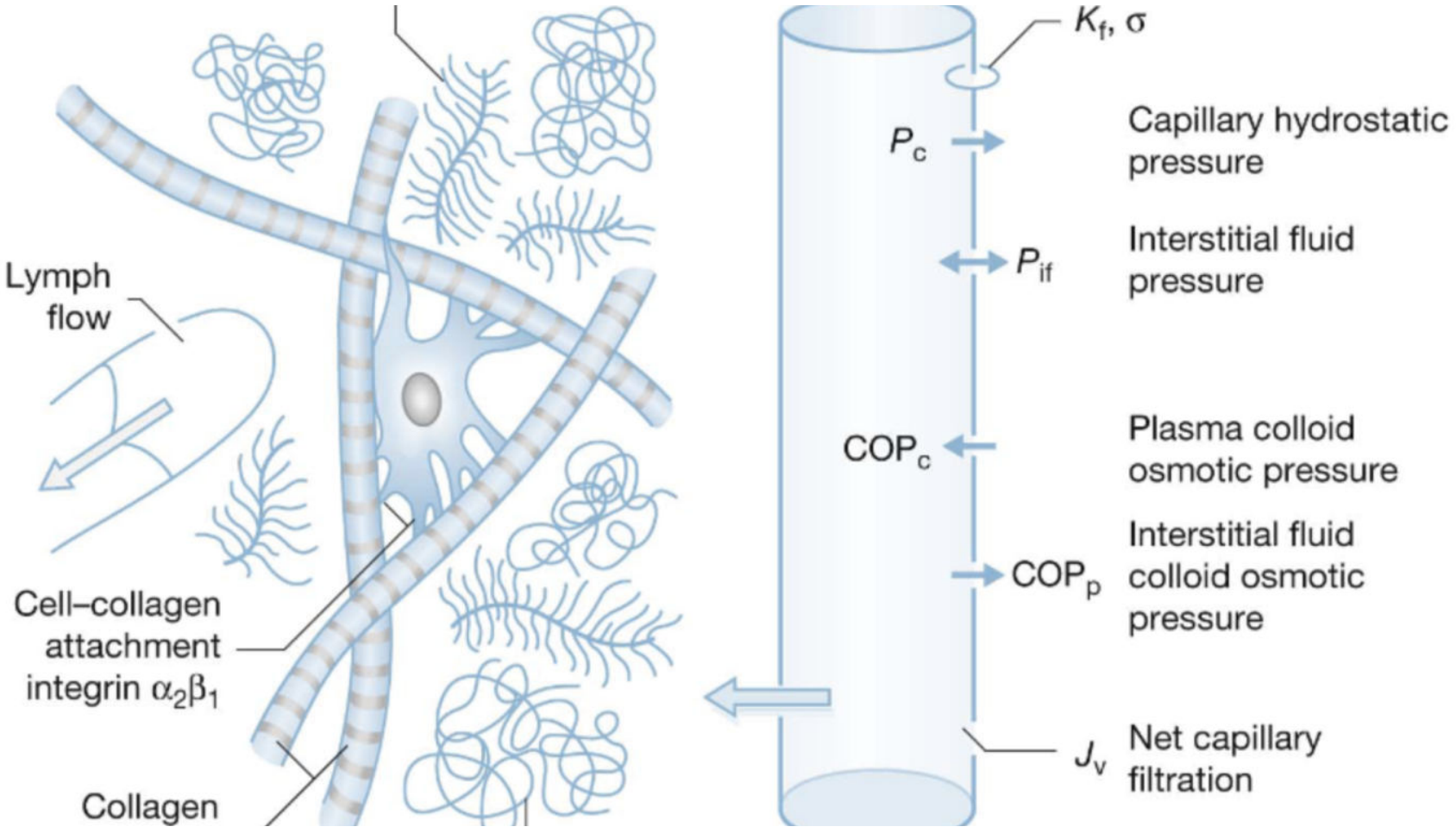
Scalds Electrical Burns, Chemical Burns, Radiation Burns, Frostbite

Dept. Of Burns & Plastic Surgery

$$J_v = K_f \left[(P_c - P_{if}) - \sigma \left(\pi_p - \pi_{if} \right) \right]$$

J_v is the volume of fluid that crosses the microvasculature barrier.
K_f is the capillary filtration coefficient, which is the product of the surface area and hydraulic conductivity of the capillary wall
P_c is the capillary hydrostatic pressure
P_{if} is the interstitial fluid hydrostatic pressure
π_p is the colloid osmotic pressure of plasma
π_{if} is the colloid osmotic pressure of interstitial fluid
σ is the osmotic reflection coefficient.
Edema occurs when the lymphatic drainage (J_L) does not keep pace with the increased J_v

