Need

Fluid status vs fluid responsiveness

S Magder
Department of Critical Care,
McGill University Health Centre
Water is 60% of total weight

Stressed Vol 1.4 L and includes RBC

Plasma water 5%

Extra-cellular Water 18%

Water is 60% of total weight
Increase in Volume to raise cardiac output

MSFP $\uparrow$
Concept of Stressed and Unstressed Volume

• Only stressed volume determines venous return.
  - Normally 1.3 to 1.5 L of total blood volume
  - ~30-45% of this volume is RBCs
• Stressed volume in systemic veins accounts for venous return and it is only about 1 Litre

Thus it is unlikely that a volume infusion > 1L is increasing intravascular volume
Patient has low urine output and is thought to be “pre-renal”

How does volume increase the flow of urine?

1. Increase renal blood flow
2. By increasing blood pressure
3. By increasing cardiac output
4. By increasing preload
5. By increasing Venous Return
What can increase $O_2$ delivery?

\[ \dot{DO}_2 = \dot{Q} \times Hb \times k \times \text{Sat}_a \]

- Volume (preload)
- Contractility
- HR
- Blood
- PO$_2$
- Usually not much gain
- Afterload
1. Is this pt volume responsive?
2. Does this pt need volume?

Version 2

1. Does this pt need volume?
2. Is this pt volume responsive?

*ie is the patient on the flat part of the cardiac function curve?*
Fluid Need

1. Condition that potentially can be fixed by giving volume
   - Raising cardiac output

2. Restore reserves
   - Unstressed volume
   - Interstitial volume

3. Correct electrolyte imbalances
What can volume fix?

1. Cardiac output
   - By increasing stressed volume, VR and thus cardiac preload
   - Presumes that the heart is on the ascending part of the cardiac function curve
2. The increase in cardiac output can then fix tissue perfusion, blood pressure, and urine output
3. Restore reserves and thus allow better “auto” adjustments
   - unstressed volume
   - Interstitial reserves
4. All these should only require < 2 L
5. Measurement of “flow” or surrogate is thus likely crucial in the complex patient
Assumptions in a fluid bolus

1. There is a need to increase perfusion
2. Heart can respond to increased preload (not on the flat part of the cardiac function curve)
3. Fluid expands the stressed vascular volume
   - Does not leak out
   - There is no stress relaxation
Fluid Challenge

1. Assess the value of Pra (NOT the wedge).

2. Give sufficient fluid to raise Pra by ~2mmHg and observe Q.

Type of fluid is not of importance if given fast enough.
Change in CVP of even 1 mmHg should be sufficient to test the Starling response

\[ Q \text{ (l/min)} = 500 \text{ ml/min/mmHg} \]
Inspiratory fall in Pra

No Inspiratory fall in Pra

Q↓Ppl

Pra

Pra
Initial Right Atrial Pressure

mmHG

No Insp Fall

+ve Insp Fall

Magder et al
JCCM 1992
Inspiratory fall

No Inspiratory Fall

L/min (delta)

+ve Resp  -ve Resp
Eg of Systolic Pulse Variation

Baseline ("apnea")

dDown

dUP

SPV
Prominent ‘y’
Consequences of increasing stressed volume

• Systemic compliance
  ~ 112 ml/mmHg
• Systemic stressed Vol
  ~1025 ml
  = MSFP ~ 9 mmHg
• Increase Vs by 1 L
  = MSFP ~ 18 mmHg

(& capillaries are upstream)
VISEP trial

= 17 L in 70 kg man.
= 40% of total volume!
Starling’s Forces

Volume increases filtration

Consider fluid shifts during routine dialysis
Can remove 3-4 L without hemodynamic consequences
Consequence of increased volume

Increased capillary filtration
Consequences of excess fluid

- Dilute clotting factors and hemaglobin
  - Can lead to excess blood use
- Slows tissue healing
- Greater risk of pulmonary congestion
- High venous pressures can compromise hepatic and renal function
- Diluted Hb can lead to decreased ScvO₂ and thus in some algorithms demand for more fluid!!
What is the cause of the shock?

1. Hemorrhagic?
   - Use blood
2. Obstructive (eg PE)
   - Fluids but NE ± inotrope
3. Perioperative
   - Potential for use of starch
4. Septic shock
   - Perhaps early – less value later
   - Should they be given over many days as in the big 3?
5. Cardiogenic shock
   - Fluids not helpful
Hypovolemia?  
? What was lost?

