

IN THE NAME OF GOD

SHOCK MANAGEMENT

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- The ability to **evaluate and manage a critically ill patient** is one of the most important skills any intensivist brings to the bedside. Patients already resident in the intensive care unit (ICU) can **decompensate** because of any of several causes; thus, the differential diagnosis is broader than it is in less complex patients.
 - Appropriate management is dependent on **correct identification** of the **cause or causes** of decompensation.

Definition

- Failure of delivery oxygen and substrates to meet the metabolic demands of the tissue beds
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SUPPLY < DEMAND

Oxygen **delivery** < Oxygen **Consumption**

$$DO_2 < VO_2$$

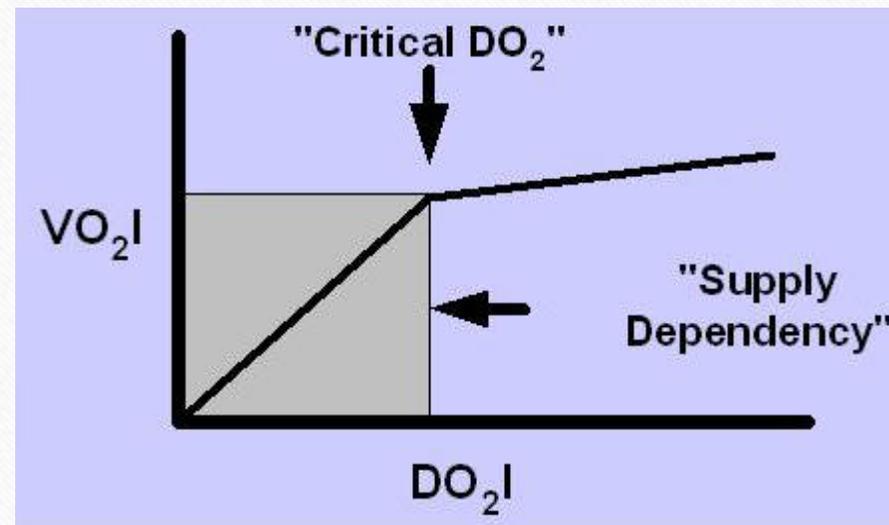
- Failure to remove metabolic end-products
- Result of inadequate blood flow and/or oxygen delivery

Oxygen delivery (DO_2)

- $DO_2 = CO \times CaO_2$
 - DO_2 : oxygen delivery
 - CO : Cardiac output
 - CaO_2 : arterial oxygen content
- $CO = HR \times SV$
 - HR: heart rate
 - SV: stroke volume
- $CaO_2 = HgB \times SaO_2 \times 1.34 + (0.003 \times PaO_2)$
 - Oxygen content = oxygen carried by HgB + dissolved oxygen

Oxygen delivery (DO_2)

$$DO_2 = CO \times CaO_2$$



Critical DO_2 : consumption depends on delivery

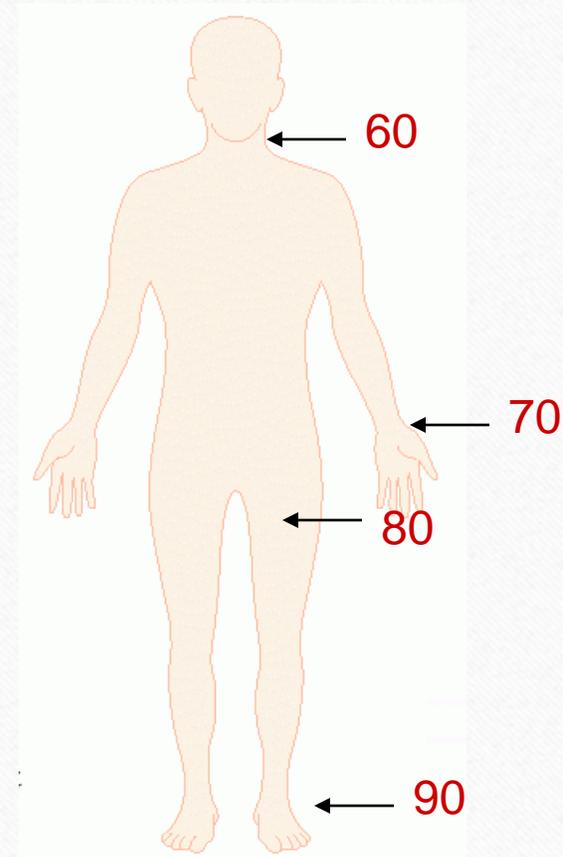
3.8 ± 1.5 mL/min/kg (266 mL/min in a 70 kg patient)

