Emergency Management of Burn Injuries

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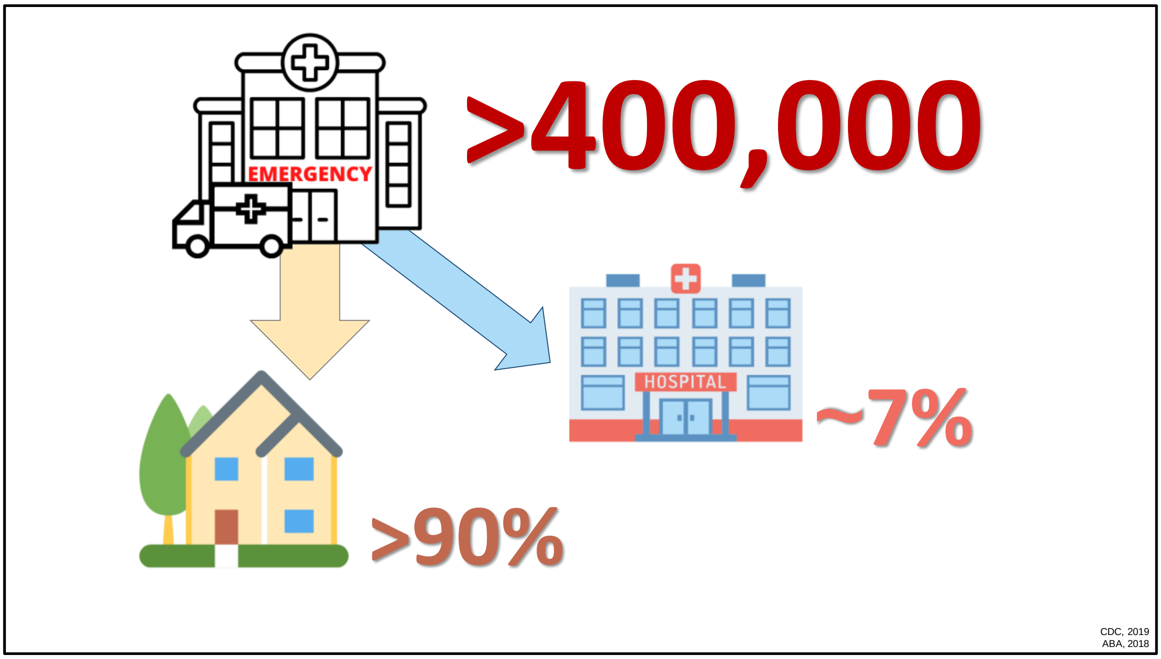
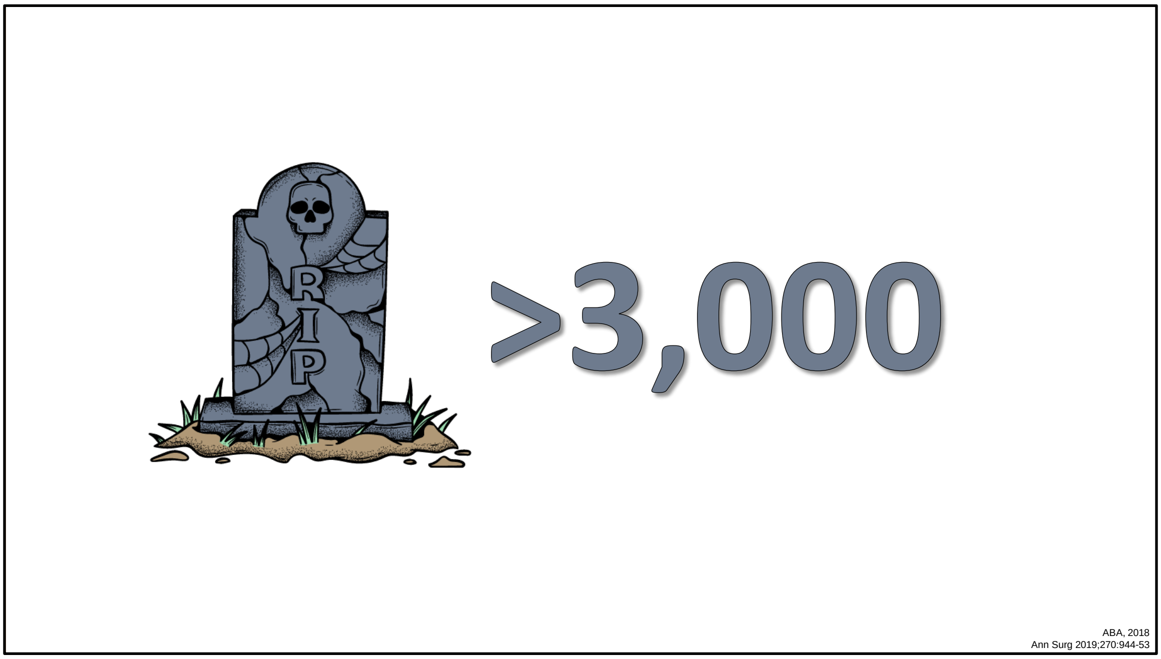


According to data from the CDC and American Burn Association, over 400,000 (closer to 420,000) burn patients present to a United State emergency department each year.

Fortunately, over 90% are discharged to home (with or without subsequent burn center follow-up); only about 7% require hospitalization or transfer.