1. Excessive diuresis
   • crystalloid

2. GI loss
   • crystalloid

3. Trauma
   • Blood and crystalloid for interstitial space

4. “Tone”
   • Starch early? Crystalloid? vasopressor?
Part = Q \times SVR \quad (+K)

**Heart**
- Heart Rate
- Afterload
- Contractility
- Preload

**Circuit**
- Stressed volume
- Compliance
- Resistance
- Pra

**Drugs**
- Dobutamine
- Milrinone

**Conditions**
- Sepsis
- Spinal
- NE
Raising the pressure with fluids vs NE
“Auto” volume reserves in the system

1. Capacitance
2. Interstitial fluid
Concept of Stressed and Unstressed Volume

![Diagram of a bathtub illustrating stressed and unstressed volume with MSFP]

- Stressed Volume
- Unstressed volume

MSFP
Vascular Capacitance

- Cannot be measured clinically
- Increased by vasodilators, $\alpha$-adrenergic blockers, and sedation
- Reserves can be determined from the “volume history of the patient”

_Beware of narcotics or vasodilators in patients with increased sympathetic tone_
Change in Capacitance (can change by 10-15 ml /kg)
“volume” balance is really about salt balance

- Osmolality is tightly regulated
- Osmolality depends upon particles
- On +v side this is essentially Na$^+$
- Thus fluid balance is really about Na$^+$ balance
- Daily recommended Na$^+$ is $\sim$2.5 to 3 G/day
- 100 ml 0.9% saline gives $\sim$ 9 G/day
  - Equivalent to a jar of Kosher dill pickles!!
Resuscitation should include interstitial Volume

3.5 L

35% Interstitial

65%

Plasma

IC

EC -12 L
Normal Saline in Patient with Large Extra-cellular Volume

The rule that saline distribution is 1/3 vascular 2/3 interstitial is no longer true.
When “volume resuscitating” consider:

- Hydration
- Extracellular volume
- Intravascular volume
What is hydration?

- The state of hydration refers to the amount of water relative to the amount of solute particles.
  - **Dehydration** -- not enough water for the solute (i.e., osmolality is above normal)
  - **Excess hydration** -- too much water for the solute (i.e., osmolality is below normal)

- Hydration is assessed by examining the solutes.
  - Primarily $\text{Na}^+$
  - Also glucose, urea, alcohol, ketones etc.
Assessment of extracellular volume

• Excess volume
  – Peripheral edema
  – Acites
  – Pulmonary edema and pleural effusions

• Decreased volume
  – Loss of skin turgor
  – Loss of sweat
Assessment of vascular volume

• Excess volume
  – Elevated jugular veins
  – S3
  – Pulmonary venous congestion

• Decreased volume
  – Postural hypotension
  – Flat neck veins
  – Tachycardia (not a strong sign)
General Principles of Maintenance fluids in “normal patient”

- Provide per day (70 kg man)
  - ~ 2-3 meq/kg Na\(^+\) (~154 meq)
  - ~ 30 ml/kg H\(_2\)O (2 L)
  - ~ 1 meq/kg K\(^+\) (80 meq)
  - ~ 100 g glucose

This is provided pretty closely by 2 L of ½ N saline in D5W
Implications from the physiology

1. Large volume resuscitations are at best only temporarily increasing cardiac output
2. Large saline infusion is just “feeding” the interstitial space
3. If goal of colloid is to increase intravascular volume, it makes no sense to give more than 1 to 1.5 L
Clinical responses to a therapy can only be in the realm of the physiological possible
Use fluids judiciously

Avoid the Michelin Man syndrome
Increased back pressure to kidney and liver
Increased back pressure to kidney and liver
Change in Capacitance

Can recruit ~ 10 ml/kg of unstressed to stressed
What happens when we increase plasma volume?

\[ MSFP \uparrow \]

\[ P \]

\[ V \]

\[ Q \]

\[ MSFP \]

\[ Pra \]
Total fluid removal 8810 L in 3 days
Decreased LV function by RV distention

Decompress the right heart

Can lead to decrease in LV function
Overfilling of the right heart can decrease cardiac function.
Looks like “downward slope” to Starling Curve

Volume does not increase cardiac output

Excess volume decreases cardiac function
“Pressure effect” of osmoles

19.3 mmHg
1 Alb has the same osmotic effect as a tiny Na$^+$

60 Kd = Na$^+$
Importance of volume

Determinants of flow

\[ Q = \frac{\text{Stressed Volume}}{C_v \times R_v} \]

Increase the initial volume

Greater flow

Stressed volume

Cv

Rv
There is a limit to cardiac filling.

