delayed return or even loss of functional capacity. Many such wounds will require revision at a later time with the patient having needlessly been denied the appropriate care. Wounds that are assessed as needing excision and closure should undergo removal as soon as possible. Jackson and associates demonstrated in the 1950s the feasibility of burn wound excision and closure in wounds of up to 30% TBSA. Present day management of burn patients has extended this approach to all but the most massive of burn injuries.

If the patient is otherwise stable burn wound excision can be carried out within a matter of hours post-injury. In the patient with a small burn delay is often related to scheduling of the operating room and the surgical team. Timely excision for the patient with a small burn reduces the period of disability and the overall cost of the injury. In patients with a large burn wound the timing and extent of the surgery is based on the patient’s relative physiologic stability and his capacity to undergo a major operative procedure. Early burn wound excision and closure in patients with large wounds shortens the length of hospitalization, reduces cost, and favorably impacts overall burn mortality. The presence of the burn wound is the primary stimulus for the ongoing problems facing the burn patient. Closure of the burn serves to ameliorate much of the post-burn pathophysiology and is one of the most effective means by which to improve a burn patient’s outcome.

Wounds that are small in size or linear in shape can be managed by excision of the burn and primary wound closure. This is of use in burns of the upper inner arm in the elderly, localized burns of a pendulous breast, abdominal burns, buttock injuries and thigh burns. Primary wound closure can also be achieved in some wounds with local tissue transfer techniques. This approach works quite well when these wounds are excised early before significant microbial colonization of the wound occurs. In such cases the burn is transformed into a healing surgical incision and creation of a skin graft donor site is avoided.

In selected cases the injury may be of such a nature that amputation of the burned part is the most appropriate plan. In the patient with significant multi-system trauma the expeditious removal of the burn injury might be seen as the best option for the patient’s overall survival. In a recent report Santaniello and associates found that the mortality in trauma victims with significant burn injuries and trauma was 28.3% whereas in patients with burns only it was 9.8% and in patients with trauma
only it was 4.3%. The management of these challenging patients requires a coordinated well-conceived plan of care that accounts for all of the patient’s injuries and integrates the treatment needs of each injury to achieve an overall satisfactory outcome. A mangled extremity, which has also suffered a severe burn that is deemed non-salvageable, should undergo early amputation. It is not necessary to extend the amputation to a level that allows closure with unburned tissue. If viable muscle is available to close the amputation site that wound bed can be resurfaced with an autogenous skin graft. A grafted amputation site can, with a modern prosthesis, function as a durable stump. In a patient who is paraplegic and suffers an extensive deep lower extremity burn injury amputation can be a viable alternative to excision and grafting.

A similar option may need to be considered for the patient in whom significant preexisting peripheral vascular disease makes the likelihood of a healed and functional extremity highly unlikely. This unfortunately has become an all too frequent occurrence in the care of elderly burn patients who have progressive complications from long standing diabetes mellitus. The amputation level should be that which will maintain maximum function. This might be a trans-metatarsal or Chopart-type amputation in patients with injuries of the distal foot. In patients confined to a wheelchair who have injuries to the leg a through-knee amputation as opposed to an above-knee amputation provides a weight-bearing platform for sitting. In the patient in whom the initial insult represents a deep composite injury repeated failed attempts at salvage are not in the patient’s ultimate best interest. Such wounds often become infected and tissue that could have been preserved now must in the end be sacrificed with the functional end result less than that which would have occurred with early amputation.

Excision and grafting will be required for wounds not amenable to primary closure. The extent of the procedure that a patient can undergo is related to the patient’s age, physiologic status and skill of the operating team. A 17% surface area burn should be a universally survivable injury in a seventeen year old patient while in a patient in the eighth decade of life the mortality can easily be 50%. An otherwise healthy individual with available donor sites can well tolerate a 20-25% total body burn excision and autografting in one procedure. Implicit in this approach is the use of experienced operating teams, an anesthesiologist who thoroughly understands the unique problems of the patient with a major body surface area burn, and an operating room fully equipped to treat such a patient, as well as ready availability of blood products and the capacity to care for the patient post-operatively. A patient having this extent of surgery in essence undergoes a doubling of
the surface area of "injury"-the now excised and grafted wound along with the partial thickness wound produced by the donor site. In patients with wounds of a larger size (>30% TBSA) or those who cannot tolerate a single procedure to achieve closure, staged excision of burned tissue is performed and the resulting wounds are closed with available cutaneous autografts or a biologic dressing.99

The technique of burn wound excision is based on the depth of the wound and anatomic site to be excised. Excision of deep partial thickness wounds to the level of a uniformly viable bed of deep dermis, by the tangential technic pioneered by Janzekovic, and immediate coverage with cutaneous autograft results in rapid wound closure with a typically excellent result.100 This can be done with an unguarded Weck knife, a Goulian guarded Weck knife, a hand held dermatome or by using a powered dermatome set at .0016-.0030 of an inch depending on the area to be excised and the age and gender of the patient. Optimally, the desired wound bed is achieved in one pass of the knife as evidenced by diffuse bleeding. If that end point is not realized another pass of the knife will be needed. A frequent error is attempting this technique in wounds of an inappropriate depth and assuming that punctuate bleeding indicates a viable bed. Such wounds will heal with a poor take of the grafted skin as the bed contains marginally viable tissue incapable of supporting the cutaneous autograft. These wounds at the initial graft dressing change may appear to be doing well only to fail at 5-10 days post-operatively. Tangential excision as originally reported was employed early in the first week post-burn, however it can be successfully applied any time to a wound that is not infected or heavily colonized. During the performance of this procedure the amount of blood loss can be minimized with the use of a tourniquet on extremity burns or sub-eschar clysis containing epinephrine. The decision that the depth of the excision is satisfactory with these adjuncts will be based primarily on the appearance of the wound, an appreciation of which most experienced burn surgeons have had to learn to some degree through trial and error.

A modification of tangential excision is wound excision via layered escharectomy. Using this technique the wound is sequentially excised to a viable bed of subcutaneous tissue and elements of deep dermis particularly at the wound margin. This allows relative preservation of body part contour, a graft with ultimately more pliability, decreased limb edema and a cosmetically more acceptable transition at the juncture of the grafted wound with the unburned skin of the wound margins.
An alternative to layered excision is to excise the wound with a scalpel or electrocautery. Using knife excision the wound is excised to the muscle fascia or to viable deep subcutaneous tissue. Bleeding can be significant with such procedures, therefore the excision and control of bleeding must be done efficiently. The use of electrocautery to perform the dissection limits the blood loss without compromising the recipient graft site. Imperative with electrocautery excision into the deep fat is avoidance and limitation of thermal injury to the wound bed, which will compromise the "take" of the applied skin graft. The use of the cutting mode with rapid dissection is necessary. In cases where excision to fascia has been performed the viability of the fascia should be assessed. The surgeon must determine if the fascia requires removal and the underlying muscle used as the graft bed. In the performance of fascial excisions caution should be exercised during the dissection to avoid entrance into a joint or bursa and injury of extensor tendons in the hand or the Achilles tendon at the ankle.

The blood loss occurring with burn wound excision is related to the time of excision post burn, the area to be excised, the presence of infection, and type of excision, i.e. fascial or tangential. Donor sites can also represent a significant portion of the blood loss. The use of the scalp or previously harvested donor sites is associated with increased bleeding. The quantity of blood loss has been estimated to range from 0.45 to 1.25-ml/cm² burn area excised. Adjunctive measures that can be used to control blood loss include elevation of limbs undergoing excision, applications of topical thrombin and/or vasoconstrictive agents in solutions to the excised wound and donor site, clysis of skin graft harvest sites and/or the eschar prior to removal, and application of tourniquets. Spray application of fibrin sealant can also reduce bleeding from the excised wound after release of the tourniquet. Blood loss will be compounded if the patient has become coagulopathic, hypothermic or acidotic during the procedure. Perioperative cold stress, which may induce hypothermia, can be reduced by maintaining the temperature of the operating room between 30 and 32 degrees centigrade and by using warmed fluids for wound irrigation. The harvest, application, and postoperative care of split thickness skin grafts and skin graft donor sites are the same as for any other surgical patient.

