MAYO CLINIC

HOSPITAL MEDICINE MEETS CRITICAL CARE

PATIENT CASES

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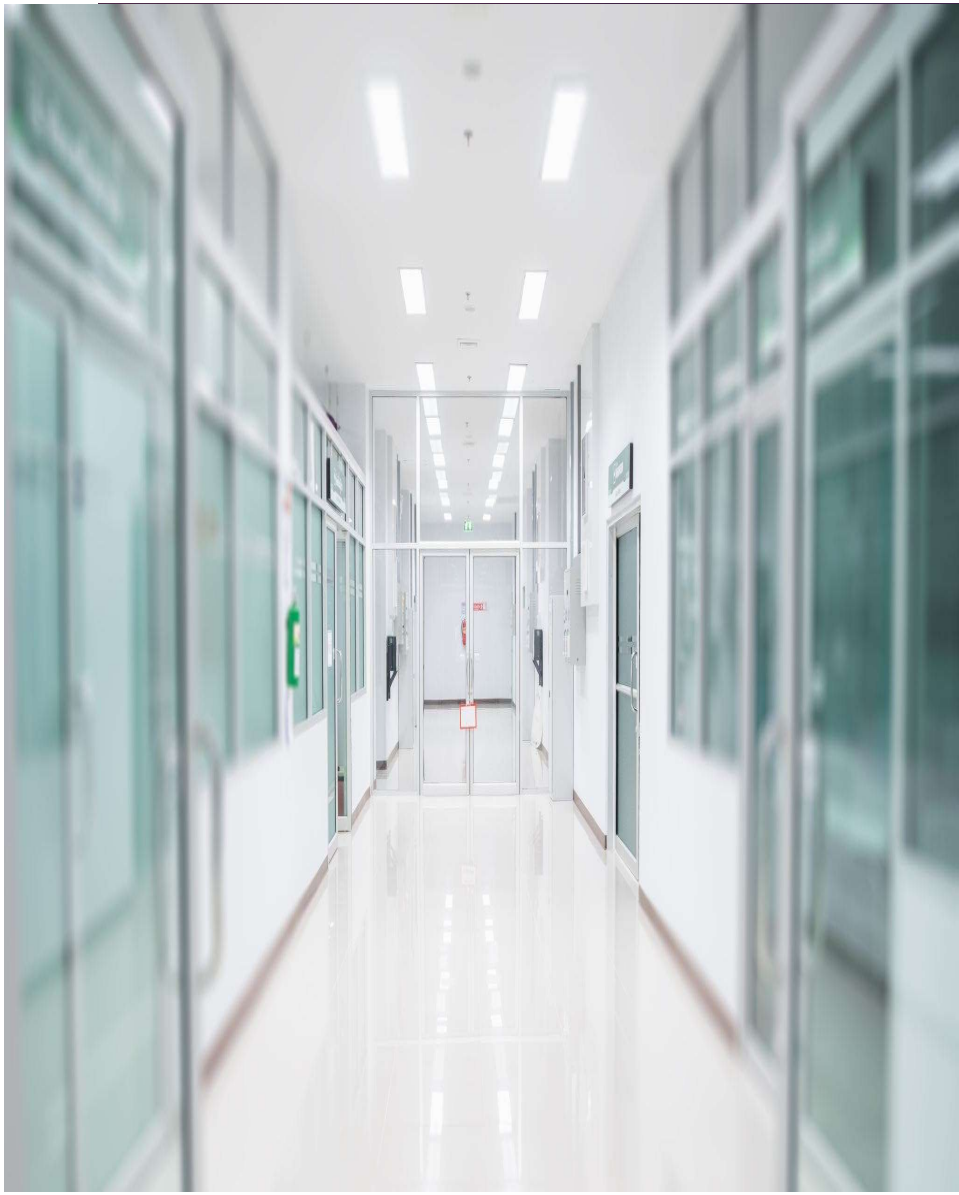
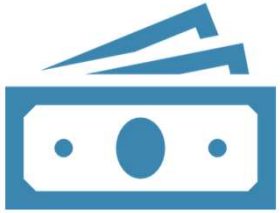


DISCLOSURES



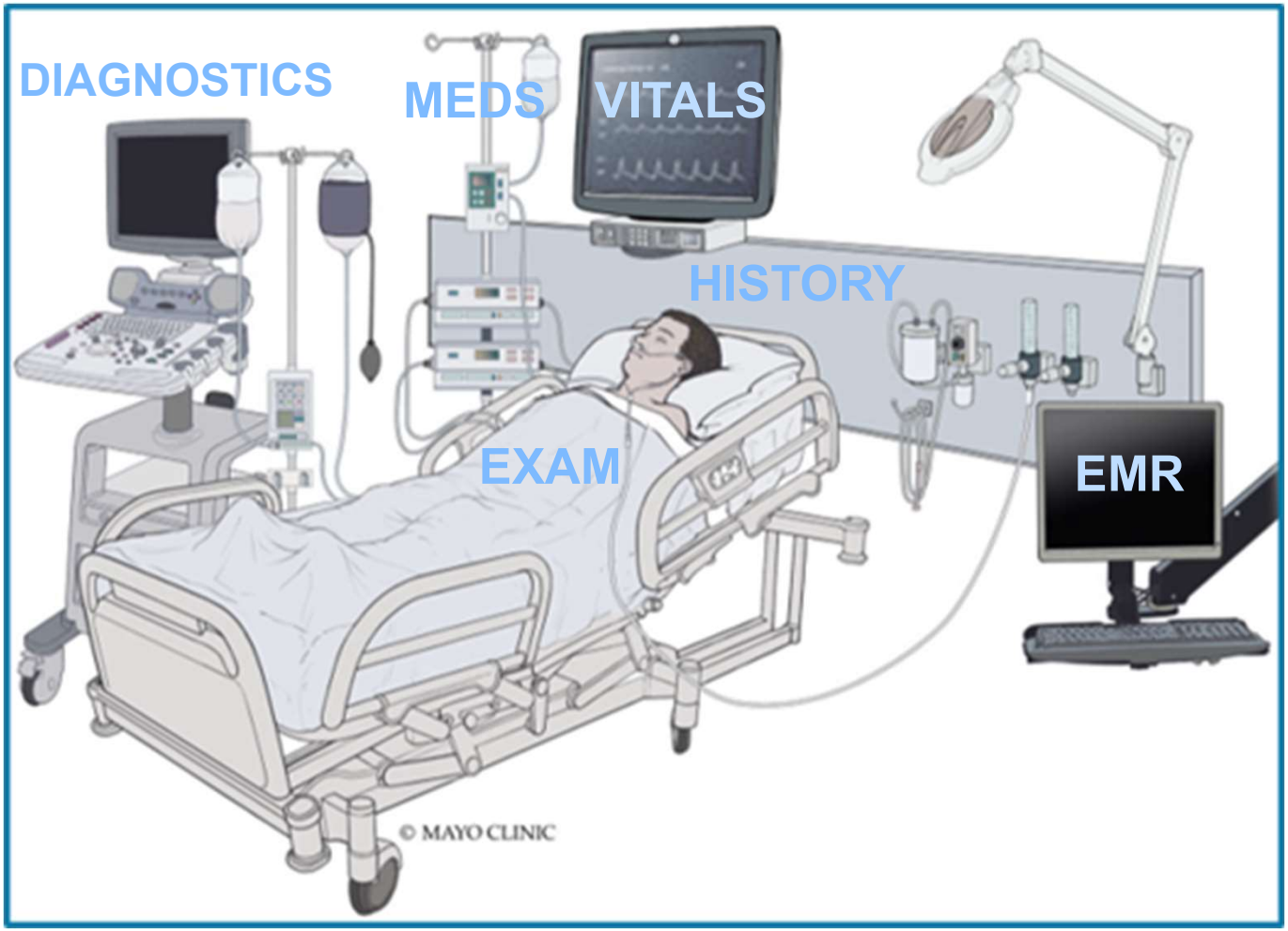
This presentation has no affiliation or financial arrangements.

Off-label use of medications will be mentioned.