Non-Indexed Sources:
Centers for Disease Control and Prevention. *National Hospital Ambulatory Medical Care Survey: 2018 Emergency Department Summary Tables*. 2019; https://www.cdc.gov/nchs/data/nhamcs/web_tables/2018-ed-web-tables-508.pdf
American Burn Association. *Burn Injury Fact Sheet*. 2018; https://ameriburn.org/wp-content/uploads/2017/12/nbaw-factsheet_121417-1.pdf



Over 3,000 deaths occur annually due to burns in the United States.

Although there are fluctuations (and an overall downward trend) in the total *incidence* of burn injuries, the adjusted mortality rate has remained constant for the last few decades.

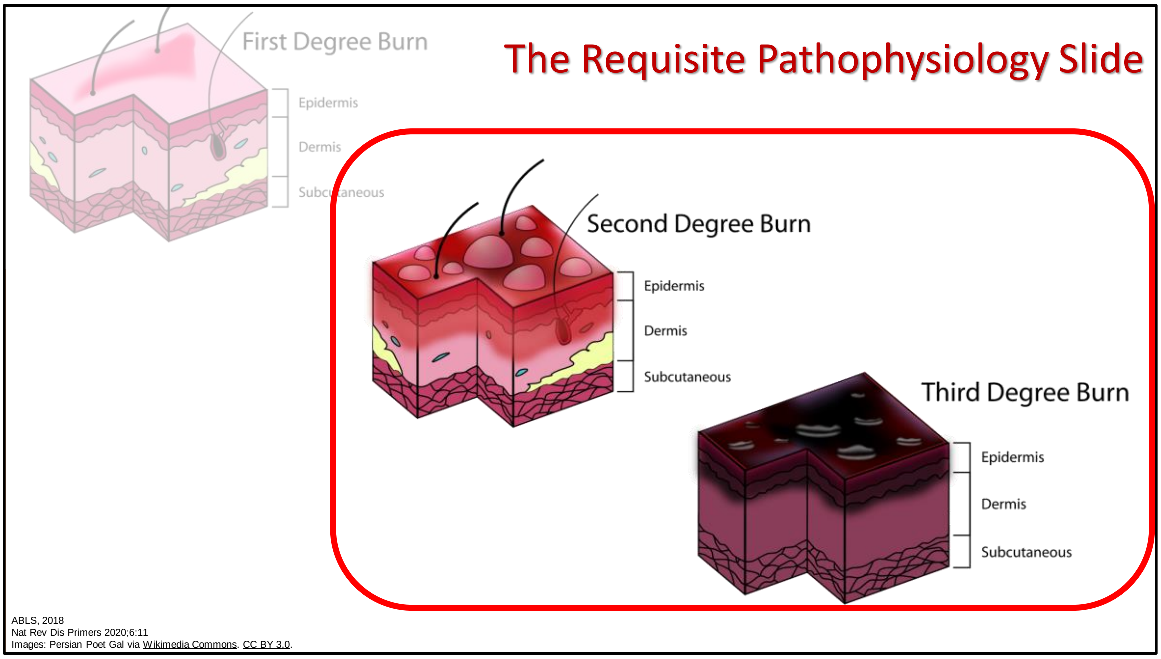
Mortality Risk Factors

- Inhalation injury
- Age
- TBSA
- Invasive management
- Complications

Burns Trauma 2018;6:24

The three **biggest predictors** of mortality are **AGE** (mortality increases with increasing age beyond the infancy period), **TBSA** (increased mortality with increasing burn area), and the presence of true **inhalational/subglottic injury**. These are factors readily identifiable by EMS and the ER.

Other factors associated with higher mortality include need for invasive management or procedures and the presence of inpatient complications, although the former may not be known in the ER, and the latter almost certainly will not be known in the acute phase of care.