Grafting of the burn wound is usually done at the time of excision. However, there are instances where it advisable to stage the skin grafting procedure. The surgeon must be aware of the patient's status throughout the surgical procedure and if necessary reassess the extent of the planned procedure. It may be best to perform the excision only and stage the timing of skin graft
application. Additionally, if the wound bed is suspect as to its viability then only excision should be performed. The wound can be dressed with a 5% sulfafmylon solution dressing or covered with allograft skin or any of several biologic dressings and subsequently reevaluated. The use of cutaneous allografts is a very useful approach when excising facial burns where the goal is to preserve all possible elements and perform the definitive grafting procedure on a "tested" recipient bed. In cases where an infected wound is being excised no attempt at placing autograft skin should be considered until the infection has been resolved following treatment with topical and systemic antimicrobial agents as determined by culture results and inspection of the wound.

The choice of the donor site in the performance of a cutaneous autograft will in some patients be limited to those skin sites that have not been injured with burns. When there is a choice of donor sites the requirements of the recipient site and the potential for donor site morbidity should be factored into selecting the site of graft harvest. In the grafting of facial burns color match is an important consideration and obtaining a graft from a site above the clavicles or the inner aspect of the thigh will provide the best result. In children harvest of a graft from the scalp results in a donor site that is not particularly painful postoperatively and has no long-term cosmetic consequences. The harvest of grafts from posterior body surfaces provides, in general, a more acceptable wound for most patients. While the anterior thigh is an often-selected site it can heal with significant hypertrophic change and cause a patient more problems and distress than the grafted burn.

The use of sheets of autograft skin for resurfacing the burn represents the gold standard. This is the only acceptable approach for burns of the face and neck and the best choice in grafting of the hands and breast. Every attempt should be made to use such autografts in children, since they provide the best long-term results. It may not be possible to achieve these objectives in patients with extensive burns or those in whom the pattern and location of the injury limits donor site availability. The use of meshed cutaneous autografts allows the surgeon to increase the area covered. Skin graft meshing devices of various design and manufacture are available with expansion ratios from 1:1 to 1:9. The wider the mesh the greater the wound area covered, however, it will take the wound longer to close by in-growth from the margins of the mesh reticulum to fill the open interstices during which time there is the very real potential for graft loss and wound infection to occur. Additionally, widely meshed autografts have a greater propensity to form hypertrophic burn scars, and may provide a skin surface with unsatisfactory mechanical stability, inadequate pliability, permanently poor cosmetic appearance, and restricted joint mobility. Despite these potential limitations, the use of
meshed cutaneous autografts is an important strategy and potentially life saving approach in patients with extensive body surface area burns.

The technique of skin graft harvesting would seem a relatively simple procedure yet it is often not done well. As noted above the harvest site should be the one that will yield a graft with the desirable characteristics and the least donor site morbidity. Grafts should be of sufficient size to achieve wound closure with a minimum of inter-graft seams. Powered dermatomes are available with up to six inch cutting widths that provide excellent sheets of skin for facial grafts or when meshed can cover a significant burn area. Donor site preparation is essential to obtain a uniform graft. Powered clysis can rapidly be accomplished over an extensive harvest site using an air powered surgical wound irrigating system equipped with a 14 or 16 gauge needle attached to three liter bags of normal saline. This provides a stable uniform surface for graft harvest and limits the difficulties encountered when harvesting over contoured surfaces or bony prominences. The thickness of the harvested graft should be related to the site to be grafted, whether the graft is to be meshed and the mesh ratio, and to some degree surgeon preference. The desired thickness of the graft also influences donor site selection, i.e., a “thick” graft should be harvested from an area of “thick” skin. Harvest of a “thick” graft from an area of “thin” skin, i.e. the inner arm, can produce a full thickness wound which will have to be grafted.

Skin grafts through which one can read printed material are primarily epithelial autografts with a minimal amount of dermis (.004-.006 inch) while those, which are more opaque contain a variably greater amount of dermis (.008-.012 inch). Thinner grafts yield a better donor site and function well on a dermal wound bed but may not do well when placed on a wound excised to fascia. In elderly patients thin grafts which contain insufficient numbers of keratinocyte progenitor cells are considered the cause of melting graft syndrome and prolong the time of re-epithelization. Thicker grafts are more pliable, heal with less contraction, and will do better than thin grafts when meshed. The thicker grafts may result in donor site scarring and delay in donor site closure especially in the elderly patient.

The harvested graft should be placed on the prepared burn wound parallel to the major flexion creases and can be attached mechanically with staples or sutures or secured with tissue adhesives such as fibrin glue. A properly placed set of grafts on an extremity should at the end of the operation be able to remain in place as the extremity is put through a gentle range of motion. One
of the most important aspects of a skin grafting procedure is the application of a proper dressing. A highly successful approach is to use multiple layers of a non-adherent linen dressing moistened with a 5% solution of mafenide acetate applied circumferentially to the excised and grafted wounds on an extremity. A bolster produced by using net dressings drawn tightly over the burn dressings and stapled to the skin is used to “fix” the grafts on torso wounds. Graft failure occurs as a result of inadequate excision, inadequate hemostasis, infection, subgraft seroma formation, mechanical sheering during post-operative care, or rarely, “upside down” application. The first dressing change is typically done 48-72 hours postoperatively. If a sheet graft is well intact at that time a non-adherent dressing is reapplied to protect the wound. In the case of meshed autografts, the moist dressings of mafenide acetate solution, changed daily or more often as required, are continued until the mesh is closed.

Skin Substitutes: While split thickness cutaneous autografts are the usual method of wound closure there is often the need for a skin substitute. Alternative wound coverings are used to achieve wound closure when the available donor surface area is not sufficient, there is a need to test the wound bed, or for primary management of selected partial thickness wounds. The goal with a skin substitute is to obtain temporary physiologic wound closure and protect the wound from bacterial invasion. The two most commonly used naturally occurring biologic dressings are human cutaneous allograft and porcine cutaneous xenograft. Human allograft skin is commercially available as split-thickness grafts in either fresh viable or cryopreserved form. Both of these preparations are capable of becoming vascularized however this best occurs with fresh allograft skin. Allograft skin can provide wound coverage for three to four weeks before rejection. Xenograft tissue is available as reconstituted sheets of meshed porcine dermis or as fresh or prepared split-thickness skin. Porcine skin impregnated with silver ions to suppress wound colonization is also available. Xenograft skin can be used to cover partial thickness injuries or donor sites, which re-epithelialize beneath the xenograft.

Various synthetic membranes have been developed that provide wound protection and possess vapor and bacterial barrier properties. Biobrane™ (Dow-Hickham, Sugarland, Texas), is one such product which has been used in the management of partial thickness and donor site wounds. This bilaminate membrane consists of a collagen gel adherent to a nylon mesh as the dermal analog to promote fibrovascular ingrowth and a thin outer silastic film as the epidermal analog to provide barrier properties. Biobrane has also been used as the scaffold for the growth of allogenic
fibroblasts that secrete, while in culture, various growth factors along with other mediators. The fibroblasts are then removed by freezing to complete preparation of the membrane. These membranes are currently approved for use in fully excised wounds, donor sites, and superficial partial thickness burns. Another collagen-based skin substitute is the dermal replacement developed by Burke and Yannas, presently in use as Integra™ (Integra LifeScience Corporation, Plainsboro NJ). This membrane consists of an inner layer of collagen fibrils with added glycosaminoglycan and an outer barrier membrane of polysiloxane. It is placed over freshly excised full-thickness wounds and once fully vascularized the epidermal analog is removed and the vascularized “neodermis” covered with a thin split thickness cutaneous autograft. A permanent skin substitute for burn care victims represents the search for the Holy Grail. Presently, cultured epithelial autografts are commercially available but are limited in their use because of suboptimal graft take, fragility of the skin surface, and high cost.