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- Shock is **traditionally** defined as “*circulatory failure that results in **inadequate cellular oxygen utilization***”
 - This definition is problematic as **oxygen delivery** has to fall to very low levels before oxygen consumption falls and most patients with “shock” have **normal levels of oxygen consumption**.
 - Ronco and colleagues determined the critical oxygen delivery threshold for anaerobic metabolism in critically ill humans while life support was being discontinued .
 - The **critical oxygen delivery threshold** was $3.8 \pm 1.5 \text{ mL/min/kg}$ (266 mL/min in a 70 kg **patient**); assuming a hemoglobin concentration of 10 g/L this translates into a cardiac output of approximately 2 L/min; it is likely that only pre-terminal moribund patients with “shock” would have such a low cardiac output

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- Furthermore, while an elevated **lactate concentration** is widely believed to be a **marker of anaerobic** metabolism, an overwhelming body of evidence suggests that in most clinical situations, that lactate is produced aerobically as part of the stress response .
 - While it is unclear how best to **define** shock, we believe “**circulatory shock**” is best defined as “***a potentially life threatening reduction in systemic organ blood flow.***”
 - The clinical diagnosis of shock is then based on a constellation of clinical and hemodynamic features which include hypotension, tachycardia, increased respiratory rate and decreased urine output. Typically, the **SBP is less than 90 mmHg or the MAP is less than 65 mmHg**. While altered mentation, notably obtundation, disorientation and confusion is common, patients may be remarkably lucid despite profound hemodynamic compromise (due to blood flow redistribution to the brain).

Shock

- Do you remember how to quickly estimate blood pressure by pulse?
- If you palpate a pulse, you know SBP is at least this number



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- The classes of shock are **cardiogenic**, **hypovolemic** (e.g., hemorrhagic), or **obstructive** (e.g., tension pneumothorax, cardiac tamponade, pulmonary embolism) **vasodilatory/ distributive** (e.g., septic, adrenal insufficiency, anaphylaxis), .
 - The first three mechanisms are characterized by **low cardiac output** while distributive shock is characterized by **decreased systemic vascular resistance**.

Shock Syndromes

🦠 Hypovolemic Shock

- blood **VOLUME** problem

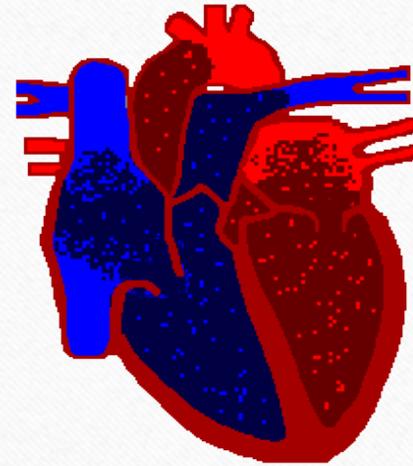
🦠 Cardiogenic Shock

- blood **PUMP** problem

🦠 Distributive Shock

[septic;anaphylactic;neurogenic]