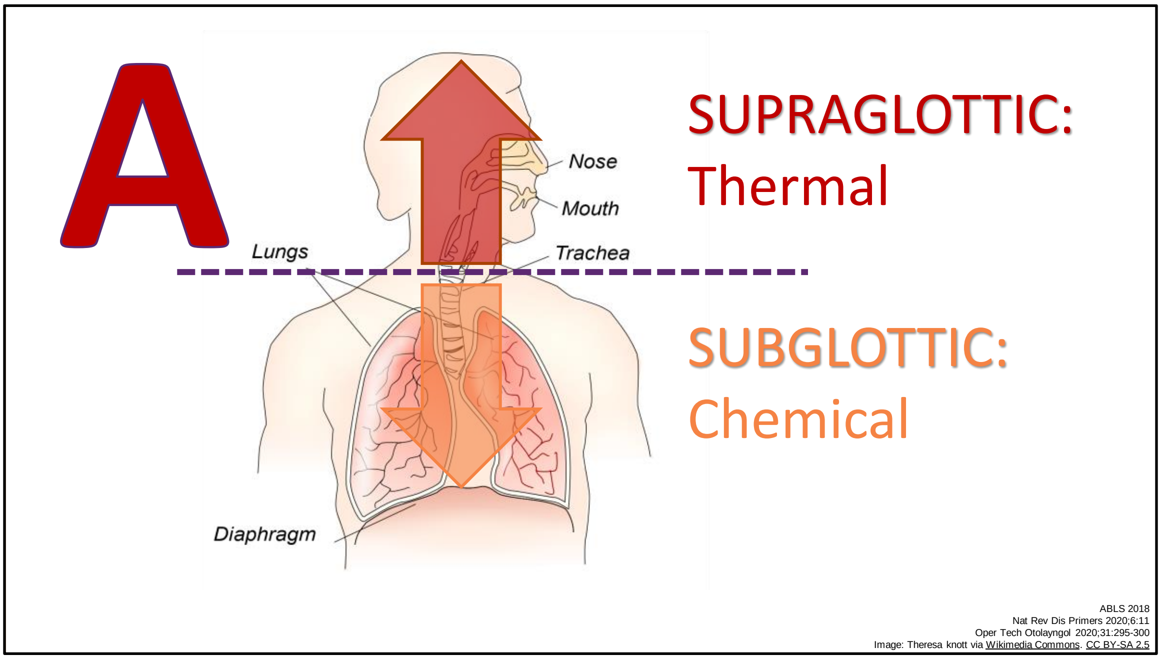


Recall that there are varying degrees of burn depth.

- **First degree**, or superficial burns, involve only the epidermal layer and are red and painful, but not blistered and do NOT have skin sloughing.
- **Second degree** burns involve both epidermal and dermal tissue and are classically characterized by painful, red skin that is blistering, wet, weeping, or edematous
- **Third degree** burns extend though the entire epidermis and dermis and may extend into subcutaneous tissue. Classically, third-degree burns are "leathery" and white or black. Although third degree burns are classically "insensate" or "anesthetic", they're closely surrounded by second- and perhaps first-degree burns, which themselves are exquisitely painful.
- There is also a **fourth degree** burn that extends through the subdermal tissue to muscle and/or bone and may appear at the surface similar to a third-degree burn, but have much more extensive underlying tissue damage.

ABLS 2018
Nat Rev Dis Primers 2020;6:11
Image: pxhere.com. CC0 1.0

The primary assessment of burn patients doesn’t differ too much from the primary assessment of *ANY* patient – begin with ABCs (airway, breathing, and circulation)



Supraglottic burns tend to be more *thermal* in nature from the actual superheated gasses being inhaled. Much like thermal burns on the skin, this leads to a significant amount of tissue edema and can result in loss of airway from glottic swelling.

Subglottic burns, on the other hand, tend to be more chemical in nature from inhaled toxins. This can lead to an inflammatory response, mucosal damage and sloughing, and can lead to pulmonary edema and ARDS in severe cases.

FACIAL BURNS
≠
AIRWAY BURNS

67-85%

<u>NO</u> FACIAL BURNS

Oper Tech Otolayngol 2020;31:295-300
Image: Nedomansky et al. Wien Klin Wochenschr 2021;133(5-6):202-208. CC BY 4.0

Just a reminder that, although facial burns *should raise suspicion for* airway burns, they do not necessarily mean the patient has an airway burn.

Consider the cause of the burn (fire vs chemical), whether the burn occurred inside or outside, the amount of time the patient was exposed to the burning agent (e.g., prolonged exposure or flash injury), etc.

And the **opposite** is true as well: an <u>airway</u> burn doesn't necessarily have to have corresponding <u>facial</u> burns. As many as 67-85% of airway burns may present without any corresponding facial burn.

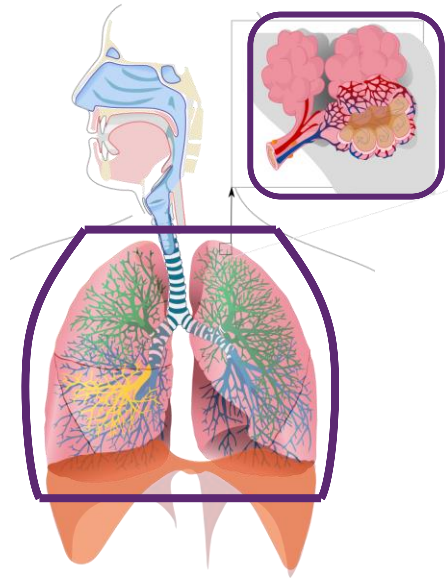
AIRWAY

WARNING SIGNS

- Stridor
- Dysphonia/hoarseness
- Carbonaceous sputum
- Respiratory distress
- Odynophagia/dysphagia
- Burns >40% TBSA

ABLS 2018
New Engl J Med 2019;380:2349-59
Nat Rev Dis Primers 2020;6:11
Oper Tech Otolayngol 2020;31:295-300
Anaesth Crit Care Pain Med 2020;39:253-67

B

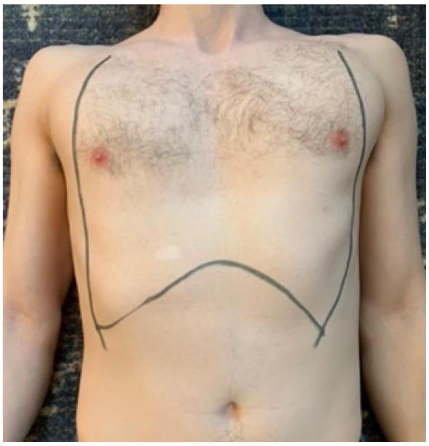


There are two major concerns with breathing:

The first is the **physical chest wall**, which may become noncompliant with circumferential high-grade burns (deep 2nd degree or 3rd/4th degree)

The second relates to **impairment of oxygen delivery** associated with carbon monoxide and/or cyanide toxicity

ESCHAROTOMY

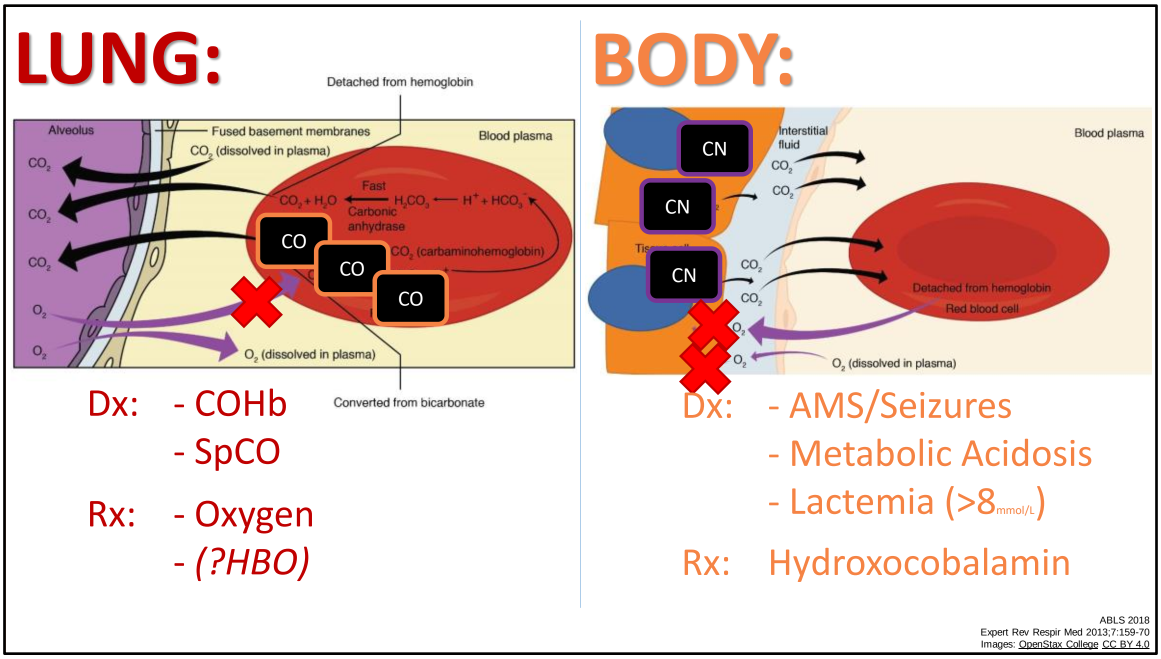


ABLS 2018
New Engl J Med 2019;380:2349-59
Anaesth Crit Care Pain Med 2020;39:253-67

A chest escharotomy can be performed to relieve tension on the chest wall and facilitate a return to normal respiratory mechanics, improved compliance, and improved gas exchange.

Unlike limb escharotomies, which can usually be delayed until a patient arrives at a burn center, chest wall escharotomy may be necessary to perform in the field or the community ER prior to transfer if the patient has significant respiratory impairment.

When performing an escharotomy, a “box” pattern is typically taught, however just incising the eschar laterally (along the anterior axillary line) and inferiorly (along the costal margin) is usually enough to improve respiratory mechanics. If possible, it may be advisable to avoid incising along the clavicles; many of these patients require prolonged mechanical ventilation (and, therefore, a tracheostomy creation) and oozing from a trach site can get into the clavicular incision and lead to infection and/or delayed wound healing.



Carbon monoxide works at the *pulmonary* level by binding with hemoglobin with a far greater affinity than oxygen, thus blocking oxygen from attaching to the hemoglobin in the RBCs and causing a hypemic hypoxia (that is, oxygen can't get on the RBC in the first place).

Carbon monoxide intoxication can be measured with arterial or venous blood gas, evaluating for carboxyhemoglobin; there are also noninvasive co-oximeters that can differentiate between oxygen and carbon monoxide and show a total SpO_2 and a fractionated SpCO.
Treatment for CO intoxication involves high concentrations of oxygen (e.g., nonrebreather mask or 1.0 FiO_2 if mechanically ventilated) which dramatically lowers the half-life of carbon monoxide bound to hemoglobin. The utility of hyperbaric oxygen therapy remains debated in the literature, and likely in the setting of a burn/inhalation injury, the risk of sending the patient to the chamber acutely will probably outweigh any potential benefits, but this remains a viable option for *isolated* carbon monoxide poisonings.

Cyanide, on the other hand, causes cellular poisoning and interferes with mitochondrial use of oxygen causing a histotoxic hypoxia (that is, oxygen is delivered to the cells, but the cells become dysfunctional and cannot use it).

Cyanide toxicity results in anaerobic metabolism and lactic acid production. Although there is no test for "cyanide levels" like there is for CO intoxication, it should be suspected with clinical history (e.g., closed-space fire and/or smoke inhalation) combined with altered mental status, metabolic acidosis, and elevated lactate despite ongoing resuscitation.
This is treated preferentially with hydroxocobalamin, first FDA approved in 2006, which binds the cyanide to form a nontoxic compound (cyanocobalamin) and is renally eliminated. Another, older treatment consists of administration of sodium nitrite, sodium thiosulfate, and amyl nitrite. Although this removes cyanide from the cell, it does so by *forming methemaglobin* (where cyanide preferentially binds), which itself can lead to hypoxia, and the antidotes (especially sodium nitrite) can lead to profound hypotension.

