VANDERBILT UNIVERSITY MEDICAL CENTER

**Guideline:** Adult Burn Fluid Resuscitation

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**I. Population:**

Adult (≥16 years old and ≥30 kg) burn patients requiring acute intravenous burn resuscitation.

**Possible Burn Resuscitation Exclusions:**

- If < 16 years of age or < 30 kg – see pediatric resuscitation protocol
- Patients with severe or symptomatic heart failure
- Active renal failure (acute, acute on chronic, chronic, ESRD)

**II. Indications:**

Adults requiring more than maintenance intravenous resuscitation will be admitted to the Burn ICU (BICU). The goal of fluid resuscitation after a severe burn injury is to reach and maintain appropriate organ and tissue perfusion. Insufficient resuscitation leads to organ failure and death while excessive resuscitation also increases morbidity and mortality.(1) For this reason, an appropriate early resuscitation is the most critical intervention in optimizing burn patient outcomes. A standardized algorithm is used to best match a continuous fluid infusion with the patient's needs, which are proportional to the size of the patient and the burn size.

**Burn Resuscitation Indications:**

- Burn injury ≥20% TBSA (2nd and 3rd degree injuries – do not include 1st degree)
- High voltage electrical injury
- Smoke inhalation with intubation
- Burn attending discretion

Please use the following guidelines to determine appropriate disposition and resuscitation:

| | **Appropriate Unit** |
|---|---|
| <10% TBSA | Admit to: burn step down<br>Resuscitation: Oral |
| 10-19% TBSA | Admit to: burn step down<br>Resuscitation: Oral + MIVF |
| ≥20% TBSA | Admit to: BICU<br>Resuscitation: Resuscitation Protocol |

**III. Definitions:**

**Depth of Burn**

Estimated percent of total body surface area (%TBSA) of partial and full thickness burns is required to calculate fluid requirements.
Superficial burns (1st degree) are <u>not included</u> in this calculation.

| **Depth of Burn** | |
|---|---|
| Superficial (1st Degree) | Erythema; skin intact ("sunburn") |
| Partial Thickness (2nd Degree) | Wet, weepy, blisters, pink, blanching |
| Full thickness (3rd Degree) | White, brown, red, black, leathery, dry |

**Calculating TBSA**

There are various methods used to estimate %TBSA. The Palmar Method(2) and/or Rule of Nines(3) are the easiest and fastest ways to estimate burn size. Regardless of the method used, these are estimated values. The Lund and Browder chart will be the final determination of TBSA.

The palmar method presumes that 1% TBSA is equal to the size of the patient's palm, from wrist crease to tip of longest finger. It is important to emphasize that the patient's hand should be used for this measurement.

The Rule of Nines divides the patient's surface area into individual sections for easier calculations (adult diagram below). However, as humans grow and their body proportions change, there are some slight changes to the Rule of Nines. To account for that, the Lund-Browder adjusts body segments based on patient age to arrive at a more accurate estimate.

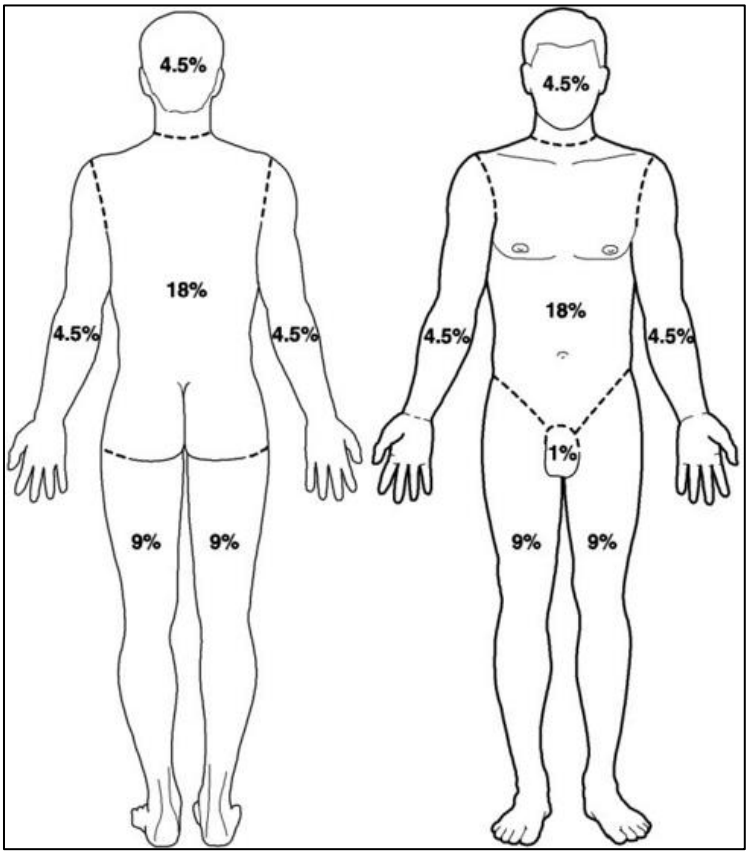
Also, if the burn does not encompass the entire area referenced in the rule, the %TBSA is counted as a proportion of the size noted in the diagram.

