MINISTRY OF THE HEALTH CARE OF UKRAINE
LVIV DANYLO HALYTSKY NATIONAL MEDICAL UNIVERSITY
General Surgery Unit

METHODICAL RECOMMENDATIONS

to practical lesson

BURNS. BURNS DISEASE. FROSTBITES. ELECTRICAL TRAUMA.

Educational discipline:
G E N E R A L S U R G E R Y

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1. ACTUALITY OF THEME

According to data of the World Health Organization, thermal injuries account near 6 % of all trauma cases in peacetime. In Ukraine approximately two to three hundred thousand of people are burnt annually, including 50 000 children. The most serious complications of burns occur in children; their account for 20 % of all lethal outcomes.

As this type of trauma is very common, doctors of all specialties need some complex of knowledge to be able to diagnose, treat and prevent complications.

This methodical recommendations include the most important material for students in studying of the topic. This methodical recommendations is intended for third – year medical students of English – speaking medium.

2. DURATION OF LESSON – 4 hours

3. TRAINING AIM.

To know (theoretical questions):

Burns are the damage to tissues due to the exposure of high temperature, chemical compounds, electrical current and radiant energy.

Hence we can distinguish the following types of burns according to their etiological factor:

- thermal
- chemical

- electrical
- radiation.

According to the circumstances the following types are distinguished:

- on-the-job burn
- domestic burn
- military burn.

In peacetime on-the-job and domestic burns occur with similar regularity. Thermal burns are the most common ones**.**

Thermal burns

In elderly people over 65 death rate due to burns is 28 %.

In 54 % of all cases among children it is children aged 1-3 years; and in 91% of cases these children get scalded by overthrowing boiling pans.

In the age-group under 5 the most common cause of burns is playing with fire – 37 % of all cases.

At the age over 5 burns can be caused by a variety of factors: fireworks, fireplace, irons, electric current and so on.

When there is a burn the degree of damage is determined by the following factors:

Exposure temperature – the higher temperature, the worse the damage. The burn can be caused by contact with an object heated to the temperature 50^0C. The worst burns are caused by open fire whose temperature is 2000-3000^0C.

Thermal conductivity of the object that comes in contact with skin – the higher the conductivity, the worse the damage. As dry heat of 100^0C does not cause burns (for example, in sauna), hot water of the same temperature causes serious deep burns.

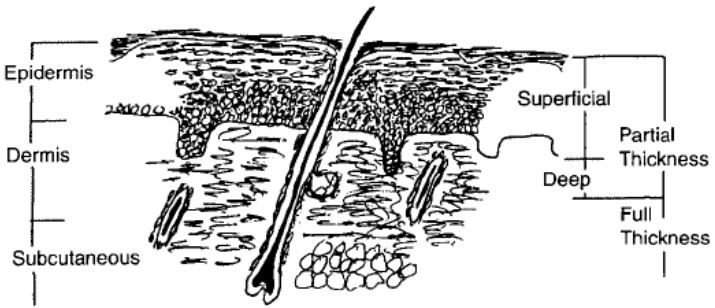
Exposure time – the longer it is, the worse the damage.

Localisation of burns and condition of skin in the area of damage – the depth of skin in different areas of the body is different. Thus we more often get deep burns on the face, anterior part of neck, medial surface of extremities, while the same type of exposure would cause more superficial burns on the back or soles. Other things being equal, burns on the face are more often life-threatening. They are often accompanied by an injury of the eyes, oral cavity and airways which aggravates the patient's condition and prognosis.

The depth of damage and its area are of most importance in the development of pathologic process and prognosis.

There are several systems classifying of burns ***according to the depth of damage.***

In Ukraine we use the four-degree classification of the burns.



Degree 1 – damage to the epidermis.

The first sign of degree 1 of burn is the development of hyperemia and edema in the area of exposure. The thermal agent causes dilation of vessels; at the same time blood plasma escapes in lot quantities through the vessel walls that have become permeable. The irritation of nerve endings causes bad pain. In several days all the signs subside, sometimes leaving skin pigmentation. A typical example of degree 1 of burn is sun burn.

Degree 2 - when the whole epithelium is injured, with formation of blisters filled with clear fluid.

The main sign of degree 2 of burn is, along with hyperemia, increased escape of plasma into tissues. The uppermost layer of epidermis rises due to blister formation which are filled with fluid that contains practically no cells. The content of blisters is usually sterile but if the blister is damaged its contents can get infected.

The pains in degree 2 are more powerful; they usually persist for 2-3 days. Getting scalded with boiling water or steam is a typical example of degree 2 of burn.

Degree 3, skin necrosis. Two stages, 3a and 3b are distinguished.

Stage 3a – necrosis of the epithelium and superficial layers of the derma.

Stage 3b – necrosis of all derma layers together with hair bulbs, sweat and oil glands, involving subcutaneous fat.

Necrotic tissues form an eschar of brown colour (after exposure to fire or sizzling objects), or of whitish-grayish colour (upon scalding) whose sensitivity is low. In a few days the eschar is divided from the living tissue with a demarcation line. Around burns of degree 3 there are always burns of degree 1 and 2. Burns of degree 3 heal with formation of crude keloid cicatrix with trophic ulcer in the centre which leads to pronounced functional and cosmetic defects.

Degree 4 of burn (charring) – necrosis of all skin layers and tissues below (subcutaneous fact, fascia, muscles and bones). It can occur in a fire in a car, plane, mine, upon contact with molten metal.

Burns of degree 1, 2 and 3a are classified as superficial, those of degree 3b and 4 – as deep. This distinction is a matter of principle. In all superficial burns the skin can close on its own as all the sources of epithelisation are preserved. In deep burns all possible sources of epithelial growth are destroyed and the defect cannot close on its own.

In other countries burns are classified into 5 degrees, so degree 3b is called degree 4, and our degree 4 corresponds to their degree 5.

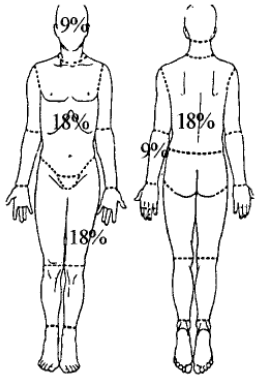
Age and burn size

A direct but inverse relationship exists between age and survival for any burn size. While the mortality of a 40% TBSA burn in a 20-year-old patient is approximately 8%, the mortality of this same injury in someone older than 70 years is 94%. The higher mortality of older patients with burn injuries is attributed to their preexisting medical conditions, including cardiac, pulmonary, renal, and hepatic dysfunction. Similarly, children younger than 1 year survive large burns at a reduced rate.

To assess the area of burn various schemes are used:

For a quick assessment the "rule of nines" or "rule of palm" is used.

"Rule of nines" (for adults): the head and each arm constitute 9 % of total body area each. The front and back surfaces of trunk – 18 % each.



AREA	0-1 YEAR	5-9 YEARS	ADULT
Head	19	11	7
Neck	2	2	2
Ant. Trunk	13	13	13
Post. Trunk	13	13	13
R. Buttock	2 ½	2 ½	2 ½
L. Buttock	2 ½	2 ½	2 ½
Genitalia	1	1	1
R.U. Arm	4	4	4
L.U. Arm	4	4	4
R.L. Arm	3	3	3
L.L. Arm	3	3	3
R. Hand	2 ½	2 ½	2 ½
L. Hand	2 ½	2 ½	2 ½
R. Thigh	5 ½	8	9 ½
L. Thigh	5 ½	8	9 ½
R. Leg	5	5 ½	7
L. Leg	5	5 ½	7
R. Leg	3 ½	3 ½	3 ½
L. Foot	3 ½	3 ½	3 ½

"Rule of palm" – the palm constitutes 1 % of total body area.

Viliavin's scheme is used at in-patient departments. It represents the scheme of anterior and posterior body surfaces where burns of varying depth are shown with different colours. In the process of healing the data on the scheme are changed accordingly.

In ***Postnikov's method*** the burnt area is covered with sterile cellophane on which the contours of the burn are plotted, then the area of the burn is calculated on the plotting paper. This method is not used nowadays.

In the patient's case history the severity of the burn is shown in the following way.

