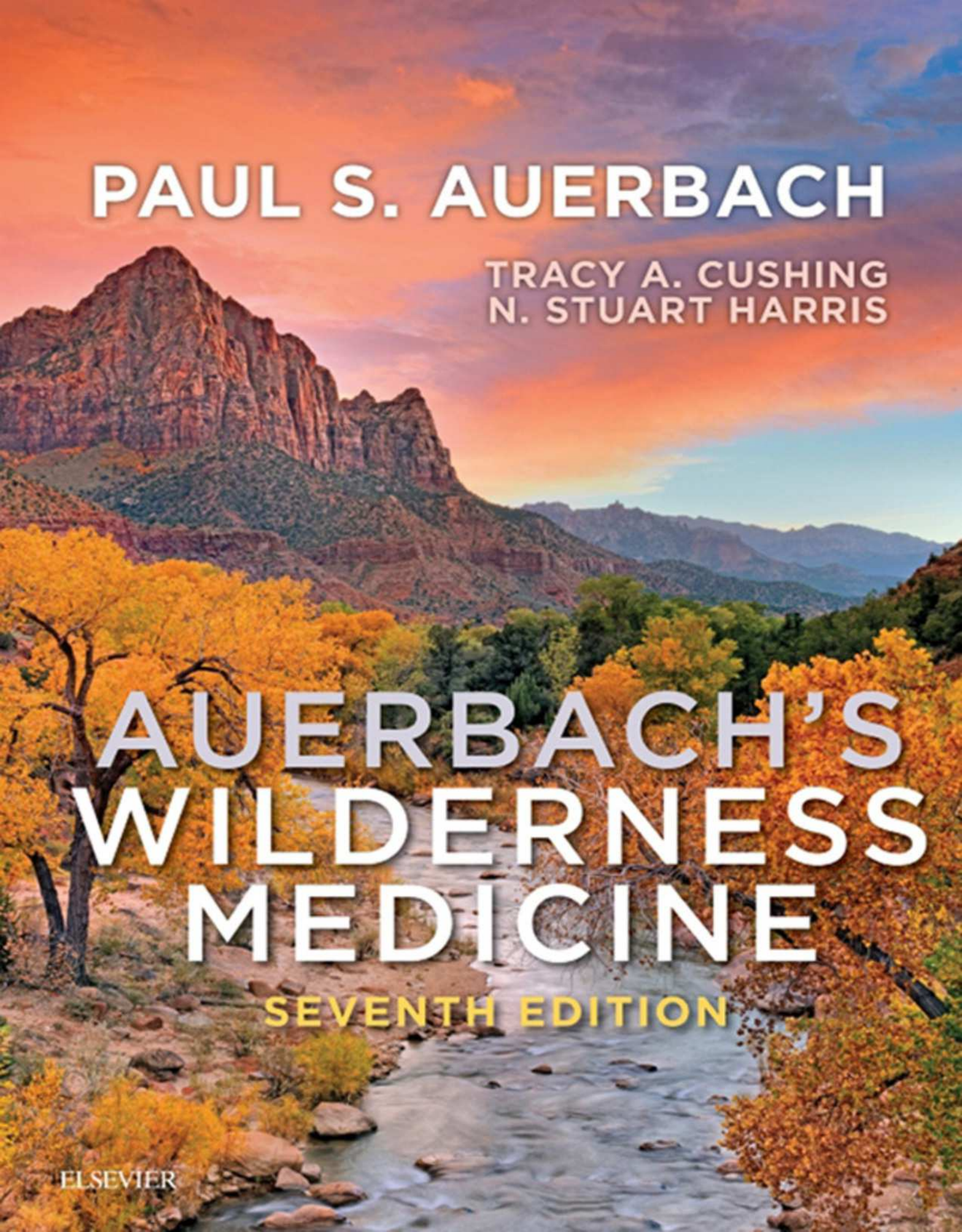
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# Foreword

Before partaking of an urban existence, men, women, and children lived in austerity in the wilderness. So, the human race is not encountering wilderness medicine for the very first time. Before the advent of such wonders as antisepsis, randomized clinical trials, and emphasis on evidence, and therefore throughout most of the history of human existence and eventually, civilization, the practice of medicine was largely improvised and based on anecdotes and dogma, rather than evolving science. Given that humans had to make do with little or nothing before they had access to tests, drugs, and devices, the history of wilderness medicine might be considered to largely be the history of medicine itself. Advances in medicine have in general paralleled other sciences, with periodic insights into its essences, but there remain geographies and circumstances where wilderness medicine is uniquely essential.

We are in the midst of a scientific revolution, but recognize that optimal urban science is not necessarily applicable in austere environments. So, as we intentionally place ourselves in wild places isolated from cities and machines, wilderness medicine comes full circle and needs to remain different in certain ways from big city medicine. From this perspective, one might identify many potential starting points for our 21st century iteration of wilderness medicine. However, thoughtful reflection identifies two prominent threads. First, today's wilderness medicine "began" when urban, high-resource medicine became too sophisticated to practice in austere environments—when improvisation was required to replace more sophisticated methodology that was not available. Second, and very significant from a definition standpoint, wilderness medicine gained true identity when men and women began in earnest to explore environments that stressed normal human physiology to the point that unique pathophysiology was discovered. This phenomenon notably occurred with human endeavors at high altitude (mountaineering and aviation), under the ocean surface (diving), at extremes of temperature (cold and heat), and at the limits of endurance posed by natural disasters or forays into the ultimate frontier of space travel.

The history of wilderness medicine could be a textbook unto itself. The following paragraphs attempt to provide key examples of how the evolution of modern medicine simultaneously drew from and shaped the specialty.

Military medicine provides many examples of this interaction. For much of history, the greatest threats to soldiers were not battlefield combatants, but weather and infectious diseases. During the American Revolutionary War, 6,200 American soldiers were killed in action, while 10,000 died of disease. Typhus, smallpox, dysentery, diarrhea, and pneumonia were prevalent. The War of 1812 generated 2,200 American combat deaths and nearly 13,000 deaths from noncombat causes. Napoleon invaded Russia in 1812 with 680,000 soldiers, and retreated back to France five months later with 27,000. Most of the remainder had succumbed to hypothermia, frostbite, and typhus. It wasn't until World War II that the number of soldiers killed in combat outnumbered those who died from other causes, many of them environmental.

*"Medicine Under Sail,"* Zachary Friedenberg's history on the subject,[1] chronicles an oft-overlooked perspective of the early history of medicine. In the same vein, Homer made reference in book 4 of the *Iliad* to a medical naval incident in the Trojan War.[2] When Menelaus was wounded by a Trojan bowman, the fleet surgeon, Machaon (son of Aesculapius, god of medicine), was called to treat the wound:

> ***Without delay he drew***
> ***the arrow from the fairly fitted belt.***
> ***The barbs were bent in drawing.***
> ***Then he loosed the plate—the armorer's work—and carefully***
> ***O'er looked the wound where fell the bitter shaft.***
> ***Cleansed it from blood, and sprinkled over it***
> ***with skill the soothing balsam of yore which***
> ***the friendly Chiron to his father gave.***

Thomas Woodall (1569-1643) perhaps deserves the title "Father of Marine Medicine" because he was ahead of his time with observations of scurvy and views on the treatment of wounds, fractures, and amputations.[1] His extensive practical experience, astute observations, and cautious judgment persuaded him that the theories of oracles, such as Galen, often offered little in the way of useful medical knowledge. As stated in his 1655 book *The Surgeon's Mate*, Woodall divided wounds into three categories: (1) puncture wounds and lacerations, (2) gunshot wounds, and (3) bone fractures.[3] His treatment recommendations have a modern ring: "…remove unnatural things forced into the wound… which should be done with the least pain to the patient and avoiding arteries, nerves, and veins." The "unnatural things" to which he referred might include wood splinters from spars and masts, fragments from cannon fire, and other foreign objects embedded into people during commerce and conflict. Anesthesia was nonexistent in this era. In the case of removal being too difficult or painful, Woodall recommended "tarry if you may, while nature helps." His suggestions to ligate specific vessels that contributed to excessive bleeding and to place dressings soaked in wine over wounds were significant departures from the usual treatment of the day, which was wound cauterization with hot oil or a red-hot searing iron.

When limb wounds were severe, Woodall was not in a rush to amputate. This approach ran counter to the prevailing custom and for several hundred years afterward. Woodall reasoned that the need for amputation should be dictated by specific criteria: one-half or more of the limb should have been dismembered or irreparably damaged; a chronic suppurating wound be present; the patient's life be imminently in danger; or the remaining portion of the limb be unserviceable. His concepts were far more conservative and reasonable than those of military surgeons who practiced for the next two centuries, such as during the American Civil War, where immediate amputation of any limb with a gunshot wound was the customary practice. Woodall's conservative principles from three centuries past seem reasonable for modern physicians providing care for trauma patients in harsh or remote environments.

It was Admiral Horatio Nelson, a senior nonmedical officer in the British Royal Navy, who near the turn of the nineteenth century brought about a revolution in medicine, particularly in disease control, practiced on the high seas. Nelson's well-documented personal medical history provides a window into certain typical maladies and injuries for the ocean-going warrior or explorer of his era. As a midshipman, he sustained partial

paralysis from an illness contracted at age 17, was stricken with malaria in the West Indies, contracted yellow fever in Nicaragua, suffered a laceration of his back and lost sight in his left eye during battles near Corsica, endured an abdominal wound during a military encounter at Cape St. Vincent, and had his right arm amputated below the shoulder after a severe injury from grapeshot during battle in the Canary Islands. His luck ran out in 1805 at Trafalgar, where a French sharpshooter's bullet delivered a fatal blow.

Largely as a result of Admiral Nelson's impressive understanding of the challenges of providing effective shipboard medical care, medical reforms in his and other navies became a reality. Much emphasis was directed at proper diet as it relates to disease prevention, a unique perspective at that time that is increasingly popular today. The success of this strategy is obvious from the historical record; the proportion of men sent sick to hospital from ships between the last decade of the 18th century and the first decade of the 19th century fell from a high of 38.4% (in 1793) to a low of 6.4% (in 1806).[4]

After the end of the American Civil War, the U.S. Army fought with many Native American tribes in the western states and territories. Military medical personnel and civilian practitioners became adept at extracting arrows and other primitive penetrating weapons. Instruments devised as early as 500 BC (such as the *belulcum*) for removing arrows became invaluable during the 1870s. These tools could dilate the point of entrance and widen the channel containing the arrow, to allow the head of the arrow to be grasped. North American Indian arrows were typically fired with great speed and force, and if not stopped by bone, could easily pass through a horse or bison. Not surprisingly, mortality from arrow wounds was high, with one 1871 report suggesting a fatality rate of approximately 30%.

The ensuing maturation of military medicine marked a turning point back to appreciation of wilderness medicine, or perhaps more appropriately, austere medicine. This occurred when the military began to move medicine to the front lines. Re-emergence of tourniquets applied in the field to extremities to control bleeding are illustrative. In the past two decades, bringing medicine to the point of action led to creation of tactical combat casualty care (TCCC)[5] and formation of the Special Operations Medical Association (SOMA), including collaboration with the Wilderness Medical Society (WMS). Soldiers with life-threatening injuries that previously would have been fatal are now stabilized on or near the battlefield before being evacuated to definitive trauma centers in their home countries.

Parallel to the contributions of military medicine were those of intrepid explorers. Many of the early "practitioners" of wilderness medicine were adventurers who accepted additional responsibilities. Although not a physician, Captain William Clark had sufficient medical knowledge to serve as the expedition doctor on the heralded Lewis and Clark expedition.[6] In the early days of exploration, expedition doctors were integral members of a dedicated team with expertise related to the logistics of the mission. The current expeditionary practice of retaining expert medical guests is a relatively recent phenomenon.

Infection from nonsterile surgical procedures and penetrating missiles was but one major challenge of this bygone era. Early explorers also had to contend with infectious diseases that could easily be passed between persons and that had the potential to bring any journey to a sudden halt. Throughout much of the course of its explorations, the Lewis and Clark expedition encountered indigenous tribes. These tribes had not been exposed to European-based diseases for many centuries, so were neither educationally nor immunologically capable of effective self-defense. The Americans learned of many settlements (especially along the Missouri River) that had been decimated by devastating epidemics of smallpox following contact with persons of European background. The concept of vaccination was gathering proponents at this time, and President Thomas Jefferson sent a sample of cowpox on the expedition with Lewis in hope that he could attempt to vaccinate the "natives."[7] Other infectious maladies of regular concern to Lewis and Clark included omnipresent venereal diseases, such as syphilis. Prior to 1800, mercury, already used as therapy for many infections and diseases, had become the treatment of choice for syphilis. It was typically taken orally or used as a topical ointment in very liberal doses. Cures were few and far between, and the patient often succumbed to mercury poisoning before the syphilis entered its secondary or tertiary phase. During this era, cinchona for malaria was one of only a handful of medications that had the ability to produce an intended result. Thus, early physicians "were like hunters going into the field and shooting blanks."[8]

As exploration of the last great terrestrial and oceanic "blanks on the map" evolved into ever more sophisticated ventures in the 19th and early 20th centuries, wilderness medical care further evolved. "Physician/naturalists," trained physicians who could multitask as field biologists, began to accompany long, arduous explorations to the ends of the earth. While their medical training was certainly superior to that of William Clark and they could be considered more competent healers, these physicians still needed to be extremely skilled outdoorsmen to participate in these demanding adventures. To appreciate the extent of the sacrifices sometimes made by these physician-explorers, one need only recall the Englishman Edward Wilson—physician, polar explorer, natural historian, painter, and ornithologist. Wilson accompanied two of Robert Scott's exploratory Antarctic voyages in the early years of the 20th century, tragically perishing with his companions in March 1912 on the Antarctic plateau during the British team's return sledge journey from the South Pole. Edward Atkinson, research parasitologist and senior expedition surgeon for Scott's final, ill-fated 1910-1913 *Terra Nova* expedition, was part of the shore party that did not accompany Scott, Wilson, and their three companions during the final push to the Pole. By March 1912, Scott's party was clearly overdue and fear mounted that they had perished. That month, Atkinson, as senior officer in command of 13 men facing 4 months of darkness and intense cold, led a futile effort to locate Scott. In October of 1912, with the sun at last shedding some light and heat on the bleak Antarctic landscape, Atkinson once again set out with a search party. On November 12, they discovered the dead explorers within Scott's tent, which was partially buried in snow. Atkinson was first to enter the tent, where he read the diary entries of the polar party's final days of privation and suffering.[9]

During the 20th century, exploration of the limits of the human body and those of the earth's physical domain altered the manner in which people viewed the world and their place in it. While the pursuits of physical exploration and medicine may seem unlikely siblings, they deeply informed each other. Dr. Charles Houston, who served as an inspiration to many current wilderness medicine researchers and clinicians, embodied the modern adventurer-physician.[10] An accomplished mountaineer and Harvard-trained physician, Houston led two attempts to summit K2, included the ill-fated attempt in 1953 that claimed the life of one team member and nearly wiped out the entire team. While a naval flight surgeon during World War II, Houston conceived, and ultimately was one of the physician/scientists in charge of, Operation Everest in 1946. This study, sponsored by the U.S. Navy, was intended to benefit the aviation community by shedding light on human adaptation to and tolerance of extreme altitudes. Such research efforts were particularly timely because they occurred at a time when airplanes became capable of flying higher than humans could tolerate without support, such as from pressurized suits or cabins. Houston's high-altitude chamber research led to the first successful simulated "ascent" to the barometric pressure equivalent of the summit of Mt Everest, proving it could perhaps be reached in real life without supplemental oxygen. His research team's findings led to great advances in understanding the challenges of altitude and etiology of high-altitude illnesses. Houston later became a founding member of the WMS, and in doing so, drew attention to its mission and potential.

Houston's achievements were part of a blossoming of science exemplified by the discoveries of other legendary figures in altitude research and mountain medicine, including Drs. Herb Hultgren, John West, Robert Schoene, and Peter Hackett. While serving as a member of the American Medical Research Expedition to Everest in 1981, Hackett accomplished a successful summit of Mt Everest, climbing alone to the top from high camp, falling

while traversing the Hillary step, and thereby fortunately uncovering a fixed rope that enabled him to self-rescue and live to tell the tale. He too became one of the founding members of the WMS. During this modern era, brave and high-spirited physician high-altitude adventurers Drs. Oswald Oelz, Charles Clarke, and Bruno Dürrer were pioneering by exploring, discovering, and innovating. There were and will be so many brilliant men and women who combine adventure with medicine.

One could write equally about the oceans that cover most of our planet, and about forests, rivers, canyonlands, or polar caps. There will hopefully always be mountains to climb, woods to wander, deserts to cross, and lagoons to explore. Each has its wilderness medicine history, from antiquity to modern times. There are lost people to find, victims of mishaps to rescue, and ever the need to make do with very little at the worst possible moments. We know more now about how to direct doctors, and how to facilitate location, stabilization, and transport of victims from remote and geographically challenging locales. Wilderness first responders are equipped with knowledge and training that allow for more advanced intervention that occurs with shorter transport times.[11] Search and rescue team training has become sophisticated and intense, in large measure because the wilderness medicine community has set the bar higher.

The logical evolution (and practical admixture) of wilderness medicine and wilderness search and rescue can be clearly seen in today's international Diploma in Mountain Medicine (DiMM). A cooperative idea initially developed in Europe in the late 1990s by the International Commission for Alpine Rescue, International Climbing and Mountaineering Federation, and International Society of Mountain Medicine, the extensive and comprehensive DiMM curriculum blends rigorous didactic and practical education in wilderness medicine with technical mountain rescue and self-sufficiency in the backcountry. Many mountain medicine organizations worldwide now offer the standard (or specialty module) DiMM curriculum, in the process bridging many nations and cultures.

The final linchpin in the modern enactment of wilderness medicine as a distinct entity is the selfless act of delivering medical care to the farthest reaches of the globe. Less fortunate people benefit from the emotionally taxing and sometimes courageous efforts of many wilderness medicine–trained volunteers who deliver medical support that ranges from immunizations during peaceful times to surgeries in the aftermath of natural disasters. Wilderness medicine breeds an ethos of service. Medical teams populated by wilderness medicine providers depart at a moment's notice to assist in humanitarian relief and disaster response. After many lessons learned from earlier treat-and-leave approaches, providers and organizations have embarked upon much more sustainable approaches to health care delivery, including vital education and training.

The modern history of wilderness medicine spawned the founding and maturation of important scientific societies dedicated to the discipline or one of its subspecialties. The WMS was formed in 1982; the International Society of Mountain Medicine in 1985; and the International Society of Travel Medicine in 1991. Phenomenal individuals who contributed to modern wilderness medicine have become too numerous to count. Modern wilderness medicine was conceptualized and organized by a dedicated and ambitious group of prime movers, and has become an enormous, growing community, reflected in part by the contributors to this textbook, some of whom have been involved in wilderness medicine for many decades. We admire the pioneers of the past, and have every confidence in the ability of today's leaders and innovators to carry us with great enthusiasm into the future.

**Robert H. Quinn, MD**
**George W. Rodway, PhD**

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# Preface

As the specialty of wilderness medicine matures, obligations grow. Education is the objective of this textbook and certainly essential, but to advance the field in all aspects, leadership and inspiration are required. In this seventh edition of *Wilderness Medicine*, the contributors are many of these leaders, and their writing and creativity are outstanding. Authors who are practitioners and researchers with an inestimable amount of experience share their knowledge and wisdom, and seek not only to teach, but to inspire those who will follow them. They have superbly pointed out not only what we already know, but also what we need to discover and learn, thereby directing a path toward observation, service, and experimentation, each of which is integral to the unique influence of wilderness medicine.

The breadth and depth of content of wilderness medicine have grown to the extent that two volumes of *Wilderness Medicine* are now required. The authors, assistant editors, and I are grateful to the publisher for using innovative techniques to create a comprehensive book with outstanding visual appeal, so that the blend of academia and art is at once logical and stimulating. In this edition, I am enormously grateful to the remarkable team at Elsevier, including Kate Dimock, Lucia Gunzel, Lauren Boyle, and Linda Belfus. My publishing family always sets the bar high and patiently helps me leap. My global academic family embraces this exciting specialty, and my biological family graciously allows me the time to pursue this endeavor.

Acquisition of new knowledge is exciting and challenging for academicians, practitioners, and students. Wilderness medicine draws not only from the timeless medical specialties of surgery, internal medicine, obstetrics and gynecology, pediatrics, and psychiatry, but from anywhere that medical science reaches out to improve the health and safety of patients. New chapters and deeper discussions within revised chapters introduce the reader to the trends that are most likely to become influential as we approach the next five years. Evidence-based medicine, genomics and personalization, and the imperative to translate all of this into how we live our lives and practice our craft in the field and hospital are new features of this edition. Other notable changes from the previous edition are a chapter on medical wilderness adventure races and expanded discussion of high-altitude medicine, improvisation, technical rescue, and wilderness medicine education, to name a few. We are proud to continue emphasis on how we approach the health of planet Earth. Wilderness conservation and preservation will remain in part the purview of wilderness medicine as we await a more concerted effort by the entire house of medicine to fulfill its obligation to play a leadership role in efforts to maintain the desired environment to support life on our planet.

The efforts of people and organizations engaged in all aspects of wilderness medicine are growing and increasingly collaborative. Wilderness medicine is firmly embedded in the activities of the military, and vice versa. As a responder to the 2015 earthquake disaster in Nepal, I witnessed once again that my remarkable colleagues in the wilderness medicine community are regularly at the front lines when calamity strikes. Whether it is an Ebola outbreak in West Africa, a typhoon in the Philippines, or a wildfire in Washington State, this book's contributors enthusiastically volunteer to serve. What I have learned since the last edition is that anywhere the ability to practice medicine in an austere setting is the task at hand, wilderness medicine knowledge and experience are essential.

In medical schools across the United States, and now in many other countries, wilderness medicine courses are taught and usually among the most popular electives. Wilderness experiences are used by undergraduate universities and medical schools to introduce students to one another early in their careers and facilitate collaborations. Competitive wilderness adventures in the cloaks of competitions and races are the backbone of reality entertainment. They all require medical planning and support. Wilderness recreation outpaces all other forms of time away from the urban work existence. Respite and renewal are inextricably linked to the wilderness. And when we seek to reach beyond ourselves, where do we go first to explore? The wilderness, of course.

At a time when humans are generally considered more of a burden to than saviors of the environment, we will more often be in the wilderness, learning its ways and hopefully not encroaching upon it. To impress upon others the need to preserve it, we will understand its offerings and document its beauty. To eliminate health care disparities, we will find a way to interact with indigenous people in a way that can preserve their surroundings and bring healing in the midst of horrific infectious diseases, violent conflicts, and post-disaster social and economic chaos. To fuel our existence, we will go beyond clear-cutting forests, pillaging oceans, and extracting fossil fuels that might never be replaced. If wilderness medicine helps make us aware that there is a wilderness and that without a concerted effort it will disappear, then that is a precious accomplishment by a noble specialty.

Judging by the quality of research, number of important publications, and attendance at educational gatherings, such as the combined sessions of the Wilderness Medical Society and International Society for Mountain Medicine, wilderness medicine is here to stay. For that, we are in great debt to those who came before us, who preached and practiced wilderness medicine long before anyone contemplated coalescing a specialty. One such visionary is Dr. Bruce Paton, whose artwork graces this Preface. We all have mentors and partners, and I have certainly had mine. Notable among them are Herb Hultgren and Charlie Houston in high-altitude medicine, Jeff Davis and Bruce Halstead in dive medicine, Warren Bowman in prehospital care, Bob Mutch in wildland fire management, Donald Trunkey in trauma care, Bruce Dixon in clinical diagnosis, Murray Fowler in veterinary medicine, Sherman Minton in envenomation, Bruno Dürrer in rescue, Steve French in bear behavior and attack, Cam Bangs and Alan Steinman in hypothermia, Joe Serra and Ed Geehr in humanism, Wongchu Sherpa in spirituality, and Ken Kizer in determination.

The spark has become a flame. I regularly see young people beam when they realize that medicine can be so enjoyable. There are hardcore science and service in wilderness medicine, but we are still "out there," away from electronic medical records, cost containment, and endless political debates about universal health care. We are in the field, responding because we are responsive, sticking our necks out to accept the adventure and risk, and then

put something back. There is heroism in medicine, and wilderness medicine has its fair share, delightfully unsung. It is brave to document the wisdom of an indigenous healer, courageous to teach mountain safety to sherpas, and selfless to assist laypersons to fill the gaps in health care that cannot be provided during a humanitarian crisis. The settings in which we practice may sometimes be uncontrolled, but it is the domain of wilderness medicine experts to bring best practices to the unique bedsides posed on the side of a mountain, in a cave during a lightning storm, or on the beach of a faraway atoll. Wilderness medicine takes everything we have learned and then adds to it the spice of life. How much fun is that? Seven editions now, and I can't wait for the eighth.

**Paul S. Auerbach**

*Aiming High*
**Bruce Paton**

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PART 1

# Mountain Medicine

CHAPTER 1

# High-Altitude Physiology

ROBERT C. ROACH, JUSTIN S. LAWLEY, AND PETER H. HACKETT

More than 40 million tourists visit recreation areas above 2400 meters (m), or 7874 feet, in the American West each year. Hundreds of thousands visit central and south Asia, Africa, and South America, many traveling to altitudes above 4000 m (13,123 feet). In addition, millions of persons live in large cities above 3000 m (9843 feet) in South America and Asia. The population in the Rocky Mountains of North America has doubled in the past decade; 700,000 persons live above 2500 m (8202 feet) in Colorado alone. Increasingly, physicians and other health care providers are confronted with questions of prevention and treatment of high-altitude medical problems, as well as the effects of altitude on pre existing medical conditions. Despite advances in high-altitude medicine, significant morbidity and mortality persist. Clearly, better education of the population at risk and those advising them is essential.

High-altitude medicine and physiology are discussed in the first three chapters of this textbook. In this chapter the reader is introduced to the basic physiology of high-altitude exposure. Chapter 2 describes the pathophysiology, recognition, management, and prevention of altitude illnesses and other clinical issues likely to be encountered in both "lowlanders" and high-altitude residents. Chapter 3 focuses on patients with preexisting medical problems who travel to high altitudes (Box 1-1).

## DEFINITIONS

### HIGH ALTITUDE (1500 to 3500 meters [4921 to 11,483 feet])

The onset of physiologic effects of diminished partial pressure of inspired oxygen ($P_{IO_2}$) includes decreased exercise performance and increased ventilation (lower arterial carbon dioxide partial pressure [$Pa_{CO_2}$]). Minor impairment exists in arterial oxygen transport (arterial oxygen saturation [$Sa_{O_2}$] at least 90%), but arterial oxygen partial pressure ($Pa_{O_2}$) is significantly diminished. Because of the large number of people who ascend rapidly to 2500 to 3500 m (8202 to 11,483 feet), high-altitude illness is common in this range of altitudes (see Chapter 2).

### VERY HIGH ALTITUDE (3500 to 5500 meters [11,483 to 18,045 feet])

Maximal $Sa_{O_2}$ falls below 90% as $Pa_{O_2}$ falls below 50 mm Hg (Figure 1-1 and Table 1-1). Extreme hypoxemia may occur during exercise, sleep, and high-altitude pulmonary edema (HAPE) or other acute lung conditions. Severe altitude illness occurs most frequently in this range of altitudes.

### EXTREME ALTITUDE (higher than 5500 meters [18,045 feet])

Marked hypoxemia, hypocapnia, and alkalosis characterize extreme altitude. Progressive deterioration of physiologic function eventually outstrips acclimatization. As a result, no permanent human habitation is above 5500 m (18,045 feet). A period of acclimatization is necessary when ascending to extreme altitude; abrupt ascent without supplemental oxygen for other than brief exposures invites severe altitude illness.

## THE ENVIRONMENT OF HIGH ALTITUDE

Barometric pressure ($P_B$) falls with increasing altitude in a logarithmic manner (Table 1-2). Therefore, the partial pressure of oxygen ($P_{O_2}$, 21% of $P_B$) also decreases, resulting in the primary insult of high-altitude: hypoxia. At approximately 5800 m (19,029 feet), $P_B$ is one-half that at sea level, and on the summit of Mt Everest (8848 m [29,029 feet]), $P_{IO_2}$ is approximately 28% that at sea level (see Figure 1-1 and Table 1-1).

The relationship of $P_B$ to altitude changes with distance from the equator. Thus, in addition to extreme cold, polar regions afford greater hypoxia at any given altitude. West[90] calculated that $P_B$ on the summit of Mt Everest (27 degrees north latitude [N]) would be about 222 mm Hg instead of 253 mm Hg if Mt Everest were located at the latitude of Denali (62 degrees N). Such a difference, he claims, would be sufficient to render impossible an ascent without supplemental oxygen.

In addition to the role of latitude, fluctuations related to season, weather, and temperature affect the pressure-altitude relationship. Pressure is lower in winter than in summer. A low-pressure trough can reduce pressure 10 mm Hg in one night on Denali, making climbers awaken "physiologically higher" by 200 m (656 feet). The degree of hypoxia is thus directly related to $P_B$, not solely to geographic altitude.[90]

Temperature decreases with altitude (average of 6.5° C [11.7° F] per 1000 m [3281 feet]), and the effects of cold and hypoxia are generally additive in provoking both cold injuries and HAPE.[59,93] Ultraviolet (UV) light penetration increases approximately 4% per 300-m (984-foot) gain in altitude, increasing the risks for sunburn, skin cancer, and snowblindness. Reflection of sunlight in glacial cirques and on flat glaciers can cause intense radiation of heat in the absence of wind. We have observed temperatures of 40° to 42° C (104° to 107.6° F) in tents on both Mt Everest and Denali. Heat problems, primarily heat exhaustion, are often unrecognized in this usually cold environment. Physiologists have not yet examined the consequences of heat stress or rapid, extreme changes in environmental temperature combined with the hypoxia of high altitude.

Above the snow line is the "high-altitude desert," where water can be obtained only by melting snow or ice. This factor, combined with increased water loss through the lungs from increased respiration and through the skin, typically results in dehydration that may be debilitating. Thus, the high-altitude environment imposes multiple stresses, some of which may contribute to, or may be confused with, the effects of hypoxia.

## ACCLIMATIZATION TO HIGH ALTITUDE

Although rapid exposure from sea level to the altitude at the summit of Mt Everest (8848 m [29,029 feet]) causes loss of consciousness in a few minutes and death shortly thereafter, climbers can ascend Mt Everest over a period of weeks without supplemental oxygen because of a process termed *acclimatization*. A complex series of physiologic adjustments increases oxygen delivery to cells and also improves their hypoxic tolerance. The severity of hypoxic stress, rate of onset, and individual physiology determine whether the body successfully acclimatizes or is

**BOX 1-1 Glossary of Physiologic Terms***

| | |
|---|---|
| $P_B$ | Barometric pressure |
| $P_{O_2}$ | Partial pressure of oxygen |
| $P_{IO_2}$ | Inspired $P_{O_2}$ (0.21 × [$P_B$ – 47 mm Hg]) (47 mm Hg = vapor pressure of $H_2O$ at 37°C [98.6°F]) |
| $P_{AO_2}$ | $P_{O_2}$ in alveolus |
| $P_{ACO_2}$ | $P_{CO_2}$ in alveolus |
| $P_{aO_2}$ | $P_{O_2}$ in arterial blood |
| $P_{aCO_2}$ | $P_{CO_2}$ in arterial blood |
| $S_{aO_2}$ | Arterial oxygen saturation ($HbO_2$ ÷ total Hb × 100) |
| RQ | Respiratory quotient ($CO_2$ produced ÷ $O_2$ consumed) |
| Alveolar gas equation | $P_{AO_2} = P_{IO_2} - (P_{ACO_2}/RQ)$ |

*Pressures are expressed as millimeters of mercury (1 mm Hg = 1 torr).

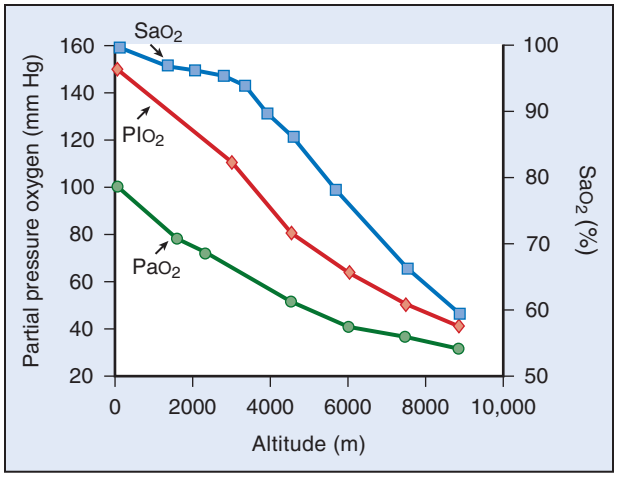


**FIGURE 1-1** Increasing altitude results in decreasing inspired oxygen partial pressure ($P_{IO_2}$), arterial $P_{O_2}$ ($P_{aO_2}$), and arterial oxygen saturation ($S_{aO_2}$). Note that the difference between $P_{IO_2}$ and $P_{aO_2}$ narrows at high altitude because of increased ventilation, and that $S_{aO_2}$ is well maintained while awake until over 3000 m (9843 feet). *(Data from Morris A:* Clinical pulmonary function tests: A manual of uniform lab procedures, *Salt Lake City, 1984, Intermountain Thoracic Society; and Sutton JR, Reeves JT, Wagner PD, et al: Operation Everest II: Oxygen transport during exercise at extreme simulated altitude,* J Appl Physiol *64:1309, 1988.)*

overwhelmed. Importantly, acclimatization is the only known means to improve physical and cognitive performance at high altitude.

The recent revolution in our understanding of the molecular mechanisms of human responses to hypoxia has focused on *hypoxia-inducible factor* (HIF). This transcription factor modulates the expression of hundreds of genes, including those involved in apoptosis, angiogenesis, metabolism, cell proliferation, and permeability processes.[20,27,67,69,88] In chronic hypoxia, HIF activation by hypoxia has the positive effect of elevating oxygen delivery by boosting hemoglobin mass. However, HIF also plays a role in carotid body sensitivity to hypoxia, which in turn largely determines the ventilatory response to hypoxia.[55,56,70] As a master regulator of the hypoxia response in humans, HIF has beneficial and harmful effects at different stages during human exposure to hypoxia and in different cells in the body.[36,47] Figure 1-2 provides an overview of some of the hundreds of processes by which the response to hypoxia is modulated by HIF.

Individuals vary in their ability to acclimatize, reflecting certain genetic polymorphisms, including HIF. Some adjust quickly, without discomfort, whereas acute mountain sickness (AMS) develops in others, who go on to recover. A small percentage fail to acclimatize even with gradual exposure over weeks. The tendency to acclimatize well or to become ill is consistent on repeated exposure if rate of ascent and altitude gained are similar, supporting the role of important genetic factors and an individual's predisposition. Successful initial acclimatization protects against altitude illness and improves sleep. Longer-term acclimatization (weeks) primarily improves aerobic exercise ability. These adjustments disappear at a similar rate on descent to low altitude. A few days at low altitude may be sufficient to render a person susceptible to altitude illness, especially HAPE, on reascent. The improved ability to do physical work at high altitude, however, persists for up to 3 weeks.[43,77] Persons who live at high altitude during growth and development appear to realize the maximum benefit of acclimatization changes; for

**TABLE 1-1 Arterial Blood Gases and Altitude***

| Population | Altitude: Meters | Altitude: Feet | $P_B$ (mm Hg) | $P_{aO_2}$ (mm Hg) | $S_{aO_2}$ (%) | $P_{aCO_2}$ (mm Hg) |
|---|---|---|---|---|---|---|
| Altitude residents | 1646[1] | 5400 | 630 | 73.0 (65.0-83.0) | 95.1 (93.0-97.0) | 35.6 (30.7-41.8) |
| Acute exposure | 2810[2] | 9219 | 543 | 60.0 (47.4-73.6) | 91.0 (86.6-95.2) | 33.9 (31.3-36.5) |
| | 3660[2] | 12,008 | 489 | 47.6 (42.2-53.0) | 84.5 (80.5-89.0) | 29.5 (23.5-34.3) |
| | 4700[2] | 15,420 | 429 | 44.6 (36.4-47.5) | 78.0 (70.8-85.0) | 27.1 (22.9-34.0) |
| | 5340[2] | 17,520 | 401 | 43.1 (37.6-50.4) | 76.2 (65.4-81.6) | 25.7 (21.7-29.7) |
| | 6140[2] | 20,144 | 356 | 35.0 (26.9-40.1) | 65.6 (55.5-73.0) | 22.0 (19.2-24.8) |
| Subacute exposure | 6500[3] | 21,325 | 346 | 41.1 ± 3.3 | 75.2 ± 6 | 20 ± 2.8 |
| | 7000[3] | 22,966 | 324 | | | |
| | 8000[3] | 26,247 | 284 | 36.6 ± 2.2 | 67.8 ± 5 | 12.5 ± 1.1 |
| | 8400[4] | 27,559 | 272 | 24.6 ± 5.3 | 54 | 13.3 |
| | 8848[3] | 29,029 | 253 | 30.3 ± 2.1 | 58 ± 4.5 | 11.2 ± 1.7 |
| | 8848[5] | 29,029 | 253 | 30.6 ± 1.4 | | 11.9 ± 1.4 |

[1]Data from Loeppky JA, Caprihan A, Luft UC: VA/Q inequality during clinical hypoxemia and its alterations. In: Shiraki K, Yousef MK, editors. *Man in stressful environments,* Springfield, Ill, 1987, Thomas; pp 199-232.
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$P_B$, Barometric pressure; $P_{aCO_2}$, arterial partial pressure of carbon dioxide; $P_{aO_2}$, arterial partial pressure of oxygen; $S_{aO_2}$, arterial oxygen saturation.
*Data are mean values and (range) or ±SD (standard deviation), where available. All values are for people age 20 to 40 years who were acclimatizing well.

**TABLE 1-2** Altitude Conversion: Barometric Pressure,* Estimated Partial Pressure of Inspired Oxygen,† and the Equivalent Oxygen Fraction at Sea Level‡

| Meters | Feet | $P_B$ | $P_{IO_2}$ | $F_{IO_2}$ at SL |
|---|---|---|---|---|
| Sea level | Sea level | 759.6 | 149.1 | 0.209 |
| 1000 | 3281 | 678.7 | 132.2 | 0.185 |
| 1219 | 4000 | 661.8 | 128.7 | 0.180 |
| 1500 | 4921 | 640.8 | 124.3 | 0.174 |
| 1524 | 5000 | 639.0 | 123.9 | 0.174 |
| 1829 | 6000 | 616.7 | 119.2 | 0.167 |
| 2000 | 6562 | 604.5 | 116.7 | 0.164 |
| 2134 | 7000 | 595.1 | 114.7 | 0.161 |
| 2438 | 8000 | 574.1 | 110.3 | 0.155 |
| 2500 | 8202 | 569.9 | 109.4 | 0.154 |
| 2743 | 9000 | 553.7 | 106.0 | 0.149 |
| 3000 | 9843 | 536.9 | 102.5 | 0.144 |
| 3048 | 10,000 | 533.8 | 101.9 | 0.143 |
| 3353 | 11,000 | 514.5 | 97.9 | 0.137 |
| 3500 | 11,483 | 505.4 | 95.9 | 0.135 |
| 3658 | 12,000 | 495.8 | 93.9 | 0.132 |
| 3962 | 13,000 | 477.6 | 90.1 | 0.126 |
| 4000 | 13,123 | 475.4 | 89.7 | 0.126 |
| 4267 | 14,000 | 460.0 | 86.4 | 0.121 |
| 4500 | 14,764 | 446.9 | 83.7 | 0.117 |
| 4572 | 15,000 | 442.9 | 82.9 | 0.116 |
| 4877 | 16,000 | 426.3 | 79.4 | 0.111 |
| 5000 | 16,404 | 419.7 | 78.0 | 0.109 |
| 5182 | 17,000 | 410.2 | 76.0 | 0.107 |
| 5486 | 18,000 | 394.6 | 72.8 | 0.102 |
| 5500 | 18,045 | 393.9 | 72.6 | 0.102 |
| 5791 | 19,000 | 379.5 | 69.6 | 0.098 |
| 6000 | 19,685 | 369.4 | 67.5 | 0.095 |
| 6096 | 20,000 | 364.9 | 66.5 | 0.093 |
| 6401 | 21,000 | 350.7 | 63.6 | 0.089 |
| 6500 | 21,325 | 346.2 | 62.6 | 0.088 |
| 6706 | 22,000 | 337.0 | 60.7 | 0.085 |
| 7000 | 22,966 | 324.2 | 58.0 | 0.081 |
| 7010 | 23,000 | 323.8 | 57.9 | 0.081 |
| 7315 | 24,000 | 310.9 | 55.2 | 0.077 |
| 7500 | 24,606 | 303.4 | 53.7 | 0.075 |
| 7620 | 25,000 | 298.6 | 52.6 | 0.074 |
| 7925 | 26,000 | 286.6 | 50.1 | 0.070 |
| 8000 | 26,247 | 283.7 | 49.5 | 0.069 |
| 8230 | 27,000 | 275.0 | 47.7 | 0.067 |
| 8500 | 27,887 | 265.1 | 45.6 | 0.064 |
| 8534 | 28,000 | 263.8 | 45.4 | 0.064 |
| 8839 | 29,000 | 253.0 | 43.1 | 0.060 |
| 8848 | 29,029 | 252.7 | 43.1 | 0.060 |
| 9000 | 29,528 | 247.5 | 42.0 | 0.059 |
| 9144 | 30,000 | 242.6 | 40.9 | 0.057 |
| 9500 | 31,168 | 230.9 | 38.5 | 0.054 |
| 10,000 | 32,808 | 215.2 | 35.2 | 0.049 |

*$F_{IO_2}$*, fraction of inspired oxygen; *$P_B$*, barometric pressure; *$P_{IO_2}$*, partial pressure of inspired oxygen; *SL*, sea level.
*$P_B$ is approximated by Exponent (6.6328 – {0.1112 × altitude – [0.00149 × altitude$^2$]}), where altitude is terrestrial altitude in meters/1000 or kilometers (km).
†$P_{IO_2}$ is calculated as $P_B$ – 47 × fraction of $O_2$ in inspired air, where 47 is water vapor pressure at body temperature.
‡The equivalent $F_{IO_2}$ at sea level for a given altitude is calculated as $P_{IO_2}$ ÷ (760 – 47). Substituting ambient $P_B$ for 760 in the equation allows similar calculations for $F_{IO_2}$ at different altitudes.

example, their exercise performance matches that of persons at sea level.[8,50]

## VENTILATION

By reducing alveolar carbon dioxide, increased ventilation raises alveolar oxygen, improving oxygen delivery (Figure 1-3). This response begins at altitudes as low as 1500 m (4921 feet) ($P_{IO_2}$ = 124.3 mm Hg; see Table 1-2) and within the first few minutes to hours of high-altitude exposure. The carotid body, sensing a decrease in $P_{aO_2}$, through a HIF-mediated process, signals the central respiratory center in the medulla to increase ventilation.[3,51,57] This carotid body function, the *hypoxic ventilatory response* (HVR), is genetically determined[89] but is influenced by a number of extrinsic factors. Respiratory depressants such as alcohol and soporific drugs, as well as fragmented sleep, depress HVR. Agents that increase general metabolism, such as caffeine and coca, as well as specific respiratory stimulants, such as progesterone[37] and almitrine,[25] increase HVR. Acetazolamide, a respiratory stimulant, acts on the central respiratory center rather than on the carotid body. Physical conditioning apparently has no effect on HVR. Numerous studies have shown that a good ventilatory response enhances acclimatization and performance,[77] and that a very low HVR may contribute to illness[61] (see Acute Mountain Sickness and High-Altitude Pulmonary Edema in Chapter 2).

As ventilation increases, hypocapnia produces alkalosis, which acts as a braking mechanism on the central respiratory center and limits a further increase in ventilation. To compensate for the alkalosis, within 24 to 48 hours of ascent, the kidneys excrete bicarbonate, decreasing the pH toward normal; ventilation increases as the braking effect of the alkalosis is removed. Ventilation continues to increase slowly, reaching a maximum only after 4 to 7 days at the same altitude (see Figure 1-3). The plasma bicarbonate concentration continues to drop and ventilation continues to increase with each successive increase in altitude. Persons with lower oxygen saturation at altitude have higher serum bicarbonate values. Whether the kidneys might be limiting acclimatization or whether this reflects poor respiratory drive is not clear.[16] This process is greatly facilitated by acetazolamide (see Acetazolamide Prophylaxis in Chapter 2).

The paramount importance of hyperventilation is readily apparent from the following calculation: the alveolar $P_{O_2}$ on the summit of Mt Everest (approximately 33 mm Hg) would be reached at only 5000 m (16,404 feet) if alveolar $P_{CO_2}$ stayed at 40 mm Hg, limiting an ascent without supplemental oxygen to near this altitude. Table 1-1 lists the measured arterial blood gas values resulting from acclimatization to various altitudes.

## CIRCULATION

The circulatory pump is the next step in the transfer of oxygen, moving oxygenated blood from the lungs to the tissues.

### Systemic Circulation

Increased sympathetic activity on ascent causes an initial mild increase in blood pressure, moderate increases in heart rate and cardiac output, and increase in venous tone. Stroke volume is low because of decreased plasma volume, which drops as much as 12% over the first 24 hours[95] as a result of the bicarbonate diuresis, a fluid shift from the intravascular space, and suppression of aldosterone.[7] Resting heart rate returns to near sea level values with acclimatization, except at extremely high altitude. Maximal heart rate follows the decline in maximal oxygen uptake with increasing altitude. As the limits of hypoxic acclimatization are approached, maximal and resting heart rates converge. During Operation Everest II (OEII), cardiac function was appropriate for the level of work performed, and cardiac output was not a limiting factor for performance.[58,76] Interestingly, myocardial ischemia at high altitude has not been reported in healthy persons, despite extreme hypoxemia. This is partly because of reduction in myocardial oxygen demand from reduced maximal heart rate and cardiac output. Pulmonary capillary wedge pressure is low, and catheter studies have shown no evidence of left ventricular dysfunction or abnormal filling pressures in humans at rest.[24,29] On echocardiography, the left ventricle is smaller than normal because of decreased stroke volume, whereas the right ventricle may become enlarged.[76] The abrupt increase in pulmonary artery pressure can cause a change in left ventricular diastolic function, but because of compensatory increased atrial contraction, no overt diastolic dysfunction results.[2] In trained

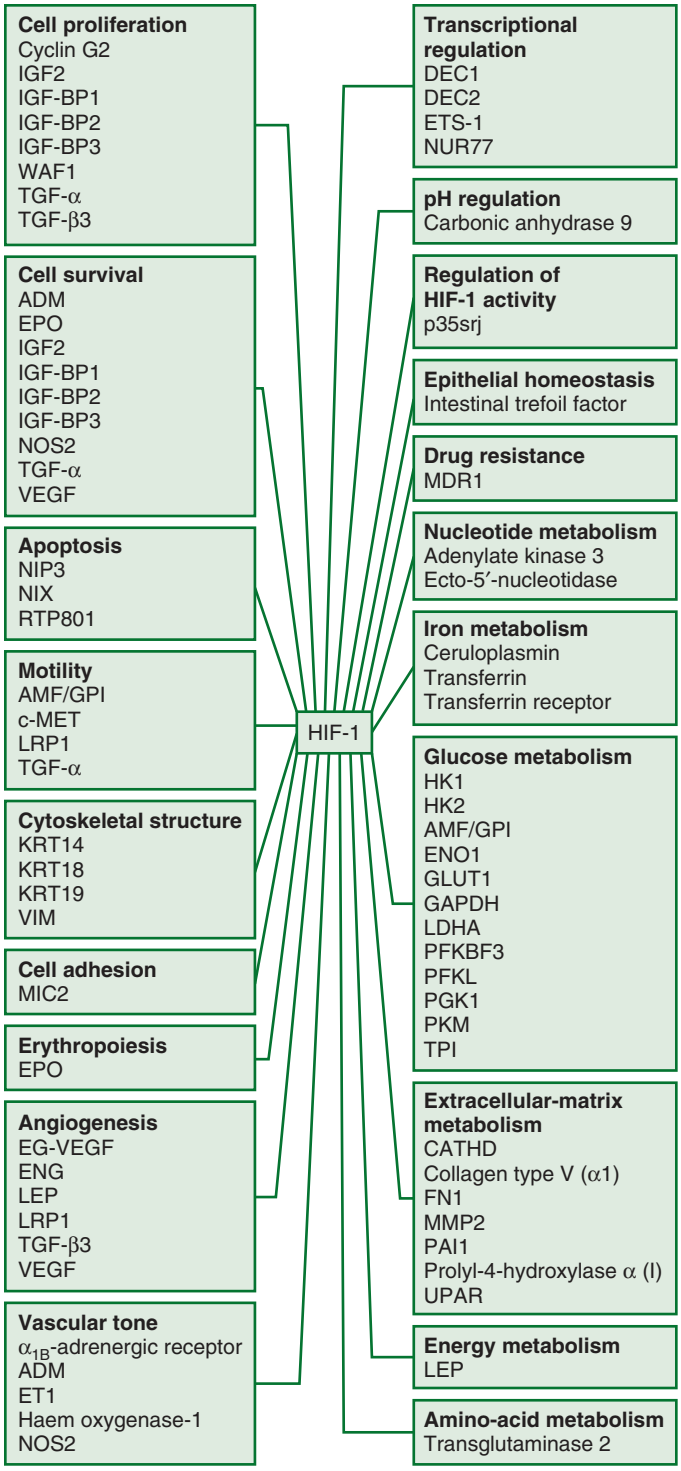


**FIGURE 1-2** Regulation of oxygen sensing by hypoxia-inducible factor (HIF). HIF is produced constitutively, but in normoxia the α subunit is degraded by the proteasome in an oxygen-dependent manner. Hypoxic conditions prevent hydroxylation of the α subunit, enabling the active HIF transcription complex to form at the hypoxia-response element (HRE) associated with HIF-regulated genes. A range of cell functions are regulated by the target genes, as indicated. ADM, adrenomedullin; AMF, autocrine motility factor; CATHD, cathepsin D; EG-VEGF, endocrine gland–derived vascular endothelial growth factor; ENG, endoglin; ENO1, enolase 1; EPO, erythropoietin; ET1, endothelin-1; FN1, fibronectin 1; GAPDH, glyceraldehyde-3-phosphate-dehydrogenase; GLUT1, glucose transporter (1, 3); HK1, hexokinase 1; HK2, hexokinase 2; IGF2, insulin-like growth factor 2; IGF-BP, IGF-binding protein (1, 2, 3); KRT, keratin (14, 18, 19); LDHA, lactate dehydrogenase A; LEP, leptin; LRP1, LDL receptor–related protein 1; MDR1, multidrug resistance gene 1; MMP2, matrix metalloproteinase 2; NOS2, nitric oxide synthase 2; PFKBF3, 6-phosphofructo-2-kinase/fructose-2,6-biphosphatase-3; PFKL, phosphofructokinase L; PGK 1, phosphoglycerate kinase 1; PAI1, plasminogen-activator inhibitor 1; PKM, pyruvate kinase M; TGF-β3, transforming growth factor-β3; TPI, triosephosphate isomerase; VEGF, vascular endothelial growth factor; UPAR, urokinase plasminogen activator receptor; VIM, vimentin. *(Modified from Semenza G: Targeting HIF-1 for cancer therapy,* Nat Rev Cancer *3[10]:721-732, 2003.)*

athletes doing an ultramarathon, the strenuous exercise at high altitude did not result in left ventricular damage; however, wheezing, reversible pulmonary hypertension, and right ventricular dysfunction occurred in one-third of those completing the race and resolved within 24 hours.

### Pulmonary Circulation

On ascent to high altitude, a prompt but variable increase in pulmonary vascular resistance (PVR) from hypoxic pulmonary vasoconstriction increases pulmonary artery pressure (PAP). Mild pulmonary hypertension is greatly augmented by exercise, with PAP reaching near-systemic values,[24] especially in persons with a prior history of HAPE.[6,19] During OEII, Groves and colleagues[24] demonstrated that even with a mean PAP of 60 mm Hg, cardiac output remained appropriate, and right atrial pressure did not rise above sea level values. Thus, right ventricular function was intact despite extreme hypoxemia and pulmonary hypertension in these well-acclimatized individuals.

Administration of oxygen does not completely restore PAP to sea level values,[45] likely because of vascular remodeling with medial hypertrophy. (See Stenmark and associates[71,72] for excellent recent reviews of molecular and cellular mechanisms of the pulmonary vascular response to hypoxia, including remodeling.) PVR returns to normal within a few weeks after descent to low altitude.

### Cerebral Circulation

Cerebral oxygen delivery, the product of arterial oxygen content and cerebral blood flow (CBF), depends on the net balance between hypoxic vasodilation and hypocapnia-induced vasoconstriction. Despite hypocapnia, CBF increases when $Pa_{O_2}$ is less than 60 mm Hg (altitude >2800 m [9186 feet]). In a classic study, CBF increased 24% on abrupt ascent to 3810 m (12,500 feet) and returned to normal over 3 to 5 days.[68] These findings have been confirmed by positron emission tomography (PET) and brain magnetic resonance imaging (MRI) studies showing both elevations in CBF in hypoxia in humans and striking heterogeneity of the CBF, with CBF rising up to 33% in the hypothalamus and 20% in the thalamus, and with other areas showing no significant change.[9,54] Cerebral autoregulation, the process by which cerebral perfusion is maintained as blood pressure varies, is impaired in hypoxia. Interestingly, this occurs with acute ascent,[31,41,81,84] after successful acclimatization,[79,82] and in natives to high altitude.[31] The uniform "impairment" in all humans who become hypoxic raises questions about the importance of cerebral autoregulation, specifically as it pertains to altitude illness (see Chapter 2 for advanced discussion on AMS and cerebral autoregulation). Overall, global cerebral metabolism seems well maintained with moderate hypoxia.[1,17,49]

## BLOOD

### Hematopoietic Responses to Altitude

Ever since the observation in 1890 by Viault[85] that hemoglobin concentration was higher than normal in animals living in the Andes, scientists have regarded the hematopoietic response to increasing altitude as an important component of the acclimatization process. On the other hand, hemoglobin values apparently have no relationship to susceptibility to high-altitude illness.

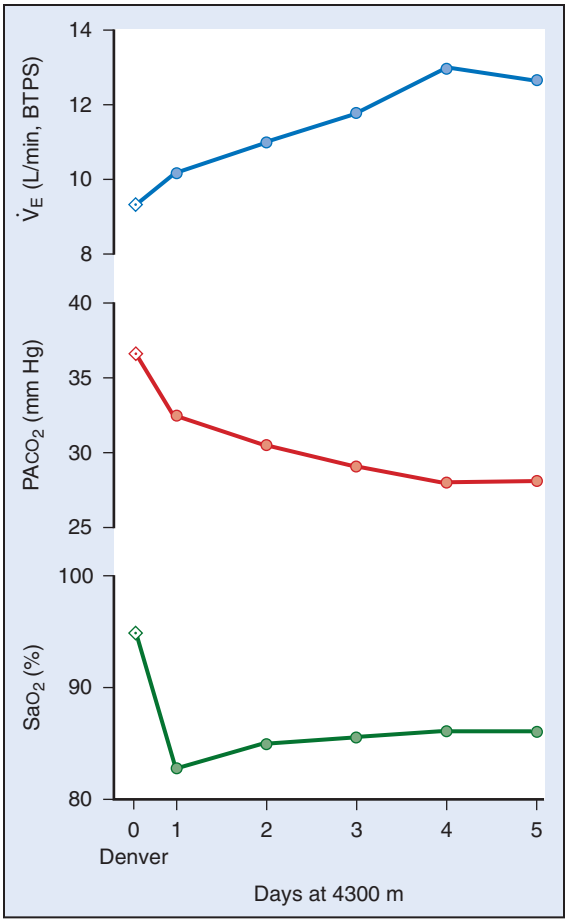


**FIGURE 1-3** Change in minute ventilation ($\dot{V}_E$), alveolar (end-tidal) carbon dioxide partial pressure ($PACO_2$), and arterial oxygen saturation ($SaO_2$) during 5 days' acclimatization to 4300 m (14,108 feet). *BTPS,* Body temperature pressure saturated. *(Modified from Huang SY, Alexander JK, Grover RF, et al: Hypocapnia and sustained hypoxia blunt ventilation on arrival at high altitude,* J Appl Physiol *56:602-606, 1984.)*

In response to hypoxemia, erythropoietin is secreted by the kidneys and stimulates bone marrow production of red blood cells (RBCs).[66] The hormone is detectable within 2 hours of ascent, nucleated immature RBCs can be found on a peripheral blood smear within days, and new RBCs are in circulation within 4 to 5 days. Over weeks to months, RBC mass increases in proportion to the degree of hypoxemia. Iron supplementation can be important; women who take supplemental iron at high altitude approach the hematocrit values of men at altitude[26] (Figure 1-4). The field of erythropoietin and iron metabolism has exploded in recent years, with discovery of two new iron-regulating hormones, hepcidin[23] and erythroferrone,[10,22,34,38] and a novel, soluble erythropoietin receptor with function directly linked to performance at high altitude.[86] How all these new findings are integrated and their responses during acclimatization to hypoxia remain to be determined.

The increase in hemoglobin seen 1 to 2 days after ascent is caused by hemoconcentration secondary to decreased plasma volume, rather than by a true increase in RBC mass. This results in a higher hemoglobin concentration at the cost of decreased blood volume, a trade-off that might impair exercise performance. Longer-term acclimatization leads to an increase in plasma volume as well as in RBC mass, thereby increasing total blood volume. Overshoot of the hematopoietic response causes excessive polycythemia, which may impair oxygen transport because of increased blood viscosity. Although the "ideal" hematocrit at high altitude is not established, phlebotomy is often recommended when hematocrit values exceed 60% to 65%. During the American Medical Research Expedition to Mt Everest (AMREE), hematocrit was reduced by hemodilution from 58% ±1.3% to 50.5% ±1.5% at 5400 m (17,717 feet) with increased cerebral functioning and no decrement in maximal oxygen uptake.[65]

## Oxyhemoglobin Dissociation Curve

The oxygen dissociation curve (ODC) plays a crucial role in oxygen transport. The sigmoidal shape of the curve allows $SaO_2$ to be well maintained up to 3000 m (9843 feet), despite significant decreases in $PaO_2$ (see Figure 1-1). Above 3000 m, small changes in $PaO_2$ cause large changes in $SaO_2$ (Figure 1-5). Because $PaO_2$ determines diffusion of oxygen from capillary to cell, small changes in $PaO_2$ can have clinically significant effects. This is often confusing for clinicians because $SaO_2$ appears relatively well preserved. At high altitude, small changes in $PaO_2$ lead to lower oxygen uptake that can have a large effect on systemic

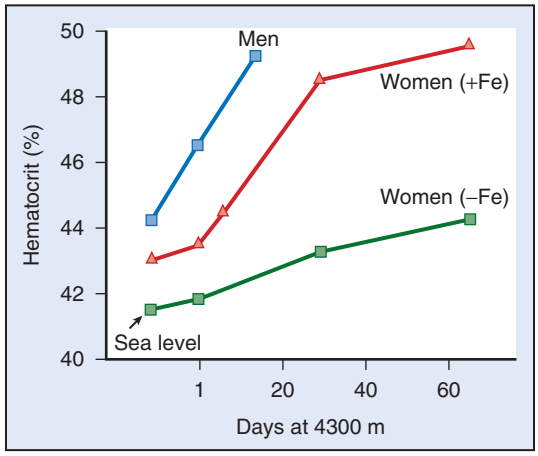


**FIGURE 1-4** Hematocrit changes on ascent to altitude in men and in women with (+Fe) and without (–Fe) supplemental iron. *(Modified from Hannon JP, Klain GJ, Sudman DM, Sullivan FJ: Nutritional aspects of high-altitude exposure in women,* Am J Clin Nutr *29:604-613, 1976.)*

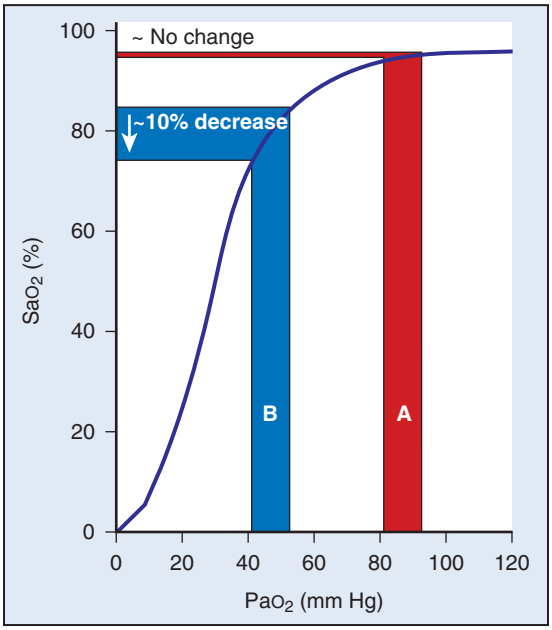


**FIGURE 1-5** Oxygen-hemoglobin dissociation curve showing effect of 10–mm Hg decrement in arterial partial pressure of oxygen ($PaO_2$) on arterial oxygen saturation ($SaO_2$) at sea level *(A)* and near 4400 m (14,436 feet) *(B).* Note the much larger drop in $SaO_2$ at high altitude. *(Modified from Severinghaus JW: Blood gas calculator,* J Appl Physiol *21:1108-1116, 1966.)*

hypoxemia, and thus on clinical status, while $Sa_{O_2}$ may appear relatively unchanged.

In 1936, Ansel Keys and colleagues[35] demonstrated an in vitro right shift in position of the ODC at high altitude, favoring release of oxygen from blood to tissues. This change, caused by increased 2,3-diphosphoglycerate, is proportional to the severity of hypoxemia. In vivo, however, the alkalosis at moderate altitude offsets this, and no net change occurs. In contrast, the marked alkalosis of extreme hyperventilation, as measured on the summit and simulated summit of Mt Everest ($Pa_{CO_2}$ = 8 to 10 mm Hg; pH >7.5), shifts the ODC to the left, facilitating oxygen-hemoglobin binding in the lung, which raises $Sa_{O_2}$ and is advantageous.[64] Persons with a very left-shifted ODC caused by an abnormal hemoglobin (Andrew-Minneapolis), when taken to moderate (3100 m [10,171 feet]) altitude, had less tachycardia and dyspnea and remarkably no decrease in exercise performance.[28] High-altitude–adapted animals also have a left-shifted ODC.

## TISSUE CHANGES

The next link in the oxygen transport chain is tissue oxygen transfer, which depends on capillary perfusion, diffusion distance, and driving pressure of oxygen from the capillary to the cell. Banchero[5] has shown that capillary density in dog skeletal muscle doubles in 3 weeks at $P_B$ of 435 mm Hg. A recent study in humans noted no change in capillary density or in gene expression thought to enhance muscle vascularity.[42] Ou and Tenney[53] revealed a 40% increase in mitochondrial number but no change in mitochondrial size, whereas Oelz and colleagues[52] showed that high-altitude climbers had normal mitochondrial density. A significant decrease in muscle size is often noted after high-altitude expeditions because of net energy deficit, resulting in increased capillary density and ratio of mitochondrial volume to contractile protein fraction as a result of the atrophy. Although there is no de novo synthesis of capillaries or mitochondria, the net result is a shortening of diffusion distance for oxygen.[42,44]

## EXERCISE

Maximal oxygen consumption drops dramatically on ascent to high altitude.[21,62] Maximal oxygen uptake ($\dot{V}O_2max$) falls from sea level value by approximately 10% for each 1000 m (3281 feet) of altitude gained above 1500 m (4921 feet). Persons with the highest $\dot{V}O_2max$ values at sea level have the largest decrement in $\dot{V}O_2max$ at high altitude, but overall performance at high altitude is not consistently related to sea level $\dot{V}O_2max$.[52,60,91] In fact, many of the world's elite mountaineers, in contrast to other endurance athletes, have quite average $\dot{V}O_2max$ values.[52] Acclimatization at moderate altitudes enhances submaximal endurance time but does not enhance $\dot{V}O_2max$ (Figure 1-6).[21] Two groups recently confirmed that acclimatization leads to improvement in submaximal work capacity using field tests,[39,77] and Subudhi and associates[77] showed that adaptation to submaximal work performance persists for up to 3 weeks after descent to low altitude. This occurs despite a marked drop in hemoglobin concentration, suggesting that other factors are involved.

Oxygen transport during exercise at high altitude becomes increasingly dependent on the ventilatory pump. The marked rise in ventilation produces a sensation of breathlessness, even at low work levels. The following quotation is from a high-altitude mountaineer:[48]

> *After every few steps, we huddle over our ice axes, mouths agape, struggling for sufficient breath to keep our muscles going. I have the feeling I am about to burst apart. As we get higher, it becomes necessary to lie down to recover our breath.*

In contrast to the increase in ventilation with exercise, at increasing altitudes in OEII, cardiac function and cardiac output were maintained at or near sea level values for a given oxygen consumption (workload).[58] Recent work attributed the altitude-induced drop to the lower $P_{IO_2}$, impairment of pulmonary gas exchange, and reduction of maximal cardiac output and peak leg blood flow, each explaining about one-third of the

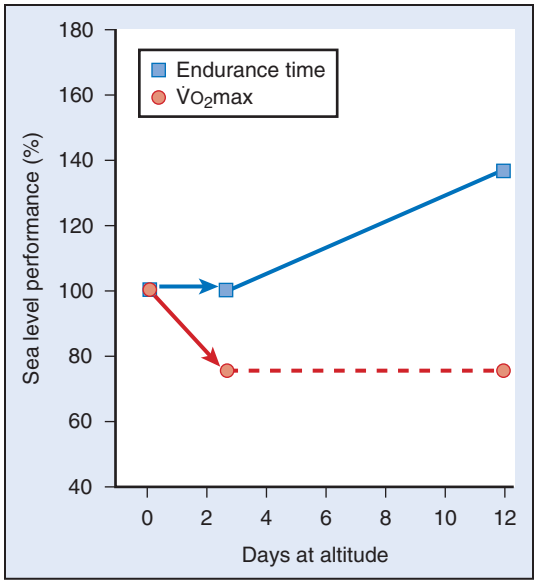


**FIGURE 1-6** On ascent to altitude, maximal oxygen consumption ($\dot{V}O_2max$) decreases and remains suppressed. In contrast, endurance time (minutes to exhaustion at 75% of altitude-specific $\dot{V}O_2max$) increases with acclimatization. *(Modified from Maher JT, Jones LG, Hartley LH: Effects of high-altitude exposure on submaximal endurance capacity of men,* J Appl Physiol *37:895-898, 1974.)*

loss in $\dot{V}O_2max$.[11] However, mechanisms to explain impaired gas exchange and lower blood flow remain elusive. Wagner[87] proposes that the pressure gradient for diffusion of oxygen from capillaries to the working muscle cells may be inadequate. Others propose that increased cerebral hypoxia from exercise-induced desaturation is the limiting factor.[15,30,78,80] Mountaineers, for example, become lightheaded and their vision dims when they move too quickly at extreme altitude (Figure 1-7).[92]

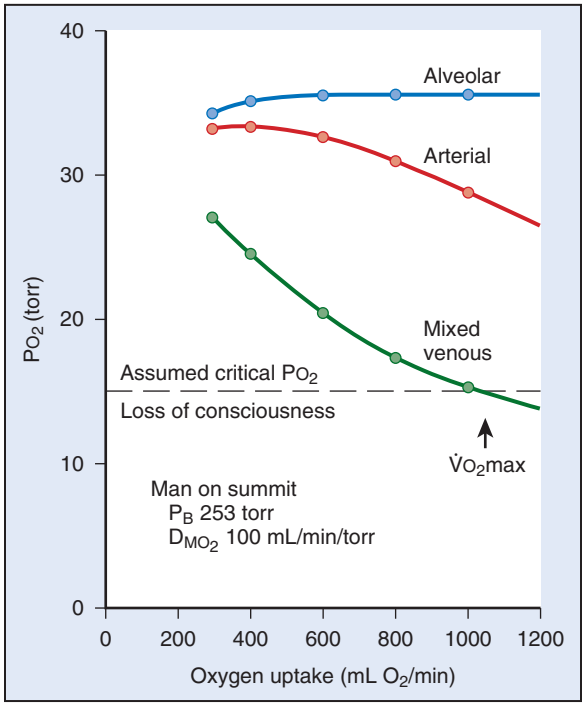


**FIGURE 1-7** Calculated changes in the oxygen partial pressure ($P_{O_2}$) of alveolar gas and arterial and mixed venous blood as oxygen uptake is increased for a climber on the summit of Mt Everest. Unconsciousness develops at a mixed venous $P_{O_2}$ of 15 mm Hg. $P_B$, Barometric pressure; $D_{MO_2}$, muscle-diffusing capacity; $\dot{V}O_2max$, maximal oxygen consumption (intake). *(Modified from West J: Human physiology at extreme altitudes on Mount Everest,* Science *223[4638]:784-788, 1984.)*

### Training at High Altitude

Optimal training for increased performance at high altitude depends on the altitude of residence and the athletic event. For aerobic activities (events lasting >3 minutes) at altitudes above 2000 m (6562 feet), acclimatization for 10 to 20 days is necessary to maximize performance.[18] For events occurring above 4000 m (13,123 feet), acclimatization at an intermediate altitude is recommended. Highly anaerobic events at intermediate altitudes require only arrival at the time of the event, although AMS may become a problem.

The benefits of training at high altitude for subsequent performance at or near sea level depend on choosing the training altitude that maximizes the benefits and minimizes the inevitable "detraining" when $\dot{V}O_2max$ is limited (altitude >1500 to 2000 m [4921 to 6562 feet]). Therefore, data from training above 2400 m (7874 feet) have shown no increase in subsequent sea level performance. Also, intermittent exposures to hypoxia seem to have no benefit.[33,83] Runners returning to sea level after 10 days' training at 2000 m (6562 feet) had faster running times and an increase in aerobic power, plasma volume, and hemoglobin concentration.[4] The "live high–train low" approach pioneered by Levine and Stray-Gundersen[40,74] has been adopted by many endurance athletes. The optimal dose for specific sports is still being determined,[12,13,94] but overall, endurance athletes believe and science supports a small but significant improvement in sea level performance after participating in a live high–train low training camp.[75] The benefit appears to result from enhanced erythropoietin production and increased RBC mass, which requires adequate iron stores and thus usually iron supplementation.[46,63,73] Some individuals do not respond to the live high–train low approach, perhaps related to genetic polymorphisms and inability to increase erythropoietin levels sufficiently to increase RBC mass and thus increase oxygen-carrying capacity.[12,14,32]

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

CHAPTER 2

# High-Altitude Medicine and Pathophysiology

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Increasingly, physicians and other health care providers are confronted with questions of prevention and treatment of high-altitude medical problems (Box 2-1). Despite advances in high-altitude medicine, significant morbidity and mortality persist (Table 2-1). Clearly, better education of the population at risk and those advising them is essential. Given the number of persons impacted, altitude illness should be considered a public health problem. In this chapter, we review recognition, pathophysiology, and management of medical problems for high-altitude visitors and residents.

## HIGH-ALTITUDE SYNDROMES

High-altitude syndromes are attributed directly to hypobaric hypoxia. Clinically, these syndromes overlap. Rapidity of hypoxia exposure is crucial in determining the syndrome. For example, sudden exposure to extreme altitude may result in death from acute hypoxia (asphyxia), whereas more gradual ascent to the same altitude may result in acute mountain sickness (AMS) or no illness at all. An example of overlap is the vague line between acute hypoxia of more than 1-hour duration and AMS, as reflected in the classic experiments of Bert.[47] For neurologic syndromes, the spectrum of illness encompasses high-altitude headache, AMS, and high-altitude cerebral edema (HACE) (Table 2-2). The pulmonary problems range from pulmonary hypertension to interstitial and alveolar edema (HAPE). These problems all occur within the first few days of ascent to a higher altitude and have many common features, and patients respond to descent or oxygen, thus reflecting some commonality of pathogenesis. Longer-term problems of altitude exposure include high-altitude deterioration in sojourners and chronic mountain sickness, pulmonary hypertension, and right-sided heart failure in high-altitude residents.

### ACUTE CEREBRAL HYPOXIA

Acute, profound hypoxemia, although of greatest interest in aviation medicine, may also occur when ascent is too rapid or hypoxia abruptly worsens. Carbon monoxide (CO) poisoning, pulmonary edema, sudden overexertion, sleep apnea, or a failed oxygen delivery device may rapidly impair blood and tissue oxygenation. Unacclimatized persons become unconscious from acute hypoxia at arterial oxygen saturation ($Sa_{O_2}$) of 40% to 60%, arterial oxygen partial pressure ($Pa_{O_2}$) of less than about 30 mm Hg, or mixed venous $P_{O_2}$ of 15 mm Hg. The effects of acute severe hypoxia are best described by Tissandier, the sole survivor of the flight of the balloon Zenith in 1875, who gave a graphic description of the effects of their overly rapid ascent to very high elevation:

> *But soon I was keeping absolutely motionless, without suspecting that perhaps I had already lost use of my movements. Towards 7500 m [24,606 feet], the numbness one experiences is extraordinary. The body and the mind weaken little by little, gradually, unconsciously, without one's knowledge. One does not suffer at all; on the contrary. One experiences inner joy, as if it were an effect of the inundating flood of light. One becomes indifferent; one no longer thinks of the perilous situation or of the danger; one rises and is happy to rise. Vertigo of the lofty regions is not a vain word. But as far as I can judge by my personal impression, this vertigo appears at the last moment; it immediately precedes annihilation—sudden, unexpected, irresistible. I wanted to seize the oxygen tube, but could not raise my arm. My mind, however, was still very lucid. I was still looking at the barometer; my eyes were fixed on the needle which soon reached the pressure number of 280, beyond which it passed. I wanted to cry*

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**BOX 2-1 Medical Problems of High Altitude**

**Lowlanders on Ascent to Altitude**
Acute hypoxia
High-altitude headache
Acute mountain sickness
High-altitude cerebral edema
Focal neurologic conditions
High-altitude pulmonary edema
Symptomatic pulmonary hypertension
High-altitude deterioration
Organic brain syndrome (extreme altitude)
Peripheral edema
High-altitude retinal hemorrhage
Impaired sleep
Nocturnal periodic breathing
High-altitude pharyngitis, bronchitis, and cough
Ultraviolet keratitis (snowblindness)
Exacerbation of preexisting medical conditions

**Lifetime or Long-Term Residents of Altitude**
Chronic mountain sickness (chronic mountain polycythemia)
Symptomatic high-altitude pulmonary hypertension with or without right-sided heart failure
Problems of pregnancy: preeclampsia, hypertension, and low-birth-weight infants
Reentry high-altitude pulmonary edema

*out "We are at 8,000 meters." But my tongue was paralyzed. Suddenly I closed my eyes and fell inert, completely losing consciousness.*[47]

The ascent to over 8000 m (26,247 feet) took 3 hours, and the descent less than 1 hour. When the balloon landed, Tissandier's two companions were dead.

The prodigious work that Paul Bert conducted in an altitude chamber during the 1870s showed that lack of oxygen, rather than an effect of isolated hypobaria, explained the symptoms experienced during rapid ascent to extreme altitude:

*There exists a parallelism to the smallest details between two animals, one of which is subjected in normal air to a progressive diminution of pressure to the point of death, while the other breathes, also to the point of death, under normal pressure, an air that grows weaker and weaker in oxygen. Both will die after having presented the same symptoms.*[47]

Bert goes on to describe the symptoms of acute exposure to hypoxia:

*It is the nervous system which reacts first. The sensation of fatigue, the weakening of the sense perceptions, the cerebral symptoms, vertigo, sleepiness, hallucinations, buzzing in the ears, dizziness, pricklings … are the signs of insufficient oxygenation of central and peripheral nervous organs. … The symptoms … disappear very quickly when the balloon descends from the higher altitudes, very quickly also … the normal proportion of oxygen reappears in the blood. There is an unfailing connection here.*[47]

Bert was also able to prevent and immediately resolve symptoms by breathing oxygen. Modern studies of acute hypoxic exposure use the measurement of "time of useful consciousness," that is, the time until a person can no longer take corrective measures, such as putting on an oxygen mask. For a sea level resident with exposure to 8500 m (27,887 feet), that time is 60 seconds during moderate activity and 90 seconds at rest.

Acute hypoxemia can be quickly reversed by immediate administration of oxygen, rapid pressurization or descent, or correction of an underlying cause, such as relief of apnea, repair of an oxygen delivery device, or cessation of overexertion. Hyperventilation increases alveolar oxygen pressure ($P_{AO_2}$) and time of useful consciousness during severe hypoxia.

## HIGH-ALTITUDE HEADACHE

The term *high-altitude headache* (HAH) has been used in the literature for decades, and studies directed toward the pathophysiology and treatment of HAH have been reported. Ravenhill, writing in 1913, first described the headache of AMS, which is the same as HAH:

*It is a curious fact that symptoms of puna do not usually evince themselves at once. The majority of newcomers have expressed themselves as being quite well on first arrival. As a rule, towards the evening the patient begins to feel rather slack and disinclined for exertion. He goes to bed, but has a restless and troubled night, and wakes up the next morning with a severe frontal headache. There may be vomiting, frequently there is a sense of*

**TABLE 2-1 Incidence of Altitude Illness in Various Groups**

| Study Group | Number at Risk per Year | Sleeping Altitude (m [ft]) | Maximum Altitude Reached (m [ft]) | Average Rate of Ascent* | Percent with AMS | Percent with HAPE and/or HACE | Reference |
|---|---|---|---|---|---|---|---|
| Western state visitors | 30 million | ~2000 (6562)<br>~2500 (8202)<br>~≥3000 (9843) | 3500 (11,483) | 1-2 | 18-20<br>22<br>27-42 | 0.01 | 178 |
| Mt Everest trekkers | 37,000† | 3000-5200 (9843-17,060) | 5500 (18,045) | 1-2 (fly in)<br>10-13 (walk in) | 47<br>23<br>30-50 | 1.6<br>0.05 | 155<br><br>310 |
| Denali climbers | 1200 | 3000-5300 (9843-17,388) | 6194 (20,322) | 3-7 | 30 | 2-3 | 148 |
| Mt Rainier climbers | 10,000 | 3000 (9843) | 4392 (14,409) | 1-2 | 67 | — | 238 |
| Mt Rosa, Swiss Alps | ‡ | 2850 (9350)<br>4559 (14,957) | 2850 (9350)<br>4559 (14,957) | 1-2<br>2-3 | 7<br>27 | —<br>5 | 270<br>99, 270, 390 |
| Indian soldiers | Unknown | 3000-5500 (9843-18,045) | 5500 (18,045) | 1-2 | ‡ | 2.3-15.5 | 409, 410 |
| Aconcagua climbers | 4200 | 3300-5800 (10,827-19,029) | 6962 (22,841) | 2-8 | 39 (LLS >4) | 2.2 | 332 |

*AMS*, Acute mountain sickness; *HAPE*, high-altitude pulmonary edema; *HACE*, high-altitude cerebral edema; *LLS*, Lake Louise score.
*Days to sleeping altitude from low altitude.
†Data for 2014, extracted from Sagarmatha National Park entry station, Jorsale Nepal, on March 26, 2015.
‡Reliable estimate unavailable.

**TABLE 2-2** Clinical Characteristics of Neurologic High-Altitude Illnesses

| | Clinical Classification | | | |
|---|---|---|---|---|
| | **HAH** | **Mild AMS** | **Moderate to Severe AMS** | **HACE** |
| Symptoms | Headache only | Headache plus one more symptom (nausea/vomiting, fatigue/lassitude, dizziness, or difficulty sleeping)<br>Symptoms of mild severity | Headache plus one or more symptoms (nausea/vomiting, fatigue/lassitude, dizziness, or difficulty sleeping)<br>Symptoms of moderate to severe intensity | ±Headache<br>Worsening of symptoms seen in moderate to severe AMS |
| LL-AMS score* | 1-3, headache only | 2-4 | 5-15 | — |
| Physical signs | None | None | None | Ataxia<br>Altered mental status<br>Papilledema; concurrent HAPE common |
| Findings | None | None | Antidiuresis<br>Slight desaturation<br>Widened A-a gradient<br>White matter edema in some (CT, MRI) | Positive chest radiograph if HAPE present,<br>Elevated ICP<br>White matter edema (CT, MRI) |

*AMS,* Acute mountain sickness; *CT,* computed tomography; *HACE,* high-altitude cerebral edema; *HAH,* high-altitude headache; *HAPE,* high-altitude pulmonary edema; *ICP,* intracranial pressure; *MRI,* magnetic resonance imaging.
*Lake Louise self-reported score. (From Roach RC, Bärtsch P, Oelz O, Hackett PH: The Lake Louise acute mountain sickness scoring system. In Sutton JR, Houston CS, Coates G, editors: *Hypoxia and molecular medicine,* Burlington, Vt, 1993, Queen City Press, pp 272-274.)

***oppression in the chest, but there is rarely any respiratory distress or alteration in the normal rate of breathing so long as the patient is at rest. The patient may feel slightly giddy on rising from bed, and any attempt at exertion increases the headache, which is nearly always confined to the frontal region.***[344]

Drawing a distinction between HAH and AMS is rather artificial because they overlap. Headache is generally the first unpleasant—and sometimes the only—symptom resulting from high-altitude exposure.[178] Headache without other symptoms is called HAH, and if associated with other typical symptoms, it is called AMS. Therefore, investigations on HAH are also to some extent studies of AMS. Headache lends itself to investigation better than some other symptoms because headache scores have been well validated.[199] One could argue that the headache itself causes other symptoms, such as anorexia, nausea, lassitude, and insomnia, as often seen in migraine or tension headaches, and that mild AMS is essentially caused by headache.

Researchers have attempted to characterize the clinical features and incidence of headache at altitude. In one study, 50 of 60 trekkers (83%) in Nepal up to 5100 m (16,732 feet) developed at least one headache when over 3000 m (9843 feet).[406] Older persons were less susceptible; women and those with headaches in daily life had more severe headaches, but no more headaches than did others. Of those with headache, 52% did not have AMS by the Lake Louise criteria. Although the clinical features were widely variable, in general the headaches were mild to severe in intensity, frontal (41%) or frontal-temporal (23%), bilateral (81%), dull (53%) or pulsatile (32%), exacerbated by exertion or movement (81%), often occurred at night, had onset in the first 24 hours at a new altitude, and resolved within the next 24 hours. The headaches were considered to have some features of increased intracranial pressure. Persons with history of migraine did not have a higher incidence of headache. In contrast, another investigator found a history of migraine associated with a higher incidence of headache at altitude.[62] Various medications alleviated the headaches, especially mild ones, in 70% of cases. Based on these investigations, the International Headache Society has defined HAH as headache developing after ascent to altitude above 2500 m (8202 feet) and resolving within 24 hours of descent and having at least two of the following characteristics: (1) bilateral, (2) mild or moderate intensity, and (3) aggravated by exertion, movement, straining, coughing, or bending.[443] This may not be useful for diagnosis, however, because headaches from a variety of causes fulfill these criteria.

### Pathophysiology

Sanchez del Rio and Moskowitz[381] have provided a useful multifactorial concept of the pathogenesis of HAH, based on current understanding of headaches in general. They suggest that the trigeminovascular system is activated at altitude by both mechanical and chemical stimuli (vasodilation, nitric oxide, and other noxious agents), and that the threshold for pain is likely altered at high altitude (Figure 2-1). If AMS and especially HACE ensue, altered intracranial dynamics may also play a role, through compression or distention of pain-sensitive structures (see AMS Pathophysiology, below). Oxygen is often immediately (within 10 minutes) effective for HAH in persons with and without AMS, which indicates a rapidly reversible mechanism of the headache, most likely related to cerebral vasodilation with increased cerebral blood flow and cerebral blood volume.[21,159] More investigation should lead to a better understanding of the pathophysiology of these often debilitating headaches, as well as new treatments.

### Prevention and Treatment

In general, HAH can be prevented by nonsteroidal antiinflammatory drugs (NSAIDs),[325] acetaminophen,[170] and the drugs typically used for prophylaxis of AMS, acetazolamide and dexamethasone (Table 2-3). Some agents appear more effective than others, with ibuprofen and aspirin apparently superior to naproxen.[57,60,63] Sumatriptan, a serotonin type 1 (5-HT1) receptor agonist, was reported to be effective for HAH prevention and treatment in some studies[61,198] but not in others.[23] Flunarizine, a specific calcium antagonist used for treatment of migraine, was not effective for HAH in one study.[38] The response to different agents might reflect multiple components of HAH pathophysiology or merely the nonspecific nature of many analgesics.

## ACUTE MOUNTAIN SICKNESS

### Epidemiology and Risk Factors

Although the syndrome of AMS has been recognized for centuries, modern rapid transportation and proliferation of participants in mountain sports have increased the number of persons affected and therefore public awareness (see Table 2-1). Incidence and

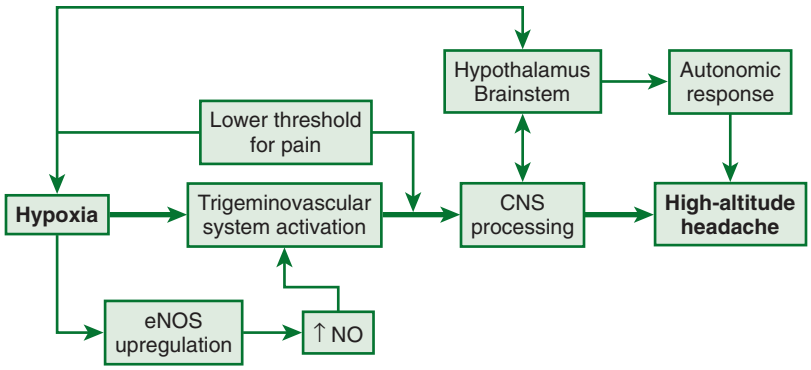


**FIGURE 2-1** Proposed pathophysiology of high-altitude headache. *CNS,* Central nervous system; *eNOS,* endothelial nitric oxide synthase; *NO,* nitric oxide. *(Modified from Sanchez del Rio M, Moskowitz MA: High altitude headache. In Roach RC, Wagner PD, Hackett PH, editors:* Hypoxia: Into the next millennium, *New York, 1999, Plenum/Kluwer Academic Publishing, pp 145-153.)*

severity of AMS depend on rate of ascent and altitude attained (especially sleeping altitude), duration of altitude exposure, level of exertion, recent altitude exposure, and inherent physiologic (genetic) susceptibility.[49,156,364,390] For example, AMS is more common on Mt Rainier because of rapid ascents, whereas high-altitude pulmonary or cerebral edema is uncommon because of short duration (<36 hours) of exposure on the mountain. Persons with demonstrated susceptibility to AMS had twice the incidence of AMS compared with nonsusceptible persons, independent of rate of ascent.[390] The basis for inherent susceptibility is still unknown.

Acclimatization induced by recent altitude exposure can be protective; 4 nights or more in the previous 2 months above 3000 m (9843 feet) reduced susceptibility to AMS on ascent to 4559 m (14,957 feet) by one-half and was as effective as slow ascent[390] (Figure 2-2). Repeated overnight normobaric hypoxia exposures before ascent may also have a preventive effect.[90,130]

Depending on the duration of stay at high altitude, the protective effects of acclimatization persist after descent to low altitude. AMS did not develop in persons who acclimatized to 4300 m (14,108 feet) for 16 days, returned to low altitude for 8 days, and then were reexposed to high altitude in a hypobaric chamber.[265] In addition, ventilation, $SaO_2$, and exercise performance at altitude were maintained for at least 7 days after acclimatization,[41,265,311] with some enhanced physical performance retained for several weeks.[53,54,122,226,227] Epidemiologic studies suggest that protection from AMS may persist for months after acclimatization for 2 or more weeks.[390,486] Retention of augmented hypoxic ventilatory response (HVR) might explain these results;[255,359] however, studies showed that HVR returned to pre-ascent values 7 days after descent.[311] Recently, the AltitudeOmics project confirmed many of these findings, with retention of acclimatization for protection from AMS and improvement in exercise performance lasting for up to 3 weeks after 16 days of acclimatization.[112,120,141,360,377,426-428] It was proposed that altered gene expression was maintained after acclimatization through DNA methylation and hypoxia-sensitive microRNAs (so-called hypoxamirs),[234,257] and that these processes may account for at least part of the memory of acclimatization.[109]

Compared with persons living at a lower altitude, residents at 900 m (2953 feet) or above had less AMS (8% vs. 27%) when ascending to between 2000 and 3000 m (6562 and 9843 feet) in Colorado.[178] Age may have an influence on incidence,[156] with persons older than 50 years somewhat less vulnerable.[363,406] In a large study in Colorado, persons older than 60 years had one-half the incidence of AMS as those under 60, whereas a study of 827 mountaineers in Europe showed no influence of age on susceptibility.[390] Different populations and physical activity may explain differing results. No study has ever shown older people to be more susceptible. The evidence regarding risk of AMS in children

**TABLE 2-3** Recommended Dosages for Medications Used in the Prevention and Treatment of Altitude Illness

| Medication | Indication | Route | Dosage |
|---|---|---|---|
| Acetazolamide | AMS, HACE prevention | Oral | 125 mg bid<br>Pediatric dose: 2.5 mg/kg/dose q12h, max 125 mg |
| | AMS treatment | Oral | 250 mg bid<br>Pediatric dose: 2.5 mg/kg/dose q12h, max 250 mg |
| Dexamethasone | AMS, HACE prevention | Oral | 2-4 mg q6h or 4 mg q12h (see text)<br>Pediatrics: should not be used for prophylaxis |
| | AMS, HACE treatment | Oral, IV, IM | AMS: 4 mg q6h<br>HACE: 8 mg once then 4 mg q6h<br>Pediatric dose: 0.15 mg/kg/dose q6h up to 4 mg |
| Nifedipine | HAPE treatment | Oral | 30 mg SR version q12h |
| | HAPE prevention | Oral | 30 mg SR version q12h |
| Tadalafil | HAPE prevention | Oral | 10 mg bid |
| Sildenafil | HAPE prevention | Oral | 50 mg q8h |
| Salmeterol | HAPE prevention | Inhaled | 125 mcg bid* |

Modified from Luks AM, McIntosh SE, Grissom CK, et al: Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Acute Altitude Illness: 2014 update, *Wilderness Environ Med* 25:S4-S14, 2014.
*AMS,* Acute mountain sickness; *HACE,* high-altitude cerebral edema; *HAPE,* high-altitude pulmonary edema; *IM,* intramuscular; *IV,* intravenous; *SR,* sustained-release; *bid,* twice daily; *q,* every; *h,* hours; *mcg,* micrograms.
*Should not be used as monotherapy and should only be used in conjunction with oral medications.