Landis Starling Equation

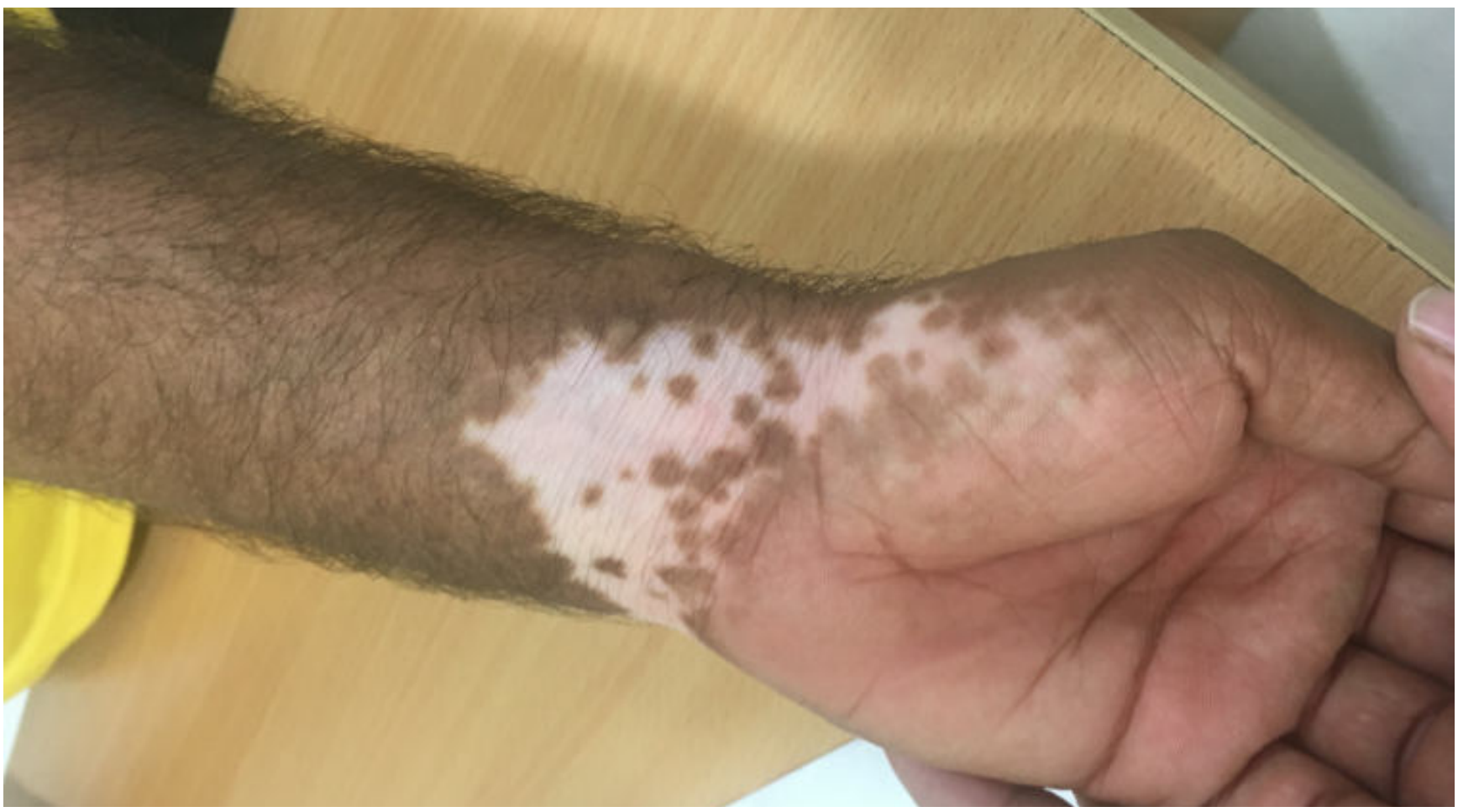


Variable	Normal or baseline	Post-burn	Δ
P_c	~25 mmHg	~50 mmHg	↑ ~25 mmHg
Π_P	20–28 mmHg	15 to 18 mmHg	↓ ~10 mmHg
P_i	−2 to 0 mmHg	~100 mmHg non-resuscitated non-perfused skin and −5 mmHg perfused skin	↓ ~100 mmHg ↓ 3–5 mmHg
Π_{if}	10–15 mmHg	13–18 mmHg in burn wound ↓ and with resuscitation hypoproteinemia in non-burned skin	↑ ~3 mmHg
σ	~0.9	~0.5	↓ ~0.4
K_f	~0.003 mL/min/mmHg/100 g (leg)	↑ 2–5×	

Skin Biology & Response to Burns

Epidermis

- Derived from ectoderm so is capable of regenerative healing.
- Keratinocytes proliferate from dermal appendages leading to re-epithelialization.
- Depleted melanocytes regenerate slowly leading to pigmentary changes
- Loss of anchoring collagen fibrils (type7) leads to blister formation



Skin Biology & Response to Burns

Dermis

- Superficial papillary
- Deep reticular
- Fibroblasts produce collagen fibrils and elastic fibers
- Dermal appendages lined by keratinocyte derived epidermal cells
- Contain capillary plexus and sensory nerves
- Derived from mesoderm so heal by scarring

Patho-phyisological changes

Jackson Zones

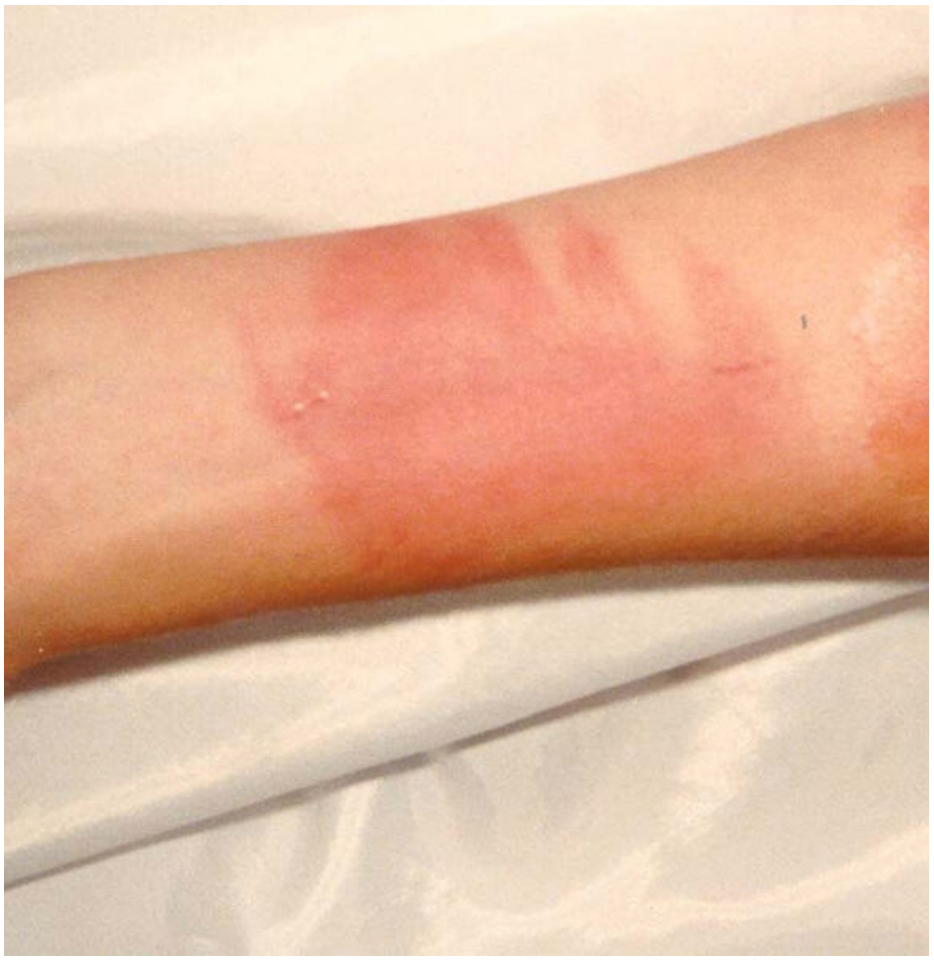
- Temperature and duration of contact have a synergestic effect
- 1s at 69^{O}C / 1 hour at 45^{o}C
- **Zone of coagulation**– Centre of the wound
- **Zone of stasis** – At risk area with mix of viable and non viable cells along with vasoconstriction
- **Zone of hyperemia**- Viable cells with vasodialtion