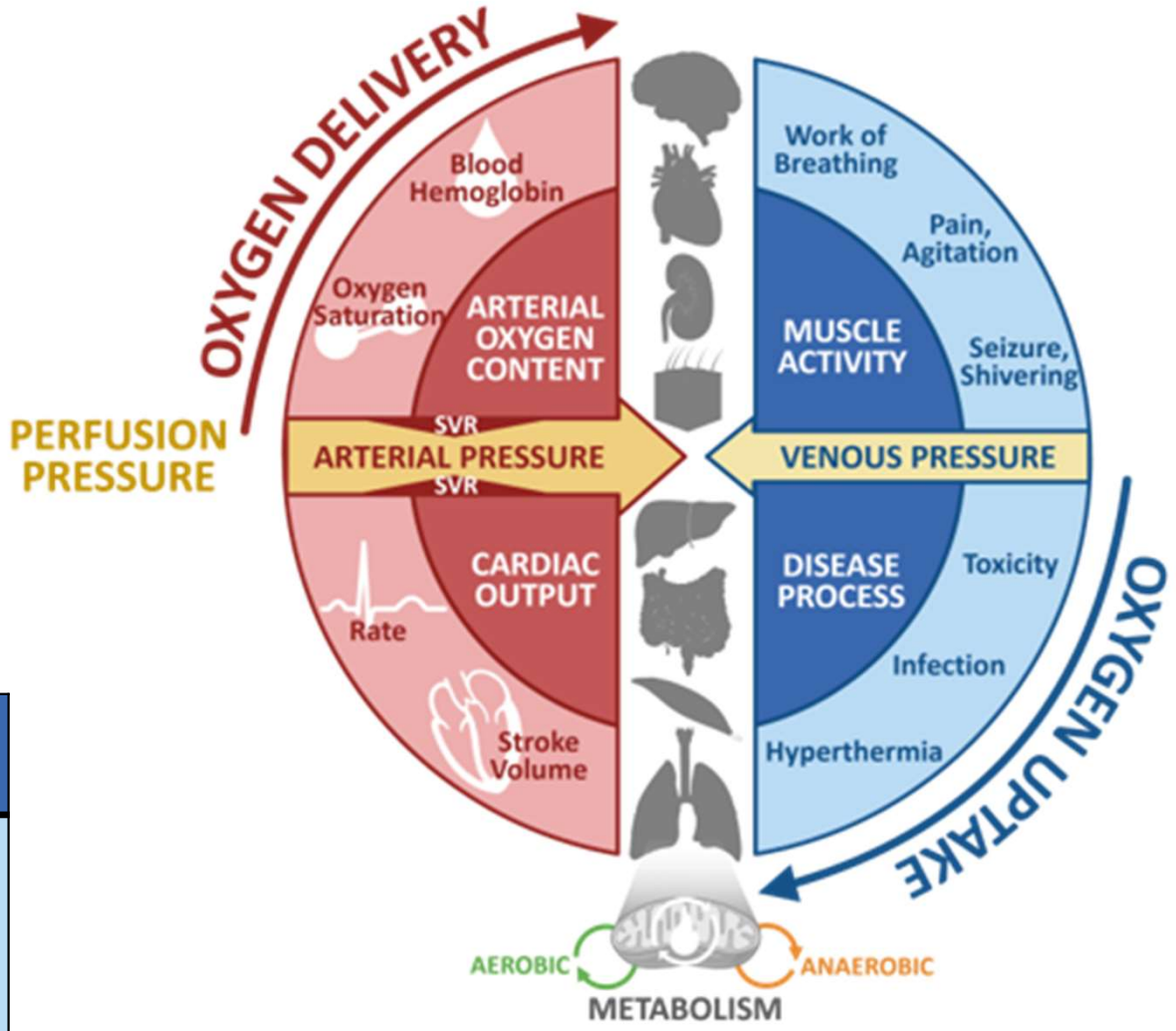
LEARNING OBJECTIVES

1. Identify patients who warrant evaluation by a critical care team and discuss early resuscitative measures.
2. Review differential diagnosis for shock state, and how to best resuscitate patients in shock.
3. Discuss how to manage a patient in decompensated liver failure.
4. Increase awareness that multiple shock types can be present at the same time.
5. List ways to monitor end organ perfusion, and therefore treatment response in shock.

APPROACH TO A “SICK” PATIENT



SHOCK



MARKERS OF SHOCK (HYPOPERFUSION)

- Altered Mental Status
- Delayed capillary refill
- Low UOP (<0.5ml/kg)
- Lactate >2.5
- Acidosis
- Elevated troponin

CAUSES:

↓ BLOOD O_2 CONTENT
- ↓ Blood (Hgb/Hct)
- ↓Oxygen Saturation (SpO_2)

↓ VASCULAR TONE (SVR)
- Sepsis
- Anaphylaxis
- Liver Dysfunction
- Adrenal Insufficiency, Autonomic Dysfunction, Acidosis
- Drugs, Devices

↓ CARDIAC OUTPUT
- Arrhythmia
- Heart Failure (↓ Function)
- ↓ Volume (Bleeding, Third Spacing, Dehydration)
- Obstruction (PE, PTX, Tamp.)
- Valvular Disease
- ↓ Filling (Thick Heart)
- ↑Afterload (SVR/BP)

↑ O_2 CONSUMPTION
- Pain, Agitation, Distress
- Exercise, Activity
- ↑ Work of Breathing
- Seizure
- ↑ Metabolism

↑ VENOUS CONGESTION
- Congestive Heart Failure
- RV Failure
- Pneumothorax
- Tamponade
- Volume Overload
- ↑ Compartment Pressures (Intracranial, Thoracic, Abdominal, Limb)

Special thanks to Hannelisa Callisen, PA-C & Dr. Bhavesh Patel for the creation of this image

TIME TO START YOUR WORK DAY!

- You are a busy hospital internal medicine APP and just arrived at work.

- You turn on your pager and within **seconds**, you recognize the familiar tone of a page…

MR. SCHRUTE

- 58M with history of nephrolithiasis, DM2, HTN, and complete heart block s/p pacemaker placement (2 years ago) who presented a few hours ago with **nausea** and vague **abdominal pain**.
- He remains hypotensive, despite 3 L of IV fluids in the ER
- A rapid response is called due to persistent hypotension

RAPID RESPONSE

You rush to bedside to find the following:

- **HR:** 60
- **RR**: 30s
- **BP**: 72/41 (51)
- **Temp**: 38.6 C
- **O2**: 93% on RA

What are some of your initial interventions?

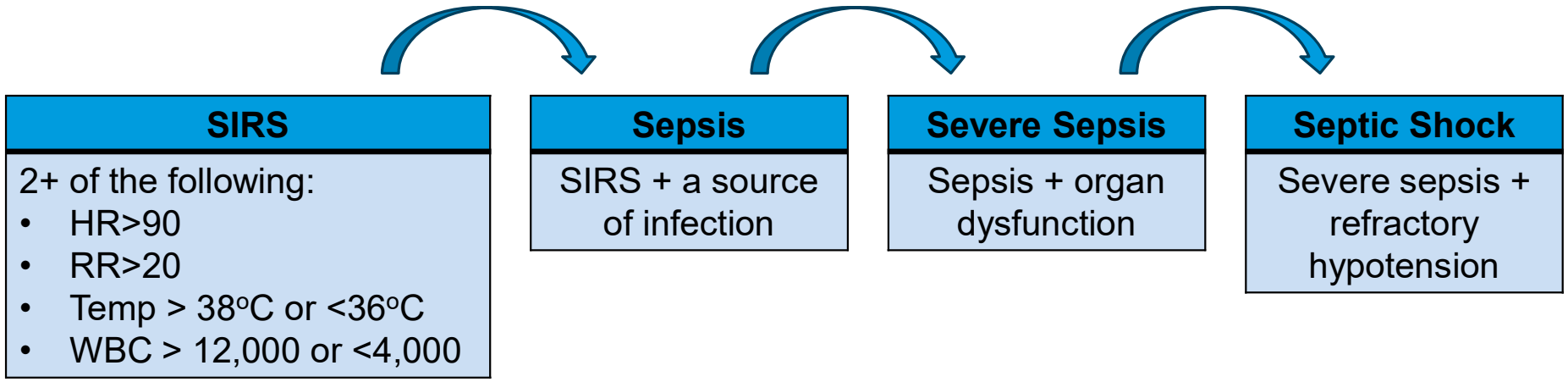
EARLY SIGNS OF SEPSIS

Fever or hypothermia
Tachypnea
Tachycardia
Hypotension
Hyper/hypoglycemia
Decreased UOP

AFP. 2013 July 1; 88 (44-53)

BATTLE OF THE SEPSIS SCORING SYSTEMS

SIRS



- Surviving Sepsis Campaign Guidelines
- SIRS criteria is fairly **nonspecific** for sepsis, but **more sensitive** at identifying septic patients
- Helpful for diagnosis/billing & coding

SEQUENTIAL ORGAN FAILURE ASSESSMENT (SOFA)

Table 1. Sequential Organ Failure Assessment Score

Variables	SOFA Score				
	0	1	2	3	4
Respiratory	PaO_2/FiO_2: > 400 SpO_2/FiO_2: > 302	PaO_2/FiO_2: < 400 SpO_2/FiO_2: < 302	PaO_2/FiO_2: < 300 SpO_2/FiO_2: < 221	PaO_2/FiO_2: < 200 SpO_2/FiO_2: < 142	PaO_2/FiO_2: < 100 SpO_2/FiO_2: < 67
Cardiovascular (doses in mcg/kg/min)	MAP ≥ 70 mm Hg	MAP ≥ 70 mm Hg	Dopamine ≤ 5 or ANY dobutamine	Dopamine > 5 Norepinephrine ≤ 0.1 Phenylephrine ≤ 0.8	Dopamine >15 or Norepinephrine > 0.1 Phenylephrine > 0.8
Liver (bilirubin, mg/dL)	< 1.2	1.2-1.9	2.0-5.9	6.0-11.9	> 12
Renal (creatinine, mg/dL)	< 1.2	1.2-1.9	2.0-3.4	3.5-4.9	> 5.0
Coagulation (platelets x $10^3/mm^3$)	≥ 150	< 150	< 100	< 50	< 20
Neurologic (GCS score)	15	13-14	10-12	6-9	< 6

qSOFA

Not very sensitive or specific for sepsis, much better used as a **prognostic** tool.

Full SOFA score is one of the best predictors of in hospital mortality.

Comput Math Methods Med. 2022 Aug 10;2022:7870434. doi: 10.1155/2022/7870434.

MODIFIED EARLY WARNING SCORE (MEWS)

Score	3	2	1	0	1	2	3
Temp	< 32	< 35	< 36	36 - 38.4	38.5 - 38.9	39 - 40.9	≥ 41
HR	< 40	40 - 44	45 - 50	51 - 100	101 - 110	111 - 129	≥ 130
RR	≤ 7	8	9	10 -14	15 - 20	21 - 29	≥ 30
SBP	≤ 70	71 - 80	81 - 100	101 - 160	161 - 180	181 - 199	≥ 200
Mental Status Change**	Unresponsive, coma	Stupor, responds to noxious stimuli	Lethargic, responds to voice or tap	Alert, calm, cooperative	Mildly agitated, confused, anxious	Very agitated, requires restraints	Extremely agitated and danger to self or others
Latest WBC	< 1*	1 - 2.9*		3 - 14.9	15 - 19.9	20 - 39.9	≥ 40

****Score of 4+ is more alarming**

Crit Care Explor. 2021 May 18;3(5):e0386. doi: 10.1097/CCE.0000000000000386.

NATIONAL EARLY WARNING SCORE (NEWS)

Physiological Parameters	3	2	1	0	1	2	3
Respiration Rate (BPM)	≤8		9–11	12–20		21–24	≥25
Oxygen Saturation (%)	≤91	92–93	94–95	≥96			
Any Supplemental Oxygen		Yes		No			
Temperature	≤35		35.1–36.0	36.1–38.0	38.1–39.0	≥39.1	
Systolic Blood Pressure (mmHg)	≤90	19–100	101–110	111–219			≥220
Heart Rate (BPM)	≤40		41–50	51–90	91–110	111–130	≥131
Level of Consciousness				Alert			U, P or V*

Note: *Unresponsive, react to pain, or loud voice.

Sensitive screening tool for predicting sepsis-related outcomes, **but not very specific**.

Outperformed other scores for **predicting hospital admission and mortality**

Infect Drug Resist. 2020 Oct 27;13:3843-3851. doi: 10.2147/IDR.S275390.
PMID: 33149629; PMCID: PMC7602891.

SOOO....WHICH SHOULD I USE?