- blood **VESSEL** problem

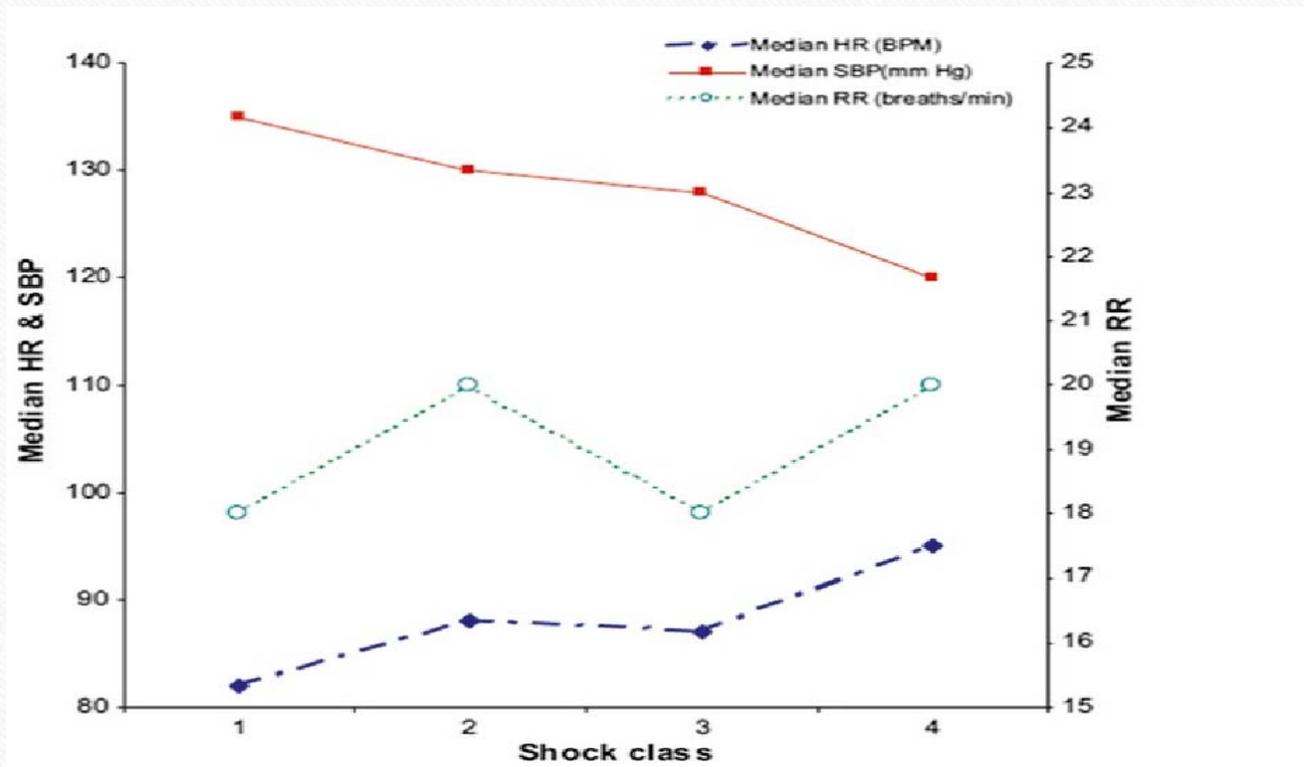


The Vital Signs: BP, HR, RR, TEMP, SaO₂ ... and SV

- With circulatory compromise the **SV falls much earlier than** any change in the other vital signs.
- This implies that a patient with an abnormality of one or more of the traditional vital signs is “in trouble”; however, **normal vital signs do not indicate that “all is well.”**

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- This is best illustrated by the study of Guly et al. using data from the **UK Trauma Audit and Research Network database** .
 - The estimated blood loss of **164,785 patients** was recorded and classified using the ATLS classes of shock The ATLS class was then correlated with the presenting SBP, HR, and RR.
 - A can be seen from Fig. patients **in Class 4 shock** (who had lost more than **40 %** of their blood volume) had vital signs within the normal range..... **REMARKABLE.**

Vital signs in each ATLS class of hemorrhagic shock.
Reproduced with permission from Elsevier
(HR ,SBP,RR)



Individual vital sign thresholds indicating potential trouble

Vital Sign	Threshold
SBP mmHg	<100
MAP mmHg	<75
HR/min	>110 or <40
RR/min	>20 or <12
SaO ₂ on room air	<90
SVI mL/m ²	<30

Treatment of Shock

- Recognize Type of Shock
 - If definite pump failure and cardiogenic shock institute cardiac protocols
- Otherwise: 2 large bore, upper extremity lines and:
 - Volume
 - Volume
 - Volume

