Hemodynamic monitoring: Guyton at the bedside

*Flow-directed approach to shock*

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An approach to hemodynamic monitoring: Guyton at the bedside

Sheldon Magder*
What are the key points that Guyton made?

• 1. Cardiac output is not just about “heart rate and stroke volume”
  – Key factor is how blood gets back to the heart
  – Guyton sited Ernest Starling

  • *venous return is dependent upon an upstream pressure which he called “mean systemic pressure”*
Increase the initial volume

Determinants of flow

\[ Q = \frac{\text{Stressed Volume}}{\text{Cv} \times \text{Rv}} \]

Importance of volume

Stressed Volume

Unstressed Volume

Greater flow

Stressed volume

2x
2. Established a method to measure “mean circulatory filling pressure” (MCFP)

3. Identified the determinants of venous return – constructed venous return curves (return function)
The height of the water determines the outflow
What are the key points that Guyton made (continued)?

4. Cardiac output is determined by the interaction of “cardiac function” and “return function”
Volume stretches the veins and creates the “recoil” pressure that drives flow back to the heart.

Heart has a “restorative” function which refills the veins.

Heart has a “permissive” function. It lowers the outflow pressure and allows veins to empty.
In contrast to your tub, the veins are the source of fluid in the circulation.
Concepts:
Capacitance and Stressed volume
Pressure vs Total Volume
Concept of Stressed and Unstressed Volume
Pressure-Volume Relationship of the Vasculature

![Graph showing the pressure-volume relationship of the vasculature with a linear increase in MSFP (mmHg) with volume (ml). The graph distinguishes between unstressed and stressed states.]
Pra = MSFP

\[ Q = 0 \]
Pra < MSFP
Principle:
The heart controls cardiac output (venous return) by regulating Pra and \textbf{not} by controlling Part.
Pra = MSFP

Pra < MSFP
$P_{RA}(CVP)$ should always be used to determine the optimal volume for *cardiac output*
Increase in Cardiac Function Curve

Higher Q for a given Pra

↑ Heart Rate
↑ Contractility
↓ Afterload
Return Function

Cardiac Function

“Return limited”

“Cardiac limited”

“working CVP”

Pra

Cardiac output

Right Atrial Pressure

Pra = MCFP

Q

Rv

Pra < MCFP

-1/Rv

MCFP

Q

A

B
Change in Cardiac Function

Increase in gradient for venous return
Change in Volume

P vs V

MCFP ↑

Q

MCFP

Pra
Change in Capacitance
(can change by 10-15 ml /kg)
Change in Capacitance

\[ \text{MCFP} \rightarrow \text{Q} \]

\[ \text{Pra} \rightarrow \text{MCFP} \]

\[ \uparrow \text{MCFP} \]

\[ \text{P} \rightarrow \text{V} \]

\[ \text{Q} \rightarrow \text{MCFP} \]

\[ \uparrow \text{MCFP} \]
Change in venous resistance:
Change in volume on plateau of cardiac function curve

No change in Q
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.

\[ \text{Slope} = 500 \text{ ml/mmHg} \]

Diagram showing:
- \( Q \) (l/min) on the y-axis
- \( \text{Pra} \) (mmHg) on the x-axis
- Plateau at \( Q = 5 \) l/min when \( \text{Pra} = 10 \) mmHg
When $Pra < \text{Pleural Pressure}$, the great veins collapse when they enter the thorax.

Lowering $Pra$ no longer effects Cardiac output

$= \text{“vascular waterfall”}$
The amount of water flowing over a waterfall is not affected by the height of the waterfall.
Lowering Pra further will not increase Q

\[ VR_{max} = \frac{MCFP}{R_v} \]

Stressed volume must increase to increase Q
When there is "venous collapse," an increase in HR decreases SV but Q does not change. Consider devices for "SV" significance.
Patient has low urine output and is thought to be “pre-renal”

You decide to try giving volume

How does volume increase the flow of urine?

1. Increase renal blood flow
2. By increasing blood pressure
3. By increasing cardiac output
For most tissues it is Oxygen Delivery (DO$_2$) that counts

**What do we have to play with?**

$$
\dot{DO}_2 = Q \times Hb \times k \times (Sat_a)
$$

- **Volume** (preload)
  - $\uparrow$ Contractility
  - $\uparrow$ HR
- **Blood**
  - $\uparrow$ PO$_2$
    - Usually not much gain
- **Afterload**
  - $\downarrow$
How do you apply these principles to manage volume clinically?

• 1\textsuperscript{st}: is there a hemodynamic problem to fix?
• Is inadequate cardiac output the problem?
• Is the inadequate cardiac output due to the heart function or return?
BP = Cardiac Output \times SVR

Measured variable

First Question to ask:

Is the cardiac output decreased

Or

Is the cardiac output normal or increased
Where does “volume” show up in the equation?

Part = Q x SVR \(( - PV)\)
If Q is normal or elevated it is a SVR problem

Causes of a low SVR:

- Sepsis (sirs)
- Drugs
- Arterio-venous shunts
- Spinal/epidural injections
- Spinal injury
- Cirrhosis
- Thyroid disease
- Anaphylaxis
- Corticosteroid deficiency
- Anemia
- Beriberi
BP = \textbf{Cardiac Output} \times \text{SVR}

\textbf{Cardiac Function:}
- Heart Rate
- Stroke Volume
- Afterload
- Contractility
- Preload

\textbf{Return Function:}
- Stressed volume
- Compliance
- Resistance
- Pra
Is it a “circuit” or a “cardiac” problem?