- They all have some utility to help screen for patients who are going to have higher mortality/need higher level of care.
- SIRS still being used for screening/diagnosis of sepsis (and billing!).
- Predicting sepsis in the ED:
  - NEWS outperformed other scores for predicting hospital admission.
  - qSOFA >> SIRS for predicting mortality in sepsis
    - Lactate is superior to qSOFA for sepsis prognostication.
    - qSOFA is **not** mean to be a diagnostic tool.
  - Full SOFA score was best predictor of in hospital mortality for sepsis patients

The American Journal of Emergency Medicine,Volume 46, 2021 (284-288),ISSN 0735-6757, https://doi.org/10.1016/j.ajem.2020.07.077.

RESUSCITATION

- Physiology stabilization and resuscitation precedes definitive diagnosis & treatment of underlying cause

- **What are your initial interventions for Mr. Schrute?**

2018 SURVIVING SEPSIS BUNDLE UPDATE

One-hour Bundle

- Measure lactate level. Remeasure if initial lactate is >2 mmol/L.
- Obtain blood cultures prior to administration of antibiotics.
- Administer broad-spectrum antibiotics.
- Begin rapid administration of 30ml/kg crystalloid for hypotension or lactate ≥4 mmol/L.
- Apply vasopressors if patient is hypotensive during or after fluid resuscitation to maintain MAP ≥65 mm Hg.

**"Time zero" or "time of presentation" is defined as the time of triage in the Emergency Department or, if presenting from another care venue, from the earliest chart annotation consistent with all elements of sepsis (formerly severe sepsis) or septic shock ascertained through chart review.*

Crit Care Med 2018;46(6):997

2021 SURVIVING SEPSIS UPDATES

- Balanced fluid >>> NS (weak recommendation)
- Do NOT use qSOFA as a single screening tool for sepsis (strong recommendation)
- Initiate antimicrobials within **1 hour** (strong recommendation)
  - Even if sepsis is just suspected, and not confirmed.
- Consider starting pressors through a peripheral line while waiting to get central access, if there is going to be a delay (weak recommendation)
- IV corticosteroids for adults with septic shock who have an ongoing vasopressor requirement
- Lots of recommendations for economic & social support screening, and ensuring adequate post ICU care/post discharge follow up.

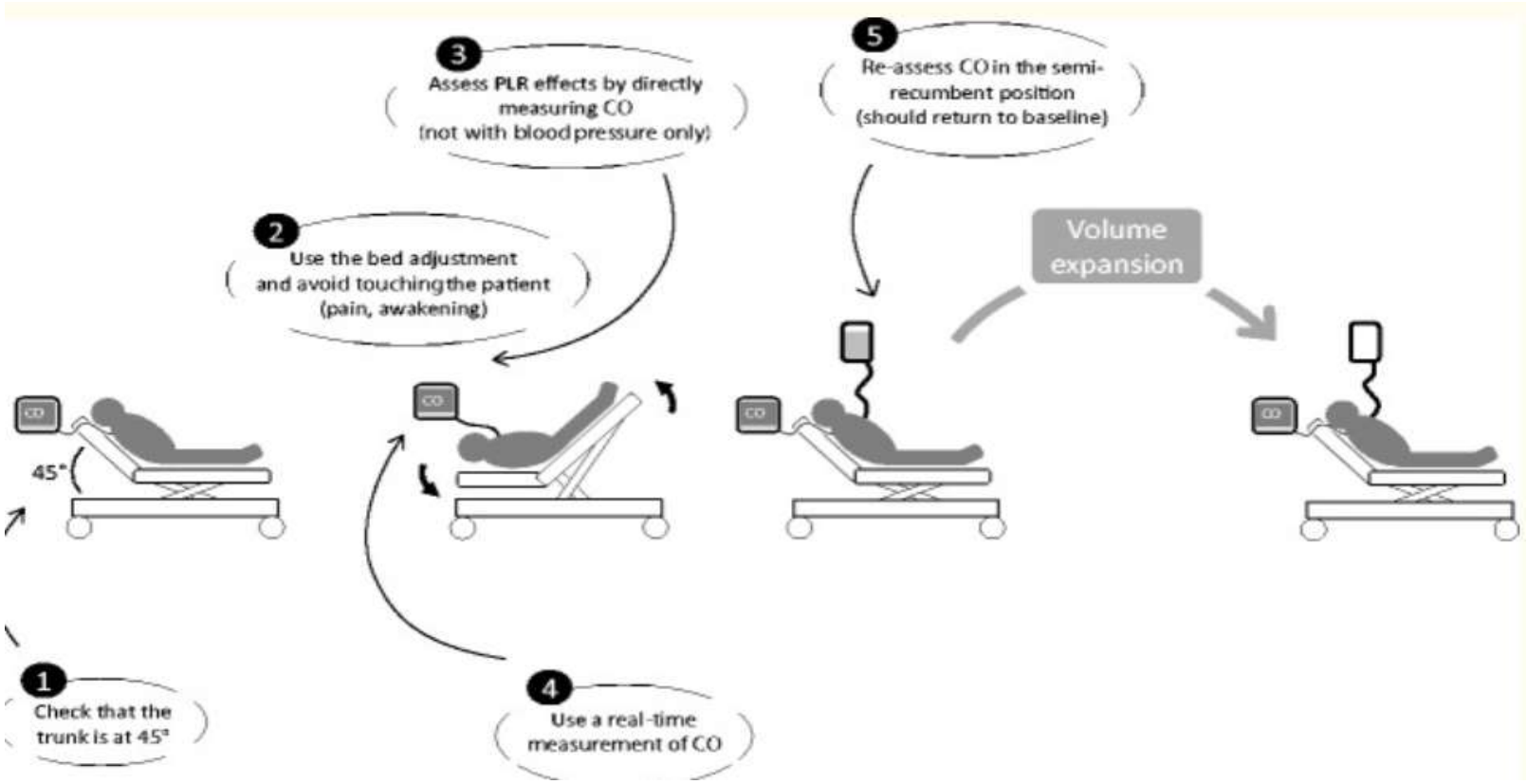
Crit Care Med. 2021;49(11):e1063–e1143. doi: 10.1097/CCM.0000000000005337.

RESUSCITATION

Mr. Schrute is still hypotensive, despite the 3L of fluid he received, what are some strategies to assess fluid status?

Fluid vs. Pressors

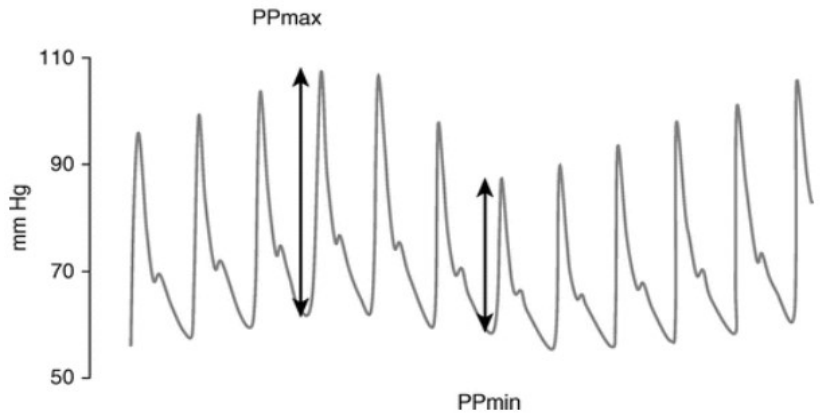
PASSIVE LEG RAISE

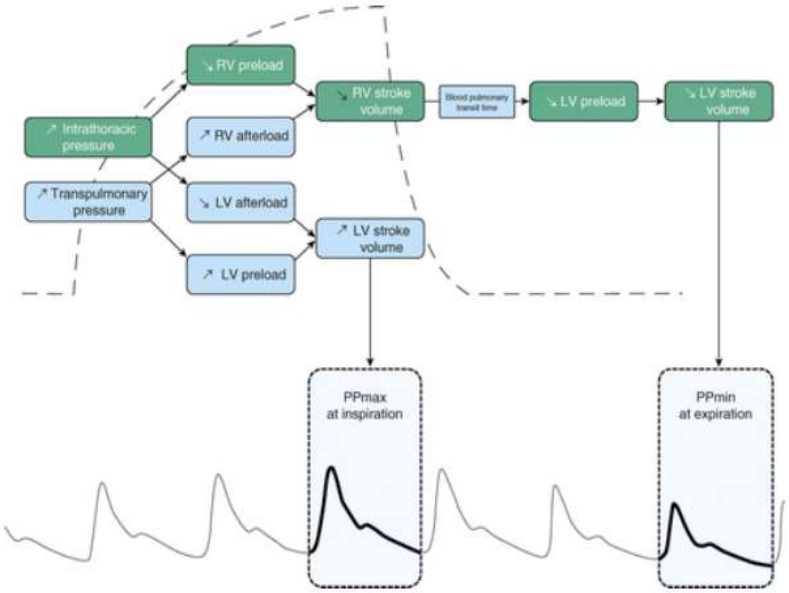


Monnet, Teboul. Crit Care. 2015; 19(1): 18.

PULSE PRESSURE VARIABILITY

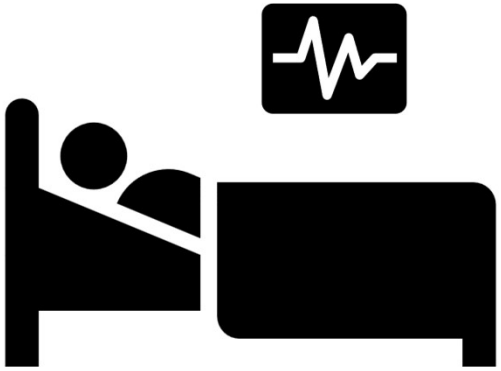
$$PPV = \frac{PPmax - PPmin}{(PPmax + PPmin) / 2}$$





Jean-Louis Teboul et al. American Journal of Resp and Critical Care Medicine. Volume 199, Issue 1. 2018. https://doi.org/10.1164/rccm.201801-0088CI

Unfortunately, Mr. Schrute remains hypotensive, plus he develops increased work of breathing…



ICU

IN THE ICU...