C

MILD TACHYCARDIA (100-120) IS **EXPECTED**

SEVERE TACHYCARDIA (>140)

OR

ANY HYPOTENSION IS **NOT NORMAL**

ABLS 2018
Anesthesiology 2018;129(3):583-9

Although a mild tachycardia into the 100-120 range is expected in adult patients, as may be some mild **hyper**tension (due to catecholamine-mediated vasoconstriction), severe tachycardia beyond 140, tachycardia >120 despite fluid resuscitation, or hypotension to ANY degree are not normal in the acute phase of burn management.

Strongly consider other sources for fluid (e.g., blood) loss, especially in the setting of concomitant trauma.

Later in the patient's course, this may well be an indication of vasoplegic "burn shock," SIRS, and/or sepsis, but in the **initial** resuscitation and stabilization in the field and ER, shocky vitals are more likely to represent hemorrhagic shock from trauma or some other acute process rather than the burn itself.

We know adequate fluid resuscitation is necessary to stay ahead of the inevitable fluid shifts that will occur during the inflammatory and vasoplegic states of burn care, but it's also important to realize that **over**-resuscitation may be as detrimental as **under**-resuscitation—so it's no wonder there are so many different formulas to target the "Goldilocks" middle zone that adequately hydrates the patient without over- or under-shooting their fluid needs.