The burn is represented with a common fraction with the affected area (deeply burnt area in parentheses) in the numerator, and the degree of burn – in the denominator. Besides, the etiology of the burn (thermal, chemical or radiation) is stated before the fraction and after it – the main affected areas. For example:

Thermal burn 10 % (5 %)/2-3 head, neck.

Determination of prognosis

The patient's life is threatened if:

- there is total burn of degree 1
- there is burn of degree 2 or 3 of 30% of body area
- there are burns of degree 3b and 4 of 10-15% of body area.

A common way to determine prognosis after burns is to use "the rule of a hundred" or Frank's index.

"The rule of a hundred":

The patient's age is added to the burnt are (in percentage to the total body area). If the resulting sum

Is under 60 – the prognosis is favourable

Is 61-80 – the prognosis is relatively favourable

Is 81-100 – the prognosis is doubtful

Is over 100 – the prognosis is unfavourable.

Frank's index is arrived at by adding the area of superficial burns with the tripled area of deep burns. If this index

Is under 30 – the prognosis is favourable
Is 31-60 – the prognosis is relatively favourable
Is 61-90 – the prognosis is doubtful
Is over 90 – the prognosis is unfavourable.

In the case of extensive deep burns the condition known as burn diseases develops.

Skin Anatomy and Function

Skin is the largest organ of the body. It has 3 major tissue layers.

Epidermis

The outermost layer, the epidermis, is composed of stratified epithelium. Epidermis has 2 components, an outer layer of anucleate cornified cells (stratum corneum) that covers inner layers of viable cells (Malpighian layers) from which the cornified surface cells arise by differentiation. The stratum corneum acts as a barrier to impede the entrance of microorganisms and toxic substances while allowing the body to retain water and electrolytes. Malpighian layers provide a continuous production of cornified cells. Malpighian layers can be further subdivided into the germinal basal cell layer, stratum spinosum, and stratum granulosum.

Dermis

Beneath the epidermis is the dermis, which is composed of a dense fibroelastic connective-tissue stroma containing collagen and elastic fibers and an extracellular gel termed ground substance. This amorphous gel comprises an acid mucopolysaccharide protein combined with salts, water, and glycoproteins; it may contribute to salt and water balance, serve as a support for other components of the dermis and subcutaneous tissue, and participate in collagen synthesis. The dermal layer contains an extensive vascular and nerve network, special glands, and appendages that communicate with the overlying epidermis.

The dermis is divided into 2 parts.

The most superficial portion, the papillary dermis, is molded against the epidermis and contains superficial elements of the microcirculation of the skin. It consists of relatively cellular, loose connective tissue with smaller, fewer collagen and elastic fibers than the underlying reticular dermis. Within the papillary dermis, dermal elevations indent the inner surface of the epidermis. Between the dermal papillae, the downward projections of the epidermis appear peglike and are termed rete pegs.

In the reticular portion of the dermis, collagen and elastic fibers are thicker and greater in number. Fewer cells and less ground substance are found in the reticular dermis than in the papillary dermis. Thickness of the dermis varies from 1-4 mm in different anatomic regions and is thickest in the back, followed by the thigh, abdomen, forehead, wrist, scalp, palm, and eyelid. Thickness varies with the individual's age. It is thinnest in the very old, where it is often atrophic, and in the very young, where it is not fully developed.

Subcutaneous tissue

The third layer of skin is subcutaneous tissue, which is composed primarily of areolar and fatty connective tissue. This layer shows great regional variations in thickness and adipose content. It contains skin appendages, glands, and hair follicles. Hair follicles extend in deep narrow pits or pockets that traverse the dermis to varying depths and usually extend into the subcutaneous tissue. Each hair follicle consists of a shaft that projects above the surface and a root that is embedded within the skin.

Apocrine and eccrine sweat glands

There are 2 types of sweat glands in skin: apocrine and eccrine.

Apocrine glands are epitrichial because they have a duct that opens into a hair follicle. Apocrine glands are largely confined to the axillary and perineal region and do not become functional until just after puberty.

Eccrine glands are simple, coiled, tubular glands usually extending into the papillary dermis. Eccrine glands are atrichial because their duct opens onto the skin surface independently of a hair follicle. Eccrine glands are found over the entire body surface, except the margins of the lips, eardrum, inner surface of the prepuce, and glans penis.

Sebaceous glands are simple or branched alveolar glands, usually connected to the hair follicles. Sebaceous glands unconnected with hair follicles occur along the margin of the lips, in the nipples, in the glans and prepuce of the penis, and in the labia minora. Depending on the depth of burn injury, epithelial repair can be accomplished from local epithelial elements and skin appendages.

When skin is burned, the damaged stratum corneum allows the invasion of microorganisms, and the Langerhans cells, which mediate local immune responses, also are damaged. In burn patients with severe injuries, their systemic immune response is diminished, making them susceptible to serious infections.

Heat transfer from heating agent to skin

Severity of burn injury is related to the rate at which heat is transferred from the heating agent to the skin. Rate of heat transfer depends on the heat capacity of the agent, temperature of the agent, duration of contact with the agent, transfer coefficient, and specific heat and conductivity of the local tissues.

Heat capacity: Capacity of a material to hold heat energy is determined by both the specific heat and the heat capacity of the material.

Specific heat of a material: This is defined as the ratio of the amount of heat required to raise a specific mass of the material 1 degree in temperature, to the amount of heat required to raise an equal mass of a reference substance (usually water) 1 degree in temperature.

Heat capacity: This refers to a quantity of heat a material contains when it comes in contact with skin. Quantity of heat stored depends on the specific heat of the material and the amount and temperature of the material.

The importance of heat capacity as a determinant of severity of burn injury is best illustrated by comparing the amount of heat stored in 10 g of 2 different materials (copper and water) heated to the same temperature (100°C). Specific heat of water is 4.2178 W xsec/g xK (watt times seconds of heat per gram mass times degrees Kelvin). If these 2 materials come in contact with skin, they give up their heat by cooling while skin accepts the heat by increasing its temperature.

If the temperature of each material decreases by 60°C, water gives up 2530 W xsec of heat, whereas copper transfers only 230 W xsec of heat. Even if the initial temperatures of the 2 materials are identical, heat available from water is much more likely to produce a severe injury. The specific heat of water (most common cause of scald burns) is the highest of all the gases, metals, and solids tested to date, with the exception of ammonia and ether.

Temperature

Initial temperature of a material at the instant of contact is also an important determinant of burn severity. Many materials (eg, water) cannot be heated beyond a certain temperature without changing state. Water can only be heated to 100°C at atmospheric pressure before it ceases to be a liquid and vaporizes. Because more joules are required to produce steam, this additional heat transfer accounts for the severe burns caused by steam injury. When other liquids reach a specific temperature, they ignite or oxidize by combining with oxygen.

The flash point of the liquid is the temperature at which the vapors of a volatile liquid mixed with air spontaneously ignite. A flammable liquid is defined as any liquid having a flash point less than 37.8°C. Liquids with a flash point above this temperature are considered combustible. In addition to their high temperatures, burning liquids also may ignite the victim's clothing, thereby further exacerbating severity of the injury.

Duration of contact

Human skin can tolerate temperatures as high as 44°C (111°F) for a relatively long time (6 hours) before irreversible injury occurs.5 Temperatures greater than this level cause an almost logarithmic increase in tissue destruction. Duration of contact between a liquid and skin depends on both the viscosity of the liquid and the manner in which it is applied to the victim's skin. When hot liquid is splashed on a person, as in a spill scald, it usually flows down the body in a rate of descent that depends on the fluid's viscosity. Although water streams to the ground unless impeded by clothing, viscous oils and greases usually cling to a victim's skin, prolonging duration of exposure and extent of injury.

In immersion scalds, duration of contact between the hot liquid and the skin is considerably longer than that with spill scalds, thereby increasing the severity of injury. Certain populations are at high risk of suffering immersion scald burns, including children younger than 5 years, older persons (65 y and older), and disabled persons. Individuals in these high-risk groups tend to have a slower reaction time and a physical inability to escape from hot water. Immersion burns commonly cover a large percentage of total body surface area (TBSA), almost twice that of other scald burns, which contributes to their high rate of morbidity and mortality.