- He becomes more tachypneic , increased work of breathing with some hypoxia
  - ABG on nasal cannula: 7.21 / 21 / 69 / 9

- Intubated & started on pressors
  - **Would you have intubated this patient??**

PROGRESSIVE SHOCK

- Central line and arterial line placed
  - Started on pressors: norepinephrine first, then vasopressin added
- Workup for source of infection only c/w abnormal UA.
  - Sent to CT for CT head/chest/abd/pelvis
  - **Would you have sent this patient to CT?**

ICU COURSE

Labs:

3.9	9.9	170
	28	

135	98	30	356
4.3	12	**4.8**	

ALT: 1455

AST: 1670

CT A/P: Obstructive R renal calculus, 8x6mm with mild R hydroureteronephrosis

STAT IR placement of R nephrostomy tube

ICU COURSE

What are some other options to treat Mr. Schrute's refractory vasodilatory shock?

Stress dose steroids

Other pressors:

- Angiotensin II?
  - ATHOS-3 trial

HAT (Hydrocortisone, Vitamin C, & Thiamine) therapy is **NOT** recommended

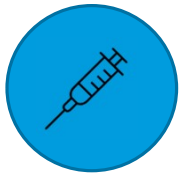
[1]Sprung CL, et al. Hydrocortisone therapy for patients with septic shock. N Engl J Med. 2008 Jan 10;358(2):111-24
[2]JAMA 2020;324(7):642
[3]JAMA 2019;322(13):1261
[4]JAMA 2021;325(8):742

ICU COURSE

*** Off-label use!**

What are some other options to treat Mr. Schrute's refractory vasodilatory shock?

Methylene Blue *

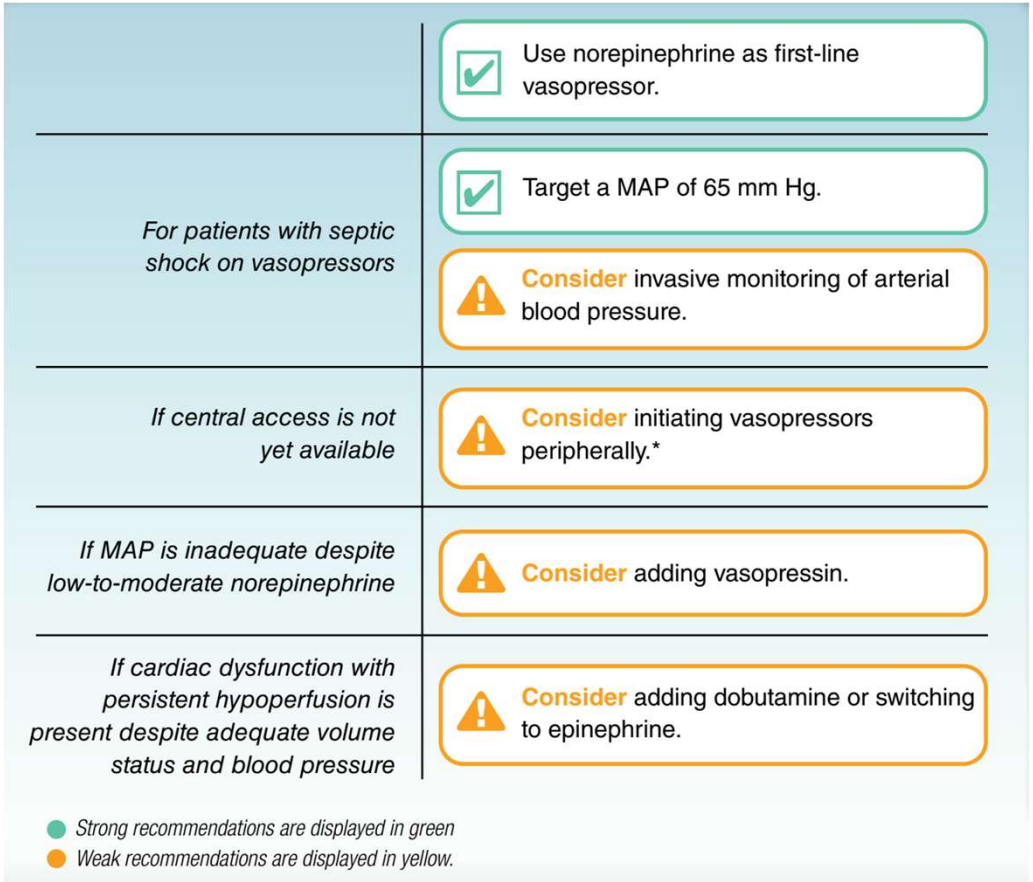


Hydroxycobalamin (CyanoKit)*

Kwok ESH, Howes D. *Journal of Intensive Care Medicine*. 2006;21(6):359-363
Park BK, Shim TS, Lim CM, et al. . *Korean J Intern Med*. 2005;20(2):123-128.
Can J Anaesth. 2017 Jun;64(6):673-674.
J Cardiothorac Vasc Anesth. 2018 Aug 11
Feih, et al. Journal of Cardiothoracic and Vascular Anesthesia, Volume 33, Issue 5, 2019,Pages 1301-130

VASOACTIVE MANAGEMENT

SURVIVING SEPSIS 2021 UPDATE



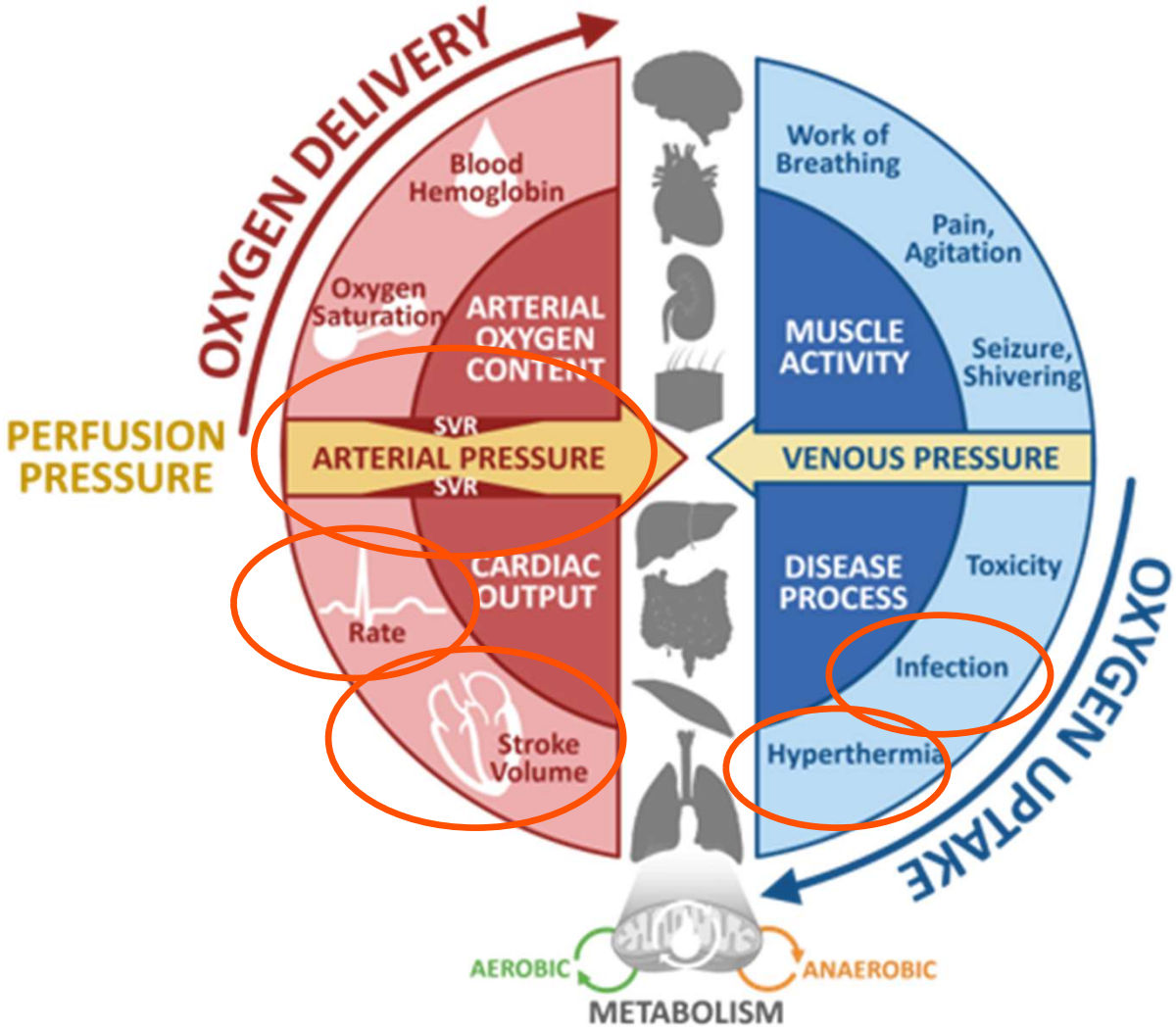
Crit Care Med. 2021;49(11):e1063–e1143.
doi: 10.1097/CCM.0000000000005337.

MR. SCHRUTE

After nephrostomy tube placement, started to improve slowly.

- UOP starting to pick up
- Pressors weaned off
- Extubated

Transferred to medical floor on hospital day #3.


OXYGEN DELIVERY
Blood Hemoglobin
Oxygen Saturation
ARTERIAL OXYGEN CONTENT
SVR
PERFUSION PRESSURE
ARTERIAL PRESSURE
SVR
CARDIAC OUTPUT
Rate
Stroke Volume
Work of Breathing
Pain, Agitation
MUSCLE ACTIVITY
Seizure, Shivering
VENOUS PRESSURE
DISEASE PROCESS
Toxicity
Infection
Hyperthermia
OXYGEN UPTAKE
AEROBIC
ANAEROBIC
METABOLISM
© Copyright 2023 Mayo Foundation for Medical Education and Research

JUST AS YOU ARE ABOUT TO GET A COFFEE…YOU GET ANOTHER PAGE.

MS. PALMER

66F with PMH of alcoholic cirrhosis, HTN, systemic lupus erythematosus (SLE) & atrial fibrillation.

Complications include hx of ascites, hepatic encephalopathy.

- Has been sober for 2 years, and her cirrhosis has been well compensated.
  - Given normal coagulation studies, and a CHADS2-Vasc score of 3, she was started on Eliquis last year after an episode of afib with RVR.

- **Home meds:** Rifaximin, Lactulose, Spironolactone and Eliquis

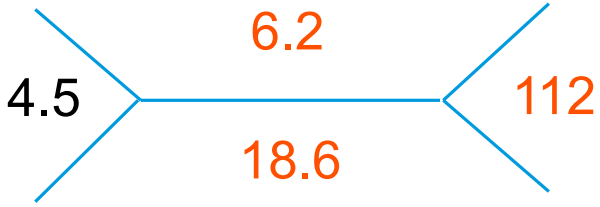
MS. PALMER

Unfortunately, she relapsed a few months ago and started drinking alcohol again. Now presents with **altered mental status** and **melena** x 2 days.