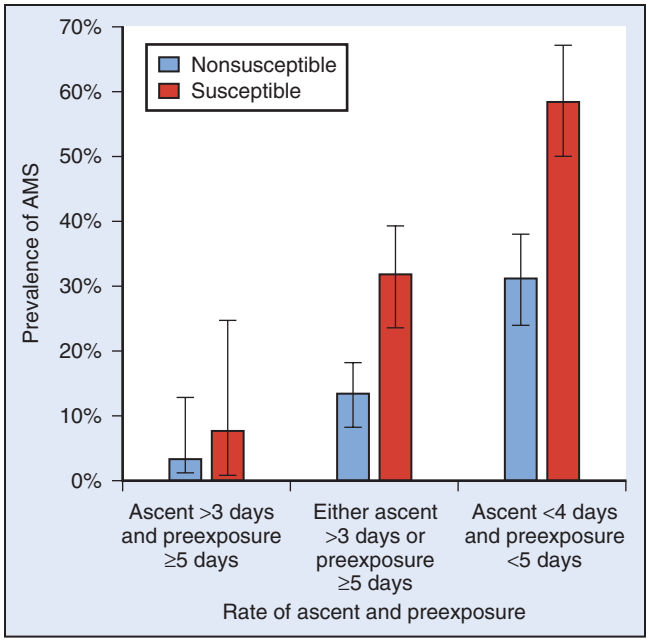


**FIGURE 2-2** Prevalence of acute mountain sickness *(AMS)* and 95% confidence intervals in nonsusceptible *(blue bars)* and susceptible *(red bars)* mountaineers in relation to the state of acclimatization defined as slow ascent (>3 days), fast ascent (≤3 days), preexposed (≥5 days above 3000 m [9843 feet] in preceding 2 months), and not preexposed (≤4 days above 3000 m in preceding 2 months). *(Modified from Schneider M, Bernasch D, Weymann J, et al: Acute mountain sickness: Influence of susceptibility, preexposure, and ascent rate,* Med Sci Sports Exerc *34:1886-1891, 2002.)*

is mixed. Children from 3 months to puberty studied in Colorado had the same incidence as did young adults,[444,494] whereas a small study of tourists in Chile found lower oxygen saturation ($Spo_2$) and higher AMS in those age 6 to 48 months at 4440 m (14,567 feet).[304] The largest study of children to date, of Han Chinese after ascent to Tibet, showed essentially the same incidence of AMS in 464 children as in 5335 adults, 34% and 38%, respectively.[480] Kriemler and colleagues[229] found a lower prevalence of AMS in children than in adolescents and adults on the first day at 3500 m (11,483 feet). The subjective nature of symptom reporting is problematic, especially in children, and must be taken into account when interpreting these studies.[416]

Women apparently have the same incidence[390] or slightly greater incidence[156,178,455] of AMS but may be less susceptible to pulmonary edema.[85,415] Most studies show no relationship between physical fitness and susceptibility to AMS. However, obesity seems to increase the risk of developing AMS.[178,355] The relationship of tobacco smoking to AMS is unclear. A recent study demonstrated that smoking was a risk factor for AMS in workers at a mine at 4000 m (13,123 feet), with an odds ratio of 1.9 for every 10 cigarettes smoked per day.[456] Although one study reported smoking as a risk factor for AMS in trekkers,[283] most studies in trekkers and tourists have found no such relationship,[178,210,355] and some have reported apparent reduction in AMS in persons who smoked.[414,484,495] There is no good evidence that the use of oral contraceptives increases risk for AMS.[210,322]

In summary, the most important variables related to AMS susceptibly are genetic predisposition, altitude of residence, altitude reached, rate of ascent, and prior recent altitude exposure.

It remains difficult to move beyond these established risk factors and accurately predict which individuals are susceptible to severe AMS, HACE, and high-altitude pulmonary edema (HAPE). Canoui-Poitrine[66] and Richalet[353] and colleagues have proposed a prediction tool that takes into account a variety of clinical and physiologic factors, including ventilatory and gas exchange responses during hypoxic exercise. This model, based on a large derivation cohort and subsequently validated in a similarly large cohort, seems to perform well. However, the requirement for conducting an exercise test in hypoxic conditions limits its wide applicability, particularly in travel clinics and primary care clinics, where many high-altitude travelers obtain consultation before their trip.

## Diagnosis

Diagnosis of AMS is based on setting, symptoms, and exclusion of other illnesses. The setting is generally rapid ascent of unacclimatized persons to 2500 m (8202 feet) or higher from altitudes below 1000 m (3281 feet), although AMS has been reported at altitude as low as 2000 m (6562 feet).[301] For persons recently acclimatized, AMS can occur from abrupt ascent to a higher altitude, overexertion, use of respiratory depressants, and perhaps onset of infectious illness.[310]

The cardinal symptom of early AMS is headache, followed in incidence by fatigue, dizziness, and anorexia.[156,178,410] The headache is usually throbbing, bitemporal, typically worse during the night and on awakening, and made worse by the Valsalva maneuver or stooping over (see High-Altitude Headache, earlier). Decreased appetite and nausea are common. These initial symptoms are highly nonspecific and similar to those seen in other situations, such as alcohol hangover and dehydration. Frequent awakening may fragment sleep, and periodic breathing often produces a feeling of suffocation. Although difficulty with sleep is almost universal at high altitude, also affecting persons without AMS, these symptoms may be exaggerated during AMS. Affected persons usually complain of a deep inner chill, unlike mere exposure to cold temperature, accompanied by facial pallor. Other symptoms may include vomiting, especially in children, and irritability. Lassitude can be disabling, with the patient too apathetic to contribute to basic needs. Although pulmonary symptoms such as cough and dyspnea on exertion are quite common at high altitude, these are not features of AMS. Worsening respiratory symptoms or dyspnea at rest should prompt further evaluation, with the primary concern being HAPE rather than AMS.

Specific physical findings are lacking in mild AMS. A higher heart rate has been noted in persons with AMS,[29,322] but Singh and associates[410] noted bradycardia (heart rate <66 beats/min) in two-thirds of 1975 soldiers with AMS. Blood pressure is normal, but postural hypotension may be present. Occasionally, localized rales may be present,[270] but this has also been observed in persons without AMS.[153] Slightly increased body temperature with AMS may be present but is not diagnostic.[267] Peripheral oxygen saturation measured by pulse oximetry ($Spo_2$) correlated poorly with presence of AMS during rapid ascent[322,363,369] but was related to AMS during trekking.[34,225] Although these studies suggest that pulse oximetry is of limited utility in diagnosing AMS, others suggest it may be useful for predicting who will develop AMS with further ascent.[206,361] It seems clear that higher-than-average $Spo_2$ is associated with wellness and lack of AMS.

Funduscopy reveals venous tortuosity and dilation; retinal hemorrhages may or may not be present and are neither diagnostic nor specific for AMS. Although more common in AMS than in non-AMS individuals at 4243 m (13,921 feet),[153] a study examining retinal hemorrhages in climbers on Muztagh Ata suggests these are not a harbinger of impending severe altitude illness.[18] Absence of the normal altitude diuresis, evidenced by lack of increased urine output accompanied by retention of fluid, is an early finding in AMS, although not always present.[30,155,253,410,433] Neurologic signs, including altered mental status and ataxia, are not a feature of AMS and, when present, should raise concern for HACE (see later).

Because there are no characteristic physical examination findings, the severity of AMS is classified solely on the basis of symptoms (see Table 2-2).

## Differential Diagnosis

Given the nonspecific nature of the symptoms, AMS is frequently confused with other conditions (Box 2-2). AMS is most often misdiagnosed as a viral flulike illness, hangover, exhaustion, dehydration, or medication or drug effect. Unlike infectious illness, uncomplicated AMS is not associated with fever and myalgia. Hangover is excluded by history, whereas dehydration

**BOX 2-2 Differential Diagnosis of High-Altitude Illnesses**

| | |
|---|---|
| Acute mountain sickness and high-altitude cerebral edema | Dehydration, exhaustion, alcohol hangover, hypothermia, carbon monoxide poisoning, migraine, hyponatremia, hypoglycemia, diabetic ketoacidosis, central nervous system infection, transient ischemic attack, ruptured cerebral arteriovenous malformation or aneurysm, stroke, seizures, brain tumors, ingestion of toxins or drugs, acute psychosis |
| High-altitude pulmonary edema | Asthma exacerbation, acute bronchitis, pneumonia, mucus plugging, hyperventilation syndrome, pulmonary embolism, heart failure, myocardial infarction, pneumothorax |

can be distinguished from AMS by response to fluid administration. AMS is not improved by fluid administration alone; contrary to conventional wisdom, body hydration does not influence AMS susceptibility.[12,68] Migraine may be difficult to distinguish from AMS but may be distinguished by observing the response to oxygen administration or descent. Hyponatremia, caused by volume depletion or more likely excessive free water intake, can mimic AMS and in severe cases HACE,[417] but it is difficult to diagnose in the field without access to laboratory studies. Seizures are much more common in hyponatremia than in HACE. CO toxicity should always be considered for persons cooking in poorly ventilated tents or snow shelters.

## Pathophysiology

The basic cause of HAH and AMS is hypoxemia (Figure 2-3). However, symptoms are somewhat worse with hypobaric hypoxia than with normobaric hypoxia, implying hypobaria plays a minor role, most likely through its effect on fluid retention.[197,212,253,254,365] Because of a time lag in onset of symptoms after ascent and lack of complete reversal of all symptoms with oxygen, AMS is thought to be secondary to the body's responses to modest hypoxia. Even though an altitude of 2500 to 2700 m (8202 to 8858 feet) presents only a minor decrement in arterial oxygen transport ($Sa_{O_2}$ still >90%), AMS is common, and some individuals may become desperately ill.

Some aspects of AMS pathophysiology are clear. Findings documented in mild to moderate AMS include relative hypoventilation,[282,302] impaired gas exchange (interstitial edema),[144,238] fluid retention and redistribution,[30,253,254,433] and increased sympathetic drive.[26,29] Physiologic and pathologic processes, including reporting of symptoms, occur concurrently and in a hypoxic dose-dependent manner; thus, epiphenomena are common. and discriminating physiology from pathophysiology is difficult.

**Mechanisms of Acute Mountain Sickness.** For decades, clinicians have postulated that AMS is a mild form of cerebral edema. Although this appears true for severe AMS,[124,166,184,230,248,281,410] it now seems unlikely that mild to moderate AMS, or high-altitude headache alone, is due to brain edema.[195,204,241,306,397] In most studies, brain volume increases slightly in most persons ascending rapidly to moderate altitude,[15,103,240,274,306] but without a clear link to HAH or AMS. Available data portray a clear picture of increased brain volume immediately on exposure to altitude, increasing over the first 10 hours, and remaining elevated through 32 hours. Spatial compensation occurs, made evident by decreased cerebrospinal fluid (CSF) volume. Thus, total intracranial volume remains normal, although with interindividual variability.[240] The link between increasing brain volume and susceptibility to AMS may be explained by the tight-fit hypothesis.

**The Tight-Fit Hypothesis.** Intracranial compliance is a measure of the brain's ability to buffer changes in volume without significant increases in pressure. However, as brain volume increases, intracranial compliance is reduced.[243] When spatial compensatory mechanisms are exhausted, intracranial pressure (ICP) rises. Spatial compensation is largely determined by CSF dynamics, particularly compliance of the spinal sac, CSF outflow resistance, and pressure gradients across the arachnoid villi. Rate and magnitude of volume expansion are mostly determined by altitude gain, ascent rate, cerebrovascular reactivity to hypoxia and carbon dioxide, and susceptibility to develop vasogenic brain edema (Figure 2-3).

Ross[375] hypothesized that persons with smaller intracranial and intraspinal CSF capacity would be disposed to develop AMS because they would not tolerate brain swelling as well as those with more "room" in the craniospinal axis. Displacement of CSF through the foramen magnum into the spinal canal is the first compensatory response to increased brain volume, followed by increased CSF absorption and decreased CSF formation. In light of our present understanding of increased brain volume on ascent to altitude, this hypothesis is still very attractive. Preliminary data that showed a relationship between preascent ventricular size or brain volume/cranial vault ratios and susceptibility to AMS support this hypothesis.[150,204,475,496] Moreover, robust evidence suggests that intracranial compliance is altered,[228,240,489] and that although ICP might not be elevated at rest, it rises abruptly with isometric maneuvers such as lifting a pack, with Valsalva maneuver, or even with turning the head, which slightly elevates sagittal venous pressure.[171,475] Pressure waves may be observed spontaneously[450] or after hypotensive stimuli because of further transient increases in intracranial blood volume caused by autoregulation.[357] Intracranial compliance and the tight-fit hypothesis in HAH and AMS deserve further study.

**Intracranial Pressure.** In individuals with moderate to severe AMS and HACE, ICP is elevated.[184,230,281,410,472] Therefore, it is certain that exposure to a hypoxic environment can lead to increases in ICP, either because intracranial volume exceeds craniospinal compensatory capacity or because sagittal sinus pressure becomes elevated, or both. It is currently unknown whether altered CSF dynamics and elevated ICP can explain susceptibility to mild forms of mountain sickness (Figure 2-3).

In one report, a neurosurgeon measured ICP changes from a self-implanted ICP-monitoring bolt in himself and in two others.[475] ICP increased slightly (~5 mm Hg) in two of the three at rest, but the single participant with AMS showed a dramatic increase in ICP even on minimal exertion. This study is consistent with recent indirect assessment of elevated ICP in AMS patients based on optic nerve sheath diameter[116,429] and magnetic resonance imaging (MRI).[240] A study that measured opening lumbar pressure revealed significant elevations in AMS patients compared with controls or after AMS resolved.[410] In contrast, one recent study of normobaric hypoxia reported no elevation of ICP in AMS patients when measured indirectly by repeat lumbar puncture after 16 hours of the hypoxia.[204] This result should not be presumed to be contradictory, but in fact may support the major role of spatial compensation in normalizing ICP during prolonged periods of hypoxia.

Hypoxia can cause intracranial venous hypertension,[240,471,473,474] but the impact on HAH and AMS is uncertain. Wilson and colleagues[473] observed modest correlation between cerebral venous sinus blood flow in normoxia and HAH, implying an anatomic venous outflow predisposition to altitude illness. However, despite quantitative analysis of venous sinus morphology, this observation has not been replicated.[240] Further research is needed to delineate the possible role of cerebral venous hypertension in the pathology of high-altitude illness.

In contrast to the steady-state condition, ICP is subject to normal and pathologic fluctuations. The best example and analog of AMS may be patients with idiopathic intracranial hypertension without papilledema. In these patients, steady-state ICP remains normal for prolonged periods, but pathologic pressure waves are observed, especially during sleep. Headache is the primary complaint in these patients.

## Treatment

Management of AMS is based on severity of illness at presentation, logistics, terrain, and experience of the caregiver. Early diagnosis is key, because treatment in the early stages of illness is easier and more successful (Box 2-3 and Table 2-3). Proceeding to a higher sleeping altitude in the presence of symptoms is contraindicated. The person must be carefully monitored for progression of illness. If symptoms worsen despite 24 hours of

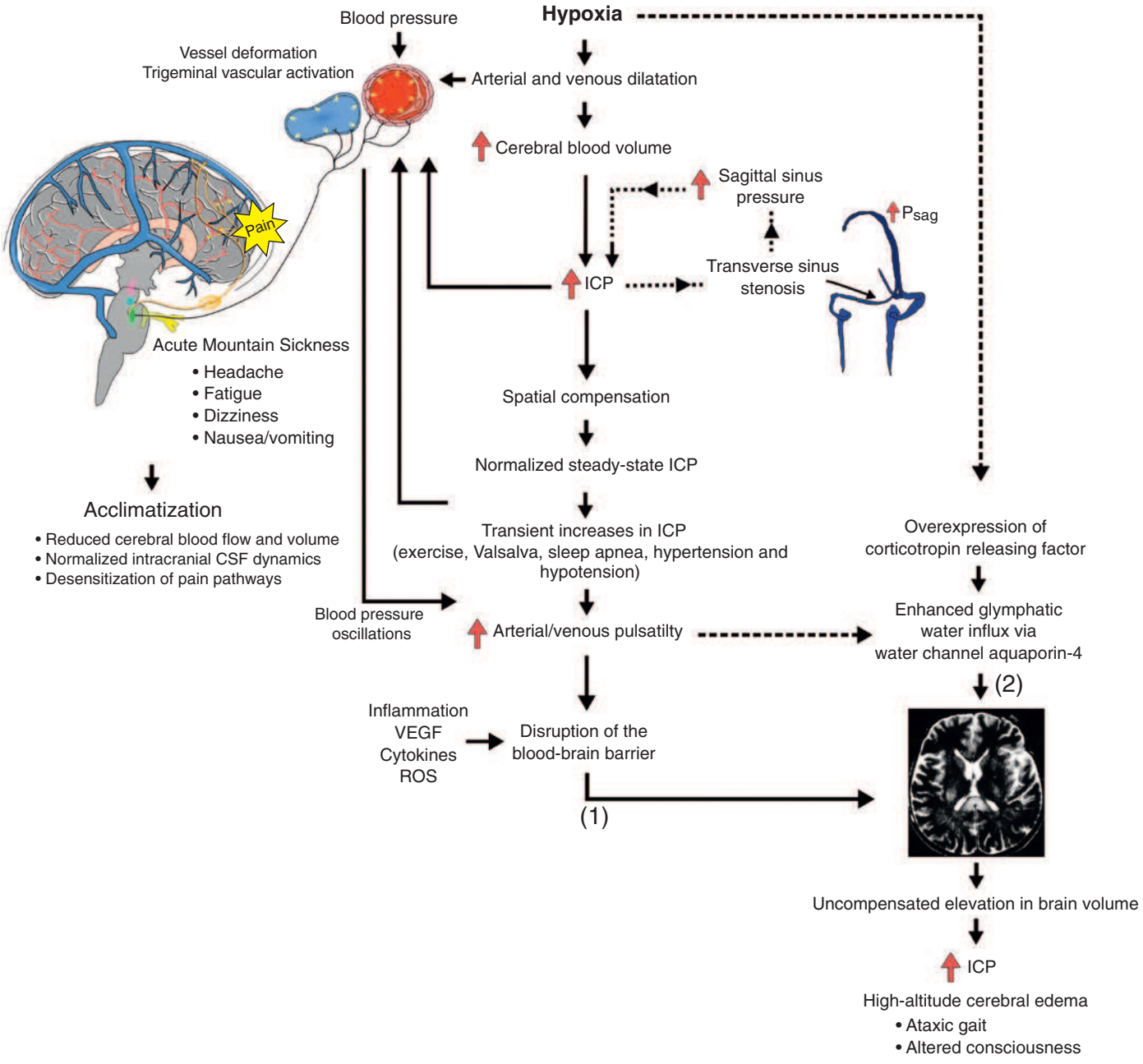


**FIGURE 2-3** Pathophysiology of cerebral forms of altitude illness. Hypoxemia initially causes an increase in arterial and venous blood volume and acute rise in intracranial pressure *(ICP)*. Over time, translocation of cerebrospinal fluid *(CSF)* into the spinal canal and increased CSF absorption (spatial compensation) returns mean ICP to normal levels. However, if transverse sinus stenosis and elevated sagittal sinus pressure *($P_{sag}$)* are present, ICP remains elevated (ICP must be higher than $P_{sag}$ to maintain CSF absorption through arachnoid villi). Irrespective of the steady-state ICP, intracranial compliance is reduced, and large transient fluctuations in ICP will occur (1) during transient increases in $P_{sag}$ (i.e., during Valsalva maneuver), (2) with increases in blood pressure, and (3) with small increases in intracranial volume after hypotensive stimuli because of autoregulation. Four hemodynamic mechanisms are proposed to independently or in combination cause vessel deformation and activation of the trigeminal vascular system, leading to headache and symptoms of acute mountain sickness (AMS). Hypoxic release of chemical mediators may also directly sensitize or activate trigeminal vascular fibers (see Figure 2-1). The fact that several factors could potentially cause headache and symptoms of AMS may explain the preponderance of headache and variability in symptom intensity with rapid ascent to very high altitude. Although dependent on ascent rate and altitude gain, persons resistant to AMS may exhibit an advantageous physiologic response at any stage in this schema (i.e., greater ventilatory drive and $PaO_2$ at any given altitude, lower cerebrovascular reactivity to hypoxia, greater spatial compensatory capacity, or a low nociceptive threshold for trigeminal activation or pain processing). Increased leakiness of endothelial tight junctions from circulating inflammatory mediators, in concert with blood pressure–dependent opening of the blood-brain barrier, may lead to fluid flux into the cerebral parenchyma from the vascular space *(1)*. Alternatively, increased brain water may originate from the CSF via the newly discovered glymphatic (paraarterial pathway) system *(2)*. Overexpression of corticotropin-releasing factor and increased arterial pulse pressure may contribute to fluid influx through the glial-bound water channel aquaporin-4 and explain the early accumulation of intracellular water seen on MRI. In contrast to opening of the blood-brain barrier, alterations in interstitial osmotic potential would "pull" more fluid into the brain, leading to brain edema and diagnosis of high-altitude cerebral edema (HACE). Brain edema may occur in many individuals who ascend rapidly and sleep at very high altitude. Brain edema would lead to further enlargement of brain volume and ICP. Persons with poor spatial compensatory capacity will experience ataxic gait, altered consciousness, and ultimately death. *Dashed lines* indicate new theoretical processes with limited available data. *ROS,* Reactive oxygen species; *VEGF,* vascular endothelial growth factor.

**BOX 2-3 Field Treatment of High-Altitude Conditions**

**High-Altitude Headache and Mild Acute Mountain Sickness**
Stop ascent, rest, and acclimatize at same altitude.
Symptomatic treatment as necessary with analgesics and antiemetics
Consider acetazolamide, 125 to 250 mg bid, to speed acclimatization.
*or* Descend 500 m (1640 ft) or more

**Moderate to Severe Acute Mountain Sickness**
Low-flow oxygen, if available
Acetazolamide, 250 mg bid, or dexamethasone, 4 mg PO, IM, or IV q6h, or both
Hyperbaric therapy
*or* Immediate descent

**High-Altitude Cerebral Edema**
Immediate descent or evacuation
Oxygen, 2 to 4 L/min; titrate to $SpO_2$ >90%
Dexamethasone, 8 mg PO, IM, or IV, then 4 mg q6h
Hyperbaric therapy

**High-Altitude Pulmonary Edema**
Minimize exertion and keep warm.
Immediate descent or hyperbaric therapy
Oxygen, 4 to 6 L/min until improving, then 2 to 4 L/min; titrate to $SpO_2$ >90%
If above measures unavailable, use one of the following:
Nifedipine, 30-mg extended-release tablet q12h
Sildenafil, 50 mg q8h
Tadalafil, 10 mg q12h
Consider inhaled β-agonist as adjunct.

**Nocturnal Periodic Breathing**
Acetazolamide, 62.5 to 125 mg at bedtime as needed

*IM,* Intramuscularly; *IV,* intravenously; *PO,* orally; *$SpO_2$,* oxygen saturation as measured by pulse oximetry; *bid,* twice daily; *q,* every; *h,* hours.

acclimatization or treatment, descent is indicated. Individuals with AMS should also descend if they begin to develop any neurologic finding suggestive of HACE, respiratory symptoms, or hypoxemia suggestive of HAPE.

Mild AMS can be treated by halting ascent and waiting for acclimatization to improve, which can take from 12 hours to 4 days. Acetazolamide (250 mg twice a day orally, or as a single dose) speeds acclimatization and thus terminates the illness if given early.[144,272] Symptomatic therapy includes analgesics such as aspirin (500 or 650 mg), acetaminophen (650 to 1000 mg every 6 hours), ibuprofen (600 mg every 8 hours),[57] or other NSAIDs for headache. Ondansetron (4-mg oral disintegrating tablet every 4 hours as necessary) is useful for nausea and vomiting. Persons with AMS should avoid alcohol and other respiratory depressants because of possible exaggerated hypoventilation and hypoxemia during sleep.

Descent to an altitude lower than where symptoms began effectively reverses AMS. Although descent should be as far as necessary for improvement, 500 to 1000 m (1640 to 3281 feet) is usually sufficient. Exertion should be minimized. Supplemental oxygen is particularly effective (and supply is conserved) if given at low flow (0.5 to 1 L/min by mask or cannula), particularly during the night (e.g., sleep). Hyperbaric chambers are effective and do not require supplemental oxygen but generally are not necessary in most cases of AMS and instead are typically reserved for patients with severe AMS, HACE, or HAPE. Lightweight (<7 kg) fabric pressure bags inflated by manual pumps can be used to simulate descent and treat AMS (Figure 2-4). Chamber inflation of 2 psi is approximately equivalent to a drop in altitude of 1600 m (5249 feet); the exact equivalent depends on initial altitude.[208,362] A few hours of pressurization result in symptomatic improvement and can be an effective temporizing measure while awaiting descent or the benefit of medical therapy.[208,309,326,366,440] Long-term (≥12 hours) use of these portable devices is necessary to resolve AMS completely.

Acetazolamide is now typically and successfully used to treat AMS, although data supporting a role in treatment are minimal.[144,272] Singh and colleagues[410] successfully used furosemide (80 mg twice daily for 2 days) to treat 446 soldiers with all degrees of AMS; furosemide induced brisk diuresis, relieved pulmonary congestion, and improved headache and other neurologic symptoms.[410] It has not since been studied for treatment and is not considered part of standard protocols at this time.[262] Spironolactone, hydrochlorothiazide, and other diuretics have not yet been evaluated for treatment (see Prevention).[262]

The steroid betamethasone was initially reported by Singh and associates[410] to improve symptoms of soldiers with severe AMS. Subsequent studies have found dexamethasone to be effective for treatment of all degrees of AMS.[121,164,214] Hackett and colleagues[164] used 4 mg orally or intramuscularly (IM) every 6 hours, and Ferrazinni and associates[121] gave 8 mg initially, followed by 4 mg every 6 hours. Both studies reported marked improvement within 12 hours, which was the first postdrug assessment, but clinical experience suggests improvement of AMS within 4 hours. There were no significant side effects. Symptoms returned slightly when dexamethasone was discontinued after 24 hours.[164] Other steroids at equivalent dosage are likely effective.

Clinicians debate whether the use of dexamethasone should also mandate descent and whether it is safe to continue ascent after treatment with dexamethasone or while taking the medication. In reality, people do continue ascent, and problems seem to be few. In our opinion, dexamethasone use should be limited to less than 72 hours to minimize side effects. This generally is sufficient time to descend, or better acclimatize, with or without acetazolamide. The exact mechanism of action of dexamethasone is unknown; it does not affect $SaO_2$, fluid balance, or periodic breathing.[248] The drug blocks the action of vascular endothelial growth factor (VEGF),[391] diminishes the interaction of endothelium and leukocytes (thus reducing inflammation),[140] and may also reduce cerebral blood flow.[203] Dexamethasone seems not to improve acclimatization, because some symptoms recur when the drug is withdrawn. Therefore, an argument could be made for using dexamethasone to relieve symptoms and using acetazolamide to speed acclimatization.[46]

## Prevention

Optimal prevention strategy is based on assessment of risk associated with the planned ascent profile (Table 2-4).[262] Graded ascent with slow increases in sleeping elevation is the surest and safest method of prevention, although particularly susceptible individuals may still become ill. Current recommendations are to avoid abrupt ascent from low altitude to sleeping altitudes greater

**FIGURE 2-4** Fabric hyperbaric (pressure) bag being used on Denali for treatment of severe altitude illness; 2 psi of pressure is equivalent to a drop of approximately 1600 m (5249 feet) in altitude.

**TABLE 2-4 Risk Categories for Acute Mountain Sickness**

| Risk Category* | Description† |
|---|---|
| Low | Individuals with no prior history of altitude illness and ascending to <2800 m (9186 ft)<br>Individuals taking >2 days to arrive at 2500-3000 m (8202-9843 ft) with subsequent increases in sleeping elevation <500 m (1640 ft)/day and an extra day for acclimatization every 1000 m (3281 ft) |
| Moderate | Individuals with prior history of AMS and ascending to 2500-2800 m (8202-9186 ft) in 1 day<br>No history of AMS and ascending to >2800 m (9186 ft) in 1 day<br>All individuals ascending >500 m (1640 ft)/day (increase in sleeping elevation) at altitudes above 3000 m (9843 ft) but with an extra day for acclimatization every 1000 m (3281 ft) |
| High | History of AMS and ascending to >2800 m (3281 ft) in 1 day<br>All individuals with a prior history of HAPE or HACE<br>All individuals ascending to >3500 m (11,483 ft) in 1 day<br>All individuals ascending >500 m (1640 ft)/day (increase in sleeping elevation) above >3000 m (9843 ft) without extra days for acclimatization<br>Very rapid ascents (e.g., <7-day ascents of Mt Kilimanjaro) |

Modified from Luks AM, McIntosh SE, Grissom CK, et al: Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Acute Altitude Illness: 2014 update, *Wilderness Environ Med* 25:S4-S14, 2014.
*AMS,* Acute mountain sickness; *HACE,* high-altitude cerebral edema; *HAPE,* high-altitude pulmonary edema.
*The risk categories described above pertain to unacclimatized individuals.
†Altitudes listed in the table refer to the altitude at which the person sleeps. *Ascent* is assumed to start from elevations below 1200 m (3937 feet).

than 2800 m (9186 feet) in 1 day, to spend at least 2 nights at 2500 to 3000 m (8202 to 9843 feet) before going higher, not to advance sleeping altitude more than 500 m (1640 feet) per day, and to take an extra night for acclimatization every 1000 m if continuing ascent.[49,262]

A study on Aconcagua reinforced the idea that individual susceptibility is a key factor, and that persons who are resistant to AMS can proceed much more quickly on the mountain safely.[332] Day trips to higher altitude, with a return to lower altitude for sleep, aid acclimatization. Alcohol and sedative-hypnotics are best avoided the first 2 nights at high altitude. Whether a diet high in carbohydrates reduces AMS symptoms is controversial.[82,169,437] Heavy exertion early in altitude exposure contributes to altitude illness,[364] whereas limited exercise seems to aid acclimatization. Because altitude exposure in the previous weeks is protective, a faster rate of ascent may be possible in individuals who use a program of preacclimatization to high altitude, although the optimal preacclimatization regimen has not been determined.[130,312,352,390,486]

**Acetazolamide Prophylaxis.** Acetazolamide is the drug of choice for AMS prophylaxis. A carbonic anhydrase inhibitor, acetazolamide slows hydration of carbon dioxide ($CO_2$). The effects are protean, involving particularly red blood cells, brain, lungs, and kidneys. By inhibiting renal carbonic anhydrase, acetazolamide reduces reabsorption of bicarbonate and sodium and thus causes bicarbonate diuresis and metabolic acidosis, beginning within 1 hour after ingestion and having full effect at 8 hours.[434,439] This rapidly enhances ventilatory acclimatization, thereby improving oxygenation. Importantly, by decreasing periodic breathing, the drug maintains oxygenation during sleep and prevents periods of extreme hypoxemia.[160,431,436,439] Because of its diuretic action, acetazolamide counteracts the fluid retention of AMS. It also diminishes nocturnal antidiuretic hormone (ADH) secretion[77] and decreases CSF production and volume and possibly CSF pressure. Acetazolamide impacts brain aquaporin channels, possibly preventing water transport into the brain.[400,441] Which of these effects is most important in preventing AMS is unclear, but most experts attribute acetazolamide's benefit to respiratory stimulation and increased alveolar and arterial oxygenation. Numerous studies together indicate that acetazolamide is approximately 75% effective in preventing AMS in persons rapidly transported to altitudes of 3000 to 4500 m (9843 to 14,764 feet).[113]

Acetazolamide prophylaxis should be considered for persons with moderate to severe risk of acute altitude illness, as indicated in Tables 2-3 and 2-4. The ideal dose of acetazolamide for prevention is debated. Many studies have shown that 250 mg two or three times a day was effective, as well as a 500-mg sustained-action capsule every 24 hours.[74,134,143,156,238,348,477] To reduce the side effects, especially paresthesia, clinicians have more recently been using smaller doses (125 mg twice daily),[290] and a number of studies now support this.[32,67,453,258] Renal carbonic anhydrase is blocked with 5 mg/kg/day, and this may be the maximum dose required, both in children and adults. Duration of medication use varies; the standard advice is to begin 24 hours before ascent. For individuals ascending to and remaining at the same elevation for a period of time, the medication can be stopped after 2 days at that elevation, although the duration might be extended if the individual ascended to that elevation at a very fast rate. For individuals climbing to a peak elevation and then descending quickly (e.g., climbing Mt Kilimanjaro), the medication is continued until descent is initiated. Acetazolamide can also be taken episodically, to speed acclimatization at any point while gaining altitude or to treat mild AMS. There is no rebound when discontinued. Although the danger of altitude illness passes after a few days of acclimatization, a single dose of acetazolamide at night may still be useful to promote more effective sleep.

Acetazolamide has side effects, most notably peripheral paresthesia and polyuria, and less often nausea, drowsiness, impotence, and myopia. Because it inhibits the instant hydration of $CO_2$ on the tongue, acetazolamide allows $CO_2$ to be tasted and can ruin the flavor of carbonated beverages, including beer. The issue of sulfonamide allergy and acetazolamide was recently reviewed.[215] As a nonantibiotic sulfonamide drug, acetazolamide is usually tolerated well by persons with a history of sulfa antibiotic allergy; approximately 10% may have an allergic reaction.[423] In persons without a history of allergy to sulfa antibiotic, the incidence of hypersensitivity reaction to a sulfonamide nonantibiotic was 1.6%.[423] The same analysis concluded that persons who are penicillin allergic are actually more likely to react to drugs such as acetazolamide than are those who are sulfa allergic. Nonetheless, it is wise to be cautious in persons with a history of allergy, especially those with anaphylaxis to either sulfa or penicillin or those planning travel into remote areas away from medical resources. Although the usual allergic reaction is a rash starting a few days after ingestion, anaphylaxis to acetazolamide does rarely occur. Although not completely ruling out a future allergic reaction, many experts recommend a trial of acetazolamide well before the altitude sojourn, to determine if the drug is tolerated.

**Dexamethasone Prophylaxis.** Dexamethasone effectively prevents AMS. A dose of 2 mg every 6 hours or 4 mg every 12 hours was sufficient for sedentary individuals,[203] but for exercising individuals at or above 4000 m (13,123 feet), this dosing was insufficient,[45,164,367] and 4 mg every 6 hours was necessary to prevent AMS.[368] The initial chamber study in 1984 was with sedentary persons.[203] Dexamethasone reduced incidence of AMS from 78% to 20%, comparable to previous studies with acetazolamide. Dexamethasone was not as effective in exercising individuals on Pike's Peak,[367] but subsequent work has shown effectiveness comparable with acetazolamide.[37,113,248,299,497] The combination of acetazolamide and dexamethasone proved superior to dexamethasone alone.[497] Because of potential serious side effects and the rebound phenomenon, dexamethasone is best reserved for treatment rather than prevention of AMS, or it can be used for prophylaxis when necessary in persons intolerant of or allergic to acetazolamide. Because of the risk of adrenal suppression and other complications, dexamethasone should not be used for prophylaxis for more than 7 days. A recent case report of severe complications in a Mt Everest climber who used

dexamethasone for altitude illness prevention for 29 days supports this recommendation.[425] If use longer than 7 days is unavoidable, the medication should not be stopped abruptly and the dose slowly tapered over time.

**Other Preventive Agents.** Studies with *Ginkgo biloba* have had inconsistent results. Four studies had positive results, ranging from 100% to 50% reduction in AMS when given either 5 days or 1 day before ascent,[135,303,371] whereas two studies were negative.[74,134] These conflicting results can possibly be explained by differences in dosing, duration of pretreatment, and varying rates of ascent, but most likely are caused by differences in preparations of ginkgo.[242,452] *Ginkgo biloba* is a complicated plant extract whose active ingredient in terms of preventing AMS is unknown. Even in "standardized" preparations (24% flavonoids and 6% terpene ginkolides), the amounts of specific elements can vary considerably. Until the active ingredient is discovered and standardized, results with ginkgo will continue to be mixed. Acetazolamide remains the superior agent and should be used rather than ginkgo when pharmacologic AMS prophylaxis is warranted.[262]

Recent studies suggest ibuprofen may have a role in AMS prevention,[325] but it has not been shown to be superior to acetazolamide and has not been adopted in recent expert guidelines on AMS prevention.[262] In addition, ibuprofen has not been shown to improve nocturnal periodic breathing or nocturnal $Sa_{O_2}$. Spironolactone,[200,237] naproxen, salicylic acid, calcium channel blockers, antacids, leukotriene receptor antagonists, sumatriptan, and medroxyprogesterone acetate have also been studied and are ineffective for AMS prevention. Despite being frequently recommended for travelers in South America for AMS prophylaxis, coca leaves, coca tea, and other coca-derived products have not been studied in a systematic manner, and it remains unclear if they provide any benefit. Bailey and Davies[14] tested an antioxidant "cocktail" for prevention of AMS. They reasoned that free radical–mediated damage to the blood-brain barrier might play a role in the pathophysiology. They preloaded nine individuals with daily L-ascorbic acid, DL-α-tocopherol acetate, and α-lipoic acid, and nine with placebo for 3 weeks, and then during a 10-day trek to the Mt Everest base camp. Those taking antioxidants had a slightly lower AMS score, higher $Sa_{O_2}$, and better appetite.[14] Another study failed to confirm any benefit of antioxidant therapy for preventing AMS.[16] (See Bärtsch and associates[20] for a detailed discussion of the antioxidant hypothesis.)

## HIGH-ALTITUDE CEREBRAL EDEMA

An uncommon but deadly condition, HACE usually occurs in persons with AMS or HAPE. Although HACE occurs most often above 3000 m (9843 feet), it has been reported at altitudes as low as 2100 m (6890 feet).[96] Reliable estimates of incidence range from 0.5% to 1% in unselected high-altitude visitors,[28,156] and incidence was 3.4% in trekkers who had developed AMS.[156] However, HACE incidence in persons with AMS likely is lower now than in this 1976 study because awareness and treatment of AMS have greatly improved. In Chinese railway workers at 4500 to 4900 m (14,764 to 16,076 feet), incidence was 0.5%. HACE occurs in 13% to 20% of persons with HAPE[131,175,189] and in up to 50% of those who die from HAPE. In addition, HAPE is very common in those diagnosed with HACE; one series from Colorado reported that 11 of 13 patients with a primary diagnosis of HACE also had HAPE. Pure cerebral edema, without pulmonary edema, appears to be uncommon at lower altitudes, but it accounted for 54 of 66 HACE cases in Tibet.[485] The mean altitude at onset was 4730 m (15,518 feet) in one survey but was lower (3920 m [12,861 feet]) when associated with HAPE.[190]

Data are insufficient to draw any conclusions regarding effects of gender, age, preexisting illness, or genetics on susceptibility to HACE. Clinically and pathophysiologically, advanced AMS and HACE are similar, so that a distinction between them is inherently blurred. A more complete discussion is available in a prior review[158] and case series.[485]

### Clinical Presentation

High-altitude cerebral edema causes encephalopathy, whereas AMS does not. The hallmarks of HACE are ataxic gait, severe lassitude, and altered consciousness, including confusion, impaired mentation, drowsiness, stupor, and coma. Headache, nausea, and vomiting occur frequently but are not always present. Hallucinations, cranial nerve palsy, hemiparesis, hemiplegia, seizures, and focal neurologic signs have also been reported.[98,167,184,410,485] However, focal neurologic deficits should not readily be attributed to HACE; if they are present without altered mental status, or persist after treatment with oxygen or descent, they should prompt concern for stroke or other issues (discussed later). Seizures are distinctly uncommon. Retinal hemorrhages are common but not diagnostic. Progression from mild AMS to unconsciousness may be as rapid as 12 hours but usually requires 1 to 3 days; HACE can develop more quickly in persons with HAPE. Arterial blood gas (ABG) determination or pulse oximetry often reveals exaggerated hypoxemia. Clinical examination and chest radiography may reveal pulmonary edema. Laboratory studies and lumbar puncture are useful only to rule out other conditions. Computed tomography (CT) may show compression of sulci and flattening of gyri, as well as attenuation of signal more in the white matter than gray matter. MRI is more revealing, with a characteristic high T2 signal in the white matter, especially the splenium of the corpus callosum, and most evident on diffusion-weighted images.[166,479,485] Initial diagnosis in the field must be made based on clinical assessment alone, but the MRI findings may be present for days after evacuation or recovery and thus can be useful if the diagnosis was previously unclear. In fact, hemosiderin deposits in the corpus callosum may be present on MRI for years after HACE.[396]

Case Study 2-1 illustrates a typical clinical course of HACE in conjunction with HAPE.

### Case Study 2-1 HIGH-ALTITUDE CEREBRAL EDEMA: CLINICAL PRESENTATION

H.E. was a 26-year-old German lumberjack with extensive mountaineering experience. Climbing Denali, he ascended to 5200 m (17,060 feet) from 2000 m (6562 feet) in 4 days and attempted the summit (6194 m [20,322 feet]) on the fifth day. At 5800 m (19,029 feet) he turned back because of severe fatigue, headache, and malaise. He returned alone to 5200 m (17,060 feet), stumbling on the way because of loss of coordination. He had no appetite and crawled into his sleeping bag too weak, tired, and disoriented to undress. He recalled no pulmonary symptoms. In the morning, H.E. was unarousable and slightly cyanotic, and was noted to have Cheyne-Stokes respirations. After 10 minutes of high-flow oxygen, H.E. began to regain consciousness, although he was completely disoriented and unable to move. A rescue team lowered him down a steep slope, and on arrival at 4400 m (14,436 feet) 4 hours later, he was conscious but still disoriented and was able to move his extremities but unable to stand. Respiratory rate was 60 breaths/min and heart rate 112 beats/min. Papilledema and a few rales were present. $Sa_{O_2}$ was 54% on room air (normal, 85% to 90%). On a nonrebreather mask with 14 L/min oxygen, $Sa_{O_2}$ increased to 88% and respiratory rate decreased to 40 breaths/min. Dexamethasone (8 mg) was administered IM at 4:20 PM and continued orally, 4 mg every 6 hours. At 5:20 PM, H.E. began to respond to commands. The next morning, H.E. was still ataxic but was able to stand, take fluids, and eat heartily. He was evacuated by air to Anchorage (sea level) at 12 PM. On admission to the hospital at 3:30 PM, about 36 hours after regaining consciousness, H.E. was somewhat confused and mildly ataxic. ABG studies on room air showed $Po_2$ of 58 mm Hg, pH of 7.5, and $Pco_2$ of 27 mm Hg. Bilateral pulmonary infiltrates were present on the chest radiograph. Brain MRI revealed white matter edema, primarily of the corpus callosum (Figures 2-5 and 2-6). On discharge the next morning, H.E. was oriented, bright, and cheerful and had very minor ataxia and clear lung fields.

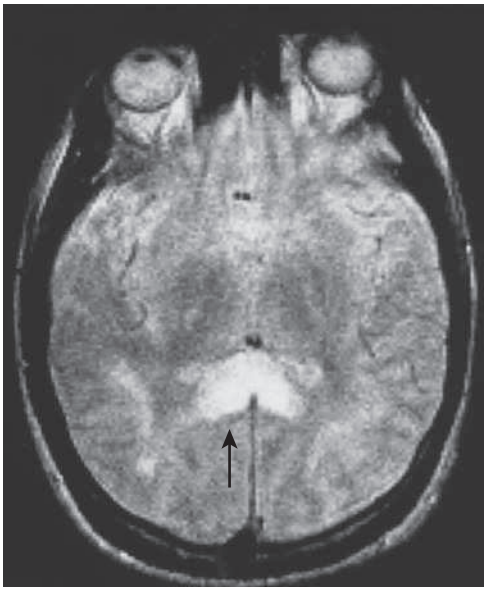

**FIGURE 2-5** Magnetic resonance image of a patient with high-altitude cerebral edema. Increased T2 signal in splenium of corpus callosum *(arrow)* indicates edema. *(Reprinted from Hackett PH, Yarnell PR, Hill R, et al: High-altitude cerebral edema evaluated with magnetic resonance imaging: Clinical correlation and pathophysiology,* JAMA *280: 1920-1925, 1998.)*

## Pathophysiology

The pathophysiology of HACE is a progression of the same mechanism noted for advanced AMS (see AMS Pathophysiology, earlier, and Figure 2-3) and appears to be mixed vasogenic and cytotoxic edema. In cases similar to this, lumbar punctures have revealed elevated CSF pressure (often >300 mm $H_2O$),[184,472] evidence of cerebral edema on CT and MRI,[166,224] and gross cerebral edema on autopsy.[98,99] Small petechial hemorrhages were also consistently found on autopsy, and venous sinus thromboses were occasionally seen.[98,99] Well-documented cases have often included pulmonary edema that was not clinically apparent.

Whereas the brain edema of reversible HACE is most likely vasogenic, as the spectrum shifts to severe, end-stage HACE, gray matter (presumably cytotoxic) edema also develops, culminating in brain herniation and death. As Klatzo[223] has noted, as vasogenic edema progresses, the distance between brain cells and their capillaries increases, so that nutrients and oxygen eventually fail to diffuse and the cells are rendered ischemic, leading to intracellular (cytotoxic) edema. Increased ICP produces many of its effects by decreasing cerebral blood flow, and brain tissue also becomes ischemic on this basis.[287] Focal neurologic signs caused by brainstem distortion and by extraaxial compression, such as third and sixth cranial nerve palsies, may develop,[372] making cerebral edema difficult to differentiate from primary cerebrovascular events. The most common clinical presentation of HACE is a change in consciousness associated with ataxia, without focal signs.[485,491]

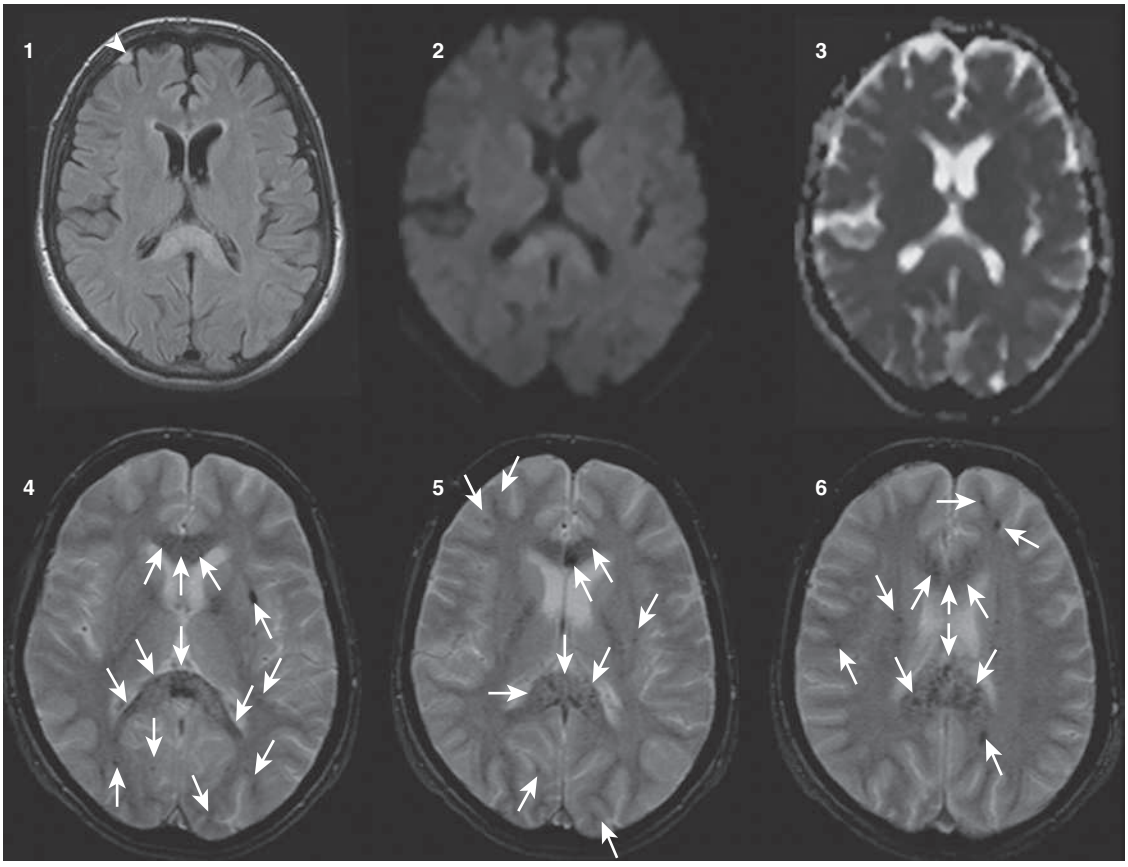


**FIGURE 2-6** Magnetic resonance imaging (MRI) scans of patient with high-altitude cerebral edema (HACE). A fluid-attenuated inversion recovery (FLAIR) image *(1)* shows edema of the corpus callosum, confirmed by diffusion-weighted imaging (DWI) *(2),* with increased values in apparent diffusion coefficient (ADC) *(3),* indicating increased water diffusivity compatible with vasogenic edema; in combination is the characteristic MR pattern consistent with HACE (*1* to *3*) that persists 6 weeks after the event. A novel finding relates to the multiple patchy hypointensities that correspond to microhemorrhages *(arrows)* displayed on the T2 images (*4* to *6*). The right frontal meningioma (visible on FLAIR [*1*]; [*arrowhead*]) is an incidental finding. *(Reprinted from Kallenberg K, Dehnert C, Dorfler A, et al: Microhemorrhages in nonfatal high-altitude cerebral edema,* J Cereb Blood Flow Metab *28:1635-1642, 2008.)*

### Treatment and Prevention

Given the sporadic nature and generally remote locations at which this disorder occurs, it is not surprising that there are no controlled trials on treatment of HACE. All experts agree that successful treatment is vastly enhanced by early recognition. At the first sign of ataxia or change in consciousness, descent should be started, dexamethasone (4 to 8 mg IV, IM, or PO initially, followed by 4 mg every 6 hours) administered, and oxygen (2 to 4 L/min by vented mask or nasal cannula) applied if available (see Box 2-3). Oxygen can be titrated to maintain $Sao_2$ at greater than 90% if oximetry is available. Comatose patients require additional airway management and bladder drainage. Hyperventilation has historically been mentioned as a tool for addressing intracranial hypertension but should be avoided because excessive hyperventilation and respiratory alkalosis can worsen cerebral perfusion and result in disastrous cerebral ischemia. Managing this issue in the field without access to end-tidal $CO_2$ monitoring or ABG measurement can be challenging and dangerous. Loop diuretics such as furosemide (40 to 80 mg) or bumetanide (1 to 2 mg) may reduce brain hydration and have been used successfully,[97,410] but maintenance of adequate intravascular volume and cerebral perfusion pressure is critical. Hypertonic solutions of saline, mannitol, or oral glycerol have been suggested but are rarely used in the field and have not been studied in a controlled manner for this purpose. Hypertonic saline has an advantage over mannitol in that it will not cause intravascular volume depletion. Controlled studies are lacking, but empirically the response to corticosteroids and oxygen seems excellent if these are given early in the course of the illness but is disappointing if these are not started until the patient is unconscious.

Coma may persist for days, even after evacuation to low altitude. Other causes of coma must be considered and ruled out by appropriate evaluation.[184] The average duration of hospital stay in one series of patients with severe HACE was 5.6 days, and average time to full recovery was 2.4 weeks, with a range of 1 day to 6 weeks.[165] Two patients of the 44 in the series by Dickinson[98] remained in coma for 3 weeks.[98] Sequelae lasting weeks are common;[166,184] longer-term follow-up has been limited, but presumed permanent impairment has been reported.[158,184] In the Chinese series of 66 patients, HACE was diagnosed early, and descent was uniformly successful. Ataxia resolved in all but one patient within 7 days, and only one patient had long-term sequelae (clonic convulsions affecting the left hand).[485]

The nonpharmacologic approach to prevention of HACE is the same as for the other forms of acute altitude illness, and the same pharmacologic measures used for AMS prevention are believed to be effective for HACE prevention.

## FOCAL NEUROLOGIC CONDITIONS WITHOUT ACUTE MOUNTAIN SICKNESS OR CEREBRAL EDEMA

Various localizing neurologic signs, transient in nature and not necessarily occurring in the setting of AMS, suggest migraine, cerebrovascular spasm, transient ischemic attack (TIA), local hypoxia without loss of perfusion (watershed effect), or focal edema.[36] Cortical blindness is one such condition. Hackett and colleagues[162] reported six cases of transient blindness in climbers or trekkers with intact pupillary reflexes, which indicated that the condition was caused by a cortical process. Treatment with breathing either $CO_2$ (a potent cerebral vasodilator) or oxygen resulted in prompt relief, suggesting that the blindness was caused by inadequate regional circulation or oxygenation. Descent provided relief more slowly. Other conditions that could be attributed to spasm or "TIA" have included transient hemiplegia or hemiparesis, transient global amnesia, unilateral paresthesias, aphasia, and scotoma.[31,36,51,69,250,478,500] The true mechanism of these focal findings is unknown and may be multifactorial. Young, healthy high-altitude sojourners are unlikely to have TIA syndrome from cerebrovascular disease. The focal findings more likely represent hypoxic/ischemic events resulting from a combination of low $Sao_2$ and low regional perfusion.

Other neurologic complications at high altitude include globus pallidum lesions, which were considered complications of AMS or HACE and reported to lead to Parkinson's disease.[201,424,432,451] Although these deficits were related to high altitude, whether altitude illness was truly the proximate cause of these lesions is unclear. The globus pallidum is sensitive to hypoxia, especially from CO poisoning, and can also be damaged by ischemia.

## CEREBROVASCULAR ACCIDENT (STROKE)

The occurrence of cerebrovascular accident (CVA, stroke) in a young, fit person at high altitude is uncommon and tragic. A number of case reports have described climbers with resultant permanent dysfunction.[76,183,413] Indian soldiers at extreme altitude have a high incidence of stroke.[202] Cerebral venous thrombosis manifests more insidiously, and diagnosis is often delayed.[149,207,379,413,447,500] Factors contributing to stroke may include polycythemia, dehydration, increased ICP if AMS/HACE is present, increased cerebral venous pressure, cerebrovascular spasm, and familial thrombophilia.[33] Stroke may be confused with HACE. Neurologic symptoms, especially focal abnormalities without AMS or HAPE that persist despite treatment with oxygen, corticosteroids, and descent, suggest a cerebrovascular event and mandate careful evaluation with a complete neurologic workup (see Case Study 2-2).

Treatment of stroke is supportive. Oxygen and corticosteroids may be worthwhile to treat any AMS or HACE component. Immediate evacuation to a hospital is indicated; thrombolytics should be withheld until neuroimaging is available to rule out cerebral hemorrhage. Persons with TIA at high altitude should probably start aspirin therapy and proceed to a lower altitude. Oxygen may quickly abort cerebrovascular spasms and will improve watershed hypoxic events. When oxygen is not available, rebreathing to raise alveolar $Pco_2$ may be helpful by increasing cerebral blood flow.

## COGNITIVE CHANGES AT HIGH ALTITUDE

Studies have documented cognitive dysfunction on ascent to altitudes greater than 5500 m (18,045 feet) in some individuals, in the absence of altitude illness and correctable with supplemental oxygen administration. Because acute central nervous system (CNS) dysfunction in this setting must be related to neuronal oxygenation, it is the tissue $Po_2$ that likely affects function. Given the marked variation in arterial and tissue oxygenation among individuals on ascent to altitude, the threshold altitude and appearance and magnitude of such cognitive changes are likely to be quite variable.

Although cerebral oxygen consumption is constant, there are well-documented but mild cognitive changes at high altitude. This phenomenon may be related to specific neurotransmitters. For example, tryptophan hydroxylase in the serotonin synthesis

### Case Study 2-2 CEREBROVASCULAR ACCIDENT (STROKE): CLINICAL PRESENTATION

**E.H., a 42-year-old male climber on a Mt Everest expedition, awoke at 8000 m (26,247 feet) with dense paralysis of the right arm and weakness of the right leg. On descent the paresis cleared, but at base camp (5000 m [16,404 feet]), severe vertigo developed, along with extreme ataxia and weakness. Neurologic consultation on return to the United States resulted in a diagnosis of multiple small cerebral infarcts, but none was visible on CT scan of the brain. The hematocrit value 3 weeks after descent from the mountain was 70%. Over the next 4 years, signs gradually improved, but mild ataxia, nystagmus, and dyslexia persisted. The hematocrit value on the mountain was greater than 70%, high enough for increased viscosity and microcirculatory sludging to contribute to ischemia and infarction. No familial thrombophilia was detected.**

pathway has a high requirement for oxygen (Michaelis constant [$K_m$] = 37 mm Hg).[81,137] Tyrosine hydroxylase in the dopamine pathway is also oxygen sensitive. It has been suggested that decrease in acetylcholine activity during hypoxia might explain lassitude.[137] One study showed that increased dietary tyrosine reduced mood changes and symptoms of environmental stress in persons at simulated altitude.[17] Further work with neurotransmitter agonists and antagonists will help shed light on their role in cognitive dysfunction at altitude and could lead to new pharmacologic approaches to improve neurologic function.

## HIGH-ALTITUDE PULMONARY EDEMA

The most common cause of death related to high altitude, HAPE is completely and easily reversed if recognized early and treated properly. Undoubtedly, HAPE was misdiagnosed for centuries, as evidenced by frequent reports of young, vigorous men suddenly dying of "pneumonia" within days of arriving at high altitude. The death of Dr. Jacottet, "a robust, broad-shouldered young man," on Mt Blanc in 1891 (he refused descent so that he could "observe the acclimatization process" in himself) may have provided the first autopsy of HAPE. Angelo Mosso wrote:

> *From Dr. Wizard's post-mortem examination … the more immediate cause of death was therefore probably a suffocative catarrh accompanied by acute edema of the lungs. … I have gone into the particulars of this sorrowful incident because a case of inflammation of the lungs also occurred during our expedition, on the summit of Monte Rosa, from which, however, the sufferer fortunately recovered.*[308]

On an expedition to K2 (Karakorum Range, Pakistan) in 1902, Alistair Crowley[86] described a climber "suffering from edema of both lungs and his mind was gone." In the Andes, physicians were familiar with pulmonary edema peculiar to high altitude,[196,252] but it was not until Hultgren and Spickard[193] and Houston[182] offered their observations that the English-speaking world became aware of HAPE (see Rennie[349] for a review). Hultgren and colleagues[194] then published hemodynamic measurements in persons with HAPE, demonstrating that it was a noncardiogenic edema. Since that time, many studies and reviews have been published, and HAPE is still the subject of intense investigation. Further information is available in several reviews on this topic.[27,89,259,266,384,388,392,422,435]

Individual susceptibility, rate of ascent, altitude reached, degree of cold,[346] physical exertion, and certain underlying medical conditions are all factors determining the prevalence of HAPE. The incidence varies from approximately 1 in 10,000 skiers at moderate altitude in Colorado to 1 in 50 climbers on Denali (6194 m [20,322 feet]) and up to 6% of mountaineers in the Alps ascending rapidly to 4559 m (14,957 feet).[24] Hultgren and associates[192] reported 150 cases of HAPE over 39 months at a Colorado ski resort at 2928 m (9606 feet). Some regiments in the Indian Army had a much higher incidence of HAPE (15%) because of very rapid deployment to the extreme altitude of 5500 m (18,045 feet) (see Singh and Roy[411] and Table 2-1). Wu and colleagues[485] reported HAPE in 0.5% of Chinese railway workers at 4000 to 4900 m (13,123 to 16,076 feet). Persons with previous HAPE had a 60% attack rate when they went to 4559 m (14,957 feet) in 36 hours, but with slower ascent, some of the same individuals climbed above 7000 m (22,966 feet) without illness.[19,25,] HAPE appears to be less common in women.[84,189,415]

Whether all persons are capable of developing HAPE (with a very rapid ascent to a sufficiently high altitude and with heavy exercise) is arguable.[27] Some studies suggest that many persons contract subclinical extravascular lung water,[85] whereas other studies contradict this.[92] Even well-acclimatized individuals with a sudden push to a higher altitude can succumb to HAPE.[85] A population of HAPE-susceptible persons with unique physiologic characteristics has been described (see Susceptibility to High-Altitude Pulmonary Edema, later). These individuals represent the small percentage of people who develop HAPE when others in the same circumstances do not. HAPE in children is additionally associated with antecedent viral infections, trisomy 21,[106] and underlying congenital cardiovascular malformations, such as unilateral absence of the pulmonary artery.[87]

### Case Study 2-3 HIGH-ALTITUDE PULMONARY EDEMA: CLINICAL PRESENTATION

D.L., a 34-year-old man, was in excellent physical condition and had been on numerous high-altitude backpacking trips, occasionally suffering mild symptoms of AMS. He drove from sea level to the trailhead and hiked to a sleeping altitude of 3050 m (10,007 feet) the first night of his trip in the Sierra Nevada. He proceeded to 3700 m (12,139 feet) the next day, noticing more dyspnea on exertion when walking uphill, a longer time than usual to recover when he rested, and dry cough. He complained of headache, shivering, dyspnea, and insomnia the second night. The third day, the group descended to 3500 m (11,483 feet) and rested, primarily for D.L.'s benefit. That night, D.L. was unable to eat, noted severe dyspnea, and suffered coughing spasms and headache. On the fourth morning, D.L. was too exhausted and weak to exit his sleeping bag. His companions noted that he was breathless, cyanotic, and ataxic but had clear mental status. A few hours later he was transported by helicopter to a hospital at 1200 m (3937 feet). On admission he was cyanotic, oral temperature was 37.8° C (100° F), blood pressure 130/76 mm Hg, heart rate 96 beats/min, and respiratory rate 20 breaths/min. Bilateral basilar rales to the scapulae were noted. Findings of cardiac examination were reported as normal. Romberg and finger-to-nose tests revealed 1+ ataxia. ABG determinations on room air revealed $P_{O_2}$ 24 mm Hg, $P_{CO_2}$ 28 mm Hg, and pH 7.45. Chest radiograph showed extensive bilateral patchy infiltrates (see Figure 2-7C). D.L. was treated with bed rest and supplemental oxygen. On discharge to his sea level home 3 days later, the pulmonary infiltrates and rales had cleared, although ABG values were still abnormal: $P_{O_2}$ 76 mm Hg, $P_{CO_2}$ 30 mm Hg, and pH 7.45. He had an uneventful, complete recovery at home. D.L. was advised to ascend more slowly in the future, staging his ascent with nights spent at 1500 m (4921 feet) and 2500 m (8202 feet). He was taught the early signs and symptoms of HAPE and advised on pharmacologic prophylaxis.

### Clinical Presentation

Case Study 2-3 illustrates typical aspects of HAPE. Patients are frequently young, fit males who ascend rapidly from sea level and may not have previously developed HAPE, even with repeated altitude exposures; the particular ascent may have been faster than any the patient had previously undertaken. HAPE usually occurs within the first 2 to 4 days of ascent to higher altitudes (>2500 m [8202 feet]), most often on the second night.[147] Decreased exercise performance and increased recovery time from exercise are the earliest indications of HAPE. The patient shows fatigue, weakness, and dyspnea on exertion, especially when walking uphill; the person often ascribes these nonspecific symptoms to various other causes. Signs of AMS are present in about 50% of cases.[189] A persistent dry cough develops. Nail beds and lips become cyanotic. Dyspnea at rest is the key clinical sign of HAPE, and tachycardia and tachypnea develop. The condition typically worsens at night. Increasing respiratory distress alerts the patient and travel partners to the development of a serious condition.

In contrast to the usual 1- to 3-day gradual onset, HAPE may strike abruptly, especially in a sedentary person who may not notice the early stages.[458] Orthopnea is uncommon (7%). Pink or blood-tinged, frothy sputum is a very late finding. Hemoptysis was present in 6% of patients in one series.[192] Severe hypoxemia may produce hypoxic encephalopathy or cerebral edema, with mental changes, ataxia, decreased level of consciousness, and coma. Hultgren and colleagues[192] reported an incidence of HACE of 14% in persons with HAPE at ski resorts.

On admission to the hospital, the patient does not generally appear as ill as expected based on ABG and radiographic

findings. Elevated temperature up to 38.5° C (101.3° F) is common. Tachycardia correlates with respiratory rate and severity of illness. Rales[194] may be unilateral or bilateral and usually originate from the right middle lobe. Concomitant respiratory infection is sometimes present.

Pulmonary edema may also manifest with predominantly neurologic manifestations caused by hypoxic encephalopathy and minimal pulmonary symptoms and findings, most likely in persons with blunted ventilatory responses to hypoxemia. Cerebral edema, especially with coma, may obscure the diagnosis of HAPE.[157] Chest radiography and pulse oximetry can be used to confirm the diagnosis. In fact, absence of hypoxemia in a patient with respiratory complaints at high altitude should prompt consideration of diagnoses other than HAPE, because previous series have shown these patients have marked hypoxemia compared to normal individuals at a given elevation. Differential diagnosis includes pneumonia, bronchitis, mucus plugging, pulmonary embolism or infarct, heart failure, acute myocardial infarction, and sometimes asthma (see Box 2-2). Complications include infection, cerebral edema, pulmonary embolism or thrombosis, and such injuries as frostbite or deep vein thrombosis (DVT) secondary to incapacitation.[190,19,157]

### Hemodynamics

Hemodynamic measurements by catheter show elevated pulmonary artery pressure (PAP) and pulmonary vascular resistance, low to normal pulmonary artery wedge pressure, and low to normal cardiac output and systemic arterial blood pressure.[191,271,330] Echocardiography demonstrates high PAP, tricuspid regurgitation, normal left ventricular systolic function, somewhat abnormal diastolic function,[7] and variable right-sided heart findings of increased atrial and ventricular size.[161,323]

The electrocardiogram (ECG) usually reveals sinus tachycardia. Changes consistent with acute pulmonary hypertension, such as right-axis deviation, right bundle branch block, voltage for right ventricular hypertrophy, and P-wave abnormalities, have been described.[189,19] Atrial flutter has been reported, but not ventricular arrhythmias.

### Laboratory Studies

Kobayashi and colleagues[224] reported clinical laboratory values in 27 patients with HAPE that showed mild elevations of hematocrit and hemoglobin, probably caused by intravascular volume depletion and plasma leakage into the lungs. Elevation of peripheral white blood cell count is common but is rarely above 14,000 cells/mL. Serum concentration of creatine phosphokinase (CPK) is increased, mostly from muscle damage, although in two patients, CPK isoenzymes showed brain fraction levels of 1% of total, which may have indicated brain damage.[224] Gao and associates[132] found that NT-ProBNP (brain natriuretic peptide) levels were moderately elevated in patients with HAPE,[132] and that levels resolved on treatment, suggesting NT-ProBNP as a potential useful HAPE biomarker.

Arterial blood gases consistently reveal respiratory alkalosis and marked hypoxemia, more severe than expected for the patient's clinical condition. Because respiratory or primary metabolic acidosis has not been reported, ABG studies are unnecessary if noninvasive pulse oximetry is available to measure arterial oxygenation. At 4200 m (13,780 feet) on Denali, the mean value of $PaO_2$ in HAPE was 28 ±4 mm Hg. Values as low as 24 mm Hg in HAPE are not unusual. Arterial oxygen saturation values in HAPE patients at 4300 m (14,108 feet) ranged from 40% to 70%, with a mean of 56% ±8%.[395] At 2928 m (9606 feet), mean $SaO_2$ was 74%.[192] Arterial acid-base values may be misleading in patients taking acetazolamide, because this drug produces significant metabolic acidosis.

### Radiographic Findings

The radiographic findings in HAPE have been described in original reports[194,275,460,461] (Figure 2-7). Findings are consistent with noncardiogenic pulmonary edema, with generally normal heart size and left atrial size, and no evidence of pulmonary venous prominence, such as Kerley B lines. The pulmonary arteries increase in diameter.[461] Infiltrates are frequently described as fluffy and patchy, with areas of aeration between infiltrates and in a peripheral rather than central location. Infiltrates may be unilateral or bilateral, with a predilection for the right middle lung field, which corresponds to the usual area of auscultated rales. Pleural effusion is rare. Radiographic findings generally correlate with illness severity and degree of hypoxemia. A small right hemithorax, absence of pulmonary vascular markings on the right, and edema confined to the left lung are criteria for diagnosis of unilateral absent pulmonary artery, a condition known to predispose to HAPE.[152,356]

Clearing of infiltrates is generally rapid once treatment is initiated. Depending on severity, complete clearing may take from 1 day to several days. Infiltrates are likely to persist longer if the patient remains at high altitude, even if confined to bed and receiving oxygen therapy. Radiographs taken within 24 to 48 hours of return to low altitude may still be able to confirm diagnosis of HAPE.

An ultrasound technique known as "comet-tail" scoring, previously validated in cardiogenic pulmonary edema, has been used to evaluate extravascular lung water at high altitude, which in the right setting might indicate clinical or subclinical pulmonary edema. The comet-tail artifacts are created by microreflections of the ultrasound beam within interlobular septa thickened by the presence of increased lymphatic fluid and consistent with interstitial and alveolar edema. Eleven patients at 4240 m (13,911 feet) with a clinical diagnosis of HAPE underwent chest ultrasound examinations using this technique.[117] Seven patients with no evidence of HAPE or other altitude illness served as controls. HAPE patients had much higher comet-tail scores and lower $SpO_2$ than did controls, and scores decreased as HAPE cleared. The comet-tail technique seemed to be able to recognize and monitor the degree of pulmonary edema in HAPE.

Another study has reported use of a satellite telemedical connection with a remote expert to guide thoracic ultrasound examinations at Advanced Base Camp on Mt Everest; high-quality images were obtained that verified increases in lung water.[324] Another group, also in Nepal, used the technique while trekking up to 4700 m (15,420 feet) and found a high prevalence of extravascular lung water, but no association with estimated PAP.[337] As technology improves and cost decreases, use of ultrasound in the wilderness setting may increase. This technique may lend itself well to answering the question of whether the oft-reported increase in extravascular lung water on ascent to high altitude and in AMS is actually a precursor to HAPE.

### Pathologic Findings

More than 20 autopsy reports of persons who died of HAPE have been published.[13,99,314,410,411,472] Of those whose cranium was opened, more than one-half had cerebral edema. All lungs showed extensive and severe edema, with bloody, foamy fluid in the airways. Lung weights were two to four times normal. The left side of the heart was normal. The right atrium and main pulmonary artery were often distended. Proteinaceous exudate with hyaline membranes was characteristic. All lungs had areas of inflammation with neutrophil accumulation. The diagnosis of bronchopneumonia was common, although bacteria were not noted. Pulmonary veins, the left ventricle, and the left atrium were generally not dilated, in contrast to the right ventricle and atrium. Most reports mention capillary and arteriolar thrombi and alveolar fibrin deposits, as well as microvascular and gross pulmonary hemorrhage and infarcts. Autopsy findings thus suggest a protein-rich, permeability type of edema with thrombi or emboli. Confirmation of HAPE as a permeability edema was obtained by analysis of alveolar lavage fluid by Schoene and associates,[395] who found a 100-fold increase in lavage fluid protein levels in patients with HAPE compared with well controls and AMS patients.[395] The lavage fluid also had a low percentage of neutrophils, in contrast to findings in adult respiratory distress syndrome. Further evidence for a permeability edema was a 1:1 ratio of aspirated edema fluid protein to plasma protein level found by Hackett and colleagues.[151] In addition, the lavage fluid contained vasoactive eicosanoids and complement proteins, indicative of endothelium-leukocyte interactions. Research using repeated bronchoalveolar lavage as HAPE was developing found that

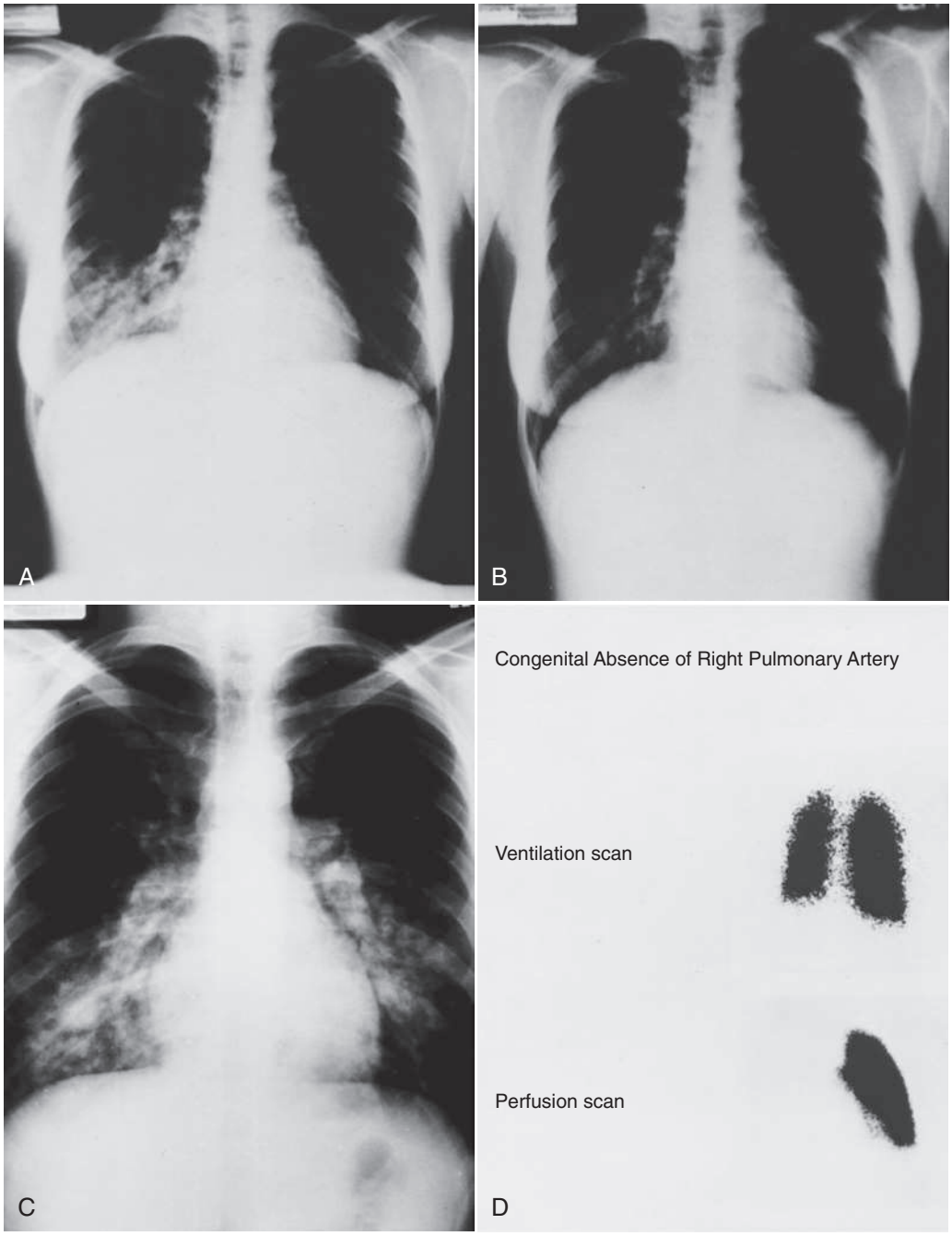


**FIGURE 2-7** **A,** Typical radiograph of high-altitude pulmonary edema (HAPE) in a 29-year-old female skier at 2450 m (8038 feet). **B,** The same patient 1 day after descent and oxygen administration, showing rapid clearing. **C,** Bilateral pulmonary infiltrates on radiograph of a patient with severe HAPE after descent (see Case Study 2-3). **D,** Ventilation and perfusion scans in a person with congenital absence of right pulmonary artery after recovery from HAPE.

inflammation was absent early, suggesting that inflammation is a response to the alveolar damage, rather than an initiating event.[438]

## Mechanisms of High-Altitude Pulmonary Edema

An acceptable explanation for HAPE must take into account three well-established facts: excessive pulmonary hypertension, high-protein permeability leak, and normal function of the left side of the heart. A mechanism consistent with these facts is failure of capillaries secondary to overperfusion and capillary hypertension caused by uneven hypoxic vasoconstriction (Figure 2-8).

**Role of Pulmonary Hypertension.** Excessive PAP is the sine qua non of HAPE; HAPE has not been reported without pulmonary hypertension. All persons ascending to high altitude or otherwise enduring hypoxia, however, have some elevation of PAP. The hypoxic pulmonary vasoconstrictor response (HPVR) is thought to be useful in humans at sea level because it helps match perfusion with ventilation and preserve gas exchange. For example, when local areas of the lungs are poorly ventilated because of infection or atelectasis, the HPVR directs blood away from those areas to well-ventilated regions. In the setting of global hypoxia, as occurs with ascent to high altitude, HPVR is presumably diffuse and pulmonary arterioles in all areas of the lung constrict, causing a restricted vascular bed and increase in PAP, which is of little if any value for ventilation-perfusion

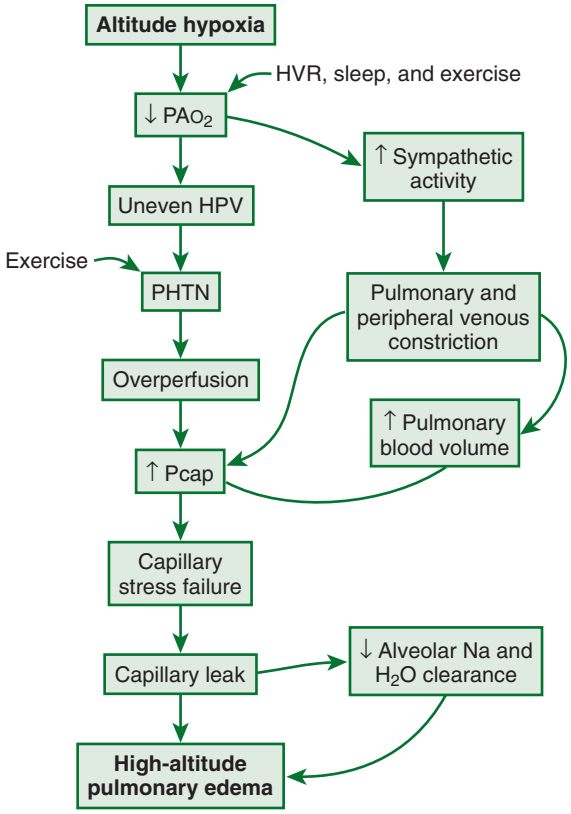


**FIGURE 2-8** Proposed pathophysiology of high-altitude pulmonary edema. *HPV,* Hypoxic pulmonary vasoconstriction; *HVR,* hypoxic ventilatory response; *$PA_{O_2}$,* alveolar partial pressure of oxygen; *Pcap,* capillary pressure; *PHTN,* pulmonary hypertension.

matching at high altitude. The degree of HPVR varies widely among individuals (as well as among species), and is most likely an inherent trait. HAPE-susceptible persons have a greater increase in PAP than do persons who are not susceptible (see later).[91,146] The relationship between the PAP increase and edema formation was well demonstrated by Swenson and colleagues,[438] who performed bronchoalveolar lavage on HAPE-susceptible and normal individuals following rapid ascent to 4559 m (14,957 feet). They demonstrated that individuals with the greatest rise in PAP had the most red blood cells and protein in their bronchoalveolar lavage fluid. Although other factors, such as the vigor of the ventilatory response and subsequent alveolar $P_{O_2}$, may help determine the ultimate degree of pulmonary hypertension, HPVR appears to be the dominant factor. Because all persons with HAPE have excessive pulmonary hypertension, but not all those with excessive pulmonary hypertension have HAPE, it appears that pulmonary hypertension is necessary, but in and of itself is not the cause of HAPE.

**Overperfusion and Capillary Leak.** To explain how pulmonary hypertension might lead to edema, Hultgren suggested that in persons who develop HAPE, hypoxic pulmonary vasoconstriction is uneven, and the delicate microcirculation in an unconstricted (relatively dilated) area is subjected to high pressure and flow, leading to leakage (edema).[439]

The unevenness of HPVR could be caused by anatomic characteristics, such as distribution of muscularized arterioles, or to functional factors, such as loss of HPVR in severely hypoxic regions.[186] Uneven perfusion is suggested clinically by the typical patchy radiographic appearance and is supported by lung CT and MRI during acute hypoxia showing uneven perfusion in persons with a history of HAPE.[93,179,459] Persons born without a right pulmonary artery are highly susceptible to HAPE (see Figure 2-7D),[152] supporting the concept of overperfusion of a restricted vascular bed as a cause of edema, since the entire cardiac output flows into one lung. Other causes of overperfusion of the pulmonary circulation that predispose to HAPE include left-to-right shunts such as atrial septal defect, ventricular septal defect, and patent ductus arteriosus. Rapid reversibility of the illness and response to vasodilators are also consistent with this mechanism. When hydrostatic pressure is reduced, alveolar fluid is quickly reabsorbed.

Other factors contributing to increased hydrostatic pressure, such as exercise or high salt load with subsequent hypervolemia, may also play a role in HAPE.[263] We have in several cases observed onset of HAPE after large salt intake. Some studies have also suggested a role for pulmonary venous constriction, which would contribute to increased capillary hydrostatic pressure.[271,161]

The end result of overperfusion and increased capillary pressure[271] is distention, increased filtration of fluid, and even rupture of the aveolocapillary membrane, called "stress failure,"[467,468] with subsequent leakage of cells and proteins.

**Alveolar Fluid Balance.** Fluid filtration into the interstitial and alveolar space, reabsorption back across the epithelial membrane, and clearance of interstitial fluid by lymph result in a dynamic balance that generally prevents alveolar flooding. On ascent to altitude, the hydrostatic pressure gradient for filtration is increased. As might be expected, multiple lines of evidence suggest that extravascular lung water is increased at altitude.[85,142,337,462] Despite this, frank alveolar flooding is uncommon. This may well be caused by differences in hydrostatic pressure in persons resistant and susceptible to HAPE. However, persons who develop HAPE appear also to have impaired ability to clear alveolar fluid. On a constitutive (genetic) basis, HAPE patients have lower activity of the epithelial sodium channel and therefore reduced ability to transport sodium across the epithelium back into the interstitial space.[273,383,385] (The sodium gradient across the epithelium determines movement of water from alveoli.) In addition, epithelial sodium transport is diminished by hypoxia, so that persons with already-impaired function become more impaired at altitude. How large a role this mechanism plays in HAPE is uncertain[180] (Figure 2-8).

**Control of Ventilation.** As in AMS, persons with HAPE had a lower hypoxic ventilatory response (HVR) than did persons who acclimatized well,[163,279] but not all persons with a low HVR become ill. Thus, low HVR appears to play a permissive, rather than causative, role in HAPE, whereas a brisk HVR appears to be protective. Persons who hypoventilate have lower $P_{AO_2}$ and presumably develop greater pulmonary hypertension. Possibly more important, low HVR may permit episodes of extreme hypoxemia during sleep.[147,163] In addition, a low HVR may allow further ventilatory depression through CNS suppression (hypoxic ventilatory depression). Such persons, when given oxygen, show a "paradoxical" increase in ventilation.[163] Despite correlation of HAPE with low HVR, pretravel identification of patients with blunted HVR has not been able to predict subsequent development of HAPE.[91]

## Susceptibility to High-Altitude Pulmonary Edema

During testing at sea level, persons susceptible to HAPE (HAPE-s) show abnormal rise of PAP and pulmonary vascular resistance (PVR) during hypoxic challenge at rest and exercise, and even during exercise in normoxia, suggesting overreactivity of the pulmonary circulation to both hypoxia and exercise.[91,111,146,209] Part of this reactivity may be related to greater alveolar hypoxemia secondary to lower HVR,[163,176,280] but other factors have been uncovered. Microneurographic recordings from the peroneal nerve during hypoxia established a direct link in HAPE-s between the rise in PAP and greater sympathetic activation,[105] indicating that sympathetic overactivation might contribute to HAPE. Smaller and less distensible lungs have been noted in HAPE-s patients, which might limit the ability to accommodate increased flow by vascular recruitment and thus result in higher PVR.[111,176,418] Another characteristic of HAPE-s is abnormal endothelial function, evidenced by reduced nitric oxide (NO) synthesis during hypoxia[44,64,389] and during HAPE[104] and higher levels of endothelin, a potent pulmonary vasoconstrictor.[43,386,419] The importance of reduced NO is reinforced by studies showing improvement in pulmonary hemodynamics when either NO or

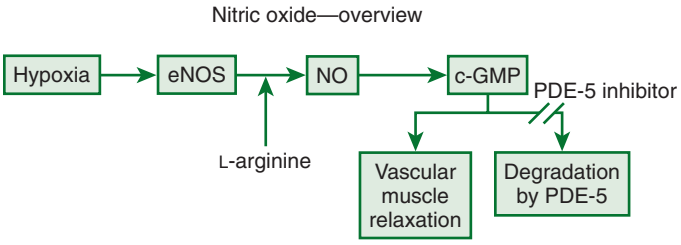


**FIGURE 2-9** Nitric oxide pathway and action of phosphodiesterase-5 *(PDE-5)* inhibitors. In the presence of an inhibitor, cyclic guanosine monophosphate *(c-GMP),* the second messenger of nitric oxide *(NO),* is not degraded, and vasodilation is therefore enhanced. *eNOS,* Endothelial nitric oxide synthase.

a phosphodiesterase-5 (PDE-5) inhibitor was given to HAPE patients[9,136,269,389] (Figure 2-9). As mentioned previously, HAPE-s patients are also characterized by impairment of respiratory transepithelial sodium and water transport, making it more difficult to reabsorb alveolar fluid.[273,383,385]

Patent foramen ovale (PFO) is more common among HAPE-susceptible than among HAPE-resistant adult mountaineers.[6] Since the initial report of Levine and co-workers,[247] the association of PFO and HAPE has been confirmed, but cause and effect have not been established. Allemann and associates[6] performed a case-control study of 16 HAPE-susceptible participants and 19 mountaineers resistant to HAPE. Presence of PFO was determined by transesophageal echocardiography (TEE) and estimated PAP by Doppler echocardiography at low altitude (550 m [1804 feet]) and high altitude (4559 m [14,957 feet]).[6] The frequency of PFO was more than four times higher in HAPE-susceptible than in HAPE-resistant participants, both at low altitude (56% vs. 11%) and high altitude (69% vs. 16%). At high altitude, $Sa_{O_2}$ before the onset of pulmonary edema was significantly lower in HAPE-susceptible participants than in the control group (73% vs. 83%). Also, in the HAPE-susceptible group, participants with a large PFO had more severe arterial hypoxemia ($Sa_{O_2}$ 65% vs. 77%) than did those with smaller or no PFO. The authors speculated that at high altitude, a large PFO may contribute to exaggerated arterial hypoxemia and facilitate HAPE.[6] Others pointed out, however, that the greater hypoxemia may have been caused by subclinical pulmonary edema,[119] or that PFO was a marker of a reactive pulmonary vascular bed.[88] HAPE-s patients are known to have exaggerated PAP response to exercise at sea level, which could maintain an open shunt. Because occurrence of PFO is so common in the general population, more compelling evidence is required before suggesting that PFO is a predisposing factor to HAPE. We do not recommend that those with PFO should consider closure of the defect before significant altitude exposure, or as a result of an episode of HAPE.[71]

## Genetics of High-Altitude Pulmonary Edema

Although many characteristics of HAPE-susceptible persons are apparently genetically determined, actual genetic studies are conflicting and difficult to interpret.* The genes associated with HAPE include those in the renin-angiotensin-aldosterone system pathway,[94,340,420] NO pathway,[1,2,101] and hypoxia-inducible factor (HIF) pathway.[39,59] Some studies showed that polymorphisms of nitric oxide synthase (NOS3),[1,2,101] angiotensin-converting enzyme (ACE),[94,420,341] heat shock protein (HSP) 70,[339] pulmonary surfactant proteins A1 and A2,[387] and aquaporin-5[403] were associated with HAPE incidence and susceptibility, but much work remains to find specific genetic factors causing HAPE.[264] Further investigations using whole-genome scanning and studies of gene expression, gene methylation, and microRNA expression in HAPE-s, and in patients during HAPE and on recovery, will lead to a much clearer understanding of the genomic contributions to HAPE.[293-295,402,464,490]

## Treatment

Treatment choices for HAPE depend on severity of illness and logistics. As with all high-altitude illnesses, early recognition vastly improves the likelihood of successful outcome (see Box 2-3). In the wilderness setting away from medical resources, persons with HAPE need to be urgently evacuated to lower altitude. However, because of augmented pulmonary hypertension and greater hypoxemia with exercise, exertion must be minimized. Because cold stress elevates PAP, the patient should be kept warm.[70] Early HAPE responds rapidly to descent of only 500 to 1000 m (1640 to 3281 feet), and the patient may be able to reascend slowly 2 or 3 days later. When descent is not feasible because of weather or other logistical factors, ill individuals can be treated with oxygen or with hyperbaric therapy using an inflatable pressure bag.[362] If mobilized in these circumstances, rescue groups should make delivery of oxygen to the patient, by airdrop if necessary, the highest priority. In high-altitude locations with oxygen supplies, such as ski resorts, bed rest with supplemental oxygen may suffice, but severe HAPE may require high-flow oxygen (4 L/min) for more than 24 hours.

Oxygen immediately increases arterial oxygenation and reduces PAP, heart rate, respiratory rate, and symptoms. The use of a mask providing resistance on expiration (expiratory positive airway pressure [EPAP]) was shown to improve gas exchange in HAPE, and this or continuous positive airway pressure (CPAP) may be useful as a temporizing measure.[394] The same airway pressure changes can be accomplished with pursed-lip breathing, but this intervention is difficult for an individual to sustain for long periods, particularly when ill. An unusual case report suggested that a climber may have saved his partner's life by using postural drainage to expel airway fluid.[50]

Drugs are of limited necessity in HAPE because oxygen and descent are so effective.[261] Medications that reduce pulmonary blood volume, PAP, and PVR are physiologically rational to use when oxygen is not available or descent delayed. Singh and associates[410] reported good results with furosemide (80 mg every 12 hours), and greater diuresis and clinical improvement occurred when 15 mg of parenteral morphine was given with the first dose of furosemide. Their use, however, has been eclipsed by recent results with vasodilators. Caution is also warranted with diuretic use because HAPE is a capillary-leak phenomenon, and many affected individuals have intravascular volume depletion at presentation. The calcium channel blocker nifedipine (30-mg sustained-release tablet every 12 to 24 hours) was effective in reducing PVR and PAP during HAPE and slightly improved arterial oxygenation.[25] Clinical improvement, however, was not dramatic. Nifedipine is well tolerated and unlikely to cause significant hypotension in healthy persons and avoids the danger of CNS depression from morphine and possible hypovolemia from diuretics. Clinical improvement is much better, however, with oxygen and descent than with any medication. Inhaled NO, a potent pulmonary vasodilator, improves hemodynamics in HAPE but is rarely available, and in any event, NO is usually given with oxygen. The PDE-5 inhibitors, which increase cyclic guanosine monophosphate (c-GMP) to produce pulmonary vasodilation during hypoxia (Figure 2-9),[136,350,351] have shown value for prevention of HAPE[268] but have not yet been studied for treatment. Whether these agents will prove to be more effective than nifedipine for treatment is unknown. A theoretical advantage is that the PDE-5 inhibitors produce less systemic vasodilation. Nifedipine, and perhaps other vasodilators, might be useful adjunctive therapy but is not a substitute for definitive treatment (see Box 2-3 and Table 2-3). β-adrenergic agonists[154,262] may also have an adjunctive role but should not be considered for monotherapy.[262]

After evacuation of the patient to a lower altitude, hospitalization may be warranted for severe cases; home oxygen is also a reasonable approach. Treatment consists of bed rest and oxygen sufficient to maintain $Sa_{O_2}$ greater than 90%; medications are rarely necessary after descent, and rapid recovery is the

*References 1, 2, 94, 100, 101, 168, 181, 235, 256, 300, 307, 340, 339, 376, 412, 421, 446, 463, 466, 499.

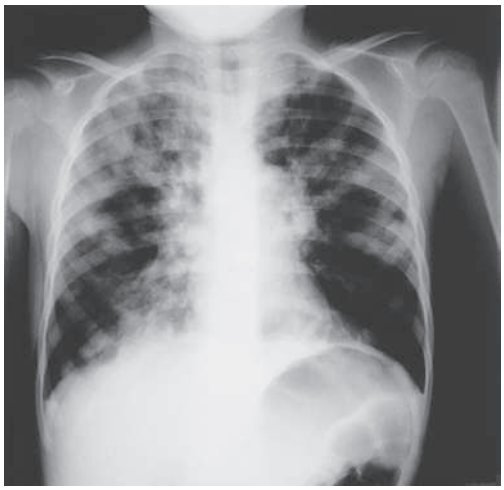

**FIGURE 2-10** Chest radiograph of severe high-altitude pulmonary edema (HAPE) in a 4-year-old girl with a small, previously unrecognized patent ductus arteriosus that predisposed her to HAPE.

rule.[502] In a series of 110 patients with HAPE from the Indian Army, Deshwal and colleagues[95] found no benefit of adding nifedipine when patients were treated with descent and oxygen. Antibiotics are only indicated when there is a high suspicion for infection. Occasionally, pulmonary artery catheterization or Doppler echocardiography is necessary to differentiate cardiogenic from high-altitude pulmonary edema. Endotracheal intubation and mechanical ventilation are rarely needed. A HAPE patient demonstrating unusual susceptibility, such as onset of HAPE despite adequate acclimatization or onset below 2500 m (8200 feet), might require further investigation, such as echocardiography, to rule out an intracardiac shunt or preexisting pulmonary hypertension, or chest radiography to evaluate for unilateral absence of a pulmonary artery. In children, undiagnosed congenital heart disease is worth considering[87] (Figure 2-10). Hospitalization until ABG values are completely normal is not warranted; all persons returning from high altitude are at least partially acclimatized to hypoxemia, and hypocapnic alkalosis persists for days after descent. Distinct clinical improvement, radiographic improvement over 24 to 48 hours, and $Pa_{O_2}$ of 60 mm Hg or $Sa_{O_2}$ greater than 90% are adequate criteria for discontinuation of oxygen therapy.