When in doubt, try a little more volume

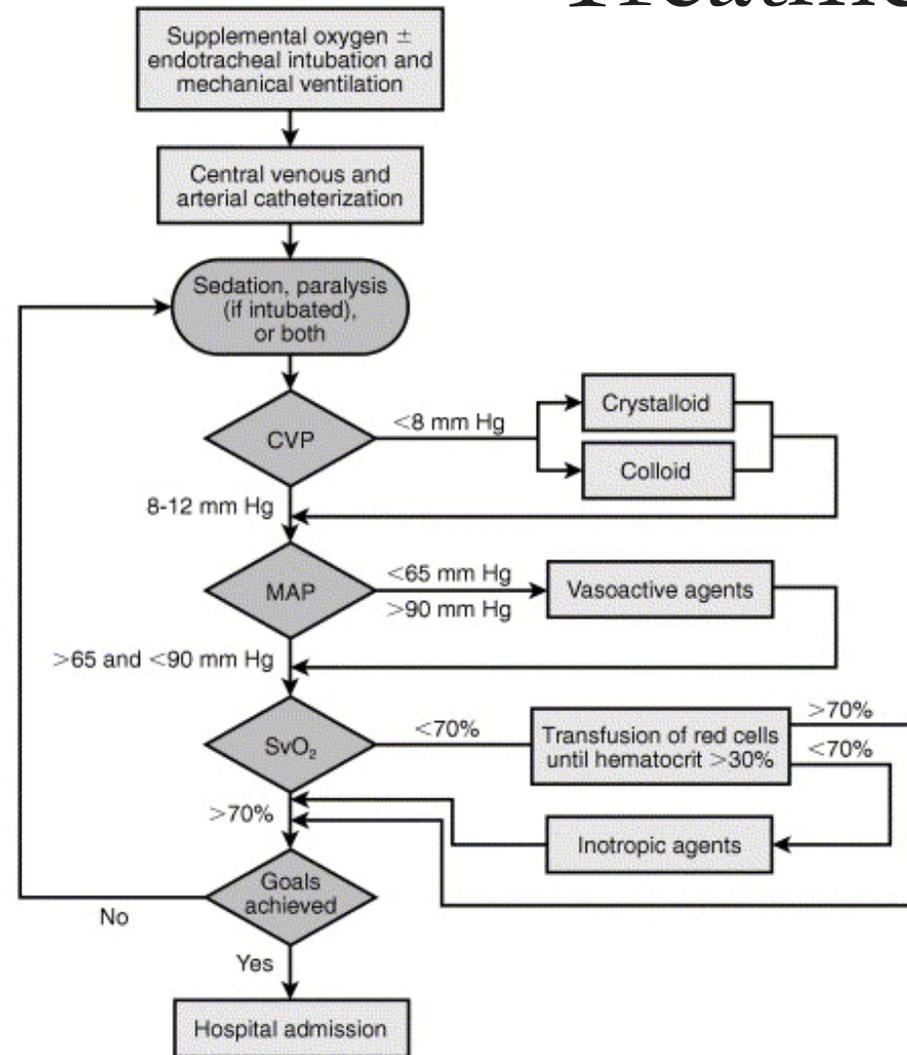


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- Current “wisdom” suggests that **aggressive fluid resuscitation** is the best initial approach to the patient with hemodynamic instability.
 - The source of this wisdom is difficult to discern, however, “Early Goal Directed therapy” (EGDT) as championed by Rivers et al. appears to have established this as the irrefutable truth.

Early Goal Directed Therapy

- Septic Shock Study 2001
 - 263 patients with septic shock by refractory hypotension or lactate criteria
 - Randomly assigned to EGDT or to standard resuscitation arms (130 vs 133)
 - Control arm treated at clinician's discretion and admitted to ICU ASAP
 - EGDT group followed protocol for 6 hours then admitted to ICU

Treatment Algorithm



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- Mixed venous or central venous oxygen saturation or content can be reliably used to make inferences about the balance of **oxygen delivery to the peripheral tissues and oxygen uptake by them.**
 - **Mixed venous oxygen saturation (SvO₂)** is measured from the **pulmonary arterial port of a pulmonary artery catheter.** For patients without pulmonary artery access, **central venous oxygen saturation (ScvO₂)** is often used as an alternative. ScvO₂ is typically measured from a **central line placed within the superior vena cava.**
 - Multiple studies have revealed **variable correlation** between SvO₂ and ScvO₂. However, Chawla et al. found that ScvO₂ drawn from a port within the right atrium had good correlation with SvO₂, although there was a bias toward **ScvO₂ being 5.2% higher** than the SvO₂. This difference can be explained by the **addition of blood from the coronary sinus, which drains the high oxygen extraction system of the heart,** into the measurement of SvO₂ but not the ScvO₂. Multiple studies focused in goal-directed resuscitation have used ScvO₂ to determine if fluid administration is sufficient.
 - A ScvO₂ greater than 70% is common in resuscitation algorithms and may improve outcomes.

Excess fluid leads to iatrogenic salt water drowning

- However, over the last decade it has become clear that aggressive fluid resuscitation is associated with **increased morbidity and mortality** across a diverse group of patients, including patients with **severe sepsis, ARDS**, as well as surgical and **trauma** patients and those with **pancreatitis**.
- Multiple RCT's and cohort studies have demonstrated that a **conservative fluid strategy** in patients undergoing **elective** non-cardiac surgery is associated with significantly **fewer complications with a lower mortality** (in the high risk patients) than patients managed with the traditional liberal fluid strategy in which fluids are administered to fill the non-existent “third space”

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- For patients with **traumatic** injuries, **high volume fluid resuscitation** as promoted by the early Advanced Trauma Life Support (ATLS) strategy, has given way to a “**damage control**” **resuscitation strategy**.
 - This approach has seen a **fall in the volume** of crystalloid delivered in the emergency department and an associated **fall in mortality**. In a prospective analysis of **3,137** trauma patients’ treated in the Emergency Department, fluid volumes of **1.5 L or more were significantly associated with mortality** that a conservative fluid strategy was associated with a lower mortality in trauma patients

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- The argument is no longer “wet or dry” but “*just the right amount of fluid*”.
 - This is not an easy determination and requires an evaluation of the patient’s hemodynamics, organ perfusion, cardiac function and an assessment of “fluid responsiveness” in addition to understanding the patients’ clinical condition.
It is no longer acceptable to pour in liters of fluid and “see what happens”.
 - In addition to understanding that we need to precisely titrate fluid volumes we have also gained much better insight into which fluid to give.
It is also important to note that at 3 h only 15 % of a crystalloid bolus remains intravascular in normal health volunteers, while in experimental sepsis models almost 100 % of the fluid bolus filters into the interstitium

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- It should be noted that hypotension and tachycardia reflect significant volume depletion. A blood loss of about **1 L** is required before a patient develops a **tachycardia** and about **1.5 L** before the blood pressure begins to drop. (**hypotension**)
 - In those patients with hypotension or evidence inadequate tissue perfusion, **fluid resuscitation is generally regarded as the first step in resuscitation.**
 - Clinical studies have, however, demonstrated that only about **50 %** of hemodynamically unstable critically ill patients (In ER, ICU or OR) are **volume responsive**

