been dictated by the "ABC" acronym that referred to the resuscitation

sequence of airway, breathing, and circulation. The recommended approach to basic resuscitation now features a "CAB" approach (circulation, airway, breathing).[9] This strategy involves immediate initiation of chest compressions for unconscious patients without spontaneous or normal cardiorespiratory function. The logic for this paradigm shift is the belief that ample oxygen is present in the lungs and arterial system for several minutes after cardiac arrest, to avoid ischemia if blood can reach the target organs. Thus, immediate circulation of blood by chest compressions will facilitate optimal oxygenation of the brain and other key organs sooner than the previous, conventional, airway-first approach. The currently recommended sequence of adult BLS can be summarized as follows:

1. *Recognition of cardiac arrest:* First responders who encounter an individual following a witnessed collapse or with unconsciousness of unknown duration should first ensure that the scene is safe to assess the patient. Factors dictating scene safety vary by specific wilderness environment, so travelers should familiarize themselves with issues specific to the locations they visit. Once scene safety has been confirmed, the patient should be assessed for responsiveness. Loud verbal engagement with concomitant physical stimulation (shoulder tapping) in the form of the question, "Are you all right?" is recommended. The responder should simultaneously assess the victim's breathing. If the victim fails to respond and is found to have absent or abnormal breathing, cardiac arrest should be assumed and resuscitation should proceed.
2. *Chest compressions—"C":* Chest compressions should be initiated immediately after confirmation of confirmed cardiac arrest and an unsuccessful precordial thump. The rescuer should place the heel of one hand on the lower half of the victim's sternum and the heel of the other hand on top of the first so that the hands are overlapped and parallel. The adult sternum should be depressed at least 5 cm (2 inches) with chest compression, and chest recoil/relaxation time should approximately equal compression time. Compressions should be performed at a rate of at least 100 per minute. The initial sequence of compressions should be performed continuously for 1 minute. For responders without prior CPR education/certification, continuous chest compressions can and should be performed indefinitely without attention to airway management and rescue breathing. Certain wilderness environments (e.g., high altitude) have been associated with reduction in quality of chest compression, underscoring the need for practice in the field prior to travel.[62]
3. *Airway—"A":* Ensuring airway patency remains an important component of the overall resuscitation effort for responders trained in BLS techniques. In the wilderness environment, the head tilt–chin lift maneuver is the optimal manual strategy for opening a victim's airway. Individuals with appropriate training may consider using an oral or nasopharyngeal airway. Cricoid pressure is not recommended.
4. *Breathing—"B":* Rescue breathing should be performed by mouth-to-mouth, mouth-to-barrier, or mouth-to-mask ventilation. Breathing should be initiated after chest compressions. To provide mouth-to-mouth rescue breaths, the victim's airway should be opened using the head tilt–chin lift maneuver. The victim's nose should be pinched to create an airtight mouth-to-mouth seal. One breath should be given over 1 second. The rescuer should take and provide a "regular" (not deep) breath to avoid becoming dizzy or lightheaded, and to prevent overinflation of the victim's lungs. The most common cause of ventilation difficulty is an improperly opened airway. If the victim's chest does not rise with the first rescue breath, the head should be repositioned by repeating the head tilt–chin lift and then giving the second rescue breath.
5. *Defibrillation:* When available, an automated external defibrillator (AED) should be applied to evaluate for presence of fibrillation that might respond to electrical defibrillation. This is rarely an option in the wilderness environment. An acceptable, although infrequently successful, method of terminating a malignant tachyarrhythmia is the *precordial thump*. To perform a precordial thump, if the environment is such that the patient's chest can be cleared of clothing to facilitate accurate assessment of anatomy, this should be done. Next, one or two firm blows should be delivered to the middle to lower third of the sternum with a closed fist from a height of 20 to 25 cm (8 to 10 inches) above the chest. A central artery should then be palpated for a pulse. If unsuccessful after one attempt, the effort should be abandoned and further BLS initiated.

Administering compressions and rescue breaths in a 30:2 ratio is recommended during resuscitation. Rescuers are advised to switch roles frequently to minimize fatigue inherent in delivering effective chest compressions. A brief resuscitation pause to assess for presence of spontaneous pulses should be performed every 2 minutes.

The success of cardiac arrest resuscitation typically requires access to ACLS equipment and trained personnel. This may not be a reality when cardiac arrest occurs in a wilderness environment. Cardiac arrest patients who do not respond to resuscitation efforts during the first 15 to 30 minutes of BLS are unlikely to recover any significant degree of brain function, even if spontaneous cardiopulmonary function is restored. The decision of when to terminate resuscitation in a wilderness setting is challenging, and recommendations for this task have been developed.[28] In general, it is reasonable to terminate resuscitation efforts after 30 minutes if a spontaneous pulse has not been restored. An extension beyond this time limit should be considered for the hypothermic patient, because hypothermia may extend the window of potential central nervous system survival. Current resuscitation guidelines provide recommendations for specific wilderness situations (Table 50-2).[58]

TABLE 50-2 Considerations for Resuscitation of Cardiac Arrest Victims in Specific Wilderness Situations

Wilderness Situation	Special Considerations
Trauma	1. A jaw thrust, not a head tilt–chin lift maneuver, should be used to establish a patent airway. 2. Ventilation should be performed using a barrier device whenever possible, especially if the victim's face is bloody. 3. Visible hemorrhage should be controlled with direct pressure and compressions to minimize blood loss.
Hypothermia	1. Wet garments should be removed if they can be replaced with dry insulating cover, ideally during early stages of the resuscitation efforts. 2. Consider prolonged resuscitation effort (>30 minutes) if it can serve as a bridge to a medical facility with active rewarming capabilities.
Avalanche burial	1. Resuscitation should not be conducted in a location with continued snow instability that could put care providers at risk. 2. Decision to initiate resuscitation efforts must be tailored to victim burial time. All individuals without clear evidence of fatal trauma (e.g., decapitation) should receive resuscitation for burial times of less than 30 minutes.
Drowning	1. Rescuers must take great care to avoid personal risk in the retrieval of a submerged or potentially drowned victim. 2. Standard resuscitation sequence applies. Rescuers should be aware that victims with respiratory arrest but intact circulation (spontaneous pulse) may respond promptly to several mechanical ventilations.
Lightning strike	1. Rescuers must take great care during a storm to avoid personal risk in the care of a victim struck by lightning. 2. Concomitant trauma to the head and spinal column is a common side effect of lightning strike and should be assumed in all victims.

There are certain situations in which resuscitation may be deferred in the wilderness environment. These include (1) any situation in which resuscitation efforts would put rescuers at risk, (2) a victim with a core body temperature of less than 15° C (60° F), (3) a victim with a frozen chest wall, (4) a victim who has been submerged in cold water for more than 60 minutes, (5) a victim with an obvious lethal injury such as decapitation, and (6) a situation in which resuscitation would significantly delay evacuation of a hypothermic patient to a situation of controlled rewarming.[28]

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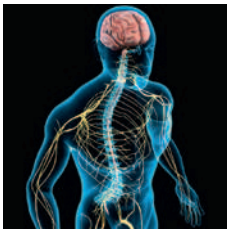
Complete references used in this text are available online at expertconsult.inkling.com.



CHAPTER 51

Wilderness Neurology

MINJEE KIM AND W. TAYLOR KIMBERLY

Common neurologic problems encountered in wilderness settings and management decision making can be divided into three main groups:

- *Acute neurologic conditions secondary to environmental extremes.* This chapter briefly reviews the distinct neurologic emergencies of high-altitude cerebral edema in mountaineers and nitrogen narcosis and decompression sickness.
- *Management of neurologic emergencies in the wilderness.* Numerous benign neurologic conditions occur in wilderness settings; true neurologic emergencies are rare. This chapter focuses on how to use the clues of history and physical examination to differentiate between benign and serious conditions. When a diagnosis is not obvious, making management decisions can be difficult. Decisions to evacuate can affect not only the patient but the entire group. Careful risk evaluation is essential.
- *Management of chronic neurologic conditions in the wilderness setting.* Examples include handling recurrent seizures in known cases of epilepsy or advising whether or not a person should take part in a trek in a remote area when the individual has been previously diagnosed with stroke, Parkinson's disease, or multiple sclerosis.

HISTORY AND EXAMINATION

Neurologic evaluation must focus on history and examination to produce a succinct and practical formulation. A careful history from the patient and witnesses to characterize the onset, duration, and range of symptoms is invaluable. Nervous system examination need not be lengthy or complex. Neurologic diagnosis is guided by the desire to pinpoint the site of the suspected lesion. Using history and examination findings, caregivers may use the following questions to make critical differentiations:

- Is the problem localized to the cerebral hemispheres or brainstem?
- Are the symptoms consistent with a lesion in the spinal cord?
- Is the problem localized to the peripheral nervous system?

A short neurologic examination takes less than 5 minutes without special equipment (Box 51-1). A reflex hammer can be improvised easily, and lack of an ophthalmoscope rarely makes a great difference. History and examination seek to localize where within the nervous system the lesion exists, because this guides the differential diagnosis. Pattern recognition—and thus some experience—is vital. Without advanced imaging, one may be able to reach only tentative conclusions in the field yet still mount appropriate clinical responses.

ACUTE NEUROLOGIC CONDITIONS SECONDARY TO ENVIRONMENTAL EXTREMES

HIGH ALTITUDE: ACUTE MOUNTAIN SICKNESS AND HIGH-ALTITUDE CEREBRAL EDEMA

Acute mountain sickness (AMS) and high-altitude cerebral edema (HACE) are considered a spectrum of the same altitude illness. AMS typically occurs within 12 to 24 hours after reaching an altitude at or above 2500 m (8202 feet), and is defined by a headache combined with anorexia, nausea, vomiting, fatigue, weakness, lightheadedness, dizziness, or difficulty falling asleep. AMS may progress to HACE, which is accompanied by ataxia and/or alteration in mental status. Risk for HACE increases at or above 5000 m (16,404 feet) and is associated with rapid ascent and/or poor acclimatization. In rare cases, HACE may also occur in well-acclimatized climbers at extreme altitudes, usually above 6500 m (21,325 feet).

If not treated, HACE can progress to coma and death. Slow ascent to accomplish prevention is very important. Acetazolamide or dexamethasone can also be used for prophylaxis. Once symptoms of high-altitude disease develop, immediate descent and administration of oxygen are critical. Detailed information on diagnosis and management of AMS and HACE is provided in Chapter 2.

DIVE MEDICINE: DECOMPRESSION SICKNESS

Decompression sickness (DCS) is caused by air bubbles in blood or tissue after rapid reduction in environmental pressure. When the sum of dissolved gas tensions (oxygen, carbon dioxide, nitrogen, and/or helium) and water vapor exceeds the local absolute pressure, air bubbles form in extravascular and intravascular spaces, leading to various degrees of mechanical, embolic, and biochemical effects. DCS has been classified as arterial gas embolism (AGE) and type 1 and type 2 DCS. Neurologic manifestations can occur in AGE and type 2 DCS. AGE can present as change in mental status and focal neurologic deficits mimicking ischemic stroke. Preexisting intracardiac right-to-left shunt (e.g., patent foramen ovale, arterial septal defect, ventricular septal defect) increases the risk of AGE.

Although AGE is not related to depth and time underwater, DCS is uncommon at depths of less than 1 additional atmosphere (33 feet of water). Risk of AGE can be reduced by avoiding breath holding and ascending slowly. Three-quarters of patients with DCS develop symptoms within 1 hour and 90% within 12 hours

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BOX 51-1 Five-Part Abbreviated Neurologic Examination

1. Look at the patient.
   - Appearance, injuries, level of consciousness, and cognition
   - Speech
   - Gait
   - Arm swinging
2. Examine the patient's head.
   - Fundi, if an ophthalmoscope is available
   - Pupils
   - Eye movements
   - Facial weakness and movements
   - Tongue
3. Examine the patient's upper limbs.
   - Posture of outstretched arms
   - Power and tone
   - Coordination
   - Deep tendon reflexes
4. Examine the patient's lower limbs.
   - Power
   - Tone
   - Deep tendon reflexes
   - Plantar responses
5. Inquire about the patient's sensation.
   - Usually, one can simply ask the patient.

of surfacing. Approximately 60% of divers with DCS develop neurologic signs, usually affecting the thoracic or lumbar spinal cord. Paresthesias and weakness may progress to paraplegia and bladder or bowel dysfunction. Cerebral dysfunction may also occur and includes ataxia, dizziness, vertigo, visual disturbances, and memory loss. Symptoms are thought to result from the formation of bubbles in the venous plexi resulting in venous obstruction.

Treatment of DCS includes administration of 100% oxygen and positioning the patient in the left lateral decubitus and mild Trendelenburg position, encouraging air to migrate toward the right ventricular cavity. Hyperbaric oxygen therapy should be initiated as soon as possible. See Chapters 71 and 72 for details regarding dive medicine.

BASIC MANAGEMENT OF NEUROLOGIC EMERGENCIES

Neurologic complaints are common, but only a minority represents true emergencies. Therefore, the main focus of neurologic evaluation in the wilderness is to distinguish between emergencies that require immediate evacuation and those that can be monitored and treated symptomatically. Although it can be challenging to reach a definitive diagnosis with limited resources, history and physical examination typically provide enough information to guide initial treatment and evacuation decisions.

HEADACHE

Headache is one of the most common neurologic symptoms and a frequent problem in wilderness settings. Headaches may be trivial, represent a persistent problem, or be indicative of serious disease.

The brain itself lacks pain receptors. Pain is caused by activation of pain receptors in the pain-sensitive structures in the head and neck, including the cranium, nerves, arteries and veins, neck and facial muscles, meninges, sinuses, eyes, ears, subcutaneous tissues, and mucous membranes. Nociceptors may be stimulated by mechanical traction or irritation of these structures, sending information via nerve fibers to the thalamus and cortex, signaling the presence of pain. The vast majority of headaches are "benign" (often self-limited and not caused by a serious disorder requiring advanced testing or referral to a specialist).

GUIDELINES FOR DIAGNOSIS OF HEADACHE: DIFFERENTIATING BENIGN FROM OMINOUS

With any headache, first establish whether there is fever, neck stiffness, change in level of arousal, or any "hard" neurologic signs. Presence of any of these factors suggests an ominous cause. Hard neurologic signs include double vision (diplopia), papilledema, visual field defects, ophthalmoparesis (e.g., new restriction in eye movement or misalignment of eyes), unilateral weakness (hemiparesis), and ataxia. If any of these is present, serious pathology must be ruled out, and advanced medical and neurologic care should be sought.

Unless the practitioner has experience with funduscopy (ophthalmoscopy), papilledema may be difficult to visualize with certainty without pharmacologically dilating the pupils. However, blind-spot enlargement can be easily tested on examination without special tools. Sit the patient 90 to 120 cm (3 to 4 feet) at the same level (height) and directly in front of you. Ask the patient to cover one eye with his palm, and close your eye on the same side (e.g., patient covers left eye and examiner closes right eye). Ask the patient to direct his gaze and stay focused on your open eye. Hold any small, bright-colored item or the tip of a pen midway between the patient and examiner. Check if both you and the patient can identify the red pin in the peripheral vision while staying focused on each other's open eye. Slowly move the pin horizontally toward the periphery (away from the noses), and ask the patient to tell you when it disappears and then reappears. The small area where the pin "disappears" in one's peripheral vision represents the physiologic "blind spot," which corresponds to the optic nerve head. In the setting of elevated intracranial pressure (ICP) that leads to papilledema, a larger blind spot than normal can develop. Compare the size of blind spot between you and the patient. If the object disappears in the patient's peripheral vision while you can still see it, the patient's blind spot is larger than yours. Repeat the test on the opposite side. In the correct clinical context, this can serve as a useful screening tool. As a relatively quick test that can be followed serially for changes, blind-spot examination serves as a rough estimate of normal versus increased ICP.

Other neurologic findings that should prompt immediate evacuation include unilateral weakness of an arm or leg or depressed mental status. The exception is the patient who has a known prior history of complicated migraines, with the neurologic symptoms characteristic of prior episodes. See later for more details about ominous causes of headache.

MIGRAINE HEADACHES

Development of sudden headache that involves "positive" visual phenomena, such as flashing lights, nausea, and vomiting, and that is consistent with prior migraine headaches is likely to be another migraine. Of the general population, 12% suffer from migraine and 10% have chronic migraine. Altered sleep-wake schedule, missing or delaying a meal, near-daily use of analgesics, dehydration, and perimenstrual hormonal changes are some of the common triggers of migraine.

Migraine treatment can be divided into two categories: acute abortive treatment for individual attacks and preventive treatment. It is critical for the patient and practitioner to understand the differences between medications in these two categories. Daily medication is key to successful preventive treatment, but excessive use of abortive medication can lead to medication overuse headache.

Acute Treatment of Migraine

A general rule of thumb is that the earlier the acute migraine is treated, the more effective the treatment will be. Therefore, the goal should be to treat patients as early as possible. Combination therapy with an antiemetic and nonopiate analgesic, together with hydration, intravenously (IV) if severe nausea, tends to be effective. In case of severe nausea, a rectal antiemetic can be administered, bypassing the oral (PO) route. Typical antiemetics include metoclopramide (5 to 10 mg PO every 6 hours), ondansetron (4 to 8 mg PO every 8 hours), prochlorperazine (10 mg

PO every 6 hours), and prochlorperazine suppository (10 to 25 mg, with a daily maximum of 50 mg). The choice of analgesic is based on severity, availability, previous response history, and relative contraindications (e.g., avoid nonsteroidal antiinflammatory drugs [NSAIDs] in patients with esophageal reflux, stomach ulcers, or renal insufficiency). For mild to moderate severity, acetaminophen (650 mg PO every 6 hours as needed, maximum daily dose of 3000 mg) or NSAIDs (e.g., ibuprofen, 400 to 800 mg PO every 6 hours as needed, maximum daily dose of 2400 mg; diclofenac, 50 mg PO every 8 hours as needed, maximum daily dose of 150 mg; naproxen sodium, 500 to 550 mg every 12 hours as needed, maximum daily dose of 1375 mg) can be given.

For moderate to severe migraine attacks and patients with NSAID failure, triptans can be first-line acute treatment. Triptans are effective (yet expensive) and should be avoided in patients with vascular disease (e.g., coronary artery disease, stroke, uncontrolled hypertension) or in a migraine associated with neurologic deficits, such as unilateral weakness. There are seven available triptans in the United States, in intranasal, subcutaneous, oral (solid tablets), and rapidly disintegrating oral (wafer) forms. Whereas they all share similar mechanism of action, individuals respond differently for reasons that are not yet well explained. There is no single "best" triptan; the aim is to find the most effective, well-tolerated, and affordable formulation for each individual. Some dosing examples are: sumatriptan 6 mg subcutaneously (SC) (daily maximum of 12 mg); sumatriptan, 20 mg intranasally (IN) (daily maximum of 40 mg); zolmitriptan, 5 mg IN (daily maximum of 10 mg); zolmitriptan, 2.5-mg wafer or tablet PO (daily maximum of 10 mg); rizatriptan, 10-mg wafer or tablet PO (daily maximum of 20 mg); eletriptan, 40 mg PO (daily maximum of 80 mg); frovatriptan, 2.5 mg (daily maximum of five mg); almotriptan, 12.5 mg (daily maximum of 25 mg); and naratriptan, 2.5 mg (daily maximum of 5 mg). The triptan should be administered as soon as headache develops. Only one dose is recommended for acute treatment, followed by a second dose if headache recurs after initial relief, 2 or more hours after the first dose. Common side effects of triptans include flushing, hot or warm sensation, paresthesia, and chest or jaw tightness or discomfort.

For all acute migraine attacks, creating a quiet and dark environment where one can also minimize head movement can be helpful. Most migraines subside within 4 to 72 hours. Once an acute attack resolves, patients should be educated on strategies to prevent a recurrent attack, including lifestyle modification (e.g., maintain adequate sleep and minimize alterations of the sleep-wake cycle; keep regular mealtimes; avoid dehydration) and trigger management (e.g., avoid trigger foods, such as red wine and chocolate; avoid exposure to loud noises and prolonged sunlight).

Preventive Treatment of Migraine

In wilderness settings, it is not practical to initiate preventive treatment of migraines. However, some travelers will be taking antimigraine preventive medications, so it is important for the wilderness practitioner to be aware of some of the basics of migraine prevention. Generally, migraine prevention is not initiated unless recurring migraine attacks significantly interfere with quality of life and occur at a frequency of four or more attacks per month. Preventive medications frequently reduce but do not necessarily eliminate migraine attacks. Classes of medications used for migraine prevention include β-adrenergic blockers, antiepileptic drugs, antidepressants, calcium channel antagonists, serotonin antagonists, and botulism neurotoxins. A drug can be chosen based on efficacy, side effect profile, the patient's comorbidities, and preference.

Topiramate can be an effective medication for overweight patients because it often results in loss of appetite and weight. The dosing is to begin with 15 to 25 mg PO at bedtime, then increase 15 to 25 mg per week to reach a total dose of 50 to 100 mg per day. If necessary, the dose can be increased to up to 200 mg per day. Dose-related cognitive clouding is often a dose-limiting side effect. Tingling of hands and feet may occur, attributed to the drug's mild carbonic anhydrase inhibition. Patients with a history of kidney stones should avoid topiramate.

Tricyclic antidepressants (TCAs) may be chosen for underweight patients with depression and insomnia because these drugs offer additional mood stabilization, weight gain, and sleep-promoting effects. Among TCAs, tertiary amines, such as amitriptyline or doxepin (for either, begin with 10 mg PO at bedtime, then increase by 15 to 25 mg per week to reach a total dose of 10 to 200 mg to achieve symptom control), are more sedating than are secondary amines such as nortriptyline (begin at 10 mg PO at bedtime, then increase by 15 to 25 mg per week to reach a total dose of 10 to 150 mg). Dry mouth, urinary retention, and orthostatic hypotension may occur, and elderly patients may develop confusion. TCAs should be avoided in patients with cardiovascular disease because of their QT prolongation effect. The nonselective β-adrenergic blocker propranolol (begin at 20 mg PO twice daily, then increase 20 mg per week until desired headache control is reached, at a total daily dose of 40 to 240 mg; can be converted to a long-acting formulation) and the selective β_1-blocker metoprolol (begin with short-acting formulation at 25 mg PO twice daily, then increase 25 mg per week to achieve a final daily dose of 100 to 200 mg as tolerated; can be converted to a long-acting formulation) are effective preventive drugs. These can cause sexual dysfunction and decreased libido and may increase the risk of depression. β-Adrenergic blockers should be avoided in individuals with known heart block or low blood pressure. Similar considerations pertain to the use of calcium channel blockers, such as verapamil, which are sometimes used as migraine prophylaxis agents.

TENSION HEADACHES

Tension-type headache is the most common form of headache and is frequently described as nonthrobbing, bilateral, dull, and pressure-like pain associated with a normal neurologic examination. The pain is general mild to moderate in severity. If the pain is localized, throbbing, or severe, other types of headaches should be considered. Tension headaches can occur infrequently or in high frequency (e.g., every other day), in which case they are classified as *chronic tension headaches*. For acute treatment of tension headache, NSAIDs such as ibuprofen (200 or 400 mg), naproxen sodium (220 or 550 mg), or aspirin (650 to 1000 mg) are reasonable choices. Acetaminophen (1000 mg) can be used in persons intolerant of NSAIDs, but may not be as effective as NSAIDs.

LOCALIZED AND CLUSTER HEADACHES

If a headache is focal, then sinusitis, glaucoma, dental pain, and cluster headache need to be considered. With glaucoma, the eye will typically be painful, and vision may be altered. Sinusitis can cause severe pain that is usually well localized within and around the affected sinus. Cluster headache causes pain of exceptional severity, often at night, and is typically perceived around and behind the eye. It is unilateral and associated with autonomic signs (flushing, sweating, or changes in pupil size on the affected side). In contrast to migraine headache, patients experiencing cluster headache are restless and often agitated as a result of the pain. Each episode will generally be short-lived, as brief as 15 minutes in duration. Cluster headache, no matter how severe, tends to resolve after 4 to 6 hours. The mainstay of treatment is 100% oxygen, preferably at 12 L/min through a nonrebreather mask for approximately 15 minutes. Symptoms may subside within the first 5 minutes. Triptans, such as sumatriptan (6 mg SC) or zolmitriptan (6 mg IN), are also effective for acute cluster headaches, although these should be avoided in patients with a history of ischemic heart disease or stroke.

HEADACHE WITH OTHER NEUROLOGIC SIGNS

Distinguishing between benign and serious headaches is necessary for triage and management. A headache associated with fever, neck stiffness, or any focal neurologic signs raises concerns for an ominous etiology. Neurologic findings such as altered mental status, somnolence, weakness, numbness, paresthesias, vertigo, speech difficulty, language disturbances, or ataxia warrant

special attention. Differential diagnosis is discussed next. Migraine, one of the most common benign headache syndromes, typically produces malaise, nausea, and visual disturbances. These symptoms typically recur with similar pattern of presentation. Dehydration and altered sleep cycle can exacerbate otherwise well-controlled migraine headaches.

Sudden Headache with Acute Neurologic Signs

Ischemic stroke or transient ischemic attack presents with acute neurologic signs and occasionally may be associated with a headache. If acute neurologic signs rapidly progress over minutes to hours, or if arousal is diminished, intraparenchymal hemorrhage, subarachnoid hemorrhage, basilar artery thrombosis, brainstem herniation, and acute hydrocephalus are in the differential diagnosis. Serial assessment of pupillary light response in an obtunded patient can provide clues to acute intracranial processes. A unilateral dilated and unreactive pupil may signal uncal herniation of the ipsilateral temporal lobe and compression of the affected oculomotor nerve resulting from cerebral edema, hemorrhage, or other space-occupying process.

Benign conditions that present with acute neurologic signs include complex migraine. Patients with complex migraine usually report a long-standing history of headache with stereotypic symptoms (e.g., hemiplegic migraine). These symptoms spontaneously resolve over several hours to a few days. Unless migraine-associated symptoms are identical to those of prior migraine attacks, it is prudent to manage the patient assuming an alternative cause.

Headache Caused by Increased Intracranial Pressure

Intracranial mass lesions displace and stretch the meninges and basal blood vessels. Pain is provoked when these structures are shifted, either by a mass or by changes in cerebrospinal fluid pressure, such as may be caused by coughing. Cerebral edema associated with brain tumors may cause further shifts of intracranial structures. Pressure headaches typically worsen when a patient is lying down or moving; however, this feature is also seen with migraine, AMS, and HACE.

Vomiting often accompanies pressure headaches. Papilledema or abnormally large blind spots may be detected on examination. Pressure headaches arise early during the pathologic process, and over the course of days to weeks when they are caused by posterior fossa masses or hydrocephalus. They typically develop over months to years when they are caused by supratentorial tumors. *Venous sinus thrombosis* (VST) can cause pressure headache with or without associated visual disturbances. Risk factors for VST include estrogen-based oral contraceptive methods, pregnancy, malignancy, hypobaric exposure, and dehydration.

Idiopathic intracranial hypertension (IIH), also called *pseudotumor cerebri,* can cause progressive pressure headache, transient or sustained vision loss, horizontal diplopia, and pulsatile tinnitus. Normal level of consciousness is preserved. It predominantly affects women of childbearing age who are overweight. Because diagnosis requires advanced neuroimaging and a lumbar puncture, this condition requires more advanced evaluation in a clinical setting.

With the exception of HACE, if a headache with increased ICP is suspected, the patient should be evacuated to obtain further evaluation with imaging studies (computed tomography [CT] and/or magnetic resonance imaging [MRI]) as soon as possible, especially if there are other progressive neurologic signs.

SUDDEN HEADACHE

If pain is focal (e.g., localized to the upper face and lasting minutes to hours), consider sinusitis, glaucoma, and cluster headache. A severe sudden headache may be associated with subarachnoid hemorrhage, migraine, bacterial meningitis, or cervical arterial dissection.

HEADACHE WITH SCALP TENDERNESS

Patches of exquisite tenderness overlying superficial scalp arteries may be caused by giant cell arteritis, also called temporal arteritis, most commonly in patients over age 50 years. Caused by chronic inflammation of large and medium-sized vessels, giant cell arteritis can produce transient or sustained visual disturbances, jaw claudication, and otherwise unexplained fever.

HEADACHE AFTER HEAD INJURY

The vast majority of headaches that occur after mild head trauma, even those that last for several months, are not caused by serious intracranial pathology. Consider subdural and epidural hematoma in the acute aftermath of serious head injury, especially if the patient has lost consciousness or is suffering repeated vomiting (see Chapter 18). Decreasing level of arousal after head injury is a warning sign that may indicate brain compression related to space-occupying effects of subdural or epidural hematoma.

SINGLE EPISODE OF SUDDEN SEVERE HEADACHE

Although the most common cause of a single, sudden, severe headache is migraine, cluster, or tension-type headache, it may also result from a serious event such as subarachnoid hemorrhage, reversible cerebral vasoconstriction syndrome, pituitary apoplexy, cervical arterial dissection, or bacterial meningitis. The term *thunderclap headache* is used to describe such an explosive and unexpected headache.

Subarachnoid hemorrhage (SAH) caused by aneurysmal rupture produces a sudden, severe headache and may also cause impaired consciousness, focal neurologic signs, neck stiffness, and nausea and vomiting. Suspicion is further raised by a history of smoking, exertion, or Valsalva maneuver immediately before onset of headache, sentinel headache (i.e., an episode of similar yet milder headache occurring days to weeks earlier, lasting for hours to days), or family history of aneurysm. If SAH is suspected, particularly if level of consciousness is impaired, immediate evacuation and evaluation with advanced imaging is necessary.

Reversible cerebral vasoconstriction syndrome (RCVS) causes a thunderclap headache with or without other neurologic signs. Seizures are common. Caused by diffuse or segmental vasospasm of intracranial arteries, RCVS is associated with the postpartum period and exposure to pharmacologic agents such as cocaine, amphetamine, 3,4-methylenedioxymethamphetamine ("Ecstasy"), selective serotonin reuptake inhibitors (SSRIs), ergotamine, triptans, and pseudoephedrine. It is often self-limited, and treatment includes cessation of provocative agents and supportive care.

A thunderclap headache with acute ophthalmoplegia and a visual field defect in both sides of peripheral vision suggests a rare condition of *pituitary apoplexy,* a sudden hemorrhage into the pituitary gland, often in the setting of pituitary adenoma. Symptoms can range from mild to severe. Immediate evacuation for brain MRI is recommended. Adrenal crisis with shock is the most serious complication, which may result from acute corticotropin deficiency. High-dose corticosteroid replacement therapy (e.g., prednisone, 60 mg PO daily) should be initiated without delay. Most patients can be managed conservatively, but careful attention to electrolytes, volume status, and hemodynamics is crucial, as are serial neuro-ophthalmologic examinations. Persistent or deteriorating ophthalmoplegia or visual symptoms are indications for surgical decompression of the pituitary.

High fever, neck stiffness, vomiting, and rash suggest acute *bacterial meningitis,* which requires immediate treatment with broad antimicrobial coverage (e.g., vancomycin, 15 mg/kg every 8 hours; ceftriaxone, 2 g every 12 hours; and ampicillin, 2 g every 4 hours). Glucocorticoids (e.g., dexamethasone, 4 mg every 6 hours) should be initiated if pneumococcal meningitis is suspected, but this may be harmful if the central nervous system (CNS) infection is caused by other bacterial pathogens. Headaches associated with bacterial meningitis tend to be of gradual onset, although they can also be sudden and severe. For afebrile persons, careful reassessment over 12 hours can usually determine whether a sudden headache was a migraine or part of SAH. Bacterial meningitis is often obvious at the onset.

HEADACHE OF SUBACUTE ONSET

The onset and progression of a headache over days to weeks, especially if accompanied by other neurologic signs, should raise suspicion for an intracranial mass or other intracranial process, Chronic cryptococcal meningitis or aseptic meningitis can cause progressively worsening headache over the course of several days. Headache with neck stiffness can be a sign of acute bacterial meningitis, especially in the setting of fever, and is discussed later in detail.

TRANSIENT ISCHEMIC ATTACK AND STROKE

Stroke (cerebrovascular accident) is an acute focal neurologic deficit that results from a disruption of normal cerebrovascular flow. It is rare in fit, young, normotensive persons. However, as increasing numbers of older people with vascular pathology risk factors (e.g., age >60 years, hypertension) take part in wilderness activities, stroke may become more common in participants. Timely diagnosis of acute stroke must occur if intravenous (IV) thrombolytic and intraarterial thrombectomy are to be employed as therapies. These techniques are effective at reversing neurologic deficits but must be initiated within the first few hours after stroke onset. With prompt diagnosis and evacuation of victims of acute stroke, significant brain tissue might be saved, resulting in improved outcomes and reduction in long-term disability. Even if a patient cannot be evacuated within the time to qualify for acute reperfusion therapy, follow-up management necessitates urgent evacuation for hospital-level care.

DIAGNOSIS OF TRANSIENT ISCHEMIC ATTACK

Transient ischemic attack (TIA) implies sudden loss of focal CNS function, usually of very rapid onset (seconds to minutes) and without preceding symptoms. By definition, a TIA lasts less than 24 hours, typically minutes to hours (Table 51-1). Transient neurologic spells of abrupt onset can be caused by TIA, seizure, or migraine. Important diagnostic clues are considerations of risk factors, age and gender, past medical history, and characteristics of individual episodes. TIA symptoms are indicative of transient loss of CNS function (e.g., weakness, visual loss, hearing loss, aphasia). In contrast, seizures and migraine typically cause "positive" symptoms (e.g., convulsive movement, seeing flashing lights, tinnitus), although there are rare exceptions (e.g., atonic or aphasic seizures). Postictal weakness after a convulsive seizure (i.e., Todd's paralysis) can mimic a TIA. *Amaurosis fugax* is defined as sudden, painless loss of vision in one eye that lasts minutes to hours; it can be caused by thromboembolism from ipsilateral carotid artery atherosclerosis.

An episode of isolated loss of consciousness is rarely, if ever, caused by a TIA. *Transient global amnesia* is a sudden loss of memory and orientation lasting minutes to hours without other signs or symptoms. It is usually not a prelude to a major stroke.

MANAGING TRANSIENT ISCHEMIC ATTACK AND STROKE

Careful neurologic, vascular, and cardiac examinations are essential. After a resolved TIA, there are no abnormal neurologic signs.