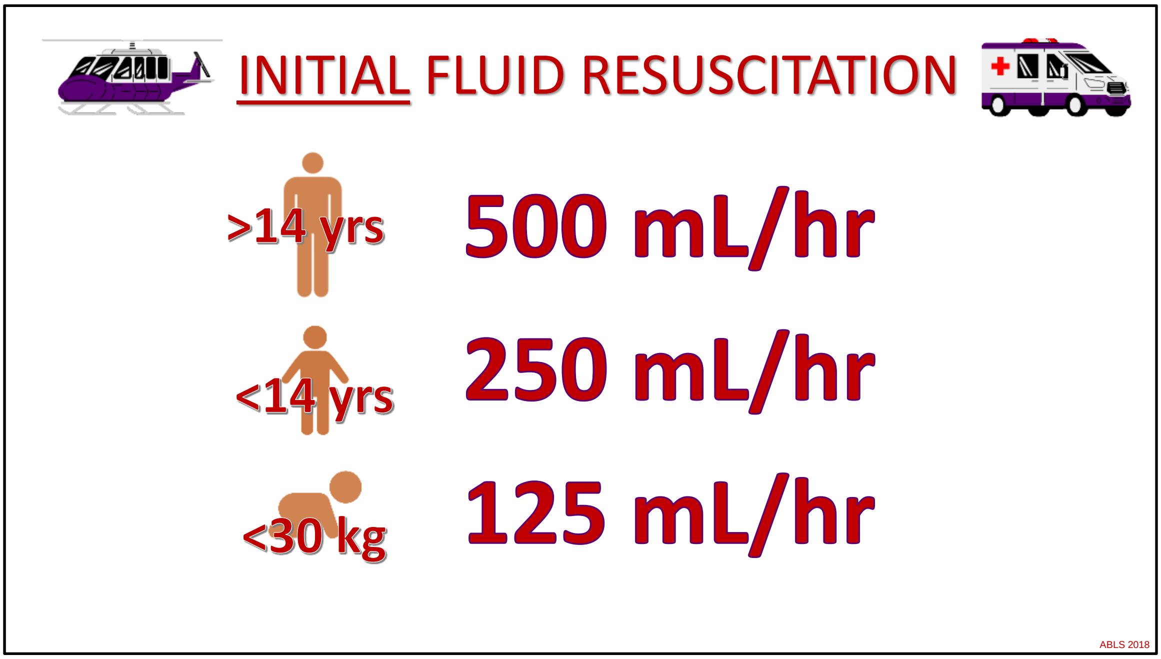
FLUID RESUSCITATION

- AGE
- WEIGHT
- TBSA BURN

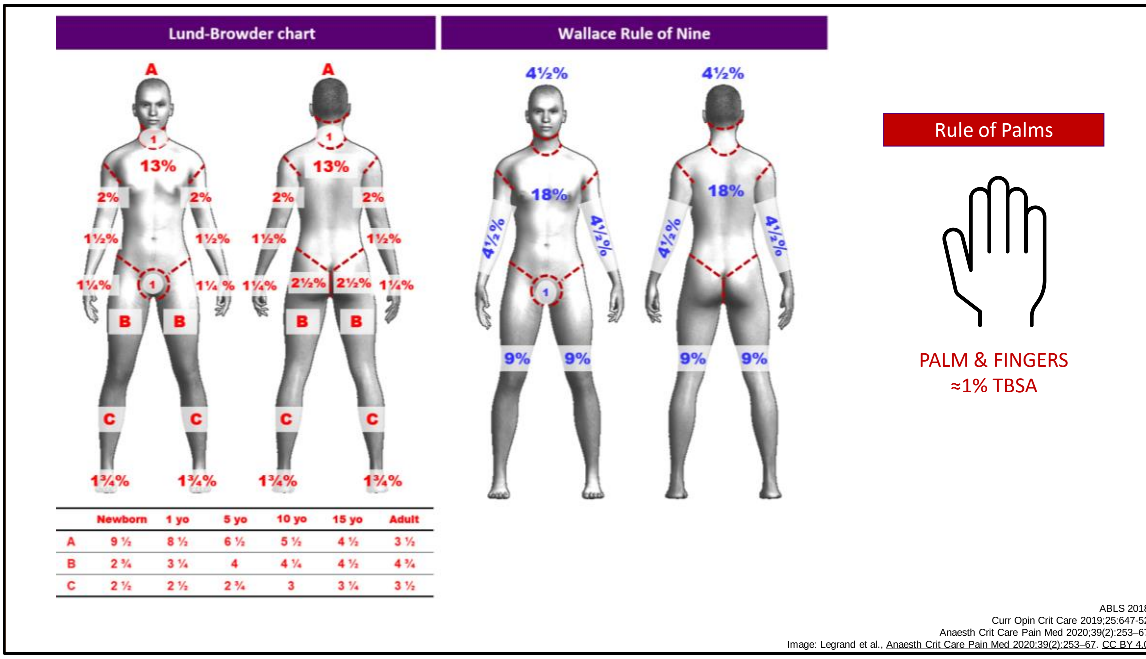
ABLS 2018
Curr Opin Crit Care 2019;25:647-52

For fluid resuscitation calculations, the ABA's Advanced Burn Life Support guidelines have made it *incredibly* simple for initial field resuscitation (e.g., by EMS), and *relatively* straightforward for the emergency department.

The only information needed is going to be the patient's age (broadly infant, child, or adult), weight (gross estimate), and TBSA burned (we'll come back to that in a bit!)



As long as estimated TBSA is >20% (2nd degree or higher), these are the ABLS-recommended initial fluid rates in the prehospital and early hospital phases.



	Newborn	1 yo	5 yo	10 yo	15 yo	Adult
A	9 ½	8 ½	6 ½	5 ½	4 ½	3 ½
B	2 ¾	3 ¼	4	4 ¼	4 ½	4 ¾
C	2 ½	2 ½	2 ¾	3	3 ¼	3 ½

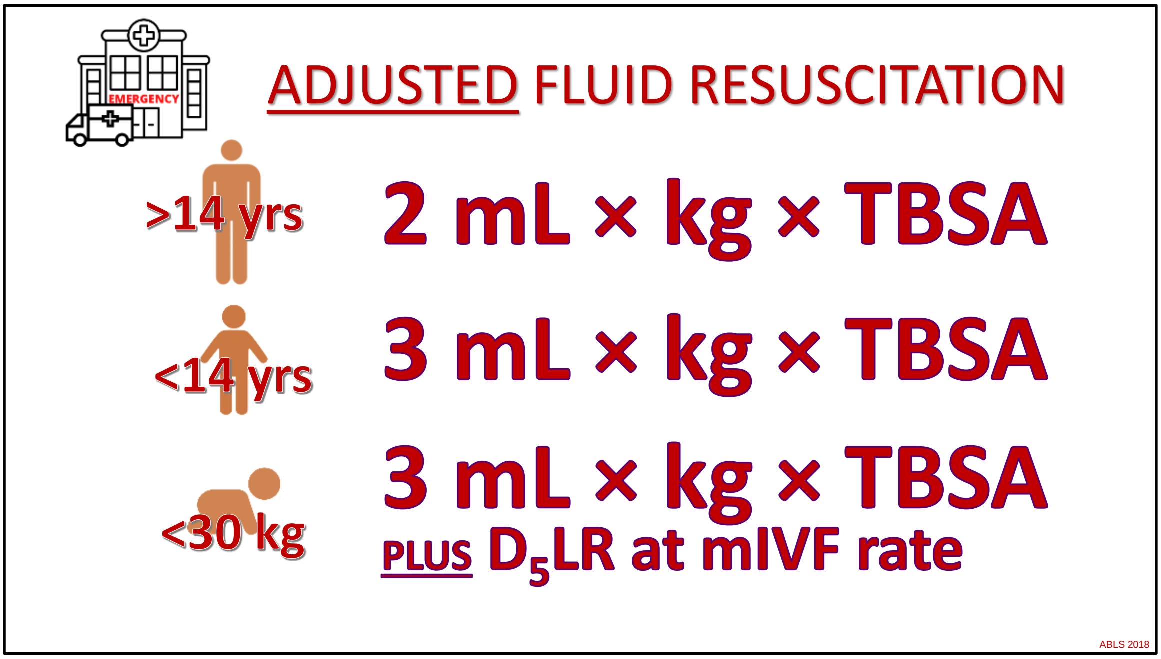
Beyond the *initial* fluids, we're going to need to be able to calculate the percent body surface area burned.

In general, most are familiar with the Rule of 9s to calculate burn area – essentially, the body is broken into 11 regions, each representing 9% body surface area

And, while that's a good approximation for adults, it risks overestimating the burned area in pediatric patients.

The Lund-Browder chart tends to be more accurate because it accounts for variations across several age groups.