For example, if half of the posterior torso is burned, that is 9% TBSA, not 18%.

The palmar method is the preferred method for estimating initial burn size until a Lund-Browder calculation can be completed for final determination.

The presence of inhalation injury and/or high-voltage electrical injury is known to lead to higher-than-expected resuscitation volumes. To account for this, the presence of either should be treated as an additional 20% TBSA of burn during resuscitation calculations.

For example, a standalone inhalation injury would be resuscitated as a 20% TBSA burn while a 15% TBSA burn with inhalation injury would be resuscitated as a 35% TBSA burn.



## IV. Resuscitation Fluids

### A. Crystalloid

Lactated ringers (LR) solution continues to be the mainstay of burn resuscitation since the 1960s. As an isotonic solution, it has been used to expand the extracellular fluid volume in the critical first 48 hours post burn. This is complicated by burn induced inflammatory and vasoactive mediators that lead to local and systemic vascular changes promoting substantial fluid loss and fluid shifts, as well as reduced cardiac output.(4) However, it remains the standard for resuscitation.

### B.

Oral resuscitation (in isolation) is primarily used in smaller burns. Larger burns with more substantial fluid requirements naturally limit oral volume delivery and may lead to emesis if attempted.

### C. Colloid

Plasma proteins exert an oncotic pressure, which balances against the hydrostatic pressure in normal homeostasis. Burn injury causes increased vascular permeability and changes the balance of plasma and interstitial oncotic pressures, driving proteins into the interstitium. This is exacerbated by increased interstitial compliance through local tissue destruction and plasma dilution through high volume crystalloid resuscitation.(5) While colloid administration may maintain the osmotic gradient in *unburned* skin and reduce resuscitation volumes while restoring cardiac output, it is not clear if this effects multi-organ failure rates, length of stay, duration of ventilation, or mortality.(1) Therefore, colloid administration has had a long and complicated history in acute burn care.

1. **FFP** – The rationale for the use of plasma in acute burn resuscitation is that it directly addresses the hypoproteinemia induced by a large burn, theoretically reducing the total crystalloid needed to restore cardiac preload and reduce burn edema. It can also act as a pH buffer and have a potential benefit as an immunomodulatory. There is also the presumed benefit of endothelial stabilization, which may avoid the microvascular leakage seen in albumin. However, allogenic plasma has a risk of transmitting pathogens and is a known risk factor in the development of acute lung injury (ALI). It also imparts a substantial financial cost to the patient and the healthcare system.(6)

2. **Albumin** – Albumin has been shown to reduce resuscitation volumes and *potentially* expand the extracellular fluid volume (when given along with LR) more efficiently than crystalloid alone.(1) It is not clear, however, that it more efficiently expands the intravascular volume than any other continuous infusion. Furthermore, potentially increased microvascular permeability in the lung and hypoproteinemia-induced loss of osmotic pressure may lead to significant pulmonary edema in spite of the administration of albumin.(7) Both early and late (or “rescue strategy”) albumin resuscitation adjuncts are clinically in use. The rescue strategy is used to obviate albumin use in the first 12 hours post burn to avoid peak microvascular permeability of *unburned* tissue. There are no data to recommend or discourage albumin boluses in burn.

**V. Resuscitation Endpoints:**

**A. Hourly Urine Output**

1. While crude, urine output is the best and least-invasive endpoint of resuscitation at our disposal. Maintenance of an hourly urine output between 30-50 mL/hr (or approximately 0.5 -1 mL/kg/hr) remains the standard in adult burn resuscitation.(4) As such, urine output should be strictly monitored. Foley catheters will be placed in all patients requiring acute burn resuscitation.

2. After the initial resuscitation rate has been calculated and initiated, further fluid management will be guided by the Burn Navigator system. This is a validated software package developed by the ISR that incorporates baseline patient characteristics with dynamic variables to adjust the infusion rate each hour.(8, 9)

3. For those patients not undergoing acute burn resuscitation (which should be the minority and *only* after discussion with the burn attending), consideration should be given to bladder scanning, possible Foley catheter placement, and alternative fluid management if the patient is unable to spontaneously void ≥4 consecutive hours during the acute phase.

4. Crystalloid boluses are **not** indicated for the management of oliguria in the hemodynamically normal patient. Hourly titrations of resuscitation volume typically correct the issue.(4)

5. Oliguria in a burn patient is most likely due to hypovolemia, but it still requires a comprehensive clinical evaluation to assess all possible etiologies. Ensure proper Foley catheter placement without obstruction, consider a bladder scan and measurement of bladder pressures.

6. Patients on home diuretic regimens should likely be restarted when they are otherwise hemodynamically appropriate (after the initial resuscitation phase).

**B. Blood Pressure**

1. The parameters for treating burn shock and the threshold of minimum blood pressure must be individualized. In the previously healthy patient, a MAP ≥60 mmHg and a heart rate below 140 BPM are considered adequate. However, age and comorbidities will require reconsideration of these metrics. Patients over 65 years and/or with a history of CV disease should have a MAP ≥65 mmHg and HR ≤130 BPM.