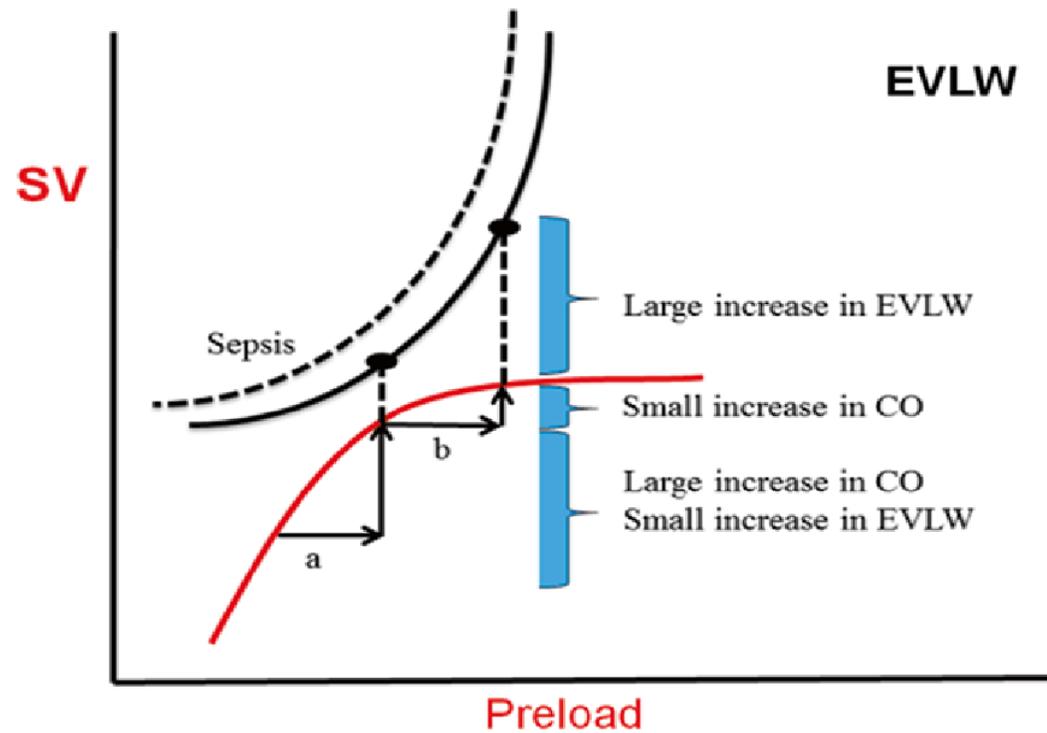
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- The **goal of evaluating circulation** is ultimately to determine whether the patient has **adequate end-organ perfusion**.
 - **Clinically, mental status** and **urine output** are widely accepted as the most reliable indicators of adequate end organ perfusion, but they may not be readily assessed in a substantial percentage of critically ill patients.
 - **Signs of hypoperfusion** may include **tachycardia** with narrow pulse pressure, **tachypnea**, **low mean blood pressure** (Mean arterial pressure (MAP) < 65 mmHg), and **low urine output**. **Encephalopathy**, a marker for cerebral perfusion, is also a predictor for higher mortality when associated with hemodynamic instability.

Poor capillary refill ,Skin perfusion/mottling ,Cold extremities (and cold knees)

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- Fluid responsiveness is generally defined as a **significant increase (> 10–15 %)** **in stroke volume** in response to a fluid challenge (usually 500 cm³). Fluid responsiveness occurs only in patients with biventricular preload responsiveness.

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- Fundamentally, the **only reason to give a patient a fluid challenge is to increase stroke volume (volume responsiveness)**.
 - If the fluid challenge *does not* increase stroke volume, volume loading serves the patient no useful benefit (and is likely harmful). **According to the Frank-Starling principle as the preload increases left ventricular (LV) stroke volume increases until the optimal preload is achieved at which point the stroke volume remains relatively constant**

Superimposition of the Frank-Starling and Marik-Phillips curves demonstrating the effects of increasing preload on stroke volume and lung water in a patient who is pre-load responsive (a) and non-responsive (b). With sepsis the EVLW curve is shifted to the left. *EVLW* extravascular lung water, *CO* cardiac output, *SV* stroke volume