TABLE 51-1 Features of Transient Ischemic Attacks

Anterior (Carotid) Circulation	Posterior (Vertebrobasilar) Circulation
Amaurosis fugax	Diplopia, vertigo, and vomiting
Aphasia	Dysarthria
Hemiparesis	Ataxia
Hemisensory loss	Hemisensory loss
Hemianopic visual loss	Hemianopic or bilateral visual loss
	Tetraparesis

After a stroke, the neurologic examination is abnormal, with features of hemiplegia, gaze preference, or aphasia. There may be an overt or subtle residual deficit (e.g., hemiparesis or body region neglect).

Stroke

When acute stroke is suspected, every effort should be made to move the patient to an advanced medical facility within 6 hours of when the patient was last witnessed to be normal.

Diagnosing the underlying cause of a stroke requires neuroimaging to distinguish between hemorrhage, infarction, or less common causes of stroke (e.g., arteriovenous malformation, vasculitis, endocarditis). In persons less than 35 years old, cervical arterial dissection is a common cause of stroke. It may be spontaneous or may follow head injury or jarring of the neck. SAH is suggested by the presence of a thunderclap headache, impaired consciousness, and neck stiffness.

If timely evacuation isn't possible, aspirin treatment may be considered. Ischemic stroke represents 85% of all stroke types. Although antiplatelet treatment may worsen a cerebral hemorrhage, administration of aspirin (75 to 325 mg PO or 300- to 600-mg suppository) should be considered. The decision whether to administer aspirin empirically may be influenced by the presence of comorbid disease such as preexisting coronary artery disease, diabetes, or hyperlipidemia. Pay special attention to the patient's airway and swallowing. Consider administering supplemental oxygen. Pulmonary aspiration of gastric contents, particularly when unilateral facial weakness is visible, is a possible complication from loss of motor function within the posterior oropharynx.

Posterior Circulation Ischemia

Posterior circulation ischemia (e.g., from basilar artery thrombosis) typically causes transient loss of consciousness in association with other neurologic deficits, such as ataxia, nystagmus, unilateral weakness, or sensory loss.

Transient Ischemic Attack

For suspected thromboembolic TIA, consider prompt evacuation and further investigation. Note that a hemorrhagic stroke will not spontaneously improve. Therefore, administering aspirin (75 to 325 mg PO) is a prudent strategy to help prevent further events while evacuating the patient.

TRANSIENT ISCHEMIC ATTACK AND STROKE IN SPECIAL CIRCUMSTANCES

Sudden neurologic events may occur after underwater dives and at high altitude in otherwise healthy individuals. There are numerous case reports of TIA and stroke-like events occurring at altitudes of more than 5000 m (16,404 feet), some in the setting of HACE and others presumably brought on by dehydration and polycythemia caused by chronic hypoxia.

Transient TIA-like episodes of aphasia, visual disturbances, and unilateral weakness are also well described, mostly at altitudes above 6500 m (21,325 feet).

1. If someone has a stroke at high altitude, administer oxygen by face mask, if available. Administer dexamethasone, 4 mg PO four times daily, and seek rapid descent. Give aspirin, 325 mg PO once daily, unless there are contraindications, such as active bleeding.
2. With a TIA-like event at altitude, hydrate the patient. Consider a short course of dexamethasone, 4 mg PO four times daily, and administer aspirin, 325 mg PO daily, as for stroke at altitude.

TRANSIENT EPISODES OF IMPAIRED CONSCIOUSNESS

A transient episode of loss of consciousness or syncope may occur, although neurological causes are uncommon. Differential diagnosis includes vasovagal syncope, seizure, cardiac arrhythmia, and rarer causes of impaired consciousness.

EPILEPSY

Epileptic seizure is a transient occurrence of signs and symptoms caused by abnormal excessive or synchronous neuronal activity in the brain. *Epilepsy* is a disorder characterized by recurrent seizures. Generalized tonic-clonic convulsion (i.e., grand mal seizure) is the most commonly recognized seizure. More than 2% of the population in developed countries is known to have one or more seizures during their lifetime; in 0.5%, epilepsy is an active problem. About 200,000 people in the United Kingdom and 1.5 million people in the United States take antiepileptic drugs.

CLASSIFICATION

Seizures are frequently described as generalized or focal, with or without impaired consciousness. A newly revised classification of epilepsy focuses on the specific underlying etiologies, such as genetic, structural, metabolic, or immune mediated.

Generalized seizures, during which the patient loses consciousness, include tonic-clonic, myoclonic, and absence seizures. Electrical abnormalities rapidly engage both hemispheres of the brain, leading to bilateral motor involvement (e.g., rhythmic convulsion of four limbs) accompanied by loss of consciousness. With a primary generalized seizure, electroencephalography (EEG) reveals bilateral abnormal electrical spike-and-wave activity.

A *focal seizure* occurs when the electrical abnormality originates within networks limited to one hemisphere. Examples include aura (e.g., sensation of flickering lights, unpleasant odor, abdominal discomfort) and rhythmic movement of one limb with preserved consciousness. A focal seizure can spread to the other hemisphere and generalize. Focal seizures accompany preserved consciousness (partial seizures) or impaired consciousness (complex partial seizures).

DIAGNOSING A SEIZURE

With a generalized tonic-clonic seizure, the typical stages are first the warning (i.e., a perception that something is about to happen but without a specific aura), which leads to the tonic (i.e., stiff or rigid) phase, during which the patient falls unconscious to the ground, sometimes first uttering a cry. Injuries may occur, and the tongue may be severely bitten. Incontinence of urine and feces may follow. The clonic (convulsive) phase begins with bilateral rhythmic limb jerking, lasting from a few seconds to several minutes. The patient may appear cyanotic during the attack, and "foaming at the mouth" is often reported. Postictal drowsiness and confusion follow, typically lasting 10 to 20 minutes, but in some cases as long as hours.

Symptoms of a focal seizure depend on where in the cortex the seizure originates. The patient may have an aura before a clinically evident event begins. An *aura* is a specific and patterned sensation caused by a focal cortical electrical discharge, without an objective clinical finding.

Patients experiencing a simple jacksonian focal motor seizure originating within the primary motor cortex may sense a vague warning (as with a primary generalized seizure), followed by an aura (e.g., unusual sensations in one limb). Onset of rhythmic jerking follows, typically in one hand or foot. Progression of this jerking ("march" of the seizure) may spread throughout the limb, to the ipsilateral other extremity and possibly to the ipsilateral face. Consciousness is not lost during a simple focal seizure. Instead of postictal drowsiness or coma, weakness of the limbs involved with the seizure may follow. This is known as Todd's paralysis and may last for several hours. A stroke may be suspected if early stages of the seizure were not witnessed or if these are unclear.

A complex partial seizure of the temporal lobe may cause an aura of familiarity with surroundings *(déjà vu)*, followed by a period during which the patient is generally unaware or disoriented. As the seizure develops, the patient has a distant expression (i.e., appears to be in a trance-like state) and does not respond to stimuli. Facial or lip twitching may occur. Recovery is typically rapid, with the patient regaining full awareness in seconds or minutes (rarely >10 minutes). The usual differential diagnosis of a temporal lobe seizure is between syncope and a nonepileptic episode of altered attention (e.g., distraction).

EMERGENCY MANAGEMENT OF SEIZURES

Field medical management of acute seizures consists of observation and protecting the patient from immediate harm. Most seizures resolve spontaneously within a few minutes. Do not place objects in the patient's mouth or attempt excessive restraint. Remain with the patient, summon help, and administer oxygen if available. Deep cyanosis may occur during a grand mal seizure, but the spontaneous return of normal breathing and oxygenation will remedy the problem.

Status Epilepticus

Status epilepticus involves occurrence of a continuous seizure more than 5 minutes in length, or recurrent seizures without fully recovering consciousness between episodes. More than 50% of cases of status epilepticus occur without a previous history of epilepsy, and it has a mortality rate of 10% to 15%.

Approximately 25% of patients with apparent refractory status epilepticus have nonepileptic "spells" of psychogenic origin. If the patient exhibits forced eye closure or side-to-side head movement, this raises suspicion for nonepileptic spells.

Status epilepticus may be nonconvulsive, creating a prolonged unresponsive state. If a postictal period after convulsive seizures lasts more than 30 minutes without sign of recovery, nonconvulsive status epilepticus may be suspected. *Focal status* describes continuous jerking of a limb or other focal seizure activity. *Epilepsia partialis continua* is a continuous seizure activity in one discrete part of the body (e.g., finger, one side of face) without a loss of consciousness. This is typically the result of a cortical neoplasm or, in the elderly patient, a cortical infarction. Focal motor status epilepticus is less serious than generalized status epilepticus, although both require prompt evacuation.

Box 51-2 describes a protocol for the treatment of status epilepticus that has been adapted for wilderness use. For the emergency kit, one method is to carry injectable and rectal diazepam,

BOX 51-2 Status Epilepticus Management

- Preserve and maintain the airway (e.g., position the patient). Consider definitive airway management with endotracheal tube as indicated.
- Treat convulsions rapidly.
- Check for head and other injuries. Maintain cervical spine precautions as indicated.
- Protect and stay with the patient.
- Establish IV access, if possible.

Drugs

- Give diazepam (5-10 mg IV at 5 mg/min or less every 5-10 min to a maximum of 30 mg).
- Use rectal diazepam (10-20 mg) or buccal midazolam (10 mg repeated once after 10 min if IV access is impossible).
- Administer oxygen, and monitor the pulse and blood pressure, if possible.
- Give IV glucose and thiamine.

If status epilepticus persists, consider other antiepileptic drugs:

- Lorazepam (4 mg at 2 mg/min IV every 10-15 min to a maximum of 8 mg)
- Phenytoin (IV bolus not exceeding 50 mg/min and adult dose of 10-20 mg/kg)
- Phenobarbital (IV bolus of 60 mg/min or less and adult dose of 10-20 mg/kg to a maximum of 30 mg/kg)
- Valproate (IV adult dose of 15-45 mg/kg at 6 mg/kg/min or less)
- Remember the potential adverse effects of antiepileptic drugs, such as hypotension, cardiac dysrhythmias, and cardiorespiratory arrest.

Data from Clarke C, Howard R, Shorvon S, et al: *Neurology: A Queen Square textbook*, Oxford, UK, 2009, Wiley Blackwell Publishing, p 235.
IV, Intravenous(ly).

injectable lorazepam, injectable phenytoin, buccal midazolam or sublingual lorazepam, a bag-valve-mask device, and several airways. One also needs to be prepared for the recurrent seizures, so oral antiepileptic drugs (e.g., phenytoin, carbamazepine) should be available for continuing therapy.

MANAGING A FIRST-TIME SEIZURE

An unprovoked, first generalized seizure is usually sufficient reason to evacuate a patient. Imaging is required to rule out a CNS mass or hemorrhage as an etiology. Seizure precautions are advised. Precautions focus on reducing risk to the patient or others if the person were to have another seizure (e.g., no swimming, climbing, or driving). Prudence argues against using antiepileptic drugs until a diagnosis of epilepsy is certain.

SYNCOPE AND RELATED PHENOMENA

Distinguishing between a seizure, syncope, and other types of transient loss of consciousness may be initiated by the history and eyewitness accounts.

SIMPLE SYNCOPE, CARDIAC SYNCOPE, AND POSTURAL HYPOTENSION

Fainting is the result of sudden reflex bradycardia with vasodilation of peripheral and splanchnic vasculature. Also known as *neurocardiogenic syncope,* it is a common response to prolonged standing, fear, venipuncture, or pain. Prodromal symptoms of palpitation, lightheadedness, or sense of doom immediately before loss of consciousness and urinary incontinence may occur. Syncope almost never occurs when an individual is in the recumbent posture, unless the vagal response is profound. Unconsciousness for less than 2 minutes and rapid recovery are typical. Brief myoclonic jerking may occur from transient lack of blood flow to the brain. This can briefly resemble an epileptic seizure and is called *convulsive syncope.* Convulsive syncope lacks a prolonged postictal period and does not warrant antiepileptic treatments or further workup for epilepsy.

Cardiac syncope caused by arrhythmia is typically abrupt in onset, with pallor and an irregular or nonpalpable pulse caused by ventricular tachycardia or asystole. It is rare in patients without underlying heart disease. Benign supraventricular tachycardias often present with palpitations and lightheadedness and rarely produce loss of consciousness.

Syncope can occur after micturition in men, particularly at night, and in either gender when venous return to the heart is obstructed by breath holding and severe coughing. Postural hypotension is also a cause of syncope. This typically occurs in elderly persons, in patients with autonomic neuropathy, and as a result of excessive doses of blood pressure medications. Postural hypotension symptoms also occur during acclimatization to altitude or severe dehydration. The immediate management of syncope or impending syncope is to lay the patient down, lift the legs, and assess the pulse.

OTHER CAUSES OF SUDDEN APPARENT LOSS OF CONSCIOUSNESS

Nonepileptic seizure, or *pseudoseizure,* is common during stressful situations and is without focal diagnostic features. Apparent loss of consciousness without prelude, followed by bizarre flailing limb movements that are not rhythmic, is typical. Cyanosis and frothing at the mouth are unusual. Epileptic patients may have both seizures and pseudoseizures, creating a diagnostic challenge.

Hypoglycemia

Sudden drop in blood glucose level can cause loss of consciousness, sometimes with a convulsion. Hypoglycemic attacks unrelated to diabetes mellitus are rare. Patients with diabetes frequently recognize their hypoglycemic prodrome: hunger, malaise, shaking, and sweating. Prompt recovery occurs with IV or oral glucose or an alternative form of sugar administration. Occasionally, injection with glucagon is necessary to elevate the blood glucose level. Prolonged hypoglycemia can cause cerebral injury.

Panic and Hyperventilation

Panic attacks are usually associated with autonomic disturbances such as tachycardia, sweating, and piloerection. Consciousness is usually preserved, so these attacks are easily recognized. Hyperventilation is common. Overbreathing causes respiratory alkalosis, which leads to dizziness, anxiety, and sometimes perioral or peripheral tingling and carpopedal spasm. On rare occasion, there is brief loss of consciousness.

Basilar Migraine

Basilar migraine involves migraine-like symptoms of lightheadedness, vertigo, limb numbness, and speech disturbance, sometimes without headache. Consciousness can be briefly lost. When these symptoms occur for the first time, malignant etiologies (e.g., SAH, ischemia, dissection) must be considered. Patients with a long history of identical presentation of symptoms suggest a benign etiology.

Carcinoid Syndrome, Pheochromocytoma, and Scombroid Poisoning

The flushing and palpitation caused by these sudden biochemical insults are sometimes mistaken for anxiety, allergy, or focal seizure.

Drug Reactions

Acute dystonic reactions, such as drug-induced oculogyric crises, are sometimes mistaken for a form of seizure. Consciousness is preserved. The history of medication ingestion, especially neuroleptic medications such as olanzapine, risperidone, and haloperidol or antiemetics such as metoclopramide, help to identify this as a cause.

Narcolepsy, Excessive Daytime Drowsiness, and Cataplexy

Narcoleptic attacks are episodes of irresistible sleep that occur in inappropriate circumstances. Physical injuries are common, and motor vehicle crash is a common cause of untimely death in narcoleptic patients. Cataplexy is the sudden loss of lower-limb tone (i.e., falling with intact awareness). Attacks are often triggered by positive emotion, laughing, crying, or sudden change in ambient temperature.

Narcolepsy can be diagnosed with or without cataplexy and is sometimes accompanied by vivid hypnagogic (on falling asleep) or hypnapompic (on waking) hallucinations and sleep paralysis, the latter being a frightening inability to move while awake. These sleep-related phenomena can occur in isolation and are of no major medical consequence. When treatment is necessary, modafinil, methylphenidate, or sodium oxybate are used for narcolepsy and cataplexy.

MENINGITIS AND ENCEPHALITIS

MENINGITIS

A CNS infection requires a particularly pragmatic approach when one is far from help. Differentiating between bacterial and viral meningitis is difficult. Bacterial meningitis may be fatal if not treated promptly and properly.

Microorganisms reach the meninges by direct extension from the ears or nasopharynx, as a result of cranial injury or congenital defect, or through the bloodstream. Patients with compromised immune systems are at particular risk.

In acute bacterial meningitis, the pia-arachnoid becomes congested with neutrophils, and a thin layer of pus forms, which can form adhesions leading to cranial nerve palsies, vasculitis, and hydrocephalus. With chronic infection (typically with tuberculosis), the brain surface becomes covered in sticky gray-green exudate with numerous meningeal tubercles. Adhesions are invariable. Brain edema occurs with any bacterial meningitis at a variable rate.

MENINGITIC SYNDROME

The classic diagnostic triad for meningeal irritation is headache, stiff neck, and fever. With acute bacterial meningitis, there is usually intense malaise, fever, rigors, headache, photophobia, and vomiting that develop within hours after infection. The patient is irritable and prefers to lie still. Neck stiffness and positive Kernig's sign (i.e., neck and back pain when the hip and knee are flexed with the patient supine) usually appear within hours. Brudzinski's sign (i.e., spontaneous flexion of the hips and knees when the neck is flexed with the patient supine) may also be present.

The diagnostic conundrum is that with bacterial meningitis, there may be few prominent meningitic features, and the clinical picture may resemble viral meningitis. For example, neck stiffness may not be evident. If in doubt, treat the patient for bacterial meningitis.

CLUES TO SPECIFIC VARIETIES OF BACTERIAL MENINGITIS

Table 51-2 lists possible infecting organisms with clinical signs. However, in wilderness practice, treatment for an unknown pyogenic organism will be required.

TABLE 51-2 Potential Sources of Meningitis

Clinical Evidence	Potential Pathogen
Petechial rash	*Neisseria meningitidis*
Skull fracture Ear disease Congenital central nervous system lesion	*Streptococcus pneumoniae, Haemophilus influenzae,* group A β-hemolytic streptococci, *Staphylococcus aureus, Staphylococcus epidermidis,* aerobic gram-negative bacilli
Immunocompromised patient	*S. pneumoniae, N. meningitidis, Listeria monocytogenes,* aerobic gram-negative bacilli
Pleuritic pain with or without rash	*Enterovirus* infection
International travel	Possible malaria or poliomyelitis
Environmental issues (e.g., polluted water, canals, sewage)	Leptospirosis

EMERGENCY WILDERNESS MANAGEMENT OF MENINGITIS

Fulminant forms of acute bacterial meningitis can be rapidly fatal. If acute bacterial meningitis is suspected, treatment must be started immediately and before evacuation is arranged.

Tables 51-3 and 51-4 indicate a practical treatment approach based on infecting organisms and age. It is important that up-to-date information about treatment protocols, potential allergies, and drug side effects is sought before embarking for a remote area. Evidence suggests that administration of dexamethasone every 6 hours reduces complications, such as brain edema and delayed deafness. The benefit is specific to pneumococcal meningitis, but the diagnosis of this specific organism is not practical in the wilderness setting.

TABLE 51-3 Empiric Antibiotics for the Treatment of Possible Adult Acute Bacterial Meningitis

Suspected Organism	Antibiotic	Alternative*
Unknown	Vancomycin (15-20 mg/kg IV every 8 to 12 hr, maximum 2 g per dose or total daily dose of 60 mg/kg; adjust dose to achieve serum trough of 15-20 mcg/mL) *plus* cefotaxime (2 g IV every 4 hr) *or* ceftriaxone (2 g IV every 12 hr) for 7-14 days	If severe β-lactam allergies and normal renal function: vancomycin *plus* moxifloxacin (400 mg IV once daily) *plus* (if *Listeria* coverage is required†) trimethoprim-sulfamethoxazole (5 mg/kg of trimethoprim component) IV every 6 to 12 hr
Neisseria meningitidis	Cefotaxime *or* ceftriaxone‡	Chloramphenicol, fluoroquinolone, aztreonam‡
Streptococcus pneumoniae	Vancomycin *plus* cefotaxime *or* ceftriaxone‡ for 14 days	Fluoroquinolone (e.g., moxifloxacin)
Haemophilus influenzae	Cefotaxime‡ *or* ceftriaxone‡ for 14 days	Chloramphenicol, cefepime, meropenem, fluoroquinolone‡
Listeria monocytogenes†	Ampicillin (2 g IV every 4 hr) *or* penicillin G (4 million units IV every 4 hr)	If severe β-lactam allergies and normal renal function: trimethoprim-sulfamethoxazole

Data from Clarke C, Howard R, Shorvon S, et al: *Neurology: A Queen Square textbook,* Oxford, UK, 2009, Wiley Blackwell Publishing, p 292.
IV, Intravenously.
*For example, in the case of allergy.
†Risk factors for *Listeria* meningitis: pregnancy, glucocorticoid therapy, immunocompromising conditions, age >50 years.
‡Dosing information for these medications can be found in the text of this chapter.

TABLE 51-4 Recommended Empirical Antimicrobial Therapy for Suspected Bacterial Meningitis Based on Age

Age	Common Bacterial Pathogens	Antimicrobial Therapy
<1 month	*Streptococcus agalactiae, Escherichia coli, Listeria monocytogenes*	Ampicillin, 100 mg/kg, *plus* cefotaxime, 50 mg/kg every 6 hr *or* Ampicillin, 100 mg/kg, *plus* an aminoglycoside (gentamicin, 2.5 mg/kg, *or* tobramycin, 2.5 mg/kg) every 8 hr
1 to 23 months	*Streptococcus pneumoniae, Neisseria meningitidis, S. agalactiae, Haemophilus influenzae, E. coli*	Vancomycin, 15 mg/kg IV every 6 hr (maximum daily dose of 4 g), *plus* cefotaxime, 100 mg/kg IV, followed by 200-300 mg/kg/day in four divided doses (maximum daily dose of 12 g), *or* ceftriaxone, 50 mg/kg IV every 12 hr (maximum daily dose of 4 g)
2 to 50 years	*N. meningitidis, S. pneumoniae*	• *Children:* Vancomycin, 15 mg/kg every 6 hr, *plus* ceftriaxone, 75-100 mg/kg every 12-24 hr, *or* cefotaxime, 75-100 mg/kg every 6-8 hr • *Adults:* Vancomycin, 15 mg/kg every 8 hr, *plus* ceftriaxone, 2 g every 12 hr, *or* cefotaxime, 2 g every 4 hr
>50 years	*S. pneumoniae, N. meningitidis, L. monocytogenes,* aerobic gram-negative bacilli	Vancomycin, 15 mg/kg every 8 hr, *plus* ampicillin, 2 g every 4 hr, *plus* ceftriaxone, 2 g every 12 hr, *or* cefotaxime 2 g every 4-6 hr

VIRAL, SUBACUTE, AND CHRONIC MENINGITIS AND ENCEPHALITIS

Most patients with viral meningitis begin to improve after several days without specific treatment. Fever, change in mental status, and seizure should raise concern for viral meningitis caused by a member of the herpesvirus family (e.g., herpes simplex, varicella-zoster), especially in the presence of preceding or concurrent rash in a dermatomal pattern. Timely administration of acyclovir (10 mg/kg IV every 8 hours) can be lifesaving. One should be familiar with vector-borne diseases transmitted by mosquitoes, ticks, and fleas that are specific to the region and season. Examples include West Nile virus and eastern equine encephalitis virus. The Division of Vector-Borne Diseases at the U.S. Centers for Disease Control and Prevention (CDC) has useful information about these diseases and their geographic distribution (http://www.cdc.gov/ncezid/dvbd/about.html). Meningitis that runs a more chronic course (e.g., with neck stiffness and drowsiness lasting several days or weeks) may be caused by tuberculosis, another chronic meningeal infection (e.g., cryptococcal meningitis, brain abscess, encephalitis), or a general medical condition (e.g., endocarditis). Evacuation is required in these situations.

PERIPHERAL NERVOUS SYSTEM CONDITIONS

Focal neuropathies, which are caused by afflictions of peripheral nerves and radiculopathies (i.e., nerve root lesions), are seen frequently in wilderness settings because of unusual postures, uncomfortable sleeping positions, heavy backpacks, carrying gear, and related activities.

MEDIAN NERVE COMPRESSION AT THE WRIST: CARPAL TUNNEL SYNDROME

Carpal tunnel syndrome is a common cause of nocturnal tingling in the hand in a median nerve distribution and involves clumsiness with weakness, typically of thumb movements (e.g., when opening bottles). Pain in the forearm is often prominent. Numbness may extend above the wrist or may be vague and poorly localized. Weakness of the abductor pollicis brevis (i.e., moving the thumb at right angles to the palm) is the earliest motor sign.

A wrist splint that holds the wrist in slight extension at night is helpful. Elevation of the arm at night may also help. Corticosteroid injection into the wrist along the median nerve is neither simple nor risk free. If symptoms persist long term, nerve conduction studies should be advised on return. Surgical carpal tunnel decompression can be curative.

ULNAR NERVE COMPRESSION AT THE ELBOW

Isolated, transient tingling in an ulnar nerve distribution is frequently benign (Figure 51-1). Persistent tingling that includes weakness of finger abduction implies substantial compression of the ulnar nerve at the elbow. Treatment with elbow splint, elbow pads, and avoidance of leaning on the elbows is advised. Symptoms lasting more than 1 week may require surgical ulnar nerve decompression and transposition.

RADIAL NERVE PALSY: COMPRESSION IN THE SPIRAL GROOVE OF THE HUMERUS

Radial nerve lesion resulting in acute wristdrop, known as "Saturday night palsy," is typically caused when an individual falls asleep with one arm draped over a hard surface. The patient awakens thinking that he or she may have had a stroke. Weakness of wrist and finger extension causes profound disability, but there is typically little or no sensory loss. Palmar wrist splinting in the position of function can be helpful. Weakness almost always resolves spontaneously in about 3 to 6 weeks.

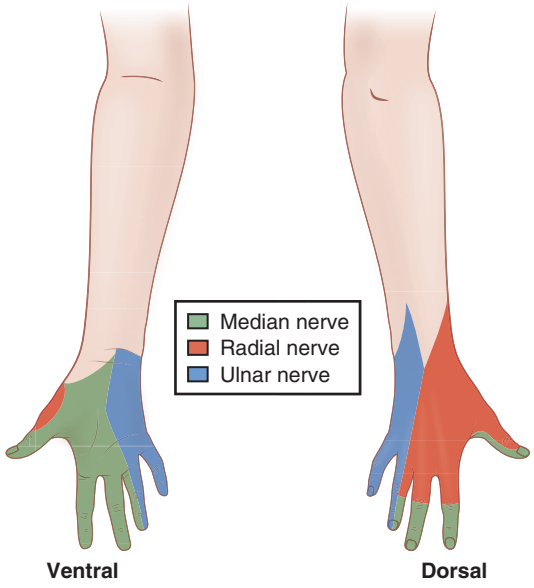

FIGURE 51-1 Typical sensory distribution of the radial, median, and ulnar nerves. The ventral surface corresponds to the palm of the hand, and the dorsal surface to the back of the hand.

BACKPACKS: NUMB HANDS AND BACKPACK PALSY

Transient numb and weak hands may occur from shoulder straps–induced trauma. Symptoms usually resolve within a few days. Occasionally, damage occurs to a branch of the brachial plexus. It is often difficult to determine the precise nerve that has been affected. True backpack palsy was first described as being caused by compression of the long thoracic nerve. This is a rarity that causes weakness of the serratus anterior muscle and leads to a "winged" scapula, which is diagnosed when the patient pushes the palm of each hand against a wall; the weak scapula protrudes from the back. The first step of treatment is to remove any potential source of compression, such as a heavy backpack, and wait for spontaneous improvement over a few days to weeks. If the symptoms worsen or new symptoms, especially new motor weakness, develop, the patient may be referred for a detailed neuromuscular examination and electromyography to exclude other causes, such as motor neuron disease or compressive mass.

THIGH PAIN AND TINGLING: MERALGIA PARESTHETICA

Painful tingling of the outer thigh in the distribution of the lateral cutaneous femoral nerve of the thigh is common, especially among persons who are obese or wear tight pants. It is also sometimes seen when the thigh swells after strenuous exercise. The problem usually resolves over a few days, and no treatment is necessary. Emptying the pants pocket on the affected side might prevent recurrence.

FOOTDROP: PERONEAL AND SCIATIC NERVE LESIONS

Peroneal nerve palsy leading to footdrop is the third most common focal motor neuropathy, after compression of the median and ulnar nerves. The peroneal nerve runs behind the head of the fibula. Palsy is usually noticed after prolonged squatting or habitual leg crossing, which results in unrelieved pressure on the peroneal nerve. There is profound weakness of the tibialis anterior, extensor hallucis longus, and peroneal muscles. The patient cannot dorsiflex and evert the ankle or extend the great toe. Deep tendon reflexes remain intact, and a small patch of numbness above the lateral malleolus is often barely noticeable. The usual course is spontaneous recovery over a few weeks. An ankle splint that supports the foot comfortably at a right angle

to the tibia may be useful. Activities or positions that put pressure on the lateral knee, such as leg crossing and leaning the affected knee against hard surfaces, should be avoided.

Sciatic nerve palsy, caused by pressure in the gluteal region, causes weakness of the hamstrings and of all muscles below the knee. Transient minor sciatic palsy is the "dead leg" that results from sitting on the edge of an uncomfortable chair and resolves within minutes. Nontransient sciatic nerve palsy is usually associated with distinct trauma to the buttock (e.g., femoral fracture with associated hematoma, penetrating trauma to the buttock). When one develops sciatic nerve palsy without a clear inciting event, an alternative diagnosis should be considered, such as lumbosacral radiculopathy from intervertebral disk herniation. If there is concurrent acute bladder or bowel dysfunction, cauda equina syndrome should be considered.

Sciatic nerve pain ("sciatica") is not uncommon. It is usually unilateral, described as sharp and burning, with intermittent shocks of shooting sensation traveling from the buttock down the posterolateral thigh and leg. It may result from stretching or irritation of the sciatic nerve as it travels through the pelvis, beneath or piercing through the piriformis muscle. In the absence of spine or disk disease, inflammation of the piriformis muscle from overuse or trauma may cause irritation of the sciatic nerve and classic pain symptoms. Pain is exacerbated by stretching the nerve (e.g., hip flexion, knee extension) and relieved by reducing tension on the nerve (e.g., walking with the foot on the affected side turned outward, which rotates the hip externally). Treatment with NSAIDs and physical therapy can be helpful.

LUMBAR BACK PAIN: LATERAL AND CENTRAL DISK PROTRUSION

Lateral lumbar disk protrusion typically causes a fifth lumbar (L5) or first sacral (S1) nerve root lesion. There is acute low back pain in a sciatic distribution that is worse with raising the straightened leg. With an L5 lesion, extension of the great toe becomes weak. With an S1 lesion, the ankle deep tendon reflex is lost. Even when there is substantial weakness, the usual course is for spontaneous recovery over days to weeks. When weakness is progressive, spinal surgery may be required to relieve compression on the affected nerve roots, so evacuation is advisable.

Central lumbosacral disk protrusion is an uncommon surgical emergency. While low back pain is a common complaint, red flags that herald more serious causes include sacral and perineal numbness and flaccid weakness of the lower limbs, usually for several hours, with the retention of urine; this is known as *cauda equina syndrome*. Loss of rectal tone and sensation are additional signs of cauda equina syndrome. History of metastatic cancer, prior IV drug use, and presence of a fever support the diagnosis of cauda equina syndrome. This is a true surgical emergency, and immediate evacuation is indicated. Even with prompt surgery, many of these patients have residual deficits.

CERVICAL DISK LESIONS

Acute Lateral Cervical Disk Protrusion

Acute lateral cervical disk protrusion causes sudden pain in the neck and down the arm. Neck movement is restricted by pain and muscle spasm. Pain may be excruciating and may require opiate analgesia. The seventh cervical (C7) root is most often affected. Root pain radiates to the myotome: in this case, the triceps, muscles deep to the scapula, and forearm extensors, all of which may be weak, with paresthesias and numbness in the C7 dermatome, which includes the middle finger. The triceps tendon reflex is lost.

Place the patient in a supine position and immobilize the neck. A premade or improvised neck splint should be used to prevent neck motion. Acute pain usually resolves within 1 week. Progressive weakness is an indication for evacuation. A minority of these patients requires diskectomy.

Neuralgic Amyotrophy (Acute Brachial Neuritis)

This intensely painful condition, which is presumed to be an acute demyelinating brachial plexopathy with an allergic basis, is mentioned because it is frequently misdiagnosed as an acute cervical nerve root lesion. Severe acute pain develops around the shoulder girdle and is followed by weakness and wasting of the supraspinatus, infraspinatus, deltoid, and serratus anterior muscles, all during the course of a week. Gradual resolution over many months is the rule. Pain management may require opiates. Corticosteroids (e.g., 50 mg of prednisone for 5 days) are often given for neuralgic amyotrophy, although there is little evidence base for this therapy.

Central Cervical Disk Protrusion (Cervical Myelopathy), Cord Compression, and Paraparesis

With central cervical disk compression of the spinal cord, the patient typically presents with ascending lower-limb numbness, spasticity, and weakness, sometimes accompanied by urinary incontinence. Spastic paraparesis implies possible spinal cord compression, potentially a neurosurgical emergency. High cervical lesions can result in life-threatening hypotension and bradycardia because of sympathetic dysfunction and inadequate respiratory mechanics. Immobilization of the neck, maintenance of a patent airway, and hemodynamic support are indicated. Acute trauma (e.g., axial loading of the spine by diving into shallow water) is a common cause of acute spinal cord injury. Although there are many causes of spastic paraparesis, including viral infection, precise diagnosis requires imaging. Evacuation is mandatory.

CRANIAL NERVE PALSIES AND VERTIGO

BELL'S PALSY

Palsy of cranial nerve VII is by far the most common, and is known as Bell's palsy. Bell's palsy is frequently the result of herpes simplex virus reactivation. Rare causes include malignant infiltration of cranial nerve VII or sarcoidosis. In endemic areas, Lyme disease can cause bilateral facial nerve palsies.

Patients with Bell's palsy present with acute unilateral (or rarely bilateral) facial weakness. Pain behind the ear is common. Patients may leak oral secretions from the side of the mouth and be unable to close the eye. Because this is a lower-motor-neuron deficit, the patient should be unable to wrinkle the ipsilateral forehead. Unilateral hyperacusis and loss of taste may be present. Other cranial nerves are not affected, and there are no other neurologic findings. Patients with Bell's palsy often complain of subjective numbness of the affected face but fail to demonstrate actual sensory deficits to testing. When there is an unequivocal loss of sensation, an alternative diagnosis is likely. Facial weakness with any other deficits is not Bell's palsy.