Patients are advised to resume normal activities gradually and that 1 to 2 weeks may be required to recover complete strength. Physicians should recommend preventive measures, including graded ascent with adequate time for acclimatization, and should provide instruction on use of acetazolamide, nifedipine, or PDE-5 inhibitors for future ascents. An episode of HAPE is not a contraindication to subsequent high-altitude exposure, but education to ensure proper preventive measures and recognition of early symptoms are critical.

## Prevention

The preventive measures previously described for AMS also apply to HAPE: graded ascent, time for acclimatization, low sleeping altitudes, and avoidance of alcohol and respiratory depressants.[262] Exertion may contribute to onset of HAPE, especially at moderate altitude. Reports from North America at 2500 to 3800 m (8202 to 12,467 feet) have included hikers, climbers, and skiers, all of whom were exercising vigorously. Menon[289] clearly showed that sedentary men taken abruptly to higher altitude were just as likely to develop HAPE. Considerable clinical experience suggests that acetazolamide prevents HAPE in persons with a history of recurrent episodes. A growing body of both animal and human research demonstrate that acetazolamide blocks hypoxic pulmonary hypertension[42,177,213,333,405,442] and provides support for this practice, although acetazolamide has never formally been studied for HAPE prevention. Nifedipine (20-mg slow-release tablet every 8 hours) prevented HAPE in persons with a history of repeated episodes.[25] The drug should be carried by such individuals and started at the first signs of HAPE or, for an abrupt ascent, started when leaving low altitude. The PDE-5 inhibitors sildenafil and tadalafil effectively block hypoxic pulmonary hypertension and therefore will prevent HAPE.[268] Tadalafil (10 mg every 12 hours) prevented HAPE with rapid ascent in HAPE-susceptible individuals. The optimal dose has not been established.[266] Regimens for sildenafil have varied from a single dose of 50 or 100 mg just before exposure for acute studies[136,350] to 40 mg three times daily for 2 to 6 days at altitude,[351,331] and for tadalafil, 10 mg every 12 hours was effective.[125,268] Rather surprisingly, dexamethasone was also effective in preventing HAPE in susceptible individuals. Maggiorini[266]gave 8 mg of dexamethasone every 12 hours, starting 2 days before exposure, and found it as effective as tadalafil in reducing PAP and preventing HAPE. Dexamethasone has many actions in the lungs; which particular action explains this observed effect is unknown.[27] As these studies demonstrate, any agent that blocks hypoxic pulmonary hypertension will block onset of HAPE, reinforcing the concept of pulmonary hypertension as the sine qua non of HAPE.

## Problems of Lifelong or Long-Term Residents of High Altitude

**Reentry Pulmonary Edema.** In some persons who have lived for years at high altitude, HAPE develops on reascent after a sojourn to low altitude.[107] Clinicians have suggested that the incidence of HAPE on reascent of altitude residents may be higher than that during initial ascent by flatlanders,[29,188] but data on true incidence are difficult to obtain. Children and adolescents are more susceptible than adults. The phenomenon has been observed most often in Peru, where high-altitude residents can return from sea level to high altitude quite rapidly. Hultgren[187] found HAPE prevalence in Peruvian natives of 6.4 per 100 exposures in persons 1 to 20 years old and 0.4 per 100 exposures in persons older than 21. Cases have also been reported throughout Colorado,[399] but reports are conspicuously rare from Nepal and Tibet,[482] perhaps because Himalayan highlanders are better adapted or because very rapid return back to high altitude was not readily available until recently.[487] Wu[482] reported a 37-year-old Tibetan man with chronic mountain sickness who developed reentry HAPE and suggested that similar cases may have been missed previously.[482] Severinghaus[401] postulated that increased muscularization of pulmonary arterioles that develops with chronic high-altitude exposure generates inordinately high PAP on reascent, causing edema. Whether uneven pulmonary vasoconstriction is present has not been investigated. Changes in fluid balance with descent and reascent might also play a role.[481] The optimal prevention strategy for reentry HAPE is not known, but clinical experience suggests acetazolamide has benefit in this regard.

**Chronic Mountain Sickness.** In 1928, Carlos Monge[296] described a syndrome in Andean high-altitude natives that was characterized by headaches, insomnia, lethargy, plethoric appearance, and polycythemia greater than expected for the altitude. Known variously as Monge's disease, chronic mountain polycythemia, or chronic mountain sickness (CMS), the condition has now been recognized in all high-altitude areas of the world. Lowlanders who relocate to high altitude as well as native residents are susceptible.[231,297,328] Chinese investigators reported that 13% of lowland Chinese males and 1.6% of females who had relocated to Tibet developed excessive polycythemia (hemoglobin level >20 g/dL).[488] The incidence in Leadville, Colorado, is also high in men older than 40 years and is distinctly low in women.[233] Increased hematopoiesis is apparently related to greater hypoxic stress, which may be caused by a number of conditions, such as lung disease, sleep apnea syndromes, and idiopathic hypoventilation. A diagnosis of "pure" chronic mountain polycythemia excludes lung disease and is characterized by relative alveolar hypoventilation, excessive nocturnal hypoxemia, and respiratory insensitivity to hypoxia.[246,297] Some studies suggest that even for the degree of hypoxemia, the red blood cell mass is excessive, implying excessive amounts or overactivity of erythropoietin.[476] International guidelines propose a hemoglobin value

greater than 21 g/dL for males and 19 g/dL for females as essential for the diagnosis of CMS, as well as residence above 2500 m (8202 feet) and absence of lung disease.[246] The reader is referred to reviews for in-depth information.[298,345,347,483] In addition, an international consensus group has defined and scored CMS.[245]

Therapy of CMS is routinely successful. Descent to a lower altitude is the definitive treatment. The syndrome reappears after return to high altitude. Supplemental oxygen during sleep is valuable. Phlebotomy is a common practice and provides subjective improvement, although without significant objective changes.[476] The respiratory stimulants medroxyprogesterone acetate (20 to 60 mg/day)[232] and acetazolamide (250 or 500 mg/day)[354] have also been shown to reduce hematocrit value by improving oxygenation. Acetazolamide (250 mg) increased nocturnal $SaO_2$ by 5%, decreased mean nocturnal heart rate by 11%, the number of apnea/hypopnea episodes during sleep by 74%, and hematocrit by 7%.[354] The fact that these responses likely occur as a result of increased ventilation after acetazolamide administration[358] emphasizes the contribution of hypoventilation and nocturnal desaturation to CMS.[329] Another approach was based on the knowledge that ACE inhibitors blunt hypoxia-mediated erythropoietin release. Plata and associates[334] showed that 5 mg/day of enalapril for 2 years reduced hemoglobin concentration, packed cell volume, proteinuria, and need for phlebotomy.[334] Pulmonary hypertension and right-sided heart failure may also occur in persons with CMS.

**Symptomatic High-Altitude Pulmonary Hypertension.** Children living at high altitude may be at risk for symptomatic high-altitude pulmonary hypertension (SHAPH)[321,329] This clinical entity has also been described in adults.[5] SHAPH has been identified in South America, North America, and Asia, with a predilection in populations not genetically adapted to altitude. Former names for SHAPH have included "pediatric high-altitude heart disease" and "subacute infantile mountain sickness." The underlying pathophysiology is pulmonary hypertension resulting in hypertrophy and dilation of the right ventricle, with eventual right-sided heart failure. Clinically, these children have poor growth, fatigue, irritability, dyspnea, and cyanosis or pallor. On physical examination, as the illness advances, hepatomegaly and leg edema signal right-sided heart failure. ABGs and pulse oximetry reveal exaggerated hypoxemia. Chest radiography shows cardiomegaly and enlarged main pulmonary artery and sometimes infiltrates. Echocardiography usually confirms the diagnosis, although ECG and cardiac catheterization can also be useful. Right ventricular hypertrophy, right atrial dilation, and persistence of PFO and patent ductus arteriosus may also be noted.

Definitive treatment of SHAPH is relocation to a lower altitude. Other, but inferior, therapies include supplemental oxygen, diuretics, and pulmonary vasodilators such as calcium channel blockers, PDE-5 inhibitors, and NO and prostaglandin inhibitors.[246] A similar phenomenon has been also been described in adults after weeks to years of high-altitude residence[5,8] and may respond to treatment with PDE-5 inhibitors.[4]

### CHILDREN AT HIGH ALTITUDE

Children born at high altitude in North America appear to have a higher incidence of complications in the neonatal period than do their lower-altitude counterparts.[319] In populations better adapted to high altitude over many generations, neonatal transition has not been as well scrutinized, but there does appear to be some morbidity. High-altitude residence does not clearly impact eventual stature, but growth and development are slowed.[321,470] In low-income countries, confounding factors, such as nutrition and socioeconomic status, make these issues difficult to assess. Children residing at high altitude are more likely to develop pulmonary edema on return to their homes from a low-altitude sojourn than are lowland children on induction to high altitude. Some of these children may have preexisting pulmonary hypertension of various etiologies.[87]

Although several studies have shown that lowland children traveling to high altitude are just as likely[336] or less likely than adults to develop AMS,[229] no data indicate that children are more susceptible to altitude illness. Studying this issue is difficult, however, because diagnosis can be more difficult in preverbal children.[493] Despite this somewhat reassuring fact, very conservative recommendations are made regarding taking children to high altitude; it should be clarified that these opinions are not based on science.[35,335] Durmowicz and colleagues[107] showed that children with HAPE had a high frequency of concomitant respiratory infections. Children with Down syndrome, past repair of congenital shunts, following chemotherapy, and with other problems are more susceptible to HAPE.[107] Acetazolamide can be used for prevention and treatment of AMS/HACE in children (5 mg/kg/day in divided doses) (see Table 2-3),[108] while dexamethasone should be reserved for treatment only and not used for prevention. Pollard and associates[336] provide an excellent consensus document on children at altitude,[336] and excellent reviews are available.[108,321,493] Healthy term newborns of lowland mothers should probably avoid sleeping altitudes greater than 2500 m (8202 feet) for the first 4 to 6 weeks of life because of concern regarding cardiopulmonary transition rather than for altitude illness.[320] Infants with viral infections, neonatal intensive care unit stay, transient oxygen treatment, Down syndrome, or any pulmonary condition or cardiac defect should be carefully evaluated by a physician regarding altitude exposure (see Figure 2-10). For all infants going to altitude, pulse oximetry monitoring should be considered. Parents need to be aware of potential problems at altitude, including symptoms of AMS unique to infants.[320]

## OTHER MEDICAL CONCERNS AT HIGH ALTITUDE

### CARBON MONOXIDE POISONING

Carbon monoxide poisoning is a danger at high altitude, where field shelters are designed to be small and windproof. Cooking inside poorly ventilated tents and snow shelters during storms is a particular hazard.[126,216,244,373,445,449] The effects of CO and high-altitude hypoxia are additive. A reduction in oxyhemoglobin caused by CO increases hypoxic stress, rendering a person at a "physiologically higher" altitude, which may precipitate AMS. Because of preexisting hypoxemia, smaller amounts of carboxyhemoglobin produce symptoms of CO poisoning. These two problems may coexist. Immediate removal of the victim from the CO source and provision of supplemental oxygen, if available, constitute the treatment of choice. Portable hyperbaric therapy might also be useful.

### HIGH-ALTITUDE DETERIORATION

The world's highest human habitation is at approximately 5500 m (18,045 feet). Above this altitude, deterioration outstrips the ability to acclimatize.[217] The deterioration is more rapid the higher one goes above the maximum point of acclimatization. Above 8000 m (26,247 feet), deterioration is so rapid that without supplemental oxygen, death can occur within days. Life-preserving tasks such as melting snow for water may become too difficult, and death may result from dehydration, starvation, hypothermia, and especially neurologic and psychiatric dysfunction.[123]

Loss of body weight is a prominent feature during expeditions to extreme high altitude. Pugh[338] reported 14 to 20 kg (30.9 to 44 lb) of body weight loss in climbers on the 1953 British Mt Everest Expedition. Almost 30 years later, with improvement in food and cooking techniques, climbers on the American Medical Research Expedition to Mt Everest (AMREE) still lost an average of 6 kg (13.2 lb).[55] This was caused in part by 49% decrease in fat absorption and 24% decrease in carbohydrate absorption. During Operation Everest II (OEII), in which the "climbers" were allowed to eat foods of their choosing ad libitum, they still had large weight losses: 8 kg (17.6 lb) overall, including 3 kg (6.6 lb) of fat and 5 kg (11 lb) of lean body weight (muscle).[374,185] At 4300 m (14,108 feet), weight loss was attenuated by adjusting caloric intake to match caloric expenditure.[65] In fact, recent work

supports hypoxic exposure as a treatment for obesity.[249,315,342,404] Thus, significant weight loss with prolonged exposure to high altitude may be overcome with adequate caloric intake, but decreased appetite is a problem.[448,211] At very high altitudes, an increase in caloric intake may not be sufficient to counteract completely the severe anorexia and weight loss, because other mechanisms may come into play.

Regarding extreme altitude, Ryn[378] reported an incidence of acute organic brain syndrome in 35% of climbers going above 7000 m (22,966 feet), in association with high-altitude deterioration. This syndrome, which features impaired judgment or even psychosis, could directly threaten survival.

## HIGH-ALTITUDE SYNCOPE

Syncope within the first 24 hours of arrival occurs occasionally at moderate altitude[316,317] but is not observed in mountaineers at higher altitudes; it is a problem of acute induction to altitude.[305] The mechanism is an unstable cardiovascular control system, and it is considered a form of neurohumoral (or neurocardiogenic) syncope.[128] An unstable state of cerebral autoregulation may also play a role.[498] These events appear to be random and seldom occur a second time. Preexisting cardiovascular disease is not a factor in most cases. Postprandial state and alcohol ingestion seem to be contributing factors. Altitude syncope has no direct relation to high-altitude illness.[36]

## ALCOHOL AT HIGH ALTITUDE

Two questions regarding alcohol are frequently asked: (1) does alcohol affect acclimatization, and (2) does altitude potentiate the effects of alcohol? Epidemiologic research indicated that 64% of tourists ingested alcohol during the first few days at 2800 m (9186 feet).[178] The effect of alcohol on altitude tolerance and acclimatization might therefore be of considerable relevance. Roeggla and co-workers[370] measured ABGs 1 hour after ingestion of 50 g of alcohol (equivalent to 1 L of beer, five mixed drinks, or five 3-oz glasses of wine) at 171 m (561 feet) and again after 4 hours at 3000 m (9843 feet). A placebo-controlled, double-blind paired design was used. For the 10 participants, alcohol had no effect on ventilation at the low altitude, but at high altitude it depressed ventilation, as gauged by decreased $Pa_{O_2}$ (from 69 to 64 mm Hg) and increased $P_{CO_2}$ (from 32.5 to 34 mm Hg). Whether this degree of ventilatory depression would contribute to AMS or whether repeated doses would have greater effect was not tested. Nonetheless, alcohol might impede ventilatory acclimatization and should be used with caution at high altitude.

Conventional wisdom proffers an additive effect of altitude and alcohol on brain function. McFarland,[285] who was concerned about the interaction in aviators, stated that "the alcohol in two or three cocktails would have the physiologic action of four or five drinks at altitudes of approximately 10,000 to 12,000 feet." Also, "Airmen should be informed that the effects of alcohol are similar to those of oxygen want and that the combined effects on the brain and the CNS are significant at altitudes even as low as 8,000 to 10,000 feet."[285] McFarland's original observations were made on two individuals in the Andes in 1936. McFarland and Forbes[386] found that blood alcohol levels rose more rapidly and reached higher values at altitude, but noted no interactive effect of alcohol and altitudes of 3810 and 5335 m (12,500 and 17,503 feet).[286] Most subsequent studies refuted the increased blood alcohol concentration data except at altitudes over 5450 m (17,881 feet). A series of chamber studies[173,174] found blood alcohol levels were similar at 392 and 3660 m (1286 and 12,008 feet), and there were no synergistic effects of alcohol and altitude.

Lategola and colleagues[239] found that blood alcohol uptake curves were the same at sea level and 3660 m (12,008 feet), and that performance on math tests showed no interaction between alcohol and altitude. In another study of 25 men, performance scores were similar at sea level and at a simulated altitude of 3810 m (12,500 feet), with blood alcohol level of 88 mg%.[79] Performance was not affected by hypoxia, only by alcohol, and older persons were more affected. When more demanding tasks were tested, a blood alcohol level of 91 mg% affected performance, as did an altitude of 3660 m (12,008 feet) during night sessions when the participants were sleep-deprived, but there was no significant altitude-alcohol interaction.[78] In the one study in which investigators were able to discern some altitude effect, there was a simple additive interaction of altitude (hypoxic gas breathing) and alcohol.[80] The authors concluded that performance decrements caused by alcohol may be increased by altitudes of 3660 m if subjects are negatively affected by that altitude without alcohol. All these aviation-oriented studies used acute hypoxia equivalent to no more than 3500 m (11,483 feet). Perhaps the highest altitude (without supplemental oxygen) at which alcohol was studied was 4350 m (14,272 feet), on the summit of Mt Evans in Colorado. Alcohol affected auditory evoked potentials the same as in Denver; that is, no influence of altitude was detectable.[127]

In summary, limited data on ABGs at altitude after moderate alcohol ingestion support the popular notion that alcohol could slow ventilatory acclimatization and therefore might contribute to AMS. Considerable data at least up to 3660 m (12,008 feet), however, refute the belief that altitude potentiates the effect of alcohol. How altitude and alcohol might interact during various stages of acclimatization in individuals at higher altitudes is unknown.

## THROMBOSIS: COAGULATION AND PLATELET CHANGES

Autopsy findings in deaths caused by altitude illness of widespread thrombi in the brain and lungs, as well as the impression that thrombosis is greater at altitude,[202] have led to many investigations of the clotting mechanism at high altitude (for a review, see Grover and Bärtsch[145]). Although changes in platelets and coagulation have been observed in rabbits, mice, rats, calves, and humans on ascent to high altitude,[172] these are generally in vitro and with very rapid ascent. In vivo studies using more realistic ascent profiles up to 4500 m (14,764 feet) in the mountains, and higher in chambers, have generally not found changes in coagulation and fibrinolysis.[145] Also, a recent field study using thromboelastography in healthy volunteers ascending to Everest Base Camp showed that coagulation may actually be slowed at high altitude.[276] Although the increased incidence of thrombosis in soldiers and others at extreme altitude can be attributed to dehydration, polycythemia, and forced inactivity,[56,454] some evidence indicates enhanced fibrin formation with stay of a few weeks above 5000 m (16,404 feet).[370] In addition, unexplained thrombosis is reported at moderate altitude of 4000 to 5000 m[72,118] and at extreme altitude.[10,218]

As for thrombosis in HAPE, Singh and colleagues[408] reported increased fibrinogen levels and prolonged clot lysis times during HAPE, attributed to a breakdown of fibrinolysis. These authors also reported thrombotic, occlusive hypertensive pulmonary vascular disease in soldiers who had recently arrived at extreme altitude.[407] A series of experiments by Bärtsch and colleagues,[22] however, carefully examined this issue in well individuals and in those with AMS and HAPE.[145] They concluded that HAPE is not preceded by a prothrombotic state and that only in "advanced HAPE" is there fibrin generation, which abates rapidly with oxygen treatment. They considered the coagulation and platelet activation as an epiphenomenon rather than as an inciting pathophysiologic factor, and likely caused by inflammation from structural damage to the capillaries or the extreme hypoxemia.

A difficult clinical question is whether ascent to altitude might result in thrombosis in persons with familial thrombophilia, such as factor V Leiden, a common anomaly, or protein C deficiency, antiphospholipid syndrome, or others. Such cases have been reported,[10,33,52,205,218,313] although cause and effect cannot be established with certainty. Schreijer and colleagues[398] demonstrated increased thrombin-antithrombin complex formation during 8 hours of hypobaric hypoxia on an airplane in patients with factor V Leiden mutation, but neither this nor other studies have shown that this is associated with increased risk of thromboembolism at

high altitude. See Chapter 3 for more detailed discussion regarding these patients and those with previous venous thromboembolism problems.

## PERIPHERAL EDEMA

Edema of the face, hands, and ankles at high altitude is common, especially in females. Incidence of edema in at least one area of the body in trekkers at 4200 m (13,780 feet) was 18% overall, 28% in females, 14% in males, 7% in asymptomatic trekkers, and 27% in those with AMS.[153] Although not a serious clinical problem, edema can be bothersome. When seen in conjunction with dyspnea or neurologic symptoms and signs, presence of peripheral edema should prompt evaluation for pulmonary and cerebral edema. In the absence of AMS, peripheral edema is effectively treated with a diuretic. Treatment of accompanying AMS by descent or medical therapy results in diuresis and resolution of peripheral edema. The mechanism is presumably similar to fluid retention in AMS but may also merely be caused by exercise.[291]

## IMMUNOSUPPRESSION

Mountaineers have observed that infections are common at high altitude, slow to resolve, and often resistant to antibiotics.[310] On the AMREE in 1981, serious skin and soft tissue infections developed. "Nearly every accidental wound, no matter how small, suppurated for a period of time and subsequently healed slowly."[382] A suppurative hand wound and septic olecranon bursitis did not respond to antibiotics but did respond to descent to 4300 m (14,108 feet) from the 5300 m (17,388 feet) base camp. Nine of 21 persons had significant infections not related to the respiratory tract. Most high-altitude expeditions report similar problems.

Data from OEII indicated that healthy individuals are more susceptible to infections at high altitude because of impaired T lymphocyte function; this is consistent with previous Russian studies in humans and animals.[288] In contrast, B cells and active immunity are not impaired. Therefore, resistance to viruses may not be impaired, whereas susceptibility to bacterial infection is increased. The degree of immunosuppression is similar to that seen with trauma, burns, emotional depression, and space flight. The mechanism may be related, at least in part, to release of adrenocorticotropic hormone, cortisone, and β-endorphins, all of which modulate the immune response.[292,115] Intense ultraviolet exposure has also been shown to impair immunity. Persons with serious infections at high altitude may need oxygen or descent for effective treatment. Impaired immunity because of high altitude should be anticipated in situations where infection could be a complication, such as trauma, burns, and surgical and invasive procedures.

## SLEEP AT HIGH ALTITUDE

Disturbed sleep is common at high altitude for a variety of reasons, which have been described in detail.[3,58,260,457,465,469] Almost all sojourners complain of disturbed sleep at high altitude, with severity increasing with the altitude. At moderate altitude, sleep architecture is changed, with a reduction in stages 3 and 4 sleep, stage 1 time increased, and little change in stage 2. Overall, there is a shift from deeper sleep to lighter sleep. In addition, more time is spent awake, with significantly increased arousals. Clinicians have reported either slightly less rapid eye movement (REM) time or no change in REM, compared with what occurs at low altitude. REM sleep may improve over time at altitude.[220] The subjective complaints of poor sleep are disproportionate to the small reduction (if any) in total sleep time and appear to result from sleep fragmentation. With more extreme hypoxia, sleep time was dramatically shortened and arousals increased, without a change in ratio of sleep stages but with a reduction in REM sleep.[11] The mechanisms of this change in sleep architecture and fragmentation are poorly understood. Periodic breathing appears to play only a minor role in altering sleep architecture at high altitude.[380] The arousals have been linked to periodic breathing in some studies but not in others. Other factors might include change in circadian rhythm and perhaps body temperature.[83] Obesity may explain susceptibility to both deranged sleep and sleep-disordered breathing in some individuals.[133] Studies of infants and children[492] and athletes in simulated-altitude devices used for training have also revealed deranged sleep quality in these groups.[221,222,327] Although a frequent complaint in high-altitude visitors, deranged sleep seems to have little relation to susceptibility to altitude illness or other serious problems. Symptomatic treatment that avoids respiratory depression is safe (see Treatment under Acute Mountain Sickness, earlier).

### Periodic Breathing

Periodic breathing is most common in early and light sleep, may occur during wakefulness when drowsy, and does not occur in REM sleep. The pattern is characterized by hyperpnea followed by apnea (Video 2-1). Unlike in obstructive sleep apnea, where respiratory efforts are made but airflow does not occur because of upper airway obstruction, the apnea of periodic breathing is central in origin, not associated with snoring, and occurs with absence of rib cage movement.

The phenomenon is caused by abnormal feedback control in CNS respiratory centers.[73,219] Hypoxemia leads to increased peripheral chemoreceptor output and a subsequent increase in minute ventilation. If the ventilatory response is large enough, $Pa_{CO_2}$ falls below the apnea threshold in the central chemoreceptors, and apnea ensues. The decrease in $Sa_{O_2}$ and increase in $Pa_{CO_2}$ that occur during the apneic period eventually trigger an increase in ventilation and termination of apnea. If this corrective response is excessive, the $Pa_{CO_2}$ again falls below the apnea threshold in the central chemoreceptor, leading to another episode of apnea. A key feature of this process is that the response to the disturbance, in this case the increase in ventilation with hypoxemia and increasing $Pa_{CO_2}$, is excessive. This fits with the observation that persons with high hypoxic ventilatory response have more periodic breathing,[48] with mild oscillations in $Sa_{O_2}$,[236] whereas persons with low hypoxic ventilatory response have more regular breathing overall but may have periods of apnea with extreme hypoxemia distinct from periodic breathing.[160] As acclimatization progresses, periodic breathing lessens but does not disappear, especially above 5000 m (16,404 feet), and sleep $Sa_{O_2}$ increases.[11,48,430]

Periodic breathing has not been implicated in the etiology of high-altitude illness, but nocturnal oxygen desaturation has been implicated.[48,129,114] Eichenberger and colleagues[110] have also reported greater periodic breathing in persons with HAPE, secondary to lower $Sa_{O_2}$. As with fragmented sleep, intensity of periodic breathing is quite variable. Total sleep time with periodic breathing has been shown to vary between 1% and 90%[501] and to increase with increasing altitude.[48] Most studies report no association between periodic breathing and AMS.[48] This may relate to the fact that persons with periodic breathing tend to have higher HVR and greater average ventilation and oxygenation.[465]

### Pharmaceutical Aids for Sleep

Acetazolamide (125 mg at bedtime) diminishes periodic breathing and awakenings, improves oxygenation and sleep quality, and is a safe agent to use as a sleeping aid with the added benefit of diminishing symptoms of AMS (Figure 2-11). Other agents include diphenhydramine (Benadryl), 50 to 75 mg, or the short-acting benzodiazepines such as triazolam (Halcion), 0.125 to 0.25 mg and temazepam (Restoril), 15 mg.[260] Although caution is warranted for any agent that might reduce ventilation at high altitude, some studies have suggested that benzodiazepines in low doses are generally safe in this situation.[102,139,318] Another option is to use both acetazolamide and a benzodiazepine. Acetazolamide (500 mg slow release PO) given with temazepam (10 mg PO) improved sleep and maintained $Sa_{O_2}$, counteracting a 20% decrease in $Sa_{O_2}$ when temazepam was given alone.[318] The nonbenzodiazepine hypnotic zolpidem (Ambien), 10 mg, was shown to improve sleep at 4000 m (13,123 feet) without adversely affecting ventilation.[40]

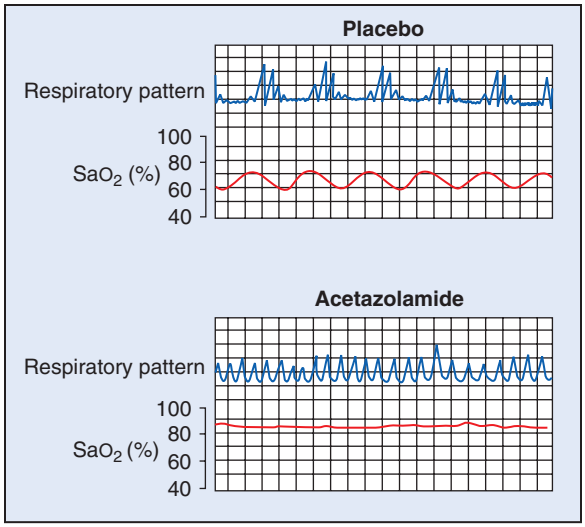


**FIGURE 2-11** Respiratory patterns and arterial oxygen saturation ($SaO_2$) with placebo and acetazolamide in two sleep studies of a person at 4200 m (13,780 feet). Note pattern of hyperpnea followed by apnea during placebo treatment, which is changed with acetazolamide.

## HIGH-ALTITUDE PHARYNGITIS, BRONCHITIS AND COUGH

Sore throat, chronic cough, and bronchitis are almost universal in persons who spend more than 2 weeks at an extreme altitude (>5500 m [18,045 feet).[75,251,277,305] All 21 members of the 1981 AMREE experienced these problems.[382] Only two of eight persons in OEII (temperature >21° C [69.8° F] and relative humidity >80%) developed cough, and only above 6500 m (21,325 feet). Only four had sore throat. Acute hypoxia directly lowers the cough threshold, thus exacerbating high-altitude cough.[278] Other factors play a role. In the field, these problems usually appear without fever or chills, myalgia, lymphadenopathy, exudate, or other signs of infection. The increase in ventilation, especially with exercise, forces obligate mouth breathing at altitude, bypassing the warming and moisturizing action of the nasal mucous membranes and sinuses. Movement of large volumes of dry, cold air across the pharyngeal mucosa can cause marked dehydration, irritation, and pain, similar to pharyngitis. Vasomotor rhinitis, quite common in cold temperatures, aggravates this condition by necessitating mouth breathing during sleep. For this reason, decongestant nasal spray is one of the most coveted items in an expedition medical kit. Other countermeasures include forced hydration, hard candies, lozenges, and steam inhalation.

High-altitude bronchitis can be disabling because of severe coughing spasms. Cough fracture of one or more ribs is not rare.[251] Purulent sputum is common. Response to antibiotics is poor; most patients resign themselves to taking medications such as codeine and do not expect a cure until descent.[343] Mean sputum production was 6 teaspoons per day. All reported that onset was after a period of excessive hyperventilation associated with strenuous activity. Although an infectious etiology is possible, experimental evidence suggests that respiratory heat loss results in purulent sputum and sufficient airway irritation to cause persistent cough.[284] This is supported by the beneficial effect of steam inhalation and lack of response to antibiotics. Many climbers find that a thin balaclava, porous enough for breathing, traps some moisture and heat and effectively prevents or ameliorates the problem. Many climbers with high-altitude cough use inhaled fluticasone and salmeterol or similar inhaled agents and report success; studies of these medications are underway. Differential diagnosis of persistent cough in these conditions can be difficult.[138]

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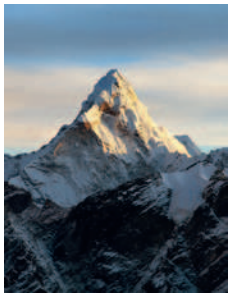
***Complete references used in this text are available online at expertconsult.inkling.com.***





CHAPTER 3

# High Altitude and Preexisting Medical Conditions

ANDREW M. LUKS AND PETER H. HACKETT

Given the prevalence of diseases such as asthma, hypertension, and diabetes mellitus in the general population, it is likely that many high-altitude travelers have one or several underlying medical conditions. Although the majority of these individuals have mild, well-controlled conditions that should not create issues during planned travel, ongoing improvements in medical care and the growing ease of travel have increased the likelihood that some high-altitude travelers have more moderate to severe forms of underlying disease that may pose challenges in this environment. Regardless of the severity of their underlying problems, patients may present to their primary care provider or a travel medicine clinic prior to travel seeking advice about the safety of their planned trip and how best to manage their disease during the sojourn. When faced with these situations, providers must ask the following two questions:

1. Will the underlying disease worsen at high altitude?
2. Will the underlying disease affect acclimatization to hypobaric hypoxia and predispose the individual to an increased risk of acute altitude illness?

In addition, clinicians working in high-altitude locations are often faced with consequences of the interaction between altitude hypoxia and common preexisting medical conditions of travelers. This chapter provides information that can be used to address these questions. After presenting a general framework for evaluating the high-altitude traveler with underlying medical conditions, we describe the available evidence regarding a wide variety of diseases likely to be encountered in such clinical situations. Additional information is available in extensive reviews on this topic,[67,93] and further disease-specific studies are cited in the appropriate sections below.

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# A GENERAL FRAMEWORK FOR EVALUATING TRAVELERS WITH UNDERLYING MEDICAL CONDITIONS

One of the challenges of evaluating individuals with underlying medical conditions who are planning high-altitude travel is that for many diseases, such as chronic obstructive pulmonary disease (COPD) or hypertension, research on how these patients fare at high altitude is limited, whereas for other diseases, there is practically no evidence to guide the pretravel assessment. In light of this situation, providers can apply the following general approach to determining whether the planned sojourn is safe, whether further evaluation is necessary before the trip, and whether risk mitigation strategies are indicated.

The initial assessment should be framed around four general questions, as follows:

1. *Is my patient at risk for severe hypoxemia or impaired tissue oxygen delivery at high altitude?* Certain categories of patients, such as those with chronic lung diseases, heart failure, cyanotic congenital heart disease, or sleep-disordered breathing, can be expected to develop increased hypoxemia beyond that experienced by normal individuals at a given altitude. This exaggerated hypoxemia will have the same effect as being at a higher altitude. Further information about the extent to which this will happen is provided in the discussion of each specific disease. More severe hypoxemia is a concern because it may lead to increased dyspnea and decreased exertional tolerance and, in some studies, has been associated with increased risk of developing acute altitude illness. In addition, it may impact comorbid conditions. Patients with severe anemia will not experience a change in arterial oxygen partial pressure ($Pa_{O_2}$) at rest compared with normal individuals, but anemia patients will have problems with decreased oxygen-carrying capacity, which can lead to worsening dyspnea and poor exertional tolerance.
2. *Can my patient mount the expected ventilatory responses to hypobaric hypoxia at high altitude?* Arterial hypoxemia at high altitude stimulates the peripheral chemoreceptors, leading to an increase in minute ventilation, the primary role of which is to defend alveolar oxygen tension ($P_{A_{O_2}}$) against the effects of decreased ambient $P_{O_2}$. Although the magnitude of this response varies between individuals, most are able to mount this response. Patients with disorders affecting respiratory mechanics, such as very severe COPD, morbid obesity, or various neuromuscular disorders, and individuals at risk for impaired chemoreceptor responses may not be able to mount the expected ventilatory response and thus may be at risk for increased levels of hypoxemia.
3. *Is my patient at risk because of the expected pulmonary vascular responses to hypobaric hypoxia at high altitude?* Decreased $P_{A_{O_2}}$ triggers hypoxic pulmonary vasoconstriction, which in conjunction with the increase in cardiac output following ascent, causes increased pulmonary artery pressure (PAP). The magnitude of this response varies between individuals and is well tolerated by most travelers but could represent a problem for patients with underlying pulmonary hypertension. This is because the further increase in PAP may worsen right-sided heart function, leading to increased dyspnea, impaired hemodynamics, and as described later, development of high-altitude pulmonary edema (HAPE).
4. *Will hypobaric hypoxia at high altitude worsen control of the underlying medical condition(s)?* Although data are lacking with regard to many chronic diseases, more information has become available in recent years regarding the effects of hypobaric hypoxia on common medical problems such as asthma, hypertension, and obstructive sleep apnea, which are reviewed in detail later. Patients should be assessed in light of the available information to evaluate the likelihood of worsening disease control after ascent (see Chapter 2).

If reassuring answers are obtained with regard to all these questions, the individual is likely safe to travel to high altitude without further evaluation. Nonreassuring answers to these questions should prompt evaluation to clarify the risk further and to assess and develop risk mitigation strategies for the sojourn, including evacuation to lower altitude. Depending on the underlying disease process, tests that may be considered as part of such an evaluation would include pulmonary function testing, hypoxia altitude simulation testing,[44] echocardiography, and cardiopulmonary exercise testing. Canceling the planned travel is a consideration if risk is deemed to be too high or if adequate risk mitigation strategies are not feasible. Details of this evaluation are provided in the discussions of specific diseases and lead to another question:

5. *At what altitude does risk increase for patients with underlying medical problems?* In general, important physiologic responses to hypobaric hypoxia, such as the ventilatory response to hypoxia and hypoxic pulmonary vasoconstriction, occur at about 2000 m (6560 feet) when $Pa_{O_2}$ falls below 70 mm Hg,[9] whereas the risk of altitude illness is thought to increase when individuals ascend above 2500 m (8200 feet). These are useful thresholds for trip planning, but one should not assume that all patients with underlying medical conditions are safe below these altitudes. Patients with unilateral absence of pulmonary arteries have developed HAPE at as low as 1500 m (4920 feet),[130] which shows that certain patients may fare poorly at elevations lower than these thresholds. In other cases, the underlying medical condition may not be an issue until ascent to elevations far above these levels. In the end, the altitude at which risk for problems increases will be a function of the particular disease and its severity.

## MITIGATING RISK WITH PLANNED ASCENT

Although the appropriate risk mitigation strategy will vary among travelers based on the underlying medical condition, all travelers with underlying medical problems should adhere to several general principles. First, the traveler must ensure that the underlying medical problem is under good control at the time of the planned trip. Patients with worsening control of heart failure or asthma, for example, should not travel to high altitude, particularly into remote areas. Second, the traveler should continue regular medications and therapies during the trip and, in some cases, such as diabetes or poorly controlled hypertension, should consider more frequent monitoring of the disease, with adjustment of medications according to a prespecified plan. Third, patients at risk for disease exacerbations, such as those with asthma, COPD, or cardiac arrhythmia, should travel with an adequate supply of rescue medications and a plan for using these therapies. Lastly, travelers should identify medical resources at their planned destination and travel with a plan to access these resources or descend in the event of severe problems. Arranging suitable travel insurance, particularly with international travel, will greatly facilitate any necessary evacuation. Further details about risk mitigation in specific conditions are provided in the following discussions.

# SPECIFIC MEDICAL CONDITIONS AT HIGH ALTITUDE

Having considered this general framework, we now discuss a variety of medical conditions that might pose problems for individuals traveling to high altitude. Table 3-1 lists specific patient groups and potential conditions that warrant consideration as well as the relative safety or risk associated with high altitude.

## RESPIRATORY DISEASES

### Chronic Obstructive Pulmonary Disease

Minimal data are available regarding outcomes for patients with COPD who travel to high altitude, and most of the available information pertains to changes in oxygenation rather than changes in airway function.[95] Aside from a single study of COPD patients at terrestrial high altitude,[65] most information about the effects of hypobaric hypoxia on these patients comes from the

**TABLE 3-1** High-Altitude Travel Risk Associated with Various Underlying Medical Conditions

| Likely No Extra Risk | Caution Required | Ascent Contraindicated |
|---|---|---|
| Children and adolescents | Infants <6 weeks old | Sickle cell anemia |
| Elderly individuals | Compensated heart failure | Severe to very severe COPD |
| Sedentary individuals | Morbid obesity | Pulmonary hypertension (systolic PAP >60 mm Hg) |
| Mild obesity | Cystic fibrosis ($FEV_1$ 30-50% predicted) | Unstable angina |
| Well-controlled asthma | Poorly controlled arrhythmia | Decompensated heart failure |
| Diabetes mellitus | Poorly controlled asthma | High-risk pregnancy |
| CAD following revascularization | Poorly controlled hypertension | Cystic fibrosis ($FEV_1$ <30% predicted) |
| Mild COPD | Moderate COPD | Recent myocardial infarction or stroke (<90 days) |
| Low-risk pregnancy | Stable angina | Untreated cerebrovascular aneurysms or arteriovenous malformations |
| Mild-moderate obstructive sleep apnea | Non-revascularized CAD | Cerebral space-occupying lesions |
| Controlled hypertension | Sickle cell trait | |
| Controlled seizure disorder | Poorly controlled seizure disorder | |
| Psychiatric disorders | Cirrhosis | |
| Neoplastic diseases | Mild pulmonary hypertension | |
| | Radial keratotomy surgery | |
| | Severe obstructive sleep apnea | |

*CAD,* Coronary artery disease; *COPD,* chronic obstructive pulmonary disease; *$FEV_1$,* forced expiratory volume in 1 second; *PAP,* pulmonary artery pressure.

literature on commercial flight. The data suggest that patients with a forced expiratory volume in 1 second ($FEV_1$) of 1.0 to 1.5 liters (L) manifest hypoxemia when exposed to the equivalent of 2340 m (8000 feet), with average $PaO_2$ values approximately 50 mm Hg (Table 3-2). Mild degrees of exertion, such as walking on flat ground or cycling on an ergometer at a low work rate, lead to further decreases below these levels. These values are significant; guidelines from the British Thoracic Society, American Thoracic Society, and Aerospace Medical Association all suggest supplemental oxygen should be employed in patients whose $PO_2$ is expected to be in this range at elevations of approximately 2340 m (8000 feet). Recognizing that these thresholds lack supporting data and are somewhat arbitrary, it is reasonable to ask whether COPD patients need supplemental oxygen with travel to similar altitudes in the mountains. How to apply such guidelines at higher elevations is unclear, however, because even normal individuals will have $PaO_2$ of approximately 50 mm Hg with acute exposure to elevations greater than 4200 to 4500 m (13,770 to 14,760 feet) and clearly do not require supplemental oxygen in these circumstances.

Predicting which patients will develop clinically significant hypoxemia at high altitude is challenging. A variety of prediction rules are available that take into account sea level blood gases, pulmonary function test results, and other variables,[30,42,61,62] but

**TABLE 3-2** Changes in Oxygenation in Lung Disease Patients Exposed to Hypoxia

| Study | Patient Population (*N*) | Disease Severity | Exposure | $PaO_2$ at Sea Level (mm Hg) | $PaO_2$ in Hypoxia (mm Hg) | $PaO_2$ with Exertion in Hypoxia (mm Hg) |
|---|---|---|---|---|---|---|
| Graham and Houston[65] (1978) | COPD (8) | $FEV_1$ 1.27 ±0.36 L | 1920 m (6300 ft) (terrestrial) | 66 ±7 | 52 ±7 | 47 ±9 (treadmill walking) |
| Christensen et al[30] (2000) | COPD (15) | $FEV_1$ 0.98 ±0.4 L | 2430 m (7970 ft) (simulated) | 88 ±9 | 53 ±7 | 44 ±6 (cycle at 20-30 W) |
| Akero et al[4] (2005) | COPD (18) | $FEV_1$ 1.5 ±0.6 L | 1830 m (6000 ft) (commercial flight) | 77 ±9 | 63 ±6 | $SpO_2$ 87 ±4% (walking in aisle) |
| Seccombe et al[140] (2004) | COPD (10) | $FEV_1$ 1.2 ±0.4 L | 2430 m (7970 ft) (simulated) | 77 ±577 ±5 | 45 ±4 | 39 ±3 (walk 50 m on flat ground) |
| | Interstitial lung disease (15) | $FEV_1$ 1.8 ±0.6 L | 2430 m (7970 ft) (simulated) | 82 ±7 | 50 ±7 | 40 ±5 (walk 50 m on flat ground) |
| Christensen et al[31] (2002) | Restrictive disease (TB, kyphoscoliosis, fibrosis) (17) | TLC 3.2 ±1 L | 2430 m (7970 ft) (simulated) | 76 ±12 | 47 ±8 | 37 ±7 (cycle at 20 W) |
| Mestry et al[106] (2009) | Restrictive disease (polio, kyphoscoliosis, neuromuscular disease) (19) | Median $FEV_1$ 0.7 L (range, 0.3-1.0 L) | 2430 m (7970 ft) (simulated) | 75 ±10 | 49 ±4 | No data |
| Fischer et al[52] (2005) | Cystic fibrosis (36) | Median $FEV_1$ 66% predicted (range, <26-107%) | 2650 m (8690 ft) (terrestrial) | 74 (range, 60-98) | 52 (range, 40-79) | 47 (range, 33-69) (cycle at 30 W) |
| Thews et al[149] (2004) | Cystic fibrosis (10) | $FEV_1$ 2.1 ±0.3 L | 3000 m (9840 ft) (simulated) | 75 ±4 | 46 ±1 | No data |

*COPD,* Chronic obstructive pulmonary disease; *$FEV_1$,* forced expiratory volume in 1 second; *$SpO_2$,* oxygen saturation as measured by pulse oximetry; *TB,* tuberculosis: *TLC,* total lung capacity; *W,* watts.

the rules are designed to predict the degree of hypoxemia during short exposures to 2340 m (8000 feet), the maximum allowed altitude in cabin aircraft. These rules have not been validated for the wide range of elevations that patients may visit or the duration of exposure they are likely to experience during their travels. An alternative approach is the *hypoxia altitude simulation test,* in which oxygenation and other parameters are monitored while a patient breathes a hypoxic gas mixture at rest.[44] This test should more accurately reflect the degree of expected hypoxemia than do the prediction rules, but it is limited by its short duration relative to the duration of exposure to high altitude, the inability to account for changes in ventilation that will occur over long stays at high altitude, and difficulties administering hypoxic gas mixtures simulating the altitude the patient will visit.

An important question that arises from the available literature is whether supplemental oxygen is necessary to counteract the expected hypoxemia, as the guidelines recommend. It is noteworthy that in the majority of studies previously cited, most patients had mild symptoms and experienced few, if any, adverse events despite having hypoxemia disproportionate to that expected for normal individuals at the same elevation. Given this issue, as well as the logistical difficulties of traveling with supplemental oxygen, a more practical alternative would be to travel with plans to monitor symptoms and oxygen saturation ($Spo_2$) on arrival using a portable pulse oximeter and a prescription for supplemental oxygen to use if difficulties arise.[91] In North America, for example, home oxygen companies accept prescriptions from physicians in any state, and oxygen is easily obtained. Patients already receiving supplemental oxygen should continue therapy during their travels but need to increase the gas flow to account for the drop in barometric pressure. A useful rule of thumb is to increase the inspiratory flow rate by the ratio of higher to lower barometric pressure. Portable oxygen concentrators provide a convenient alternative to oxygen gas cylinders, provided patients can recharge the batteries. However, these devices may not deliver the same inspired oxygen concentration at altitude as at sea level.

Except for the hypoxemia issue, no information in the literature addresses the risk of acute altitude illness in COPD patients. Use of acetazolamide for altitude illness prophylaxis in COPD patients has not been studied. Caution is necessary when considering acetazolamide in patients with $FEV_1$ of less than 25% predicted, because it may lead to impaired carbon dioxide elimination and worsening hypercarbia.[95,97] In patients whose COPD is complicated by pulmonary hypertension, further increases in PAP after ascent could theoretically increase the risk of HAPE or right-sided heart dysfunction. As discussed later, this issue has not been formally studied in this patient population. No evidence suggests that patients with bullous emphysema are at increased risk for pneumothorax or other forms of pulmonary barotrauma after ascent, or that ascent to high altitude leads to deterioration in markers of pulmonary function.[95] Patients should continue taking their baseline medications at high altitude and travel with an adequate supply of rescue inhalers and prednisone in the event of an exacerbation during their trip, particularly if visiting an area remote from medical care.

### Interstitial Lung Disease

Compared with COPD patients, fewer studies are available regarding the risks of hypobaric hypoxia in patients with interstitial lung disease (ILD), such as idiopathic pulmonary fibrosis or sarcoidosis. Limited evidence from the literature on hypoxemia during commercial flight demonstrates that patients with a variety of ILDs and $FEV_1$ of about 50% to 55% predicted develop significant hypoxemia with exposure to 2430 m (8000 feet), which worsens with exertion (Table 3-2).[31,140] Prediction rules to assess the risk of hypoxemia in ILD patients are available as for COPD patients, but utility is limited by the same considerations noted earlier, and the hypoxia altitude simulation test likely remains the best tool for assessing risk. Similar principles on traveling with a prescription for supplemental oxygen and monitoring symptoms and pulse oximetry after arrival should likely be applied in ILD patients as well.

### Asthma

Doan and Luks[45] recently reviewed the issue of asthma management in the wilderness, particularly at high altitude. From a theoretical standpoint, multiple factors at high altitude could affect airway function in asthmatic patients. On the one hand, lower air density and decreased number of dust mites[155] would be expected to lead to better control. On the other hand, many asthmatic patients experience worsening control when exercising, particularly in the cold, dry air typical of the high-altitude environment. A high incidence of asthma and asthma-like symptoms has been documented in epidemiologic studies of cross-country skiers; this activity is associated with high minute ventilation in a cold environment.[81] Other studies have documented a 50% incidence of exercise-induced bronchoconstriction (EIB) in highly trained ski mountaineers in the Alps, even though almost three-quarters of these individuals had never been diagnosed with EIB.[47]

Current evidence suggests that despite these concerns, the majority of well-controlled asthmatic patients do well with exposures to altitudes as high as 6000 m (19,685 feet). Several studies have documented decreased bronchial hyperresponsiveness to hypoosmolar aerosols and methacholine after travel to altitudes of 4559 and 5050 m (14,950 and 16,560 feet)[5,32] and no increase in symptoms, need for medications, exacerbations, or risk of acute mountain sickness (AMS) with ascent to elevations of 5895 to 6400 m (19,335 to 20,990 feet).[74,147] Other studies have noted small decreases in $FEV_1$ or peak expiratory flow at high altitude, but these were not associated with any changes in patient symptoms and likely were not clinically significant.[89,105,] In a survey study of asthmatic patients seen in a travel clinic, almost 75% of whom traveled to high altitude, 43% reported worsening asthma control during their trip, and 37% experienced the "worst asthma attack of my life."[60] This study, however, did not account for the altitudes attained by these patients or the fact that many high-altitude trips require transit through large cities such as Kathmandu or Bangkok or environments with very poor air quality. Aside from concerns about asthma control, the available literature has not reported an increased incidence of acute altitude illness in asthmatic patients.[74,147] Acetazolamide use also appears to be safe in this patient population.[107]

In summary, patients with mild, well-controlled asthma can travel to altitudes over 6000 m (19,685 feet). Individuals with worsening control at the time of their planned trip or with persistent, moderate to severe disease should avoid high-altitude travel, particularly travel into remote areas with inadequate access to medical care. All patients should continue their baseline medication regimen and carry an adequate supply of rescue inhalers and prednisone in the event control worsens before they can access care. Patients with known EIB or cold-induced bronchoconstriction can consider adding or increasing the intensity of their controller therapy or taking preexercise short-acting β-agonists or leukotriene receptor blockers. Further details on asthma control in the wilderness are provided in Chapters 53 and 94, and several reviews on this topic are available.[33,45]

### Cystic Fibrosis

Although data on the effect of high altitude on pulmonary function in cystic fibrosis (CF) patients are inconsistent,[52,133,149,] CF patients experience more severe hypoxemia than normal individuals when exposed to altitudes of 2000 to 3000 m (6560 to 9840 feet), with the largest decrements seen in patients with greater degrees of impaired pulmonary function[52,133] (Table 3-2). As with the studies in COPD patients previously noted, these exaggerated levels of hypoxemia were not associated with increased symptoms or an increased risk of AMS,[52,133] although the duration of exposure was short. There are reports of patients with severe CF ($FEV_1$ ~1 L) developing pulmonary hypertension and cor pulmonale during ski trips to this environment.[145] Prediction rules are available to assess the likelihood of severe hypoxemia at high altitude but are poor predictors of $Pao_2$ at altitude in CF patients and are limited by the same issues noted with COPD.[52,77,118,] Given significant improvements in the quality of CF care in the past decade, it is also unclear whether older data and prediction rules still apply to the current population of CF

patients. There is no evidence that exposure to high altitude leads to an increased incidence of CF exacerbations.

As with asthmatic patients, CF patients should only travel to high altitude when their disease is under good control. High-altitude travel should be avoided in patients with worsening symptom control, recent exacerbations, or $FEV_1$ of less than 30% predicted. Given difficulties with the prediction rules for hypoxemia, patients with $FEV_1$ of 30% to 50% predicted should travel to high altitude with a prescription for supplemental oxygen that can be filled in the event symptom and pulse oximetry monitoring raise concerns. All patients should continue their baseline antibiotic and mucolytic regimens and scheduled airway clearance strategies.

### Pulmonary Hypertension

The decrease in $P_{AO_2}$ after ascent leads to hypoxic pulmonary vasoconstriction, which in conjunction with the rise in cardiac output leads to an increased PAP. Most individuals tolerate this rise in pressure without difficulty, but patients with underlying pulmonary hypertension or right-sided heart dysfunction may be at risk for capillary stress failure (pulmonary edema) or worsening right-sided heart function. Multiple case reports and case series, for example, have documented development of HAPE after ascent in patients with pulmonary hypertension as a result of both anatomic and nonanatomic factors.[48,68,112,130,153] Other reports have documented right-sided heart failure in patients with morbid obesity and severe kyphoscoliosis, two problems associated with pulmonary hypertension, during commercial flight.[115,151] The degree of baseline pulmonary hypertension necessary to increase risk is not clear because these patients had a broad PAP range. How high an individual needs to ascend to increase risk is also unclear, although in several reports, HAPE developed with ascent to altitudes between 1500 and 2000 m (4920 and 6560 feet), lower than the altitudes typically associated with HAPE.[48,130]

Despite the lack of firm data to guide clinicians, a reasonable approach is that persons with mean PAP greater than 35 mm Hg or systolic PAP greater than 60 mm Hg at baseline should probably avoid travel to sleeping altitudes above 2500 m (8200 feet). However, if such travel is undertaken, supplemental oxygen should be recommended. Patients with milder degrees of pulmonary hypertension may travel to altitudes below 3000 m (9840 feet) but should consider prophylactic measures, including pulmonary vasodilators or supplemental oxygen, if not already receiving such therapy.[90] Instituting these measures is relatively straightforward when it is known beforehand that the patient has pulmonary hypertension. The challenge arises in patients with conditions known to predispose to pulmonary hypertension, such as bronchopulmonary dysplasia, cirrhosis, collagen vascular diseases (e.g., scleroderma), congenital heart disease, and recurrent venous thromboembolism. These patients may not be recognized as having pulmonary hypertension at the time of their planned trip. Strong consideration should be given to screening such patients for pulmonary hypertension with echocardiography or undertaking a hypoxia altitude simulation test with concurrent echocardiography to measure the PAP response to hypoxia. Patients found to have pulmonary hypertension at rest in normoxia or larger-than-expected PAP responses to hypoxia[66,40] should be managed according to the measures previously outlined.

### Sleep-Disordered Breathing

Two issues warrant consideration for patients traveling to high altitude with sleep-disordered breathing: (1) the effect of high altitude on their apnea-hypopnea index (AHI) and (2) the effect of recurrent apnea on nocturnal oxygen saturation ($S_{O_2}$). With AHI, an earlier chamber study using a normobaric hypoxia model demonstrated a decrease in the number of obstructive apnea episodes from 25.5 per hour at 60 m (200 feet) to 0.5 per hour at 2750 m (9020 feet) that was offset by a significant increase in the number of central events (0.4/hr at 60 m [200 feet] vs. 78.8/hr at 2750 m [9020 feet]).[29] A subsequent field study by Nussbaumer-Ochsner and colleagues[117] confirmed the marked increase in central apnea at 2590 m (8495 feet) in patients with moderate to severe obstructive sleep apnea (OSA) but showed

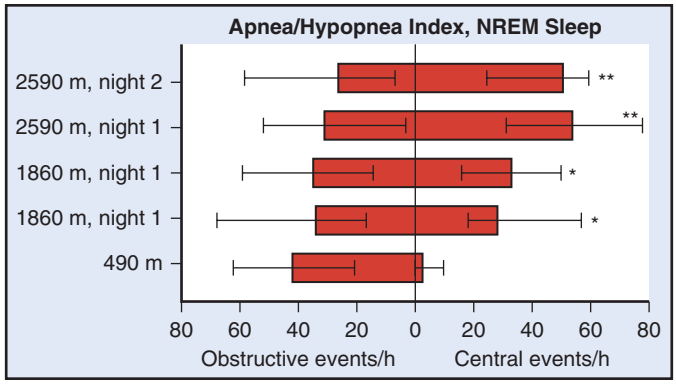


**FIGURE 3-1** Changes in the number of obstructive and central apnea and hypopnea events during non–rapid eye movement (NREM) sleep at increasing elevations. The width of each bar represents the median values; the *horizontal lines* denote the quartile ranges. With increasing altitude, the number of obstructive apnea/hypopnea events remains unchanged, but there is a significant increase in the number of central events. *(From Nussbaumer-Ochsner Y, Schuepfer N, Ulrich S, Bloch KE: Exacerbation of sleep apnoea by frequent central events in patients with the obstructive sleep apnoea syndrome at altitude: A randomised trial,* Thorax *65:429-435, 2010.)*

no significant change in the number of obstructive events after ascent (Figure 3-1). These results support a smaller study from Colorado that demonstrated a decrease in the number of central apnea episodes but persistence of obstructive events when patients with untreated OSA traveled to lower elevations.[123]

Apnea events at sea level are frequently associated with fluctuations in nocturnal $S_{O_2}$. Average nocturnal $S_{O_2}$ worsens following ascent to high altitude. Nussbaumer-Ochsner and colleagues,[117] for example, showed a statistically significant decrease in $S_{O_2}$ from 94% at 490 m (1607 feet) to 90% at 1860 m (6100 feet) and 86% on the first night at 2590 m (8495 feet). Burgess and associates[29] reported a mean $S_{O_2}$ of 85% ±4% at 2750 m (9020 feet) compared to 94% ±1% at 60 m (200 feet). Increases in AHI and worsening hypoxemia may have implications for patients; the field study also demonstrated impaired tracking performance during simulated driving at 2590 m compared to 490 m.[117] In addition, the authors found increased systolic blood pressure, likely related to the increased number of nocturnal arousals, and increased cardiac arrhythmias related to increased sympathetic stimulation from greater hypoxemia.

Because obstructive events may persist following ascent, patients with OSA traveling to high altitude should strongly consider traveling with their continuous positive airway pressure (CPAP) machines when access to electrical power can be ensured. Strong consideration should also be given to adding acetazolamide to CPAP therapy; this combination has been shown to decrease the number of central apnea events and AHI and to improve nocturnal oxygenation compared with autotitrating CPAP alone.[82] Even if logistical issues prevent continuation of CPAP after ascent, acetazolamide is still effective at improving oxygenation, AHI, and overall sleep quality and should be strongly considered in patients with OSA.[116]

## CARDIOVASCULAR CONDITIONS

### Hypertension

Studies have generated mixed results regarding the effects of hypobaric hypoxia on blood pressure (BP) in individuals with known hypertension. Some studies demonstrate BP increases on exposure to various altitudes,[121,132,135,159] and others show small, non–statistically significant changes.[41,143] The majority of studies have examined patients with mild to moderate disease at modest elevations (<3500 m [11,483 feet]) and thus may not inform evaluation of the full range of hypertensive patients who may travel to high altitude. On average, the increases in systolic BP are modest (<15 mm Hg) and appear to be exaggerated by exercise.[41,135] There is significant interindividual variability at rest, with

some persons experiencing marked rises in BP compared to others,[132] but a priori identification of persons at risk for such responses remains challenging. Several studies suggest that the initial BP rise is followed by a decline over days to weeks.[132,159] A more recent study in normotensive individuals, however, suggested that BP may remain elevated for several weeks as individuals steadily proceed to higher elevations.[122]

Patients receiving antihypertensive therapy should continue their medications while traveling at high altitude. Because BP increases are generally mild and do not appear to be associated with significant side effects, the majority of individuals should not monitor their BP after ascent. Monitoring should be reserved for those with very labile or difficult-to-control hypertension. These patients should adjust their medications according to a prearranged plan for systolic BP above 180 mm Hg or diastolic BP above 120 mm Hg and symptoms such as chest pain, vision changes, or headache, or for systolic BP above 220 mm Hg or diastolic BP above 140 mm Hg in the absence of symptoms.[92] Travelers who adjust their medications following ascent should return to their normal regimen on descent, because BP is expected to fall as the altitude-induced sympathetic stimulation wanes.

The appropriate medication for managing severe hypertension remains unclear. Since the BP rise is likely related to increased α-adrenergic activity, α-blockers would appear to be a good choice, but no data support this practice. Data are also lacking regarding calcium channel blockers, the use of which could provide the further benefit of prophylaxis against HAPE. A recent field study suggested that angiotensin receptor blockade lowered BP at 3400 m (11,150 feet), but this study was performed in normotensive individuals. Whether the same response would be seen in a hypertensive patient at high altitude, particularly one with difficult-to-control disease, is unclear.[122]

## Coronary Artery Disease

The primary concern in patients with underlying coronary artery disease (CAD) is that myocardial oxygen demand may outstrip oxygen supply in the setting of hypobaric hypoxia, particularly with exertion, thereby leading to myocardial ischemia. This problem may be exacerbated because coronary vasodilation is impaired in these patients, and paradoxical vasoconstriction may even occur during exercise.[64] Despite these theoretical concerns, patients with stable CAD appear to tolerate exposure to moderate altitudes without significant complications. Levine and associates,[85] for example, demonstrated a decrease in the ischemia threshold, denoted by the double product and treadmill workload necessary to produce 1 mm of ST-segment depression on the electrocardiogram (ECG), on acute exposure to high altitude, which resolved after several days at the same elevation. Despite these changes in the ischemia threshold, no new wall motion abnormalities were detected by echocardiography, and individuals did not manifest evidence of high-grade ectopy or arrhythmia. Similarly, Erdmann and colleagues[49] performed cardiopulmonary exercise test (CPET) at 1000 m (3280 feet) and 2 days after ascent to 2500 m (8200 feet) on 23 patients with myocardial infarction or revascularization within the preceding 4 months and with low ejection fraction (<45%), and noted the same double product and systolic BP at peak exercise between testing sites, with no evidence of ischemia or arrhythmia. DeVries and associates[38] performed CPET at 4200 m (13,776 feet) on eight patients with single-vessel disease and preserved ejection fraction; they found no changes in wall motion score indices of left ventricular function and no evidence of arrhythmia or other adverse events.

These data suggest that patients with stable, well-controlled CAD tolerate exposure to and exertion at altitudes as high as 4000 m (13,120 feet). Given the changes in the ischemia threshold noted by Levine and associates,[85] it may be prudent to decrease the level of physical activity in the first few days at altitude, although the study by Erdmann and colleagues[49] suggests heavy exertion may be possible as soon as 2 days after arrival at high elevation. Patients should not engage in de novo exercise at high altitude if they are not already engaged in an exercise program at lower elevation. Sleeping at moderate altitude before any exercise at high altitude may also reduce the risk of complications, including the risk of sudden cardiac death

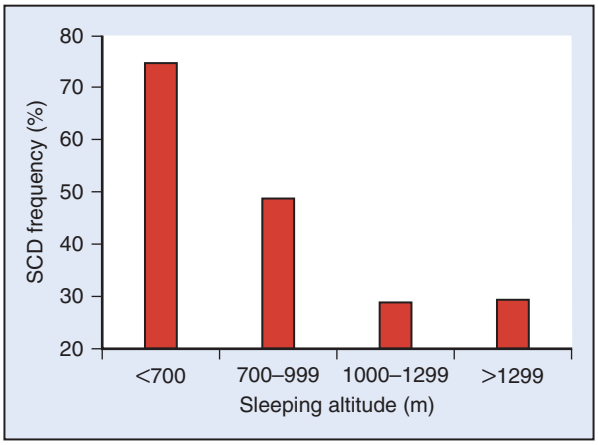


**FIGURE 3-2** Frequency of sudden cardiac death (SCD) on the first day of physical activity at moderate altitudes (2000 to 3000 m [6560 to 9840 feet]) stratified according to the sleeping altitude. The higher the individual sleeps on the first night at altitude, the lower the frequency of SCD. *(From Lo MY, Daniels JD, Levine BD, Burtscher M: Sleeping altitude and sudden cardiac death,* Am Heart J *166:71-75, 2013.)*

(Figure 3-2).[87] Patients with unstable angina, ischemia at low workloads (<80 W or 5 METs), or recent myocardial infarction should delay high-altitude travel until they have undergone revascularization. Patients with prior ventricular tachycardia or fibrillation should also avoid travel unless stable on a recent exercise test. Travel should be delayed for 4 months or longer after revascularization procedures, because studies have examined and demonstrated the safety of exertion in hypobaric hypoxia only at 4 to 18 months after such interventions. Symptom-limited exercise tests can be used in these patients, as well as those thought to be at risk for CAD, to gauge the safety of a planned ascent. Patients should continue their medication regimen throughout their planned trip. Further information on these issues is available in several reviews.[11,39]

## Heart Failure

Heart failure patients face several physiologic challenges at high altitude, including increased PAP that may lead to right-sided heart strain, increased sympathetic nervous system activity, and increased systemic BP. Together with the decreased ventilatory reserve and pulmonary compliance seen in some patients, these issues theoretically increase the risk of decompensated disease with high-altitude travel.[12,129] Only a limited number of studies have evaluated this risk in detail. Erdmann et al[49] examined patients with CAD and stable heart failure (ejection fraction <45%) and noted no greater decrement in exercise performance compared to controls. In contrast, Agostoni and associates[2] performed CPET at simulated altitudes up to 3000 m (9840 ft) in heart failure patients with varying degrees of functional limitation and noted larger decrements in maximum work rate compared to healthy controls, with the greatest decrease seen in those who were most limited at baseline. No tests had to be interrupted for ischemia in either study. Schmid and colleagues[137] performed CPET with echocardiography in New York Heart Association (NYHA) Class I and II patients (mean ejection fraction, 28.8% ±5.4%), and found that although peak oxygen consumption declined at high altitude by 22%, no patient developed ischemia, pulmonary edema, or echocardiographic evidence of left or right ventricular dysfunction. One patient developed self-limited ventricular tachycardia during maximal exercise.

These studies may seem reassuring, but the duration of exposure to high altitude before performance of exercise testing was short relative to the duration of time patients may spend at high altitude. This concern is supported by anecdotal evidence from physicians in resort areas who have noted a tendency toward acute decompensation in heart failure patients within 24 hours of arrival. The etiology for this is not clear but may relate to alterations in sodium and fluid balance that can develop after ascent.[148] Development of AMS may pose a similar risk to heart

failure patients, because AMS is often associated with fluid retention, which in turn can cause volume overload, worsening peripheral edema, and possibly pulmonary edema.

Patients with stable, well-compensated heart failure can travel to moderate altitudes (<3000 m [9840 feet]), but travel should be avoided in patients with NYHA Class III or IV disease, high-grade ventricular arrhythmias, recent exacerbation or evidence of worsening symptoms, or worse fluid balance before their planned sojourn. Patients should not engage in exercise at high altitude if they are not already active at sea level. They should continue their baseline medications but should travel with a prearranged plan to adjust their diuretics and antihypertensive regimens in response to adverse changes in body weight or systemic BP.

Among the β-blockers used in heart failure patients, carvedilol may be associated with blunted ventilatory responses, greater hypoxemia, and a greater decrement in exercise capacity compared to placebo and nebivolol at a simulated altitude of 2000 m (6560 ft).[3] The duration of exposure to hypoxia in this study was short, however, and data are insufficient to recommend patients change to alternative β-blockers for the purpose of high-altitude travel. When used for altitude illness prophylaxis, acetazolamide may have the added benefit of improving overall fluid balance because of its diuretic effect. Beyond such medication concerns, it is important to remember that many heart failure patients also have implanted pacemakers and defibrillators. Although limited evidence suggests that pacemaker ventricular stimulation thresholds remain unchanged with short hypoxic exposures,[156] a 4% incidence of defibrillator discharges has been noted at altitudes above 2000 m [6562 feet].[79] Whether this latter finding is related to changes in the device's defibrillation threshold or to the patient's underlying condition remains unclear at this time.

### Congenital Heart Disease

Many patients with congenital heart disease (CHD) are living longer and are able to engage in a variety of activities as a result of improvements in medical care, but the evidence base guiding management of these patients at high altitude remains limited. In perhaps the only study focused on CHD patients at high altitude, Garcia and associates[56] demonstrated that patients with a history of the Fontan procedure for correction of tricuspid atresia tolerated submaximal exercise at 3050 m (10,000 feet) despite lacking a functional right ventricle. Case series and reports have documented an increased risk of HAPE in patients with unilateral agenesis of a pulmonary artery,[68,130] and a single study suggests that patent foramen ovale (PFO) may be associated with an increased risk of HAPE;[6] however, a causal link between PFO and HAPE has not been definitively established. Because HAPE-susceptible patients are known to have exaggerated pulmonary vascular reactivity,[40,66] the PFO may occur because of the large PAP responses to hypoxia and exercise and thus may be a phenomenon associated with HAPE rather than a link in the causal pathway.

Aside from the limited evidence base, another challenge in evaluating CHD patients before planned high-altitude travel is the wide variety of congenital disorders and their associated repairs, which makes assessment of anticipated changes in hemodynamics and gas exchange at high altitude difficult. Nevertheless, two groups of patients warrant increased attention. As previously noted, patients whose congenital defects are associated with pulmonary hypertension may be at increased risk for HAPE or worsening right ventricular function, whereas patients with cyanotic diseases will be at risk for marked hypoxemia, particularly when engaged in any physical activity.[96] Whether the latter group can maintain adequate tissue oxygen delivery in the face of severe hypoxemia will be a function of their underlying cardiac function[22] and whether they have secondary polycythemia as a result of chronic hypoxemia.[28] Greater hypoxemia secondary to intracardiac or intrapulmonary shunting will likely increase the risk of altitude illness.

These concerns can be evaluated before any planned trip using echocardiography to determine PAP, hypoxia altitude simulation testing to assess the likelihood of severe hypoxemia, or CPET in normoxia or hypoxia to assess exercise tolerance and risk of exercise-induced hypoxemia, while being aware of the limitations of these tests as discussed earlier. High-altitude travel should be avoided in patients with moderate to severe pulmonary hypertension, low maximum exercise capacity ($\dot{V}O_2$max <20 mL/kg/min), or cyanotic disease and anemia and/or impaired cardiac output.[96] There is no evidence to support closing a PFO before ascent to high altitude to prevent HAPE.

### Arrhythmia

From a theoretical standpoint, increased sympathetic activity in response to hypoxemia would be expected to increase the incidence or severity of tachyarrhythmia at high altitude. Although there is evidence of sinus tachycardia, atrial and ventricular ectopy, and atrial flutter in healthy individuals traveling to high altitude,[85,158] little is known about the effects of hypobaric hypoxia on persons with preexisting arrhythmias, such as atrial flutter or other supraventricular tachycardias. In one of the few studies on this issue, Wu and associates[159] performed ECG and 24-hour ambulatory monitoring on 42 individuals with preexisting arrhythmias at 1 week and at 3 months after arrival at 4500 to 5050 m (14,760 to 16,564 feet) and found only a single case of asymptomatic Wolff-Parkinson-White syndrome. No participants experienced life-threatening arrhythmias or exacerbation of their preexisting arrhythmia. Given the lack of documented problems, it is likely safe for arrhythmia patients to continue to go to high altitude, provided their arrhythmia is under adequate control at the time of their sojourn. Patients should continue their medication regimen through the duration of their trip and should have a plan in place in the event of an exacerbation.

## HEMATOLOGIC DISEASES

### Hypercoagulable States

Because of scant evidence, it is unclear whether hypobaric hypoxia increases the risk of thromboembolic disease in individuals with a history of venous or arterial thromboembolism or known coagulopathy. Many case reports document thromboembolic events at high altitude in which the patient was known or subsequently found to have evidence of coagulopathy, such as protein C deficiency,[26] antiphospholipid antibody syndrome,[16] hyperhomocysteinemia,[8] or S/C hemoglobinopathy,[69] but little information exists about the relative risk of thromboembolism at high altitude in such patients compared to sea level. In one of the few studies that provides some insight into this issue, Schreijer and associates[139] exposed healthy individuals to hypobaric hypoxia equivalent to 1800 to 2100 m (5904 to 6888 feet) and found that levels of thrombin-antithrombin complexes were increased only in individuals with factor V Leiden mutation and oral contraceptive use, suggesting that a preexisting coagulopathy might affect the response to hypobaric hypoxia. Other studies examining changes in markers of coagulation during hypoxic exposure in normal individuals have also failed to yield consistent evidence regarding bleeding times,[13,46] fibrinolytic activity,[14,103] platelet count and function,[73,141] or thrombin formation.[15,20] However, a recent study using thromboelastography actually showed evidence of slowed coagulation at high altitude rather than a hypercoagulable state.[104]

Given the evidence that risk of thrombosis is not increased by hypobaric hypoxia high altitude, there is no indication for individuals with underlying coagulopathy who are not already taking anticoagulants to start warfarin or antiplatelet therapy before high-altitude travel. Individuals receiving anticoagulation therapy should continue their regimen at high altitude, and patients taking warfarin should arrange to monitor prothrombin time during or immediately after stays lasting over 2 weeks, because some evidence suggests that these values may change as a result of changes in elevation.[154] No data are available regarding the effects of hypobaric hypoxia on direct thrombin inhibitors. Limiting activities in remote areas should be considered to reduce the risk of trauma and severe hemorrhage. Regardless of whether they use anticoagulation, patients with a history of thromboembolism or coagulopathy should maintain hydration and adequate mobility throughout their stay, because these factors may affect the risk of thrombosis independent of the effects of hypoxia.

### Hemoglobinopathy

Sickle cell anemia patients should avoid travel to high altitude because hypoxia can trigger red blood cell sickling and vasoocclusive crises.[53] Even travel to modest elevations may be associated with risk. Previous work has demonstrated a 20% incidence of vasoocclusive and splenic crises in patients with sickle cell anemia or hemoglobin S/C traveling above 2000 m (6560 feet) or flying in pressurized aircraft.[102] Beyond the risk of vasoocclusive crises, sickle cell anemia patients can develop pulmonary hypertension as a consequence of long-standing disease,[84] which could predispose to HAPE or worsening right-sided heart function in hypobaric hypoxia. If high-altitude travel cannot be avoided, patients should travel with supplemental oxygen. Unlike patients with COPD or other lung diseases, who might travel with a prescription for supplemental oxygen and use it only if they have problems at high altitude, sickle cell anemia patients should plan on having oxygen available immediately on arrival.

The risk of problems in patients with sickle cell trait has not been specified, but several reports document the occurrence of splenic crises in patients traveling between 1600 and 3660 m (5250 and 12,005 feet).[80,54,63,150] Such reports should probably not preclude high-altitude travel in all sickle cell trait patients, but development of left upper quadrant pain following ascent to high altitude should prompt urgent medical evaluation and descent to lower elevation.

Thalassemia has not been reported to cause problems at high altitude.

### Anemia

Depending on its severity, anemia impairs oxygen delivery and limits exercise tolerance at high altitude. No study has assessed whether anemia increases the risk of high-altitude illness or has defined specific hemoglobin thresholds that are safe for high-altitude travel. Depending on the anticipated length of stay and level of exertion at high altitude, blood transfusion can be considered before the planned trip, although the appropriate target hemoglobin concentration after transfusion is unclear. Anemic patients planning trips longer than 2 weeks should consider oral iron supplementation.

### Polycythemia Vera

As with anemia, evidence is scant regarding polycythemia vera (PV) patients at high altitude. From a theoretical standpoint, higher hemoglobin concentrations would be expected to improve oxygen delivery in the setting of hypoxemia. On the other hand, individuals traveling at high altitude are at increased risk for dehydration because of lower humidity and altitude-induced diuresis. In PV patients, this could raise hemoglobin concentration further, thereby worsening blood viscosity, impairing cardiac output and oxygen delivery, and increasing risk of thrombosis.[146] The effect of hypobaric hypoxia on risk of gastroduodenal erosions in PV patients is unknown.[152] Given these uncertainties, PV patients can travel to high altitude but should maintain adequate hydration and mobility and should consider using low-dose aspirin to reduce the risk of thrombotic events. Patients with a history of gastroduodenal erosions should avoid aspirin, as well as dexamethasone, because either medication might increase the risk of upper gastrointestinal bleeding.

## NEUROLOGIC DISORDERS

### Headaches

A history of nonmigrainous headaches at lower elevation may predispose to headaches at high altitude[23] and has been associated with increased severity of headaches when they occur after ascent.[142] Hypoxia may trigger migraine headaches at high altitude in patients with a known history of the disorder[138] (or a family history of migraine), and these headaches may be of greater severity and associated with focal neurologic deficits.[111] Anecdotal reports[67] and systematic studies[127] also suggest that a history of migraines may also predispose to acute altitude illness after ascent. An important issue for these patients is distinguishing a migraine headache from headaches associated with AMS, with the key distinguishing factors being whether the patient has an aura and focal neurologic deficits—features typically lacking in AMS-associated headaches—and the character of the headache compared to the typical migraine at lower elevations. Headaches that are different in character than typical migraines or that do not respond to standard migraine headache treatment, such as sumatriptan, should be treated as AMS. Response to oxygen breathing can also be helpful, because altitude headaches typically improve in 10 to 15 minutes, whereas migraine headaches do not.

### Seizures

High altitude may rarely induce seizure in healthy persons and sometimes unmask preexisting seizure disorders.[98] Individuals with known seizure disorders well controlled with medication are not thought to be at increased risk for increased frequency or severity of seizures after ascent. They can trek or do other activities at high altitude but should avoid technical climbing or roped travel on glaciated terrain because of the risks to the individual or travel partners if a seizure occurs in either setting. Individuals who use topiramate as part of their seizure regimen should use dexamethasone rather than acetazolamide for AMS prophylaxis at high altitude; topiramate has carbonic anhydrase activity similar to that of acetazolamide, and concurrent use of the medications for more than a few days can lead to severe metabolic acidosis and nephrolithiasis.[97] Persons with a past history of more than one seizure who are not taking antiseizure medications should be cautious on ascent to altitude and should pay close attention to ensuring an adequately slow ascent rate.

### Cerebrovascular Diseases

Minimal information exists about the risk of high-altitude travel in patients with cerebrovascular disease. As with patients who have recently had a myocardial infarction and revascularization, it is prudent to delay high-altitude travel after a recent transient ischemic attack or cerebrovascular accident (stroke) for at least 90 days.[18] Patients taking warfarin, clopidogrel, or a direct thrombin inhibitor as part of primary or secondary stroke prevention should be cautious with travel into remote areas, where trauma and bleeding can have significant consequences if the individual cannot access care in a timely manner. Patients with known, unsecured intracranial aneurysm or arteriovenous malformation should probably avoid high-altitude travel because of a theoretical risk of rupture from cerebral vasodilation and increased flow, or if systemic BP increases significantly after ascent. If high-altitude travel cannot be avoided, efforts should be made to limit exertion during the sojourn.

## DIABETES MELLITUS

There are multiple issues for diabetic patients traveling to high altitude. It is important to note that the literature on short-term exposure to high altitude is based largely on studies done in well-controlled type 1 diabetic patients engaged in climbing expeditions.[1,76,110,124] Significantly less information is available about patients with poorly controlled type 1 or with type 2 diabetes of any severity, so care must be used in extrapolating available data to these individuals.

### Acclimatization and Risk of Altitude Illness

Multiple studies from climbing expeditions suggest that the ventilatory and hematologic responses to acute hypoxia, risk of acute altitude illness, and summit success rates are similar in well-controlled type 1 diabetic patients and healthy individuals. Pavan and colleagues,[125] for example, measured blood gases and hematologic parameters in well-controlled type 1 diabetic patients at 3700 and 5800 m (12,135 and 19,025 feet) during an ascent of Cho Oyu and noted no difference in $PaO_2$, $PaCO_2$, bicarbonate, or hematocrit. Multiple studies have reported no difference in the incidence of AMS or Lake Louise AMS scores between diabetic and nondiabetic climbers during ascents of Aconcagua (6962 m [22,835 feet]), Cho Oyu (8201 m [26,890 feet]), or Kilimanjaro (5895 m [19,335 feet]).[76,110,124] In only one of the studies did diabetic climbers fail to summit the mountain, but summit success rates were low in the nondiabetic climbers as well,

making it difficult to attribute the lack of summit success to diabetes alone. In one of the few studies examining type 2 diabetic patients, del Mol and associates[36] reported low AMS scores in 13 patients with no history of diabetes-related complications during a 12-day trek to the summit of Mt Toubkal (4167 m [13,665 feet]) in Morocco.

### Insulin Requirements and Glycemic Control

Multiple factors impact glycemic control at high altitude, including changes in diet, degree and duration of exercise, sympathetic nervous system responses to hypoxia, and other forms of stress when traveling in this environment. The majority of studies on insulin requirements and glycemic control report increased requirements during high-altitude travel,[1,35,124] whereas a single study has noted decreased insulin requirements.[110] Interpretation of these data is difficult, however, because ascent rates, levels of exertion, altitudes attained, and dietary factors varied significantly between studies. These studies also involved travel to extreme elevations (>5800 m [19,025 feet]) and may not be relevant to most high-altitude travelers or to situations where individuals are not engaged in the physical exertion of expedition climbing.[128] Given the uncertainties about what will happen with insulin requirements in most individuals, travelers with diabetes should plan on checking blood glucose levels more frequently than they would at home and should be prepared to change insulin dosing as necessary.

### Glucometer Function

Monitoring glucose levels more closely at high altitude requires accurate glucometers. Many earlier studies reported issues with glucometer accuracy at high altitude,[51,57,59,110,126] possibly because older monitors were glucose oxidase–based systems. Some of the more recent-generation devices operate on a glucose dehydrogenase reaction, which is not oxygen dependent and theoretically less susceptible to the effects of altitude. Thus far, however, it remains unclear which system performs best. Oberg and associates,[119] for example, found that the glucose dehydrogenase meters performed better at simulated altitudes of 2500 and 4500 m (8200 and 14,780 feet), whereas Olateju and colleagues[120] noted no difference in performance at a simulated altitude of 2340 m (8000 feet). De Mol and associates[37] noted no difference at simulated altitude but found that at terrestrial high altitude, one of the tested glucose dehydrogenase systems was most accurate. The measurement errors are generally not clinically significant. When readings fall near the low end of normal, however, small measurement errors may have greater implications because travelers may fail to recognize true hypoglycemia. For this reason, diabetic travelers at high altitude should err on the side of caution and react more readily to values near the low end of normal rather than using the typical thresholds for intervention.

### Insulin Pumps

Diabetic patients are increasingly using insulin pumps rather than intermittent subcutaneous injections for blood glucose regulation. A single study has examined function of these pumps in hypobaric hypoxia and found that bubbles formed and expanded within the system during commercial flight and in a hypobaric chamber, with the potential to cause excess insulin administration on ascent.[78,88] Therefore, diabetic travelers using insulin pumps should monitor closely for bubble formation in the system and should consider either decreasing the dose administered by the pump or switching to intermittent subcutaneous infection for the duration of the trip.

### Retinal Disease

Retinal hemorrhage is a common complication of travel at moderate to extreme altitudes in normal individuals.[25] Two studies specifically examined this issue in diabetic patients, who are at increased risk for retinal disease. Using ophthalmoscopy, Moore and colleagues[110] found asymptomatic hemorrhage in 2 of 15 diabetic climbers during an ascent of Kilimanjaro; Leal and associates[83] used retinography and noted development of asymptomatic hemorrhage in one of seven climbers ascending a 7100-m (23,290-foot) peak; the one climber had known diabetic retinopathy. These incidence rates are comparable to those seen in the general population climbing at moderate to extreme elevation, so without control groups, these small studies do not establish that diabetes increases the risk of retinal hemorrhage. Given this ongoing uncertainty, there is no reason to restrict high-altitude travel in patients with retinal disease except in cases of severe diabetic retinopathy.[100] Aspirin or other nonsteroidal antiinflammatory drugs (NSAIDs) do not appear to increase the risk of retinal hemorrhage at low elevations, but this has not been studied at high altitude. It may be prudent to rely on acetaminophen as the first-line agent to treat AMS symptoms in patients with retinopathy.

## OBESITY

One of the first studies to provide information on the risk of high-altitude travel in obese individuals was that of Honigman and colleagues,[70] who reported a higher incidence of AMS among obese members of a general tourist population traveling to altitudes of 1920 to 2950 m (6300 to 9675 feet) in Colorado. Ge and associates[58] exposed obese and nonobese individuals to a simulated altitude of 3660 m (12,005 feet) for 24 hours and noted a higher incidence of AMS and lower nocturnal $So_2$ in the obese participants (Figure 3-3). Together, these studies suggest a link between obesity and AMS but do not clarify the causal mechanism behind this link or the degree of obesity necessary to increase risk. Whether the efficacy of acetazolamide for AMS prophylaxis is similar between obese and nonobese individuals is unclear, although no theoretical rationale exists to suggest there should be a difference.

Morbidly obese individuals who develop the obesity hypoventilation syndrome, a disorder marked by alterations in pulmonary mechanics and ventilatory control, may be at risk for additional problems beyond AMS at high altitude. These patients often have pulmonary hypertension caused by chronic alveolar hypoxia, so further rises in PAP after ascent could predispose to HAPE or worsening right-sided heart function. Sleep-disordered breathing is another common problem in these patients. Sleep quality and subsequent daytime function may worsen as a result of increased central apnea events and increased nocturnal hypoxemia. Given these risks, morbidly obese individuals with pulmonary hypertension should avoid high-altitude travel. If travel cannot be avoided, individuals should travel with supplemental oxygen or a prescription that can be filled at high altitude based on symptoms or pulse oximetry monitoring. Individuals using nocturnal CPAP or

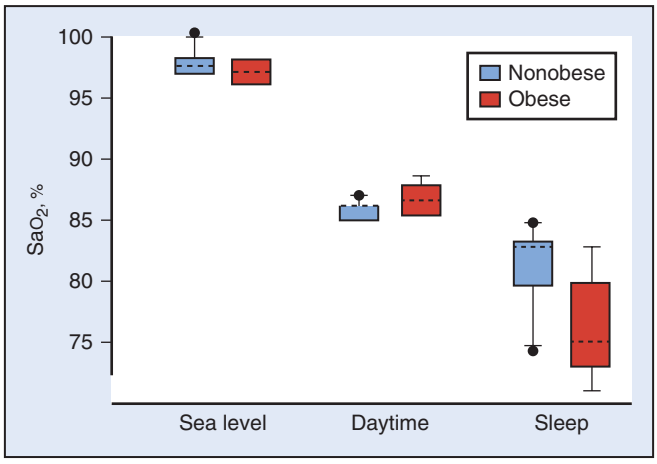


**FIGURE 3-3** Oxygen saturation ($Sa_{O_2}$) in normal *(blue boxes)* and obese individuals (BMI >30 kg/m$^2$) *(red boxes)* while awake at sea level and while awake and sleeping at simulated high altitude (3658 m [12,000 feet]). The bottom of each box represents the lowest 25th quartile of $So_2$ and the top of the box represents the 75th quartile. The *dashed line* represents the median; the *vertical lines* show the largest and smallest values for each group of patients under each condition. *(From Ge RL, Chase PJ, Witkowski S, et al: Obesity: Associations with acute mountain sickness,* Ann Intern Med *139:253-257, 2003.)*

noninvasive positive-pressure therapy should continue these therapies at high altitude for the duration of electrical power access.[95]

## GASTROINTESTINAL DISEASES

### Gastrointestinal Bleeding

Several studies have raised concern about the risk of gastrointestinal (GI) bleeding at high altitude. Wu and colleagues[159] studied more than 13,000 workers between 3500 and 4900 m (11,480 and 16,070 feet) on the Qinghai-Tibet railroad and noted a 0.49% incidence of hematemesis, melena, or hematochezia, and Liu[86] reported an incidence of 0.8% among Chinese soldiers stationed between 3700 and 5380 m (12,135 and 17,550 feet) over a 1-year period. Saito[134] documented GI bleeding in 5 of 52 Mt Everest climbers. Fruehauf and associates[55] performed upper endoscopy on 26 asymptomatic individuals before and after ascent to 4559 m (14,950 feet) and noted new gastric or duodenal erosions/ulcers, hemorrhagic gastritis or duodenitis, and reflux esophagitis in 28% and 61% of individuals on the second and fourth days at high altitude, respectively. Although these studies do not definitively establish that high-altitude travel increases the risk of GI bleeding, they do suggest that individuals with a recent history of GI bleeding or poorly controlled esophagitis, gastritis, or peptic ulcer disease may be at risk for problems at high altitude. It would be prudent for such individuals to avoid extended use of aspirin or NSAIDs for prevention of AMS or treatment of other conditions at high altitude.

### Cirrhosis

No studies have examined how patients with liver cirrhosis fare at high altitude, but there are strong theoretical reasons to suspect that two groups of cirrhotic patients may be at risk for problems: those with portopulmonary hypertension and those with hepatopulmonary syndrome. As noted earlier, patients with portopulmonary hypertension, a form of pulmonary arterial hypertension seen in up to 16% of cirrhotic patients,[21] may be at risk for HAPE or worsening right-sided heart function when PAP increases after ascent. Patients with hepatopulmonary syndrome, a disorder seen in up to 32% of cirrhotic patients[136] and marked by hypoxemia from the presence of pulmonary arteriolar vascular dilation, are at risk for exaggerated hypoxemia. Patients with portopulmonary hypertension should likely avoid high-altitude travel, or if travel is unavoidable, should receive pulmonary vasodilator therapy or travel with supplemental oxygen. Hepatopulmonary syndrome patients should travel with plans to monitor pulse oximetry after ascent and fill a prearranged prescription for supplemental oxygen in the event of severe symptoms or hypoxemia. For cirrhotic patients not known to have these disorders, screening for their presence should be considered before any high-altitude travel.

Cirrhotic patients should not use acetazolamide for AMS prophylaxis or treatment because this may increase the risk of hepatic encephalopathy.[97] Although development of altered mental status at high altitude should always raise concern for HACE, a broader differential should be considered in cirrhotic patients and include the possibility of hepatic encephalopathy provoked by infection, GI bleeding, or medication nonadherence. Cirrhotic patients with altered mental status should not simply be given dexamethasone, but instead be evacuated to a lower elevation or health facility to undergo thorough evaluation.

## OPHTHALMOLOGIC CONDITIONS

### Refractive Error Surgery Patients

Of patients who had the older refractory surgery options, those with radial keratotomy appear to fare the worst at altitude, because hypobaric hypoxia can cause uneven corneal swelling, hyperopic shifts, and impaired vision after more than 24 hours at altitudes as low as 3000 m (9840 feet).[100] Individuals with a history of this procedure who travel to high altitude should travel with glasses with increasing plus power to compensate for such shifts. Corneal swelling can occur with photoreactive radial keratotomy, but the swelling is more uniform and does not lead to refractive errors.[99] Laser-assisted in situ keratomileusis (LASIK) is now performed more often than either radial keratotomy procedure. The available data suggest that high-altitude travel, even to extreme elevations, is tolerated in these patients. Small myopic shifts have been demonstrated in controlled studies[113] and case reports,[24,157] but climbers have still been able to reach summit objectives at extreme elevations. In a detailed report by Dimmig and Tabin,[43] six climbers ascended above 7900 m (25,910 feet) after LASIK in both eyes, and four reached the summit. Five of the six had no visual changes up to 8000 m (26,240 feet), while two climbers reported blurred vision at 8200 and 8700 m (26,900 and 28,535 feet), which improved with descent. Further details on this issue are available in a recent review.[101]

### Glaucoma

The effect of high altitude on intraocular pressure (IOP) is unclear, with studies reporting an increase, decrease, or no change in this parameter during exposure to hypoxia.[19,27,50] In one of the better-controlled studies, Somner and colleagues[144] examined 76 individuals and found statistically significant but clinically insignificant increases in IOP after an exertion-free ascent to 5200 m (17,050 feet), which resolved after 7 days at that elevation. There is no evidence that hypobaric hypoxia provokes acute narrow-angle glaucoma or worsens open-angle glaucoma. Acetazolamide would be a useful means for pharmacologic prophylaxis against AMS in glaucoma patients, given its ability to decrease intraocular fluid production through carbonic anhydrase inhibition.[100]

## PREGNANCY

Because pregnancy-associated hypertension, preeclampsia, and small-for-gestational age infants are more common among high-altitude residents,[108,109] the issue arises of whether short-term exposure to hypobaric hypoxia poses risks to pregnant lowland residents during high-altitude travel. Data are sparse, but limited evidence suggests moderate altitude exposure is likely safe.[71,72] There is no evidence that short-term exposures increase the risk for pregnancy-induced hypertension, spontaneous abortion, placental abruption, or placenta previa.[114] Artal and colleagues[7] performed submaximal and maximal exercise tests on seven sedentary women at 34 weeks' gestation at sea level and after 2 to 4 days of acclimatization at 1830 m (6000 feet) and documented the expected decrease in maximal aerobic work, but no difference from sea level in fetal heart rate responses or maternal lactate, epinephrine, or norepinephrine concentration. In another study of 12 pregnant women exercising after ascent to 2225 m (7300 feet), Baumann and associates[17] also found no evidence of abnormal fetal heart rate responses. Additional studies have demonstrated that the fetus tolerates acute hypoxia at high altitude, provided that placental fetal circulation is normal.[10,34,131]

When viewed together, the limited literature suggests that physical exertion during short-term exposure to altitudes up to 3000 m (9840 feet) is likely safe for women with normal, low-risk pregnancy.[71,75] High-altitude travel should be avoided, however, in certain groups of patients, particularly after the 20th week of pregnancy (Box 3-1).[75] Before any high-altitude travel, pregnant

**BOX 3-1 Contraindications to High-Altitude Travel in Pregnancy**

Anemia
Chronic hypertension
Impaired placental function
Intrauterine growth retardation
Maternal heart or lung disease
Preeclampsia
Pregnancy-induced hypertension
Smoking

Data from Jean D, Moore LG: Travel to high altitude during pregnancy: Frequently asked questions and recommendations for clinicians, *High Alt Med Biol* 13:73-81, 2012.

women should have a checkup, including ultrasound, to ensure their pregnancy is low risk. Those who travel to high altitude should not exceed levels of exertion done at home, maintain adequate hydration, and avoid travel to remote areas. Care should be taken to avoid high-altitude illness, because severe hypoxemia during HAPE could impair fetal oxygenation. While pharmacologic options are available for prophylaxis and treatment of altitude illness (Table 3-3), slow ascent should be the primary prevention strategy and descent the primary treatment strategy.