- Protection of zone of stasis is achieved with adequate fluid resuscitation, avoidance of vasoconstrictors and prevention of infection

Depth of Burn

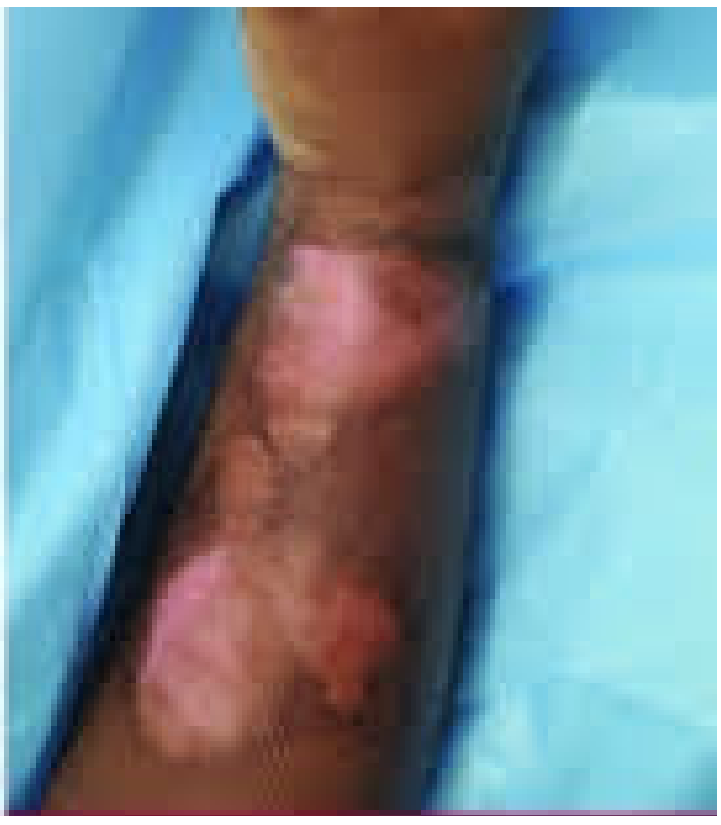
1st Degree

- Burns involving only the epidermis.
- Erythematous and very painful but do not form blisters.
- Sunburns fit this category of superficial, epidermal injury.
- Within 3–4 days, the dead epidermis sloughs and is replaced by regenerating keratinocytes.



2nd degree (Superficial dermal burns)

- Extend into the papillary dermis and characteristically form blisters.
- Appearance is pink, wet and hypersensitive to touch.
- Painful as uncovering the wound allows currents of air to pass over it.
- These wounds blanch with pressure as the blood flow to the dermis is increased due to vasodilation.
- Superficial dermal burns usually heal within 2–3 weeks without risk of scarring and therefore do not require operation.

3rd degree (Deep Dermal Burns)

- Extend into the reticular dermis and generally will take 3 or more weeks to heal.
- They also blister, but the wound surface appears mottled pink and white
- The patient complains of discomfort and pressure rather than pain.
- When pressure is applied to the burn, capillaries refill slowly
- **Partial-thickness burns that are predicted not to heal by 3 weeks should be excised and grafted.**

4th Degree (Full Thickness)

- Full-thickness burns involve the entire dermis and extend into subcutaneous tissue.
- Their appearance may be charred, leathery, firm, and depressed when compared to adjoining normal skin.
- These wounds are insensitive to light touch and pinprick.
- Non-charred full-thickness burns can be deceptive as they may have a mottled appearance
- Must be excised and grafted early