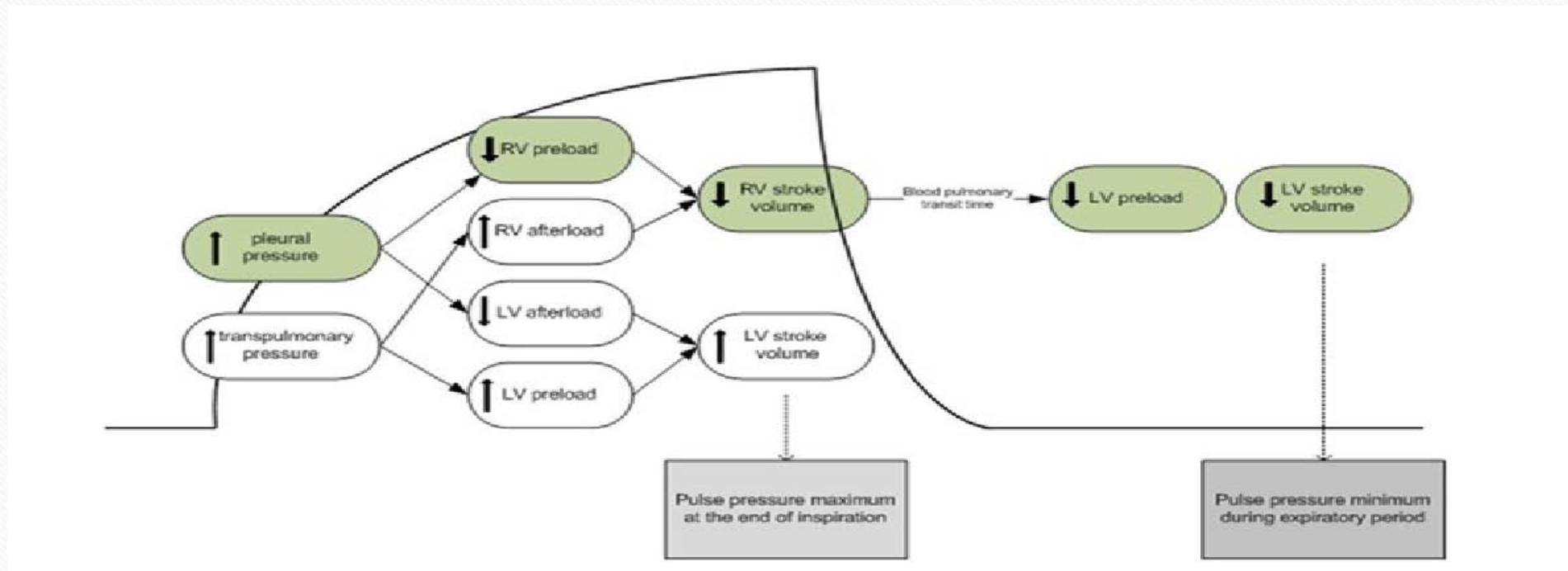


How to predict fluid responsiveness?

- After Hughes and Magovern described the technique of central venous pressure (CVP) monitoring in 1959 this method became a standard tool for guiding fluid therapy
- It has now been clearly established that there is a **poor relationship between the CVP and intravascular volume status** and **no relationship between the CVP and fluid responsiveness**
- 2–5 CVP Rule
According to this scheme, the CVP is measured at 10 min intervals.
If the change in CVP was < 2 mmHg, the infusion was continued,
if it was in the 2–5 mmHg the infusion was interrupted and reevaluated after a 10 min wait.
If the change was > 5 mmHg the infusion was stopped

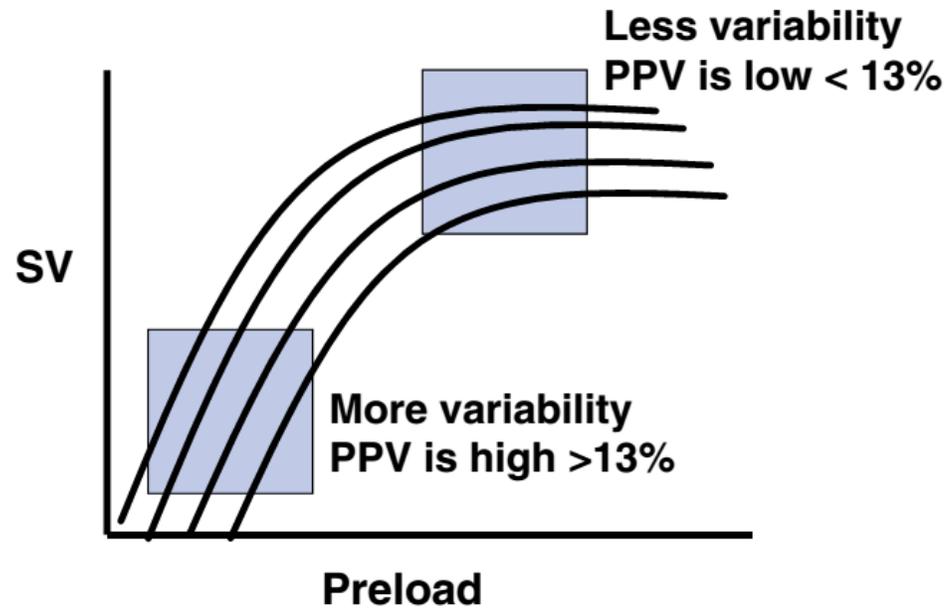
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- In 1970 the flow-directed **pulmonary artery catheter (PAC)** was developed by **Swan and Ganz** allowing measurement of the pulmonary artery occlusion pressure (**PAOP**). However, the PAOP suffers from the same limitation as the CVP and multiple studies have demonstrated that, like the CVP, the PAOP is unable to predict fluid responsiveness.