## MEDICATION CONSIDERATIONS IN HIGH-ALTITUDE TRAVELERS WITH UNDERLYING MEDICAL PROBLEMS

Recently updated expert guidelines provide recommendations for pharmacologic approaches to prevention and treatment of acute altitude illness.[94] Dosing, efficacy, and safety of the medications used for these purposes have been established largely on the basis of studies done in healthy individuals; little information exists about their use in patients with chronic underlying medical conditions. These issues have been reviewed in detail.[97] Table 3-4 summarizes some key considerations for common medications.

**TABLE 3-3** Safety of High-Altitude Medications During Pregnancy and Lactation

| Medication | Safety During Pregnancy* | Safety During Breastfeeding |
|---|---|---|
| Acetazolamide | Category C | Not established |
| Dexamethasone | Category C | Debate about safety |
| Nifedipine | Category C | Compatible |
| Tadalafil | Category B | Not established |
| Sildenafil | Category B | Not established |
| Salmeterol | Category C | Not established |

Modified from Luks AM, Swenson ER: Medication and dosage considerations in the prophylaxis and treatment of high-altitude illness, *Chest* 133:744-755, 2008.
*Pregnancy Category B: presumed safe based on studies in animals, but no data from humans.
Pregnancy Category C: no human studies demonstrating harm, but animal studies show evidence of teratogenicity.

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

**TABLE 3-4** Dose Adjustments and Other Medication Considerations for High-Altitude Travelers with Underlying Medical Conditions

| Medication | Dose Adjustments: Renal Insufficiency | Dose Adjustments: Hepatic Insufficiency | Other Considerations |
|---|---|---|---|
| Acetazolamide | Avoid use in patients with GFR <10 mL/min, metabolic acidosis, hypokalemia, hypercalcemia, hyperphosphatemia, or recurrent nephrolithiasis. | Acetazolamide use is contraindicated. | Avoid in patients taking chronically high doses of aspirin.<br>Avoid in patients with ventilatory limitation ($FEV_1$ <25% predicted).<br>Use caution in patients with documented severe sulfa allergy.<br>Limit concurrent use with topiramate and ophthalmic carbonic anhydrase inhibitors to 3-5 days. |
| Dexamethasone | No contraindication and no dose adjustments necessary | No contraindication and no dose adjustments necessary | May increase blood glucose values in diabetic patients<br>Avoid in patients at risk for peptic ulcer disease or upper gastrointestinal bleeding.<br>Use caution in patients at risk for strongyloidiasis. |
| Nifedipine | No contraindication and no dose adjustments necessary | Best to avoid<br>If use is necessary, give at reduced dose (10 mg twice daily) | Use caution in patients taking medications metabolized by CYP-450 3A4 and 1A2 pathways.<br>Use caution during concurrent use with other antihypertensive medications. |
| Salmeterol | No contraindication and no dose adjustments necessary | Insufficient data | Potential for adverse effects in patients with coronary artery disease prone to arrhythmia<br>Avoid concurrent use of β-blockers.<br>Avoid concurrent use of monoamine oxidase inhibitors or tricyclic antidepressants. |
| Sildenafil | Dose adjustments necessary if GFR <30 mL/min | Dose reductions recommended<br>Starting dose: 25 mg three times daily<br>Avoid use in patients with known esophageal or gastric varices. | Increased risk of GER<br>Use caution in patients taking medications metabolized by CYP-450 3A4 pathway.<br>Avoid concurrent use of nitrates or α-blockers. |
| Tadalafil | Dose adjustments necessary if GFR <50 mL/min<br>If GFR 30-50 mL/min, use 5-mg dose, maximum 10 mg in 48 hr<br>If GFR <30 mL/min, no more than 5 mg | Child's Class A and Class B: maximum 10 mg daily<br>Child's Class C: do not use tadalafil. | Increased risk of GER<br>Use caution in patients taking medications metabolized by CYP-450 3A4 pathway.<br>Avoid concurrent use of nitrates or α-blockers. |

Modified from Luks AM, Swenson ER: Medication and dosage considerations in the prophylaxis and treatment of high-altitude illness, *Chest* 133:744-755, 2008.
*CYP-450*, Cytochrome P-450; *$FEV_1$*, forced expiratory volume in 1 second; *GER*, gastroesophageal reflux; *GFR*, glomerular filtration rate.

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# CHAPTER 4
# Avalanches

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An avalanche is a mass of snow that slides down a mountainside. In the United States, approximately 100,000 avalanches occur annually, of which about 100 cause injuries, death, or destruction of property. In the 10 years from 2003 to 2013 in the United States, each winter an estimated 350 persons were caught in avalanches, 90 of whom were partly buried, 40 fully buried, 40 injured, and 28 killed.[1] The median yearly direct losses to property are estimated at $100,000, although this varies significantly from year to year. Annual losses from 2003 to 2013 ranged from a low of about $30,000 to a high of $6 million for destruction caused by a 2008 avalanche that destroyed a portion of the high-power transmission lines supplying Juneau, Alaska. This chapter describes the properties of the mountain snowpack that contribute to avalanche formation and discusses avalanche safety techniques, rescue, and the physiology and treatment of avalanche victims.

## PROPERTIES OF SNOW

### SNOW CLIMATES

Snow cover varies on both the broad geographic scale (e.g., Antarctic snow differs from snow found in the Cascade Mountains of North America, which is different from the snow in the southern Rocky Mountains of the United States) and the microscale (e.g., snow conditions may vary greatly from one side of a rock or tree to the other). All snow crystals are made of water molecules, but local and regional environmental conditions control the type and character of the snow found at a given location.

To better understand geographic differences that affect avalanche initiation, it is helpful to consider some basic climate conditions. The character of the snow and avalanche propensity in different mountain ranges around the world can be described as one of three types—maritime, continental, or transitional—on the basis of the average snow conditions of that particular region.

Heavy snowfall and relatively mild temperatures characterize the *maritime* snow climate. The snow cover is deep, and the new snow is dense as a result of moist ocean storms coming ashore at lower altitudes. Rain is common and a significant cause of avalanches when it falls on deep fresh snow. Arctic air intrusions are uncommon but can occur each winter. In general, the snowpack gains strength quickly with time in the maritime snow climate. In North America, examples of maritime ranges include the Sierras, Cascade Mountains, and Coast Range of British Columbia.

Far removed from coastal areas, the *continental* snow climate is characterized by low snowfall, cold temperatures, and higher altitudes. Snowfall is light and of low density, and wind is a key instigator of avalanches. Avalanche cycles are often the result of buried structural weaknesses that occur in shallow snowpacks and may cause avalanche danger to persist for days, weeks, or even months. Avalanches released from these old, persistent, weak layers are a distinguishing trait of a continental climate. In this climate, especially during the early months of winter, the shallow snow cover loses strength with time. Continental ranges include the Canadian Rocky Mountains, southern Rocky Mountains, and Brooks Range.

In between the maritime and continental regions is a *transitional* snow climate that, in North America, is often referred to as the "intermountain" snow climate. Many mountain regions in this class tend to exhibit intermediate features that reflect both maritime and continental snow climates (Table 4-1). Examples of these mountain ranges include the Wasatch Range in Utah, Teton Range in Wyoming, and Columbia Mountains of British Columbia.

### PHYSICAL PROPERTIES

Although snow cover appears to be nothing more than a thick, homogeneous blanket that covers the ground, it is in fact one of the most complex materials found in nature. It often exists concurrently in solid, liquid, and gaseous phases. Snow is highly variable; it may go through significant changes during relatively short periods as a result of environmental factors.

At any single site, the seasonal snow cover varies from top to bottom and results in a complex layered structure, the study of which is referred to as *stratigraphy*. Individual layers may be quite thick or very thin, and may vary greatly in strength and their ability to adhere to one another. In general, thicker layers represent consistent conditions during one storm, when new snow crystals that fall are of the same type, when wind speed and direction vary little, and when temperature and precipitation are fairly constant. Thinner layers that are perhaps only millimeters in thickness often reflect conditions between storms. Examples include a melt-freeze crust or sun crust formed during fair weather and a hard wind crust formed during a period of strong winds. A brittle, buried surface-hoar layer represents what were once delicate, feather-shaped crystals of surface hoar on the surface of the snow that were deposited from the moist atmosphere onto the cold snow surface during a clear night. These crystals are fragile and weak; after they are buried by subsequent snowfalls, they may be persistent and major contributors to avalanche formation.

One property of snow is strength or hardness, which is of great importance in terms of avalanche formation. Snow can vary from light and fluffy, easy to shovel, and especially suitable for skiing, to heavy and dense, impossible to penetrate with a shovel, and firm enough to make it difficult for skiing. Arrangement of the ice skeleton (i.e., the lattice of ice crystals within the snowpack) and the changing density (i.e., the mass per unit volume, typically represented as kilograms per cubic meter [$kg/m^3$]) produce this wide range of conditions. In the case of snow, density is determined by the volume mixture of ice crystals and air. In general, the denser the snow layer, the harder and stronger it becomes, as long as it is not melting.

Density of new snow can have a wide range of values. This depends on how closely the new snow crystals pack together, which is controlled by the shapes of the crystals. The initial crystals that fall from the atmosphere have a variety of shapes, and some pack more closely together than others (Figure 4-1). For example, needle crystals pack more closely than stellate crystals (stellars); thus the density of snow made of compressed needles may be three to four times greater than that of snow composed of stellars.

Wind can alter the shapes of new snow crystals and break them into much smaller pieces that pack very closely together to form wind slabs. Such slabs may possess a density that is 5 to 10 times that of new stellars falling in the absence of wind. Because these processes occur at different times and locations at the surface of the snow cover and are buried by subsequent snowfalls, a varied heterogeneous layered structure results. Minor variations in atmospheric conditions can have an important influence on the properties of snow on the ground.

After snow has been deposited on the surface, snow density increases as the snow layer compacts vertically because of the

**TABLE 4-1** Characteristics of Maritime, Transitional (Intermountain), and Continental Snow Climates on the Basis of 15-Year Means

| Type | Total Precipitation (mm) | Air Temperature in °C (°F) | Snow Depth (cm) | New Snow Density (kg/m³) |
|---|---|---|---|---|
| Maritime | 1286 | –1.3° (29.7°) | 186 | 120 (12%) |
| Transitional | 854 | –4.7° (23.5°) | 170 | 90 (9%) |
| Continental | 550 | –7.3° (18.9°) | 113 | 70 (7%) |

Data from Armstrong R, Armstrong B: Snow and avalanche climates of the Western United States: A comparison of maritime, transitional, and continental conditions. In *Proceedings of the Davos Symposium*. Davos, Switzerland, 1986, Swiss Avalanche Institute.

effects of gravity, weather, and crystal metamorphosis (settlement). Because increase in density often equals increase in strength, the rate at which this change occurs is important with respect to avalanche potential. Snow is composed predominantly of air pockets within an ice skeleton of crystals, and it is therefore highly compressible. In a typical layer of new snow, 85% to 95% of the volume is composed of air pockets, and snow can settle under its own weight. Individual ice crystals can move and slide past each other, and because the force of gravity causes them to move slowly downward, the layer shrinks. A heavier snow layer or warmer temperature speeds settlement.

At the same time, the complex and intricate shapes that characterize the new snow crystals begin to change. They become more rounded and suitable for closer packing. Intricate crystals (e.g., stellars) possess a shape that is naturally unstable and changes quickly. New snow crystals have a large surface area–to–volume ratio and are composed of a crystalline solid that is close to its melting point. In this regard, snow crystals are unique among materials that are found in nature. Surface energy physics dictate that this unstable condition will lead to a change in the crystalline shape toward an energy equilibrium; in other words, the warmer the temperature, the faster the change. Under very cold conditions, the original shapes of the snow crystals are sometimes still recognizable after they have been in the snow cover for several days, or even after 1 or 2 weeks. However, as temperatures warm and approach the melting point, such shapes disappear within a few hours to a day. Changes in the shape or texture of snow crystals are examples of *metamorphism,* which in geologic terms defines changes that result from the effects of temperature and pressure. As the crystal shapes simplify, they can pack more closely together, thereby enhancing further settlement and strength (Figure 4-2).

Metamorphic changes in new snow generally occur within hours to a few days. However, the structure of a seasonal snow cover changes over weeks to months through other processes. Settlement, which may initially have been rapid, continues at a much slower rate. Other factors begin to exert dominant influences on metamorphism throughout the snowpack. These factors include the difference in temperature measured upward or downward in the snow layer, which is called the *temperature gradient.*

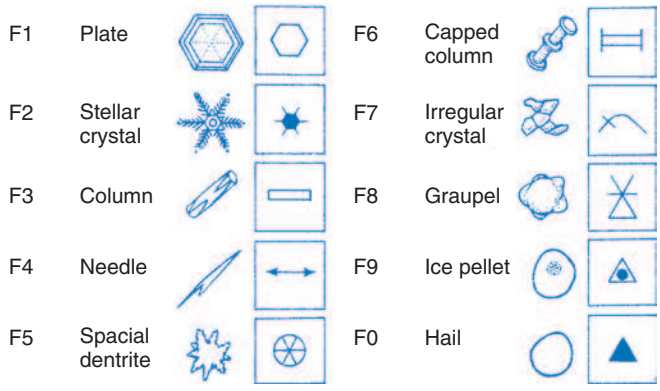
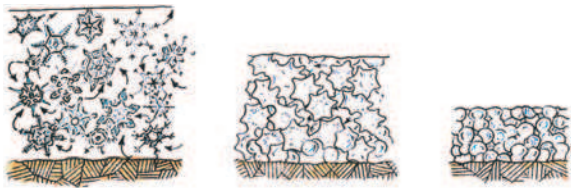



**FIGURE 4-1** International classification of solid precipitation: shape of crystals *(left)* and shorthand recording method *(right). (From the International Association of Scientific Hydrology, with permission.)*



Densification and strengthening of snowpack

**FIGURE 4-2** Settlement. As the crystal shapes become more rounded, the crystals can pack more closely together, and the layer settles or shrinks in thickness.

Averaged diurnally, snow temperatures are generally coldest near the surface and warmest near the ground at the base of the snow cover, which creates a temperature gradient across a snow layer that is sandwiched between cold winter air and the relatively warm ground (Figure 4-3). The effect of temperature gradients is an ongoing dynamic process that can cross ice, large empty spaces filled with air, and dense snow.

Warm air contains more water vapor than does cold air; this holds true for air is trapped within the snow cover. The greater the amount of water vapor, the greater is the pressure; therefore both a pressure gradient and a temperature gradient exist through the snow cover. When a pressure difference exists, it naturally tends to equalize, just as adjacent high and low atmospheric pressure centers cause the movement of air masses. Pressure differences within snow cause vapor to move upward through the snow layers. The air within the layers of the snow cover (in the pore spaces between grains) is saturated with water vapor, with a relative humidity of 100%. When air moves from a warmer to a colder layer, the amount of water vapor that can be supported in the pore spaces diminishes. Some vapor changes to ice and is deposited on the surrounding ice grains. A similar process occurs when warm, moist air in a heated room comes in contact with a cold windowpane. The invisible water vapor is cooled to its freezing point, and some of the vapor changes state and is deposited as frost on the window.

Figure 4-4 shows how the texture of the snow layer changes during this temperature-gradient process. Water molecules sublimate from the upper surfaces of a grain. The vapor moves upward along the temperature (and vapor) gradient and is deposited as a solid ice molecule on the underside of a colder grain above. This process will continue as long as a strong temperature gradient exists. If the gradient continues long enough, all grains

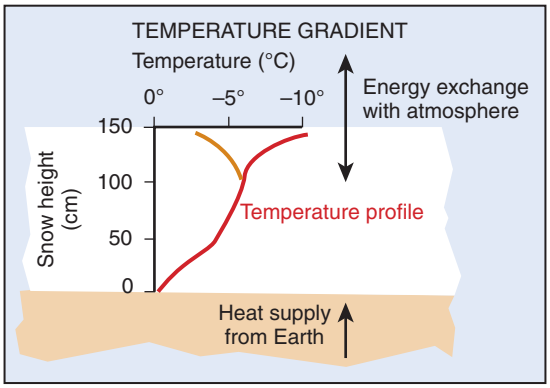



**FIGURE 4-3** As the insulating property of snow separates the warm ground (0°C/32°F) from the cold air, temperature gradients develop across the snow layer. This graph depicts an example of the diurnal variation in temperature gradients in the upper snowpack (20-30 cm) affected by atmospheric energy change. The orange line represents daytime heating by incoming short-wave radiation; the red line depicts nighttime cooling by outgoing long-wave radiation.

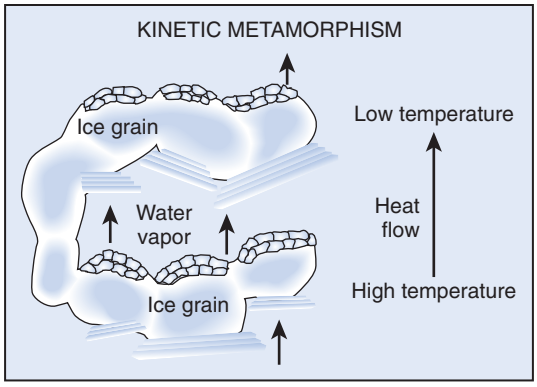



**FIGURE 4-4** In the temperature-gradient process, ice sublimates from the top of one grain, moves upward as water vapor, and then is deposited on the bottom surface of the grain above. If conditions allow this process to continue long enough, all the original grains are lost as recrystallization produces a layer of new crystals.

in the snow layer are transformed from solid to vapor and back to solid again; in other words, they recrystallize. The new crystals are completely different in texture and shape from their initial form. They become loose, coarse crystals with faceting, straight sides and sharp angles (also known as *faceted crystals* or *sugar snow*) and may eventually evolve into a large, striated, hollow-cup form. Examples of these crystals are shown in Figure 4-5. This process is called *temperature-gradient metamorphism* or *kinetic metamorphism*. Well-developed crystals that typically form at the basal layer of the snow cover are commonly known as *depth hoar*. Depth hoar and faceted crystals are of particular importance to avalanche formation because these crystals are very weak, with little or no cohesion (bonding) at the grain contacts. They can form the weak layer that fails under a slab and causes an avalanche.

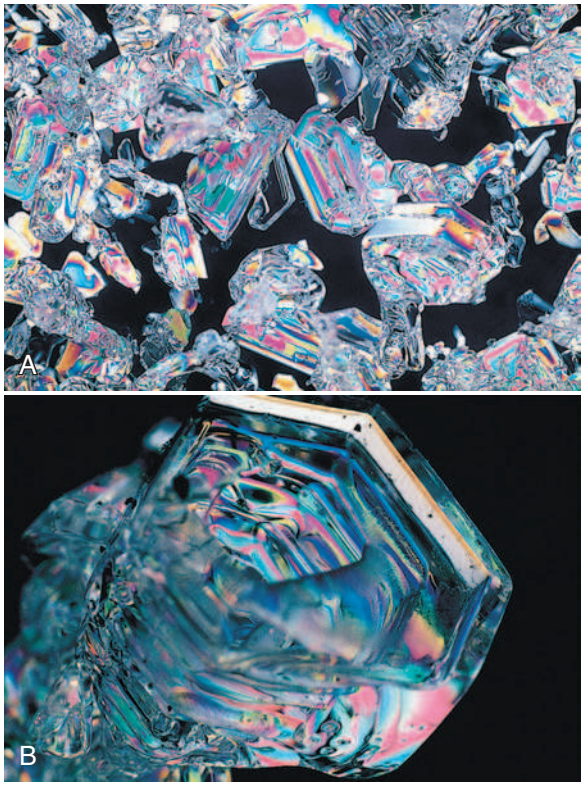



**FIGURE 4-5** **A,** Mature depth-hoar grains. Facets and angles are visible. Grain size, 3 to 5 mm (0.12 to 0.20 inch). **B,** Advanced temperature-gradient grains attain a hollow, cup-shaped form. Size, 4 mm (0.16 inch). *(Courtesy Doug Driskell.)*

## KINETIC METAMORPHISM

In the presence of a strong temperature gradient (e.g., ≥1°C [1.8°F] per 10 cm [4 inches]), kinetic metamorphism can occur anywhere within a snowpack. Typically, temperature gradients are described as extending upward from the warm ground toward the colder snow surface; however, very strong temperature gradients on the order of 2°C (1.8°F) per centimeter or more (20°C [18°F] per 10 cm [4 inches]) can extend for a couple of centimeters from a warm melt-freeze crust into colder snow. Strong temperature gradients can exist in the upper 10 to 30 cm (4 to 12 inches) of the snowpack, which is often related to the effects of alternating incoming solar radiation (short-wave radiation) and outgoing radiational cooling (long-wave radiation) resulting in near-surface facets. These coarse and poorly bonded loose crystals, which are typically 1 to 3 mm (0.04 to 0.12 inch) in size, have straight sides and sharp angles. Backcountry skiers often describe this snow as "recycled powder," because it often forms on colder and northerly facing slopes with prolonged periods of cold and clear conditions. A temperature gradient forms because radiational cooling (which occurs often, but not exclusively, on clear nights) causes the surface snow to become very cold relative to the snow 10 to 30 cm below the surface, which changes more slowly and may still be under the effects of daytime warming.

In the colder temperatures of continental snow climates, strong temperature gradients often exist just above the warm ground and can persist for months when the snow cover remains shallow, typically less than 1 m (3.3 feet) deep. The persistent gradient and warm basal snow layer temperature can cause very large and complex faceted grains to form as well-developed depth hoar. These large, cup-shaped crystals, typically 4 to 10 mm (0.16 to 0.4 inch) in size, are characterized by straight sides, sharp angles, and multiple layers of faceted faces. The grains can be described as resembling etched crystal glass, and their organized striations can make them sparkle.

The strong temperature gradients that drive kinetic metamorphism are aided by low-density new snow because the larger pore spaces allow for easier migration of water vapor. Typical faceted crystals or grains, previously referred to as *temperature-gradient snow,* are commonly called *squares*. Depth hoar is reserved for well-developed, cup-shaped, and large-faceted crystals. Any faceted layer in the snow cover can lead to a persistent weak layer, especially layers of larger grains, which are slower and more resistant to change or to gaining strength.

## EQUILIBRIUM METAMORPHISM

In the absence of a strong temperature gradient, a totally different type of snow texture develops. When the gradient is less than about 1°C (1.8°F) per 10 cm (4 inches), there are still vapor pressure differences, but upward movement of vapor through the snow layers occurs at a much slower rate. As a result, water vapor deposited on a colder grain tends to cover the total grain in a more homogeneous manner, rather than showing the preferential deposition that is characteristic of faceted crystals. This process produces a grain with a smooth surface of a more rounded or oblong shape. Over time, vapor is deposited at the grain contacts (concavities) as well as over the remaining surface of the grain (convexities). Connecting bonds that are formed at the grain contacts give the snow layer strength over time (Figure 4-6). Bond growth, which is called *sintering,* yields a cohesive texture, in contrast to the cohesionless texture of depth hoar and other forms of faceted crystals. This type of grain has been referred to by various terms: *equilibrium snow, equitemperature snow, equilibrium metamorphism,* or simply *rounds*. These grains can generally be described as fine and well-sintered (bonded) snow; Figure 4-7 shows such bonded and interconnected grains. Weak temperature gradients and high-density new snow force water vapor molecules to form bonds and thus drives equilibrium metamorphism.

The previous discussion of kinetic and equilibrium metamorphism provides an overview of what happens to snow layers after they have been buried by subsequent snowfalls. If the temperature layer is below freezing and no melting is taking

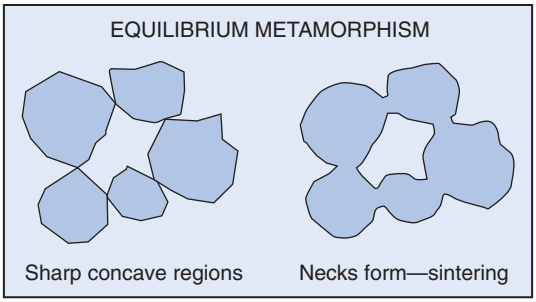



**FIGURE 4-6** Equitemperature grain growth. In the presence of weak temperature gradients, bonds grow at the grain contacts.

place, one of these two processes is occurring, or perhaps a transition exists between the two. Within the total snow cover, these metamorphic processes may occur simultaneously, but only one can take place within a given layer at a given time, depending on the strength or weakness of the temperature gradient. Both processes accelerate with warmer snow temperature because more water vapor is involved.

## AVALANCHE TYPES

There are two basic types of avalanche release. The first is a point-release avalanche or a loose snow avalanche (Figure 4-8). A loose snow avalanche involves cohesionless snow; it is initiated at a point, and spreads out laterally as it moves down the slope to form a characteristic inverted-V shape. A single grain or a clump of grains slips out of place and dislodges those below on the slope, which in turn dislodge others. The avalanche continues as long as the snow is cohesionless and the slope is steep enough. In dry snow, this type of avalanche usually involves only small amounts of near-surface snow. However, in wet snow, which is caused by warm air temperatures or rain, these avalanches can be very large and destructive.

The second type of avalanche, the slab avalanche, requires a cohesive layer of stronger snow over a layer of weaker snow. The cohesive blanket of snow breaks away simultaneously over a broad area (Figure 4-9). A slab release can involve a range of snow thicknesses, from the near-surface layers to the entire snow cover down to the ground. Slab avalanches can occur in dry or wet snow. In contrast to a loose snow avalanche, a slab avalanche has the potential to involve very large amounts of snow.

Because dry slab avalanches are responsible for 95% of U.S. fatal accidents, these avalanches receive the interest of researchers. The majority of the information in this chapter deals with dry slab avalanches or dry, loose snow avalanches. Because wet snow avalanches have received scant research attention, relatively little is known about the processes that cause these avalanches.

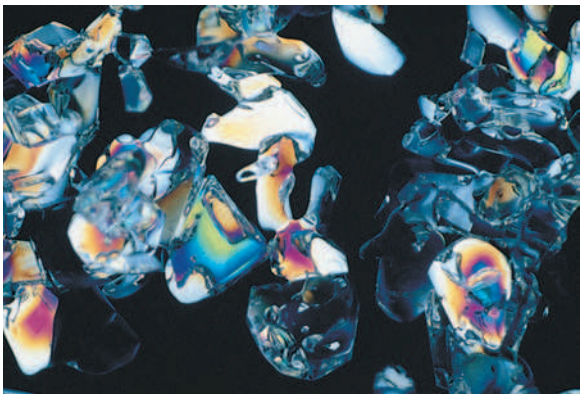


**FIGURE 4-7** Bonded or sintered grains that result from equitemperature metamorphism. Grain size, 0.5 to 1 mm (0.02 to 0.04 inch). *(Courtesy Doug Driskell.)*

## SLAB AVALANCHE FORMATION

To understand the conditions of snow cover that contribute to dry slab avalanche formation, it is essential to reemphasize that snow cover develops layer by layer. The layered structure is directly tied to the two ingredients that are essential to the formation of slab avalanches: the cohesive layer of snow and weak layer beneath. If the snow cover is homogeneous from the ground to the surface, slab avalanche danger is low, regardless of the snow type. If the entire snow cover is sintered, dense, and strong, stability is very high. Even if the entire snow cover is composed of depth hoar, there is still no hazard from slab avalanches, because the cohesionless character precludes formation of a slab, which is one of the essential ingredients. Loose snow avalanches may still occur in this situation on steep slopes. The combination of a basal layer of depth hoar with a cohesive layer above provides the ingredients for slab avalanche danger.

For successful evaluation of slab avalanche potential, information is needed about the entire snowpack and not just its surface. A hard wind slab at the surface may intuitively appear strong

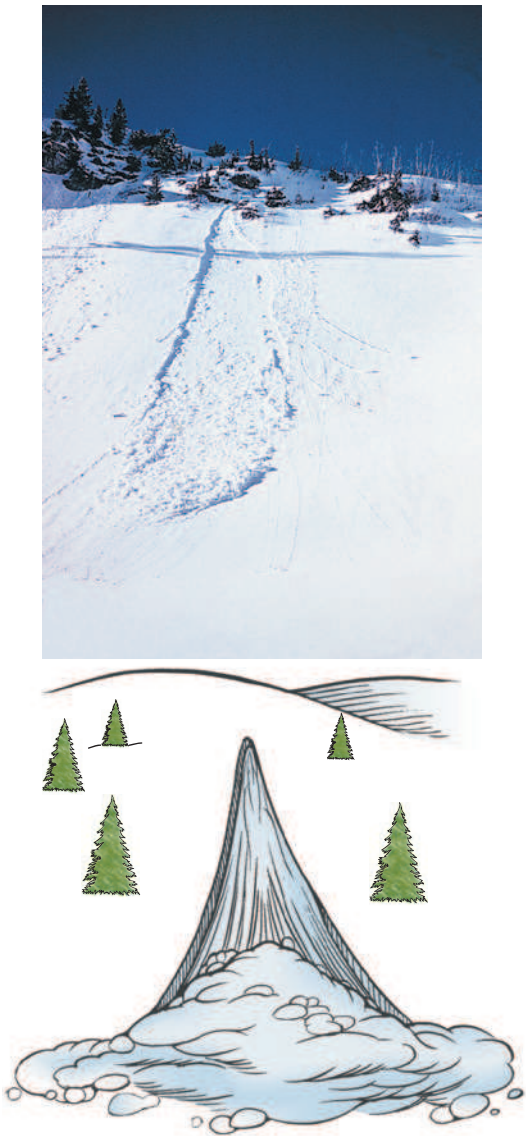


**FIGURE 4-8** Loose snow avalanche or point-release avalanche. *(Courtesy US Department of Agriculture Forest Service.)*

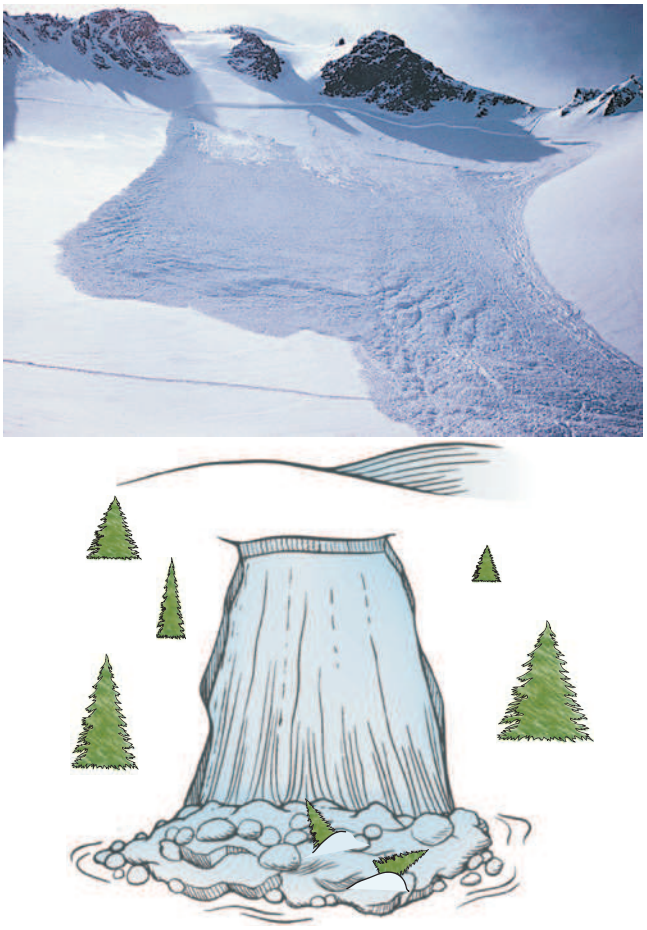


**FIGURE 4-9** Slab avalanche. *(From US Department of Agriculture Forest Service; Williams K, Armstrong B:* The snowy torrents, *Jackson, Wyo, 1984, Teton Bookshop, with permission.* Top, *Courtesy Alexis Kelner.)*

and safe. However, when it rests on a weaker layer that may be well below the surface, it may fail under the weight of a skier and be released as a slab avalanche. Many snow structure combinations can contribute to slab avalanche formation, but again, the prerequisite conditions are a cohesive layer over a weak layer sitting on a bed surface. Figure 4-10 describes other combinations that result in brittle or cohesive layers of snow on a weak layer.

## MECHANICAL PROPERTIES: HOW SNOW DEFORMS ON A SLOPE

Almost all physical properties of snow can be easily seen or measured in the field. A snow pit provides a wealth of information about these properties, layer by layer, throughout the thickness of the snow cover; however, even detailed knowledge of these properties does not provide all the information necessary to evaluate avalanche potential. The current mechanical state of the snow cover must be considered. Unfortunately, for the average person, these properties are virtually impossible to measure directly.

Mechanical deformation occurs within the snow cover just before its failure and the start of a slab avalanche. Snow cover tends to settle simply from its own weight. When this occurs on level ground, the settlement is perpendicular to the ground, and snow layer increases in density and gains in strength. The situation is not so simple when snow rests on a slope. The force of gravity is divided into two components: one that tends to cause the snow layer to shrink in thickness and a new component that acts parallel to the slope and tends to pull the snow down the slope. Downslope movement within the snow cover occurs at all times, even on gentle slopes. The speed of movement is slow, generally on the order of a few millimeters per day, but it can be up to millimeters per hour within new snow on steep slopes or with warming temperatures. The evidence of these forces is often clearly visible in the bending of trees and damage to structures built on snow-covered slopes. Although the movement is slow, when deep snow pushes against a rigid structure, the forces are significant, and even large buildings can be pushed off of their foundations.

Snow deforms in a highly variable manner and is described as a *viscoelastic* material. Sometimes it deforms as if it were a liquid (viscous), and at other times it responds more like a solid (elastic). Viscous deformation implies continuous and irreversible flow. Elastic deformation implies that, after the force causing the deformation is removed, some part of the initial deformation is recovered. The elasticity of snow is not so obvious, primarily because the amount of rebound is very small compared with more familiar materials.

With regard to avalanche formation, it is important to know when snow acts primarily as an elastic material and when it responds more like a viscous substance. These conditions are shown in Figure 4-11. Laboratory experiments have shown that conditions of warm temperatures and slow application of force favor viscous deformation. One sees examples of this as snow slowly deforms and bends over the edge of a roof or sags from a tree branch. In such cases, the snow deforms but does not crack or break. By contrast, when temperatures are very cold or when force is applied rapidly, snow reacts like an elastic material. If enough force is applied, it fractures. We think of such a substance as "brittle"; release of stored elastic energy causes fractures to move through the material. In the case of snow cover on a steep slope, forces associated with accumulating snow or the weight of a skier may increase until the snow fails. At that point, stored elastic energy is released and is made available to drive brittle fractures over great distances through snow slab.

The slab avalanche provides the best example of elastic deformation in snow cover. Although the deformation cannot actually be seen, evidence of the resultant brittle failure is clearly present in the form of the sharp, linear fracture line and crown face of the slab release (Figure 4-12). The crown face is almost always

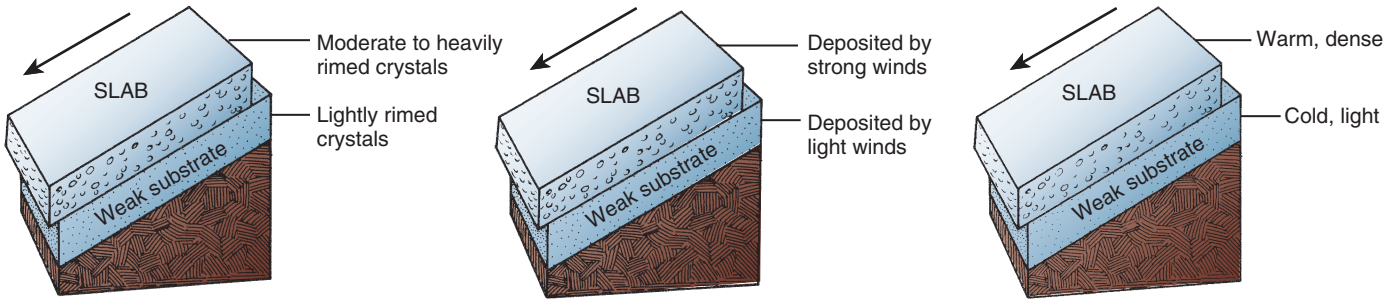



**FIGURE 4-10** Snow-layer combinations that often contribute to avalanche formation.

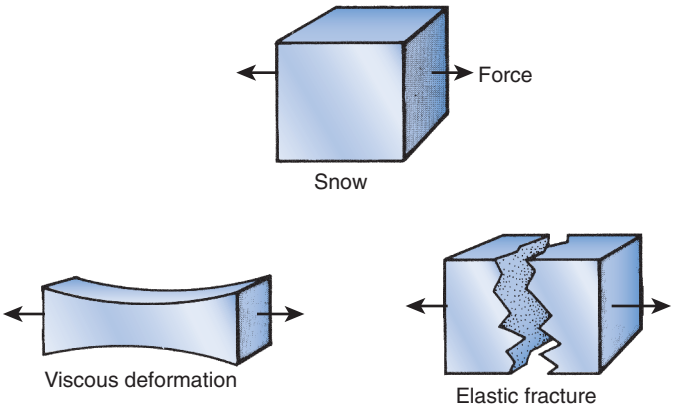



**FIGURE 4-11** Depending on prevailing conditions, snow may deform and stretch in a viscous or flowing manner, or it may respond more like a solid and thus fracture.

perpendicular to the bed surface, which is evidence that snow has failed in a brittle manner.

A full understanding of the slab avalanche condition or stability of the snow cover requires consideration of its mechanical state. Snow is always deforming in a downslope manner, but throughout most of the winter, the strength of the snow is sufficient to prevent an avalanche. The snow cover is layered, and some layers are weaker than others. During periods of snowfall, blowing snow, or both, an additional load or weight is being applied to the snow in the starting zone, the snow is creeping faster, and these new stresses are beginning to approach the strength of the weakest layers. The weakest layer has a weakest point somewhere within its continuous structure. If the stresses caused by the load of the new snow or the weight of a skier reach the level at which they equal the strength of the weakest point, the snow fails completely at that point (Figure 4-13); this means that the strength at that point immediately goes to zero. This is analogous to what would happen if someone on a tug-of-war team were to let go of the rope. If the remainder of the team was strong enough to make up for the lost member, not much would change immediately. The same situation exists with snow cover. If the surrounding snow has sufficient strength to make up for the strength at the weakest point having now gone to zero, nothing happens beyond perhaps a localized movement or collapse in the snow, often heard as a "whumpf" sound. If the surrounding snow is not capable of compensating, however, the area of snow next to the initial weak point fails, then at the next area, and so forth, until a propagating chain reaction begins.

**FIGURE 4-12** The consistent 90-degree angle between the crown face and bed surface of the avalanche shows that slab avalanches result from an elastic fracture. *(Courtesy A. Judson.)*

As the initial crack forms in the now unstable snow, the elastic energy is released, which in turn drives the crack further and releases more elastic energy. The ability of snow to store elastic energy is essentially what allows large slab avalanches to occur. As long as the snow properties are similar across the avalanche starting zone, the crack will continue to propagate, thereby allowing entire basins that are many acres in area to be set in motion within a few seconds.

## AVALANCHE DYNAMICS

The topic of avalanche dynamics includes how avalanches move, how fast they move, and how far and with how much destructive power they travel. The science of avalanche dynamics is not well advanced, although much has been learned during the past few decades. Measured data for avalanche velocity and impact pressure are still being studied. Although any environmental measurement presents its own set of problems, it is clear that opportunities are extremely limited for making measurements inside a moving avalanche.

Avalanche paths exist in a variety of sizes and shapes, but they all have three distinct parts with respect to dynamics (Figure 4-14). In the *starting zone,* which is usually the steepest part of the path, the avalanche breaks away, accelerates down the slope, and picks up additional snow. From the starting zone, the avalanche proceeds to the *track,* where it remains essentially constant and picks up little or no additional snow as it moves. The average slope angle becomes less steep, and frequently the snow cover is more stable than in the starting zone; however, a significant amount of snow can be entrained into the avalanche from the track. Small avalanches often stop in the track. Larger avalanches travel down the track and into the *runout zone,* where the avalanche motion ends. Most avalanches stop quickly, within seconds, although very large avalanches tend to decelerate slowly across a gradual slope, such as an alluvial fan. As a general rule, the slope angle of starting zones is 30 to 45 degrees, track is 20 to 30 degrees, and runout zone is less than 20 degrees.

Avalanches may appear to be turbulent rivers of snow with fluid-like characteristics; however, avalanches are granular flows that move much more like a sliding block than like water. Few actual measurements of avalanche velocities have been made, but enough data have been obtained to provide some typical values for the various avalanche types. For highly turbulent dry powder avalanches, velocities are typically in the range of 34 to 45 meters per second (m/s), or 75 to 100 miles per hour (mph), with rare examples in the range of 67 to 89 m/s (150 to 200 mph). Such speeds are possible for powder avalanches because large amounts of air in the moving snow greatly reduce the forces that result from internal friction. As the snow in the starting zone becomes dense, the terrain becomes less steep and movement becomes more flow-like, with typical velocities slowing to the range of 22 to 34 m/s (50 to 75 mph). During spring conditions, when the snow contains large amounts of liquid water, speeds may reach only about 11 m/s (25 mph).

In most cases, the avalanche simply follows a path down the steepest route on the slope while being guided or channeled by terrain features. However, the higher-speed avalanche may deviate from this path. Terrain features (e.g., side walls of a gully) that would normally direct the flow of the avalanche around a bend may be overridden by a high-velocity powder avalanche (Figure 4-15). The slower-moving avalanches, which travel near the ground, tend to follow terrain features, thereby giving them somewhat predictable courses.

Because avalanches can travel at very high speeds, the resultant impact pressures can be significant. Smaller and medium-size events with impact pressures of 1 to 15 kilopascals (kPa) have the potential to heavily damage wood-frame structures. Extremely large avalanches with impact pressures of more than 150 kPa possess the force to uproot mature forests and even to destroy structures made of concrete.

Some reports of avalanche damage describe circumstances that cannot be easily explained simply by the impact of large amounts of fast-moving dense snow. Some observers have noted that as an avalanche passed, some buildings seemingly exploded,

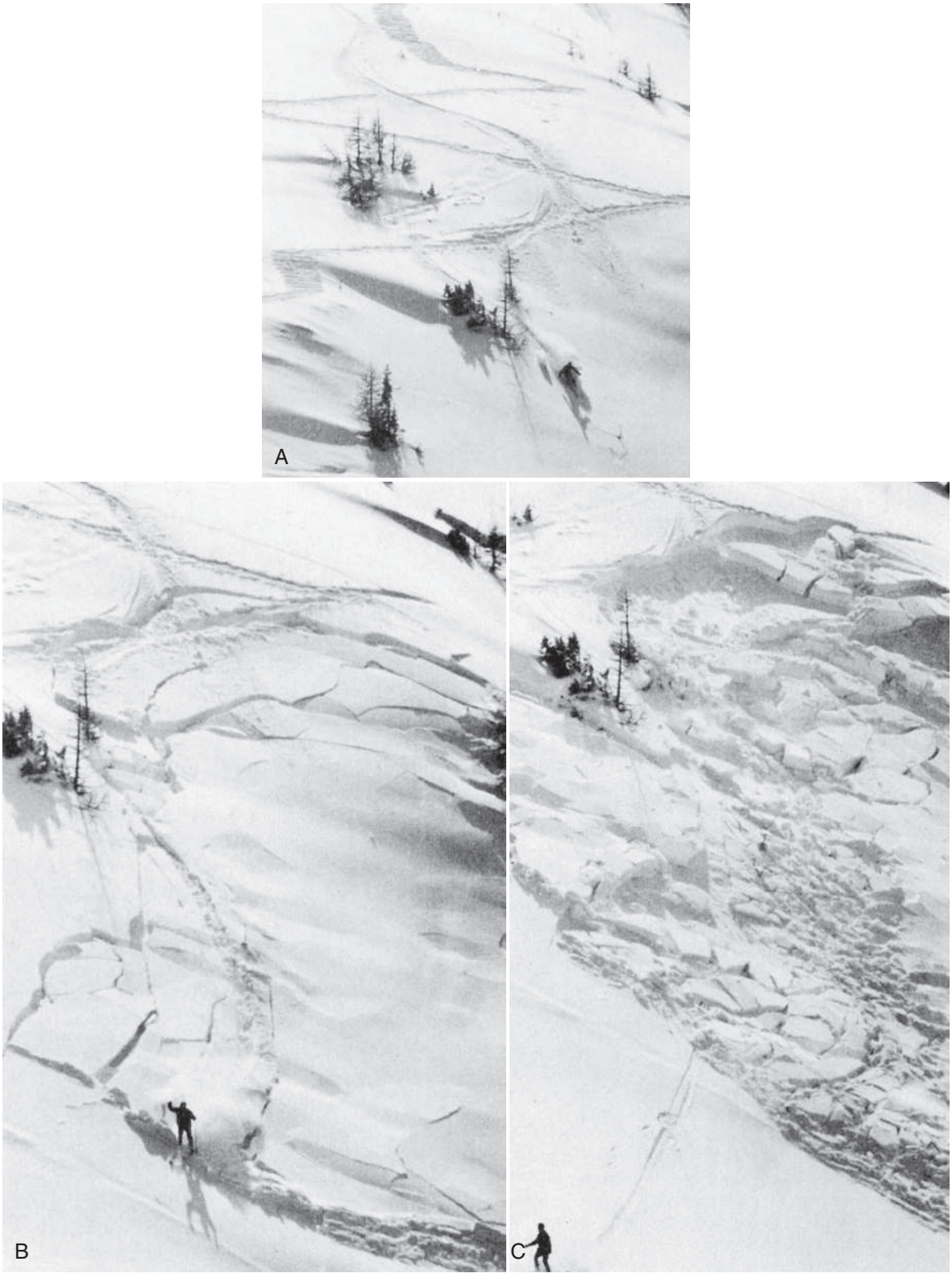



**FIGURE 4-13** Slab avalanche released by a skier. A skier is descending a slope **(A)** and causes the release of a slab of avalanche **(B),** but is able to ski off to the side to escape **(C).** *(Courtesy R. Ludwig.)*

perhaps from some form of vacuum created by the fast-moving snow. Other reports have indicated that a structure was destroyed by the "air blast" preceding the avalanche, because there was no evidence of large amounts of avalanche debris in the area. This damage more likely resulted from the powder cloud, which may be composed of only a few inches of settled snow, yet contributes significantly to the total impact force. Presence of snow crystals can increase air density by a factor of three or more. A powder cloud that is traveling at a moderate dry avalanche speed of 27 m/s (60 mph) could have the impact force of an 80-m/s (180-mph) wind, which is well beyond the destructive capacity of a hurricane.

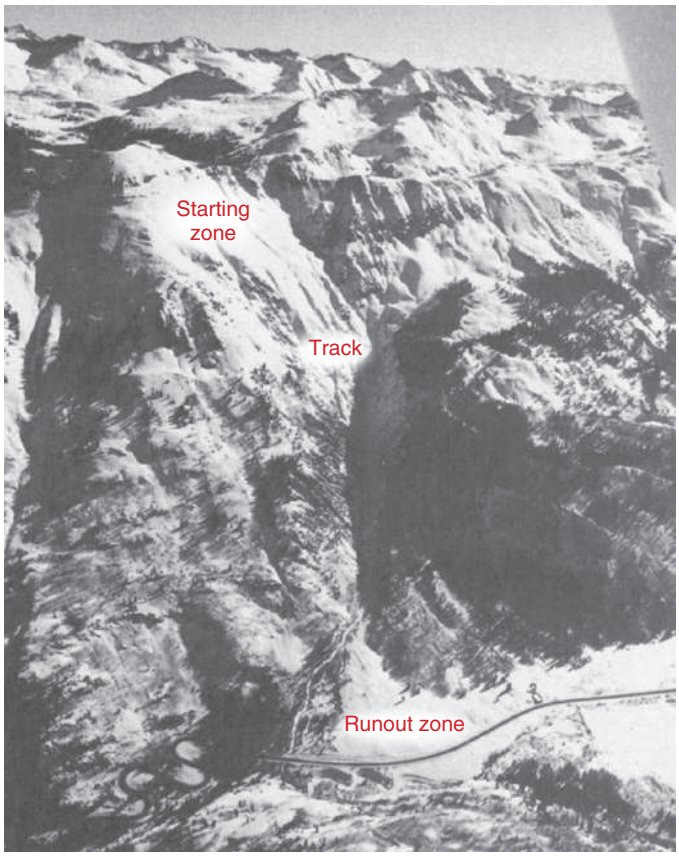



FIGURE 4-14 Three parts of an avalanche path: the starting zone, track, and runout zone. *(Courtesy B. Armstrong.)*

## IDENTIFYING AVALANCHE TERRAIN

The essential ingredients of an avalanche—snow and a steep-enough slope—are such that any mountain or even a small hillside can produce an avalanche if conditions are exactly right. To be a consistent producer of avalanches, a mountain and its weather must work in harmony.

FIGURE 4-15 The large powder cloud associated with a fast-moving, dry snow avalanche. *(Courtesy R. Armstrong.)*

### SLOPE ANGLE

Avalanches occur with greatest frequency on slopes of 30 to 45 degrees. These are the angles at which the balance between strength (i.e., the bonding of the snow trying to hold it in place) and stress (i.e., the force of gravity trying to pull it loose) is most critical. The easiest way to create high stress is to increase the slope angle; gravity works that much harder to stretch the snow and to rip it from its underpinnings. A slope of 45 degrees produces many more avalanches than does a slope of 30 degrees. On even steeper slopes (>45 degrees), the force of gravity wins; snow generally rolls or sloughs off, thus preventing buildup of deep snowpacks. Exceptions exist, especially in maritime snow climates or when strong winds plaster damp snow onto steep slopes. Dry snow avalanches have occurred on slopes of 22 to 25 degrees, which is the angle of repose for granular round substances such as sand. These are rare, however, because snow grains are seldom round and seldom touch without forming bonds. Although an avalanche release requires a steep slope, it is possible to trigger an avalanche from shallow and even flat slopes at the bottom of steep slopes. A collapse in a persistent weak layer in these areas could send fractures upslope, thus releasing the avalanche. This is analogous to pulling a log out from the bottom of a wood pile.

When snow is thoroughly saturated with water, a slush mixture is formed, and an avalanche can release on low-angle terrain. For example, a wet snow avalanche in Japan occurred on a beginner slope at a ski area. The slope was only 10 degrees, but the avalanche was large enough to kill seven skiers. This extreme situation applies only to a water-saturated snowpack, which behaves more like a liquid than a solid.

### ORIENTATION

Avalanches occur on slopes facing every point of the compass. Steep slopes are equally likely to face in any direction. In the northern hemisphere, certain factors cause more avalanches to occur on slopes that are facing north, northeast, and east than on those facing south through west; these relate to slope orientation with respect to sun and wind. In the southern hemisphere, more avalanches occur on east, southeast, and south-facing slopes. The sun angle in northern hemisphere winters causes south slopes to receive much more sunshine and heating than north slopes, which frequently leads to radically different snow covers. North-facing slopes have deeper and colder snow covers, often with a substantial layer of depth hoar near the ground. South slopes usually carry a shallower and warmer snow cover that is laced with multiple ice layers that are formed on warm days between storms. Most ski areas are built on predominantly north-facing slopes to take advantage of deeper and longer-lasting snow cover. At high latitudes (e.g., in Alaska), the winter sun is so low on the horizon and the heat input to south slopes is so small that there are few differences in the snow covers of north-facing and south-facing slopes.

The effect of the prevailing west wind at middle latitudes is important. Storms most often move west to east, and storm winds are most frequently from the western quadrant (i.e., southwest, west, or northwest). Storm winds pick up fallen snow and redeposit it on slopes that are facing away from the wind (i.e., northeast, east, and southeast slopes). These are the slopes that are most often overburdened with wind-drifted snow. The net effect of sun and wind is to cause more avalanches on north-facing through east-facing slopes.

### AVALANCHE TERRAIN PATHS

The frequency with which a path produces avalanches depends on a number of factors, with slope steepness being a major one. Again, the easiest way to create high stress is to increase the slope angle; gravity works that much harder to stretch out the snow and rip it from its underpinnings. A 45-degree slope produces many more avalanches than does a 30-degree slope; however, specific terrain features are also important.

Broad slopes that are curved into a bowl shape and narrow slopes that are confined to a gully efficiently collect snow. Slopes

that have a curved horizontal profile, such as a bowl or gully, trap blowing snow that is coming from several directions; the snow drifts over the top and settles as a deep pillow. Alternatively, the plane-surfaced slope collects snow efficiently only if it is being blown directly from behind. A side wind scours the slope more than it loads the slope.

The surface conditions of a starting zone often dictate the size and type of avalanche. A particularly rough ground surface (e.g., boulder field) does not usually produce avalanches early in the winter, because it takes considerable snowfall to cover the ground anchors. After most of the rocks are covered, avalanches pull out in sections, with the area between two exposed rocks running one time and the area between another two other rocks running another. A smooth rock face or a grassy slope provides a surface that is usually too slick for snow to grip; therefore full-depth avalanches are distinctly possible. If the avalanche does not run during the winter, it is likely to run to ground in the spring after melt water percolates through the snow and lubricates the ground surface.

### VEGETATION

Vegetation has a mixed effect on avalanche releases. Bushes provide anchoring support until they become totally covered. At that point, bushes may provide weak points in the snow cover, because air circulates around the bush and provides an ideal habitat for growth of depth hoar. It is common to see that the fracture line of an avalanche has run from a rock to a tree to a bush, because these are all places of healthy depth-hoar growth.

A dense stand of trees can easily provide enough anchors to prevent avalanches. Reforestation of slopes that are devoid of trees as a result of logging, fire, or avalanche is an effective means of avalanche control. Scattered trees on a gladed slope offer little if any support to hold snow in place. Isolated trees may do more harm than good by providing concentrated weak points on the slope.

## FACTORS THAT CONTRIBUTE TO AVALANCHE FORMATION

The factors that contribute to avalanche release are terrain, weather, and snowpack. Terrain factors are fixed, but the states of the weather and snowpack change daily or even hourly. Precipitation, wind, temperature, snow depth, snow surface, weak layers, and settlement are factors that determine whether an avalanche will occur.

### SNOWFALL

New snowfall is the event that leads to most avalanches. More than 80% of avalanches run during or just after a storm. Fresh snowfall adds weight to existing snow cover. If the snow cover is not strong enough to absorb this extra weight, avalanche releases occur. The size of the avalanche is usually related to the amount of new snow. Snowfalls of less than 15 cm (6 inches) seldom produce avalanches. Snows of 15 to 30 cm (6 to 12 inches) usually produce a few small slides, and some of these harm skiers who release them. Snows of 30 to 60 cm (1 to 2 feet) produce avalanches of larger size that present considerable threats to skiers and pose closure problems for highways and railways. Snows of 60 to 120 cm (2 to 4 feet) are much more dangerous, and snowfalls of more than 120 cm (4 feet) produce major avalanches that are capable of large-scale destruction. These predictions are guidelines that are based on data and experience and must be considered with other factors to arrive at the true hazard. For example, a snowfall of 25 cm (10 inches) whipped by strong winds may be serious; a fall of 60 cm (2 feet) of featherlight snow in the absence of wind may produce no avalanches.

### SNOWFALL INTENSITY

The rate at which snowfall accumulates is almost as important as the amount of snow. A snowfall of 90 cm (3 feet) in one day is much more hazardous than the same amount of snow falling over 3 days. As a viscoelastic material, snow can absorb slow loading by deforming or compressing. Under a rapid load, the snow cannot deform quickly enough and is more likely to crack, which is how slab avalanches begin. A snowfall rate of 2.5 cm (1 inch) or more per hour that is sustained for 10 hours or more is generally a red flag with regard to avalanche danger. The danger worsens if the snowfall is accompanied by wind.

### RAIN

The age of the surface snow is an important factor with regard to changes in stability when rain falls. Within hours of light rain falling on fresh or recent snow (i.e., within the last several days), avalanching can occur for reasons that are not well understood. The rain somehow causes a loss of strength in the new snow. Significant rain, usually about 2.5 cm (1 inch), falling on a snow surface several days or more old produces few or no avalanches. Despite the apparent addition of weight caused by rain (2.5 cm of rain is the equivalent in weight to 25 to 30 cm [10 to 12 inches] of snow), rain tends to drain down through the older snow grains and invariably freezes into an ice crust, which adds strength to the snow cover. Later, the smooth crust could become a sliding layer beneath new snowfall. Heavy rain (usually >2.5 cm) greatly weakens the snow cover. It adds weight to the snowpack, but adds no internal strength of its own (in the form of a skeleton of ice, as new snow would create). Rain dissolves bonds between snow grains as it percolates through the top snow layers, thus reducing strength even further.

### NEW SNOW DENSITY AND CRYSTAL TYPE

A layer of fresh snow contains only a small amount of solid material (ice); the majority of the volume is occupied by air. It is convenient to refer to snow density as a percentage of the volume occupied by ice. New snow densities usually range from 7% to 12%, depending on the snow climate. In the high elevations of Colorado, 7% is an average value; in the more maritime climates of the Sierras and the Cascades, 12% is a typical value. Density becomes an important factor in avalanche formation when it varies from average values. Avalanche danger increases when heavier, denser snow falls on lighter, less dense snow, which can occur from storm to storm or even within a single storm (sometimes called an *upside-down storm*).

Wet snowfalls or falls of heavily rimed crystals (e.g., graupel, also called "soft hail") may have densities of 20% or more. *Graupel* is a type of snowflake that has been transformed into a pellet of soft ice because of riming inside a cloud. A layer of heavier-than-normal snow presents a danger because of excess weight. Snowfall that is much lighter than normal (e.g., 2% to 4%) can also present a dangerous situation. If the low-density layer quickly becomes buried by snowfall of normal or high density, a weak layer has been introduced into the snowpack. Because of its low density, the weak layer has a marginal ability to withstand the weight of layers above, thereby making it susceptible to collapse. Storms that begin with low temperatures but then warm up produce an unstable layer of weak, light snow beneath a stronger, heavier layer, thereby acting as a slab and thus as one of the necessary ingredients for an avalanche.

Density is closely linked to crystal type. Snowfalls that consist of graupel, fine needles, and columns can accumulate at high densities. Snowfalls of plates, stellars, and dendritic forms account for most of the lower densities.

### WIND SPEED AND DIRECTION

Wind can transport snow into avalanche starting zones at much greater rates than can snow falling from clouds. Wind drives fallen snow into drifts and cornices from where avalanches begin. Winds pick up snow from exposed windward slopes and drive it onto adjacent leeward slopes, where it is deposited into sheltered hollows and gullies; this can quickly turn a 1-foot snowfall into a 3-foot drift in a starting zone. The rate at which blowing snow collects in bowls and gullies can be impressive. In one test

at Berthoud Pass, Colorado, the wind deposited snow in a gully at a rate of 45 cm (18 inches) per hour.

A speed of 7 m/s (15 mph) is sufficient to pick up freshly fallen snow. Higher speeds are required to dislodge older snow. Speeds of 9 to 22 m/s (20 to 50 mph) are the most efficient for transporting snow into avalanche starting zones. Speeds of more than 22 m/s (50 mph) can create spectacular banners of snow streaming from high peaks, but much of this snow is lost to sublimation in the air or is deposited far down the slope away from the avalanche starting zone.

Winds also increase the avalanche potential, because blowing snow is denser after deposition than before transport. Snow grains are subjected to harsh treatment in their travels; each collision with another grain knocks off arms and rounds sharp angles, thereby reducing the grain's size and allowing the pieces to settle and pack together into a denser layer. The net result of wind is to fill avalanche starting zones with additional, heavier, and more cohesive snow than if no wind had blown.

## TEMPERATURE

The role of temperature in snow metamorphism occurs over days, weeks, and even months. The influence of temperature on the mechanical state of the snow cover is more acute, with changes occurring in minutes to hours. The actual effect of temperature is not always easy to interpret. Whereas a temperature increase may contribute to stabilization of the snow cover in one situation, it might at another time lead to avalanche activity.

In several situations, increased temperature clearly produces increased avalanche potential. In general, these conditions include (1) a rise in temperature during or immediately after a storm and (2) a prolonged period of warm, fair weather, such as occurs with spring conditions. In the first example, temperature at the beginning of a snowfall may be well below freezing, but as the storm progresses, temperature increases. As a result, the initial layers of new snow are light, fluffy, low density, and relatively low in strength, whereas the later layers are warmer, denser, and stiffer. Thus the essential ingredient for a slab avalanche is provided within the new snow layers of the storm: a cohesive slab resting on a weak layer. If the temperature continues to rise, the falling snow turns to rain. This situation is seen in lower-elevation coastal mountain ranges. When this occurs, avalanches are almost certain. As rain falls, additional weight is added to the avalanche slope, but no additional strength is provided, as might be by a new layer of snow.

The second example may occur after an overnight snowstorm that does not produce an avalanche on the slope of interest. By morning, the precipitation stops, and clear skies allow the morning sun to shine directly on the slopes. The sun rapidly warms the cold, low-density, new snow, which begins to deform and creep down slope. The new snow layer settles, becomes denser, and gains strength. At the same time, it is stretched downhill, and some of the bonds between the grains are pulled apart; thus the snow layer becomes weaker. If more bonds are broken by stretching than are formed by settlement, there is not enough strength to hold the snow on the slope, and an avalanche occurs.

In these first two examples, the complete snow cover generally remains at temperatures that are below freezing. A third example occurs when a substantial amount of the winter's snow cover is warmed to the melting point. During winter, sun angles are low, days are short, and air temperatures are cold enough that the small amount of heat gained by the snow cover during the day is lost during the long, cold night. As spring approaches, this pattern changes, and eventually enough heat is available at the snow surface during the day to cause some melt. This melt layer refreezes again that night, but the next day more heat may be available, so eventually a substantial amount of melting occurs, and melt water begins to move down through the snow cover. As melt water percolates slowly downward, it melts the bonds that attach the snow grains, and the strength of the layers decreases. At first the near-surface layers are affected, with the midday melt reaching only as far as the uppermost few inches and with little or no increase in avalanche hazard. If warm weather continues, the melt layer becomes thicker, and the potential for wet snow avalanches increases. The conditions that are most favorable for wet slab avalanches occur when the snow structure provides the necessary layering. When melt water encounters an ice layer or an impermeable crust, or in some cases a layer of weak depth hoar, wet slab avalanches are likely to occur.

## DEPTH OF SNOW COVER

Of the snowpack factors that contribute to avalanche formation, the depth of the snow cover is the most basic. When the early-winter snowpack covers natural anchors (e.g., rocks, bushes), the start of the avalanche season is at hand. However, a word of caution is necessary for backcountry travelers in the weak, depth-hoar–prone, continental snow climate. The early-winter avalanche danger starts not when the natural anchors are covered, but when the snow fills in the spaces between the obvious anchors. North-facing slopes are usually covered before other slopes. A scan of the terrain usually suffices to discern this clue, but another method can be used to determine the time of the first significant avalanches. Long-term studies show a relationship between snow depth at a study site and avalanche activity. For example, along Red Mountain Pass, Colorado, it is unlikely that an avalanche large enough to reach the highway will run until almost 90 cm (3 feet) of snow covers the ground at the University of Colorado's snow study site. At Alta, Utah, when 130 cm (52 inches) of snowpack has built up, the first avalanche to cover the road leading from Salt Lake City can be expected.

## WEAK LAYERS

Any layer that is susceptible to failure and fracture because of the overburden of additional weight is a weak link. Of the snowpack contributory factors, this is the most important, because a weak layer is essential to every avalanche. Fracture in the weak layer propagates along what is called the *failure plane, sliding surface,* or *bed surface.*

The most troubling layers are called *persistent weak layers,* which are usually made of snow crystals and include larger-faceted snow, depth hoar, and surface hoar. These crystals are slow to change shape and gain strength, so they may persist for days, weeks, months, or even all season. One common weak layer is an old snow surface that offers a poor bond for new snow. Another weak layer that forms on the snow surface is hoar frost or surface hoar (see Physical Properties, earlier). On clear and calm nights, this hoar forms a layer of feathery, sparkling flakes that grow on the snow surface. The layer can be a major contributor to avalanche formation when it is buried by snowfall as a result of its frequent persistence in the snowpack. Many avalanches have been known to release from a buried layer of surface hoar, and sometimes this layer is more than 1 month old and 180 cm (6 feet) or more below the surface.

A weak layer that is almost always found deep within the snowpacks that blanket the Colorado Rocky Mountains (continental snowpack) is large temperature-gradient snow (facets) or depth hoar. One way to decide whether a temperature-gradient layer is near its collapse point is to test the strength of the overlying layers and the support provided by specific stability testing performed near the edge of the slope. This is no easy task, and results may be unreliable because of the spatial variability of weaknesses on the slope. In addition, most field stability tests do not test for deep instabilities in the snowpack. Another method is to try jumping on your skis while standing on a shallow test slope of similar aspect. Collapse is a good indication that comparable snow cover on a steeper slope will produce an avalanche. Often, skiers and climbers cause inadvertent collapses while skiing or walking on a depth-hoar–riddled snowpack. The resulting "whumpf" sound is a warning of weak snow below.

In the past couple of decades, study has focused on the frequency of faceted snow layers near the surface of the snowpack and their relation to avalanches, particularly in the deeper snow cover of the transitional (intermountain) snow climate. Several mechanisms involved in the evolution of these layers may

account for the majority of avalanches in this region, because significant depth-hoar–related avalanches are less common.

Graupel (pellet-like heavily rimed crystals) can act as ball bearings after being buried in the snowpack and can be responsible for the layer involved in avalanche initiation.

Finally, a weak layer can be created within the snow cover when surface melting or rain causes water to percolate into the snow and then fan out on an impermeable layer, thereby lubricating that layer and destroying its shear strength. This phenomenon can be seen during the winter months in the maritime snow climate of the West Coast mountains.

Combining the constellation of contributory factors on a day-by-day basis is the avalanche forecaster's art. Every avalanche must have a weak layer on which to release, so knowledge of snow stratigraphy or layering and what level of applied load will cause a layer to fail form the essence of forecasting.

## SAFE TRAVEL IN AVALANCHE TERRAIN

The first major decision often faced in backcountry situations is whether to avoid or confront a potential avalanche hazard. A group that is touring with no particular goal in mind will probably not challenge avalanches. For this group, education to recognize and avoid avalanche terrain is sufficient. At the other extreme, mountaineering expeditions that have specific goals and are willing to wait out dangerous periods or take severe risks to accomplish their objective need considerably more information. Traveling safely in avalanche terrain requires special preparations, including education and carrying safety and rescue equipment. The group should have the skills required to anticipate and react to an avalanche.

### IDENTIFYING AVALANCHE TERRAIN

Because most avalanches release on slopes of 30 to 45 degrees, judging angle is a prime skill for the recognition of potential avalanche areas. An *inclinometer* can be used to measure slope angles. Some compasses are equipped for this purpose; a second needle and a graduated scale in degrees can be used to measure slope angles. A ski pole may be used to judge approximate slope angle. When dangled by its strap, the pole becomes a plumb line from which the slope angle can be "eyeballed."

Evidence of fresh avalanche activity (i.e., the presence of fracture lines and rubble of avalanche snow on the slope or at the bottom) identifies avalanche slopes. Other clues are swaths of missing trees or trees that are bent downhill or damaged, especially with the uphill branches removed. Above the tree line, steep bowls and gullies are almost always capable of producing avalanches.

### ROUTE FINDING

Good route-finding techniques are necessary for safe travel in avalanche terrain (Figure 4-16). Route finding in avalanche country should avoid avalanches, be efficient, and take into account the abilities and desires of the group to choose a route that is not overly technical, tiresome, or time-consuming. The safest way to avoid avalanches is to travel above or below them and at a distance from them. When taking a route above avalanche terrain, the traveler should choose a ridgeline that is above the avalanche starting zones. It is safest to travel the windward side of the ridge. The snow cover is usually thinner and wind packed, with rocks sticking through; this does not make for the most pleasant skiing, but is safe. Cornice collapses present a very real hazard; they should be avoided by staying on the roughened snow toward the windward side. When ascending a potential avalanche path, a safe route (e.g., a dense forested area that is well anchored, a low-angle approach) should be selected.

Skiers who are taking a route in the valley below avalanche terrain should not linger in the runout zones of avalanche paths. Although it is unlikely that a skier who is traveling along the valley could trigger an avalanche high up on the slope, it is possible to trigger an avalanche from below if one is traveling close to the compression zone, thereby initiating a fracture that propagates upward. Slopes of 30 degrees or more should be approached with caution. By climbing, descending, and traversing only in gentle terrain, avalanche risk can be avoided.

### CROSSING AVALANCHE SLOPES

Travel through avalanche country always involves risk, but certain travel techniques can minimize that risk. Proper travel techniques might not prevent an avalanche release but can improve the odds of survival. The timing of a trip is closely involved with safety. Most avalanches occur during and just after storms. Waiting a full day after a storm has ended can allow the snowpack to react to the new snow load and gain strength.

Before crossing or venturing onto a potential avalanche slope, the skier, hiker, or snowmobiler should tighten up clothing; zip up zippers; and put on a hat, gloves, and goggles. Backpacks should be worn normally, because they may afford protection from back trauma and because the items necessary for survival after a rescue can be stored inside. If use of a large mountaineering pack will make a person top heavy and more likely to fall, another route should be considered. The skier should remove pole wrist straps and ski runaway straps, because poles and skis attached to the avalanche victim hinder extremity motions and only serve to drag the victim under. A person who is wearing a rescue transceiver should always be certain that it is transmitting.

When crossing potential avalanche slopes, it is always better to cross as high or low as possible and to avoid the middle. All persons should traverse in the same track, spaced sufficiently apart to expose only one person at a time. This not only reduces the amount of work required but also disturbs less snow, which lowers the chance of avalanche release. Should the slope fracture when individuals are crossing higher up, most of the sliding snow will be below, and the chance of staying on the surface of the moving avalanche will be greater. Invariably the person who is highest on the slope runs the least risk of being buried.

A person who must climb or descend an avalanche path should keep close to its sides. Should the slope fracture, escaping to the side improves the chance of survival. Only one person at a time should cross, climb, or descend an avalanche slope; all other members should watch from a safe location. Two commonsense principles underlie this advice. First, only one group member is exposed to the hazard, thereby leaving the others available as rescuers. Second, less weight is put on the snow. Snowmobilers all too often fall prey to multivictim accidents because an additional rider will drive up a steep slope to help a friend who is stuck near the starting zone.

Skiers, snowboarders, climbers, and snowmobilers should never drop their guard on an avalanche slope. They should stop only at the edge of the slope or beneath a point of protection (e.g., rock outcropping); they should *not* stop in the middle of a slope or in the runout zone. The second, third, or even tenth person traversing or traveling down a slope may trigger the avalanche. Trouble should always be anticipated, and an escape route (e.g., moving to the side, grabbing a tree) should be kept in mind.

### STABILITY EVALUATION TESTS

People who are traveling on snow-covered slopes should perform hands-on tests of snow strength and instability. Testing the strength of snow helps with location of weak layers, and checking for instability demonstrates how well layers are adhering to one another. Several simple but meaningful tests can be performed without digging holes in the snow, although much more information can be learned by digging snow pits.

A fast and simple test to find significant weak layers is to push a ski pole into the snow; this should be done handle-end first if the snow is dense. This helps the individual to feel the major layering of the snowpack. For example, the skier may feel the layer of new snow, and stronger or weaker layers in the middle of the pack can be appreciated by the ease of the push. Hard-snow layers and ice lenses may resist penetration altogether, but

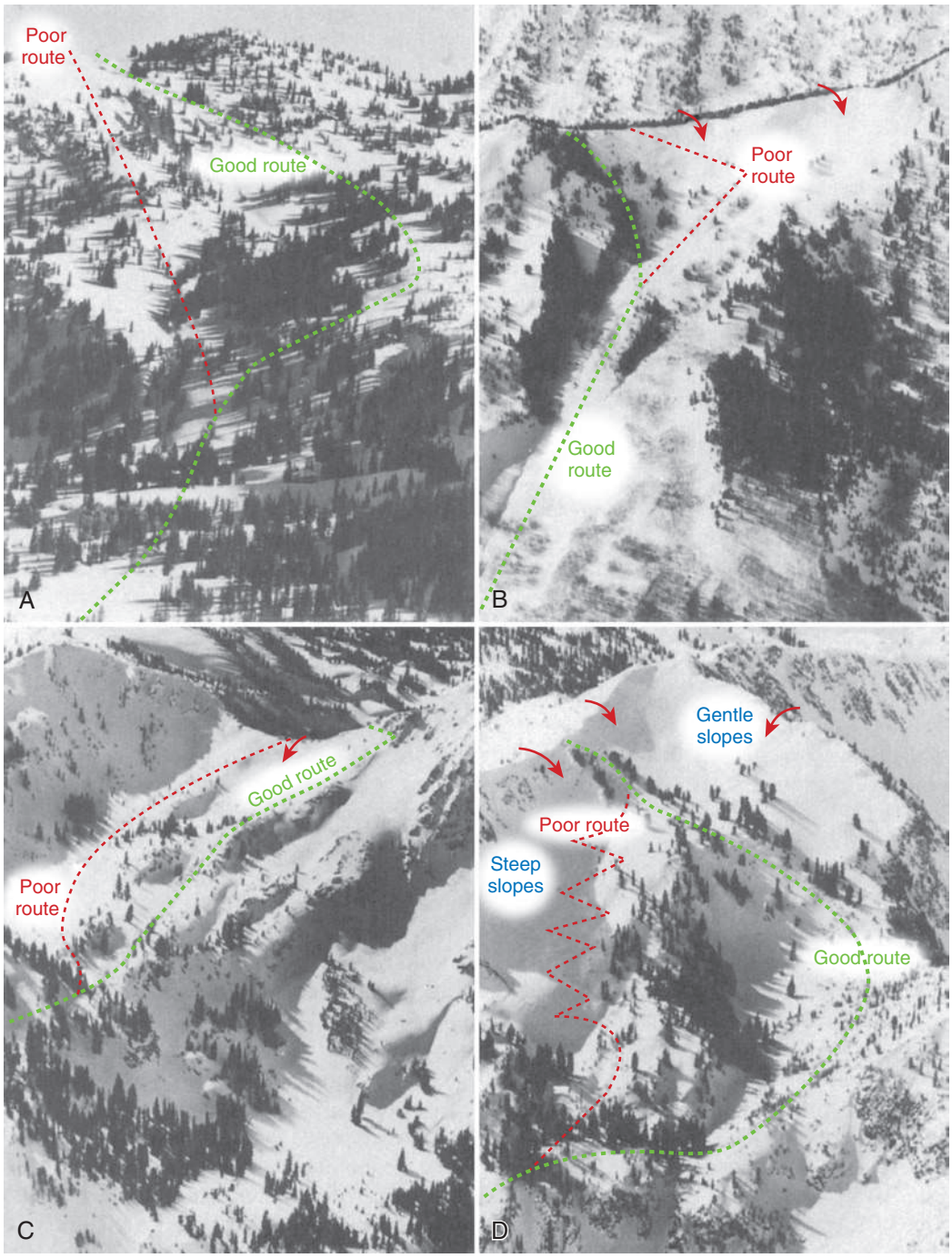



**FIGURE 4-16** Four ski-touring areas showing the safer routes *(green dashed lines)* and the more hazardous routes *(red dashed lines). Arrows* indicate areas of wind loading. *(From US Department of Agriculture Forest Service:* Avalanche handbook, *Agricultural Handbook 489, Washington, DC, 1976, US Government Printing Office, with permission. Courtesy Alexis Kelner.)*

these can sometimes be felt as resistance by pulling out the pole slowly along the side of the hole. This test reveals only the gross layers within the reach of the pole. Thin weak layers, such as buried surface hoar or a poor bond between any two layers, cannot be detected. Although of limited value, the ski pole test can provide useful information that must then be correlated with other testing.

The simple but useful ski cut test reveals how a similar slope may react to the additional weight of a skier, snowboarder, or snowmobiler. On a small slope that is not too steep (and therefore probably not avalanche prone), the skier can try a ski cut by skiing along a shallow traverse and then setting the ski edges in a hard check. Any cracks or collapse noises indicate instability and that slopes of similar aspect and elevation, if steeper, would have probably avalanched. Ski cuts can be made on steeper suspect slopes by starting a diagonal straight path from an area of safety in a continuous cut to another planned area of safety on the other side of the slope. This can be a dangerous maneuver, but the momentum—even if an avalanche is initiated—increases the chances of escaping from the starting zone to a safe position. An experienced snowmobiler can perform a similar test by riding across the test slope.

A much better way to observe directly and test snowpack layers is to dig a hasty snow pit. In a spot as near as possible to a suspected avalanche slope, without putting the traveler at risk, a pit 120 to 150 cm (4 to 5 feet) deep and 90 cm (3 feet)

wide should be dug. With the shovel, the uphill wall is shaved until it is smooth and vertical. The layers of snow can now be observed and felt. The tester can see where the new snow touches the layer beneath, poke the pit wall with a finger to test hardness, and brush the pit wall with a brush to see which layers are soft and fall away, and which are hard and stay in place after being brushed. Buried surface hoar, faceted grains, graupel, and crusts are better appreciated; in shallow snowpacks, basal depth hoar can also be easily reached and evaluated.

The shovel *shear test* (ST) is a simple and quick test used to locate weak layers (especially very thin layers). However, its small sample size and subjective nature make it less reliable for determining stability of the snowpack. A column of snow is isolated from the vertical pit wall. Both the sides and the back of the column are cut with a saw (a shovel or a ski will suffice) so that the column is freestanding. The dimensions are a shovel's width on all sides. The tester inserts the shovel blade at the back of the column and gently pulls forward on the handle. A cohesive layer of the column will shear on a weak layer and make a clean break; the poorer the bond, the easier the shear. A five-point scale is used to rate the shear: (1) "very easy" (STV) if it breaks as the column is being cut or as the shovel is being inserted; (2) "easy" (STE) if a gentle pull on the shovel does the job; (3) "moderate" (STM) if a slightly stronger pry with the shovel is required; (4) "hard" (STH) if a solid tug is required; and (5) "no shear" (STN) if no shear failure is observed. Generally, "very easy" and "easy" shears indicate unconditionally weak snow, "moderate" means conditionally weak, and "hard" and "no shear" mean strong. The value of the ST is that it can find thin weak layers that are undetectable by any other method, even on flat areas. The ST's major shortcoming is that it is not a true test of stability, because it does not indicate the amount of weight or compression required to cause shear failure.

The *compression test* (CT) is an effective way to find weak layers near the surface (~1 m [3.3 feet]), and works well in soft, new snow. The test also gives a good indication of snow stability because it stresses the weak layer. The CT involves isolating a block similar to that created for the ST on sloping terrain. Using the shovel blade turned upside down and held on the top of the column, successive taps are made on the shovel: 10 taps from the wrist using the fingertips, 10 from the elbow using the fingertips, and finally 10 from the shoulder using the palm or the fist. The number of taps required to produce a shear is recorded. For example, a shear after 5 taps from the elbow would be recorded as CT15. Interpretation of the stability of the weak layer that is discovered is defined as "easy" (taps 1 through 10), "moderate" (taps 11 through 20), or "hard" (taps 21 through 30). The CT evaluates the ability of weak layers in the upper snowpack to fail in shear and can be performed quickly, thereby allowing for multiple assessments of different aspects and elevations during backcountry travel (Figure 4-17).

Most tests, including the ST and the CT, assess fracture initiation (i.e., additional force or stress needed to start fractures), but do not assess fracture propagation (i.e., propensity for fractures to spread through a weak layer in the snowpack). Without assessing for fracture propagation, tests may conceal the real problem layer in a weak snowpack. In a stable snowpack, a fracture may be easy to initiate, but no avalanche may result because the fracture does not propagate across the weak layer. In an unstable snowpack, a fracture may be easy or difficult to initiate. After it has been initiated, however, the fracture can propagate across the weak layer and result in an avalanche.

To better assess fracture initiation, propagation, and instability, many backcountry travelers and avalanche professionals use the *Rutschblock test* (RB). This test is calibrated to the skier's weight and to the stress that the skier would put on the snow. A snow pit is dug with a vertical uphill wall. The pit must be about 240 cm (8 feet) wide. By cutting into the pit wall, the skier isolates a block of snow that is approximately 210 cm (7 feet) wide (i.e., a ski length) and that goes back 120 cm (4 feet; i.e., a ski pole length) into the pit wall. Both the sides and the back are cut with a shovel or ski so that the block is freestanding. The test receives a score of RB1 if the snow fails while the individual is isolating the block. While wearing skis, the skier climbs around

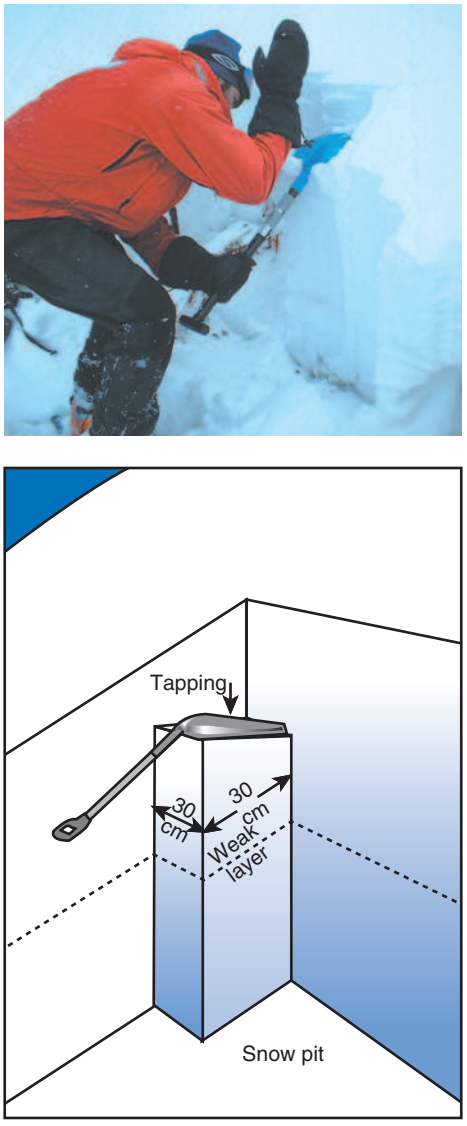



**FIGURE 4-17** Photo and schematic of a shovel compression test. *(From The American Avalanche Association, with permission.* Top, *Courtesy Doug Richmond.)*

and well uphill from the isolated block and carefully approaches it from above. With skis across the fall line, the skier gently steps onto the block, first with the downhill ski and then with the uphill ski, so that the person is standing on the isolated block of snow. If the slab of snow fails at this time, the score is RB2. Gently flexing the knees applies a little more pressure (RB3). The tester then jumps and lands in the same compacted spot (RB4). The jump is then repeated (RB5). If no failure occurs, the skier moves to the middle of the block and repeats the flexing and jumping (RB6). If no reasonably smooth failure is produced at this time, the test is considered an RB7.

The results are interpreted as "extremely unstable" if the block fails while the skier is cutting it, approaching it from above, or merely standing on it (RB1 or RB2); "unstable" if it fails with a knee flex or one gentle jump (RB3 or RB4); "moderately stable" if it fails after repeated jumps (RB5); and "very stable" if it never fails but merely crumbles (RB6 or RB7). These are somewhat objective results that may help answer the bigger question of whether the snowpack will fail under the weight of a person, and that may help the mountain traveler with risk evaluation. Limitations include the time required to isolate the block; inability to test deep, weak layers; spatial variability of the results; and the problem of relying on the RB as a one-step stability evaluation.

A more practical test developed during the late 2000s is the *extended column test* (ECT), which does a remarkable job of effectively discriminating between unstable and stable slopes by extending the size of the column beyond the size of the loading area. Similar to the loading of the CT, stress can be transmitted across the slab. If conditions are unstable, fracture will propagate across the column's weak layer. An excellent video tutorial can be viewed on the Utah Avalanche Center website at https://utahavalanchecenter.org/how-do-extended-column-test.

To prepare an ECT, a snow pit is dug with a vertical uphill wall and a column isolated that is 90 cm (3 feet) across the slope by 30 cm (1 foot) up the slope. The column is then loaded from one side with use of the same successive tap technique used in the CT. The result reporting emphasizes what happened to the snow column (i.e., whether a fracture propagated across the column or not). The coding is recorded as ECTPV ("very easy") if the fracture propagates across the entire column during isolation. As with the CT, the number of taps is reported as ECTP## when a fracture initiates and propagates across the entire column. If a fracture initiates on ## tap but does not propagate across the entire column, or if more than one additional tap is required after initiation to cause complete propagation, the result is noted as ECTN##. If no fracture occurs, the result is noted as ECTX.

The *propagation saw test,* developed and tested in Canada and Switzerland, has the advantage of being able to assess the propensity of a preidentified persistent weak layer and slab combination to propagate a fracture. Only a shovel and snow saw are needed, and the test's interpretation is relatively straightforward. A column of snow 30 cm (1 foot) wide by at least 100 cm (3.3 feet) uphill is isolated. After the persistent weak layer is identified and marked, the blunt edge of the saw is directed uphill through the weak layer until a fracture propagates. The spot where the saw was located at the time of the propagation is marked. Fracture propagation in a similar snowpack is considered "unlikely" if it requires sawing more than 50% of the weak layer to initiate propagation, or if the fracture fails to reach the end of the column. Propagation would be considered "likely" if the fracture reaches the end of the column, or if it requires sawing less than 50% of the weak layer to fracture. Unlike many other stability tests, the propagation saw test allows assessment of the slab propagation propensity in deep instabilities.

Interpreting stability from a single snow pit or stability test is dangerous because of spatial variability. Snow cover is not homogeneous in its stability; rather, it has a patchwork-quilt–like pattern of stronger and weaker snow. Even within a known combination of an unstable slab and a weak layer, there are variations along the slope where one can trigger or not trigger an avalanche (i.e., the false-stable rate). The implications of spatial variability are significant. On a suspected avalanche slope, explosives set in an area of strength will not result in an avalanche. It might then be assumed, incorrectly, that the snow in a ski area is stable and safe, only to have a skier hit a weak spot and trigger an avalanche. Likewise, a backcountry skier might dig a snow pit and find strong snow, only to trigger an avalanche on the entire slope after skiing a few turns.

All tests are prone to misleading results. Research has shown that the CT and RB tests, which are favored by most avalanche professionals, have a relatively high false-stable rate (i.e., a stable test result on an unstable slope) of about 10% to 20%.[4] On paper, being correct 80% to 90% of the time may sound encouraging. In practice, however, the false-stable rate is still too high, because the consequences of an error can be fatal. The false-stable rate of 3% with the ETC is more encouraging, but researchers caution that more studies are needed.[39]

## AVALANCHE RESCUE EQUIPMENT

### Shovel

The first piece of safety equipment that an individual entering avalanche terrain should bring is a shovel, which can be used to dig snow pits for stability evaluation, dig snow caves for overnight shelter, and rescue a buried partner. A shovel is mandatory for digging in avalanche debris because of the snow's high density. Such snow is extremely difficult to move with hands or skis.

The shovel should be sturdy and strong enough to dig in avalanche debris, yet light and small enough to fit into a pack. There is no excuse for not carrying a shovel. Collapsible shovels made of aluminum are available in mountaineering stores.

### Probe

Several pieces of equipment are designed specifically for finding buried avalanche victims. The first is a collapsible probe pole. Organized rescue teams keep rigid poles in 3- to 4-m (10- to 12-foot) lengths as part of their rescue caches. The recreationist can buy probe poles of tubular aluminum or carbon fiber that come in 45-cm (18-inch) sections that attach together quickly by pulling a stiffening cable to construct a full-length probe. Some ski poles with removable grips and baskets can be screwed together to make an avalanche probe. Probes are used to search for buried victims by spot probing in likely burial areas or to confirm transceiver findings before shoveling (see Small-Team Rescue [Companion Rescue], later).

### Avalanche Rescue Transceiver

Avalanche rescue transceivers (beacons) are one of the best personal rescue devices for quickly finding buried companions. With practice and proper use, transceivers are a fast and effective way to locate buried victims. Worldwide, these devices have become standard gear for ski area patrollers involved in avalanche work and for helicopter or other mechanized skiing guides and clients. Transceivers are also frequently used by highway departments and search and rescue organizations and should be used by anyone traveling into avalanche terrain. Since transceivers were first introduced in the United States in 1971, they have been credited with saving multiple lives each winter.

Transceivers act as electromagnetic transmitters that emit a signal on a standard frequency of 457 kHz. A buried victim's unit emits this repetitive signal in radial "flux lines," which the rescuers' units receive and analyze when switched to "receive" mode. The signal carries approximately 30 to 50 m (100 to 150 feet). When audibly or visibly detected, the signal can guide searchers specifically to the buried unit in less than 5 minutes.

Transceiver technology has evolved dramatically. Current transceivers offer a significant technologic advantage over the original analog devices. Two major categories of transceivers can be found: (1) analog, which processes the receiving signal in a basic electromagnetic convergence of induction pulses to allow for a stronger (visual and audible) signal as the receiving transceiver approaches the sending unit, and (2) digital, which uses a computer chip to microprocess the receiving analog signal in order to display a digital readout of the distance, strength (audible and visual), and in some units (i.e., dual and triple antenna), general direction to the buried unit. The newer triple-antenna transceivers not only provide data about distance, direction, and signal strength, but also can more easily identify multiple burials and their approximate location in relation to each other. The triple-antenna transceivers reduce occasional misleading signals, which can be a confusing issue with single-antenna and dual-antenna devices in the final few meters of the search. Transceivers are audiomagnetic-induction devices and not radio devices; the directional arrows point radially along a flux line of the sending unit's magnetic field (Figure 4-18). Likewise, the displayed distance represents the distance along the flux line rather than a direct distance to the sending unit; the closer the distance displayed, the closer the flux line from the sending unit.

Both analog and digital transceivers operate on the same internationally standardized frequency and are compatible with one another. However, slightly different search techniques may be necessary to use each type most efficiently, and special training and practice are required before the user attains proficiency. Experience has shown that digital units have a significantly faster learning curve; these are routinely used by helicopter skiing services and backcountry guides for clients with limited experience. Multiantenna digital transceivers have largely replaced pure analog units because of their relative ease of use and enhanced information regarding burial location.

