Organ system failure resulting from burn sepsis has greatly diminished during the past 30 years, because of the introduction of techniques for early excision and grafting of the burn wound. Removal of the burn wound decreases infection and reduces the need for reconstructive surgery. Grafting is done with split-thickness skin grafts; dermal substitutes, which serve as a bed for cell repopulation, may be used in large full-thickness burns.

Injury to the airways and lungs may develop within 24 to 48 hours after the burn and may result from the direct effect of heat on the mouth, nose, and upper airways or from the inhalation of heated air and noxious gases in the smoke. Water-soluble gases, such as chlorine, sulfur oxides, and ammonia, may react with water to form acids or alkalis, particularly in the upper airways, producing inflammation and swelling, which may lead to partial or complete airway obstruction. Lipid-soluble gases, such as nitrous oxide and products of burning plastics, are more likely to reach deeper airways, producing pneumonitis.

In burn survivors the development of hypertrophic scars, both at the site of the original burn and at donor graft sites, and itching may become long-term, difficult-to-treat problems. Hypertrophic scarring is a common complication of burn injury marked by excessive deposition of collagen in the healing wound bed; its etiology is not well understood.

MORPHOLOGY

Grossly, full-thickness burns are white or charred, dry, and painless (because of destruction of nerve endings), whereas, depending on the depth, partial-thickness burns are pink or mottled with blisters and painful. Histologically, devitalized tissue reveals coagulative necrosis, adjacent to vital tissue that quickly accumulates inflammatory cells and marked exudation.

Hyperthermia

Prolonged exposure to elevated ambient temperatures can result in heat cramps, heat exhaustion, and heat stroke.

- *Heat cramps* result from loss of electrolytes via sweating. Cramping of voluntary muscles, usually in association with vigorous exercise, is the hallmark. Heat-dissipating mechanisms are able to maintain normal core body temperature.
- *Heat exhaustion* is probably the most common hyperthermic syndrome. Its onset is sudden, with prostration and collapse, and it results from a failure of the cardiovascular system to compensate for hypovolemia caused by dehydration. After a period of collapse, which is usually brief, equilibrium is spontaneously re-established if the victim is able to rehydrate.
- *Heat stroke* is associated with high ambient temperatures, high humidity, and exertion. Older adults, individuals undergoing intense physical stress (including young athletes and military recruits), and persons with cardiovascular disease are at particularly high risk for heat stroke. Thermoregulatory mechanisms fail, sweating ceases, and the core body temperature rises to more than 40°C, leading to multiorgan dysfunction that can be rapidly fatal. The hyperthermia is accompanied by marked generalized vasodilation, with peripheral pooling of blood and a decreased effective circulating blood volume. Hyperkalemia, tachycardia, arrhythmias, and other systemic effects are common. Particularly important, however, are sustained contractions of skeletal muscle that can exacerbate the hyperthermia and lead to muscle necrosis (rhabdomyolysis). These phenomena appear to stem from nitrosylation of ryanodine receptor 1 (RYR1), which is located in the sarcoplasmic reticulum of skeletal muscle. RYR1 regulates the release of calcium from the sarcoplasm. Heat stroke deranges RYR1 function and allows calcium to leak into the cytoplasm, where it stimulates muscle contraction and heat production. Inherited mutations in *RYR1* occur in the condition called *malignant hyperthermia*, characterized by a "heat-stroke–like" rise in core body temperature and muscle contractures following exposure to common anesthetics. *RYR1* mutations may also increase the susceptibility to heat stroke in humans and produce heat intolerance in mice. Of interest, mice with *RYR1* mutations are protected from heat stroke by drugs that inhibit calcium leakage from the sarcoplasm, suggesting that it may be possible to develop specific therapies for those who develop or are at high risk for heat stroke and malignant hyperthermia.

Hypothermia

Prolonged exposure to low ambient temperature leads to hypothermia, a condition seen all too frequently in homeless persons. High humidity, wet clothing, and dilation of superficial blood vessels resulting from the ingestion of alcohol hasten the lowering of body temperature. At a body temperature of about 90°F, loss of consciousness occurs, followed by bradycardia and atrial fibrillation at lower core temperatures.

Hypothermia causes injury by two mechanisms:

- *Direct effects* are probably mediated by physical disruptions within cells by high salt concentrations caused by the crystallization of intra- and extracellular water.
- *Indirect effects* result from circulatory changes, which vary depending on the rate and duration of the temperature drop. Slow chilling may induce vasoconstriction and increase vascular permeability, leading to edema and hypoxia. Such changes are typical of "trench foot." This condition developed in soldiers who spent long periods of time in waterlogged trenches during the First World War (1914-1918), frequently causing gangrene that necessitated amputation. With sudden, persistent chilling, the vasoconstriction and increased viscosity of the blood in the local area may cause ischemic injury and degenerative changes in peripheral nerves. In this situation, vascular injury and edema become evident only after the temperature begins to return to normal. However, during the period of ischemia, hypoxic changes and infarction of the affected tissues (e.g., gangrene of toes or feet) may develop.

Electrical Injury

Electrical injuries, which are often fatal, can arise from contact with low-voltage currents (i.e., in the home and

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