BURNS (SECOND OR THIRD DEGREE)

Tissue injury classified as a *burn* may be caused by thermal, electrical, radioactive, or chemical agent denaturation of cellular protein resulting in cell injury or death. The degree of damage depends on the type, duration, and intensity of the agent.

The severity of the injury is gauged against the extent of the injury (percentage of total body area affected and the depth of injury). *Burns* are generally classified as three types: first, second and third degree.

The damage sustained from a *first-degree burn* is limited to the outer layer of epidermis; symptoms include erythema, sensations of warmth or heat, palpation tenderness, and overt pain.

Damage sustained from a *second degree or partial thickness burn* extends through the epidermis and involves the dermis, but leaves the sweat glands and hair follicles intact to provide cells necessary for rapid regeneration of the epithelium. *Second-degree burn* symptoms include intense pain and the formation of vesicles, blebs and bullae.

A third degree (or full thickness burn) causes destruction of both the epidermis and dermis to the tissue level where sweat glands, hair follicles and nerve endings are destroyed. Symptoms of the *third degree burn* include charring, or (as in cases of scalding) coagulation of the skin; the *third degree burn* site is initially painless and insensitive to touch, but intense pain and tactile hypersensitivity develop with the regrowth and exposure of nerve endings.

Severe or extensive *burns* may initially be accompanied by the symptoms characteristic of primary shock, including varying degrees of low blood pressure, weak thready pulse, cold and clammy extremities, pale face, cold sweats, increased respiration, restlessness, confusion, anxiety, and oliguria. More life threatening is the insidious secondary shock that may develop with increased capillary permeability resulting from blood vessel wall damage, which allows copious amounts of fluid to exude into the wound area. The lost fluid is made up of water, crystalloids and plasma protein that may comprise two-thirds of the volume.

Of constant concern is the possibility of colonization of the *burn* site by opportunistic bacterium: pseudomonas aeruginosus, staphylococcus aureus, and streptococci are most likely to attack sites in the upper trunk and upper extremities, while coliform and clostridium may be a threat below the waist. Complicating matters is the fact that the severe tissue stress involved provokes an autogenic high output of adrenocortical steroids, making bacterial invasions more dangerous by suppressing the autoimmune defenses of the body, rendering the patient more vulnerable to infection and subsequent septicemia (the greatest threat to the patient's life). IgA and IgG immunoglobulins may fall to extremely low levels in the first forty-eight hours following the burn incident, to gradually be restored to normal levels over a seven to fourteen day period. Cellular immunity becomes severely depressed in the post-burn period and may remain depressed for up to two months.

There is a remote possibility that the patient may develop nephrosis, as well as Curling's ulcers of the stomach, induced by stress, from high steroid levels; such ulcerations carry the threat of hemorrhage or perforation of the stomach wall.

S*econd degree burn* site healing (if there are no complications) follows the pattern of wound healing: (1) acute inflammation with inflammatory exudate to clear wound site of microorganisms and tissue debris, (2) contraction of wound edges, (3) in-growth of capillary buds, (4) fibroblast proliferation, (5) synthesis and aggregation of tropocollagen, (6) reepithelization, and (7) maturation and contraction of scar tissue. The *third degree burn* healing may depend on skin grafting to provide the cells necessary for reepithelization.

Treatment

Much of the physical treatment of the *second or third degree burn* in the recovery stage revolves around the *careful* debridement of necrotic tissue from the *burn* site without causing additional hemorrhage or damage to granulating tissue (usually performed in meticulous fashion with blunt, broad probes, with every effort being made to avoid damaging the tops of the granulating tissue); debridement is performed to reduce the warm debris cover that may serve to enhance bacteria incubation and to facilitate the healing process.