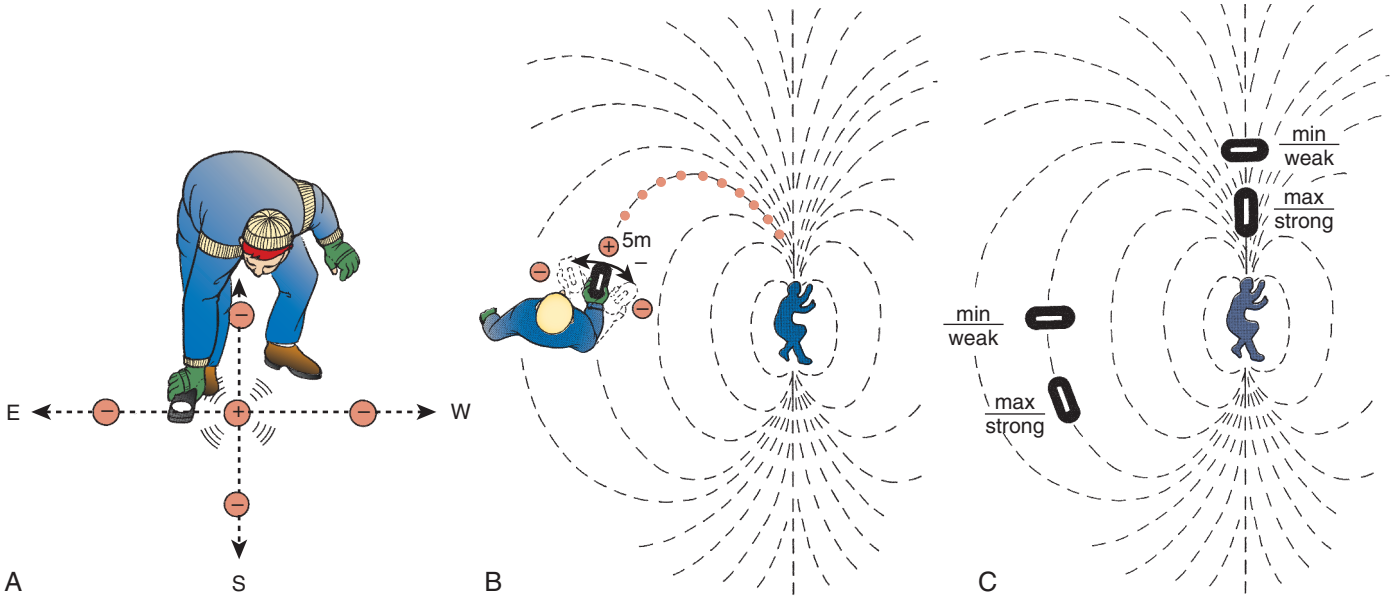



**FIGURE 4-18** Induction ("tangent") line search method. **A,** Arrangement of the electromagnetic flux lines (induction lines) emitted from a buried victim. The signal received by the searching transceiver along the transmitted flux line is strongest when oriented in parallel and is weakest when oriented perpendicularly. **B,** The searcher moves in short (3 to 5 m [9.8 to 16.4 feet]) "tangents" and then orients the transceiver to the strongest signal. In this way, the receiving transceiver follows a flux line toward the victim. The sensitivity (loudness) of the beacon should be adjusted downward as the victim is approached so that the searcher can discern the strongest signal before proceeding in a new direction. **C,** The "pinpoint" search is performed when the buried person is within 3 m (9.8 feet); this typically occurs when the transceiver is at its loudest with the sensitivity turned all the way down. It is a "grid" search on a much smaller scale that is carried out close to the snow surface. The loudest signal is found along one axis (E to W) and followed by the perpendicular axis (N to S) to the likely burial position. A probe is then used to confirm the victim's location and depth. *(From Auerbach PS, Constance BB, Freer L, et al.:* Field guide to wilderness medicine, *4th ed, Philadelphia: Elsevier; 2013.)*



Merely possessing a transceiver does not ensure its lifesaving capability. Experience shows that the chances of surviving full avalanche burial with a transceiver and small-team (companion) rescue are reported to be 27% to 45%.[1,10,26] Frequent practice is required to master a transceiver-guided search, which may not be as straightforward as the directions suggest. In this regard, "beacon parks" with automated practice burials have become a popular method for fine-tuning one's ability. Skilled practitioners can typically find a buried unit in less than 5 minutes once a signal is achieved. Because speed is essential in avalanche rescue, transceivers can certainly be lifesavers. Therefore, the minimal rescue equipment required for a rapid small-team (companion) recovery is a transceiver, a collapsible probe or pole to confirm and pinpoint the suspected location of the victim, and a shovel for extrication (Box 4-1 and Figure 4-18).

## Avalanche Airbag

The primary purpose of an avalanche airbag device is to prevent complete burial. German industrialist Peter Aschauer introduced the backpack-integrated avalanche airbag system (ABS) in 1985 (Figure 4-19). It was designed specifically for guides and ski patrollers but rapidly became popular in Europe for use by recreationalists venturing into avalanche terrain. Accident data involving avalanche airbag devices[38] have been compiled and analyzed since 1990 by the Swiss Federal Institute for Snow and Avalanche Research in Davos, Switzerland. From 1990 to 2010, 295 avalanche airbag–equipped persons were caught in avalanches; 255 victims survived and seven victims died with deployed airbags (two deaths caused by full burial from secondary avalanches, three deaths from full burial in large avalanches, and two deaths from trauma). In the other 33 victims, the airbags failed to work properly, or the user did not or could not deploy the airbag; of these, 10 died. Overall, of 295 persons caught in an avalanche wearing an avalanche airbag device, 17 deaths occurred (94% survival). However, it must be noted that in 67 persons involved in the same accidents who were *not* wearing an airbag, 17 died (75% survival) from complete burial. From this initial information, avalanche airbags provide some survival benefit. However, the exact increase in survival benefit imparted by the use of an avalanche airbag device is uncertain.

Early reports found that avalanche airbag devices reduced the likelihood of critical burial from 39% to 16.2% and lowered mortality from 23% to 2.5%.[11] A "critical burial" is defined as a partial or complete avalanche burial where the victim is at risk for asphyxiation. Among all persons caught in an avalanche, early

**FIGURE 4-19** The German 170-L (5.6-foot$^3$) dual-airbag system (ABS). This device utilizes two wing-like 85-L (3-foot$^3$) balloons inflated by pulling a T-shaped ignition handle, thereby rapidly releasing nitrogen from a canister that draws air into the balloons by a Venturi effect. Although this configuration favors a final prone position close to the surface of the debris, possibly compromising the airway, the manufacturers suggest that the bilateral and posterior position of the balloons affords horizontal stability in the laminar flow of the avalanche.

## BOX 4-1 Avalanche Transceiver Search

### Initial Search

1. Safely access the slide path and debris area, and have everyone switch their transceivers to "receive" and turn the volume to "high."
2. If enough people are available, post a lookout to warn others about possible additional slides.
3. Should a second slide occur, have rescuers immediately switch their transceivers to "transmit."
4. Have rescuers space themselves no more than 30 m (100 feet) apart and walk abreast along the slope.
5. If a single rescuer is searching within a wide path, he or she should zigzag across the rescue zone and limit the distance between crossings to 30 m (100 feet).
6. For multiple victims, when a signal is picked up, have one or two rescuers continue to focus on that victim while the remainder of the group carries out the search for additional victims.
7. For a single victim, when a signal is picked up, have one or two rescuers continue to locate the victim while the remainder of the group prepares shovels, probes, and medical supplies for the rescue.

### Locating the Victim

With practice, the induction line search is more efficient than the conventional grid search for getting close to the sending beacon, but the conventional grid search is still necessary to fine-search for the buried victim. A probe pole can be used to pinpoint the victim's exact location. The induction line search is very similar to the flux line search that is used by digital transceivers. Users should always study the owner's manual to learn the best techniques for the specific brand of avalanche rescue beacon that they are using.

#### *Induction Line Search (Preferred Method)*

When an induction line search is used, the rescuer may initially follow a line that leads away from the victim (see Figure 4-18A,B). Remember to lower the transceiver volume if it is too loud, because the ear detects signal strength variations better at lower volume settings.

1. After picking up a signal during the initial search, hold the transceiver horizontally (parallel with the ground), with the front of the transceiver pointing forward (see Figure 4-18C).
2. Holding the transceiver in this position, turn until the signal is maximal (maximum volume), then walk five to seven steps (~5 m [16.4 feet]), stop, and turn again to locate the maximum signal (see Figure 4-18). When locating the maximum signal, do not turn yourself (or the transceiver) more than 90 degrees in either direction. If you rotate more than 90 degrees to locate the maximum signal, you will become turned around and will follow the induction line in the reverse direction.
3. Walk another five steps, as just described, and then stop and orient the transceiver toward the maximum signal. Reduce the volume.
4. Continue repeating these steps. You should be walking in a curved path along the induction line toward the victim (see Figure 4-18B).
5. When the signal is loud at the minimum volume setting, you should be very close to the victim and can begin the grid search.

#### *Grid Search*

1. When a signal is picked up, stand and rotate the transceiver, which is held horizontally (parallel with the ground) to obtain the maximum signal (loudest volume). Maintain the transceiver in this orientation during the remainder of the search.
2. Turn the volume control down until you can just hear the signal. Walk in a straight line, down the fall line from where the signal was first detected. (If the signal fades immediately, walk up the fall line.) If you are headed in the right direction, the signal will increase; turn the volume control down until the signal fades. Take an extra couple of steps to be sure that the signal truly fades. If the signal increases, continue until it fades.
3. When the signal fades, mark the point, and then turn 180 degrees and walk back toward the starting position. The signal will increase in volume and then fade again; take two additional steps to confirm the fade. Walk back to the middle of the two fade points; this spot may or may not be the point of loudest volume and maximum signal. If you experience two maximum signals, go to the midpoint between the two maximums.
4. At this point, turn 90 degrees in one direction or the other. From that position, reorient the transceiver (held parallel with the ground) to locate the maximum signal. After orienting the transceiver to the maximum signal, reduce the volume, and begin walking forward. If the signal fades, turn around 180 degrees, and begin walking again.
5. As the signal volume increases, repeat steps 3 and 4 until you have reached the lowest volume control setting on the transceiver. (Be sure always to take an extra step or two to confirm the fade point.) This time, when you return to the middle of the fade points, you should be very close to the buried victim and can now begin pinpointing the person.
   a. While stationary, orient the transceiver to receive the maximum signal (loudest volume). At this point, turn the volume control all the way down.
   b. With the transceiver just above the surface of the snow, continue doing the grid search pattern two to four more times. Always sweep the transceiver a couple of feet beyond the fade point to confirm the fade point.
   c. Find the signal position halfway between the fade points (i.e., at the loudest signal). At this point, you should be very close to the victim's position, and you can begin to mechanically probe. Speed is essential. With practice, the transceiver will be accurate to less than one-fourth the burial depth. For example, a 4-foot burial should result in about a 1-foot square at the surface.
   d. Depending on the brand, pinpointing with a digital transceiver will involve the use of a slight variation or a combination of the induction line and grid techniques. Be sure to study the owner's manual.

studies reported that avalanche airbags reduced mortality from 18.9% without an airbag to 2.9% with an airbag.[10] This apparent dramatic reduction in mortality presumably occurs because the avalanche airbag device effectively prevents complete or critical burial, and very few (~3% to 5%) noncritically buried or unburied victims die in avalanches.

Recent examination of the effectiveness of avalanche airbag devices[24] used a retrospective, multivariate regression analysis on a comprehensive avalanche accident data set from Europe, Canada, and the United States that included avalanches from 1994 to 2012 with the potential for mortality. Each accident in the analysis involved at least one airbag user compared with either nondeployed-airbag users or nonusers from the same incident. The multivariate analysis for critical burial and mortality after protocol exclusions included 189 seriously involved individuals from 61 accidents. The adjusted "risk of critical burial" was 47% with noninflated airbags or no airbag compared to 20% with inflated airbags. The adjusted mortality was 44% for critical burial and 3% for noncritically buried victims. Finally, the adjusted mortality reduction with an inflated airbag dropped from 22% without use of the bag to 11% (i.e., mortality was reduced by half with inflated airbags). Interestingly, overall noninflation rate was 20%, the majority resulting from deployment failure by the user. These findings, despite inherent limitations of the heterogeneous data set and the retrospective nature, support that avalanche airbags are a valuable safety device, although possibly of less impact than previously reported.

The airbag works as a result of *granular convection* (the "Brazil nut effect," similar to shaking a large bowl of mixed nuts to have the largest nuts rise to the top) (Figure 4-20). An avalanche in motion is composed of many different-sized particles of snow and other objects moving in layers of granular flow. When these particles and objects begin to segregate, smaller particles sink to the lower portion of the flow and larger particles rise to the surface. The process of granular convection depends primarily on the relative sizes of the particles within this granular

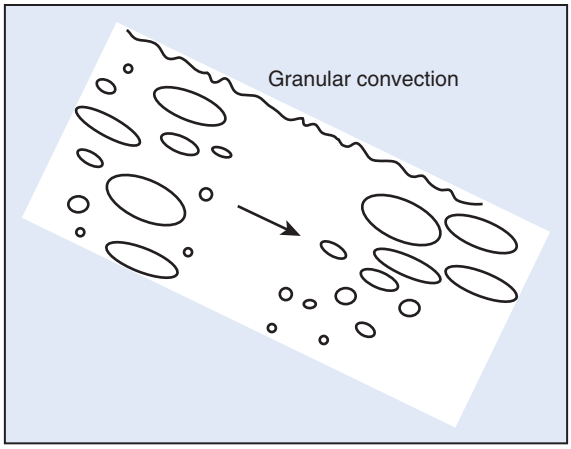



**FIGURE 4-20** Granular convection. The physical unmixing of flowing particles is based on size, which distributes larger particles in the upper layers and forms the basis for successful functioning of avalanche airbag systems. This is similar to the "Brazil nut effect," where shaking a bowl of mixed nuts causes the largest nuts to rise to the top.


**FIGURE 4-21** The JetForce Avalanche Airbag Pack (Black Diamond Equipment) uses a battery-powered fan instead of a compressed gas cylinder. The fan inflates a 200-L (6.9-foot$^3$) airbag in 4 seconds, then automatically deflates the bag after 3 minutes. This system can be deployed, repacked, and ready for use without having to refill a compressed gas cylinder.



flow. A person who is carried in an avalanche may be a medium or small particle compared with a particulate slab and therefore may be buried under the surface. However, the airbag device creates a greater surface area (typically a total of 150 to 170 L [5.2 to 6.0 feet$^3$] when inflated) for the avalanche victim, making the user a large enough particle to allow for greater separation effect toward the surface, thus reducing the risk of full burial. Buoyancy plays no role in the efficiency of this system. In addition, during partial burial, the brightly colored balloons may be easily visible on the surface, making a transceiver search unnecessary.

Since 1995, avalanche airbag devices have undergone significant improvements with regard to device size, balloon technology, and cartridge weight. Avalanche airbags are available from four companies: ABS (with partners Osprey, Dakine, Ortovox, and The North Face), Snowpulse/Mammut, Backcountry Access, and Black Diamond Equipment. The airbag is usually integrated into a special backpack, and the user deploys it by pulling a ripcord-like handle. This action releases a cartridge of compressed air or nitrogen that escapes at high velocity and draws in outside air.

Different avalanche airbag device configurations include a pack with two separate airbags, a pack with a single airbag, and a vest with a single airbag. Configurations are two approximately 75- to 85-L (2.6- to 3.0-foot$^3$) airbags or one 150-L (5.2-foot$^3$) airbag. These inflate in 2 to 3 seconds with use of the Venturi effect. In 2014, one manufacturer released a 200-L (6.9-foot$^3$) airbag built into a pack that inflates in 4 seconds using an electric-powered fan instead of a compressed-gas cylinder (JetForce Avalanche Airbag Pack, Black Diamond Equipment) (Figure 4-21).

Avalanche airbags may deploy as a large bag or in a horseshoe shape around the back of the victim's head toward the anterior chest. Avalanche airbag systems that cover the back of the neck and head or surround the back and sides of the head and anterior chest (Figure 4-22) have the theoretical advantage of preventing head trauma and may allow for a greater chance of a head-up partial burial. However, there is no evidence to support prevention of head or chest trauma with specific avalanche airbag system configurations. This is an area for future study.

Unlike the original avalanche airbag system (ABS), none of the new systems has been independently tested by the Swiss Federal Institute for Snow and Avalanche Research or have a

**FIGURE 4-22** Example of an airbag system that surrounds the head and anterior chest when deployed. This configuration, unlike the ABS, seems to favor a head-up, airway-favorable final position and may afford some theoretical protection to the head, neck, and chest. Critics of this balloon design suggest that travel in the avalanche might be more vertical, increasing the chances of lower-extremity trauma and a possible "straining effect" from terrain features that would disrupt the process of granular convection.


**FIGURE 4-23** The AvaLung is a breathing device worn over all other clothing that is intended to prolong survival during avalanche burial by diverting expired air away from inspired air drawn from the snowpack. The *white arrows* show the flow of inspiratory air; the *red arrows* show the flow of expiratory air. The person breathes in and out through the mouthpiece (*A*). Inhaled air enters from the snowpack through the one-way inspiratory valve on the side of the housing inside the mesh-protected harness on the chest (*B*). Expired air leaves the lungs via the mouthpiece and travels down the respiratory tubing to the housing and then passes through an expiratory one-way valve located at the bottom of the housing (*B*). It then travels via respiratory tubing inside the harness around to the back (*C*). *(Courtesy Jill Rhead and Black Diamond Equipment, Salt Lake City, Utah.)*

documented history of use in avalanche accidents. Questions remain about the different-shaped balloons on the market regarding ease of deployment, efficacy in preventing critical burial, and protection from head trauma. Only the German ABS has been rigorously tested and has undergone extensive use in avalanche accidents. The new airbag designs are too recent to make any sound recommendations at this time.

### AvaLung

In 1996, after testing a homemade prototype on himself, Dr. Thomas Crowley received a patent for an emergency breathing device to extract air from the snow surrounding a buried avalanche victim. Black Diamond Equipment in Salt Lake City, Utah, performed research and development on the device and secured distribution rights in 2000 for the original external vest format of the AvaLung. The company redesigned the device in 2002 for easier and more comfortable use when worn outside of the clothing like a bandoleer (Figure 4-23). The most recent version, which is the most popular, is incorporated into various-sized backpacks with a stowable mouthpiece kept in the shoulder strap (Figure 4-24).

The AvaLung functions mainly by separating inhaled from exhaled air in the surrounding burial snowpack, with a goal of prolonging survival time by slowing the process of asphyxiation. Ambient air is 21% oxygen ($O_2$) and has virtually no carbon dioxide ($CO_2$). Expired air is 16% $O_2$ and 5% $CO_2$. The AvaLung prevents rebreathing expired air, which is the major cause of asphyxiation during avalanche burial, by diverting expired air away from inspired air that is drawn from the snowpack[20] (see Avalanche Victim Physiology and Medical Treatment after Rescue, later). Survival after complete avalanche burial with use of the AvaLung has occurred in a number of cases.[34]

The AvaLung system has proved effective in numerous simulated burials by allowing persons to breathe for 1 hour in tightly packed snow of similar density to avalanche debris. In control burials without the AvaLung, participants were only able to breathe from 4 to 19 minutes using a 500-mL (30.5-inch$^3$) air pocket.[20,35] In addition, several cases of survival have occurred, although burial time was fairly short. In a dramatic hallmark case, a helicopter skier survived a 120-cm (4-foot) burial of 35 to 45


**FIGURE 4-24** The AvaLung backpack. A stowable mouthpiece is located in the shoulder strap along with the inspiratory and expiratory valve housing. Inspiratory air enters through a one-way valve in the valve housing located in the shoulder strap and flows through respiratory tubing and the mouthpiece into the lungs. Expiratory air flows through the mouthpiece into the shoulder strap and through a second one-way expiratory valve. Expired air then travels through the respiratory tubing and through the pack to the lower back on the opposite side, where expiratory air is expelled. *(Courtesy Black Diamond Equipment, Salt Lake City, Utah.)*

minutes by breathing with an AvaLung. Two other skiers buried in the same avalanche a few feet apart at a similar depth were not wearing the device, and both died of asphyxiation. Rescue times were similar for the three victims, none of whom had suffered any trauma.[13]

The ABS and AvaLung are designed as adjuncts to the basic companion (small-team) rescue equipment, which includes a transceiver, probe, and shovel. These devices should never be used to justify taking additional risks. Surviving any avalanche is always uncertain, and no equipment should ever replace sound judgment.

# AVALANCHE RESCUE

## INDIVIDUAL RESCUE (SELF-RESCUE)

### Escaping to the Side

During the moment that the snow around a person begins to move, there is a split second during which that person can potentially move off the avalanche to more stable snow (see Figure 4-13). Escaping to the side of an avalanche is unlikely, however, and should not be considered a viable rescue or safety plan. If the skier happens to be next to a tree when an avalanche begins, holding onto the tree may help prevent the person from being carried, but the force of an avalanche usually carries the person. As such, skiing in an area with trees should not be used as a prevention strategy. Trying to outrun an avalanche by turning the skis or the snow machine downhill is impractical and dangerous; an avalanche invariably overtakes its victims.

If caught, a victim should shout "Avalanche!" to alert companions, then close the mouth and breathe through the nose to prevent inhaling a mouthful of snow. If the individual is wearing an AvaLung, the mouthpiece should be quickly placed in the mouth, which also helps to prevent oropharyngeal snow impaction. If the person is wearing an ABS, the ripcord should be pulled and the airbag activated.

### Actions during the Slide

For years, it was taught to "swim to the surface" (i.e., struggling with the arms and legs flailing) if caught in an avalanche. The idea is that the victim may be able to climb to the surface as well as increase the apparent surface area. The efficacy of these actions has been questioned. The process of granular convection (see Avalanche Airbag, earlier) rather than swimming may be more important in keeping the victim on or near the surface. Nevertheless, many survivors claim that "swimming" kept them on or close to the surface. If the victim sees or feels an object, such as a tree or rock band, the person should make every attempt to grab and hang on, allowing some of the avalanche forces to bypass.

Skiers should try to discard their skis and poles. With any luck, the avalanche will strip away the skis. Backpacks should be kept, if possible, because they make the victim a slightly larger particle for granular convection and increase the likelihood that the person will be closer to the surface. Packs might offer some protection to the thoracic spine. If the victim ends up on the surface, the equipment will be useful. However, the equipment's fate will usually be decided by the avalanche. Packs slide off easily when a person is turned upside down, even with tight straps. Snowmobile riders should try to stay on their machine. Once separated from the machine, avalanche victims are twice as likely to be buried than if they had stayed with the machine.

Visual clues are the quickest method to find a buried avalanche victim. If the victim senses that he is close to the surface, he should thrust a hand or a foot toward that direction. Any visual clue on the surface will give rescuers a possible indication of location and greatly improve odds of survival.

Creating an air pocket is a key to survival. Without an air pocket, the time to asphyxiation is much more rapid. If the victim is not close to the surface or the position is unknown, the person should place one or both hands in front of the face to create a space, or air pocket, when the avalanche is slowing. This action should be performed early because the avalanche may stop abruptly, at which time no movement will be possible.

## SMALL-TEAM RESCUE (COMPANION RESCUE)

All backcountry users should carry appropriate avalanche rescue equipment to aid a fellow group member or another group. This includes a transceiver, shovel, and probe, as well as knowledge of and practice using this equipment. At resorts, ski patrol members monitor the avalanche danger and attempt to trigger sensitive avalanches. Therefore, avalanches are rare in these areas. However, avalanche management is not a perfect science, and skiers have become caught, injured, and even killed at ski resorts. Therefore, some skiers wear avalanche transceivers and carry avalanche rescue gear inside the ski resort for increased safety. Basic medical skills to care for and manage a partner who has been buried in an avalanche are equally important. Every backcountry user should attend a cardiopulmonary resuscitation (CPR) course and wilderness-oriented first-aid course.

### Calling for Help

If the accident site may be within the range of cell phones or other communication methods, a call or text should be attempted immediately. Search and rescue teams as well as medical assistance can be summoned and can be en route during on-site search for the victim. If team members are certain that no cell reception or other communication is possible and that travel to communications is more than 30 minutes away, all should remain on-site for the search and rescue. If the accident occurs in or near a ski area and there are several companions, one person can be assigned to leave the scene and immediately notify the ski patrol.

### Marking the Last-Seen Area

A companion or eyewitness to an accident needs to act quickly and positively. Rescuer actions over the next several minutes may mean the difference between life and death for the victim. Companions must assess the terrain to ensure that they and other rescuers are safe from secondary slides. The bed surface of an avalanche that has recently run is usually safe to enter. Occasionally, however, an avalanche fracture line has broken at midslope, which leaves a large mass of snow, sometimes called "hang fire," still positioned above the fracture that could avalanche onto the rescuers.

First, the victim's last-seen area should be fixed and marked with a piece of equipment or clothing, tree branch, or any item that can be seen from a distance downslope. If the victim was seen riding the avalanche before the last-seen area, a line of trajectory can be visualized that could narrow down likely areas of burial. Persons equipped with transceivers should travel to the last-seen area to begin their search (see Probing after Transceiver Search, later). Victims who are fully buried without transceivers likely face an unsurvivable situation.

## INITIATE SEARCH AND SCAN FOR CLUES

All companions must immediately switch their units to receive mode. At the same time as initiating this transceiver search, the fall line (i.e., the line of trajectory) should be quickly scanned below the last-seen area in the flow line for any clues of the victim. A glove, ski pole, or other object could lead rescuers directly to the victim. If an obvious clue is seen, shallow probing should be done into likely burial spots with an avalanche probe (i.e., spot probing). All backcountry users should carry a proper avalanche probe. A ski, ski pole, or tree limb could be used to search if necessary or in a ski resort, where probes may not be carried. Likely spots are the uphill and downhill sides of trees and rocks and benches or bends in the slope where snow avalanche debris piles up. The toe of the debris should be searched thoroughly because many victims are found in this area.

### Rescue Transceivers

While making the quick visual search for clues, companions should search the debris using their transceivers, as described in Box 4-1 and Figure 4-18. When a signal is detected, companions must narrow the search area quickly. If they are skilled with a transceiver, companions can pinpoint the burial site in a few minutes and should confirm the location with a probe.

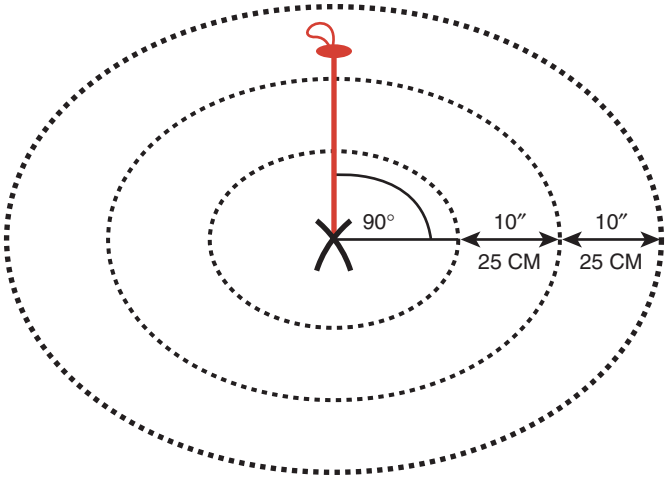



**FIGURE 4-25** Concentric probing method. Probing should proceed from inward to outward in concentric circles spaced 25 cm (10 inches) apart.

### Probing after Transceiver Search

When the victim's location has been pinpointed, probing should begin. The probe should be placed perpendicular to the slope at the location of the highest transceiver signal and pushed deeply, usually 2 to 3 m (6.6 to 10 feet; the majority of probe lengths). If this does not strike the victim, the probe should be removed and probing continued in a spiral or concentric-circle pattern until the victim is contacted (Figure 4-25). When the victim is located by a probe strike, the probe should be left in place and shoveling should begin.

### Shoveling Techniques

Shoveling will, unfortunately, take much longer than the transceiver search. Depending on the number of rescuers and technique used, this aspect of the rescue could be the difference between life and death. Efficient shoveling techniques can make this process much quicker, increasing the chances of survival.

**Strategic Shoveling.** During companion rescue, only one to three shovelers might be available. The strategic shoveling technique[14] increases digging efficiency (Figure 4-26). With the probe left in place, shovelers begin digging downslope about 1½ times the burial depth, as determined by markings on the probe. Stand away from the probe, and do not stand above the buried victim, if at all possible. Quickly dig a waist-deep starter hole about one wingspan wide (i.e., the distance between the fingertips when the arms are held out to the sides). If two shovelers are digging, they should work in tandem and side by side rather than one digging behind the other. Throw the snow to the sides. Move to the starter hole, and continue digging downward and forward. As depth increases, snow can be cleared to the back rather than lifted and tossed to the sides.

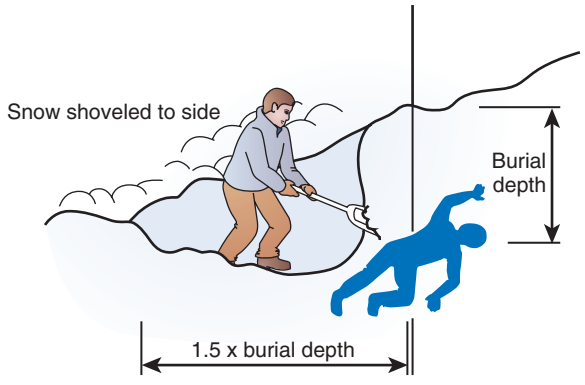



**FIGURE 4-26** Strategic shoveling technique for one or two rescuers. *(Courtesy Dale Atkins and National Ski Patrol, Lakewood, Colo.)*

When close to the victim, use a scraping action to clear snow. Use the first body part to estimate the location of the head, then use the hands to clear away snow from the victim's face and airway, and continue to clear snow off the chest. The most important feature of efficient shoveling is to create a ramp or platform that leads to the probe (and the victim) instead of digging a hole straight down around the probe. Extrication and resuscitation of the victim are made easier by creating a flat surface with space to work on the victim, because the air pocket is not compromised and raising the victim is not necessary.

**V-Shaped Conveyor Belt.** With a professional (organized) rescue, when hours or days have elapsed, the debris is often much harder as a result of age hardening. Typically, more shovelers are available in a professional or organized rescue. In this situation, the V-shaped conveyor belt method[16] works effectively to clear snow quickly (Figure 4-27). Starting downslope from the probe, rescuers are arranged in a wedge-shape or inverted-V pattern. The lead shoveler chops out blocks of snow and scoops the snow downslope. The other shovelers use paddling-like motions to clear out snow through the center of the V to create a platform. When nearing the buried victim, an additional shoveler may join the lead to increase the working space. Shovelers may rotate clockwise every 5 minutes to decrease fatigue. After they reach the victim, the rescuers should locate the head and chest and use their hands to clear a space for the airway.

Avalanche rescue courses should teach efficient shoveling techniques, and the method should be practiced as often as transceiver searches to reduce the total time to extrication.

### Calling for Professional Resources

A cell phone or other communication method should have already been used to alert outside agencies. If communication could not be made, on-site rescuers often face a difficult question of when to leave the scene to summon outside help. If only one or two companions are present, the correct choice is more difficult. The best advice is to search the surface quickly but thoroughly for clues before anyone leaves to notify the patrol or obtain outside assistance.

If the avalanche occurs in the backcountry far from any professional rescue team (organized rescue), all companions should remain at the site. The guiding principle in backcountry rescues is that companions search until they cannot or should not continue. When deciding when to stop searching, safety of companions must be weighed against the decreasing survival chances of the buried victim. If a small team (companions) rescues a team member who has been determined to be dead, the team should attempt to stay on scene until a professional (organized) rescue team arrives or other appropriate arrangements are made. This allows professional rescue teams and medical examiners to assess the situation and assist in evacuation.

## PROFESSIONAL RESCUE

### Incident Command System

In 2003, Presidential Homeland Security Directive 5 required that all emergencies be managed with the Incident Command System (ICS; visit www.fema.gov for more information). To meet this mandate, members of organized rescue teams should have an awareness of ICS. ICS only changes how incidents are managed; it does not change how avalanche rescuers do their job.

No matter how an incident is managed, all avalanche search and rescue operations have four key functional components, which are often being organized and managed simultaneously. One component is *search,* the goal of which is quickly finding and extricating any buried victims. The first team dispatched to the avalanche, which is known as the *immediate search team,* should consist of skilled and swift-traveling rescuers who are competent not only in avalanche rescue but also in route finding and hazard evaluation. Basic search and rescue tools are transceivers, probes, shovels, avalanche rescue dogs, the RECCO system (described later), and basic first-aid equipment. The immediate search team performs the initial search and looks for clues with the hope of making a quick find. If they have no success, the team determines the most likely burial areas. The

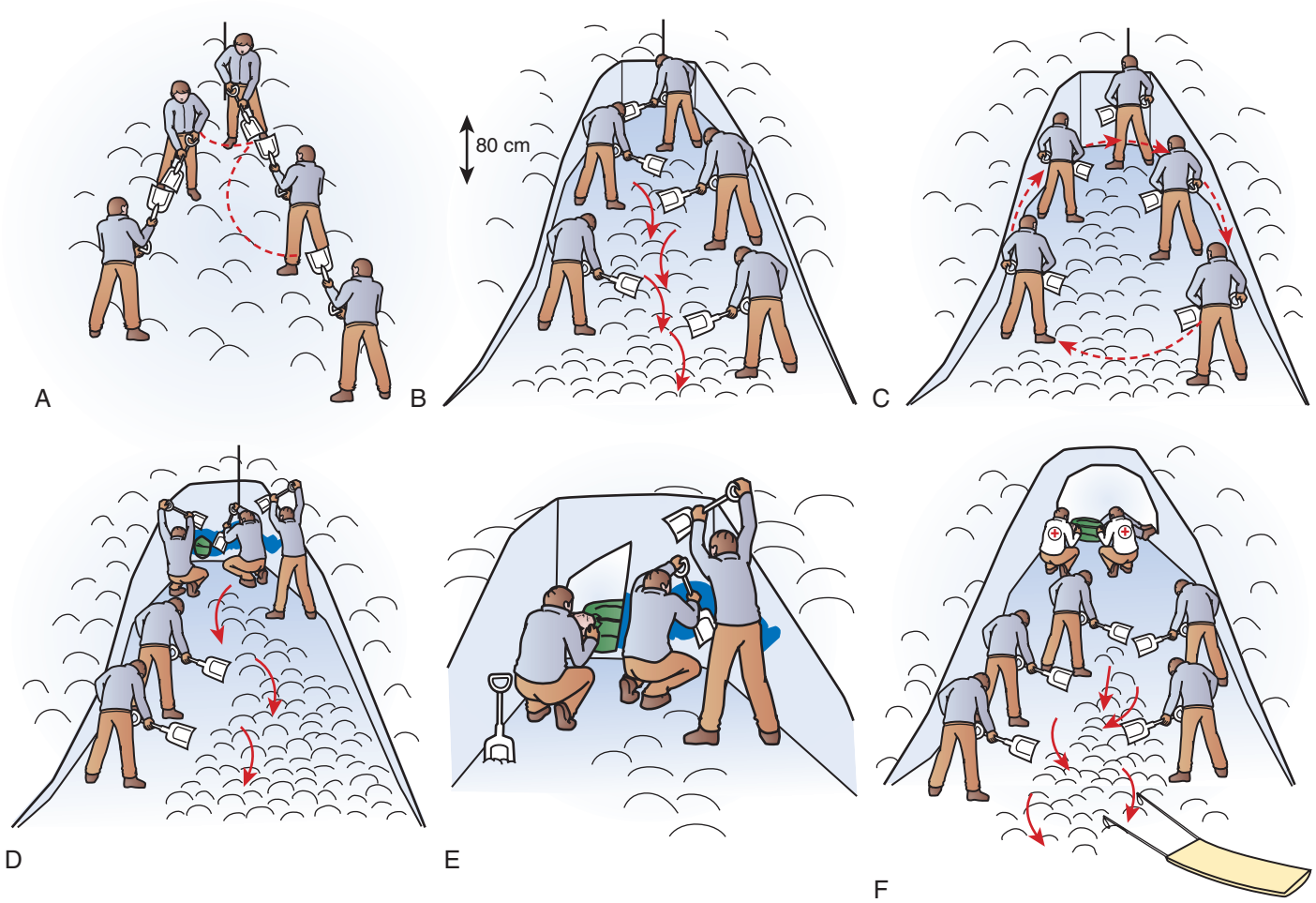



**FIGURE 4-27** V-shaped conveyor belt shoveling approach. **A,** Positioning of rescuers, with a quick measurement of the distance between shovelers. **B,** Working in sectors on the snow conveyor belt; snow is transported with paddling motions. **C,** Clockwise rotation is initiated by the front person; job rotation maintains a high level of motivation and minimizes early fatigue. **D,** The buried victim is first seen. More rescuers are needed at the front, and the snow conveyor belt only needs to be kept partially running. **E,** Careful work occurs near the buried victim, while some shovelers aggressively cut the side walls to adapt the tip of the V to the real position of the victim. **F,** Interface to organized rescue. More space is shoveled only after medical treatment of the victim has begun. *(Courtesy Manuel Genswein. From Genswein M, Eide R: V-shaped conveyor belt approach to snow transport,* Avalanche Rev *20:20, 2008, with permission.)*

person who is reporting the avalanche should meet rescuers at the accident site, or the reporting person should be returned to the same vantage point from which the accident was witnessed to best guide rescuers to locate last-seen areas. Arriving rescuers will meet the site leader and continue the immediate search until sufficient clues can steer rescuers to positioning probe lines.

The second component is *emergency care* to provide medical care for victims who are found. One or two small teams are dispatched with resuscitation, medical, and simple evacuation equipment. Ideally, the medical team should be sent within minutes of fielding the first search teams to ensure that necessary medical and evacuation equipment reach the site in a timely manner.

The third component is *transportation,* which includes travel for additional rescuers to and from the accident site and for evacuation of victims. The transportation effort must begin immediately so that rescuers and equipment can be moved efficiently and quickly. Likewise, coordination of helicopters, snow machines, and rescuers is essential to a fast and safe evacuation.

The fourth component is *support,* which sustains the entire operation, especially when a rescue is prolonged. This support may include additional rescuers (to take over for cold and tired searchers), hot food and drinks, tents, warm clothing, and lights for nighttime searching.

## Organized Probing Search Techniques

Organized probing is a simple but slow method of searching for buried victims. For more than 40 years, the traditional probe line (closed course probing) used by rescue teams was composed of about a dozen rescuers standing elbow to elbow with a probe pole that was 3 to 3.5 m (10 to 12 feet) long. The rescuers probe once between their feet, with each probe entering the snow about 75 cm (30 inches) from the neighbors on either side; the line then advances 70 cm (28 inches), and the rescuers probe again. Open course probing involves the rescuers being an arm's length apart and probing twice (once in front of each foot) before stepping forward. The probability of detection with these methods was thought to be about 70%. If the probe line missed the victim on the first pass, which tended to happen, the area was probed again and again. Behind the probe line, shovelers stood ready to check out any possible strike. The line did not stop in such an event but continued to march forward with a methodic "probe down, probe up, step forward" cadence.

The optimal combination of probe-grid spacing and search time, as determined by computer analysis, is a grid spacing of 50 cm by 50 cm (20 inches by 20 inches).[2] This probe-grid spacing yields the best combination of probability of detection and fast search time. For a three-holes-per-step probe, probers stand with their arms out, wrist to wrist. Probers first probe

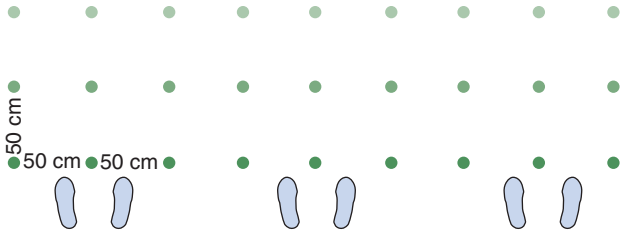



**FIGURE 4-28** Three-probe spacing for 50-cm by 50-cm, three-hole-per-step probe method.

between their feet, then probe 50 cm (20 inches) to the right and 50 cm (20 inches) to the left (Figure 4-28). At a command from the leader, the line advances 50 cm (20 inches; i.e., one step). This method gives an 88% chance of finding the victim on the first pass, with an estimated time to discovery almost identical to that of traditional spacing. Ski patrols and mountain rescue teams who have adopted the three-holes-per-step style of probe with a 50-cm by 50-cm (20-inch by 20-inch) grid have noted improved efficiency and effectiveness of the probe line.

Because these rescuers insert more probes per rescuer, fewer rescuers can be used on a probe line. Short probe lines are easier to manage and work faster than long probe lines. Five rescuers doing three holes per step at 50-cm intervals form a slightly longer probe line than nine rescuers using the traditional method. To ensure proper spacing, it is most helpful to use a guidon cord with marked 50-cm intervals. Suspended between two rescuers, the lightweight guidon cord positions rescuers and probes at the correct spots, thereby allowing the line to move smoothly and efficiently without interruption. When a guidon cord is not used, the probe line must be frequently stopped and reassessed to ensure proper spacing. If the victim is missed on the first pass, the second pass increases the probability of detection to 99%.

The third effective organized probe search is called the slalom probing technique (Figure 4-29).[17] In this method, organized rescuers probe three areas in a left-to-right pattern after two lateral steps, then step forward, and then probe three areas in a right-to-left pattern. This pattern is repeated for the debris field that must be searched. The main advantage of the slalom probing method is that all probing is performed directly in front of the rescuers, which has been determined to be stronger and more efficient than probing to the side, as required in the three-hole-per-step technique. Also, forward steps are more time-consuming and variable in distance than lateral steps. The main disadvantage is that it is surprisingly difficult to train novices to perform the slalom probing technique well.

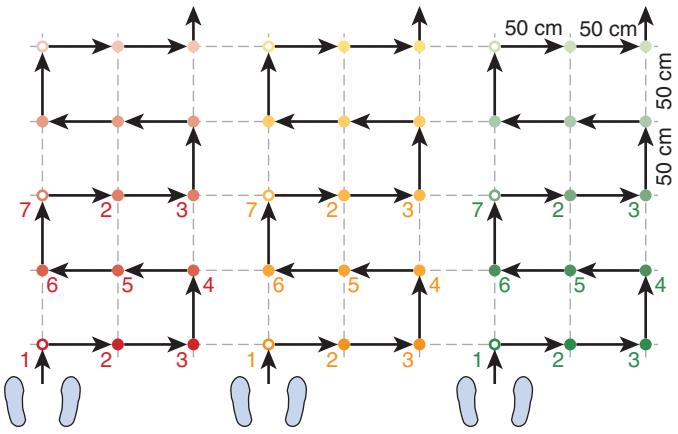



**FIGURE 4-29** Slalom probing technique. On direction by the incident commander, each rescuer probes in the sites indicated by color *(red, yellow, green)* in the rescuer's respective area. *(From Genswein M, Letang D, Jarry F, et al: Slalom probing—A survival chance–optimized probe line search strategy. Paper presented at the International Snow Science Workshop, 2014, Banff, Alberta, Canada.)*

Rescue teams use probe lines to find most avalanche victims who are not equipped with transceivers or RECCO reflectors, or when an avalanche rescue dog fails to locate the victim. However, these search methods should all be used concurrently. Because probe lines are time intensive, few victims are found alive using this technique alone (see Rescue Statistics, later).

### Avalanche Rescue Dogs

Trained search dogs, which are traditionally used in Europe early during avalanche rescues, are capable of locating buried victims very quickly. Since 1950, there have only been nine reported live recoveries using dogs. Six of these involved trained dogs: four at ski areas, one along a highway, and one in the backcountry. Three other avalanche burials involved personal dogs who found their owners in the backcountry. Burial depths of avalanche victims found alive ranged from 0.3 to 1.5 m (1 to 5 feet); burial depths of victims killed ranged up to 4.5 m (15 feet).[1]

The number of trained certified search dogs has increased substantially. Search and rescue teams and law-enforcement agencies work closely with search-dog handlers, and trained avalanche dogs are now common fixtures at ski areas in the western United States. A trained avalanche dog moving rapidly over avalanche debris and using its sensitive nose to scan for human scent (i.e., rafts or shed dead skin cells) diffusing up through the snowpack can search more effectively in 30 minutes than can 20 searchers in 4 hours with the use of course probing. Dogs are not infallible, and their ability to find buried human scent may be affected by several factors, such as the length of burial, weather, snow density, and contamination of the debris field with spit, urine, cigarettes, or gasoline from snowmobiles or generators. Dogs have found bodies buried 10 m (33 feet) deep, but have also passed over persons buried only 2 m (6.6 feet) deep. Dogs find "scent" and not people, although sometimes they find both. On numerous occasions, dogs signaled an alert in the vicinity, which may extend out as far as 9 to 12 m (30 to 40 feet) from a buried victim. These scent clues then led to searchers finding victims with other technologies.

### RECCO

The electronic rescue system called RECCO (www.recco.com) enables organized rescue teams to find victims who are equipped with reflectors (Figure 4-30). The system consists of two parts: a detector used by the rescue team (either on the ground or from helicopters) and a reflector worn by the recreationist. About the size and weight of a notebook computer, the detector is easily transported to the accident site. The detector transmits a directional radar signal. When it hits the reflector, the signal's frequency is doubled and reflected back to the detector, and the rescuer can follow the signal to the buried person. The reflectors are small and passive (i.e., no batteries) electronic transponders that are fitted into outerwear, ski and snowboard boots, and helmets. The system will detect some electronic equipment (with diminished range), such as cell phones, electronic cameras, radios, and even turned-off avalanche rescue beacons, so RECCO should be used by rescue teams together with avalanche rescue dogs, rescue beacons, and probers as part of the first response to any avalanche rescue. A detector operator can search as quickly as if using an avalanche beacon. In North America, more than 130 ski areas, mountain rescue teams, and national parks have detectors. Worldwide, more than 600 ski resorts and helicopter rescue teams use them.

## THE AVALANCHE VICTIM

After reaching a 20-year low in the late 1980s, avalanche deaths in the United States soared during the 1990s (Figure 4-31) and spiked twice (2007-2008 and 2009-2010) with 36 deaths, the greatest number killed during the modern era (i.e., after 1950). Figure 4-31 also presents the 5-year moving average. Since the end of the 1980s, the average number of fatalities per winter rose from 11 to 30, but decreased slightly in the mid-2000s. In recent years, the 5-year average has edged back upward to 30 deaths per winter. As more people head into the winter backcountry, avalanches continue to be deadly. In the United States since 1950,


**FIGURE 4-30** **A,** The RECCO reflector is a thin circuit card covered in soft plastic. It does not need batteries and does not need to be turned on or off. The reflector can be attached to jackets, pants, boots, and helmets. **B,** The RECCO detector consists of a transmitter and a receiver. It can also be used from a helicopter. *(Courtesy RECCO AB, Sweden.)*

avalanches have claimed 993 lives, and 28% (281) of those victims died during the 10 winters up to 2012-2013.[1]

From 2004 to 2014, almost all avalanche victims (94%) were pursuing some form of recreation at the time of the accident (Figure 4-32). Snowmobilers constitute the largest group of avalanche victims, primarily because snow machines with powerful engines and better tracks can climb steep, avalanche-prone terrain and can cover significantly more terrain in a day than human-powered backcountry users, thereby exposing snowmobilers to greater avalanche risk. The combined "backcountry tourer" and "sidecountry rider" categories accounted for more avalanche deaths than in snowmobilers (112 vs. 101). Backcountry tourers and sidecountry riders are "out of bounds" skiers and snowboarders who venture into areas that are not part of a ski resort and thus are not managed for avalanches. The backcountry tourer category includes ski mountaineers, helicopter skiers, riders on split boards, and snowcat skiers. In-bounds riders are skiers and boarders who are on open terrain within the ski area boundary that is managed for avalanches; in-area avalanche fatalities are rare. Among nonrecreation groups, avalanche deaths occur in residents of avalanche-hit homes, highway personnel (motorists and plow drivers), and ski patrollers. Since 2004, 13 states have registered avalanche fatalities (Figure 4-33).

## STATISTICS OF AVALANCHE BURIALS

Survival during avalanche burial depends on the grade and duration of burial and the pathologic processes of asphyxia, trauma, and hypothermia.[9] The grade of burial is defined as critical or noncritical. *Critical burials* are those in which asphyxiation may occur and include full burial or partial burial, where the head is under the snow and breathing is impaired.[24] Duration of burial

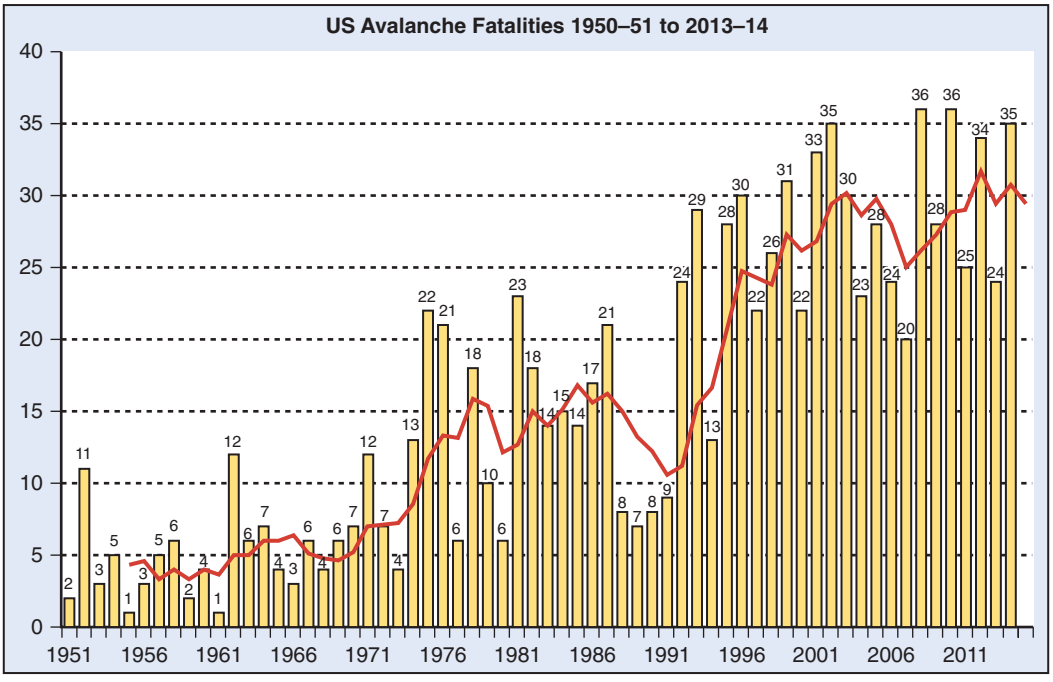



**FIGURE 4-31** Avalanche deaths in the United States in the modern era (i.e., after 1950) by winter, by season, and showing a 5-year moving average. *(Data from Dale Atkins and Colorado Avalanche Information Center.)*

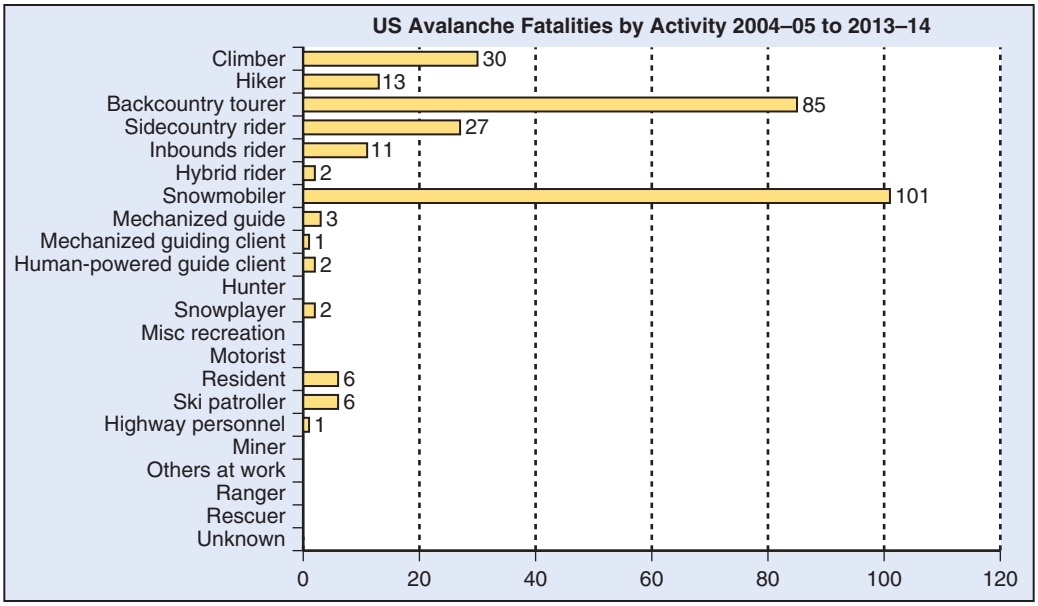



**FIGURE 4-32** U.S. avalanche fatalities by activity, winter 1999-2000 to winter 2008-2009. *(Data from Dale Atkins and Colorado Avalanche Information Center.)*

can be influenced by depth of burial, clues on the surface that make the avalanche victim easier to locate, available rescue equipment, and competence of the rescuers. Body position, traumatic injury, snow density, and presence and size of an air pocket influence risk of asphyxiation.

A victim who is uninjured and able to fight on the downhill ride usually has a better chance of being only partly buried, or if completely buried, a better chance of creating an air pocket for breathing. A victim who is severely injured or rendered unconscious will more likely be rolled, flipped, and twisted during the slide. Being trapped in an avalanche is a life-and-death struggle.

Avalanches kill in two ways. First, traumatic injury may occur as the victim tumbles down the avalanche path. Trees, rocks, cliffs, and the wrenching action of snow in motion can produce blunt or penetrating trauma. Up to one quarter of all avalanche deaths are caused by trauma, especially to the head and neck. Second, snow burial causes asphyxiation in at least three quarters of avalanche deaths from either airway obstruction or hypercapnia and hypoxemia as a result of rebreathing expired air. A very small percentage of avalanche victims succumb to hypothermia (see Avalanche Victim Physiology and Medical Treatment after Rescue, later).

A victim who is swept down in the churning maelstrom of snow has difficulty breathing. Inhaled snow clogs the mouth and nose; if the victim is buried with the airway already blocked, asphyxiation will occur more quickly. Snow that was light and airy when a skier carved turns becomes viselike in its new form. Where the snow initially might have been 80% air, it might be less than 50% air after an avalanche and is much less permeable to airflow, thereby making it more difficult for the victim to breathe.

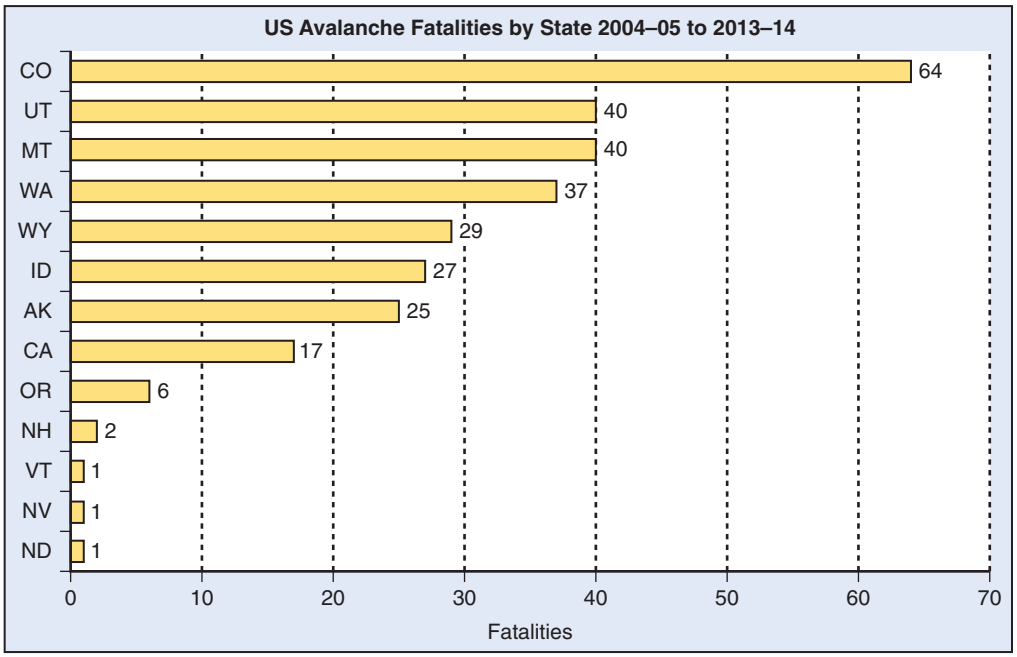



**FIGURE 4-33** U.S. avalanche fatalities by state, winter 1999-2000 to winter 2008-2009. *(Data from Dale Atkins and Colorado Avalanche Information Center.)*

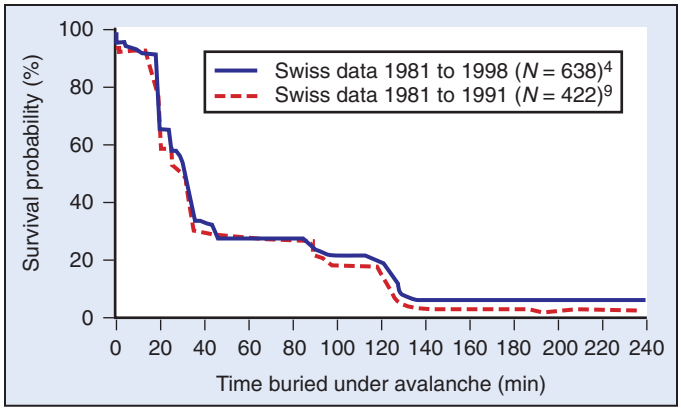



**FIGURE 4-34** The *solid blue line* indicates data from Switzerland for survival probability for completely buried avalanche victims in open areas from 1981 to 1998 ($n = 638$) in relation to time (minutes) buried under the snow. The median extrication time was 37 minutes. The *dashed red line* represents survival probability for completely buried avalanche victims in open areas ($n = 422$) on the basis of the Swiss data for 1981 to 1991. *(Data from Falk M, Brugger H, Adler-Kastner L: Avalanche survival chances,* Nature *386:21, 1994. From Brugger H, Durrer B, Adler-Kastner L, et al: Field management of avalanche victims,* Resuscitation *51:7, 2001, with permission.)*

Snow sets up hard and solid after an avalanche. It is almost impossible for victims to dig themselves out, even if they are buried less than 30 cm (1 foot) deep. Hard debris also makes recovery very difficult, so a sturdy shovel is essential. The pressure of the snow in a burial of several feet is sometimes so great that the victim is unable to expand the chest to draw a breath.[41] Warm exhaled breath freezes on the snow immediately in front of the face, eventually forming an ice lens that cuts off all airflow and contributes to asphyxiation in some buried avalanche victims.

Statistics regarding survival are derived from a large number of avalanche burials (Figure 4-34). When compiling these figures, only persons who were totally buried in direct contact with the snow were included. Victims who were buried in the wreckage of buildings or in vehicles are not included, because they can be shielded from the snow in situations in which sizable air pockets may be present. Under such favorable circumstances, some victims have been able to live for days. In 1982, Anna Conrad lived for 5 days in Alpine Meadows, California, in the rubble of a demolished building; this is the longest avalanche victim survival on record in the United States.

A completely buried victim has a poor chance of survival, which is related to both time and depth of burial. Survival diminishes with increasing burial depth, because it takes longer to uncover a victim who is buried deeper. To date, no one in the United States who has been buried deeper than 3 m (10 feet) has been recovered alive; however, in Europe, two victims survived burials of 6 to 7 m (20 to 23 feet).[1,42]

Burial depth is important in avalanche survival, but time is the enemy of the buried victim. Figure 4-34 shows survival probability function calculated from European data, which has become a classic demonstration of decreasing survival chances with increasing burial time. During the first 15 minutes, most people (~90%) are found alive. At 30 minutes, an equal number are found dead and alive. After 30 minutes, more are found dead than alive, and the survival rate continues to diminish with longer burial time. Speed is essential to the search. Buried victims can live for several hours beneath the snow under favorable circumstances. A miner in Colorado who was buried by an avalanche near a mine portal was able to dig himself free using his hard hat from nearly 1.8 m (6 feet) of debris after approximately 22 hours. In 2003, two snowshoe hikers caught near Washington's Mt Baker survived burials of 23 and 24 hours. Such long survival times are a reminder that no rescue should be abandoned prematurely on the assumption that the avalanche victim is dead.

## RESCUE STATISTICS

A buried victim's chance of survival directly relates not only to the depth and length of time of burial, but also to the type of rescue. Table 4-2 shows the statistics for survival as a function of the rescuer. Buried victims rescued by a small team (companions or groups nearby) have a much better chance of survival than those who are rescued by professional (organized) teams. Small teams of companions can potentially rescue a victim in minutes, whereas it may take hours to mount a professional rescue. Of people found alive, 78% were rescued by small teams, and 13% were found by a professional rescue team.

Table 4-3 compares the results of rescue by different techniques. Of victims (36 of 70) buried with a body part (e.g., hand) or an attached object (e.g., ski tip) protruding from the snow, 51% were found alive. In most cases, this was simply good luck, but in some cases, it was the result of actively fighting with the avalanche or of thrusting a hand upward when the avalanche stopped. Either way, this statistic shows the advantages of a shallow burial: less time required to search, shorter digging time, and the possibility of attached objects or body parts being visible on top of the debris. Of the fatalities in this category, many of the individuals were skiing or snowboarding alone with no companion present to identify the hand or ski tip and provide rescue. Organized probe lines have found more victims than any other method, but because of the time required, most victims (93%) are recovered after they are dead.

**TABLE 4-2** Type of Rescue for Buried Avalanche Victims in Direct Contact with Snow Based on a Sample of 360 Burials in the United States from Winter 2003-2004 to Winter 2012-2013

| | Individual (Self) | Small Team (Companions) | Professional (Rescue Team) | Total |
|---|---|---|---|---|
| Found alive | 9 | 83 | 14 | 106 |
| Found dead | — | 116 | 138 | 254 |
| Survival | 100% | 42% | 9% | 29% |

Data from Atkins D: 10 years of avalanche rescues in the United States, 2003/04 to 2012/13. *Avalanche Rev* 33(3):22-24, 2015.

**TABLE 4-3** Method of Rescue for Buried Avalanche Victims Based on a Sample of 274 Avalanche Burials in the United States from Winter 2003-2004 to Winter 2012-2013

| Method | Found Alive | Found Dead | Total |
|---|---|---|---|
| Attached object or body part | 36 (51%) | 34 (49%) | 70 |
| Spot probe | 3 (18%) | 14 (82%) | 17 |
| Probe line | 3 (7%) | 40 (93%) | 43 |
| Rescue transceiver | 41 (27%) | 109 (73%) | 150 |
| Avalanche dog | 2 (7%) | 21 (93%) | 23 |
| Voice | 10 (100%) | 0 | 10 |
| Digging | 2 (14%) | 12 (86%) | 14 |
| RECCO | 0 (0%) | 4 | 4 |
| Melted out | 0 (0%) | 14 | 14 |
| Not found, not recovered | 0 (0%) | 4 | 4 |
| Inside vehicle | 0 (0%) | 0 | 0 |
| Inside structure | 0 (0%) | 2 | 2 |
| Total | 97 (28%) | 254 | 351 |

Data from Atkins D: 10 years of avalanche rescues in the United States, 2003/04 to 2012/13. *Avalanche Rev* 33(3):22-24, 2015.

During the 10 years of winter 2003/04 to 2012/13, the avalanche transceiver was used to find more victims than any other method; it is the best method for finding the completely buried victim if it is carried and used correctly. The bad news is that the mortality rate over those 10 years was 73% (see Table 4-3). Unfortunately, most companions cannot use a transceiver fast enough to save a life. Even in textbook rescues (i.e., signal quickly located and victim dug free in a short time), many victims did not survive.

Organized probe lines are an effective way to find a buried victim, but because this method requires many rescuers and much time, most victims (93%) are recovered dead. Despite the sound-insulating properties of snow, 10 victims who were shallowly buried were able to yell and be heard by rescuers (i.e., voice contact; see Table 4-3).

These statistics point out the extreme importance of rescue skills. Professional (organized) rescue teams (e.g., ski patrollers, mountain rescuers) must be highly practiced. They must have adequate training, manpower, and equipment to perform a hasty search and probe the likely burial areas in a minimal amount of time. For backcountry rescues, the buried victim's companions (small-team rescue) remain the best hope for survival. The need to seek outside rescue units means a much lower chance (but not zero) for live recovery.

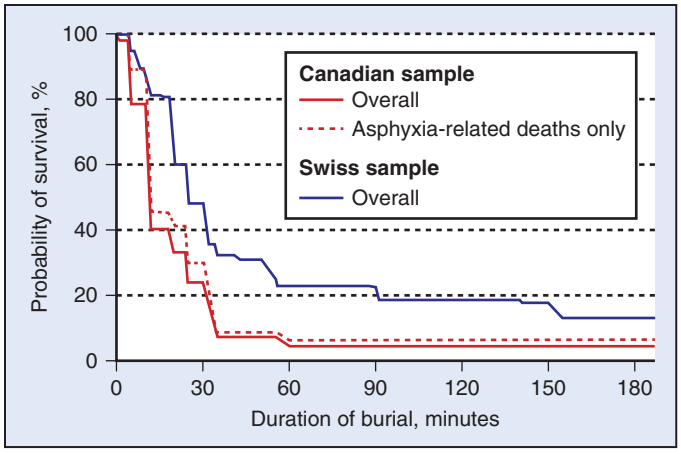



**FIGURE 4-35** Comparison of the Swiss avalanche survival curve *(blue line)* and the Canadian survival curve *(red line)* over the same 25-year period. Note the rapid drop after 10 minutes in the Canadian curve, although maintaining the same morphologic survival phases. *(From Haegeli P, Falk M, Brugger H, et al: Comparison of avalanche survival patterns in Canada and Switzerland,* CMAJ *183(7):789-795, 2011.)*

# AVALANCHE VICTIM PHYSIOLOGY AND MEDICAL TREATMENT AFTER RESCUE

## MORBIDITY AND MORTALITY

Asphyxiation is the most common cause of death during avalanche burial. More than 75% of avalanche deaths result from asphyxiation; less than 25% result from trauma, and very few result from hypothermia.[7,22,25,28,30,41,42] Because asphyxiation is the major cause of death during avalanche burial, time to extrication is a major determinant of survival. In a large Swiss data set, fully buried avalanche victims have a more than 90% chance of survival if they are extricated within 15 to 18 minutes, but this chance drops to only 30% to 34% after about 35 minutes (see Figure 4-34).[8,15] This emphasizes the need for a swift companion rescue at the avalanche site to avoid asphyxiation. Survival beyond 30 minutes of burial requires a patent airway and adequate air pocket for breathing. If the air pocket is large enough, avalanche victims may survive for hours and develop severe hypothermia.

The mortality statistics, in particular relating to trauma, vary with geographic location and the terrain over which the avalanche slides. Avalanches that run through heavily forested regions may cause significant trauma to victims compared with avalanches that run in open bowls above tree line. In Canada, trauma is the primary cause of death in 24% and asphyxiation in 75% of avalanche deaths. In 13% of the primary asphyxia avalanche deaths in Canada, there was evidence of major trauma, which may have contributed to asphyxiation.[7] Data from Utah and Austria show a much lower mortality from trauma, about 5% and 6%, respectively, and a much higher mortality from asphyxiation, about 95% and 92%.[25,30] A difference in geography between countries may be one reason why trauma mortality in avalanche victims is higher in Canada than in the United States and Austria.

In an example of geographic variability as it pertains to avalanche survival, recent comprehensive examination of full-burial survival patterns in Canada was compared to an updated Swiss data set over the same 25-year period using similar statistical methodology (Switzerland, $n = 946$; Canada, $n = 301$).[23] There was no difference in overall survival between the two countries (46.2% in Canada, 46.9% in Switzerland). The Canadian survival curve, however, was characterized by earlier and more rapid drop in survival in the early stages of burial, after only 10 minutes (Figure 4-35). Trauma accounted for more than half the deaths in victims extricated in the first 10 minutes, and two thirds of trauma-related deaths involved collision with trees. Overall trauma-related avalanche mortality in Canada was 18.9%, compared with about 6% in Europe and the United States.[25,30]

Traumatic injury to avalanche victims depends on the terrain over which the avalanche occurred. If the victim is carried through trees or over rock bands, trauma is more likely and may result in death. An early study of both partial and complete burials in Utah and Europe reported that traumatic injuries occurred among 25% of avalanche survivors.[22] The most common traumatic injuries were major orthopedic, soft tissue, and craniofacial injuries (Table 4-4). In a report of injuries in 105 avalanche victims in Austria,[25] most were only minor (Table 4-5). A high incidence of lower-leg fractures and shoulder dislocations occurred, which were thought to result from attached skis and poles causing additional mechanical leverage on the extremities. Spine fractures were found in 7% of victims.

A review of autopsy reports from 28 avalanche deaths in Utah over a 7-year period[27] showed that among 22 avalanche victims who died from asphyxiation, one-half experienced mild or moderate traumatic brain injury. The authors argued that this could cause a depressed level of consciousness and contribute to death from asphyxiation. All six of the avalanche deaths that resulted from trauma involved severe traumatic brain injury. In another review of 56 avalanche deaths in Utah, all three fatalities caused solely by trauma showed evidence of head injury.[30] Although these studies demonstrate the role of head injury in avalanches,

**TABLE 4-4** Injuries among Survivors of Avalanche Accidents (Partial and Total Burials)

| | Utah | Europe |
|---|---|---|
| Total injuries | 9 (total of 91 avalanche accidents) | 351 (total of 1447 avalanche accidents) |
| Major orthopedic injuries | 3 (33%) | 95 (27%) |
| Hypothermia requiring hospital treatment | 2 (22%) | 74 (21%) |
| Skin and soft tissue injuries | 1 (11%) | 84 (25%) |
| Craniofacial injuries | — | 83 (24%) |
| Chest injuries | 3 (33%) | 7 (2%) |
| Abdominal injuries | — | 4 (1%) |

From Grossman MD, Saffle JR, Thomas F, et al: Avalanche trauma, *J Trauma* 29:1705, 1989.

**TABLE 4-5** Pattern of Injury among 105 Avalanche Victims

| Trauma | Frequency (*n*) |
|---|---|
| Cerebral trauma | 2 |
| Chest trauma: all | 18 |
| Chest trauma: sternum or rib fracture (*n* = 16) | |
| Chest trauma: pneumothorax or hemothorax (*n* = 6) | |
| Spinal fracture: all | 7 |
| Spinal fracture: cervical (*n* = 3) | |
| Spinal fracture: thoracic (*n* = 1) | |
| Spinal fracture: lumbar (*n* = 3) | |
| Abdominal trauma | 1 |
| Pelvic fracture | 1 |
| Extremity trauma: all | 20 |
| Extremity trauma: lower-leg fracture (*n* = 8) | |
| Extremity trauma: shoulder dislocation (*n* = 6) | |
| Extremity trauma: femoral fracture (*n* = 4) | |

From Hohlrieder M, Brugger H, Schubert HM, et al: Pattern and severity of injury in avalanche victims, *High Alt Med Biol* 8:56, 2007.

the report from Austria of injuries in avalanche victims noted that the two fatalities caused solely by trauma in their series resulted from isolated fracture and dislocation of the cervical spine.[25] This reveals the force that an avalanche applies to the human body and the vulnerability of the cervical spine in an avalanche, because repeated hyperflexion and hyperextension can occur. In addition, it emphasizes the need for proper cervical spine stabilization during the rescue and resuscitation period. Avalanche victims can sustain virtually any type of trauma during their often turbulent descent in the avalanche flow, and certainly if involved in collisions with trees or rocks. The head and neck are especially vulnerable to enormous forces, and these areas appear to be the cause of much of the traumatic mortality during avalanche accidents.

## RESPIRATORY PHYSIOLOGY OF AVALANCHE BURIAL

Asphyxiation occurs during avalanche burial because inhaled snow occludes the upper airway or because expired air is rebreathed. Acute upper airway obstruction that results in asphyxiation is one of the causes of asphyxiation during the first 15 to 30 minutes of avalanche burial. Asphyxiation caused by rebreathing expired air may also occur during this time if there is no air pocket for breathing, or it may be delayed if an air pocket is present. Inspired air contains 21% $O_2$ and less than 0.03% $CO_2$; expired air contains about 16% $O_2$ and 5% $CO_2$. Rebreathing expired air in an enclosed space results in progressive hypoxia and hypercapnia that eventually causes death from asphyxiation. The larger the air pocket, the greater is the surface area for diffusion of expired air into the snowpack and for diffusion of ambient air from the snowpack into the air pocket, and thus survival time is longer before death occurs from asphyxiation. An ice mask is formed when water in the warm and humid expired air freezes on the snow surface in front of the face. Because this barrier is impermeable to air, it accelerates asphyxiation by preventing diffusion of expired air away from the air pocket in front of the mouth.

The physiology of asphyxiation from breathing with an air pocket in the snow was demonstrated in a study where participants sat outside a snow mound and breathed through an airtight mask connected by respiratory tubing to 1- or 2-L (0.9- or 1.8-quart) air pockets in the snow.[12] The snow had a density similar to that of avalanche debris (i.e., 150 to 600 kg/m$^3$ or 15% to 60% water). The initial fraction of inspired oxygen ($F_{IO_2}$) in the air pocket was 21%, and the initial fraction of inspired carbon dioxide ($F_{ICO_2}$) was near 0%. As expired air was rebreathed in the air pocket over about 30 minutes, the $F_{IO_2}$ decreased to about 10% and the $F_{ICO_2}$ increased to about 6% (Figure 4-36). These changes in $O_2$ and $CO_2$ levels in the air pocket resulted in decreased arterial $O_2$ saturation as measured by pulse oximeter and increased end-tidal partial pressure of $CO_2$. Most participants were not able to complete the entire 30 minutes of the study and had to stop as a result of dyspnea, hypercapnia, and hypoxia, but some reached an equilibrium where $F_{IO_2}$ and $F_{ICO_2}$ stabilized and hypoxemia and hypercapnia were tolerable. These few individuals lasted the full 30 minutes of the research protocol, demonstrating how breathing with an air pocket can prolong survival during avalanche burial.

Hypoxemia and hypercapnia occur as expired air is rebreathed. A smaller air pocket or lower-porosity snow (usually higher density) causes more rapid development of hypoxia and hypercapnia. Larger air pockets and higher-porosity snow (usually lower density) allow for more mixing of expired air with ambient air within the air-filled pore spaces between grains of snow in the snowpack. This results in longer survival times before hypoxia and hypercapnia become severe enough to cause death from asphyxiation. Porosity of snow, which is a dimensionless value, refers to the volume of air within a sample of snow compared to its total volume. It relates to the ability of gases to diffuse through snow, and roughly correlates to snow density (i.e., the mass per unit volume, typically kg/m$^3$; see Physical Properties, earlier). Higher-density snow generally, but not always, has lower

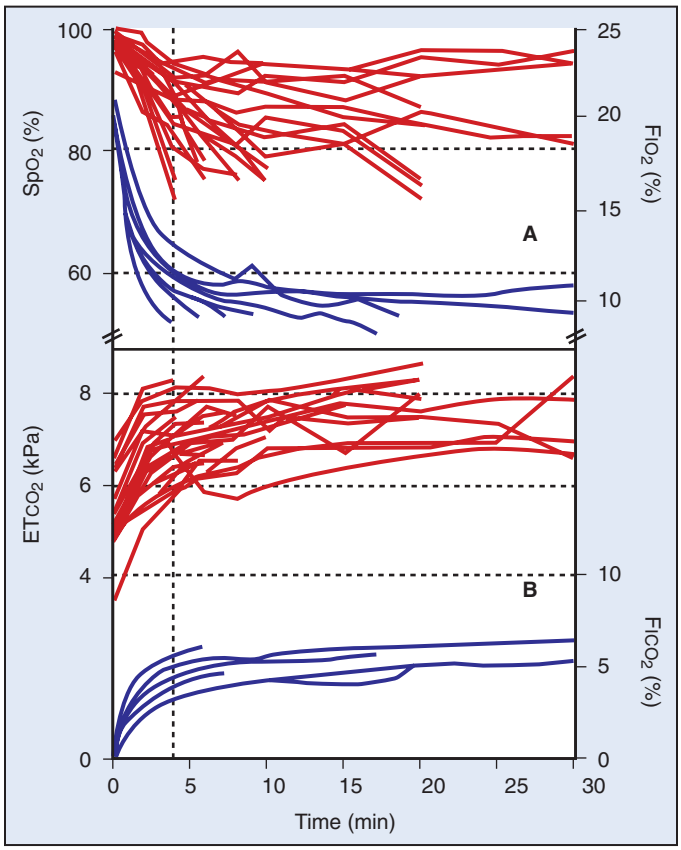



**FIGURE 4-36** Curves of individual respiratory parameters in persons breathing with a tight-fitting face mask connected to respiratory tubing running into 1- or 2-L air pockets in dense snow (*n* = 28). The *x*-axis represents time in minutes. Some participants did not complete the 30-minute study because of dyspnea or hypoxia. **A,** Arterial oxygen saturation ($SpO_2$ [%]) as measured by a digital pulse oximeter on the left *y*-axis *(red lines)* and fraction of inspired oxygen ($FIO_2$ [%]) on the right *y*-axis *(blue lines)*. **B,** Partial pressure of end-tidal carbon dioxide ($ETCO_2$ [kPa]) on the left *y*-axis *(red lines)* and fraction of inspired carbon dioxide ($FICO_2$ [%]) on the right *y*-axis *(blue lines)*. *(From Brugger H, Sumann G, Meister R, et al: Hypoxia and hypercapnia during respiration into an artificial air pocket in snow: Implications for avalanche survival,* Resuscitation *58:81, 2003, with permission.)*

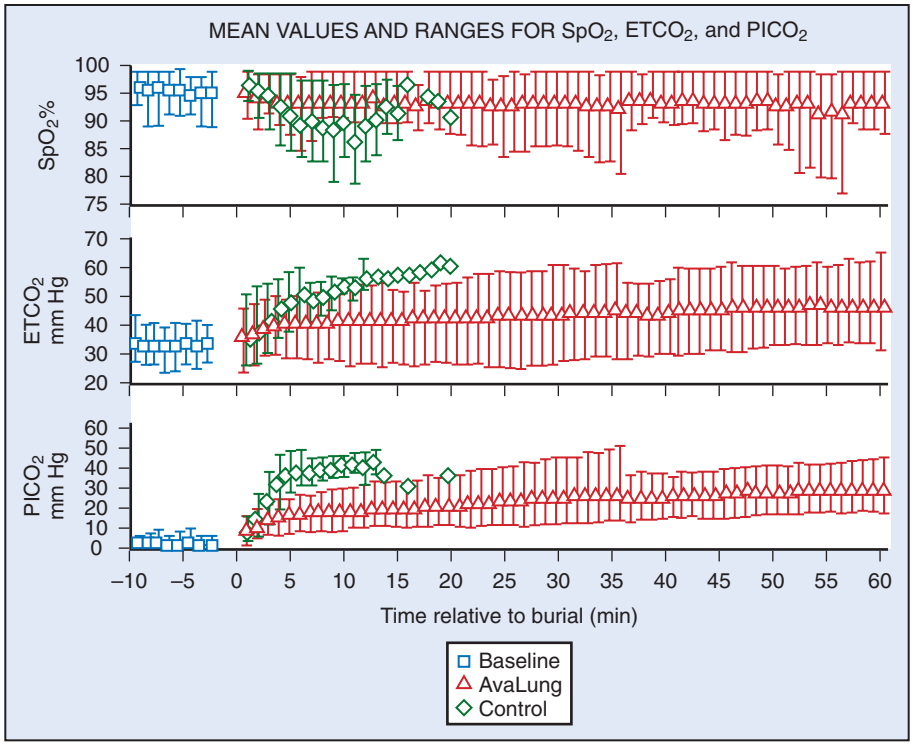



**FIGURE 4-37** Mean data and ranges are shown for physiologic parameters at baseline breathing ambient air (□), during the AvaLung burial (△), and during the control burial without the AvaLung (◇) ($n = 14$). The *x*-axis represents time relative to full burial in minutes for all panels. The *y*-axis represents percent saturation of hemoglobin with oxygen ($SpO_2$ [%]) for panel 1, end-tidal partial pressure of carbon dioxide ($ETCO_2$ [mm Hg]) for panel 2, and inspired carbon dioxide partial pressure ($PICO_2$ [mm Hg]) for panel 3. Some mean data points at the end of the control burial are missing or do not have ranges because of participant dropout (i.e., burial times of 5 to 19 minutes). *(Data from Grissom CK, Radwin MI, Harmston CH, et al: Respiration during snow burial using an artificial air pocket,* JAMA *283:2261, 2000; and Grissom CK, Radwin MI, Harmston CH, et al: Respiration during snow burial using an artificial air-pocket. In Schobersberger W, Sumann G, editors:* The annual yearbook of the Austrian Society of Mountain Medicine, *Austria, 2001, Austrian Society of Mountain Medicine.)*

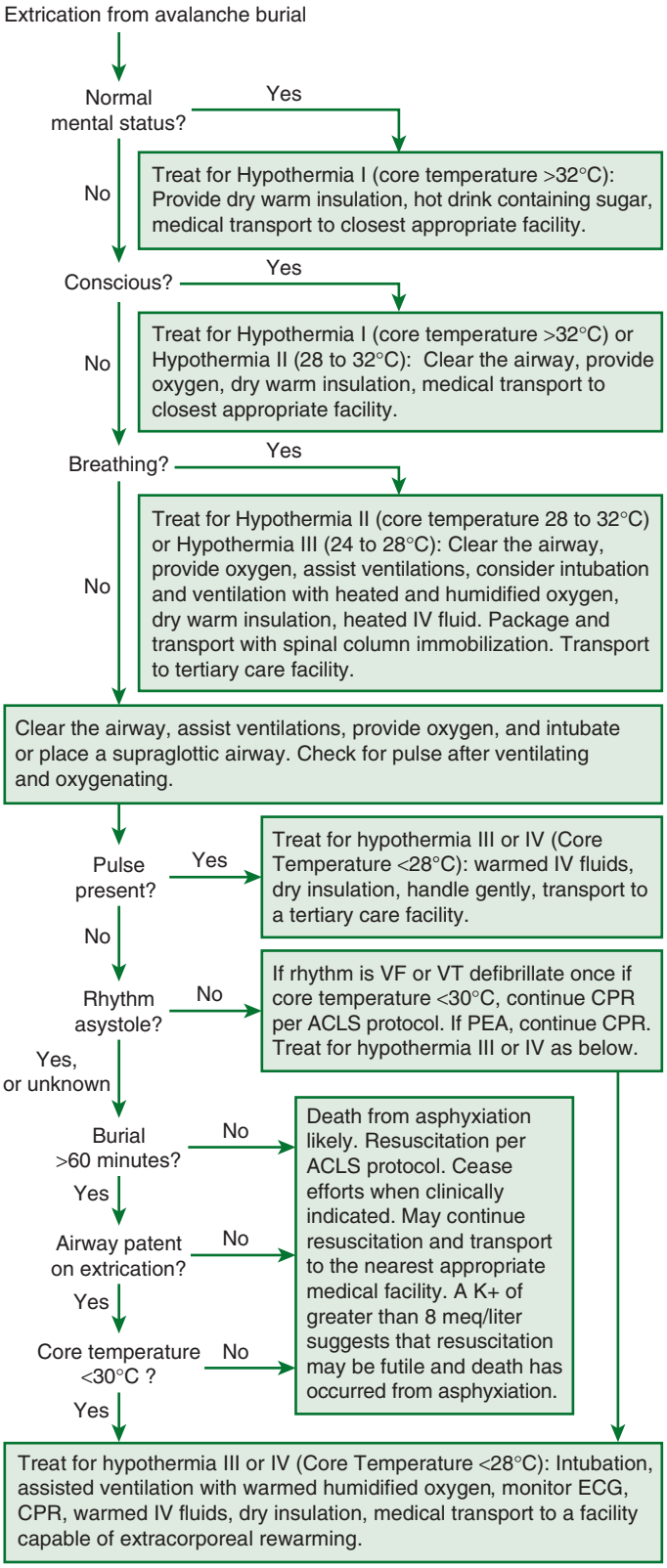
porosity because of complex variables. Since porosity of snow is very difficult to measure but density is frequently measured in the field, the avalanche literature refers to snow density as a surrogate value in relation to asphyxiation during avalanche burial.[12,20,23] An equilibrium occurs when expired air diffuses out of the air pocket and is replaced by fresh air that diffuses in from the snowpack, depending largely on snow porosity. This results in $FIO_2$ and $FICO_2$ reaching plateaus within a physiologically tolerable range, prolonging survival of the avalanche victim. This may occur even with small air pockets, which has been observed when extricating avalanche burial survivors of up to 2 hours' duration (likely in highly porous snow).[8]

Even densely packed snow contains sufficient ambient air to permit normal oxygenation and ventilation as long as all expired air is diverted out of the snowpack. This was demonstrated in a study of persons who were totally buried in dense snow and who inhaled air directly from the snowpack (density, 300 to 680 kg/m$^3$ or 30% to 68% water) through a two-way nonrebreathing valve attached to respiratory tubing that diverted all expired air to the snow surface. Participants maintained normal oxygenation and ventilation for up to 90 minutes.[35] This study demonstrated that there is sufficient air for breathing in snow with a density similar to that of avalanche debris as long as expired air is not rebreathed. This is the principle behind the AvaLung (see earlier), which has been designed to prolong survival during avalanche burial (see Figure 4-23). Although the device prevents formation of an ice mask, the expired air permeates around the buried person's body and through the snow, and it eventually contaminates inspired air. The AvaLung has been well studied using a human model of burial in snow of similar density to avalanche debris.[18-21] In the initial study,[20] breathing with the AvaLung while buried in dense snow was compared to breathing without the device but with a 500-cc (0.45-quart) air pocket in the snow. Mean burial time was 58 minutes when breathing with the AvaLung and 10 minutes when breathing with a 500-cc air pocket in the snow. Development of hypoxia and hypercapnia was significantly delayed by breathing with the AvaLung (Figure 4-37). The AvaLung has resulted in survival during actual avalanche burials.[13,34]

## MEDICAL TREATMENT AND RESUSCITATION OF AVALANCHE BURIAL VICTIMS

A published algorithm is available from the International Commission for Mountain Emergency Medicine (ICAR MEDCOM) for resuscitation of avalanche burial victims.[6,9] We recommend an algorithm for evaluating an extricated avalanche burial victim that incorporates key decision points from the ICAR MEDCOM algorithm for treatment and triage of avalanche victims in cardiac arrest. However, our algorithm also incorporates recommendations from the Wilderness Medical Society Practice Guidelines for the Out-of-Hospital Evaluation and Treatment of Accidental Hypothermia.[44]

Figure 4-38 presents the key points regarding assessment and treatment of an extricated avalanche burial victim. An initial impression of the level of consciousness is made as the head is exposed and cleared of snow. Opening the airway and ensuring adequate breathing are the primary medical interventions. Every effort should be made to clear the airway of snow as soon as possible and to provide assisted ventilation if breathing is absent or ineffective. These measures should be instituted as soon as possible and should not be delayed until the entire body is extricated. If traumatic injury to the spinal column is suspected, or if there is evidence of head or facial trauma, the spinal column




Notes:
Burial for 60 minutes: The provider should use the 60-minute threshold as a general, but not absolute, guide. Circumstances such as a presumed large air pocket could allow for a longer survival time without development of severe hypothermia. Core temperature: Providers may not have access to core temperature thermometers in the field. In this circumstance, the severity of hypothermia may be estimated with the use of Swiss hypothermia stages I through IV as determined by the clinical presentation (see Box 4-2)

**FIGURE 4-38** Assessment and medical care of extricated avalanche burial victims.

is immobilized as the airway is opened, adequate breathing ensured, and oxygen provided. When the avalanche burial victim is unconscious, maintaining the airway may be challenging because of space limitations with the opening of the snow leading to the victim. If endotracheal intubation is required for the unconscious apneic patient who is not yet fully extricated from snow burial, the inverse intubation technique may be required.[36] With this technique, the laryngoscope is held in the right hand while straddling the victim's body and facing the head and face. While facing the victim, insert the laryngoscope blade into the oropharynx with the right hand so that the larynx and cords can be visualized by leaning over and looking into the victim's mouth. The endotracheal tube is then passed through the vocal cords with the left hand.

After an adequate airway and breathing are established and supplemental oxygen is provided, circulation is assessed. The conscious patient is assumed to have a perfusing rhythm, and further treatment is directed at treating hypothermia and traumatic injuries. A patient who is found unconscious but with a pulse may have moderate or severe hypothermia and should be handled gently to avoid precipitating ventricular fibrillation (VF). The medical treatment of this patient is focused on (1) ensuring adequate oxygenation and ventilation, either noninvasively with a bag-valve-mask device or with a supraglottic airway device or endotracheal tube, as clinically indicated, and (2) immobilizing the spinal column for transport and treating any obvious signs of trauma. Intravenous (IV) access may be obtained and warmed isotonic fluids infused. Treatment of hypothermia is described in the next section.

If a pulse is not present after opening the airway and ventilating the patient and after checking for a pulse for up to 1 minute, CPR is begun.[9,44] Before CPR is initiated, careful evaluation for the presence of a pulse should occur. Avalanche burial victims are hypothermic, which causes peripheral vasoconstriction and makes pulses difficult to palpate. In addition, moderate to severe hypothermia causes bradycardia and depression of respiration.

In a pulseless avalanche burial victim, electrocardiographic monitoring should be used to assess cardiac rhythm, or, alternatively, an automatic external defibrillator may be applied. In the moderately or severely hypothermic patient in VF with a core body temperature of less than 30° C (86° F), defibrillation should be performed according to published guidelines,[3,43] with the caveat that, after unsuccessful defibrillation(s), further defibrillation may be deferred until rewarming is underway as chest compressions continue.[44] Drugs may be administered as part of advanced cardiac life support in accordance with published guidelines.[43]

The likelihood of the successful resuscitation of an avalanche burial victim who is in cardiac arrest at the time of extrication depends on whether cardiac arrest occurred from asphyxiation or from hypothermia. Factors that may be used to indicate the likelihood of survival in an extricated avalanche burial victim in cardiac arrest are burial time, airway patency, core temperature, and serum potassium level.[9,43] Avalanche victims in cardiac arrest after burials of less than 60 minutes are unlikely to be resuscitated, because death has most likely occurred as a result of asphyxiation. A patent airway is essential for survival beyond 60 minutes.[9] Even if duration of burial is less than 60 minutes or the airway is occluded, resuscitation should always be attempted for a limited time if feasible and safe. Avalanche victims extricated from burials of more than 60 minutes with a patent airway, who have no signs of life but who are moderately or severely hypothermic (i.e., core temperature of <30° C [86° F]), may be considered for ongoing resuscitation and transport to a medical facility that is capable of extracorporeal rewarming (Figure 4-38). Avalanche burial victims who are extricated in cardiac arrest after more than 60 minutes of burial and who have an obstructed airway have most likely died from asphyxiation, and continuing resuscitation efforts until rewarming occurs is unlikely to result in survival. In one study, none of 13 avalanche victims who were found in cardiac arrest after burials of 30 to 165 minutes' duration survived.[28] These results suggest that cardiac arrest was the result of asphyxiation rather than hypothermia.

An air pocket for breathing and a patent airway must be present for an avalanche burial victim to survive long enough to develop severe hypothermia. If an air pocket for breathing is not present or if the airway is obstructed, the avalanche victim who is extricated from snow burial in cardiac arrest has most likely died from trauma or asphyxiation. This is not meant to discourage initial attempts at resuscitation, but rather to suggest that prolonged CPR may be a futile exercise. It is always warranted initially to start CPR to see if return of circulation can be achieved in a reasonable time. This is because the rescuer can never know when the avalanche burial victim went into cardiac arrest; it may have been minutes or hours before extrication. The case of an 11-year-old boy in Utah who was extricated in cardiac arrest after 40 minutes of full burial demonstrates the importance of immediately starting CPR. Return of circulation was achieved 5 minutes after the initiation of CPR, and the boy went on to full recovery.