2. Crystalloid boluses are **not** the preferred method of management for hypotension during the initial resuscitation phase per the ABA. If hypotension (MAP < 60 mmHg or signs of end-organ ischemia) persists despite appropriate escalation of fluid, then other pharmacologic agents should be considered. Norepinephrine is usually the preferred vasopressor in the absence of known pulmonary hypotension. Vasopressin should be

considered with escalation of norepinephrine, or possibly as a first-line agent in a patient with known pulmonary hypertension or right ventricular dysfunction. If the patient continues to fail resuscitation despite escalating doses of vasopressors and maintenance fluids, heart failure and/or burn-mediated cardiogenic shock should be considered.

3. Hypotension in a burn patient requires a comprehensive clinical evaluation to assess all possible etiologies. Noninvasive blood pressure measurements may also be inaccurate when tissue edema or thick, leathery burns are present. Consideration should be given to arterial line placement early in the resuscitation and it is expected in the setting of hemodynamic difficulties.

4. As abdominal compartment syndrome can cause both hypotension and decreased urine output, and further fluid administration will only exacerbate this etiology, it is important that *every* resuscitation that is failing include an assessment for intra-abdominal hypertension.

**C. Lactate**

1. Lactate makes a poor independent metric of resuscitation compared to urine output. However, both the initial lactate and the ability to clear it have been shown to prognosticate mortality in burn resuscitations.(10, 11) Lactate clearance at 24 hours has been shown to predict improved mortality. As such, any persistent lactatemia mandates comprehensive clinical evaluation to assess all possible etiologies.

2. Serum lactate reflects a balance of production and clearance (primarily hepatic) . As such, persistent lactatemia may represent a complication separate from resuscitation, such as liver failure, mesenteric ischemia, compartment syndrome, etc. Therefore, other causes of elevated lactate should be ruled out before using this as an independent metric of the trajectory of a burn resuscitation.(12)

**D. Other Endpoints**

1. Various invasive and non-invasive endpoints of resuscitation have been studied with inconsistent or unimpressive results. Central venous pressures and pulmonary artery catheter pressures have not been shown to improve mortality and may lead to greater over-resuscitation than urine output alone. Ventricular–arterial coupling measurements, pulse contour analysis, tissue perfusion monitors, and other measures of cardiac output and perfusion are similarly of questionable use at this time and should primarily be used as adjuncts when patient factors, such as renal failure or cardiomyopathy, make urine output unreliable.

**E. Resuscitation Failure**

1. Causes of resuscitation failure include incorrect burn size estimation, incorrect evaluation of burn depth, compression syndrome from eschar, electrical injury mechanism, concomitant trauma or inhalation injury,

significant comorbidities (especially those that make UOP an unreliable metric of resuscitation). Patients at extremes of age and those with prior dehydration or delayed start of resuscitation are at particular risk of a failed resuscitation.(13, 14) Absence of a strict fluid titration protocol is another risk factor.

2. Defining burn resuscitation failure is difficult, especially in its early stages. Any evidence of inadequate end-organ oxygen delivery despite reaching or exceeding predicted fluid resuscitation volumes may be considered evidence of resuscitation failure. Irreversible burn shock is the primary contributor to burn mortality.(15)
   Signs of failure include:
   - Persistent oliguria despite escalation of fluid resuscitation up to twice the initial calculated rate
   - Persistent hypotension (MAP<60) requiring escalating doses of vasopressor agents
   - Persistent elevation of lactate over 2.5(10, 11)

   *Note: any of these could be present in isolation or together to be considered potential resuscitation failure, though each of these in isolation would require elimination of other causes.*

