Merck &21: Disorders Due to Physical Agents

Chapter 253: Reactions to Sunlight

The skin responds to excessive sunlight with an acute reaction (sunburn); a chronic reaction that may lead to skin cancer after many years; or an unusual photosensitivity that may be due to the ingestion or application of certain drugs or chemicals, which may be indicative of systemic disease, or may be idiopathic.

Etiology and Predisposing Factors

Solar radiation that reaches the earth's surface ranges in wavelength from 290 to 1850 nanometers **(nanom)** or 2900 to 18500 angstroms **(Å).** The character and amount of such radiation varies greatly with the seasons and with changing atmospheric conditions. Sunburn-producing rays - those below 320 nanom (3200 Å) - are filtered out completely by ordinary window glass and to a great extent by smoke and smog; most are filtered out during winter months in northern temperate zones, especially in urban areas. Large amounts of sunburn-producing rays may pass through light clouds, fog, or 1 ft of clear water and many persons unwittingly sustain severe reactions under such conditions or while swimming. Snow, sand, and bright sky enhance exposure by reflecting the rays.

Following exposure to sunlight, the epidermis thickens and the melanocytes produce melanin at an increased rate, providing some natural protection against further exposure. Persons differ greatly in their reactivity to sunlight. Uneven melanin deposition occurs in many fairheaded individuals and results in freckling. Pigmentation does not occur in the skin of albinos because of a defect in melanin metabolism, nor in areas of vitiligo because of the absence of melanocytes. Blondes and redheads are especially susceptible and should avoid overexposure. Blacks and other nonwhites are not immune to the effects of the sun and can become sunburned with prolonged exposure. In recent years "tanning parlors" that utilize artificial light sources to induce tanning without sunburn have become popular. As all such light sources contain some UVB (see below), some long-term deleterious effects should be expected.

Acute Sunburn

Ordinary sunburn results from overexposure of the skin to ultraviolet rays of 280 to 320 nanom, UVB (2800 to 3200 Å). **Symptoms and signs** appear in 1 to 24 h and, except in severe reactions, pass their peak in 72 h. Skin changes range from mild erythema with subsequent evanescent scaling, to pain, swelling, skin tenderness, and blisters from more prolonged exposure. Sunburn affecting the lower legs, particularly the pretibial surfaces, is especially uncomfortable and often slow to heal. Constitutional symptoms (fever, chills, weakness, shock), similar to a thermal burn, may appear if a large portion of the body surface is affected; these may be due to heatstroke or heat exhaustion (see Ch. 255).

Secondary infection and miliaria-like eruptions (seen in Ch. 242) are the most common late complications. Following exfoliation, the skin may be hypervulnerable to sunlight for one to several weeks.

Prophylaxis

Simple precautions will prevent most cases of severe sunburn. Initial summer exposure to bright midday sun should not be > 30 min, even in persons with dark brunette skin. In temperate zones, exposure is less hazardous before 10:00 am and after 4:00 pm because sunburn-producing wavelengths are usually filtered out. In winter, the greatest danger of sunburn (and snow blindness) comes during foggy days that may be deceptive and have almost as much UVB as clear days on fresh snow; the danger is increased at high altitude.

Formulations of 5% aminobenzoic acid **(PABA)** as its esters in ethyl alcohol in a gel or in a cream are very effective in preventing sunburn. They take about 30 min to bind strongly to the skin, and therefore should be applied about 30 to 60 min before sun exposure so that wash off from perspiration or swimming will be minimized. PABA products rarely cause allergic or photoallergic contact dermatitis. Those who cannot tolerate PABA or its esters may use a benzophenone sunscreen. Opaque formulations containing zinc oxide or titanium oxide physically block and prevent radiation from reaching the skin. When suitably colored with agents such as iron salts, they are cosmetically acceptable. Newer, highly effective, nonopaque lotions containing both a PABA ester and benzophenone are now available. **Sunscreens** are now rated in the USA by the FDA's SPF (sun protection factor) numbers: 15 is the most protective and 1 the least. In some other countries 10 is maximum and equal to US 15. Patients with photodrug reactions are rarely protected with these products.

Treatment

Further exposure should be *avoided* until the acute reaction has subsided. Topical corticosteroids are no more effective than cold tap-water compresses in relieving symptoms. Ointments or lotions containing local anesthetics such as benzocaine and other sensitizing agents should be *avoided.*

Early treatment of extensive and severe sunburn with a systemic corticosteroid (i.e. prednisone 10 mg orally qid for 4 to 6 days for adults or teenagers) will decrease the discomfort considerably. (For treatment of heatstroke and heat exhaustion, see Ch. 255.)

Chronic Effects of Sunlight

Chronic exposure to sunlight ages the skin. Wrinkling and elastosis (yellow discoloration with small yellow nodules) and pigment alterations are the most common troubling consequences of long-term exposure, especially for women. The atrophic effects in some persons may resemble those seen after x-ray therapy. Precancerous keratotic lesions **(actinic keratoses)** are a frequent, disturbing consequences of many years' overexposure. Blondes and redheads are particularly susceptible, blacks are rarely affected. The keratoses are usually hard and sharp on palpation, and gray to dark in color. They should be differentiated from warty brown *seborrheic* keratoses, which increase in number and size with age but occur on covered as well as uncovered areas of the body and are not premalignant.

The incidence of squamous and basal cell carcinoma of the skin in fair, white-skinned persons is directly related to the amount of yearly sunlight in the area. Such lesions are

especially common in those who were exposed as children and teenagers and in sportsmen, farmers, ranchers, sailors, and sunworshipers. Malignant melanomas may also increase in incidence with increasing sun exposure.

Treatment

If there are only a few lesions, cryotherapy, i.e., freezing with liquid nitrogen, is the most rapid and satisfactory treatment for actinic keratosis. If there are too many lesions to freeze, actinic keratosis usually responds dramatically to small amounts of 5-fluorouracil **(5-FU),** applied to the affected area nightly. For face lesions, 1% 5-FU in propylene glycol is best; elsewhere (i.e., on the arm), 2 or 5% 5-FU cream can be used; if no response is seen within 10 days, 0.1% tretinoin solution should be applied a few hours before the 2 or 5% 5-FU application. Treatment is continued for at least 2 wk or until a brisk reaction with redness, scaling, and slight burning is seen, often including patches with no previously detected gross changes. If the reaction is too brisk, application should be suspended for 2 or 3 days. Masking cosmetics can make the treatment more acceptable. Topical 5-FU therapy is free of significant adverse effects but is so disfiguring and painful during treatment that freezing of individual lesions is returning to popularity. 5-FU treatment is no more lasting than individual destruction and has concealed serious underlying basal cell cancers. It should not be used to treat basal cell cancers.

Photosensitivity Reactions

In addition to the acute and chronic effects of sunlight, a variety of unusual reactions may occur after only a few minutes' exposure: i.e., areas of erythema or frank dermatitis; urticarial and erythema multiforme-like lesions; bullae; and chronic, thickened, scaling patches.

Numerous factors (many unknown) may contribute to increased photosensitivity: (1) SLE or cutaneous LE - unless the cause is obvious, every patient with pronounced photosensitivity should be studied for these conditions; (2) ingestion of a variety of drugs (i.e., sulfonamides, tetracyclines, thiazides, griseofulvin), though sensitivity appears in only a small percentage of patients taking such compounds; (3) external application of or contact with various substances (see also Ch. 230), including toilet waters and bergamot containing perfumes, sulfonamides, coal tar, soaps containing halogenated salicylanilides, and certain plants (i.e., meadow grass, parsley); (4) xeroderma pigmentosum and certain porphyrias are less common but serious diseases also associated with photosensitivity.

Polymorphous light eruptions are unusual reactions to light that are not associated with systemic disease or drugs, as far as can be determined. Eruptions may be papular or plaque-like, dermatitis, urticarial, or erythema multiforme-like, and appear on sun-exposed areas. They are more common in people from northern climates when first exposed to spring or summer sun than in those who get sun year around. Direct immunofluorescence of a biopsied lesion and of normal-appearing skin is negative, as opposed to LE, where the result is usually positive. Diagnosis is by exclusion or by reproduction of the lesions with artificial or natural sunlight when the patient is not using any medication (systemic *or* topical).

Prophylaxis and Treatment

Avoidance of sunlight is important, and the patient should wear protective clothing (i.e., hats and long-sleeved shirts) when outdoors on sunny days. Sunscreening preparations (see Acute Sunburn, above) are sometimes helpful. Other treatment is directed to the underlying cause, where possible. Polymorphous eruptions manifested as papules, plaques, or dermatitis may respond to topical corticosteroids. In patients with polymorphous photosensitivity or cutaneous LE, prolonged (2 to 4 mo) administration of hydroxychloroquine 200 to 400 mg/day orally often reduces or completely suppresses photosensitivity and may be tried if treatment is required and sunscreens are not effective. Potential eye toxicity should be watched for by an ophthalmologist particularly by examining visual fields. PUVA (psoralens plus UVA) is also effective in preventing some cases of polymorphous light eruptions if used before sun exposure but should not be used in LE.

Chapter 254: Burns

Tissue injury caused by thermal, chemical, or electrical contact results in protein denaturation, burn wound edema, and loss of intravascular fluid volume due to increased vascular permeability. Systemic effects, such as hypovolemic shock, infection, or respiratory tract injury pose a greater threat to life than do local effects.

In spontaneous burn wound healing, dead tissue sloughs off as new epithelium begins to cover the injured area. In **superficial burns,** regeneration occurs rapidly from uninjured epidermal elements, hair follicles, and sweat glands; little scarring results unless infection occurs. With **deep burns** (destruction of the epidermis and much of the dermis), reepithelialization starts from the edges of the wound or from the scattered remains of integument. The process is slow, and excessive granulation tissue form before being covered by epithelium. Such wounds generally contract and develop into disfiguring or disabling scars unless treated promptly by skin grafting.

Symptoms, Signs, and Assessment of Burn Injury

The **severity** of the burn is judged by the quantity of tissue involved. This quantity is represented by the percentage of the **body surface area (% BSA)** burned and by the **depth** of the burn. A reasonable classification of burns by severity is: small, or < 15% BSA; moderate, or 15 to 49% BSA; large, or 50 to 69% BSA; and massive, 70% or greater BSA.

The depth of the burn may be described as first, second, and third degrees. **First degree** burns are red, very sensitive to the touch, and usually moist. There are no blisters and the surface markedly and widely blanches to light pressure. **Second degree** burns may or may not have blisters. The bases of the blisters may be erythematous or whitish with a fibrinous exudate. The wound base is sensitive to touch and may blanch to pressure. **Third degree** burns may but generally do not present with blisters. The surface may be white and pliable when pressure is applied or it may be black, charred, and leathery. Third degree burns may be pale in color and mistaken for normal skin, but the subdermal vessels do not blanch to pressure. The wound may be bright red due to fixed hemoglobin in the subdermal region. The third degree burn is generally anesthetic or hypoesthetic. Hair may be pulled from their follicles easily. Often, the distinction between deep second and third degree burns can be

made only after 3 to 5 days of observation.

Respiratory tract ventilation injury accompanying thermal burns is due to inhalation of the incomplete products of combustion, which are potent chemical irritants to the respiratory mucosa. Only steam inhalation causes actual thermal damage to the respiratory tract. Inhalation of hot gases cause immediate upper airway obstruction; airway edema can produce a slower developing upper airway obstruction; and small airway alveolar capillary injury can cause delayed progressive respiratory failure. Symptoms and signs of respiratory tract injuries are described under Initial Treatment, below.

In **electrical burns,** injury results from the generation of heat up to 5000 °C. Since most of the resistance to electric current is at the point of skin contact with the conductor, electrical burns usually involve the skin and subjacent tissues and may be of almost any size and depth. Progressive necrosis and sloughing are usually greater than the original lesion would indicate. Electrical injury, particularly from alternating current, may cause immediate respiratory paralysis, ventricular fibrillation, or both (see Cardiac Arrest and Cardiopulmonary Resuscitation in Ch. 27).

Chemical burns may be due to strong acids and alkalies, phenols, cresols, mustard gas, or phosphorus. All produce necrosis that may extend slowly for several hours.

About 85% of burns are small and can be treated in outpatient facilities. Criteria establishing when to treat a burn victim as an outpatient are given below, following a description of the evaluation and treatment of the more extensively burned patient (> 15% BSA burns in adults, > 10% BSA in small children).

Initial Treatment of the Burn Victim

Immediate care requires establishment of an adequate airway for ventilation and oxygenation, stopping the burning process, replacement of acute plasma volume loss, recognition and management of any associated life-threatening major trauma, diagnosis of metabolic abnormalities, and protection from bacterial contamination.

Ventilation injuries, if severe, can be treated with tracheal intubation (the nasotracheal route is preferred) and mechanical ventilation. **Absolute indications for intubation** include rapid and shallow ventilation with tachypnea of 30 to 40 breaths/min; inadequate ventilation indicated by a respiratory rate of < 8 to 10 breaths/min; mechanical airway obstruction from trauma, edema, or laryngospasm; or signs of respiratory failure with arterial blood determinations of pH < 7.2, PO_2 < 60 mm Hg, or PCO_2 > 50 mm Hg. **Relative indications for intubation** may include history of an enclosed space explosion or fire; singed nasal hairs or oral mucosa, erythema of the palate, or soot in the mouth, larynx, or in the sputum; edema associated with a burn of the face or neck; and signs of respiratory distress such as nasal flaring, respiratory crowing or stridor, anxiety, agitation, or combativeness. If ventilation mechanics seem adequate, then O_2 may be administered by face mask or nasal cannula.

Stopping the burning process involves removing all clothing, especially any smoldering material such as melted synthetic shirts or hot tar-laden material. All chemical agents should first be flushed off the skin with copious amounts of water. **Acid and alkali**

burns and burns caused by organic compounds such as **phenols or cresols** should be diluted with copious amounts of water. **Phosphorus burns** should be immersed immediately in water to avoid contact with air. Phosphorus particles are removed gently under water and the wound is washed with 1% copper sulfate solution to coat any residual particles with a protective film of copper phosphide; these fluoresce and can be readily removed in a darkened room. Care must be taken to avoid excess absorption of copper. Following initial treatment, chemical burns should be treated as thermal burns of comparable size and extent.

Immediate volume replacement: Shock should be *anticipated* in third degree and extensive second degree burns involving > 25% BSA of adults or > 30% BSA of children. When hypovolemic shock is present, **volume replacement** should begin immediately with the establishment of a 14- to 16-gauge venous cannula in 1 or 2 veins. Although central lines may not be necessary initially, their later placement may be difficult. Therefore, if the need for central access is anticipated for fluid or K replacement or for hyperalimentation, a subclavian or internal jugular line should be placed early. If necessary, central or peripheral lines may be placed through burn eschar. A "cutdown" is avoided, since it more likely destroys the vein and precludes its future use, but more importantly, it carries a high risk of infection. Blood should be obtained for determination of Hb, Hct, blood type, and cross-match.

The immediate resuscitation fluid is Ringer's lactate. A rapid estimate of the extent of burn injury to determine the initial flow rate can be obtained by the **rule of fourths,** in which the number of fourths of the body that are burned is determined. The area of the body burned is estimated as 1, 2, 3, or 4 fourths and multiplied by the flow rate of 1 L/h. For example, if the face and anterior chest are burned, then 1/4 of the body is burned. The fluid delivery rate is 1 x 1 L/h, or 1 L/h. This infusion rate can be used for the first 1 to 2 h, when a more detailed physical examination and accurate calculation of the fluid requirements can be done (see below). Proper fluid replacement is determined by careful monitoring of the patients and should be modified to optimize BP, pulse, and urinary output.