Use of any biologic dressing requires that the excised wound and the dressing that has been applied be meticulously examined on at least a daily basis. Submembrane suppuration or the development of infection necessitates removal of the dressing, cleansing of the wound with a surgical detergent disinfectant solution, and even re-excision of the wound if residual nonviable or infected tissue is present. Following such wound care, the biologic dressing can be reapplied and if it remains adherent and intact for 48-72 hours without suppuration, that biologic dressing can be removed and the wound closed definitively with cutaneous autografts.

The proper management of the patient’s burn wounds is critical to achieve the optimum cosmetic and functional outcome and the timely return of the patient to full activity. In patients with major burns the wound must be properly cared for and closure achieved expeditiously to lessen the level of physiologic disruption that accompanies a major burn. Failure to do so can result in invasive wound infection, chronic inflammation, erosion of lean body mass, progressive functional deficits and even death.

The Treatment of Special Thermal Injuries

Electric Injury

The principal mechanism by which electricity damages tissue is by conversion to thermal energy. Currents of 1000 volts and above are classified as high voltage. Upon contact with such currents, the body acts as a volume conductor with small differences in conductivity among tissues of little
consequence. At contact points where current density is greatest, tissue charring may limit current
flow but at very high voltages current flow persists until the contact is interrupted. The electric
current may induce cardiac and/or respiratory arrest necessitating cardiopulmonary resuscitation at
the site of injury, on the way to the hospital, and in the emergency department. Arrhythmias may
also occur after admission to the hospital necessitating EKG monitoring for at least 24 hours after
the last recorded episode of arrhythmia.

Two characteristics of high voltage electric injury increase the incidence of acute renal failure in
patients with such injury. First there may be only a small charred contact site evident with
extensive inapparent subcutaneous tissue injury in a limb underlying unburned skin. The limited
cutaneous injury may lead to gross underestimation of resuscitation fluid needs. Secondly, the
mass of muscle injured by the electric current may liberate large amounts of hemochromogens that
may damage the renal tubules. In addition to the electric injury, arcing of the current may ignite
the patient's clothing to cause conventional thermal burns in addition to the electric injury.

Resuscitation fluids should be based on the extent of burn visible plus the estimated daily needs of
the patient and adjusted according to the patient's response. If the urine contains hemochromogens
(dark red pigments) fluid should be administered to obtain 75-100 ml of urine per hour with
sodium bicarbonate added to the fluids to alkalinize the urine. If the hemochromogens do not clear
promptly, or the patient remains oliguric, 25 grams of mannitol should be given as a bolus and 12.5
grams of mannitol added to each liter of lactated Ringers until the pigment clears. After
administration of mannitol urinary output is no longer an index of resuscitation adequacy and other
indices of physiologic well being must be monitored to assess resuscitation status.

When the body functions as a volume conductor, current flow is proportional to the cross sectional
area of the body part involved. Consequently, severe tissue destruction may occur in a limb with
relatively small cross section area and relatively little tissue damage occur as current flows through
the trunk. With cessation of current flow body parts that acted as volume conductors now act as
volume radiators with periosseous tissue deep in the limbs being exposed to higher temperatures
for longer periods of time. Damage to the muscle in a limb is often associated with marked
increase in the pressure within the compartment containing the damaged muscle which, if
unrelieved, may cause further tissue necrosis. A limb muscle compartment which is stony hard to
palpation should alert one to the need for surgical exploration. Evidence of extensive deep tissue
necrosis, development of a compartment syndrome, and persistent or progressively severe
hyperkalemia mandate operative intervention. At the time of exploration, the investing fascia is widely opened and the muscles of the involved limb thoroughly examined including the periosseous muscles of the limb which can be necrotic yet overlain by more superficial viable muscle (Fig.8). The extent of destruction may necessitate amputation at the time of exploration, particularly if the nonviable muscle is the source of persistent hyperkalemia. If the extent of muscle necrosis in a limb is indefinite, an arteriogram may be helpful. The identification of "pruning" of the intramuscular branches of the arterial tree identifies injured muscle and defines the level of amputation required to encompass the nonviable tissue.109 Following debridement or amputation the wound should be dressed open. The patient is returned to the operating room in 24-36 hours for reinspection and further debridement of nonviable tissue if necessary. When all tissue in the wound is viable it may be closed definitively.

A detailed neurologic examination must be performed on all patients with high voltage electric injury at the time of admission and at scheduled 24-48 hour intervals thereafter. Signs of peripheral nerve and central nervous system impairment may be evident immediately after injury or may appear later. It is uncommon for a nerve directly injured by electric current to regain function. The immediate functional impairment caused by nondestructive injury to which motor nerves are more sensitive than sensory nerves commonly resolves. Late occurring parasthesias and other polyneuritic symptoms causing deficits in the function of peripheral nerves remote from the sites of electric contact have been attributed to electroporation, the cellular effects of millivoltage electric fields.110 Immediate neurologic impairments caused by direct nerve damage of the spinal cord more commonly resolve than do spinal cord deficits of later onset. Delayed onset spinal cord dysfunction can range from quadriplegia to localized nerve deficits with signs of ascending paralysis and even an amyotrophic lateral sclerosis-like syndrome.111

Remote organ injury is rare in patients with high voltage injury, but intestinal perforation, gall bladder necrosis, and direct liver injury have all been reported. Delayed hemorrhage from medium to large sized vessels has been attributed to electric injury induced arteritis but inadequate debridement of injured tissue or transmural necrosis of the vessel wall as a consequence of exposure and dessication appear to be more likely causes.

The formation of cataracts has also been associated with high voltage electric injury, particularly in those patients with a contact site on the head or neck. The patient should be informed that such
cataracts may occur, commonly three or more years after the injury, but often much sooner. In patients with a head contact site exfoliative debris may be evident in the anterior chamber of the eye immediately after injury. Such debris is slowly cleared and typically requires no specific treatment.

Tissue damage can also be caused by low voltage house current. Burns of the oral commissure occur in young children who bite electric cords or suck on the end of a live extension cord or an electric outlet. The lesion may have the characteristics of full thickness tissue damage but early surgical debridement may only accentuate the defect and should be avoided. These injuries will usually heal with minimal cosmetic sequelae which can be addressed electively if needed. In the course of spontaneous healing, labial artery bleeding may occur. The parents should be warned that such bleeding, which can be impressive, may occur and instructed in how to apply manual pressure for temporary control until the vessel can be ligated.

Cardiopulmonary arrest is particularly common in patients struck by lightning and necessitates the immediate institution of cardiopulmonary resuscitation. Subsequent ECG abnormalities are uncommon and signs of acute myocardial damage, though rare, may become evident later. Coma immediately following injury is common, but typically transient. Keraunoparalysis (lightning paralysis) characterized by parathesias and paralysis typically involving the lower limbs may develop over several days after lightning injury in association with vasomotor disorders. This paralysis typically resolves without residual deficit. Lightning injury of the skin is generally superficial with a “splashed on” arborescent and spidery appearance. The tip-toe sign refers to the small, circular full thickness burns on the tips of the toes that are common in patients with lightning injury. Prompt institution of cardiopulmonary resuscitation has increased the survival rate of patients struck by lightning to 70% and improved management of the systemic effects of lightning injury has reduced the incidence of acute renal failure and other complications.

