Background
Epidemiology

**USA**
2,000,000 burns py
70,000 admissions py
8,500 deaths py

**Israel**
40,000 burns py
2,500 admissions py
150 deaths py
Epidemiology

Approximately 50% of burn deaths occur within the first 10 days
Epidemiology
Pathophysiology

- Thermal injury to the skin
- Burn Shock and Edema
- Mediators of burn injury
- Hemodynamic Consequences
- Systemic consequences
Thermal injury to the skin

Three zones of burn injury (Jackson’s)

Coagulation
Stasis
Hyperemia
Coagulation zone

- Necrosis
- Irreversible injury
  Caused directly from heat, Chemical insult or electrical current
Zone of Stasis

- Viable but vulnerable
- Ischemia Stasis d/t:
  - Vasoconstriction
  - Thrombosis
  - Fibrin deposition
- Can convert to necrosis
Zone of Edema (Hyperemia)

- Vasodilatation d/t mediators
- Complete recovery expected
Burn Shock and Edema

Starling equation

\[ J_v = K_f [P_c - P_{if}] - \sigma (\Pi_p - \Pi_{if}) \]

*J_v* = Volume of fluid  
*k_f* = Capillary filtration coefficient  
*P_c* = Capillary pressure  
*P_{if}* = interstitial hydrostatic pressure  
*\sigma* = Osmotic reflection coefficient  
*\Pi_p* = Plasma colloid osmotic pressure  
*\Pi_{if}* = Interstitial osmotic pressure
\[ J_v = K_f [P_c - P_{if}) - \sigma (\Pi_p - \Pi_{if})] \]

- Net force to move fluid from capillary to interstitium grows dramatically

**Net force:**
- Normal: \(-6\) mmHg filtration
- 5 min postburn: \(-56\) mmHg filtration

- decreased \(\sigma\);
- increased \(K_f\);
- increased \(P_c\);
- decreased \(P_{if}\);
- decreased \(\Pi_p\); and
- increased \(\Pi_{if}\)
Mediators of burn injury

Histamine
- Early phase increased permeability
- mainly from mast cells, monocytes

Prostaglandins (PGI$_2$, PGE$_2$)
- Vasodilatation and permeability

Thromboxane
- Vasoconstrictor contribute to ischemia
Mediators of burn injury

Serotonin
smooth muscle constrictor may increase
vascular permeability

Catecholamines
vasoconstriction in the arterioles may
partially inhibit the increased
permeability
Mediators of burn injury

oxygen radicals
  Vascular injury (damage to endothelial cells) Xanthine oxidase (Allopurinol)
Platelet aggregation factor
  Increases capillary permeability
Mediators of burn injury

Angiotensin 2 & Vasopressin
Potent vasoconstrictors of terminal arterioles Largely responsible to increased systemic vascular resistance

Corticotrophin-releasing factor (CRF)
May be powerful natural inhibitor of protein extravasation
Hemodynamic Consequences

Myocardial dysfunction
  Rt +Lt increased afterload
  decreased preload
  depression of contractility

Increased systemic vascular resistance
  Peripheral vasoconstriction & increased blood viscosity dt hemoconcentration
Hemodynamic Consequences

Pulmonary edema
- hypoproteinemia
- pulmonary vascular resistance
- no vascular increase permeability

Fluid Shift
- Massive from intravascular to interstitium

Systemic Edema
Hemodynamic Consequences
+
“"Myocardial depressant"” factor

Cardiac output depression
Systemic consequences

Immunosuppression

Loss of barriers

Dose dependent damage to chemotaxis, phagocytosis, intracellular killing power

Impaired complement activation

Suppressor cells are increased \( 7–14 \) days post burn

B cell function compromised

Loss of immunoglobulins
Systemic consequences

Immunosuppression (works both ways)

• Cellular immunity is decreased with increased allograft survival

• Significantly low levels of IL-2 which has a key role in immune response
Systemic consequences