Spontaneous resolution within 6 weeks is usual in about 80% of cases. It is essential to protect the eye (e.g., cornea), especially during sleep, by taping the lid closed. Treat superficial eye infection with antibiotic drops or ointment (see Chapter 48). Drug treatment with valacyclovir (1000 mg PO three times daily for 1 week) and a corticosteroid (prednisone, 60 to 80 mg PO daily) is often given.

ACUTE VERTIGO

Sudden rotary vertigo is a distinctly unpleasant symptom. *Vestibular neuritis* (VN) is a presumed viral infection. VN causes severe prostrating rotary vertigo with vomiting. Jerk nystagmus (i.e., oscillating fast-slow nystagmus) is typically present. Severe symptoms last for a few days before their gradual resolution.

Benign paroxysmal positional vertigo (BPPV) is believed to be caused by loose otolith fragments within the semicircular canals. Onset of rotary vertigo is typically sudden and provoked by head movement (e.g., rising from bed). Neurologic examination is typically unremarkable until positional testing is done. The Dix-Hallpike maneuver tests for dysfunction of the posterior semicircular canal, which is the most common cause of BPPV. With the patient sitting, the neck is extended by 30% with the head turned to one side. Rapidly lay the patient back, so that his head hangs

over the edge of a stretcher or bed. Make sure that the patient keeps the eyes open in this position. Observe the eye movement until nystagmus occurs or 30 seconds have passed without nystagmus. Then, return the patient to upright position while maintaining the same head position, and observe for another 30 seconds for nystagmus. Then, turn the head to the other side and rapidly lay the patient back, and repeat the maneuver. For patients with BPPV, the Dix-Hallpike maneuver provokes vertigo and rotatory nystagmus after a few seconds of latency, and the nystagmus usually lasts less than 1 minute. The nystagmus has a characteristic trajectory, upward and rotatory, with the upper poles of the eyes beating toward the ground when the posterior canal dysfunction is present in the lower ear (e.g., if such nystagmus occurs when the patient is rapidly lowered to supine with the head turned to the right, the right posterior canal is the culprit). After the patient sits up, nystagmus will recur, after a few seconds of delay, in the opposite direction. The maneuver should be repeated to the same side. The intensity and duration of nystagmus will subside ("fatigue") with each repetition. With a positive Dix-Hallpike test, the patient typically complains of severe nausea and may vomit.

Once the diagnosis of BPPV is confirmed, repositioning maneuvers should be attempted for immediate treatment. One example is the Epley maneuver (Figure 51-2). Begin with the patient sitting up with the neck extended and head turned to the side that provoked the most severe symptoms and nystagmus. Rapidly lay the patient supine while maintaining the head position so that the head is turned to the right and hangs over the edge of the bed. This may prompt nystagmus and nausea. After the nystagmus resolves, rotate the head to the unaffected side so that the other ear is now facing the floor. Hold the position for 30 seconds. Then, have the patient roll in the same direction onto his or her side while the examiner rotates the head until the nose is angled toward the floor. Hold the position for 30 seconds. Bring the patient into a sitting position. The entire sequence of the Epley maneuver can be repeated until no nystagmus occurs. This often requires several repetitions to be effective, and the patient may report slight improvement of vertigo and nausea with each repetition.

Vertigo can have other causes, including migraine and CNS lesions of the brainstem or cerebellum, such as stroke, a multiple sclerosis lesion, or tumor, but these causes rarely lead to isolated vertigo. Ménière's disease presents with vertigo, tinnitus, and deafness. Vertigo guidelines include the following:

- The diagnosis is likely to be VN or BPPV if there are no other neurologic symptoms or signs.
- Presence of any additional signs (e.g., cerebellar ataxia, diplopia) suggests a structural lesion such as infarction, hemorrhage, tumor, or demyelination. Urgent evacuation to allow medical investigation is recommended.
- If the patient is without other neurologic signs, and Dix-Hallpike maneuvers elicit positional vertigo and nystagmus, BPPV is the likely diagnosis. Attempt Epley maneuvers. If this does not cure the problem immediately, repeat the procedure more vigorously, because this may dislodge the otolith fragment.

SLEEP

Problems sleeping are common in wilderness settings, often from a combination of crossing time zones, physical discomfort, anxiety or depression, and disrupted daily routine (e.g., long watches at sea, predawn starts on climbs, precarious bivouac sites). At altitude, hypoxia causes disrupted sleep patterns, irregular respirations, and episodes of sleep apnea.

Prolonged sleep deprivation causes exhaustion, poor judgment, and even mild confusion. Seizures may be provoked in persons with a low seizure threshold. Sleepwalking, jerking episodes, and movements in sleep, which are common events among the normal population, may become more evident and worrisome in the close confines of a wilderness living situation, such as within a tent.

SLEEP MANAGEMENT

1. Ensure that team members have adequate sleep duration (7 to 8 hours at a stretch for adults; 9 to 11 hours for children).
2. Ensure that sleeping accommodations are as comfortable as possible. A pillow, warm headgear, dry nightclothes, and earplugs can be remarkably helpful.
3. Avoid sedatives unless they are really necessary. Recognize repeated early-morning awakening as a possible feature of emotional depression.
4. Avoid caffeine, if possible, for 6 hours or more before any attempt to sleep.

MANAGEMENT OF CHRONIC NEUROLOGIC CONDITIONS IN AUSTERE SETTINGS

Presence of chronic diseases does not preclude engaging in meaningful outdoor pursuits. For most conditions, medical risks (i.e., those that would cause an underlying condition to worsen or cause death) are limited and generally less than the generally known risks of operating in a wilderness environment (e.g., danger of avalanche on a mountaineering expedition).

When giving patients advice, one should be encouraging but should consider and mitigate risk as much as possible. With

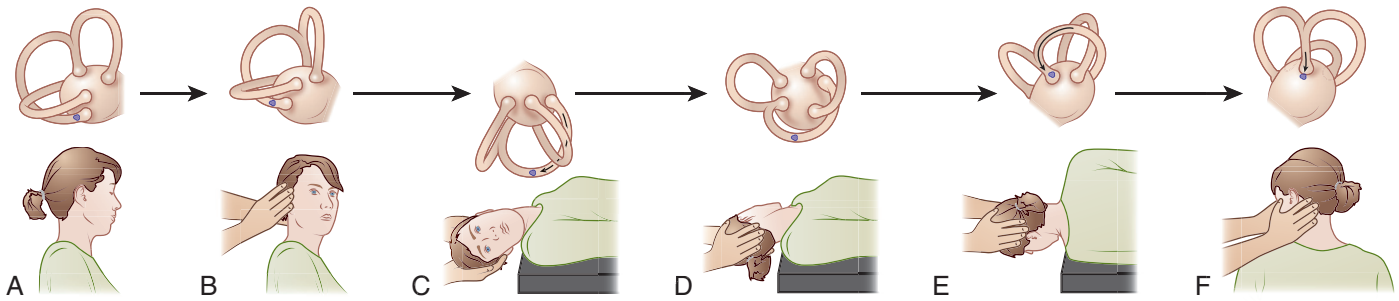

FIGURE 51-2 Epley maneuver to treat benign paroxysmal positional vertigo. The patient sits up on a bed or surface elevated above the ground (**A**). The examiner then turns the patient's head toward the affected side (**B**) and rapidly lowers the head below the level of the bed as the patient sits back (**C**), making sure to cradle the head to avoid injury. The examiner waits 30 seconds, then turns the patient's head to the unaffected side (**D**). The examiner waits another 30 seconds, then has the patient roll the torso to the lateral decubitus position. This will allow the patient's head to face downward (**E**). After another 30 seconds, the patient is allowed to sit up (**F**). The maneuver may need to be repeated for better therapeutic response. *(Modified from Kim J, Zee DS: Clinical practice: Benign paroxysmal vertigo,* N Engl J Med *370:1138-1147, 2014.)*

proper preparation, patients with such disorders as epilepsy, brain tumor, multiple sclerosis, and cerebrovascular disease may take part in prolonged expeditions. In one instance, a climber with a cerebral glioma traveled to Mt Everest and climbed to 7000 m (22,966 feet).

Patient education on potential risks and outcomes is critical. On extended or remote expeditions, group leaders and medical staff should be involved in discussions. The full range of medical opinions and ethical and cultural considerations should be discussed. Potential medical malpractice ramifications and evacuation plans should be addressed. Travel insurance companies are often reluctant to accept any increased level of risk and may deny policies to these individuals.

EPILEPSY

Well-controlled epilepsy should not be a barrier to wilderness activity. Risk assessment must include assessment of danger to other members of the party as well as to the patient.

When a patient with a history of seizures is assessed for suitability to join a trek at altitudes of 4500 to 5500 m (14,764 to 18,045 feet), consider the following:

- *Certainty of diagnosis*. Epilepsy tends to be overdiagnosed. Have all appropriate investigations (e.g., EEG, imaging) been carried out? Has there been a diagnostic assessment by a specialist in epilepsy? If not, suggest one.
- *Technical risks*. If the trekking route involves high footbridges or other obstacles where even a brief loss of conscious could result in a fatal injury, prudence would argue against patient participation. Rock climbing, sea kayaking, and diving (other than snorkeling in company) are usually prohibited.
- *Seizure control*. More than one seizure every few months, previous status epilepticus, and poor drug compliance would be reasons to reject an applicant for participation in certain wilderness activities.
- *Liaison with the treating neurologist*. Obtain recommendations about anticonvulsant drug treatment in the event of a seizure. Consider plans for evacuation.

There is no evidence that seizures are provoked by exercise, AMS, or HACE.

CEREBROVASCULAR DISEASE

Well-controlled hypertension usually remains so in wilderness settings and at high altitude. Patients who have suffered a TIA have a 25% chance of having a TIA, ischemic stroke, cardiac event, or vascular death within the following 90 days. Travel to high altitude or a wilderness setting should be discouraged during this period. Afterward, individual risks should be factored into the decision to travel to high altitude, depending on the mechanism of TIA. For example, a TIA with severe narrowing or occlusion of carotid, vertebral, basilar, or subclavian artery with recurrent symptoms of ischemia may cause the patient to experience recurrent symptoms or a new stroke at high altitude, possibly related to hyperviscosity, hypoxia, or dehydration. Patients who have recovered well after ischemic or hemorrhagic stroke should discuss with their neurologists their risks for recurrent stroke or other vascular events at high altitude and need for any special precautions (e.g., close blood pressure monitoring, antiplatelet therapy, maintenance of adequate hydration) before the excursion, because individual risks vary depending on the mechanism of stroke, age, and comorbidities. Anticoagulation therapy is not an absolute contraindication to travel in the wilderness, as long as the person uses caution walking on uneven surfaces and during activities where there is a risk of a fall or becoming injured, because minor injuries can lead to significant bleeding. Patients taking warfarin should be counseled regarding potential drug interaction with certain food items (e.g., leafy greens) and other medications (e.g., antibiotics) that may be encountered during the excursion. Portable monitors can allow safe titration of warfarin dosing in the field.

It is not uncommon for people to report TIA-like events (e.g., transient hemiparesis or aphasia) at high altitude (usually >7000 m [22,966 feet]). Symptoms can be attributed to hyperviscosity, incipient brain edema, thromboembolism, or a benign cause such as migraine. Age, cardiovascular risk factors, and postexpedition CNS imaging can help risk-stratify patients considering returning to extreme elevations. Practically, mountaineers become experienced by accepting major risks and will remain generally averse to restrictive advice based on their health status without concrete evidence. Conversely, the typical recreational trekker is much more likely to be risk averse and will likely adopt a cautious attitude.

MULTIPLE SCLEROSIS

Multiple sclerosis (MS) is an immune-mediated disorder against myelin in the brain, spinal cord, and optic nerves, affecting more than 2.3 million people worldwide. MS symptoms may range from infrequent and mild to fulminant CNS injury that causes severe disabilities.

Relapsing and remitting MS is characterized by clearly defined relapses with full or partial recovery. Disease-modifying therapies decrease relapse rate and slow down accumulation of brain lesions. A minor infection (e.g., common cold, gastroenteritis) can trigger an acute MS flare, usually treated with high dose glucocorticoids, rest, and the treatment of infection. For patients with chronic progressive MS, a significant increase in disability is unlikely during an expedition. Each patient must be assessed individually, with clear explanations given regarding the potential lack of hospital treatment.

Uhthoff's phenomenon describes acute worsening of MS symptoms caused by heat. This is the result of slow conduction in nerve fiber pathways that have been stripped of myelin. This is a temporary problem that occurs as body temperature increases. Weakness, ataxia, and visual symptoms may be provoked by strenuous exercise, exposure to high environmental temperature, or fever. Deficits resolve within hours with rest and cooling.

PARKINSON'S DISEASE AND OTHER NEURODEGENERATIVE CONDITIONS

Patients with Parkinson's disease who have a mild form of the condition have normal exercise tolerance and preserved motor skills. For example, they may be able to rock-climb at a high standard, and such exercise may be emotionally therapeutic. Asymmetric resting tremor ("pill-rolling"), cogwheel rigidity, hypographia (small handwriting), and masked facies are some of the key features. The slow tremor often subsides when the patient initiates movement of the affected limb. Fine motor coordination can be affected. Many patients with mild Parkinson's disease benefit from symptomatic drug therapy, such as levodopa-carbidopa, which can be taken three to four times daily. Although postural instability, autonomic dysfunction (e.g., orthostatic hypotension), and slowed reaction time in the event of losing balance may increase the risk of falls and associated injuries, patients usually can tailor their activities without compromising safety. Assistive walking devices can be beneficial.

SUGGESTED READINGS

Suggested readings for this chapter can be found online at www.expertconsult.inkling.com.

SUGGESTED READINGS

Clarke C, Howard R, Shorvon S, et al. Neurology: A Queen Square textbook. Oxford, UK: Wiley Blackwell Publishing; 2009.

Clarke ML. Neurological disease. In: Clark ML, Kumar PJ, editors. Clinical medicine. 7th ed. St Louis: Saunders; 2009.

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CHAPTER 52

Mental Health in the Wilderness

BRIAN STAFFORD

The "wilderness" is frequently considered to be a place of danger. This is ironic considering that, by almost all accounts, Western culture is perhaps the most environmentally dangerous and emotionally pathologic culture in history. Many believe that our urbanized and unsustainable culture has created ever-widening gaps in wealth, intractable political wars, shallow and omnipresent entertainment, certain inadequate educational systems, and increasing rates of mental health issues. Much of the cultural system's rampant destruction can be attributed to the lack of balance between time spent and familiarity with the natural world in comparison to the cultural world.

Most Western cultures primarily view nature and the attendant wilderness as a vehicle for recreational and adventure pursuits or for tangible financial benefit. When a culture primarily values the wilderness for its anthropocentric use, that culture is less likely to protect it, recreate within it, or participate in the scientific and cultural or spiritual aspects of wilderness environments.

Generally, wilderness environments have beneficial effects on the overall emotional and spiritual health of many individuals (Table 52-1). Individuals who consciously choose to pursue wilderness activities may be seeking relief from the stress of their hectic urban lives or looking for adventure lacking in their domesticated existence. Others pursue wilderness activities while, perhaps subconsciously, looking for deeper meaning to their lives. Although remote wilderness environments may provide opportunities for physical, emotional, psychological, and spiritual renewal, they can also present unique stressors. Many emotional and behavioral problems improve in the wilderness, but behavioral and emotional symptoms might emerge or become worse in response to the demands of wilderness experiences. Another way of thinking about wilderness is that it can provide a "eu" stress, or "good" stress, leading to an increase in attention and interest that might lead to optimal arousal and performance (Figure 52-1). When preparation is less than optimal or the challenge appears to be or is insurmountable, overwhelming signs of distress, such as fear and anxiety, may occur.

This chapter begins by discussing the benefits of wilderness and nature immersion from emotional, psychological, and psychospiritual perspectives. The role of the wilderness guide or wilderness medical provider in prevention, identification, and focused evaluation of symptoms is described, as well as modes of initiating psychosocial and, if necessary, psychopharmacologic support and managing and coordinating the expedition team with outside resources. Common psychiatric symptoms and medications are then reviewed, with a more focused discussion on disaster and survival psychology. The chapter concludes with a brief discussion about children and adolescents and future trends.

MENTAL HEALTH BENEFITS: ECOTHERAPY AND WILDERNESS THERAPY

The concepts of deep ecology and ecopsychology have entered the public consciousness, and ecotherapy and wilderness therapy as treatments have entered the clinical arena in the past 40 years. *Deep ecology* is a contemporary ecologic and environmental philosophy characterized by its advocacy of the inherent worth of living beings regardless of their instrumental utility to human needs, and advocacy for a radical restructuring of modern human societies in accordance with such ideas.[9] The field of *ecopsychology* seeks to develop and understand ways of expanding the emotional connection between individuals and the natural world, thereby assisting individuals to develop sustainable lifestyles and remedy alienation from nature.[2]

Outdoor behavioral health care includes ecotherapy, wilderness therapy, and adventure therapy.[4] *Ecotherapy* refers to healing and growth nurtured by healthy interaction with the earth. *Wilderness therapy* is use of wilderness expeditions for the purpose of therapeutic intervention. *Adventure therapy* involves the combination of physically and psychologically demanding activities, usually (but not always) conducted in a group setting.

Although defining these therapies and demonstrating evidenced-based outcomes for these programs is still a work in progress, anecdotal reports abound, and many European countries now authorize these therapies as standard or adjunctive treatments. It is hoped that there will be continuing progress with specific mental health and developmental benefits of nature experiences for children and adolescents, as well as incorporation of specific approaches to being in wilderness settings to foster greater psychic benefits for adults.[11]

ROLE OF WILDERNESS GUIDE/ MEDICAL PROVIDER IN EMOTIONAL AND BEHAVIORAL HEALTH

With regard to mental health, the role of the guide or medical provider in the wilderness or during an expedition encompasses areas of prevention, identification, evaluation, initiation, management, coordination, and collaboration (Table 52-2).

PREVENTION

Prevention of mental health issues during wilderness excursions can take two routes: one before ever entering the wilderness and one taken in the field. Screening forms for medical and mental health issues are mandatory for most wilderness excursions, if not by the guide, then by the insurer. The prospective participant's "Medical History Form" should include the person's previous psychiatric or mental health history of psychotherapy or counseling, medications, suicide attempts, psychiatric hospitalizations, prior substance use (hospitalizations, treatment, history of withdrawal, support groups, alcohol, tobacco, cannabis, painkillers, psychedelics, stimulants, methamphetamines, etc.), current stressors/losses, and previous wilderness experience (Table 52-3). Ensure that persons with chronic medical and psychiatric problems bring extra medications.

Any positive response that an individual is receiving current mental health care should initiate a request for the individual to have a letter from the primary mental health provider stating that the individual's coping strategies are such that the excursion is likely to be of benefit and not overwhelming. Because many participants do not report a history of psychiatric or substance treatment, even when given the opportunity, or they may be unaware that they may have symptoms or problems, this is an opportunity for screening and subsequent psycho-education. A simple and helpful online screening tool can be accessed at https://whatsmym3.com, completed by the participant, and returned with other trip registration materials.

Frequently, a phone call discussion with the prospective participant with regard to physical and psychological readiness for a wilderness immersion experience can be of benefit. Despite these precautions, individuals frequently do not report an

TABLE 52-1 Benefits of Nature Immersion

Benefit	Description	Examples
Psychological well-being	Positive effect on mental processes	Increased self-esteem Improved mood Reduced anger/frustration Psychological well-being Reduced anxiety Improved behavior
Cognitive	Positive effect on cognitive ability or function	Attentional restoration Reduced mental fatigue Improved academic performance Education/learning opportunities Improved ability to perform tasks Improved cognitive function in children Improved productivity
Physiologic	Positive effect on physical function and/or physical health	Stress reduction Reduced blood pressure Reduced cortisol levels Reduced headaches Reduced mortality rates from circulatory disease Faster healing Addiction recovery Perceived health/well-being Reduced cardiovascular/respiratory disease and long-term illness Reduced occurrence of illness
Social	Positive social effect at an individual, community, or national scale	Facilitated social interaction Enables social empowerment Reduced crime rates Reduced violence Enables interracial interaction Social cohesion Social support
Spiritual	Positive effect on individual religious pursuits or spiritual well-being	Increased inspiration Increased spiritual well-being
Tangible	Material goods that an individual or cultural can accrue for wealth or possession	Food supply Money

Modified from Keniger LE, Gaston KJ, Irvine KN, Fuller RA: What are the benefits of interacting with nature? *Int J Environ Res Public Health* 10:927, 2013.

accurate medical or psychiatric history. Therefore, it is also useful to inquire about recent stressors or losses or change in mental health status during check-in for an expedition.

Prevention through Effective Orientation, Leadership, and Teamwork

Wilderness activities are usually not solo affairs. Outdoor adventures can range from an 8-km (5-mile) day hike with a social group to an expedition that involves dozens of people who climb to the summit of Mt Everest. For adventures to be successful and safe, planning, preparation, leadership, judgment, and guidance are paramount. People who share wilderness activities together frequently experience the same types of group interactions that

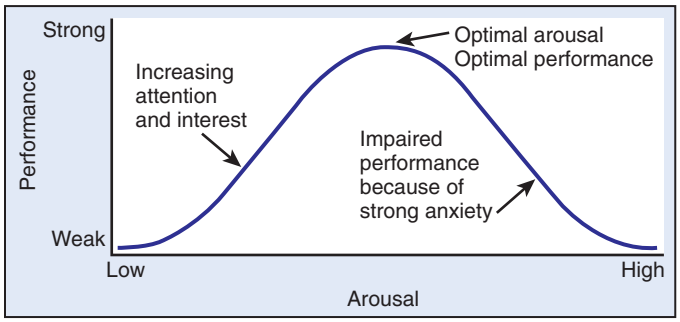

FIGURE 52-1 Stress bell curve.

TABLE 52-2 Role of Wilderness Guide or Medical Provider in Emotional/Behavioral Health

Health Care Area	Description/Activities
Prevention	Intake form Screening Approval from physician, therapists, psychiatrist Effective orientation Creating a safe container Grounding in wholeness Group dynamics
Identification	Tracking the emotional and behavioral field Participant direct or indirect report
Evaluation	Brief interview Rule out medical/physiologic/neurologic etiology Provisional assessment
Initiation	Brief support Access wholeness Psychopharmacologic support Confrontation Evacuation
Management	Ongoing support Evacuation
Coordination Collaboration	With other group or expedition members With outside resources

TABLE 52-3 Mental Health History*

Hospitalizations
Suicide Attempts
Outpatient Counseling
Psychiatric Medication
Substance Use History
Inpatient Treatment
Outpatient Treatment
History of Withdrawal
Substance Use:
Caffeine
Nicotine
Marijuana
Painkillers
Stimulants (Ritalin)
Psychedelics
Cocaine
Methamphetamine
PCP
Other
Current Stressors or Recent Losses

*Medical history form should include the following: previous psychiatric history, including psychotherapy or counseling, medication, suicide attempts, and psychiatric hospitalizations; previous substance use history, including hospitalizations, treatment, history of withdrawal, support groups, alcohol, tobacco, cannabis, painkillers, psychedelics, stimulants, methamphetamine, etc.; current stressors/losses; and previous wilderness experience.

occur among co-workers, families, and many other groups. A safe and rewarding wilderness adventure begins before the actual trip. Careful planning includes attention to numerous issues, including food, equipment, maps, screening of participants, impact on the environment, and necessary permits.[6] The planning process itself may involve a group working together. Subsequent to the adventure, some type of communication may be essential to bring social, emotional, or logistical closure to the experience.

The manner in which groups interact can result in a safe, challenging, and rewarding adventure, or a conflicted, dangerous, and disorganized affair. Trips in the wilderness sometimes involve people who may not yet know one another very well, yet are thrown into close quarters and frequent contact for an extended period. Regular close contact inevitably leads to annoyances and conflicts, but it can also result in pleasurable camaraderie. Leadership is one key ingredient to a successful wilderness experience. A group in the wilderness without a leader or a plan can be a recipe for disaster. Groups without direction or guidance tend to deteriorate; they often break into factions or drift aimlessly. Morale suffers and tempers flare if arguments and fights erupt. Such situations tend to bring out the worst in people. However, an effective leader can bring out the best in everyone. The type of leadership that is needed for a day hike differs from that needed for a difficult expedition. However, even the short hike takes coordination. What if someone breaks a leg on the hike? Who will go for help, administer first aid, and reassure upset members of the group?

Leadership skills should be taught and developed; otherwise, the leader may make the same mistakes repeatedly.[6] Good leaders have a vision for the outings but are sufficiently flexible to adapt according to exigencies of evolving circumstances. Successful leaders care about their people and build teams that make use of the strengths of each member of the group. Caring, sensitivity, and empathic listening are crucial components of a successful and effective leader. When effective leaders convey caring and empathy to an entire group, the results are increased cohesiveness and solidarity. Sometimes, leaders emerge during a crisis or disaster and are critical to survival of a group.[6] Leadership and interactions among group members evolve during the course of a trip. Leadership is often fluid, because individuals with different skills take leadership roles in different functional areas, such as building fires, reading maps, and maintaining good humor and morale. People can be remarkably courageous, kind, and ingenious, but they can also be petty, mean, and destructive. A group on a wilderness journey is a microcosm of society; in other words, the group is complex and multifaceted. This type of situation reflects the full range of strengths and weaknesses of both men and women.

Group Dynamic Creation and Monitoring

Successful expeditions and wilderness immersions require knowledge of the terrain, comprehensive preparation, and the ability to read the weather and maps, as well as other technical skills and physical abilities. They also require the leader or wilderness medical provider's ability to facilitate the process of a collaborative group. Process skills include the components of building trust, creating a mutually supportive environment, conflict resolution, and effective communication.[3] As with most medical professionals, wilderness guides typically have innate ability and extensive technical training but are short on training in interpersonal processes. A strong ability and focus on creating a supportive group is likely to decrease the likelihood of individual members having emotional decompensation or the group becoming dysfunctional. Additionally, identification of a group purpose and validation of each individual's unique hopes for the excursion will help cohesiveness. Clarifying individual responsibilities (personal care, feeding, enjoyment) and group responsibilities (mutual support, clear communication, allowing for differences, resolving conflict) can lead to success for individuals, the group, and the guide.

IDENTIFICATION

Identification of emotional and behavioral issues can occur in several realms. Individuals may approach the guide with concerns of their own or about another participant. In addition, the guide may notice a disturbance in the group field and become curious about the dynamic between individuals or an individual and the group. Also, the guide may have an intuition—through a gut feeling, felt sense, or a "knowing"—of a disturbance in the group field.

Tracking the emotional and behavioral field is akin to, and just as important as, tracking the weather or reading snow or river conditions. The optimal guide or wilderness medical provider is one skilled at reading multiple "fields," including knowing themselves, reading the field of participant-participant interaction, participant-group interaction, group-to-group interaction, and individual and group interactions with agencies and the local populace. In addition, guide skills include having a stance that is not detached, permissive, or authoritarian but primarily authoritative. *Authoritative* guides are issue oriented and pragmatic, rather than motivated by an external, absolute standard. They tend to adjust their expectations to the needs of the participant, group, and circumstance. Authoritarian guides exert control through power and coercion. Detached and permissive guides have a "laissez-faire" attitude to leadership—an "it's all good" persona that can lead to serious consequences.

EVALUATION

The process of cognitive, emotional, and behavioral evaluation in the wilderness consists of three steps: (1) a brief interview, (2) ruling out of medical, physiologic, or neurologic illness, and (3) the provisional assessment. The interview starts with what the informant shares or what has been noticed by the guide. First, obtain a description of the problem through a brief interview with the following questions:

- When did the problem start (before the excursion, after the event, in childhood)?
- If not of new onset, what has been done previously in terms of evaluation and treatment?
- What circumstances make the problem better or worse?
- How is the participant's ability to participate in individual and group tasks affected?
- How does the symptom or behavior affect the group dynamics?

- What attempts by the participant have been made to alleviate the problem?
- Do others have opinions about the symptom or concern?

Many emotional, cognitive, and behavioral symptoms that are consistent with a psychiatric disorder often have medical, neurologic, or other physiologic abnormalities that can cause or contribute to these symptoms, and these must be ruled out in the second step of evaluation.

Triage

Although mental illness is common, most emotional problems in the wilderness are mild and do not require evacuation or restraint. One of the most serious errors that can be made when assessing an individual with apparent mental problems is to fail to exclude an organic cause of the change in mental status. People can become agitated and irritable, for example, in response to encephalitis or dehydration. It is important to not forego the fundamentals of assessment, such as taking vital signs and obtaining a thorough medical history before the expedition and reviewing it during the evaluation. As with assessment and treatment of any medical problem in the wilderness, a careful systematic approach will yield the best triage decisions. Finally, as the third step in the wilderness evaluation, a provisional assessment is made and plan of action drafted.

Discerning between normative and impaired human emotions, cognitions, and behaviors is the first step in triage of these problems. It may be a challenge to assess which symptoms and behaviors are adaptive and which are maladaptive. All emotions are indeed valuable to the individual and need to be experienced and understood at some point, but under physical duress and without suitable psychosocial support, not all emotions should be expressed on an expedition. In addition, difficult personality states may be triggered that interfere with the expedition. For example, at the end of a physically challenging day of backpacking with a group, one member of the group may break down in tears. Such crying may be a healthy and adaptive response, or it may be a sign of depression or panic. The process of crying can relieve stress and result in the tearful backpacker receiving needed emotional support. Alternatively, such tears may suggest that the person feels mentally and physically overwhelmed. The person might be approaching a panic state, and if appropriate interventions are not undertaken, the person's mental state may deteriorate. The challenge is to determine whether a pat on the back and some validating and encouraging words are sufficient, or if the individual needs to be assisted out of the wilderness to prevent further emotional deterioration.

The medical provider then develops an etiologic hypothesis based on the information gathered:

1. The emotion or behavior falls within the range of normal.
2. The emotion or behavior is related to medication noncompliance.
3. The behavior is related to medical, physiologic, or neurologic impairment.
4. The emotion or behavior is a normal reaction to stressful circumstances (e.g., medical illness, change in family structure, loss of a loved one).
5. The problem is primarily a reflection of dysfunctional expedition dynamics (e.g., the participant is the symptom bearer, scapegoat, or identified patient for the group).
6. The problem indicates a possible psychiatric disorder.
7. The problem is primarily, or complicated by, an underlying medical condition.
8. Some combination of the previous etiologies.

To help with this assessment, Box 52-1 summarizes the recommendations made in this chapter regarding simple triage and treatment decisions for a variety of mental health problems in the wilderness.

TREATMENT INITIATION

Treatment is initiated based primarily on severity of the impairment. If the symptom impairs the participant's ability to function to the degree that it jeopardizes the safety of that individual or others in the group, the participant should be evacuated. If the impairment from the symptom is moderate, support from the guide or medical officer and the group should be offered. In addition, one can encourage such participants to access their own wholeness: can they find an aspect of their own being to comfort and reassure themselves? In some circumstances, one should attempt nonjudgmental confrontation in response to behaviors that jeopardize the individual or group's goals.

Brief support can stem from empathy, to an encouraging word, to reassurance. It is best to validate the feelings of individuals at the start rather than to talk them out of their feelings. For example, for an anxious and exhausted participant, "I totally get why you feel afraid and exhausted and why you don't want to attempt this; there is a part of me that feels the same way. But this is why you and I are here, and you have overcome many obstacles already, and the team and I believe you can manage this one as well."

Accessing Wholeness

Frequently, when faced with a participant who is caught in a frightened or wounded state with panic as the main emotion, support can be offered by talking the person into accessing the inner strength that has nurtured others when they have been frightened.

Confrontation can include discussing the participant's goals for the expedition and how the person's current state may be sabotaging these goals as well as the group's goals. Increasing levels of confrontation with direct consequence, should such behavior continue, needs to be followed through with ending the expedition for that individual.

Psychopharmacologic support in the wilderness can be initiated in unique circumstances, primarily in patients with overwhelming and paralyzing anxiety (Box 52-2), as well as psychotic symptoms related to delirium or a psychotic disturbance. Antidepressants (e.g., selective serotonin reuptake inhibitors [SSRIs], serotonin-norepinephrine reuptake inhibitors [SNRIs], tricyclic

BOX 52-1 Preparing to Deliver Psychological First Aid: Core Actions

- Contact and engagement
- Safety and comfort
- Stabilization
- Information gathering: current needs and concerns
- Practical assistance
- Connection with social supports
- Information on coping
- Linkage with collaborative services

BOX 52-2 Signs and Symptoms of Anxiety

Psychologic
Fears and worries
Increased dependence on others
Avoidance of anxiety-producing stimuli
Decreased performance
Increased self-doubt and irritability

Psychomotor
Motoric restlessness and hyperactivity
Sleep disturbances
Decreased concentration
Ritualistic behaviors (e.g., washing, counting)

Psychophysiologic
Autonomic hyperarousal
Dizziness and lightheadedness
Palpitations
Shortness of breath
Flushing, sweating, dry mouth
Nausea and vomiting
Panic
Headaches and stomach aches

TABLE 52-4 Triage, Support, and Treatment of Emotional, Behavioral, and Cognitive Concerns in the Wilderness

Areas of Concern	Management Approach
Anxiety disorders	Provide reassurance, support, and lorazepam.
Mood disorders, mild	Provide support and encouragement.
Mood disorders, severe (e.g., mania, suicidal ideation, violence)	Observe constantly, contain or restrain, give lorazepam and haloperidol, and evacuate.
Psychotic disorders	Observe constantly, contain or restrain, give lorazepam and haloperidol, and evacuate.
Organic mental disorders	Identify the cause and eliminate it if possible, observe constantly, contain or restrain, give haloperidol or other antipsychotic, and evacuate.
Personality disorders, mild	Avoid overreacting to annoying behaviors and intercede to prevent conflicts within the group.
Personality disorders, severe (e.g., violence, extreme behaviors)	Expel the individual from the group if possible or obtain assistance and terminate the outing.
Substance abuse (i.e., no delirium or psychosis)	Confiscate drugs and alcohol.
Substance abuse or withdrawal, severe (e.g., unstable vital signs, delirium, psychosis)	Give lorazepam for withdrawal and haloperidol or other antipsychotic for psychosis or delirium, observe constantly, contain or restrain, and evacuate.
Suicidal or violent behaviors	Observe constantly, contain or restrain, give lorazepam and haloperidol or other antipsychotic, and evacuate.
Somatic symptoms of psychological origin	Exclude an organic cause and provide support, reassurance, and firm expectations.
Disasters and posttraumatic stress disorder	Ensure physical and psychological security and safety, provide support, and provide psychological first aid.
Wilderness survival	Stop, take time to develop a plan, attend to physical security (e.g., shelter, warmth, water), and avoid panic.

antidepressants [TCAs], dopamine reuptake inhibitors [DRIs]), helpful in the treatment of depressive and anxiety disorders, frequently require 2 to 3 weeks before any benefit is seen. Benzodiazepines work quickly to reduce anxiety. Likewise, neuroleptic medications (typical and atypical antipsychotics) also work relatively quickly to combat both agitation and psychosis.

