LECTURE 6

BURNS.CLASSIFICATION.ELECTRICAL BURNS. FROST BITE.

I. Actuality of theme. The incidence of burn injury has declined steadily over the past several decades in developed countries. According to the American Burn Association, each year in the United States, 1.1 million burn injuries require medical attention.
Approximately 50,000 of these require hospitalization, and roughly half of those burn patients are admitted to a specialized burn unit.
Each year, approximately 4,500 of these people die.
Up to 10,000 people in the United States die every year of burn-related infections; pneumonia is the most common infectious complication among hospitalized burn patients. Twenty years ago, burns covering half the body were routinely fatal; today, patients with burns covering 90 percent of the body can survive (but often with permanent impairments).
Practices that have contributed to this improvement include advances in resuscitation, wound cleaning and follow-up care, nutritional support, and infection control. Seventy-five percent of those hospitalized have burns covering less than 10 percent of the body-surface area. Such burns rarely cause hemodynamic problems or death except in the elderly or in those with smoke inhalation. But these common small injuries cause major, persistent morbidity because burn injury to the reticular dermis is associated with the development of unsightly, restrictive, and uncomfortable hypertrophic scars. The lack of understanding of the biologic basis of these scars and therefore of their effective prophylaxis or treatment is a major unresolved problem.

II. Aims of lecture :
Educational:
- To describe the history of treatments of burns (β=I);
- To expound the epidemiological, demographic and outcome characteristics of burn injury,classification of burns (β=II);
- To describe pathophysiology of burn shock (β=II);
- To give the sudents knoweledge about fluid resuscitation and early management the burns (β=II);
- To substantiate the treatment of infection in burns (β=II);
- To characterize the operative burn wounds management (β=II);
- To expound pathophysiology , diagnostic and treatment of electrical injuries and frostbites (β=II);
- To study the students the main principles of evidence-based medicine according the subject of lecture (β= IV).

Educative:
1. To form for students skills of clinical thought in the process of intercourse with the patients. To teach students to adhere to principles of medical

deontology and bioethics in the process of socializing with a patient, his relatives, and also with colleagues.

III. Plan and organization of structure of lecture

№	Basic stages of lecture and their maintenance	Aims are in the levels of abstraction	Type of lecture, methods and facilities of activation of students, equipment	Division of time
1	Preliminary stage. Determination of educational aims and motivation.		Items I, II	5%
2	**Basic stage. Teaching of lecture's material** History of Treatments of Burns. Epidemiological, Demographic and Outcome Characteristics of Burn Injury.Classification of Burns Pathophysiology of Burn Shock and Burn Edema. Fluid Resuscitation and Early Management the Burn. Evaluation of the Burn Wound: Management Decisions Treatment of Infection in Burns Operative Burn Wounds Management Electrical Injuries Frostbite	I II II II II II II II II	Type of lecture – thematic (with controversial elements – critical analysis of results of meta-analyses, randomized controlled, trials, guidlines which are devoted for the problem of burns and cold injuries). Facilities of activation of students are a questions, controversial situations, illustrative material	85%

3	Final stage (resume of lecture, general conclusions, answers to the possible questions, task for students for preparation for practical classes)		List of literature, question, task for students	10%

IV. Subject of a lecture

Burn is a type of injury to the skin caused by heat, cold, electricity, chemicals, or radiation

Classification of Burns

Superficial Partial Thickness:

- Are very painful with redness of the skin and blister formation
- Usually heals satisfactorily with little or no scarring.

Deep Partial Thickness:

- Contact with hot liquids, flames or chemicals.
- Severe pain, skin discolouration and blister formation.
- Produces moist and mottled skin.
- Heals slowly with scarring may need skin grafts.

Full Thickness:

- Direct contact with flames or hot liquids
- Produces white, leathery, charred and dry skin.
- Destroys hair follicles, blood vessels and nerve endings.
- Causes tissue coagulation with little or no pain.
- Will require a skin graft.
- Cosmetic and functional impairment.

Beyond simple erythema, burns are either partial or full thickness depending on whether the basement membrane has been lost. On examination a full thickness (3rd degree) burn is usually pale, bloodless and insensitive to the firm touch of a sterile needle. Partial thickness burns can be further divided into superficial (1st degree) and deep (2nd degree), which refers to the depth at which the dermal layer is injured. Sensation is preserved and healing of the skin more likely.Fourth-degree burns are devastating full-thickness burns that extend into muscle and bone.