When laboratory testing equipment is available, serum potassium level can be measured and used as a prognostic indicator for avalanche burial victims in cardiac arrest.[6,37,43] In a hypothermic adult avalanche victim in cardiac arrest, a serum potassium level of greater than 8 mmol/L indicates that resuscitation efforts should be terminated. If the serum potassium is greater than 12 mmol/L in an adult or a child, resuscitation efforts should be terminated.[6]

## HYPOTHERMIA IN THE AVALANCHE BURIAL VICTIM

In avalanche burial victims who are extricated alive, hypothermia is a major medical problem that requires treatment.[6,9] Box 4-2 provides clinical definitions of hypothermia severity correlated with core body temperature. As the patient is extricated from snow burial, warm dry insulation is provided during packaging, and wet clothing is removed as soon as is practical. If the patient is unconscious, moderate or severe hypothermia is suspected, and handling should be gentle to avoid precipitating VF. When treatment of moderate or severe hypothermia is clinically indicated, IV access is obtained, and warmed (42°C [107.6°F]) isotonic fluid is infused. If the patient is unconscious, endotracheal intubation is appropriate to provide adequate oxygenation and ventilation with heated (40° to 45°C [104° to 113°F]) humidified oxygen. The goal of treating moderate to severe hypothermia before reaching the hospital is to limit temperature afterdrop during medical transport to the location where definitive rewarming can occur. The shivering patient with mild hypothermia can rewarm in the field if dry insulation is provided[18] (Figure 4-39).

**BOX 4-2 Hypothermia Clinical Prehospital Evaluation: Swiss Society of Mountain Medicine Definitions**

Hypothermia I: Patient alert and shivering (core temperature ≈35°-32°C [95°-90°F])
Hypothermia II: Patient drowsy and not shivering (core temperature ≈32°-28°C [90°-82°F])
Hypothermia III: Patient unconscious (core temperature ≈28°-24°C [82°-75°F])
Hypothermia IV: Patient not breathing (core temperature <24°C [75°F])

Data from Brugger H, Durrer B, Adler-Kastner L: On-site triage of avalanche victims with asystole by the emergency doctor, *Resuscitation* 31:11, 1996; Brugger H, Durrer B, Adler-Kastner L, et al: Field management of avalanche victims, *Resuscitation* 51:7, 2001; and Zafren K, Giesbrecht GG, Danzl DF, et al: Wilderness Medical Society Practice Guidelines for the Out-of-Hospital Evaluation and Treatment of Accidental Hypothermia, *Wilderness Environ Med* 25(4 Suppl):S66-S85, 2014.

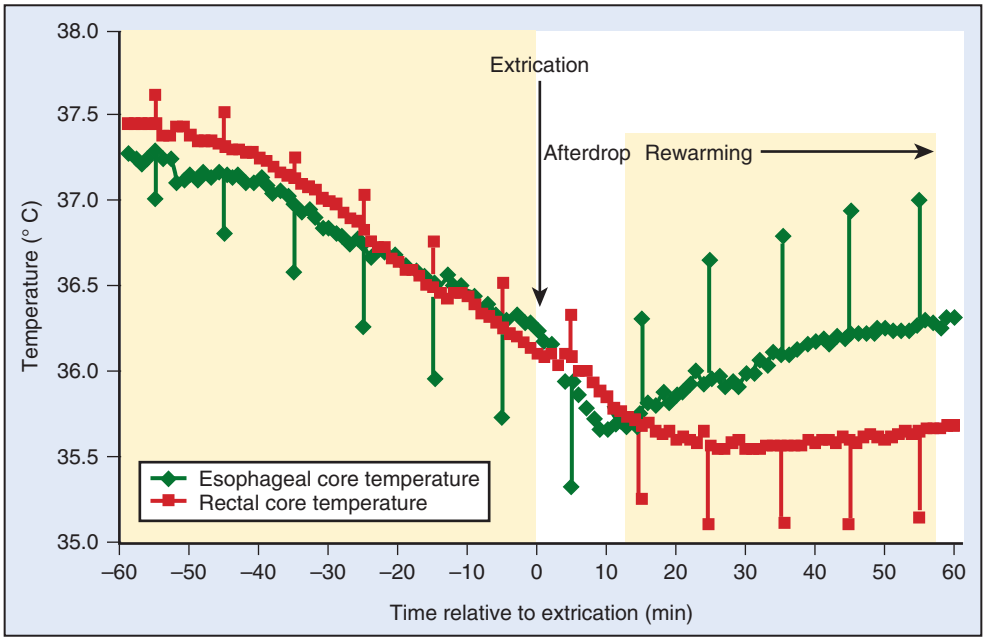



**FIGURE 4-39** Esophageal (◆; Tes) and rectal (■; Tre) core temperatures in °C ± standard deviation during snow burial and after extrication during passive rewarming. Time zero is relative to extrication from snow burial. At extrication from snow burial, both Tes and Tre show an afterdrop, but the afterdrop of Tes is attenuated and the Tes shows rewarming of the core as insulation is provided to the shivering person. Tre lags behind Tes during rewarming, which represents temperature gradients from the body core to the body shell as rewarming occurs. These findings suggest that core cooling rates during avalanche burial will cause only mild hypothermia during burials of up to about 1 hour in duration, but significant afterdrop can occur among avalanche victims after they are extricated from snow burial. Insulation should be provided as quickly as possible. Avalanche burial victims who are awake and shivering after extrication from snow burial can be rewarmed in the field by spontaneous endogenous rewarming (i.e., providing insulation and allowing shivering to occur to rewarm the avalanche burial survivor). *(From Grissom CK, Harmston CH, McAlpine JC, et al: Spontaneous endogenous rewarming of mild hypothermia after snow burial,* Wilderness Environ Med *21:229, 2010.)*

If the patient is conscious and alert, warm fluids that contain sugar may be given by mouth. If possible, patients with mild hypothermia should remain supine until rewarming has occurred, to prevent a greater temperature afterdrop from ambulation. Optimally, the severity of hypothermia is determined by core body temperature measurement in the field, preferably with use of an esophageal probe, but tympanic membrane sensors or rectal probes may also be used. If core body temperature cannot be measured in the field, the severity of hypothermia may be estimated with use of Swiss hypothermia stages I through IV, as determined by the clinical presentation (see Box 4-2).[44]

Severity of hypothermia may also be estimated from the time that the victim was buried and the average rate of core temperature cooling during burial. In a controlled experiment of human volunteers wearing a lightweight clothing insulation system, fully buried in compacted snow of similar density to avalanche debris, and breathing with an AvaLung for up to 60 minutes, core body temperature cooling rate was about 1.3°C (2.3°F) per hour.[21]

Retrospective studies of core temperature on hospital arrival[28] suggest that the average core temperature cooling rate is 3°C (5.4°F) per hour in survivors (range, 0.75° to 4.75°C [1.4°F to 8.6°F]). The higher rate of cooling in this study is based on a known time of burial and known arrival time at the hospital where core body temperature was recorded, and it does not differentiate between core temperature cooling rate during snow burial and afterdrop cooling rate during medical transport.

Burial cooling rates are accelerated by hypercapnia. Cooling rates measured in hypercapnic and in normocapnic buried individuals were 1.28°C and 0.97°C (34.3°F and 33.7°F), respectively,[19,21] In addition, minute ventilation, respiratory heat loss, total metabolic rate, and respiratory muscle metabolic rate were greater for the hypercapnic persons. Afterdrop cooling rate increases transiently after extrication from snow burial.[18,19] Afterdrop cooling rate increased up to fourfold for a mean of 12 minutes after extrication in those buried in snow breathing with an AvaLung for 60 minutes[18] (Figure 4-39). Accelerated cooling rate during and after extrication places avalanche victims at greater risk for complications caused by hypothermia. Rescue personnel should make every effort to prevent further heat loss in avalanche victims as soon as possible during extrication from the snow.

Core temperature cooling rate during avalanche burial may also be estimated from anecdotal reports of prolonged survival (Table 4-6). In one case, a 25-year-old male snowboarder with a large air pocket in front of his body survived 20 hours of avalanche burial.[21] At extrication, he had a tympanic core body temperature of 25.6°C (78.1°F) in snow at 5°C (23°F) and was spontaneously breathing; he had a Glasgow Coma Scale score of 8, heart rate of 35 beats/min, and a palpable pulse. Core body temperature cooling rate in this anecdotal report was about 0.6°C (1.1°F) per hour. The large air pocket probably allowed for adequate diffusion of expired air away from inspired air and prevented asphyxiation. In that same rescue, the survivor's companion was found dead from asphyxiation after 20 hours of burial with a tympanic core body temperature of 6°C (42.8°F), which is an average core body temperature cooling rate of about 1.5°C (2.7°F) per hour.

The highest reported rate of core body temperature cooling in an avalanche burial victim is 9°C per hour, which was reported for a 29-year-old man who was buried for 100 minutes and extricated alive with an epitympanic core temperature of 22°C.[31] He was endotracheally intubated and ventilated, but went into VF during helicopter transport and was transported with ongoing CPR (except for 15 minutes during the initial helicopter evacuation from the scene to a local hospital). He was in cardiopulmonary arrest for 150 minutes before defibrillation on the fifth attempt resulted in return of spontaneous circulation after extracorporeal rewarming. He made a full recovery.

These anecdotal reports demonstrate that core body temperature cooling rates during avalanche burial may vary significantly, and that survival after long-duration avalanche burial with severe hypothermia is possible. Survival of avalanche victims after cardiac arrest, however, is possible when the arrest is witnessed after extrication and caused by severe hypothermia. Resuscitation efforts should continue, and these victims should be transported to centers capable of extracorporeal rewarming. Avalanche victims extricated after unwitnessed cardiac arrest most likely were asphyxiated, and resuscitation to a perfusing rhythm is very unlikely.[29]

## SUMMARY

Asphyxiation is the major cause of death during avalanche burial and is time dependent. Traumatic injuries can be immediately fatal or can shorten the time for asphyxiation to occur. Avalanche victims who have not succumbed to a traumatic fatality and who are extricated within 15 minutes have a greater than 90% chance of survival. However, this "survival phase" decreases to about 30% if they are extricated after 30 minutes, thus emphasizing the need for small-team (companion) rescue with use of avalanche transceivers, probes, and shovels. Survival beyond 30 minutes of burial depends on the presence of an air pocket for breathing. The larger the size of the air pocket, the longer survival is possible during burial. Asphyxiation during avalanche burial is caused by rebreathing expired air that contains 16% oxygen and 5% carbon dioxide. A larger air pocket provides more surface area for diffusion of expired air away from an avalanche burial victim, allowing more ambient air from the snowpack to diffuse into the air pocket for inspiration. Opening the airway, ensuring adequate ventilation, and providing supplemental oxygen are the primary medical interventions for an extricated avalanche burial victim. If the victim is conscious, mild hypothermia is most likely, and treatment consists of providing warm, dry insulation and

**TABLE 4-6** Core Temperature Cooling Rates in Survivors of Avalanche Burial

| Author (Year) | Study Population | Conditions | Cooling Rate |
|---|---|---|---|
| Locher and Walpoth[28] (1996) | 16 survivors (3 cardiac arrest) of avalanche burial | Retrospective review, avalanche burial victims | 2.9°C/hr (range, 0.75°-4.75°C/hr), burial to hospital arrival |
| Spiegel[40] (2002) | Case report, 25-yr-old male avalanche victim | 20-hr avalanche burial with a large air pocket | 0.6°C/hr |
| Grissom et al[21] (2004) | 12 healthy participants with lightweight clothing | Simulated avalanche burial for up to 60 min | 1.3°C/hr (range, 0.6°-2.4°C/hr) |
| Putzer et al[33] (2010) | Case report, 28-yr-old male avalanche victim | 90-min avalanche burial; 25-min transport | 5°C/hr, burial to hospital arrival |
| Oberhammer et al[31] (2008) | Case report, 27-yr-old male | 95-min avalanche burial; cardiac arrest after extrication | 9°C/hr tympanic membrane temperature, burial to extrication |
| Paal et al[32] (2013) | 8 piglets, anesthetized, intubated, buried in a simulated avalanche | Breathing into an air pocket or ambient air | 4.6°C/hr with air pocket and 4.8°C/hr with ambient air |
| Boue et al[5] (2014) | Two case reports: first case, 17-yr-old male; second case, 41-yr-old male | First case, complete burial for 6 hr<br>Second case, complete burial for 7 hr | First case, 2.3°C/hr<br>Second case, 1.8°C/hr |

warm, sugar-containing liquids. Rewarming will occur through shivering. Avalanche burial victims who are not shivering have progressed to moderate or severe hypothermia and require medical transport to a hospital for definitive rewarming. Unconscious avalanche burial victims who are breathing may require endotracheal intubation for airway control and assisted ventilation and are most likely moderately to severely hypothermic. Victims who are extricated in asystolic cardiac arrest have most likely died from asphyxiation, and resuscitation to a perfusing rhythm is very unlikely. Avalanche victims extricated with a perfusing rhythm who subsequently have a witnessed cardiac arrest from severe hypothermia have a good chance of resuscitation to a perfusing rhythm, and resuscitation efforts should be continued while they are transported to a medical facility capable of extracorporeal rewarming. Head and cervical spine trauma should be anticipated, although any injury pattern can occur. Many avalanche victims have no traumatic injuries.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***Complete references used in this text are available online at expertconsult.inkling.com.***



# CHAPTER 5
# Lightning-Related Injuries and Safety

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## HISTORICAL OVERVIEW[104,155,325,381]

Lightning has caused injuries to people since humans evolved on Earth. It has played a major part in almost every ancient religion and culture. Priests, some of whom were the earliest astronomers, also became proficient at weather prediction, interpreting changes in weather as omens of good or bad fortune, sometimes to the advantage of their political mentors. To this day, lightning continues to engender stories, perceptions, and myths and is a popular topic for the press and science and weather documentaries.[155,325]

Lightning was often depicted in the art of ancient cultures and religions and has long been feared as an atmospheric flash of supernatural origins. A roll seal from Akkadian times (2200 BC) portrays a goddess holding sheaves of lightning bolts in each hand. Next to her, a weather god drives a chariot and creates lightning bolts by flicking a whip at his horses, while priests offer libations. A relief found on a castle gate in northern Syria (900 BC) depicts the weather god Teshub holding a three-pronged thunderbolt (Figure 5-1).

Beginning around 700 BC, Greek artists began to incorporate lightning symbols representing Zeus's tool of warning or favor. Zeus, the king of the gods, could control the weather. Early Greeks believed that lightning was his weapon and that lightning striking the Earth was a direct sign of Zeus's presence or influence. The ancient Greek poet Hesiod called Zeus the "cloud-gatherer" and the "thunderer." Zeus was also concerned with hospitality; if you treated a guest or stranger badly, you could outrage him (Figure 5-2).

Because lightning was a manifestation of the gods, any spot struck by lightning was regarded as sacred. Temples often were erected at these sites, where the gods were worshipped in an attempt to appease them.

Roman mythology regarded lightning as more ominous than did the Greeks, with Jupiter using thunderbolts as tools of vengeance and condemnation. Romans killed by lightning strikes were considered damned by Jupiter and denied burial rituals. Several Roman emperors wore laurel wreaths or sealskin to ward off lightning strikes. Important matters of state were often decided on observations of lightning and other natural phenomena. Both Seneca and Titus Lucretius discussed lightning in their treatises on natural events, and Plutarch noted that sleeping persons, having no spirit of life, were immune to lightning strikes.

The Navajo Indians of North America have a story about the hero Twins who used "the lightning that strikes straight" and "the lightning that strikes crooked" to kill several mythic beasts that were plaguing the People (Navajo), and in the process created the Grand Canyon.

Bantu tribesmen in Africa believed that lightning was caused by the flashing feathers of Impundulu, the lightning bird-god, whose flapping wings produced the sound of thunder (Figure 5-3). They hold that lightning has great power in their healing rituals. Even today, their medicine men go out in storms and bid lightning to strike far away. Different charms, herbs, or other materials may be buried under a house to protect it from lightning. Sand paintings show the lightning bolt as a wink in the thunderbird's eye. Lightning is associated with wind, rain, and crop growth.

The art of native Australians incorporates lightning symbols. Their lightning spirit is depicted as having axes attached to his joints, which beat together to make thunder.

Lightning also played a role in Buddhist symbolism. Early statues of Buddha show him carrying a thunderbolt with arrows at each end. Although lightning is most frequently rendered as fire, it has also been represented as stone axes hurled from the heavens. In the pantheistic Hindu religion, Indra was the god of heaven, lightning, rain, storms, and thunder (Figure 5-4). The Maruts used thunderbolts as weapons.

The Yakuts of eastern Asia regard rounded stones found in fields hit by lightning as thunder axes and often use the powdered stones in medicines and potions. In Chinese mythology, the goddess of lightning, Tien Mu, used mirrors to direct bolts of lightning. She was one of the deities of the "Ministry of Thunderstorms" of ancient Chinese religion.

Scandinavian mythology alludes to Thor, the thunderer, who was the foe of all demons (Figure 5-5). Thor tossed lightning bolts at his enemies, and Thursday is named for him. For the Vikings, lightning was produced by Thor as his hammer struck an anvil while riding his chariot across the clouds.

Throughout early Europe, church bell ringers would make as much noise as possible, hoping to scare away the storms from holy dwellings, because steeples were struck frequently

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**FIGURE 5-1** Teshub, the Syrian weather god.

**FIGURE 5-2** Zeus, king of gods who hurls the lightning.

**FIGURE 5-3** Impundulu, the lightning bird. *(Copyright artbybluedaisy.)*

by lightning. French peasants carry a pierre de tonnerre, or lightning stone, to ward off lightning strikes. Even Santa Claus gets into the act with his reindeer Donner (thunder) and Blitzen (lightning).

People in both developed and Third World countries often regard lightning and thunder with great fear as mysterious, uncontrollable, and unmanageable. Cambodians believe that by doing good deeds, they can avoid lightning strikes, and that people with moles on their calves are susceptible to lightning strikes, as are people who have broken promises. To resuscitate a victim of lightning strike, Cambodian villagers may drape the person's body with a white cloth, or jump over it three times, or place the victim in a bed and light a fire underneath it.

In South Africa, there is a belief that lightning can be "sent" to steal something, or from a traditional healer to someone else. In the Venda and Tsonga cultures of South Africa, families may use gasoline (petrol) to bomb the house of an enemy whom they believed called down lightning on a relative who was struck. In Africa in the 1990s, when only the members of one soccer team were injured, it was ascribed to witchcraft on the part of the other team. Other African beliefs state that burying a hippopotamus hide 2.4 m (8 feet) underground will protect their area. Wearing red is believed to be protective, but mirrors are thought to attract lightning, not only in Africa but in other cultures, including the United States.

South Africans believe that the *Syringa* tree attracts lightning (Figure 5-6). So strong is this belief that some farmers will cut down every *Syringa* tree on their property. Other trees are believed to be protective and homes are preferentially built under or near these trees.

In "civilized" Western societies, lightning can take on mystical significance. When lightning struck an English cathedral just

**FIGURE 5-4** Indra, the Hindu god of heaven, lightning, rain, storms, and thunder.

**FIGURE 5-5** Thor, the Norse god of thunder.

FIGURE 5-6 So strong is the belief among South Africans that the *Syringa* tree attracts lightning that farmers will cut down every *Syringa* tree on their farms. *(From http://www.bushveld.co.za/wildsyringa-tree.htm.)*

before enthronement of a controversial bishop in the late 20th century, some regarded it as an omen. Bullock[53,54] advances a case that the conversion of Saul on the road to Damascus resulted from a lightning strike.

## MODERN LIGHTNING MYTHS AND MISCONCEPTIONS

Medicine and meteorology are replete with myths, false impressions, and misunderstandings. In meteorology, these are based on a combination of lack of insight and understanding, limited personal experience, wishful thinking, and faulty outside opinion. This problem exists with regard to tornadoes, hurricanes, winter storms, perceived local effects, rare-event frequency, apparently cyclic storms, and so forth. Lightning is among the worst phenomena in this regard. These myths and misunderstandings are not idle issues; they can result in fatalities, maltreatment of patients, and erroneous court testimony. Therefore, some common misconceptions are worth addressing.

### LIGHTNING LORE

To dispel myths and misconceptions, it is helpful to understand the basic facts: lightning appears during thunderstorms that are often small and local, so the related time and space scales are short and limited. The effect of lightning begins with a 1-inch-diameter channel that has an extreme but spatially limited area of influence in most cases and only lasts a fraction of a second. As with other environmental threats, avoidance activities often need to be taken before lightning appears a certain threat.

The most important myth is that something can be done to make people completely safe from lightning wherever they may be. In more developed countries, almost 100% of lightning casualties occur because people do not take advantage of two generally safe locations: (1) a large, substantial, and enclosed, frequently occupied structure and (2) a fully enclosed, metal-topped vehicle. In the event of lightning strike, these locations conduct energy around the person to the ground through structural metal (e.g., plumbing, wiring, metal vehicle body). If a person is not inside one of these two reliably safe places, lightning becomes a threat. Outside these places, no posture, location, or action will make a person completely safe. In developed areas, these safe places are available to most people most of the time. When people choose to be away from these safe places, they have put themselves at risk for lightning injury. Thus, proper planning is important.

It is nearly impossible to protect all people in developing countries because there are times when lightning-safe structures or vehicles are unavailable. At night, homes may provide no lightning protection because of rudimentary construction. During the day, people may be at risk because schools, markets, and work structures may not be safe. Workers are also vulnerable because of labor-intensive manual agriculture, mining, and other unprotected work situations.

A second safety myth is that a person is safe if it is not raining hard or thundering frequently. Most people seek shelter from rain when storm clouds roll overhead. However, if shelter is not sought, injuries occur even in locations where there are relatively low flash rates. Protection from the rain is not the same as protection from lightning. Approximately 10% of cloud-to-ground strikes occur when no rain is falling at the time or location of the ground strike.

At least one-third of lightning casualties occur as the thunderstorm is approaching, because people misjudge its location or speed of approach, do not pay attention to warning signs, or want to finish an activity before seeking shelter.[89,190] Lightning may travel horizontally as far as 16 km (10 miles) or more in nearly any direction from the edge of a thunderstorm. As a result, when the sun is shining where the person is located, lightning may flash "out of a clear blue sky." Approximately one-third of victims are struck at the end of a thunderstorm because they have gone outside too soon.

One of the most consistently dangerous locations of injury is standing near a tree or other tall object. Tents, regardless of their construction, do not prevent lightning injury. Standing in an isolated flat area larger than 15 to 30 m (49 to 98 feet) in diameter is a recipe for disaster. The takeaway is that no place outside is safe from lightning threat when thunderstorms are in the area.

Small structures are almost always dangerous. It should be assumed that a small structure is not lightning safe, although it provides rain or sun protection. Although beach, sun, rain, bus, and golf shelters and sheds, including a hiker's lean-to, can be protected to some extent against lightning strikes by adhering to National Fire Protection Association (NFPA) lightning codes (NFPA780), this does not mean the people inside are safe.[229,291,292] In fact, it is likely that using such shelters may actually increase a person's risk for injury because of side flash and ground current. One should think of these shelters as capacitors with air insulation between the capacitor plates (roof and floor). It is easy to understand that someone standing inside the shelter decreases the amount of energy needed to discharge the capacitor and send lightning energy through the occupant. Sheltering within a shallow cave on a mountainside may protect from rain but may be very dangerous from a lightning perspective.

"When thunder roars, go indoors" is a primary teaching phrase in many lightning safety education efforts, including for the National Weather Service's Lightning Safety Awareness Week (http://www.lightningsafety.noaa.gov).[295] Although one might suppose that seeing lightning is a reliable warning, this is not always the case. On the Great Plains and in the western United States, lightning may be seen up to 160 km (100 miles) away, but in a forested area, lightning may not be seen at all before people are in danger. Seeing lightning is highly variable and may overestimate or underestimate the risk. Thunder is a more reliable tool for estimating distance and danger, because it is seldom audible from more than 16 km (10 miles) away. Unfortunately, in many cases, such as in a city, heavy traffic area, or noisy sports stadium, thunder may be inaudible.

The flash-to-bang method refers to seeing lightning, then hearing thunder. It is useful to understand in concept, even though it is decreasingly used by the general public, The rule is frequently misremembered, and there may be substantial uncertainty in linking flashes to thunder. The 30-30 rule from the late 1990s addresses the beginning and end of thunderstorms from a lightning safety point of view.[43,295] The first "30" is for counting the seconds from seeing lightning to hearing its thunder at the beginning of a storm. If the count to thunder is 30 seconds or less, lightning is 10 km (6 miles) distant or closer, indicating that a person is in danger and should be seeking safe shelter. U.S. Lightning Safety Week leaders replaced the first "30" for the general public with the phrase, "When thunder roars, go indoors," because they realized that people were using this time first to count rather than simultaneously seek shelter.[295] The second "30" indicates the minutes elapsed to reach the conclusion of the storm after seeing the last lightning or hearing the last thunder. One should wait these 30 minutes before going outside and

resuming activity. Although the 30-30 rule is not used as often as previously, the concept of a rule for both the beginning and end of a thunderstorm is the basis for safety rules used at many large venues, such as airports, mines, sports stadiums, and industrial sites. When objective lightning detection network data provide accurate information on lightning times and locations for large venues, these data should be used instead of imprecise colloquialisms.[292,295]

In developed countries, lightning injuries may occasionally occur indoors, but they are rarely, if ever, fatal. The causes are contact injury or side flashes from plumbing fixtures, computers, hard-wired telephones or electronic devices, and other appliances.[11,125,126] With a hard-wired phone, persons may suffer acoustic damage, neurocognitive deficits, death, or other lightning-related problems.[32,277] These occur because the telephone system in most houses is not grounded to the house's electrical system, but rather acts as a conduit for lightning either to come into or to exit from the home. Cell phones and cordless indoor phones offer complete protection from indoor electrical effects of lightning.[6,25,140,173,174,274,295] In developing countries where safe housing is not available, a dozen or more people may be killed indoors during a single incident.

## MEDICAL MYTHS AND MISCONCEPTIONS

A persistent myth is that lightning strikes are invariably fatal (Box 5-1). In truth, mortality rate may be as low as 5% to 10%.[67] One study of lightning cases occurring since 1900 found that lightning strike carries a mortality rate of 30% and morbidity rate of 70%.[87] A slightly different interpretation of the same data yielded a mortality rate of 20%.[33] Because peer-reviewed publications tend to reflect severe or interesting cases, case reviews likely overestimate mortality rate.

A prevalent myth is that the lightning victim retains an electrical charge and is dangerous to touch because the person is still "electrified." This myth may have led to unnecessary deaths by delayed resuscitation.[89] A person does not retain a charge like a battery or a capacitor. However, a person in contact with a live electrical power wire may transmit electricity.

A myth regarding treatment is that lightning injuries should be treated as are other high-voltage electrical injuries. In developed countries, the injuries seen with lightning are very different from high-voltage injuries and should be treated differently if iatrogenic morbidity and mortality are to be avoided.[23,88]

Although burns are commonly thought to be the major cause of death, this is not the case. Less than one-half of lightning survivors have any signs of burns or marks on their skin. The "crispy critter" myth, where someone struck by lightning bursts into flames or is reduced to a pile of ashes, is science fiction, not fact.[89] Lightning frequently flashes over the outside of a victim, sometimes damaging or disintegrating clothes, but leaving few external signs of injury and few, if any, burns. The only cause of immediate death is from cardiac arrest, sometimes following respiratory arrest.[87] Persons who are stunned or lose consciousness without cardiopulmonary arrest are highly unlikely to die, although they may have serious long-term sequelae.[90,97,281,320]

### BOX 5-1 The Most Common Myths and Facts about Lightning

**No Truth**

- Lightning injuries are always fatal.[12,26,33,87]
- Lightning is spelled with an *e*, "lightening."
- The cause of death from lightning injury is burns.[12,26,33,87]
- Burns are a major component of lightning injury.[12,26,33,87]
- Nothing is left of a person after a lightning strike except a pile of ashes.[87,88]
- Lightning victims have "entry" and "exit" points.[33,88]
- Lightning victims have internal burns.[88]
- One can predict the degree of injury from the voltage, amperage, or polarity of the strike.
- Metal (on the body or not) attracts lightning.
- Lightning does not hit outside the rainstorm.[295]
- It is safe to wait until the rain arrives to evacuate.[295]
- It is safe to finish the game if lightning is nearby.[190,295]
- As soon as the rainstorm passes and rain stops falling, it is safe to resume activity.[190,295]
- If you can see blue sky, lightning danger is minimal.
- Lightning never strikes the same place twice.
- It is safe to seek shelter and dryness under a tree.[295]
- Tall objects provide a 45-degree "cone of protection."
- The majority of persons injured are golfers.[190,295]
- Lightning injuries cannot occur inside a building.
- Rubber tires (shoes, raincoats, sitting on a backpack) protect a person from lightning.[295]
- Cell phones, iPods, and other electronic devices attract lightning.[6,25,140,173,174,274,295]
- Golf, picnic, bus, rain, beach, and other shelters are safe.[229,295]
- Grounding a building or shelter makes it safe for people from lightning injury.[229]
- Grounding a building makes it safe from structural damage.[229,292,325]
- Lightning victims remain electrified and dangerous to touch.
- Lightning victims are easier to resuscitate than other cardiac arrest victims.[372]
- If there are no outward signs of lightning injury, the damage cannot be serious.[89,90]
- Lightning victims usually require treatments similar to those for high-voltage electrical injuries, with aggressive fluid resuscitation, alkalinization, and fasciotomies.[88,93]
- Lightning survivors have few permanent problems.[90,105,281,320]

**Some Truth**

- Lightning always hits the highest object.
- The "pointier" an object, the more likely it will be hit.

**True**

- No place outside is safe when thunderstorms are in the area.[295]
- It is better to be "proactive" rather than "reactive" with lightning injury avoidance.[295]
- The top activities and places for lightning casualties in the United States are all outdoors[295]:
  Near trees (or other tall object)
  Open fields and elevated locations
  Water-related activities (e.g., swimming, boating, fishing)
- Safe shelter is a typical house or other fully enclosed and substantially constructed building with properly earthed plumbing and wiring.[295]
- Another safe shelter is a fully enclosed, solid, metal-topped vehicle, such as a car or bus.[90,105,281,295,320]
- Open picnic, bus, golf, and rain shelters, as well as tents and other camping cover, offer absolutely no protection from lightning and may actually increase the risk for injury.[229,292,295]
- Having and following a lightning safety plan can decrease the number of lightning deaths and injuries.[295]
- There is nothing that a person can do that will substantially decrease his or her lightning injury risk if "caught" in a thunderstorm.
- The lightning crouch does not significantly decrease the chance of injury or death.[295,342]
- Sitting on a backpack or sleeping pad does nothing to decrease an injury.
- Lying flat on the ground increases injury by increasing ground current effect.[104]
- The primary cause of death is cardiac and respiratory arrest at the time of the injury.[87]
- Ninety percent of lightning victims survive, but often with disability.[90]
- Lightning victims are safe to touch and do not retain an electrical charge.[292,295]
- Automated external defibrillators (AEDs) have been useful in some resuscitations.[103,106,290]
- Cell phones, iPods, and other small electronic devices do not attract lightning.[6,19,25,140,173,174,274,295]
- Lightning can strike the same place more than once.

**BOX 5-2 Lightning Myths Commonly Cited as Facts in Litigation**

These myths apply to electrical injury as well as lightning injury.

**Behavior of Current in the Body**

- Current seeks the earth.
- Current seeks the path of lowest resistance.
- Nerve tissue is a good conductor, or alternatively, current is preferentially conducted by nerve tissue.
- When a person touches a source of potential, current flows through the skin to other parts of the skin in contact with the same conductor (i.e., current passage is local only).

**Severity of Electric Shock**

*Myth:* If the following are absent, the shock cannot be severe and no deleterious effect can result:

- Being thrown (i.e., if a person is not thrown, the shock is not severe).
- Burns (i.e., if burns are not present, the shock is not severe).
- If the only surface change is a blister, the shock was not severe.
- Entry and exit wounds:
  Must be present.
  Demonstrate the current path.
  If not present, indicate no shock occurred.
- Fuses being blown (i.e., if a fuse does not blow, the shock is not severe enough to harm a victim).
- Low voltage cannot harm.
- Electroconvulsive therapy does not give long-term post–electric-shock symptoms (also false), so any other electric shock cannot be harmful.

**Investigations**

- If computed tomography (CT) and magnetic resonance imaging (MRI) scans are normal, there are no injuries.
- Negative investigations (e.g., nerve conduction study, electroencephalogram, CT, MRI) mean a victim has not sustained an electric shock.
- Neuropsychological testing is objective and easily interpreted.
- Burns of electrical origin can be distinguished on histologic examination.

**Remote Symptoms**

- Remote symptoms do not exist.
- Remote symptoms are proportional to the size of the shock.
- Symptoms of the shock that are not present immediately after the shock are not related to it.
- A person experiencing remote symptoms was psychologically vulnerable all the time.

**Miscellaneous**

- Litigation increases the potency of the claimed symptoms. *Corollary:* Resolving litigation terminates symptoms.
- Medical specialists understand electricity or lightning.
- Electrical experts can predict lightning or electrical injury.
- A diagnosis of depression, posttraumatic stress disorder, adjustment disorder, or other psychological problem negates an electrical injury causation.
- Residual current devices (RCDs) eliminate the possibility of all shocks. *Corollary:* A shock occurring when an RCD breaks the circuit cannot be severe.

Delayed causes of death include suicide induced by depression, perhaps related to disabilities wrought by lightning.[90,97] Unfortunately, burns in developing countries seem to be more severe in nature, perhaps because victims suffer temporary paralysis from lightning (keraunoparalysis) and may not be able to escape from falling, burning thatch.

Two other myths are, "If you're not killed by lightning, you'll be OK," and "If there are no outward signs of lightning injury, the damage can't be serious"[89] (Box 5-2). Older medical literature, lacking longitudinal or follow-up reports, implies that there are few permanent sequelae of lightning injury. However, reports in the last two decades consistently document several permanent sequelae, including nervous system injury (e.g., peripheral neuropathy, chronic pain syndromes) and neuropsychological symptoms (e.g., severe short-term memory difficulty, difficulty processing new information, attention deficit, depression, posttraumatic stress disorder (PTSD).[297,315,319,320,323,326,330]

Medical literature and practice are plagued by myths that arose from misread, misquoted, or misinterpreted data. These myths continue to be propagated, such as the "suspended animation" tenet that lightning victims who have resuscitation for several hours or who have been in cardiac arrest for a prolonged period without resuscitation may still successfully recover. This concept, credited to Taussig,[372] appeared some time before her article. The case reports a longer resuscitation period than usual, but not as miraculous as reported in Taussig's paper or as mentioned in subsequent references to her article.

A study of lightning survivors showed prolongation of the QT interval, raising the theoretical possibility of torsades de pointes as a mechanism for the suspended animation reports.[23] There is evidence from animal experiments to support the teaching that respiratory arrest may persist longer than does cardiac arrest.[12,108,109] A study of Australian sheep struck by simulated lightning showed histologic evidence of damage to respiratory centers located beneath the fourth ventricle.[12] Prolonged assisted ventilation may be successful after cardiac activity has returned, but this is obviously difficult to test in a human clinical study.[92,95,301]

Another series of animal experiments with simulated lightning strikes to hairless rats showed that it is possible to obtain skin changes (keraunographic markings), primary and secondary cardiac arrest with prolonged respiratory arrest, and temporary lower-extremity paralysis.[108,109,111]

## METEOROLOGIC LORE

Some people believe that small rivers, lakes, buildings, and hills can influence the formation or path of thunderstorms. Cumulonimbus clouds that produce lightning are, at the very least, several miles high and wide, and usually much larger. They move and transform because of large-scale factors exerted on the local atmosphere from great distances. Winds aloft arrive from various directions and usually are sufficiently strong that air within a thunderstorm may well have originated in another state or even country as recently as within the past 6 hours. A natural feature such as a small lake that is 1 km (0.6 mile) wide will have a thunderstorm traverse it at a typical velocity of 30 km/hr (18 miles/hr) over approximately 2 minutes. A very large mountain or ocean shoreline poses a different situation because of stationary large surface temperature contrasts.

"Lightning never strikes the same place twice," noted Benjamin Franklin in the late 1700s. In fact, tall buildings, communications towers, and wind turbines are struck many times a year. If the same meteorologic circumstances that caused the original lightning strike to occur are present, it is likely that lightning will strike repeatedly in the same place.

## MYTHS REGARDING ELECTRIC CURRENT CONDUCTION

The myth that "electricity seeks the least resistant path between two points" leads to the conclusion that there is only one line or tissue in which current will travel and produce damage. To the contrary, electric current is carried along all tissues and paths in inverse proportion to the resistance within the tissue/path. All structures between contact points are at risk, and there may be multiple paths between any two points traversed within a human.

Another myth is that lightning always seeks the earth, and furthermore, seeks the shortest path to the earth. Electrical energy may be conducted between two points on a body, neither of which needs be connected to the earth (e.g., the shock delivered to a person with each hand touching separate conductors). When a person is touching the earth, part of the current may flow through the person to earth and part may flow between two conduction points.

People commonly assume that no psychological effect will occur if lightning/electric current does not transit the brain. This

argument ignores the fact that multiple pathways exist for current conduction. Current conducted between two hands will be accompanied by electricity in the head and brain as a parallel pathway. In addition, there may be PTSD from the injury, blunt injury, and other factors (e.g., cortisol release) that can cause profound effects on the brain. Certainly, peripheral electric shocks have been shown to have effects on brain tissue.[241]

Many people try to simplify a complex phenomenon by using a much simpler phenomenon as an analogy. For instance, they may use a household circuit to explain lightning or electricity and assume that the effects are linear and scalable. Persons with knowledge of physics may insist on using Ohm's law. Lightning and electric currents are highly nonlinear and not amenable to simple circuit analysis. Lightning stroke current is probabilistic. It cannot be modeled using ordinary electrical components and principles and should not be analyzed in this way. Furthermore, the body and its tissues do not support this approach.

## MISCELLANEOUS LORE

Innumerable myths, superstitions, and misconceptions about lightning based on local customs, beliefs, and stories exist. An example is that using a cell phone increases a person's risk of lightning injury. With almost universal use of cell phones, it is common for people to be injured while using them (Figure 5-7). There is no scientific evidence that electromagnetic waves, the metal in a phone, or any other factor increases the risk for lightning strike. Injury probably occurs because people who should be seeking safety inside a substantial building or fully enclosed metal vehicle are outside and distracted from paying attention to the weather while they are talking or texting.

### Other Specific Myths and Misconceptions

- *Victims may have internal burns.* False; there may be cellular and nervous system damage, but rarely, if ever, internal burns typical of high-voltage electrical injuries.
- *Wearing rubber-soled shoes is protective.* False; similar erroneous concepts include wearing a raincoat or sitting on a foam pad or backpack. These do not protect a person or reduce the severity of the injury.
- *The rubber tires on a vehicle protect a person.* False; electrical energy travels along the outside of a metal conductor (the car body). It dissipates through the rainwater to the ground or flashes off the axles or bumper of the car (Figure 5-8). Tires may be torn or exploded.
- *Metal attracts lightning.* False; wearing metal does not attract lightning or increase one's risk. Metal objects, such as fences and bleachers, conduct electricity and lightning *after* they have been struck, but do not inherently attract lightning. Metal objects worn by a victim may become heated and thereby cause burn injuries. Tall objects tend to be made of metal, which are tall, isolated, and pointed. Their composition is not itself a significant factor for the propensity to be struck. The primary factors that affect where lightning will strike follow:
  1. Height of an object
  2. Isolation of an object from taller objects
  3. Pointed object (not a factor for people)
- *Lightning always hits the highest object.* False; lightning is only affected by objects within approximately 30 to 50 m (98 to 164 feet) from its leading tip. In addition, several pictures exist of lightning striking halfway down a flagpole or in a parking lot next to tall, isolated, and pointed light poles. Someone standing in the middle of a football field will not be protected by the goalposts if lightning is coming down directly overhead. There are many photographs of lightning branching to meet the earth in several places. These images portray the immense difficulty in being sure of the path of a cloud-to-ground lightning event.
- *Carrying an umbrella increases risk.* False; compared with the mile or two that lightning has already traveled to reach a person, increasing or decreasing one's height by a foot or two has a very minor effect.
- *The lightning "crouch" position can be used to significantly protect someone caught in a thunderstorm.* False; the change in height by approximately a foot has a very minor effect. A statistical evaluation indicated that it may decrease risk of the very infrequent direct strike by approximately 50%, but substantial risk remains from mechanisms of ground current, side flash, direct contact, and upward streamers.[341] The crouch cannot offer the almost complete protection afforded by preplanning, proactive avoidance of lightning, and staying in a substantial building or completely enclosed metal vehicle.
- *Lightning may occur without thunder.* False; whenever there is lightning, there is thunder, and vice versa. Sometimes it will appear that lightning is visible without thunder, since thunder is seldom heard more than 16 km (10 miles) from the lightning stroke. Thunder can be difficult to hear because of wind, trees blowing in the wind, noisy activities, and sound being blocked by buildings or mountains. There is also distraction from conversation, mobile phones, and other electronic devices. Survivors who are very close to a strike may not hear or remember the thunder, but it was there.

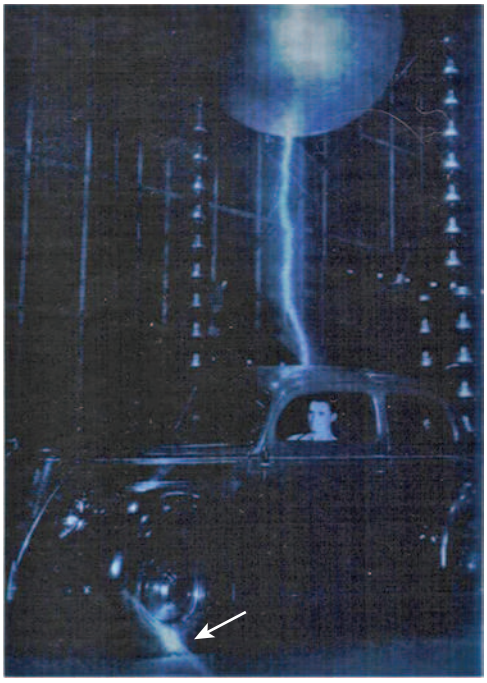

**FIGURE 5-8** Simulated lightning in a high-voltage laboratory travels across the external metal body of the car, escaping through the axle, hubcaps, or other metal closer to ground *(arrow)*. The Faraday cage effect of the metal body of the car protects the person from injury but does not protect the tires or wiring system of the vehicle.


**FIGURE 5-7** Warning on the Great Wall of China.

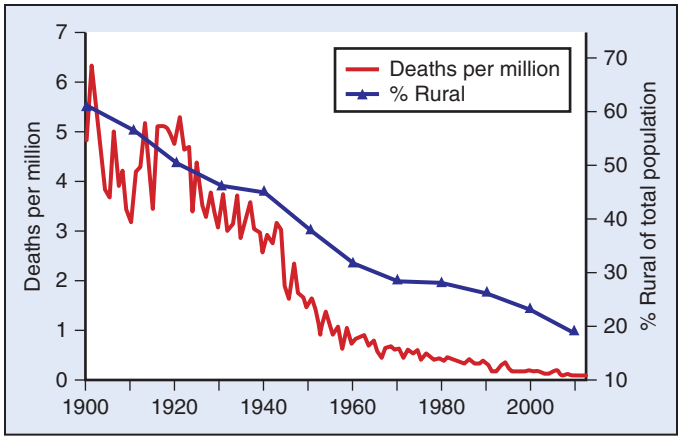


**FIGURE 5-9** U.S. lightning deaths per million people from 1900 to 2013 *(red)*. Percent rural population *(blue)*. *(From Lopez RE, Holle RL: Changes in the number of lightning deaths in the United States during the twentieth century,* J Climate *11(8):2070-2077, 1998.)*

## INCIDENCE OF INJURY

The number of lightning fatalities in the United States and other developed countries is well known, but the global figures are not reliable. The basis for global estimates comes from well-studied data from the United States and other developed countries that can be extrapolated. The U.S. lightning casualty history is an instructive example of what might be achieved globally, recognizing the trend from an agricultural to urban economic milieu.

### U.S. LIGHTNING CASUALTIES AND LIGHTNING

The number of annual lightning fatalities in the United States decreased greatly from a maximum of more than 400 deaths early in the 20th century to less than 30 deaths in recent years.[114,199,259,291,340] Figure 5-9 shows that the lightning fatality rate decreased from as high as six fatalities per 1 million people per year early in the 20th century to less than 0.1 per 1 million people per year at present.[259] This trend occurred simultaneously with a decrease from a 60% rural population in 1900 to the current rural population below 20%.

During this period, in addition to the rural to urban shift, there was also a significant increase with respect to lightning safety in the quality of dwellings, workplaces, schools, and other public and private buildings.[192] Also important was a huge increase since the early 20th century in availability of fully enclosed, metal-topped vehicles that provide safety from lightning.[192] Additionally, a significant lightning safety education and awareness campaign has been sustained across the United States for more than a decade.[100,213]

On average, 10 lightning-related injuries require medical treatment per lightning fatality over a large geographic area and a long period.[67] Males (84%) account for a much higher number of fatalities in the United States than do females.[114] This male majority has been found in most regions of the world over the last two centuries. The most common situation is for a single victim to be involved in a lightning incident.

Lightning deaths by U.S. state for the decade from 2005 to 2014 are shown in Figure 5-10, *left*. The general pattern is somewhat similar to the distribution of lightning in Figure 5-11. In general, there are more fatalities in the southeast, but more populous states also have larger total numbers of fatalities. Fatality information, rather than injuries, is used for these maps, because of greater uncertainty due to injury underreporting.[260] The lightning hazard is shown more clearly when population is taken into account (Figure 5-10, *right*). There are two maxima, one in the southeast and the other in the northern Rocky Mountain states. The most populous states, such as Pennsylvania, no longer necessarily have high ranks. Similar tabulations were recently made by Ashley and Gilson,[35] and maps by U.S. state have been published for earlier years.[114]

A large number of maps of cloud-to-ground lightning flashes for the U.S. have been prepared for three decades from data collected by the National Lightning Detection Network (NLDN)[113,286] (Figure 5-11). More than 20 million cloud-to-ground flashes occur annually in the lower 48 states.[304] Existing technology detects more than 90% of all cloud-to-ground flashes within the contiguous United States.[113,286] The most recent detailed 10-year climatology of cloud-to-ground lightning from the NLDN shows three locations on peninsular Florida, with the largest density of flashes per area in the United States. Flash density decreases northward and westward from Florida, although there are many variations that depend on well-identified meteorologic factors of strongly heated land surfaces producing strong upward atmospheric motions. Similar important features occur along the coast of the Gulf of Mexico. In the mountainous western states, where there are large changes in elevation, strong upward atmospheric motions are produced during many summer days.[188]

A series of NLDN studies has explored monthly and diurnal distributions of lightning, so these aspects of the lightning threat are well known across the continental United States. Approximately two-thirds of cloud-to-ground lightning flashes and casualties occur between noon and 1800 Local Standard Time.[114] Similarly, approximately two-thirds of cloud-to-ground lightning flashes, as well as lightning casualties, occur during the summer months of June, July, and August.[114,195] However, there are significant and sometimes unexpected variations from these general results.

The first steps to combining population with lightning data were made by noting that U.S. lightning fatalities tend to be concentrated in urban areas.[35] A new study of lightning fatality

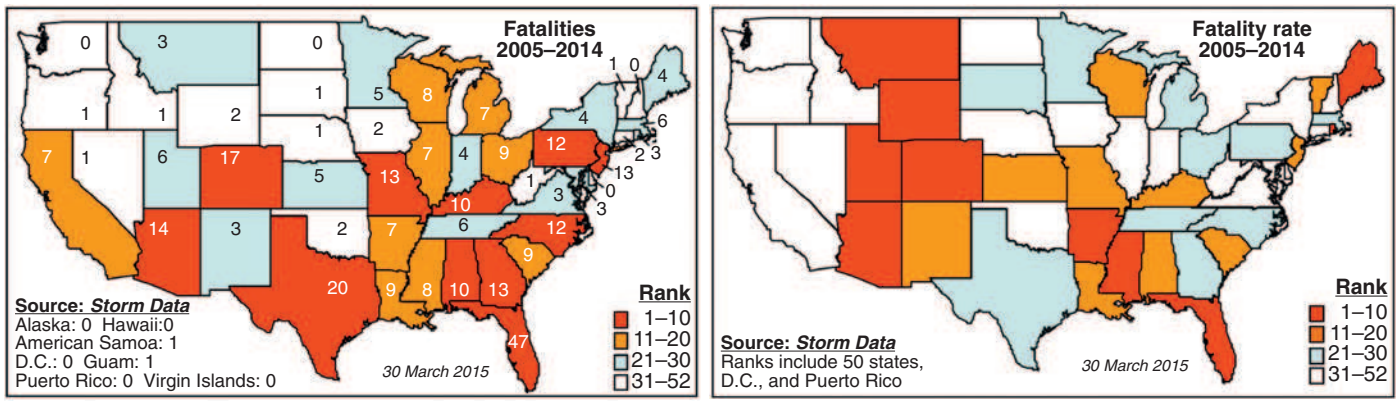


**FIGURE 5-10** Lightning fatalities *(left)* and fatality rate *(right)* by U.S. state from 2005 through 2014.

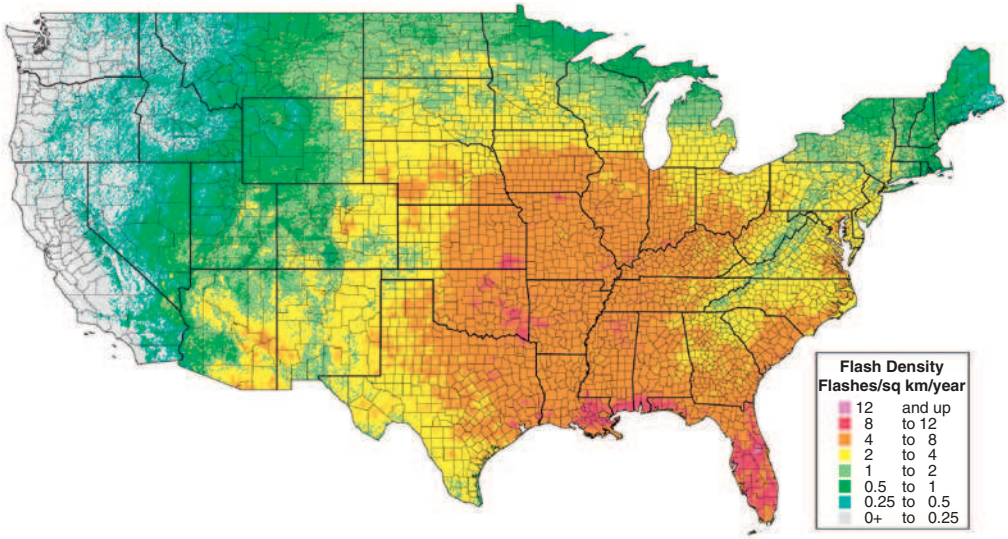


**FIGURE 5-11** Cloud-to-ground flash density at 2-km resolution from 2006 through 2015, based on the U.S. National Lightning Detection Network. *(Courtesy Vaisala Inc.)*



risk combines lightning frequency from a detection network with U.S. population data.[347]

## GLOBAL LIGHTNING CASUALTIES AND LIGHTNING

No reliable complete global information exists regarding fatalities or injuries. Two recent global lightning fatality studies estimate 6000 fatalities and 24,000 fatalities per year.[59,198] Underreporting of lightning fatalities, and especially injuries, results from a number of factors, including the situation that many events involve only one person and may not be reported or considered newsworthy.[260] In past population studies, the practice has been to assume that injuries are 10 times as frequent as are fatalities,[67] although this may not be the case in developing countries. There is a limited number of published formal and informal papers in recent years that summarize lightning fatalities on national scales. Even though single-incident cases dominate data sets from more developed countries, events with large numbers of deaths and injuries that may be more common often occur in developing countries. In addition, it is not uncommon in developing countries for multiple fatalities to occur in what would be considered an 'indoor' environment.

Figures 5-12 through 5-17 summarize all published national studies of fatality rates, with low lightning fatality rates in yellow, medium in orange, and red for highest. Fatality rates by decade since the 1800s have been published,[189] but no summary over recent years has been made. The most common shading is white, which indicates that no national lightning casualty summaries have been published. It is apparent that the highest rates are in Africa and South America. The lowest rates are evident in North


**FIGURE 5-12** Lightning fatality rate per million people per year in Africa. *Red* shading indicates rate >5.0 fatalities/million/yr, and *orange* is 0.6 to 5.0. *White* indicates no national summaries have been published for data sets ending in 1979 or later.


**FIGURE 5-13** Lightning fatality rate per million people per year in Asia. *Orange* shading indicates rate of 0.6 to 5.0 fatalities/million/yr, and *yellow* shading indicates <0.5. *White* indicates no national summaries have been published for data sets ending in 1979 or later.

America, Western Europe, Japan, and Australia. This is probably because of the availability of more substantial housing, metal vehicles, lightning education, and lower lightning density in temperate zones. Regional, local, short-period,[1] and Internet-based reports are not included. The most recent published results include the following:

- Africa: South Africa,[47] Malawi,[282] Swaziland,[128] Uganda,[5] and Zimbabwe[81,376]
- Asia: China,[262,398] India,[207] Japan,[224a] Malaysia,[2] and Singapore[306]
- Australia[84]
- Europe: Austria,[231] France,[159] Greece,[311] Lithuania,[149] Poland,[256] Turkey,[371] and United Kingdom (including Ireland and Wales)[133,134]
- North America: United States,[114,200,259] Canada,[275] and Mexico[322]
- South America: Brazil[59] and Colombia[288]


**FIGURE 5-14** Lightning fatality rate per million people per year in Australia. *Yellow* shading indicates rate <0.5 fatalities/million/yr. *White* indicates no national summaries have been published for data sets ending in 1979 or later.

Figure 5-18 shows Global Lightning Network (GLD360) strokes for 2011 to 2014.[317,318,351] Unlike prior lightning detection networks, which did not provide good data over large bodies of water, GLD360 is a real-time network that seamlessly covers continents as well as oceans to show lightning on monthly, regional, and annual scales. Because 80% of GLD360 lightning events are cloud-to-ground strokes, the actual lightning threat is now much better known around the globe. Land areas clearly comprise most of the lightning threat to people and infrastructure.

Lightning is not uniformly distributed. Some locations on land, especially along the slopes of tall mountains and along tropical coastlines, have much more lightning at certain times of day and year than do others. This knowledge can be useful to improve understanding of lightning threat. Studies have been made in a number of countries of the variability of lightning in time and space. The opportunity now exists for global studies using lightning detection data sets with substantially uniform areal detection.

## TRENDS IN LIGHTNING FATALITIES

The maps show that the highest population-weighted annual rates of lightning fatalities occur in countries with the following features:

- Fewer lightning-safe dwellings, workplaces, school, and other facilities than in more developed countries
- Fewer easily available, fully enclosed, metal-topped vehicles
- High rate of labor-intensive manual agriculture, mining, and other activities
- Lack of awareness or data about the lightning threat and its avoidance
- Unavailability or delay in medical treatment

The global population living in these situations may be increasing in absolute number, but statistics are difficult to obtain. Also, the trend toward a reduced percentage of the population in rural areas has not occurred in many areas of the world. For example, it has been reported that 97% of lightning fatalities in China occur in rural areas.[262] As a result, it is likely that the number of lightning fatalities and injuries globally is steady or may be increasing, which will continue until more people have ready access to safe dwellings, structures, and vehicles and spend less time in labor-intensive agriculture and other outdoor occupations. The lightning fatality and injury rates per population are

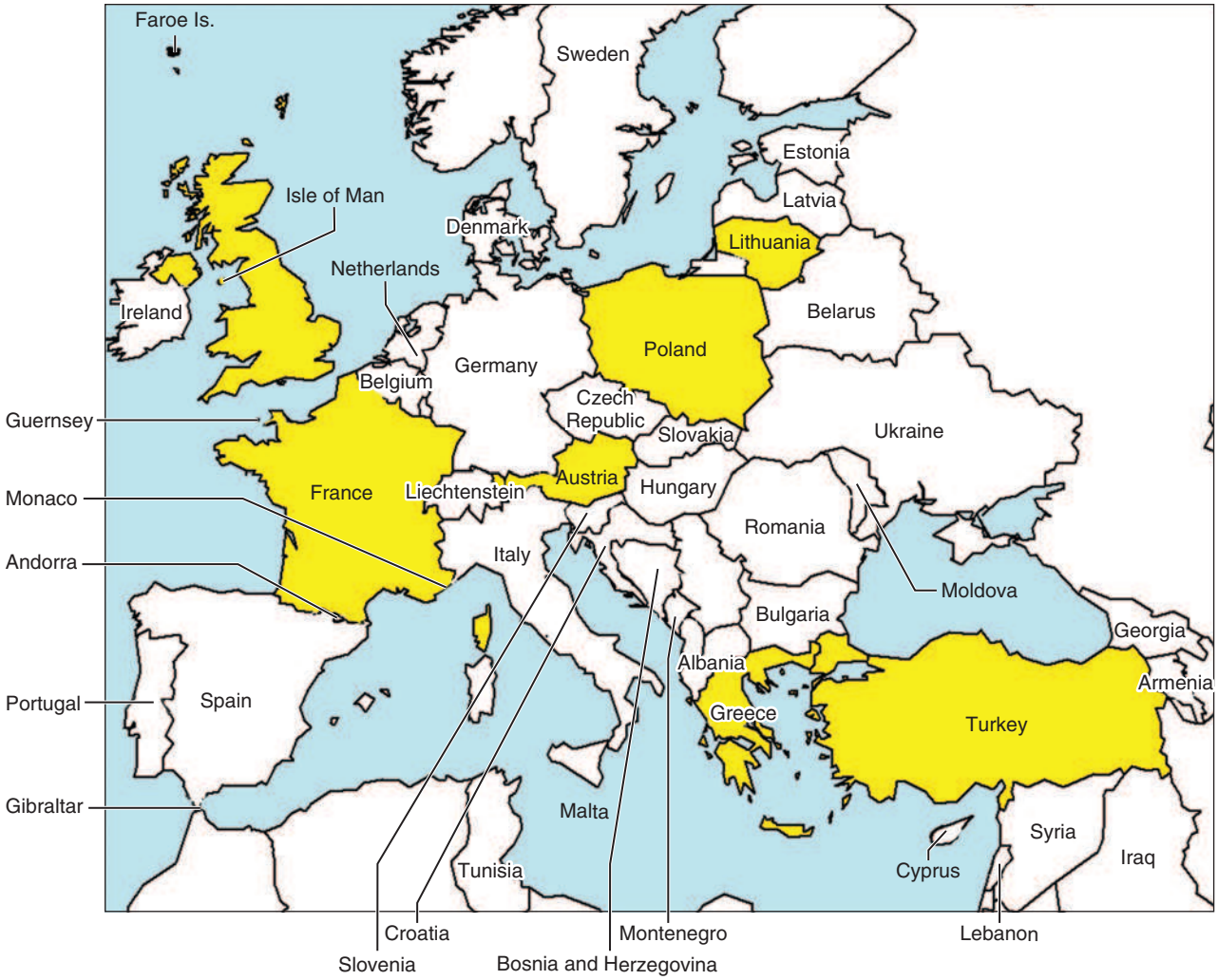


**FIGURE 5-15** Lightning fatality rate per million people per year in Europe. *Yellow* shading indicates rate <0.5 fatalities/million/yr. *White* indicates no national summaries have been published for data sets ending in 1979 or later.

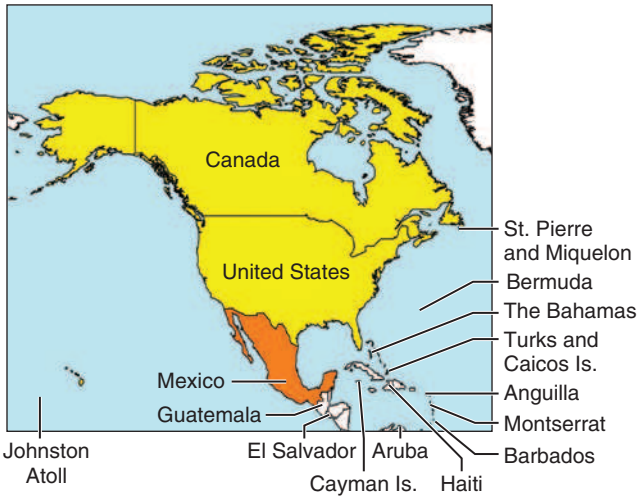


**FIGURE 5-16** Lightning fatality rate per million people per year in North America. *Orange* shading indicates rate of 0.6 to 5.0 fatalities/million/yr, and *yellow* is <0.5. *White* indicates no national summaries have been published for data sets ending in 1979 or later.


**FIGURE 5-17** Lightning fatality rate per million people per year in South America. *Orange* shading indicates rate of 0.6 to 5.0 fatalities/million/yr. *White* indicates no national summaries have been published for data sets ending in 1979 or later.

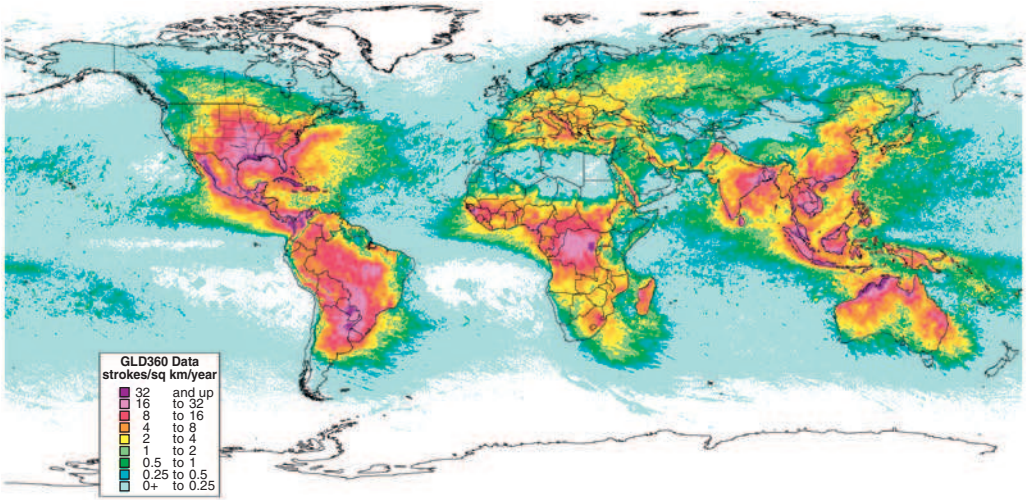


**FIGURE 5-18** Map of 3,639,467,075 Global Lightning Dataset GLD360 strokes for 3 years through December 2015. *(Courtesy Vaisala Inc.)*

thought to be very high in many of the countries that have no published national fatality data shown in Figures 5-12 through 5-17. For example, India is documented to have an average of 1755 deaths per year from 1967 to 2012,[207] which represents 78,975 fatalities during this period.

## CONCLUSIONS

The impacts of lightning vary greatly between developed and less developed countries. In the United States the population-weighted rate of lightning fatalities and injuries has greatly decreased from a maximum approximately a century ago. The two priorities in less developed countries to achieve a decrease in lightning deaths and injuries are protecting people working in labor-intensive agriculture and providing lightning-safe dwellings, buildings, and vehicles. Data from global lightning detection networks can help identify areas with the highest density of lightning. Through a combination of sound science, education, and uniform global lightning data, advances can be made to reduce the impacts of lightning on people and assets. To achieve improvements, the infrastructure of these countries likely must be significantly improved, accompanied by inexpensive lightning protection systems that can be easily installed and maintained.

Initial activities have been taken with regard to education and awareness of lightning threat in several Asian countries through the Centre of Excellence on Lightning Protection (CELP)[96,156,157,212] and are beginning in Africa by the newly formed African Centres for Lightning Electromagnetics (ACLE).

# EARLY SCIENTIFIC STUDIES AND INVENTION OF THE LIGHTNING ROD

The study of electrical phenomena is often traced to publication of Gilbert's *De Magnete* in London in 1600. Experiments in France and Germany and by members of the Royal Society of London led to invention of the Leyden jar in 1745.

Benjamin Franklin is generally regarded as the father of electric science and during his lifetime was known as the American Newton. He was accepted into the French and English courts around the time of the American Revolution not because he was an ambassador from America but because he was considered one of the foremost scientists of his time. Franklin was elected to every major scientific society at the time and received medals of honor from France and England for his scientific contributions.[83,325] Before his work, it was thought that two distinct types of electrical phenomena existed. Franklin's work[148] unified these two aspects and is responsible for renaming them "positive" and "negative." He went on to prove that lightning is an electrical phenomenon and that thunderclouds are electrically charged, as demonstrated by the famous kite and key experiment.[147] Because of the damage he saw to buildings, he invented the lightning rod and announced its use in 1753 in *Poor Richard's Almanack:*

> It has pleased God in his Goodness to Mankind, at length to discover to them the Means of securing their Habitation and other Buildings from Mischief by Thunder and Lightning. The Method is this: Provide a small Iron Rod (It may be made of the Rod-iron used by the Nailers) but of such a Length, that one End being three or four Feet in the moist Ground, the other may be six or eight Feet above the highest Part of the Building. To the upper End of the Rod fasten a Foot of brass Wire the Size of a common Knitting-needle, sharpened to a fine Point; the Rod may be secured to the House by a few small Staples. If the House or Barn be long, there may be a Rod and Point at each End, and a middling Wire along the Ridge from one to the other. A House thus furnished will not be damaged by Lightning, it being attracted to the Points, and passing thro the Metal into the Ground without hurting any Thing. Vessels also, having a sharp pointed rod fix'd on the Tops of their Masts, with a Wire from the Foot of the Rod reaching down, round one of the Shrouds, to the Water, will not be hurt by Lightning.

In the 1750s and 1760s, use of lightning rods became prevalent in the United States for protection of buildings and ships. Some scientists in Europe urged installation of lightning rods on government buildings, churches, and other tall buildings. Religious advocates at the time, unfortunately, maintained that it would be blasphemy to install such devices on church steeples, which received "divine protection." Some groups chose to store munitions in churches, leading on more than one occasion to significant destruction and loss of life when the buildings were struck by lightning.

Part of the delay in installing lightning rods in England has been attributed to distrust of scientific theories originating in the upstart, newly independent United States. Years and numerous unsuccessful trials with English designs were required before the Franklin rod became accepted on Her Majesty's ships and buildings.[58]

At one time (and still believed by some laymen today), lightning rods were theorized to diffuse electrical charge, neutralizing storm clouds and averting lightning. This may have been an outgrowth of the observation of St Elmo's fire, an aura appearing around the tip of lightning rods, noses of aircraft, and ships' masts during a thunderstorm, caused by an electron discharge that results from the strong electromagnetic field induced around the glowing object. Properly installed lightning rods and lightning protection systems neither "diffuse" nor "attract" lightning, but rather protect a building by providing a preferential attachment

**FIGURE 5-19** Damage to the roof of a clubhouse from direct lightning strike. The damage was extensive and included structural damage to the clubhouse. *(Copyright Ian R. Jandrell.)*

**FIGURE 5-21** Damage to curtaining material covering a window of the clubhouse in Figure 5-19. *(Copyright Ian R. Jandrell.)*

point for the lightning stroke, allowing the current to be harmlessly directed through the system to the ground.[52,291,295] Lightning can otherwise travel into or through the building and cause extensive damage (Figures 5-19 to 5-21).

The first Lightning Rod Conference was held in London in 1882. Recommendations from this conference were published that year and again in 1905. Several countries developed codes of practice for lightning protection (Germany, 1924; United States, 1929; Britain, 1943; British colonies, 1965).[115] In the past decade, NFPA 780 (U.S. National Fire Protection Act) has been accepted worldwide as a reliable standard.[291]

Building codes and industrial standards may require particular structures to have lightning protection systems.[52,291] Including a system in the initial design and construction is always easier and less expensive than modifying a completed building. Other factors to be considered include relative frequency of strikes in an area; height, construction, and design of the building; and degree of protection desired, depending on whether the building is a storage shed, house, school, hospital, or munitions factory. In cases not covered by code, a lightning protection system, despite high exposure to lightning, may not be worth the expense, such as for a mountain cabin that is seldom visited.[228,295]

At present, the most important economic impacts from lightning include electrical utility interruption; interruptions at airports and other outdoor locations; loss or corruption of financial, security, and other databases and control systems; and downtime of industrial equipment. For humans, the impact of being struck by lightning includes loss of work ability, unemployment, relationship decay, depression, anxiety, and loss of cognition, and this is of economic importance as well. Lightning may pose significant danger not only to individuals, but also to larger groups, such as when hundreds of miners are trapped deep underground after lightning has made elevators, ventilation systems, and water pumps nonoperational.[295]

At home, the most reliable way to protect electronic equipment is to unplug it from the wall before arrival of a


**FIGURE 5-20** Damage to the ceiling (**A**) and internal wall (**B**) of the clubhouse in Figure 5-19 with resultant damage to the electrical and electronic devices connected to the electrical system. *(Copyright Ian R. Jandrell.)*

thunderstorm. Surge protectors, which may be effective for minor household electrical surges, are seldom completely effective in eliminating the effects of lightning, despite manufacturers' claims. Lightning protection needs to be done correctly, and it is recommended that it be installed by a licensed, bonded, insured, and experienced specialist using proven methods according to accepted codes. The best source of information on lightning risk for people is the Lightning Protection Institute (www.lightning.org).

# PHYSICS OF LIGHTNING STROKE

## LIGHTNING DISCHARGE

The study of lightning discharge and formation is complex and has led to development of a separate specialization within physics and meteorology This section describes the simplified and most common mechanisms of thundercloud formation and lightning strike.

Thunderstorms can be formed in a number of ways to produce the necessary vertical updrafts. These ingredients are afternoon heating of warm moist air, large-scale upward atmospheric motions (Figure 5-22A), sea and lake breezes, lifting of deep layers of the atmosphere by mountains, and cold fronts.[188,194]

As warm air rises, turbulence and induced friction cause complex redistribution of charges within the cloud (Figure 5-22B). Although the ground temperature may be very warm to hot, thunderstorms are tall enough that their highest parts are colder than freezing. In fact, all lightning comes from clouds that have ice aloft at temperatures colder than freezing; at these temperatures, water droplets and ice particles of several types within the cloud acquire and increase their individual charges as they interact and transfer charge. The varying sizes and shapes of the frozen snow and ice crystals, supercooled water droplets, and hail are moving vertically at different speeds because of their different fall speeds. The result is separation of charge into several layers. A large potential difference develops between layers as a result of interaction of charged water and ice particles, updrafts that vary in time and space, and internal and external electric fields within the cloud.

Lower layers of the cumulonimbus cloud generally become negatively charged relative to the earth. The earth, which normally is negatively charged relative to the atmosphere, has a strong positive induced charge as the negatively charged thunderstorm passes overhead. The induced positive charge tends to flow as an upward current from trees, tall buildings, poles, people, or sometimes very small objects or flat open ground beneath the overhead thunderstorm cloud and may move up in upward-propagating leaders.[325]

Normally, the discharge of the potential difference is discouraged by the strong insulating nature of air. However, when the potential difference between charges within the clouds or between the cumulonimbus cloud and the ground becomes too strong, the molecular structure of the intervening air may break down under the influence of the electric field that has developed, and the charge is then dissipated as lightning.

A downward stroke begins as a relatively weak and slow downward leader from the cloud (Figure 5-22C). Although the

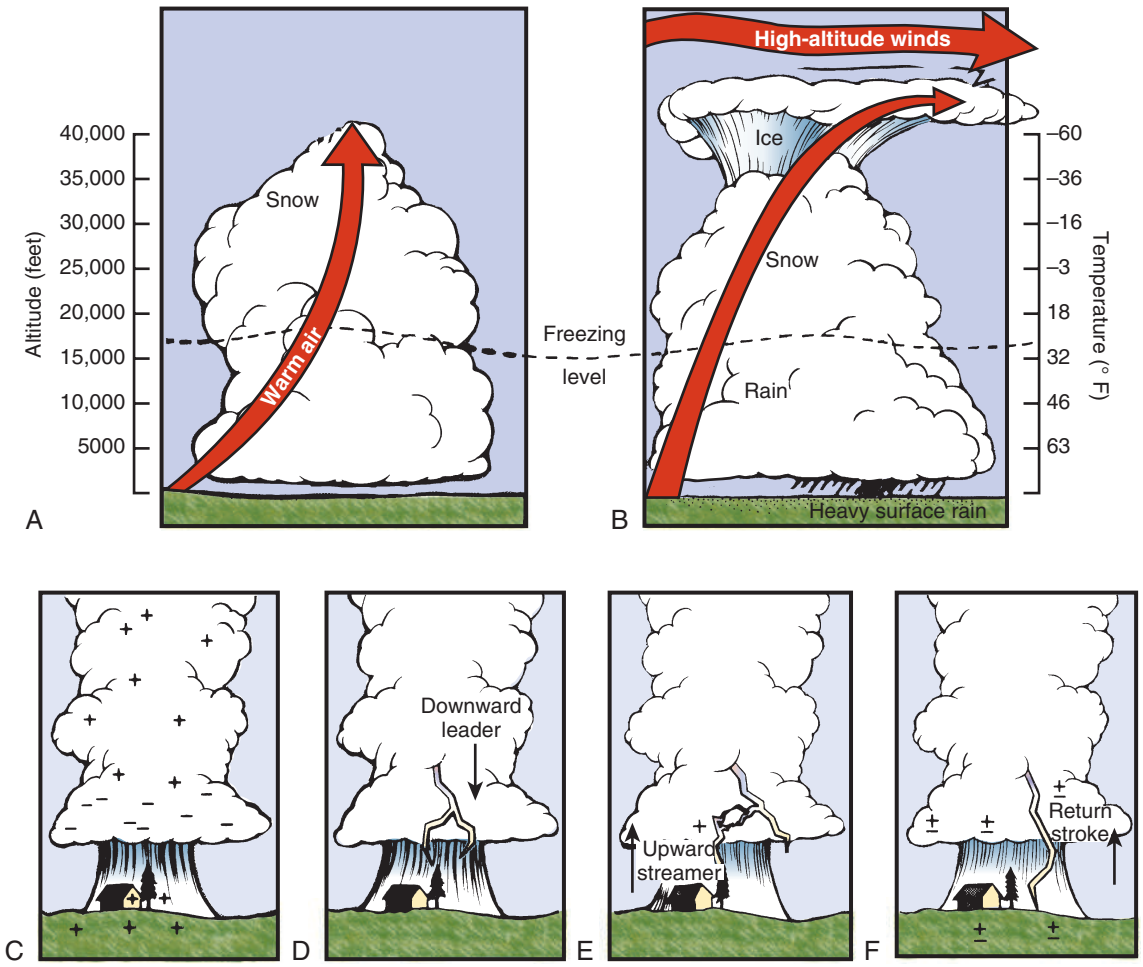


**FIGURE 5-22** **A,** Air rises and condenses into a cumulonimbus cloud. **B,** Typical anvil-shaped thundercloud. **C,** Water droplets within the cloud accumulate and layer charges. **D,** Stepped downward leader initiates the lightning strike. **E,** Positive upward streamer releases from the ground to meet the stepped leader. **F,** Return stroke travels back up the channel made by the stepped leader.

tip of the leader may be luminous, the stepped leader itself is barely discernible with the unassisted eye. Very-high-speed video has shown this bright tip and a more faint trailing leader as it travels to the ground.[349,353] The leader travels at about one-third the speed of light ($1 \times 10^8$ m/sec), and the potential difference between the leader's lower tip and the earth ranges from 10 to 200 million volts (V). The leader ionizes a pathway that contains superheated ions, both positive and negative, and forms a plasma column of very low resistance. The leader travels in relatively short branched steps downward about 50 m (164 feet) and then retreats upward. The next time the leader goes downward toward the ground, it fills the original ionized path but branches at the end to go down another 50 m and then retreats again. This up-and-down multiple-branching process continues until the leader comes to within 30 to 50 m (98 to 164 feet) of the ground. Because lightning follows this ionized path, its lower tip only can sense the existence of nearby objects within a radius of about 30 to 50 m, meaning that lightning will not be affected by the existence of a hill or tower farther away. For human safety, being within 50 m (164 feet) of the lowest tip of a cloud-to-ground lightning flash as it comes to ground is extremely unsafe.

## DIAMETER AND TEMPERATURE OF LIGHTNING

Although many techniques have been used to measure the diameter and temperature of lightning, all measurement techniques have artifact problems. Visual measurements of the lightning stroke using standard photography usually show the diameter of the main body of the stroke to be about 2 to 3 cm (0.8 to 1.2 inches).

The diameter of the channel is sometimes measured indirectly, using measurements of holes and strips of damage that lightning produces when it hits aircraft wings, buildings, or trees. Measurements vary from 0.003 to 8 cm (0.001 to 3.15 inches), depending on the material that was destroyed. Hard metallic structures sustain smaller punctures than do relatively softer objects such as trees. The ionized sheath around the tip of the bright leader stroke has not been measured but is estimated to have a diameter of 3 to 20 m (9.8 to 66 feet).

The temperature of the lightning stroke varies with the diameter of the stroke and has been calculated to be approximately 8000°C (14,400°F). Other estimates of the temperature are as high as 50,000°C (90,000°F). After a few milliseconds, the temperature falls to 2000° to 3000°C (3600° to 5400°F), similar to the temperature of a high-voltage electric arc.

## FORMS OF LIGHTNING

Lightning can be divided into cloud-to-ground and cloud (intracloud) flashes (Figure 5-23). Cloud-to-ground flashes contain one or more return strokes. The flickering often seen in a cloud-to-ground flash is caused by the return strokes, which average three to four per flash. Cloud-to-ground flashes contact the surface of the earth at one or more locations, because one of the subsequent return strokes often takes a different path to the surface, up to a few kilometers from the first return stroke; the average is 1.47 ground contact points per flash.[367] The term *total lightning* describes the sum of cloud-to-ground and cloud flashes. An extremely small portion (<0.01 of 1%) of all lightning travels from ground to cloud when this is induced by the special circumstances of high towers or mountains.

**FIGURE 5-23** Cloud-to-ground flash *(right)* and cloud flash *(center)*. *(Copyright Ronald Holle.)*

Cloud flashes can travel between clouds, within clouds, from cloud to cloud, and in all combinations of these paths. The same flash can simultaneously strike ground at one or more locations and travel a long distance in-cloud (Figure 5-23). There are several times as many cloud flashes as those that reach the ground. Cloud flashes have been measured to extend 305 km (189 miles) in horizontal length and last 5.7 seconds.[244] From the point of view of a person on the ground, cloud flashes may appear to travel in long streaks across the sky, or the channels may be obscured and visible only by the brightening within clouds along their paths. Such cloud flashes are seen more often in regions with low cloud bases in humid areas.

The most unusual and least understood type of lightning is *ball lightning*. It is usually described as an orange, blue, or white globe between the size of a softball and a basketball. It has been observed to enter planes, ships, or houses and to travel down narrow spaces such as hallways. Ball lightning very infrequently injures people and objects and often exits through a door, chimney, or window.[313] It may explode with a loud bang or exhibit other bizarre behavior.

Cloud-to-ground lightning flashes usually lower negative charge to the ground.[325] The less frequent (5% to 10%) positive cloud-to-ground flashes tend to occur during the winter, at the end of thunderstorms, on the U.S. high plains, or in relatively shallow thunderstorms. Positive flashes usually have one return stroke. It is not known if positive cloud-to-ground flashes cause a different injury profile, although they tend more to have long, continuing current that may impart more energy to a person than do the usually shorter-pulsing, negative cloud-to-ground flashes.

## THUNDER

Thunder is formed when shock waves result from the almost explosive expansion of air heated and ionized by the lightning channel. Understanding some basic features of thunder is important because of its usefulness in many lightning safety recommendations.[295,384] The following are accepted features of thunder:

- Cloud-to-ground lightning flashes produce the loudest thunder.
- Thunder is seldom heard more than 16 km (10 miles) away, except under extremely quiet conditions.
- The time interval between the perception of lightning and the first sound of thunder can be used to estimate the distance from the lightning channel, because the sound travels at a rate of 3 sec/km (5 sec/mile).
- Wind, rain, man-made noise such as traffic, vegetation, and intervening buildings, hills, and mountains reduce audibility of thunder.
- The pitch of thunder deepens as the rumble persists, because only lower-frequency sounds remain at greater distances.
- Atmospheric turbulence reduces audibility of thunder.

The thunderclap from a close lightning flash is heard as a sharp crack. Distant thunder rumbles as the sound waves are refracted and modified by the thunderstorm's turbulence. Because there is a large difference between the speed of light and speed of sound, the distance to lightning can be estimated by a person on the ground. The estimation is made by the flash-to-bang method of counting the seconds between seeing a flash and hearing thunder from the same flash, when the two can be matched. The time interval between lightning and thunder is 3 sec/km (5 sec/mile). For example, if the difference is 30 seconds between when a flash is seen until its thunder is heard, the flash is 10 km (6.2 miles) away. This time interval is part of the basis for the 30-30 rule described later in Precautions for Avoiding Lightning Injury.

# MECHANISMS OF INJURY BY LIGHTNING*

## ELECTRICAL INJURY PHYSICS REVISITED

In many texts dealing with electrical injury, Kouwenhoven's six factors (AC vs. DC, voltage, amperage, duration, pathway, and resistance) are often discussed. However, these factors are neither useful clinically nor useful predictively and have led more to confusion and misunderstanding of electrical and especially lightning injuries than to understanding or usefulness in the medical setting. In addition, simplistic application of this list leads to even more misunderstanding by attorneys, insurance companies, and others regarding the disabilities that typically occur, with the unfortunate outcome of denial of legitimate claims for real injuries and their sequelae.

## CONCEPTS IN ELECTRICITY

Voltage can be regarded as an external force or pressure applied to an object to force it to conduct electric current, like water pressure applied at one end of a pipe to cause water flow through the pipe. When voltage is applied, a current (measured in amperes) flows through the conductor (i.e., the object). The amount of current is inversely proportional to the resistance of the object. For a given applied voltage, the higher the resistance of the object, the smaller the resulting current. For technical or generated electrical injuries, the voltage is externally selected, the resistance of an object is a given, and the current is the result.

Resistance is a given property of an object. It can be thought of as the ease or difficulty with which something flows through the object and is analogous to the diameter of the water pipe or the friction within the pipe. Resistances of various tissues in the body have been measured, but these change as integrity of the tissue changes. If it is assumed that resistance is predictable, then resistance, voltage, and current can be linearly related, making modeling, and therefore prediction, possible. Voltage, current, and resistance are related by Ohm's law (voltage = resistance × current). However, as tissue reacts to injury, resistance changes. Although theoretically these changes can be modeled for tissues, the uncertainty that creeps into nearly every clinical situation makes application of these calculations to explain or predict injury to an individual patient much too complex and fraught with error to be used on a day-to-day basis. Even worse, complex calculations such as these can easily be misapplied in a court of law, taking the focus away from objective clinical evidence of injury and disability.

Another treatment of Ohm's law would be to specify current and resistance, making voltage the calculated or measured outcome. Generated electricity is in the first category, a "voltage-driven" phenomenon, where the equation is driven by voltage. Resistance changes as injury occurs, and current is the calculated or measured dependent variable. Lightning is quite different and in the second category, a "current-driven" phenomenon. Although the simplest form may be governed by Ohm's law, the resistance of the body is not uniform. The result is that advanced calculation methods must be used to find the voltage across the body when struck and subjected to lightning current, including the phenomenon of *flashover*. To make the situation even more complex, once lightning attachment occurs and an open channel is made, the voltage drops to zero, making Ohm's law meaningless as an avalanche of current begins to flow.

Another concept to consider is that of an electric field. When a voltage is applied across a definite area of space (e.g., the "gap" between a cloud and the ground), an electric field results. Air generally has a high resistance and is a good insulator, conducting very little current under these circumstances. *Electric field* is defined as the voltage across the gap, divided by the width of the gap. If the magnitude of the field is increased, it reaches a size where the intervening insulator will "break down." That is, an avalanche of electrons will occur in the gap. Before this happens, a minor amount of current will be conducted under the influence of the field. Beyond this point, new processes come into play, causing the physical structure of the conducting medium to be disrupted. The actual moment that this disruption occurs varies with the voltage applied and width of the gap. The voltage necessary to flash over an air gap is about 4000 V/cm (10,000 V/inch), depending on humidity and other factors. A large, noisy flashover may be observed if the gap is sufficiently large and rapid expansion of superheated air forms the thunderclap.

### Technical Electricity Supply vs. Lightning Current

Technical electricity is provided to a household at a constant voltage; current flows in devices connected to this supply. Flashover is rare at relatively low voltage in domestic use. The behavior of any current that results is much more linear, although not perfectly so when it involves the human body. The possibility for conduction of large internal currents exists, and for longer periods of time, because flashover is absent. Heat generated by this current is much more likely to result in burning. Conduction is likely to be much more prolonged. Resultant muscular reactions for a prolonged period locks muscles through contraction to the source, prolonging the contact. Prolonged conduction internally can expose the heart to prolonged, damaging passage of current. Ventricular fibrillation (VF) arises proportionate to the amount of conducted current and the duration of time over which it is conducted. The current flowing for a given time allows one to estimate the likelihood of VF.[205] A victim who is still in contact with the electrical source continues to be dangerous for the human rescuer to touch.

Lightning current, on the other hand, is of very short duration (microseconds). Burning is therefore minimal. Flashover is highly likely, making internal conduction minimal. Once the discharge is completed (fractions of a second), a victim is safe to touch. Current, though short-lived, is large. Some sequelae of both types of current passage are similar otherwise, particularly the psychological sequelae.

## MECHANISMS OF INJURY

### General Conduction Effects

Lightning is dangerous to humans predominantly because of heat and less because of concussive force. Lightning may injure indirectly through forest fires, house fires, explosions, or falling objects. Only injuries directly caused by lightning are discussed here. When lightning current is injected into an individual, current is initially transmitted directly through the individual for microseconds until internal structures (capacitances) become charged, and flashover occurs over the surface of the individual as the breakdown field is reached. After that, internal current reduces dramatically.[301]

Lightning current may initially be inflicted on a person in several ways, described in more detail later (Box 5-3 and Figure 5-24).[226] The internal current phase may cause cardiac and respiratory arrest, particularly if the pathway primarily includes the heart. As the body's electric potential builds up in response to

**BOX 5-3 Mechanisms of Lightning Injury**

**Electrothermal Effects**

1. Direct strike
2. Contact potential
3. Side flash, sometimes called "splash"
   (1 to 3 may include surface arcs over the body surface.)
4. Step voltage (also termed *earth potential rise* or *ground current*)
   a. Transmitted through the ground
   b. Surface arcing
5. Upward streamer current[3,75] (also called *fifth mechanism*)

**Blunt Force Trauma Effects**

6. Barotrauma
7. Concussive injury
8. Musculoskeletal injury from muscle contraction; falls

*References 8, 10, 26, 27, 41, 63, 75, 91-93, 99, 104.

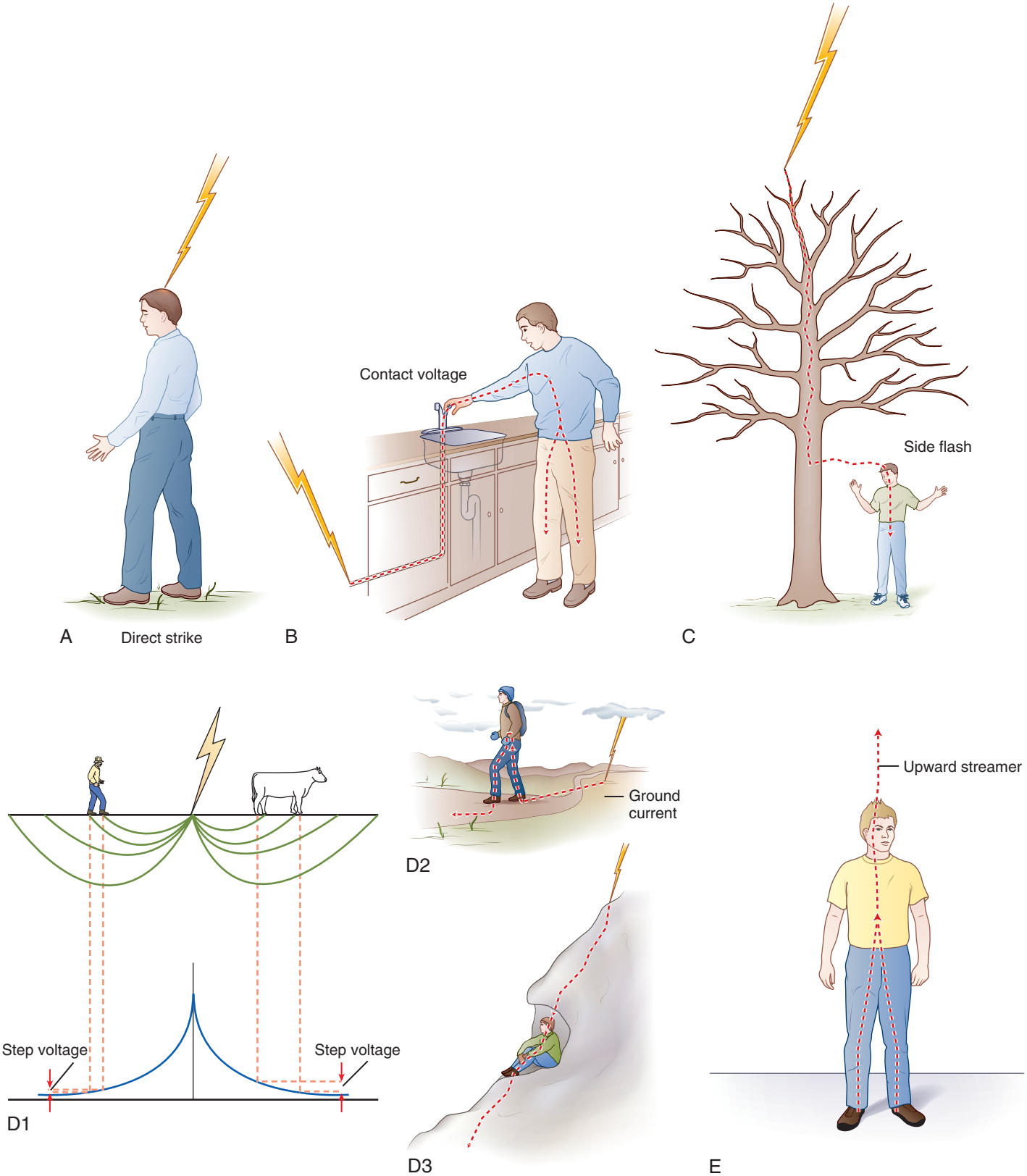


**FIGURE 5-24** Mechanisms of lightning injury. **A,** Direct. **B,** Contact. **C,** Side splash/flash. **D1,** Ground current through the earth. **D2,** Ground arcing. **D3,** Ground arcing cave. **E,** Upward streamer.

internal current, it produces an electric field over the surface of the body. Our understanding of what happens to a lightning-struck human is assisted by what happens with conduction in wood on telephone and electrical supply poles.[118] The consistency and resistance of wood are much closer to that of the human body than one might expect. This allows us to conclude that after flashover occurs, internal current drops dramatically. Current can be directed to be conducted within the wood or conducted externally over the wood, but not both ways. In wood pole design, metal implants from outside to inside the wood enhances the ability to swap between these routes. As the current continues to increase, the surface flashover bridges the strike

point and the ground. When this happens, most of the lightning current flows as an arc current through the air outside the body (flashover effect). Only a tiny fraction flows through the body and may not be sufficient to cause cardiac and respiratory arrest unless it has already done so.

## Specific Strike Mechanisms

There are five mechanisms by which lightning current may impinge on a body. Several authors have attempted to estimate the percentage of injuries connected with each mechanism; this is speculative[28,102,104] Similarly, mortality attributed to each mechanism is speculative and not derived from studies.