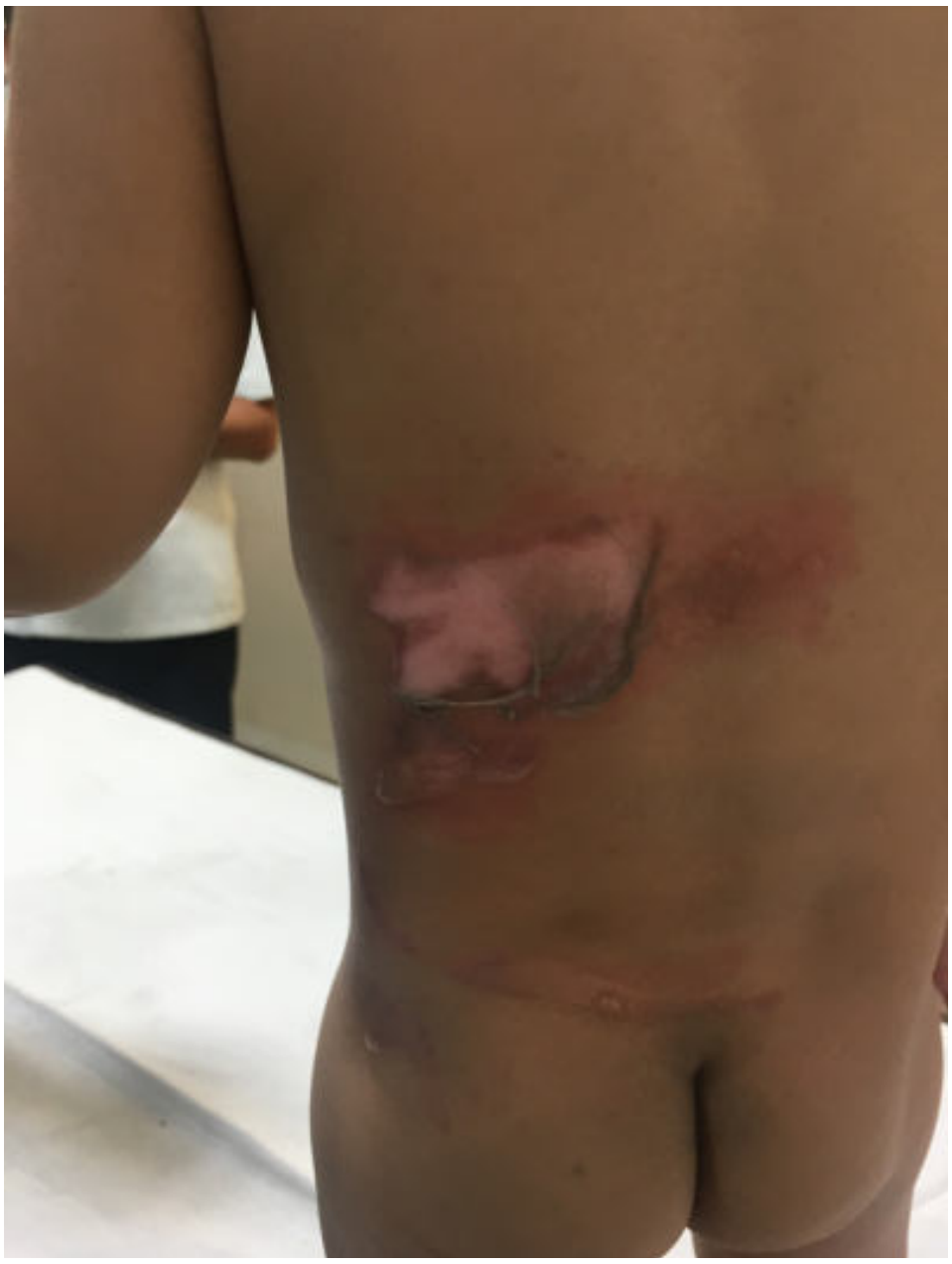
SCALD INJURY

Scald Injury

- The depth of scald injury depends on the water temperature, the skin thickness and the duration of contact.
- Boiling water often causes a deep dermal burn, unless the duration of contact is very short.
- Soups and sauces, which are thicker in consistency, will remain in contact longer with the skin and invariably cause deep dermal burns.
- Exposed areas tend to be burned less deeply than clothed areas.
- Clothing retains the heat and keeps the liquid in contact with the skin longer.

Scalds are a mosaic of dermal burns

- A common example is a toddler who reaches above head level and spills hot water on himself. His face bears a superficial burn, his trunk burn is of indeterminate thickness, and his skin under his diaper has a deep dermal burn.



Immersion Scalds

- Immersion scalds are often deep because of the prolonged skin exposure.

They occur in individuals who

- Cannot perceive the discomfort of prolonged immersion (i.e. a diabetic patient soaking his foot in hot water)
- Those who are not able to escape from the hot water (i.e. young children, the elderly, or people with physical and cognitive disabilities).

Atypical Scalds

- Grease and cooking oils cause deep dermal and full thickness injuries
- Tar and asphalt are ‘special scald’ injuries because they have to be first removed before the depth of wound can be assessed
- Tar can be removed by application of petroleum-based ointment under a dressing.
- The dressing is changed and ointment reapplied every 2–4 h until the tar has dissolved

RADIATION BURN

Radiation Injuries

- Damage to biological tissue by ionizing radiation is due to

1) Electromagnetic radiation (e.g. X-rays and gamma rays)

2) Particulate radiation (e.g. alpha and beta particles or neutrons).

- The severity of tissue damage is determined by the energy deposited per unit track length, known as Linear Energy Transfer (LET).

How to measure Radiation Injury

- Electromagnetic radiation passes through tissue almost unimpeded by the skin and are called low LET since little energy is left behind.
- Neutron exposure has high-LET, resulting in significant energy absorption within the first few centimeters of the body.
- Alpha and low-energy beta particles do not penetrate the skin, and represent a hazard only when internalized by inhalation, ingestion or absorption through a wound.

How to measure Radiation Injury

- The biological effect of radiation is measured by rad (Radiation Absorbed Dose)
- 1 Gy= 100 rad
- Is 1Gy of X Ray = 1 Gy of Neutron??
- rem(Roentgen Equivalent Man)= dose in rads*Quality Factor(QF)
- QF takes into account linear energy transfer so QF for X ray=1 & neutron=10
- 1 (Sv) Seivert= 100 rem
- **So now 1Sv of X ray= 1Sv of neutron**

Incidence of radiation injury

The majority of radiation accidents are from

1) Radiation devices such as accelerator
2) Highly radioactive sources used for industrial radiography.
3) Radioisotope accidents involving radioactive materials which are unsealed, such as tritium, fission products, radium and free isotopes used for diagnosis and therapy.

How does Radiation Injury Occur?

Radiation accident is defined as

- Whole body doses >25 rem (0.25 Sv)
- Skin doses > 600 rem (6 Sv)
- Absorbed dose > 75 rem (0.75 Sv) to other tissues or organs from an external source
- Internal contamination >one-half the maximum permissible body burden (MPBB) as defined by the International Commission on Radiological Protect (different for each radionuclide)

Radiation Effect

- Ionizing radiation causes formation of free radicles which injure DNA, nuclear and cellular membrane
- Cells are most sensitive when undergoing mitosis so that those that divide rapidly such as bone marrow, skin and the gastrointestinal tract are more susceptible to radiation damage.
- Radiation to an organ such as brain or liver, which has parenchymal cells with a slow turnover rate, results in damage to the more sensitive connective tissue and microcirculation.

Localized Injury-Skin

- Erythema is equivalent to a first-degree thermal burn and occurs in two stages.

1) Mild erythema appears within minutes or hours following the initial exposure and subsides in 2–3 days.
2) The second onset of erythema occurs 2–3 weeks after exposure and is accompanied by dry desquamation of the epidermal keratinocytes.

- Epilation may occur as soon as 7 days post injury.
- It is usually temporary with doses less than 5 Gy but may be permanent with higher doses.