Child abuse and immersion scald burns

Child abuse accounts for a large proportion of immersion scald burns. Immersion burns caused by child abuse can be distinguished from accidental burns by the pattern and site of the burn, histories given by the caretaker and patient, and a medical history of scars representing previous abuse. Nonaccidental burns often have clear-cut edges, as found in "stocking" scalds, where a child's foot has been held in scalding water. Spill scald burns, on the other hand, more often have uneven, fuzzy edges as a result of the victim's attempts to escape the hot liquid. Burns from abuse tend to occur on the back of hands and feet, the buttocks and perineum, and legs. Accidental burns, such as those caused by a child spilling a cup of coffee, more often cause burns on the head, trunk, and palmar surface of hands and feet. Physical evidence of previous injuries, such as craterlike cigarette burn scars or bruises, also suggests abuse.

Heat transfer

Even when a substance possesses sufficient heat to cause a burn injury, it will not do so unless its heat can be transferred to the skin. This ability to transfer heat between 2 different materials is regulated by the heat transfer coefficient, which is defined as the amount of heat that passes through a unit area of contact between 2 materials when the temperature difference between these materials is 1 degree.

Three different methods of heat transfer exist: conduction, convection, and radiation. The simplest method of heat transfer is conduction, which occurs when a hot solid object comes in direct contact with the skin.

Convection is the transfer of heat by a material that involves the physical movement of the material itself and is determined by heat conduction and by energy storage and mixing motion. Convection is most important as the mechanism of energy transfer between skin and a heated liquid or gas. Hot water spilling on skin transfers heat by convection between the water droplets and the skin surface. Steam or very hot air also transfers heat to the skin by convection.

Tissue conductivity

Conductivity of the specific tissue involved has a significant influence on the extent of burn injury. Heat transfer within skin is influenced by the thermal conductivity of the heated material, the area through which heat is transferred, and the temperature gradient within the material. Water content, natural oils or secretions of the skin, and the presence of insulating material (eg, cornified keratin layer of skin) influence tissue conductivity. In addition, alterations in local tissue blood flow produce a profound effect on heat transfer and distribution. Inability to conduct heat away from a contact point efficiently results in varying degrees of tissue injury.

Because skin is a relatively poor conductor of heat, it provides an extensive barrier to heat injury. The degree to which it resists injury depends on its anatomic configuration. Its uppermost layer, the epidermis, is relatively uniform in thickness in all body regions (75-100 mm) except for the soles and palms, where it attains a greater thickness (0.4-0.6 mm). The rarity of full-thickness injury to the palms and soles of the feet can be attributed to their thick epithelial cover.

The ultimate outcome of a burn injury also is influenced by the depth of epidermal appendages in the burned tissue, which varies according to the age of the patient. Very young and old individuals have superficial appendages, which make both groups more susceptible to full-thickness burn injury. By contrast, the epidermal appendages of the human scalp and male beard are very deep, making these sites more refractory to severe burn injury.

Burn wound injury

During the first day after burn injury, 3 concentric zones of tissue injury characterize a full-thickness burn: zones of coagulation, stasis, and hyperemia.8 The central zone of coagulation has the most intimate contact with the heat source. It consists of dead or dying cells as a result of coagulation necrosis and absent blood flow. It usually appears white or charred. The intermediate zone of stasis usually is red and may blanch on pressure, appearing to have an intact circulation; however, after 24 hours, circulation through its superficial vessels often has ceased. Petechial hemorrhages may be present. By the third day, the intermediate zone of stasis becomes white because its superficial dermis is avascular and necrotic. The outer zone of hyperemia is a red zone that blanches on pressure, indicating that it has intact circulation. By the fourth day, this zone has a deeper red color. Healing is present by the seventh day.

Transformation of the zone of stasis to coagulation occurs and has been related to many factors, including progressive dermal ischemia. Experimental studies have implicated prostaglandins, histamine, and bradykinin as the chemical mediators of this progressive vascular occlusion. They can produce edema by altering endothelial cell and basement membrane function to enhance permeability. When this ischemia persists, the zone of stasis eventually becomes a full-thickness burn injury.

When Robson et al discovered various prostaglandin derivatives in burn wounds, they suggested that an imbalance in the vasoconstrictive and vasodilatory prostanoids produces a progressive tissue loss in the zone of stasis. In acute burn wounds, an increased level of oxygen free radicals, such as xanthine oxidase, appeared to be involved in the formation of burn edema. This edema formation can be attenuated by pretreatment with xanthine oxidase inhibitors.

Systemic inflammatory response

In patients whose burns exceed 30% of TBSA, cytokines and other mediators are released into the systemic circulation, causing a systemic inflammatory response. Because vessels in burned tissue exhibit increased vascular permeability, an extravasation of fluids into the burned tissues occurs. Hypovolemia is the immediate

consequence of this fluid loss, which accounts for decreased perfusion and oxygen delivery. In patients with serious burns, release of catecholamines, vasopressin, and angiotensin causes peripheral and splanchnic bed vasoconstriction that can compromise in-organ perfusion. Myocardial contractility also may be reduced by the release of inflammatory cytokine tumor necrosis factor-alpha.

In deep third-degree burns, hemolysis may be encountered, necessitating blood transfusions to restore blood loss. A decrease in pulmonary function can occur in severely burned patients without evidence of inhalation injury from the bronchoconstriction caused by humoral actors, such as histamine, serotonin, and thromboxane A2. A decrease in lung and tissue compliance is a manifestation of this reduction in pulmonary function. Burned skin exhibits an increased evaporative water loss associated with an obligatory concurrent heat loss, which can cause hypothermia.

A significant proportion of the morbidity and mortality of severe burns is attributable to the ensuing hypermetabolic response. This response can last as long as a year after injury and is associated with impaired wound healing, increased infection risk, erosion of lean body mass, impaired rehabilitation, and delayed integration of the burn patient into society.

Pharmacologic and nonpharmacologic strategies are being used to reverse the catabolic effect of thermal injury. Nonpharmacologic approaches include early excision and wound closure, aggressive management of sepsis, elevation of the environmental temperature, continuous high carbohydrate/high protein enteral feeding, and early institution of resistive exercise programs. Pharmacologic modulation of the postburn hypermetabolic response has been achieved through administration of recombinant human growth hormone, low-dose insulin infusion, use of synthetic testosterone analog (oxandrolone), and beta blockade with propranolol.

Nutritional support

Because burn injury causes a hypermetabolic state that is characterized by a dramatic increase in resting energy expenditure, nutritional support is essential, especially via the enteral route, to reduce intestinal villous atrophy. Deitch et al reported a syndrome of decreased bowel mucosal integrity, capillary leak, and decreased

mesenteric blood flow, which allowed bacterial translocation into the portal circulation. These translocated bacteria significantly alter hepatocyte function and spread systemically to cause systemic sepsis. Adequate resuscitation that ensures mesenteric blood flow can prevent potential development of multisystem organ failure. Enteral nutrition with glutamine has a tropic effect on the enterocytes that preserve mucosal integrity.

Infection

In patients with major burn injuries, infection remains the major cause of death. Immune consequences of this injury have been identified and are specific deficits in neutrophil chemotaxis, phagocytosis, and intracellular bacterial killing. Cell-mediated immunity, as measured by skin testing, also is compromised and has been related to both decreased lymphocyte activation and suppressive mediators present in the serum of burn patients. A reduction in immunoglobulin synthesis also has been encountered in these seriously ill patients.

Burn disease is a complex of clinical manifestations, general body reactions and dysfunction of internal organs upon thermal injury of the skin and underlying tissues.

The signs of burn disease are observed if superficial burns cover over 15-20% of body area or if deep burns cover over 10%. In old people and children burn disease develops when as little as 5% of skin is affected.

The course of burn disease is divided into stages:

- Burn shock
- Acute burn toxemia
- Burn septicemia
- Convalescence.

Burn shock begins immediately after the trauma and lasts for up to 3 days.