**Direct Strike.** A direct strike occurs when the lightning stroke attaches directly to the victim (Figure 5-24A). This most often occurs in the open when a person has been unable to find a safe location and occurs in no more than 3% to 5% of fatalities in developed countries. Even though it seems intuitive that direct strike would be the most likely to cause fatalities, there are no studies on the relative fatality rate for each strike mechanism. This low rate of occurrence is estimated on the basis of examination of thousands of cases (Figure 5-25).[101]

**Contact (Touch Potential) Injury.** Contact injury, or touch potential injury, occurs when a person touches or holds onto an object to which lightning attaches. A voltage gradient is created on that object from strike point to ground or to another point of contact, and the individual in contact with the object is subject to the voltage between his or her contact point and the earth (Figure 5-24B), resulting in a current flowing through them. Contact injury probably occurs also in about 3% to 5% of strikes.

Static electricity may be discharged when a person reaches for a car door or stands close to a metal window or door frame during a thunderstorm, because the surrounding electric field induces static electrical charges. These are not lightning injuries. Although people may be startled when this happens, these discharges are unlikely to be any more dangerous than static electricity discharges experienced in the winter months from shuffling across the carpet and reaching for a door handle.

**Side Flash.** A more frequent cause of injury, accounting for as many as 30% of lightning-related fatalities, is a side flash, also termed "splash." Side flashes occur when lightning that has hit an object, such as a tree or building, travels down that object before a portion "jumps" to a nearby victim (Figure 5-24C). Safety protocols stress that standing under or close to trees or other tall objects is dangerous and to be avoided. Current divides itself between the two paths in inverse proportion to their resistances. Side flash may also occur from person to person.

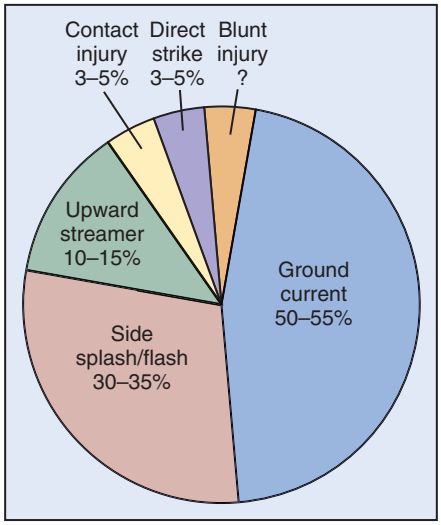


FIGURE 5-25 Chart showing the frequencies of primary lightning fatality mechanisms. *(From Cooper MA, Holle RL: Mechanisms of lightning injury should affect lightning safety messages. Preprints, International Lightning Meteorology Conference, Orlando, Florida, Vaisala, 5 pp.)*

**Earth Potential Rise.** Also called "step potential," "ground current," and a number of other similar terms, earth potential rise (EPR) occurs because the earth, modeled ideally as a perfect conductor, is not so in reality. When lightning current is injected into the earth, it travels through the earth just as it would within any other conductor. Earth has a defined resistance, and thus voltages are set up in the ground, decreasing in size with distance from the strike point. The voltage (or potential) of the earth is raised, thus the term. EPR can damage a person in several ways. If a person is standing in an area where EPR is active, that is, near the location of a cloud-to-ground lightning strike, a voltage will appear between the feet, and current will flow through the legs into the lower part of the body. Four-legged animals are likely to sustain even more serious damage if the current goes between back and front legs, where the path may involve the heart (Figure 5-24D1,D2).

A special case occurs when a person is injured inside a building as lightning strikes nearby.[11,30,125] The person, along with the environment (including the dwelling) around the person, is raised in potential via EPR. For example, if a telephone line is not locally earthed (grounded), the person touching the telephone is at the same voltage as the environment. Current is transmitted from the local EPR-raised environment to the remote unaffected earth using the human as the medium between the local environment and the distant earth.

All local electrical apparatuses, including telephones, should be well grounded locally. The grounds of all local structures (e.g., power, telephone, plumbing, structural steel) should have a common grounding point (i.e., should be bonded) to eliminate any voltage differences developing between separated ground points for each system. For the special case of indoor or telephone injury, EPR may account for 80% or more of the injuries; all these relate to hard-wired phones. Cell phones, not being hard-wired or distantly grounded, provide no connection or EPR effect to a person during a lightning strike. Any practices that bring earth voltage more easily inside a dwelling will make this worse: plumbing, house base metal reinforcing, and high earth resistivity.

Kitagawa and colleagues[226] have identified further subdivisions of the EPR phenomenon. Not only can EPR occur as discussed, but it can also occur in a manner similar to the surface flashes over a body, with arcs developing over a ground surface (Figure 5-26). Despite mathematic modeling that assumes the earth is homogeneous, the grounding earth is not homogeneous and provides arc generation points.

Irregularities occur on mountainsides. If the terrain is markedly irregular, spreading lightning current may reach the surface. A surface arc discharge may develop along with flow of the conduction current in the ground. Because arcs carry considerable energy, a person exposed to a surface arc discharge is more likely to sustain a more severe effect, including thermal injuries, temporary paralysis, or even death. This mechanism of injury makes it particularly dangerous for someone on a mountainside to shelter inside a shallow cave or under a small cliff or outcropping of terrain, where surface arcing is much more likely to occur, injuring the person just as the person thinks some degree of safety has been achieved (Figure 5-24D3). All these factors illustrate the danger of being outdoors in the presence of lightning.

Ground current effects are more likely to be low level and less likely to produce fatalities. However, multiple victims with injuries are frequently found in an arc around the strike point to earth. Large groups have been injured on baseball fields, at racetracks, while hiking or camping, and during military maneuvers.[34,58,61] Shocks via telephones can produce significant long-term medical problems, and the majority of these are via EPR.[30,125,281]

**Upward Streamers.** Upward streamers generate the fifth mechanism of lightning injury.[8,61,91] Injury may occur when a victim serves as the conduit for one of the usually multiple upward leaders induced by a downward-stepped leader and its field (Figure 5-24E). Streamers occur even when there is no attachment between them and the stepped leader. Although one might think that these streamers are weak in energy compared

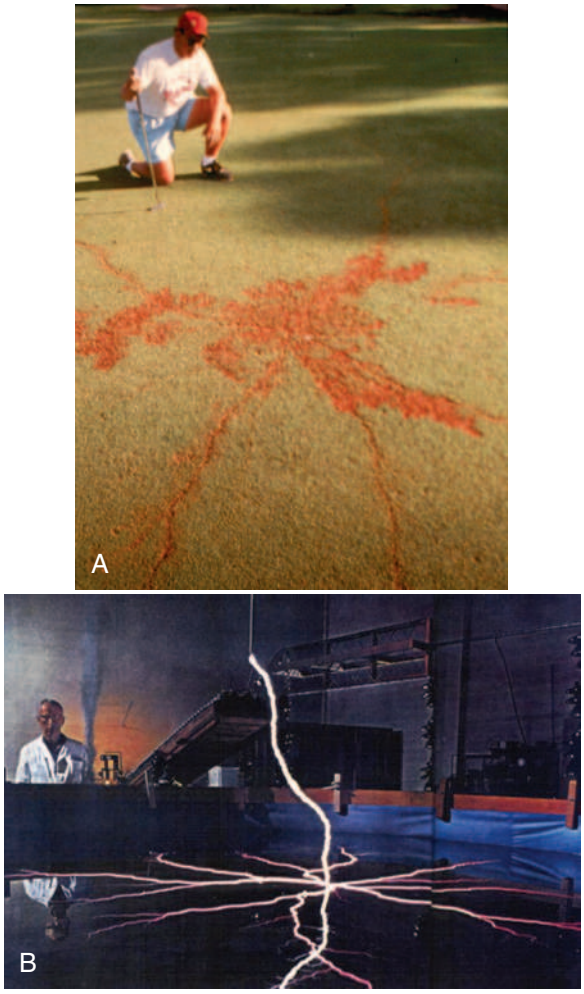


**FIGURE 5-26** **A,** Lightning injury and ground current effect to a golfing green, photographed a few days after the strike when the grass had died. **B,** Laboratory lightning onto a pool surface.

with the full lightning strike, they may carry significant and dangerous current through or around the victim. Upward streamer injury is probably the most underestimated mechanism of lightning injury.

It is difficult to separate the incidence of injury due to EPR from that attributed to upward streamers. Where direct strike may account for 5% of individual strikes, contact potential approximately 5%, and side flash approximately 30%, the combined incidence of EPR and streamer shock may be up to 60%. There are no better figures in the literature, and these represent a consensus, at least in terms of relative rankings. Many cases with multiple casualties are likely a combination of many of these effects, with the majority of them from EPR and upward streamers, sometimes complicated by side flashes if people or animals are standing too close together (Figures 5-27 and 5-28).

## Barotrauma and Blunt Injury from Lightning

The prior five mechanisms are strictly electrical mechanisms. Injuries may also be characterized by more indirect, nonelectrical mechanisms, such as concussive injuries, blast injuries, and blunt force injury from being thrown.

Persons may sustain blunt injury either by being close to the concussive force of the shock wave produced as lightning strikes nearby or if ground current or some other mechanism induces an opisthotonic contraction. Witnesses have reported victims being thrown tens of yards by either mechanism.

A curious but common and often diagnostic finding is the tearing, melting, magnetizing, or signs of arcing seen in clothing, watches, and other objects worn by the victim. Torn clothing may raise the suspicion of foul play if the incident was unwitnessed. The clothing is typically ripped as if by some internal explosion. Similarly, belts and boots may be ruptured from the inside by vapor explosion of sweat or water in socks.[355] There are two theories to explain this:

1. *Flash moisture vaporization theory*.[227,300,301] Blast injury results from the explosive vaporization of superheated water along the path of the surface flashover. Lightning blast injury to the skull, brain, and viscera has been demonstrated in animals.[300]
2. *Concussive/explosive force,* from being close to the lightning channel. As lightning superheats and the air expands explosively, a "pressure-shock wave" can occur. One can hear thunder from as far away as 14 km[225] to 25 km[325] (8 to 15 miles), indicating a tremendous amount of energy is involved in generation of thunder. Even before the noise is produced, a pressure blast wave can affect people close by the lightning channel. During a lightning strike, the channel temperature will be raised to about 25,000 kelvins (K) (24,727° C [44,541° F]) in a few microseconds, and as a result, the pressure in the channel may increase to several atmospheres (atm). The resulting rapid expansion of the air creates a shock wave. This shock wave can injure a person in the vicinity of the lightning flash.[112]

Halldorsson and Couch[169] believed that this explosion/implosion phenomenon may cause trauma that mimics blast injuries seen in bomb blast victims. The sudden, cylindrical-shaped pressure shock wave that arises from the rapid volume

**FIGURE 5-27** Example of contact and side flash lightning casualties.

**FIGURE 5-28** Example of side flash or "splash" lightning casualties.

FIGURE 5-29 Mechanism of concussive, explosive barotrauma.

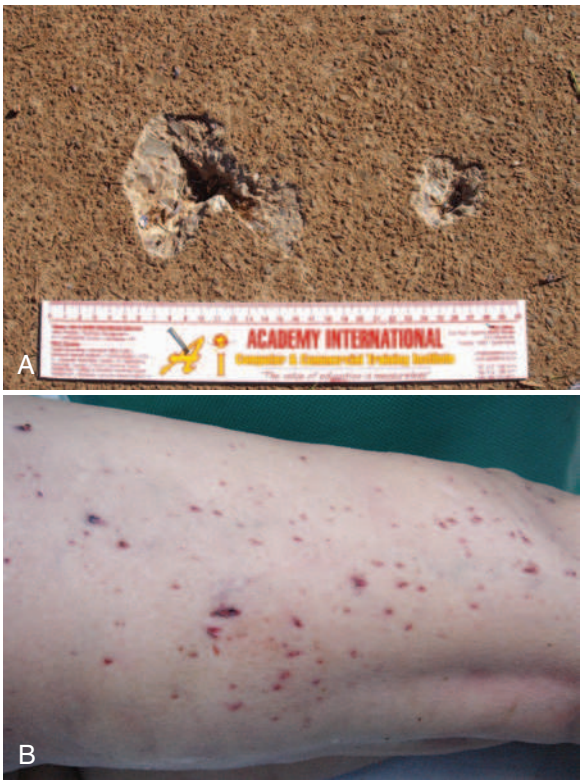


FIGURE 5-30 **A,** Concrete pavement that was struck by lightning. **B,** Secondary missile injury caused by lightning striking concrete pavement. *(Copyright the American Journal of Forensic Medicine and Pathology; from Blumenthal, R: Secondary missile injury from lightning strike,* Am J Forensic Med Pathol *33(1):83-85, 2012.)*

expansion may reach pressures of more than 10 to 20 atm (1013 to 2026 kilopascals [kPa]) in the vicinity of the lightning bolt channel[112,180,245,265,335,400] (Figure 5-29). It has been known to cause shrapnel injury; one victim had multiple small fragments of shattered concrete pavement embedded in her skin[49] (Figure 5-30).

Lightning's pressure blast wave has been reported to tear and tatter clothing, fracture bones, and cause tympanic membrane rupture and lung contusions. Tympanic membrane rupture may make the diagnostic difference in difficult cases. A study by Richmond and colleagues[334] suggests a minimum threshold of about 20 kPa to produce minor eardrum rupture. The threshold for lung damage occurs at about 103 kPa of blast overpressure.[152]

Pneumomediastinum and bleeding lung in a tracheostomized patient have both been reported.[50,169,280,365] As the blast wave impacts the human body surface, a pressure differential is generated at that surface, resulting in rapid acceleration and movements of the body surface, with propagation of shear and stress waves through the tissue[390] (Figure 5-31). The force of the pressure blast wave may be another mechanism that causes falls, in addition to head, brain, and other blunt trauma.

No evidence suggests that lightning victims sustain severe blast-related disfigurement, such as blast-related cavitation. About 690 kPa is the minimum threshold for serious damage to humans,[152,223,355] although, paradoxically, the human body can at other times survive relatively high blast overpressure without experiencing barotrauma.[266]

Theoretically, it is possible to estimate the distance from which a human could be at risk from lightning's pressure blast wave. A 4.5-kg TNT–equivalent bomb would rupture the eardrum of a 70-kg (154-lb) person within approximately 10 m (33 feet); lung damage would occur at about 5 m (16.5 feet), and the body would be injured at about 3 m (10 feet).[152]

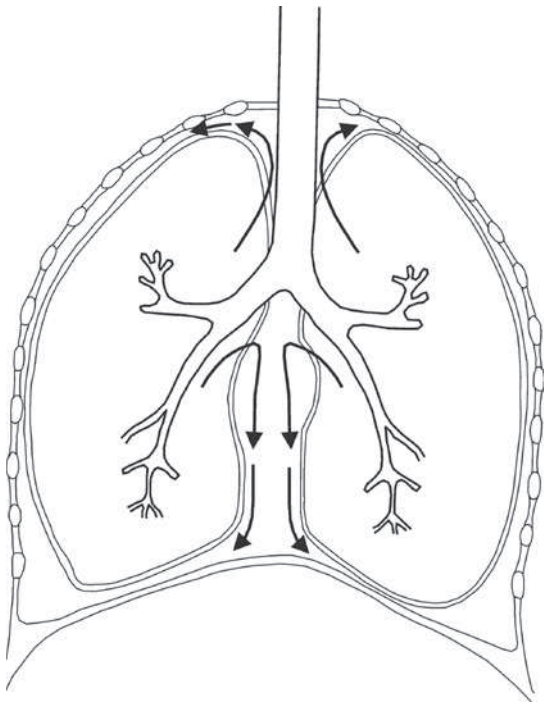

FIGURE 5-31 Graphic representation of the pathogenesis of traumatic emphysema (pneumomediastinum): Lightning's blast wave may cause widespread distribution of fine air bubbles within the mediastinal soft tissues, extending around the esophagus and pericardial sac. The theory is that barotrauma may cause disruption of the normal architecture of the pulmonary parenchyma, which may then cause air to dissect into the surrounding soft tissues. *(Graphic courtesy Ryan Blumenthal, Department of Forensic Medicine, University of Pretoria.)*

## PATHOPHYSIOLOGY OF LIGHTNING INJURY

This section describes the way in which lightning and electric current produces alterations in human physiology.

### ELECTRICAL INJURY PHYSICS REVISITED

This section examines the voltages, currents, and pathways that are estimated to occur in a lightning strike by the five mechanisms referred to above.[26,87,104,208]

#### Electric Field Effects

Figure 5-32 shows 20 kilovolts (kV) applied to a 1.8-m (6-foot) man, causing current to traverse source to ground. This produces

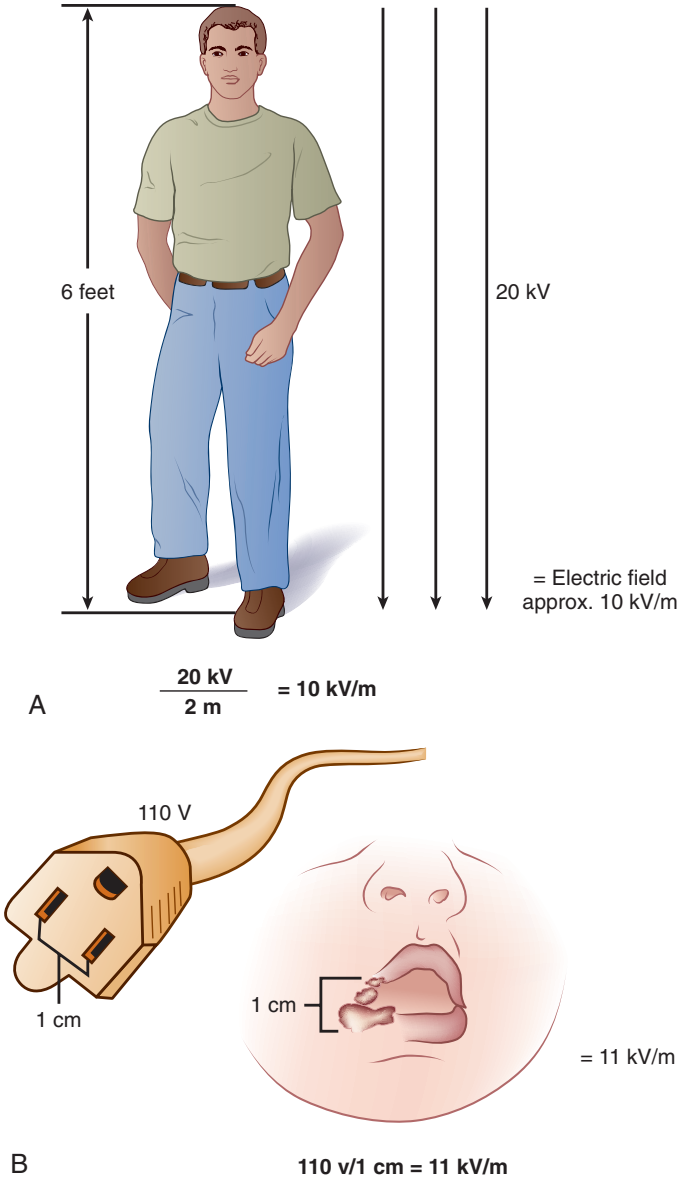


**FIGURE 5-32** **A,** If 20 kV is applied to a 1.8-m (6-foot) man source to ground, an electric field strength of approximately 10 kV/m is produced. **B,** When a child chews on the end of an extension cord, the applied voltage of 110 V across 1 cm produces an electric field strength of 11 kV/m—higher than the classic "high-voltage" injury. **C,** This explains the deep full-thickness burns the child receives, which one would not predict given the "low-voltage" source.

an internal electric field strength of approximately 10 kV/m. When a child chews on an electric cord and suffers a lip burn, the field strength is approximately the same 110 V applied to 1 cm (0.4 inch) of a child's lip and generates a field strength of 11 kV/m. Even though no one would classify the child's injury as being caused by "high" voltage, it is from a high electric field strength and produces the same tissue destruction in a small localized area, much as would a high-voltage injury to the standing man. In both cases, the voltage can be applied for an arbitrary amount of time. The difference is the distance over which the field is applied and the localization of the field. High electric fields of approximately this size, if applied for a sufficiently long period, can rupture cell membranes by generating pores in cell membranes (electroporation). Cell death, or at least dysfunction, results. Although Lee[247] has discussed the importance of internal electric field calculations in describing electroporation damage, this has not been studied for lightning injury, and the time scale is quite different.

## Characteristics of Lightning Current vs. Industrial Electricity

The danger of industrial electrical current versus lightning current is discussed earlier.

Industrial current is usually inflicted at low voltage, although voltage is a poor predictor of injuries.[82] It is usually alternating current (AC). If the supplied voltage is measured at any one location, the voltage swings negative and positive in a sinusoidal manner. Long ago, AC was chosen for electrical distribution because of ease of generation and transmission. Current flowing constantly in one direction is called direct current (DC) and is the current one encounters from a DC battery.

Lightning is neither direct nor alternating current. The best description of lightning is that it is a "unidirectional massive current impulse." The cloud-to-ground impulse results from breakdown of a large electric field between cloud and ground, measured in millions of volts. Once connection is made with the ground, the voltage difference between cloud and ground disappears, and a large current flows largely in a single direction impulse over a very short time. The study of massive electrical discharges of such short duration, particularly their effects on the human body, is not well advanced, although recently the threshold for VF induction for short duration impulses has been addressed.[22] Linear equations, such as Ohm's law ($V = I \times R$) and power calculation ($P = V \times I$), do not apply.

Energy dissipated in a given tissue is determined by the current flowing through the tissue and its resistance:

$$\text{Energy (heat)} = \text{Current}^2 \times \text{Resistance} \times \text{Time}$$

where a current flows through a resistance for a period of time. The energy is produced in the tissues, subject to the current, and largely appears as heat. This is often referred to as *joule heat* but is seen as temperature rises in the tissues if current is applied for a sufficiently long time.

As resistance increases, such as in the high resistance of skin, so does the heat generated by passage of the same current. In humans, when low current levels are encountered for a given time, much of the electrical energy may be dissipated by the skin, so that superficial burns are often not accompanied by internal injuries. Similarly, high-level current injuries, such as lightning, applied for a very short interval will cause little burn damage.

Although lightning occasionally creates what appear to be discrete entry and exit wounds, these are less severe and extensive than one would expect from a "high-voltage" injury. There is no voltage after lightning attachment occurs. Lightning more frequently causes only superficial streaking burns. Burns, discussed later, are a most important distinction between lightning and industrial electric shocks. The exception to this is when a long, continuing current stroke occurs. This is a prolonged stroke lasting up to 0.5 second that delivers a tremendous amount of energy, capable of exploding trees and setting fires. Other, poorly understood factors may contribute to the formation of deep burns, although deep burns similar to those seen with high-voltage electrical injuries are quite rare with lightning. Skin at the

site of a direct strike can also be mechanically or electrostatically disrupted. However, skin breakdown should not be the expectation for all lightning strikes, because direct strikes occur in only 3% to 5% of cases.[102] The absence or presence of skin breakdown should not be used to deny that injury occurred or to diagnose a direct strike clinically.

## ESTIMATION OF LIGHTNING CURRENTS

It takes a finite amount of time for skin to break down when exposed to heat or energy. Generally, lightning is not present long enough to cause skin to break down. A large portion of current travels along the outside of the skin as "flashover." Some experimental evidence indicates that a portion of the current may enter cranial orifices: the eyes, ears, nose, and mouth.[14] This pathway would help explain the myriad eye and ear symptoms reported with lightning injury.

Functional consequences of lightning on cardiorespiratory function showed that entry of current into cranial orifices allows passage of current directly to the brainstem. In a sheep study, specific damage to neurons at the floor of the fourth ventricle was demonstrated in the location of the medullary respiratory control centers.[12] Blood and cerebrospinal fluid (CSF) are preferential paths that conduct current to impinge directly on the myocardium. Histologic damage to the heart, consistent with a number of autopsy reports of inferior myocardial necrosis, was demonstrated in similar experiments.[12,32]

An alternative hypothesis can be tested with a mathematic technique.[26] Certain assumptions are made in any model, usually based on accepted principles. Figure 5-33 shows a model for skin resistance *(A)* and its connection to the internal body milieu *(B)*. The internal body structures are regarded as purely resistive, whereas the skin contains significant elements of capacitance.[46]

In the model, for the infrequent direct strike, the sequence of events during the strike begins with the stroke attaching initially to the victim's head. For a brief moment, current flows internally as the skin becomes charged. At a voltage taken as 5 kV, the skin was assumed to break down. Once the internal current increased, the voltage across the body to the earth built up, and external flashover across the body occurred when the field reached the breakdown strength of air, 4000 V/cm (10,000 V/inch).

The results of modeling these events are shown in Figure 5-34, and the relative magnitudes of the various voltage components can be seen with their time scale. On this scale, the times to breakdown are short, and most events occur early in the course of the stroke. In summary, in this model, lightning applies a current to the human body. This current initially is transmitted internally, and then skin breaks down quickly and external flashover occurs. Support for this model is shown in waveforms recorded from measurements in the experimental application of lightning impulses to sheep.[12] Further modeling of step voltage injury verified that, for the erect human, this mechanism is less dangerous than direct strike.[226]

Further experimental evidence suggests that "a fast flashover appreciably diminishes the energy dissipation within the body and results in survival."[301] In addition, Ishikawa and colleagues[208] obtained experimental results with rabbits similar to the human data noted in Cooper's study.[87] Cooper and associates[107-109] carried her studies to animals in developing a model of lightning injury and successfully showed primary cardiac arrhythmias, prolonged ventilatory arrest, secondary cardiac arrest, keraunographic skin changes, and temporary lower-extremity paralysis.

As current flashes over the outside of the body, it may vaporize moisture on the skin and blast apart clothes and shoes, leaving the victim nearly naked, as noted by Hegner in 1917:

> The clothing may not be affected in any way. It may be stripped or burned in part or entirely shredded to ribbons. Either warp or woof may be destroyed leaving the outer garments and the skin intact. . . . Metallic objects in or on the clothing are bent, broken, more or less fused or not affected. The shoes most constantly show the effects of the current. People are usually standing when struck, the current then enters or leaves the body through the feet. The shoes, especially when dry or only partially damp, interpose a substance of increased resistance. One or both shoes may be affected. They may be gently removed, or violently thrown many feet, be punctured or have a large hole torn in any part, shredded, split, reduced to lint or disappear entirely. The soles may disappear with or without the heels. Any of the foregoing may occur and the person not injured or only slightly shocked.[175]

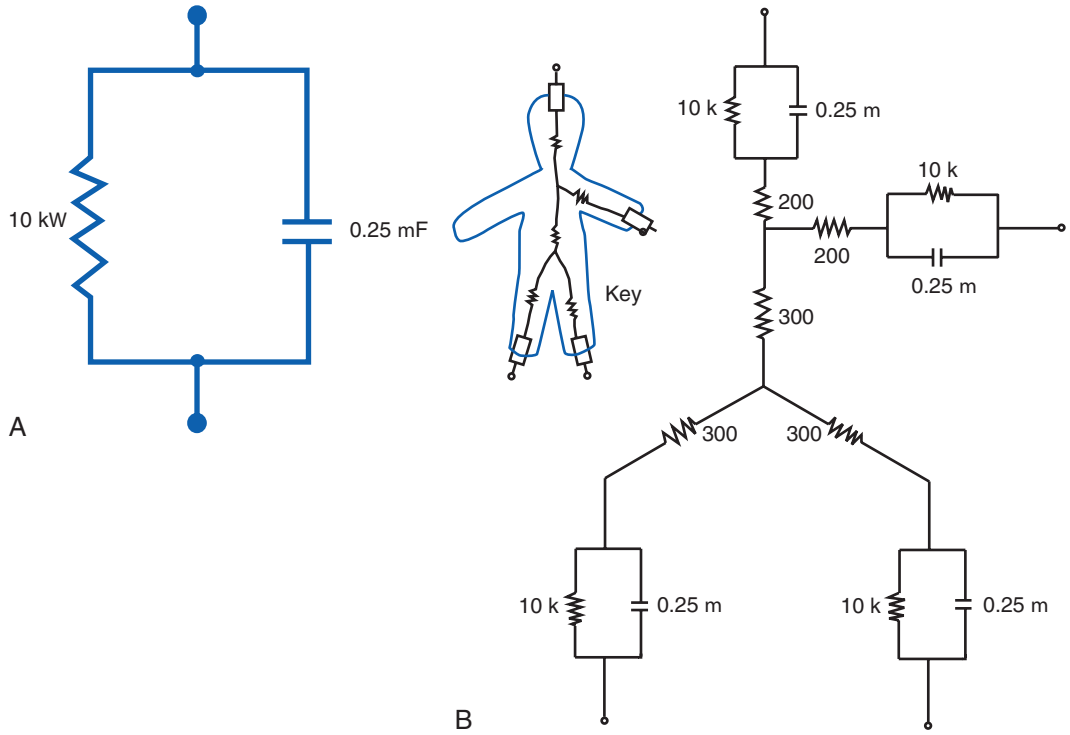


**FIGURE 5-33** **A,** Electrical model for human skin impedance. **B,** Model of human body for the purpose of examining current flow during lightning strike.

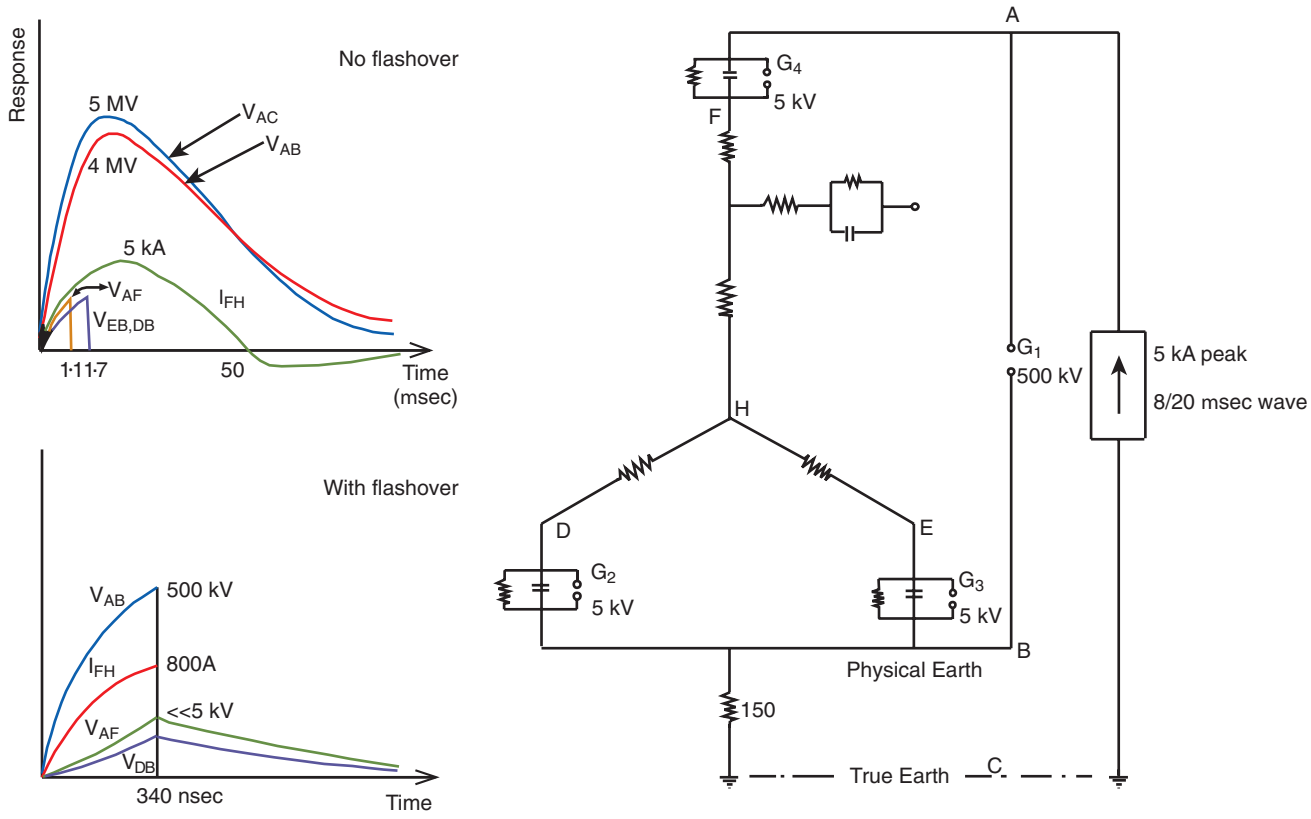


**FIGURE 5-34** Model of human body adapted for the circumstance of direct lightning strike. Responses of the body model are shown for cases of direct strike with and without subsequent flashover.

The amount of damage to clothing or the surface of the body is not an index of the severity of injuries sustained within a human. Either may be disproportionately great or small. However, in unwitnessed situations, forensic evidence of damage to shoes and clothing, sometimes accompanied by tympanic membrane rupture, may be the most important and reliable indicator in determining whether lightning caused a healthy person's death when no other cause is apparent.[47,218,389]

The factor that seems most important in separating the effects of lightning from high-voltage electrical injuries is the duration of exposure to the current. This conclusion is reached both because lightning is not present long enough to cause tissue breakdown in the classic burn sense and because of the results of the mathematic modeling describing the path of the energy and how long it is in contact with the body.

## ESTIMATES OF STREAMER CURRENTS

Upward streamer mechanism currents have been estimated.[8-10,91] This mechanism recognizes that as a stepped leader steps toward the earth from a cloud, an upward leader will emanate from several objects that are possible points of attachment. Of these, a return stroke will evolve from perhaps only one. Alternatively stated, there will be several upward leaders that dissipate without attachment.

The current needed to establish and maintain any upward leader nonetheless must be supplied from the earth, and if a person is the source of an upward leader, current must flow through the person. If this streamer meets the downward leader, a direct strike results. If attachment does not occur, then once the return stroke is established through another upward streamer elsewhere, the subject upward streamer collapses back through the victim. Thus an initial unidirectional current occurs in this case, followed by a reverse collapse.

Becerra and Cooray[41] studied the amount of current to which a victim of upward leader current may be exposed. They identified two processes that are important to model. First, current flows through a victim to establish the upward streamer and space charge around the victim's head, under the influence of the electric field resulting from the stepped leader's downward path. Second, once a separate leader from the one affecting the person makes connection with the stepped leader, the electric field collapses rapidly. In that case, the charge in the unattached upward leader rapidly returns to the earth.

The first process is relatively prolonged and regular, and the second is rapid and impulsive. In an analysis using a human model and a 30-kiloampere (kA) lightning strike grounding just beyond the striking distance, current rises to approximately 20 amperes (A) over 150 microseconds (μsec). At the start of this rise, there is an extremely short pulse of about 5 A. This represents a body-charging component before the propagation current for the actual streamer. This is a significant current; however, the duration is extremely short. In fact, the flow of any substantial current internally occurs for less than 10 μsec before the current escapes externally and flashover occurs. The potentially fatal effect of this current, conducted internally within a victim for such a short time, theoretically depends on its timing to the vulnerable period of the cardiac cycle. For an 80-kA return stroke, the peak current is approximately 80 A, which again is significant.

Once the aborted leader collapses, there is a current pulse of opposite polarity. This is exquisitely short. Little is known about the collapse of an aborted leader, and assumptions are made regarding the time constant of the collapse. This ranges from 0.1 to 1 μsec. Cases are examined for a 30-kA and an 80-kA return stroke. The peak current can be large, depending on the size of the stroke and time constant. The least peak current is computed at about −600 kA, with the least energy deposited being 0.22 kilojoule (kJ). Again, these are highly significant currents that are large enough to produce significant injury and death.

## BEHAVIOR OF ELECTRIC CURRENT IN TISSUE

High-voltage or low-voltage electric current may be carried through tissue in a direct conduction manner, obeying simple linear equations such as Ohm's law. The result is heating of tissues under Joule's law, with thermally-induced cellular death and

dysfunction. Simple passage of current may interfere with neural and muscular function.[248] It is a feature of the nonlinearity of tissue that as conduction takes place, tissue modifies and its properties change. Conduction in a biologic material such as wood is discussed earlier, and the various tissue dispersions in operation during conduction are examined later with current research.

## MAGNETIC FIELD EFFECTS

It has been stated that some effects of lightning might be magnetically mediated.[27,75] A case cited in support of this involved a golfer under a tree in the company of three other persons. It was stated that death occurred without evidence of current entering or leaving the index case. On the other hand, one accompanying golfer showed evidence of current traversal, but survived. It was also stated that three methods of lightning shock may have existed—direct strike, side flash, and ground potential/streamer potential—but no evidence of any was seen. It is uncertain what evidence may have been expected from the latter two mechanisms, which may have been more likely causes. Contact potential was not considered relevant. In this case, with persons under a tree, it would seem possible to explain deleterious effects without resorting to a magnetic hypothesis, but this bears examination if only because it is a recurrent question. In the case under consideration, the stroke was considered as a line current 1 m (3.3 feet) distant from the victim, and peak fields and their effects were calculated.

In considering this contention, it is useful to consider the stroke as a single line current; however, one must also have an idea about how far from a victim such a stroke will act. If the stroke is close to a victim, attachment to the victim takes place and electrical effects apply as described earlier. If farther away, the magnetic field is operative without attachment and magnetic effects need to be examined. Ground potential also exists at this 1-m (3.3-foot) distance. It is necessary to find the minimum distance away from a victim that a stroke can reach ground without attachment to the victim. This gives the worst-case distance from a victim (the worst case being the closest) at which a pure magnetic field acts without lightning attachment and its electrical effects. The standard striking-distance formula gives such a distance,[120] as follows:

$$d_s = 10 \times I^{0.65}$$

where $d_s$ (m) is the striking distance and I is the stroke current in kiloamperes. This represents the distance at the last turn of the downward-stepped leader, such that if an object lies inside this distance, attachment of the leader to the object will take place.

For illustrative purposes, assume a stroke has a peak current of 18,000 A (50th percentile of lightning strike currents) so that $d_s$ is 65.5 m (215 feet). Pure magnetic effects are applicable at this distance and beyond. Inside this distance, the victim is subject to electric current effects. By comparison, the ground potential between two points 1 m (3.3 feet) apart at 60 m (197 feet) from a stroke of 18 kA is about 60 V, assuming Earth resistivity of 100 ohm-meters.

In examining the magnetic fields involved at this distance, assume an 18-kA stroke at a distance of 65 m (213 feet) from an individual. The peak magnetic B field (the "magnetic induction" formally quantifying the force on a moving charge in its influence) is:

$$B_{peak} = \frac{\mu_0 I_{peak}}{2\pi d_s}$$

At 65 m (213 feet), the peak B field is 88 μT.

For comparison, the earth's magnetic field is about 1 μT, and the magnetic fields causing concern for power-line fields are in the 1 to 100 μT range. (Magnetic field strength is measured in Teslas [T], with 1 μT being $1 \times 10^{-6}$ T.) The magnetic fields used in magnetic resonance imaging (MRI) are 2 to 5 million times these levels. As with other exposures, effects from chronic exposures may be quite different from acute or momentary exposures. Power-line fields, for instance, are arguably dangerous only if chronic. If one is concerned about a lightning stroke magnetic field, it would be wise to be concerned about MRI fields as well, where to date no effect has been found in practice.

Certainly, the time-varying nature of any B field is important, both in terms of the rate of change of the field and of movement of a conductor within this field. If one assumes that the above B field is generated in about 2 μsec, the time rate of change for the B field is about 22 T/sec. Andrews and colleagues[27] applied this field to a model heart and found that an entirely noninjurious current resulted.

It is concluded that magnetic field danger in normal circumstances does not seem to exist.[27] Certainly, special circumstances, such as the presence of a pacemaker or of an arrhythmic pathway, might exist, but in normal terms, magnetic effects would not seem to be clinically significant during occurrences of lightning strike.

## X-RAY AND GAMMA RAY EFFECTS

In recent years, both x-rays and gamma rays associated with lightning have been detected. As with magnetic effects, when calculated, both x-rays and gamma rays are much too small, short-lived, and usually far too distant to cause harm to people.

## THE FARADAY CAGE

It has been stated that the safest place for personal protection from lightning is in a solid vehicle or enclosure. This is a specific example of the general Faraday cage.

Any hollow symmetric shape made of conducting material is such that charge introduced at any one point of its surface will distribute quickly over the entire surface. The surface, by virtue of its conduction properties, remains at a constant potential (voltage). The potential of one side of the shape will be the same as that of the opposite side. Because current flow depends on a difference of potential, there can be no internal current flow. A person inside such a container is safe from whatever impinges on the outside, with the following conditions:

1. Provided the shape is surrounded by conducting material, the same principles apply to a nonsymmetric shape, such as a car body.
2. Provided the walls of a building or structure are sufficiently conductive, the same principles apply to a solid building, but not an open-sided building.
3. Full metal will form such a cage; however, wire meshing or metallic framing is sufficient to achieve the same end.

Conductivity of building walls is enhanced by metal internal wall framing and addition of metal piping, external down-piping, metal roofing, and any continuous metal or framing enclosing the space. These principles can be applied to other structures to assess their protective ability.

# INJURIES FROM LIGHTNING[26,68,88,104]

There are marked differences between injuries caused by high-voltage electrical accidents and lightning (Table 5-1). Exposure to high-voltage–generated electricity tends to be more prolonged, particularly if the victim "freezes" to the circuit. In this context, "long" could mean only a few to several seconds of contact. With skin breakdown, electrical energy surges through the tissues with little resistance to flow, causing massive internal thermal injuries that sometimes require major amputations. Myoglobin release may be pronounced, and renal failure may occur. In addition, compartment syndromes requiring fasciotomy may occur. Burns may be severe.[88,234a]

In contrast, lightning contact with the body is almost instantaneous, often leading to a flashover with very little energy going through the body, if at all, particularly because the vast majority of lightning injuries are indirect, as with side flash and ground current. Serious burns and deep injury are uncommon. Fluid restriction and expectant care are usually the standard.[88] Lightning injuries are primarily neurologic, not burns.

There have been at least two useful categorizations of lightning injuries by severity of injury, based on (1) the initial presentation and (2) the neurologic outcome (Box 5-4).

**TABLE 5-1** Lightning Injuries Compared with High-Voltage Electrical Injuries

| Factor | Lightning | High Voltage |
|---|---|---|
| Energy level | 30 million volts (V), 50,000 amperes (A) | Usually much lower* |
| Time of exposure | Brief, instantaneous | Seconds |
| Pathway | Flashover, orifice | Deep, internal |
| Burns | Superficial, minor | Deep, major injury |
| Renal | Rare myoglobinuria or hemoglobinuria | Myoglobinuric renal failure common |
| Fasciotomy | Rarely if ever necessary | Common, early, and extensive |
| Blunt injury | Explosive thunder effect | Falls, being thrown |

*Range is 500 V up to millions of volts in transmission lines.

## INITIAL PRESENTATION MODEL

### Minor Injury

These patients are awake and may report symptoms such as dysesthesia in an extremity or a feeling of having been hit on the head, or they may recall having been in an explosion. They may or may not have perceived lightning or thunder. At the scene, they often suffer confusion, amnesia, temporary deafness or blindness, or temporary unconsciousness.[88] They seldom demonstrate cutaneous burns or paralysis, but may complain of confusion and amnesia lasting from hours to days. Paresthesias, muscle pain, and headaches may last for days to months. Victims may sustain tympanic membrane rupture from the explosive force of the lightning shock wave. Vital signs are usually stable, although occasionally, victims demonstrate transient mild hypertension. They may experience soreness for a few days; recovery is usually gradual and may or may not be complete. Permanent neurocognitive damage may occur. Some victims may suffer PTSD and other psychological sequelae.

### Moderate Injury

Moderately injured lightning victims may be disoriented, combative, or comatose. They often exhibit *keraunoparalysis* (a term introduced by the French neurologist Charcot), which is motor paralysis, particularly of the lower extremities, with mottled skin and diminished or absent pulses. Nonpalpable peripheral pulses may indicate arterial spasm and sympathetic instability, which should be differentiated from hypotension. If true hypotension occurs and persists, the victim should be scrutinized for fractures and other signs of blunt injury. Spinal shock from cervical or other spinal fractures, although rare with lightning, may also account for hypotension.

Occasionally, victims have suffered temporary cardiopulmonary standstill, although it is seldom documented. Spontaneous recovery of the pulse is attributed to the heart's inherent automaticity. However, respiratory arrest that often occurs with lightning injury may be prolonged and may lead to secondary cardiac arrest from hypoxia or some other, yet-to-be-elucidated cause. Seizures may occur.

Burns are uncommon in lightning victims. First- and second-degree burns not prominent on admission may evolve over the first several hours. Rarely, third-degree burns may occur. Tympanic membrane rupture should be anticipated and, along with hemotympanum, may indicate a basilar skull fracture.

Whereas the clinical condition often improves within the first few hours, victims are prone to permanent sequelae, such as sleep disorders, irritability, difficulty with fine psychomotor function and attention, chronic pain and dysesthesia, generalized weakness or easy fatigability, sympathetic nervous system dysfunction, chronic headaches, and sometimes PTSD. A few cases of atrophic spinal paralysis have been reported.[26,68,88,104,390a]

### Severe Injury

Patients with severe lightning injury may be in cardiac arrest with either ventricular standstill or fibrillation when first examined. Cardiac resuscitation may not be successful if the victim has sustained a prolonged period of cardiac and central nervous system (CNS) ischemia. Direct brain damage may occur from the lightning strike or blast effect. Tympanic membrane rupture with hemotympanum and CSF otorrhea is more common in this group.

The prognosis is usually poor in the severely injured group, worsening further with any delay in initiating cardiopulmonary resuscitation (CPR), thus causing anoxic injury to the brain and other organ systems. There are anecdotal reports of successful resuscitation of lightning victims with automated external defibrillators (AEDs).[159a,290,364]

**BOX 5-4** Lightning Injuries

**Immediate**
- Cardiac arrest and cardiac injuries
- Pulmonary injuries
- Neurologic signs
- Seizures
- Deafness
- Confusion, amnesia
- Blindness
- Dizziness
- Contusion from shock wave
- Chest pain, muscle aches
- Tympanic membrane rupture
- Headache, nausea, postconcussion syndrome

**Delayed**
- Neurologic symptoms and signs
- Neuropsychological changes
- Memory deficits
- Attention deficit
- Coding and retrieval problems
- Distractibility
- Personality changes
- Irritability
- Chronic pain
- Seizures

## NEUROLOGIC OUTCOME MODEL

Cherington[66,68,80] proposed a classification of lightning injury focusing on neurologic complications. The classification consists of four groups, based on time of onset, duration of symptoms, and severity of the clinical situation.

### Immediate and Transient Symptoms

These transient symptoms may include loss of consciousness, amnesia, confusion, headache, paresthesias, and weakness. Many patients experience temporary paralysis of their limbs; keraunoparalysis is a brief paralytic state with loss of sensation that affects the lower more than the upper limbs. Muscle strength and sensation usually return to normal within a few hours. Paralysis is accompanied by pallor, severe vasoconstriction, and hypertension. Some have suggested that this peculiar state is caused by transient outpouring of catecholamines.

### Immediate and Prolonged or Permanent Symptoms

Patients have structural lesions that may be seen on imaging studies or on postmortem examination. The great majority of these neurologic complications involve the CNS, including post-hypoxic ischemic encephalopathy, intracranial hemorrhage, cerebral infarction, cerebellar syndromes, and spinal cord injuries.

### Possible Delayed Neurologic Syndromes

Delayed neurologic disorders attributed to lightning include motor neuron disease and movement disorders. These sequelae have followed lightning strikes from days to years, although the cause-and-effect relationship between lightning and all subsequent delayed neurologic complications remains open to question.

### Lightning-Linked Secondary Trauma from Falls or Blast

Lightning can cause trauma when the patient is thrown or falls. Injuries include epidural, subdural, and subarachnoid hemorrhages. Blunt and blast effects can damage the brain and other organs and cause tympanic membrane rupture, the most common blast effect seen in victims of lightning strike.

## CARDIOPULMONARY ARREST AND CARDIAC INJURIES*

### Cardiac Arrest

The most common cause of death in a lightning victim is cardiopulmonary arrest. In fact, a victim is highly unlikely ($p$ <0.0001) to die unless cardiopulmonary arrest is sustained as an immediate effect of the strike. In the past, almost 75% of persons who sustained cardiopulmonary arrest from lightning injuries died, mainly because CPR was not attempted.[87]

A primary cardiac arrest occurs immediately after the lightning discharge and is manifested as asystole and respiratory standstill. Because of the heart's automaticity, myocardial contractions generally resume, and return to a spontaneous circulation is possible within a short time. Unfortunately, respiratory arrest caused by unknown factors may persist, and unless the victim receives immediate ventilatory assistance, attendant hypoxia may induce arrhythmias and secondary hypoxic cardiac arrest.[29] Primary and secondary cardiac arrests had previously been hypothesized and confirmed in animal studies.[12,99,107-109] For example, sheep sustain initial asystole followed by resumption of a short run of bradycardia, then tachycardia, followed by atrioventricular block or bradycardia, and finally asystolic cardiac arrest.[12] Prolonged respiratory arrest has been confirmed in hairless rats.[107,234]

It is unknown whether cardiac arrest and arrhythmias induced by lightning are a result of damage to central cardiac and respiratory centers in the brain, to the carotid body and other pacemakers along the cardiac control paths, to feedback control mechanisms within the autonomic nervous system (ANS), to the heart, or to a combination of these.[95,107] Certainly, clinical evidence of general damage to ANS regulation has been well documented and confirmed in the laboratory.[107]

Asystole and ventricular fibrillation have been reported with lightning strike.[14,159a,170,222,232,275a,396] As noted in animal studies, asystole seems to be both the first and the last response to the strike, as the secondary agonal arrest rhythm of VF deteriorates.

### Other Cardiac Injuries

Multiple mechanisms, such as direct thermal damage, coronary artery spasm, increased circulating catecholamine levels, myocardial ischemia secondary to arrhythmia, and coronary artery ischemia as part of a generalized vascular injury, have been suggested to explain the cardiovascular events following lightning strike.[172,269]

Immediate prolongation of the QT interval has been shown in up to 64% of cases evaluated, with consequent predisposition to arrhythmias.[24] In the study by Lichtenberg and colleagues[253] of four patients with direct injuries, three had a prolonged QTc, whereas none of the patients with splash or ground strike had a prolonged QTc.

The electrocardiogram (ECG) pattern after initial shock may show tachycardia or bradycardia and the pattern of myocardial infarction. In the months and years following lightning strike, arrhythmias, such as tachycardia, bradycardia, premature ventricular contractions, and atrial fibrillation, have been reported.[106,130,249,253,305,363] Postural tachycardia also has been reported, implicating the ANS.[166]

Although the initial ECG may be unreliable for reasons given next, it is not uncommon to find ST changes consistent with ischemia and myocardial injury compromising any coronary vascular segment.[209,253,272] However, the ST-segment and T-wave changes generally occur on the inferior side of the heart.[7,26,135,170,209,220,253,350] Creatine phosphokinase (CPK), MB isoenzyme, and troponin are used as markers of myocardial damage. The first report of troponin level elevations and cellular damage in a lightning survivor was published by a high-altitude pulmonary physiologist who was injured while climbing in the Alps.[40]

Saglam and associates[350] presented a typical case of infarct changes with normal coronary angiography, summarizing the widespread cardiac consequences of lightning strike: "the victim may develop hypertension, tachycardia, nonspecific electrocardiographic changes (including prolongation of the QT interval and transient T-wave inversion), and myocardial necrosis with the release of creatine phosphokinase–myocardial band fraction." Unfortunately, their case of a "13-year-old boy with myocardial infarction secondary to an indirect lightning strike" resulted in early death, so the progress of the changes was not able to be reported. Others tend to show resolution of infarct changes.[160,348] Many of these abnormalities are accompanied by normal coronary angiography and nuclear medicine scans.[209,220,350,396]

The ECG changes may not occur until the second day following the strike,[307,337] making the initial ECG a poor screening tool for ischemia. Several clinicians stress that cardiac symptoms may not be apparent on initial presentation. Premature ventricular contractions were reported in one patient nearly 1 week after presentation. Whereas most ECG changes resolve within a few days, some may persist for months.[209,307]

Several reports of cardiomyopathy exist. Rivera and associates[337] refer to "stunned myocardium" and document a 42-year-old victim with severe cardiogenic shock and left ventricular dysfunction. This was demonstrated clinically with ECG changes and scan changes. The victim recovered fully with no long-term disability. The authors concluded that "the exact mechanism of abnormal contractility in the absence of direct electrofulguration is unknown but may be explained by release of oxygen-free radicals, proteolysis of the contractile apparatus, and cytosolic overload of intracellular calcium, followed by reduced myofilament sensitivity to calcium." There was a similar case in a 36-year-old man who presented with severe compromise of ventricular function with ejection fraction of 15%, and rapid recovery during a 10-day hospitalization.[364]

Takotsubo-shaped hypokinesis with aneurysmal dilation (transient left ventricular apical ballooning syndrome) with associated cardiomyopathy has been described.[172] This is noted in the setting of ECG changes mimicking myocardial infarction, but with minimal enzyme release.[131,160] Recovery from dramatic dysfunction is completed over days to weeks.

Some clinicians have theorized that vascular spasm is a cause of cardiac damage not dissimilar to keraunoparalysis.[306] However, ECG changes are not always consistent with cardiovascular supply patterns. Areas of focal cardiac necrosis have been reported in autopsies, and histologic changes have been shown in sheep hearts.[14]

## PULMONARY INJURIES

Pulmonary edema may accompany severe cardiac damage.[124a,131] Pulmonary contusion, with hemoptysis and pulmonary hemorrhage, may result from blunt injury or direct lung damage.[365] Blunt thoracic trauma may also cause pneumomediastinum.[169]

## NEUROLOGIC INJURIES[37,68-71,90,217,224]

Lightning injury is primarily neurologic, with damage possible to central, peripheral, and sympathetic nervous systems. Injury to the nervous system far and away causes the greatest number of long-term problems for survivors. Tools commonly used in evaluation and treatment include functional scans, such as single-photon emission computed tomography (SPECT, usually demonstrating an abnormality); positron emission tomography (PET); anatomic scans such as computed tomography (CT) and MRI (rarely demonstrating gross abnormality); neuropsychological assessment; cognitive retraining; pharmacotherapy; and psychotherapy. Electroencephalography is often mentioned in the literature but is seldom helpful.[32,88]

*References 39, 87, 93, 96, 103, 106, 112, 232, 396.

### Central Nervous System Injury[37,68-71,76-80,90,224]

Almost 72% of victims in one study had loss of consciousness.[87] Nearly three-fourths of these victims also suffer cardiopulmonary arrest. Persons with cranial burns are two to three times more likely to sustain immediate cardiopulmonary arrest and have a three to four times greater probability of death. Persons who are stunned or lose consciousness without cardiopulmonary arrest are highly unlikely to die, although they may still have serious sequelae.

The victim of prolonged cardiopulmonary arrest may suffer anoxic brain injury that is not specific to lightning injury.[80a] Whether by this mechanism or others, cell loss and infarction of various CNS areas (e.g., cerebellum,[37] cerebrum) have been reported. Autopsy findings include meningeal and parenchymal blood extravasation, petechiae, swelling and herniation of the brainstem, dural tears, scalp hematomas, and skull fractures).[170,265a,284,300,374]

Gross structural changes to the brain have been reported, including coagulation of brain substance, formation of epidural and subdural hematomas, paralysis of the respiratory center, and intraventricular hemorrhage. In hemorrhagic injuries, there is a pattern of compromise to the basal ganglia.[4,60,167,305,368] Intracranial hemorrhages related to blunt trauma do not tend to occur in the basal ganglia, and therefore mechanical trauma is a less likely explanation. Studies in animals suggest that direct cellular damage occurs to basal ganglia, the respiratory center beneath the fourth ventricle, and the anterior surface of the brainstem. This occurs because the path of current flow in direct-strike patients is through orifices of the head (eyes, nose, ears), and the current travels caudally from the neocortex toward the basal ganglia, pituitary, hypothalamus, and brainstem.[12,14] As a result, signs and symptoms of endocrine dysfunction, respiratory or cardiac arrest, and sleep disturbances can be reasonably expected to occur. Cerebellar compromise may eventually manifest as parkinsonism, extrapyramidal syndrome, or other involuntary movement disorders.[37,66,69,79,80,297]

Although MRI or CT may show diffuse edema, intracranial hemorrhage, or other injuries, they are most often normal.[79,80,124] Hemorrhage has been found on MRI in a few acutely injured victims.[79,383]

Seizures may accompany initial cardiorespiratory arrest as a result of hypoxia or intracranial damage. Electroencephalography may show epileptogenic foci in the acute phase. These patterns may be focal or diffuse, varying with the site and type of injury. However, most patients do not experience seizures during hospitalization. Some victims, including children, develop delayed seizures (months to 1 or 2 years later), some of which manifest as "absence spells," memory loss, or blackouts that are often diagnosed as "pseudoseizures."

In Cooper's study[87] of severely injured victims, nearly two-thirds had some degree of lower-extremity paralysis (keraunoparalysis), usually demarcating around the waist or pelvis, and approximately one-third had upper-extremity paralysis. The affected extremities appear cold, clammy, mottled, insensate, and pulseless.[26] This is probably the result of sympathetic instability and intense vascular spasm, similar in appearance to Raynaud's phenomenon. It usually clears after several hours.[26,88] Fasciotomies are seldom indicated for lightning injuries, because signs of compartment syndrome or distal ischemia usually clear with patient observation. Pulses can sometimes be elicited with Doppler examination. Atrophic spinal paralysis has been reported, as have persistent paresis, paresthesias, incoordination, delayed and acute cerebellar ataxia, hemiplegia, aphasia, quadriplegia (immediate or delayed), and progressive muscle atrophy of the upper extremities.

Whether or not victims have suffered loss of consciousness, they almost universally demonstrate anterograde amnesia and confusion, which may last for several days. Retrograde amnesia is less common. Although victims may carry on a conversation and remember their actions before the strike, they are often unable to assimilate new experiences for several days, even when there is no external evidence of lightning burns on the head or neck. Early after the strike, symptoms resemble postconcussion syndrome.

Survivors may have persistent sleep disturbances, difficulty with fine mental and motor functions, dysesthesias, headaches, mood abnormalities, emotional lability, storm phobias, decreased exercise tolerance, and PTSD.[90] Centrally derived pain and psychological syndromes have been reported.[66,69,79,80,297]

Spinal cord injury developing over months after the injury has been reported.[242,243] Paresthesias are frequently seen, often in the area of keraunoparalysis. There may be evidence of autonomic neuropathy.

### Peripheral Nerve Injury*

The peripheral nervous system may be affected. Pain and paresthesias are prominent features of lightning injury, particularly in the line of presumed current passage, although this is difficult to ascertain from external burns or other injury. Symptoms may be delayed by weeks to months. Any peripheral nerve can be involved.

### Autonomic Nervous System Injury[85,176,177,323,357,358]

Central hyperadrenergic state with superimposed autonomic storms and diffuse degeneration of the peripheral autonomic system have been reported.[309,387] Autonomic dystrophy, also called sympathetic dystrophy or sympathetically mediated pain syndrome, may occur. Such chronic pain syndromes are now subsumed under the classification of complex regional pain syndromes, which are of type 1 (reflex sympathetic dystrophy) or type 2 (previously causalgia).

Complex regional pain syndromes are long-term neurologic sequelae that may be caused by even minor injuries to nerves. These are characterized by pain, edema, autonomic dysfunction, trophic changes (including atrophy secondary to disuse from pain), and movement disorder.

### Posttraumatic Headache[93,375]

Many victims of lightning injury exhibit severe, unrelenting headaches for the first several months, along with other symptoms, including gastrointestinal distress, that resemble postconcussion symptoms. Acupuncture may be effective. Many patients complain of nausea and unexpected, frequent vomiting spells early in the recovery period. Dizziness and tinnitus from eighth cranial nerve injury are also common complaints, especially with telephone-transmitted lightning strikes.[30,90,104,388,392]

## BURNS†

Most people assume that because of the tremendous energy discharge involved, a lightning victim will be "flash-cooked."[58,89] Fortunately, the physics of lightning flashover, as well as the vast majority of injuries occurring from indirect strikes, spares most victims from suffering more than minor burns. Less than one-third of lightning survivors have any signs of burns or skin marks. Although extensive third- and fourth-degree burns may occur in combination with skeletal disruption, these are quite rare. The incredibly short period of exposure may explain the lack of significant burn injury. Burn location provides a prognostic indicator. Cranial and lower-extremity burns are associated with a fourfold and fivefold increase in mortality, respectively, compared to burns in other locations.[87]

Skin injuries are influenced by amount of moisture on the surface, type of clothing, and presence of metal objects worn or carried during the strike. Superficial partial- or full-thickness burns can be isolated or combined. The morphology can be linear, punctuate, flower-like, tattooing, or imprint burn mark from contact with metal objects; feathering; fern-leaf mark; or classic first, second, or third degree.[74,272,385]

### Entry, Exit, and Types of Burns

Discrete entry and exit points are uncommon with lightning. The burns most frequently seen may be divided into five categories:

*References 66, 68-70, 85, 176, 177, 217, 315, 319, 320, 323, 330, 357, 368, 378, 384, 392.

†References 38, 61, 86, 93,104, 181, 206, 268, 283, 284, 360, 366, 380.

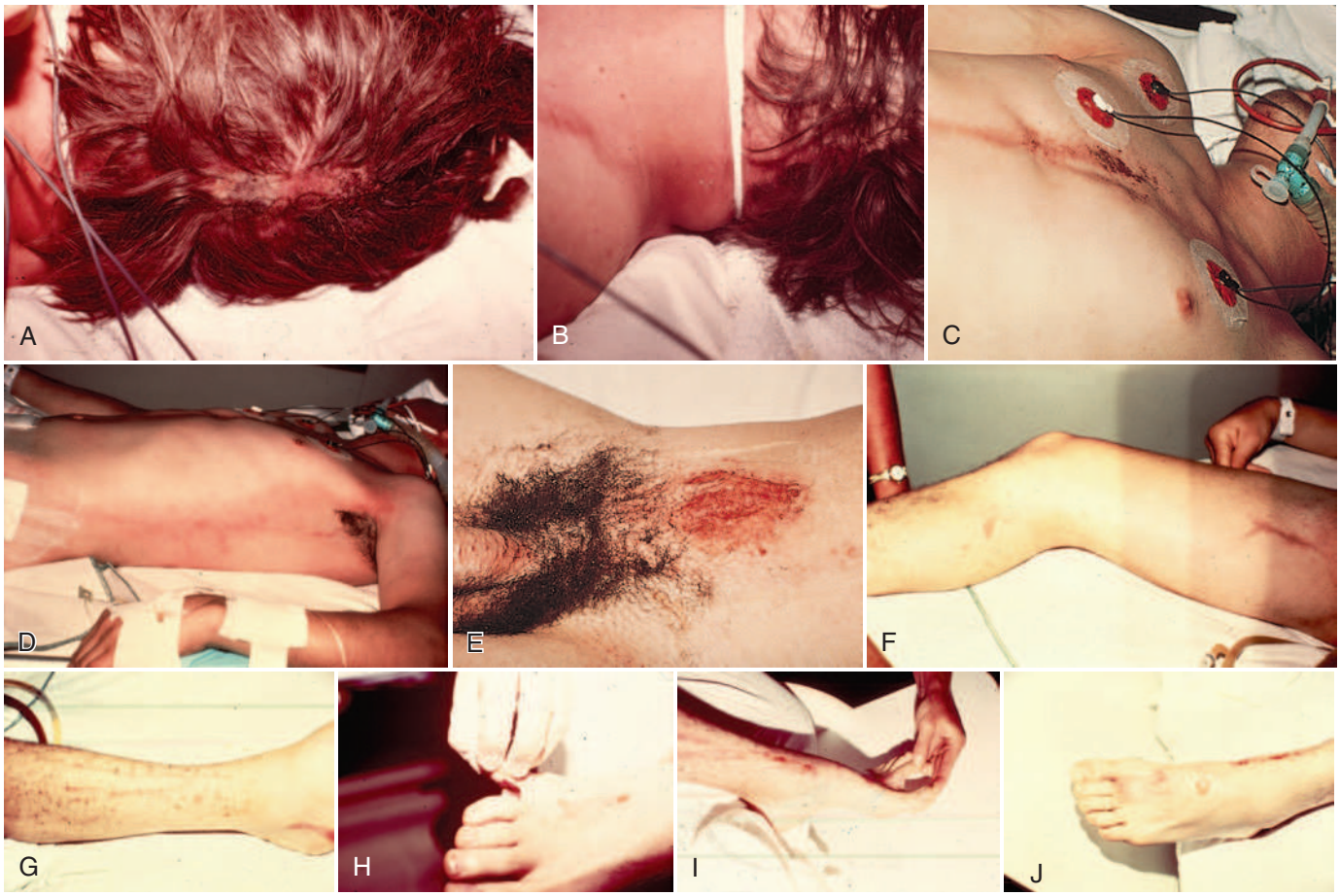


**FIGURE 5-35** Linear burns from a fatal 1977 lightning injury to 22-year-old baseball player. Most of these marks (those with small eschar) did not appear until a few hours to a few days after the injury. **A,** The mark that matured on the back of the head by the third day. **B,** The linear marks continuing down the side of the neck. **C** and **D,** Continuing marks down the anterior and lateral torso. Note that these are the normal "sweat lines" that a baseball player would develop. Also note the burn to the antecubital fossa, where sweat would accumulate under the baseball jersey as the player stood crouched and ready to catch near second base. Note that all these burns are partial thickness with sparse blistering. **E,** More extensive burns where a metal belt buckle or athletic supporter may have been causing secondary thermal burns or electrical discharge to the skin from the metal. **F** to **H,** Damage to the right leg and foot. Note the parallel marks on the foreleg, which would correspond to sweat accumulation or wetness in the ribbing of the athletic socks. Note also the mark on the heel, which may have been from the metal heel cup in the shoe or contusion from the shoe being ripped off by the vapor explosion of the flashover. The socks were destroyed or exploded off below the ankles, and the shoes were never found. Blunt injury from the explosion caused nonburned ripping of the fifth toe web (**H**). **I** and **J,** Damage to the left lower leg and foot. *(Copyright Mary Ann Cooper.)*

linear; punctate, full thickness; feathering, or flowers; thermal from ignited clothing or heated metal; and combinations.

*Linear burns* often begin at the victim's head and progress down the chest, where they split and continue down both legs (Figure 5-35). The burns generally are 1 to 4 cm (0.5 to 1.5 inches) wide and tend to follow areas of heavy sweat concentration, such as beneath breasts, down the middle chest, and in the midaxillary line.[88] Linear burns are usually first- and second-degree burns that may be present initially or may develop as late as several hours after the lightning strike. They are not primary lightning injuries, but more likely steam burns caused by vaporization of sweat or rainwater on the victim's skin as flashover and current flow occur around the body. In patients wearing thin or cotton clothing, the steam tends to escape through the clothing, causing less damage than in areas where the clothing is bunched (e.g., axilla, antecubital fossa [see Figure 5-35C], groin) or under nonporous material, such as leather jackets or belts (Figure 5-36).

*Punctate burns* are discrete circular burns that individually range from a few millimeters to 1 cm (0.4 inch) in diameter, and may be multiple and closely spaced (Figure 5-37). They may be full thickness and resemble cigarette or cinder burns, and are usually too small to require grafting.

Particularly useful for forensic investigation is explosion of clothing, such as occurs with vapor explosion of wet socks in shoes. This may blast shoes off with subsequent contusion and avulsion injuries to parts of the foot (see Figure 5-35H). Examination of clothing may show singed fibers at the edge of the damage and absence of very fine fibers on material, such as cotton, because these were vaporized or burned away (Figures 5-38 and 5-39; see also Figure 5-33B). With a handheld 10× magnifier, plasticized or polyester materials may show frank melting. Even in cotton clothing with no apparent damage to the material, examination of the threads joining pieces at the seams will often show a tiny droplet of plastic, because thread contains a polyester or nylon core for strength and durability.

*Feathering* or *fern-leaf mark figures* are pathognomonic of lightning and known by such names as Lichtenberg flowers or figures, filigree burns, arborescent burns, ferning, and kerauno-graphic markings[73,74,86,129,203,239,263,332,380] (Figures 5-40 and 5-41). These

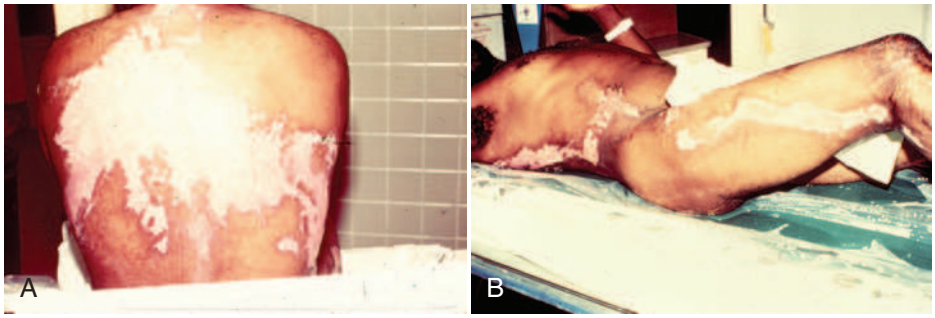

**FIGURE 5-36** Steam burns on a motorcyclist who was wearing a leather jacket, belt, and pants. This man was injured 3 weeks after and only a few miles away from the man in Figure 5-35. **A,** Note the more extensive burn to the back where the steam was held against the skin longer by the leather jacket than would occur with more porous clothing. **B,** More linear burns where the jacket was not as close to the skin. Note that the burns take a right angle under the belt at the waist before splitting at the groin and continuing laterally down the leg. Old scarring on the right knee was from a previous airplane crash and not part of the lightning injury. *(Copyright Mary Ann Cooper.)*

markings are not true burns, usually appearing as transient pink to brownish, sometimes lightly palpable, arborescent marks that follow neither the vascular pattern nor nerve pathways. The pattern found is similar to that on a photographic plate exposed to a strong electric field and has been compared with fractals.[181] Sometimes the most superficial skin over the areas will slough or flake off after a few days. Although many pictures exist of these marks, they have never been described histologically. The most current theory is that they represent blood cells forcefully extravasated into the superficial layers of the skin from contracting capillaries, similar to a superficial bruise, which is consistent with their rapid resolution and pattern of color changes. Experimentally, these marks follow the flashover current lines seen in Cooper's animal model.[102,104]

Deep burns may be caused by metal worn close to skin.[104,215,385] Figure 5-35E shows a burn resulting from a metal belt buckle or athletic supporter worn by a young man who was struck by lightning while playing softball. Figure 5-42 shows a necklace burned into the skin with permanent tattooing. Another theory for these burns involves discharge of current from metal to underlying skin. On rare occasions, clothing is ignited by lightning, causing severe thermal burns.[26,88] Victims of lightning may exhibit various combinations of burns, as demonstrated in Figure 5-35.

In developing countries, reports often describe lightning victims as "charred" or "burned beyond recognition."[94] Since these reports are frequently written with no first-hand knowledge or investigation, it is unclear if these are from the reporter's expectations or if the character of burns from lightning in developing countries is different or more severe. Recent newspaper reports accompanied by pictures or YouTube documentation show that buildings where people were injured have been almost

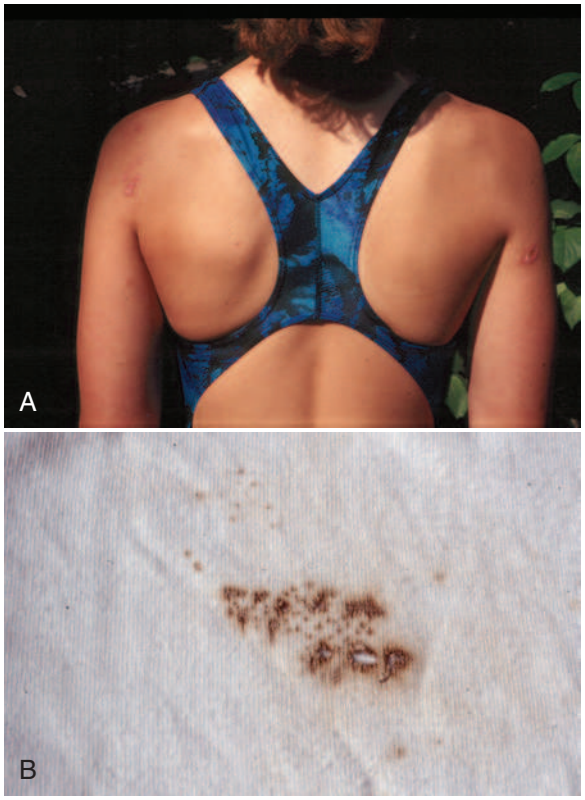

**FIGURE 5-37** **A,** Punctate burns to the shoulder and arm of a 12-year-old girl who was at a campfire with friends. **B,** Note singeing of the cotton T-shirt that she was wearing at the time over the area of the right arm burn. *(Copyright Mary Ann Cooper.)*

**FIGURE 5-38** Damage to the socks of a fatally injured farmer. *(Copyright Mary Ann Cooper.)*

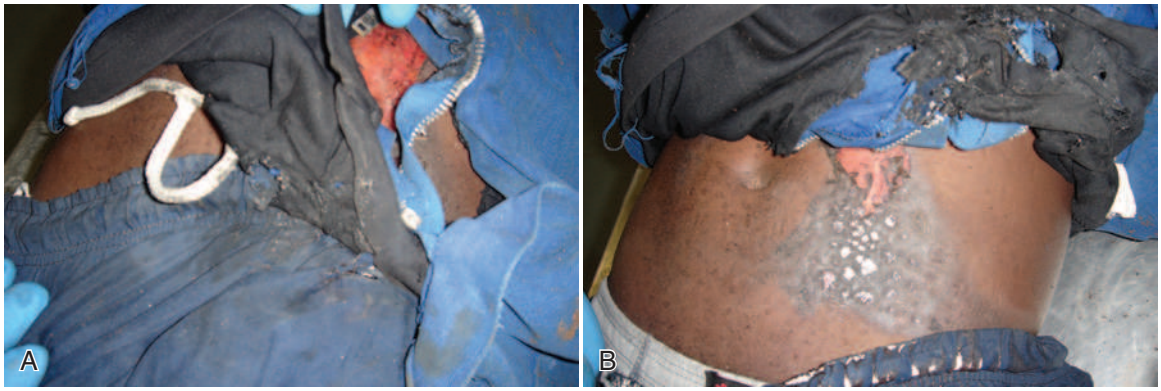


**FIGURE 5-39** **A,** Melting of synthetic clothing material from lightning damage. **B,** Underlying damage to skin from zipper and melted material. *(Copyright Ryan Blumenthal.)*

completely destroyed. Most homes and businesses in developing countries have no protective Faraday cage effect and do not provide safe shelter from lightning. Because of the high incidence of acute keraunoparalysis, it is hypothesized that even young healthy victims may be paralyzed temporarily after lightning injury and unable to evacuate from mud and thatch structures (Figure 5-43).

## BLUNT, CONCUSSIVE, AND EXPLOSIVE (BLAST) INJURIES

As with explosive injuries, lightning may cause several types of injuries, which can be similarly classified. Primary explosive injuries, similar to the shock wave formed on a battlefield, an explosion, or barotrauma, mainly manifest with damage to organs containing air or gas in their interior (ear, lungs, intestines), or in areas with air-liquid and air-solid interphases. Barotrauma from lightning has been well described and includes pulmonary effects,[221,280,365] pneumomediastinum,[169] gastrointestinal perforation[36,142,221] or contusion,[170] and tympanic membrane damage (see Ear Injuries, later). Injuries caused by fragments that impact and penetrate the body are similar to injuries secondary to explosion, but are rare in lightning injury.[377] Tertiary injuries from explosion are closed and concussive, and usually produced by falls.

Back and spinal injuries unrelated to the electrical effects of lightning injury may result from these mechanisms. A variety of

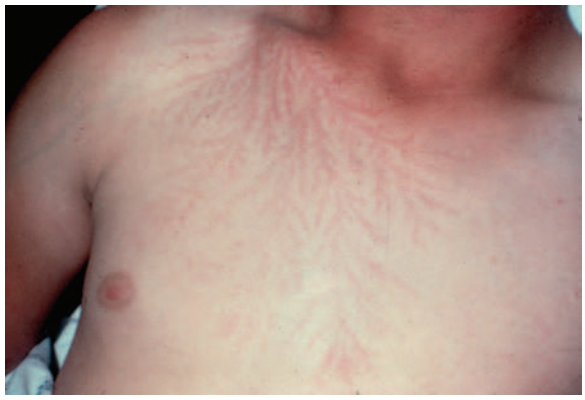

**FIGURE 5-41** Lichtenberg figures in a teenager who survived lightning strike. *(Courtesy/copyright Mary Ann Cooper.)*

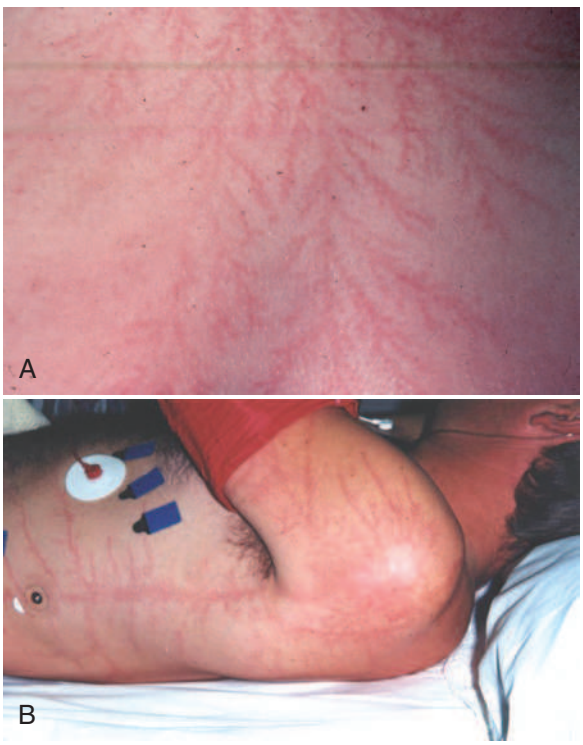


**FIGURE 5-40** **A,** Feathering marks. **B,** Ferning developed after the victim received a side flash lightning strike. *(**A** courtesy Mary Ann Cooper; **B** courtesy Sheryl Olson, RN.)*

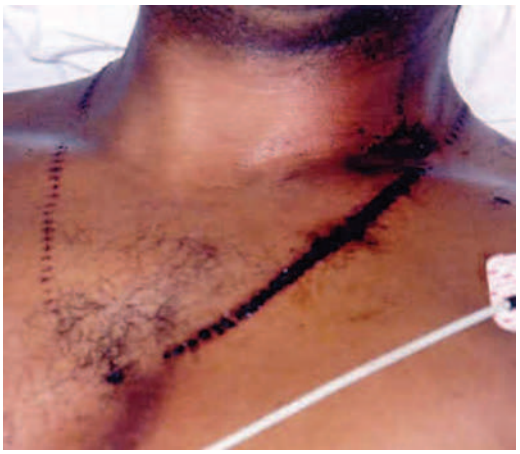

**FIGURE 5-42** Metal necklace burned into skin by lightning with permanent tattooing (nonfatal injury). *(Copyright Mary Ann Cooper; courtesy R. Washington.)*

**FIGURE 5-43** Rondavel in South Africa where this woman's family member died by fire. *(Courtesy Ronald Holle.)*

fractures, including skull, ribs, extremities, and spine, have been reported in lightning victims.[219,255] Unfortunately, these are often missed, particularly in the workup of persons with chronic pain. Rarely, a burst-like injury of soft tissue occurs and discloses extensive underlying injuries, especially in the feet, where boots or socks may explode as a result of vapor expansion (Figure 5-44; see also Figures 5-35H and 5-38). Persistent hypotension should alert the physician to blunt injuries to the chest, spine, lungs, heart, and intestines that may lead to complications of prolonged coma, pulmonary contusions,[365] heart failure, and ischemic bowel.

Other findings, such as hemoglobinuria and myoglobinuria, are seldom reported.[312,314] Multifactor rhabdomyolysis may be diagnosed due to elevation of CPK.[287,289,386] When these occur, they are usually transient. Myoglobinuric renal failure is quite rare.[171,269,302,314]

Several victims have complained of jaw pain. A number have suffered loss of teeth or fillings or necrosis of the jaw and teeth, and many describe a metallic taste in the mouth for months after the acute injury. At least one was found to have a styloid process fracture. Many recovering victims believe that premature arthritis may be a result of their injury.

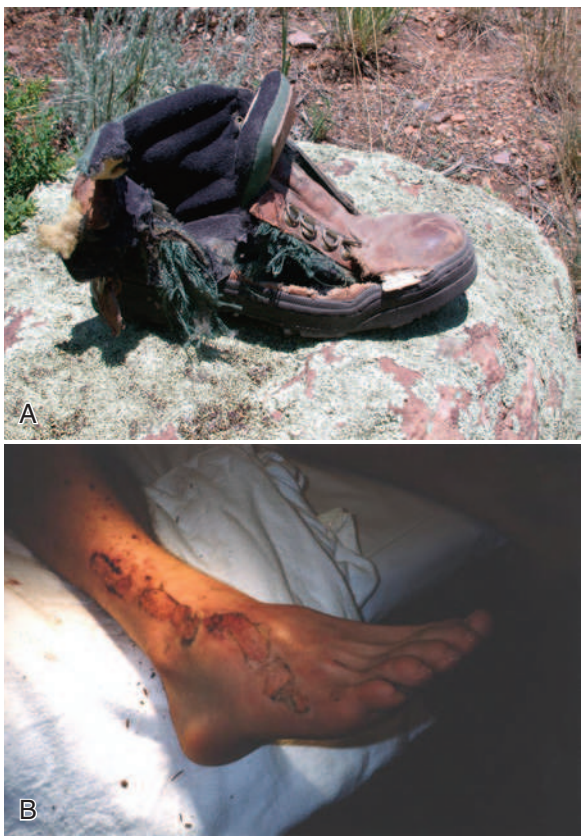


**FIGURE 5-44** The heat from a lightning strike caused water in the wet boot to instantaneously turn to steam, exploding the boot off the foot (**A**) and causing burns (**B**). *(Courtesy Sheryl Olson, RN.)*

## EYE INJURIES*

Ocular injuries may be caused by direct thermal or electrical damage, intense light, contusion from the shock wave, or combinations of these factors.

Although cataracts most often develop within the first few days, they may occur late and are often bilateral.† While the cataracts may be the typical anterior midperipheral type, posterior subcapsular opacities and vacuolization seem to occur more often with lightning injuries.[56] Corneal lesions, hyphema, uveitis, macular holes, iridocyclitis, vitreous hemorrhage, choroidal rupture, chorioretinitis, retinal detachment, macular degeneration, macular hole,[61,327] optic atrophy, diplopia, loss of accommodation, and decreased color sense have been reported.

Autonomic disturbances of the eye, including mydriasis, Horner's syndrome, anisocoria, and loss of light reflexes, may be transient or permanent. Transient bilateral blindness of unknown etiology is not uncommon. Intense photophobia may be present as the victim recovers. An interesting paper conjectures that the conversion of St Paul on the Damascus road and his subsequent blindness were caused by lightning.[53,54]

Dilated or nonreactive pupils should never be used as a prognostic sign or criterion for brain death in a lightning victim until all anatomic and functional lesions have been excluded.[64,65]

## EAR INJURIES‡

Between 30% and 50% of more severely injured lightning victims may have ruptured one or both tympanic membranes from the shock-wave effect, concomitant basilar skull fracture, or direct burn damage because of current flow into this orifice.[26,87] Otorrhea of CSF or hemotympanum rarely occurs. Disruption of the ossicles and mastoid has been reported. All these injuries are manifested as conductive hearing loss. Many cases of permanent deafness are noted in older literature and are seldom found today. The mechanism of sensorineural hearing loss may be explained by microhemorrhage and microfracture in the cochlea or by the hypoxia theory.[285]

Facial palsies, both acute and delayed, may occur from direct nerve damage by lightning. Balance problems and tinnitus are common, probably from eighth cranial nerve injury, and nystagmus and ataxia may occur. Otologic injury from hard-wired telephone–transmitted lightning strikes is common, but probably occurs less often now with the advent of mobile phones.[11,30,31]

## FETAL SURVIVAL

The fetus of a pregnant woman struck by lightning has an unpredictable prognosis. Of 11 cases reported, nearly one-half of the pregnancies ended in full-term live births, with no recognizable abnormality in the child. Approximately one-fourth resulted in live births with subsequent neonatal death; the remainder was stillbirths or deaths in utero. There has been one report of ruptured uterus after lightning strike.[87,144,150,329]

*References 56, 62, 127-126, 137, 165, 204, 216, 230, 276, 293, 294, 336, 366, 395.

†References 56, 126, 127, 137, 165, 168, 230, 276, 336, 366.

‡References 42, 44, 57, 141, 153, 158, 163, 216, 238, 277, 285, 298, 299, 328, 369, 388.

## HEMATOLOGIC ABNORMALITIES

Several unusual hematologic complications have been attributed to lightning injuries in isolated case reports. These include disseminated intravascular coagulation (DIC), transiently positive Coombs test, and Di Guglielmo syndrome, a type of erythroleukemia characterized by erythroblastosis, thrombocytopenia, and hepatosplenomegaly. Although there have been anecdotal reports of increased hypersensitivity, development of allergies, and increased risk for cancer in lightning victims, perhaps indicating an immunologic component to lightning injury, these have not been studied or validated with controlled longitudinal studies.

## ENDOCRINE AND SEXUAL DYSFUNCTION

Other neurologic-endocrine findings have anecdotally been reported, including cerebral salt wasting syndrome and inappropriate secretion of antidiuretic hormone (SIADH).[136,303]

Decreased libido for both men and women and impotence for men are common complaints. Sexual dysfunction may be caused by neural, spinal cord, endocrine (including hypoadrenalism and hypogonadism), autonomic, or neuropsychological injury; side effects of therapeutic drugs; and possibly other causes and should not be discounted. A report of male hypersexuality after a lightning strike has not been authenticated but can also be explained by a specific brain injury mechanism. Some patients have reported amenorrhea or menstrual irregularities lasting up to 2 years.

## PSYCHOLOGICAL AND NEUROCOGNITIVE DYSFUNCTION*

A person hit by lightning may rest at home for a few days, assuming that he or she is supposed to feel "bad" after being hit by lightning. The victim may not see a physician until family members insist or symptoms worsen or do not abate. Neurocognitive deficits may not become apparent until a victim attempts skilled mental functions or returns to work. Often the person returning to work, because of decreased work tolerance, short-term memory problems, easy fatigability, and difficulty assimilating new information, will be unable to continue the prestrike occupation. Chronic pain syndromes are cited as commonly overlooked diagnoses in psychiatric and functional terms and can lead to depressive symptoms.[24,177] If the survivor does not obtain useful medical and psychological help, the person often lapses into a low-functioning state, unable to work and with multiplying psychological dysfunctions.

Although some medical clinicians have been historically suspicious of victims' complaints of psychological and neurocognitive dysfunction, evaluation of many such patients shows a vast commonality of psychological symptoms. Although every syndrome has its pretenders, there is a tendency, both medically and legally, to discount survivors' complaints as evidence of malingering, excessive reaction, conversion reaction, personality problems, or manifestations of "weak" coping strategies. The psychological disability has been examined for its depth and pervasiveness.[17,279,315,316,320,321] Patients are often young, previously healthy, and generally productive, with young families. Serious injury may change the family structure, family economic expectations, social structures, and future planning. As with other serious illnesses, relationship breakdown may occur. There is both economic and social cost to society, the workplace, and the home.

Survivors often complain of symptoms listed in Table 5-2 and others, including anxiety, depression, and learning disorder.[17] In a long term study, Andrews[18] noted problems with learning and executive function (Table 5-3).

### Functional Issues

**Memory Disturbance.** Individuals show marked diminution of short-term memory ability. They require shopping lists and reminder lists. Memory for recent names and places is diminished to the point of disability. Individuals tend to self-isolate, stop mixing socially, and avoid new circumstances.[90]

**Concentration Disturbance (Adult Attention Deficit Disorder).** Individuals show deficits in their working memory, are unable to focus attention for more than a short period, and are easily distracted. In particular, reading and understanding are poor. Job training is detrimentally affected. This is worsened by sleep disturbance.

**Cognitive Function.** Individuals report diminished mental agility. Calculation, estimation, and managing accounts and telephone calls become erratic. This affects both family and work performance. Ability at mental manipulation and problem solving is greatly decreased.

**Higher Executive Functioning.** Individuals are neither able to coordinate multiple tasks simultaneously nor able to follow orders for complex tasks they used to perform easily before the injury. One patient described it as if "the office manager of my brain had quit."

### Behavioral Issues

**Emotional Lability and Aggression.** Individuals find that they are more aggressive than before. They are easily frustrated and may have outbursts of temper. The strains on a partnership are significant, and marital and relationship dysfunction is common. An increased state of arousal and anxiety may further complicate distractibility, as well as proper recognition and assimilation of new learning.

**Sleep Disturbance.** Easy fatigability, sleep disturbance, or hypersomnolence (usually early after injury) is common and may last for years. Flashbacks and nightmares may be experienced as part of PTSD.

**Phobic Behavior.** Avoidance of thunderstorms and electricity is common, along with other, less specific phobias.

**Depression.** Depression, both from biologic injury and secondary to chronic pain, loss of work, sleep deprivation, decreased

**TABLE 5-2 Frequent Complaints of Lightning Survivors**

| Symptom | % |
|---|---|
| Memory disturbance | 71 |
| Concentration disturbance | 63 |
| Aggression and irritation | 67 |
| Wariness and phobia | 58 |
| Loss of mental powers | 50 |
| Social isolation | 38 |
| Sleep disorder | 38 |

**TABLE 5-3 Learning Deficits in Lightning Survivor**

| Findings | % |
|---|---|
| **Memory and Learning Deficits** | |
| Globally | 19 |
| Visual | 35 |
| Visuospatial | 38 |
| Auditory | 62 |
| **Other Deficits** | |
| Verbal learning | 54 |
| Verbal fluency | 46 |
| Concentration | 46 |
| Attention | 42 |
| Executive function | 38 |
| Reduced executive speed | 62 |

*References 69, 70, 104, 315, 319, 320, 326, 378, 393, 394.

mental abilities, or changes in family dynamics, is almost universal and should be anticipated. Antidepressant medication may be useful. Formal neuropsychological testing may be used to attempt to validate the injury, quantify a functional baseline, and design cognitive therapy. Very few studies have formally examined the syndrome.[320,378] Primeau and co-workers[320,321] point to research difficulties, including sample bias and heterogeneity, methodology (cross-sectional rather than longitudinal or prospective), and the essential difficulties of determining premorbid status or current independent psychiatric status. The authors considered somatoform disorders as a cause, noting patients' tendency to be preoccupied with the injury and to overattribute subsequent symptoms to lightning injury. This may result partly from physicians' general lack of knowledge of the problems and the lack of longitudinal controlled studies, so victims do not know what to expect in the future.

**Other Behavioral Issues.** Conversion reaction may also be considered, although it cannot account for the symptomatology of lightning and electrical disability.[90] Good neuropsychological testing can detect this facet. One difficulty with neurocognitive testing is use and interpretation of the Minnesota Multiphasic Personality Inventory (MMPI). Although it was not developed to characterize patients with chronic problems, particularly those with chronic pain, the MMPI has been applied to them, often resulting in erroneous conclusion of conversion reaction or preoccupation with physical complaints. It should surprise no one that a person suffering from chronic pain and neurologic injury that hampers normal preinjury activities will rank higher on the "preoccupation with physical complaints" and "conversion" scales than will uninjured normal individuals.

Andrews and Darveniza[30] recognized three postinjury syndromes in telephone-related lightning injury and correlated these with the postinjury periods of 1 week, 1 week to 3 months, and 3 months to 3 years. Primeau and co-workers[320] add a fourth syndrome, in persons experiencing longer and perhaps lifetime dysfunction.[163] Cherington[68] and colleagues[80] have further classified lightning injury by its permanent sequelae.