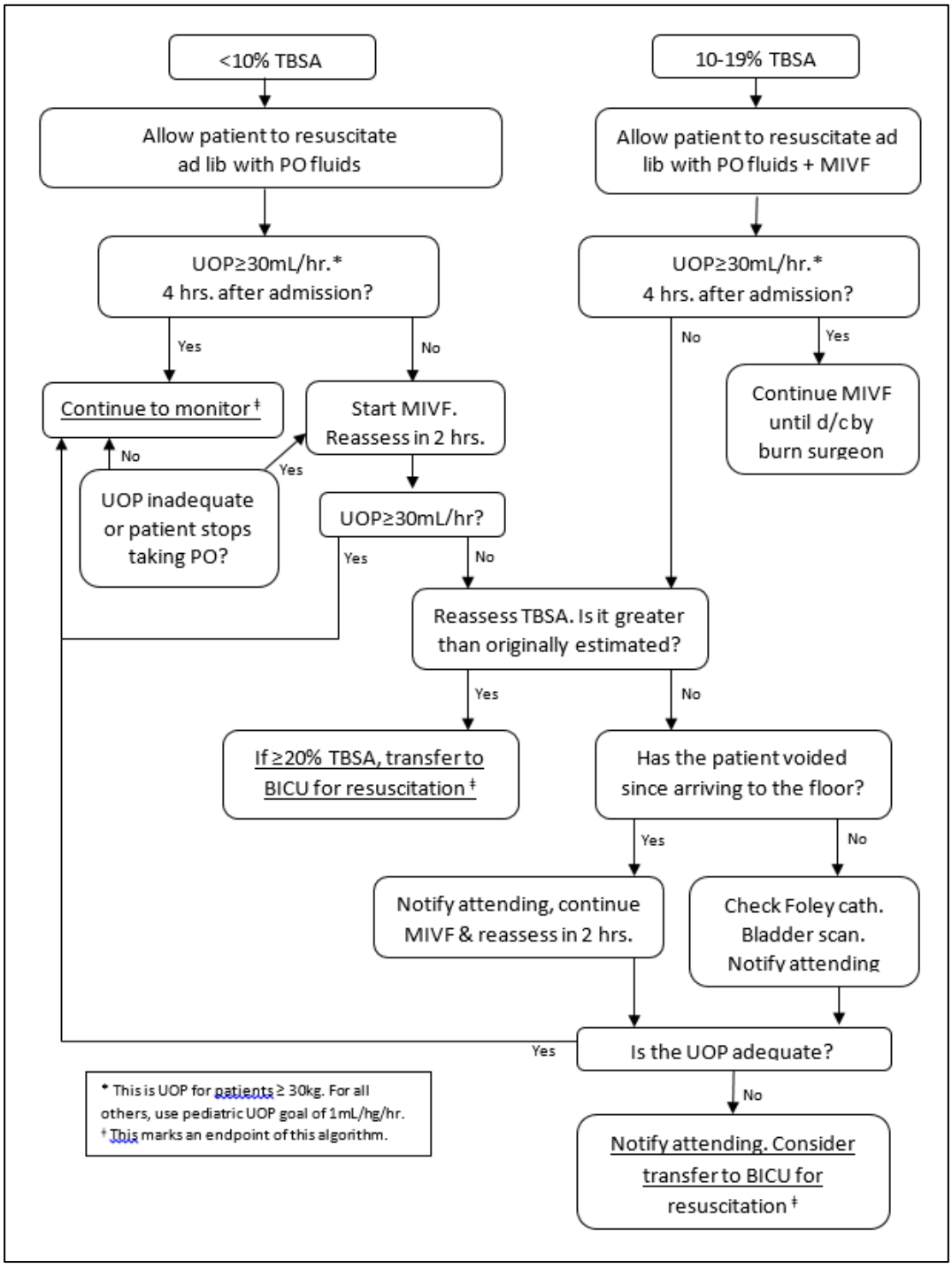
3. Mitigation: A single instance of hypotension or decreased urine output does not define failure, but steps must be taken to correct the trajectory of the resuscitation. Oliguria, hypotension, and/or lactatemia must be treated with an increase in basal resuscitation fluid rate per protocol. However, some portion of the population will have a poor outcome from resuscitation, even with rescue efforts embedded in the algorithm. In these cases, other possible methods of mitigation include:
   - Vasoactive agents (15-17)
   - Addition of colloid (bolus or drip) – used to expand intravascular volume, reduce total resuscitation volume, decrease edema in *unburned* tissue, and other potential effects that are less well understood (stabilization of endothelium, cardiac output restoration, etc.)(7)
   - Hemofiltration (reduces inflammatory burden and manages renal failure)
   - Very-early burn excision – within 24-48 hours (reduce inflammation)(16, 17)
   - Plasma exchange/plasmapheresis (reduces inflammatory burden)(18, 19)

## VI. Intervention/Treatment:

### A. Labs at admission

CBC with differential, CMP, serum lactate, magnesium, phosphorous, ionized calcium, type & screen, and coagulation labs should be drawn upon admission. **For Burns greater than 20%**, draw CBC, BMP, lactate, magnesium, phosphorus, and calcium every six hours until the resuscitation is complete.

**B. FLUID MANAGEMENT**
**If TBSA <20% (flowchart)**




**Burns 10-19% TBSA:**

Consider a feeding tube
Consider calorie counts on at-risk patients (e.g., elderly, recent weight loss, homeless)
Track I & O's and consider placing a Foley if concerns for progressing AKI.

**If TBSA ≥20%**

**Preparation**

Ideally, 2 large bore (18 gauge or larger) IVs would be placed in unburned skin for resuscitation as well as a Foley catheter. The patient's TBSA, Baux score, and expected mortality should be documented on the resuscitation table and should also be discussed with the burn attending, bedside nurse, and charge nurse.

