

Burn Shock

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Objectives

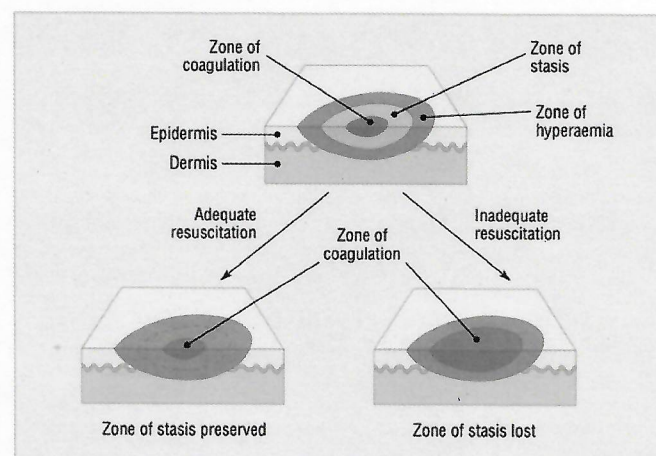
- What is burn shock?
- How to recognize burn shock.
- How to treat burn shock.

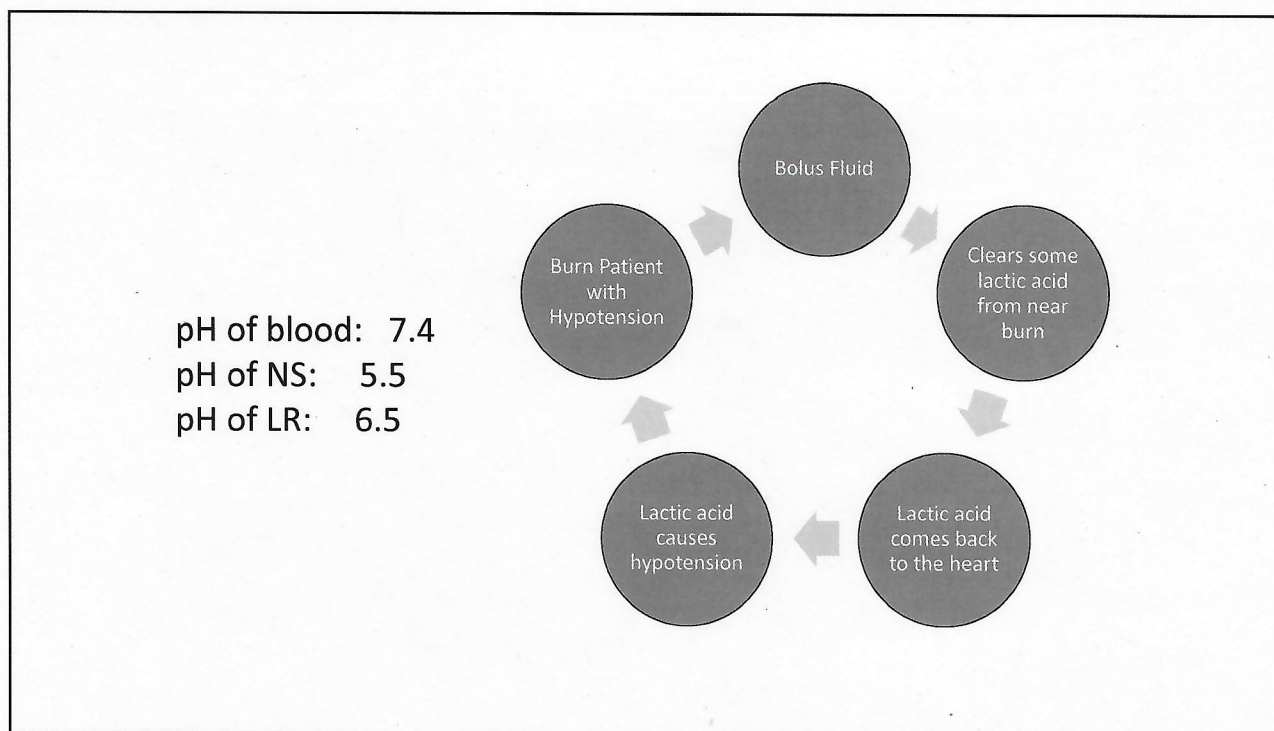
Burn Shock

- Burn shock is a **unique combination of hypovolemic and distributive shock, accompanied by cardiogenic shock.**
- Burns initially causes capillary leakage syndrome, resulting in severe hypovolemia and massive edema