Table 1. A description of the traditional and current classifications of burns

Nomenclature	Traditional nomenclature	Depth	Clinical findings
Superficial thickness	First-degree	Epidermis involvement	Erythema, minor pain, lack of blisters
Partial thickness	Second-degree	Superficial	Blisters, clear fluid, and pain

— superficial		(papillary) dermis	
Partial thickness — deep	Second-degree	Deep (reticular) dermis	Whiter appearance, with decreased pain. Difficult to distinguish from full thickness
Full thickness	Third- or fourth-degree	Dermis and underlying tissue and possibly fascia, bone, or muscle	Hard, leather-like eschar, purple fluid, no sensation (insensate)

Definition of a Major Burn

A major burn can defined as any burn that requires intravenous fluid resuscitation (10% Body Surface Area (BSA) in a child, 15% in an adult) and / or a burn to the airway.

The mechanism of the burn can be classified into six categories

Contact - direct contact with a hot surface.
Scald - hot fluid/gas usually causing a superficial burn.
Flash - a brief burn, usually partial thickness.
Flame - usually full thickness.
Chemical
Electrical
Burn Pathology

The classic description of the burn wound and surrounding tissues is a system of several circumferential zones radiating from primarily burned tissues, as follows:

Zone of coagulation - A nonviable area of tissue at the epicenter of the burn

Zone of ischemia or stasis - Surrounding tissues (both deep and peripheral) to the coagulated areas, which are not devitalized initially but, due to microvascular insult, can progress irreversibly to necrosis over several days if not resuscitated properly

Zone of hyperemia - Peripheral tissues that undergo vasodilatory changes due to neighboring inflammatory mediator release but are not injured thermally and remain viable

The tissues in ischemic areas can potentially be salvaged by proper resuscitation in the initial stages and by proper burn wound excision and antimicrobial therapy in the convalescent period. Underresuscitation can convert this area into deep dermal or full-thickness burns in areas not initially injured to that extent. Reevaluation of these threatened areas over the first several days is used to determine when the first burn excision should be performed (ie, when the depth of burn has become

apparent and decisions about which areas are deep dermal or of full thickness are clear).

The severity of the burn is also judged by the amount of body surface area (BSA) involved. Health care workers use the **"rule of nines"** to determine the percentage of BSA affected in patients more than 9 years old: each arm with its hand is 9% of BSA; each leg with its foot is 18%; the front of the torso is 18%; the back of the torso, including the buttocks, is 18%; the head and neck are 9%; and the genital area (perineum) is 1%. This rule cannot be applied to a young child's body proportions, so BSA is estimated using the palm of the patient's hand as a measure of 1% area.

Pathophysiology of burn

The underlying process involved is both a local and systemic inflammatory reaction, the end result of which is an almost immediate shift of intravascular fluid into the surrounding interstitial space. This occurs as a consequence of changes in vascular permeability as the normal capillary barrier is disrupted by a host of mediators, including histamine, serotonin, prostaglandins, platelet products, complement components, and members of the kinin family. This process occurs in burned tissues and, to lesser extent, in unburned tissues. The margination of neutrophils, macrophages, and lymphocytes into these areas is associated with the release of a rich milieu of these mediators, which affect both local and systemic capillary permeability.

Local

Within moments the capillaries of the injured tissue become leaky. Plasma is lost, drawing water with it. This continues for between 3 and 36 hours and results in oedema of the tissues involved. Local airway swelling may lead to loss of the airway by both internal and external oedema. Chest wall oedema may make ventilation difficult and oedema of the limbs may cause ischaemia leading to limb loss (especially if the burn is circumferential).

Hypovolaemia and haemoconcentration of the blood leads to a rising haematocrit which will result in poor systemic tissue perfusion. This is **'Burns Shock'**. Red blood cells are lost both directly in the burn and as a result of increased fragility.

Systemic

Damaged tissue will release 'middle molecules' (leukotrienes, prostaglandins, oxygen free radicals and histamine) into the circulation leading to a systemic increase of capillary permeability.

Burn Shock is defined as the inability of the circulation to meet the needs of tissues for oxygen and nutrients and the removal of the metabolites. The clinical picture of severe shock consists of pale cold skin and a rapid yet thready pulse. Respiration is rapid and shallow leading to gasping ('air hunger'). Urine output falls

and the patient becomes increasingly restless and disorientated. Often consciousness is lost only as a preterminal event.

Pathophysiology

After a burn, fluid accumulates rapidly in the wound and, to a lesser extent, in unburned tissues. If the burns involve at least 15 to 20 percent of the body-surface area, hypovolemic shock will develop unless there is effective and rapid intervention. Edema formation is most rapid in the first 6 to 8 hours after injury but continues for 18 to 24 hours. Inflammatory mediators are elaborated locally in part from activated platelets, macrophages, and leukocytes and contribute to local and systemic hyperpermeability of the microcirculation. Histologically, gaps appear in the venular and capillary endothelium; the wound capillary-protein reflection coefficient falls by 50 percent or more. Regional blood flow increases and is accompanied by an early increase in capillary pressure. Physicochemical alterations in the extravascular, extracellular matrix, such as degradation and unraveling of the collagen triple helix due to its partial denaturation from loss of cross-linking, may further increase wound edema by forming osmotic and hydrostatic gradients. Erythrocytes also are invariably extravasated, but substantial early loss of blood is rare, and transfusion is not required or desirable.