Pain from minor burns can usually be relieved by codeine 30 to 60 mg orally or s.c. and aspirin 650 mg orally q 4 to 6 h. In severe burns with peripheral vasoconstriction, morphine 0.1 mg/kg or meperidine 1.0 mg/kg should be given IV q 3 h.

A **tetanus toxoid booster,** 0.5 to 1 mL s.c. or IM, may be given to patients immunized within 4 to 5 yr; otherwise, tetanus immune globulin (human) 250 u. IM should be given (and repeated every 6 wk as necessary), and concomitant active immunization should be started.

Bacterial invasion occurs whenever the epidermis is broken. Dead tissue, warmth, and moisture provide ideal conditions for bacterial growth. Streptococci and staphylococci usually predominate shortly after a burn, and gram-negative bacteria after 5 to 7 days, but mixed flora are always found.

Penicillin G 5 million u. IM is given daily for 3 days as prophylaxis against streptococcal cellulitis.

Long Range Burn Treatment

Problems and therapeutic solutions should be identified after initial resuscitation. This requires a detailed history and physical examination with a careful evaluation of the injury, calculation of fluids required, monitoring vital signs and urinary flow to adjust the resuscitative fluids given, consideration of escharotomies, detection and treatment of metabolic abnormalities, and topical wound care.

History and physical examination: Accurate data about the burn episode usually comes from sources such as the ambulance driver, accompanying family member, coworker, police, or firefighter. The following details are important: (1) Where was the patient when the burn occurred? Was he in a closed space? (2) What exactly was the patient doing when the burn occurred? Was there an explosion? (3) What was the burn source (i.e., thermal, electrical, chemical)? (4) How long was the patient exposed? (5) What exactly was done to eliminate the burn mechanism? (6) Did anything happen to the patient that would suggest the presence of any associated injuries?

Further medical history seeks information about allergies, medications, the presence of heart, pulmonary, or renal disease, diabetes, or any other medical or psychiatric disorders. Smoking and drinking habits should also be noted.

A **complete physical examination** is performed to detect any pulmonary, cardiac, hepatic, neurologic, or renal disease and to determine the extent and severity of injury. This must be done before maturation of burn injuries, when physical findings may be more difficult to discern. A best estimate for height and weight is recorded to allow calculation of the patient's BSA. Often the height can be measured immediately and preburn weight estimated by a family member. Details of the body surface burned and the estimated depth are recorded on a burn diagram. The area involved is outlined, and the depth of the burn indicated by the type or style of the markings. **In adults,** the extent of the burn (% BSA) is estimated by comparing the burn diagram with the **rule of nines:** head and neck, 9% of BSA; each hand and arm (including deltoid), 9%; each foot and leg as far up as the inferior gluteal fold, 18%; anterior and posterior trunk including buttocks, 18% each; perineum, 1%. **In children,** a more accurate estimate for the % BSA involved may be obtained using the Lund-Browder chart (see Fig. 254-1).

Calculation of volume replacement: The object is to maintain normal physiology as reflected by urine, vital signs, and mental status. The fluid needed is related to the extent of the burn. Most formulas suggest 2 to 4 mL/kg/% BSA for the first 24 h. The colloid requirement (plasma, albumin, etc) is a matter of judgment. Whether to use colloid and when to start depends on the size of the burn, the patient's age, and concomitant diseases. Patients with small burns seldom require colloid. Patients with large or massive injuries, young children, elderly patients, and those with cardiac disease will require colloid in the first few hours. The exact volume and rate of crystalloid and colloid administration depends on each patient's response to the fluid delivery. Patients who have inadequate urine output despite administration of a high volume of crystalloid often respond to colloid. The object is to maintain normal physiology as reflected by urine volume, vital signs, mental status, and ventilatory function. Precalculated figures are seldom correct for the total period of resuscitation; formulas are used *only as a guide.*

A general formula that may be used as a guide for the first 24 h is:

0.5 mL/kg/% BSA of colloid
1.5 mL/kg/% BSA of Ringer's lactate
100 mL/h of maintenance Ringer's lactate

One half is given in the first 8 h, 1/4 in the second 8 h, and 1/4 in the third 8 h. For the first 4 h, only Ringer's lactate is given. The colloid is begun during the second 4 h.

A sample calculation for a 70-kg man with a 40% BSA burn is:

0.5 x 70 x 40 = 1400 mL colloid (fresh frozen plasma)
1.5 x 70 x 40 = 4200 mL Ringer's lactate
2400 mL maintenance Ringer's lactate

Total fluid = 8000 mL fluid over the first 24 h

This should be given by the following schedule:

First 4 h:

1050 mL Ringer's lactate
100 mL/h maintenance Ringer's lactate

Second 4 h:

700 mL colloid
1050 mL Ringer's lactate
100 mL/h maintenance Ringer's lactate

Second 8 h:

350 mL colloid
1050 mL Ringer's lactate
100 mL/h maintenance Ringer's lactate

Third 8 h:

350 mL colloid
1050 mL Ringer's lactate
100 mL/h maintenance Ringer's lactate

Rounding off the calculation, the sample orders would read:

Please administer the following IV solutions:

For the first 4 h:

Ringer's lactate at 360 mL/h

For the second 4 h:

Fresh frozen plasma at 180 mL/h
Ringer's lactate at 360 mL/h

For the second 8 h:

Fresh frozen plasma at 45 mL/h
Ringer's lactate at 220 mL/h

For the third 8 h:

Fresh frozen plasma at 45 mL/h
Ringer's lactate at 220 mL/h

Monitoring: Many parameters must be followed closely to prevent or recognize incipient problems as early as possible. Therefore, it is good procedure to establish a flow chart listing the items discussed here. The goal of fluid replacement is to maintain an adequate BP and a urine output of > 50 mL/h in an adult or 1 mL/kg/h in a child while avoiding overloading the circulation. An indwelling Foley catheter should be placed to monitor the urine output. Insufficient therapy can be recognized by a decline in urine volume, an increase in hemoconcentration, and symptoms of shock. Pulmonary edema and congestive heart failure should be prevented by monitoring the pulse, respiration, BP, and neck vein distention or the central venous pressure. The lung bases should be auscultated frequently for rales.

Patients with preexisting cardiovascular-renal disease present a special problem. Fluid, electrolyte, and colloid administration should be limited to amounts sufficient to produce minimal adequate urinary output (25 mL/h) and the patient watched for signs of circulatory overload.

Pulse, BP, temperature, ECG, and arterial blood gases should be monitored in severe burns, in the elderly, or in patients with preexisting disease. Most cardiac arrhythmias are caused by hypovolemia, hypoxia, acidosis, or hyperkalemia. These metabolic disorders should be evaluated and corrected before cardiac drugs are used. Ventricular tachycardia and fibrillation are exceptions, which should be treated immediately while underlying metabolic abnormalities are evaluated. If 0.5% silver nitrate is used as a topical antibacterial agent, it is well to remember that such a solution is hypotonic and leeches Na and Cl and some K from the tissues into the wet dressing. Losses may result in severe **hyponatremia, hypochloremia,** and **hypochloremic alkalosis.**

Serum K is aggressively maintained above 4 mEq/L. **Hypokalemia** is common in the early resuscitation period for 3 reasons: (1) Many patients present with depleted K stores secondary to prior diuretic therapy. (2) K is not generally included in the early vigorous fluid replacement. (3) Some K is lost into the hypotonic 0.5% silver nitrate dressings.

Hypoalbuminemia results from the combination of dilutional effects of crystalloid replacement therapy and enhanced loss of protein into the subeschar edema fluid. Colloid solution is continued throughout the resuscitation period at the rates previously described in order to maintain albumin levels at about 3 gm/dL and a total protein > 5 gm/dL. Most Ca in the serum is reversibly bound to albumin. **Hypocalcemia** may therefore be a result of hypoalbuminemia. The ionized fraction of serum Ca is usually normal but should be measured periodically. Replacement quantities of Ca, phosphate, and magnesium are given daily.

Generalized poor tissue perfusion, a result of hypovolemia or cardiac failure, results in **metabolic acidosis.** Blood pH < 7.2 should be treated with sodium bicarbonate IV (see Metabolic Acidosis in Ch. 84). Focal poor tissue perfusion can result from a constricting eschar or fascia that should be treated with escharotomy and fasciotomy (see Operative Management below).

Myoglobinuria can result from ischemic constrictions of muscle and from deep thermal or electrical burns of muscle, and should be treated by alkalinization of the urine with 50 mEq sodium bicarbonate q 4 to 8 h with frequent monitoring of both serum and urine pH. The goal is a urinary pH > 8. In severe myoglobinuria, mannitol 12.5 gm is given IV q 4 to 8 h for osmotic diuresis until the myoglobinuria clears. **Hemoglobinuria** may result from erythrocyte destruction following burns and is treated identically as myoglobinuria. Both myoglobinuria and hemoglobinuria may result in renal tubular necrosis if not promptly and accurately managed.

Hb is determined q 3 to 4 h for the first 72 h, and therapy is regulated so that the Hb does not rise above 16 or fall below 11 gm/100 mL. Hct should be maintained at about 40%.

Body temperature should be closely followed because hypothermia occurs frequently in the extensively burnt patient. Those with rectal temperatures < 97 °F (36 °C) are treated by warming the resuscitation fluids. Rewarming patients from temperatures < 91 °F (33 °C) may be associated with fatal dysrhythmias. These patients should be warmed slowly and monitored continuously (see Ch. 256).

Escharotomies: Indications for escharotomies are liberal in circumferential third-degree burns. The loss of a previously palpable pulse, or a nonpalpable pulse in a single extremity with easily palpable pulses in the remaining extremities, are indications for escharotomies. Peripheral ischemia is suspected when a single extremity is cooler than the others and has a poor capillary refilling time. A tense eschar is released even in the presence of Doppler pulses if peripheral ischemia is suspected. With injuries to the skin that do not involve deeper tissue, the depth of the escharotomy incision extends only through the dermis, excluding the hypodermis or fat. The incision should extend well beyond the tense region of eschar to assure complete release. Some full thickness eschars retain pain sensation, making the releasing incision painful. Anesthesia may be obtained with 1% lidocaine.

Topical wound care: Under aseptic conditions, the burned surface is washed with soap and water, cultured, treated with the appropriate topical agent, and covered with sterile dressings. Some of the common topical agents used are 0.5% silver nitrate solution, mafenide acetate, and 1% silver sulfadiazine. To use silver nitrate, the wounds are covered with as many as 8 layers of cotton roll bandages and the silver nitrate is poured over the dressing at 2-h intervals. This keeps the dressing moist and the concentration of the silver nitrate at the skin at about 0.5%. A lower concentration probably is not bactericidal. A higher concentration, which is a result of evaporation, can further burn the skin. Both the mafenide and silver sulfadiazine are directly applied to the wound as a cream and then may be covered with a few layers of cotton rolls. It is important to completely remove the old topical agents before the new application each day. Following topical applications of silver nitrate, patients may develop excessive Na losses, hypokalemia, hypochloremia, alkalosis, and methemoglobinuria. Mafenide acetate cream applied topically inhibits carbonic anhydrase activity and may produce compensated metabolic acidosis and, occasionally, proximal renal tubular acidosis.

Operative management: Excision or removal of the burn eschar creates a clean wound bed that can be covered with grafting material. Naturally, deep second- or third-degree burns separate and slough wound eschar over a period of time leaving a raw wound bed. Excision is best done during the first 1 to 4 days after the burn **(early excision).** This removes devitalized tissue, avoids subeschar sepsis, and allows early wound closure, which shortens hospitalization and improves the functional result of the burn wound.

In early excision, it is first determined whether a burned region is a deep second- or third-degree burn and thus requires grafting; then which part of the body should be excised first. The largest area of involvement is removed first, in order to remove the largest eschar possible, but no more than 20% BSA is excised at a single sitting. The back, chest, and abdomen are excised first and in that order. These are the most successful areas for graft acceptance. The neck and upper chest should be excised early to provide skin coverage for future central line placement. The upper extremities including the hands are excised before the lower extremities. The face is rarely excised.

Excision results in a burn wound bed that requires closure with graft material. Currently available graft materials are (1) autografts, the patient's own skin; (2) allografts, viable skin usually from cadaver donors; and (3) xenografts, skin from porcine sources. In the future, artificial skin will provide early wound closure, elimination of the inflammatory response in the burn wound bed, and subsequent wound bed contraction. Autografts can be transplanted as either sheet or meshed grafts. A sheet graft is a solid piece of skin. Mesh grafts are used when donor skin is scarce. To obtain mesh grafts, small incisions are made at regular intervals in the sheet of donor skin with a skin meshing instrument that permits the graft to cover a larger area. Meshed grafts heal with an uneven gridlike appearance. Autografts are histocompatible and therefore are not rejected. Sufficient autograft material is not available in burns > 50% BSA. However, autograft may be taken from the same donor site region repeatedly at 14-day intervals, which expands the autograft supply in time. Both allografts and xenografts are temporary and will be rejected at 10 to 14 days. They must be replaced with autograft, but their use is lifesaving in massive burns.

Physical and occupational therapy: Positioning, exercising, splints, and pressure garments help preserve function and appearance as burn wounds heal. Body surfaces with high skin tension and movement such as the face, joints, upper legs, and chest are most susceptible to formation of scars and contractures.

Splints to prevent contractures and keep specific joints in the position of function should be applied as soon as possible after admission. They must be fitted properly and constantly assessed in the early stages of treatment to avoid constriction of the extremity that may result in increasing edema. Later, as edema subsides, the splints often need to be remodeled for a closer fit. In extensive burns, splints are worn continuously until the area is grafted and shows substantial healing. Joints are also maintained in functional position throughout convalescence with dressings and blanket rolls.

With extensive burns, the total body position is considered. The **neck** is extended using soft foam pads fashioned to conform to the skull. The **axilla** is flexed about 30 to 60 degrees with arm troughs and should be abducted. The **elbow** is positioned and splinted at extension or slight flexion. The **wrist** is slightly extended 20 to 30 degrees. The **metacarpophalangeal joints** are flexed at 90 degrees. The **proximal and distal interphalangeal joints** are extended completely to decrease the stretch of the dorsal skin over them and the possibility of ischemic necrosis. The **thumb** is in slight apposition to the palm. The **hips** should be abducted and are prevented from external rotation by lateral extensions of the ankle splints. The **ankle** is splinted at 90 degrees dorsiflexion to stretch the achilles tendon.

Joints are put through active and passive range of motion exercises once or twice a day before grafting to preserve function. Both exercise and positioning become easier as the initial edema subsides. After skin grafting, the grafted part is usually kept immobile for 5 to 10 days awaiting stability of the graft before starting preoperative exercises.

Nutrition: Aggressive metabolic support is indicated in patients with > 20% BSA burns, preinjury malnutrition, complications such as sepsis or associated injury, and a weight loss in excess of 10% of the premorbid weight. This support begins 3 to 4 days after the fluid resuscitation phase of the burn therapy.

Oral feeding is preferred because of fewer complications and lower cost, but anorexia, facial burns, or dysphagia may make it difficult or impossible. If oral feeding is inadequate, but GI motility and absorption are normal, then **tube feedings** are used as a supplemental or total caloric source. **Parenteral nutrition** is indicated in those patients with prolonged gastric and colonic ileus related either to the burn wound, repeated operations, or sepsis. Complications are more likely in parenteral nutrition than with the enteral route. For further details of providing adequate nutrition, the use of elemental or defined formula diets, and parenteral nutrition, see Ch. 79.