It develops due to the irritation of a large amount of nerve elements in the affected area. In the initial stage of burn shock the patient is conscious, suffering from bad pains and thirst. Skin areas outside the burn are

pale and cold. In a few hours the consciousness is clouded, the patient grows indifferent and can develop cramps. Vomiting and diarrhea are a very bad prognostic sign. Urination decreases, sometimes anuria develops.

The pulse is rapid, blood pressure drops to 50 mm Hg which a bad prognostic sign, too. Along with rapid respiration and tachycardia the cardiac output decreases, cyanosis increases and collapse can develop. If they do not receive treatment within 12-48 hours, patients die of cardiovascular collapse.

Loss of plasma is the main pathogenetic factor in burn shock. The disorder of vascular permeability develops immediately after the burn reaching its peak in 6-8 hours.

The shock always develops if the burnt area is over 50% and it is the main cause of death.

According to the clinical course ***3 degrees of burn shock*** are distinguished:

Burn shock of degree 1 develops when 15-20% of body surface is burnt. The patient is somewhat agitated, the heart rate is up to 90 beats per minute. BP is slightly higher or normal. The diuresis is normal. If infusion therapy is delayed for 6-8 hours, oliguria and haemoconcentration develop.

Burn shock of degree 2 develops upon the injury of 21-60% of body surface. The patients are conscious but retarded. Tachycardia is 100-120 beats per minute. Blood pressure tends to be lower. Body temperature lower than the norm. Diuresis is decreased.

Burn shock of degree 3 develops when over 605 of the body is affected. The patient's condition is very bad. In 1-3 hours soporific state develops. The pulse is thready, blood pressure – under 80 mm Hg. Nausea, hiccups, vomiting are possible. Urine is characterized by macrohematuria, and then anuria develops. Body temperature drops. If cardiac insufficiency aggravates, the patients often die.

Treatment of burn shock begins with first-aid measures:

- discontinue the exposure to thermal agent
- cool the affected area with an ice-bag or cold water for 10-15 minutes. First, cold inhibits lactate production and acidosis, thereby promoting catecholamine function and cardiovascular homeostasis, inhibits burn wound histamine release, which in turn blocks local and remote histamine-mediated increases in vascular

permeability. This minimizes edema formation and intravascular volume losses; cold suppresses the production of thromboxane, implicated as the mediator of vascular occlusion and progressive dermal ischemia after burn injury.

Burn victims often complain of feeling chilled as a result of the loss of water and heat through the burned skin. Heat loss can be minimized by first placing a clean sheet under the patient and then covering the patient with another clean sheet, followed by clean blankets. The inside of the transport vehicle should be heated enough to make the patient comfortable. If the vehicle with advanced life support capability can transport the burn patient to a specialized burn treatment facility within 30 minutes, the burn patient should be transferred directly to this facility, bypassing other hospitals. If the transport time to the specialized burn treatment facility is longer than 30 minutes, transport the patient to the nearest ED with advanced life support capability.

- apply aseptic dressing
- give analgesia and start anti-shock therapy: narcotic analgesics if possible, infusion therapy, immediate evacuation.

Treatment of burn shock in the prehospital setting should consist of elevating the patient's legs 12 inches off the ground and administering humidified oxygen. If rescue personnel have advanced life support capability and transport time may be prolonged, these treatments are complemented by intravenous (IV) fluid administration. Fluid resuscitation need not be initiated if patient is transported to the hospital in less than 30 minutes. When transport time is longer than 30 minutes, the indications for fluid resuscitation are thermal injuries involving greater than 20% of TBSA or evidence of burn shock.

Fluid resuscitation is not recommended for children at the scene of the accident because of the difficulties encountered in cannulating small veins. When fluid resuscitation is indicated in an adult, administer lactated Ringer solution or normal saline without glucose though a large-bore percutaneous catheter, preferably inserted

through unburned skin. The arm is the preferred site for cannulation. Determine IV flow rates by the patient's clinical status.

In hospital the order of measures for grave patients is as follows:

- make sure that airways are patent
- give narcotic analgesics in combination with antihistamine andsedative drugs
- catheterize the central vein and start infusion
- do toilet of the burnt area (unless the patient's condition is very bad): cleanse the skin around the burn, remove the detached epithelium, cleanse the surface with hydrogen peroxide, apply dressing.
- Catheterize the bladder
- Introduce a tube into the stomach.

Further treatment is based on continuation of analgesia, parenteral administration of crystalloid and protein solutions.

The volume of solutions to be infused is determined according to the Evans formula: body weight in kilos x (multiply by) burn area in percentage + 2000 ml = (equals) the volume in ml. a half of the estimated volume should be infused to the patient within the first 8 hours. The ratio of crystalloid and protein solutions should be 2:1.

In a favourable course of the disease the burn shock passes to the ***toxemia stage*** on 10-15 days' duration.

Various substances formed upon the trauma act as toxins – histamine, serotonin, lipoproteides, prostaglandins, antigen-specific glycoproteides, toxic oligopeptides, and also products of erythrocyte hemolysis and fibrinolysis.

The first sign of toxemia is the increase of body temperature to 38-39^{0}C. Apart from fever, other signs of intoxication are noted: pallor of skin, tachycardia, weakness, nausea.

Clinical tests reveal leucocytosis with a shift to the left, anemia, hypoproteinemia, hyperbilirubinemia, transaminase increase.

In the second stage of burn disease the components of therapy are:

- infusion therapy
- disintoxication therapy
- treatment of acute renal failure
- correction of acidosis

Infusion therapy in this stage can be less in volume than on the first day but apart from colloid and protein preparations it should include:

- energy replenishment with glucose solution and fat emulsion
- administration of disintoxicating drugs – albumin, hemodes.
- as a rule, blood transfusion is required of 250-500 ml of packed red cells 2-3 times a week

The treatment of renal failure consists of infusion therapy and administration of diuretics. If there is no reaction to diuretics, that is, anuria develops, then the only possibility lies in administering extracorporal detoxication – hemodialysis.

Almost in all burns acidosis develops. In minor injuries it is compensated for by hyperventilation of lungs, however, it is often necessary to administer 4-5% soda solution or another alkaline buffer intravenously.

In burn disease with deep burns when rejection of necrotic tissues begins in 10-14 days, ***septicemia*** develops. The duration of this stage is 2-3 months.

The patients manifest all signs of suppurative intoxication: fever, chills, weakness, lack of appetite.

In this stage anemia continues that is due to the suppression of erythrocyte production. The white blood count manifests a shift to the left. Biochemical changes are associated with protein deficit due to great protein loss through the damaged skin and due to decreased protein synthesis in the liver suffering from intoxication (toxic hepatitis is manifested by moderate jaundice, enlarged liver and increase of transferase).

Typical complications in septipyemia are:

- pyelonephritis
- pneumonia
- acute ulcers of alimentary tract with gastrointestinal bleeding (Curling's stress ulcer)

3-4 weeks after the burn the patients often develop ***burn dystrophy*** with the absence of wound healing or resistance to infection. Burn sepsis becomes quite probable.

Therapy against septipyemia should begin in early stages of burn disease as preventive measures are most effective. These include:

- ***preventive administration of antibiotics*** beginning on day one to all patients with deep burns of more than 10% of body area. Cephalosporines are the drugs of choice.
- ***Stimulation of immune system***: active stimulation with staphylococcus anatoxin, passive stimulation with antistaphylococcal plasma, burn convalescent plasma, γ-globulin, vitamins.
- ***Measures aimed at early separation of necrotic tissues and closing of wound surface***

These therapeutic measures can be conservative or surgical.

Conservative treatment can be performed by an open or closed method.

In the ***open technique*** the aim is the formation of dry eschar which is a biological dressing of sorts. To prevent infection of wound surface the burnt area or the whole patient is placed in the so-called controlled antibacterial medium – a room with a constant stream of sterile air heated to 30-34^{0}C.

Another modification of this method is the use of a special bed with an air-cushion.

The open method is used in case of extensive burns or when the face, neck or perineum is affected where application of dressing is difficult and excision of necrotic tissues is undesirable.

The closed technique is the most common one nowadays.