Major burns precipitate a systemic inflammatory response that if overly prolonged or exaggerated can lead to organ dysfunction, sepsis, or both. The local mediators listed appear within minutes to hours after the injury. Among the systemic mediators, plasma levels of interleukin-1, 2, and 8 are elevated very early. Increases in the level of interleukin-6 occur with sepsis. Transient elevations of tumor necrosis factor are associated with a poor prognosis. Interferon- levels tend to peak about 10 days after the burn. Many factors in addition to the extent of the burn, especially age, determine the severity of injury.

Management Principles

The severity of the burn will determine not only the type of treatment, but also where the burn patient should receive treatment. Minor burns may be treated at home or in a doctor's office. These are defined as first- or second-degree burns covering less than 15% of an adult's body or less than 10% of a child's body, or a third-degree burn on less than 2% BSA. Moderate burns should be treated at a hospital. These are defined as first- or second-degree burns covering 15%-25% of an adult's body or 10%-20% of a child's body, or a third-degree burn on 2%-10% BSA. Critical, or major, burns are the most serious and should be treated in a specialized burn unit of a hospital. These are defined as first- or second-degree burns covering more than 25% of an adult's body or more than 20% of a child's body, or a third-degree burn on more than 10% BSA. In addition, burns involving the hands, feet, face, eyes, ears, or genitals are considered critical. Other factors influence the level of treatment needed, including associated injuries such as bone

fractures and smoke inhalation, presence of a chronic disease, or a history of being abused. Also, children and the elderly are more vulnerable to complications from burn injuries and require more intensive care.

First aid

Remove the casualty from further injury. Extinguish flames, remove clothing, turn off the electrical source, or douse the chemically burnt patient with water. Flames ascend so lie the patient down. Cover the burn with a clean dressing, avoid the patient getting cold and transfer to a hospital as soon as possible. Additional oxygen should be given during transfer.

Primary management

Airway - check the airway is clear. Endotracheal intubation is necessary if there are deep burns to the face and neck, soot in the nostrils, burns of the tongue and pharynx, stridor or hoarseness.

History including time and nature of the incident (Wet or dry burn/chemical/electrical/inside or outside).

Weigh the patient.

Examine the burn and assess the size with the 'rule of nines' to give a %BSA.

Intravenous access - obtain large bore venous access, even through burnt tissue.

Blood sampling -samples for haematocrit, electrolytes, crossmatch, arterial blood gases and carboxyhaemoglobin levels.

Analgesia - intravenous morphine, ketamine.

Catheterise - assess urine output as a gauge of tissue perfusion and adequate resuscitation.

Reassess the patient thoroughly at regular intervals and also the burn.

Fluid resuscitation

This should be instituted as soon as possible. There are two simple protocols that both depend upon the %BSA, time passed since injury and patients weight. The rule of nines may over-estimate the BSA, but the Lund and Browder chart gives a more accurate assessment. Fluid requirements may be greater than the protocols suggest.

Parklands: Crystalloid resuscitation with Hartmanns

24 hour fluid requirement = 4 x %BSA x Wt (Kg)

Give half over the first 8 hours, and the remainder over the next 16 hours

Although there may be pronounced generalised oedema initially, as large volumes are required, it is cheap and produces less respiratory problems later on.

Muir and Barclay: Colloid resuscitation with plasma

The first 36 hours are divided into time periods of 4,4,4,6,6,12 hour intervals

Each interval = 0.5 x %BSA x Wt (Kg)

With colloid resuscitation, less volume is required and the blood pressure is better supported. However they are expensive, often unavailable and tend to leak out of the circulation and may result in later oedema especially in the lungs.

Controversy remains as to which fluid should be used. Inhalational injury may increase fluid requirements by 50%. Both regimes require regular assessment as to the adequacy of resuscitation. This includes blood pressure, pulse, capillary return, urine output, level of consciousness and haematocrit. Additional fluid should be given if resuscitation is inadequate.

Water loss is related to evaporative and other extrarenal losses and may lead to a hypernatraemia. Salt intake should be balanced against the plasma sodium concentration, but is usually about 0.5mmol/kg/%BSA. If the burn is left exposed in an hot environment, sodium free water intake must be increased, but only to achieve a moderate hypernatraemia. Aggressive water load may lead to a low plasma sodium and result in 'burn encephalopathy'. Hyperkalaemia usually associated with severe muscle damage may require correction with insulin and dextrose.
Due to the high morbidity associated with high-volume resuscitations, an interest exists in using various colloid solutions to both decrease edema and volume requirements and blunt the myocardial depression phenomena observed with large burns. An important consideration for adding colloid in the first 24 hours is the loss of capillary integrity during early burn shock. This process occurs early and is present for 8-24 hours depending on which authority is referenced. A strategy for testing whether the capillary leak has begun to resolve involves substituting an equal volume of albumin solution for RL solution. An increase in urine output suggests that at least some of the leak has resolved and that the further introduction of colloid can help decrease the fluid load.