Localized Injury-Skin

- Moist desquamation is equivalent to a second-degree thermal burn and develops after a latent period of about 3 weeks with a dose of 12–20 Gy.
- The latency period may be shorter with higher doses.
- Blisters form, which are susceptible to infection if not treated.
- Full-thickness skin ulceration and necrosis are caused by doses in excess of about 25 Gy

ARS

- Physiological effects of whole-body radiation are described as the acute radiation syndrome (ARS).
- Prodromal symptoms include nausea, vomiting, diarrhea, fatigue, fever and headache.
- There then follows a latent period, the duration of which is related to the dose.
- Hematopoietic and gastrointestinal complications follow this.

ARS

Three Sub Syndromes of ARS-

- **Hematopoietic syndrome**

Opportunistic infections result from the granulocytopenia and spontaneous bleeding results from thrombocytopenia.

- **Gastrointestinal syndrome**

Epithelial damage results in loss of transport capability, bacterial translocation with sepsis, bowel ischemia and bloody diarrhea.

- **Neurovascular syndrome**

Due to endothelial injury there is release of NO and other vasodilator mediators.

This leads to neurological symptoms, respiratory distress, cardiovascular collapse and death.

Treatment

- First aid (airway, breathing , circulation)
- Irrigation with running water until Geiger Muller counter shows minimum radiation count
- Exposures >100 rem require full evaluation in hospital.
- Patients with exposures >200 rem or who have symptoms of ARS should preferably be sent to specialist centers with facilities to treat bone marrow failure.

Treatment..

- Burn wound is managed as per the depth of wound
- Pain is severe and can be treated by opiates
- Radiation injury causes severe nausea and vomiting which can be managed by ondensetaron (safe in children)
- Diethylene triamine pentaacetic acid (DTPA) and intravenous administration of DTPA for workers exposed to plutonium

FROSTBITE

Frostbite

- Traumatic injury caused by the failure of normal protective mechanisms against the thermal environment, resulting in local tissue temperatures falling below freezing
- At risk populations are

Mentally ill

Homeless

Alcohol and Drug Intoxications

Wilderness activities(Treking /Camping)

- Acral areas are typically affected

Pathophysiology

Direct Cellular Damage

- Intracellular formation of Ice crystals

D/t rapid cooling and leads to severe cell injury

Histamine release occurs which causes flushing and formation of blisters

- Extracellular formation of Ice crystals

D/t slow cooling and leads to injury to cell membrane

Leads to gradual dehydration of cell as osmotic imbalance occurs

Pathophysiology

Microvascular Occlusion

- Cold injury causes vasoconstriction
- With rewarming capillary blood flow resumes but with presence of microemboli
- Certain areas close to injury will have complete cessation of blood flow within 20 min
- The remaining area is at risk due to endothelial injury and accumulation of inflamatory mediators similar to Jackson Model

Classification

- **First-degree injury**

It is superficial, without formation of vesicles or blebs.

There may initially be an area of pallor with surrounding erythema.

- **Second-degree**

Partial dermal involvement with generally favourable prognosis

Associated with light-colored blisters and subsequent epidermal sloughings.

- **Third-degree**

Has dark or hemorrhagic blisters that evolve into thick, black eschar over 1–2 weeks.

- **Fourth-degree**

Injury involves bone, tendon or muscle and uniformly results in tissue loss.

Management

- Rewarming in the field should not be pursued unless the ability to maintain the affected tissue in a thawed state is certain
- Injured areas should be mechanically protected from trauma because they are typically insensate and are at high risk for further injury
- Management of Hypothermia (Temp<32) should be started before Management of frostbite

Management

- Rewarming is done with the help of water bath at temperatures of 40- 42^{o}C
- Duration of rewarming is usually 30 min
- Clinically until sensation returns and flushing in the most distal part of tissue
- Blisters may be debrided as they help in assessment of the deeper tissue
- Blisters contain high amount of $PGF_{2Aalpha}$ &TXA_2

Non Surgical Management

- Systemic NSAIDs and topical aloe vera to address the inflammatory chemokines coupled with systemic penicillin as prophylaxis against Gram-positive infection
- Pentoxifylline improves red blood cell flexibility, which may limit microvascular sludging and thereby diminish thrombus formation in small vessels.

Role of Thrombolytics

- Use of Thrombolytic therapy can enhance survival of digits
- t-PA only appears to be efficacious within 24 hours of thaw, meaning that this may not be an option for patients who are injured in extremely remote environments.
- Additionally, although digit salvage has been improved with thrombolytics, the actual long-term functional results of this salvage have not been documented.

HBO

- Studies of hyperbaric oxygen are limited but have some of the most promising functional results of an adjunctive therapy for frostbite.
- One of the early documented uses of HBO for therapy in frostbite involved four Alpine mountaineers, all of whom presented 10 or more days following injury and all of whom demonstrated good tissue preservation with HBO

Management

- Prevalent clinical practice remains to time surgery anywhere from 4 weeks to 3 months following injury, once tissues have clearly demarcated to an experienced clinical eye.

CHEMICAL BURNS

Chemical Burns

- The 3D structure of biological proteins depends on hydrogen bonding and weak Van der walls forces
- Chemicals substances can destabilize a protein and alter its function by changing the pH or dissolving the lipids thus altering the hydrogen bonding
- Thermal injury also occurs by breaking the hydrogen bonds and causing protein denaturation

Severity of Chemical Burn

- Quantity of Chemical Agent
- Concentration of Chemicals
- Manner and Duration of skin contact
- Extent of penetration
- Mechanism of action

Mechanism Of Action

1.*Reduction*: Act by binding free electrons in tissue proteins, causing denaturation.

Eg Alkyl mercuric compounds, Ferrous iron, and Sulphite compounds.

2. *Oxidation*: Oxidizing agents are oxidized on contact with tissue proteins. Byproducts are often toxic and continue to react with the surrounding tissue.

Eg Sodium hypochlorite, Potassium permanganate, Peroxide.

3. *Corrosive agents*: Corrosive substances denature tissue proteins on contact and form eschar and a shallow ulcer.

Eg. Phenols, Cresols, White phosphorus, sodium metals, lyes, sulphuric acid, and hydrochloric acid, Alkalis .

Mechanism Of Action

4. *Protoplasmic poisons*: These agents produce their effects by binding or inhibiting calcium or other organic ions necessary for tissue viability and function.

Eg. Acetic acid, Formic acid, Oxalic, Hydrofluoric, and hydrazoic acid.

5. *Vesicants*: Vesicant agents produce ischemia with necrosis at the site of contact.