EVACUATION

Evacuation may be necessary when the safety of the individual is such that it cannot be ensured or that the individual's condition threatens the safety of the entire team. Evacuation may also be necessary if the condition fails to respond to other supportive measures and appears to be worsening.

SPECIFIC DISORDERS AND SYMPTOMS

The *Diagnostic and Statistical Manual of Mental Disorders,* 5th edition (DSM-V) is the most recent and widely used compendium of the nosology of psychiatric disorders.[1] It is widely criticized by mainstream mental health experts,[5,9] mental health advocates, and proponents of other more holistic, integrative, and nonperjorative or nonstigmatizing approaches. This manual is of little practical use in the wilderness.

The DSM approach is based on a medical model of the human psyche, with symptoms being placed into several relevant categories. In its current iteration, the broad categories are (1) anxiety disorders, (2) mood disorders, (3) schizophrenia spectrum and other psychotic disorders, (4) stress- or trauma-related disorders, (5) organic mental disorders, (6) neurodevelopmental disorders, (7) obsessive-compulsive and other related disorders, and (8) somatic and other related disorders.

Lifetime prevalence of a mental disorder is as follows: any depressive disorder, 20.8%; any anxiety disorder, 28.8%; impulse control disorder, attention-deficit/hyperactivity disorder (ADHD), oppositional defiance or conduct, or related disorder, 24.8%; and any substance use disorder, 14.6%. Based on previous DSMs, 1-year prevalence rate for adults, for any psychiatric disorder is 14.9%; for anxiety disorder, 11.8%; for any mood disorder, 5%; for bipolar spectrum disorders, 0.7%; for schizophrenia, 1%; and for any substance abuse disorder, 6%. The 1-year prevalence rate for any psychiatric or substance use disorder is 18.5%.[7] No studies have compared prevalence of mental health issues in the general population and in persons on wilderness excursions. One assumes that the rates of types of disorder are similar, even though individuals on wilderness excursions are assumed to have greater financial and social resources than does the general population.

Neither in-depth diagnostic assessment using DSM-V nor completion of a psychological test in a wilderness setting is considered practical. As previously stated, appropriate evaluation for triage and management in a wilderness setting depends primarily on the level of impairment of the individual as it affects the individual or the wilderness group, and much less so on classifying emotional and behavioral problems into one or more specific diagnoses within these broad categories.

In the following sections, five important topical areas for assessment and management of specific emotional problems in the wilderness are discussed: (1) the common characteristics of the six major categories of psychiatric disorders, (2) suicide and violence potential, (3) psychosomatic complaints, (4) impairment, and (5) response to crisis (i.e., disasters and survival psychology). Table 52-4 summarizes the triage of emotional, behavioral, and cognitive concerns in the wilderness.

ANXIETY DISORDERS

Fear is an emotion when there is an identifiable, precise, and well-defined threat. *Anxiety* is a set of responses to an unknown, imprecise, poorly defined threat, or perhaps even absent threat. Anxiety is a universal emotion that is usually a normal and adaptive response to everyday life. The experience of anxiety is both physical as well as psychological (see Box 52-2). Increased heart rate, blood pressure, and respirations; sweaty palms; and muscular tension are common physical manifestations that accompany the psychological feelings of anxiety and tension. Rarely is anyone totally free of anxiety; however, for most people, anxiety is mild.

The several types of anxiety disorders include generalized anxiety disorder, specific phobias, and panic disorder. Obsessive-compulsive disorder (OCD) is no longer considered an anxiety disorder, and posttraumatic stress disorder (PTSD) is now considered a stress and trauma-related disorder. People with generalized anxiety disorder worry excessively. Their anxiety is out of proportion to what most people would experience in similar circumstances. The afflicted individual may find it difficult to keep worrisome thoughts from interfering with attention that needs to be paid to current activities. The focus of the worries

among people with generalized anxiety disorders are mundane and include such concerns as awakening on time for work, making appointments, and performing chores. These individuals experience somatic symptoms, such as sweating, nausea, and diarrhea. The annual prevalence of generalized anxiety disorders is approximately 1.5%.

In a wilderness setting, the foci of worries might continuously shift from bear attacks, to getting lost, to not keeping up with the group, to flash floods, and so forth. These anxieties will likely diminish the person's enjoyment of a wilderness experience, affect concentration, and drain the individual's energy. People with generalized anxiety may negatively affect group morale. However, generalized anxiety problems are not emergencies; they are chronic problems that can be managed in the field. Psychologically, individuals with these pervasive symptoms have an inability to determine appropriate threat, nurturingly reassure themselves, or tolerate their own anxieties, leading to a reinforcing loop. People with a generalized anxiety disorder may require ample reassurance. A good leader can nonjudgmentally empathize with the individual, provide reassurance, and appoint other group members to assist with reassuring the anxiety-ridden person. The ability to access one's nurturing self is also recommended. Building rapport and trust with the afflicted person is a good first step to providing help. Trying to talk someone out of his or her worries is unlikely to be productive. In the field, if the symptom becomes impairing, a benzodiazepine (e.g., 0.5 to 2 mg of lorazepam two to three times daily) can provide immediate and short-term relief of anxiety-related symptoms. Side effects include sedation, memory difficulties, and impaired motor coordination. Sustained benzodiazepine use of several weeks' duration can lead to physical dependence and withdrawal symptoms with abrupt discontinuation. Therefore, such medications must be used cautiously in the field.

One of the more common specific phobias encountered during a wilderness outing is a fear of heights or exposure (e.g., to an edge with a drop-off). Other common phobias are to snakes, spiders, and water. A specific phobia is an unreasonable fear in anticipation of or on exposure to a particular object or situation. The intensity of anxiety with the exposure may vary from relatively mild to extreme panic. In some cases, phobias of heights cannot be overcome in the wilderness. For example, a trail that crosses a bridge over a chasm or that involves a section of precipitous exposure might be more than can be managed by an individual with a phobia to heights. Sometimes, gentle reassurance or distraction may be all that is required. Engaging height-phobic people in distracting conversations while backpacking in areas that involve dizzying exposure can be successful. However, if an individual is likely to respond with extreme anxiety and become unbalanced or flee, it is not safe to use this psychotherapeutic measure.

Individuals with panic disorder experience recurrent and unexpected panic attacks. A panic attack occurs during a discrete period that lasts approximately 10 to 30 minutes, manifesting with one or more of the following symptoms: sensation of a pounding heart, sweating, trembling, chest pain, nausea, dizziness, numbness, chills, hot flashes, and shortness of breath. During a panic attack, a person may fear that he or she is dying, having a heart attack, going crazy, or losing control. Such individuals often visit emergency departments out of fear that they have had a heart attack or that something is seriously, physically wrong with them. Any person with one of the anxiety disorders described in this section may experience a panic attack, but the person with a panic disorder experiences recurrent and unexpected panic attacks. These attacks occur without warning and seem to come "out of the blue," in contrast to being in response to a specific trigger (e.g., as with specific phobias, described previously). As a result of recurrent attacks, people may develop avoidance behaviors, because they do not want to be in situations where escape might be difficult or embarrassing or where help might not be available (e.g., agoraphobia). When these people are caught in these situations, they have a desperate desire to flee.

Panic disorders occur in approximately 1.6% to 5% of the population.[6] Treatment with selective serotonin reuptake inhibitors (SSRIs; e.g., fluoxetine, sertraline) or benzodiazepines (e.g., alprazolam, clonazepam), or a combination of the two, and with psychotherapeutic intervention can be successful in controlling panic disorder. People with panic disorder whose symptoms are not sufficiently severe as to be problematic may safely enjoy a wilderness adventure without difficulty. However, remote locations, bad weather, or physical challenges may increase the stress on such individuals, thereby causing them to experience a resurgence of symptoms. The afflicted person may want to escape from the wilderness and may be intolerant of being left alone, even for brief periods.

A person who is having a panic attack in the wilderness may present a diagnostic dilemma. The person may appear to be having a heart attack or experiencing acute respiratory distress caused by pulmonary edema. A careful history showing that the person has a history of panic disorder and does not have a history of heart disease may help. However, for an older person who may have risk factors for heart disease, the distinction between panic disorder and symptoms of a myocardial infarction may be nominal. People who have a panic attack usually begin to calm down within 30 minutes to 1 hour. Symptoms often respond to a benzodiazepine (e.g., lorazepam), reassurance, and support. Providing regular doses of benzodiazepines to an individual with a panic disorder or increasing the dosage of currently prescribed benzodiazepines may be enough to allow completion of the trip.

OBSESSIVE-COMPULSIVE DISORDERS

A person on a wilderness trip is observed constantly stopping to wash his hands in a stream or lake or with water that he is carrying. His hands are observed to be raw from frequent washing, and the time consumed by this activity is slowing the group's progress to a crawl. When asked about the handwashing, the man apologizes and acknowledges that his behavior is irrational but explains that he is deathly afraid of germs and cannot cease the behavior. Another person on a wilderness trip is frequently overheard counting in a whispered voice. When asked about the counting, the woman (with considerable embarrassment) explains that she is afraid that she will attack someone, so every time that she has a violent thought, she counts to 100.

The obsessions and compulsions of a person with OCD are usually seen as irrational by the person who is experiencing them, but at the same time the individual feels helpless to stop them. If prevented from performing ritualistic obsessions and compulsions, the person with OCD may feel panic briefly or may experience a full panic attack. A person with OCD may not be able to adhere to the requirements of a wilderness trip. For instance, the chronic handwasher may slow a group so profoundly that the group cannot make reasonable progress. People with OCD are not typically violent, but a person who is observed to be constantly counting to ward off violent behaviors may have difficulty fitting in with a group.

In the general population, the prevalence of OCD is 1% to 2.5%. People with OCD are often successfully treated with clomipramine or high-dose SSRIs and psychotherapy. It is unlikely that OCD symptoms will appear suddenly or for the first time on a wilderness trip. Frank disclosure to a wilderness group about the OCD symptoms of a group member can diminish the group's anxieties about the person's odd behaviors and make the person less likely to be isolated by the group.

MOOD DISORDERS

Sadness, moodiness, happiness, and elation are normal emotions within the human experience. However, when these become extreme, prolonged, or appear without response to experience or setting and interfere with normal functioning, these emotional responses are considered mood disorders. People with severe depression or unstable bipolar disorder (i.e., manic-depressive illness) usually will not and should not be encouraged to venture into the wilderness. However, people who are being successfully treated or who have relatively mild symptoms may participate in wilderness adventures. Seriously depressed people feel sad, useless, and bad about themselves and the world. Their view of

the world is dark, and they have difficulty believing that their lives will improve.

Depressed people have difficulty sleeping, little or no appetite for food, poor energy levels, and poor or absent concentration. They typically withdraw socially, cry for no reason, and have difficulty enjoying anything. The most common diagnostic term for this type of depression is *major depression*; the prevalence of this disorder is 2.5% to 10%. At their worst, people with severe depression may become psychotic and suicidal (see Psychotic Disorders next and suicide discussion later). People with mild problems are unlikely to develop severe symptoms rapidly, because severe symptoms gradually appear over weeks to months.

The more likely problem on a wilderness adventure is finding someone who started out with mild symptoms that are becoming worse, related either to the stress of the activity or, more likely, lack of compliance with medication. Such individuals may already be taking antidepressant medications (e.g., citalopram, paroxetine). These medications work slowly over weeks, so a change in dose during a wilderness trip probably would not provide significant benefit, although restarting medication is recommended. The most common problem that the wilderness group may face with a depressed person is that person's overall impact on group morale. Emotional outbursts, crying for no apparent reason, and offhand comments about suicide will likely concern and distress others in the group. If group members provide encouragement and emotional support, this may help the depressed person finish the trip safely. However, severe symptoms may not respond to ordinary support. A lack of response can anger others, who may begin to feel conflicted, because they recognize that they are punishing someone who is already suffering. As the symptoms grow worse, it may become necessary to hasten the depressed person's departure from the wilderness, especially if there is suicidal ideation.

Individuals with bipolar personality disorder (BPD) experience episodes lasting up to a few months of major depression alternating with periods of mania, often with normal functioning between the abnormal episodes. BPD is also considered a spectrum disorder, with a lifetime prevalence of the variants as follows: BPD I, 0.6%; BPD, 0.04%; subthreshold BP, 0.8%; and bipolar spectrum, 1.5%.[8] During periods of normal mood (euthymia) or the early stages of a manic phase (i.e., hypomania), bipolar persons may participate in wilderness adventures with little difficulty. During a period of hypomania, a person with BPD is very positive, productive, hardworking, energetic, and expansive. However, as the person becomes manic, problems become readily apparent. There is often rapid and pressured speech that is difficult to interrupt.

Persons with BPD might not sleep at all, or they may be excessively gregarious, become extremely irritable, and begin to believe they have superhuman powers. An individual may try to awaken a wilderness group in the middle of the night to hike up a nearby peak. When rebuffed, the manic person might take off alone with no water or protection from the weather because of the belief that he is superhuman. A guide described a participant on a wilderness trip who became manic and could not be dissuaded from climbing to the top of a mountain to speak to God. An individual with mania who is in the wilderness should be considered a medical emergency and evacuated as rapidly as possible.

Careful questioning may reveal that the manic person is taking psychiatric medications such as mood stabilizers (e.g., lithium, divalproex, lamotrigine), antipsychotic agents (e.g., risperidone, olanzapine), or both. People who take lithium must avoid dehydration, which can contribute to lithium toxicity. Lithium toxicity begins with tremulousness and can proceed to seizures and death. If lithium toxicity is suspected, the lithium should be discontinued and the patient well hydrated. The patient may require dialysis to resolve the toxicity, so evacuation is required. People with bipolar disorder sometimes stop their medications because they enjoy how they feel when they are hypomanic. The use of a benzodiazepine (e.g., lorazepam) or an antipsychotic medication (e.g., risperidone) can control some of the symptoms. Risperidone (2 to 6 mg) and lorazepam (4 to 8 mg) spread over the course of a day may be required to keep a manic individual calm.[5] It may be possible to coerce a manic person out of the wilderness using encouragement and enticements. Manic individuals can be extremely irritable and sometimes aggressive. Weapons should be confiscated. If a manic individual will not voluntarily leave the wilderness, then assistance—including engaging law enforcement—should be obtained as soon as possible. An evacuation may require the individual to be forcibly restrained and medicated. While waiting for help to arrive, members of the group must constantly contain the manic individual's excesses to keep both the affected individual and the group safe.

PSYCHOTIC DISORDERS

Individuals with severe mental illnesses (e.g., schizophrenia, schizoaffective disorder) do not typically venture into the wilderness as part of a group, especially if they are in a deteriorated state. Their overall functioning is too low for them to organize themselves to the point that they could join a wilderness group. Those persons who have been able to benefit from treatment, usually in the form of antipsychotic medications, sometimes venture with groups into the wild. The prevalence of schizophrenia is approximately 1% worldwide.[5] Tragically, the illness often manifests during young adulthood. The hallmark of these disorders is a substantial period, usually at least 1 month, during which the individual becomes unable to perceive reality rationally. A person who believes that a sinister group worshiping the devil has implanted a transmitter in his mouth (delusion), and who hears mysterious people saying that he is pathetic and should kill himself (auditory hallucination), is considered psychotic.

People with delusions and hallucinations cannot be talked out of these beliefs. People who are successfully treated with medications may still have some unusual or even delusional beliefs, but the psychotic symptoms have receded into the background and are not an active concern for the individual. In addition to psychotic symptoms, people with schizophrenia or a schizoaffective disorder often seem awkward or distant interpersonally. Their thoughts may be jumbled and disorganized. Concrete thinking that is literal or obvious with little ability for abstraction is common. If they are receiving adequate treatment, such individuals might enjoy a wilderness adventure with no difficulty, although their personal contribution to the group may be nominal. Antipsychotic medications, especially older drugs such as haloperidol, that are required for treatment of symptoms cause a wide range of side effects, including extrapyramidal symptoms (e.g., parkinsonism), acute dystonias, insomnia, sedation, and orthostatic hypotension.[8] Medication side effects alone may preclude a person from participating in a wilderness adventure, but people who have been taking medication for weeks or months may have adapted to the side effects. Newer antipsychotic medications (e.g., aripiprazole [Abilify]) have fewer side effects.

Problems arise for the wilderness group if a member becomes acutely or floridly psychotic. Symptoms may range from mild issues (e.g., a person asking the group why they are always talking about him) to paranoid beliefs that snipers are in the woods trying to kill the individual. Very mild symptoms may be tolerable, or the individual may respond to periodic gentle reassurance (i.e., reality orientation). Increasing the dose of the individual's already-prescribed antipsychotic medication may help, or a benzodiazepine may have a calming effect, at least temporarily. If psychotic symptoms become more severe with prominent paranoia or agitation, or if the person begins to make bizarre accusations toward others in the group, the situation should be treated as a medical emergency, and the person should be evacuated as soon as possible. Psychotic individuals' behaviors may be unpredictable and violent toward themselves or others. The many causes of psychotic symptoms include schizophrenia, schizoaffective disorder, mania, illicit drugs (e.g., methamphetamine), and organic causes (e.g., brain tumor, metabolic disturbance). A thorough history may help flesh out the differential diagnosis, but it may be difficult in the field to determine the cause of psychosis.

ORGANIC MENTAL DISORDERS

Many medical problems in the wilderness can cause behavioral symptoms, ranging from mild confusion associated with high-altitude hypoxemia to delirium associated with high-altitude cerebral edema (HACE). A person in the wilderness may have fallen and hit his head on a rock, which initially results in a minor scrape and a headache; however, this may progress to a subdural hematoma and delirium that may become life threatening. It is crucial to recognize which signs of delirium in the wilderness are true medical emergencies. A person with delirium has a fluctuating level of consciousness; he or she may be awake and alert for a few minutes and then become inexplicably sleepy. People with delirium are often confused and disoriented with regard to date, time, and place. Concentration, memory, and calculation ability may be impaired, and visual, tactile, or other types of hallucinations may appear. Paranoid delusions may occur.[5] The key sign for differentiating delirium from psychosis is that the psychotic person remains alert with no change in level of consciousness, whereas the delirious person's level of consciousness fluctuates over minutes or hours.

If delirium is suspected, a cause must be determined as rapidly as possible. Dehydration, hypoglycemia, HACE, head injury, meningitis, encephalitis, drug or alcohol withdrawal, and heat illness—to name but a few conditions—can cause delirium. In their confusion, people with delirium can easily injure themselves, so they must be constantly watched. Especially with delirious patients, physical restraint may be needed. In the wilderness, physical restraint can be accomplished by applying duct tape over cloth (to prevent self-injury from struggling against the restraints) to each limb and possibly to the trunk as well, with the person being restrained in a side- or face-down position to prevent aspiration. Physical restraint is potentially hazardous and should only be done as a last resort. Grand Canyon rangers have had to duct-tape individuals who are combative and delirious (as a result of dehydration) to litters before putting them on a helicopter for evacuation. If an easily treatable cause of the delirium cannot be identified and managed, evacuation is necessary.

PERSONALITY DISORDERS

Personality disorders are psychiatric disturbances that can cause considerable difficulty in the wilderness, although they rarely present as emergencies. Personality disorders are common, with an approximate 10% to 12% prevalence.[5] There are 10 different categories of personality disorders described in the DSM-V. People with personality disorders have enduring maladaptive patterns of behavior that usually date from adolescence. The use of a narrow set of adaptive strategies inflexibly leads these individuals to have chronic problems at school and work and in their interpersonal relationships. Ironically, many people with personality disorders do not tend to see themselves as having problems; rather, they blame others for their difficulties. Individuals with personality disorders unwittingly create their own special circle of misery.

Depression and substance abuse are common in this population. In DSM-V, individuals are described as having the essential features of a personality disorder, including significant impairments in self (identity or self-direction) and interpersonal (empathy or intimacy) functioning, and one or more pathologic personality trait domains or trait facets.

People with *schizoid* personality disorders are loners, and they tend to avoid relating to others, except on a shallow and superficial level. People who are schizotypal have odd and eccentric preoccupations and manners. They may be enamored of astrology, spiritual vortices, or numerology and tend to relate in an awkward or uncomfortable way. Annoying, aloof, and unable to join in comfortably with a group, these individuals do not usually cause major problems.

People with a *paranoid* personality disorder are constantly suspicious of others and believe that others have malevolent intentions toward them. Their suspicions are not totally absurd because their behaviors may make companions uncomfortable. For example, these individuals may believe that fellow travelers do not like them or will steal from them, but they do not believe that transmitters have been implanted in their brains or that the Federal Bureau of Investigation is conspiring to kill them. A person with persistent suspicions of others can be problematic for a wilderness group, because the successful functioning of such a group depends on trust. Group leaders need to work doubly hard to draw such individuals into the group and to avoid having the group isolate these people; such isolation serves only to amplify the paranoia and discomfort between the affected person and group.

Another group of personality disorders is sometimes referred to as the "dramatic and erratic" personality disorders. The person with *narcissistic* personality disorder is one who considers himself special and unusually talented and therefore deserving of special praise or attention. The person with a *histrionic* personality disorder is colorful and dramatic and demands to be the center of attention. The individual may be perceived as vain and shallow and often exaggerates physical complaints; for example, a small blister can cause excruciating pain and discomfort. The person with a *borderline* personality disorder is unstable and has a fragile sense of identity. Relations with a borderline person tend to be stormy; she may be exquisitely sensitive, and then react with rage at the slightest sign of rejection. The person with an *antisocial* personality disorder is a "confidence man" who may manipulate, intimidate, or steal to obtain what he wants. He can be superficially charming, but is irresponsible, unreliable, and sometimes aggressive and violent if his wishes are thwarted.

A third group of personality disorders includes the avoidant, dependent, and obsessive-compulsive personality disorders. The person with an *avoidant* personality disorder is constitutionally shy but desires relationships with others. The person with a *dependent* personality disorder is someone who looks to others to make decisions and care for the person. The person with an *obsessive-compulsive* personality disorder is task oriented and hardworking, but also a perfectionist, moralistic, and humorless. The obsessive-compulsive personality disorder is to be contrasted with OCD, described previously.

A psychologically intuitive leader may be able to run interference between the affected person and rest of the group. At other times, the maladjusted individual can be so disruptive that the leader or group must insist that the person depart. If this is the case, the person should always be assisted with exiting the wilderness. These individuals may not willingly accept being dismissed from the group, and they may become physically or verbally combative. They may be enraged and may state that they are going to kill themselves, file a lawsuit, or seek another form of retaliation. Nevertheless, if the individual cannot safely function as part of a wilderness group, the leader must remain firm in dismissing the person from the group and assist him or her safely out of the wilderness.

SUBSTANCE ABUSE DISORDERS

People who abuse illicit drugs or alcohol should not be on wilderness adventures; they are a hazard to themselves and others. A wilderness traveler may appropriately enjoy a small quantity of alcohol after a long day; however, using illicit drugs, such as methamphetamines, cocaine, heroin, or lysergic acid diethylamide (LSD), on a wilderness trip is dangerous. Legal use of marijuana is a new phenomenon in several states. Guides should have policies to distribute before the expedition regarding their company's position on using cannabis.

If the policy is that, for safety reasons, no drugs should be used, and if someone is unwilling to abide by the policy or is unable to stop using a prohibited drug(s), he or she should be escorted out of the wilderness. More problematic is a person who is prescribed narcotic medications (e.g., oxycodone) to control pain. Narcotics are central nervous system depressants that can impair concentration and coordination. In modest doses for medical indications, such medications may be reasonable. However, at higher doses or if abused, narcotics can compromise safety. If narcotic use poses a danger, the user should be escorted out of the wilderness.

Some people may be unaware that they have a substance abuse problem when they begin a wilderness trip, only realizing

their predicament after they begin to experience physical or psychological withdrawal. A careful history can establish if someone is experiencing alcohol or drug withdrawal. Alcohol withdrawal is considered a medical emergency because of the possibility for seizures, psychotic symptoms, and delirium tremens.[2] Withdrawal symptoms usually begin with shakiness and increased heart rate and blood pressure. Withdrawal from barbiturates or benzodiazepines can manifest similarly to alcohol withdrawal and can also be life threatening, if not directly, then in the risks posed by the environment and potential for accidents. If withdrawal is suspected, a benzodiazepine (e.g., lorazepam) can be given; several milligrams may be needed before vital signs stabilize.

Alcohol can be used to treat alcohol withdrawal symptoms, if necessary and available. People who abuse cocaine, methamphetamines, or narcotics will not experience life-threatening withdrawal, but their symptoms can be extremely uncomfortable and disabling. Withdrawal ("crashing") from substances such as cocaine is associated with extreme irritability and fatigue. Aches and pains that are typical of influenza are seen in association with withdrawal from narcotics. Withdrawal symptoms may last several days. Individuals who are experiencing alcohol or drug withdrawal should be evacuated.

On a wilderness trip, someone may experiment with natural plants or bring other plant-based entheogens in hopes of achieving an induced high or spiritual enlightenment. Tea made with jimsonweed or certain other plants can induce florid psychosis and delirium. Such individuals may shed clothes, develop severe sunburns, become dehydrated, and expose themselves to all manner of risks. Plants that can cause psychotic symptoms and delirium are common and should be avoided (see Chapter 65). People who are experiencing psychotic symptoms or delirium as a result of the ingestion of toxic plants must be watched constantly until they are evacuated to a hospital.

SUICIDE, VIOLENCE POTENTIAL, AND ANGER MANAGEMENT

Suicide is a major public health problem. The suicide rate in the United States is approximately 11.1 per 100,000 population, with a recent increase in suicides occurring among young people.[5] Depression and alcoholism are frequently associated with suicide. People who are depressed, lonely, or physically ill sometimes commit suicide. Rejection, unemployment, and legal problems are often associated with suicide. In the context of a wilderness trip, factors to consider are depression and recent loss. When completing a gear check immediately before an excursion, it is helpful to inquire about any recent medical or psychiatric changes and monitor what nonessential items, in particular weapons, are being brought in the backpack.

For example, a wilderness traveler who has been divorced, widowed, or fired from his or her job shortly before the trip may be at higher risk for suicide. A family history of suicide or history of suicidal behavior increases suicide risk. If there is a concern that someone might be suicidal, the person should be asked about suicidal thinking in a straightforward and concerned manner.

Asking about suicide does not increase suicide risk. People who kill themselves often think about or become preoccupied with ending their lives. They plan how they will kill themselves, so they should be asked about their plans for suicide. If the suicide potential is judged to be significant, the person must be watched closely. People who are delirious or psychotic can suddenly become impulsively suicidal during their confusion and frenzy. Suicidal individuals should be evacuated. More than 40 people have killed themselves below the rims of the Grand Canyon. Wilderness locations can be inviting places for people to end their lives, initially because of the beauty and then because of access to effective means (e.g., high cliffs). Many more people think about suicide than those who attempt or complete suicide; however, no reliable means is yet available for quantitatively assessing the seriousness of an individual's suicide risk. Extreme caution should be the watchword when someone is assessed as being even mildly suicidal in a wilderness setting.

Anger is an adaptive and universal human experience. With modulation, expressions of anger can signal distress, conflict, fatigue, or fear. Allowing people who are not typically angry to have time to "cool off" and then approaching them in a supportive and concerned manner can lead to rapid conflict resolution. Asking an individual who has been angry how he or she is doing can be a good beginning to dealing with the person. These individuals may report fatigue, fear about some aspect of the journey, or conflict with someone in the group. Active and nonjudgmental listening can often identify the problem and help lead to a resolution. For example, the wilderness trip may need to be slowed down to allow rest for someone who has become exhausted or to provide empathy for a person who is struggling to keep up on the journey. More problematic are people with psychiatric disorders, especially those with borderline or antisocial personality disorders, who have maladaptive ways of expressing anger and hostility. These individuals may be chronically angry, hostile, sarcastic, and even menacing on a wilderness journey.

Management of this type of anger should be characterized as attempting damage control. Intensive psychotherapy using cognitive behavioral techniques can be useful, but this is beyond what can be provided in the field.[6] People with these more serious forms of anger can have a very negative impact on a group, sometimes splitting the group into factions and causing an absence of camaraderie and undercurrent of tension. Periodically taking problematic individuals aside in private, providing support, and allowing them to ventilate while setting firm limits on their more troublesome behaviors can help diminish the impact that these individuals might have on a group. For extreme behaviors that involve blatant threats or actual violence, more draconian interventions may be needed, as described later.

The U.S. culture is much more violent than that of other industrialized nations. Rape, child abuse, homicide, and other violent crimes are far too common. Men are 10 times more likely than women to be violent, and young men are particularly prone to violence.[6]

Substance abuse is highly correlated with violence. People who have a history of acting violently in the past are more likely to do so in the future, and people who are going to be violent often signal their intentions. They act aggressively, clench their fists or jaws, and speak loudly or shout. They may have thoughts of acting violently, so they should be asked if they are thinking about hurting anyone. People who have a personality disorder or who are psychotic are also at an elevated risk for violence.

If a concern exists about someone being violent in the wilderness, weapons should be removed, secured, and guarded, and everyone should be vigilant. The potentially violent person should be evacuated or assisted out of the wilderness, with the help of law enforcement personnel if necessary.

SOMATIC COMPLAINTS

In previous iterations, these "somatoform" disorders had overlapping symptoms and lack of clarity about diagnostic boundaries. DSM-V reduces the number of diagnoses as well as the subcategories. The core feature of "somatic symptom disorders" is either somatic symptoms or the belief that one has an illness, with persistent thoughts, anxiety, and time and energy devoted to these complaints.

Some people are more sensitive to physical irritations and discomfort than others. For most people, a headache, minor abrasion, or sore muscle is not cause for alarm. For some individuals, however, a headache is a sure sign of a brain tumor, the abrasion will undoubtedly become infected and gangrenous, and sore muscles are evidence of a smoldering systemic illness. Simple reassurance that no major pathologic process is present may be enough for many people, but others continue to worry despite solid medical evidence to the contrary; these people suffer from hypochondriasis.[1] Insomnia, gastric distress, and fatigue are common complaints associated with travel and are usually not cause for concern. However, persons who tend to magnify the significance of common somatic complaints may worry excessively and may voice their concerns to the entire

group. Occasionally, this can have the effect of creating a kind of "mass hysteria," where other members of the group begin to experience the same symptoms.

For a person who is very worried about his or her health, repeated reassurance might be successful to ameliorate the problem. The difficulty lies in discriminating symptoms of little consequence from genuine and potentially serious organic medical problems. A woman on a wilderness trip in the mountains had a minor fall and became nonresponsive to painful stimuli. She was evacuated to a hospital, where she was released from the emergency department with no ill effects and no diagnosed cause for her unresponsiveness. This woman probably had a conversion disorder, which is rare but can manifest as sudden blindness, paraplegia, or unresponsiveness with no apparent etiology. Taking a good history to exclude an organic explanation for symptoms is important before concluding that the complaints are primarily of psychological origin. The Grand Canyon Park Service Rangers at Phantom Ranch and Indian Gardens regularly respond to individuals who report that they cannot physically hike out of the canyon. After taking a history to exclude organic illness, the rangers use a firm approach and tell these individuals that they can and will hike themselves out of the canyon. These individuals are encouraged to hike slowly, avoid hiking during the hottest part of the day, and remain hydrated, but are not given a helicopter or mule ride to the rim.

INSOMNIA

Complaints of insomnia are exceedingly common in U.S. culture, with its poor entrainment with the natural rhythms of the day. Insomnia complaints can approach a 1-year prevalence rate of 30% to 45%.[1] Although individuals may complain of poor sleep and daytime sleepiness, sleep laboratory assessments frequently demonstrate that these individuals sleep better than they subjectively report. Common insomnia-related complaints include difficulty falling asleep, frequent awakenings, and early awakenings. As people age, they normally sleep less and more lightly and have more frequent awakenings. This leads to frequent sleep complaints in elderly persons. Even for persons without baseline sleep problems, traveling long distances across several time zones to wilderness locations might lead to insomnia problems as a result of disruption of circadian rhythms. Just as often, individuals report that being away from noise, technology, screens, and the stresses of civilization leads to improvement in sleep. Individuals with insomnia often have poor sleep hygiene. They nap during the day, drink caffeine and alcohol, do not exercise, watch television or stare at a computer screen while falling asleep, have irregular sleep schedules, and sleep in noisy and uncomfortable settings. In wilderness settings, sleep problems can be accentuated as a result of heat, cold, humidity, high altitude, uncomfortable sleeping arrangements, and fears of bugs, snakes, and wild animals.

However, wilderness settings can also have a salutary effect on sleep because of the associated quiet, darkness, exercise, and freedom from the stresses of work and other responsibilities. Several suggestions can be made for persons who report difficulty with sleep in wilderness settings: no napping, no caffeine or alcohol, regular exercise, comfortable sleeping space in a tent or cabin, reassurance regarding wild animals, and a regimented wake time. It is best for people to cue their biologic sleep rhythm to the time that they usually awaken. In other words, if someone normally awakens at 6 AM, they should get out of bed and begin their day at 6 AM in the local time zone, even if they only slept a couple of hours the night before. By cueing to this awake time, sleep quality should gradually improve over subsequent nights. No one can sleep in a tent with a gale force wind blowing all night, but losing a night of sleep will not have any substantial effect on functional ability the next day, other than some increased sleepiness.

Sleep medicines are frequently used for insomnia but can have deleterious effects on wakefulness and coordination. Uncommon side effects are unusual behaviors (e.g., driving a car in the middle of the night with no destination) that are associated with memory loss for the event. Thus, medications to relieve insomnia should only be used if more conservative sleep hygiene suggestions fail. Drugs should also be instituted if an individual has a history of bipolar disorder because a lack of sleep and circadian disruption constitute a major risk factor for an episode.