Albumin is the plasma protein that most contributes to intravascular oncotic pressure. When administered intravenously as a 5% solution from pooled plasma product, approximately half the volume remains intravascularly, as opposed to 20-30% of crystalloid solutions. Alternatively, some centers prefer using fresh frozen plasma over using albumin because of the theoretic advantage of replacing the whole range of plasma proteins that are lost rather than just the albumin fraction. Guidelines for this infusion have been reported as 0.5-1 mL/kg per percentage burn during the first 24 hours, beginning 8-10 hours postburn as an adjuvant to RL solution resuscitation.

Dextran is a solution of polymerized, high molecular weight glucose chains with almost twice the oncotic pressure of albumin. An increase in microcirculatory flow is also produced by reducing erythrocyte aggregation. Proponents of dextran point to the reduction of edema in nonburned tissues as justification for its use. The edema-reducing properties are maintained for as long as the infusion is continued, but upon withdrawal and subsequent metabolism of the glucose, rapid loss of fluid occurs back into the interstitium if the capillary leak is still present. Demling and others have used dextran 40 successfully in the early postburn period (first 8 h) at 2 mL/kg/h along with RL solution before switching to some albumin or fresh frozen plasma plus RL solution combination for the second 18-hour phase.

Hypertonic saline solutions, ranging in concentration from 180-300 mEq sodium per liter, have many theoretic benefits. These benefits are achieved by the reduction in volume requirements by mobilizing intracellular fluid into the vascular space by the increased osmotic gradient. The intracellular depletion of water that results is a debated concern, but it appears to be well tolerated. Close monitoring of serum sodium levels is mandatory, and serum sodium levels should not be allowed to increase to greater than 160 mEq/dL.

As a compromise strategy to limit the risk of hypernatremia and sodium retention, some institutions use RL solution with 50 mEq amps of sodium bicarbonate per bag, for a fluid approaching 180 mEq sodium per liter during the initial 8 hours of the resuscitation, rather than using the more concentrated saline solutions. Then, after the first 8 hours, the fluid is changed to RL solution to complete the resuscitation. Hypertonic saline management must be titrated closely to both urine output and serum sodium checks and probably should not be used routinely outside of tertiary burn centers.

The safety and benefits of hypertonic saline resuscitation extend to both the pediatric and geriatric populations, but using solutions at the lower end of tonicity is probably safer. The greatest benefit may ultimately be for those patients with the most limited cardiopulmonary reserves, those with inhalation injury, and those with larger burns approaching 40% or more.

Exactly when or whether to add colloid to resuscitation fluids is a confusing issue. As mentioned previously, most of the mainstream burn formulas add colloid during the resuscitation, at least in the second 24-hour period. However, what must be recognized is that despite a general consensus that colloid use is both beneficial and appropriate, especially in burns greater than 40% TBSA, demonstrating improved outcomes in morbidity or mortality has been difficult. In fact, some studies have demonstrated harmful effects secondary to increased pulmonary edema and some evidence of renal dysfunction as manifested by a decreased

glomerular filtration rate. For smaller burns (ie, 20-40% without inhalation injury), expectant management with RL solution titrated to urine output is a safe and well-tested strategy.

The patients who benefit the most from lower-volume resuscitations aided by colloid are those with larger burns (>40%), those with preexisting heart disease, geriatric patients, and those with burns with associated inhalation injuries
At 24-30 hours after the insult, the patient should be resuscitated adequately, with near complete resolution of the transcapillary leak with fluid requirements. At this point, some authorities recommend a change in fluid management from RL solution to a combination fluid infusion involving albumin and D5W. The rational for this is the massive protein losses that have occurred from the burn wound during the first 24 hours. Replacing this deficit with a steady infusion of 5% or 25% albumin solution can serve to maintain a serum albumin concentration greater than 2, which can help reduce tissue edema and improve gut function. Associated insensible losses of free water from the injured skin barrier can be met by replacing the deficit with an electrolyte-free fluid such as D5W solution, which also serves to restore the extracellular space to an isotonic state, especially if hypertonic solutions were used during the resuscitation.

<u>The formula for the estimate for 5% albumin infusion is as follows:</u>
0.5 mL/kg per percentage burn = mL albumin for 24 hours
<u>The formula for the free water estimate is as follows:</u>
(25 + percentage burn) X BSA (m2) = mL/h of free water required

There is no evidence from randomised controlled trials that resuscitation with colloids reduces the risk of death, compared to resuscitation with crystalloids, in patients with trauma, burns or following surgery. As colloids are not associated with an improvement in survival, and as they are more expensive than crystalloids, it is hard to see how their continued use in these patients can be justified outside the context of randomised controlled trials.