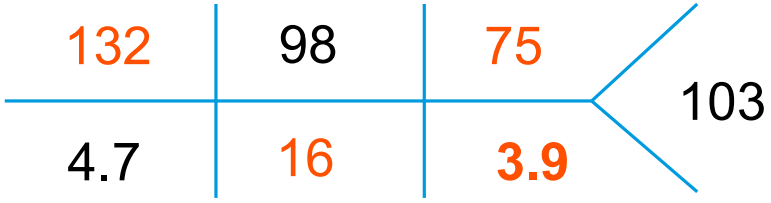
HR: 118 **RR:** 34 **BP:** 80/45 (57) **O2:** 97%. **Temp:** 37.3^{o}C

What is going through your mind? DDX? First interventions?

LABS



AST: 122
ALT: 64
Alk Phos: 233
Tbili: 4.3



INR: 2.4
Fibrinogen: 189

HEMORRHAGE CLASSIFICATION

Variable	Class I	Class II	Class III	Class IV
Systolic BP	Normal	Normal	↓	↓
HR, beats/min	<100	>100	>120	>140
RR, breaths/min	14-20	20-30	30-40	>35
Mental status	Anxious	Agitated	Confused	Lethargic
Base deficit (mEq/L)	0 to -2	-2 to -6	-6 to -10	-10 or less
Blood loss (%)	<15	15-30	30-40	>40
Need for blood products	Monitor	Possible	YES	Massive transfusion protocol

**Off-label use!

DOAC REVERSAL

Andexanet alfa (Andexxa®)

- Recombinant inactivated coagulation factor Xa – decoy for factor Xa-inhibitors
- Onset: 2-5 minutes, peak effect 15-30 minutes (around end of infusion)
- Duration: 2 hours post infusion
- Monitoring: **thrombosis**

4-factor, prothrombin complex concentrate (Kcentra®)**

- Factors II, VII, IX, X, proteins C and S – replenishes clotting factors, no direct effect on DOACs
- Onset: rapid, within 10 minutes
- Duration: 6-8 hours
- Monitoring: INR 15 minutes and 12-hours post infusion, **thrombosis**, hypersensitivity reactions

Idarucizumab (Praxbind®)

- Monoclonal antibody that binds specifically to dabigatran and its metabolites

Activated charcoal

- 50 grams once
- Only if last dose of DOAC within 2 hours

Br J Cardiol. 2022 Jan 12;29(1):1. doi: 10.5837/bjc.2022.001. PMID: 35747314; PMCID: PMC919607
Ann Transl Med. 2019 Sep;7(17):411. doi: 10.21037/atm.2019.07.101. PMID: 31660310; PMCID: PMC6787376

VKA REVERSAL

4-FPCC (Kcentra)

- Onset: rapid, within 10 minutes
- Duration: 6-8 hours
- Monitoring: INR 15 minutes and 12-hours post infusion, **thrombosis**, hypersensitivity reactions

FFP

- Less effective at reversal than 4-FPCC
- Longer infusion time, higher risk of transfusion related fluid overload and lung injury when compared to PCC

Vitamin K (Phytonadione)

- Onset: 1-2 hours (IV) and 6-10 hours (oral), peak effects at 12-14 hours (IV) and 24-48 hours (oral)
- Monitoring: INR, hypersensitivity reactions, thrombosis

Drugs **79**, 1557–1565 (2019). https://doi.org/10.1007/s40265-019-01179-w

MS. PALMER

Focus was first on stabilizing hemorrhagic shock:

- 2 units PRBCs ordered
- A dose of KCentra & Vitamin K given.
- IV fluids started while waiting for blood products.

Hepatology/GI service consulted urgently.

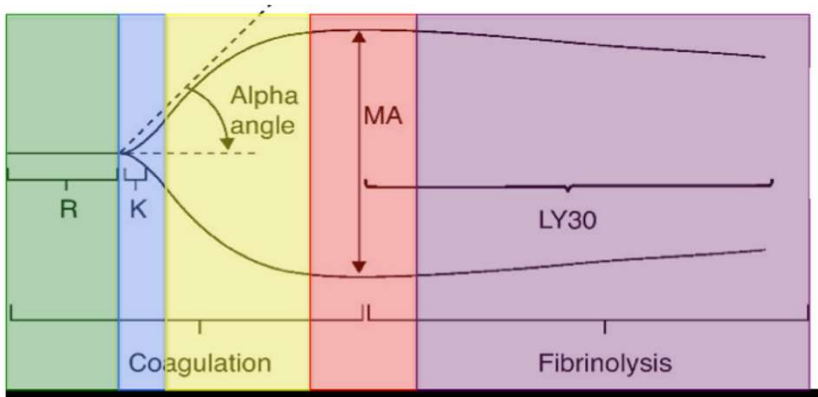
IV access with 2 large bore peripheral IVs.

UPPER GI BLEED TREATMENT

VARICEAL BLEEDING

- NPO, 2 large bore peripheral IV's
- Transfusion:
  - For severe bleeding immediately transfuse blood products **in 1:1:1 ratio of PRBC: FFP : Plts**
  - Hemodynamic instability despite crystalloid resuscitation, transfuse 1-2 units RBCs
  - Transfuse for Hgb <7 (or <8 for high risk patients like CAD), or active bleeding
  - Avoid over-transfusion with possible variceal bleeding
  - Give FFP for coagulopathy or after transfusing 4 units of RBCs; give platelets <50,000 or platelet dysfunction (eg, chronic aspirin therapy) or after transfusing 4 units of RBCs
- Immediate consult for GI/hepatology. IR or Surgery for large scale bleeding
- **Pantoprazole** 80 mg IV if active bleeding (40 mg IV if no active bleeding), followed by 40mg IV BID
- **Octreotide** 50mcg IV bolus followed by 50 mcg/hr continuous infusion
- IV antibiotics (eg. **Ceftriaxone**)
- Balloon tamponade as a temporizing measure (eg. Blakemore tube, Minnesota tube)

THROMBOELASTOGRAPHY (TEG)



rebelem.com

Thromboelastogram (TEG)

Components	Definition	Normal Values	Problem with...	Treatment
R Time	Time to start forming clot	5 – 10 minutes	Coagulation Factors	FFP
K Time	Time until clot reaches a fixed strength	1 – 3 minutes	Fibrinogen	Cryoprecipitate
Alpha angle	Speed of fibrin accumulation	53 – 72 degrees	Fibrinogen	Cryoprecipitate
Maximum Amplitude (MA)	Highest vertical amplitude of the TEG	50 – 70 mm	Platelets	Platelets and/or DDAVP
Lysis at 30 Minutes (LY30)	Percentage of amplitude reduction 30 minutes after maximum amplitude	0 – 8%	Excess Fibrinolysis	Tranexemic Acid and/or Aminocaproic Acid

ALTERED MENTAL STATUS

- **A** Alcohol, ammonia, Alzheimer's
- **E** Endocrine, electrolyte abnormalities
- **I** Infection, intoxication
- **O** Opiates, oxygen, C**O**2
- **U** Uremia
- **T** Tumor, trauma, toxins
- **I** Insulin
- **P** Psych/psychogenic
- **S** Stroke, seizure, syncope, shock

How do we assess if a patient is able to “protect their airway”?

HEPATIC ENCEPHALOPATHY

DIAGNOSIS

West Haven Criteria

Stage	Features
0	No abnormality detected
1	Trivial lack of awareness, euphoria or anxiety, shortened attention span, impaired performance of addition
2	Lethargy or apathy, minimal disorientation for time or place, subtle personality change, inappropriate behavior, impaired performance of subtraction
3	Somnolence to semistupor, but responsive to verbal stimuli Confusion, Gross disorientation
4	Coma (unresponsive to verbal or noxious stimuli)

Hepatol Int **12** (Suppl 1), 135–147 (2018). https://doi.org/10.1007/s12072-017-9812-3

HEPATIC ENCEPHALOPATHY

TREATMENT

- Correction of precipitating cause
  - GI bleed, infection (including SBP), renal failure, hypokalemia, metabolic alkalosis, acute worsening of liver failure, including HCC
- Correct hypokalemia
- Lower blood ammonia:
  - **Lactulose** (titrate to 2-3 loose BM's per day)
  - Rifaximin (neomycin is an alternative)
- **Dialysis!** (If ammonia is >3-4x upper limit of normal)
  - Concern with extremely high ammonia levels is brain edema.

Clinical Gastroenterology and Hepatology, Volume 18, Issue 4, 2020 (800-812.e25)
https://doi.org/10.1016/j.cgh.2019.08.047.

HEPATIC ENCEPHALOPATHY

TREATMENT

- Other treatment options (for refractory cases) :
  - Flumazenil (only short term improvement!)
  - Zinc
  - Polyethylene glycol (PEG) - one study actually found it to be more effective than lactulose
  - Acarbose
  - L-ornithine L-aspartate

JAMA Intern Med. 2014;174(11):1727.
Cochrane Database Syst Rev. 2017;7:CD002798. Epub 2017 Jul 26.
Hepatology. 2022; 75: 1194–1203. https://doi.org/10.1002/hep.32255

MS. PALMER

- Ms. Palmer ended up having to be intubated for airway protection.
- Underwent EGD with banding of her esophageal varices, and stabilized from a hemorrhagic shock standpoint.
- Once no longer bleeding, we had enteral access placed and started treatment for her hepatic encephalopathy.

- Unfortunately , she continued to be extremely hypotensive requiring 2 pressors (norepinephrine and vasopressin).

WHY DO YOU THINK THAT IS?!

VASODILATORY SHOCK

Differential for vasodilatory shock:

?

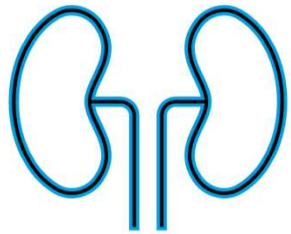
- **S**epsis
- **A**drenal insufficiency
- **L**iver Disease
- **A**naphylaxis, Autonomic Dysfunction, Acidosis
- **D**rugs (and devices)

MS. PALMER

- Ms. Palmer's renal function continues to worsen and she stops making urine.
  - Initiated on CRRT.