Outpatient Burn Treatment

Superficial burns that involve a small body surface area and no inhalation injury may be treated in outpatient facilities. The general criteria are (1) first and superficial second-degree burns of < 15 to 20% BSA (adults) or < 10% BSA (small children), (2) moderate to

deep second-degree burns involving < 10% BSA, and (3) third-degree burns of < 1% BSA. An estimate is made of time required for wound closure. The patient is admitted if a wound is not expected to heal spontaneously in 3 wk or less. Admission is planned either at the first encounter with the patient or at follow-up a few days later. Patients are more likely to be admitted if (1) the face, hand, perineum, or feet are involved with the deeper aspects of the burn, (2) if poor compliance with elevation and dressing changes is anticipated, or (3) the patient is < 2 yr of age or > 70 yr of age.

Outpatient treatment of burns requires a history and physical examination as described above (including estimates and recording of both the depth and the extent of the burn in terms of % BSA); cleansing and debriding the wound; topical therapy; splinting and positioning the wound; administration of medications; explanation of home care instructions; and arrangement for outpatient follow-up visits.

Wound cleaning, removing the burning agent, and debridement: Small burns should immediately be immersed in cold water, if possible. Chemical wounds should be copiously irrigated with water. The burn wound should be cleaned with soap and water.

The blisters of the burn wound should be sharply debrided away if they are already broken or are likely to be broken. If the depth of the burn is in question, blisters should be removed and the wound base examined for full thickness injury. All debris should be carefully removed from the wound. For deeply embedded dirt the wound may be anesthetized with a local infiltration of 1 to 2% lidocaine and scrubbed with a stiff brush and soap.

Topical therapy: Once the wound is clean, a topical agent may be applied. Silver sulfadiazine cream is commonly used. One layer of the cream is applied with sterile technic, using a sterile tongue blade as an applicator and the wound is covered with gauze roll bandages.

Splinting and positioning: If the burn involves a joint and is at least of second-degree depth, splinting is required (see also above). The hand is splinted by wrapping each finger individually with cotton roll gauze over the hand and wrist in a "figure 8" pattern. The palm should receive extra padding to maintain the metacarpophalangeal and interphalangeal joints in slight flexion. The wrist or elbow may be splinted by using an arm sling. The lower extremities are not usually splinted in outpatient care.

The most important therapeutic maneuver is elevation of the extremity, especially in patients with a lower extremity or hand burn. The extremity should be placed above heart level at all times except for brief periods of ≤ 20 min during the day.

Medications: Patients with burns of second-degree or more are often given penicillin V 1 to 2 gm/day orally in 4 divided doses as prophylaxis for streptococcal cellulitis over the first few days. Erythromycin 1 to 2 gm/day orally in 4 divided doses may be used in patients with a penicillin allergy. Active immunization against tetanus is given as described above. Analgesic is prescribed as necessary.

Home care instructions: The patient should be advised to: (1) keep the wound clean and dry; (2) keep the wound elevated; (3) change the dressing twice daily as directed; (4) take

the antibiotics as directed; and (5) the patient should know when and where to return for follow-up. The patient should especially understand to clean the wound completely with water to remove all the residual topical medication before applying a new layer.

Outpatient follow-up visits are required to: (1) monitor compliance with wound care; (2) debride the wound; (3) examine for cellulitis; (4) further assess the burn depth; (5) evaluate and arrange for ambulatory, occupational, and physical therapy; and (6) consider excisional therapy. The first follow-up visit is usually within 24 to 48 h after the burn or immediately if signs of sepsis occur. Subsequent outpatient visits are q 24 to 72 h as necessary, depending upon the severity of the burn depth and the ability of the patient to care for the wound.

Chapter 255: Heat Disorders

Heat Stroke and Heat Exhaustion

Mild to grave reaction to high temperature due to inadequate or inappropriate responses of heat-regulating mechanisms.

Etiology

Prolonged exposure to high ambient temperatures may lead either to excessive fluid loss and hypovolemic shock (heat exhaustion) or to failure of heat loss mechanisms and dangerous hyperpyrexia (heat stroke). Dehydration, excessive sweating, vomiting, diarrhea, age, and debility predispose to either; high humidity, strenuous exertion, poor ventilation, and heavy clothing contribute. Many drugs (i.e., antihistamines, anticholinergics, phenothiazines, numerous psychotropic drugs, and cocaine) increase susceptibility to heat illness, particularly heat stroke. Though stemming from the same cause, heat stroke and heat exhaustion are sharply different (see Table 255-1).

Table 255-1. Differentiation Between Heat Stroke and Heat Exhaustion

	Heat Stroke	Heat Exhaustion
Cause	Inadequate or failure of heat loss	Excessive fluid loss - hypovolemic shock
Warnings	Headache, weakness, loss of consciousness	Gradual weakness, nausea, sudden anxiety, excess sweating, syncope
Appearance and Signs	Hot, red, dry skin; little sweating; hard rapid pulse; very high temperature	Pale, grayish, clammy skin; weak, slow pulse; low BP; faintness
Management	Emergency cooling by wrapping or immersion in cold water or ice; immediate hospitalization	As for simple syncope: head down; replace lost salt and water (usually orally, rarely IV)

Prophylaxis

Common sense is the best preventive; strenuous exertion in a very hot environment, inadequately ventilated space, or heavy, insulating clothing should be avoided; loss of fluid and electrolytes (often imperceptible in very hot, very dry air) should be replaced by continuous oral fluids slightly salty to taste (i.e., near isotonic). Sometimes exertion in a hot environment cannot be avoided; every effort should then be made to replace lost fluid and salt and to keep the skin temperature cool by evaporation. Salt tablets occasionally cause gastric distress and are less desirable than lightly salted beverages and foods.

Heat Stroke

(Sunstroke; Hyperpyrexia; Thermic Fever; Siriasis)

Symptoms and Signs

An abrupt onset is sometimes preceded by prodromal headache, vertigo, and fatigue. Sweating is usually but not always decreased, and the skin is hot, flushed, and usually dry. The pulse rate increases rapidly and may reach 160 to 180; respirations usually increase, but BP is seldom affected. Disorientation may briefly precede unconsciousness or convulsions. The temperature climbs rapidly to 40 or 41 °C (1o4 or 106 °F) and the patient feels as if burning up. Circulatory collapse may precede death; after hours of extreme hyperpyrexia, survivors may have permanent brain damage.

Diagnosis and Prognosis

Hot, dry, flushed skin, high body temperature, and rapid pulse in a person exposed to a hot environment are usually enough to distinguish heat stroke from food, chemical, or drug poisoning. Heat stroke is a *serious emergency* and unless promptly and energetically treated, results in convulsions and death or permanent brain damage. Core temperature of 41 °C (106 °F) is a grave prognostic sign; temperature a degree higher is often fatal. Old age, debility, or alcoholism worsen the prognosis.

Treatment

Heroic measures should be instituted immediately. If distant from a hospital, the patient should be wrapped in wet bedding or clothing, immersed in a lake or stream, or even cooled with snow or ice while waiting for transportation. Warning: *The temperature should be taken every 10 min and not allowed to fall below 38.3 °C (101 °F) to avoiding converting hyperpyrexia to hypothermia.* Once in hospital, more exact control measures are instituted and the core temperature is monitored continuously to avoid hypothermia. Stimulants and sedatives including morphine are avoided; diazepam or a barbiturate may be given IV if convulsions are not otherwise controllable. Electrolyte determinations should guide IV therapy. Bed rest is desirable for a few days after severe heat stroke, and temperature lability may be expected for weeks.

Heat Exhaustion

(Heat Prostration, Collapse, or Syncope)

Symptoms and Signs

Because of excessive fluid loss, this disorder gives adequate warning by increasing fatigue, weakness, anxiety, and drenching sweats, leading to circulatory collapse with slow thready pulse, low or imperceptible BP, cold, pale, clammy skin, and disordered mentation followed by a shock-like unconsciousness. Temperature is usually *below* normal and the picture is that of simple syncope.

Diagnosis and Prognosis

Heat exhaustion causing vasomotor collapse is more difficult to differentiate from insulin shock, poisoning, hemorrhage, or traumatic shock than is heat stroke. Usually the history of heat exposure, failure of hydration, absence of other apparent cause, and response to treatment are sufficient for diagnosis. The condition is usually transient and the prognosis is good unless circulatory failure is prolonged.

Treatment

Heat exhaustion requires restoration of normal blood volume and assurance of adequate brain perfusion. The patient should be placed flat or with head down. Small amounts of cool, slightly salty fluids should be given orally every few minutes to restore normovolemia. Isotonic saline IV, cardiac stimulants, or plasma volume expanders (albumin, dextran) are seldom needed, and should be given cautiously to avoid overloading an embarrassed circulatory system.

Heat Cramps

Severe cramps of striated muscle resulting from excessive sweating due to exertion and/or high ambient temperatures.

Etiology

Heat cramps are due to excessive loss of sodium chloride by profuse sweating during strenuous activity at high atmospheric temperatures (> 38 °C (100 °F)). It is common in manual laborers (i.e., engine room personnel, steel workers, and miners), in mountaineers or skiers overdressed against the cold, and in those not acclimatized to hot, dry climates where excessive sweating is almost undetected because or rapid evaporation.

Symptoms and Signs

Onset is often abrupt, with muscles of the extremities affected first. Severe pain and carpopedal spasm may incapacitate the hands and feet. Often episodic, the cramping makes the muscles feel like hard knots. When the cramps affect only the abdominal muscles, the pain may stimulate an acute abdomen. Vital signs are usually normal. The skin may be either

hot and dry or clammy and cool, depending on the humidity.

Prophylaxis and Treatment

In most instances, heat cramp is prevented and also rapidly relieved by drinking fluids or eating foods containing sodium chloride. If the patient cannot take food or drink orally, 0.9% sodium chloride IV may be necessary. Sodium chloride tablets are often used for prophylaxis, but can cause stomach irritation, and overdose may lead to edema. Awareness of the problem is usually sufficient to prevent it.

Chapter 256: Cold Injury

(Frostnip; Frostbite; Accidental Hypothermia; Exposure; Immersion or Trench Foot; Chilblains; Pernio)

Injury by cold causing structural and functional disturbances of small blood vessels, cells, nerves, and skin; or generalized lowering of body temperature.

Etiology

Exposure to damp cold (temperatures around freezing) causes **frostnip** and **immersion (trench) foot.** Exposure to dry cold (temperatures well below freezing) causes **frostbite** and **accidental hypothermia.** Loss of body heat is by conduction (wet clothing, contact with metal), convection (windchill), and radiation (flow of heat from warm to cold object). Susceptibility to cold injury is increased by dehydration; drug or alcohol excess; impaired consciousness; exhaustion; hunger; anemia; impaired circulation due to cardiovascular disease, constricted vessels or polycythemia; and in very young or old age.

Hypothermia occurs when the body cannot sustain normal temperatures. Wind chill, wet or inadequate clothing, substance abuse, or debility may cause dangerous hypothermia even though ambient temperature is no lower than 17 to 20 °C (50 to 60 °F). As shivering ceases, the body becomes unable to warm itself and the core temperature falls (see also Accidental Hypothermia in Ch. 264).

Pathology

Ice crystals may form within or between cells, interfering with the sodium pump, thus rupturing cell walls; RBCs clump, and platelet micro-emboli form to cause thromboses; neurovascular impulses shunt blood, often sacrificing an injured part to save the whole. Any or all of these events may produce from mild to severe injuries. **Dry cold injury** is usually superficial: the hard carapace of dry gangrene is often only a few mm thick over healthy tissue. **Wet gangrene** is often complicated by infection and tends to be deeper. **Immersion** or **trench foot** causes physiologic aberrations such as edema, blotchy cyanosis, increased sweating and paresthesias, rather than tissue loss. The pathology of long-lasting symptoms from cold injury **(Chilblains)** is unknown.

Symptoms, Signs, and Diagnosis

Frostnip manifests as firm, cold, white areas on face, ears or extremities. Peeling or blistering (as from sunburn) may occur in 24 to 72 h, and occasionally mild hypersensitivity to cold persists. In **immersion** or **trench foot,** the extremity is pale, edematous, clammy, cold, and numb; tissue maceration and infection are likely. Increased sweating, pain, and hypersensitivity to temperature change may persist for years due to autonomic disturbance. In **frostbite,** the area is cold, hard, white, and anesthetic; on warming it becomes blotchy red, swollen, and painful. Depending on the extent of injury, the area may recover normally or deteriorate to soft wet gangrene or to the black carapace of dry gangrene. In **hypothermia,** the falling core temperature leads to lethargy, clumsiness, mental confusion, irritability, hallucinations, slowed or arrested respiration, and slowed, irregular, and finally arrested heartbeat. Rectal temperature below 34 °C (93 °F) will help to differentiate hypothermia from cardiac disease, diabetic coma or hyperinsulinism, CVA, or substance abuse that may also be present.

Prophylaxis

Preventive measures, though obvious, are often ignored. Several layers of warm clothing, and protection against wetting and wind are important, even though weather may not seem to threaten cold injury. Gloves and socks should be kept as dry as possible, and insulated boots that do not impede circulation are essential in very cold weather. Warm head covering is particularly important, since 30% of heat loss is from the head. Ample fluid and food should be taken. Being alert for and warming cold numb parts may prevent damage. As the body cools, shivering, exertion, warm clothing, and hot drinks may prevent hypothermia.

Treatment

Frostnip is treated by warming with an unaffected hand or warm object. **Frostbitten** extremities should be warmed rapidly in warm water, being careful not to burn the anesthetic tissue. Frozen feet should not be warmed if the victim must walk some distance to care, because trauma to thawed tissue increases the damage; refreezing after thawing is also dangerous. Warm drinks, snuggling with a warm companion, or warming hands or feet against a warm abdomen or axilla may be all that is possible in the field.

In the hospital, extremities should be warmed in large containers of water kept at 38 to 43 °C (100 to 110 °F) while overall assessment is made. Reserpine (0.5 mg) may be given into the brachial or femoral artery of the affected extremity to dilate vessels and decrease sludging; pit viper venom is preferred by European doctors. Tetanus toxoid should be given if damage is extensive and antibiotics if infection is apparent. After warming, extremities should be kept dry, open to air, and as sterile as practicable. Phenoxybenzamine 10 mg/day orally, increasing by 10 mg/day at 4- to 6-day intervals may decrease tissue loss. Heparin and dextran appear less effective. Oxygen is helpful only at altitude. Chemical or surgical sympathectomy is rarely needed. Nutrition and morale require special attention. Surgery should be delayed as long as possible, since the ominous black carapace often will be shed leaving viable tissue; "Freeze in January, operate in July" remains a valid adage. Whirlpool baths followed by gentle drying, rest, and time are the best long-term management. No

treatment for the prolonged symptoms following immersion foot or frostbite is known.

In **hypothermia,** when shivering stops and lethargy and other symptoms increase, *a major emergency is imminent.* The victim is often found unconscious some distance from shelter. At the scene, further heat loss should be prevented and rapid assessment made. If the patient is conscious, warming by any means available may proceed while evacuation is planned. If unconscious, many factors must be considered, and there is debate whether warming should precede or follow removal to hospital. If a pulse is detectable, however faint, transport is preferred, preventing further heat loss and taking extreme care to avoid jarring or sudden motion which may trigger ventricular arrhythmia to which the cold heart is prone. If no pulse is detected, cardiopulmonary resuscitation takes priority, but it may be difficult to restore sinus rhythm. Ventricular tachycardia or fibrillation may occur. Some authorities prefer to control fluids, pH, and oxygenation before resuscitation, providing this can be accomplished rapidly. Unconscious hypothermic victims cannot generate enough heat to warm spontaneously and must be warmed externally.