<u>Airway management</u>
A high index of suspicion is required regarding the patient's airway. Laryngeal oedema develops from direct thermal injury leading to early loss of the airway. With signs of an airway burn (soot in the nostrils/stridor/hoarse voice) consider early intubation of the patient. If in doubt it is better to protect the airway (and be able to provide tracheo-bronchial toileting) than to risk losing the airway altogether. A tracheostomy may be necessary if there is any delay in securing the 'at risk' airway.

The airway is further endangered by an associated loss of respiratory drive due to a depressed level of consciousness (eg head injury or carbon monoxide poisoning). Again intubation may be required.

Burn wound management

Dressings are necessary to reduce infection and adsorb exudate. Bactericidal agents, such as silver sulphadiazine 1% and silver nitrate are used. Antibiotic preparations should be avoided to prevent resistant colonisation developing. Regular, often daily, dressing changes are recommended, and the patient should be washed with clean warm water.

Surgery

Circumferential burns will require immediate surgery to improve circulation to distal extremities or to permit adequate breathing if the chest wall is burnt. Early excision and grafting is preferred as it minimises infection and hastens wound healing. The whole burn should be excised with in 48 hours. Regular dressing changes, further excision and grafting may be required. It should be remembered that blood loss may be excessive at these times. Blood loss can be reduced by using diathermy and/or applying gauze soaked in adrenaline (1: 200,000) during the burn excision.

The problem with excision of a large burn is often the lack of donor skin to cover the excised burn. The patient's donor skin can be meshed, so as to increase the size. It can then be covered with cadaveric skin which acts as a biological dressing with growth stimulating properties. Artificial bovine skin (such as *Integra*®) may also be used but are expensive.

Full-thickness burns — those covering about 25 percent of the body-surface area or less in young or previously healthy patients — have a low mortality rate. Such injuries should be treated as soon after resuscitation as is feasible by excision of the eschar and skin grafting. There is no benefit in delay. Sufficient autologous skin grafts should be available to close the wounds at the same operation, particularly if the grafts are meshed and expanded. Excision and skin grafting are bloody procedures, however, especially if they are prolonged. An experienced surgical and anesthetic team and adequate amounts of blood matched to the patient's blood type are essential.

Some deep partial-thickness burns are also treated surgically as soon as their depth can be estimated. Advocates believe that this approach results in better joint function and less severe hypertrophic scar than more conservative management, which requires a period of at least three weeks for wound epithelialization. Although surgical therapy clearly results in earlier return of function and a shorter convalescence, its long-term results are probably similar to those of expectant therapy.

Grafting skin on established, granulating wounds from which the eschar has sloughed (a phenomenon due primarily to bacterial proteases) was once the norm, but it is currently the poorest surgical option. This approach is sometimes necessary, however, in the presence of severe illnesses or systemic complications.

The impediments to early surgical closure increase in parallel with the increasing extent of the burn. In trials comparing early with delayed surgical therapy at the same institution, the hospital stay was reduced with early surgery only when the burn area averaged 6 percent of the body-surface area. The incidence of other problems, such as the frequency of septic episodes, was lower with early surgical therapy, however.

Treatment of Extensive Deep Burns

For deep burns too extensive to be closed in one procedure, wound excisions can be staged — typically at intervals of about one week — as sufficient autologous skin grafts become available to close the excised wound. Alternatively, the burns can be completely excised within the first several days after injury, and a temporary skin substitute used to close the wound remaining after all available autologous skin has been harvested and grafted. Fresh or cryopreserved allogeneic skin from cadavers is the most reliable wound cover, although its use has a small risk of disease transmission. In severely burned children the systemic administration of recombinant human growth hormone can speed reepithelialization of skin-grafting sites and permit earlier reharvest.

Alternatives for Wound Closure

Every year, millions of people experience burns, suffer from nonhealing wounds, or have acute wounds that become complicated by infection, dehiscence or problematic scarring. Effective wound treatment requires carefully considered interventions often requiring multiple clinic or hospital visits. The resulting costs of wound care are staggering, and more efficacious and cost-effective therapies are needed to decrease this burden. Unfortunately, the expenses and difficulties encountered in performing clinical trials have led to a relatively slow growth of new treatment options for the wound management. Research efforts attempting to examine wound pathophysiology have been hampered by the lack of an adequate chronic wound healing model, and the complexity of the wound healing cascade has limited attempts at pharmacological modification. As such, currently available wound healing therapies are only partially effective. Therefore, many new therapies are emerging that target various aspects of wound repair and the promise of new therapeutic interventions is on the immediate horizon.