The conservative wound-closing technique is used on superficial burns. It includes application of ointments with water-soluble base (suspensions like levomicole, levocine). The dressings are changed in 2-3

days. If suppurative inflammation develops, wet dressings with antiseptic solutions are applied (dioxine, chlorhexidine, boric acid).

In deept burns of degree 3b or 4 ***combined conservative and operative therapy*** is used.

Synthetic dressings

First-generation film dressings (eg, thin films, hydrocolloidal hydrogel foams) are based on the concept that epidermal regeneration occurs best in a moist environment. Second-generation microenvironmental wound dressings combine the fluid-retaining properties of film dressings with the absorptive properties of the hydrocolloid. In theory, a central membrane absorbs wound fluid through the porous inner layer. An external layer allows moisture vapor to escape, yet is impermeable to exterior fluids and bacteria.

Second-generation dressings have many desirable features but are relatively expensive. A simple, less costly alternative to these synthetic dressings has been proposed that can be reliably used in minor burns. After cleansing and debridement, strips of sterile fine meshed gauze (type 1) soaked in 0.9% sodium chloride are placed over the entire wound. This layer of gauze is then covered by multiple layers of fluffed 4 X 4 inch coarse mesh gauze (type 6) secured by an inelastic roller gauze dressing. The gauze dressing is attached to unburned skin using microporous tape.

When possible, elevate the site of injury above the patient's heart. Elevation of the injured site limits accumulation of fluid in the interstitial space of the wound. A healing burned extremity with little edema resumes normal function more rapidly than does the markedly edematous extremity. Early mobilization of the injured area within 24 hours after injury limits the development of joint stiffness, a particularly challenging problem in both older persons and heavy laborers.

Routinely prescribe oral analgesics for patients with painful burns and give wound care instructions to the patient before departure. Schedule a follow-up appointment in 2-3 days.

If the patient returns to the ED for a follow-up visit, use an aseptic technique to gently remove the outer layers of the dressing to visualize the bottom (fine mesh gauze) layer. If the fine mesh gauze adheres to a relatively dry and pink burn wound, it should be covered again by layers of 4 X 4 inch coarse mesh gauze secured by roller gauze dressing. Instruct patient to return in 5-7 days for reevaluation. Because most superficial partial-thickness burns heal in 10-14 days, spontaneous separation of the gauze from the healing burn wound should be evident at the time of the next dressing change.

If the burn wound exhibits a purulent discharge, remove the fine mesh gauze and cleanse the burn wound with saline or poloxamer 188. Apply silver sulfadiazine cream twice daily to the burn wound, and dress the area with sterile roller gauze dressing. Instruct the patient to gently wash the burn wound in clean water to remove this cream before reapplying additional cream. If the topical cream is not removed completely at each dressing change, multiple layers of the cream accumulate on the burned skin and predispose the wound to infection.

Review potential complications of minor burn injury with each patient. The patient must be aware that burn wound infection is a continual threat that can cause a partial-thickness burn to be converted to a full-thickness burn. Threat of hypertrophic scar formation, as well as pigmentary skin changes, also should be discussed. Remind patient to use a sun-blocking agent over the healed wounds for at least 6 months after injury to prevent the development of permanent pigmentary changes caused by sun exposure.

Antiseptic dressings are changed mostly under general anesthesia. During the procedure ***bloodless necrectomy*** of the softened eschar is performed. Proteolytic enzymes can be used to accelerate the separation of necrotic tissues but these drugs cannot be applied to more than 10% of body area as they melt the eschar and increase intoxication.

In circulatory necrosis of large areas which compresses the body surface ***necrotomy*** is performed – longitudinal incisions through the necrotic tissues until blood wells up.

In specialized burn centers ***early necrectomy with wound closing*** is the method of choice. It is a major operation performed on day 3-5 after the trauma when the shock has been resolved and suppurative complication

has not developed. Necrotic tissues are removed completely until only healthy tissues are left which is effective prevention of toxemia and septicemia. However, the resulting wound surface is quite large and it should be closed as much as possible otherwise the patient will lose much plasma through it and infection will develop.

The wound surface is closed in the following ways:

- ***local tissue rearrangement:*** after mobilising the edges of the wound the raised flaps are placed on the wound surface and fixed with stitches. Unfortunately, this technique cannot close extensive skin defects.
- ***Free skin grafting.*** A thin layer of skin 0.6 mm deep is raised from an intact area (the thighs, shins, abdomen). They used to do it with a blade, now a dermatome is used. The flaps are placed on the wound surface. To increase the closed area the flaps are perforated and stretched. In a few days the flap takes root and starts growing covering the whole affected area.
- ***Temporary biological closing of defect.*** In this case donor or dead body skin (allodermoplasty) is used; it can also be skin taken from piglets or calves (xenotransplantation), or it can be synthetic materials (polycapronolactone, hydron, epiguard, sinkaver). Such synthetic skin stops plasma loss; it is a protective barrier against infection; it helps to gain time till gradual autodermoplasty.

When the main areas of the wound have been closed, the stage of treating deep cosmetic defects begins which uses thick skin flaps. The following techniques are used:

- full thickness skin grafting
- pedicle flap grafting
- grafting flaps with vascular peduncles with the help of microsurgery technology

Upon complete restoration of skin integuments ***the 4th stage of burn disease – convalescence –*** begins which can last for more than a year.

The natural result of wound healing is scar formation. This is very common for burn's patients. Scarring after burns very often needs plastic and reconstructive surgical treatment.

Inhalation injury

Inhalation injury has a significant impact on the survival of burn patients.

3 components:

- upper airway swelling,
- acute respiratory failure,
- carbon monoxide intoxication.

The natural history of upper airway burn injury is the development of edema that narrows the airway 12-24 hours after injury. Intubation rather than observation is recommended in patients with signs of upper airway injury, such as stridor, inspiratory grunting, wheezing, or tachypnea.

Fiberoptic bronchoscopy is a simple, safe, and accurate method of diagnosing acute inhalation injury. It reveals the anatomic level and severity of large airway injury; identification of supraglottic and infraglottic involvement is helpful in predicting ultimate pulmonary complications, also may aid in intubating patients with inhalation injury. It has been associated with the development of severe hypoxemia, especially in elderly burn patients, administer supplementary oxygen through the bronchoscope. The ventilatory system for fiberoptic bronchoscopy can deliver a warmed, humidified, and measured fraction of inspired oxygen either for spontaneous or mechanically assisted ventilation.

Further treatment includes maintenance of pulmonary toilet, relief of mechanical restriction of chest wall motion, and prevention of respiratory failure. When collagen is burned, it loses its elasticity, shortens its fibers, and becomes rigid. This can occur very quickly in fourth-degree and severe third-degree burns. When combined with accumulation of burn edema in interstitial spaces, respiratory insufficiency or ischemia of an extremity may result.

The compressive effect of a full-thickness burn of the neck may contribute to airway compromise. Without tracheostomy, tight neck eschar accentuates pharyngeal edema and draws the neck into flexion, compressing the pharyngeal airway. A vertical incision through the eschar extending from the sternal notch to the chin helps maintain a patent airway. If respiratory insufficiency is caused by a constricting eschar of the anterior thorax that limits respiratory excursion, escharotomy is imperative. Lateral incisions are made in the anterior axillary lines that extend 2 cm below the clavicle to the 9th or 10th rib. The top and bottom of the incisions are then joined to form a square across the anterior chest.

Mechanical ventilation

If respiratory failure ensues, mechanical ventilation is necessary. Airway resistance is often increased after inhalation injury resulting from edema, debris within the airway, or bronchospasm. The goal of mechanical ventilation should be to accept a slightly acidic environment (pH >7.32) to minimize the mean airway pressure required for ventilation. To keep airway pressures to a minimum, ventilator settings may need to be adjusted to slightly higher respiratory rates (16-20 breaths/min) and smaller tidal volumes (7-8 mL/kg).

Experimental evidence has demonstrated in baboons with moderate smoke inhalation that the barotrauma index (rate times pressure product) is significantly increased during regular ventilation compared with high-frequency flow interruption ventilation. Significantly greater histologic damage of pulmonary parenchyma also occurred in the group treated with conventional ventilation.