In the hospital rapid warming by total immersion in a tub of water kept at 45 to 48 °C (113 to 118 °F) is preferred. Inhalation of warm moist air or oxygen, heating pads, or thermal blankets should be used while the bath is prepared. In desperate cases, peritoneal dialysis with large volumes of warm saline may be lifesaving. Acidosis can be expected during warming, and pH, K, and Na levels should be monitored q 2 h until consciousness and rectal temperature of at least 35 °C (95 °F) are present. The ECG should be monitored to detect arrhythmia.

Chapter 257: Radiation Reactions and Injuries

The harmful effects - acute, delayed, or chronic - produced in body tissues by exposure to ionizing radiations.

Etiology

Harmful sources of ionizing radiation once were limited primarily to high-energy x-rays used for diagnosis and therapy and to radium and other naturally occurring radioactive materials. Present sources of potential radiation injury include nuclear reactors, cyclotrons, linear accelerators, alternating gradient synchrotrons, and sealed cobalt and cesium sources for cancer therapy. In addition, numerous radioactive materials have been produced artificially for use in medicine and industry.

Accidental escape of large amounts of radiation from reactors has occurred several times. The most publicized event in the USA was the accident involving the nuclear power plant at Three Mile Island in Pennsylvania on March 28, 1979. More recently, on April 26, 1986, a major accident occurred in the nuclear power complex at Chernobyl in the Ukraine, USSR. This accident resulted in several deaths due to radiation and a large number of injuries. In addition, significant radiation from the accident was detected in most of eastern Europe and parts of western Europe, Asia, and the USA. This event may have been unique because of the differences in design of Russian reactors and the lack of a containment vessel. Radiation exposure from such accidents during the first 40 yr of nuclear energy use up to 1985 (not

including Chernobyl) resulted in 35 serious exposures (whole body irradiation ≥ 100 rems) with 10 deaths; none were associated with power plants. The Three Mile Island accident did not result in any major radiation exposure. Estimated doses received ranged from 73 mrem at 0 to 1 mile from the plant to 0 to 2 mrem at 10 to 20 miles. Doses received by the population in the immediate area of the Chernobyl plant were considerably higher. Table 257-1 puts these doses in perspective. Nuclear power generators in the USA must meet stringent federal standards that limit effluent radioactivity to extremely low levels. Although background radioactivity in the earth and in its atmosphere increased after the years of atmospheric nuclear weapons testing, it appears to have stabilized at present levels.

Ionizing radiation - whether in the form of x-rays, neutrons, protons, alpha or beta particles, or gamma-rays - acts either directly or by secondary reactions to produce biochemical lesions that initiate a series of histologic changes and physiologic symptoms and signs that vary with the radiation dose and time.

The biologic effect of a given dose of radioactivity is also dependent on the rate at which it is administered. A single rapid dose of radioactivity may be fatal, while the administration of the same dose over a period of weeks or months may be tolerated with little measurable acute effect. Relationships between the degree of damage caused and the healing or death of a cell are quite complex. In addition to the early somatic effects of large doses (observable within days), changes in the DNA of rapidly proliferating cells may become manifest as a disease or as a genetic effect in offspring many years later.

Radiation usually is characterized in the lay press as low level or high level, with doses in the range of 20 to 30 rads often referred to as low level radiation. Medical doses are usually < 5 rads and frequently < 1 rad.

Total dose and dose rate determine somatic or genetic effects. The units of measurement commonly used in determining radiation exposure or dose are the roentgen, the rad, and the rem. The **roentgen (R)** is a measure of quantity of x or gamma ionizing radiation in air. The **radiation absorbed dose (rad)** is the amount of energy absorbed in any tissue or substance from exposure and applies to all types of radiation. The R and the rad are nearly equivalent for practical purposes. The **rem** is used in describing the observation that some types of radiation, such as neutrons, may produce more biological effect for an equivalent amount of absorbed energy; thus the rem is equal to the rad time a constant called the "quality factor". For x and gamma radiation, the rem is equal to the rad. The rad and the rem are currently being replaced in the scientific nomenclature by 2 units that are comparable with the International System of Units, namely, the **gray (Gy),** equal to 100 rads, and the **Sievert (Sv),** equal to 100 rem.

The **dose rate** is the radiation dose/unit of time. From the very low dose rates of unavoidable background radiation (about 0.1 rad/yr), where no effect can be detected, the probability of measurable effects increases as the dose rate and/or total dose increases. An observable effect becomes quite certain after a single dose of several hundred rads, but may require higher doses if given at a low dose rate or intermittently. Large doses are of concern because of their immediate somatic effects, while low doses are of concern because of their potential for late somatic and long-term genetic effects. The effects of radiation exposure on an individual are cumulative.

The **body area** exposed is also an important factor. The entire human body can probably absorb up to 200 rads without fatality; however, as the whole-body dose approaches 450 rads the death rate will approximate 50% (i.e. LD_{50}), and a total wholebody dose of > 600 rads received in a very short time will almost certainly be fatal. By contrast, thousands of rads delivered over a long period of time (i.e. for cancer treatment) can be tolerated by the body when small volumes of tissue are irradiated. **Distribution of the dose** within the body is also important. For example, protection of bowel or bone marrow by appropriate shielding permits survival of the exposed individual from what would otherwise be a fatal whole-body dose.

Pathophysiology

Tissues vary in response to immediate radiation injury according to the following descending order of sensitivity: (1) lymphoid cells, (2) gonads, (3) proliferating cells of the bone marrow, (4) epithelial cells of the bowel, (5) epidermis, (6) hepatic cells, (7) epithelium of the lung alveoli and biliary passages, (8) kidney epithelial cells, (9) endothelial cells (pleura and peritoneum), (10) nerve cells, (11) bone cells, (12) muscle and connective tissue. Generally, the more rapid the turnover of the cell, the greater the radiation sensitivity.

If the absorbed dose of radiation is sufficiently high, necrosis of any living cell will occur. Large but sublethal doses of radiation may produce disturbances in cell proliferation: (a) the rate of mitosis is decreased and (b) DNA synthesis is impaired in 2 ways - first, the rate of synthesis is slowed; second, cells may continue DNA synthesis and become polypoid. These and other ill-defined effects of radiation are reasonably certain to occur after significant doses in tissue categories 1 to 4, above, are received.

Diminished production of new cells in tissues that normally undergo continual renewal (i.e. enteric mucosa, marrow, gonads) results in progressive hypoplasia, atrophy, and eventually fibrosis, depending on the dose. Some cells, injured but still capable of mitosis, may be so damaged that they will pass through 1 or 2 generative cycles, producing abnormal progeny such as giant metamyelocytes and hypersegmented neutrophils before they die.

Most of our estimates of the effects of very low levels of radiation exposure (< 10 rads) are obtained by extrapolation from studies of higher doses. These extrapolations usually are based on a linear relationship. Some workers believe there may be a threshold effect, citing experiments in which animals subjected to extremely low levels of increased radiation actually had *prolonged* survival compared to animals receiving radiation from background sources only. Regardless, there are very few objective data documenting the somatic and genetic effects of doses of radiation below 10 rads.

Symptoms and Signs

The disruption of cell renewal systems and direct injury of other tissues produce clearly defined clinical syndromes:

1. Acute radiation syndromes can be divided into cerebral, GI, and hematopoietic categories, depending on dose, dose rate, area of the body, and time after exposure.

The **cerebral syndrome,** produced by extremely high total body doses of radiation (> 3000 rads), is always fatal, and consists of 3 phases: a prodromal period of nausea and vomiting; then listlessness and drowsiness ranging from apathy to prostration (possibly due to nonbacterial inflammatory foci in the brain or to radiation-induced toxic products); and, finally, a more generalized component characterized by tremors, convulsions, ataxia, and death within a few hours.

The **gastrointestinal syndrome** (400 or more rads) occurs when the total body dose of radiation is smaller but still high. It is characterized by intractable nausea, vomiting, and diarrhea that lead to severe dehydration, diminished plasma volume, vascular collapse, and death. The GI syndrome results from the initial "toxemia" due to necrosis of tissue and is perpetuated by progressive atrophy of the GI mucosa. Ultimately, the intestinal villi are denuded, with massive loss of plasma into the intestine. Regeneration of intestinal epithelial cells may be possible after large doses of radiation. Massive plasma replacement and antibiotics during the first 4 to 6 days will keep patients alive until the epithelium regenerates. However, even if the patient does survive, the respite is temporary, since hematopoietic failure will ensue within 2 or 3 wk.

The **hematopoietic syndrome** (200 to 1000 rads), characterized by anorexia, apathy, nausea, and vomiting, may be maximal within 6 to 12 h. Symptoms then subside, and within 24 to 36 h after exposure the subject is asymptomatic. During this period of relative well-being, lymph nodes, spleen, and bone marrow begin to atrophy, leading to pancytopenia. This atrophy is the result of 2 distinct processes - direct killing of radiosensitive cells and inhibition of new cell production. In the peripheral blood, lymphopenia commences immediately, becoming maximal within 24 to 36 h. Neutropenia develops more slowly. Thrombocytopenia may be prominent within 3 or 4 wk.

Increased susceptibility to infection develops due to (1) a dose-dependent decrease in circulating granulocytes and lymphocytes, (2) a dose-dependent impairment of antibody production, (3) impairment of granulocyte migration and phagocytosis, (4) decreased ability of the reticuloendothelial system to kill phagocytized bacteria, (5) diminished resistance to diffusion in subcutaneous tissues, and (6) hemorrhagic areas of the skin and bowel that encourage entrance and growth of bacteria. Susceptibility to infection by both saprophytic and pathogenic organisms is present. Hemorrhage is mainly due to the thrombocytopenia.

With acute total body radiation doses of > 600 rads, hematopoietic or GI malfunction will be fatal; with doses < 600 rads, the probability of survival is inversely related to the total dose.

2. Acute "radiation sickness" following therapeutic irradiation (particularly of the abdomen) is characterized by nausea, vomiting, diarrhea, anorexia, headache, malaise, and tachycardia of varying severity. The discomfort subsides within a few hours or days; its cause is not understood.

3. Delayed effects: (a) *Intermediate effects:* Prolonged or repeated exposure to low dose rates from internally deposited or external sources of radiation may produce amenorrhea, decreased fertility in both sexes, decreased libido only in the female, anemia, leukopenia, thrombocytopenia, and cataracts. More severe or highly localized exposure causes loss of hair,

skin atrophy and ulceration, keratosis, and telangiectasia, and ultimately may cause squamous cell carcinomas. Osteosarcomas may appear years after ingestion of radioactive bone-seeking nuclides such as radium salts.

Serious injury to exposed organs may occur occasionally after extensive radiotherapy for cancer. Renal functional changes include a decrease in GFR and tubular function. Clinical manifestations may occur acutely after extremely high doses (after a latent period of 6 mo to 1 yr) and may include proteinuria, renal insufficiency of varying degree, anemia, and hypertension. When cumulative kidney exposure is > 2000 rads in less than 5 wk, radiation fibrosis and oliguric renal failure will occur in about 37% of cases. The remainder will develop variable changes over a prolonged time. Large accumulated doses to muscles may result in painful myopathy with atrophy and calcification. Very rarely, these changes may be followed by a neoplastic change, usually a sarcoma. Radiation pneumonitis and subsequent pulmonary fibrosis may be severe when lung metastases are irradiated, and can be fatal after a cumulative dose of > 3000 rads if treatment is not spread over a sufficient period. Radiation pericarditis and myocarditis have been produced by extensive mediastinal radiotherapy. Catastrophic myelopathy may develop after a segment of the spinal cord has received cumulative doses > 4000 rads. Following vigorous therapy of abdominal lymph nodes for seminoma, lymphoma, or ovarian carcinoma, chronic ulceration, fibrosis, and perforation of the bowel may develop. Skin erythema and ulceration were observed fairly often during the era of orthovoltage x-ray therapy, but the high-energy photons produced by modern cobalt-60 units and linear accelerators penetrate deeply into tissues and have virtually eliminated these complications.

(b) *Late somatic and genetic effects:* Radiation alters the "information system" of proliferating somatic and germ cells. With somatic cells this may be manifested ultimately as somatic disease - i.e. cancer (leukemia, thyroid, skin, bone) or cataracts - or, as suggested in animal models, by nonspecific shortening of life. Leukemia from substantial radiation in humans has been observed. It is asserted, but not proved, that there is no "threshold" dose for leukemia, and that the incidence increases with dose. Thyroid carcinoma has been observed 20 to 30 yr after x-ray therapy for adenoid and tonsillar hypertrophy, and x-ray treatment for nonmalignant conditions is now considered inappropriate except in highly unusual situations. However, several large studies have failed to show increased thyroid cancer in persons receiving up to 8 rads to the thyroid delivered by radioisotopes.

With germ cell exposure, mutations are increased. When mutations are perpetuated by procreation, animal studies indicate that an increasing number of genetic defectives will be expressed in the course of generations. Although this has not been observed in man as a direct result of germ cell irradiation, the possibility presents serious medical, ethical, and philosophic problems with respect to unborn generations. It imposes a moral obligation to limit radiation exposure to that which is absolutely necessary for valid diagnostic or therapeutic purposes, and to strictly control occupational exposure. The potential harm, however, should be kept in perspective. Some investigators believe that no measurable effects will occur below a certain threshold, while others insist that any radiation is potentially harmful. The long-term probability of a measurable genetic or somatic effect appearing in a given individual is estimated to be 10^{-4}/rad.

Diagnosis and Prognosis

When a person is receiving therapeutic radiation or has been exposed during a radiation accident, the etiology is obvious. Prognosis depends on the dose, the rate at which it was received, and its distribution within the body. Review of the data and serial hematologic and bone marrow studies to gauge the severity of marrow injury are necessary to accurately determine the prognosis.

When the cerebral or GI syndromes are present, the diagnosis is simple but the prognosis grave. Death occurs with the cerebral syndrome within hours to a few days; with GI symptoms within 3 to 10 days; and with hematopoietic symptoms in 8 to 50 days. In the latter, death may occur from a supervening infection in 2 to 4 wk or from massive hemorrhage between 3 and 6 wk.

In **chronic** cases, where external exposure is either unknown or overlooked, a diagnosis may be difficult or impossible. A search for possible occupational exposure is required. In institutions licensed by federal or state governments, records of exposure to radiation are maintained. Serial chromosome studies can be performed to watch for types and frequency of chromosomal abnormalities that are likely to occur after significant radiation exposure, but such abnormalities may have preexisted or been induced by nonradiation sources. Periodic examination for early cataracts is appropriate in chronic radiation exposure of the eye, especially from neutrons.

Cases of alleged exposure to radiation are difficult to evaluate since emotional or psychological factors tend to predominate. Unless the individual has received a documented external or internal dose, exact diagnosis is probably impossible. Normal hematologic values and the absence of objective clinical illness would permit reassurance of the patient and others concerned.

Prophylaxis

Many drugs and chemicals increase the survival rate in animals if given prior to irradiation; i.e. sulfhydryl compounds. However, none are currently of practical value in man. The only certain way to avoid fatal or serious overexposure is the rigorous enforcement of protective measures and adherence to the maximum permissible dose (MPD) levels. These values are listed in *Basic Radiation Protection Criteria,* NCRP Report No. 39, published by the National Council on Radiation Protection and Measurements.