The skin substitute Integra47 is a bilaminate membrane composed of a porous lattice of cross-linked chondroitin 6-sulfate engineered to induce neovascularization as it is biodegraded. An outer layer of silicone serves to close

the wound while permitting water-vapor transfer. This layer is peeled off after about 14 days and replaced with ultrathin (0.01 to 0.015 cm [4/1000 to 6/1000 of an inch]) autologous skin grafts. These donor sites can be reharvested after about one week.

Autologous keratinocytes cultured in vitro from a small (2 cm2) biopsy of unburned skin obtained shortly after injury offer another option for the treatment of extensive burns. Culture of the keratinocytes for three weeks results in multilayered epithelial sheets, which are then applied on freshly excised burns. The rates of permanent engraftment of these cells have been poor in most centers, however, and the technique is extraordinarily expensive. In patients with burns involving more than 70 percent of the body-surface area, there was an engraftment rate of only 31 percent and only 4.6 percent of the body-surface area burned was closed, at a mean cost of $60,000 per patient.

It remains unclear whether the early total excision of extensive burns (those involving more than 40 percent of the body-surface area) improves survival and so is preferable to staged excision. In the most recent randomized trial, early total excision was associated with a marginal increase in survival in burns involving at least 40 percent of the body-surface area, but only in young adults without inhalation injury. However, the increasingly rapid pace of development of skin substitutes and of transplantation biology increases the likelihood that early total excision of the burn accompanied by one or more means of permanent, reliable wound closure will soon become routine in the care of patients with extensive deep burns.

Anaesthesia

If there is any concern over the airway a gas induction following pre-oxygenation or a fibre-optic intubation are the safest options. Suxamethonium should be avoided after the first 48 hours up to 2 years after a major burn because it may result in a large increase in serum potassium. Analgesia requirements are increased. Give ketamine or morphine (titrated to response).

Consideration should be given to altered pharmacokinetics:

- Volume of distribution increases for water soluble drugs (resistance to non-depolarising agents occurs.)
- Increased extracellular fluid:intracellular fluid ratio
- Albumin falls - less protein binding
- Increased metabolic rate / temperature leading to altered half life

Monitoring must include vital signs, temperature and urine output. Invasive monitoring may be necessary. The ECG leads can be placed with the use of staples if the chest wall is burnt.

Postoperatively the patient should be admitted to a high dependency unit, so that the continuing fluid loss following burn excision can be maintained.

Electrical burn

An electrical injury can occur to the skin or internal organs when a person is directly exposed to an electrical current.The outcome of an electric shock to an individual depends on the intensity of the voltage to which the person was exposed, the route of the current through the body, the victim's state of health, and the speed and adequacy of the treatment.

Causes

- Accidental contact with exposed parts of electrical appliances or wiring
- Young children biting or chewing on electrical cords, or poking metal objects into the electrical outlet
- Lightning
- Flashing of electric arcs from high-voltage power lines
- Machinery or occupational-related exposures

Symptoms may include:

- Skin burns
- Numbness, tingling
- Weakness
- Muscle contraction
- Muscular pain
- Bone fractures
- Headache
- Hearing impairment
- Seizures
- Heart arrhythmias
- Cardiac arrest
- Respiratory failure
- Unconsciousness

Patogenesis:

Electrical injury may cause disruption of the body's normal electrical activities. The neurologic system is affected most commonly. Neurologic dysfunction is present in some form, even if only temporary, in virtually all patients. Mass depolarization of the brain may lead to a loss of consciousness, amnesia, and coma. Spinal cord involvement may result in transverse myelitis. Transverse myelitis may have delayed onset and is associated with poor prognosis for recovery.

Electrical injuries also may affect the heart. As many as 25% of patients with electrical injuries have cardiac dysrhythmia. Sudden death from an alternating

current electrical injury is usually the result of ventricular fibrillation, although asystole and other dysrhythmias are common.

Another mechanism of injury is related directly to the amount of heat generated by the flow of electrical current through body tissue. At higher voltages, higher temperatures are achieved, resulting in greater direct thermal injury. High-tension voltages cause devastating injuries from huge amounts of internal thermal damage.

Vascular injury occurs as a result of vascular spasm. Heat generated by the injury also can cause coagulation and vascular occlusion. Damage to the vascular wall may produce delayed thrombosis and bleeding. Compartment syndrome may develop as a result of acute ischemic insult to the musculature.

Renal injuries may occur as a result of rhabdomyolysis. Rhabdomyolysis causes myoglobinuria from massive release of myoglobin. Myoglobin crystallization in the kidney tubules may cause acute renal failure.

Imaging Studies:

Chest radiograph is indicated for patients with history of syncope, cardiac arrest, chest pain, or shortness of breath.

Extremity radiographs are indicated for swelling, deformity, or pain.

Computed tomography of the head is indicated for patients with altered level of consciousness or a history of possible blunt or concussive trauma associated with the electrical injury.