Hypothermia
  No barrier, evaporation, No end organ in regulating temp

Hyperthermia
No end organ in regulating
Systemic consequences

Pain

Known to alter immune system

Pain control with Methadone very efficient in adults as well as in pediatric

Adult 5-15 mg q8h + 1 x prn
Child 0.2mg/kg q8h + 1 x prn
Systemic consequences

Coagulopathy
- Minor burns - Hypercoagulability
- Major burns - Consumption coagulopathy

Sensorium
- Intoxicating gases inhaled
- Rule out CO intoxication
- Intracranial edema
Systemic consequences

Gastrointestinal

Stress ulcers
Gastrointestinal bleeding 48h
Acute gastric dilatation
Motility disorders
Young children - early feeding via FT
Resuscitation

History

1897 - First advocating of saline to severe burn

1940 - Hypovolemic shock the leading cause of death after burn injury

1942 - Evans formula
Immediate care

Early cooling
Can reduce the depth of the burn
Do not cool if more than 25% TBSA
Too rapid cooling can lead to hypothermia and VT

Chemical burn
removing clothes, brushing,
irrigation with copious amounts of water
Immediate care

A B C according to ATLS as in Trauma

Early intubation Clinical decision:

- Suspected inhalation injury (circumstantial)
- Large TBSA burned
- Dyspnea, Coughing, Hoarseness
  Black sputum
- facial burn (hair, lips)
Cont’

• PO2 < 60
• CO > 25%

Absolute indication for intubation:

• Burn in palate, tongue, pharynx
• Edema in glottis, posterior pharynx
Fluid resuscitation

**Indications for IV**

Pedriatic  >10% TBSA

Adult  >20% TBSA
Fluid resuscitation
Types of fluids

**Crystalloid**  isotonic cheap no need of proteins

**Hypertonic**  240-300 mEq/l NaCl
supposed to form less edema
Na should not allowed to exceed 160 mEq/l
Fluid resuscitation
Types of fluids

**Colloid** restore fast the rapid hypoproteinemia that aggravates edema especially effective in young pediatric, old, more than 50% TBSA (Three schools)
Albumin contraindicated, FFP diseases

**Dextran** High molecular weight polysaccharides
# Fluid resuscitation

## Types of formulas

<table>
<thead>
<tr>
<th>Colloid formulas</th>
<th>Electrolyte</th>
<th>Colloid</th>
<th>D5W</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evans</td>
<td>Normal saline 1.0 ml/kg/% burn</td>
<td>1.0 ml/kg/% burn</td>
<td>2000 ml</td>
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<tr>
<td>Brooke</td>
<td>Lactated Ringer’s 1.5 ml/kg/% burn</td>
<td>0.5 ml/kg</td>
<td>2000 ml</td>
</tr>
<tr>
<td>Slater</td>
<td>Lactated Ringer’s 2 l/24 h</td>
<td>Fresh frozen plasma 75 ml/kg/24 h</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Crystalloid formulas</th>
<th>Lactated Ringer’s</th>
<th>4 ml/kg/% burn</th>
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</thead>
<tbody>
<tr>
<td>Parkland</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Modified Brooke</td>
<td>Lactated Ringer’s</td>
<td>2 ml/kg/% burn</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Hypertonic saline formulas</th>
<th>Volume to maintain urine output at 30 ml/h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertonic saline solution (Monafo)</td>
<td>Fluid contains 250 mEq Na/l</td>
</tr>
<tr>
<td>Modified hypertonic (Warden)</td>
<td>Lactated Ringer’s + 50 mEq NaHCO₃ (180 mEq Na/l) for 8 hours to maintain urine output at 30–50 ml/h</td>
</tr>
<tr>
<td></td>
<td>Lactated Ringer’s to maintain urine output at 30–50 ml/h beginning 8 hours postburn</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dextran formula (Demling)</th>
<th>Dextran 40 in saline – 2 ml/kg/h for 8 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lactated Ringer’s – volume to maintain urine output at 30 ml/h</td>
</tr>
<tr>
<td></td>
<td>Fresh frozen plasma – 0.5 ml/kg/h for 18 hours beginning 8 hours postburn</td>
</tr>
</tbody>
</table>
Fluid resuscitation in Pediatric patient

Anatomy & physiology differences:

- Size
- Surface area to body volume ratio
- Anthropomorphic differences (body proportion)
- Skin, fat, muscle mass,
- Metabolism
- Heat loss, evaporative loss
Alterations in Resuscitation

Formula for Fluids resuscitation:
5000ml/m² body surface area burned
+
2000ml/m² body surface area - Maintenance

for first 24 hours
half in first 8 hours
achieve urine output of 0.5-2ml/kg
Calculating BSA burned
Calculating BSA burn pedriatic
Monitoring
Monitoring

Vital signs - HR BP TEMP RR
Pulse oxymeter

Urine output

*Adult* 30-50ml/h, *Children* 1-2 ml/kg/h

Weight

Infection: sepsis - CBC, peripheral circulation
Pedriatic vital signs

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Heart Rate (beats/min, range)</th>
<th>Blood Pressure(^\ast) (mm Hg, range)</th>
<th>Urinary Output (mL/kg/h)</th>
<th>Respiratory Rate (breaths/min, range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N.B.</td>
<td>3.5</td>
<td>100–180</td>
<td>50–80</td>
<td>2</td>
<td>30–60</td>
</tr>
<tr>
<td>1</td>
<td>10</td>
<td>80–150</td>
<td>72–110</td>
<td>1.5</td>
<td>24–40</td>
</tr>
<tr>
<td>4</td>
<td>15</td>
<td>80–130</td>
<td>80–114</td>
<td>1</td>
<td>20–30</td>
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<tr>
<td>8</td>
<td>25</td>
<td>70–110</td>
<td>84–122</td>
<td>1</td>
<td>18–25</td>
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<tr>
<td>12</td>
<td>40</td>
<td>60–110</td>
<td>94–136</td>
<td>0.5</td>
<td>14–20</td>
</tr>
</tbody>
</table>

\(^\ast\)Estimation of BP: 50% percentile systolic BP $= 80 + 2($age in years$)$; 5% percentile systolic BP $= 70 + 2($age in years$) = \textbf{shock}.$

From Children’s Hospital of Pittsburgh, Benedum Pediatric Trauma Program: Pediatric Field Reference, April 1994.
Monitoring *inhalation injury*

- Bronchoscopy
- CXR - pulmonary injury (not in 24h)
- Sputum
Electrical burns

Deep-tissue damage exceeds the surface damage observed after electrical (especially high-voltage) injury.

The incidence of associated injury is quite high.

Myoglobinuria due to extensive muscle injury should be treated with forced diuresis 100/h 2/kg/h. Manitol & bicarbonate
Electrical burns

Massive CNS damage -
  Head CT
Peripheral nerve damage
Chemical Burns

- Differs in immediate care
- Fluid resuscitation
- Check up for liver function
- Electrolytes
Pitfalls or failure

- Delayed resuscitation
- Electrical
- Inhalation
- CO poisoning
- Pre existing
The American Burn Association has identified the following injuries as those usually requiring a referral to a burn center. Patients with these burns should be treated in a specialized burn facility after initial assessment and treatment at an emergency department. Question with the burn center.

**Second and third degree burns >10% body surface area (BSA) in patients <10 or >50 years old.**

**Second and third degree burns >20% BSA in other groups.**

**Second and third degree burns with serious threat of functional or cosmetic impairment** that involve face, hands, feet, genitalia, perineum, and major joints.

**Third-degree burns >five% BSA in any age group.**

**Electrical burns**, including lightening injury.

**Chemical burns with serious threat of functional or cosmetic impairment.**

**Inhalation injury with burn injury.**

**Circumferential burns with burn injury.**

**Burn injury in patients with pre-existing medical disorders that could complicate management, prolong recovery, or affect mortality.**

Any burn patient with concomitant trauma (for example fractures) in which the burn injury poses the greatest risk of morbidity or mortality. However, if the trauma poses the greater immediate risk, the patient may be treated in a trauma center initially until stable, before being transferred to a burn center. Physician judgement will be necessary in such situations, and should be in concert with the regional medical control plan and triage protocols.

**Hospital without qualified personnel or equipment for the care of children should transfer burned children to a burn center with these capabilities.**
Thank you