Treatment

Contamination of the skin by radioactive materials should be immediately removed by copious water irrigation and special chelating solutions containing EDTA when available (Radiac Wash). Small puncture wounds must be treated vigorously to remove contamination. Irrigation and debridement are indicated until the wound is free of radioactivity. Ingested material should be removed promptly by induced vomiting or lavage if exposure is recent. If radioiodine is inhaled or ingested in large quantities, the patient should be given Lugol's solution or saturated solution of potassium iodide to block thyroid uptake for days to weeks, and diuresis should be promoted. Monitoring of exposed patients is mandatory, using hand-

type rate-meter probes or sophisticated whole-body counting. Urine should be analyzed for nongamma-emitting radionuclides if exposure to these agents is suspected. Radon breath analysis can be done in cases of suspected radium ingestion.

Since the **acute cerebral syndrome** is uniformly fatal, treatment is palliative and directed toward combating shock and anoxia, relieving pain and anxiety, and sedation for control of convulsions.

Symptoms of **radiation sickness due to therapeutic irradiation of the abdomen** can be diminished by an antiemetic (i.e. prochlorperazine 5 to 10 mg orally or IM qid) and may be prevented by administering the drug beforehand. Attention to nutrition and fluid balance through close cooperation between radiotherapist and referring physician is mandatory. Most difficulties can be avoided or minimized by careful planning of the overall management (i.e. dose, time interval between treatments, supportive therapy).

If the **gastrointestinal syndrome** develops after external whole-body irradiation, the type and degree of therapy will depend on the severity of the symptoms. After modest exposure, antiemetics and sedation may suffice. If oral feeding can be started, a bland diet is tolerated best. Fluid, electrolytes, and plasma, by appropriate routes, may be required in large volumes. The amount and type will be dictated by blood chemical studies (especially electrolytes and proteins), blood pressure, pulse, fluid exchange, and skin turgor.

Management of the **hematopoietic syndrome,** with its obvious potentially lethal factors of infection, hemorrhage, and anemia, is similar to therapy of marrow hypoplasia and pancytopenia from any cause (see Hypoplastic (Aplastic) Anemia under Normochromic Normocytic Anemias in Ch. 96). Antibiotics, fresh blood, and platelet transfusions are the main therapeutic aids. Rigid asepsis during all skin-puncturing procedures is mandatory as is strict isolation to prevent exposure to pathogens.

Concurrent antineoplastic chemotherapy or use of other marrow-suppressing drugs, unless strongly indicated because of some preexisting clinical condition or sudden complication should be *avoided* because of the potential for further suppression of blood-forming elements in bone marrow.

Bone marrow transplants have proved helpful in genetically identical animals. If a dose > 200 rads is suspected, tissue typing and search for a compatible bone marrow should be made. If an identical twin is available, a marrow transplant will increase the probability of survival. If granulocytes and platelets continue to decrease at a constant rate and fall to < 500 and 20.000/microL, respectively, homotransplantation of marrow should be considered, though the likelihood of success is small and the transplant may be followed by a potentially fatal immunologic graft-vs-host reaction. (See Bone Marrow Transplantation in Ch. 22.)

In dealing with **late somatic effects due to serious chronic exposure,** removal of the patient from the radiation source is the first step. With radium, thorium, or radio-strontium deposition in the body, prompt administration of oral and parenteral chelating agents (EDTA) will increase the excretion rate. However, in the late stages these agents are useless. Radiation ulcers and cancers require surgical removal and plastic repair. Radiation-induced leukemia is treated like any similar spontaneous leukemia. Anemia is corrected by whole-blood

transfusion. Thrombocytopenic bleeding may be reduced by platelet transfusions. However, these measures are of only temporary value since the probability is slight that an extensively damaged bone marrow will regenerate. No effective treatment for sterility, or for ovarian and testicular dysfunction, except for hormonal supplementation, has been devised.

Chapter 258: Electric Shock

Injury caused by an electric current passing through the body. The electricity may be atmospheric (lightning) or man-made, i.e., high-voltage transmission and low-voltage lines. Potential injuries include physiologic aberrations and burns.

Pathogenesis

Factors that determine the form and severity of injury (which may range from a small minor burn to death) include (1) the type and magnitude of current, (2) the resistance of the body at the point of contact, (3) the current pathway, and (4) the duration of current flow.

The **type and magnitude of current** have a profound influence upon the injury sustained. In general, **direct current (DC),** which has zero frequency (although it may be intermittent or pulsating), is less dangerous than **alternating current (AC),** the type generally used in the USA. AC, particularly of the common 50-60 Hz (cycles/second) variety, is 3 to 5 times more dangerous than DC of the same voltage and amperage. DC tends to cause a convulsive contraction, often forcing the victim away from further current exposure. The effects of AC on the body depend to a great extent on the frequency - low-frequency currents, 50 to 60 Hz, usually being more dangerous than high-frequency currents. AC, 60 Hz, causes muscle tetany, often "freezing" the hand to the circuit as the fist clenches the current source resulting in prolonged exposure with severe burns. *Generally, the higher the voltage and the amperage, the greater the damage from either type of current.* Both AC and DC may affect the body by either altering physiologic functions (involuntary muscular contractions and seizures, ventricular fibrillation, respiratory arrest due to CNS injury or muscle paralysis, etc) or by producing thermal, electrochemical, or other damage (burns, necrosis of muscle and other tissue, hemolysis, coagulation, dehydration, vertebral and other skeletal fractures, muscle and tendon avulsion, etc). Electric shock often causes a combination of these effects.

The **threshold of perception** for DC entering the hand is about 5 to 10 milliamperes (mA); for AC, 60 Hz, about 1 to 10 mA. The maximum current that can cause contraction of the flexor musculature of the arm but still permit the subject to release his hand from the current source is termed the **"let-go" current.** For DC this value is about 75 mA; for AC, about 15 mA and varies with muscle mass. A low-voltage (110 to 220 volts) 60-Hz AC travelling through an intact skin and a transthoracic pathway for a fraction of a second can induce **ventricular fibrillation** at currents as low as 60 to 100 mA; about 300 to 500 mA of DC are required. If the current has a direct pathway to the heart (i.e. via a cardiac catheter or pacemaker electrodes), much smaller currents (< 1 mA, AC or DC) can produce fibrillation.

Body resistance (measured in ohms/cm^2) is concentrated primarily in the skin, and varies directly with the skin's condition. Dry, well-keratinized, intact skin has an average resistance of 20.000 to 30.000 ohms/cm^2, whereas the resistance of moist, thin skin is about

500 ohms/cm^2. If the skin is punctured (i.e. from a cut or abrasion, or by a needle), or if current is applied to moist mucous membranes (i.e. mouth, rectum, vagina), the resistance may be as low as 200 to 300 ohms/cm^2. A calloused palm or sole may have a resistance of 2 to 3 million ohms/cm^2. As current passes through the skin, much energy may be dissipated at the surface if the skin resistance is high, and large surface burns can result at both the entry and exit points with charring of tissues in between (heat = $amperage^2$ x resistance). Tissues are also burned internally, depending on their resistance, nerves, blood vessels, and muscles conduct electricity far better than the denser tissues of fat, tendon, and bone. If the skin resistance is low, the patient may have few if any extensive burns but may still suffer cardiac arrest from the current reaching the heart.

The **pathway of current** through the body can be crucial in determining human injury. Conduction from arm to arm or between an arm and a foot at ground potential is much more dangerous than contact between a leg and ground since the current may traverse the heart. Electrical injuries to the head may cause seizures, intraventricular haemorrhage, respiratory arrest, ventricular fibrillation or asystole, and cataracts. The most common entry point for electricity is the hand followed by the head. The most common exit is the foot. Lightning rarely, if ever, has entry and exit wounds.

The **duration of current flow** through the body is important. While the heart is vulnerable to small currents at relatively low voltages, in general, the amount of injury to the body is directly proportional to the duration of exposure because tissue breakdown occurs with longer durations allowing internal current flow. Heat is produced by current flow through tissues, causing severe burns, protein coagulation, vascular thrombosis, and tissue necrosis.

When a victim freeze to a circuit (see "let-go" current above) he may suffer severe burns. Conversely, a lightning victim rarely suffers external or internal burns, despite the higher voltage, because the short duration of current is not enough to cause skin breakdown. It "flashes over" the victim, producing little internal damage other than electrical short-circuiting of systems (heart- asystole, brain - confusion and loss of consciousness).

Symptoms and Signs

The effects and clinical manifestations of electrical injuries depend on the complex interaction of the factors discussed above. Electricity can startle a person and cause him to fall down or to drop objects. It may cause severe, spastic stimulation and contraction of the muscles with accompanying fracture, dislocations, and loss of consciousness. Both respiratory paralysis (apnea) and cardiac arrhythmias or arrest may occur. Sharply demarcated electrical burns may be present on the skin and extend well into the subjacent tissues.

High voltage may cause coagulation necrosis of the internal tissues between the entry and exit points of the current. Massive edema may supervene as the veins coagulate and the muscles swell. Fluid and electrolyte disturbances as well as severe myoglobinuria may cause acute renal failure. Dislocations, fractures, and blunt injuries may be present from powerful muscle contractions or falls secondary to the electric shock.

"Bathtub" accident victims, where a wet (grounded) individual contacts a 110 v circuit, may show no burns but suffer cardiac arrest.

Lightning, because of its short duration, rarely leaves entry or exit wounds and seldom causes muscle damage or myoglobinuria. Coma and other neurologic sequela may be present, but usually resolve within hours or days. Death is most frequently due to cardiopulmonary arrest or severe brain damage.

Prevention

Prevention of electrical injuries entails proper design, installation, and maintenance of all electrical devices. Education and compliance, as well as common sense and respect in dealing with electricity, are essential. Any electric device that touches or may be touched by the body and has life-threatening potential should be properly grounded and incorporated in circuits containing fail-safe equipment. **Ground-fault circuits breakers,** which trip at current leakage to ground levels of as low as 5 mA, are excellent safety devices and are readily available.

Treatment

Treatment consists of (1) separating the patient from the current source, (2) reestablishing vital functions immediately, and (3) supportive care as required.

Breaking contact between the victim and the current source can be done either by **shutting off the current** or by **removing the person from contact with it.** The best method is to cut off the source, if it can be done rapidly (i.e. throwing a circuit breaker or switch, disconnecting the device from its electrical outlet, or cutting the wires, using insulated tools); otherwise, the victim must be removed from the source. *For low voltage,* the rescuer should first ensure that he himself is well insulated from ground, and then should use an insulating material (i.e. cloth, dry wood, rubber, leather belt) to pull the person free. *If it is suspected that high voltage lines are involved (> 1000 v), it is best to leave the victim alone until the power can be shut off,* since high and low voltage lines are not always easily differentiated.

Once it has been established that it is safe to touch the victim, **a rapid examination for vital functions should be performed** (i.e. radial, brachial, or carotid pulses; respiratory function; level of consciousness). Airway stabilization is the first priority. If spontaneous respiration is not observed or cardiac arrest has occurred, immediate resuscitation is required. Heart-lung resuscitation technics are detailed in Cardiac Arrest and Cardiopulmonary Resuscitation, in Ch. 27. The treatment of shock and other manifestations of massive burns are discussed in Ch. 254.

Once vital functions have been reestablished, the full nature and extent of the injury must be evaluated (see Pathogenesis, above) and treated. A search for dislocations, fractures, cervical-spine and blunt injuries should be made. If myoglobinuria is present, fluid loading is essential and mannitol or furosemide may be indicated.

Lightning injuries are usually superficial, but victims may require cardiac resuscitation, monitoring, and supportive care. Fluid restriction is the rule due to potential brain edema. After being struck by lightning, patients without vital signs for 15 min or even longer have been revived by CPR, although this is unusual.

Tetanus prophylaxis is required for any burn. An ECG, cardiac enzymes, CBC, and a urinalysis especially for myoglobin are baseline determinations for all electrical injuries. Other tests may be indicated as necessary. Any suggestion of cardiac damage, arrhythmias, or chest pain requires monitoring for at least 24 h. Any deterioration in the level of consciousness mandates a CT scan to rule out intracranial hemorrhage.

Chapter 259: Motion Sickness

A disorder caused by repetitive angular and linear acceleration and deceleration and characterized primarily by nausea and vomiting. Sea-, air-, car-, train-, swing-, and space-sickness are specific forms. Prevention is easier than treatment.

Etiology

Excessive stimulation of the vestibular apparatus by motion is the primary cause. There is great individual variation in susceptibility. The pathways of the afferent impulses from the labyrinth to the vomiting center in the medulla are undefined, but motion sickness only occurs when the 8th nerve and cerebellar vestibular tracts are intact. Visual stimuli (i.e. a moving horizon), poor ventilation (fumes, smoke, and carbon monoxide), and emotional factors (i.e. fear, anxiety) commonly act in concert with motion to precipitate an attack. Motion sickness in space travel is referred to as the **space adaptation syndrome;** weightlessness or zero gravity is an etiologic factor. This syndrome is a major problem in the efficiency of astronauts in the first few days of space flight, but adaptation occurs over several days.

Symptoms and Signs

Cyclic nausea and vomiting are characteristic. They may be preceded by yawning, hyperventilation, salivation, pallor, profuse cold sweating, and somnolence. Aerophagia, dizziness, headache, general discomfort, and fatigue may also occur. Once nausea and vomiting develop, the patient is weak and unable to concentrate. With prolonged exposure to motion, individuals may adapt and gradually return to well-being. However, symptoms may be reinitiated by more severe motion or by recurrence of motion after a short respite.

Prolonged motion sickness with vomiting may lead to arterial hypotension, dehydration, inanition, and depression. Motion sickness can be a serious complication in patients who are already ill.

Prophylaxis and Treatment

Susceptible individuals should minimize exposure by positioning themselves where there is the least motion (i.e. amidships or, in airplanes, over the wings). A supine or semirecumbent position with the head braced is best. Reading should be avoided. Keeping the axis of vision at an angle of 45 degrees above the horizon will reduce the susceptibility. Avoiding visual fixation on waves or other moving objects is helpful to some. A well-ventilated cabin is important and going out on deck for a breath of fresh air is helpful. Alcoholic or dietary excesses before or during travel increase the likelihood of motion sickness. Small amounts of fluids and simple food should be taken frequently during extended

periods of exposure; if the exposure is short, as in air travel, food and fluids should be avoided. In the space adaptation syndrome, movement aggravates the symptoms.

Prophylactic drugs should be given before nausea and vomiting occur. One hour before departure, susceptible individuals may be given diphenhydramine, meclizine, or cyclizine 50 mg orally; promethazine 25 mg or diazepam 5 to 10 mg orally; or scopolamine HBr 0.6 mg orally to minimize the vagal mediated GI symptoms. A smaller dose of scopolamine can be delivered via a dermal patch from which 0.5 mg is released over 3 days. If emotional factors are significant, phenobarbital 15 to 30 mg may be given orally 1 h prior to departure. Sedation with pentobarbital 100 mg orally to induce light sleep may be helpful if alertness is not required. However, sedation should be mild enough to allow mental clarity when the passenger arrives at the destination. All dosages should be appropriately modified for prolonged exposure. After vomiting begins, medication must be given rectally or parenterally to be effective. With prolonged vomiting IV fluids and electrolytes may be required for replacement and maintenance.

Chapter 260: Medical Aspects of Air Travel

Commercial aviation safely transports millions of passengers each year but imposes a variety of potential medical stresses. While absolute prohibitions on flying exist for very few medical conditions, planning and precautions are necessary for some patients who travel by air. Commercial pilots undergo rigorous screening and physical examinations and accidents due to their illness or to substance abuse are extremely rare.