Eg. Mustard gas (sulphur and nitrogen), and Lewisite.

6. *Desiccants*: cause damage by dehydrating tissues and exothermic reactions causing the release of heat into the tissue.

Eg. Calcium sulphate , Silica gel

Alkalis more dangerous than Acids

- Acids cause coagulation necrosis with precipitation of protein, whereas the reaction to alkali is ‘liquefaction’ necrosis allowing the alkali to penetrate deeper into the injured tissue.
- The presence of hydroxyl ions within these tissues increases their solubility, allowing alkaline proteinates to form when the alkalis dissolve the proteins of the tissues.

Treatment

Removal of the Chemical

- This requires removal of all contaminated clothing and copious irrigation.
- Irrigation of chemical burns requires protection of healthcare providers to prevent additional injuries.
- Wounds should not be irrigated by placing the patient into a tub, thereby containing the chemical and spreading the injurious material.
- Irrigation should be large volume and drained ‘to the floor,’ or out of an appropriate drain

Treatment

- Do not use neutralizing agents
- They cause exothermic reactions causing further thermal damage
- Protect from hypothermia as unwarmed lavage fluid is being used
- Most of these patients require excision with grafting as the chemical burn wound tend to be deeper than they appear

Management of Hydrofluoric Acid Burn

- HF is used as cleaning agent in petroleum industry, for glass etching and removal of rust
- Acid component causes coagulation necrosis
- Fluoride ion then gains a portal of entry that chelates calcium and magnesium, resulting in hypocalcemia and hypomagnesemia.
- Efflux of intracellular calcium down concentration gradient occurs with resultant cell death.

HF Burn Management…..

- Death is mostly due to systemic toxicity
- When concentration of exposure is >20% or duration of exposure is prolonged Calcium gluconate injections are to be given topically, subcutaneously and intra Arterially
- 10% Calcium Gluconate 0.5ml/cm^2
- 10 ml of 10% Calcium Gluconate in D5 to be infused over 2-4 hrs

Vesicant Chemical Warfare agents

- Lewisite, Mustard gas – Affect all epithelial tissue including eyes and respiratory epithelium
- Cause burning in eyes & throat along with blister formation on skin
- Dimercaprol is used as Cheliating agent for Lewisite
- Sodium thiosulfate & N-Acetylcysteine for management of Mustard gas poisoning

ELECTRIC BURN

Electric Burn

Pathophysiology

- High Voltage burns (>1000V) are associated with deep extension and tissue damage like crush injury
- Low Voltage(<1000V) cause injury mostly around the area of contact point
- Current = Voltage / Resistance

Pathophysiology

Burn severity is determined by

- voltage
- current (amperage),
- type of current (alternating or direct),
- path of current flow,
- duration of contact,
- resistance at the point of contact

Pathophysiology…

- Alternating current causes tetanic muscle contractions, which may either throw victims away from the contact or draw them into continued contact with the electrical source – the ‘no-let-go’ phenomenon
- This phenomenon occurs because both flexors and extensors of the forearm are stimulated by current flow.
- However, the muscles of flexion are stronger, making the person unable to let go voluntarily

Pathophysiology…

Electrical Injury is divided into

1) Joule Heating (J) =I^2(Current)×R(Resistance)

- Tissue resistance is from lowest to highest in nerves<blood vessels< muscle< skin< tendon< fat < bone
- Severity of injury is inversely proportional to the cross-sectional area of the body part involved.
- Thus the most severe injuries are often seen at the wrist and ankle

Pathophysiology…

2) Electroporation

- It is the formation of aqueous pores in lipid bilayers exposed to a supraphysiologic electrical field.
- The formation of these pores allows calcium influx into the cytoplasm and triggers a subsequent cascade leading to apoptosis

Pathophysiology…

3) Electroconformational denaturation

- The transmembrane proteins change in polarity of amino acids in response to exposure to electrical fields.
- Voltage-gated channel proteins were found to change their conductance and ion specificity after exposure to a powerful pulsed field

Pathophysiology…

- Low voltage alternating current injury is usually localized to the points of contact,
- On prolonged contact, tissue damage may extend into deep tissues with little lateral extension, as seen in high-voltage wounds.
- These wounds are treated by excision to viable tissue and appropriate coverage based on wound depth and location.

Management

- Besides the ATLS guidelines 3 points are to be considered

1) Identify patients who will require ECG monitoring

2) Fluid therapy for Myoglobinurea

3) Identify patients at risk for compartment syndrome

ECG Abnormality

- Due to skeletal muscle injury along with injury to myocardium it is problematic to use cardiac biomarkers such as CK-MB
- Non-specific ST-T changes are the most common ECG abnormality and atrial fibrillation is the most common dysrhythmia
- Low voltage injury patients normally do no not require ECG monitoring

Criteria for 24 ECG monitoring

(1) Loss of consciousness

(2) ECG abnormality

(3) Documented dysrhythmia either before or after admission to the emergency room

(4) CPR in the field.