Sleep disorders, such as narcolepsy, sleepwalking, and sleep apnea, could prove hazardous in a wilderness setting. *Narcolepsy* is characterized by sudden sleep attacks, even during the day, during which the afflicted person suddenly falls asleep with little or no warning. Narcoleptic patients are also subject to *cataplexy,* which means that they suddenly lose muscle tone, often in response to strong emotions. People who sleepwalk may not be aware of their behavior, which occurs during the deepest phase of sleep. Narcolepsy and cataplexy could present serious hazards on wilderness journeys, where individuals could inadvertently fall off a cliff or cause someone else to fall and injure themselves. *Sleep apnea* is associated with airway blockage caused by enlarged tonsils or uvula. This causes hundreds of awakenings each night, of which the individual may or may not be aware. Sufferers often snore, complain of fatigue and sleepiness, and are at risk for heart failure and stroke. At higher altitudes, symptoms associated with sleep apnea may worsen as a result of hypoxia and may lead the individual to be severely sleep-deprived. The person may have difficulty paying attention or may even fall asleep in hazardous situations.

RESPONSE TO CRISIS, TRAUMA, AND DISASTERS

In response to threat or danger, an extreme form of anxiety that may be triggered is the "fight, flight, or freeze" response. This response occurs automatically and without conscious control. Athletes, extreme sports enthusiasts, military combatants, crime victims, and anyone confronting serious injury or death may have this response triggered. The fight, flight, or freeze response is mediated through the amygdala and autonomic nervous system, which when triggered, results in the release of cortisol, increased blood pressure and blood glucose, and release of epinephrine and norepinephrine. The response causes blood to be diverted from the internal organs to the skeletal musculature. A person in a fight, flight, or freeze state often acts with little or no reflective thinking to preserve his or her life or the lives of others. Memories of these episodes may be unreliable because an individual is often on "automatic pilot." Episodes of autonomic arousal in response to danger or threat are normal. However, some people experience anxiety in ways that interfere with their ability to function, complete everyday tasks, and respond appropriately to danger. A person in a "freeze" state has unconsciously "reasoned" that there is no way out and "dissociates" during the episode as a survival mechanism. Avalanches, storms, rockfalls, climbing, white-water accidents, and many other untoward events can lead to the death or serious injury of one or more group members. Most individuals who venture into the wilderness usually do not anticipate someone being seriously injured or killed. However, the extreme nature and risk of certain expeditions, combined with individuals venturing out unprepared and expecting to be readily in communication and rescued, can lead to extreme consequences, including death. Most persons who have taken wilderness first-aid courses do not enter the wilderness expecting the worst, even though there are many descriptions of outdoor trips that have involved crises.

The U.S. culture is extremely avoidant of death and grief. Thus, when a crisis or tragedy occurs, it is almost always an emotional shock. When one watches a climbing partner fall to his death or a friend drown when his kayak flips in white water, the usual reaction is horror, dread, and disbelief. The situation is overwhelming, and survivors or witnesses of these types of tragedies often describe a sense of unreality associated with the horrible event. People who are involved in tragedies often need to replay the event to put it into some type of meaningful personal context. People who are relatively intact psychologically and who have good social support may be able mentally to process a trauma successfully without long-term mental health problems. As the size of a disaster grows, and its impact on a

TABLE 52-5 Psychotropic Drug Types and Side Effects

Class	Type of Disorder and Symptoms Treated	Withdrawal: Nonadherence on Wilderness Program	Typical Side Effects	Intoxication
Stimulants	Attention-deficit/hyperactivity disorder	Return of symptoms	Anxiety Anorexia	Hypertension, palpitations, insomnia, psychosis
Antidepressants	Depression Anxiety Obsessive-compulsive disorder	Return of symptoms Paresthesias for paroxetine	Nausea, dry mouth, somnolence, or insomnia	Neuromuscular excitation, autonomic stimulation, changed mental state
Atypical antipsychotics	Psychotic disorders Adjunctive for mood disorders	Return of symptoms	Weight gain, somnolence, hypotension	Extrapyramidal side effects
Typical antipsychotics	Psychotic disorders	Return of symptoms	EPS, tachycardia, rash, lactation, leukopenia, NMS	Extrapyramidal side effects
Mood stabilizers	Bipolar disorder Mood disorder	Return of symptoms	Variable	Lithium toxicity
Anxiolytics: Benzodiazepines	Anxiety Sleep	Return of symptoms Physiologic withdrawal	Sedation	Sedation
Somnolents	Insomnia	Return of symptoms Physiologic withdrawal	Sedation	Sedation

EPS, Extrapyramidal symptoms; *NMS,* neuroleptic malignant syndrome.

person or a community's support networks increases, the likelihood of long-term mental health problems grows. People who handle trauma by psychological avoidance and casting blame may have poorer outcomes.

In response to natural disasters, some individuals develop trauma and stressor-related disorders, the new category for these conditions in DSM-V. They are at risk specifically for acute stress disorder (ASD) or posttraumatic stress disorder (PTSD). Estimates of the frequency of these disorders after disasters (man-made or natural) vary from 2% to more than 50%. The symptom clusters include intrusive recollections, avoidance of stimuli, negative alteration in mood and cognitions, and hyperarousal. The individual may attempt to avoid reminders of the traumatic event and may have greatly increased anxiety during exposure to such reminders. The person may be flooded with uncontrollable memories, including bad dreams and flashbacks (i.e., feeling and behaving as if the trauma were happening again). The person may feel a sense of emotional detachment from others. He may have difficulty concentrating and sleeping, may feel restless, and may be easily startled. Symptoms can begin immediately after a disaster. If they persist for more than 3 days but for less than 1 month, the disorder is called ASD; if the symptoms are present for more than 1 month, the disorder is called PTSD. For most people, symptoms diminish as time passes, although symptoms usually intensify around the 1-year anniversary of the tragedy.

Symptoms may become chronic and require ongoing psychological and psychiatric care. The presence of ASD and PTSD in first responders and expedition teams is becoming more readily recognized and discussed.

There is a growing evidence base for mental health interventions during the immediate aftermath of a disaster. Specifically, in the first 4 days after an event, psychological first aid, especially when combined with psycho-education about psychological trauma, normalization of current symptoms, and social support, is being recommended as being of "some benefit" by many trauma specialists. In addition, there is reputed to be significant benefit in delivering trauma-informed cognitive behavioral therapy (CBT) in days 4 to 30 after the event. Previously, many believed that psychological debriefing was beneficial. All available evidence suggests that is not helpful and, for many, harmful.

The immediate and first response to a wilderness disaster should emphasize ensuring safety and security of the survivors, including provision of food, water, warmth, and shelter. Second, efforts should be made to calm everyone who was involved in the disaster. Survivors should be accurately informed about what has happened and about what rescue efforts are underway. It is important to advise survivors about the status of others who were involved in the disaster and about the safety of family and friends. Communication with relatives should be facilitated as soon as possible. Victims should be provided with a calm setting to decrease arousal as much as possible. Encouraging deep breathing and muscle relaxation may be helpful. Reassurance should be provided regarding any distortions that a survivor may experience, such as feelings of weakness or guilt or catastrophic perceptions that the world is a malevolent and dangerous place.

Medication use continues to be controversial. There is no evidence that benzodiazepines or atypical antipsychotics are helpful in the immediate aftermath of a disaster. Using psychotropic medications during the immediate aftermath may not be necessary and may even be harmful, particularly if the sedative properties interfere with survival behavior or motivation (Table 52-5). This is not true, however, for opioids in the treatment of pain because opioids may block the memory and traumatic response. In addition, trials with β-adrenergic blockers (e.g., propranolol) and α-adrenergic agonists (e.g., clonidine), which decrease tachycardia, may assist in decreasing the likelihood of developing PTSD after trauma or disaster.

For formal PTSD treatment, trauma-focused psychotherapy that includes the components of exposure and cognitive restructuring is beneficial. In addition, using SSRIs (e.g., paroxetine, fluoxetine) as well as SNRIs (e.g., venlafaxine) should be considered once an individual has developed a full PTSD disorder. These agents should not be used immediately after the disaster as a preventive treatment unless the individual has a history of recurrent major depression. It is expected that survivors will be emotional after a disaster, but the experience of those emotions may be naturally adaptive, so artificially dampening them with medications can be counterproductive. Survivors should be approached with the expectation that they will recover psychologically, and they should be reassured about their future mental health. Further interventions are required in the initial aftermath of a disaster.

Promoting a sense of self-effectiveness and self-worth is recognized as an important component of recovery efforts, and this includes providing survivors with continuing information about the effects of the disaster and the means to contact other survivors. Making psychological intervention accessible can be very useful during the weeks and months after a disaster. Cognitive behavioral treatments have also demonstrated considerable value for survivors of disasters. Finally, promoting a sense of connectedness with the broader community and instilling a sense of hope (i.e., avoiding social withdrawal and hopelessness) can be crucial for people who are recovering from a disaster. (See http://www.nctsn.org/sites/default/files/pfa/english/1-psyfirstaid_final_complete_manual.pdf.)

SURVIVAL PSYCHOLOGY

It is not uncommon for people to become lost in the wilderness. The best prevention is a proper orientation to the surroundings and only leaving the group if one is carrying appropriate gear (food, water, water purifier, emergency blanket, warm clothes, matches). Human spatial orientation abilities are limited; we often function under the illusion that we know exactly where we are, when that is generally not the case. People rationalize disorientation for too long before they become convinced that they are completely lost. At that point, they may experience intense fear, panic, and disorientation unique to this type of dilemma. Lost people have been shown to tend to walk in circles. Even people with considerable experience in the wilderness may demonstrate inexplicable behaviors, such as abandoning provisioned backpacks or other potentially valuable items. Some have even removed clothing, which increased their exposure to the elements.

After becoming lost, some persons develop a strategy and carry it through; however, when this plan fails, these individuals may abandon hope and resign themselves to fate. The best preparation for getting lost is to discuss the possibility of an individual becoming lost and making sure that whenever individuals wander away from the group, they have the necessary survival supplies in their pack. Ironically, children who are less than 6 years old fare better when they are lost compared with older children and adults. The explanation for this paradox might be that younger children act on instinct and seek shelter when cold and water when thirsty, whereas older children and adults panic and sometimes overlook obvious and simple means to help them to survive. In groups who become lost, good leadership is often a key component of the group's survival. Perhaps the most famous survival story is that of Ernest Shackleton's ill-fated 1914 voyage to reach the South Pole. His ship, the Endurance, broke up in the ice. Under his leadership, the group survived months of incredible hardship. Against all odds, all 27 men survived.

CHILDREN AND ADOLESCENTS IN THE WILDERNESS

As technology continues its rapid infiltration into the lives of children, there is a growing movement to counteract this trend and have children spend more time in natural settings. As with adults, the time that children spend in nature has a growing evidence base for their current well-being as well as their development. In addition, many children and adolescents who fail traditional mental health treatment are referred for wilderness or other nature-based therapies. Although imprecise in definitions, the core components include (1) the wilderness as a restorative environment; (2) basic outdoor life, incorporating sequenced and intentional tasks and challenges; (3) individualized, structured therapeutic work; and (4) establishment of a supportive peer group and provision of group therapy. Anecdotal reports are that wilderness therapy is quite successful for individuals and families deemed recalcitrant to treatment. Adventure, wilderness, and other nature-based psychospiritual expeditions are also becoming more common among the medical profession as a way to counter burnout.

Very little is known about the mental health of children in the wilderness and on expeditions. The principles espoused for adults are somewhat transferrable to young individuals. The incidence of psychiatric disorders in children and adolescents is currently 22%.[10] In addition, one must remember that most adult psychiatric disorders begin in childhood. For example, the average age of first onset for an anxiety disorder is 6 years; for a behavioral disorder, 11 years; for mood disorder, 13; and for substance use disorder, 16. Anxiety disorders were most common (31.9%), followed by behavior disorders (19.1%), mood disorders (14.3%), and substance use disorders (11.4%), with approximately 40% of participants with one class of disorder also meeting criteria for another class of lifetime disorder. The overall prevalence of disorders with severe impairment and/or distress was 22.2% (11.2% with mood disorders, 8.3% with anxiety disorders, and 9.6% behavior disorders).

LOOKING TOWARD THE FUTURE

With rates of work-related and urban life–induced burnout on the rise, the time spent in the wilderness diminishing, and the level of "self-determination" limited by changes in the structure of health care delivery, it may be instructive for the field of wilderness medicine to evaluate the positive mental benefits of expeditions and other outdoor experiences rather than hoping that no mental health issues arise on these journeys. Integrated nature-based programs are designed to bring ecotherapy and adventure therapy to the medical and health care professions. Further education and training on adapting and delivering psychological first aid is a next step for the field. Also, a study of mental health issues in wilderness participants, both before and after wilderness contact, would be a welcomed step. Although mental health issues can occur in the wilderness, it is much more likely that positive mental health enhancement occurs that balances the challenges and negative occurrences.

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Complete references used in this text are available online at expertconsult.inkling.com.

CHAPTER 53

Chronic Diseases and Wilderness Activities

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The focus of this chapter is the prevention and treatment of complications caused by chronic medical conditions during wilderness activities. For this purpose, we define *wilderness activities* as any excursion into an outdoor environment where usual medical services are not available, which include outings ranging from a day to several months. Wilderness activities may occur close to home, in the country of residence, in foreign developed countries, or in foreign developing countries. Issues relating to chronic medical conditions during wilderness activities are important to primary care and specialty physicians

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because patients often seek advice on whether a certain wilderness trip or activity is appropriate, given their medical condition, and how they should manage their disease while in the wilderness.

Medical problems may occur during wilderness activities as a result of an acute injury, acquired infectious illness, or environmentally caused illness (heat, cold, or high-altitude illness). A preexisting medical condition may also cause complications during a trip or activity or may predispose the patient to environmentally-caused illness. Some studies that report the epidemiology of medical problems in the wilderness show that most are caused by traumatic injuries, and that most deaths result from falls or drowning.[58,82,147] A review of the responses of emergency medical services in U.S. National Parks, however, showed an equal number of medical events as trauma events.[44] Cardiac disease is the most common medical illness that causes death in the wilderness. Cardiac disease, asthma, and diabetes are also reported as causes of medical illness during wilderness activities. Medical illness caused by environmental exposure, such as heat, cold, or altitude illness, depends on the environment.[58,120]

The first issue to consider in evaluating a patient with a chronic medical condition who is planning a wilderness trip or activity is how the environment will affect the patient and his or her medical condition. In a wilderness environment, patients with chronic medical conditions will potentially be exposed to heat, cold, high altitude, and a greater level of exercise. Wilderness activities occur in remote areas where medical care is impossible to obtain. These factors require evaluation in relation to the patient's chronic medical condition. First, the physician and patient need to address whether the planned wilderness activity is appropriate for the patient. Second, the physician should provide advice to the patient on how to manage his or her medical condition in the remote wilderness environment.

Deciding on whether a potential wilderness activity is appropriate for a patient is a shared responsibility between the physician and patient. "Wilderness" implies that medical care will be remote, so if a major medical problem arises, the consequence may be severe or even include risk for death. Risk is an inherent part of any wilderness activity, so persons who venture into the wilderness should make conscious decisions about acceptable levels of risk and how they can exercise good judgment to reduce those risks. The same process is used with regard to a chronic medical condition: acknowledging risk, deciding whether it is acceptable, and changing behavior to minimize risk. Although it is important for the physician to help the patient understand issues and to make medically responsible recommendations, the physician should recognize patient autonomy and leave most decisions to the patient.

CONSIDERATIONS FOR WILDERNESS TRAVEL

INCREASED PHYSICAL ACTIVITY

Although some wilderness activities are of the sedentary variety (e.g., car-based safaris), many other activities involve physical exertion. The level of exertion may be comparable with that which the person already performs on a regular basis, but in some cases may involve more activity than the person's typical baseline. As a result, all wilderness travelers with underlying medical problems must consider the level of activity on their planned trip and whether they are capable of doing the work necessary for that activity.

Many commercial outfitters label trips with a difficulty rating that takes into account the level of exertion, remoteness, and exposure to high altitude. Ratings generally include easy, moderate, and strenuous categories. This can be useful to the physician evaluating the patient before the trip, especially if the physician is not familiar with the environment or the activity that will be pursued; however, the physician must be aware that these ratings are not based on hard criteria. What constitutes a "moderate" trip for one company may be labeled as a "strenuous" trip by another. Some companies that offer adventure trips require potential clients to complete a medical history questionnaire and may also have a consultant physician review the questionnaire and provide advice to select clients.[92,93]

Erb[59] has developed a scale of trip difficulty and correlated this with objective parameters of exercise capacity required to complete the type of activity (see Table 93-2, Chapter 93). "Extreme-performance ventures" are the most physically demanding and require above-average exercise capacity for participation. This category would include activities such as mountaineering at high altitude and alpine climbing. "High-performance ventures," such as high-altitude trekking or hunting or jungle trekking, are the next most demanding category of activities and require an average exercise capacity. The third level of wilderness trips, "recreational activities," requires just below-average exercise capacity and includes activities such as hiking on mild-moderate terrain in a variety of environments. The last category of wilderness trips is "therapeutic activities," which may be appropriate for persons with chronic cardiovascular or pulmonary disease that limits activity. Even though Erb defines categories of wilderness trips and assigns objective exercise capacity parameters to them, formal cardiac stress testing or cardiopulmonary exercise testing is not required in most cases to determine if a patient has adequate exercise capacity to complete a planned wilderness trip. The most important predictor is successful completion by the participant of similar activities in the past. Still, formal cardiopulmonary exercise testing may be useful in patients with chronic medical conditions to objectively define maximum exercise capacity to select the appropriate level of a wilderness trip. This helps ensure a more enjoyable and safe experience for patients and their trip partners.

TRIP PREPARATION

Persons with chronic medical conditions have an obligation to inform the other people involved in a trip, or the trip leader in the case of a commercial trip, about medical issues that might affect their ability to complete the planned itinerary. This is important because chronic medical conditions may influence not only the affected person but also other members of the party. Notification will allow trip leaders or other participants to anticipate situations in which the underlying medical problem may worsen, facilitate monitoring of the person's condition, and allow preparations to alter the group's plans or arrange an evacuation when serious problems arise. It is also important that everyone in the group accept the limitations of a chronic medical condition and make conservative decisions during the trip that prevent the persons from overexerting themselves.

Pretrip preparation includes identifying the closest medical facility and resources available should evacuation become necessary. Rescue insurance and repatriation medical insurance are available and should be strongly considered by all participants. Persons with chronic conditions should carry an adequate supply of medications and keep medications with them at all times to ensure that their drugs do not become lost (e.g., pack in carry-on baggage rather than checked baggage). Patients should travel with a list of their medications and a concise list of their medical issues, including a baseline electrocardiogram for patients with cardiac problems, and contact information for their regular health care providers. They must also recognize that in remote settings, they may encounter facilities not equipped for, or physicians lacking experience dealing with, complex medical problems.

ENVIRONMENTAL EXTREMES AND CHRONIC MEDICAL CONDITIONS

Thermoregulation, hypothermia, heat illnesses, and high altitude are extensively reviewed in earlier chapters. For the purpose of understanding how these environmental extremes might affect chronic medical conditions, some key points are worth emphasizing.

COLD

Exposure to cold causes peripheral vasoconstriction, increased systemic blood pressure, and tachycardia and, as a result, increases myocardial oxygen consumption. When core body temperature drops below normal, shivering ensues as a metabolically demanding activity that occurs as a means to generate heat and defend against further drops in temperature. Although such physiologic responses are important for protecting the individual, learned behavioral responses, such as avoiding moisture or donning dry clothing, also play an important role during cold exposure. Even with appropriate preparation, however, some chronic medical conditions still increase the risk for accidental hypothermia or frostbite. Alternatively, cold conditions may exacerbate the underlying chronic medical condition (see Chapters 7 to 11). Situations in which such problems may arise include the following:

- Cardiovascular disease (angina or congestive heart failure may occur because of increased systemic vascular resistance, afterload, and myocardial oxygen demand)
- Hypertension (exacerbated by cold-induced peripheral vasoconstriction)
- Peripheral arterial disease (increased risk for frostbite)
- Raynaud's phenomenon (increased frequency of Raynaud's attacks and potentially increased risk for frostbite)
- Asthma (cold air– and exercise-induced bronchospasm)
- Older adults (impaired thermogenesis)
- Endocrine disease (impaired thermogenesis)
- Iron deficiency (impaired thermogenesis)
- Neuromuscular disease (impaired shivering thermogenesis)
- Malnutrition (decreased body surface area/mass ratio increases risk for heat loss and hypothermia)
- Diabetes (hypoglycemia may impair thermogenesis; peripheral neuropathy may predispose to cold injury)

HEAT

Peripheral vasodilation is a primary response to heat exposure that leads to sweat production and evaporative heat loss. In high-humidity conditions, however, heat loss from sweating is diminished. Radiant heat loss has no usefulness when environmental temperature exceeds skin temperature. Unlike cold adaptation, heat adaptation depends more on physiologic acclimatization than on behavior. Prior heat exposure reduces the skin temperature at which vasodilation and sweating occur, thus initiating cooling earlier. Physical training improves heat tolerance through the same mechanisms and improves efficiency of the cardiovascular system. Poor physical fitness, obesity, lack of heat acclimatization, and dehydration decrease heat tolerance. Chronic medical conditions increase the risk for heatstroke, whereas increased temperature may exacerbate chronic medical conditions, as follows:

- Diabetes (the disease may predispose to dehydration, whereas dehydration may increase the risk for hyperglycemia)
- Cardiovascular disease (limited cardiac output in the face of peripheral vasodilation decreases heat tolerance and may lead to syncope or presyncopal symptoms)
- Skin disease (inadequate sweating predisposes to heatstroke)
- Anticholinergic drugs or diuretics (decrease heat tolerance)
- Sickle cell trait (increased risk for heatstroke and rhabdomyolysis with physical exertion)
- Obesity (low ratio of body surface to mass increases risk for hyperthermia)

Susceptible individuals can improve heat tolerance through a program of heat acclimatization before or during their trip.[7] Before a wilderness trip to a hot environment, exercising daily in the heat for limited periods of at least 1 hour's duration for at least several days improves heat tolerance. If such pretrip training is not feasible, the individual should restrict exertion to limited periods during cool parts of the day for the first week of the trip. Regardless of the acclimatization program, once a trip commences, individuals should maintain volume status through adequate intake of water or electrolyte drinks, with copious clear urine output being a good indicator of adequate hydration status. Individuals engaged in prolonged bouts of exercise (lasting several hours or more) should supplement water intake with meals or salty foods to maintain electrolyte balance and prevent hyponatremia (see Chapters 12 and 13).

HIGH ALTITUDE

The primary physiologic insult at high altitude is hypobaric hypoxia. This leads to a decrease in the partial pressure of inspired oxygen (P_{IO_2}), which in turn leads to drops in alveolar and arterial oxygen tension (P_{O_2}). For persons with normal gas exchange, significant decreases in arterial oxygen saturation (Sa_{O_2}) will not occur on acute exposure until they are higher than approximately 3000 m (9843 feet), because of the sigmoidal shape of the oxyhemoglobin dissociation curve. In acclimatized individuals, Sa_{O_2} decreases below 90% at elevations above approximately 3500 m (11,483 feet). Any individual with gas exchange abnormalities or right-to-left shunts at sea level may become very hypoxemic at significantly lower elevations, whereas individuals with diseases exacerbated by hypoxia (e.g., severe coronary artery disease or heart failure) may have significant problems in this environment as well (see Chapters 1 to 3).

Regardless of their underlying health status, all individuals undergo physiologic responses that facilitate acclimatization to hypobaric hypoxia. Individuals with certain chronic conditions, however, may either fail to mount these responses or suffer adverse consequences as a result of the responses. For example, low arterial P_{O_2} triggers an increase in minute ventilation, referred to as the *hypoxic ventilatory response*. This response, which occurs within hours of ascent to altitudes greater than 2000 m (6562 feet) and varies in magnitude between individuals, helps to raise alveolar and arterial P_{O_2} and may affect susceptibility to altitude illness. Persons with chronic diseases that restrict ventilatory capacity (e.g., extremely severe COPD, obesity hypoventilation syndrome, neuromuscular diseases with respiratory system involvement) may be unable to raise their minute ventilation and may experience severe hypoxemia and exercise intolerance.

Low alveolar P_{O_2} also trigger hypoxic pulmonary vasoconstriction and a rise in pulmonary artery pressure. The response is seen above 2000 m (6562 feet) in elevation and varies in magnitude between individuals. Most people tolerate the rise in pulmonary artery pressure, but individuals with underlying pulmonary hypertension or right-to-left shunts may be at risk for high-altitude pulmonary edema, worsening right-sided heart function, or increased hypoxemia.

Important cardiovascular responses also occur that may not be tolerated well by all individuals. Increased sympathetic tone occurs acutely after ascent to high altitude and increases heart rate and blood pressure. Heart rate and blood pressure gradually decrease over several days at high altitude, but remain higher than sea level baseline values for the duration of stay at high altitude. Despite the increase in sympathetic tone, most persons with mild to moderate cardiovascular disease do well after ascent to moderate altitudes of approximately 2500 m (8202 feet),[122,170] although individuals with unstable angina, severe cardiomyopathy, or poorly controlled hypertension might not tolerate such changes.

Increased erythropoiesis is a long-term adaptation to high altitude, requiring weeks to complete, that increases red blood cell (RBC) mass and oxygen-carrying capacity of the blood. Persons with severe iron deficiency or chronic marrow suppression are unable to increase erythropoiesis to complete the normal long-term hematologic acclimatization.

The following chronic diseases may worsen at high altitude or predispose to impaired tolerance of hypobaric hypoxia:

- Chronic obstructive pulmonary disease (COPD; inadequate ventilatory acclimatization, worsened hypoxemia)
- Asthma (potential for exacerbation from hyperventilation of cold, dry air)
- Cardiac disease (cardiac ischemia or congestive heart failure precipitated by hypoxia)
- Pulmonary hypertension (hypoxia precipitates a rise in pulmonary artery pressure that may predispose to high-altitude pulmonary edema or worsening right-sided heart function)

- Morbid obesity (hypoventilation increases risk for hypoxemia and high-altitude illness)
- Neuromuscular disease (respiratory muscle involvement impairs ventilatory responses and acclimatization)
- Hematologic disease (hypoxia may worsen sickle cell disease and thalassemia; anemia or bone marrow suppression can impair erythropoietic responses)

CHRONIC MEDICAL CONDITIONS AND WILDERNESS TRAVEL

ASTHMA

Asthma is a disorder of reversible airflow limitation marked by the presence of cough, wheezing, chest tightness, and shortness of breath. Affected individuals can have long, symptom-free periods punctuated by exacerbations and worsening symptoms often triggered by stimuli such as respiratory infections, exercise, or allergen exposures. The topic of travel in the wilderness with asthma has been recently reviewed in detail,[50] and the key points are summarized here.

Despite the high prevalence of asthma in the general population, and the subsequent high likelihood that many wilderness travelers have this disorder, few data are available as to how wilderness travel affects these patients. In the only prospective study of asthma patients engaged in wilderness travel, Golan and colleagues[80] studied 203 patients with mild to moderate asthma presenting to a travel clinic before departure; 43% reported an exacerbation during their trip, 20% reported worsening asthma control, and 16% reported the worst exacerbation of their life. The leading risk factors for exacerbations during the trips were frequent rescue inhaler use (>3 times per week) before the trip and participation in intense physical activity during the trip. Pretrip exercise testing with pretest and post-test spirometry was not useful in predicting which patients would develop an exacerbation.

Because the potential for exacerbations exists, and pretrip identification of patients likely to experience such problems is difficult, all asthma patients should strongly consider pretravel evaluation to ensure that their disease is under adequate control at the time of their trip and to establish the means to monitor for and respond to exacerbations. This is not necessary before every daylong or weekend excursion, but should take place before any long trips, particularly those to international or remote destinations.

Evaluation of a patient with asthma planning a wilderness activity should begin with the basic approach used in any patient with asthma: monitoring of symptoms, pharmacologic therapy, avoidance of triggers, and patient education. The first part of this evaluation is to review the state of the patient's current symptoms and determine if the patient is receiving the appropriate pharmacologic regimen, because appropriate treatment at baseline will help prevent attacks during the sojourn. The U.S. National Institutes of Health (NIH) *Guidelines for the Diagnosis and Management of Asthma*[145] provides definitions of categories of severity for patients with asthma and the appropriate treatment (Box 53-1 and Table 53-1). These guidelines can inform this evaluation and help the evaluator to determine whether it is necessary to escalate therapy before the planned trip. For example, a patient who is only taking short-acting β-adrenergic agonists but has been using them more frequently than in the past may consider adding an inhaled corticosteroid for improved control. Ideally, this change should be made far enough in advance of the trip to evaluate the response to therapy. For patients with marked worsening of their baseline control, strong consideration should be given to postponing the planned trip.

Because exercise may be a primary trigger for asthma on a wilderness trip, consideration can be given to adding the leukotriene receptor blocker montelukast to the patient's controller regimen, because this has been shown to be effective adjunctive therapy for exercise-induced asthma.[121] Patients can also use short-acting β-agonists before and during exercise.[140] Further details about management of exercise-induced bronchoconstriction are available in recently published guidelines.[153]

BOX 53-1 Classification of Asthma Severity in Patients 12 Years of Age and Older

Mild Intermittent (Step 1 Treatment)
Symptoms <2 days/wk
Nights with symptoms: <2/mo
Short-acting β-agonist use <2 days/wk
No interference with normal activity
Normal FEV_1 between exacerbations
FEV_1 >80% predicted
FEV_1/FVC normal

Mild Persistent (Step 2 Treatment)
Symptoms >2 times a wk but less than daily
Nights with symptoms: 3-4/mo
Short-acting β-agonist use >2 days/wk but not daily and not more than once daily
Minor limitation of normal activity
FEV_1 >80% predicted
FEV_1/FVC normal

Moderate Persistent (Step 3 Treatment)
Daily symptoms, exacerbations affect activity
Daily use of short-acting β-agonist
Nights with symptoms: >1 wk but not nightly
Some limitation of normal activity
FEV_1 >60% but <80%
FEV_1/FVC reduced 5%

Severe Persistent (Step 4 or 5 Treatment)
Continual symptoms throughout the day, limited physical activity
Frequent exacerbations
Extremely limited activities
Nights with symptoms: often 7/wk
Short-acting β-agonist use several times per day
FEV_1 <60% predicted
FEV_1/FVC reduced >5%

Modified from National Asthma Education and Prevention Program: Expert Panel Report 3 (EPR-3): Guidelines for the diagnosis and management of asthma—Summary report 2007, *J Allergy Clin Immunol* 120:S94, 2007.
FEV_1, Forced expiratory volume in 1 second; *FVC*, forced vital capacity.

As noted previously, many exacerbations are triggered by identifiable factors such as allergens, respiratory infections, irritants, chemicals, exercise, and emotional stress. The pretravel assessment also provides an opportunity to review the triggers that could contribute to worsening control during the trip. Patients should realize, however, that on certain wilderness trips, it might be difficult to avoid certain triggers, such as breathing cold, dry air during exercise on a mountaineering expedition. In such cases, effort can be made to minimize exposure by, for example, wearing and breathing through a balaclava or pretreating with asthma medications before exertion. Asthma patients should also anticipate exposure to triggers that might not be an issue at home. For example, adventure travel often requires transit through major international cites, such as Bangkok, Thailand, or Kathmandu, Nepal, where local air quality may be poor and contribute to worsening control before the actual adventure activity begins.

Another important part of the pretravel assessment is devising a program for objectively monitoring disease status during the trip. Asthma patients usually monitor asthma control using *peak expiratory flow* (PEF), an objective parameter measured after the patient inhales to total lung capacity and then forcefully exhales into a peak flow meter. Patients establish their baseline peak flows when their disease is under good control by performing the maneuver several times a day and recording the results in a diary. The highest measured PEF becomes the baseline for the patient, and comparison of further measurements with that baseline can be used to identify disease exacerbation and therefore escalate therapy. NIH guidelines recommend using a zone scheme for categorizing results of PEF: green is a PEF greater than 80% of personal best, yellow is a PEF 50% to 80% of

TABLE 53-1 Stepwise Approach to Managing Asthma in Patients 12 Years of Age and Older

Step	Pharmacotherapy
Intermittent Asthma	
1	Short-acting β_2-agonist as needed
Persistent Asthma	
2	*Preferred:* Low-dose inhaled corticosteroid *Alternative:* Cromolyn, leukotriene receptor antagonist, nedocromil, or theophylline
3	*Preferred:* Low-dose inhaled corticosteroid plus long-acting β_2-agonist OR medium-dose inhaled corticosteroid *Alternative:* Low-dose inhaled corticosteroid plus either leukotriene receptor antagonist or theophylline
4	*Preferred:* Medium-dose inhaled corticosteroid plus long-acting β_2-agonist *Alterative:* Medium-dose inhaled corticosteroid plus either leukotriene receptor antagonist or theophylline
5	*Preferred:* High-dose inhaled corticosteroid plus long-acting β_2-agonist AND consider omalizumab for patients who have allergies
6	*Preferred:* High-dose inhaled corticosteroid plus long-acting β_2-agonist plus oral corticosteroid AND consider omalizumab for patients who have allergies

Modified from National Asthma Education and Prevention Program: Expert Panel Report 3 (EPR-3): Guidelines for the diagnosis and management of asthma—Summary report 2007, *J Allergy Clin Immunol* 120:S94, 2007.
Each step: Patient education, environmental control, and management of comorbidities.
Steps 2-4: Consider subcutaneous allergen immunotherapy for patients who have allergic asthma.
Quick relief of symptoms for all patients: Short acting β_2-agonist as needed for symptoms. Intensity of treatment depends on severity of symptoms: up to three treatments at 20-minute intervals as needed. Short course of oral systemic corticosteroids may be needed.
Use of short-acting β_2-agonist more than 2 days/wk for symptom relief (not prevention of exercise-induced bronchospasm) generally indicates inadequate control and the need to step up treatment.
Step down, if possible, if asthma is well controlled at least 3 months.

personal best, and red is a PEF less than 50% of personal best.[145] A PEF in the green zone indicates the patient should continue maintenance medications, whereas a PEF in the yellow or red zone requires adjustments in treatment according to a predetermined plan, as well as seeking evaluation by a physician.