HEPATORENAL SYNDROME

- Caused by arterial vasodilatation in the splanchnic circulation, which is triggered by portal hypertension
  - ↑ activity of vasodilators (NO being one of the most important) → ↓ in SVR → ↓ kidney perfusion
- Diagnosis of exclusion! Need to r/o other AKI/CKD DDX.



World J Nephrol. 2015 May 6;4(2):277-86. doi: 10.5527/wjn.v4.i2.277.

HEPATORENAL SYNDROME
TREATMENT

- Stop antihypertensives, including beta blockers
- **If not in the ICU:**
  - Terlipressin + 25% albumin

    - OR –

  - Midodrine + octreotide + albumin
- **In the ICU:**
  - Norepinephrine (+/- vasopressin) + albumin

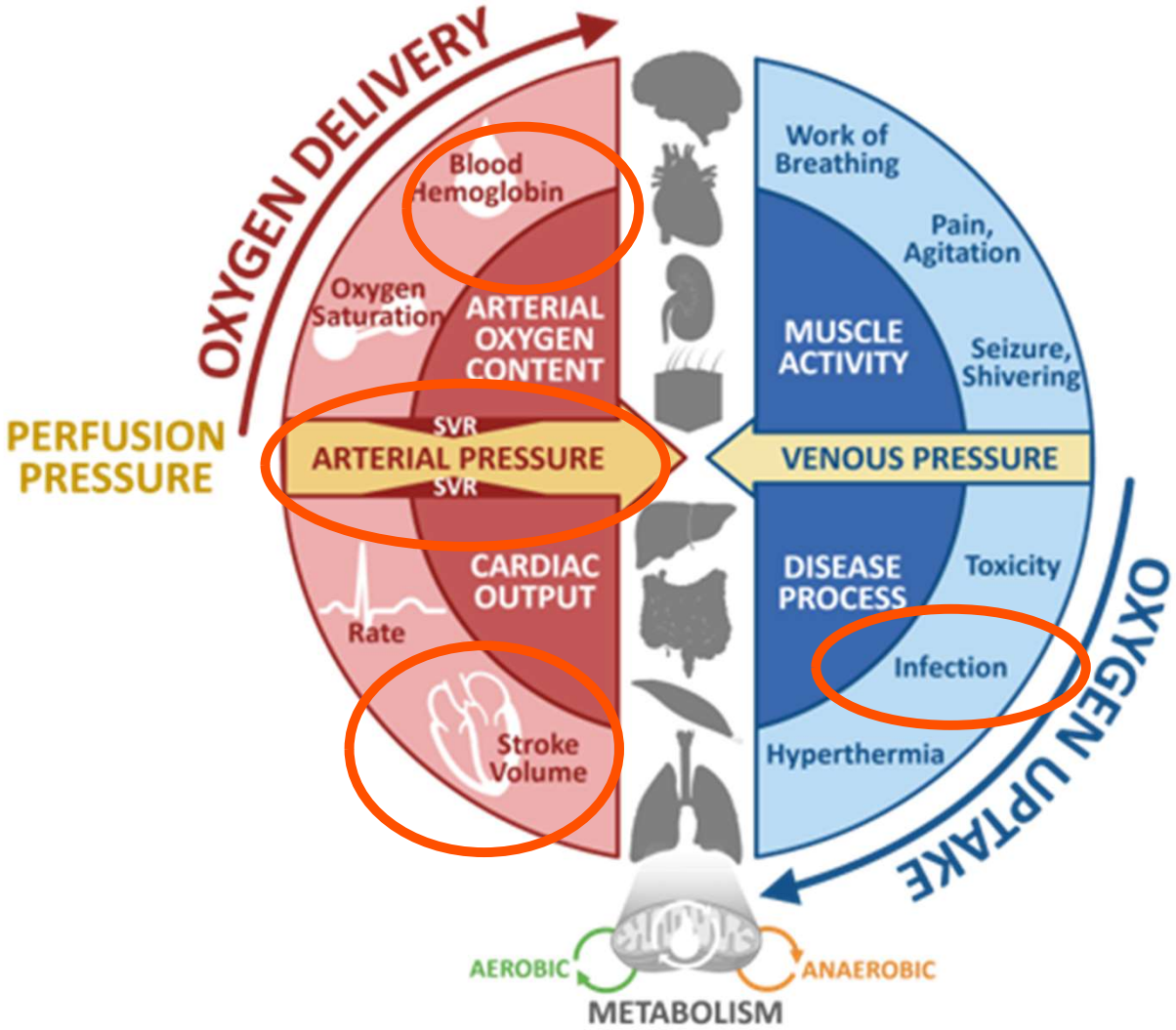
- TIPS (transjugular intrahepatic portosystemic shunt) can also be considered if patient is well enough to receive it

World J Nephrol. 2015 May 6;4(2):277-86. doi: 10.5527/wjn.v4.i2.277.
UpToDate: Hepatorenal Syndrome

MS. PALMER

- ☑ Variceal GI bleeding, treated with banding. **Hemorrhagic** shock resolved.
- ☑ Dialysis started (CRRT), which helped renal failure and hepatic encephalopathy (as well as lactulose, rifaximin, etc.)
- ☑ Steroids started for possible **adrenal** insufficiency
- ☑ Paracentesis performed, SBP + , treated with Ceftriaxone. (which improved any underlying **septic** component)
- ☑ Supportive care to otherwise optimized her end stage liver disease.

Ms. Palmer stabilized enough to be extubated and come off pressors.
Unfortunately, she was unable to come off dialysis (though she did transition to iHD).
Discharged home with plans to complete intensive outpatient program for alcohol use disorder, with hopes to be listed for kidney/liver transplant if she can maintain her sobriety.


OXYGEN DELIVERY
PERFUSION PRESSURE
Blood Hemoglobin
Oxygen Saturation
ARTERIAL OXYGEN CONTENT
SVR
ARTERIAL PRESSURE
SVR
CARDIAC OUTPUT
Rate
Stroke Volume
Work of Breathing
Pain, Agitation
MUSCLE ACTIVITY
Seizure, Shivering
VENOUS PRESSURE
DISEASE PROCESS
Toxicity
Infection
Hyperthermia
OXYGEN UPTAKE
AEROBIC
ANAEROBIC
METABOLISM
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MR. SCOTT

- 57yo male admitted for a burn injury – he burned his foot while grilling bacon on his George Foreman grill.
- The burn quickly improved but unfortunately, his course was complicated by an **aspiration** event that required a brief stay in the ICU where he received **mechanical ventilation** x 3 days for an aspiration PNA.
- He is now on the hospital medicine service, where he continues to improve on **piperacillin/tazobactam.**

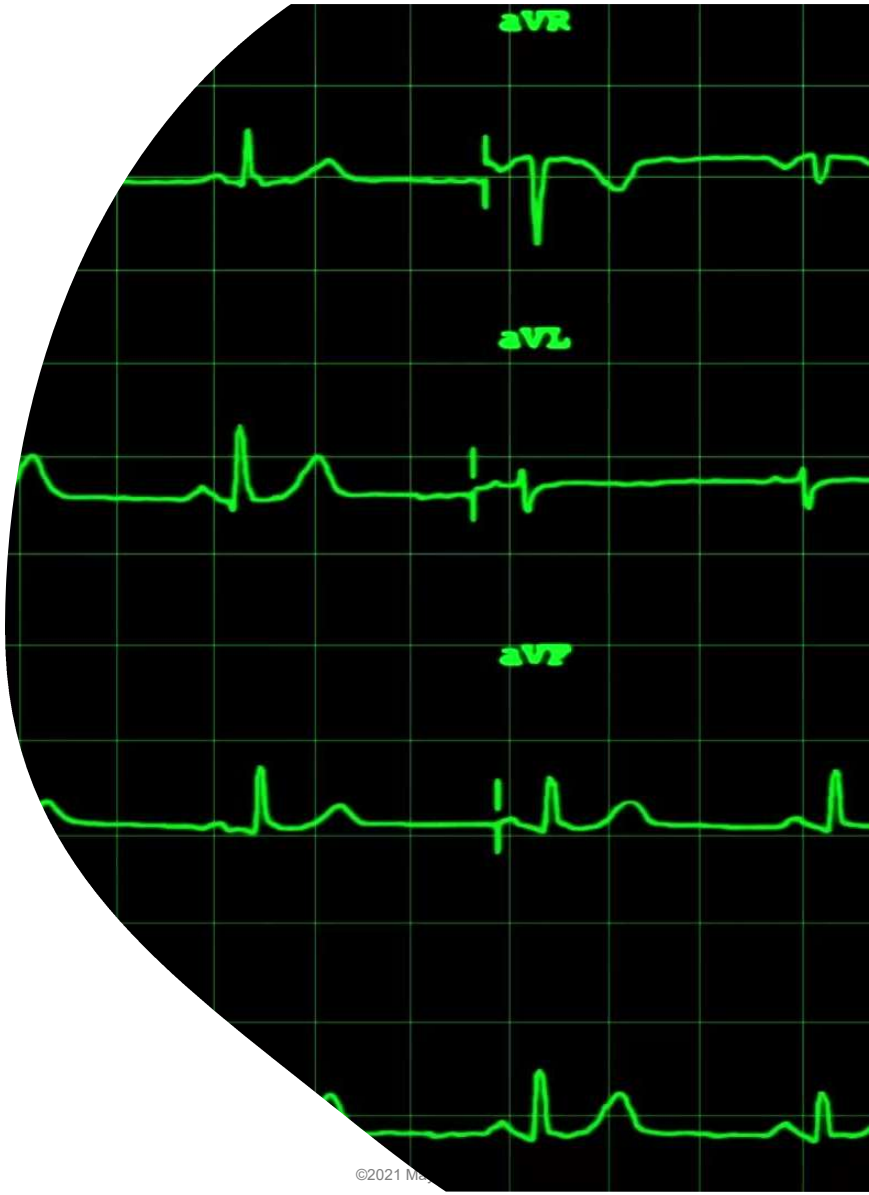
MR. SCOTT

- Your page is from the patient's nurse, stating he had a fever of 38.9^{o}C four hours ago.
- She tried to call the attending and didn't get through, so nothing has been done for the fever.

<u>His other vitals:</u>

HR 110 **BP** 83/52 **RR** 30 **SpO2** 99% on 2L NC

WHAT'S YOUR NEXT STEP?