Myoglobinurea

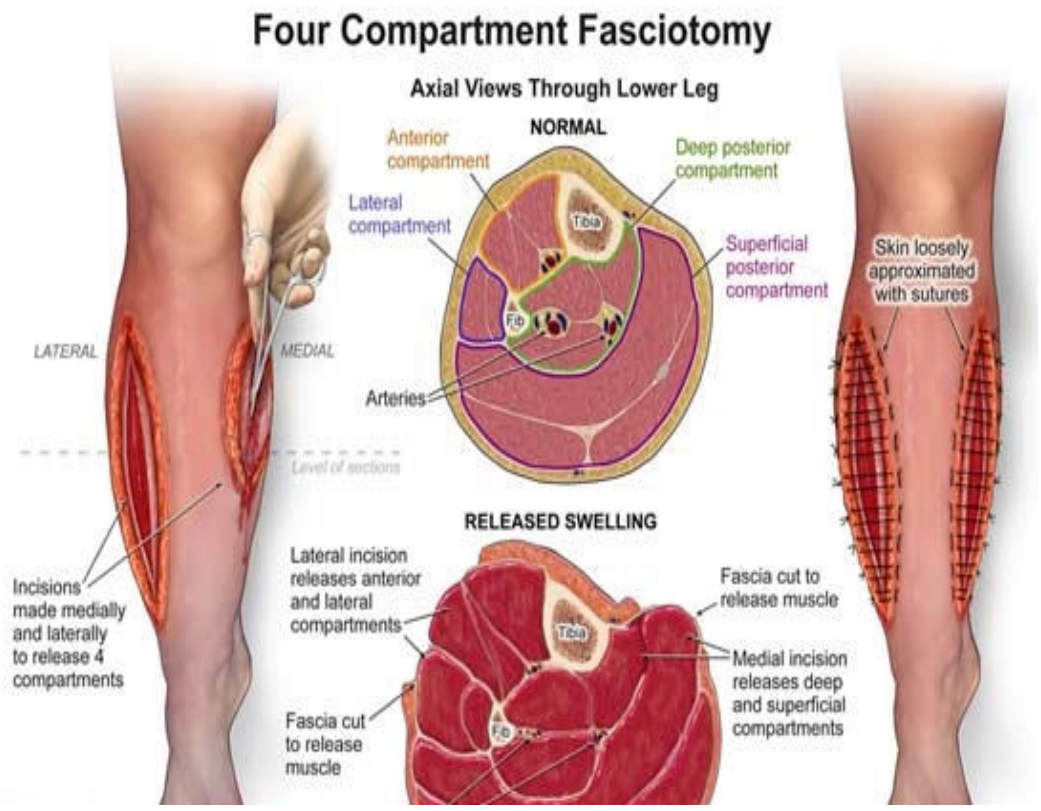
- Light pink urine indicates myogloninurea
- Recussitation with RL to maintain urine output>100ml/hr
- Alkalinization of the urine with a sodium bicarbonate
- Osmotic Diuresis with Mannitol

Compartment Syndrome

- Damaged muscle, and swelling in the investing fascia of the extremity, may increase pressures to the point where muscle blood flow is compromised.
- Loss of pulses is one of the last signs of a compartment syndrome, unlike the early loss of pulses occurring in a circumferentially burned extremity requiring escharotomy.
- A high index of suspicion is paramount for an early diagnosis, usually by serial examinations.
- Compartment pressure measurement is generally not necessary and may even be misleading

Compartment Syndrome

- Four compartment fasciotomies of the lower leg
- Anterior as well as posterior fasciotomies of the upper extremity are performed in the operating room under general anesthesia.
- Upper extremity decompression will generally always include a carpal tunnel release, as this is usually the location of the most severe injury.



Low Voltage Injuries

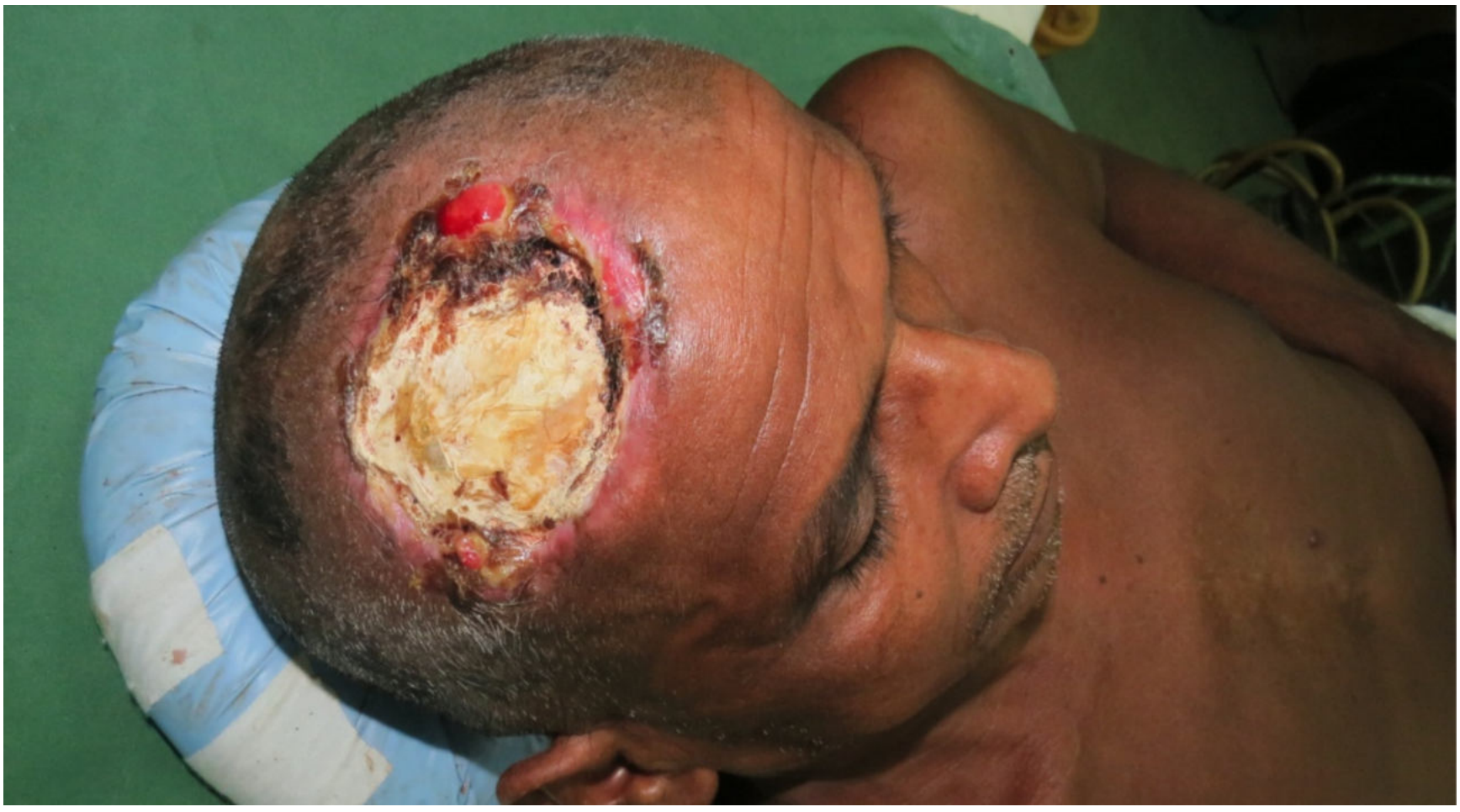
- Burns of the oral cavity are the most common type of serious electrical burn in young children.
- Injuries involving only the oral commissure are almost never excised, as the extent of injury is difficult to predict
- Gentle stretching and the use of oral splints gives good cosmetic and functional results in most patients, with reconstructive surgery being reserved for the remainder