Documentation

- Time and Date of Injury
- Pre-hospital IV fluid totals
- Pre-hospital urine output
- Admission height and weight

Nutrition

- Place Dobhoff tube (DHT) – consider bridle
  - Start a 1.0 TF formula
  - Advance TF to a goal of 1cc/kg/hr using the patient's actual weight (not AIBW)
  - Consult dietician
- Start metoclopramide for burn induced gastric ileus
  - Metoclopromide 10mg IV q6 x 48 hours
  - Consider advancing DHT to post-pyloric position if ongoing ileus

**FLUID MANGEMENT**

Step One: Fluid Requirement

- Pre-hospital and hydro room rate: 500mL/hr of LR
- Calculate the burn size (TBSA) and depth ($2^{nd}$ and $3^{rd}$ degree)
- Document burn on a **Lund and Browder** burn diagram
- Document Baux Score (Age +%TBSA [+17 for inhalation injury]); use nomogram to estimate mortality
- Obtain accurate height and weight at admission
- Calculate **IBW**
  - IBW (men) = 50 + 0.91(height in cm – 152.4)
  - IBW (women) = 45.5 + 0.91(height in cm – 152.4)
- Calculate **ABWI (Adjusted Body Weight Index)** = IBW + **0.3**(actual body weight – IBW)
- Obtain Body Surface Area (entire patient – not just burn)
  - Derived from nursing record upon entering correct height and weight
- Calculate resuscitation cessation rate: mIVF + Insensible loss =
  - ( ____ kg + 40 mL) + [(25+ ____ %TBSA) x____ BSA (Body Surface Area)= _____ mL/hr
- Calculate **Alarm Rate** (= 2 x initial starting rate) and **Ivy Index** (= 250 mL/kg AIBW)

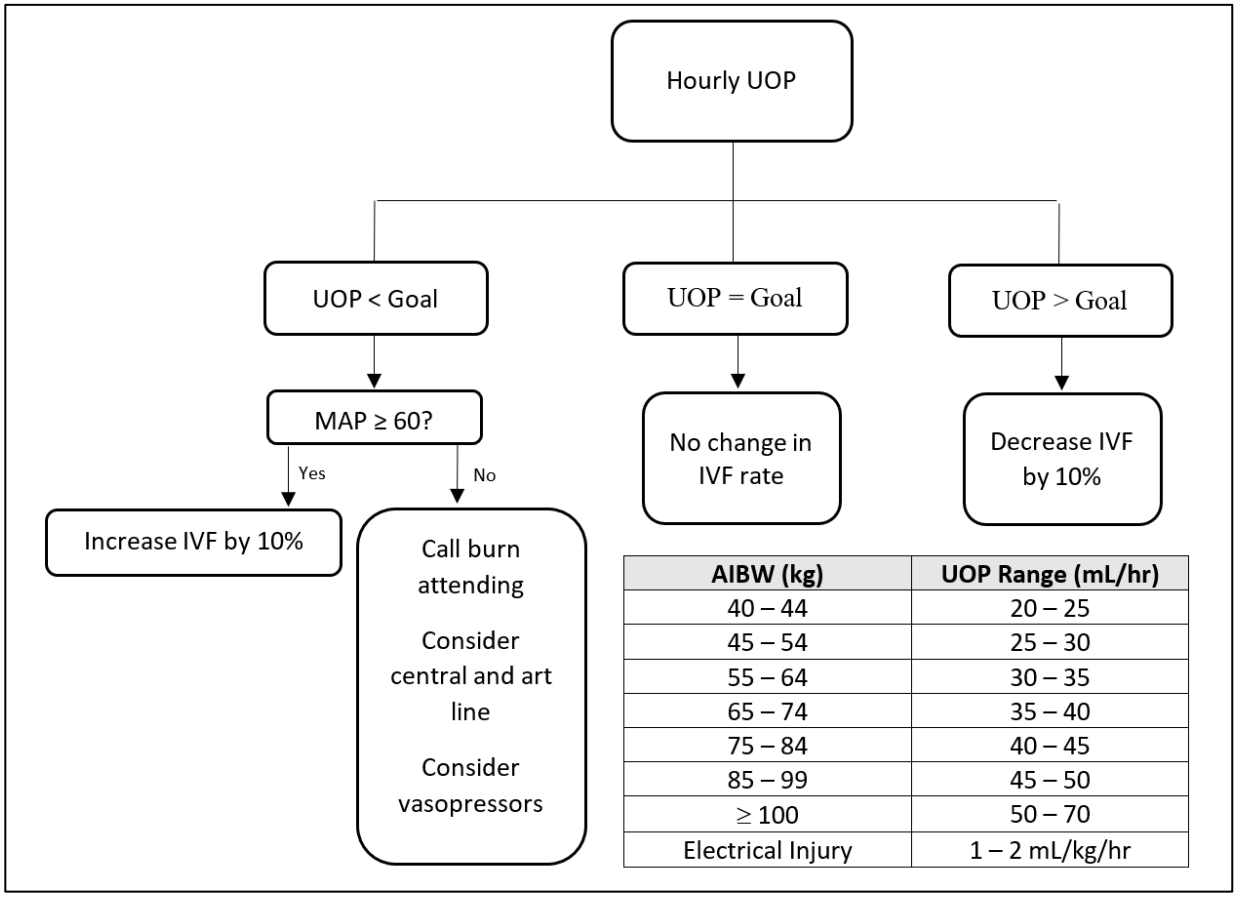
Step Two: Resuscitation

- **≥30%-50% TBSA** burns – give **1 unit of FFP** bolus on admission
- **>50% TBSA** burns – give **2 units of FFP** bolus on admission