Chemical Injuries
A variety of chemical agents can cause tissue injury as a consequence of an exothermic chemical reaction, protein coagulation, dessication, and delipidation. The severity of a chemical injury is related to the concentration and amount of chemical agent and the duration with which it is in contact with tissue. Consequently, initial wound care to remove or dilute the offending agent takes priority in the management of patients with chemical injuries (Fig.9). Immediate copious
water lavage should be instituted while all clothing, including gloves, shoes, and underwear exposed to the chemical are being removed. The lavage is continued for at least 30 minutes or until dilution has lowered the concentration of the agent below that which will cause tissue damage or until testing the involved surface with litmus paper confirms that the agent has been removed. For patients in whom extensive surface injury has occurred, the irrigation fluid should be warmed to prevent the induction of hypothermia. Although seldom needed, if a patient with concentrated alkali injuries requires prolonged irrigation and is hemodynamically stable he can be cared for while sitting in a chair under a shower.

The appearance of skin damaged by chemical agents can be misleading. In the case of patients injured by strong acids, the involved skin surface may have a silky texture and a light brown appearance which may be mistaken for a sunburn rather than the full thickness injury that it is. Skin injured by delipidation caused by petroleum distillates may be dry, show little if any inflammation, and appear to be undamaged but found to be a full thickness injury on histologic examination.

Variable degrees of pulmonary insufficiency may occur in patients with cutaneous injuries caused by volatile chemical agents which can also be inhaled, such as anhydrous ammonia, the ignition products of white phosphorus, mustard gas and chlorine, and even the vapors of strong acids. Additionally, pulmonary insufficiency may be caused by the inhalation of the gaseous products of petroleum distillates as may occur in patients who sustain delipidation injuries due to partial immersion in gasoline and other petroleum products.

In the case of patients with anhydrous ammonia injury, any powdery condensate adherent to the skin should be brushed off prior to irrigation. Hydrofluoric (HF) acid injury is most common in those involved in etching processes, the cleaning of air conditioning equipment, patio grills and other metallic objects with spray products containing HF, and petroleum refining. After contact with hydrofluoric acid there is a characteristic pain-free interval of variable duration with subsequent appearance of focal pallor which progresses to penetrating necrosis, typically accompanied by severe pain. Immediately after injury, calcium gluconate gel should be applied topically, or prolonged irrigation with a solution of benzalkonium chloride instituted. The persistent severe pain that may occur in digits injured by hydrofluoric acid can be relieved by injecting 10% calcium gluconate into the artery supplying that finger. Local tissue injection of
calcium gluconate is an alternate route of delivery but may in itself compromise the blood supply of the involved digit. Persistent pain caused by subungual HF is best treated by removal of the nail under digital block anaesthesia. The pain typically relents and the nail grows back with little or no deformity. If these measures fail to control pain, local excision and skin grafting will be needed to remove the damaged tissue and achieve pain relief. Extensive hydrofluoric acid injury may induce systemic hypocalcemia which is treated by intravenous infusion of calcium.

Burns caused by phenol should be treated with immediate water lavage to remove, by physical means, the liquid phenol on the cutaneous surface. Following that lavage, the involved area should be washed with a lipophilic solvent such as polyethylene glycol to remove any residual adherent phenol which is only slightly soluble in water. Intensive systemic support is required for patients with extensive phenol burns, in whom absorption of the agent can cause central nervous system depression, hypothermia, hypotension, intravascular hemolysis, and even death.

Injuries caused by white phosphorous are usually discussed with other chemical injuries but are actually conventional thermal burns caused by the ignition of the particulate phosphorus. These injuries are most commonly encountered in military personnel injured by explosive antipersonnel devices (grenades) that may cause mechanical tissue damage and drive fragments of white phosphorus into the soft tissues. All wounds containing white phosphorus particles should be covered with a wet dressing which is kept moist to prevent ignition of the particles by exposure to air. If the interval between injury and definitive wound care will be so long as to permit dessication of the wet dressings, the wounds can be briefly washed with a freshly mixed dilute 0.5 - 1% solution of copper sulfate followed by copious rinsing. Such treatment generates a blue-gray cupric phosphide coating on the retained phosphorus particles which both impedes ignition and facilitates identification. Whatever form of topical treatment is employed, the wound should be debrided and all retained phosphorus particles, which can be readily identified with an ultraviolet lamp, removed. The removed particles should be placed under water to prevent them from igniting and causing a fire in the operating room.

Strong acids and alkali can cause devastating ocular injuries and must be treated immediately, even before leaving the scene of the injury, by irrigation with water, saline, or phosphate buffer. In the hospital eye irrigation must continue until the pH of the eye surface returns to normal. The rapid penetration of ocular tissue by strong alkalis necessitates prolonged irrigation (12-72 hours). Such
irrigation is best carried out with a modified scleral contact lens with an irrigating side arm. The effects of iritis induced by chemical ocular injury are minimized by installation of a cycloplegic such as 1% atropine following irrigation. If irrigation and removal of the offending agent is delayed, the entire globe may be so damaged as to lose turgor and all visual function. Even with early irrigation, corneal damage can be severe and late complications of symblepharon and xerophthalmia may occur. An ophthalmologist should be involved in the care of such patients from the time of admission.

**Bitumen Burns:** Bitumen injuries are commonly caused by hot tar coming in contact with the skin. The injury that results is a thermal contact burn which is not associated with any significant component of a chemically mediated injury. There is no significant absorption of materials unless the patient is in an explosion and has ingested or inhaled the material. The primary initial treatment is urgent cooling of the molten material with no attempt made to remove the tar. By cold application the transfer of heat can be limited and the degree of tissue damage minimized. There are various agents that have been advertised as being effective for the removal of tar and asphalt products. These have varied from mayonnaise to simple petroleum-based jellies and seem to be similar in terms of efficacy. Considering that the initial temperature of liquid tars and asphalts are typically in excess of 600 degrees Fahrenheit early concerns about infection would seem to be unfounded and offer no support for urgent removal with potential destructive consequences to underlying otherwise viable tissue. It is preferable to apply an emulsifying petroleum based ointment and allow the tar to separate during the first day or two after admission.¹¹⁴

**Cold Injuries:** Injuries occurring secondary to environmental exposure can result in local injuries, frostbite, or systemic hypothermia. During the wintertime in urban environments, the most common mechanism of injury involves homeless persons or an elderly patient who has become disoriented and wandered from home. The pathophysiology of the local injuries consists essentially of crystal formation due to freezing of both extracellular and intracellular fluids. Consequently, the cells dehydrate and shrink and blood flow is altered to the exposed area resulting in tissue death. During the thawing of damaged tissues, micro emboli that have formed further occlude the microvascular circulation adding insult to injury.¹¹⁸ It is important to note that the initial clinical presentation of the patient is not likely representative of the ultimate degree of tissue loss. Patients presenting with frostbite will have coldness of the injured body part with loss of sensation and proprioception. On initial exam, the limb may well appear pale, cyanotic or have a yellow white
discoloration. During rapid rewarming at 40-42°C in water for 15-30 minutes, hyperemia will occur followed by pain, paresthesias and sensory deficits. Over the subsequent 24 hours, edema and blistering will develop and it may be the better part of a week before one can determine the true depth and extent of the injury. In the initial management of the patient, re-warming is critical but it must be done only when there is no chance for an episode of re-freezing. If blisters appear whether they should be preserved or debrided has proponents on both sides of the answer. Some authors suggest that white blisters can be debrided while purplish blue blisters should be left intact. The injured extremity should be elevated in an attempt to control edema and padded to avoid pressure-induced ischemia as a secondary insult. Administration of pain medication is based on the patient’s response. Frostbite wounds are tetanus prone wounds and therefore tetanus toxoid should be administered based on the patient’s immunization status.

Before any definitive plans are made for surgical intervention sufficient time should be allowed to pass so that a clear demarcation between viable and nonviable tissue is apparent (Fig.10). However, it is not in the patient’s best interest to follow the adage of “freeze in January and amputate in June”. While it will take some time for definitive delineation of the depth of the injury, once the wounds have begun to mummify the thought that there will be tissue salvage seems more than naive. Patients suffering frostbite injuries should be evaluated for other potential trauma and treated for systemic hypothermia if it is present. The post-hospitalization disposition of cold injury patients requires a clear understanding of their preexisting health status and the factors that predisposed them to injury such as dementia or major psychological disease.