For patchy burns, the palmar surface of the **patient's** own hand (<u>including</u> the fingers) can be used to estimate 1% TBSA



Once the patient's estimated weight and estimated burn area is calculated, the IV fluid rate is then *adjusted* to the calculated fluid rate using these calculations (so, much closer to modified Brooke than to Parkland for thermal burns).

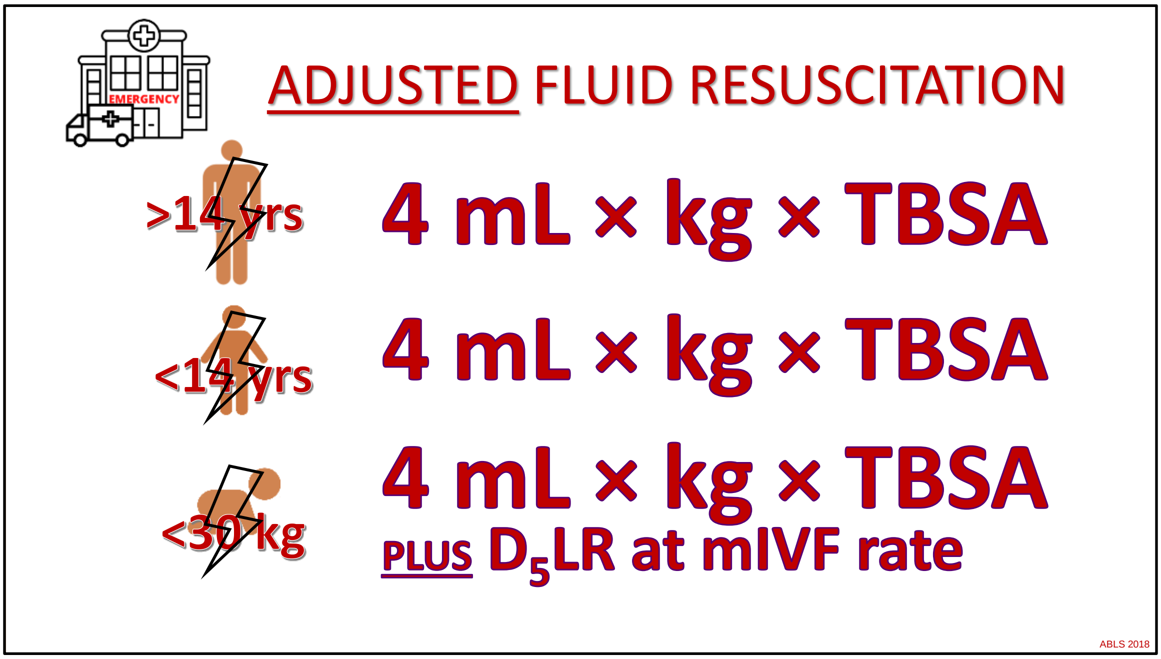
As a reminder, these calculations provide the 24-hour total; the recommendation remains to infuse half of that volume in the first 8 hours *from the time of the burn*.

So, first, you need to divide this volume in half to get the first-8-hour volume.

Then, account for fluids already given (e.g., from the initial resuscitation) and account for any time "catch up" for delayed presentation.

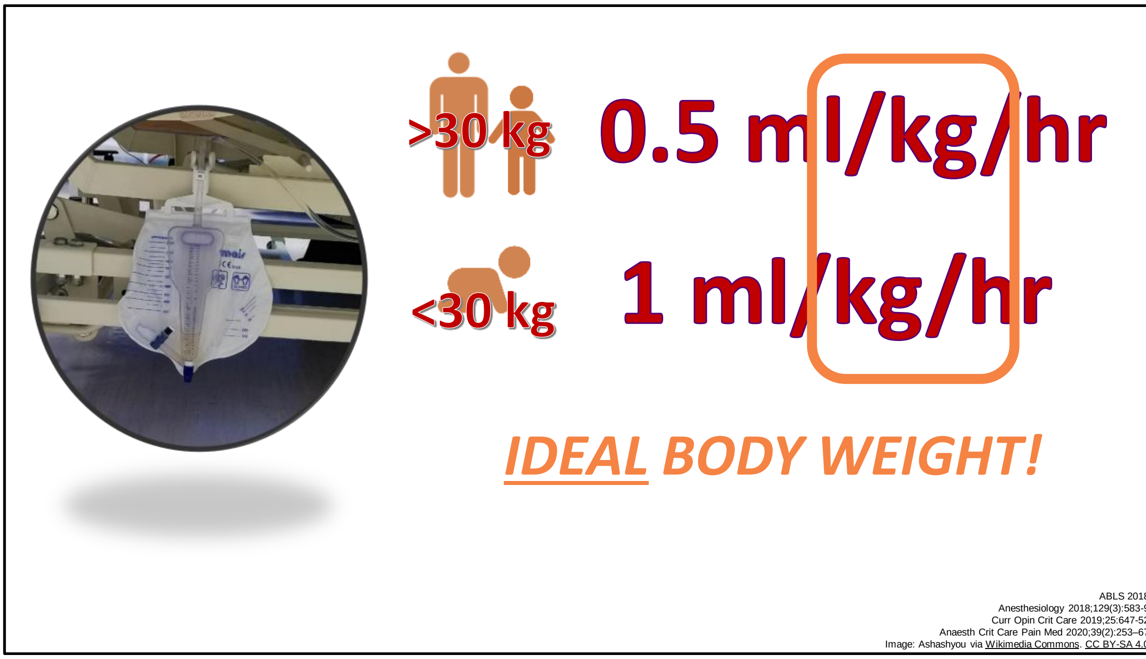
Let's consider a case of an adult patient weighing 80 kg suffering 20% 2nd-degree burns to his body after falling into a bonfire. He was intoxicated and did not call EMS for about an hour after the injury. During transport, EMS administered about 250 mL of fluid, and he arrives to your hospital 2 hours after his burn.