**Use the Rule of 10's to calculate the fluid starting rate using the AIBW (attending discretion)**

a. < 80kg: 10 x _____ (%TBSA)= _____ mL/hr
b. ≥ 80kg: 10 x _____ (%TBSA) + _____ (100 mL for every 10kg over 80 kg) = _____ mL/hr
c. **+/- Electrical injury**: Treat as **20% TBSA minimum** unless >20% cutaneous TBSA – then treat at calculated TBSA% for resuscitation. (Do NOT add 20% to all TBSAs)
d. +/- **Inhalation injury:** Every patient with proven or highly suspected inhalation injury must have an acute burn resuscitation (treat as **20% TBSA minimum).** For patients with **≥3% cutaneous TBSA, simply add 17% to calculated TBSA** then use above "Rule of 10s" calculation.
e. Follow the algorithm or the Burn Navigator by increasing or decreasing the total fluids by 10% hourly based on the urine output goals (LR only – do not titrate the FFP)
f. **Electrical injury:** Keep UOP 1-2 mL/kg/hr until urine is clear & yellow and CK levels have plateaued and are decreasing
g. Record the hour-to-hour changes in the resuscitation flow sheet
h. We will expect to see a correction in the lactate as well as improvement in hemoconcentration if the resuscitation is going well
i. Call burn attending for any questions or concerns

**If TBSA ≥ 20% Flowsheet**




| AIBW (kg) | UOP Range (mL/hr) |
|---|---|
| 40 – 44 | 20 – 25 |
| 45 – 54 | 25 – 30 |
| 55 – 64 | 30 – 35 |
| 65 – 74 | 35 – 40 |
| 75 – 84 | 40 – 45 |
| 85 – 99 | 45 – 50 |
| ≥ 100 | 50 – 70 |
| Electrical Injury | 1 – 2 mL/kg/hr |

Step Three: FFP Drip

Population: All burns > 20% TBSA at 8 hours post burn

**FFP Drip calculations:**

**Runs for 8 hours – from hour 9-16 post burn**

Total FFP gtt Volume = 0.5mL x _____ (AIBW in kg) x _____ (%TBSA)=_____ mL

FFP gtt Rate = _____ (Total FFP Volume) / 8hrs = _____ mL/hr

Step Four: Alarm Indications

**Contact the Burn Attending:**

a. Prior to initiation of:
   -Vasopressors
   -Diuretics
   -Steroids
   -Antibiotics
b. Inability to wean IV fluids X 4 hours
c. Low UOP X 2 hours consecutively
d. Hypotension: MAP<60 or hemodynamic instability
e. Patient has reached alarm rate (2 X initial starting rate) of IVF
f. Total resuscitation projected to reach the Ivy Index: 250 mL/kg AIBW
g. Any other concerns that the patient is off the expected course

**Monitoring Resuscitation**

**Vital Sign Monitoring & Interventions for Hemodynamic Abnormalities**

Heart rate and blood pressure are monitored throughout the resuscitation. At 6 hours of inpatient protocolized resuscitation, admission labs should be repeated.

The trajectory of the resuscitation at the end of the first 6 hours should be reviewed by the resident and APP. Both parties will sign off their satisfaction (or lack thereof) with the resuscitation. This information should be conveyed to the attending burn attending and documented on the flowsheet and in the patient's chart.

This should occur even if the resuscitation is going well.

Any 2 consecutive hours without meeting UOP goals or with signs of worsening end-organ perfusion require re-evaluation by the resident/APP and the nurse, review of the labs and hemodynamics, and a call to the burn attending.

Labs (CBC, CMP, and lactate) will be redrawn every 6 hours throughout the resuscitation.

If at any point either the bedside nurse or the resident feel that the trajectory of the resuscitation is poor, they **MUST** let the burn attending know that the patient is exhibiting signs of a potentially failing burn resuscitation. This must be documented.

Step Five: Cessation of Resuscitation

a. Calculate the resuscitation cessation fluid rate using mIVF and insensible loss estimate using the following calculation:
   ( ____ kg + 40 mL) + [(25+ ____ %TBSA) x____ $m^2$ BSA]= _____ mL/hr
b. Once the patient's total fluids have been decreased to the resuscitation cessation rate for **4 consistent hours**, they have completed resuscitation.

c. **Document** the end time of the resuscitation on the patient's chart and on the resuscitation flow sheet.
d. Discontinue foley catheter within 4-6 hours post resuscitation completion.

***Use caution when discontinuing IV fluids in patients who still have the inflammatory drive and insensible losses of the burn injury.***

## VII. Failing Resuscitations

**A.** **Projected Failure:** Delineated into 3 phases based on presentation/recognition: Early (first 8 hours), In-resuscitation (8-24 hours), and late (after 24 hours). No specific criteria, but generally accepted as significant deviations from calculated protocol without improvement. If at any time the total resuscitation fluids is projected to be > 250 mL/kg AIBW (Ivy Index), there is a high likelihood of failure.