Pra is the clue:

• *Actual level is only of some help*
  – CVP > 10-12 mmHg usually indicates cardiac limit, but limit can be higher or lower
• Examine the response to a fluid challenge or
• Examine the respiratory variation in Pra
Three examples of low CVP

1. Normal cardiac function and blood volume
   - $Q_{\text{Pra}} = 0$ mmHg
   - $Q = 5$ L/min

2. Depressed cardiac function but low blood volume
   - $Q_{\text{Pra}} = 0$ mmHg
   - $Q = 3$ L/min

3. Decrease in volume with normal cardiac function
   - Give volume
   - Give inotrope
1. Increase volume with normal cardiac function

2. Decrease cardiac function and normal blood volume

3. Decrease RVR without change in cardiac function (?sepsis)

Three examples of high CVP
BP ↓ = Q ↓ x SVR

*CVP fell* with the fall in Q

Decrease in volume with normal cardiac function

*Give volume*
$BP \downarrow = Q \downarrow \times SVR$

*CVP rose with the fall in Q*

Decreased cardiac function - Give inotrope
1. Decrease in cardiac output
   - decrease in return function (decreased volume)

2. Decrease in cardiac output
   - decrease in cardiac function

Fall in Q with fall in CVP

Fall in Q with rise in CVP
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
Inspiratory fall in Pra

No Inspiratory fall in Pra
Inspiratory fall

No Inspiratory Fall

+ve

-ve

L/min (delta)

-1.0

-0.5

0.0

0.5

1.0

1.5

2.0

2.5

+ve Resp -ve Resp

L/min (delta)

+ve Resp

-ve Resp

Graph showing data points for Inspiratory fall and No Inspiratory Fall.
Part = Q x SVR (+K)

Dobutamine
Milrinone

Heart
Heart Rate
Afterload
Contractility
Preload

Circuit
Stressed volume
Compliance
Resistance
Pra

Sepsis
Drugs
Spinal
NE

Volume
NE

NE
Fluids after cardiac surgery: A pilot study of the use of colloids versus crystalloids*

Sheldon Magder, MD; Brian J. Potter, MD; Benoit De Varennes, MD; Steve Doucette, Msc; Dean Fergusson, PhD; for the Canadian Critical Care Trials Group

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Fluid Protocol

CI < 4 and CVP ≤ 12?

Yes

Protocol Fluid Bolus

Check CI and CVP

CVP incr. < 2 and CI incr. < 0.3
CVP incr. ≥ 2 and CI incr. < 0.3
CVP incr. < 2 and CI incr. ≥ 0.3
CVP incr. > 2 and CI incr. ≥ 0.3

Inadequate challenge

Review fluid criteria

Yes

Catecholamine Protocol

No

Saline

Total Protocol Fluid >1L/24hr?

Observe or wean

Cardiac response ok yet

CI < 2.2 or MAP < 70

UO < 20

Pt not volume responsive

triggers

CI < 2.2 or MAP < Target or SBP < Target or CVP < 3 or Urine < 20 cc/hr

CI < 2.2 or MAP < Target or SBP < Target or CVP < 3 or Urine < 20 cc/hr
Primary Outcome
Catecholamines between 8:00 and 9:00 AM

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Clinical Scenario

- Post operative cardiac surgery
- Initial blood pressure is 80 mmHg

*What do you want to know*
- CI = 3.2 l/min/m$^2$, Pw = 12 mmHg, Pra = 10 mmHg,

*What is wrong and what do you want to do?*
Clinical Scenario

• Patient comes back from aorto-coronary bypass surgery. The initial hemodynamics are:
  – \( Q = 2.2 \text{ l/min/m}^2 \), \( \text{Pra} = 12 \text{ mmHg} \), \( \text{Pw} = 8 \text{ mmHg}, \text{ Part} = 110/70 \text{ mmHg} \)

• One hour later
  – \( Q = 1.8 \text{ l/min/m}^2 \), \( \text{Pra} = 6 \text{ mmHg} \), \( \text{Pw} = 7 \text{ mmHg}, \text{ Part} = 90/70 \text{ mmHg} \)

What would you do?
Clinical Scenario

- Patient comes back from aorto-coronary bypass surgery. The initial hemodynamics are:
  - \( Q = 2.2 \text{l/min/m}^2 \), \( P_{ra} = 8 \text{ mmHg} \),
  - \( P_w = 6 \text{ mmHg} \), \( Part = 110/70 \text{ mmHg} \)
- One hour later
  - \( Q = 1.8 \text{l/min/m}^2 \), \( P_{ra} = 12 \text{ mmHg} \),
  - \( P_w = 8 \text{ mmHg} \), \( Part = 80/60 \text{ mmHg} \)

What would you do?
1. Normal cardiac function and blood volume

\[ Q = 5 \text{ L/min} \]
\[ P_{ra} = 0 \text{ mmHg} \]

2. Depressed cardiac function but low blood volume

\[ Q = 3 \text{ L/min} \]
\[ P_{ra} = 0 \text{ mmHg} \]

3. Decrease in volume with normal cardiac function
1. Increase volume with normal cardiac function

2. Decrease cardiac function and normal blood volume

3. Decrease RVR without change in cardiac function (sepsis)
Part = Q \times SVR \ (+_K)

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

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

Drugs
- Sepsis
- Spinal
- NE
- Dobutamine
- Milrinone
Inspiratory fall in Pra  No inspiratory fall in Pra
Importance of volume

Determinants of flow

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

Increase the initial volume

Unstressed Volume

Stressed Volume

Greater flow

Stressed volume

C_v

R_v