• T. Ishikawa, H. Maeda, *Encyclopedia of Forensic Sciences* 2013, Pages 47-53

Ischemia-Reperfusion injury





Conventionally, these patients would get bolused fluid

• Kirkpatrick, A.W., Ball, C.G., Nickerson, D. *et al.* Intraabdominal Hypertension and the Abdominal Compartment Syndrome in Burn Patients. *World J Surg* **33**, 1142–1149 (2009). <https://doi.org/10.1007/s00268-009-9995-4>



Capillaries are damaged and weak

- They leak in burned and unburned areas
- They leak into the lung interstitium and interfere with ventilation
- Compartment syndrome:
 - Extremities
 - Abdomen
 - Neck
 - Eyes

myocardial dysfunction in thermal injury

- Slowed relaxation of the heart in diastole
- And impaired contractility
- Decreased diastolic compliance of the left ventricle (when the heart gets coronary perfusion)
- Decrease in cardiac output and initial metabolic rate with compensatory increments in heart rate and peripheral vascular resistance
 - which increase myocardial oxygen demand, leading ultimately to right and left heart deficits.
- Depending on the extent of the burn, this deficit might result in a state of cardiogenic shock, which has been identified as a major cause of failed resuscitation.

- increased mortality associated to cardiac dysfunction during the acute hospitalization
- prolonged derangement can lead to an increase in long-term morbidity.
- burn-related dysfunction that was believed to be transient, with maximal dysfunction apparent 18 – 30 hours postburn followed by recovery of cardiac function 48 – 60 hrs postburn,
- has now been proven to last for up to 2 years in burned children

Abu-Sittah GS, Sarhane KA, Dibo SA, Ibrahim A. Cardiovascular dysfunction in burns: review of the literature. *Ann Burns Fire Disasters*. 2012 Mar 31;25(1):26-37. PMID: 23012613; PMCID: PMC3431724.

Hemodynamic features of burn shock comprise a decrease in:

Cardiac output (in the order of 40-60%)

Stroke work

Stroke volume

Myocardial oxygen consumption

Venous return

Myocardial metabolic activity

Coronary blood flow

Myocardial oxygenation (ischemia)

Peak systolic blood pressure

Myocardial contractility

Mean arterial pressure

Myocardial compliance

Estimated myocardial work

Cytokines

- TNF- α (tumor necrosis factor) is a multifunctional cytokine detected in several human cardiac related conditions, including congestive cardiac failure and septic cardiomyopathy, and has been implicated as well in cardiac dysfunction following burns
- TNF- α was shown to depress cardiac contractility, intracellular calcium currents and induce programmed cell death (apoptosis) of cardiomyocytes in experiments simulating ischemic conditions of the heart
- Apoptosis resulting in rapid and reversible declines in contractile function is directly proportional to TNF- α levels

Contributing issues

- Leaky capillaries
- Denatured proteins and byproducts of dead tissues and near dead tissues entering circulation
- Loss of proteins from leaky capillaries, + lots of fluid
- Ongoing losses from damaged skin

What does burn shock look like?

- Why is the pressure 70/30?
- Is that pressure right?
- Are we maxed on levo AND vaso? Why isn't it working?
- Are they in atrial fibrillation ... or is that sinus tach at 150?
- Why is the heart rate 32?

Burn Shock is a conglomerate

- Not only Blood Pressure
- Whole body poor perfusion
- May have neurologic signs
- Bowel ischemia- bacteria can eventually translocate

How to treat burn shock.