This type of self-monitoring could easily be used on a wilderness trip. For example, a predetermined treatment plan for PEF in the yellow zone associated with symptoms of asthma might include acute treatment with an inhaled short-acting β-agonist followed by reevaluation in 1 hour, and if not improved to the green zone, initiation of oral corticosteroids and an increase in the dose of inhaled corticosteroids. A treatment plan for PEF in the red zone associated with symptoms of an asthma exacerbation would specify immediate therapy with inhaled β-agonists, to be repeated at frequent intervals, and initiating an oral corticosteroid. Persistent symptoms associated with PEF in the red zone suggest that evacuation from the wilderness environment should be initiated.

An alternative to peak-flow monitoring is the PiKo-1, which measures both PEF and forced expiratory volume in 1 second (FEV_1). This electronic device has a 2-year battery life and is small and easy to pack.[173] Patients should be aware that the PiKo-1 and PEF meters, particularly variable orifice peak flow meters, may generate readings significantly lower than actual PEF in cold temperatures or at high altitude.[156,162] If concern exists about such problems on a trip, the individual should emphasize the trends in the measured PEF rather than the absolute values.

Finally, all patients should travel with an adequate supply of rescue inhalers (i.e., short-acting bronchodilators) and a supply of prednisone to treat an exacerbation during the trip. If traveling in a cold environment, patients should be instructed to keep their inhalers warm, because cold exposure can decrease their effectiveness.[164] Extreme warm temperatures, on the other hand, do not appear to affect inhaler function.[96]

Beyond these general guidelines, two specific activities warrant further attention in the asthma patient: high-altitude travel and diving. The effect of high-altitude exposure on asthmatic patients has not been extensively studied, but available evidence suggests that patients with mild to moderate disease that is well controlled at the time of their trip, can tolerate significant altitude exposure. Several studies have shown that exposure to elevations as high as 5000 m (16,404 feet) is associated with decreased bronchial hyperresponsiveness.[2,39] In terms of clinical responses, a small study of patients with mild to moderate disease climbing Mt Kilimanjaro found a non–statistically significant improvement in PEF between 2700 and 4700 m (8858 and 15,420 feet), no difference in the incidence of acute mountain sickness or summit success compared with nonasthmatic patients, and no evidence of exacerbations during the excursion.[193] Another field expedition showed that well-controlled asthmatic patients with mild disease could trek as high as 6400 m (21,000 feet) without increased symptoms or need for medications.[98] Although these studies suggest high-altitude travel is safe, patient-specific factors must also be taken into consideration during the pretravel evaluation. Patients whose disease is triggered by breathing cold, dry air may have difficulty during mountaineering or ski excursions that include significant exposure to such conditions. Epidemiologic evidence suggests, for example, that asthma incidence is increased among cross-country skiers and ski mountaineers, athletes whose activities entail high levels of minute ventilation in cold, dry environments.[53,116] Patients whose disease is triggered primarily by allergens, on the other hand, may actually fare better at altitude,[203] where the number of dust mites decreases with increasing elevation.

The primary concerns with scuba diving in patients with asthma are the potential risk for increased airway hyperresponsiveness,[38,187] and the fact that active airflow obstruction could lead to air trapping and significantly increase the risk for pulmonary barotrauma with changes in barometric pressure on ascent back to the surface. As discussed further in Chapter 71, asthma was previously considered an absolute contraindication to diving but is now permitted provided the patient is (1) an asymptomatic adult with a childhood history of asthma, (2) has well-controlled disease with known triggers, (3) has normal pulmonary function tests with a less than 20% change in PEF after exercise, and (4) no evidence of cold- or exercise-induced bronchospasm or symptoms provoked by emotion.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

A syndrome of progressive airflow limitation, COPD is caused by chronic inflammation of the airways and lung parenchyma. The prevalence of COPD is approximately 4 to 7 per 1000 persons in developed countries.[8] The extent to which COPD limits an individual's planned wilderness activities is a function of disease severity, assessment of which can be made using criteria specified by the Global Initiative for Chronic Obstructive Lung Disease (GOLD).[79]

According to the most recent version of these guidelines, assessment of disease severity takes into account three factors: the decrement in the patient's postbronchodilator FEV_1 from a forced vital capacity (FVC) maneuver (Table 53-2), the severity of their symptoms, and the number of exacerbations per year. Based on the patient's status with regard to each of these factors, the patient is placed in one of four categories of disease severity (Figure 53-1). Category A patients will probably do well with wilderness activity, provided the planned activity does not far exceed their usual exercise tolerance. Patients in category B, C, or D, however, warrant careful evaluation to determine their suitability for the planned activity. Cardiopulmonary exercise testing should be considered to determine exercise capacity, which can then be compared with the expected level of exertion on the planned trip.[6] Of note, patients whose disease may not appear too severe based on their pulmonary function test results may have significant air trapping during exercise that impairs

TABLE 53-2 GOLD Classification of Severity of Airflow Obstruction in COPD

Gold Category	Severity	Degree of Airflow Obstruction*
1	Mild	FEV_1 ≥80% predicted
2	Moderate	50%≤ FEV_1 <80% predicted
3	Severe	30%≤ FEV_1 <50% predicted
4	Very severe	FEV_1 <30% predicted

Data from Global Initiative for Chronic Obstructive Lung Disease (GOLD): Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: Updated 2014. http://www.goldcopd.org/uploads/users/files/GOLD_Report_2014_Jun11.pdf. From the Global Strategy for Diagnosis, Management and Prevention of COPD 2016.
COPD, Chronic obstructive pulmonary disease; *FEV_1,* forced expiratory volume in 1 second, reported as percentage predicted; *FVC,* forced vital capacity.
*Values are based on FEV_1 after bronchodilator. Categorization applies to patients with FEV_1/FVC <0.7.

TABLE 53-3 Choosing Initial Pharmacologic Regimen for COPD Patients Based on Disease Severity

Severity Class	First-Line Treatment Regimen
A	Inhaled short-acting anticholinergic or short-acting β-agonist as needed
B	Inhaled long-acting anticholinergic or long-acting β-agonist
C	Inhaled corticosteroid *plus* long-acting β-agonist *or* long-acting anticholinergic
D	Inhaled corticosteroid *plus* long-acting β-agonist *or* long-acting anticholinergic

Long-acting anticholinergic: tiotropium.
Long-acting β-agonist: formoterol or salmeterol.
Short-acting anticholinergic: ipratropium.
Short-acting β-agonist: albuterol or levalbuterol.

pulmonary mechanics and leads to significant exercise limitation.[151] Patients with carbon dioxide retention or right-sided heart failure should be advised against any wilderness activity in which exertion above their baseline level of tolerance is expected, or if the planned trip is to a higher altitude than their current residence. Such patients may, however, tolerate car or horse-led activities, such as safaris or fishing, at low elevation that do not require much physical exertion. In considering suitability for travel, it is important to remember that many patients with COPD have comorbid conditions, such as coronary artery disease, that may also affect their tolerance for a planned activity and that also require attention in the pretravel assessment.

Once a patient is deemed suitable for a given activity, several important aspects of disease management should be addressed before the start of the trip. First, the patient's pharmacologic regimen should be reviewed to ensure the person is receiving appropriate therapy with adequate symptom control (Table 53-3). Patients whose symptoms are not under good control before their planned wilderness trip may require escalation of therapy. Any such changes should be made several weeks before the planned departure, because the benefits of such agents as inhaled corticosteroids or tiotropium are not maximized until 1 or more weeks after initiation of therapy. Although theophylline is sometimes used as an adjunctive therapy in patients whose disease is not well controlled with the other agents, it has a narrow therapeutic window and increased risk for toxicity compared with other therapies and should not be added to a patient's regimen before a wilderness trip. Patients already taking theophylline on a regular basis should have their level checked before departure and should be counseled regarding how to recognize symptoms and signs of toxicity.

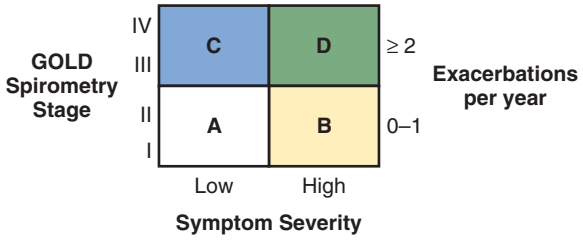

FIGURE 53-1 Revised classification scheme from the Global Initiative on Obstructive Lung Disease for determining the severity of chronic obstructive pulmonary disease (COPD). Based on the spirometry stage, level of symptoms, and number of exacerbations per year, patients are placed in one of four boxes, with *A* representing the least severe category and *D* representing the most severe category. Symptom severity can be assessed using either the modified Medical Research Council (mMRC) or the COPD assessment test (CAT) scoring systems. Patients in group A are likely safe to engage in wilderness travel without further evaluation, whereas patients in groups B, C, or D should undergo pretravel assessment. *(From Global Initiative for Chronic Obstructive Lung Disease (GOLD): Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: Updated 2014.* http://www.goldcopd.org/uploads/users/files/GOLD_Report_2014_Jun11.pdf. *From the Global Strategy for Diagnosis, Management and Prevention of COPD 2016.)*

After reviewing the adequacy of the medication regimen, a plan should be created for responding to disease exacerbations that might occur during the planned sojourn. Often triggered by viral or bacterial infections, such exacerbations are characterized by increased dyspnea and a change in the frequency or character of sputum production and can lead to worsening hypoxemia and overt respiratory failure. Because the incidence of COPD exacerbations during wilderness travel is unknown, all COPD patients traveling into the wilderness should prepare for this complication. They should identify medical facilities close to the area in which they will be traveling and arrange an evacuation plan in the event of severe symptoms. They should also carry a supply of rescue medications, including short-acting bronchodilators, prednisone, and oral antibiotics (azithromycin, levofloxacin, doxycycline, or trimethoprim-sulfamethoxazole), particularly when traveling in remote areas with limited access to health facilities, and instructions regarding when and how to use them. With proper technique, short-acting bronchodilators given by metered-dose inhaler therapy can be of equal efficacy to nebulized solutions typically used in emergency departments (EDs). Oral prednisone also has bioavailability when administered as intravenous steroid preparations in the ED inpatient setting and should be useful in the field. Formoterol has a fast onset of action and can be used as a rescue inhaler if albuterol is not available, but its long half-life precludes use more frequently than every 12 hours. Onset of salmeterol is too slow to be of use in rescue situations.

Patients with COPD receiving supplemental oxygen at baseline should continue this therapy on any planned wilderness trip. Other patients may not require this therapy depending on the circumstances of their trip. The traditional supplemental medical oxygen delivery system is a continuous flow of 100% oxygen from a compressed gas cylinder delivered by nasal cannula. The disadvantage to this system is its inefficient use of oxygen, because only a small percentage of the oxygen delivered to the nose actually reaches the lungs. In a wilderness setting, the weight and space required to carry medical oxygen cylinders create a significant burden and limit the trip duration. A more efficient alternative is for the patient to use a pneumatic, nonelectronic demand valve that delivers flow of oxygen only on inspiration.[199] Portable liquid oxygen units with demand valves are an alternative to oxygen cylinders and offer the advantage of lighter weight for a comparable amount of oxygen, but are more expensive. Any patient using a demand valve system for a wilderness trip should be evaluated during rest and exercise to ensure that adequate oxygenation is maintained.[198] Also, portable oxygen concentrators are available that obviate the need for liquid or compressed gas cylinders and increase patient mobility. However, their usefulness is limited by short battery life.[125] Depending on the length of the planned trip, access to power

sources, and ability to carry spare batteries, these may not represent a suitable alternative.

Important logistic issues can affect plans to travel with supplemental oxygen. For persons traveling by car to their destination, there should be few problems bringing oxygen, but persons traveling by airplane may encounter significant problems. As a general rule, patients are not allowed to bring liquid or compressed gas oxygen cylinders on board aircraft as either carry-on or checked baggage. The Federal Aviation Administration permits the use of small, portable oxygen concentrators on aircraft in the United States, but use may not be permitted on all airlines worldwide. Unfortunately, standard practices for supplemental oxygen vary across the airline industry, with the availability of service, feasibility of using personal systems, and fees varying between countries, airlines, and domestic and international flights.[128,208] Patients planning to obtain oxygen sources on arrival at their destination should confirm in advance whether such sources are available, because access varies based on whether the person is traveling in low-income or high-income countries. Even in high-income countries, access to supplies might be limited in more remote settings.

Similar to the considerations for asthma patients, two specific wilderness activities that deserve further attention in patients with COPD are high-altitude travel and diving. The issue of high-altitude travel with COPD has been reviewed extensively.[125,127] The primary concern regarding high-altitude travel is the potential for worsening hypoxemia. Few data are available about COPD patients in actual mountain environments, but data from the literature on COPD patients and commercial airline flights clearly indicate that patients with FEV_1 values of 1 to 1.5 L experience significant hypoxemia when exposed to altitudes equivalent to 2440 m (8000 feet), with further drops in PaO_2 with minimal exertion, such as walking on flat ground or cycling at very modest work rates (20 to 30 W).[125,127] Patients already using supplemental oxygen at home should increase their flow rates at high altitude and can consider portable pulse oximetry as a means to decide on the appropriate adjustment. Depending on their baseline disease severity, patients not already receiving supplemental oxygen should undergo pretravel assessment to determine the need for oxygen at high altitude. This can be done using either high-altitude simulation testing[49] or a variety of prediction rules that take into account various factors such as the arterial oxygen tension (PaO_2) while breathing ambient air at sea level, FEV_1, or target altitude.[37,81,143] Patients who develop symptomatic hypoxemia (PaO_2 <50 to 55 mm Hg) during high-altitude simulation testing or who are predicted to have a PaO_2 in this range should consider supplemental oxygen at high altitude. Decisions to use oxygen should not be based on the PaO_2 alone, but should also reflect whether or not the patient develops associated symptoms (dyspnea, lightheadedness, dizziness, altered mental status, exercise intolerance). Patients who become hypoxemic (PaO_2 <50 to 55 mm Hg) but remain asymptomatic with preserved exertion tolerance can travel without supplemental oxygen but should monitor symptoms and oxygen saturation on arrival at high altitude using portable pulse oximetry (SpO_2) or through periodic medical evaluation, and they should carry a prescription for supplemental oxygen that can be filled at their destination if problems develop after arrival. Patients whose PaO_2 remains above these thresholds can travel without supplemental oxygen but may also consider monitoring symptoms and SpO_2 during their sojourn.[125]

There are no data on the frequency of exacerbations at high altitude, whereas data on measures of airflow obstruction are limited and conflicting. As a result, any COPD patient traveling to high altitude must be prepared for the possibility of exacerbations as previously described. No evidence suggests that patients with severe bullous disease are at increased risk for pneumothorax at high altitude, despite the fall in barometric pressure.[127]

Diving carries the same concerns for COPD patients as for asthma patients. Airflow obstruction could lead to significant air trapping, which would increase the risk for barotrauma when trapped air expands in response to decreasing barometric pressure on ascent. Any blebs or bullae that do not adequately communicate with the environment could expand and rupture with ascent to the surface. For these reasons, COPD is considered a contraindication to diving.

SLEEP APNEA

Sleep-disordered breathing refers to respiratory disturbances that occur during sleep and includes entities such as obstructive sleep apnea (OSA), central sleep apnea (CSA), and sleep-related hypoventilation. OSA is the most common form of sleep-disordered breathing and is marked by repeated reduction (hypopnea) or cessation (apnea) of airflow that results from partial or complete occlusion of the upper airway during sleep. Present in up to 28% of the general population, OSA is more common in men and among older individuals and may occur in people who lack other underlying medical problems.[215] CSA is also marked by recurrent apnea or hypopnea, but unlike in OSA, alterations in airflow occur because of changes in respiratory signaling and effort rather than upper airway occlusion. Although idiopathic forms of CSA occur, it is most frequently seen among individuals with severe cardiomyopathy or after severe neurologic injury. As indicated in Chapter 3, CSA is also common among otherwise healthy people at high altitude.

Sleep-related hypoventilation refers to abnormally high arterial carbon dioxide tension ($PaCO_2$) levels during sleep and is usually seen in the context of morbid obesity, obstructive lung diseases, and various neuromuscular disorders, such as muscular dystrophy or amyotrophic lateral sclerosis. The various forms of sleep-disordered breathing are significant not only because of their ability to disrupt sleep quality, but also because of their adverse effects on daytime function, including excessive daytime somnolence and impaired concentration. In addition to treatments directed at the underlying disease (e.g., heart failure), the standard treatment approach for each of these disorders is nocturnal use of noninvasive intermittent positive-pressure ventilation (NIPPV) or continuous positive airway pressure (CPAP), depending on the particular condition and its severity.

Individuals with these disorders who seek to travel into wilderness environments must consider several key issues before their trip, including (1) what will happen to the underlying disorder in that environment, (2) whether it is necessary to continue treatment while engaged in the wilderness activity, and (3) how to facilitate continued treatment if such treatment is deemed necessary. With regard to the first question, little is known about what happens to the various patterns of sleep-disordered breathing in the wilderness. While there is little reason to expect changes in the incidence and severity of these problems when sleep is conducted at or near the same elevation at which the patient normally resides, changes may occur because of barometric pressure changes following ascent to high altitude. Changes in the severity of preexisting CSA after ascent to high altitude have not been studied, but data are available regarding OSA. Whereas two earlier studies suggested that the number of obstructive events decreased in both normal individuals exposed to terrestrial high altitude (5050 m [16,570 feet])[27] and individuals with moderate OSA (baseline apnea-hypopnea index >15) exposed to simulated high altitude equivalent to 2750 m (9024 feet),[26]more recent, larger field studies demonstrate that the number of obstructive events remains largely unchanged after ascent.[149] In all these studies, however, there were marked increases in the frequency of central apneas. Additional studies in normal individuals further suggest that these central events persist even following prolonged acclimatization (>2 weeks at altitude >3730 m [12,238 feet]).[16] The reason for the observed increase in central events was not elucidated but is likely related to the impact of ventilatory responses to hypoxia on central respiratory control. Because the various forms of sleep-disordered breathing are generally associated with intermittent nocturnal hypoxemia, it can be expected that high-altitude travel will lead to greater degrees of nocturnal desaturation,[149] with the magnitude of hypoxemia likely being a function of the altitude attained, as well as the duration of apneas and hypopneas.

Whether individuals with sleep-disordered breathing should continue treatment while engaged in wilderness activity will be a function of the severity of their disease, as well as associated

symptoms and characteristics of their planned activities. For example, individuals with OSA but minimal or no daytime symptoms may be able to forego CPAP for several days while away on a trip, although excessive snoring may be a nuisance to other people on their excursion. On the other hand, individuals with severe daytime symptoms or who expect to perform activities that require high levels of concentration may find it necessary to continue therapy.

For individuals who plan to continue NIPPV or CPAP, the most important issue is ensuring reliable access to power supplies. When such access is available (e.g., hotel, generators), travel should be relatively straightforward, because most machines are small and lightweight enough to facilitate travel and can be brought on airplanes as carry-on luggage. Significant challenges arise, however, when power access is not available. Some manufacturers make devices with rechargeable lithium batteries, but battery life is short, and use for more than 1 to 2 days would require access to recharging facilities or ample numbers of backup batteries. An alternative is to obtain a special power cord from the device manufacturer that allows one to draw power off a 12-volt battery (deep-cycle marine batteries offer the best battery life), but this option is limited by the size and weight of these batteries, expense, and logistic issues associated with obtaining a battery at the destination or traveling with a battery in hand.[181] Individuals should contact the device manufacturer for information about the power consumption of their device to guide anticipated battery needs and should be aware that use of heated humidity systems will increase power needs. Finally, although most commercially available machines can run off the voltage levels used with outlets in the United States (110 to 120 V) and Europe (220 to 240 V), individuals should confirm the range for their machine and ensure that they are carrying the appropriate plug adapters.

Patients with OSA traveling to high altitude should be aware that changes in barometric pressure with ascent can affect the delivered pressure[74] and leak detection systems on older CPAP machines. Most current-generation machines have pressure-compensating features to account for changes in barometric pressure, but these systems may not be designed to work above certain elevations. Individuals should consult their sleep medicine provider or device manufacturer in advance of the trip to clarify whether their device will function at the intended destination. OSA patients traveling to high altitude can also consider addition of acetazolamide (250 mg PO three times a day) to their CPAP regimen, because combination therapy with these interventions has been shown to decrease the apnea-hypopnea index and improve nocturnal So_2 at 2590 m (8500 feet) compared with use of autotitrating CPAP alone.[117]

DIABETES

Approximately 21 million people with a diagnosis of diabetes live in the United States (6.7% of the U.S. population), so it is likely that diabetes will be encountered in persons pursuing wilderness activities.[31] Diabetes encompasses the disorders of type 1 diabetes, previously known as insulin-dependent or juvenile-onset diabetes, and type 2 diabetes, previously known as non–insulin-dependent or adult-onset diabetes. Approximately 5% to 10% of diabetic patients have type 1 disease, and 80% have type 2, with the remainder of cases from other causes. The pathophysiology of type 1 diabetes results from inadequate insulin production, whereas the pathophysiology of type 2 diabetes is caused by peripheral resistance to insulin action. Patients with type 1 diabetes must be treated with insulin, whereas those with type 2 diabetes may be treated with diet and exercise, oral and injectable hypoglycemic agents, or insulin.

A number of issues need consideration in patients with diabetes pursuing wilderness activities. Diabetic patients may be remote from medical help, and they need to ensure that adequate medication is available. Carrying two or three times as much medication as anticipated and extra devices (syringes, glucometer, glucose/ketone test strips) and splitting up medication and medical device supplies among group members will mitigate against theft, loss, or unanticipated treatment or travel delays on longer trips (Table 53-4). Anticipate erratic meals, time changes, and increased levels of physical activity, and factor how patients may change medication regimens. Engaging in nonroutine activities also creates certain safety issues for patients with diabetes, so advising them on how to manage diabetes appropriately during exercise in an outdoor environment is essential. High-risk wilderness activities, such as mountaineering or rock climbing, where loss of focus and concentration may result in death, would not be appropriate for a diabetic patient who is susceptible to hypoglycemia, unless the person is constantly monitored by a

TABLE 53-4 Diabetes-Specific Supplies for Wilderness Activities

Insulin Supplies	Wilderness Pack Description
Insulin	Three times the amount anticipated for each type of insulin, stored at nonextreme temperatures
Insulin pens and needles (if applicable)	One extra pen and three times the anticipated number of needles
Pump supplies (if applicable)	Three to five times the amount anticipated
Syringes	Enough to cover the entire trip if on the pen or pump; two to three times the anticipated requirement if using syringes alone
Glucose meter	Two different meters with extra batteries for each
Glucose strips and lance/lancets	Three times anticipated number of strips for each meter, two lances, and three times the anticipated number of lancets; a supply of visually read strips should also be taken as a backup in the event of meter failure
Ketone strips	Two packages
Carbohydrates	
Dextrose tablets (rapid-acting carbohydrate)	One package (50 g) per day
Dried fruit and cookies (slower-acting carbohydrates)	Several individually wrapped packages per day
Glucagon kit (this must be protected from breakage and from freezing of the vehicle)	Two kits
Intravenous setup	One complete kit
Single-use sterile needles and syringes	Several 18-g and 10-mL syringes, respectively, in the event that medical treatment is required in a hospital or clinic with limited resources
Insulated packs	Enough to carry all supplies
Letter from physician	Listing supplies and their necessity, for international border crossings

Modified from Brubaker PL: Adventure travel and type 1 diabetes: The complicating effects of high altitude, *Diabetes Care* 28:2563, 2005.
Note: Supplies should be packed and carried in a minimum of two independent sites (carried personally at all times by two people or by one person with the second set in a separate travel bag and/or at a nearby hotel).

climbing partner, does not climb in the lead, and is always secured by a rope. Similarly, solo wilderness activities may not be appropriate for diabetic patients who may become hypoglycemic.

Both type 1 and type 2 diabetic patients require evaluation for several issues before a wilderness trip. The extent of increased exercise that will be undertaken on the trip needs to be determined relative to the patient's current level of physical activity and glucose control. Increased physical activity is beneficial for diabetic patients, particularly those with type 2 disease, and wilderness activities may be encouraged as part of lifestyle modification. In addition to exercise, the specific activity and associated travel are likely to effect a change in diet. Screening for conditions associated with diabetes, such as cardiovascular disease, peripheral and autonomic neuropathies, proliferative retinopathy, and nephropathy, should be done. These conditions may significantly influence the ability of diabetic patients to pursue wilderness activities safely and may require further evaluation and testing before the trip. Lastly, the effect of the change in environment on the diabetic patient is considered, including potential problems with exposure to heat or cold.

The effect of increased exercise is important for both type 1 and type 2 diabetic patients because it may precipitate hypoglycemia or hyperglycemia, depending on the timing of the last dose of insulin and blood glucose level at the onset of exercise.[84,210] The normal response to exercise in nondiabetic persons is a decrease in insulin secretion as serum glucose levels fall because of uptake of glucose by exercising muscle, and an increase in hepatic glucose release in response to catecholamines, glucagon, and growth hormone to maintain blood glucose levels. Patients with type 1 diabetes who are hypoinsulinemic when they exercise (as may occur if they excessively decrease their insulin in an effort to accommodate the increased physical activity) may become hyperglycemic because of increased hepatic release of glucose into the blood, in addition to insufficient insulin to allow glucose to enter the cells. Thus, hyperglycemia results in decreased exercise capacity, because exercising muscle is depleted of glucose. Under these conditions, the substrate for fuel becomes free fatty acids released from adipocytes, with generation of ketone bodies by the liver. Hypovolemia may result from glycosuria; if the process persists, diabetic ketoacidosis may ensue.

Patients with type 1 diabetes who are administered exogenous insulin in excess may become hypoglycemic when they exercise. Exercise results in increased insulin secretion, rather than the expected physiologic decrease in insulin with exercise. Increased insulin levels enhance glucose uptake by the muscles and decrease release of glucose from the liver. Both result in a lowering of blood glucose to a greater degree than in nondiabetic persons during exercise.[52] Type 1 diabetic patients also have defective glucagon secretion that will augment defective counter-regulatory responses to hypoglycemia. Delayed hypoglycemia may occur hours after exercise as the resting muscle takes up glucose from the blood to replenish glycogen stores. Because the kidneys metabolize insulin, dehydration can result in decreased renal insulin clearance and prolong insulin action. Therefore, monitoring glucose levels, nutrition, and hydration and adjusting the dose of insulin are also important after exercise.

Another factor contributing to hypoglycemia in the exercising patient with type 1 diabetes is increased exogenous insulin mobilization from subcutaneous tissue because of increased blood flow.[72,105] Inadvertent intramuscular injection would exaggerate this phenomenon. It is important for patients with type 1 diabetes to administer their dose of subcutaneous insulin before exercise in a location away from exercising muscle. They should avoid injections into the arms and legs, instead using the abdomen or back of the neck. Insulin absorption is fastest and most consistent when it is injected into the abdomen. Absorption is generally slower from the arms and slowest from the thighs or buttocks, but the rate may be more inconsistent depending on the type of exercise. If the planned exercise uses primarily arm muscles, such as sport climbing, it may be beneficial to give the insulin injection in the abdomen to avoid increased absorption from the exercising arm muscles. Likewise, if the planned activity is hiking or skiing, it would be beneficial to use the arm or abdomen to avoid increased absorption from the exercising leg muscles. Absorption of subcutaneous insulin during exercise may also depend on the type of insulin used. A study of the long-acting insulin analog glargine, injected subcutaneously into the thigh on the evening before an intense 30-minute exercise session in patients with type 1 diabetes, did not show an increased rate of absorption, but plasma glucose fell during exercise.[159]

Another measure to prevent exercise-associated hypoglycemia is to reduce the dose of insulin that will be in effect during exercise.[134] The best strategy for a patient with type 1 diabetes is to monitor blood glucose before, during, and after exercise to predict changes, and adjust insulin doses accordingly. This means that before a wilderness trip, the type 1 diabetic patient should exercise daily at a level of physical activity similar to that anticipated on the wilderness trip, so that adjustments in insulin dosing can be made. Differences in nutritional intake during the wilderness trip should also be anticipated when planning insulin regimens. Patients with type 1 diabetes may engage in strenuous physical activity without experiencing problems, but it is important for each patient to focus on the timing of exercise in relation to meals and insulin dosing.[106,196] It is also important to anticipate the nature of the exercise that will be undertaken. Long endurance activities have different implications for type 1 diabetes management (more risk for hypoglycemia) than do short bursts of high-intensity exercise (more risk for hyperglycemia).[84]

Another consideration is whether regular insulin or a rapidly acting insulin analog (lispro or aspart) is used for prandial dosing. Rapidly acting insulin alters the timing of exercise-related hypoglycemia. Patients who exercise early in the postprandial period (1 to 3 hours after a meal) require a decrease in the dose of rapidly acting insulin, whereas those who exercise later (3 to 5 hours after a meal) require a smaller or no change.[94] A predictable exercise schedule on a wilderness trip would be useful for the patient with type 1 diabetes, although this may be difficult depending on the type of trip and the environment.

Other members of the group need to be aware of signs and symptoms of hypoglycemia in the diabetic patient and how to render appropriate treatment. This is especially important for patients with type 1 diabetes, but also is significant for patients with type 2 diabetes who are taking oral hypoglycemic drugs or insulin, because this group also experiences a decrease in serum glucose during exercise.[161,210] Preventive measures include ingesting extra food in the form of 15 to 30 g (0.5 to 1 oz.) of quickly absorbed carbohydrate (e.g., glucose tablets, whole milk, hard candy, juice), which should be taken 15 to 30 minutes before exercise and approximately every 30 minutes during exercise.[83,84] Patients are also at risk for late hypoglycemia 4 to 8 hours after the termination of exercise because of replenishment of depleted glycogen stores. This can usually be avoided by ingesting slowly absorbed carbohydrates (dried fruit, granola bars, or trail mix) immediately after exercise.[188] Fluid intake should also be increased to ensure adequate hydration, because dehydration prolongs insulin action by decreasing insulin clearance from the blood.

The acute symptoms of hypoglycemia are mediated by the adrenergic autonomic nervous system and are manifested by shakiness, tremors, palpitations, cold sweats, hunger, confusion, and nervousness/anxiety. If these are ignored, not sensed (because of hypoglycemic unawareness), or cannot be treated immediately, the person may experience neuroglycopenic symptoms because of inadequate amounts of glucose reaching the brain. Neuroglycopenic symptoms consist of headache, loss of concentration, irritability, and if allowed to proceed long enough, seizures and loss of consciousness. Hypothermia is a common sign of hypoglycemia. All persons with diabetes should be questioned about their history of hypoglycemic reactions (frequency and severity) as well as their ability to sense the reactions (history of hypoglycemic unawareness). Travel into the wilderness should be discouraged in persons with a history of severe or frequent reactions or a history of hypoglycemic unawareness. Companions should be taught to recognize the signs and should be knowledgeable in the treatment of hypoglycemia.

If alert and cooperative, the diabetic patient may be treated with 15 g (0.5 oz.) of a rapidly absorbed carbohydrate (e.g.,

glucose tablets, juice, hard candy). The blood glucose should be checked in 15 minutes to ensure that the glucose level has increased to a safe level (>100 mg/dL) before continuing with the physical activity. The person should be closely watched for evidence of recurrent symptoms. If the person becomes uncooperative or loses consciousness, a glucagon injection kit should be available. Glucagon releases glucose stored in the liver and helps raise the glucose level to the point at which the person becomes alert, so that treatment may be continued with an oral, rapid-acting carbohydrate. The dose of glucagon is 1 mg (1 ampule) given either subcutaneously or intramuscularly. Another route for delivering glucose is to deposit glucose formulations, such as table sugar, under the tongue.

Activity limitations for diabetic patients also depend on associated complications. Patients with proliferative retinopathy should avoid activities that can rapidly or explosively elevate intraocular pressure, because vitreous hemorrhage may occur. Bleeding from damaged retinal capillaries may occur during Valsalva maneuver, while lifting heavy objects, or during a collision. An ophthalmologist should examine for early signs of retinopathy at least once a year.[51,206]

Patients with peripheral neuropathy are at risk for pressure ulcers of the feet and should avoid traumatic weight-bearing exercise (long-distance running or prolonged downhill skiing). Well-fitting protective footgear helps prevent this complication. Diabetic patients with peripheral neuropathy are also more susceptible to cold injury of the feet in a cold environment. Patients need to monitor their feet meticulously. In a cold-weather environment, it is especially important to have well-insulated warm footgear with dry socks. Diabetic patients should check all surfaces of their feet at least once a day. A mirror may be required to visualize the plantar surface. Any sign of inflammation, including blistering or abrasion, should be promptly addressed. The importance of well-broken-in footwear and the need to wear clean and dry socks at all times should be emphasized. Diabetic patients should be advised to never walk barefoot.

Autonomic neuropathy also poses problems with increased activity in a wilderness environment. Cardiac responsiveness may be delayed during rapidly changing exercise levels. Gastroparesis may make absorption of carbohydrates unpredictable and could result in a mismatch in timing between insulin and glucose peaks.

Certain drugs that may be used at high altitude also need special consideration in diabetic patients. Acetazolamide is often used for prevention of acute mountain sickness through a mechanism of inhibition of renal carbonic anhydrase, causing bicarbonate diuresis and non–anion gap metabolic acidosis that result in compensatory hyperventilation. Acetazolamide could worsen metabolic acidosis and promote volume depletion in diabetic patients. Dexamethasone is frequently used to treat severe acute mountain sickness or high-altitude cerebral edema and, as a glucocorticoid, increases insulin resistance and predisposes to hyperglycemia. Lastly, metformin is often used to treat type 2 diabetes. It is contraindicated in clinical conditions that may cause hypoxemia. This may be a consideration in travel to high altitude.

Ensuring that insulin does not freeze and glucose testing equipment works properly are important for the insulin-dependent diabetic patient in the wilderness. Strategies to ensure that insulin does not freeze include carrying the medication inside a jacket next to the body or storing insulin in an insulated container and putting the container in the sleeping bag at night. Glucose-testing equipment should be reliable, rapid, and used frequently to help identify impending hypoglycemia. The accuracy of glucose-testing equipment can be affected by high altitude and cold. Both overestimation and underestimation of glycemia and of standard glucose control solutions have been reported for all types of glucose meters.[23] Glucose meters using the oxygen-insensitive enzyme glucose dehydrogenase may perform better at high altitude than those using the enzyme glucose oxidase, but both types perform poorly at low temperatures.[150] High glucose levels seem to be misreported more at altitude than low to normal glucose levels.[23] The use of multiple meters with control glucose solutions can lend some confidence. Carrying glucose-monitoring equipment next to the skin may prevent the problems associated with battery malfunction at cold temperatures.