When reached, further increases in preload with a colloid will not increase cardiac output and can only do harm.
Cardiac function concept

• Start at right atrium --- out from aorta
• Heart lung treated as unit
• LV can only put out what the RV gives it
• RV filling limited at ~ 6-10 mmHg
• Importance of where transducer is leveled
• Pulmonary artery occlusion is a useful value for determining best filling pressure
Oncotic effect from plasma proteins

\[ \text{+} = \text{Na}^+ \]
\[ \text{-} = \text{Cl}^- \]
Colloid osmotic pressure (Oncotic Pressure)

Vessel walls are impermeable to plasma proteins.
- only 0.5% of total osmotic pressure
- But osmotic effect of these proteins prevents movement of water out of the vasculature

• 85% of this activity is due to Albumin
  (.: Think of the impact of a low albumin)
Concept of Limits

- Limit of “return function”
  - Lowering $Pra$ will not increase $VR$
- Limit of “cardiac function”
  - Raising $Pra$ will not increase $Q$
\[ VR_{\text{max}} = \frac{\text{MSFP}}{R_v} \]

**Increase HR**
- SV decreases
- Q does not change

\( \text{Pra} \)
Importance of MW in starches

Initial size is a determinant of number of subsequent particles - also affected by rate of breakdown.

Cleared by kidney when small enough (approx 60 Kd)

Amylopectin

α-1,6 bond

www.sbu.ac.uk/water/hysta.html
Value of colloids

- Expand *intra*vascular volume
- If stressed volume is only 1.4-1.5 L then why would you give more?
- Do not help the interstitial reserves
Shock is inadequate blood flow and oxygen delivery for tissue needs
What does CVP tell you?

- Is the CVP high and therefore it is unlikely that volume loading will help?
  - There is no “optimal” CVP
  - Concept should be “high” vs “low” CVP

- What happened to the CVP in relation to a change in hemodynamics (especially cardiac output)
It is often argued that CVP is of no value

It is true!!

• CVP must be used in the context of the hemodynamics (especially Q) and the changes over time

• *Think of use of PCO\textsubscript{2} – it must always be in the context of the pH*
What determines cardiac output?

(What makes the blood go around?)
Cardiac output depends on Cardiac and Return functions.

Cardiac function

Return Function

MSFP

Stressed volume

Unstressed volume

Pra

Rv

Cardiac output depends on Cardiac and Return functions.
The height of the water determines the outflow
Heart has a "restorative" function. Volume stretches the veins and creates the "recoil" pressure that drives flow back to the heart. Heart has a "permissive" function. It lowers the outflow pressure and allows veins to empty, refilling them.
BP = Cardiac Output x SVR

BP can be increased by an increase in flow
or
an increase in SVR

Are they the same?
Pra = MSFP

Pra < MSFP

"working Pra"
Increase in blood flow with no change in blood pressure must mean that there is a decrease in resistance
2. Volume responsiveness does not mean volume need

Your CVP sitting in this lecture is likely < 0 mmHg

You do not need a saline bolus!
Cardiac limited “Wasted preload”

Cardiac output

Pra/CVP

Cardiac Preload

Gradient for VR
1. Capacitance
2. Interstitial fluid
General Schema

Plasma: 35%
Interstitial: 65%
IC
EC
Consider the following:

In the sitting position the CVP of a normal person is $< 0$ mmHg, yet cardiac output (Q) and blood volume are normal.

&

*There is no need for fluid infusion*
Determinants of Fluid Filtration (Starling Forces)

\[ J_v = K_{f,c} \left[ (P_c - P_t) - \sigma_d (\pi_c - \pi_t) \right] \]

where:
- \( J_v \) = vol flow across the wall
- \( K_{f,c} \) = filtration coefficient
- \( P_c \) = Capillary pressure
- \( P_t \) = tissue pressure
- \( \pi_c \) = capillary oncotic pressure
- \( \pi_t \) = tissue oncotic pressure
- \( \sigma_d \) = reflection coefficient
Circulatory Model

Heart

Rv

stressed volume

unstressed volume

MCFP
BP = Cardiac Output x SVR

Cardiac Function:
- Heart Rate
- Stroke Volume
- Afterload
- Contractility
- Preload

Return Function:
- Stressed volume
- Compliance
- Resistance
- Pra

CO must = Venous Return
Change in CVP of even 1 mmHg should be sufficient to test the Starling response.

Given:
- Slope = 500 ml/min/mmHg
- Plateau

Diagram:
- Q (l/min)
- Pra (mmHg)
- Slope = 500 ml/min/mmHg
- Plateau
Approach:

1. Assess adequacy of inspiratory effort from wedge
2. Evaluate the change in Pra

Eg of no fall in Pra with inspiratory effort

Magder et al JCCM 1992
Initial Right Atrial Pressure

mmHG

No Insp Fall +ve Insp Fall

0 4 8 12 16 20