Radiation Injury: Radiation exposure secondary to the detonation of a thermonuclear device is not as likely as is exposure from an industrial or medical accident, misuse of radiation materials, or acts of terrorism. The dispersal of radioactive substances can take several forms including accidents during storage and mishandling, accidents during transportation of radioactive materials, intentional dispersal either alone or in combination with other agents, and intentional dispersal through an explosive device. In both storage and transport accidents, the dispersal and subsequent exposure to radioactive materials is usually limited to the people immediately involved and is well contained geographically once the event is recognized. It is typically difficult to expose large numbers of individuals to significant doses of radiation at any given time and the risks are limited to those involved in a given incident. Small dose radiation exposure does not affect health for many years and is associated with few acute problems although it is still a significant health risk.
In the event of intentional radiation dispersal, the risk of exposure and injury as well as the source involved need to be evaluated. The risk of trauma is related to the primary explosive device itself as well as trauma related to the secondary effects of the explosion such as shell fragments, structure collapse, or injury from debris. Psychological trauma due to either patients witnessing the primary event or the experience of living through the event with the associated physical manifestations may pose a further problem in the handling of a significant number of injured victims.

Exposure risk is related to primary contamination from the particles released from the explosive device, secondary contamination from particles that have become mixed with debris, debris dust and fallout, and tertiary contamination from exposure to particles in contact with patients. Ionizing radiation is composed of two types: radiation that has mass and that which is energy only. Exposure to alpha particles which are relatively large, highly charged particles, slow moving and penetrate only a few microns into tissue can be effectively shielded with ordinary substances such as paper, cardboard or clothing. Alpha particles can be a source of secondary and tertiary contamination. Beta particles made up of either positively or negatively charged species have greater energy and can penetrate more deeply into tissues and require shielding with material such as aluminum to prevent exposure. Both alpha and beta particles result from the decay of a radioactive source. Gamma and x-rays are produced by radioactive decay or an x-ray source; they have neither mass nor charge however they penetrate deeply and shielding requires the use of such materials as lead, steel or thick cement. Following removal from the source of radiation no further exposure occurs and the patient poses no danger to those providing care. Radiation due to neutrons requires special consideration. Nuclear reactors are the major source of neutron emission and create radiation that penetrates deeply causing widespread damage to underlying tissues.

Radiation exposure of two to four gray (Gy) can cause nausea and vomiting, hair loss and bone marrow injury leading to death from infection up to two months after exposure. Exposures of six to ten gray result in the destruction of the bone marrow, and injury to the gastrointestinal tract with a mortality approaching 50% within one month. When the exposure is ten to twenty Gy there is severe injury to the gastrointestinal tract and death may occur in as little as two weeks. When exposure is greater than thirty gray, cardiovascular and nervous system damage occur primarily as a result of hypotension and cerebral edema. There is almost immediate nausea, vomiting, prostration, hypotension, ataxia and convulsion and death can occur in a matter of hours. At present there appears to be no effective treatment following radiation exposure. For treatment to
be effective, it would need to be given prior to the exposure. In cases of accidental exposure, treating bone marrow suppression while successful has not prevented death, which usually occurs from radiation pneumonitis, GI tract injury and hepatic and renal failure.121, 122

The burn injuries resulting from radiation exposure are usually localized and represent a high radiation dose to the skin. They appear identical to a thermal burn and may present with erythema as with a first-degree burn which will heal following some sloughing of the skin. With higher dose exposures, blisters may occur as with a partial thickness burn and healing occurs in a similar manner. When the radiation exposure has been significant such as twenty gray, radionecrosis occurs. If the event leading to the radiation exposure causes surface contamination, decontamination needs to be done prior to dealing with the wound. This consists of saline irrigation of the wound and treatment with standard aseptic techniques. It is not necessary to excise the wound urgently unless it is contaminated with long-life radionuclides such as alpha emitting particles. Patients who have greater than a one gray whole body exposure should be considered for early wound closure so that the wound itself does not become the site of a lethal infection.123

To manage radiation-exposed victims effectively a hospital must have a well-organized plan in place and the appropriate decontamination facility within the emergency room. The goals are to save the patients life and to prevent further injury. The decontamination must be done so that the personnel providing care to the patient do not become exposed. All contaminated materials must be carefully handled to prevent contamination of the hospital and its facilities and the public sewage system.

Toxic Epidermal Necrolysis (TEN): TEN is a rare life threatening mucocutaneous form of exfoliative dermatitis that is often secondary to drug sensitivity. The incidence of TEN has been estimated at 0.4-1.2 cases per million population per year.124 These patients may give a history of sore throat, burning eyes, fever, and malaise and present with systemic toxicity. Physical findings can include rash, bullae, and diffuse exfoliation with the large areas of separation having the appearance of a partial thickness burn. When lateral stress is applied to the involved skin it separates at the dermal-epidermal junction, Nikolsky’s sign. The resulting wounds give the appearance of a wet surface as seen in a second-degree burn. The mechanism of injury is thought to be keratinocyte apoptosis induced by interactions between the cell surface death receptor Fas
and its receptor FasL or CD95L. Lyle in 1956 was the first to describes two entities in the initial description of toxic epidermal necrolysis consisting of staphylococcal scalded skin syndrome (SSS) and what today is recognized as TEN. Staphylococcal scalded skin syndrome is a generalized exfoliative dermatitis due to infections with staphylococcal organisms. In SSS the lesion is at the intra-epidermal layer with blister formation followed by desquamation of large sheets of skin with relatively rapid re-epithelialization over 7-10 days. The outcome in patients with SSS is significantly better than that in TEN patients. In TEN there is necrosis of all layers of the skin and a mortality between 30 and 40% while with SSS it is 3-4%. Stevens Johnson Syndrome (SJS) is an entity in which there is also extensive epidermolysis often presenting with target shaped skin lesions with differentiation from TEN related to the extent of cutaneous involvement. One current delineation classifies patients with less than ten to thirty percent cutaneous involvement as SJS and those with greater than ten to thirty percent as TEN particularly if it involves oral-genital and ocular mucosa. Whether SJS and TEN represent the same process differing only in the extent of cutaneous involvement and sites affected or are pathologically distinct entities has not been answered with any degree of certainty.

Patients with TEN have wound care needs identical to those of patients with extensive second-degree wounds. They exhibit significant fluid losses and have specialized nutritional needs. Care of these patients in a Burn Center by experienced surgeons has resulted in a significant improvement in outcome. General principles of management in these patients include the cessation of potential precipitating drugs, the discontinuance of systemic steroids if recently initiated, ophthalmologic evaluation, and skin biopsy confirmation of the diagnosis. Additionally, systemic antibiotics should be reserved for those cases in which infection is highly likely. Replacement of fluid and electrolytes and provision of nutritional support and aggressive wound care are critical elements in the care of these patients. Wound care may consist of the application of a biologic dressing once all of the nonviable tissue is fully debrided or the use of silver impregnated dressings (Fig.1). The most frequent mistakes in the care and management of these patients are underestimating the extent of the cutaneous involvement, airway compromise, and not understanding how rapidly these patients can become critically ill. To date, the results of studies of various modalities that can be employed to control the degree of skin slough have been too inconsistent to recommend their general use.
Mechanical Injury: The combination of burn injury and multi-system trauma occurs in up to 4 - 5% of all burn patients. Patients suffering combination injuries are typically male with their injuries having occurred from a flame ignition during an assault or motor vehicle crash. Victims suffering a combination of burns and trauma tend to have a higher incidence of inhalation injury, higher mortality, higher Injury Severity Score and longer length of stay despite no differences in total body surface area burned when compared to patients with only burns. Trauma victims with burns with an inhalation injury have a near three-fold increase in their mortality rate. Those victims not surviving their injuries typically are significantly older, have a higher ISS and a larger body surface area burn compared to trauma victims with burns who survive their injuries. The management priorities in patients suffering burns plus trauma must be as for patients with trauma. Understanding the mechanism of injury is vital in determining the probability of associated injuries and provides a guide in the work-up of the patient. A formal trauma evaluation should be performed on all burn victims when the history of the event points to the possibility of combined mechanisms of injury.