High-frequency flow interruption ventilation appears useful in its ability to recruit damaged, collapsed alveoli and keep them open in expiratory ventilation. Maintaining alveolar recruitment at low mean alveolar pressures helps minimize barotrauma and allows improved distribution of ventilation. Two retrospective studies demonstrate a decreased incidence of pneumonia and mortality in patients with inhalation injury when high-frequency percussive ventilation is used, compared with conventional "volume-limited" ventilation. Oscillating ventilator, which superimposes high-frequency ventilation onto conventional tidal volume breaths, may be an

even better method of ventilation after smoke injury. This method reduces barotrauma and aids in the removal of airway casts by causing vibratory air movement. Airway cast and plug formation can be decreased by nebulized heparin treatments (5000 U in 10 mL of normal saline every 4 hours), which inhibit fibrin clot formation in the airway.

Carbon monoxide

Carbon monoxide (CO) is present in smoke and has 280 times the affinity for hemoglobin as oxygen. Obtain a CO level in all patients with suspected inhalation injury. Patients should receive 100% oxygen until their carboxyhemoglobin (COHb) level is less than 10% because the elimination half-life for COHb depends on oxygen tension. In room air, the half-life of CO-bound hemoglobin is 4 hours. Under 100% oxygen, the half-life of CO-bound hemoglobin decreases to 45 minutes. Administration of 100% oxygen increases the gradient for oxygen binding to hemoglobin, and unbound CO is exhaled through the lungs.

Patients who have elevated COHb levels associated with a pH of less than 7.4 should be treated with hyperbaric oxygenation. Because serum COHb levels do not reflect tissue levels, evaluate clinical symptoms when considering hyperbaric oxygen therapy. These include a history of unconsciousness, the presence of neuropsychiatric abnormalities, and the presence of cardiac instability or cardiac ischemia.

Cyanide poisoning

Specific therapy for cyanide poisoning in patients with inhalation injury is another consideration. Cyanide causes tissue hypoxia by uncoupling oxidative phosphorylation by binding to mitochondrial cytochrome a-a3. Consider empiric treatment for cyanide toxicity for patients with unexplained severe metabolic acidosis associated with elevated central venous oxygen content, normal arterial oxygen content, and a low COHb level.

Chemical injuries are commonly encountered following exposure to acids and alkali, including hydrofluoric acid (HF), formic acid, anhydrous ammonia, cement, and phenol. Other specific chemical agents that cause chemical burns include white phosphorus, elemental metals, nitrates, hydrocarbons, and tar.

Most chemical agents damage the skin by producing a chemical reaction rather than hyperthermic injury. Although some chemicals produce considerable heat as the result of an exothermic reaction when they come into contact with water, their ability to produce direct chemical changes on the skin accounts for most significant injury. Specific chemical changes depend on the agent, including acids, alkalis, corrosives, oxidizing and reducing agents, desiccants and vesicants, and protoplasmic poisons. The concentration of toxic agent and duration of its contact primarily determine the degree of skin destruction. When the skin is exposed to toxic chemicals, its keratinous covering is destroyed, and underlying dermal tissues are exposed to continuous necrotizing action.

Both inorganic and organic acids denature the proteins of the skin, resulting in a coagulum, the color of which depends on the acid involved. Nitric acid burns result in a yellow eschar, whereas sulfuric acid eschar is black or brown. Burns caused by hydrochloric acid or phenol tend to range from white to grayish-brown. Following the initial exposure, cellular dehydration and further protein denaturation/coagulation occur. This dehydrative effect results in the characteristic dry surface of acid burns. The methods of neutralization used in the treatment of acid burns depend on the nature of the acid.

Alkali burns are those caused by lime (cement), ammonia, and caustics (sodium hydroxide, potassium hydroxide). Alkali dissolves protein and collagen, resulting in alkaline complexes of these molecules. Cellular dehydration (as in acid burns) and saponification of fatty tissue also occurs. Whereas acid burns are characterized as "dry" burns, with little fluid loss or edema, alkali burns present with marked edema and extensive fluid loss. Neutralization of alkali exposure is accomplished by first irrigating the burned site with a large amount of water to dilute any unreacted alkali remaining on the wound surface. This protects the wound from further damage caused by heat released during the neutralization reaction.

After skin contact, absorption of some agents may cause systemic toxicity. Dichromate poisoning produces liver failure, acute tubular necrosis, and death. Oxalic acid and HF injuries may result in hypocalcemia. Tannic and

phosphorous burns may be followed by nephrotoxicity. Absorption of phenol may be associated with CNS depression and hypotension. Inhalation injury may result from exposure to toxic fumes, particularly when the exposure occurs within a closed space.

Chemical burns continue to destroy tissue until the causative agent is inactivated or removed. For example, when hydrotherapy is initiated within 1 minute after skin contact with either an acid or alkali, severity of the skin injury is far less than when treatment is delayed for 3 minutes. Early treatment is followed by a return of skin pH to normal. When contact time exceeds 1 hour, the pH level of a sodium hydroxide (NaOH) burn cannot be reversed. Similarly, brief washing of a hydrochloric acid (HCl) burn more than 15 minutes after exposure does not significantly alter acidity of damaged skin.

Because contact time is a critical determinant of the severity of injury for skin exposed to a toxic liquid chemical, an exposed person or a witness to the injury must initiate hydrotherapy immediately. When workers' clothes are soaked with such agents, valuable time is lost if their clothing is removed before copious washing commences. Gentle irrigation with a large volume of water under low pressure for a long time dilutes the toxic agent and washes it out of the skin. During hydrotherapy, the rescuer should remove the patient's clothes and wear powder-free, latex-free, emergency medical examination gloves to prevent hand contact with the chemicals.

Water is the agent of choice for decontaminating acid and alkali skin burns.

Alkali substances are the most toxic chemicals, and anhydrous ammonia appears to be the worst offender. Even alkali burns that seem mild can result in devastating injury, because alkalis tend to react with the lipid in corneal epithelial cells to form soluble soap that penetrates corneal stroma. Alkali moves rapidly through the stroma and endothelial cells to enter the anterior chamber. Anhydrous ammonia can penetrate the anterior chamber in less than 1 minute.

Alkali usually kills each tissue layer of the anterior segment of the eye that it contacts. This results in occlusive vasculitis around the corneoscleral limbus, which makes repair of these tissues difficult. As the tissues of the

anterior segment of the eye degenerate, perforation follows with the development of endophthalmitis and loss of the eye. If perforation can be prevented, recovery of sight may be possible through eventual corneal transplantation. Recent experimental studies conclude that destruction of corneal stroma can be minimized by drug therapy (eg, N-acetylcysteine, steroids). However, drug therapy has limited therapeutic usefulness because of the need for frequent applications, significant number of clinical failures, and potential adverse effects.

Electricity is the flow of electrons from atom to atom. Movement of electrons is comparable to the way water is passed along in a bucket brigade. Electrons, which comprise the current, are passed along from atom to atom. Amperage is the term used for the rate of flow of electrons. Every time 6.242 x 1015 electrons pass a given point in 1 second, 1 ampere of current has passed. The current is what can kill or hurt a victim of an electric injury. One ampere is roughly equivalent to the amount of current flowing through a lighted 100-watt light bulb. In most materials, a number of electrons are free to move about at random until a driving force termed voltage propels them to move in one direction. A large voltage exerts a greater force, which moves more electrons through the wire at a given rate of time. Electric voltage of 380 volts or less is considered low voltage and above 380 volts, high voltage. High voltage is generated at the power plant and is transformed down to approximately 120 volts for most wall outlets in homes.

Resistance of the human body has been likened to that of a leather bag filled with an electrolyte fluid, with high resistance on the outside and lower inside. Skin resistance also varies depending on moisture content, thickness, and cleanliness. Resistance offered by the callused palm may reach 1,000,000 ohms/cm2, while the average resistance of dry normal skin is 5000 ohms/cm2. This resistance may decrease to 1000 ohms/cm2 if hands are wet. Skin resistance is encountered primarily in the stratum corneum that serves as an insulator for the body. The voltage gradient in skin cannot be increased indefinitely and breaks down at low voltages. Exposure of the skin to 50 volts for 6-7 seconds results in blisters that have a considerably diminished resistance.