The first 12 months after injury are crucial to recovery. It is during this period that the most recovery is seen, with possible mild improvement up to 3 years after injury. Van Zomeren and associates[378] provide one of the few thorough examinations of the syndrome in lightning-injured patients. The main complaints were fatigue, energy loss, poor concentration, irritability, and emotional lability. Impairment of memory, attention, and visual reaction times as well as depression and PTSD were documented ($p$ <0.001). The authors stated that the lasting complaints and mild cognitive impairments could not be explained on the basis of anxiety reactions or depression, and summarized their findings under the heading of "vegetative dystonia."

# RECOGNITION AND ACUTE TREATMENT OF LIGHTNING INJURIES[88,90]

## DIAGNOSIS

Diagnosis of lightning injury may be difficult. The history of thunderstorms, witnesses who can report having seen the strike, and typical physical findings in the victim make diagnosis easier but are not always present.

Lightning can strike on a relatively sunny day, thunder may not be appreciated, and sometimes the victim is alone when injured. A diligent history taking and careful physical examination at the earliest opportunity may help determine the true cause. Any person found with linear burns, mental status changes, ruptured tympanic membrane, and clothes exploded off should be treated as a victim of lightning strike. Tympanic membrane rupture may make the diagnostic difference in difficult cases.

Diagnosis is made doubly difficult because burns, which most people expect to accompany lightning injuries, are often absent. Feathering marks, also called Lichtenberg flowers, are pathognomonic of lightning strike but are rare.[73,74,86,129,332,380] Other signs or symptom complexes include linear or punctate burns, tympanic membrane rupture, confusion, and outdoor location. Because several cases of side flash or contact injury from indoor plumbing and telephones have occurred, the physician should suspect lightning strike in persons found confused and unconscious indoors after or during a thunderstorm.[11,26,30,88]

Documentation of lightning activity to support legal or insurance claims is available from commercial sources.

## INITIAL FIRST AID AND TRIAGE OF VICTIMS[26,88,103,106]

Ensuring scene safety is paramount. Rescuers are at risk if thunderstorms are in the area.[373] If the situation makes it possible, the victim is transferred to a safe area quickly, or to a location under lesser risk.

If the victim is unresponsive with no breathing or no normal breathing (i.e., only gasps), the person has suffered a cardiac arrest. Rescuers should immediately activate the emergency response system, if possible. CPR should be started immediately with a CAB (chest compressions, airway, breathing) sequence. AEDs have been helpful in some cases.[159a,290] Cardioversion, defibrillation, and medications should proceed according to the most recent advanced cardiac life support (ACLS) guidelines.[379]

The victim will probably die unless pulse and respirations resume spontaneously in a short time. The heart may resume activity but may slip into secondary cardiac arrest. It is unknown whether the secondary arrest is caused by primary brain damage, hypoxia from prolonged respiratory arrest, primary cardiac damage, autonomic nervous system damage, or any of a number of other mechanisms. If no pulse is obtained within 20 to 30 minutes of starting CPR, it is reasonable to stop further resuscitation efforts; 77% of victims do not respond to CPR.[87] If a pulse is obtained, ventilation should be continued until spontaneous adequate respirations resume, the victim is pronounced dead, continued CPR is deemed unfeasible because of rescuers' exhaustion, or there is danger to rescuers' survival.

When lightning strike involves multiple victims, resources and rescuers may not meet the demand, and triage must be instituted. Normally, the rules of triage in multiple-casualty situations dictate bypassing dead persons for those who are moderately or severely injured and can benefit from resuscitation efforts. However, "resuscitate the dead" is the rule in lightning incidents,[120a,397] because victims who show some return of consciousness, or who have spontaneous breathing, are already on the way to recovery. The most vigorous CPR attempts should be directed to the victims who appear to be dead, because they may ultimately recover if properly resuscitated.[106,159a,379] Survivors should be routinely stabilized and transported to the hospital for more thorough evaluation.

The probability that lightning victims can recover after prolonged CPR is low. No evidence suggests that survival after a longer period than normal without resuscitation is possible in the victim of lightning injury. Often, in a remote setting, the rescuer is emotionally tied to the victim by age and friendship and may tend to continue resuscitation past the point of futility. In pronouncing a victim dead, the rescuer must ensure that other problems, such as hypothermia, are not contributing to the victim's response to CPR efforts.

Other procedures, such as airway control or intubation, and institution of intravenous (IV) fluids and oxygen may be required. Because lightning victims may suffer traumatic blunt injuries, rescuers may need to perform spinal immobilization and splinting of fractures, if possible, before transport.

Most lightning injury victims can be treated and discharged from the emergency department (ED), but more complex lightning injuries are considered one of the criteria for being referred and assessed at a burn unit. If this is not possible because of geographic location or distance, the receiving institution must have the capacity to treat victims with complex trauma, promptly assemble a multidisciplinary group of specialists and paramedical staff with experience in these types of injuries, and access a consolidated group of rehabilitation, physical, and occupational therapists.

## HISTORY AND PHYSICAL EXAMINATION

An eyewitness report is helpful because lightning victims are often confused and amnestic.[87,88] History that includes a description of the event and the victim's behavior following the strike may be helpful.

After scene safety has been secured, the victim should be undressed to search for hidden signs of injury. Special note should be taken of vital signs, temperature, and level of consciousness. Because many victims are struck during a thunderstorm, they may be wet and cold. Hypothermia should be anticipated and treated appropriately (see Chapter 7).

The awake patient should be assessed for orientation and short-term memory. A cursory mental status examination of the lightning victim may reveal good ability to carry on a "social conversation" that easily hides deficits in fine neurocognitive skills. It is easier to detect deficits when the victim cannot assimilate information, perseverates, or asks repetitive questions.

The scalp should be meticulously checked for hidden lesions. The victim's eyes should be examined for pupillary reactivity and ocular injury. Pupillary dilation or lack of pupillary reaction should not be taken as a sole indicator of death. Tympanic membrane rupture is an important indicator of lightning strike. Ossicular disruption may be one explanation for a victim's lack of appropriate response to verbal stimuli.

The cardiovascular examination should include distal pulses in all extremities, because assessment of just one extremity, usually the one most compromised, can result in the examiner considering the absence of distal pulses a sign of poor hemodynamic state. Cool, mottled, and pulseless extremities as a manifestation of keraunoparalysis may occur in up to two-thirds of victims.[87] Evaluating proximal pulses (femoral and carotid) may help avoid confusion. In the setting of an overall absence of distal pulses, cardiogenic, hypovolemic, and spinal shock should be ruled out.[106] Detecting arrhythmias may be difficult in a wilderness setting far from clinical resources. Repetitive evaluation of pulses and auscultation may determine the presence of some arrhythmias. Although the pulmonary system may be affected by cardiac arrest, pulmonary edema, or adult respiratory distress syndrome, it is uncommon to witness these initially.

The victim's abdominal examination occasionally demonstrates absent bowel sounds, which suggests ileus or indicates acute traumatic injury, such as contusion of the liver, bowel, or spleen.

The examiner should document skin changes. The victim's skin may show mottling, especially below the waist. Burns may be present or may require hours to evolve. However, most are minor injuries. Notation of pulses, color, and movement and sensory examination of the victim's extremities are important. Keraunoparalysis is common, but usually improves or resolves in a few hours.

The physical findings and mental state of minimally and moderately injured victims tend to change considerably over the first few hours; careful observation and documentation delineate the course so that therapy can be modified. The minimally injured victim can almost always be discharged to a responsible person or requires only overnight observation, whereas the severely injured person may require intensive care with mechanical ventilation, antiarrhythmic medications, and invasive interventions and monitoring techniques, or referral to a burn center.

## LABORATORY TESTS AND RADIOGRAPHIC EXAMINATION

The more severely injured victim may require tests such as complete blood count, electrolytes, blood urea nitrogen, creatinine, testing for cardiac damage, and urinalysis. If the victim is to be placed on a ventilator, arterial blood gas (ABG) determinations will be necessary. If intracranial pressure monitoring is used, determination of serum osmolality may be required.

An ECG is desirable for evaluating most lightning victims. Although several types of acute arrhythmias have been reported, they are uncommon. QT prolongation is an abnormality associated with lightning injury.[24,253,307,350] The increased risk of developing torsade de pointes ventricular tachycardia (VT) must be considered, so monitored admission of lightning strike victims with QT prolongation is probably prudent. Cardiac enzyme elevations have rarely been reported with lightning injury, but measurement is recommended with any cardiac symptomatology or abnormal ECG.

Radiographs and other imaging studies may be obtained depending on the presentation and history. Cervical spine imaging should be performed if there is evidence of cranial burns, contusions, loss of consciousness, or change in mentation that would make the physical examination unreliable, or if there is any suspicion of a fall or being thrown. The victim who is unconscious, confused, or has a deteriorating level of consciousness requires brain CT or MRI to identify trauma or ischemic injury. Other studies to rule out fractures, dislocations, and other bony abnormalities may be obtained as clinically indicated.

The serum creatine kinase (CK) level is typically used as a biochemical marker of rhabdomyolysis and prognosticator of the risk for acute renal failure, even though CK shows a wide range among individuals. Although measurement of plasma or urine myoglobin can also be used as a confirmatory test, these are not widely used in clinical practice.

## TREATMENT[87]

### Fluid Therapy

Intravenous access is required for the victim who shows unstable vital signs, unconsciousness, or disorientation. If the victim is hypotensive, fluid resuscitation with normal saline or Ringer's lactate solution may be indicated. Fluid restriction in normotensive or hypertensive victims is recommended because of the risk for cerebral edema, particularly if intracranial injury is suspected.

In seriously injured or unstable patients, intensive care unit (ICU) types of monitoring, including arterial or central venous pressure, may be indicated. Careful intake and output measurements are necessary in the severely injured patient and require placement of an indwelling urinary catheter. Myoglobinuria, unless caused by blunt injury or other mechanism, is rarely attributed to lightning injury alone. There are no reports in the literature of investigations about the usefulness of mannitol-induced diuresis, alkalinization, or aggressive fluid therapy in lightning injuries, unlike what is known about the treatment for high-voltage electrical injuries. However, if burns are severe and extensive, which is rarely the case in developed countries, fluid resuscitation may be required.

### Fasciotomy Not Needed

Intense vascular spasm with lightning is usually transient and caused by sympathetic instability. The presence of keraunoparalysis should not be treated like similar-appearing traumatized extremities caused by high-voltage electrical injury.[26,88,267] Steady improvement in the mottled and cool extremity, with return of pulses in a few hours, is the rule rather than the exception. Fasciotomies are rarely indicated unless the extremity shows no signs of recovery and increased intracompartmental tissue pressures are documented.

### Antibiotics and Tetanus Prophylaxis

Prophylactic antibiotics are not indicated. Antibiotic therapy should follow culture and identification of pathogens, except in obvious cases involving open fractures or cranial fractures that violate the dura. Appropriate tetanus prophylaxis should be given if burns or lacerations are present.

### Cardiovascular Therapy[103,106,144,232,253]

Management of cardiac arrest, including use of an AED, is standard. In the lightning victim who is not in cardiac arrest, vasospasm may make peripheral pulses difficult to palpate. When pulses are present, it is usually easiest to feel them in the femoral, brachial, or carotid arteries. Doppler examination may help to locate peripheral pulses and record blood pressure.

If a victim remains hypotensive, fluid resuscitation may be necessary to establish adequate blood pressure and tissue perfusion. Causes of hypotension include major fractures, blood loss

from abdominal or chest injuries, spinal shock, cardiogenic shock, and occasionally deep burns similar to high-voltage electrical burns. As soon as an adequate central blood pressure is achieved, fluids should probably be restricted because of the incidence of cranial injuries, cerebral edema, and postarrest anoxic brain injury.

Mechanical ventilation is required for the victim who is without spontaneous or adequate respirations until the person resumes adequate ventilation, brain death is declared, or the physician and family decide to cease efforts.

If lightning did not cause immediate cardiac arrest, there is very low risk of death. However, observation, cardiac monitoring, and serial cardiac marker (e.g., troponin) measurements are indicated if there is any sign of cardiac ischemia or arrhythmia, or if the victim complains of chest pain. The indications for antiarrhythmic drugs and pressor agents are the same as for suspected myocardial infarction.[88] No systematic study has addressed proper duration of observation for lightning survivors.

Transient hypertension may be so short-lived as not to require acute therapy. However, several victims developed hypertension 12 to 72 hours after lightning strike and seemed to respond well to antihypertensive medications. No systematic study of antihypertensive agents has been undertaken for lightning survivors.

### Central Nervous System Injury*

Every lightning victim should have a screening neurologic examination. If there is history of loss of consciousness or if the victim exhibits confusion, hospital admission is warranted. The victim with tympanic membrane rupture, cranial burns, or loss of consciousness, or who shows decreasing level of consciousness, should undergo cervical spine imaging, brain CT, and possibly brain MRI.

Cerebral edema may be managed with standard therapies, which may include mannitol, furosemide, fluid restriction, and cerebral pressure monitoring. Although hypothermia prior to resuscitation efforts was reported to contribute to complete recovery in one victim, there is no evidence that this would benefit all victims.[222]

Early seizures are probably caused by anoxia. If there is evidence of CNS damage, or if seizures continue after adequate oxygenation and perfusion have been restored, standard pharmacologic intervention with diazepam, fosphenytoin, phenobarbital, and other anticonvulsants should be considered. The response to specific individual drugs has not been studied.

If paralysis does not improve, causes other than lightning, including blunt injury from a fall, may be responsible. Spinal artery syndrome has been reported, perhaps caused by arterial spasm or undiagnosed fractures. A physical therapy program should be initiated before discharge.

### Burns†

Lightning burns may be apparent at admission but more often develop within the first few hours. Burns occur in less than one-half of lightning survivors and are generally superficial, unlike high-voltage electrical burns, and seldom cause massive muscle destruction. Myoglobinuria is infrequently seen, so vigorous fluid therapy and mannitol diuresis are not usually indicated. In the rare instance of myoglobinuria, the decision must be made by the treating physician, according to the case. Overzealous hydration with resultant cerebral edema following presumption of similarity to high-voltage injuries has probably harmed more lightning victims than has myoglobinuric renal failure.

Lightning burns are generally so superficial that they do not require treatment with topical agents. In the unusual instance of deep injury, topical therapy is standard. The findings that lightning burns are superficial and do not require active surgical intervention is reinforced by Matthews and Fahey.[268] Their paper provides a useful guide to the remote need for surgical intervention in lightning injury.

### Eye Injuries*

Eye injuries are myriad. Visual acuity should be measured and the victim's eyes thoroughly examined. Cataracts may develop in the first few weeks or months. Eye injuries should be treated in standard fashion and may require referral to an ophthalmologist. Case reports exist of successful treatment of optic neuritis with high-dose corticosteroids similar to those used with spinal cord injury, but because there were no controls, it is not known if recovery would have occurred without drug use.

### Ear Injuries†

Loss of hearing mandates otologic evaluation. Blast as well as direct injury may occur. Simple tympanic membrane rupture is usually handled conservatively with observation until the victim's tissues heal. Sensorineural damage to the auditory nerve, resulting in hearing changes, dizziness, and permanent tinnitus and facial nerve palsies, is not uncommon. Ossicular disruption or more severe damage may necessitate surgical repair. Otorrhea and hemotympanum suggest basilar skull fracture. Complaints of pain around the angle of the victim's jaw may indicate occult fracture of the styloid process and other musculoskeletal damage.

### Pregnant Victims[144,150]

If a pregnant woman is struck by lightning, fetal viability should be assessed, including fetal heart tones, ultrasonography to observe fetal activity, and other standard methods.

### Other Considerations

Gastric irritation is sometimes seen.[146] A nasogastric tube is appropriate if ileus or hematemesis occurs. Extended Focused Assessment with Sonography for Trauma (EFAST) is indicated in comatose patients who remain hypotensive. Abdominal CT scan is preferred in hemodynamically stable patients, because intestinal contusions and hemorrhage have been reported. It is common for survivors to report continuing nausea for several weeks to months.

Endocrine dysfunction, perhaps as a result of pituitary or hypothalamic damage, including amenorrhea, impotence, hypogonadism, and decreased libido, has occurred in some victims. Spinal cord or sympathetic nervous system injury or postinjury depression, as well as side effects of medications, may also cause impotence, decreased libido, and sexual dysfunction after lightning injury.

### Pronouncing the Victim Dead[55,64,65,101,103,106]

Dilated pupils should not be taken as a sole sign of brain death in the lightning victim. It is always necessary to exclude other causes of pupillary dysfunction and eliminate them before death is declared. Hypothermia with lightning injury may cloud end-of-life decisions. If the victim has not regained a pulse after 20 to 30 minutes of resuscitation, it is reasonable to cease CPR.

### Long-Term Care‡

In the long run, there is no specific treatment for lightning injury. Lightning is a nervous system injury that can involve chronic pain, neuropathy, and brain injury, sometimes complicated by initially unrecognized musculoskeletal injury. Most symptoms can be treated in standard fashion, including cognitive therapy, pain management, job retraining, and counseling, as indicated by the survivor's signs and symptoms. As with any

*References 26, 66, 68, 71, 78-80, 89, 90, 393, 394.

†References 38, 86, 93, 104, 129, 181, 206, 268, 283, 284, 359, 360, 366, 380.

*References 56 ,62, 126, 127, 137, 165, 166, 204, 216, 230, 276, 293, 294, 336, 366, 395.

†References 14, 42, 44, 57, 153, 158, 164, 216, 239, 277, 298, 299, 328, 369, 388.

‡References 69, 107, 111, 243, 319, 359, 393, 394.

**TABLE 5-4** Drugs That May Be Useful in Lightning Patients

| Symptoms | Suggested Drugs |
|---|---|
| Pain | Nonsteroidal antiinflammatory drugs |
| | Acetaminophen |
| | Duloxetine |
| | Gabapentin and similar drugs |
| Social avoidance | Venlafaxine |
| | Desvenlafaxine |
| | Duloxetine |
| | Older tricyclic antidepressants, including clomipramine |
| Others | Fluoxetine |
| | Paroxetine |
| | Escitalopram |
| Resistant cases | Monoamine oxidase inhibitors |

serious illness, the caregiver and family may be the unsung heroes and need support, recognition, and counseling, as well as respite.

### Pain Control

Neuropathy and autonomic pain syndromes may develop in survivors. These may respond to chronic pain management therapies, such as combinations of nonsteroidal antiinflammatory drugs (NSAIDs), antiepileptic agents, antidepressants, and ganglionic blocks[26,30,176,177,320] (Table 5-4). Acupuncture may be helpful for resistant posttraumatic headaches that often accompany lightning injury.

### Psychological Problems and Cognitive Deficits

In recent years, neuropsychological deficits from lightning have become better appreciated.[17,230,315,319,320,378] Heightened anxiety states, hyperirritability, memory deficits, attention deficit, aphasia, sleep disturbance, PTSD, and other evidence of brain damage should be assessed.[30] Some of these deficits are similar to those suffered by victims of blunt head trauma. A rehabilitation program should be instituted early for these patients to return them to as functional a state as possible.[69,319,394,395]

Often, the victim's family and co-workers have difficulty understanding the change in personality that affects many of these victims. Neuropsychological testing to define the injury, establish a baseline, and plan appropriate cognitive therapy may be helpful. It is unknown if certain personality types may predispose to more pronounced neuropsychological symptoms. Other aids in assessment may include occupational therapy and psychiatric evaluation.

In some cases, the feeling of isolation and change that victims sometimes experience can lead to depression, substance abuse, and suicidal ideation. Because of unfamiliarity with lightning injuries and their sequelae, many physicians are poorly equipped to manage long-term care of lightning victims or may be so skeptical that the victim and family become frustrated and angry with their care. Although it is helpful if the specialists have familiarity with lightning survivors and the literature, it is not mandatory if they are willing to work with the survivors and their families and read the lightning literature.

It has been shown that clinical depression and electrical injuries are associated with decreased hippocampal mass and hippocampal cell atrophy.[51,116,241,352,362] The conclusion that untreated depression can cause brain damage necessitates almost mandatory antidepressant use. In electrical injury survivors, litigation has not been shown to be a factor,[178,179,370] but similar studies have not been done in lightning survivors. Regardless, treatment should not be delayed until litigation is complete. Depending on the symptomatology, some pharmacologic recommendations are included in Table 5-4.

A mainstay of treatment is ongoing psychologist consultation and support.[319,393,394] Therapy should be multifactorial and guided by the individual's symptomatology, as follows:

- For reestablishment of personal image and personal integrity in the face of lost function
- Aids and techniques for living with memory dysfunction
- Aids and techniques for concentration and motivation assistance
- Consideration of adapting to other limitations
- Social support
- Family and relationship counseling
- Adjunctive treatment for depression and anxiety

Treatment may also be needed to address adaptation to a new life circumstance if the injury is severe enough that the individual cannot return to previous employment and family situation.

Less conventional treatment methods, such as eye movement desensitization and reprocessing (EMDR) therapy, may have a place, but these should be performed in the hands of an experienced practitioner and tailored to the individual.

### Referral to Support Groups and Other Information Sources

Lightning Strike and Electric Shock Survivors International (LSESSI), a support group founded in the late 1980s, has numerous materials for survivors and their families (PO Box 1156, Jacksonville, NC 28541-1156; Tel: 910-346-4708; http://www.lightning-strike.org).[110,111] Other useful websites are http://www.uic.edu/labs/lightninginjury and http://www.lightningsafety.noaa.gov.

## FORENSIC INVESTIGATION

An unwitnessed lightning event can be one of the most difficult clinical presentations to diagnose.[72,148] The forensic examination of a critical lightning event can be divided into five stages[48,261,356,391]:

1. Case history
2. Scene investigation
3. Physical and/or autopsy examination
4. Special procedures
5. Collation

### CASE HISTORY

If a witness is available, it is important to answer the following questions:

- Was there a storm?
- Was there lightning?
- Did the witness actually see the lightning strike the victim?
- Was death immediate, or not?
- Where was the deceased person at the time of the strike (e.g., under a tree, on an open golf course)?
- Was any attempted resuscitation applied?
- What was the activity of the deceased person before death?
- A meticulous description of the lightning event must be given.
- How many people were involved?
- Were there any survivors? If so, where are they?
- What was the medical history of the deceased person? Specifically, were there any cardiac problems?

A history of electrical storm activity should be ascertained from the weather service, because lightning network detection and location systems should be able to assist with the exact time and location of the strike.[138]

### SCENE INVESTIGATION

Attending a critical lightning incident is a very specialized activity that crosses many disciplines. Insurance investigators, electrical engineers, scene reconstruction experts, and/or investigating officers will be called to review the scene of a lightning strike. Signs of lightning strike on the scene can be subtle or blatant.[210]

Lightning scene investigation can be divided into the following:

1. Environmental signs of direct lightning strike
2. Structural signs of direct lightning strike
3. Trace evidence signs of direct lightning strike

### Environmental Signs of Direct Lightning Strike

- At the scene, there may be damage to nearby trees, such as splitting or removal of bark.
- Arc marks may be present on the walls or nearby structures.
- The ground may display a fern pattern.
- Soil may show fulgurite formation—bore or tube-like structures formed in sand or rock by lightning.
- Often a crater will be exposed in the earth, with rock and sand being flung far afield. Craters of up to 2 m (6.6 feet) in diameter have been reported.
- To preserve the case history for scientific purposes, a relevant academic institution or other expert in the field should be advised of the incident, particularly if there is any indication of intent to file a lawsuit by a surviving party.

### Structural Signs of Direct Lightning Strike

Jandrell and associates[210] described the effects of direct lightning strike to housing structures in southern Africa.[161,270,271] Figure 5-19 shows damage to the roof of a clubhouse from direct lightning strike. The damage was extensive and included structural and internal damage (see Figure 5-20). Figure 5-21 shows damage to curtaining material covering the window. Thatched structures have been known to ignite following lightning strike. Thatch is very combustible, so inhabitants are at greater risk for severe injury and burns.[161,187]

### Trace Evidence Signs of Direct Lightning Strike

A direct strike can be very difficult to prove. Cindering on clothing or arc marks on metallic structures may be seen. In "zincification" and "cuprification," metal with a lower melting point vaporizes, leaving the other metal behind. Magnetization of metallic objects has been mentioned in the literature, although current thinking is that this may be a myth. Reports of metallic chains being magnetized and "sticking" to metallic postmortem trays have yet to be verified.

## PHYSICAL AND/OR AUTOPSY EXAMINATION

A complete postmortem examination of a lightning victim should be performed.

- The external examination should include a meticulous description of clothing and any evidence of resuscitation (Figure 5-38).
- Metal objects may have burned underlying skin or may have been marked by the heat of electrical arcing but did not attract the lightning strike (Figure 5-45). Figure 5-39 shows damage to clothing and underlying burns. Figure 5-42 shows permanent tattooing from a metal necklace burned into skin in a nonfatal injury.[215]
- Metal objects may show signs of fusing, zincification, or cuprification. Always check for magnetization. Metallic objects, such as tooth fillings, spectacles, belt, buckles, coins, and pacemakers, should be specifically described.
- The type, pattern, and distribution of any cutaneous thermal injuries, including clusters of punctuate burns, blisters, or charred burns, should be noted. Figure 5-44 shows damage to a hiking shoe with underlying damage to the skin of the foot.[26]
- Determine if there has been tympanic membrane rupture. Barotrauma, including pneumomediastinum, has been cited as one of the injuring mechanisms of lightning.[169]
- Note singed or scorched hair.
- Ocular injuries, such as retinal detachment, should be determined. Cataracts may be difficult to demonstrate postmortem.
- Unique arborescent or fern-like injuries (Lichtenberg figures) should be noted (see Figures 5-40 and 5-41).

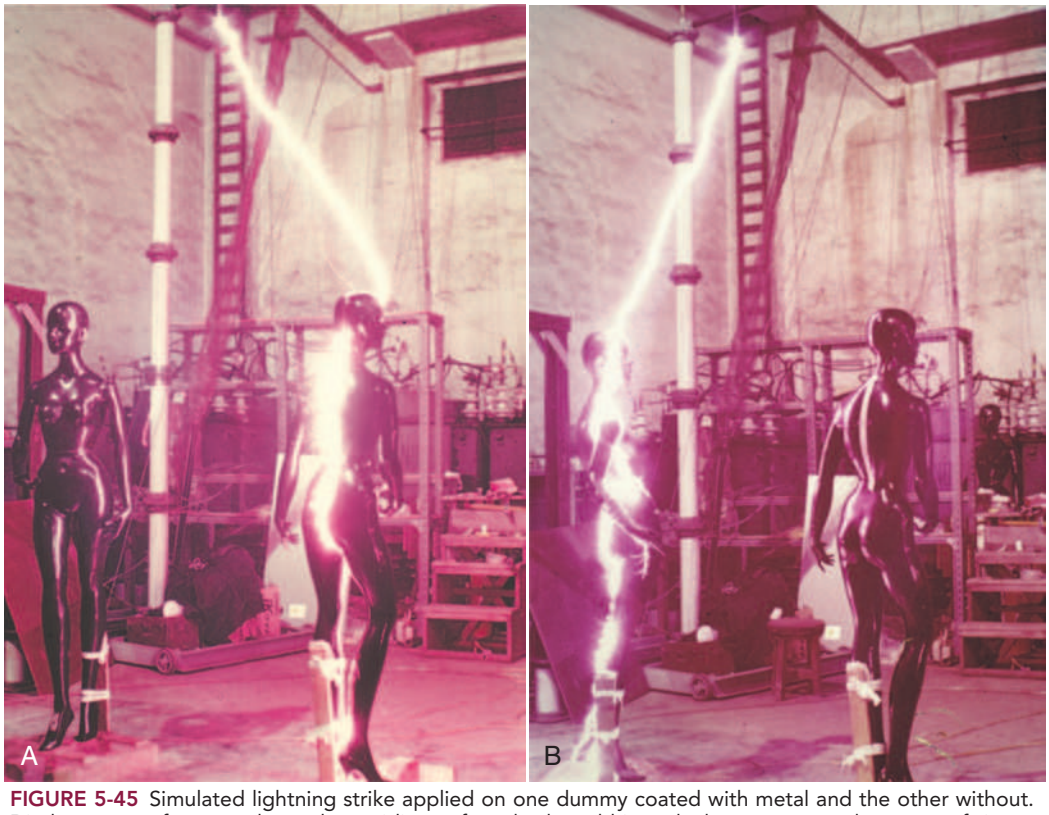


**FIGURE 5-45** Simulated lightning strike applied on one dummy coated with metal and the other without. Discharges ran from an electrode equidistant from both and hit each dummy an equal amount of times, showing that metal does not attract lightning, including anything a person wears. *(Copyright Nobu Kitigawa; courtesy Mary Ann Cooper.)*

- Determine if there are lightning wounds on the bases of the feet—the "tiptoe" sign typically on the base of the foot.[283]
- The procedure for internal examination is identical to that for any forensic autopsy.
- In female victims, ascertain if the victim was pregnant, and if so, carefully examine the fetus macroscopically and microscopically for injuries.[150]

## SPECIAL PROCEDURES

### Diagrams and Photographs

Where possible, diagrams of the pattern and distribution of the lightning injury to the body should be constructed to provide graphic documentation of the nature and extent of the electrothermal injury patterns. Close-up and distance photographs should be taken to document all injuries.

### Radiographs

Radiographic examination may be helpful. Certain fractures, dislocations, and subluxations may be missed at autopsy.[211] CT or MRI might add to the body of knowledge that constitutes postmortem, noninvasive, and virtual analyses. Certain keraunopathologic findings, such as pneumomediastinum, may be missed at autopsy.[169]

### Histologic Examination[209,213]

Skin burn wounds should always be microscopically examined for signs of electrothermal injury patterns, such as vacuolation in the epidermis, eosinophilia, and elongation and streaming of nuclei in the lower epidermis. Histologic staining of the heart with hematoxylin and eosin may prove useful. The heart should also be carefully examined microscopically for signs such as "waviness" of the myofibers,[211] necrosis, and contraction bands.[396] Special preparation of the heart with each of Mallory, Weigert elastic, Movat pentachrome, and acid fuchsin orange stains, as well as immunohistochemical staining with monoclonal anti–complement C9 antibodies, may aid the diagnosis of myofiber breakdown, an antemortem change that may be a distinct finding in electrothermal injury cases.[143] Any neuropathologic condition should be specifically investigated and addressed.

### Toxicologic Studies

Ethanol, recreational drugs, and carbon monoxide (CO) levels are the minimum toxicologic investigations required in lightning cases. From a mitigating-circumstance point of view, ethanol and recreational drugs are always important to know. CO levels will be valuable, especially if there is a thermal component to the injuries.

### Collection of Evidence

Investigators should collect and preserve evidence or specimens, because equivocal cases may require electrical testing of equipment by an expert. Nearby damaged electrical equipment should be sent to an electrical engineer for testing. Unwitnessed lightning cases are typically complex, and unusual situations may arise occasionally. The approach to all these cases should be multidisciplinary. Only by means of a careful forensic investigation, with strict adherence to guidelines, will the truth be revealed. This becomes even more important in determining whether a lightning strike was the cause of a later medical condition.

## COLLATION

> ***If the fresh facts which come to our knowledge all fit themselves into the scheme, then our hypothesis may gradually become a solution.***
> SHERLOCK HOLMES
> *The Adventures of Wisteria Lodge*

Data become information, which becomes knowledge, which becomes scientific opinion. Scientific opinion depends on experience, cognitive ability, and facts. At the end of the investigation, investigators should collate their findings with the known physics and effects of lightning.[325]

# PRECAUTIONS FOR AVOIDING LIGHTNING INJURY

## LIGHTNING SAFETY GUIDELINES*

Most lightning casualties in the United States involve only one person at a time. The possibility for multiple injuries exists where large crowds gather for fireworks, holiday beach outings, sporting events, concerts, and similar situations. Lightning injury prevention behavior should be proactive, rather than reactive, after the threat becomes imminent. Clearly defined education is important, *in advance,* to help make proper decisions at the critical times when lightning threatens. Prevention is more important than cure, as is apparent from the impacts of lightning injuries described in the medical discussions of this chapter. There are 20 to 25 million cloud-to-ground lightning flashes in the United States every year, and one-half have a subsequent return stroke coming to ground at a different location up to a few kilometers from the first stroke.[367] For this reason, it is impractical for the National Weather Service (NWS) to warn of every potentially dangerous lightning flash, so the key to safety is individual education and responsibility.[84,250,252,384] Only one-quarter of U.S. lightning fatalities are associated with tornado or severe thunderstorm warnings issued by the NWS.[35] As a result of this situation, a broad collection of lightning safety guidelines for minimizing the lightning risk is available from a multiagency group coordinated by the NWS[295] at http://www.lightningsafety.noaa.gov.

Cloud-to-ground lightning data from the NLDN have shown that lightning deaths and injuries occur in generally equal portions before, during, and after the strongest lightning activity in a thunderstorm.[201,250] Weak thunderstorms are at least as likely to result in casualties as are moderate or strong storms.[193] A detailed study by Lengyel and associates[250] found that almost half of lightning victims had enough warning to reach safety from nearby lightning before they became a lightning casualty, so that anticipation would have been beneficial. A recent publication documents in detail two cases in Rocky Mountain National Park involving low-flash-rate storms that killed people on successive days.[183]

A multidisciplinary group of lightning safety experts met in 1998 to develop guidelines that had not been adjusted in any meaningful way for several decades.[201,399] Development of national lightning detection networks and availability of other meteorologic data sets had caused major rethinking of existing guidelines. Since 1998, most of the guidelines have been supported, others have been evaluated, and some have been adjusted further or placed into a more limited context. At this point, most pre-1998 recommendations are considered to be based on false assumptions and have become obsolete.

## LIGHTNING SAFETY PLAN†

For a person watching the sky who is prepared by being aware of the forecast, lightning rarely appears so suddenly that precautions could not have been taken to minimize the risk. Thunderstorms take tens of minutes to develop or move into an area. A surprise is avoided by a series of steps in a lightning safety plan. The plan includes knowing the safest place to reach, how long it takes to reach it, how far in advance action should be taken, who makes the decision, and backup plans when people or situations change. The National Athletic Trainers' Association (NATA) has recently published a full description of how to make such decisions.[384] The National Collegiate Athletic Association (NCAA) website has a succinct version at http://www.ncaapublications.com/productdownloads/MD10.pdf.[43] A checklist for lightning safety has been developed for large venues, stadia, and other situations and can be accessed via a toolkit at http://www.lightningsafety.noaa.gov/more.htm. The NWS works with groups to tailor large-venue situations to their setting. Their Storm

*References 43, 104, 105, 117, 132, 196, 201, 251, 295, 338, 344-346, 384, 399.

†References 43, 105, 117, 132, 201, 295, 343, 346, 344, 384, 399.

Ready Program covers most outdoor storm threats and is useful for camps and hiking programs and in planning for many other wilderness or near-wilderness situations (http://www.nws.noaa.gov/stormready/).[296]

Before working in the outdoors or going on a recreational trip, be aware of weather forecasts and conditions. If thunderstorms are forecast for later in the day, pay attention to updates by using National Oceanic and Atmospheric Administration (NOAA) weather radio, cell phone lightning alerts, websites, and tailored information from private weather providers. Because access is available for much of the United States via mobile methods, there should be fewer surprises with regard to storms that are growing or moving into an area. Users, however, should be aware that many sources of weather data, particularly free ones, may be delayed by several minutes.

Thunderstorm and tornado warnings issued by the NWS indicate that thunderstorms are almost certain to occur in the area, and most likely in surrounding counties. However, most lightning does not occur within a warning area, but appears in less intense, yet frequently nonsevere, thunderstorms.[35]

Most lightning occurs during the summer months of June, July, and August (Figure 5-46A).[188] Furthermore, most lightning occurs between noon and 6 PM (Figure 5-46B).[187] These general figures make it much more apparent when to avoid long, risky exposure to lightning. Storms begin before noon on some days, particularly in locations such as over the high mountains of Colorado, where a few flashes can occur by 10 AM on active days. In such a location, starting hikes very early in the morning helps to eliminate lightning threat, while beginning a hike in late morning on active thunderstorm days results in increased vulnerability to lightning.[186,397] Summer lightning may linger into the evening in almost all regions of the country; unfortunately, this is the time of day when sports and recreation users are more likely to be outdoors.[43]

## AN APPROACHING THUNDERSTORM

Pay more attention to lightning than rain. Approximately 10% of all cloud-to-ground lightning strikes occur without rain at the location of the ground strike, so waiting for rain to arrive does not provide certain protection from lightning when a thunderstorm approaches. Thunder can be heard up to about 10 miles away in quiet conditions, but not nearly that far in the presence of wind or traffic, or when inside a structure.

A simple rule at the beginning of a storm is, "When thunder roars, go indoors."[182,292,-342-344,346] This rule removes any doubt about whether it is time to take action and is effective as a thunderstorm approaches. Although there is some overwarning because of distant lightning that may not reach a location, it is an effective and easy way to manage the lightning threat.

For a more objective approach, use the 30-30 rule developed at the 1998 lightning safety meeting.[43,105,132,193,264,384,399] The first 30 refers to the time in seconds between seeing lightning and hearing thunder from that flash (the second 30 refers to the wait time; see next section). If the interval from flash to bang is 30 seconds or less, people are in danger from lightning and should actively seek a designated safe place. This count of 30 seconds indicates lightning to be no more than 10 km (6.2 miles) away, using the speed of the sound of thunder of 5 sec/mile. Ten kilometers (6.2 miles) includes about 80% of all subsequent

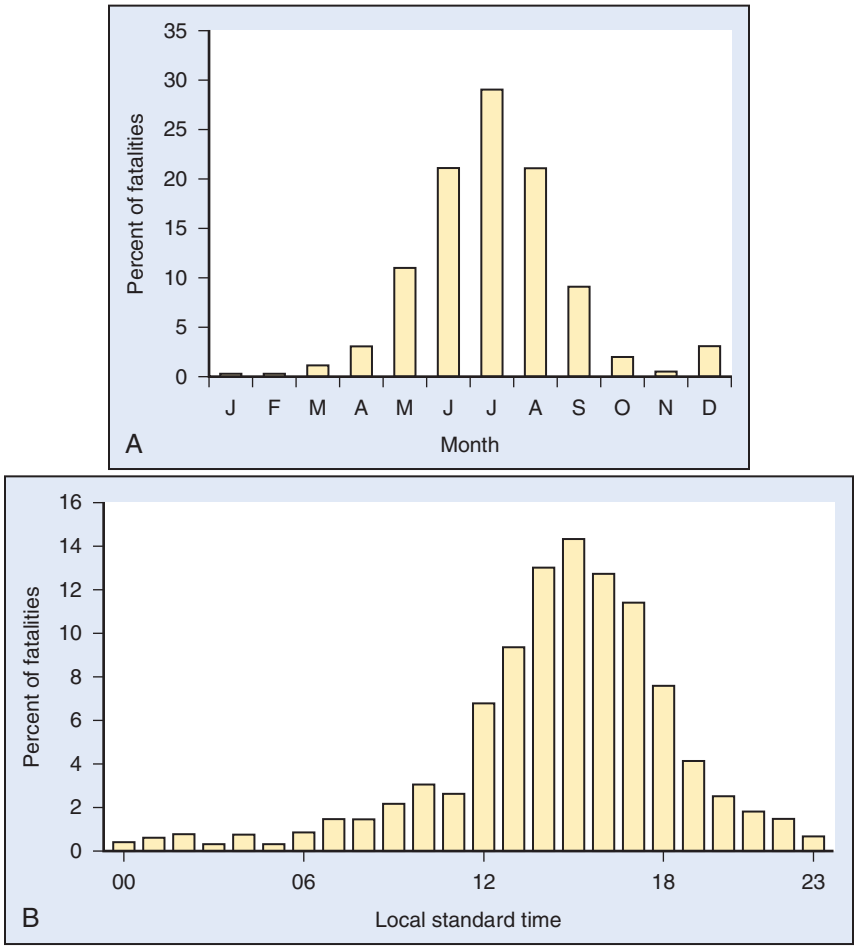


**FIGURE 5-46** **A,** Lightning fatalities per month from 1959 through 1994 for the United States. **B,** Hourly distribution of U.S. lightning fatalities. *(From Curran EB, Holle RL, López RE: Lightning casualties and damages in the United States from 1959 to 1994,* J Climate *13(19):3448-3464, 2000.)*

cloud-to-ground lightning flashes in a storm.[202] Variations of the 30-second rule are widely used at military and civilian airports for radii between 8 and 16 km (5 and 10 miles), where validated and accurate cloud-to-ground lightning detection systems are used. In such situations, too many warnings from a large radius around a point may result in a lack of trust in the method, whereas too small a radius misses too many storms and leads to more injuries.

Comparison of the two methods indicates that "when thunder roars, go indoors" is a useful approach for everyday use. The first 30 of the 30-30 rule corresponds to a 10-km (6.2-mile) radius and is more objective than the previous phrase. It may be adjusted based on local preferences to balance downtime with operational efficiency at a facility such as an airport or mine. With either approach, when the rule indicates that lightning is a threat, attention should focus on lightning rather than rain. Outdoor activity should be stopped, with immediate evacuation to a designated safe place.

## END OF THUNDERSTORM

Do not underestimate the danger of lightning at the end of a thunderstorm. As many people are killed or injured by lightning at the end as at the start or during the middle of a storm.[202,250] A number of people are killed every year when going outside into the backyard of a home too soon because of impatience, or crossing a field or parking lot before the storm is finished.[187,191]

The second 30 of the 30-30 rule says to wait 30 minutes after the last lightning is seen or thunder is heard before resuming outdoor activities.[43,105,132,201,399] At night, flashes may be visible low on the horizon inside tall thunderstorms up to 80 km (50 miles) away, but these are not of much concern unless the lightning channel itself is visible to the ground.

Since the 1998 meeting, it has become apparent that 30 minutes is longer than is needed in most situations because the lightning threat is minimal after 15 minutes.[202] In a large-group situation, however, where a long evacuation time is required, the full 30 minutes is needed to avoid returning people to a field or stadium too soon, then needing again to send them to safety a few minutes later when the lightning threat returns. In one's own backyard, a person can wait 15 minutes after lightning and thunder and be quite safe, although a lingering flash can cause return to a dwelling in a matter of a few seconds if necessary. In practice, most airports and other industrial situations use a warning expiration time of 15 minutes, although local high levels of safety concerns may make 30 minutes preferable. Less than a 10-minute wait time is not recommended. There are exceptions to all rules when a large, overhead thunderstorm lingers for hours over a location.[214] In such a situation, any policy may need to balance between efficiency and safety of outside workers.

## SAFE PLACES INSIDE

There are two reliable places to be safe from lightning: inside a large, substantial building and inside a fully enclosed, metal-topped vehicle.

### Buildings

Large, substantial buildings where people live and work are very safe from lightning in more developed countries. Although such structures are often hit directly by lightning, fatalities inside them are extremely rare, considering the amount of time spent by people inside them. In a study of U.S. dwellings and buildings, the only lightning-caused fatalities among many hundreds of cases were to older adults, very young persons, or persons with physical or intellectual disabilities who were unable to leave the dwelling when a fire broke out, almost always at night.[187] There were no fatalities inside offices, schools, or other large buildings. As a result, adequate safety is attained by directing people to go inside a large, substantial building, including a dwelling, rather than staying outside.[292]

The protection in modern buildings is provided by grounded wiring and plumbing, as well as metal structural members inside the buildings that carry the charge of a strike to the structure around people and into the ground. Contact with conducting paths of wiring, plumbing, corded telephones, and large openings, such as garages and doors, can result in injuries. Such contact needs to be avoided during the presence of lightning to avoid potentially serious injury, but these are not known to lead to fatalities in well-constructed and grounded buildings.


**FIGURE 5-47** Lightning-unsafe picnic shelter in Tucson, Arizona. Note warning sign on roof, which states: "WARNING: This structure is unsafe for shelter during lightning storms. Seek suitable shelter elsewhere."

Unsafe indoor locations are small structures such as those used as golf, beach, sun, rain, school, agricultural, or bus shelters (Figure 5-47). All these structures should be considered unsafe and must be abandoned for a larger, safe building. It is possible to make such small structures safe, but their protection must be designed, installed, and approved by a knowledgeable, experienced, bonded insured lightning protection specialist.[229] Also on this list of unsafe structures are all tents that only provide rain protection but no barrier to lightning.[190] Pads on the ground surface inside a tent are of no value for lightning protection; composition of the tent structure and poles provides no protection.

In less developed countries, dwellings and workplaces may be thatched-roofed huts that provide no protection from lightning because they are often ungrounded and made with nonconducting material.[187] Note that it is possible to provide low-cost lightning protection for such unsafe structures by using simple towers and natural local materials for grounding; however, these must be designed and installed by knowledgeable lightning protection specialists using internationally accepted, scientifically based standards to ensure their efficacy.[240]

### Vehicles[191,214,292,399]

Fully enclosed, metal-topped vehicles are safe from lightning and should be used as a safe place when no large substantial building is available. In a study of hundreds of vehicle incidents, there were injuries to vehicle occupants in less than one-half the cases; the majority said the experience was frightening, but they emerged unscathed. There have been no documented "electrical" injuries to occupants, with the exception of those involving direct wiring, such as from older handheld police radios.[191] The only unambiguous fatality was an older adult who was startled or incapacitated by a nearby flash and drove into oncoming traffic. Damage to vehicles ranged from minor, such as antennas vaporizing, to a few cases of major engine failure and electrical fires; nevertheless, all passengers escaped the vehicles.

Unsafe vehicles are those without the safety of fully enclosed metal surroundings that would otherwise act in a manner consistent with a Faraday cage.[99] Safety is provided from a direct vehicle strike by the lightning energy traveling across the outside shell of the metal vehicle and around anyone inside, with

subsequent arcing to the ground through bumpers or axles. This path may account for the mistaken impression that tires are the safety feature; they are sometimes blown apart by current passing through the vehicle's metal frame (nearest to the ground) through the tires. Unsafe vehicles include those with cloth tops (convertibles) or fiberglass or plastic bodies. Others include golf carts and four-wheeled conveyances with open sides. Strikes to such vehicles have no defined path to ground, and it is not safe for people to be inside them. Although it is possible to design lightning protection for unsafe golf carts and other open-sided vehicles, there is no such commercially available product; such an approach requires a specific configuration and correct behavior of people inside them.[119]

Any place outside such a vehicle is as unsafe as anywhere else outside.[191] Particularly dangerous is *step voltage,* when a person is in contact with both the vehicle and the ground. This situation occurs when stepping into or out from a vehicle when lightning strikes the vehicle, because the step potential between the energized vehicle and the ground is very large. Conversely, a nearby ground strike will travel to a person with one foot on the ground and the other in contact with an unaffected vehicle. Other situations outside a vehicle include frequent cases in parking lots, waiting for buses, and law enforcement and other people near but not inside disabled vehicles.

A fully enclosed, metal-topped vehicle can be used as a safe place at a school or sports event. If flashes will strike the ground at such a venue, it is an easy choice to be inside such a vehicle rather than outside at the same location.

## ALWAYS UNSAFE OUTSIDE[281]

There are no reliable places outside to be safe from lightning. Almost complete safety can be achieved by being inside a large, safe building or a fully enclosed, metal-topped vehicle, as described in the previous two sections. Some recommendations continue to emphasize the speculative and ultimately unsuccessful safety approaches outside, at the expense of noting the reliable safety that is often present in more developed countries in nearby substantial buildings and fully enclosed, metal-topped vehicles.

One of the most important misconceptions of such outdoor safety advice is the expectation that the direct strike is the most common mechanism of lightning injury. As described earlier, it is estimated that only about 3% to 5% of lightning injuries and deaths are caused by direct strikes. The result of this mistaken direct-strike approach is the recommendation that lowering one's height is sufficient to be safe, rather than recognizing the other, more likely mechanisms and using reliable safety plans and evacuation.

The reliable lightning safety approach is to recognize the lightning threat early and go to the known safe places of buildings and vehicles. The sooner a person reaches the safety of one of these locations, the sooner the lightning threat is ameliorated. As a result, the everyday lightning situation encountered by an individual is to anticipate the lightning threat, know where a safe building or vehicle is located and how long it will take to reach that location, and complete the plan by reaching safety before lightning arrives.

Lightning is not predictable in its path to ground, and exactly what it strikes is not predictable with any certainty. Case reports have indicated that the prior speculative advice of seeking a small tree among larger ones is unreliable. Recent very-high-speed video shows lightning traveling toward ground in multiple branches. The first branch that contacts the surface of the earth is the only important one, whereas the rest dissipate in the air. Which branches reach the surface appears to be random within the flash.[354]

Roeder[342] examined the relative value of common advice on outdoor lightning avoidance according to the five mechanisms of lightning injury (see Specific Strike Mechanisms, earlier). It was found that if every precaution were to be followed, and some are quite difficult, only a 50% reduction in risk would be achieved. The other 50% of the time would result in an injury or fatality. As mentioned, one of the least effective approaches is to stress the direct strike by lowering one's height, because the direct-strike situation is scarce.

### The Difficulty of Wilderness Situations

No action will achieve certain safety from lightning in the wilderness away from a substantial building or fully enclosed, metal-topped vehicle. The two approaches to lightning safety in the wilderness are to avoid the risk in the first place and to accept that a lightning risk exists.

The amount of time spent by hikers and climbers in multiday situations is quite small. The more common situation is a hike or climb lasting a day or less, often on a weekend day.[190] For this common situation, the first approach of avoiding the risk is most manageable, since a vehicle or sometimes a sufficiently large building is likely to be available at the trailhead. Pay close attention to the forecast for the day, and avoid the daytime period when lightning is most prevalent. Over high mountains of the western United States, for example, storms start as early as 10 or 11 AM local time, so hikers should be off the higher parts of the mountain by that time. Otherwise, postpone the hike to another day. Such an approach does not necessarily preclude hiking on most days; a study over Colorado's Rocky Mountains[271] showed that no location had lightning on more than one-third of summer days, and almost none during spring and fall months.[194,258] Therefore, hikers can manage the risk by choosing the time and place of a day hike. Once the hike is underway, they should pay attention to evolving cloud formations that indicate future lightning, stop the hike or climb, and return to safety when indicated (see An Approaching Thunderstorm and End of Thunderstorm, earlier).

The pressure of a schedule or other factors may be such that a wilderness activity will take place despite the possibility of a very real lightning threat. Thus, a hiker or climber has accepted the personal risk in the same manner as risks from dangerous animals, falls, or other natural hazards. Roeder[342] analyzed the relative risk when no acceptable buildings or vehicles are nearby. The five mechanisms of lightning injury discussed earlier help to identify actions that can be taken, such as crouching (minimal value) and staying away from tall objects. The results of the Roeder analysis show a reduction in risk to about one-half the full lightning exposure. In other words, following all precautions does not prevent half the lightning risk. Because the steps must be performed correctly, remembered under duress, and reduce the risk for death or injury by only one-half, they are not to be considered when lightning safety can be readily attained by entering a safe building or vehicle.

## SAFETY OF LARGE GROUPS

An individual can respond to lightning threat quickly by going inside a large, substantial building or fully enclosed, metal-topped vehicle. In less than a minute in the backyard of a dwelling, for example, quick action places the person in safety. When the threat is over, using a rule such as 15 minutes for the cessation of lightning and thunder, an individual can return to the backyard. If the lightning threat returns, a person can quickly go back into the dwelling or a safe vehicle until the threat passes.

For larger crowds, all these steps are more difficult. Steps include accepting that lightning threat is important, knowing how to identify lightning threat, where safe locations are located and how long it will take to reach them, recognizing when the threat is over, and who makes important decisions.[43,105,117,201,258,264,295,384]

In the case of a neighborhood youth soccer game, for example, safety can be reached in minutes inside nearby fully enclosed, metal-topped vehicles. When game officials or others make the decision to go to safety, players and spectators need to go immediately to the cars, vans, or buses. Note that small venues may have rest rooms or concession stands that are likely to be unsafe from lightning, and people in those locations need to be informed to abandon them for the safety of vehicles commonly located in parking lots around the field (see Buildings section of Safe Places Inside, earlier). At a somewhat larger school sporting event, including practices, there are likely to be nearby lightning-safe large buildings. Sports and custodial staff need to be informed

to leave doors unlocked or be available to open doors when players and spectators need to get inside in a hurry.

When the crowd is large at a sporting or other outdoor event, the situation becomes more complex. Advance planning is essential. The first step is convincing a venue owner or manager in advance of the potential lightning risk. Many lightning threats are accompanied by rain, strong winds, hail, and other significant weather, so that placing lightning into the context of the thunderstorm threat may be more acceptable. In the United States, the lightning threat at collegiate football games was examined by Gratz and Noble,[162] who found that a single game's threat is not large, but the large number of games per year makes a lightning incident at a football game inevitable over a period of years. Holle and Krider[196] present a case study of an individual game's response to the lightning threat. Edwards and Lemon[132] emphasized the potential for major storm-related disruptions at auto, dog, and horse races; professional and collegiate sporting events; and outdoor concerts, examining situations such as close calls of tornadoes passing near large crowds. However, their warnings have often been met with silence, skepticism, or limited positive responses.

There are now lightning safety recommendations for schools and U.S. athletic programs from the NCAA and NATA.[43,378] Nearly all institutions follow these recommendations, so it has become common for all types of U.S. collegiate sporting events, as well as many professional sporting events, to be delayed, suspended, or canceled. This wisdom has spread worldwide such that lightning safety policies are now followed globally in athletic events, at least to some degree. There have been almost no lightning casualties over the last decade in the United States at organized sports events. It is a useful tactic in convincing venue operators that they should follow the lead of the NCAA and NATA in accepting the lightning risk. This recommendation applies equally to practices and rehearsals.

Once there is acceptance of the lightning threat, a method of identifying the existence of lightning is needed. For a large crowd, the sound of thunder is probably not applicable. Reliable, proven, and accurate cloud-to-ground lightning data are available on cell phones, websites, and other portable devices that objectively measure the presence of lightning. Any purchaser or user of these devices should be certain that lightning data are real time. Rules need to be established for behavioral triggers at a particular stadium or event site. Stadium managers need to know how long a lead time is needed to evacuate people to safety. Once that time is decided, it determines the distance to approaching lightning for action to be taken. The rule needs to be followed objectively on days when the threat exists. Programs may choose to use the checklist for lightning safety that has been developed for large venues, stadia, and other situations by invoking a toolkit at http://www.lightningsafety.noaa.gov/more.htm. The NWS has worked with many groups to tailor large-venue lightning safety to their settings.

When an announcement is made of a lightning threat, the plan goes into action. At a football game, for example, players and coaches may try to stay on the field, which gives the impression that the situation is not really important. In every case, the game or performance area needs to be empty and nothing shown on video screens except the current safety announcement, so that the event no longer provides an attraction to the crowd. In many stadiums, there is adequate room beneath the stands.[196] At other locations, a nearby gymnasium or school building may be the best alternative, so plans need to include permission to enter those buildings. In the case of a golf tournament, the first concern of organizers is for player and sponsor safety; they can be sent quickly to a safe building or vehicle. However, spectators need a much longer time to reach the safety of their vehicles and may need to walk several miles.

While at the safe location, information needs to be given to the audience and participants concerning the status of warnings. This can be difficult in many situations, so warning horns or message boards may be the best approach. Informing people waiting in diverse locations involves complete and active planning in advance on the part of the venue operator. The safe location also needs to be comfortable enough for the crowd to accept a long wait. Appropriate signage or inclusion of lightning safety information in event programs may also be useful in communicating safety information.

The "all clear" at the end of the lightning threat needs to be identified as objectively as is the warning at the beginning (see End of Thunderstorm, earlier). As crowds become larger, a longer wait time until the all-clear signal is appropriate. Nothing is more ineffective and reduces confidence more in the evacuation process than sending people back to their seats, making them return to a safe place again a short time afterward, only to return once again to their seats. For a large stadium or golf tournament, for example, at least 30 minutes should be used for the wait time, and still longer times may well be needed to avoid back-and-forth crowd movement. At a fireworks show, people may be unwilling to leave a favored spot on a field, so crowd control and security specialists may be needed to address the situation. However, in many cases after the first suspension of a concert, for example, some of the crowd does not return, so the problem may be reduced.

Instead of moving a large group from one place to another, it is possible, and usually much less expensive than managers might expect, to provide safety in place. The approach is to place large, properly grounded poles and overhead wires that divert the flash from striking a crowd by using these down-conducting paths into the ground.[15] The principles are the same as those routinely applied in lightning protection of buildings, towers, utility poles and lines, and many other aspects of modern infrastructure. This approach has long been used for space vehicles and other critical activities and structures. There are great benefits to installing such a system, such as no evacuation, no need for safe places to be identified, and no uncertainty in the process and its execution. However, people must stay at the correct locations within the protection provided by such a system and trust that it will work as intended. Flags or banners at the top of the protection poles can make the installation as unobtrusive as possible for the crowd and media (Figure 5-48).

### Lightning Protection in Situ[15]

The Franklin rod is often the basis of lightning protection schemes. Many existing structures can be turned into such rods with ease without detracting from appearances. It is emphasized that the following examples are illustrative only, and proper design is needed when these are being implemented.

Figure 5-49 shows a pathway with light poles of a given height. Spacing of these poles may be selected to protect the pathway, depending on the height of the poles and width of the path. Figure 5-48A shows the "natural" protected zone around a temporary stand that might form an evacuation region if other risks are accepted. Franklin rods added along the back of the stand extend this zone and also add some in situ protection (Figure 5-48B). Useful Franklin rods are formed by flagpoles that have the required "look." A horizontal wire at a given height above a stand also provides in situ protection (Figure 5-48C). These can be made almost unnoticeable, especially to media cameras. Alternatively, they can carry banners, lights, or media cameras.

Figure 5-50 shows horizontal wires providing protection to a group of stands. The width of the protective corridor depends on the height of the catenary, and the position of the corridor can be altered as a design parameter for effectiveness.

Figure 5-51 shows a stadium for a field game (e.g., baseball), where an existing stand provides some protection, as do lightning pylons. The addition of overhead wires can complete the coverage.

Figure 5-52 shows the main stadium stylistically and how open stands at either end of the stadium can be protected by fine wires, which are all but invisible.

## CONTROVERSIES AND ONGOING RESEARCH IN LIGHTNING INJURY[92,95]

Much remains to be learned about lightning injury, and knowledge at present remains partial. Specific areas of controversy,

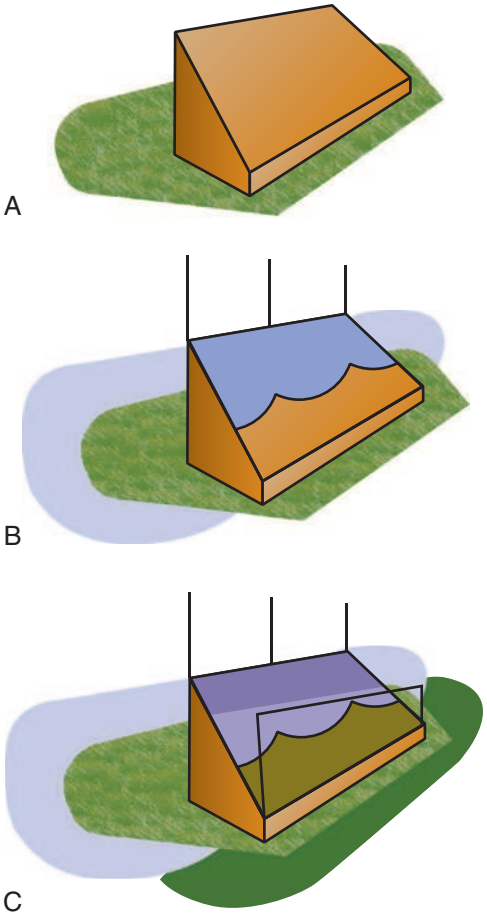


**FIGURE 5-48** **A,** Natural protection zone of a stand. **B,** The effect of Franklin rods disguised as flagpoles. **C,** The added effect of a catenary added to a stand. *(Redrawn from Crowd protection strategies: Experience from the Sydney Olympic Games, 2000. Presented at International Conference on Lightning and Static Electricity, Blackpool, UK, 2003.)*

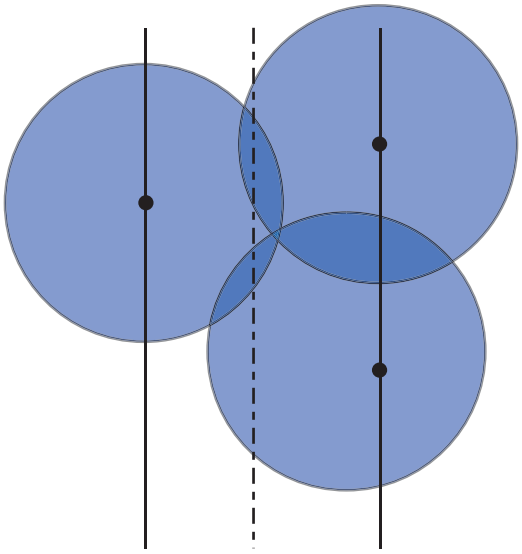

**FIGURE 5-49** Protected zones on a pathway from light poles of varying height. *(Redrawn from Crowd protection strategies: Experience from the Sydney Olympic Games, 2000. Presented at International Conference on Lightning and Static Electricity, Blackpool, UK, 2003.)*

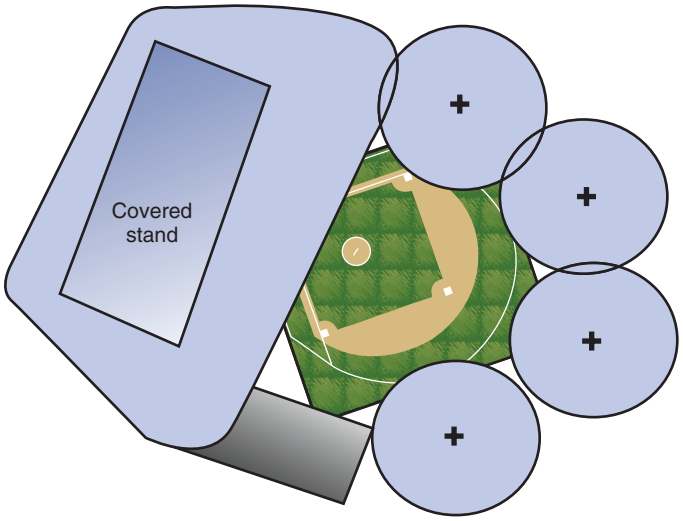


**FIGURE 5-51** Effect of existing covered stands and lighting stanchions. *(Redrawn from Crowd protection strategies: Experience from the Sydney Olympic Games, 2000. Presented at International Conference on Lightning and Static Electricity, Blackpool, UK, 2003.)*

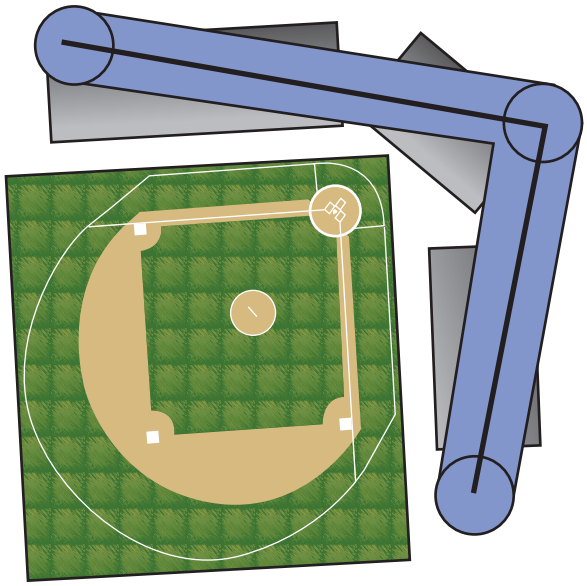

**FIGURE 5-50** Use of catenaries to protect several stands. *(Redrawn from Crowd protection strategies: Experience from the Sydney Olympic Games, 2000. Presented at International Conference on Lightning and Static Electricity, Blackpool, UK, 2003.)*

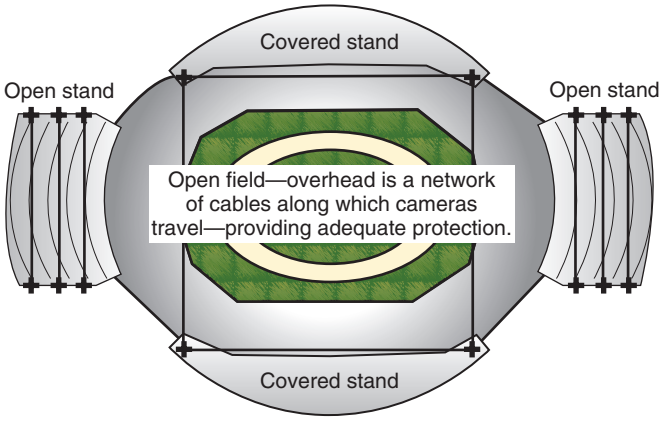


**FIGURE 5-52** Open field—a properly installed network of overhead cables that can double for movable cameras can also provide adequate protection. *(Redrawn from Crowd protection strategies: Experience from the Sydney Olympic Games, 2000. Presented at International Conference on Lightning and Static Electricity, Blackpool, UK, 2003.)*

limitations of present research, and currently debated features point the way for future research.

There are presently two major thrusts in *keraunomedicine,*[13] the interaction of lightning with humans and their protection: (1) the interaction between electric current and humans—the pathophysiologic changes that are induced and how they underlie lightning injury symptomatology—and (2) the protection of individuals and crowds from lightning injury. Lightning experts note the significant reduction in strikes and morbidity/mortality as good indices of success in this area. An entirely uninvestigated area is whether there are ways to mitigate the process of injury once it has occurred so that common sequelae can be avoided.

## PROBLEMS WITH EXPERT REPORTING[92,98]

At regular stages, medicolegal reporting, including to workers' compensation entities, is required for documentation and evaluation of an injury.

### Totality of the Injury

Rarely is there a report on the totality of an electrical or lightning injury. Most reports focus only on the section of an injury that is in a particular medical specialist's realm. The orthopedic aspects are often reported by an orthopedic surgeon as if the injury was the result of simple trauma rather than a complex electrical injury. Similarly, neurologists may report on neurologic aspects, rarely giving a view of the entire injury complex. They may take their knowledge from neurologic trauma coupled with misguided assumptions about neural vulnerability. There are three consequences:

1. There is seldom expert assessment of the physical/engineering aspects of the injury.
2. There is little perception of the total injury complex and its nuances.
3. There is an assumption, often erroneous, as to the precise physical aspects of the injury.

Recent vascular research[308] has demonstrated the importance of vascular damage in contrast to more commonly assumed neural damage. Current passage through body fluid (blood and CSF) is important and is much more supportable in theoretical terms than is neural transmission. In addition, release of humoral factors may well affect brain functions, with the cortisol-HPA (hypothalamic-pituitary-adrenal) axis particularly implicated (see next).

### Presumption of Site of Injury

Electrical injuries are uncommon and lightning injuries rare in most practices. Unless the physician has a special interest in them, they will usually make up only a tiny part of any physician's work. Because of unfamiliarity with the overall picture, physicians, both general practitioners and specialists, will often and quite naturally default to diagnostic tests with which they are familiar. Unfortunately, these tests and assumptions about the site of injury may not apply to the sequelae experienced by the survivor.[17]

It can be frustrating to the patient, family, and physician and devastating in disability cases when a physician reports, "None of the testing I ordered showed damage," or, "All of the patient's tests are normal." Unfortunately, although the physician is saying, "The tests are normal," the patient, family and courts often hear, 'There is nothing wrong," rather than that the wrong tests may have been ordered.

For example, nerve conduction studies (NCS) test only the largest nerve trunks, but do not test pain pathways, which usually contribute a large component of the lightning injury survivor's complaint. Fatigue, weakness, and sensory disturbances may be mediated by neural end-plate damage or sensory terminal damage, neither of which is detected by NCS. Functional deficits that may be readily apparent by history, talking with the family, neuropsychological testing, or other functional testing are not detected by anatomic tests, such as CT and MRI. Unfortunately, testing for damage to finer nerves and end plates, using functional MRI and other tests applicable to a survivor's symptoms, may not have been undertaken for technical, monetary, or other reasons. This does not mean the symptoms are imagined, only that appropriate diagnostic testing is not available at this time. Advancing knowledge may shed light on various new modalities to test, such as hippocampal size.[241]

### Limits to Reporting

Physicians may be more comfortable with or may be constrained to use codes (e.g., DSM) for existing and known diagnoses. This may lead to diagnoses of PTSD, adjustment disorder, and depression because they are secondary to the uncoded overall syndrome, which is more correctly a post–electric shock syndrome. These diagnoses only describe a portion of what the patient may be experiencing. It is not surprising that an electrical event should give rise to a trauma response or to difficulty adjusting to new limitations, brain injury, or chronic pain, but this is not the total picture. Unfortunately, psychiatric or psychological diagnoses may distract from recognition and treatment of underlying organic damage.

## RESEARCH METHODOLOGY PROBLEMS

Cooper[92,95] highlights further features of what is not yet known about lightning injury, documenting methodology difficulties. Without knowledge of the basic physiology of the injury, only general symptomatic aftercare can be rendered, instead of more specific treatments and early interventions that might stop or change the course of the injury cascade precipitated by the initial injury. Therefore, medical treatment is frequently empirical and based on symptomatology. Treatment of depressive symptoms is particularly important and should not be delayed until more exact pathophysiology is determined.

### Bias in Research

Research on lightning injuries is difficult (Box 5-5). No public health regulations require reporting, so cases are difficult to collect and survivors difficult to locate in any systematic manner.[67,198,273]

Although it may be convenient to target certain populations, such as members of LSESSI, as study participants, people who join support groups differ from those who do not. They represent a subset of survivors who may have systematic biases developed from the services and materials that LSESSI supplies to its members.[92,95,254] In addition, only limited research can be done with this group because of ethical limitations on human research, national dispersion, and lack of funding for research, among other difficulties.

**BOX 5-5 Research Problems**

**Human Research**
Recruitment of cases
Study biases
Dispersion of participants
Cases must be free from:
- Diabetes and other neuropathic illnesses
- Drug history
- Psychiatric history
- Blunt head trauma

**Animal Research**
Expensive—both animals and equipment
Large number of animals for some studies
Difficult signal-processing problems
Monitoring equipment design
Shock timing control considerations
Definition of dose
Standardization of dose
Flashover effect

**Molecular Biology**
Cell culture, blood levels of indicators of injury, etc.
Requires specialized techniques
Bioengineering, collaboration
Expensive

To separate lightning electrical effects from other etiologies, it is necessary to obtain relatively recently injured patients (no more than 6 to 12 months) with an otherwise uncomplicated history. There should be no past history of alcohol or drug abuse, head injury, psychiatric problems, or concurrent medical problems that cause neuropathic pain or psychiatric symptoms, and no blast effect during the injury that would confound investigation of the traumatic effects of lightning. Unfortunately, this narrows the available participant pool.

### Experimental Vehicles

Because it is difficult to recruit volunteers, credible animal work is a reasonable alternative.[12,14,208,226,301] Cooper and colleagues[107-109] have developed a reasonable rat model that demonstrates most of the clinical signs seen in humans. Unfortunately, such research is expensive, time-consuming, and sometimes requires large numbers of animals to show significant differences. Most problematic is the delivery of a "standardized dose" of simulated lightning that can reliably reproduce a specific ("standard") pattern of injury.

### Equipment Requirements

Many hurdles must be overcome in research equipment design, anesthetic choice, animal care, and shock timing and delivery. Monitoring equipment must be electrically isolated from the animal and shocking platform, or the connections may preferentially transmit the lightning shock to the equipment, not the animal, clouding the experiment and destroying the equipment. Some anesthetics are neuroprotective, and others affect cardiac function. Temperature control in anesthetized rats is a significant issue. Animals that are studied for neurocognitive injury using water maze and other behavioral models need a standardized quiet environment, which is not always possible in the cramped quarters of most animal facilities. In survival or cardiac studies, timing of the shock either to target or to avoid the "vulnerable period" must be done on a statistically predictive basis, because direct sensing of the specific targeted cardiac cycle would result in damage to the equipment.

### Survival Statistics

Box 5-6 lists factors concerning the unknown issues as to why the vast majority of lightning casualties survive.[67] Characteristics of a lightning strike or possibly the mechanism by which the current impinges on the individual may be different for lightning fatalities. Timing of the lightning strike may be the important factor, particularly if it hits during a more vulnerable portion of the cardiac cycle. Although possible, it is unlikely that the physical characteristics of people differ sufficiently to cause a difference in mortality. A more likely explanation is that many strikes may not meet thresholds (see IEC advances in next section) in particular cases.

### Remote and Psychological Symptoms

A further area of active research is the etiologic origin of symptoms not directly in the line of passage of current.[20] This includes industrial electrical injuries as well.[278] Even when there is no indication of current near the brain or evidence of blast/blunt injury, patient histories and neuropsychological tests consistently suggest an organic origin to the deficits.[17]

**BOX 5-6 Why Do 70% to 90% of Lightning Strike Victims Survive?**

- Lightning characteristics (possible factor)
  - "Type" of lightning
  - "Dose" of lightning delivered
- Timing of hit during cardiac cycle or other factors
- Pathway of lightning
- Other meteorologic conditions (possible factor)
- Physical characteristics of those "hit" (possible factor)
  - Comorbidities
  - Rescue/resuscitation efforts
  - Where people were/what they were doing (possible factor)

**BOX 5-7 Possible Injuring Forces**

- Blunt trauma—explosive injury
- Structural changes—direct damage
- Pathway of the injury
- Orifice entry
- Flashover—how much goes through versus around
- Neurochemical changes
- Autonomic nervous system effects
- Electrical effects
- Electroporation
- Cellular level mechanical effects
- Cellular level enzymatic effects
- Subcellular organelle damage

Awareness of the role of the spinal cord in nociception and existence of proximally directed neural pathways may provide a fruitful connection. Release of humoral transmitters may play a role. The importance of premorbid personality predisposition is unknown and difficult to measure.

Some note that the neuropsychological constellation resembles the psychological effects of other syndromes, such as traumatic brain injury and the response to autoimmune disorders. It is possible that the symptom complex represents a "final common pathway" of brain injury from many etiologies.[17] Box 5-7 lists other possible factors yet to be verified.

Andrews[20,21] and Reisner[330,331] have specifically developed cogent theories of the psychological injury. To be credible, these theories need to explain several facets, including the constellation of symptoms, its organicity, symptoms developing over time with some being time-lagged, and the specific underlying organicity localizing the injury.

Both theories are similar. Although some current will pass through the brain in any shock, it will often be minute. It is probable that a chemical generated in the current pathway will affect the brain; a likely substance is cortisol. The interaction between cortisol as a cytotoxic, brain-derived neurotrophic factor and recognition that the hippocampus is one of only two sites in the brain capable of cellular regeneration are noted parts of one theory.[21] Insights from the theory of depression implicate the hippocampus as a site of cell loss,[51,116,310,352,361,362,382] which is supported by both PTSD and psychiatric depression research.[310] The same cell loss occurs in industrial electrical injury.[241] Also, a substance yet to be identified is released from vascular epithelium with an electric shock, which can act at substantial distance on other epithelia.[308] The site in the hippocampus and limbic systems creates a good localization for the symptoms. Reisner's theory[331] is similar but implicates oxidative free radicals as a mediating influence. Research in this area is supported by advances in imaging technology provided by high-field MRI scanning.

### Technical Matters

The degree of lightning injury, or "dose," varies considerably with the mechanisms of injury (e.g., direct, splash, contact).[16] Thresholds are being quantified more accurately by the International Electrotechnical Commission (IEC) for short-duration pulses.[205] Other technical factors regarding interaction of a lightning stroke with the body, such as the effect of multiple return strokes and any "capacitive memory," are poorly known. Although flashover occurs, the amount and path of energy going through victims versus around them and the duration of energy flow initially or with return strokes are known only by implication.[16] The degree that flashover electricity can induce opposite charge, damage, or arrhythmias inside the body is unknown. Although these factors have been calculated based on engineering assumptions, actual measurements remain to be done. Finite-element modeling may offer the best alternative.

The IEC[205] recently discussed extension of pulse safety in two areas. It is now believed that with present knowledge, safety standards can be extended to cover the safety of lightning pulse widths from about 1 to10 milliseconds down to 1 microsecond.

This development is paralleled by extension of waveform frequency standards up to approximately 150 kHz, which is relevant to lightning circumstances and the cell membrane filter time constant.[151]

Knowledge of conduction mechanisms at shorter pulse widths and higher frequencies draws on tissue conduction dispersions, as well as known conduction and membrane excitation properties.

### Electroporation

In terms of the damaging influences of current on tissue, certain matters remain to be investigated. Lee[246] has corroborated electroporation of muscle cell walls with high-voltage electrical injury that generates high internal electric fields. Applicability of electroporation to lightning injury has not been established.[88,92,246]

### Sites of Injury in the Body

Microscopic studies are needed to examine actual fine loci of injury within the body. For example, what might be the effect on motor end plates and the neuromuscular junction? Does this underlie the symptoms of muscular weakness and fatigue? It is unknown if the findings of Koshima and associates[233] are as applicable to lightning injury as to high-voltage injury.

### Predictability of Lightning and Forecasting

Researchers are able to describe the characteristics of lightning, how it forms, and how it acts. Wide variability in atmospheric conditions gives rise to equally intense lightning events, and it must always be remembered that lightning is stochastic when its parameters are quantified. Modeling of lightning behavior must have this probabilistic nature in mind, particularly where protection is involved.[139,154,324,325]

### Lightning Danger Warnings

In the United States, NWS is charged with providing severe-weather warnings to minimize property and personal injury. One of the difficulties in forecasting is the compromise between absolute safety that results from issuing many warnings that may later turn out to be "false" and lesser guarantees of safety with fewer but more reliable warnings. People learn not to pay attention when too many warnings prove false. An additional complication is that because of the small areas where thunderstorm cells arise, it is impossible for regional forecasts to cover all of them. Individuals should be aware of the general weather forecasts and incorporate their own knowledge of local weather patterns, along with an eye and ear to the sky, when fast-changing weather is predicted, to notice rapid development of nearby storms that may contain dangerous lightning. Preplanning, alertness, and knowing the lightning safety rules, such as "When thunder roars, go indoors," and "No place outside is safe when thunderstorms are in the area," can save lives.

## LIGHTNING EXPOSURE AND SAFETY BEHAVIOR

Unfortunately, some risks have no halfway measures that will significantly improve safety. Lightning is one of those. Currently, there is an outcry for advice on improving lightning safety for wilderness situations. However, *no* place outdoors is safe when thunderstorms are in the area. Responsible outdoor behavior involves always having an escape route to safety in mind. Avoidance of the risk is the only prudent recourse. One must look at the risk/benefit ratio in considering any other response.

During the vast majority of their life, most people are within a very short distance of safety from lightning. Many routine outdoor adventures are on frequently hiked paths, common ascents of mountains, and gatherings at tent-only campgrounds and scenic overlooks. Planning to avoid being in these areas during times of high risk (summer afternoons) can minimize but cannot entirely exclude the chance of injury.

In addition to avoidance of risk, the other part of the "when thunder roars, go indoors" rule is seeking safe shelter. Modified metal shipping containers are used in many countries in industrial, heavy construction and mining sites and are moved as needed by truck or helicopter (Figure 5-53). Discarded containers

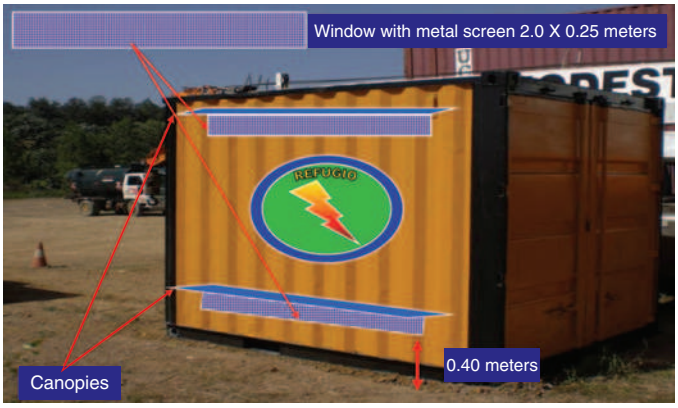


**FIGURE 5-53** Metal shipping container modified for use as a safe shelter in mining areas of New Guinea, Tanzania, and Peru. *(Courtesy Richard Kithil, National Lightning Safety Institute.)*

are sometimes adapted to become open stalls in public markets. These could be used in many wilderness situations to provide lightning-safe shelters within easy hiking distance of common paths, particularly in areas such as summer camps and parks. Development of inexpensive shelters that could be backpacked in by a scout group and set up as a summer project is potentially possible, but carries design, testing, maintenance, and liability issues, particularly in the United States. Other considerations would be location, accessibility, expense, signage, ownership, vandalism, and permits, not to mention resistance from some purists about altering the environment for what may not be perceived as a likely or major risk.

## LIGHTNING SAFETY RESEARCH

There are a variety of unresolved issues relating to improving lightning safety recommendations. The topic includes a mixture of scientific, social, economic, and demographic variables that are often difficult to identify. Although the phenomenon of lightning has become better understood in the last two decades, many features of how lightning results in fatalities and injuries remain elusive. In addition, more examination is needed to determine if there are differences in the injury pattern between developing and developed countries.

The mechanisms of lightning injury have been categorized more completely than ever before, and identifying this mixture of mechanisms has made a difference in lightning safety advice. Most importantly, the common perception of a direct strike is now thought to represent only 5% or less of lightning injury. However, the estimates of Cooper[95] and others are based on subjective experience of researchers studying large numbers of cases, showing the need to better identify the ratio of mechanisms causing lightning injury. Distribution of injuries between the five types may be different in developing countries but has not been investigated. It appears that fires originating in thatched dwellings and other structures are quite common in developing countries, so that confusion and paralysis of people inside may hinder them from escaping in time; this is not an issue inside substantial structures of the more developed world. The issue of the upward leader with regard to human injury needs more study. Postevent recognition of streamer shock (vs. EPR) needs particular attention, although it is difficult to see how these will be differentiated.

Roeder[339] provides a set of questions and comments on research needed to improve lightning safety. The requirements can be divided into several main groups: safety of buildings and vehicles, determining the range of safe distance from lightning, and demographics.

### Building and Vehicle Safety

Lightning safety recommendations now state that safety is reached inside large, well-constructed buildings with plumbing

and electrical wiring in the walls, or within fully enclosed, metal-topped vehicles.* Although fatalities in these locations are rare in developed countries, multiple injuries per incident continue to occur in developing countries, where there may be no substantial buildings or metal vehicles for miles in any direction. Small structures are always problematic, so recommendations stress staying away from them, although it is possible to make them safe in certain situations.[229] A related question is the degree of interruption of the lightning strike by metal meshwork as opposed to the solid conductive sheeting that makes a Faraday cage.

The amount of time people spend outdoors is a key factor. People may now spend more of their time inside safe buildings and vehicles than in the past. Although social data are extremely difficult to quantify, knowing the answers may explain a portion of the steady decrease in lightning fatality rates in developed countries studied in recent years.[189,198]

In less developed regions, safe buildings and vehicles are not available to many people much of the time. Lack of such a safe place nearby is a likely contributor to the higher lightning fatally rate in such regions compared with more developed countries.[189]

### Safe Distance to Lightning

Real-time cloud-to-ground lightning data have been available across the United States since 1989, and monthly maps have been compiled.[112,194] Similar regional or national lightning detection network data are also available to a varying extent in many other regions of the world, and a global lightning data set is becoming accessible everywhere.[122] These data sets have been considered in some situations for determining the distance between flashes and the time of day and year when flashes occur.[202]

How long a lead time to seek safety is needed for the general public, at airports, sports events, and other locations? The lead time includes identifying the safe places that are nearby and the time to reach them. What amount of risk is acceptable for each application? For example, a munitions depot has no tolerance for any direct lightning strike, whereas other situations accept more lightning exposure in exchange for economic efficiency.

The role of cloud lightning in the warning process is also not fully understood. Does an overhead flash without a cloud-to-ground flash demand the same response? What are the safe distances to anvil lightning? With development of total lightning detection, it is becoming possible to address these questions in a more systematic way.[123]

Thunder distancing is often recommended for safety when lightning detection network data are not available. Few if any studies of thunder have been made in the last few decades, so audibility of thunder is not well understood from the practical viewpoint of lightning safety. The issues involved are the normal distance that thunder can be heard, effect of nearby noise on that distance, role of cloud lightning in producing thunder to its use in warnings, transmission velocity of thunder, and relationship of hearing thunder to the actual lightning threat.[339]

### Demographics

Lightning fatality and injury rates are quite well known in developed countries. The global impact of lightning, however, is not well investigated. Lightning casualty data are beginning to be collected systematically in some very populous countries, but data collection methods are in early stages and will need to be established over longer periods.[333]

Global lightning impacts were first estimated by Holle and López[197] to be 24,000 deaths and 240,000 injuries annually around the world. This estimate is based on minimal or no data from Africa, Southeast Asia, and India, where lightning casualty rates appear to be quite high and flash densities are coincidentally often large. Another estimate is 6000 deaths and 60,000 injuries per year globally.[59] Local organizations examining lightning are being established.

*References 43, 105, 117, 119, 120, 187, 201, 264, 292, 384.

Availability of safe versus unsafe buildings and vehicles is important in understanding data that may be collected in less developed areas of the world. It is important that related casualty data be collected concerning activity, location, gender, age, nearby structures, and other issues related to fatality and injury data, in order to separate the influences that have been outlined here and in Roeder's work.[339] Such information can be used to develop relevant safety recommendations and strategies.

## LIGHTNING DETECTION AND DATA APPLICATIONS

### DETECTION

Cloud-to-ground lightning over the United States has been detected and located in real time since the late 1970s. Pulses from lightning in the very low frequency (VLF) and low frequency (LF) range that propagate along the earth are used to detect and locate return strokes in cloud-to-ground flashes. Such sensors can also locate distant lightning in the VLF range because signals propagate thousands of kilometers between the Earth and ionosphere.[113]

The U.S. National Lightning Detection Network has gone through several stages of improvements to combine direction finding and time-of-arrival location methods. Networks with some or most of the NLDN capabilities operate in real time in more than 40 countries; most are owned and operated by national meteorologic services or electric utilities. The NLDN has been operating continuously since 1989 and has become the benchmark for cloud-to-ground lightning detection; typical distances between its sensors are 300 to 350 km (180 to 210 miles). NLDN has been calibrated by rocket-triggered lightning, tower strikes, and camera studies to determine location accuracy over the contiguous United States of 300 to 500 m and flash detection efficiency exceeding 90%.[45,113,145] The NLDN also provides polarity and peak current and measures the quality of each flash location. Approximately 70% of the multiple-return strokes within a cloud-to-ground flash are also located by the NLDN, and half of cloud flashes contain pulses that are strong enough to be detected and located.[113]

Direction finding and time-of-arrival methods have been combined into an advanced method that allows global coverage to detect lightning over extremely long distances.[121] The Global Lightning Dataset GLD360 achieves 70% cloud-to-ground flash detection efficiency with a horizontal location accuracy of 2 to 5 km (1.2 to 3.1 miles). Several lightning detection satellites make twice-daily or regional passes; the new GOES-R lightning sensor scheduled for launch in 2016 will cover much of the western hemisphere at an expected 8-km (5-mile) resolution.

In addition to cloud-to-ground lightning detection networks, detailed cloud lightning can be mapped by regional very high frequency (VHF) networks that use direction finding and/or time-of-arrival techniques over line-of-sight distances.[122,185,235-237,257] These networks track the horizontal branching associated with cloud-to-ground flashes, and some provide vertical resolution. Such cloud lightning networks have closer spacing between sensors than does the NLDN, because they use line-of-sight detection in the VHF range. Some of the cloud flashes seen by these networks are immense and have major implications for lightning safety. One cloud flash in Texas was measured to have a continuous horizontal channel measuring more than 300 km (180 miles) in length and lasting 5.7 seconds.[244]

Continued deployment of larger global and smaller regional networks has occurred beyond the medium-area coverage of wide-area VLF/LF networks. These technologies indicate the need for different techniques or frequency ranges to meet differing demands for lightning information.[113]

### APPLICATIONS

The first operational U.S. lightning detection network was developed for early recognition of lightning-caused fires. Eventually, large networks were established to detect cloud-to-ground

lightning across the western United States, Canada, and Alaska. Cloud-to-ground lightning is the largest cause of transients, faults, and outages in electric power transmission and distribution systems in lightning-prone areas. As a result, this was the other early application of such wide-area networks.[113] In these situations, each flash or stroke is carefully identified as the cause, for example, of a power-line fault on a transmission or distribution line. The initial motivation leading to development of real-time lightning detection was concern about lightning during ground activities and launches at Kennedy Space Center in the 1970s.[113]

A very broad array of applications of lightning detection data has developed over the last three decades. There are more than 3000 published papers on the applications and operations of real-time lightning detection networks in the United States and elsewhere. The applications can be separated into four categories: those of the direct threat from lightning, and lightning as an indicator, substitute, or covariate.

### Direct Threat from Lightning

The direct threats to forest and utility concerns are described in the previous section. In addition, archived and real-time lightning data are used in many forensic and insurance applications. Other direct threats include munitions, ground safety in aviation and defense operations, recreational and workplace safety, spacecraft launches, and shipping and navigation. However, linkage of lightning network data with human casualties is only occasionally made; such studies show a tendency in many recreational situations to involve weak storms with short lifetimes.[183,184,202,214,250]

### Indicator Lightning

Climatologies of lightning have been developed over a variety of regions and time periods.[194] Such annual, monthly, or hourly maps can also be divided according to the type of meteorologic regime at the time to provide information about where thunderstorms and associated impacts are most likely. Lightning climatologies have been found to be easier to compile than many other data sets, such as radar reflectivity and satellite data, since VLF and LF lightning detection is unaffected by terrain, and the data sets are relatively compact for long periods and large areas.

In remote regions where conventional radar and surface observations are not available, tracking thunderstorms and assessing tropical and nontropical cyclone status are important challenges in weather prediction for civilian and defense purposes. Thunderstorms over the ocean represent a threat to aircraft routing and ocean shipping and are usually beyond the range of ground-based meteorologic radars. Current research with long-range lightning data has indicated presence of sporadic cloud-to-ground lightning in the inner cores of tropical cyclones that may be useful for assessing their intensity changes.

Meteorologists use lightning data in many other ways to indicate the state of the atmosphere. These include identifying the difference between showers and thunderstorms; deciding when convection has become strong enough to produce the first flash in a developing thunderstorm; identifying storm orientation, size, and shape; location of frontal boundaries; and when excessively high lightning rates are occurring in a thunderstorm that may indicate significant weather of many types. Isolated versus organized thunderstorms can sometimes be assessed better with lightning data than with radar and satellite information. Lightning data can also be used to indicate location and timing of a developing low-pressure region over the oceans, as well as presence of very cold air aloft. In addition, some winter storms have distinct bands of ice and snow that include lightning where the heaviest precipitation is falling.

### Substitute Lightning

In many situations, such as over oceans and less developed countries, desired meteorologic data are not available to identify situations that might lead to significant or severe weather. For example, over the oceans beyond radar range of about 300 km (186 miles), aircraft can use lightning as a radar substitute to identify turbulence. Similarly, meteorologic radar information is lacking in mountainous areas of the United States below mountaintops because the radar beam is blocked, so lightning can be used to identify thunderstorms in rugged terrain. Rainfall can be estimated with lightning information in regions, times, or areas where radar, rain gauge, or other information is missing during flash floods, tropical cyclones, and other severe weather. National meteorologic agencies also use lightning data along borders of neighboring countries without accessible radar data. Lightning data have proved valuable in geophysical research concerning the natural versus man-made production of nitrogen oxides.

### Covariate Lightning

When radar, satellite, and surface data are available, real-time lightning detection data can be used to identify trends that occur simultaneously with, or earlier or later than, other data, as well as spatially displaced from other information. Examples include lightning jumps combined with radar in severe weather situations and hurricane eyewall outbreaks as the storm changes intensity. More complex methods are also being actively pursued with respect to assimilation of lightning network information into numeric weather prediction models to help initialize them by better locating convection.

There is promise that lightning can be combined with radar or satellite data to more accurately locate heavy and excessive rainfall, because VLF and LF lightning network data do not degrade from terrain effects. Changes of cloud-to-ground lightning polarity and rate changes, as well as ratios of cloud to cloud-to-ground lightning, are topics that require large, accurate, and complex data sets and statistical analyses over multiple regions. They hold promise for many applications.

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