Life-threatening injuries must be promptly treated, fractures immobilized, and the resuscitation fluid needs of the patient should be calculated to include the burn wound mandated needs and those of the associated trauma. Blood is not part of the initial resuscitation for patients with only burn injuries but when there is multiple trauma blood transfusions may be necessary in the early management of the patient. Often the presence of a major burn wound results in the patient being viewed as having only a burn and the standard assessment of a trauma patient is not done. Patients with impaired neurological status should undergo a computerized axial tomographic scan to rule out intracranial pathology along with evaluation for a spinal injury. This is particularly important if the patient jumped from a burning building to escape the fire, was injured in an industrial accident, or involved in a motor vehicle crash. Potential thoracic, abdominal or pelvic injuries should be evaluated with chest, abdominal and pelvic roentgenograms as well as with abdominal CT and FAST examinations. Diagnostic peritoneal lavage may also be used in the unstable patient to verify the presence of an injury requiring exploratory laparotomy. The nonoperative management of significant injuries of the spleen or liver requires thoughtful consideration in patients with a major burn and it maybe prudent to opt for surgical management particularly if the abdominal wall is extensively burned. In patients with major long bone injuries, early operative intervention with stabilization will facilitate the patient’s overall management as well as that of the
burn. In selected circumstances, early burn excision with skin graft wound closure may be the best approach to facilitate the operative management of the orthopedic injury.

The management of patients with significant burn injuries in conjunction with mechanical trauma requires a highly coordinated plan of care. The patient must be continuously reassessed to avoid missing an injury and the surgeon vigilant to the development of trauma related complications.

Metabolic and Nutritional Support

Estimation and Measurement of Metabolic Rate

Burn injury alters central and peripheral thermoregulatory mechanisms, the predominant route of heat loss, the distribution and utilization of nutrients, and metabolic rate. All of these postburn metabolic changes must be considered in planning the metabolic support and nutritional management of the hypermetabolic burn patient necessary to minimize loss of lean body mass, accelerate convalescence, and restore physical abilities. Metabolic support includes patient care procedures and environmental manipulations in addition to the provision of adequate nutrition.

The perceived temperature of comfort of burn patients (on average 30.4°C) is higher than that of unburned control patients and necessitates maintaining the ambient temperature at that level in the patient’s room to prevent the imposition of added cold stress which would exaggerate an already elevated metabolic rate. Physical therapy with active motion to the extent possible and passive motion to stretch muscles in the absence of spontaneous motion is instituted on admission to minimize muscle wasting secondary to disuse. Analgesic and anxiolytic agents should be used as needed to prevent pain and anxiety-related increases in circulating catecholamine levels which can further increase metabolic rate. Assiduous monitoring is necessary to facilitate early diagnosis and prompt treatment of infections and thereby reduce their metabolic impact. The importance of excision and grafting of the burn wound, has been emphasized by recent studies showing that such treatment reduces resting energy expenditure in burn patients, even if the entire wound cannot be excised and grafted at a single sitting.

Even though metabolic rate can be reduced by pharmacologic means, studies indicating that the hypermetabolic response to burn injury is wound directed speak for meeting caloric needs rather than reducing nutrient supply to the burn wound by pharmacologic intervention. One must determine the resting energy expenditure in order to calculate the nutrients required to meet the
Bedside indirect calorimetry is the most accurate means of determining metabolic rate, but a bedside metabolic cart may not always be available. A number of formulas permit one to make close approximations of daily energy expenditure in a variety of surgical patients. A formula based on studies of extensively burned patients is useful in estimating burn patient calorie needs.\(^{134}\)

\[
EER = [BMR \times (0.89142 + 10.01335 \times TBS)] \times m^2 \times 24 \times AF
\]

- **EER** = estimated energy requirements
- **BMR** = basal metabolic rate
- **TBS** = total burn size
- \(m^2\) = total body surface area in square meters
- **AF** = activity factor of 1.25 for burns

A rule of thumb estimate for nutritional needs of patients whose burns involve more than 30% of the body surface is 2000-2200 kilocalories and 12-18 grams of nitrogen per square meter of body surface per day.\(^{39}\)

**Nutritional Support**: Meeting the metabolic needs of the burn patient can be accomplished by providing nutritional support via the gastrointestinal tract or by the intravenous route. After determining what the metabolic needs will be for an individual burn patient the next question is will the patient be capable of meeting the needs by oral intake? In patients who can eat it is not likely that a standard hospital diet will meet the calculated needs and it is often necessary to supplement the patient’s intake with various nutritional supplements. A calorie count should be recorded to verify that the patient is capable of consistently meeting the daily nutrient intake goal. In the patient who is incapable of achieving the necessary nutrient intake or cannot eat, one must decide how to deliver the feedings. Total parenteral nutrition in the past provided a way by which patients could receive the majority or all of their calorie and protein needs but at present has largely been supplanted by the use of enteral nutritional support. As compared to total parenteral nutrition, enteral nutritional support is technically easier to accomplish, lower in cost, supports the health of the gastrointestinal tract, and ameliorates the systemic inflammatory response syndrome.\(^{135, 136, 137, 138, 139}\)

At the time of admission, a patient who will require specialized nutritional support should have either a nasogastric or nasoduodenal tube placed. Patients can safely and effectively be fed by
either of these routes with appropriate precautions. It is not required that one use custom made feedings to meet the patient’s nutrient needs. It is possible by using combinations of currently available commercial products to obtain the necessary blend of nutrients, feeding density, water, and protein requirements while avoiding the cost of compounding specialized enteral feedings. It is preferable to start enteral feedings soon after the patient is admitted. The patient should be fed with the head of the bed elevated to 30° with feeding residuals checked frequently to avoid gastric distention and possible aspiration. A potential advantage of early enteral feedings is modulation of the hypermetabolic response although the actual ability of early feedings to achieve this goal has been called into question. \(^{140,141,142}\) When feedings are initiated early post-injury the desired rate of administration can typically be reached within twenty-four to forty-eight hours of admission. There are multiple recommendations regarding the initial concentration, rate, incremental increase and the frequency of the increases. Starting a tube feeding of standard concentration at twenty to forty ml per hour and advancing the rate a similar amount every four hours works well in most patients. The most important issues are that the nursing staff understands the goals, knows how to monitor for feeding intolerance, and appreciates the attention to detail necessary to achieve consistent delivery of the feedings.

If a patient is intolerant of gastric feedings and gastric aspirate volume exceeds the total of two hourly feedings, the administration of metaclopramide will often resolve the problem. If the patients fails to respond to metaclopramide an attempt should be made to place either a nasoduodenal or nasojejunal feeding tube, which will minimize this feeding difficulty and lessen the risk of aspiration. Patients who become septic will often manifest changes in feeding tolerance along with new onset hyperglycemia or changes in insulin needs as early signs pointing to this problem. In patients receiving central vein alimentation the risk of catheter sepsis must be evaluated as an etiology for the patient’s septic process. In patients who become intolerant of enteral feedings or develop gastrointestinal complications that prevent use of the gastrointestinal tract, total parenteral nutrition will be required. However, with careful attention to detail and a well designed, patient specific enteral feeding protocol, this should rarely be needed in the care of a burn patient.

**Monitoring:** The complications associated with the use of enteral or parenteral support in the burn patient are in large part similar. Burn injury induces insulin resistance which may lead to hyperglycemia. The maintenance of blood glucose values with aggressive insulin replacement has
a favorable impact on the outcome of critically ill patients. In critically ill patients the preferable route of administration of insulin is intravenously with the goal of maintaining plasma glucose values between 80-110 mg/dl. There is a well-recognized limit to the caloric load that a critically ill patient can tolerate from carbohydrates and for the 70-kilogram patient this is approximately 1800 kilocalories per day from glucose. Excessive amounts of glucose can result in RQ values >1 which may cause hepatic steatosis and complicate ventilatory management.