- Correctly monitor the patient
- In the hospital: Arterial lines, upper torso central line for CVP monitor, +/-hemosphere? Prehospital: Check Bp, Check perfusion of fingers and toes. Keep them warm.
- Correct fluids at the correct rate. Resuscitation guidelines are an estimate. They are not the exact science we want them to be.
 - ABA Guidelines:Prehospital >20% TBSA burn?
 - Adult: 500cc/hr LR
 - 6-14 yr old: 250cc/hr LR
 - 0-6 yr old: 125cc/hr LR
- May need a colloid
 - Albumin
 - Plasma
- Pressors/ Bicarb infusions

Fluids

- Why we like Lactate Ringers
 - More physiologic- more like what we normally have
 - Isotonic, balanced and buffered
 - Sodium, chloride, potassium, calcium, and lactate-in the form of sodium lactate, mixed into a solution with an osmolarity of 273 mOsm/L and pH of about 6.5. (normal saline has a pH of 5.5)
 - Historically made for renal patients
 - We are doing a large volume resuscitation
 - Large volumes of normal saline can cause metabolic acidosis on their own, causing hypotension
 - Hypernatremic, hyperchloremic metabolic acidosis is iatrogenic

Vanderbilt study

- Looked at using NS vs LR for large volume resuscitation in ICU
- More death and acute kidney injury in NS group (by only about 1% in the study...but significant)
- Vanderbilt switched from 40-50% use of NS in their ICU's to about 5%

Matthew W. Semler, M.D., Wesley H. Self, M.D., M.P.H., Jonathan P. Wanderer, M.D., Jesse M. Ehrenfeld, M.D., M.P.H., Li Wang, M.S., Daniel W. Byrne, M.S., Joanna L. Stollings, Pharm.D., Avinash B. Kumar, M.D., Christopher G. Hughes, M.D., Antonio Hernandez, M.D., Oscar D. Guillamondegui, M.D., M.P.H., Addison K. May, M.D., et al. for the SMART Investigators and the Pragmatic Critical Care Research Group. Balanced Crystalloids versus Saline in Critically Ill Adults. *N Engl J Med* 2018; 378:829-839 DOI: 10.1056/NEJMoa1711584

Plasmalyte A

- Not enough studies.
- Cappuyns L, Tridente A, Stubbington Y, Dempsey-Hibbert NC, Shokrollahi K. Review of Burn Resuscitation: is Plasmalyte® A Comparable Alternative to Ringer's Lactate? *J Burn Care Res.* 2022 Aug 2;irac106. doi: 10.1093/jbcr/irac106.
- More basic than LR, but does not significantly change acidosis in patient.
- Does reduce ionized calcium in burn resuscitation when compared to LR controls
- Chaussard M, Dépret F, Saint-Aubin O, Benyamina M, Coutrot M, Jully M, Oueslati H, Fratani A, Cupaciu A, Poniard A, Asehnoune K, Dimby SF, Mebazaa A, Houze P, Legrand M. Physiological response to fluid resuscitation with Ringer lactate versus Plasmalyte in critically ill burn patients. *J Appl Physiol* (1985). 2020 Mar 1;128(3):709-714. doi: 10.1152/jappphysiol.00859.2019.

Colloids in burns

- We like albumin
- Colloid oncotic properties can better maintain intravascular volume than crystalloids, reducing fluid volume demands
- Albumin has the same sodium profile as NS (Na of 145-155)
- Initial studies of albumin alone by Baxter™ were stopped early (why?)
- Started fluids in combination with colloid
- Albumin infusion was associated with reduced mortality. odds ratio=0.34
- Albumin administration was also accompanied by decreased occurrence of compartment syndrome. odds ratio=0.19

• Navickis RJ, Greenhalgh DG, Wilkes MM. Albumin in Burn Shock Resuscitation: A Meta-Analysis of Controlled Clinical Studies. *J Burn Care Res.* 2016 May-Jun;37(3):e268-78. doi: 10.1097/BCR.0000000000000201.

Plasma

- Long history of use of plasma, from the time of Parkland and Baxter, Israeli and West Penn resuscitation strategies
- Adults with burns > 40% TBSA or > 25% TBSA with a smoke inhalation injury were randomized to either 2000 ml/24 hours of LR plus 75 ml/kg FFP/24 hours titrated to a urinary output of 0.5 ml/kg/hour or to 4 ml/kg/%burn/24 hours (half given over the first 8 h postburn) and also titrated to 0.5 to 1 ml/kg/hour of urine output.
- i.e. 100kg man would get 7.5L of plasma in 24 hours
- There was a profound and significant reduction in fluid administered to the FFP group (0.14 liter/kg) compared with the crystalloid group (0.26 liter/kg) by 24 hours post injury.
- I would get angry calls from the blood bank. (supply issues)