Other Tests:

Cardiac monitoring while in the emergency department is recommended in patients who are either symptomatic for or have a history of high-voltage electrical or lightning injury.

Electrocardiogram is recommended for patients with any of the following signs or symptoms: (1) cardiac arrest, (2) dysrhythmia or loss of consciousness in the field, (3) a hand-to-hand path of current, or (4) a mechanism of enhanced conduction associated either with tetany and the locked-on phenomenon or with wet skin. Admission for continued cardiac monitoring is rarely necessary following low-voltage electrical injury and is safely reserved for patients with a history of cardiac arrest or loss of consciousness, dysrhythmia, or abnormal electrocardiogram findings.

Procedures:

Foley catheters are indicated for patients with high-voltage injuries to allow continuous measurement of urine output.

Treatment: Remove the patient from the source of the injury while maintaining the safety of all rescuers and caregivers. Standard burn care and tetanus prophylaxis should always be provided.

Airway: Maintain airway patency. The airway itself usually is unaffected unless it is a specific site of contact.

Breathing

Maintain adequate ventilation. Intubation and mechanical ventilation may be required as indicated. Patients may have central apnea if struck by lightning.

High-voltage electrical injuries may be associated with life-threatening rhythms or multisystemic involvement requiring ventilatory support.

Circulation

Maintain adequate perfusion to tissues. Intravenous access and, possibly, central venous access may be indicated depending on the severity of the injury. Low-voltage injuries rarely cause significant thermal injury, and in general, patients do not require large amounts of fluid.

Patients with high-voltage injuries have proportionately larger amounts of injured tissue and require large volumes of fluid. If peripheral access is inadequate, attempt either intraosseous or central venous catheter insertion. Accurately estimating the BSA involved is very difficult because the internal depth and degree of injury may be much greater than would be assumed from external appearances.

One approach to fluid resuscitation is to attempt to estimate the TBSA involved and to use a burn-resuscitation formula. The two most commonly used are the Brooke and the Parkland formulas.

A second approach to fluid resuscitation is to begin with 20 mL/kg boluses and to continue fluids to maintain a minimum urine output of 1 mL/kg/h.

The fluid of choice is lactated Ringer solution, which is recommended over isotonic sodium chloride solution because the latter tends to cause significant hyperchloremia.

Disability: After obtaining a baseline neurologic examination, prevent any further injury. If potential exists for a cervical spine or back injury, completely immobilize the patient pending radiographic studies.

Exposure: Thoroughly undress and examine patients, taking into consideration that hypothermia has particularly deleterious effects on patients with burns or who have experienced trauma. Cover patients, and keep them clean and dry. Use external warming sources early in the course of care if necessary.

Renal function

Patients with high-voltage injuries are susceptible to rhabdomyolysis and myoglobinuria. Myoglobin is toxic to the kidney because it may crystallize, occlude urine flow, and result in acute renal failure.

Surgical Care:
Vascular access is necessary in all patients with high-voltage injury or direct lightning strikes. If peripheral access is inadequate, attempt insertion of either an intraosseous or a central venous catheter.
Fasciotomies may be necessary relatively early in treatment and should be considered in patients with extremity injuries and compromised neurovascular status to relieve tight compartments and compartment syndrome. Gross myoglobinuria is a predictor of the need for fasciotomies. Removal of devascularized tissue is necessary. Once the wound is clear of necrotic debris, autografting may be performed. Some wounds may necessitate amputation, and the tissue may continue to evolve and require revisions over several days. Multiple methods, including arteriography, have been used to estimate the appropriate level of amputation with mixed results.
Escharotomies usually are not necessary because severe thermal burns of the skin are uncommon.
For oral commissure burns, delayed reconstruction is reported to provide better cosmetic results.
Frostbite is a cold-related injury characterized by freezing of tissue. Most cases are encountered in soldiers, in those who work outdoors in the cold, and among winter outdoor enthusiasts. Mountain frostbite is a variation observed among mountain climbers and others exposed to extremely cold temperatures at high altitude. It combines tissue freezing with hypoxia and general body dehydration.

Risk factors of frostbite include using beta-blockers and having conditions such as diabetes and peripheral neuropathy.

Cold exposure leads to ice crystal formation, cellular dehydration, protein denaturation, inhibition of DNA synthesis, abnormal cell wall permeability with resultant osmotic changes, damage to capillaries, and pH changes. Rewarming causes cell swelling, erythrocyte and platelet aggregation, endothelial cell damage, thrombosis, tissue edema, increased compartment space pressure, bleb formation, localized ischemia, and tissue death. Underlying responses to these injuries include generation of oxygen free radicals, production of prostaglandins and thromboxane A2, release of proteolytic enzymes, and generalized inflammation. Tissue injury is greatest when cooling is slow, cold exposure is prolonged, rate of rewarming is slow, and, especially, when tissue is partially thawed and refreezes. Symptoms affecting frostbitten body part include the following: coldness and firmness, stinging, burning, numbness, clumsiness, pain, throbbing, burning, or electric

current-like sensations on rewarming.While hands and feet are affected most frequently, shins, cheeks, nose, ears, and corneas may be involved. As in thermal burns, frostbite injuries may be classified by degree.