INITIAL RESUSCITATION

30 mL/kg crystalloids within the first 3 hours if evidence of hypoperfusion

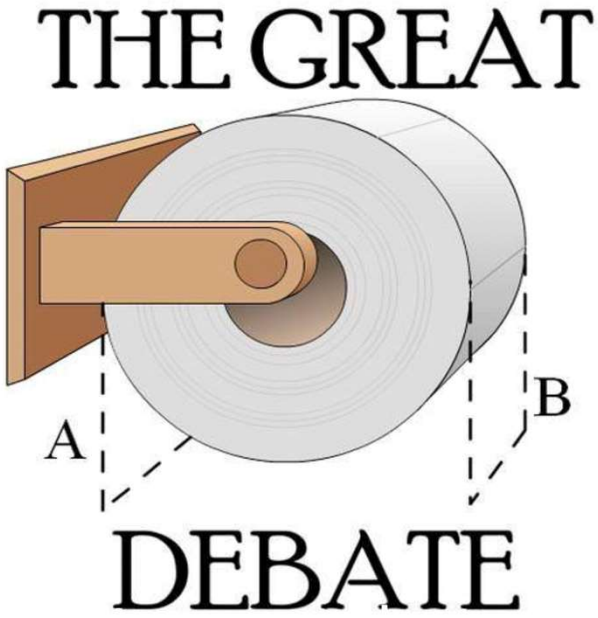
Dynamic reassessment of volume status

Target MAP ≥ 65 mm Hg

Goal is to clear lactate

Crystalloids first!

WHICH IV FLUID SHOULD I USE?



WHICH IV FLUID SHOULD I USE?

SMART Trial, 2018

Balanced crystalloids (LR, Plasma-Lyte) >>> NS

- Lower 30-day mortality
- Less need for renal replacement therapy
- Lower rate persistent renal dysfunction

N Engl J Med 2018;378:829 & NEJM 2014;370(15):1412 7 *AMA*. 2013;310(17):1809-1817.

BUT WAIT!

BaSICS trial, 2021

Plasma-lyte vs. NS

- No difference in 90-day survival
- No difference in incidence of AKI, need for RRT, hospital or ICU death, length of stay

JAMA. 2021;326(9):818–829.

THE VERDICT

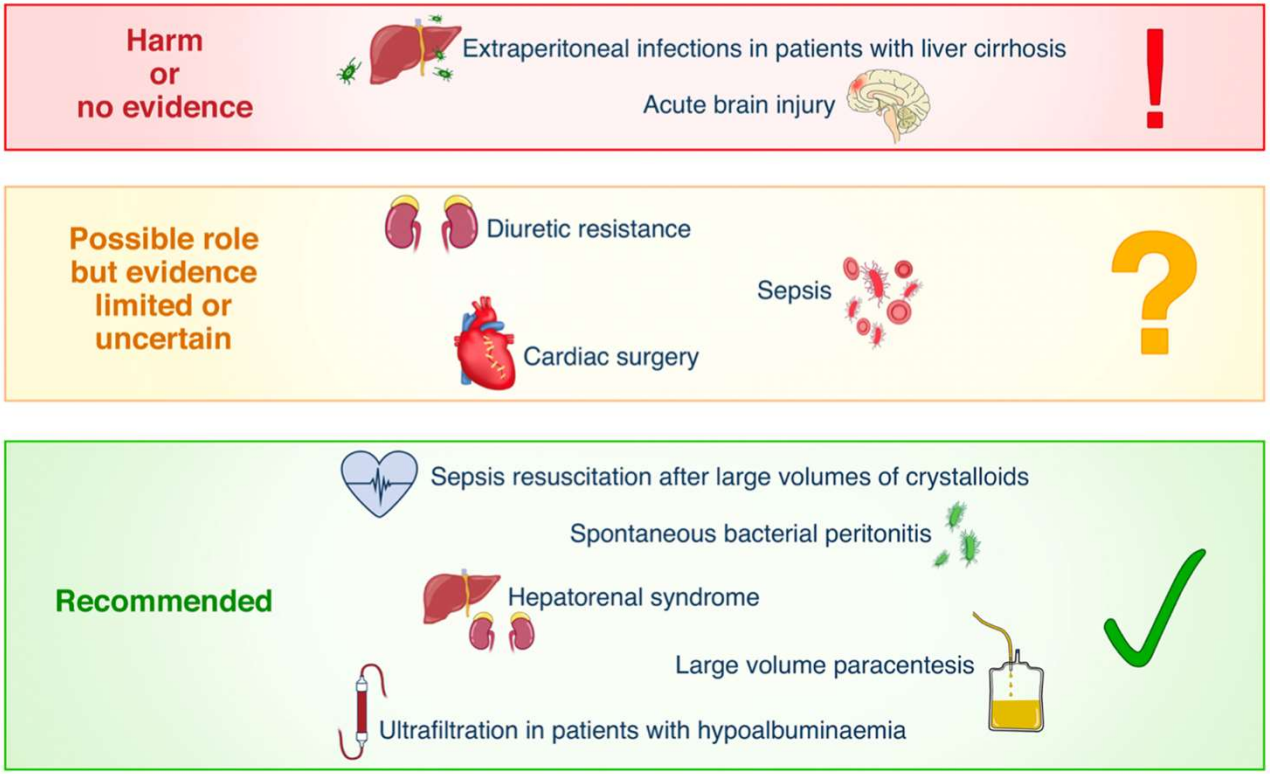
- NS may not be as bad we thought!

- But in large volume resuscitation, consider using a balanced crystalloid

	Na	Cl	K	Mg	Ca	HCO3	Glucose	Acetate	Osm	pH
Plasma	140	104	4.5	1.25	2.5	24	0.08		290	7.4
0.9% NaCl	154	154							308	5.5
0.45% NaCl	77	77							406	
LR	130	109	4		1.5	28 (as lactate)			273	6.5
Plasma-Lyte	140	98	5	1.5				27	294	7.4
D5W							5		278	
Albumin	130-160	100-130	<2						309	

1 L of NS = 9 g Na
WHO recommendation = 2g/day
Average Na intake in the US = 3.3 g/day

WHAT ABOUT ALBUMIN?

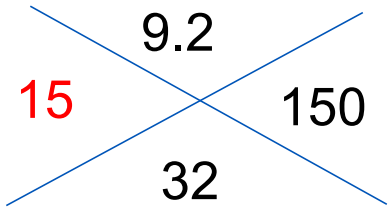


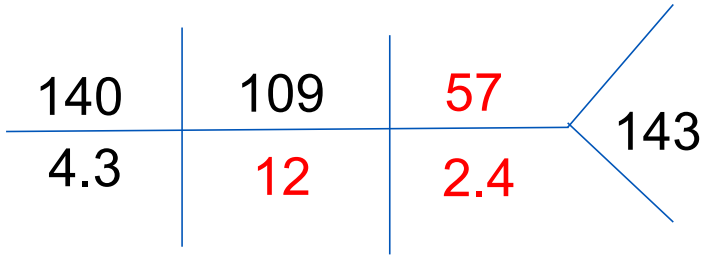
Intensive Care Med (2022) 48:602–605

WHICH IV FLUID SHOULD I USE?

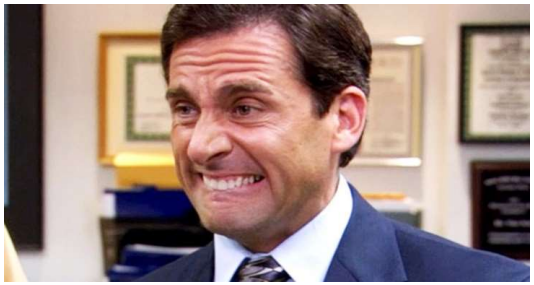
Type of Fluid	Hospital Cost	Patient Cost
NS	$ 1.25	$78.40
LR	$ 2.00	$82.00
Plasma-Lyte	$ 2.00	$82.00
500 mL 5% albumin	$33.25	$266.00
100ml 25% albumin	$66.30	$234.00

MR. SCOTT





Lactate: 8.7 **Procalcitonin: 5**



BICARB?

BICAR-ICU trial, 2018

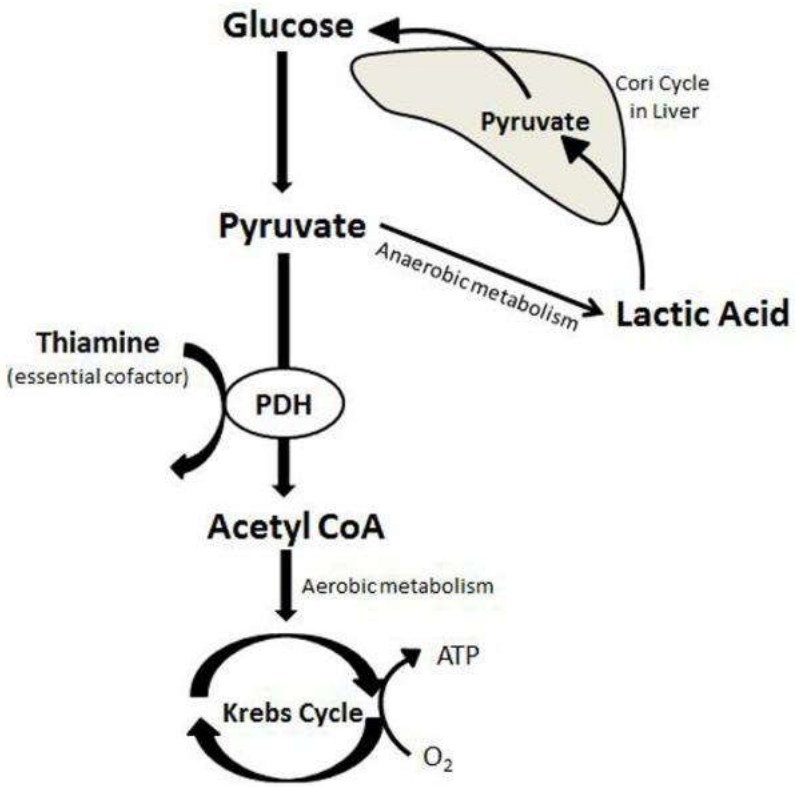
When do we give bicarb in metabolic acidosis?

- 28 day mortality was no different (bicarb vs. placebo).
- No difference in LOS, ICU days
- ?less RRT

JAMA. 2021;326(9):818–829.

LACTATE

- Produced by most tissues (mostly muscle)
- Cleared mostly by the liver.