• O'Mara MS, Slater H, Goldfarb IW, Caushaj PF. A prospective, randomized evaluation of intra-abdominal pressures with crystalloid and colloid resuscitation in burn patients. *J Trauma* 2005;58:1011-8

• Robert Cartotto, MD, FRCS(C), Jeannie Callum, MD, FRCP(C), A Review on the Use of Plasma During Acute Burn Resuscitation, *Journal of Burn Care & Research*, Volume 41, Issue 2, March/April 2020, Pages 433-440, <https://doi.org/10.1093/jbcr/jrz184>

Pressors

- Best info is from the US military (Iraq/Afghanistan conflict)
- Interestingly, documented use of pressors, 48% in the BRG group and 34% in the control group, was associated with increased survival (OR, 6.309; CI, 1.466–27.137; $p = 0.013$).
- First line, first 24h: Vasopressin
- Second line: Levophed/Norepinephrine
- Third line Dobutamine
 - Military generally healthy prior to injury.

• Ennis, Jody L. RN, BSN; Chung, Kevin K. MD; Renz, Evan M. MD, FACS; Barillo, David J. MD, FACS; Albrecht, Michael C. MD; Jones, John A. BS, BBA; Blackbourne, Lorne H. MD; Cancio, Leopoldo C. MD, FACS; Eastridge, Brian J. MD, FACS; Flaherty, Steven F. MD, FACS; Dorlac, Warren C. MD; Kelleher, K.S. MD; Wade, Charles E. PhD; Wolf, Steven E. MD, FACS; Jenkins, Donald H. MD; Holcomb, John B. MC. Joint Theater Trauma System Implementation of Burn Resuscitation Guidelines Improves Outcomes in Severely Burned Military Casualties. The Journal of Trauma: Injury, Infection, and Critical Care: February 2008 - Volume 64 - Issue 2 - p S146-S152 doi: 10.1097/TA.0b013e318160b44c

Difficult resuscitation guidelines

Table 1 Recommendations for the Difficult Fluid Resuscitation

At 12–18 h postburn, calculate the projected 24-h resuscitation if fluid rates are kept constant. If the projected 24-h resuscitation requirement exceeds 6 mL/kg/%TBSA then the following steps are recommended.

1. Initiate 5% albumin early as described previously in the Emergency War Surgery Handbook.
2. Check bladder pressures every 4 h.
3. If Urine Output (UOP) <30 mL/h, strongly consider the placement of a Pulmonary Artery (PA) catheter to guide resuscitation with specific Pulmonary Capillary Wedge Pressure (PCWP) and Mixed Venous Saturation (SvO₂) goals. (Goal PCWP 10–12, SvO₂ 65%–70%). If PA catheter placement is not practical then consider monitoring Central Venous Pressures (CVP) from a subclavian or IJ along with Central Venous (ScvO₂) saturations. (Goal CVP 8–10, ScvO₂ 60%–65%).
 - a. If CVP or PCWP not at goal then increase fluid rate.
 - b. If CVP or PCWP at goal then consider vasopressin 0.04 Units/min to augment MAP (and thus UOP) or Dobutamine 5 mcg/kg/min (titrate until SvO₂ or ScvO₂ at goal). Max dose of Dobutamine is 20 mcg/kg/min.
 - c. If both CVP or PCWP and SvO₂ or ScvO₂ at goal then stop increasing fluids (EVEN if UOP <30 mL/hr). The patient should be considered hemodynamically optimized and the oliguria is likely a result of established renal insult. Some degree of renal failure should be tolerated and expected. Continued increases in fluid administration despite optimal hemodynamic parameters will only result in "resuscitation morbidity", that is oftentimes more detrimental than renal failure.
4. If the patient becomes hypotensive along with oliguria (UOP <30 mL/hr), then follow the hypotension guidelines.
5. Every attempt should be made to minimize fluid administration while maintaining organ perfusion. If UOP >50 mL/h, then decrease the fluid rate by 20%.

After 24 h, LR infusion should be titrated down to maintenance levels and albumin continued until the 48 h mark.