General aviation, a rapidly growing area for business and recreational flying, also present a number of potential health hazards. The FAA requires periodic medical examination of private pilots by specially designated physicians, but most of these approximately 1 million pilots licensed in the USA receive their medical care from private practitioners. Of the more than 4000 accidents that occur each year in general aviation, most happen in recreational aircraft and are often related to the injudicious use of alcohol or drugs. The physician may counsel his pilot patient in these matters, caution about side effects of medications (i.e. antihistamines), and provide current tetanus immunization.

Aircraft of all types present increasing **environmental health hazards** in terms of urban noise and air pollution, occasional large scale disasters, and toxic contamination in agricultural areas. For patients living near large airports, continuous high level noise and air pollution can aggravate a wide variety of medical conditions. Local disaster facilities near airports should be prepared to provide initial management of major trauma and burns of air crash survivors. Physicians in agricultural communities should know the toxic manifestations of the chemicals used in aerial spraying that may accidentally contaminate farm workers or nearby populated areas (see Ch. 289).

Clinical Manifestations

Major problems imposed on the air traveler relate to (1) changes in barometric pressure, (2) decreased O_2 tension, (3) turbulence, (4) circadian dysrhythmia, and (5) psychologic stress.

Changes in barometric pressure: Modern jet aircraft, including the supersonics, maintain cabin pressure equivalent to between 5000 and 8000 ft. At such altitudes free air in body cavities tend to expand by about 25% and may aggravate certain medical conditions. The occasional accidental loss of cabin pressure and the fact that some airplanes are unpressurized must be kept in mind. Upper respiratory inflammation or allergy may obstruct eustachian tubes or sinus ostia, resulting in **barotitis media** (see Ch. 206) or **barosinusitis** (see also Ch. 208). Frequent yawning or closed-nose swallowing during descent, decongestant nasal sprays, and antihistamines taken before or during flight often prevent or relieve these conditions. Children are particularly susceptible to barotitis media and should be given oral fluids or feedings during descent to encourage swallowing (chewing gum or hard candy is more effective than eating). Facial pain of dental origin may occur with air pressure changes. Air travel is *contraindicated* in the following: patients with pneumothorax or potential for its development (i.e. large pulmonary blebs or cavities); and where air or gas is trapped and even modest expansion may cause pain or stress tissue (i.e. incarcerated bowel or recent (< 10 days) laparotomy). Patients with a colostomy should wear a large bag and expect frequent filling.

Decreased O_2 tension: Cabin pressure at a 7500-ft altitude-equivalent results in a PaO_2 of about 70 mm, which is well tolerated by healthy travelers. Problems may arise, however, in a number of conditions, including moderate or severe **pulmonary disease** (i.e. asthma, emphysema, cystic fibrosis, etc), **congestive heart failure, anemia** with a Hb < 8.5 gm/dL, severe **angina pectoris, sickle-cell disease** (but not trait, see Ch. 96), and some **congenital heart diseases.** Patients with these conditions can usually fly safely with continuous O_2, available from most airlines if arranged for in advance (all commercial aircraft carry O_2 for use during an in-flight emergency). Patients recovering from **myocardial infarction** may fly when stable, often within 10 to 14 days. Ankle edema is common on long flights and should not be confused with increased congestive heart failure. **Hypertension** is affected by psychologic stress of flight and may be controlled by a mild pre-flight tranquilizer. Smoking can aggravate mild hypoxia and should be avoided. The impact of alcohol may be increased by fatigue and hypoxia. In general, anyone able to walk 100 yd or climb one flight of stairs and whose disease is stable should tolerate normal cabin conditions without additional O_2.

Turbulence may cause air sickness (see Ch. 259) or injury and can occur at any time. Passengers should keep their seat belts fastened at all times while seated.

Circadian dysrhythmia ("jet lag"): Rapid travel across multiple time zones creates many biologic and psychologic stresses; after long trips, travelers should plan on 24- to 48-h rest upon arrival and avoid major commitments or decisions during this adjustment period. A gradual shift in sleeping and eating patterns (a high protein, low calorie intake is recommended) before departure may partly alleviate the problem. Some therapeutic regimens require alteration to compensate for circadian dysrhythm, i.e., diabetics using long-acting insulin may need to change to regular insulin until they have accommodated to the destination time, available food, and activity. Other medication schedules may require adjustment based on elapsed rather than local time.

Psychological stress: Fear of flying and claustrophobia are psychologic and not influenced by logic or reason; hypnosis and behavioral modification psychotherapy have

reduced the fear of flying in some. Fearful passengers may benefit from mild sedation before and during flight. Hyperventilation may provoke unconsciousness, tetany-like convulsions, or simulate cardiovascular disease; physician-passengers may be asked to volunteer Good Samaritan services in such inflight situations. Psychotic tendencies may become more acute and troublesome in flight, and patients with violent or unpredictable tendencies must be accompanied by an attendant and be appropriately sedated.

Other considerations: (1) Thrombophlebitis is a possibility for anyone sitting for long periods, especially during pregnancy and for patients with venous diseases; frequent (q 1 to 2 h) walks around the cabin and short-movement exercises should be practiced while seated. **(2) Dehydration** may develop because of very low cabin humidity, but can be avoided by adequate intake of fluids and avoidance of alcohol. **(3) Wired jaw:** Maxillofacial injury immobilized by fixed wires, unless fitted with a special quick-release devise, is a *contraindication* to air travel since air sickness may result in aspiration of vomitus. **(4) Pacemakers and metal prostheses:** Newer models of pacemakers are effectively shielded from interference from security devices; older units may be affected. Battery life sufficient for the length of travel should be assured. The metal contents of pacemakers and of metal prostheses may trigger a security alarm; a physician's letter should be carried to avoid security difficulties. **(5) Communicable disease:** Patients with any communicable disease that may endanger others in a crowded aircraft are not acceptable as passengers. International immunization requirements change frequently; current information may be obtained from local or state health departments. **(6) Contact lens** wearers should instilled artificial tears frequently to avoid corneal irritation resulting from low cabin humidity. **(7) Medications and records:** The experienced traveler carries on his person essential medications sufficient to assure continued therapy in the event of lost baggage, theft in hotels, delayed arrival, or local unavailability. Patients who must carry narcotics should have a verifying letter from their physician to avoid possible security complications. A summary of a patient's medical record (including ECG) may be invaluable should a patient become ill away from home. Patients subject to disabling illness (i.e. epilepsy) or who are at high risk should wear a medical identification bracelet or necklace (i.e. as provided by Medic-Alert Foundation, Turlock, CA 95380). A recent dental checkup and carrying extra glasses are wise precautions. **(8) Uncomplicated pregnancy** through the 8th mo is acceptable, high-risk patients must be individually evaluated if travel is planned. Acceptance during the 9th mo usually requires a physician's written approval dated within 72 h of departure and indicating delivery date. Seat belts should be worn across the thighs. Thrombophlebitis is a specific risk when sitting for long periods (see above). **(9) Children:** Infants under 7 days of age are not accepted for travel. For children with chronic disease (i.e. congenital heart disease, chronic lung disease, anemia), the same precautions apply as for adults. **(10) Elderly and the handicapped:** There is no upper age limit and airlines make all reasonable efforts to accommodate patients with handicaps. Wheel chair and litter patients can often be accommodated on commercial aircraft; otherwise, air ambulance service is necessary. Some airlines will accept patients requiring special equipment (IV fluids, respirators, etc) provided appropriate personnel accompany the patient and arrangements have been made in advance. **(11) Special foods,** including low-sodium, low-fat, and diabetic diets are usually available upon advance request.

Further advice regarding air travel may be obtained from the medical department of major airlines or from the FAA Regional Flight Surgeon. Special arrangements (i.e. O_2, wheelchair, etc) can be made through regular reservations clerks, but at least 72 h advance

notice is usually required.

Foreign travel may involve significant difficulties in case of illness. Millions travel abroad yearly; about 1 in 30 requires emergency care. Many insurance plans, including Medicare, are not valid in foreign countries; overseas hospitals often require a substantial cash deposit regardless of insurance. Special insurance programs, including some that will arrange for emergency evacuation (i.e. NEAR, 1900 N. MacArthur, Oklahoma City, OK 72127) are available. Directories listing English-speaking physicians in foreign countries are available from several organizations (i.e. InterMedic, 777 Third Avenue, New York, NY 10017, and International Association for Medical Assistance to Travelers, 736 Center Street, Lewiston, NY 14092); US Consulates may also assist in obtaining emergency medical services. *The Traveler's Medical Manual*, by Scotti and Moore, Berkley Books, 1985, contains valuable information for persons travelling abroad, including those with special health needs.

Chapter 261: Near-Drowning

Pathophysiology

Near-drowning victims, because of aspiration or laryngospasm, usually sustain significant hypoxemia, with the consequent danger of respiratory failure and hypercapnea. Acute reflex laryngospasm may result in asphyxia without aspiration of water. Aspiration of fluid and particulate matter may cause chemical pneumonitis, damaging cells lining the alveoli, and may impair alveolar secretion of surfactant, resulting in patchy atelectasis.

The perfusion of nonaerated, atelectatic areas of the lungs leads to intrapulmonary shunting of blood and aggravates hypoxemia. The more fluid aspirated, the greater the surfactant loss, atelectasis, and hypoxemia. Aspiration of large quantities of water may cause sizable areas of atelectasis resulting in stiff noncompliant lungs and respiratory failure (see Ch. 35). Respiratory acidosis with hypercapnea and hypoxemia can occur. A concomitant metabolic acidosis may also result from tissue hypoxia. Hypoxemia and tissue hypoxia often result in pulmonary edema and even cerebral edema. On x-ray, pulmonary edema may simulate atelectasis and the 2 conditions may coexist.

The mammalian **diving reflex** in cold water allows survival after long periods of submersion. The diving reflex, first identified in seagoing mammals, slows the heartbeat and constricts the peripheral arteries, shunting oxygenated blood away from the extremities and the gut to the heart and brain. In cold water the O_2 needs of the tissues are reduced, extending the possible time of survival.

Respiratory insufficiency is more critical than changes in electrolytes and blood volume, which vary in magnitude depending on the type and volume of aspirated fluid. Sea water may cause a mild elevation of Na and Cl, but the levels are rarely life-threatening. By contrast, aspirating large quantities of fresh water can cause a sudden increase in blood volume, profound electrolyte imbalance, and hemolysis. Victims may succumb to the effects of these changes, asphyxia, and, possibly, ventricular fibrillation, at the scene of the tragedy. Cardiac arrest, usually preceded by fibrillation, causes many of the deaths attributed to drowning. However, current belief is that the pulmonary edema following near-drowning is a direct result of hypoxemia and analogous to pulmonary edema of high altitude, i.e.,

noncardiogenic pulmonary edema.

Prevention

Eating and drinking shortly before swimming should be avoided. Children require proper supervision at beaches and near pools or ponds. All swimmers should be accompanied by an experienced swimmer or swim only in guarded areas. Nonswimmers and small children should wear flotation jackets when in boats or playing near bodies of water. Children should be taught to swim as early as possible, and adults and children over 12 should be familiar with the basics of resuscitation. Infants, children, the debilitated, and the elderly should not be left unattended in bathtubs.

Treatment

The key factors for surviving submersion without permanent injury appear to be duration of submersion, water temperature, age of the individual (the diving reflex is more active in children), and speed of resuscitation efforts. Survival depends more on the prompt correction of hypoxemia and acidosis (ventilatory insufficiency) than correction of electrolyte imbalance, the goal being to prevent pulmonary and cerebral edema due to tissue hypoxia.

If near-drowning takes place in very cold water, the victim *may be hypothermic.* Because of the diving reflex and the reduced metabolic needs associated with hypothermia, vigorous attempts should be made to resuscitate victims (especially children) even though they have been submerged for periods up to 1 h or longer. The management of hypothermia is discussed in Ch. 256.

Emergency mouth-to-mouth resuscitation should begin immediately if the victim is apneic - in the water, if necessary. If heart beat and carotid pulse cannot be detected, closed chest cardiac massage (see Cardiac Arrest and Cardiopulmonary Resuscitation in Ch. 27) is initiated as soon as artificial ventilation is started. Mechanical ventilators, which supply higher inspired O_2 concentrations, should be used if available. Electrical defibrillation may be necessary.

Time should not be wasted in attempts to drain water from the lungs in a fresh-water victim, because the hypotonic fluid passes rapidly into the circulation. Sea water, being hypertonic, draws plasma into the lung, and the Trendelenburg position may promote drainage.

Hospitalization is mandatory for all victims. Resuscitation should continue during transport, regardless of the patient's condition. Consciousness is not synonymous with recovery, since *delayed death from hypoxia can occur.*

Initial emphasis in the hospital continues to be intensive pulmonary care to achieve adequate arterial blood-gas and acid-base levels. Required measures range from simple O_2 administration for a spontaneously breathing patient to continuous ventilatory support of an apneic patient by tracheal intubation with a cuffed tube connected to a mechanical ventilator. Sodium bicarbonate IV is usually indicated, since metabolic acidosis almost invariably accompanies the tissue and cellular hypoxia. Further bicarbonate administration, ventilatory

support, and proper inspired O_2 concentrations are determined by monitoring blood gases. High supplemental levels of O_2 inhalation must be continued until the arterial blood-gas studies indicate that lesser O_2 concentrations are adequate.

Frequent manual hyperinflation of the lungs is indicated to re-expand atelectatic alveoli. Beta2-agonists by inhalation or injection help to reduce bronchospasm. Since near-drowning with fluid aspiration is a form of aspiration pneumonitis, corticosteroids and antibiotics may be considered depending on the individual case.

Fluid and electrolyte solutions are required to correct significant electrolyte imbalance. A large quantity of fluid may be extravasated into the lungs, producing a reduced blood volume that may be reflected by lowered central venous pressure; infusion of volume expanders may be indicated. Fluid restriction is usually not advisable, since the pulmonary and cerebral edema caused by hypoxia are related to direct pulmonary epithelial damage or osmotic gradients rather than to circulatory overload as in congestive heart failure. RBC replacement to increase the O_2-carrying capacity of the blood, and forced diuresis to facilitate excretion of the free plasma Hb may be necessary if there is significant hemolysis.

The patient who develops acute respiratory distress syndrome requires mechanical ventilation. Positive end-expiratory pressure (PEEP) may help to maintain patency of alveoli, prevent alveolar collapse, and expand collapsed alveoli (see Ch. 35). Pulmonary care may be necessary for hours or days, depending on the arterial blood - gas and pH analyses. Permanent brain damage from hypoxemia and tissue hypoxia may be a residual problem in some cases. Cerebral resuscitation measures and the role of neurologic classification for brain injury require further study. The following measures may be beneficial, but carry intrinsic risks: hyperventilation, hyperoxygenation with hyperbaric chambers, hypothermia, barbiturate coma, and steroids. Acetazolamide IV has been effective in relieving cerebral edema due to hypoxia.

Chapter 262: Medical Aspects of Diving and Work in Compressed Air

Serious errors in diagnosis and treatment may occur when illness arises from diving or other activities involving **increased environmental pressure.** Diving with **scuba** (self-contained underwater breathing apparatus) has grown enormously as a popular sport and in commercial and scientific applications. As a result, many individuals are now potential victims of conditions that were once confined to deep sea divers and construction workers in tunnels or caissons.

A patient with almost any disorder that develops during, or especially following, exposure to increased pressure could have decompression sickness or gas embolism and urgently need recompression. Physicians who see such patients must have a high index of suspicion and be ready to seek advice. This chapter provides basic information, and the **Divers Alert Network (DAN),** coordinated by the Duke University Medical Center, Durham, NC, provides consultation at any hour (919-684-8111).