Degree of injury

First-degree injury - Erythema, edema, waxy appearance, hard white plaques, and sensory deficit

Second-degree injury - Erythema, edema, and formation of blisters filled with clear or milky fluid and which are high in thromboxane (These blisters form within 24 hours of injury.)

Third-degree injury - Presence of blood-filled blisters, which progress to a black eschar over a matter of weeks

Fourth-degree injury - Full-thickness damage affecting muscles, tendons, and bone, with resultant tissue loss

Symptoms

Generally, frostbite is accompanied with discoloration of the skin, along with burning and/or tingling sensations, partial or complete numbness, and possibly intense pain. If the nerves and blood vessels have been severely damaged, gangrene may follow, and amputation may eventually be required. If left untreated, frostbitten skin gradually darkens after a few hours. Skin destroyed by frostbite is completely black and looks loose and flayed, as if burnt.

Lab Studies:

Lab studies are not important in the initial diagnosis and management of frostbite; however, they may be helpful in identifying delayed systemic complications, such as wound infection with sepsis or underlying hypothermia.

Commonly encountered lab findings include evidence of hemoconcentration and depressed liver function.

Imaging Studies:

Tc-99m (Technetium 99) pertechnetate scintigraphy is sensitive and specific for tissue injury. Some studies have shown good correlation between scintigraphy findings at 48 hours after injury and ultimate extent of deep-tissue injury. In addition, scintigraphy is useful in assessing the response of damaged tissue to therapy.

Radiographs identify clinically suspected fractures or dislocations but are otherwise rarely useful in initial evaluation. They may assist in the diagnosis of long-term complications, such as osteomyelitis.

Arteriography is of limited value because it only images large vessels, not microvasculature. It cannot be used to estimate bone cell perfusion or viability.

Prehospital Care:

Address life-threatening conditions first.

Replace wet clothing with dry soft clothing to minimize further heat loss.

Initiate rewarming of affected area as soon as possible. Do not attempt rewarming if a danger of refreezing is present. Avoid rubbing affected area with warm hands

or snow, as this can cause further injury. If affected body part is an extremity, wrap it in a blanket for mechanical protection during transport. Avoid alcohol or sedatives, which can enhance heat loss and impair shivering.
It is better to walk with frozen feet to shelter than to attempt rewarming at the scene; however, walking on frostbitten feet may cause tissue chipping or fracture.
Emergency Department Care:
Address life-threatening conditions first. Fluid resuscitation, especially in persons with mountain frostbite, enhances blood flow and tissue perfusion.
Rapidly rewarm affected body part, avoiding further trauma.
An appropriate warming technique is the use of a whirlpool bath or tub of water at 40-42°C. Mild antibacterial soap may be added. Avoid warmer temperatures or dry heat because of the risk of thermal injury.
If a tub is not available, use warm wet packs at the same temperature.
Avoid massaging the affected area, as this can cause further injury.
Administer analgesics, such as morphine sulfate, as needed for pain.
Thawing usually takes 20-40 minutes and is complete when distal tip of affected area flushes. Once thawed, keep body part on sterile sheets, elevated, and splinted when possible. A cradle may be used over an injured lower extremity to avoid pressure or trauma.
Debride clear blisters to prevent thromboxane-mediated tissue injury. Leave hemorrhagic blisters intact to reduce risk of infection.
In patients with an associated dislocation, perform reduction as soon as thawing is complete. Manage fractures conservatively until postthaw edema has resolved.
Only indication for early surgical intervention is debridement of blisters or necrotic tissue and fasciotomy in the case of compartment syndrome. Early surgical decision for amputation is rarely needed.It often takes 1-3 months for frostbitten tissue to be declared viable. Affected area generally heals or mummifies without surgery; therefore, unless guided by scintigraphy, delay amputation as long as possible.Lower extremity involvement, infection, and delay in seeking medical attention are associated with an increased risk of operative therapy.

In addition to therapies described below, there are useful therapies that have not been prospectively validated and doses not standardized. Some of these include low molecular weight dextran of which daily infusions may be beneficial. This agent may prevent erythrocyte clumping in cold-injured blood vessels. In addition, low-dose infusions of heparin may prevent microthrombosis. Marcaine also has been used either for cervical or lumbar sympathetic blockade to decrease sympathetic tone and relieve pain, but its efficacy is unclear.

V. Materials of activation of students

(questions, tasks, controversial situations, illustrative materials and other).

VI. Materials of selftraining of students on the topic of lecture: literature, questions, tasks.

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