The dermis offers low resistance, as do almost all internal tissues except bone, which is a poor conductor of electricity. Other factors that affect the flow of electrons are the nature and size of the substance through which it passes. If the atomic structure of the material is such that the force of attraction between its nucleus and outer electrons is small, little force is required to cause electron loss. These substances (eg, copper, silver) in which electrons are loosely bound are termed conductors, because they readily permit the flow of electrons. Materials such as porcelain and glass are composed of atoms that have powerful bonds between their nuclei and the outer electrons. These materials are termed insulators because electron flow is restricted.

Resistance is a measure of how difficult it is for electrons to pass through a material and is expressed in a unit of measurement termed an ohm. The resistance offered to the flow of electricity by any material is directly proportional to its length and inversely proportional to its cross-sectional area. Electricity is transmitted by a high-voltage system, because it allows the same amount of energy to be carried at lower current, which reduces electrical loss through leakage and heating. The relationship between current flow (amperage), pressure (voltage), and resistance is described in Ohm's law, which states that the amount of current flowing through a conductor is directly proportional to voltage and inversely related to resistance.

Current (I) = Voltage (E)/Resistance (R)

Electrons set in motion by the current force (voltage) may collide with each other and generate heat. The amount of heat developed by a conductor varies directly with its resistance. Power (watts) lost as a result of the current's passage through a material provides a measure of the amount of heat generated and can be determined by Joule's law.

Power (P) = Voltage (E) x Current (I)

Because E = I x R (resistance), the above equation becomes P = I(squared) R. Consequently, the heat produced is proportional to the resistance and the square of the current. Commercial electric currents usually are generated with a cyclic reversal of the direction of electric pressure (voltage). Pressure in the line first pushes and then pulls electrons, resulting in alternating current. Frequency of current in hertz (Hz) or cycles per second is the

number of complete cycles of positive and negative pressure in 1 second. The usual wall outlet (120 volts) provides a current with 120 reversals of the direction of flow occurring each second and is termed 60-cycle current. Frequency of alternating current can be increased to a range of millions of cycles per second. In direct current, electron travel is always in the same direction.

Alternating current has almost entirely superseded direct current, since it is cheaper and can be transformed easily into any required voltage. Most machines in industry and appliances in the home use alternating currents; therefore, workers and consumers are mainly at risk from this current. Direct current usage is primarily restricted to the chemical and metallurgical industries, ships, streetcar systems, and some underground train systems.

Electric arc

Contact with high-voltage current may be associated with an arc or light flash. An electric arc is formed between two bodies of sufficiently different potential (high-voltage power source and the body, which is grounded). The arc has an intense, pale-violet light and consists of ionized particles that are driven by the voltage pressure between the two bodies and are present in the space between them. Temperature of the ionized particles and immediately surrounding gases of the arc can be as high as 4000°C (7232°F) and can melt bone and volatilize metal. As a general guide, arcing amounts to several centimeters for each 10,000 volts. Burns occur where portions of the arc strike the patient. The electric arc remains the cause of most high-voltage electrical burn injuries. Because of its high frequency, the electric arc has become the basis for many standard safety precautions.

Effects of electricity on the body

Effects of electricity on the body are determined by 7 factors:

- type of current,
- amount of current,

- pathway of current,
- duration of contact,
- area of contact,
- resistance of the body,
- voltage.

Low-voltage electric currents that pass through the body have well-defined physiologic effects that are usually reversible. For a 1-second contact time, a current of 1 milliampere (mA) is the threshold of perception, a current of 10-15 mA causes sustained muscular contraction, a current of 50-100 mA results in respiratory paralysis and ventricular fibrillation, and a current of more than 1000 mA leads to sustained myocardial contractions.

Humans are sensitive to very small electric currents because of their highly developed nervous system. The tongue is the most sensitive part of the body. Using pure direct current and 60-cycle alternating current, the first sensations are those of taste, which are detected at 45 microamperes. When subjected to 60-cycle alternating current, the threshold of perception in the hands of men and women, which is usually a tingling sensation, is approximately 1.1 mA. Using pure direct current applied to hands, the first sensations are those of warmth in contrast to tingling, detected at 5.2 mA.

Skin offers greater resistance to direct current than alternating current, thus 3-4 times more direct current is required to produce the same biologic effect elicited by alternating current. With increasing alternating current, sensations of tingling give way to contractions of muscles. The magnitude of the muscular contractions enhances as the current is increased. Finally, a level of alternating current is reached for which the subject cannot release the grasp of the conductor. The maximum current a person can tolerate when holding a conductor in one hand and still let go of the conductor using the muscles directly stimulated by the current is termed the "let-go" current. This tetanizing effect on voluntary muscles is most pronounced in the frequency range of 15-150 Hz. Such strong muscular reactions may cause fractures and/or dislocations. Numerous reports of bilateral scapular fractures and shoulder dislocations and fractures in electric accidents attest to this occurrence. As the frequency

increases above 150 Hz, the potential for this sustained contraction is lessened. At frequencies from 0.5-1 megacycle, these high-frequency currents do not elicit sustained contractions of the skeletal muscles. For 60-cycle alternating current, the let-go threshold for men and women is 15.87 mA and 10.5 mA, respectively. The lower value for women may result from their generally somewhat poorer muscular development compared to men.

Electrical accidents involving power frequency (50-60 Hz) and a relatively low voltage (150 V/cm) occasionally can result in massive trauma to the victim. Skeletal muscle and peripheral nerve tissue are especially susceptible to injury. Historically, Joule heating, or heating by electrical current, was viewed as the only mechanism of tissue damage in electrical trauma. Yet in some instances, Joule heating does not adequately describe the pattern of injury observed distant to the sites of contact with the electrical source. These victims exhibit minimal external signs of thermal damage to the skin, while demonstrating extensive muscle and nerve injury.

Recently, electroporation of skeletal muscle and nerve cells was suggested as an additional mechanism of injury in electrical burns. This mechanism is different from Joule heating, even though it is influenced by temperature. It is the enlargement of cellular-membrane molecular-scale defects that results when electrical forces drive polar water molecules into such defects. Experimental studies have documented that electroporation effects can mediate significant skeletal muscle necrosis without visible thermal changes.

High-voltage accidents

The national electric code defines high-voltage exposure as greater than 600 volts. In the medical literature, high-voltage exposure is judged as greater than 1,000 volts. In high-voltage accidents, the victim usually does not continue to grasp the conductor. Often, he or she is thrown away from the electric circuit, which leads to traumatic injuries (eg, fracture, brain hemorrhage). The infrequency with which sustained muscular contractions occur with high-voltage injury apparently occurs because the circuit is completed by arcing before the victim

touches the contact. Currents that cause subjects to "freeze" to the circuit despite their struggle to be free are frightening, painful, and hard to endure, even for a short time.

Turning off power source

Consequently, a witness of the accident must turn off the power source as soon as possible. If this is not possible, the victim must be disengaged from the electric current. Wearing lineman's gloves, trained electricians must separate the victim from the circuit by a specially insulated pole. Looping a polydacron rope around the injured patient is another method of pulling him or her from the electric power source. Ideally, the first responder should stand on a dry surface during the rescue.

Muscular contractions

Tests using gradually increasing amounts of direct current produce sensations of internal heating rather than severe muscular contractions; however, sudden changes in the magnitude of direct current produce powerful muscular contractions. At the instant of interruption of the direct current, the subject occasionally falls back a considerable distance; the impact of the fall may cause a fracture. As the alternating current strength increases above 20 mA, a sustained contraction of muscles of respiration of the chest occurs.

Normal respiration returns after the current has been turned off, provided that the duration of current flow is less than 4 minutes. If sustained contractions last longer than this time interval, death from asphyxiation occurs, unless the current is stopped and mouth-to-mouth ventilation on the breathless patient is started. The pathway of current flow, involved in tetanic contractions of the muscles of respiration, is usually arm to arm or arm to leg and does not pass through the respiratory center located in the medulla of the brainstem. This center is injured in executions in the electric chair, leading to permanent respiratory arrest.