Depths and Pressures

Increased pressure at depth results from the weight of water, just as barometric pressure on land reflects the weight of the atmosphere above. Pressures in diving are often

expressed in units of depth or atmospheres absolute **(atm abs; ATA).** A diver at 33 ft (10 m) in sea water is exposed to a pressure of 14.7 psi, 760 mm Hg, or one atmosphere **(atm)** greater than the barometric pressure at the surface. The **total pressure** at 33 ft, 2 atm abs, includes both the weight of the water and the barometric pressure at the surface. Every additional 33 ft descent adds one atm of pressure. In a caisson or tunnel, compressed air is used to exclude water from the worksite and the interior pressure reflects the head of water outside.

Pathophysiologic Effects of Increased Pressure

Medical problems caused by exposure to increased pressure involve one or more of the following mechanisms:

1. Local differences in pressure ("squeeze). When external pressure on the body increases with depth, the pressure of gas in the lungs and airways increases accordingly. If the eustachian tubes can be opened normally (i.e. by swallowing or yawning), pressure in the middle ear can be kept equal to the increasing external pressure. If a structural anomaly, allergic or vasomotor rhinitis, or URI prevents such equalization, the excess external pressure is exerted directly on the eardrum. The external pressure is also fully transmitted to all blood vessels of the body including those in the mucosa of the middle ear, where if the pressure remains lower than the external pressure, the capillaries may dilate, leak, and rupture. If edema fluid and extravasated blood do not occupy enough space to equalize the pressure, the eardrum may rupture. Middle ear infection often follows such **barotitis media** (see also Ch. 206).

Barotrauma by the same mechanism may also occur in the paranasal sinuses. It is signaled by local pain or, in sphenoid sinus "squeeze", by pain referred to the occiput, vertex, or frontal area. Mucosal congestion causing inability to equalize pressure in the ears or sinuses may respond to local or systemic decongestants, but persistent efforts to dive without free equalization of pressure will usually produce some injury.

Any rigid or semi-rigid airspace attached to the body can also become the site of local "squeeze". Face masks are equalized by air from the nose, but goggles and some diving suits can cause discomfort, local hemorrhage, and tissue damage. Ear plugs form a closed space in the auditory canal and must *not* be used in diving.

2. Compression and expansion of gas. Boyle's Law indicates that the volume of a given mass of gas changes inversely with the absolute pressure; i.e. 1 L of air at the surface (1 atm abs) is compressed to 1/2 L at 33 ft of depth (2 atm abs). Equalization of pressure in body airspaces during descent must compensate for such compression. Compression of lung gas limits the safe depth of a "breath-hold" dive, but breathing from a diving helmet or scuba regulator compensates for compression of gas in the respiratory system.

Changes in pressure and gas volume in the middle ear can produce **vertigo** by at least 3 different mechanisms. (1) If the eardrum ruptures when a diver is bareheaded in cold water, the effect is like that of a **caloric test** (see in Ch. 204) and can produce severe and potentially disastrous vertigo, disorientation, nausea, and vomiting. (2) Unequalized pressure differences in the middle ear may affect the inner ear via the round window and produce **alternobaric**

vertigo, a possible cause of the dysequilibrium sometimes experienced by divers upon starting ascent. (3) **Perilymph fistula,** an uncommon but serious cause of vestibular disorder, requires prompt surgery. It can be confused with inner-ear decompression sickness when it becomes evident following a dive.

Compression of gas at depth produces **increased gas density** proportional to the pressure in atm abs. Since a scuba diver breathes at about the same rate and tidal volume at depth as during comparable work at the surface, the number of gas molecules respired per minute at depth increases in proportion to the pressure. Thus at 2 atm abs it is twice that at the surface. Not only does the diver's air supply duration decrease proportionately, but breathing becomes difficult at greater depths because of the gas-flow limitations of the diver's airways and of his breathing apparatus. Respiratory limitation can accentuate overexertion, respiratory exhaustion, and general fatigue - potentially significant problems of diving even under ideal conditions.

Life-threatening complications can arise from the expansion of pulmonary gas on ascent. If a diver breathes compressed gas at depth and then fails to let it escape freely on ascent, the expanding gas may overinflate the lungs. Possible consequences include **pneumothorax, mediastinal and subcutaneous emphysema,** and **gas (air) embolism;** the latter is an extreme emergency and a leading cause of death among scuba divers (see below and Table 262-1).

3. Partial-pressure effects. The partial pressure of a gas is proportional to the number of molecules of that gas present in a given volume of gas. The concentration of O_2 in air is about 21%, and the partial pressure of O_2 in air at surface (1 atm abs) is about 0.21 atm. The concentration of O_2 in air remains the same at depth, but the partial pressure reflects the increasing pressure and compression of the gas. At 2 atm abs, the number of O_2 molecules per unit volume is twice what it is at the surface, and the partial pressure is double.

The physiologic effects of gases are related to their partial pressure and change according to depth. Toxic effects appear as the partial pressure of O_2 increases. **Pulmonary oxygen toxicity** can cause lung damage with extended exposure to a P_{O2} much above 0.6 atm (equivalent to 60% O_2 at surface or 30% O_2 at 33 ft). **Oxygen convulsions** may occur, especially in working dives, as the P_{O2} approaches or exceeds 2 atm (100% O_2 at 33 ft or 50% at 99 ft).

Increased partial pressures of N_2 produce **nitrogen narcosis,** a condition resembling alcohol intoxication. In divers breathing air, this becomes noticeable at 100 ft or less. It is generally incapacitating at about 10 atm abs (300 ft), where it produces an anesthetic effect resembling that of 30% nitrous oxide at sea level. Helium lacks this anesthetic property and is used in place of N_2 as the diluent for O_2 in deep diving.

Partial pressure of O_2 and CO_2 in alveolar gas are modified by the pressure of depth in **breath-hold diving** and in underwater swimming without breathing apparatus. The impulse to return to the surface and resume breathing depends largely upon CO_2 buildup in the body. When a breath-holding diver hyperventilates beforehand to extend his time underwater, he blows off CO_2 but adds little to his stores of O_2. He may then become **unconscious from hypoxia** without warning before his P_{CO2} rises enough to become an effective stimulus.

Diving to a significant depth during the breath-hold complicates the situation by elevating the P_{O2} and permitting extended O_2 uptake at depth. A diver who has "pushed his limits" under those circumstances may lose consciousness when his alveolar P_{O2} falls to a low level on ascent. This phenomenon is probably responsible for many unexplained drownings among spearfishing competitors and others who do extensive breath-hold diving. The term **shallow-water blackout** is sometimes applied, but it is best reserved for its original meaning in connection with **CO_2 excess** in rebreathing types of scuba. **Hypoxia** is also a potential problem in rebreathing units.

Carbon dioxide poisoning. In normal individuals on land, hyperpnea and breathlessness usually provides ample warning of increased CO_2 in inspired gas. SUch a response may be more the exception than the rule underwater, especially where a high P_{O2} and exertion are also factors. Some individuals develop spontaneous **CO_2 retention** through an inadequate increase in pulmonary ventilation during exertion. Whatever the source, abnormally high P_{CO2} per se can cause **loss** or **impairment of consciousness** at depth and can also increase the likelihood of **O_2 convulsions** and augment the severity of **nitrogen narcosis.** The tendency to retain CO_2 may be suspected in divers who frequently experience post-dive headaches or pride themselves on low air-use rates.

Inert-gas uptake in the blood and tissue occurs whenever the partial pressure of gases such as N_2 is increased. When the pressure is subsequently reduced by ascent, bubbles may form with various consequences (see Table 262-1, and Decompression Sickness, below).

4. Pressure per se. Certain neuromuscular and cerebral abnormalities comprise the **high pressure neurologic syndrome (HPNS),** which is seen in deep diving and may appear at about 600 ft on descent. HPNS is attributed to hydrostatic pressure without reference to gas compression or partial pressures. It has no evident medical importance at shallower depths.

5. Complicating factors in diving include poor visibility, currents requiring excessive effort, and cold. **Hypothermia** can develop rapidly in water, and early effects may include crucial loss of judgment and dexterity. Cold water can trigger fatal **cardiac arrhythmias** in susceptible individuals. **Hypoglycemia** is a serious hazard in insulin-dependent diabetics and in those who indulge in alcohol while neglecting adequate food intake. **Drugs,** including medications as well as **alcohol** and other drugs of abuse, may have unanticipated effects at depth.

Conditions Requiring Recompression

Gas Embolism

(Air Embolism)

A disorder resulting from overinflation of the lungs by expanding pulmonary gas during reduction of surrounding pressure (i.e. ascent from depth in diving), generally characterized by abrupt loss of consciousness and/or other CNS manifestations, and attributed to cerebral gas emboli originating in the lungs. (See Table 262-1.)

Etiology

Overinflation of the lungs is the usual cause of **arterial gas embolism.** The victim is most commonly a scuba diver who holds his breath during ascent from a dive. Running out of air at depth is a common precipitating event. Even swimming-pool depths are sufficient to cause gas embolism if the individual has access to a source of gas and takes even a single breath underwater. Gas inspired at any depth expands on ascent and, if not allowed to escape freely, overinflates the lungs and elevates alveolar pressure resulting in escape of gas into pulmonary veins returning blood to the heart. If this gas reaches the carotid arteries, embolization of the cerebral vessels is almost inevitable.

Symptoms, Signs, and Diagnosis

Immediate **loss of consciousness,** with or without convulsions or other cerebral manifestations, is the typical consequence of gas embolism. A diver who loses consciousness during or very shortly after ascent *must be assumed to have gas embolism and should be promptly recompressed* (see Recompression Treatment, below). Milder symptoms and signs, ranging from behavioral changes to hemiparesis, may also be seen.

Overinflation of the lungs also can produce mediastinal and subcutaneous emphysema, alone or with gas embolism. Pneumothorax is less frequent but more consequential. Hemoptysis or bloody froth suggests pulmonary injury. Iatrogenic arterial gas embolism is not unknown. It may be suspected, i.e., when a patient fails to regain consciousness after heart surgery.

Emergency Treatment

The patient must be transported to a suitable chamber for recompression without any delay for nonessential procedures. Transport by air may be justified if it will save significant amount of time, but exposure to reduced pressure at altitude must be minimized. Most authorities recommend the Trendelenburg position when gas embolism is suspected. Life-saving emergency measures take precedence where indicated (i.e. CPR, control of bleeding, attention to the airway), but these, together with administration of a maximal concentration of O_2 and fluid therapy, can usually be carried out during transport. Recompression treatment is discussed below.

Decompression Sickness

(Caisson Disease; The Bends)

A disorder resulting from reduction of surrounding pressure (as in ascent from a dive, exit from a caisson or hyperbaric chamber, or ascent to altitude), attributed to formation of bubbles from dissolved gas in blood or tissues, and usually characterized by pain and/or neurological manifestations.
(See Table 262-1.)

Pathophysiology

A diver or compressed-air worker breathing air under increased ambient pressure takes up additional quantities of O_2 and N_2 in solution in the blood and tissues. O_2 is utilized continuously, but N_2 (or any other "inert" gas present) leaves the body only via the reverse of its entry through the lungs and circulation. Gradients of partial pressure govern uptake and elimination of the gas, but the degree of **supersaturation** (excess of tissue gas pressure over ambient pressure) is crucial in determining whether symptomatic bubbles will form in the body during or after ascent.

The consequences of bubble formation from dissolved gas are known as **decompression sickness, caisson disease, or "the bends"** (see Table 262-1). Although "the bends" strictly refers to painful manifestations, it is often used as a synonym for decompression sickness.

Consequential bubble formation can usually be avoided by (1) restricting the uptake of gas, as by limiting the depth and duration of dives to a range that does not require decompression stops on ascent ("no-decompression limits"; see Table 262-2), or (2) using a standard air **decompression table,** as found in the *US Navy Diving Manual* (obtainable from the Superintendent of Documents, US Government Printing Office, Washington, DC 20402). The table provides a pattern of ascent that normally allows excess inert gas to escape harmlessly. Decompression sickness rarely occurs after dives within the limits given in Table 262-2 or when US Navy decompression tables are followed; but the diver's account of depth, duration, and decompression procedure is not necessarily reliable. Many divers incorrectly believe that large "safety factors" are built into US Navy diving tables and do not follow them accurately.

Repetitive dives are a major source of difficulty. Some divers are unaware that an excess of inert gas remains in the body after every dive and increases with each subsequent exposure. If the interval between dives is < 12 h, special repetitive dive tables (as provided by the *US Navy Diving Manual*) must be used.

US Navy decompression tables have not been tested for adequacy in females or in older divers; such persons should use them with caution. Dives conducted at **altitude** and **flying after diving** require special procedures or precautions.

Symptoms and Signs

Local pain ("the bends") is present in a large proportion of cases of decompression sickness, but it is often accompanied by neurologic abnormalities. In divers, pain is most commonly reported in or near an arm joint. In compressed-air workers, joints of the leg are more often affected. The pain is unusual, hard to describe, and often poorly localized. "Deep" and "like something boring into the bone" are expressions sometimes used. At first, pain may be mild or intermittent, but it is apt to increase steadily and may become very severe. Local inflammation and tenderness are usually absent and the pain may not be affected by motion.

Neurologic manifestations may accompany pain or be present independently. They are much more common following dives with scuba than in "hard-hat" diving or caisson

work. Currently, the proportion of neurologic problems among cases of decompression sickness probably exceeds 50%. The spinal cord is especially vulnerable, and many divers are unaware of the dire significance of seemingly minor manifestations such as weakness or numbness in the extremities.

Neurologic symptoms and signs are exceedingly variable. They range from mild paresthesia to major cerebral problems. Vestibular involvement may produce severe vertigo and may be difficult to differentiate from **perilymph fistula.** Spinal cord lesions leading to paraplegia are a particular hazard, and delay in treatment may render the condition irreversible.

The condition known as **the chokes,** or **respiratory decompression sickness,** is rare in occurrence but grave in significance. It arises from massive bubble-embolization of the pulmonary vascular tree. Some cases resolve spontaneously, but rapid progression to circulatory collapse and death is not uncommon without prompt recompression. Substernal discomfort and coughing on deep inspiration or inhalation of tobacco smoke are often early manifestations of chokes. In animal studies, chokes are strongly associated with exposure to altitude soon after diving. Chokes and other serious manifestations appearing at **altitude** are not necessarily cured by return to ground level and may require prompt chamber recompression.

Other manifestations of decompression sickness include **itching,** skin **rash,** and exceptional **fatigue.** These have not customarily been treated by recompression, but they are sometimes forerunners of much more serious problems. Divers complaining of them should at least be kept under observation. One hundred percent O_2 by mask may relieve such symptoms. **Cutaneous edema** is a rare occurrence and probably reflects obstruction of lymphatic channels by bubbles. It deserves recompression if progressive or persistent. **Mottling** ("marbling") of the skin is uncommon but may precede or accompany conditions that require recompression. **Abdominal pain** may reflect bubble formation at the site; but, especially in the form of **girdle pain,** *it can be an important signal of spinal cord involvement.*

Late manifestations of decompression sickness include **aseptic bone necrosis** (dysbaric osteonecrosis). This is much more common in compressed-air workers than in divers, but divers are not immune. Bone necrosis is presumably a consequence of bone infarction by bubbles, as in inadequate or delayed treatment of bends. Lesions adjacent to articular surfaces are most common in the shoulder and hip and can cause great damage to the joint with chronic pain and severe disability. Bone necrosis is an insidious hazard because it becomes symptomatic or is detected by x-ray months or years after the responsible insult, which may be a single improper decompression.