**B.** **Tachycardia**: Frequently multifactorial in large burns, but if the patient develops a sustained heartrate of >140 BPM please contact the burn attending. Pain and anxiety should be addressed first. Intoxication should be considered if UOP is adequate and pain/anxiety have been addressed. If tachycardia is accompanied by **hypotension** (defined as a MAP of <60 mmHg), the most likely diagnosis is burn shock due to inadequacy of resuscitation and the patient should be started on a vasopressor agent along with a simultaneous increase in the hourly infusion rate by 20%. If vasopressors are initiated, the burn attending must be notified.

**C.** **Oliguria** (UOP <30 mL/hr): If oliguria persists despite 2 hours of escalation on the protocol, contact the burn attending. A bolus of 50 mL of 25% albumin may be considered. The burn attending must be notified in any of these cases. A discussion should be initiated regarding additional strategies to mitigate a failing burn resuscitation.

**D.** **Lactatemia**: Although lactatemia can have multiple causes, increasing or persistently elevated lactate should not be ignored. Hepatic function should be evaluated with a persistent lactatemia. Also, concerns for ischemia (e.g., abdominal or limb compartment syndrome) or inadequate oxygen delivery should be immediately assessed. If elevated lactate (serum lactate persistently above 2.5 or up-trending) persists after 4 hours of acute resuscitation, there should be strong consideration for beginning a vasopressor agent along with a simultaneous increase in the hourly infusion rate by 20%. If vasopressors are initiated, the burn attending must be notified.

## VIII. Mitigation of Failure

Consideration of Causes: Patients who are failing their resuscitation should have a comprehensive physical examination and evaluation of all of their potential risk factors for failure, as described above. This should be performed immediately as it is identified, irrespective of previous evaluations. Labs, including CBC with differential, CMP, serum lactate, and coagulation labs, should be repeated at this time. The evaluation should also include evaluation of cardiac function, such as point of care ultrasound or echocardiogram. Results of this evaluation should be discussed with the burn attending in case any of them

can be intervened upon without significant alteration to the resuscitation protocol. In all others, and only with the agreement of the burn attending, consider the following:

**A. Vasoactive medications**

There are no prospective, randomized control trials regarding the use of vasopressors in burn. The rare, descriptive studies and protocols using vasopressors tend to agree that they may have a role in patients with sustained hypotension and *potentially* in sustained oliguria.

1. Ideal Population: Sustained MAP ≤60, despite fluid resuscitation by protocol.
2. Avoid in: Under-resuscitated patients.
3. Potential medications (starting doses):
    a. Norepinephrine: 5 mcg/min
    b. Vasopressin: 0.04 units/min,
    c. Epinephrine: 2 mcg/min
    d. Each should be titrated to effect
    e. Phenylephrine is NOT first line and should only be used by approval of burn attending

**B. Further Fluid Administration**

Patients who are failing their resuscitation are likely to already have had significant escalation in their fluid administration. It is very likely that these patients are no longer fluid responsive and further escalation would increase the risk of excessive resuscitation and increase mortality. Any further escalation in resuscitation should be done with this in mind and should only be done under the direction of the burn attending.

1. Ideal population: intravascular deplete (US, PAC, etc.) and fluid responsive (pulse pressure variation, leg raise, dynamic US changes, etc.). Consider in patient dehydrated at admission.
2. Avoid in: patients with anasarca and evidence of worsening intra-abdominal hypertension or abdominal compartment syndrome.

**C. Hemofiltration**

Continuous renal replacement therapy may play a role in mitigating failing burn resuscitation through removal of inflammatory mediators. It is known that CVVH can reasonably filter out many small, water-soluble immune mediators efficiently and may improve mortality in some populations.(20) In the burn population, CVVH may improve shock and decrease vasopressor requirements, but its outcome on mortality is unclear.(21-23) For now, CVVH should be considered in patients with a need for RRT and/or escalating shock. This requires evidence of adequate intravascular resuscitation, placement of an appropriate vascular catheter, and consultation with the nephrology service.

1. Ideal population: Patients who present with renal failure or those who have isolated oliguria and worsening renal function. Patients must be close to euvolemic and will continue to require resuscitation. Unclear stopping point, but cessation should be considered at 24 hours and should not need to go for more than 48, except in patients with pre-existing or worsening renal failure.
2. Avoid in: Under-resuscitated patients.

**D. Very Early Burn Excision** (within 48 hours)

The decision to excise a patient before they have completed their resuscitation is challenging and should be based on clinician judgment. The vast majority of patients should be through their resuscitation and through the diuretic phase of resuscitation before an operation. Very early excision has been shown to decrease pro-inflammatory cytokines and improve mortality in select patients. While there are real limitations on very early excision (e.g., need to continue resuscitation in the OR; higher blood transfusion requirements), it is reasonable and should be considered.

**E. Hypertonic Solutions**

There are very few studies addressing the use of hypertonic agents in burn resuscitation. For the most part, these studies have not demonstrated any benefit and there is good reason to suspect potential harm with the administration of hypertonic fluids for resuscitation purposes.(24) Therefore, these solutions **should not be used** during the resuscitation phase unless there is a different indication (e.g., concern for raising ICP or symptomatic hyponatremia) and only after discussion with the burn attending.