Sufficient protein to meet metabolic demands must be provided. To estimate protein needs twenty-four hour urine urea nitrogen is measured to which an additional 0.1-0.2-gm of nitrogen per % TBSA burn remaining is added. These determinations can be done on a weekly basis unless there is a special need to perform them more frequently. Numerous studies have been done to determine precise protein needs and the optimum balance of protein to nonprotein calories. In adult patients 1.5-2.0 g of protein per kg lean body mass per day is a reasonable goal and for children 3g of protein per kg lean body mass. A nonprotein calorie to nitrogen ratio of 100:1 provides the patient with sufficient calories to support protein synthesis in the face of ongoing protein breakdown and reduces net protein loss. The provision of dietary protein at these levels has been shown to positively impact patient outcome. An increasing blood urea nitrogen level must be evaluated in terms of nitrogen over feeding and the protein load recalculated to avoid uremia and an associated diuresis. Measurements of visceral proteins such as serum transferrin and albumin can be used to monitor the impact of the nitrogen content of the diet on the patient’s nutritional status. Those proteins are simply markers that can be followed over time and are probably best utilized in a trend analysis based on weekly determinations since albumin has a half-life of twenty days and transferrin eight days. Thyroid pre-albumin with a half-life of two days and retinal binding protein with a twelve hour half-life can be used to track short-term responses in selected patients.

To prevent the development of essential fatty acid deficiencies, lipids must be included in the diet but should not exceed more than 40% of the total calorie load or more than 3 grams per kg BW per day. Most enteral diets will contain adequate fat to prevent the development of essential fatty acid deficiency and parenteral diet formulations typically contain long chain fatty acids. The serum triglyceride concentration and the triene/tetraene ratio should be measured weekly to assess fatty acid status. If that ratio is greater than 0.4 an essential fatty acid deficiency exists which
necessitates adjustment of the dietary fat content. Supplemental medium chain triglycerides can be given enterally but are associated with increased ketone production and may cause diarrhea.

Complications: Serum electrolytes must be monitored to make necessary adjustments in the amount of free water, sodium, chloride, potassium, phosphorus, calcium and magnesium provided to the patient. Laboratory values should be obtained at initiation of the feedings and daily during the stabilization phase and with each change in the patient's clinical status. During the first several days after admission, and with the initiation of nutritional support, there can be dramatic shifts in serum and plasma values of electrolytes and minerals. As noted above, hypernatremia can develop if free water replacement is insufficient to account for insensible water loss through the burn wound, which can be 2.0-3.1 ml/kg body weight/% burn/day. Hypernatremia can also develop with persistent febrile episodes if free water replacement does not match the patient’s needs. Hyponatremia may represent under replacement of sodium but typically is related to free water excess. Correction of hyponatremia should be attempted with restriction of free water intake. In adults an increase in body weight of more than 400 grams per day reflects water loading and should prompt a review of fluid intake and output records and adjustment of fluid administration.

Potassium and phosphorous must be given to meet the patient’s needs which often exceed initial estimates particularly when large loads of glucose are being given along with exogenous insulin.

In the course of the patient’s care as the open wound area decreases and the hypermetabolic state slowly resolves, the nutrient load should be adjusted so that balance is maintained between metabolic needs and substrates delivered and the patient is not overfed. Alternatively, if a patient is found to have lost more than 10% of his/her admission weight, it is likely that caloric estimates are not being achieved or were under estimated. While most experienced clinicians possess the skill to assess patient needs accurately the performance of bedside indirect calorimetry can provide objective information as to the patient’s resting energy expenditure, respiratory quotient, oxygen consumption and carbon dioxide production. The results may indicate the need to adjust the total calorie load if the resting energy expenditure has been under-estimated or modify the fuel substrate load if the respiratory quotient is approaching or greater than one.

The patient should receive increased amounts of vitamin C, at recommended doses of a gram per day in adults and 500 mg. per day in children, which will aid in wound healing. In patients with
burns of greater than 20% of the total body surface area zinct at doses of 220 mg/day will support wound healing as well as white cell function. The routine provision of these nutrients avoids complications related to insufficient delivery and obviates the need to measure their levels in the patient.

In patients with prior surgery or preexisting medical conditions special attention may be required to monitor for feeding intolerance and to insure that adequate amounts of iron, folate and vitamin B12 are being effectively delivered. In patients who have received extended courses of broad-spectrum antibiotics vitamin K replacement beyond standard recommendations may be required to avoid the development of nutritionally related coagulopathy. The preservation of lean body mass requires more than just the appropriate amounts and blend of nutrients. Physical activity is important in directing the nutrients to muscle and reducing truncal fat deposition and the risk of hepatic steatosis.

In addition to providing appropriate calorie, protein and nutrient loads to burn patients, it is now possible to modulate the metabolic response. Administration of beta antagonists in children has been shown to be safe and have a significant positive effect on outcome. The administration of growth hormone, which is depressed following burn injuries, has met with variable results. Herndon et al have reported a positive effect in burned children given growth hormone but a recent multi-center trial from Europe in critically ill patients showed an increased mortality in treated patients. An alternative strategy that seems not to be associated with problems in adults and is efficacious in children is the use of the drug oxandrolone, although a recent study reported that the agent was associated with prolonged need for mechanical ventilation in trauma patients. Additional strategies that might be utilized are the provision of selected nutrients in increased amounts. Glutamine, arginine, nucleotides and omega-3 fatty acids have all been used in attempts to improve immune function above that seen with the optimal use of standard nutritional formulations. The routine use of these measures requires a full understanding of the therapeutic benefits and the potential adverse consequences of each. Additionally, some studies have found such supplements to be ineffective.

In patients who have established chronic renal failure or develop renal insufficiency during their course of care, changes in the nutritional formulation will have to be made to accommodate their altered clinical status. In patients who require dialysis, the frequency of dialysis should be adjusted so that the protein intake needed to meet metabolic needs can be given. In patients with significant
injuries who are receiving large amounts of feeding through the gastrointestinal tract, the health of the GI tract itself must be continuously monitored. The development of major gastrointestinal complications while not common can adversely impact the patient’s outcome. Complications can include ischemic necrotic bowel disease, intestinal obstruction, the development of clostridium difficile colitis and non-infectious diarrhea. The patient’s clinical status should be continuously monitored and any changes in abdominal findings on physical examination should be aggressively followed up with appropriate diagnostic radiographic studies, endoscopy, stool cultures and abdominal exploration before the patient deteriorates and develops an irreversible condition.

TRANSPORTATION AND TRANSFER
Many important advances have been made in the care and management of burn injured victims during the past 50 years. One of the more significant advances has been the recognition of the benefits of a team approach in the care of critically injured burn patients. The American College of Surgeons and the American Burn Association have developed optimal standards for providing burn care and a burn center verification program that identifies those units that have undergone peer review of their performance and outcomes. Patients with burns and/or associated injuries and conditions listed in Table II should be referred to a burn center.

Once the decision has been made to transfer a patient to a Burn Center, there should be physician-to-physician communication regarding the patient’s status and need for transfer. Institutions should have pre-existing inter-hospital transfer policies in place to facilitate communication and patient transfers. It is critical that the patient be properly stabilized in preparation for the transfer. The flight transfer team should have the capability of providing the care required for a critically injured, severely burned patient throughout the entire transfer procedure. A surgeon, a respiratory therapist, and a licensed practical nurse, all experienced in burn care comprise such a team for long distance, fixed wing aircraft transfers. For short distance transport by rotary wing aircraft, inclusion of a burn physician in the flight team optimizes the safety and quality of care of extensively burned patients, but patients with lesser burns may be adequately cared for by non-physician helicopter flight team members (a flight nurse and/or an advanced paramedic) who are in ready contact with medical control. A flight team roster should be maintained and published so the surgeons and other members of the team will be available when needed. Physicians and other team members should be assigned to the flight (transfer) team only after six to twelve months experience
at a burn center which will enable them to become familiar with the complications that occur in burn patients during resuscitation and develop competence in the prevention, treatment, and resolution of those problems.