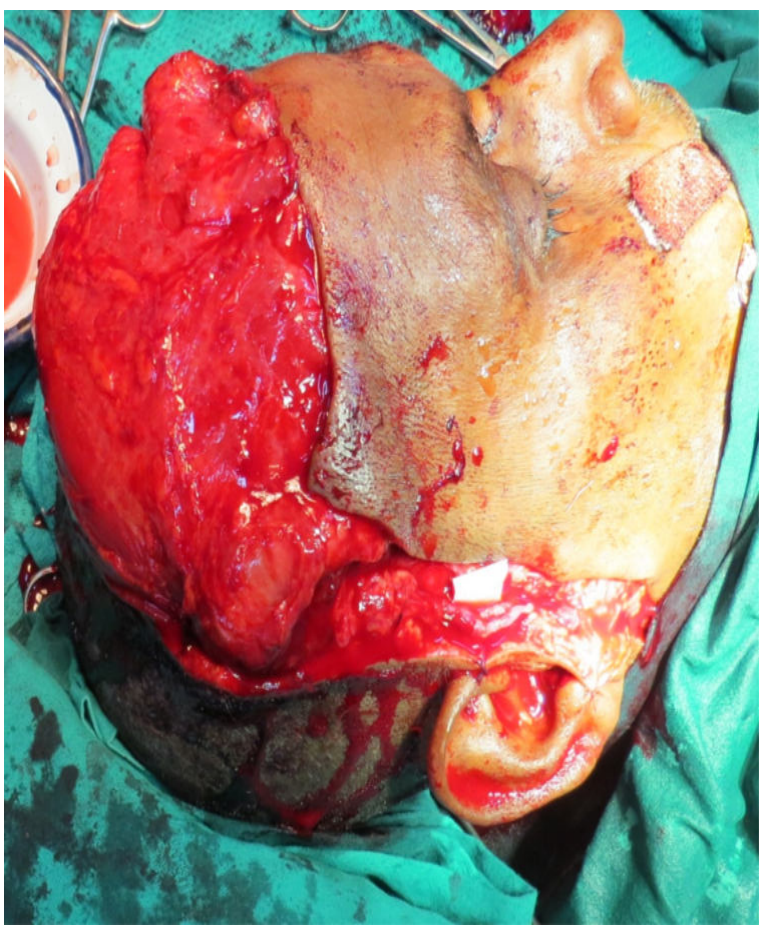
Low Voltage Injuries

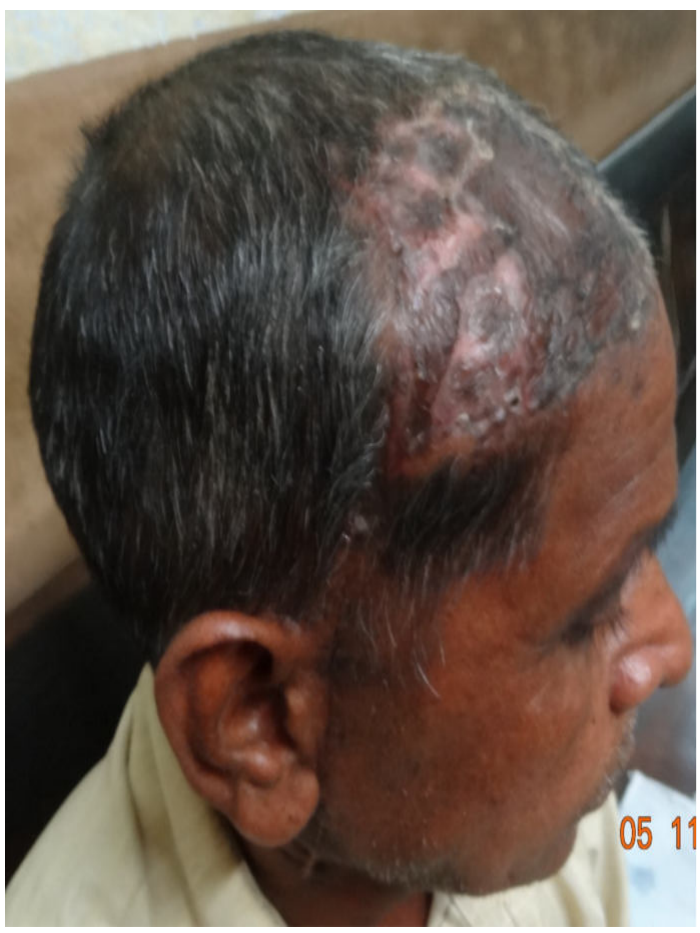
- Most cases require excision and grafting at the point of contact only
- Burns to the fingers are also mostly seen in children as they insert their fingers in power sockets

Problem Areas

- Scalp Injury where loss of outer table has occurred
- Costal chondritis is the most frequent complication of deep chest wall burns, often becoming a source of long -term morbidity, requiring multiple debridements.
- Abdominal wounds provide the potential for internal injuries, both directly under contact points and remotely as the result of late ischemic necrosis.
- Changes in their abdominal examination and/or feeding tolerance mandates investigation & laparotomy.







Lightning Strike..

- Neurologic complications are relatively common and include unconsciousness, seizures, paresthesias and paralysis, which may develop over several days after injury
- Surgically treatable lesions, including epidural, subdural and intracerebral hematomas, may occur, mandating a high index of suspicion for altered levels of consciousness.
- Prognosis of many lightning-caused neurologic injuries is generally better than for other types of traumatic cause

Lightning Strike

- The pathognomonic sign of a lightning strike is a dendritic, fern-like branching erythematous pattern on the skin.
- Lichtenberg figures (also known as keraunographic markings), consist of extravasation of blood in the subcutaneous tissue which appears within an hour of injury and fades rapidly, much like a wheal and flare reaction.
- Full-thickness isolated burns on the tips of the toes have also been reported as characteristic.
- Lightning may cause both respiratory and cardiac stand-still, for which CPR is especially effective when promptly initiated.

Thank You