Hypotension guidelines

Table 2 Hypotension Guidelines

The optimal minimum blood pressure for burn patient must be individualized. Some patients will maintain adequate organ perfusion (and thus have adequate UOP) at MAPs lower than 70. True hypotension must be correlated with UOP. If a MAP (generally <55 mm Hg) is not adequate to maintain the UOP goal of at least 30 mL/h then the following steps are recommended.

1. Start with Vasopressin 0.04 units/min drip (DO NOT TITRATE).
2. Monitor CVP (goal 8–10).
3. If CVP not at goal then increase fluid rate.
4. If CVP at goal then add Levophed (norepinephrine) 2–20 mcg/min.
5. If additional pressors are needed, consider the placement of a PA catheter to guide resuscitation with specific PCWP and SVO₂ goals (goal PCWP 10–12, SVO₂ 65%–70%). These patients may be volume depleted but a missed injury should be suspected.
 - a. If PCWP not at goal then increase fluid rate.
 - b. If PCWP at goal then consider Dobutamine 5 mcg/kg/min (titrate until SvO₂ at goal). Max dose of Dobutamine is 20 mcg/kg/min.
 - c. If hypotension persists, look for missed injury.
 - d. Consider adding epinephrine or neosynephrine as a last resort.
6. If the patient is exhibiting catecholamine-resistant shock, consider the following diagnoses.
 - a. Missed injury and on-going blood loss.
 - b. Acidemia. If pH <7.20 then adjust ventilator settings to optimize ventilation (Target PCO₂ 30–35). If despite optimal ventilation, patient is still has a pH <7.2, consider bicarb administration.
 - c. Adrenal insufficiency. Check a random cortisol and add start hydrocortisone 100 mg every 8 hours.
 - d. Hypocalcemia. Maintain Ionized Calcium >1.1.

European resuscitation

- Survey of ICUs in Europe
- During early resuscitation phase:
- Early use of colloid >60%
- Use of norepinephrine >80%
- Heterogeneous results were reported regarding monitoring strategies, early vasopressors, and albumin use between burn centers and nonspecialized centers

• Soussi, S., Berger, M.M., Colpaert, K. *et al.* Hemodynamic management of critically ill burn patients: an international survey. *Crit Care* 22, 194 (2018).
<https://doi.org/10.1186/s13054-018-2129-3>

Pitfalls

- Carbon Monoxide
 - Also contributes to shock by reducing oxygen availability and causing anaerobic respirations
 - Build up of lactate/metabolic acids with hypotension
 - Carbon monoxide (CO) poisoning, by creating a more hypoxic intracellular environment, further complicates myocardial damage from smoke inhalation. It manifests as ECG changes, dysrhythmias, congestive heart failure or hypotension
 - CO makes platelets more thrombotic
 - Animal and human studies demonstrated the histology of CO-induced myocardial injury.
 - focal areas of hemorrhage and necrosis, frequently involving the subendocardium and papillary muscle
 - -must treat with 100% FiO₂
- Abu-Sittah GS, Sarhane KA, Dibo SA, Ibrahim A. Cardiovascular dysfunction in burns: review of the literature. *Ann Burns Fire Disasters*. 2012 Mar 31;25(1):26-37. PMID: 23012613; PMCID: PMC3431724.

Pitfalls

- Hydrogen Cyanide
 - Also impairs oxygen utilization on the cellular level
 - Treat preferably with cyanocobalamin (cyanokit)
 - Can increase blood pressure temporarily
 - May be associated with some acute kidney injury down the road.
 - Should be treated within 30 minutes to be effective

Burn Shock

- Hypovolemic Shock: treat with steady fluid infusions. Burn patients drip to death, they don't hemorrhage.
- Distributive shock: treat with pressors
- Cardiogenic shock: treat with fluids and pressors, supportive care
- Consequences of the patient being in shock, or hypoxic/ CO/ Cyanide toxicity from anerobic respiration (metabolic acidosis): treat with 100% oxygen, treat with IV bicarbonate.

Burn Shock

Is caused by one event, but is a multimodal disease

- Can be difficult to treat
- Can be addressed by multiple therapies, and no two patients will be exactly alike
- Correct and prompt treatment can reduce complications down the road