During transport the need to perform life saving interventions such as endotracheal intubation or reestablishing vascular access may be very difficult to accomplish in the relatively unstable and limited space of a moving ambulance or a helicopter in flight (Fig.12). That difficulty makes it important to institute hemodynamic and pulmonary resuscitation and achieve “stability” prior to undertaking transfer by either aeromedical or ground transport. A secure large-bore intravenous cannula must be in place to permit continuous fluid resuscitation. Patients should be placed on 100% oxygen if there is any suspicion of carbon monoxide exposure. If there is any question about airway adequacy an endotracheal tube should be placed and mechanical ventilation instituted before transfer begins. In-flight mechanical ventilatory support can be provided by a transport ventilator with oxygen supplied from a lightweight kevlar tank transported in back-pack fashion by the respiratory therapist. Patient safety during transport may necessitate chemical paralysis of the patient to prevent loss of the airway or vascular access. In-transit monitoring for helicopter transfer includes pulse rate, blood pressure, EKG, pulse oximetry, end tidal CO₂ levels, and respiratory rate. For long distance transfer, the same physiologic indices should be monitored. In addition, the ultrasonic flow meter should be used to assess the presence and quality of pulsatile flow in all four limbs on a scheduled basis and excursion of the chest wall should be monitored to identify a need for limb or chest escharotomy respectively. The hourly urinary output should also be monitored with fluid infusion adjusted as necessary. All patients should be placed NPO and those with a greater than 20% body surface area burn require placement of a nasogastric tube. In essence, a mini ICU should be established for the duration of the long distance flight.

The burn wound should be covered with a clean and/or sterile dry sheet. The application of topical antimicrobial agents is not necessary prior to transfer, since they will have to be removed on admission to the burn center. Maintenance of the patient’s body temperature is vital. Wet dressings which can lead to hypothermia, particularly in small adults and children, should be avoided. The patient should be covered with a heat reflective space blanket to minimize heat loss. Pain medication is given in sufficient dosage to control the patient’s pain during transport while avoiding respiratory depression, airway compromise or hypotension. Burn wounds, as tetanus prone wounds, mandate immunization in accordance with the recommendations of the American College
of Surgeons. As in the case of the transfer of any trauma victim, documentation must be thorough, flow sheets should be clearly marked, and a listing of all medications, including IV fluids that have been given must be provided to the receiving physician. In the case of a patient suffering from significant multi-system trauma and burn injuries, it may be necessary to treat the patient’s life threatening mechanical injury prior to transfer if the transport time will be of long duration or the patient is unstable.  

Survival Data

During the course of the past half century, early postburn renal failure as a consequence of delayed and/or inadequate resuscitation, has been eliminated and inhalation injury as a co-morbid factor has been tamed. Invasive burn wound sepsis has been controlled and early excision with prompt skin grafting and general improvements in critical care have reduced the incidence of infection, eliminated many previously life threatening complications, and accelerated the convalescence of burn patients. All of these improvements have significantly reduced the mortality of burn patients of all ages. At the mid-point of the last century, a burn of 43% of the total body surface would have caused the death of 50 of 100 young adult patients (15-40 years) with such burns. Since that time, the extent of burn causing such 50% mortality (the LA₅₀) in 21-year-old patients has increased to 82% of the total body surface and in 40-year-old patients to 72% of the total body surface. In children (0-14 years) the LA₅₀ has increased from 51% of the total body surface in the 1950s to 72% today, and in the elderly (>40 years) the LA₅₀ has increased from 23% of the total body surface area to 46% (Table V). Not only has survival improved, but the elimination of many life threatening complications and advances in wound care have improved the quality of life of even those patients who have survived extensive severe thermal injuries.
Legends

Figure 1: The burns on this abused child show the typical distribution (feet, legs, posterior thighs, buttocks, and genitalia) of injury caused by intentional immersion scalding. Note the pink to red color and moist surface of the partial thickness injuries on the proximal legs and distal thighs. The pallor, hemorrhagic discoloration, and eschar caseation of the full thickness injuries of the feet and distal legs are characteristic of third degree burns which required skin grafting for closure.

Figure 2: Diagram of the skin, adnexa, and subcutaneous tissue showing, by stippled shading, the depth of tissue injury that defines both first degree and second degree partial thickness burns and third degree full thickness burns.

Figure 3: Example of a form used for documenting extent of burn. Figure outlines are filled in with a blue pencil and a red pencil to indicate distribution of partial thickness and full thickness burns respectively. Note the columns indicating how the percentage of total body surface area represented by body part surface changes with time.

Figure 4: The dashed lines indicate the preferred sites of escharotomy incisions for the limbs (mid-lateral and mid-medial lines), thorax (anterior axillary lines and costal margin), and neck (lateral aspect). The thickened areas of the lines on the limbs emphasize the importance of carrying the incisions across involved joints.

Figure 5: Endoscopic view of a bronchus in a patient with severe inhalation injury. Note bright white areas reflecting light from edematous mucosa, erythema and focal ulceration of other areas of the mucosa and the extensive black carbonaceous material from the smoke deposited on the endobronchial surface.

Figure 6: Microbial control can be achieved in partial thickness burn wounds by application of a polyethylene mesh coated with a nanocrystalline film of pure silver ions as shown here. When in contact with wound fluids, the silver is released continuously to limit bacterial proliferation on and in the wound.

Figure 7: A topical antimicrobial cream is applied to the entirety of the burn wound as shown here after the daily wound cleansing and inspection procedure. The topical agent is reapplied 12 hours later to maintain microbial control.

Figure 8: High voltage electric injury caused the edema evident in the muscles bulging above the edges of the fasciotomy incision on the forearm and dorsum of the hand. Note the focal areas of charring on the exposed muscle (white arrows), the deep injury and fixed flexion of the thumb, index finger, and long finger, and the burn in the anticubital area caused by arcing (black arrow).

Figure 9: Failure to remove footwear and institute water lavage to dilute and remove concentrated lye, which had spilled into the boot of this patient, resulted in severe tissue injury during transportation to the hospital. Note extensive liquefaction of tissue, thrombosed vessels
(white arrow), and edema of the extensor tendons exposed at the mid-metatarsal level on the dorso-medial aspect of the foot (black arrow).

**Figure 10:** Spontaneous healing of frostbite injury proximal to the discolored skin on the dorsum of this foot is indexed by decreased hair growth in that area. The demarcation of nonviable tissue shown here permitted amputation at a mid-foot level and salvaged the heel pad.

**Figure 11:** The back, buttocks, and upper thighs of this patient with toxic epidermal necrolysis (TEN) have been covered with a translucent collagen-based skin substitute, Biobrane® following cleansing with saline and gentle debridement of exfoliated epidermis. Note focal areas of adherent darkly pigmented epidermis which were left in place and covered with the bilaminate membrane which provides barrier function, reduces pain, and prevents dessication of the exposed dermal surface to promote healing. The undressed wounds of the arms and legs were covered with Biobrane® after this photo was obtained.

**Figure 12:** The transfer of patients to burn centers is often done by helicopter as shown here. Note the shiny metallic inner surface (black arrow) of the “space blanket” in which the patient has been wrapped to conserve heat and prevent excessive cooling during transport. The burn surgeon and burn nurse, sitting adjacent to the patient, monitor urinary output and, as needed, adjust the rate of infusion of the fluids suspended above the patient. The vibration, noise, poor light, and limited space which conspire to make monitoring and therapeutic intervention difficult, mandate preflight physiologic stabilization of each patient who is to be transferred.