CAUSE OF ELEVATED LACTATE

Shock

Post-cardiac arrest

Regional tissue ischemia

- Mesenteric ischemia
- Limb ischemia
- Burns
- Trauma
- Compartment syndrome
- Necrotizing soft tissue infections

DKA

Drugs/toxins

- Alcohols
- Cocaine
- CO
- Cyanide

Thiamine deficiency

Medications

- Linezolid
- NRTIs
- Metformin
- Epinephrine
- Propofol
- Acetaminophen
- Beta2 agonists
- Theophylline

Anaerobic muscle activity

- Seizure
- Heavy exercise
- Increased WOB/asthma exacerbation

Malignancy

Liver insufficiency

Mitochondrial disease

Type A vs. Type B?!

Type A: due to hypoperfusion and hypoxia, which occurs when an oxygen consumption/delivery mismatch occurs →anaerobic glycolysis

Type B: not having to do with tissue Hypoperfusion or hypoxia (problem with metabolism of lactate)

LACTATE CLINICAL PEARLS

- Lactate ≥4mmol/L is associated with an increased mortality within 72 hours
- Lactate clearance has a greater prognostic value than the initial lactate level
- Can obtain by either arterial or venous samples

WHICH IV FLUID SHOULD I USE?

Does LR affect lactate levels?

No!

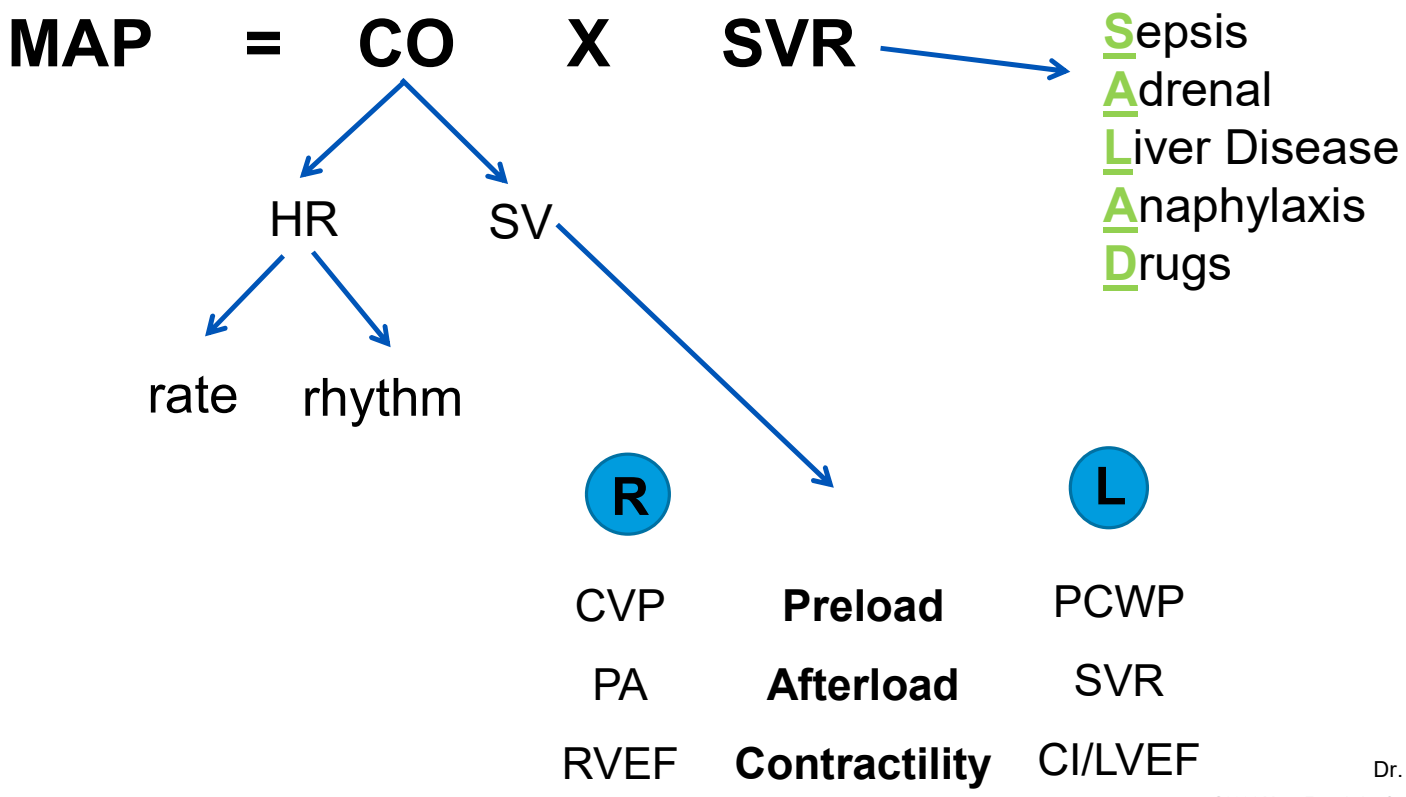
- LR contains *sodium lactate* (not lactic acid), and is therefore not acidotic
- Lactate is very rapidly metabolized to bicarbonate
- Also you can use it even in hyperkalemia (it doesn't have a ton of K and NS can actually worsen HyperK if hyperchloremic acidosis created)

MR. SCOTT

- He continues to have a fever of 38.7C even after a dose of Tylenol.

- After you give him a 500cc fluid bolus…his BP is still low at 87/49.

HYPOTENSION



Dr. Bhavesh Patel & Dr. Ayan Sen

SVO2/SCVO2

SVO2 = venous O2 saturation

- The amount of O2 “left over” after the tissues have used up everything they need
- Normal = 65-70%

A true SVO2 is drawn from a PA catheter, but you can get an ScVO2 from any central line (including PICC)

SVO2

HIGH SVO2

- ↑ **O2 delivery** (increased FiO2)
- ↓ **O2 demand** (hypothermia, anesthesia)
- **High flow states** (sepsis, hyperthyroid, etc.)

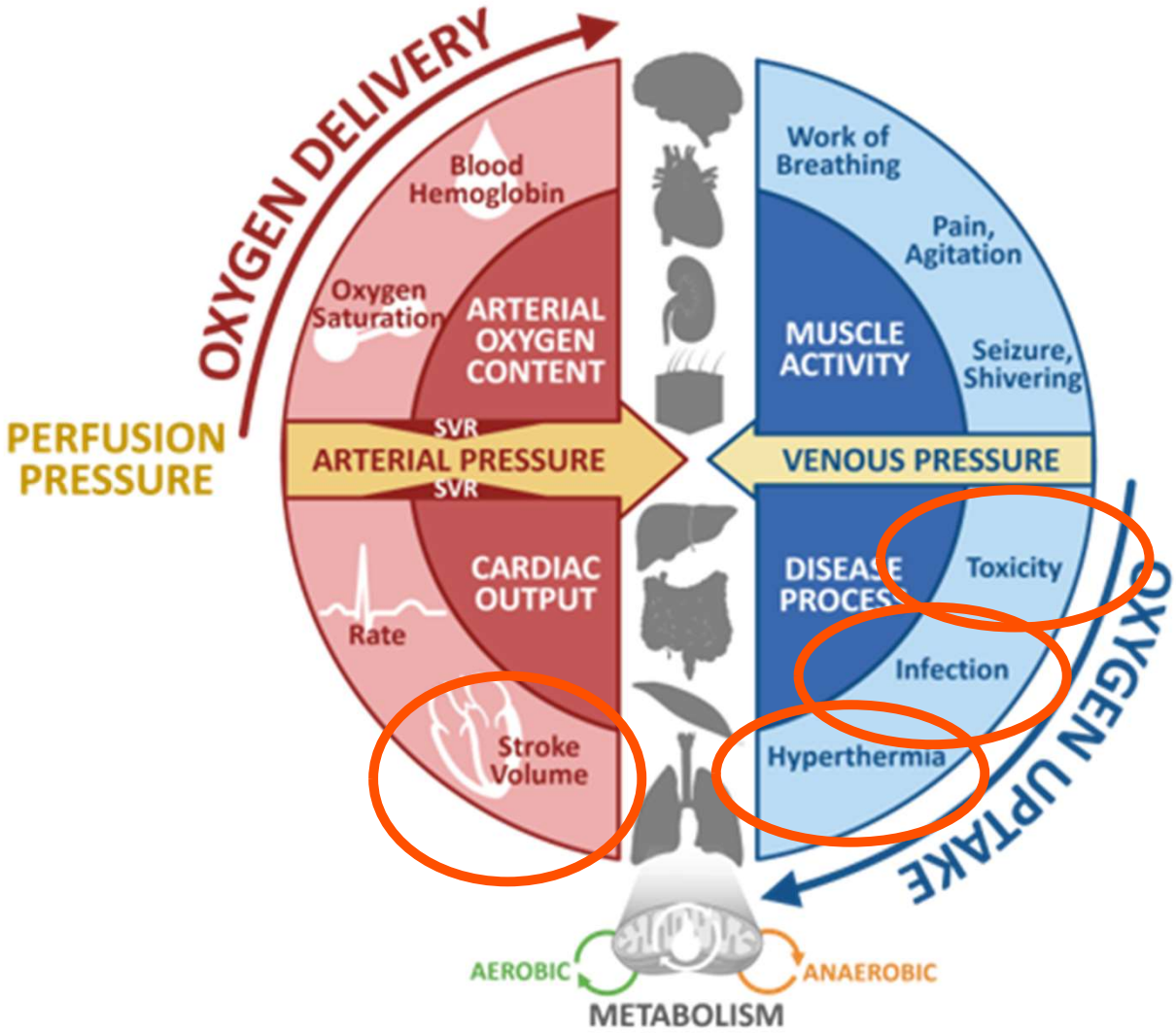
LOW SVO2

- ↓ **O2 delivery**
  - ↓ Hgb
  - ↓ SaO2 (hypoxemia)
  - ↓ forward flow (heart failure)
- ↑ **O2 demand**
  (hyperthermia, shivering, pain, seizures)

MR. SCOTT

Blood cultures started growing GPC within three hours

- ScVO2 came back at 39%
- Stat echo showed an EF of 25% (from a previous normal)



HYPOTENSION CLINICAL PEARLS

Monitor clinical response by:

- UOP
- Peripheral perfusion assessment
- Mental status
- Lactate/acidosis

Not all patients with hypotension have shock!!

TIME TO GO HOME!

QUESTIONS?

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