Treatment at the scene

When current flow increases above 30-40 mA, ventricular fibrillation may be induced. Numerous factors can influence the magnitude of electric current required to produce ventricular fibrillation. Factors found to be of

primary importance are duration of current flow and body weight. The threshold for ventricular fibrillation is inversely proportional to the square root of the shock duration and directly proportional to body weight.
When the heart is exposed to currents of increasing strength, its susceptibility to fibrillation first increases and then decreases with even stronger currents. At relatively high currents (1-5 amps), the likelihood of ventricular fibrillation is negligible with the heart in sustained contraction. If this high current is terminated soon after electric shock, the heart reverts to normal sinus rhythm. In cardiac defibrillation, these same high currents are applied to the chest to depolarize the entire heart.
If disconnecting the victim from the electric circuit does not restore pulses, the first responder must start cardiopulmonary resuscitation to restore breathing and circulation. Ideally, when they arrive at the scene of the accident, paramedics will continue this resuscitation. Field intervention should include advanced life support treatments delivered under the direction of a physician at the hospital base station using telemetered communication. Telemetered monitoring of these patients is recommended throughout transport to the advanced life support hospital facility.
These life-threatening consequences of low-voltage electric burns usually occur without any lesions of the skin at entrance and exit points of the current. An absence of local lesions indicates that the surface area of contact (current density) is large and that the heat is insufficient to produce a thermal injury; however, the smaller the surface area of the contact, the greater the density of the current and the more energy is transformed into heat that can cause local burn injury.
Severity of bony injury
Severity of bony injury from a low-voltage electric burn may vary considerably. Injury may be isolated to a single tooth or involve many orofacial structures. Composite dysplasia involving both dentine and enamel of a tooth may be evident. The injured tooth has a vital pulp, a shorter root than crown, and a wrinkled, pitted, brownish labial surface. Abnormal growth of the orofacial structures causes dental changes, such as crossbite,

crowding, and retrusion of the bite. Severity of electric burns of the mouth can be categorized into 3 groups: minor, moderate, and severe.

1. Minor injury involves less than one third of either the upper and lower lip without commissure injury or less than one sixth of both lips with commissure injury.
2. Moderate injury affects more than one third of either lip without commissure injury or more than one sixth of each lip with commissure injury. Moderate injuries are limited to skin around the lips with minimal mucosal loss and do not involve the buccal sulcus.
3. Severe injury includes a significant loss of skin and muscle and mucosal involvement of buccal sulcus.

In burns from an electric arc, the current courses external to the body from the contact point to the ground. Circumscribed burns occur where the portions of the arc contact the patient. These contact points may be multiple, single, or diffuse and vary in their depths. The most common contact points for the current are the hands and skull, while the most common ground areas are the heels. Entry points on the flexor surfaces often produce "kissing" entry lesions, resulting from severe tetanic muscle contractions and causing extensive tissue damage. The most common of these lesions is the circumscribed deep wound on the volar surface of the forearm in association with contact wounds of the palm. A flame may complicate this burn injury if the flashes of an arc ignite the victim's clothing.

The other burn injury is from an electric current that passes between the power source and the anatomic point of contact (entrance wound), and between the patient (exit wound) and the grounding mechanism, causing hidden destruction of deeper tissues. Such electrically conductive burns are simply thermal injuries occurring when the electric energy is converted to thermal energy. The extent of the electric burn is related to the magnitude, frequency, and duration of the current flow and the volume and resistance of the tissue.

Resistance of living tissue changes as the current flows. Skin represents an initial barrier to flow of current and serves as insulation to the deeper tissues. Once an electric current contacts skin, the amperage rises slowly, followed by an abrupt and rapid climb. This change in flow coincides with a progressive decline in skin

resistance. Once this skin resistance breaks down, current enters the underlying tissue whose internal tissue resistance, with the exception of bone, is negligible to current flow. Within seconds, electric current in tissue peaks and then falls precipitously to zero. Current ceases to flow when the heat-producing tissue carbonization (eschar) volatilizes tissue fluid. Termination of current flow is signaled by the appearance of an arc or flash.

Current pathways

Low-voltage current generally follows the path of least resistance (ie, nerves, blood vessels), yet high-voltage current takes a direct path between entrance and ground. The volume of soft tissue through which current flows behaves as a single uniform conductor, thus is a more important determinant of tissue injury than the internal resistance of the individual tissues. Current is concentrated at its entrance to the body, then diverges centrally, and finally converges before exiting. Consequently, the most severe damage to the tissue occurs at the sites of contact, which are commonly referred to as the entrance and exit wounds.

Entry and exit wounds

High-voltage electric entry wounds are charred, centrally depressed, and leathery in appearance, while exit wounds are more likely to "explode" as the charge exits. High-voltage electrical burns often leave a black metallic coating on the skin that is mistaken for eschar, from vaporization of the metal contacts and electroplating of the conductive skin surface. Cleansing of the coating usually reveals only superficial skin injury. Electric current chooses the shortest path between the contact points and involves the vital structures in its pathway. Fatalities are high (nearly 60%) in hand-to-hand current passages and are considerably lower (20%) in hand-to-foot current passages. Severity of damage to the tissue is greatest around the contact sites.

Consequently, anatomic locations of the contact sites are critical determinants of injury. Most of this underlying tissue damage, especially muscle, occurs at the time of initial insult and does not appear to be progressive. Microscopic studies of electric burns demonstrate that this initial destruction of tissues is not uniform. Areas of total thermal destruction are mixed with apparently viable tissue. Between the entrance and exit points of the

electric current, widespread anatomic damage and destruction may be seen. An electric current can injure almost every organ system.

Complications

- Sequelae peculiar to this type of injury are important determinants in the choice of therapy. Various complications related to the damage to the various organ systems are now clearly identifiable.
- Characteristic entry and exit wounds in extremities usually signal local destruction of deeper tissues, the magnitude of which often cannot be predicted.
- Bone has a high resistance, thus readily transforms current to heat production, which may result in periosteal necrosis or even melting of the calcium phosphate matrix.
- This injured underlying tissue has several consequences.
- Necrosis of the entire limb is the most serious complication, necessitating amputation usually within 2-3 days after injury.
- More commonly, the extent of underlying tissue injury involves a portion of the superficial and deep muscles of one or more compartments. Vessels within these electrically injured tissues exhibit increased vascular permeability. This permeability change allows extravasation of fluids into the wounds, resulting in a reduction in intravascular volume that must be corrected by an intravenous (IV) infusion of Ringer lactate solution without glucose.

Treatment

The quantity of fluid sequestered in the injured tissue usually cannot be estimated using skin surface measurement, because the magnitude of damage to the underlying tissue often is grossly underestimated. Consequently, titrate the quantity of fluid administration to maintain an adequate urinary output.

In contrast to flame injury, completion of fluid resuscitation can be predicted by the patient's hematocrit and plasma volume. When extracellular fluid is restored, the hematocrit and plasma volume returns to normal, if significant hemolysis has not occurred.

In acute electric injuries in the adult with underlying devitalized tissue, administer Ringer lactate solution without glucose at a rate sufficient to maintain a urinary output of 50-100 mL/h.
In the presence of hemochromogens in the urine, the rate of fluid infused must be sufficient to maintain a minimum urinary output of 100 mL/h. This rate and volume of fluid administration is continued until the urine is free of pigment. Alkalization of the urine by adding sodium bicarbonate to the IV fluid increases the solubility and clearance rate of myoglobin in the urine.
Transfusions are unnecessary during the first 24 hours unless multiple escharotomies and/or fasciotomies result in significant blood loss. An almost immediate loss of intravascular fluid into an electrically burned extremity results in considerable swelling of the muscle lying within a relatively inelastic fascial compartment. Such intense swelling of the injured muscle may cause noticeable changes in the circumference of the extremity. More frequently, fascial investments may limit the swelling to such a degree that minimal external enlargement of the limb occurs.

Rehabilitation.

The period immediately after discharge from the burn unit is often extremely difficult for patients and their families. In fact, for many burn patients, the first 18 months after discharge is more difficult than the acute stay. The principal rehabilitation goals at this time include the following:

- Progressive ranging and strengthening
- Evaluation of evolving problem areas
- Specific postoperative therapy after reconstructive operations
- Scar management.

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