Provided that the insulin-treated patient with diabetes has reviewed all the factors associated with a wilderness trip that may be complicated by diabetes and has chosen to undertake the preparation required and accept the risks, a treatment plan needs to be generated. The most challenging wilderness trips for maintaining glucose control in diabetic patients are those where the level of exercise is consistently increased above usual. Intensive diabetes management can be achieved with multiple daily injections of insulin or an insulin pump and numerous checks of blood glucose throughout the day (Table 53-5 and Table 53-6). A standard strategy to offset the increased efficiency of insulin caused by exercise is to ingest 15 to 30 g (0.5 to 1 oz.) of carbohydrate for every ½ hour of moderate aerobic exercise. Monitoring blood glucose during periods of exercise of several hours' duration is important.[84,210] Adjusting the dose of insulin downward may be required if carbohydrate supplementation does not prevent hypoglycemia. It may be necessary to decrease the dose of insulin by 30% or more.[51,134] Blood glucose should be checked more often early during a wilderness trip to assess how to balance insulin, carbohydrate intake, and exercise. It is also critical to prevent delayed hypoglycemia associated with exercise, which often occurs after muscle and liver glycogen stores have been depleted and not replenished. The use of intermediate-acting insulin may need to be shifted from the afternoon or early evening to bedtime, or a long-acting insulin preparation that does not peak may be used. Adequate hydration is essential to ensure that the duration of action of insulin is not increased.

Diabetic patients may pursue the same wilderness activities as do other individuals, provided they have the experience necessary to participate safely. This includes activities such as

TABLE 53-5 Matching Insulin Treatment Schedules with Exercise Schedules

Treatment Type	Advantages	Disadvantages
Standard: two injections, mixed intermediate- and short-acting insulins	Easy to perform	Poor match with exercise, rigid time restraints, least likely to give good metabolic control and health
Intensive: three or more injections a day	Better control, more flexible timing, less hypoglycemia in the evening	More frequent testing, harder to learn
Extended: glargine for basal plus lispro/aspart for meals	Least amount of time rigidity, most protection against hypoglycemia, excellent metabolic control	Much more effort to master and do well
Continuous infusion (pump)	Most flexible (no injections most days), low hypoglycemia risk overnight, best metabolic control Basal insulin infusion rate can be adjusted to accommodate increased insulin sensitivity due to increased activity	Needs expensive device, harder to master, must remove pump for some activities; need to carry syringes in case of mechanical failure Risk for infusion site infections

Modified from Draznin MB: Type 1 diabetes and sports participation: Strategies for training and competing safely, *Phys Sportsmed* 28:49, 2000.

TABLE 53-6 Types, Names, and Uses of Insulin

Insulin	Onset	Peak	Duration	Uses	Notes
Rapid-acting (insulin analogs lispro, aspart, glulicine)	≤0.5 hr	0.5-3 hr	3-5 hr	Meal coverage if taken up to 15 min before eating; adjust for elevated blood glucose levels	Insulin of choice for pumps. Rapid action may not be desirable during periods of physical activity because it may lead to more hypoglycemia
Short-acting (human regular)	0.5-1 hr	2-5 hr	Up to 12 hr	Meal coverage if taken 30-45 min before eating	May last too long, leading to hypoglycemia
Intermediate-acting (human NPH)	1.5-4 hr	4-12 hr	Up to 24 hr	Meal coverage (lunch) if taken in the morning	More active early in the dosage or can linger longer than anticipated; either can cause hypoglycemia
Long-acting (insulin analogs glargine, detemir)	0.8-4 hr	None	Up to 24 hr	Basal	Once-daily injection; cannot be mixed with other insulins; soluble until injected

Modified from Draznin MB: Type 1 diabetes and sports participation: Strategies for training and competing safely, *Phys Sportsmed* 28:49, 2000; Hirsch IB: Insulin analogues, *N Engl J Med* 352:174, 2005; and Vijan S: Type 2 diabetes, *Ann Intern Med* 152:ITC31, 2010.
NPH, Neutral protamine Hagedorn.
Notes: A typical intensive schedule of injections would be a mixture of NPH with lispro before breakfast, lispro alone at dinner, and NPH at bedtime.
Testing is done before meals and at bedtime regardless of the type of insulin used. To establish the dose (given by injection or by pump) for long-acting insulin plus lispro for meals, the patient also tests blood glucose 2 hours after each meal for 3 to 4 weeks. Careful record keeping will determine if assumptions are correct and which doses work best. Testing should be done initially every 30 to 60 minutes during exercise to determine the effect of the activity on the blood glucose levels. Testing should be done every 2 hours after the completion of exercise for 6 to 12 hours to monitor for late-onset hypoglycemia.
The time course of action of each insulin may vary among persons or at different times in the same person. Because of this variation, the time periods indicated here should be considered general guidelines only.

high-altitude mountaineering.[119] On an expedition to Cho Oyu, an 8201-m (26,906-foot) peak in the Nepal Himalayas, six type 1 diabetic patients participated in the expedition, and one reached the summit.[154,155] These climbers were highly motivated and adhered to careful glucose monitoring, and all completed the expedition safely and free from long-term complications. Their cardiovascular parameters were comparable with those of nondiabetic control climbers on the same expedition, although the patients with diabetes had worsening in metabolic control and required higher insulin doses than at sea level.

PERIPHERAL ARTERIAL DISEASE

Peripheral arterial disease (PAD) is caused by atherosclerotic occlusion of the arteries of the legs. The most common symptomatic presentation is intermittent claudication, which is leg muscle discomfort on exertion that is relieved with rest. Many patients present with atypical symptoms, including leg fatigue, difficulty walking, and leg pain not typical of claudication.[212] Among men over age 60, 2% to 3% have symptomatic PAD, as do 1% to 2% of women. The prevalence of asymptomatic PAD, proved by an ankle-brachial index (ABI) of 0.9 or less, is three to four times as great as that of symptomatic disease.[4] Approximately 25% of PAD patients have symptoms of claudication, and approximately 10% have critical ischemia.[192] Because PAD is common, it may be present in persons intending to pursue wilderness activities, especially elders and those with other manifestations of systemic atherosclerosis. Severity of PAD is closely associated with the risk for myocardial infarction, ischemic stroke, and death from vascular causes. The major risk factors for PAD are older age (>40), cigarette smoking, and diabetes mellitus. Hyperlipidemia, hypertension, and hyperhomocysteinemia are also important risk factors.[91]

When evaluating a patient with PAD for a wilderness activity, it is helpful to characterize the severity of the disease and how it affects functional capacity. The most useful method for evaluating severity is the ankle-to-arm ratio of systolic blood pressure (ABI), which is easily obtained with a standard blood pressure cuff and a Doppler device.[91,212] Systolic blood pressure is measured by Doppler ultrasonography in each arm and in the dorsalis pedis and posterior tibial arteries in each ankle. The higher of the two arm pressures is selected, as is the higher of the two pressures in each ankle to calculate a left and right ABI. An ankle/arm pressure ratio of 0.91 to 1.3 is normal. A ratio of 0.41 to 0.9 indicates mild to moderate PAD, and less than 0.4 is severe. A ratio greater than 1.3 indicates a calcified noncompressible vessel. Patients with claudication, defined as walking-induced pain in one or both legs (primarily in the calves) that does not go away with continued walking and is relieved by rest, typically have ABI in the mild to moderate range. Patients with critical ischemia have ABI in the severe range.

Wilderness activities are associated with increased exercise. This may be a limiting factor for patients with peripheral vascular disease who suffer claudication. Hiking and trekking, however, are reasonable activities for patients with claudication if they engage in a regular exercise training program. The benefit of exercise in improving functional capacity in patients with claudication is well proven,[87,139,192] and greater physical activity is associated with lower mortality.[76] Exercise training improves exercise treadmill performance, pain-free walking time, and walking distance.[75,113,152] Exercising at least twice weekly, even in the presence of pain from intermittent claudication, increases walking time. Walking through the claudication pain is not harmful and increases walking distance.[87] Even self-directed walking exercise or alternative exercise programs may be of equal benefit to supervised exercise programs.[118,138]

Exercise-induced increases in functional capacity and lessening of claudication symptoms primarily result from improvements in endothelial vasodilator function, skeletal muscle metabolism, blood viscosity, and biomechanics of walking. Increases in leg blood flow and oxygen delivery may occur, but do not appear to account for the large improvements in exercise capacity. A supervised exercise program several times per week is recommended, but a regular self-directed exercise program can also have benefit.[118,138] The key elements of each session include a warm-up period followed by walking on a track or treadmill at a workload that causes claudication in about 5 minutes. One continues at that workload until claudication of moderate severity occurs, then rests standing or sitting for a brief period to allow symptoms to subside, and repeats the exercise-rest pattern for the duration of the session, lasting initially approximately 30 minutes, but working up to about 1 hour. This exercise strategy might also be used while hiking, repeating periods of exercise and rest as gauged by claudication symptoms, and could potentially lead to improvements in exercise capacity over time. A walking or hiking exercise program needs to be done regularly and sustained over months, or the benefits diminish.

Patients with PAD who want to pursue wilderness activities or an exercise program should undergo evaluation for heart disease, hypertension, hyperlipidemia, and diabetes. Appropriate treatment and risk factor modification (including smoking cessation) should be initiated.[87] In addition, an exercise test with

12-lead electrocardiographic monitoring should be performed to evaluate for cardiac ischemia. Patients with claudication may be limited by leg pain during a maximal exercise test, thus limiting stress on the heart, but information gained regarding heart rate and blood pressure response, work level at which claudication occurs, and exercise capacity is useful for estimating the type of wilderness activity that may be pursued and in formulating a prescription for exercise training.

Treatment of claudication should include a formal walking-based exercise program. In addition, several options exist for drug therapy. The Ninth ACCP Consensus Conference on Antithrombotic and Thrombolytic Therapy recommends lifelong antiplatelet therapy in patients with PAD with aspirin or clopidogrel.[4] Clopidogrel is a thienopyridine drug that inhibits platelet activation, is an alternative to aspirin, and has fewer hematologic side effects than the related drug ticlopidine. Aspirin is significantly less expensive than clopidogrel and ticlopidine.[87] Clopidogrel has U.S. Food and Drug Administration (FDA) approval for prevention of ischemic events in patients with PAD.[4,91]

Cilostazol and pentoxifylline are the two drugs approved in the United States for treating claudication. Cilostazol is a phosphodiesterase inhibitor that suppresses platelet aggregation and is a direct arterial vasodilator. Cilostazol improves walking distance in patients with intermittent claudication[10] and is recommended for patients with more severe disabling claudication.[4] Because other oral phosphodiesterase inhibitors used for inotropic therapy have caused increased mortality in patients with advanced heart failure, cilostazol is contraindicated in heart failure of any severity. Pentoxifylline is a methylxanthine derivative that improves deformability of RBCs, lowers plasma fibrinogen concentrations, and has antiplatelet effects. It is not as effective in improving walking ability as is cilostazol.[43] The Ninth ACCP Consensus Conference did not recommend pentoxifylline for treatment of PAD.[4]

Patients with symptomatic PAD can pursue activities in the wilderness that are appropriate for their functional capacity. Such activities should include walking or hiking and allow for periods of rest when necessary. An exercise program combined with drug therapy can improve ability to participate in wilderness activities. Environmental extremes, such as cold and high altitude, may worsen limb ischemia and predispose to frostbite.

RAYNAUD'S PHENOMENON

Raynaud's phenomenon is a disorder of abnormal vasomotor control marked by the onset of pallor or cyanosis in the distal extremities resulting from vasospasm and decreased arterial blood flow.[214] It can occur as an isolated problem (primary Raynaud's phenomenon) or as part of collagen vascular diseases such as systemic sclerosis or systemic lupus erythematosus (secondary Raynaud's phenomenon). Primary Raynaud's phenomenon is more common in women than men and occurs in approximately 5% of the U.S. population.[214] Raynaud's phenomenon is episodic, with attacks usually occurring in response to triggers such as cold (exposure of the extremity to cold or a when the patient's body becomes cold), moisture, vibration, and emotional stress. Attacks begin with an ischemic phase marked by well-demarcated pallor that progresses to cyanosis of the digits (Figure 53-2). Changes typically start in one or several digits and spread symmetrically to all digits in the hands and/or feet, with the hands being a more common site of involvement than the feet in most individuals. These symptoms are often accompanied by pain, numbness, and burning sensations and are usually, although not always, followed by a hyperemic phase as circulation is restored to the affected areas. The classic three-phase color change occurs in approximately two-thirds of affected patients, whereas the rest have only pallor and cyanosis.[18] In most cases the attack is self-limited and resolves after rewarming the hands or feet. In severe secondary Raynaud's phenomenon, superficial ulceration or deep tissue necrosis with gangrene and amputation can occur.

Certain aspects of wilderness travel may pose risks for patients with Raynaud's phenomenon. Intense vasoconstriction can make the hands nonfunctional for performing basic tasks essential for survival in a cold-weather environment, such as zipping a coat, lacing boots, or putting on crampons. Affected individuals may be forced to rely on others to accomplish these tasks.

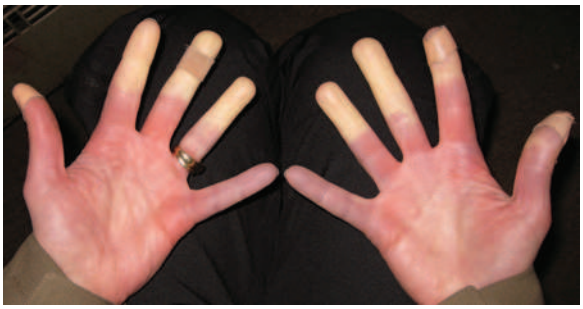

FIGURE 53-2 Active Raynaud's phenomenon. Sharply demarcated pallor results from closure of digital arteries in the second, third, and fourth digits of both hands. Cyanosis of the fifth fingers on both hands is seen. Note the bandages over fingers covering nonhealing wounds resulting from arterial insufficiency. *(Courtesy Colin K. Grissom, MD.)*

Concern has been raised about the risk for frostbite in Raynaud's patients, because they have been shown to have lower finger temperatures with body cooling,[169] slower rate of skin temperature rise with rewarming,[207] and diminished cold-induced vasodilation ("hunting response")[101] compared with healthy controls. Few studies, however, have determined whether the incidence of frostbite is increased relative to normal controls. Ervasti and colleagues[61] compared Finnish military recruits with and without the disorder and found that the odds ratio for all degrees of frostbite among people with Raynaud's phenomenon was 2.05 (95% confidence interval, 1.72 to 2.44). The risk for "deep frostbite" (blisters, ulcers, and gangrene), however, was not different between the two groups. In another study of high-altitude travelers with primary Raynaud's phenomenon, Luks and colleagues[126] reported an incidence rate of 15%, but this study lacked a control group, and the data were based on self-report rather than formal confirmation.

In addition to the issues of cold exposure in the wilderness, there is also theoretical concern that high-altitude travel could adversely affect Raynaud's patients. Any impairment in oxygen delivery stemming from arterial vasoconstriction might be worsened by low ambient oxygen conditions at high altitude, thereby increasing the severity or frequency of attacks and the risk for frostbite and gangrene. In the only study to address this question, Luks and colleagues[126] surveyed 142 people with Raynaud's phenomenon, 98% of whom had primary disease, and did not find evidence of worse problems at high altitude. Survey respondents spent 5 to 7 days per month at elevations greater than 2400 m (7874 feet) and engaged in a wide variety of activities, including winter sports (89% of respondents). Only 22% reported changing their pattern of activities in the mountains because of their disease. Of note, there was significant heterogeneity in the respondents' perceptions of the frequency, duration, and severity of their disease at altitude, compared with their home elevation. Although the survey approach made it difficult to separate out the effects of hypobaria and cold at high altitude, this study does at least suggest that motivated individuals with primary Raynaud's phenomenon can safely engage in many different activities, including winter sports at high elevations.

To prevent problems in cold or high-altitude environments, Raynaud's patients should take steps to minimize the risk for serious attacks. For example, they might choose to travel in warmer climates or in the summer rather than the winter months. If winter activities are pursued, a location with a less severe winter environment can be chosen. For example, high-altitude mountaineering in South America is more equatorial and takes place in a warmer climate than does mountaineering in North America, Asia, or Europe. Appropriate cold-weather gear and clothing are essential to keep the entire body warm and help mitigate attacks. High-quality plastic mountaineering boots or ski boots and expedition-type mittens or gloves with space for disposable chemical hand warmers help in winter environments.

Once on a trip, patients might avail themselves of a variety of strategies to prevent attacks. In the study by Luks and colleagues,[126] for example, 89% of the respondents used multiple strategies on any given trip, with the most common tactics being wearing gloves or mittens at all times, chemical hand warmers, the use of liner gloves, and limiting exposure to moisture. Climbers with Raynaud's phenomenon should also exercise good judgment and retreat early, before becoming overextended and exhausted. Nicotine and other drugs with peripheral vasoconstrictive effects, such as over-the-counter decongestants, should be avoided.

Patients might also consider the use of pharmacologic prophylaxis. Calcium channel blockers[197] and phosphodiesterase inhibitors,[29] for example, have been shown to decrease the frequency and severity of episodes of Raynaud's phenomenon and may be used on an as-needed basis during the trip.[29] Other pharmacologic agents that can be used for prevention include the α_1-antagonist prazosin, the serotonin uptake inhibitor fluoxetine, the angiotensin receptor inhibitor losartan, and the direct vasodilator isoxsuprine.[90,211] Calcium channel blockers are the pharmacologic agents of choice, but for patients intolerant of these, other agents may be tried. Benefits may also be idiosyncratic, so lack of response to calcium channel blockers is also an indication to try an alternative agent. Once an attack sets in, efforts should be made to warm the affected extremities and, in some cases, the entire body. By way of example, individuals can move to a warmer environment, don dry gloves and change into dry clothing, use chemical hand warmers, or place hands in warm water or on a warm surface.

OSTEOARTHRITIS

Osteoarthritis is a major cause of disability in adults and most often affects the joints of the hands, hips, knees, and cervical and lumbar spine. Infrequently affected joints include the shoulder, elbow, and wrist. Factors in the evolution of osteoarthritis include initiation in either previously injured or susceptible joints; development, which is biochemically mediated and biomechanically driven; and clinical expression, which may be modified by factors such as weight and gender.[41] The primary symptom of osteoarthritis is pain that is typically exacerbated by activity and relieved by rest. With more advanced disease, pain may occur with progressively less activity. Osteoarthritis can be inflammatory or noninflammatory. Patients with noninflammatory osteoarthritis complain primarily of joint pain and disability. Physical findings in affected joints include tenderness, bony prominence, and crepitus. Patients with inflammatory osteoarthritis complain of articular swelling, morning stiffness, and night pain. Signs of inflammation include joint effusion on examination or radiography, warmth on palpation of the joint, and synovitis on arthroscopic examination.

The degree of disability caused by hip or knee osteoarthritis is an important consideration for wilderness activities. Patients should be guided toward activities that are within their functional capability. Wilderness activities that cause increased weight bearing on lower-extremity joints should be avoided. For example, hiking or trekking with a light pack or bicycling is recommended over activities such as a multiday backpacking trip or mountaineering, where carrying heavy loads might be required. Risk for development of hip or knee osteoarthritis is also relevant to wilderness activities. There is increased risk for lower-limb osteoarthritis associated with repetitive, high-impact sports, and this risk increases with joint injury.[41] When assessing risk for osteoarthritis from wilderness activities, the nature and intensity of the activity, presence of previous injury, and body mass index should all be considered. Recreational running does not appear to increase the risk for knee osteoarthritis,[41,114] but how this applies to trail running is not known. The evidence that obesity is strongly associated with development of knee and probably hip osteoarthritis, and that weight loss improves joint pain and function,[100,146] are relevant to wilderness activities where carrying heavy loads is required. Wilderness activities that repeatedly require carrying a heavy pack or squatting and kneeling maneuvers may cause progression of knee or hip osteoarthritis, especially with previous injury to a joint or associated joint muscles, ligaments, or tendons.[186]

Recreational rock climbers do not appear to be at risk of developing osteoarthritis of the hands, but elite climbers and competitive sport climbers have a greater incidence of osteoarthritis of the hands.[3,172,194]

Measures that can be taken to improve functional capacity for wilderness activities include nonpharmacologic and pharmacologic treatments. The goal of management of osteoarthritis is to control pain and improve function and health-related quality of life, with avoidance of therapeutic toxicity. Potential treatments include weight loss, exercise, biomechanical techniques, pharmacologic therapy, and surgery. Table 53-7 outlines a stepwise approach to management of osteoarthritis.[100]

Exercise is a primary nonpharmacologic intervention for lower-limb osteoarthritis. There is good evidence that strengthening and aerobic exercise can reduce pain and improve function and health status in patients with knee osteoarthritis.[62,68,69,107,171] Well-conditioned muscles and muscular balance are needed to attenuate impact loads and provide joint stability. Muscular conditioning can prevent exercise-related osteoarthritis.[186] Muscular conditioning is achieved through well-designed exercise programs performed with supervision or as home exercise routines that include range-of-motion and flexibility exercise, muscle conditioning, and aerobic cardiovascular exercise.[63,64,204]

Biomechanical treatments for knee osteoarthritis are relevant to wilderness activities, such as hiking and trekking, and are helpful at reducing symptoms. For appropriate application, consultation with a physiatrist or sports medicine physician may be required. Shock-absorbing footwear reduces impact loading, heel wedges reduce loading of the medial knee joint surface, neoprene support sleeves increase proprioception and reduce feelings of instability, dynamic bracing controls lateral instability, and taping allows repositioning of the patella.[63,64] Hiking poles are an additional method to help unload lower-extremity joints while hiking or trekking and may be helpful for patients with osteoarthritis of the hip and knee.[104] During downhill walking when not carrying an external load, the use of poles reduces forces on the knees,[70,178] and with an external load, reduces forces on the ankles, knees, and hips.[17]

The major pharmacologic treatments for osteoarthritis include analgesics, nonsteroidal antiinflammatory drugs (NSAIDs), and intraarticular corticosteroids. The major goal of treatment with these agents is relief of pain, which usually is achieved with nonopioid analgesics. The nonprescription analgesic acetaminophen at doses up to 3 g/day is the recommended primary treatment.[19,95,100,216] Patients with osteoarthritis who have mild to moderate pain will obtain a similar degree of pain relief with

TABLE 53-7 Stepwise Management of Osteoarthritis

Management	Symptom Severity
Nonpharmacologic Education, exercise, weight loss, appropriate footwear ↓ **Further Nonpharmacologic** Physiotherapy, braces Simple analgesics Acetaminophen ↓ **Pharmacologic** NSAIDs, opioids If effusion is present, aspirate and inject intraarticular corticosteroids ↓ **Surgery** Osteotomy, joint replacement	Mild ↓ Severe

NSAIDs, Nonsteroidal antiinflammatory drugs.

acetaminophen as with NSAIDs.[63,64,200] Although it is one of the safest analgesics, acetaminophen can prolong the half-life of warfarin and can cause hepatic toxicity at therapeutic doses in patients with chronic alcohol abuse.

For patients who do not obtain adequate symptom relief with acetaminophen, self-limited use of nonselective NSAIDs is an alternative after considering the risk for upper gastrointestinal (GI) and renal toxicity. Cyclooxygenase-2–selective NSAIDs are an alternative with less potential risk for GI toxicity, but with an increased risk for serious cardiovascular events. The risk for adverse cardiovascular events may also apply to the nonselective NSAIDs, and they are recommended only for short-term use in the relief of pain.[15]

Another alternative therapy for osteoarthritis is the combination of glucosamine and chondroitin. These are compounds extracted from animal products that are absorbed by the GI tract and are purported to be capable of increasing proteoglycan synthesis in articular cartilage. Although some studies concluded that these compounds may be efficacious for treatment of osteoarthritis and have no significant side effects,[42,100,135] a recent meta-analysis concluded that there are no clinically significant benefits.[209]

In persons with osteoarthritis of the hand or knee who have mild to moderate pain, use of topical analgesics, such as capsaicin cream, is appropriate as adjunctive treatment or monotherapy.[42,100]

Evidence supports short-term (up to 2 weeks) improvement in symptoms of osteoarthritis of the knee after intraarticular corticosteroid injection. Significant improvement may also occur for up to 16 to 24 weeks with a dose equivalent to 50 mg of prednisone.[5,42,100] There is concern that multiple injections of intraarticular corticosteroids may promote disease progression; further study is required. Corticosteroid injection for knee osteoarthritis to provide benefit for the duration of a wilderness trip for up to 2 weeks seems reasonable[129] and may provide enough pain relief and increase in function to make wilderness activity safer and more enjoyable.

Surgical treatment of osteoarthritis is usually considered after failure of nonsurgical treatments. Four categories of surgical treatment are available: osteotomy, arthroscopy, arthrodesis, and arthroplasty. Osteotomies are performed in persons with early osteoarthritis and may relieve symptoms and slow the rate of progression. Arthroscopic debridement and lavage can also successfully alleviate symptoms, particularly in the case of degenerative meniscal tears in the presence of mechanical symptoms. When there is substantial joint space narrowing, however, arthroscopic surgery has limited benefit. Arthrodesis, or joint fusion, successfully alleviates pain and is usually performed in the spine and in small joints of the carpus, hand, and foot. Arthrodesis of the major proximal joints of the upper and lower extremities is not well tolerated because of the functional deficits associated with loss of motion.

Total joint arthroplasty represents the most significant advancement in the treatment of osteoarthritis in the past century. It is the mainstay of surgical treatment for advanced osteoarthritis of the hip, knee, and glenohumeral joints. For older persons, total joint replacement is a highly successful procedure that will probably last for the duration of their life. The pain and disability of end-stage osteoarthritis can be eliminated, restoring patients to near-normal function. In recent years, total hip replacement and hip resurfacing have become efficacious treatment options for middle-aged persons with severe osteoarthritis of the hip, and may return function to a level where participation in high-intensity sports is possible.[141] Total knee replacement also appears to have good durability in younger active persons.[124]

After total joint arthroplasty, patients should be encouraged to remain physically active for general health and also for the quality of their bones. There is evidence that increased bone quality improves prosthesis fixation and decreases the incidence of early loosening. To recommend a certain activity after total knee or hip replacement, factors such as wear, joint load, intensity, and the type of prosthesis must be taken into account for each patient and sport. Because load exponentially influences the amount of wear, only activities with low joint loading, such as swimming, cycling, or walking, should be performed regularly for exercise. If an activity is performed intermittently for recreation, activities with higher joint loads, such as skiing or hiking, may be acceptable. It is unwise to begin technically demanding wilderness activities after total joint replacement, because the joint loads and risk for injuries are generally higher for these activities in unskilled individuals. Activity recommendations differ after total knee and hip replacements. During activities such as hiking or jogging, high joint loads occur between 40 and 60 degrees of knee flexion, where many knee designs are not conforming and high stress will occur. It is prudent to be more conservative after total knee arthroplasty than after total hip arthroplasty for activities that exhibit high joint loads in knee flexion. After total knee replacement, patients should alternate activities such as walking and cycling. For mountain hiking, patients are advised to avoid descents or at least use hiking poles. Jogging or sports involving running should be discouraged after total knee replacement,[110,111] although some higher-impact activity, such as tennis, may be well tolerated.[182]

HEMATOLOGIC CONDITIONS

Anemia

The most common hematologic condition encountered is anemia. Although specific anemias have certain concerns discussed below, the universal effect of mild to moderate anemia is a reduction in exercise capacity. As a rule, for every 1% fall in hematocrit, maximum oxygen consumption ($\dot{V}O_2max$) decreases 1% and endurance decreases by 2%.[28,57,78,201] Thus, anemic patients should be counseled that their ability to perform strenuous exercise will be less than that of traveling companions; they may not be able to keep up the same pace or hike as far.

Thalassemia Trait. Thalassemia trait is the most common inherited hemoglobin disorder and can be encountered in patients from diverse ethnic origins. β-Thalassemia trait is autosomal dominant and results in mild microcytic anemia (mean corpuscular volume [MCV] approximately 60 to 70 fL) and hematocrit in the 30% range. It is diagnosed by hemoglobin electrophoresis showing elevated hemoglobin A_2 in the presence of normal iron stores. The genetics of α-thalassemia trait are more complex, with the most common forms being electrophorectically silent and autosomal dominant.[160] α-Thalassemia trait leads to microcytosis (MCV approximately 70 fL) with hematocrit at the lower end of normal range. A severe recessive variant, hemoglobin H disease, predominantly affects people of Southeast Asian origin. This type of α-thalassemia results in hemolytic anemia with hematocrit that can be in the high 20% to low 30% range.

The presence of either α- or β-thalassemia trait does not lead to any specific problems except for the anemia. Diagnosis is important to avoid inappropriate therapy with iron and to establish the need for potential genetic counseling during family planning.

Iron Deficiency. Iron deficiency is very common, affecting up to 40% of women and 1% to 5% of men. In athletes, especially runners, the incidence is increased to 50% to 80% of women and 10% to 17% of men.[144,174] This increase in iron deficiency may reflect iron loss through GI bleeding,[30,35,136] sweat losses, urinary iron loss via hemolysis,[30,86] and blockage of iron absorption by inflammation.[157] Iron deficiency has multiple impacts on exercise ability. As previously noted, lower hematocrit decreases exercise ability. Lack of iron by itself has a negative effect on exercise beyond the decrement in hematocrit. Studies have shown that repletion of iron improves $\dot{V}O_2max$, exercise endurance, and strength, suggesting that lack of tissue stores of iron is detrimental.[21,22,24,73,174] This may be caused by iron deficiency first leading to loss of muscle iron stores because of the bone marrow iron demand.[25,108] Iron deficiency has been shown to impair cold tolerance, perhaps because of alteration in thyroid hormone metabolism.[9,20]

Going to altitude puts additional stresses on body iron stores. Although the initial increase in hematocrit at altitude is caused by contraction of plasma volume, RBC production increases several days later. Studies have shown that after as soon as 1 week at altitude, serum ferritin falls as iron is consumed to make

more RBCs.[12,89,165] For example, Berglund and associates[12] showed that after 10 days of breathing 14% oxygen, a 10% increase in hemoglobin was associated with a 46% decrease in ferritin.

Although serum ferritin is the best test for iron deficiency, the range of "normal" listed on the laboratory report may not be the appropriate level for athletes. Improvement can be seen in exercise ability and fatigue with the ferritin above 50 ng/mL.[25,205] Climbers with low ferritin levels may not be able to mount an adequate hematocrit response to altitude and may have impaired exercise ability. In one study, climbers with ferritin levels above 50 to 100 ng/mL were the ones who performed the best.[165] In theory, 250 mg of storage iron is required for every 1-g/dL increase in hemoglobin; that amount of iron is equivalent to 25 to 32 ng/mL of serum ferritin. It has been suggested that "ideal" hemoglobin for altitude should be 2.5 g/dL higher than the usual range; increasing the hemoglobin by that amount would require a serum ferritin level of 62 to 80 ng/mL.[12]

Iron replacement therapy should be prescribed for any person with a serum ferritin under 50 ng/mL who is planning an expedition, or if serum ferritin is under 100 mg/mL in a person planning a prolonged trip to altitude. Given that 100 mg of oral iron decreases iron deficiency and improves vigor in women undergoing basic training for 9 weeks, women undergoing major treks or expeditions can consider this option.[137] The best method of iron replacement is to start with a dose that contains at least 18 mg of elemental iron once a day; the gut cannot absorb more iron, and additional daily doses can lead to GI intolerance.[45] Taking the iron with 500 units of vitamin C can aid absorption. People taking iron should avoid certain foods, such as fiber and tea, within several hours of iron ingestion. Those who need iron replacement but who cannot tolerate or absorb oral iron can accomplish intravenous iron therapy.[65]

Hemolytic Anemias. Many patients have well-compensated congenital hemolytic anemias. The laboratory findings are mild to moderate anemia with elevated reticulocyte count, indirect bilirubin, and lactate dehydrogenase. RBC membrane defects, such as hereditary spherocytosis, are the most common causes of inherited hemolytic anemia.

Any patient with hemolysis is prone to folate deficiency, so adequate intake (400 to 800 mcg/day) should be part of nutrition planning. Patients with hemolytic anemia may have increased hemolysis with fevers. The incidence of gallstones is increased in these patients, so consideration should be given to obtaining a screening ultrasound before prolonged expeditions away from medical care.

Glucose-6-Phosphate Dehydrogenase Deficiency. G6PD deficiency is an important cause of hereditary hemolytic syndromes with wilderness implications.[13] G6PD deficiency is gender linked and most often affects males. The defect is in the hexose monophosphate shunt and renders the RBC unable to withstand oxidative stress. Most G6PD patients have hemolysis only with such stressors as infections and intake of oxidative drugs. The two main subtypes of G6PD deficiency are African and Mediterranean. In the African type, the enzyme is unstable, and older cells have diminished activity. Therefore, when these patients have hemolysis, it is self-limited because as the reticulocyte count increases, G6PD activity returns to normal. The Mediterranean type is caused by a defective enzyme and thus tends to be more severe because with oxidative stress, G6PD activity does not keep pace with the reticulocyte count increase, and fatal hemolysis may result. G6PD deficiency is of concern because many medicines used in travel medicine can lead to sudden and severe hemolysis (Box 53-2). Many patients are unaware that they are G6PD deficient; their first symptom may be fulminant hemolysis with antimalarial drugs. In the field, acute hemolysis manifests as back pain and dark urine. Management is stopping the suspect drug and hydrating the patient. Ingestion of fava beans (an oxidative stress) in patients with the Mediterranean type of G6PD deficiency may cause severe hemolysis, which may prove fatal.

Sickle Cell Anemia and Trait. About 8% of black persons and 0.08% to 0.5% of Caucasians have sickle cell trait.[67,132,185] It is mostly clinically silent, so there is a potential for problems in the wilderness. Sickling can occur in trait patients under moderately hypoxic conditions. Multiple case reports describe splenic crisis occurring at altitude or during airplane travel.[67,132] Patients present with acute onset of severe left upper quadrant pain that may have a pleuritic component, nausea, and vomiting. Therapy is descent, oxygen, and pain control. In cases of large splenic infarction, splenectomy may rarely be required. Curiously, Caucasians carriers of sickle cell trait are more susceptible to high-altitude splenic infarction.