**F. Plasma Exchange/Plasmapheresis**

Plasma exchange (PlEx)and plasmapheresis are extracorporeal blood purification methods that fractionates whole blood and returns components free of, in this case, inflammatory mediators to the patient. PlEx has been shown to decrease fluid requirements to reach goal UOP in difficult burn resuscitations, as well as improve base deficit, lactate, and hematocrit.(25)

1. Ideal Population: Patients with large, survivable burn wounds exhibiting oliguria despite escalation of resuscitation. Especially in patients at greater than BFR 4.
2. Avoid in: The vast majority of patients are unlikely to need this.

## IX: Complications/Comorbidities

**A. Heart Failure**

Burn induced inflammatory mediators can reduce cardiac output and increase systemic and pulmonary vascular resistance. Patients with preexisting heart failure can quickly exhaust their cardiac reserve in the setting of a large burn. These patients may benefit from more formalized metrics of resuscitation such as a pulmonary artery catheter (PAC) or echocardiogram.

1. PAC is only useful in the hands of a skilled and experienced provider who is able to reliably place the catheter, analyze the data, and make appropriate interventions. The target cardiac output (CO), peripheral vascular resistance (PVR), and mixed venous oxygen saturation ($S_vO^2$) should be decided by the burn attending.
2. CVVH may be indicated for patients who need resuscitation but cannot tolerate the volumes required due to heart failure.
3. Medications to consider in this population include the following:
    a. ACE inhibitor – offload cardiac stress - expected increase in creatinine
    b. Dobutamine started at 2.5 g/kg/min and titrated to effect
    c. Epinephrine started at 2 mcg/min
    d. Other: β-1 agonist +/- phosphodiesterase inhibitor

**B. Renal Failure**

In patients with renal failure, whether acute or chronic, decisions must be made regarding an alternative metric of resuscitation and management of renal failure. Alternative metrics of resuscitation in these patients include hemodynamic changes, mental status, labs indicating acid-base status and oxygen availability, and invasive monitors, such as PAC.

1. In patients who meet criteria for renal replacement, or those on outpatient dialysis, CVVH should be used for the acute resuscitation to minimize hemodynamic perturbations of hemodialysis.
2. BMP should be regularly obtained to evaluate BUN and $K^+$. A baseline EKG should be obtained.

**C. Liver Failure**

The presence of liver disease is an independent predictor of mortality in burn patients. Patients with chronic liver disease often have a reduced SVR at baseline and are highly-dependent on increased cardiac output to meet their oxygen delivery. If these patients experience a significant burn injury, it is very difficult for them to meet the increased demand for oxygen delivery. Also, the liver is responsible for mobilizing glucose stores and synthesizing proteins needed for the acute phase reactions needed to maintain normal physiologic function. Liver patients have varying degrees of coagulopathy, which will complicate the clinical course of a burn patient. Of note, most tests of coagulation (e.g., INR) are **NOT** good measures of coagulation status in a patient with liver disease. When possible, a more comprehensive test, such as a TEG should be considered.(26)

**D. Hypothermia**

Significant hypothermia is common in burn patients. If severe, it can cause alterations in oxygenation and electrolyte balance. Interventions include:

Hypothermia Management

| | |
|---|---|
| <35$^{o}$ | • Forced Air Warmer (BAIR Hugger)<br>• Increase Room Temp<br>• Heat Lamps<br>• Plastic Covering<br>• Thermal Surgical Cap<br>• Fluid Warmer<br>• Heated Ventilator Circuit |
| 35-36$^{o}$ | • Forced Air Warmer (BAIR Hugger)<br>• Increase Room Temp<br>• Heat Lamps<br>• Plastic Covering<br>• Fluid Warmer<br>• Heated Ventilator Circuit |
| >36$^{o}$ | • Forced Air Warmer (BAIR Hugger)<br>• Increase Room Temp<br>• Continuous Monitoring |

E. **Compartment Syndrome**

Patients receiving high-volume resuscitations are at risk for developing ocular, abdominal, and extremity compartment syndrome. Any patient who cannot communicate vision changes should have an ophthalmology consult to measure intraocular pressures and intervene if elevated. If there is suspicion of abdominal or extremity compartment syndrome, notify burn attending immediately. All patients with circumferential full thickness burn on an extremity should receive hourly neurovascular assessments until discontinued by the burn attending.

F. **Electrical Injury and (concomitant) Inhalation Injury**

These mechanisms have separate practice management guidelines, but from a resuscitation standpoint, it is important to remember that the %TBSA will underestimate the fluid requirements in these patients as the underlying tissue damage is greater than what is visible on presentation.

Electrical Injury patients are also at a greater risk of compartment syndrome and acute renal injury due to underlying muscle damage. This may require supra-normal urine output targets for the resuscitation and a lower threshold for compartment decompression.

Inhalation injury increases fluid requirements relative to %TBSA estimate in patients with burns >25% TBSA. As such, the %TBSA alone would be an inadequate metric to estimate to total fluid requirement. (27)

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