Heart lung interaction during mechanical ventilation



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- Intermittent positive-pressure ventilation induces **cyclic changes** in the loading conditions of the left and right ventricles. Mechanical insufflation **decreases preload** and **increases afterload** of the right ventricle (RV). The reduction in RV preload and increase in RV afterload both lead to a **decrease in RV stroke volume**, which is at a minimum at the end of the inspiratory period. The inspiratory reduction in RV ejection leads to a **decrease in left ventricular (LV) filling** after a phase lag of two or three heart beats. The cyclic changes in RV and LV stroke volume are greater when the ventricles operate on the steep rather than the flat portion of the Frank-Starling curve

PPV and position on Frank-Starling Curve



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- A pulse pressure variation (PPV) or stroke volume variation (SVV) of greater than 13 % were shown to be predictive of fluid responsiveness

It soon became apparent that a large number of clinical factors interacted to limit the accuracy of the PPV/SVV in predicting fluid responsiveness

Sinus Rhythm

Volume cycled ventilation with V_t of at least 8 ml/kg IBW

No ventilator-patient dyssynchrony

Heart rate/respiratory rate ratio >3.6

Normal chest wall compliance (Δ intra-pleural pressure)

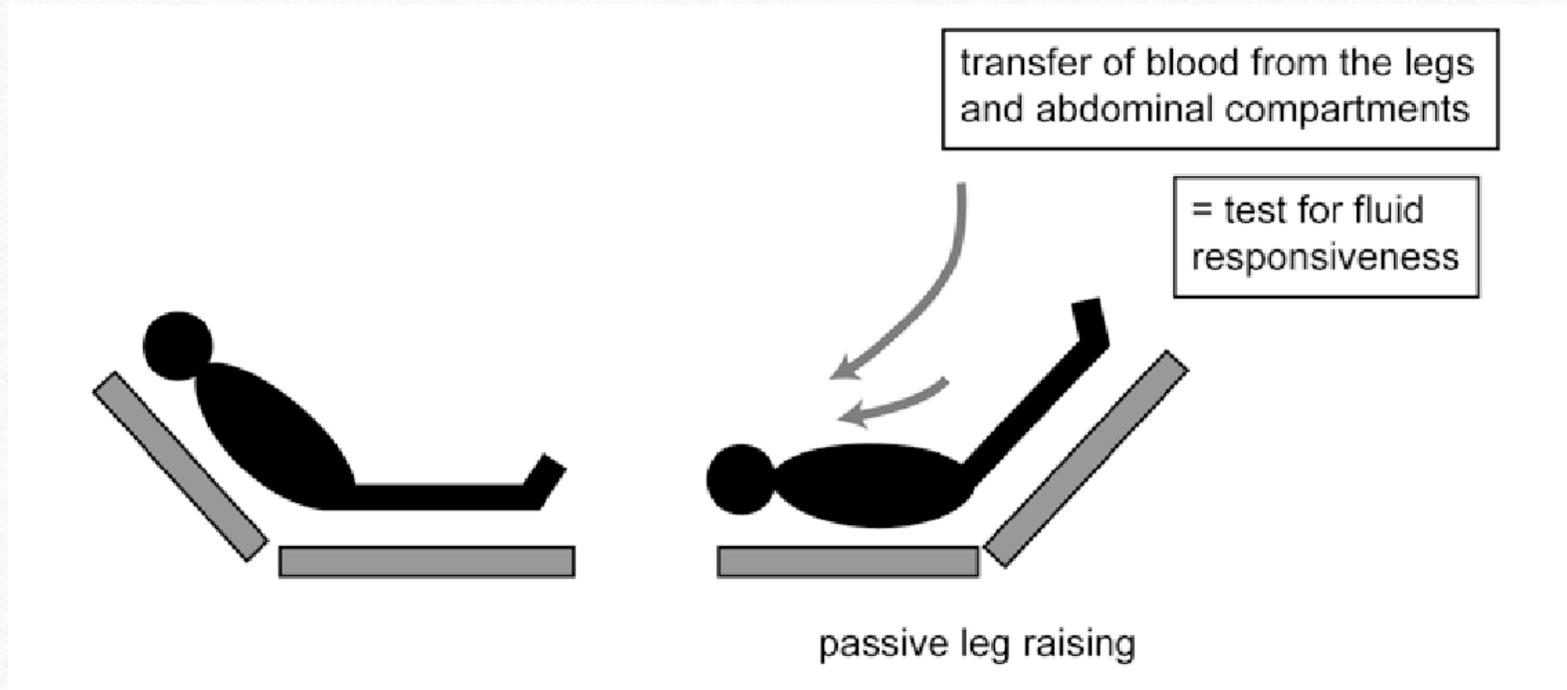
No evidence of cor pulmonale- pulmonary hypertension