BOX 53-2 Drugs That May Precipitate a Hemolytic Crisis with G6PD Deficiency

Acetanilide
Dapsone
Furazolidone
Isobutyl nitrate
Methylene blue
Nalidixic acid
Naphthalene
Nitrofurantoin
Pamaquine
Phenazopyridine
Phenylhydrazine
Primaquine
Quinolones
Sulfacetamide
Sulfamethoxazole
Sulfanilamide
Sulfapyridine

G6PD, Glucose-6-phosphate dehydrogenase.

Patients with sickle trait are more prone to dehydration because of a renal concentration defect. Maintenance of adequate fluid intake is very important. One study showed that just 45 minutes of exercise without fluids led to sickling in trait patients walking briskly in warm (33°C [91.4°F]) weather.[11] Because patients cannot maximally concentrate their urine, the color of the urine cannot be used as a guide to hydration. If patients with sickle trait develop diarrhea or vomiting, close attention should be paid to hydration status.

Concern about the risk for "sudden death" in people with sickle cell trait with strenuous exercise is valid.[54,177] Data collected in the 1960s from army recruits in boot camp showed a 28-fold increased risk for death (absolute risk, 1:3200).[183] Review of these cases revealed that most patients had heatstroke, with resulting severe rhabdomyolysis causing their deaths. Practical advice for patients with sickle cell trait is to be cautious about dehydration and to avoid strenuous exercise in the heat, especially if they are deconditioned. Cramping or especially weakness of the legs can be an early warning sign of sickling and a sign to immediately stop exercising and begin to rehydrate.[55,177]

Patients with sickle cell anemia often have end-organ damage that may complicate wilderness travel. Older patients may have chronic pain syndromes because of multiple bony infarcts. The leading causes of overall mortality in adults are complications of pulmonary hypertension, which result in hypoxia and impair lung function. Pain crises are unpredictable and can be provoked by heat extremes, dehydration, or hypoxia—all features found in wilderness travel.

Patients with sickle cell anemia who are going on limited trips to the wilderness need to be reminded about the importance of adequate fluid intake and to bring sufficient pain medicine to manage both chronic pain and any crisis.[190] When considering longer-term or adventure travel, patients should be screened for pulmonary hypertension. Physician documentation about the patient's narcotics supply and needs should be carried. Patients should be reminded to comply with folic acid supplementation. Because most adults with sickle cell anemia are functionally asplenic, they should take the same precautions as outlined later for asplenic patients.

Hemophilia

Deficiency of factor VIII or IX occurs in 1 of 5000 males.[189] Patients with severe hemophilia are at risk for severe bleeding, even with minor trauma. Bleeding most often occurs in muscles

TABLE 53-8 Guidelines for Factor Replacement

Site of Bleed	Hemostatic Level	Hemophilia A	Hemophilia B
Joint	80% acutely, then 40% every other day until resolved	40 units/kg initially, then 20 units/kg every other day until healed	80 units/kg initially, then 40 units/kg every other day or third day as needed
Muscle	40%-50%	20-40 units/kg per day until healed	40-60 units/kg, then 20-30 units/kg every other day as needed
Oral	100%*	50 units/kg*	100 units/kg*
Nose	Initially 80%-100%, then 30% until healing	40-50 units/kg, then 30-40 units/kg per day	80-100 units/kg, then 35-40 units/kg per day
Gastrointestinal	Initially 100%, then 50% until healing	50 units/kg, then 30-40 units/kg per day	100 units/kg, then 30-40 units/kg per day
Genitourinary	Initially 100%, then 30% until healing	50 units/kg, then 30-40 units/kg per day	100 units/kg, then 30-40 units/kg per day
Central nervous system	Initially 100%, then 50%-100% for 14 days	50 units/kg, then 25 units/kg every 12 hr	100 units/kg, then 50 units/kg per day
Surgery/trauma	Initially 100%, then 80%-100% until wound healing begins, then 30% until suture removed	50 units/kg, then 40-50 units/kg every 12 hr adjusted according to healing	100 units/kg, then 50 units/kg per day adjusted according to healing

Modified from DiMichele D, Neufeld EJ. Hemophilia. A new approach to an old disease, *Hematol Oncol Clin North Am* 12(6):1315-1344, 1998.
Note: For severe or persistent minor bleeding, factor levels should be followed.
*Antifibrinolytic agents are useful for oral bleeding.

and joints, but intracranial hemorrhage is the leading cause of fatal hemorrhage. Patient can have severe arthritis because of repeated bleeding. A general rule is that patients with less than 1% of normal levels of factor VIII or IX can have spontaneous bleeding, those with 1% to 15% of normal levels may have bleeding with minor trauma, and those with greater than 15% of normal factor levels may have bleeding with major trauma. Factor concentrates to correct the coagulation defects are available; Table 53-8 provides a suggested dosing scheme. The World Federation of Hemophilia website is an invaluable guide to resources available for the traveler in many different countries (http://www.wfh.org).

Hemophilia does not preclude travel or expedition travel. However, patients with hemophilia who are planning travel should take factor replacement with them, especially if they will be away from health care facilities. Only certain factors listed in Box 53-3 can be stored without refrigeration. The patient should also have supplies, such as alcohol wipes and needles, needed to inject the factors. At least one member of the traveling party should be trained to infuse factor in case the patient is incapacitated.

The greatest functional limitation to the hemophilia patient is joint disease. Arthritis from joint bleeds is common and can be disabling. Joint bleeds can occur with minor injuries or ankle twists. Before travel, patients should work to strengthen their muscles to provide joint protection. Simple measures to prevent ankle sprains, such as wearing high-top boots, should be taken. Damaged joints should be splinted to prevent reinjury.

BOX 53-3 Factor Concentrates That Do Not Require Refrigeration

Factor VIII Concentrates
Alphanate
Eloctate
Hemofil M
Koāte-DVI
Monarc-M
Recombinate
ReFacto

Factor XI Concentrates
AlphaNine SD
Alprolix
BeneFix
Mononine
Profilnine SD

Bypass Factor for Factor VIII or IX Inhibitors Patients
NovoSeven (rFVIIa)

Von Willebrand Disease

The most common inherited bleeding disorder is von Willebrand disease; this may affect up to 1 in 10,000 people. It is characterized by easy bruising and mucocutaneous bleeding. Joint bleeding is unusual for most patients. Patients with von Willebrand disease can have significant bleeding with trauma. There are multiple types of von Willebrand disease, but the 80% of patients with type 1 respond to desmopressin, which is available as a nasal spray (Stimate); the dose is one squirt in each nostril, with the effect lasting up to 24 hours. One needs to be specific when prescribing Stimate, because the generic nasal desmopressin used for enuresis has insufficient desmopressin per dose to be effective for bleeding disorders. Estrogen can also raise levels of von Willebrand factor; use of oral contraceptives can normalize levels in mildly affected women. The patient with rarer types of von Willebrand disease may require specific replacement therapy with Humate-P, a factor concentrate that contains von Willebrand factor. This concentrate must be kept refrigerated.

Thrombocytopenia

Immune thrombocytopenia is the most common autoimmune hematologic disease, affecting 1 in 50,000 people. Patients with modest (>20,000 platelets/μL) thrombocytopenia are not at greater risk for bleeding except with extreme trauma, so no specific precautions are needed. Patients with chronic immune thrombocytopenia and stable platelet counts can be cleared for wilderness travel. For the patient with a history of recurrent severe thrombocytopenia who is planning prolonged travel, a dose of "pulse" dexamethasone (40 mg orally for 4 days) can be prescribed to be carried and started if the patient notices sudden onset of petechiae and bleeding.

There are multiple causes of congenital thrombocytopenia and platelet function defects. Some patients have mild thrombocytopenia with no bleeding defects, whereas other may have both platelet number and function defects. One group of such patients is those with mildly decreased platelets (75,000 to 150,000/μL) but no bleeding defects. These patients require no special precautions. Most patients with congenital platelet defects have a mild bleeding diathesis. Many of these patients respond to desmopressin and can carry the nasal form (Stimate) with them.

Anticoagulation

Indications for chronic anticoagulation are increasing, especially because of recommendations for lifelong anticoagulation for

idiopathic and recurrent deep vein thrombosis (DVT). Patients on vitamin K antagonists such as warfarin pose special challenges.[168] The risk of anticoagulated travelers bleeding in one study was 6.5%, with a 0.9% risk of thrombosis.[167] A varied diet, increased exertion, and potential travel-related illness can dramatically alter the level of anticoagulation. Unless the patient has access to a point-of-care international normalized ratio (INR) monitor, there may be no way to monitor the level of anticoagulation.

Well-controlled patients with stable INR can safely go several weeks without an INR check. This assumes that their diet is the same as their regular diet and there are no concurrent illnesses. For patients who plan to travel longer or for those who will be away from health care, the use of point-of-care INR monitors is advised. Patients should know how to operate the machine and should be able (or have a nomogram) to adjust their warfarin dose. Table 53-9 provides a suggested nomogram. Vitamin K tablets should be carried to allow treatment of very high INR.

There is little consensus about the type of activities in which patients taking anticoagulants should participate.[88] A commonsense approach would be to avoid activities with high potential for major body contact and to wear a helmet when there is a risk of head injury. Furthermore, others should be prepared to evacuate emergently any anticoagulated patient after head trauma caused by risk of intracranial hemorrhage.

In the past 4 years, direct oral anticoagulants (DOACs) have been approved for use in treatment and prevention of thromboembolic events[166] (Table 53-10). These agents have the advantage of fixed dosing, no monitoring requirements, and no food and minimal drug interactions. Disadvantages are the inability to use them in dialysis patients and patients with mechanical heart valves. Concerns about "irreversibility" have been shown not to be valid, because outcomes after bleeding are the same or better as those in patients bleeding while taking vitamin K antagonists. Abundant data show the overall risk of fatal and serious bleeding is less with DOACs.[33] A traveler taking a DOAC should ensure carrying an adequate supply. For patients receiving vitamin K antagonists who are planning long trips, one option is to change to a DOAC for travel and then resume the vitamin K antagonist on their return, to avoid the challenges of INR monitoring.[166]

TABLE 53-9 Maintenance Warfarin Adjustment Nomogram

INR	Dose Change
1.1-1.4	*Day 1*: Add 10%-20% TWD *Weekly*: Increase TWD by 10%-20% *Return*: 1 week
1.5-1.9	*Day 1*: Add 5%-10% of TWD *Weekly*: Increase TWD by 5%-10% *Return*: 2 weeks
2.0-3.0	No change *Return*: 4 weeks
3.1-3.9	*Day 1*: Subtract 5%-10% TWD *Weekly*: Reduce TWD by 10%-20% *Return*: 2 wk
4.0-5.0	*Day 1*: No warfarin *Weekly*: Reduce TWD by 10%-20% *Return*: 1 week
>5.0	Stop warfarin until INR <3.0 Decrease TWD by 20%-50% *Return*: Daily

Modified from DeLoughery TG: Oral anticoagulants. In Goodnight SH, Hathaway WE, editors: *Disorders of hemostasis and thrombosis: A clinical guide*, New York, 2001, McGraw-Hill Professional, pp 533-566.
INR, International normalized ratio; *TWD*, total weekly dose.

Travel Thrombosis

Airplane travel has been considered a risk factor for DVT for decades, but only recently has flight been rigorously studied as a DVT risk factor. Case-control studies suggest a relative risk for thrombosis of threefold to fourfold with prolonged (>4 to 6 hours) travel, with a higher risk for travel times over 8 hours.[1,34,103,130,195] The absolute risk for thrombosis is uncertain. For example, the overall risk for symptomatic pulmonary embolism is estimated to be 0.4 per 1 million passengers, rising to 4 per million in the highest-risk group. One study showed a DVT risk of 1 in 4600 following flight.[109,115,158] In contrast, small prospective trials showed a calf vein thrombosis rate of 2% to 10%.[179,180] The presence of risk factors, such as history of DVT or underlying hypercoagulable state, is important. Up to 70% to 90% of persons with thrombosis had other risk factors.[97,133]

Pathogenesis of "traveler's thrombosis" is controversial. Venous stasis appears to be the primary risk factor. Prolonged sitting is a risk factor for thrombosis, perhaps by increasing venous stasis. Although mild hypoxia caused by the low air cabin pressure is often blamed in the popular press, there are currently no data to show activation of coagulation with mild hypoxic

TABLE 53-10 Direct Oral Anticoagulants

	Apixaban	Dabigatran	Edoxaban	Rivaroxaban
Mechanisms	Xa inhibition	IIa inhibition	Xa inhibition	Xa inhibition
Half-life	12 hours	12-17 hours	9-11 hours	5-9 hours
Dosing	Acute VT: 5 mg bid (after 7 days of 10 mg bid) Prophylaxis: 2.5 mg bid AF: 5 mg bid	Acute VT: 150 mg bid (after 5 days of heparin) AF: 150 mg bid	Acute VT: 60 mg daily (after 5 days of heparin) Prophylaxis: 15 mg bid AF: 60 mg daily	Acute VT: 20 mg daily (after 21 days of 15 mg bid) Prophylaxis: 10 mg daily AF: 20 mg daily
Drug interactions	Azoles HIV protease inhibitors	Azoles Dronedarone	Azithromycin Azoles Clarithromycin Cyclosporine Erythromycin HIV protease inhibitors Quinidine Verapamil	Azoles HIV protease inhibitors
Renal dosing	2.5 mg bid if two of these three: age over 80, creatinine >1.5, weight less than 60 kg (132 lb) Contraindicated if CrCl <15	Dose reduced to 75 mg bid if CrCl <30 Contraindicated if CrCl <15	30 mg/day if CrCl 30-60 Contraindicated if CrCl <30	Dose reduced to 15 mg/day if CrCl 50-15

AF, Atrial fibrillation; *bid*, twice daily; *CrCl*, creatinine clearance; *HIV*, human immunodeficiency virus; *VT*, venous thrombosis.

BOX 53-4 Risk Factors for "Traveler's Thrombosis"

Older than 65
Cancer
Carrier of a hypercoagulable state
Estrogen use
History of lower-extremity deep vein thrombosis
Lower-leg cast
Obesity (>2 × ideal body weight)
Pregnancy
Recent surgery (previous 6 weeks)

exposure.[46] Preexisting risk factors for thrombosis are important. Most studies indicate that people who develop travel-related thrombosis have other risk factors, such as history of thrombosis or estrogen use. The presence of long travel and these thrombotic risk factors raise the risk for thrombosis by 16-fold.[133]

The best method of prophylaxis is still to be determined. Ideally, people should try to be up and exercising their legs at least once an hour. However, given crowded cabins and the current security climate, this is unrealistic. Aspirin remains a popular recommendation but has been demonstrated in a randomized trial not to be effective for preventing traveler's thrombosis and thus should not be relied on for this purpose.[32,77,102] Elastic stockings provide protection in trials but have the side effect of discomfort and superficial thrombosis.[180] A single prophylactic dose of a DOAC, such as 10 mg of rivaroxaban, before the flight is an approach that can be used for high-risk patients.

A reasonable approach is first to assess a patient's risk for thrombosis when traveling by plane for over 6 hours (Box 53-4). For most low-risk people, one could encourage foot movement and avoiding dehydration. For medium- and high-risk patients, stockings (knee-high, 20 to 30 mm Hg compression pressure) should be recommended. In addition, the feasibility of adding a DOAC on a case-by-case basis for high-risk patients should be considered.

The Asplenic Patient

Asplenic patients pose unique travel risks. Asplenic patients may have no other health problems but are at risk for overwhelming infections from a diverse group of infectious agents.[175] The risk for overwhelming sepsis varies by indications for splenectomy, but appears to be about 1%.[14,112] Use of pneumococcal vaccine and recognition of this syndrome have helped lessen the risk, but travel can expose asplenic persons to novel (for them) infectious organisms.

The classic bacterial pathogen is the pneumococcus, but in older patients, gram-negative bacteria are a common cause of infections.[56] Unusual organisms causing overwhelming infections, such as *Capnocytophaga* from dog bite or babesiosis from tick bite, have been reported.[47,213]

Asplenic patients need to be counseled about the risk for overwhelming infections and should be vaccinated against pneumococcus, meningococcus, and *Haemophilus influenzae*.[85,142,175] Patients previously vaccinated for pneumococcus should be revaccinated every 3 to 5 years.[112] In addition, because of concerns about greater severity of malaria, they should be scrupulous with antimalarial prophylaxis.[131,148] The role of prophylactic antibiotics is controversial for adults, but patients under age 18 should receive penicillin VK, 250 mg orally twice daily. Patients should be advised to start antibiotics and seek medical care if they have a fever, shaking chills, or lower respiratory tract infections. A reasonable oral antibiotic for self-medicating is amoxicillin/clavulanic acid, 875 mg twice daily.[40] Patients who are bitten by dogs should also take antibiotics.[175] Asplenic patients should wear an identification bracelet to alert health care providers.[60]

ONCOLOGY

The numbers of cancer survivors is increasing, and many of these patients pursue wilderness activities. Chemotherapy can lead to short-term side effects such as nausea and neutropenia, but many side effects can last months or years after chemotherapy has ended.

Chemotherapy

Common side effects of most chemotherapeutic agents are nausea and marrow suppression. The nausea is short term and managed with 5-hydroxytryptamine blockers. The highest-risk time for neutropenia is usually 10 to 21 days after onset of chemotherapy. Patients may want to hike or go camping between chemotherapy sessions. Neutropenic patients can be cleared for this type of activity as long as they can seek medical care if they become febrile. A good rule of thumb is that patients should not be more than 2 hours away from an ED. The vast majority of infections in these patients result from endogenous organisms and not from the environment, but some precautions, such as wearing a mask when in crowded areas and avoiding ingestion of fresh fruits or vegetables, should be suggested. Thrombocytopenia is rarer with most chemotherapy regimens. Platelet transfusion should be performed for platelet count under 10,000/μL.

Chemotherapy can cause a variety of long-term side effects (Box 53-5). Anthracyclines such as doxorubicin (Adriamycin) can lead to cardiac damage that may be well compensated until another stressor, such as altitude, is added. The risk for chronic heart failure is highest with doses over 300 mg/m^2 but can be seen with any dose. Agents such as vincristine or bortezomib can lead to neuropathy that causes loss of dexterity.

Of particular concern is bleomycin, an antineoplastic agent that is part of a curative regimen for both germ cell tumors and Hodgkin's disease. Bleomycin can lead to subtle lung damage that results in loss of diffusing capacity. For very competitive athletes with germ cell tumors, effective non–bleomycin-containing regimens can be used. Bleomycin can also interact with high concentrations of oxygen to cause fulminant lung toxicity. Fatal cases have been reported up to many months after cessation of therapy. Controversy remains as to whether patients who have had bleomycin can safely go scuba diving because of the high pressures of oxygen to which they will be exposed. For example, during a dive to 20 m (66 feet), the P_{IO_2} is 0.63 atm. It will be 0.84 atm at 30 m (99 feet), potentially leading to lung toxicity.[99] This notion has recently been challenged, such that diving can be considered for patients 6 months after receiving bleomycin if they did not suffer pulmonary toxicity.[48] Patients who have received bleomycin should wear an identification bracelet in case emergency surgery is needed, so that the lowest possible oxygen concentration is used.

The cancer survivor planning to go on a high- or extreme-performance expedition should undergo careful screening.

BOX 53-5 Chemotherapy Agents: Long-Term Side Effects

Cardiac Dysfunction
*Anthracyclines (doxorubicin, daunorubicin, epirubicin, mitoxantrone, idarubicin)

Pulmonary Toxicity
*Bleomycin
*Radiation therapy
*Nitrosoureas
Cyclophosphamide
Methotrexate

Neuropathy
*Bortezomib
Cisplatin
*Oxaliplatin
*Vincristine
*Taxol

Raynaud's Phenomenon
Bleomycin
Cisplatin

*Common.

Patients who have received anthracyclines should have cardiac function screened, and pulmonary function needs to be checked in patients who have received bleomycin or other potential pulmonary toxic agents. Any patient who received upper chest or neck radiation should have thyroid function screened.

Certain agents used for hematologic malignancies, especially chronic lymphocytic leukemia, can lead to profound and prolonged T cell immunosuppression that persists for months to years and puts patients at risk for unusual infections. These agents are fludarabine, cladribine, and alemtuzumab. Measuring CD4 counts can monitor the degrees of immunosuppression. Patients with counts under 200/μL are severely immunocompromised, and travel is not recommended.[36,176] Patients with higher CD4 counts still may have residual immunosuppression and may be prone to herpes zoster outbreaks for years after chemotherapy.

Increasingly, patients are using oral "targeted" agents for cancer, such as imatinib for chronic myelogenous leukemia and sorafenib for renal tumors. Patients need to be sure that they have an adequate supply of these medications for the trip and "guard" them closely, because they are not available in most countries and often cost several thousands of dollars for a month's supply. Certain agents, especially vemurafenib and dabrafenib, can be associated with photosensitivity, so patients on these agents need to use sunscreen and protect themselves from intense sunlight (Box 53-6).

BOX 53-6 Targeted Antineoplastic Agents Associated with Phototoxicity

Less Common (<10%)
Axitinib
Cabozantinib
Cetuximab
Dasatinib
Erlotinib
Gefitinib
Imatinib
Lapatinib
Nilotinib
Panitumumab
Pazopanib
Pertuzumab
Ponatinib
Regorafenib
Sorafenib
Sunitinib
Trametinib
Vandetanib

Very Common (>30%)
Dabrafenib
Vemurafenib

Modified from http://www.cancernetwork.com/cancer-management/dermatologic-adverse-events-associated-systemic-anticancer-agents.

Stem Cell ("Bone Marrow") Transplantation

Stem cell transplantation is an increasingly common therapy. Currently, three different types of stem cell transplants are performed. Autologous transplants involve harvesting and preserving the patient's stem cells and then administering radiation and/or chemotherapy (conditioning) to ablate the marrow. This is followed by reinfusion of the stored stem cells. Allogeneic transplant involves infusion of another person's marrow. Increasingly, nonmyeloablative allogeneic transplants ("mini-transplants"), which involve only modest conditioning, are used. "Conditioning" is a course of chemotherapy or radiation therapy intended to eradicate the immune system and any cancer in preparation for a bone marrow transplant. The major long-term complication of allogeneic transplant is graft-versus-host disease (GVHD) from the donor immune system attacking the recipient. Chronic GVHD can be debilitating and lead to chronic skin disease, restrictive pulmonary disease, and increased propensity for infection.

When counseling the stem cell transplant patient about travel, one needs to know what type of transplant the patient received, time since transplant, and whether they have GVHD. In general, a patient who has received an autologous transplant and has recovered his or her blood counts can be cleared for travel 6 months after transplant. For the patient who has received an allogeneic transplant, the main concerns are restoration of immunity and presence of GVHD. A reasonable rule of thumb is that patients will be immunosuppressed for 12 to 24 months after they stop taking immunosuppressants and should always be treated as functionally asplenic. The transplant (especially allogeneic) patient who is considering a high- or extreme-performance trip should undergo pulmonary and cardiac screening.

Timing of travel immunizations for transplant patients is an uncertain area.[40,123] If given too soon, the immature immune system will not mount a response. Live vaccines are also a concern if immune function is not normal. Table 53-11 offers one approach.

TABLE 53-11 Vaccine Recommendations After Bone Marrow Transplant

Vaccine	Allogeneic Transplant	Autologous Transplant	Timing	Live
Tetanus toxoid	Recommended	Recommended	6-12 mo	
Diphtheria	Recommended	Recommended	6-12 mo	
Inactivated poliovirus	Recommended	Recommended	6-12 mo	
Pneumococcal	Recommended	Recommended	6-12 mo	
H. influenzae	Recommended	Recommended		
Influenza	Recommended	Recommended		
Measles	No earlier than 24 mo			Yes
Rubella	No earlier than 24 mo			Yes
Hepatitis B			6-12 mo	
Hepatitis A			6-12 mo	
Inactivated polio			6-12 mo	
Oral polio	No earlier than 24 mo			Yes
MMR	No earlier than 24 mo			Yes
Typhoid (tY21a)	No earlier than 24 mo			Yes
Varicella	No earlier than 24 mo			Yes
Yellow fever	No earlier than 24 mo			Yes
Japanese encephalitis V			6-12 mo	
Meningococcal vaccine			6-12 mo	

From Committee to Advise on Tropical Medicine and Travel (CATMAT): The immunocompromised traveler: An Advisory Committee Statement(ACS), *Can Commun Dis Rep* 33:1, 2007; and Ljungman P, Cordonnier C, Einsele H, et al: Vaccination of hematopoietic cell transplant recipients, *Bone Marrow Transplant* 44:521, 2009.

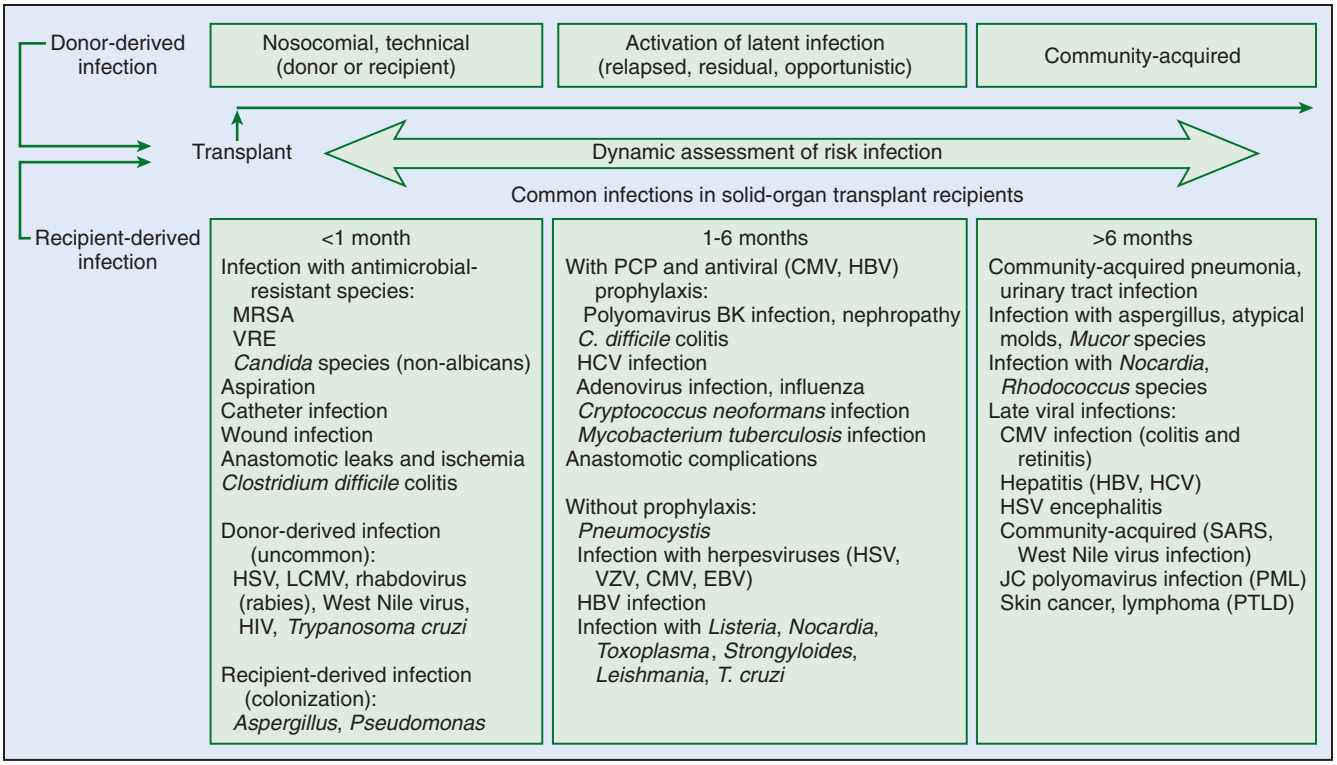

FIGURE 53-3 Changing timeline of infection after organ transplant. Infections occur in a generally predictable pattern after solid-organ transplant. The development of infection is delayed by prophylaxis and accelerated by intensified immunosuppression, drug toxic effects that may cause leukopenia, or immunomodulatory viral infections such as infection with cytomegalovirus (CMV), hepatitis C virus (HCV), or Epstein-Barr virus (EBV). At transplantation, a patient's short-term and long-term risks for infection can be stratified according to donor and recipient screening, the technical outcome of surgery, and the intensity of immunosuppression required to prevent graft rejection. Subsequently, ongoing assessment of the risk for infection is used to adjust both prophylaxis and immunosuppressive therapy. *HBV,* Hepatitis B virus; *HIV,* human immunodeficiency virus; *HSV,* herpes simplex virus; *LCMV,* lymphocytic choriomeningitis virus; *MRSA,* methicillin-resistant *Staphylococcus aureus; VRE,* vancomycin-resistant *Enterococcus faecalis; PCP, Pneumocystis carinii* pneumonia; *PML,* progressive multifocal leukoencephalopathy; *PTLD,* post-transplantation lymphoproliferative disorder; *SARS,* severe acute respiratory syndrome; *VZV,* varicella-zoster virus. *(Data from Fishman JA: Infection in solid-organ transplant recipients,* N Engl J Med *357:2601, 2007.)*

Patients with stem cell transplants who remain on immunosuppression have special concerns. It should be remembered that their family members and close contacts should also not receive live vaccines because of concern about disease transmission to these patients.[184] New medications can interfere with immunosuppression, so consideration should be given to starting any new medications (e.g., malaria prophylaxis) at home to monitor levels of immunosuppressive medications.

SOLID-ORGAN TRANSPLANT RECIPIENTS

Solid-organ transplant recipients include persons who have received heart, lung, kidney, liver, or pancreas transplants. Concerns for the solid-organ transplant recipient engaging in wilderness activities include risk for infection, availability of local medical care knowledgeable about transplant recipients when traveling outside the United States, carrying adequate supplies of medication and storage of medications on extended trips (immunosuppressive medications and prophylactic medications for infection), and increased level of physical activity.

Improvements in immunosuppressive agents have decreased the incidence of rejection of transplanted organs in solid-organ transplant recipients but have increased the risks for infection and malignancy.[66] Newer immunosuppressive approaches, including use of sirolimus, mycophenolate mofetil, and T cell and B cell depletion, have largely replaced high-dose corticosteroids and azathioprine. Sources of infections include donor-derived infections, recipient-derived infections, nosocomial infections, and community-acquired infections. A predictable timeline for risk for infection is dictated by the time from transplant and the immunosuppressive regimen (Figure 53-3). In the first month after transplant, donor-derived infection, recipient-derived infection, and nosocomial infection are the categories for infection risk. In the first 6 months after transplantation, infectious risk is determined by whether *Pneumocystis* (trimethoprim-sulfamethoxazole) and antiviral prophylaxis (valacyclovir, high-dose acyclovir, ganciclovir, or valganciclovir) medications are used. After 6 months, the primary risk is community-acquired infections, some of which are "unusual" pathogens (e.g., *Aspergillus,* atypical molds, *Mucor* spp., *Nocardia*, *Rhodococcus*, rare viruses).

Wilderness activities and travel in rural areas or low-income countries can increase risk for exposure to potential infectious pathogens. Transplant recipients should wash their hands after food preparation and contact with soil or feces. They should avoid well water and lake water (which may contain *Cryptosporidium* or *Giardia* spp.), undercooked meats, unwashed fruits and vegetables, and unpasteurized dairy products (which may contain *Escherichia coli* or *Listeria monocytogenes).*[66] Despite these concerns, solid-organ transplant recipients can travel safely outside the United States or Canada. In one study of 303 solid-organ transplant recipients, 94% of whom were further than 1 year after transplant, only 24 (8%) became ill during travel.[202] The most common illness was upper respiratory infection. Illness during travel was more common among those who visited high–infection risk areas (18.4% became ill during travel to Asia, Central or South America, Africa, or the Middle East), compared with low–infection risk areas (6.1% became ill). No travelers were diagnosed with "tropical" infections. Three of 24 ill travelers

returned home early. Two travelers to high–infection risk areas developed transplant-related illness, one with post-transplant lymphoproliferative disease and the other with acute allograft rejection. Only 18% of travelers in this series were in rural areas, and only one traveler was camping, so risk for infection may be greater during some wilderness activities.

Vaccination is an important element of prevention of infection for transplant recipients. Immunization status should be carefully reviewed before transplant to ensure that all immunizations are up to date and any needed vaccinations administered while the patient is immunocompetent. Vaccination is generally less effective during immunosuppression. Pneumococcal vaccine is recommended every 3 to 5 years, and influenza vaccine is recommended annually. The immunologic protection from vaccines may be limited. Other vaccines are appropriate for patients who travel to regions where certain illnesses are endemic, and patients should seek advice from transplant and travel medicine specialists. Live vaccines are generally contraindicated after transplantation, because they may cause disseminated infection in immunocompromised hosts.[66]

Wilderness activities usually require increased exercise; solid-organ transplant recipients in general have lower-than-predicted exercise capacity.[191] This includes recipients of heart, lung, liver, and kidney transplants. Although $\dot{V}O_2max$ is decreased, cardiopulmonary function is appropriate for the level of work performed. Cardiac output is normal in response to exercise, oxygen desaturation does not occur, and ventilation is not a limiting factor. Anemia may play a role in decreasing oxygen-carrying capacity, although the degree of anemia is generally mild and does not explain the decrease in exercise capacity. Peripheral muscle work capacity and oxygen utilization by the tissues are proposed as causes of decreased exercise capacity. Indeed, many solid organ transplant recipients have endured long illnesses before transplant and have deconditioned muscle. Supervised exercise programs improve $\dot{V}O_2max$, but it still may be lower than predicted.[191] Immunosuppressive drugs have also been suggested as a cause of peripheral myopathy. Despite results from studies showing decreased maximal exercise capacity in solid-organ transplant recipients, clearly some are capable of high-performance athletic endeavors, including mountaineering, competitive recreational sports, and even competition at the Olympic level. Most transplant recipients return to a level of function at least as good as before transplant.

In general, solid-organ transplant recipients are capable of pursuing wilderness activities but should consult with their transplant specialist medical providers for recommendations on specific issues. If traveling outside the United States, transplant recipients should wait until their immunosuppressive regimen is stable for at least 6 months after transplant and consult with a travel medicine specialist for specific vaccination and prophylaxis recommendations.

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