Based on these guidelines, his **24-hour volume** of fluid would be estimated at 2 x 80 (kg) x 20 (%TBSA) = 3200 mL
HALF of this volume should be infused over the first **8 hours** post-burn = 1600 mL
Then, consider that the patient **already received** 250 mL, so the balance to infuse = 1350 mL
Last, this volume infuses over 8 hours *post-burn*, but the patient did not arrive for 2 hours after his injury, so in this case, that volume infuses over the next (8-2=**6 hours**)
So, 1350 ÷ 6 hrs = **225 ml/hr** adjusted fluid rate



For patients of <u>any</u> age with electrical injury, initial fluid rate is based on 4 ml/kg/TBSA (plus maintenance rate for children <30 kg).

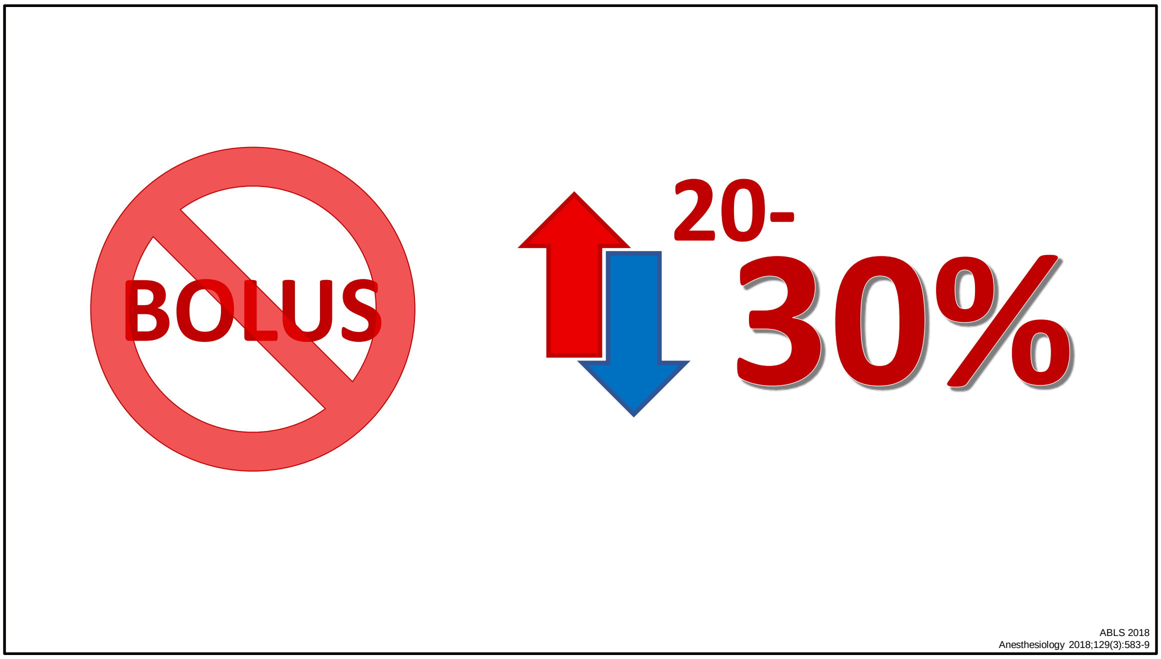
Electrical injury typically has less visible thermal burn and is at a much higher risk of developing rhabdomyolysis (myoglobinuria), thus the increased fluid rate.



Regardless of the starting fluid rate, ongoing infusion rates are based on urinary output, with goal urinary output of 0.5 ml/kg/hr in adults and older children, and 1 ml/kg/hr in children less than 30 kg.

If there is evidence of myoglobinuria, urinary output is ideally augmented to 75-100 ml/hr (for adults) until the urine clears, then it can be returned to the previous goal of 0.5 ml/kg/hr.

Also, importantly, urine output is based on **IDEAL** body weight – not actual body weight (i.e., a 200 kg adult does not need a urine output of 100 ml/hr, unless they have evidence of myoglobinuria)
For most adults, this will come out to 30-50 ml/hr of urine output.



If urinary output is not at goal, guidelines advise AGAINST bolusing fluids. Rather, the rate of infusion should be increased by about 20-30%
(ABLS recommends about a 1/3, or 33%, change up or down in response to urine output below or above goal, respectively)

KEEP WARM!

AVOID HYPOTHERMIA

Hypothermia can cause local vasoconstriction and potentially worsen the extent of the burned area by propagating ischemic tissue damage.

→ Cover the burned area with a clean, dry sheet (does not *need* to be sterile!); this can help prevent hypothermia (especially in larger burns) and may prevent air currents from causing additional pain in partial thickness burns.
→ Do NOT cool burns greater than 5% TBSA (risk of hypothermia outweighs potential benefits)

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