Normal intra-abdominal pressure

Normal pulmonary compliance

Echocardiographic Assessment of Fluid Responsiveness

Passive leg raising (PLR) maneuver



The Fluid Challenge

- The **gold standard** to determine fluid responsiveness is the **change in SV following a fluid challenge**
- The disadvantage of this technique is that a **bolus of fluid is given to a patient who may not benefit**. However, the non-responder should receive no more fluid; and the small volume given should “hopefully” do little harm.
- As crystalloids redistribute very quickly the fluid bolus should be given as **quickly** as possible. A bolus of between **200 and 300 cm³** is given, the volume determined in part by the rate of infusion.
- Muller et al reported that a “mini-fluid” challenge with **100 ml colloid over 1 min** was highly predictive of fluid responsiveness

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- A change in **blood pressure** following a PLR or fluid challenge is a poor guide to fluid responsiveness.
 - **SV** may increase without a significant change in blood pressure

Table 9.1 Techniques for assessing fluid responsiveness

Static pressure and volume parameters (ROC ~ 0.5–0.6)

Central venous pressure (CVP)
Pulmonary artery occlusion pressure (PAOP)
Inferior vena cava (IVC)/superior vena caval (SVC) diameter
Flow corrected time (FTc)
Right ventricular end-diastolic volume (RVEDV)
Left ventricular end-diastolic volume (LVEDV)
SVC/IVC variation during mechanical ventilation

Dynamic techniques based on heart-lung interactions (ROC ~ 0.7–0.8)

Pulse pressure variation (PPV)
Stroke volume variation (SVV)
Pleth variability index (PVI)
Aortic blood flow (Doppler or echocardiography)

Techniques based on real or virtual fluid challenge (ROC ~ 0.9)

Passive leg raising (PLR)
Rapid fluid challenge (200–300 cm³)

ROC area under receiver operator characteristic curve

The Patient is fluid responsive. So What Next!

Nothing

- Do not need to increase CO

- Increased lung water

Fluid bolus of 250–500 cm³ LR

Give vasoconstrictor

- increase venous return secondary to α -agonist mediated venoconstriction (sepsis and anesthesia)

Targets of resuscitation

- MAP >65 mmHg (consider > 75 mmHg in those with preexistent hypertension)
CI >2.5 l/m²
Heart rate < 110/min (sinus rhythm)
Adequacy of organ perfusion
Lactate or lactate clearance should not be used as an end-point of resuscitation

Having no precedence to guide me I injected ounce after ounce of fluid closely observing the patient.

Thomas Latta, Physician, (1798–1833)

- The technique of fluid resuscitation described by Dr. Latta nearly 200 years ago has stood the test of time; and is the **ONLY** way to resuscitate patients.... Give a small volume of fluid (LR) and observe the patient (what a remarkable concept).
- This is best done by giving 250–500 ml boluses of LR and closely monitoring the response.
- Furthermore, unlike the fluid challenge which must be given rapidly (to assess fluid responsiveness) fluid boluses are best given over about 30 min (to prevent excessive short lived cardiac chamber enlargement).
- The idea of giving large fluid boluses of 20–30 ml/kg is absurd and likely to lead to severe volume overload.

So, Which Fluid?

- The data presented above suggests that in almost all circumstances **LR** should be the fluid of choice for fluid resuscitation. There are however a few exceptions to this rule:
- **Hyponatremic** dehydration (0.9 % NaCl).
Patients with **acute cerebral insults** at risk of cerebral edema (hypertonic solutions are preferable in these patients (0.9 % NaCl or hypertonic saline).
0.9 % NaCl is considered the initial fluid of choice in patients with **diabetic ketoacidosis** before switching to 0.45 % NaCl. Most patients are switched at some point to one-half isotonic saline to replace the free water loss induced by the glucose osmotic diuresis. LR does not appear to have a role in the treatment of DKA .
- A solution of 1 L **D5W with 2–3 amps of sodium bicarbonate**; severe metabolic acidosis due to ethylene glycol or severe metabolic acidosis due to loss of HCO_3^- (diarrhoea, renal tubular acidosis).
This solution can also be considered in patients with **renal failure and severe metabolic acidosis**

Electrolyte composition of normal saline and lactated ringers solution

	LR	0.9 % NaCl
Sodium (meq/l)	131	154
Chloride (meq/l)	111	154
Calcium (meq/l)	2	–
Potassium (meq/l)	5	–
Lactate (meq/l)	29	–
Measured osmolality [82] (mmol/kg H ₂ O)	257	285

TARGETS OF TREATMENT

- **Mean arterial blood pressure** is a primary indicator of hemodynamic instability.
- End-organ perfusion is maintained over a wide range of mean arterial pressures (MAPs) because of **autoregulation**. In patients with **chronic hypertension**, the autoregulation curve is shifted to the right, indicating that these patients require higher MAPs to achieve adequate end-organ perfusion. Because of its close association with sufficient perfusion, MAP goals become a target for the initial resuscitation efforts in shock.
- Recent literature suggests that a MAP of **65** mm Hg is sufficient for patients with septic shock and is generally associated with good outcomes in critically ill patients.

Alvin Toffler, Writer and futurist (1928)

- *The illiterate of the 21st Century will not be those who **cannot read and write**, but those who cannot learn, unlearn and relearn.*

THANKS, ANY QUESTION?