Permanent neurologic defects, i.e., **paraplegia,** are frequently attributable to delayed or inappropriate treatment of early signs of spinal cord involvement. In some instances, the initial damage may be too severe to remedy even with prompt and well-chosen treatment. However, repeated treatments with **hyperbaric oxygen** (see in Ch. 267) assist in recovery. The prognosis appears to be considerably more favorable in spinal cord injury from decompression sickness than from other forms of trauma.

Emergency Treatment

Decompression sickness requires recompression. *Transport to a suitable recompression facility takes precedence over any procedure that can be conducted during transport or postponed without serious risk to life.* Transport should not be delayed even in cases that appear mild, since more serious manifestations may develop. O_2 in maximal concentration should be administered by mask. Shock may develop, especially in severe cases with delayed treatment. Adequate fluid intake should be ensured and both intake and output recorded along with the vital signs.

Recompression Treatment

(See also Ch. 267)

Recompression is imperative in both decompression sickness and gas embolism and should be accomplished as soon as possible to avoid serious and lasting injury. Its objective is to compress bubbles to asymptomatic size, redissolve them, and restore adequate O_2 to affected tissues. Regardless of the distance to a chamber or how long the delay, it is likely to be beneficial. Unnecessary recompression involves far less risk than palliative treatment prescribed in the hope that the problem will subside without recompression.

Recompression in the water *must not* be attempted. Divers themselves, and medical facilities and rescue and police units in popular diving areas, should know the location of the nearest suitable chamber, the means of reaching it rapidly, and the most appropriate source of consultation by telephone. In the absence of such local forethought, the DAN number (919-684-8111) is invaluable.

When a physician recommends recompression but cannot be sure that the patient will receive proper treatment in a suitable chamber, he should obtain signed and witnessed acknowledgement of his recommendation. What constitutes a "suitable" chamber is currently being debated. Many cases can be treated successfully in one-man hyperbaric chambers available in many areas; however, the need for "hands-on" access to the patient or for greater pressure capability often cannot be foreseen and may be vital.

The need for recompression is determined by the fact that the individual has been exposed to increased pressure and has symptoms or signs suggestive of decompression sickness or gas embolism. Details of the history, physical examination, and laboratory findings usually add little of value. Sometimes, a conclusive diagnosis cannot be made without a "test of pressure". At some point, at least before leaving maximum treatment pressure, a careful **neurologic examination** is important to identify any defect that might require modifying the course of treatment.

Failure to provide prompt and appropriate treatment of decompression sickness or gas embolism entails entirely unacceptable risk of serious and lasting injury.

Tables for treatment and guidelines for their selection and use are found in the *US Diving Manual.* The main difference in treatment between gas embolism and decompression sickness is that tables specifically designed for the former include an excursion to 6 atm abs

with the aim of rapid compression of cerebral bubbles. Otherwise, treatment of both conditions normally relies upon breathing O_2 at pressures < 3 atm abs.

The US Navy treatment tables achieve a high percentage of success in cases of decompression sickness that follow ordinary diving with air as the breathing medium. Dives involving unusual gas mixtures or extraordinary depths or durations may require special therapeutic procedures.

Medical adjuncts to recompression: Patients will sometimes need medical or surgical procedures in addition to recompression. If it is impossible to provide both simultaneously, recompression takes precedence over any measure that can be postponed without serious risk to life. Intensive care, when required, can be provided in a well-equipped chamber.

Adequate fluid intake with monitoring is important. If IV fluids are required, 0.9% sodium chloride is generally preferred. The possibility of bladder paralysis and need for catheterization should be kept in mind. Periodic measurement of Hct is desirable, and measurement of central venous pressure and circulating blood volume may be necessary in severely ill patients.

Corticosteroids (i.e. dexamethasone sodium phosphate 20 to 40 mg IV, then 4 mg IM q 6 h) may be useful in curbing inflammation from decompression sickness and in controlling CNS edema. Additional measures for reducing brain/cord swelling are indicated when CNS manifestations are present, especially if the response to recompression is not prompt.

Sedatives and narcotics may obscure symptoms and cause respiratory insufficiency. They should be avoided before and during treatment or used in minimum effective dosage when urgently needed.

Evaluation of Fitness for Diving

Although asked to judge the fitness of individuals for diving or related pursuits, physicians usually cannot bar anyone from diving and function largely as advisors. It is advisable not only to explain unwelcome findings and their implications but to make them a matter of record with signed acknowledgment by the individual concerned.

In the absence of uniform standards, a few self-evident considerations can be cited:

(1) A diver must be able to take care of himself over a wide range of conditions. Diving can involve unusually **heavy exertion** even for individuals who do not plan to participate in any arduous underwater activities. Air cylinders are heavy, currents can necessitate strenuous swimming, etc. Divers should be free of any significant cardiac or pulmonary disease and possess normal to superior **aerobic capacity.** Gross **obesity** is often associated with poor exercise tolerance and increased susceptibility to decompression sickness. **Physical handicaps** must be assessed in terms of the individual's ability to aid a "diving buddy" as well as to function as a diver with minimal assistance himself.

Rigid **age limits** are inappropriate, but older aspirants deserve special scrutiny especially in **cardiopulmonary fitness.** Family history and coronary risk factors should be

considered. Certain **cardiac arrhythmias,** including some that are acceptable in other sports, are contraindicated in diving even in younger people.

(2) **Divers must be able to equalize pressure uneventfully in all body air spaces.** Pulmonary conditions that involve air trapping may cause gas embolism on ascent. *Absolute contraindications* include lung cysts, asthma, emphysema, and a history of spontaneous pneumothorax. Chronic nasal congestion, perforated eardrum, and certain otologic operations are *contraindications.* Diving should be avoided during respiratory infections and exacerbations of vasomotor or allergic rhinitis. Habitual air-swallowing and a tendency toward regurgitation have unfavorable implications.

(3) **Divers must not be subject to impairment of consciousness, alertness, or judgment.** Such lapses, even if momentary, can lead to underwater mishaps endangering the diver and his companion. Epilepsy, syncope, insulin-dependent diabetes, and alcohol or drug abuse are incompatible with diving. Medications that cause drowsiness or reduce alertness are undesirable and may potentiate N_2 narcosis. Lack of emotional stability is perilous for a diver and his associates. It is suspected when motivation seems inappropriate or when the history suggests accident-proneness or impulsive behavior.

Women have taken up scuba diving in large and increasing numbers, and present knowledge suggests that, with only a few exceptions, fit and healthy women can dive as safely as men. One probable exception concerns susceptibility to decompression sickness; women should be even more conservative about **decompression** than men. Another exception concerns **pregnancy,** with the likelihood that diving increases the incidence of birth defects and fetal death. Safe limits of exposure cannot be specified with confidence, so diving is best avoided by women who are, or may be, pregnant.

Evaluation of professional divers and others at unusual risk warrants special procedures including pulmonary function testing, stress electrocardiography, audiometry, and bone x-rays.

Adequate training is an absolute necessity for safe diving, and the physician should emphasize its importance. Courses under the auspices of national organizations are widely available.

Chapter 263: High-Altitude Illness

(Acute Mountain Sickness (AMS); High-Altitude Pulmonary Edema (HAPE); High Altitude Cerebral Edema (HACE); Soroche; Puna; Mareo)

Syndromes due to decreased O_2 at high altitudes.

Etiology, Pathology, and Pathophysiology

Atmospheric pressure decreases as altitude increases but the percentage of O_2 in air remains constant; as a result the partial pressure of O_2 decreases with altitude and at 18,000 ft (5500 m) is about 1/2 that at sea level. About 15% of persons ascending above 9000 ft (2700 m) in less than a day will develop symptoms and signs of altitude illness in which the

dominant form may vary. Persons who have had one attack are slightly more susceptible to another under similar conditions, but there is great variation between individuals and even in the same person at different times. Children under six and women in the premenstrual phase are especially vulnerable. Very rapid ascent (as in unpressurized aircraft, balloons, or a decompression chamber) causes a different form of hypoxic illness.

Hypoxia stimulates breathing, which increases tissue oxygenation but also causes respiratory alkalosis, which contributes to the symptomatology until it is partially compensated by loss of bicarbonate in urine. Hypoxia impairs the O_2 dependent "sodium pump", resulting in the accumulation of Na and water within, and the movement of K out of cells; it is thought that the resultant swelling of cells is the basic pathophysiology of altitude sickness. ADH secretion may be increased by hypoxia in some individuals causing further water retention.

No specific pathology has been demonstrated in AMS. Pulmonary artery pressure is invariably elevated by hypoxia, but systemic pressure may or may not increase. Interstitial edema precedes frank alveolar edema in HAPE; the edema fluid resembles plasma, which presumably seeps through lung capillary walls. Edema and petechial hemorrhages are found in the brains of patients dying from HACE and HAPE. Platelet and fibrin microemboli or gross thromboembolism are common in severe cases. Retinal hemorrhages, and splinter hemorrhage beneath nailbeds are often seen above 16,000 ft (5000 m), but nosebleed is rare. Endocrine glands are normal, though their function is often altered. Liver, kidneys, and heart are normal and passive congestion is not seen.

Symptoms, Signs, and Diagnosis

Various forms of altitude illness may occur separately or in combination. **Acute mountain sickness (AMS)** is the most common and may appear at altitudes as low as 6500 ft (2000 m). It is characterized by headaches, fatigue, nausea, dyspnea, sleep disturbance, and rapid, forceful heartbeat. Exertion aggravates the symptoms. Unless dehydration is severe or hyperventilation is excessive, AMS usually subsides within a few days. Laboratory studies are nonspecific and rarely required for diagnosis.

High-altitude pulmonary edema (HAPE) is less common but more serious, usually developing 24 to 72 h after rapid ascent above 9000 ft (2700 m). Long time high-altitude residents, returning after a brief stay at low altitude appear to be at a slightly greater risk. Children under six are much more susceptible than adults. HAPE is characterized by increasing dyspnea; irritative cough that becomes productive of frothy, often bloody sputum; weakness; ataxia; and later coma. Cyanosis, tachycardia, and low grade fever are common, and together with fine and coarse rales (often audible without a stethoscope) may lead to a misdiagnosis of pneumonia. Chest x-ray shows Kerley lines and a patchy distribution of edema quite different from that seen in congestive heart failure. Atrial pressure is normal, but pulmonary artery pressure is even greater than that found in normal subjects during hypoxia. HAPE may worsen rapidly, and coma and death may occur within hours.

The absence of one pulmonary artery is a rare congenital anomaly that greatly increases the risk of HAPE even as low as 5000 ft (1500 m), probably because of the resulting mismatch between perfusion and ventilation. Persons who develop HAPE repeatedly or at unusually low altitude, should be studied for unsuspected pulmonary artery pathology.

High-altitude cerebral edema (HACE) is probably present to some degree in all altitude illness, but occasionally is seen in severe form with headache, mental confusion, gait ataxia, clumsy hand movements, and diplopia. Occasionally, coma develops rapidly after few symptoms or signs, but usually ataxia and hallucinations give warning. Stiff neck is not seen and papilledema is not necessary for diagnosis. HACE must be differentiated from other causes of coma (such as infection, vascular accident, ketoacidosis) by history, absence of significant fever or paralysis, and by normal blood and spinal fluid studies.

High altitude retinal hemorrhages (HARH) are common above 17,000 ft (5200 m) but if seen at lower altitudes should be considered due to other illness. HARH may occur in the absence of any other altitude illness but are more common with HAPE and HACE. They are asymptomatic unless in the macular region and are absorbed without sequelae. "Cotton wool spots" occur rarely.

Prophylaxis

Altitude illness is best prevented by slow ascent, no faster than 1000 ft (300 m) a day from 5000 to 10,000 ft (1500 to 3000 m) and more slowly above this. Physical fitness and climbing experience, though they enable greater exertion for less O_2 consumption, do not protect against any form of altitude illness. Strenuous effort should be avoided for several days, but bed rest is less beneficial than mild exercise. Because of great individual variation, those going to altitude should learn how fast they can ascend without symptoms; a climbing party should be paced at the rate of its slowest member. Young children, and altitude residents returning from a sojourn at low altitude should be especially alert for symptoms.

Water loss is greatly increased by overbreathing the dry air at altitude, and dehydration with some degree of hypovolemia aggravates symptoms. Drinking much more water than usual is important, but additional salt should be avoided. Alcohol seems to worsen AMS. A light diet, high in easily digested carbohydrates (fruits, jams, starches) improves altitude tolerance and is recommended for the first few days.

Acetazolamide is an effective prophylactic for AMS. Dosage recommended is 250 mg orally q 8 h the day before, during, and a day after ascent; sustained release capsules may be used. Acetazolamide inhibits carbonic anhydrase and allows increased ventilation and better O_2 transport with less alkalosis; it halts periodic breathing (almost universal during sleep at altitude) thus preventing sharp falls in blood O_2. Low flow O_2 during sleep accomplishes the same but is inconvenient. Aspirin may relieve headache and possibly decrease the risk of HAPE by preventing platelet emboli. Prochlorperazine 10 mg orally q 6 h, a popular preventive in Europe, has not been widely used in the USA. Antacids are worthless. Phenytoin is currently being reevaluated because of its value as a membrane stabilizer. Dexamethasone 4 mg orally q 6 h may also prevent AMS.

Acclimatization: Persons exposed to altitude gradually develop an integrated series of responses that restore tissue oxygenation toward normal. Full acclimatization takes more time the higher the altitude, and above 18,000 ft (5500 m) deterioration is more rapid and there are no permanent residents. Major features of acclimatization include moderate sustained hyperventilation with slight alkalosis and decreased alkaline reserve, normal or low cardiac output, increased hemoglobin and myoglobin, increased tissue capillaries, and changes in cell

enzyme activity. After many generations at altitude, different populations have acquired slightly different strategies of acclimatization. Although acetazolamide has been used to enhance acclimatization, its value is not proven.

Treatment

AMS seldom requires treatment other than fluid, analgesics, light diet, mild activity, and (rarely) descent. Acetazolamide 250 mg orally q 4 h is helpful in AMS and HAPE, but more controlled studies are needed. When **HAPE** is suspected, bed rest and O_2 may be tried, but if condition worsens, *immediate descent is essential.* O_2 is less effective than descent. Although morphine may be effective, respiratory depression may outweigh its value. Digitalis, phlebotomy, and limb tourniquets are of no value since congestive heart failure is not at fault. Furosemide (20 to 40 mg orally q 2 h or 20 mg slowly IV) has been effective. (Caution: *Brisk diuresis may cause hypovolemic shock, since the patient is often already dehydrated; oral fluid replacement is essential.*) In hospital, treatment is based on ruling out other causes of pulmonary disease; adequate oxygenation, perhaps by intubation and Positive End Expiratory Pressure (PEEP); bedrest; judicious diuresis; postural drainage; and antibiotics, if superimposed infection is suspected. Recovery is usual within 24 to 72 h. Persons who experience one episode of HAPE are likely to have another and should be warned. In severe **HACE,** dexamethasone (8 mg IV q 4 h) has been used and IV acetazolamide has been dramatically effective. **HARH** require no treatment, generally resolving during stay at altitude.

Chronic mountain sickness (CMS or Monge's disease), is an uncommon condition which affects longtime altitude residents; it is characterized by fatigue, dyspnea, aches and pains, excessive polycythemia, thromboembolism, and ultimately cardiac failure. CMS resembles **alveolar hypoventilation** (formerly called pickwickian syndrome) and both are thought caused by an inadequately sensitive respiratory center. The victim should descend to sea level; recovery is slow and return to altitude may cause recurrence. Repeated phlebotomy has been helpful but may not be the most desirable management.