Fluid Responsiveness

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OBJECTIVES

**Resuscitation** often requires the **infusion of iv fluid** in the attempt to reverse organ dysfunction by increasing **Stroke Volume**.

1. To show the physio-pathological background of fluid responsiveness in critically ill patients.
2. To illustrate the capabilities of ultrasound approach to drive appropriately fluid responsiveness.
3. To show modalities for combining the ultrasound approach with clinical exam.
4. To provide practical information to drive fluid responsiveness and tolerance in the acute care.
The formula of fluid responsiveness

The adequate oxygen delivery ($DO_2$) to prevent/treat organ dysfunction

$DO_2 = CaO_2 \times CO$

$CO = SV \times HR$

$MAP = CO \times SVR$

$SV = EDV - ESV$

$CaO_2 =$ Oxygen content of arterial blood

Determinants of SV:
1. LV contractility
2. Afterload
3. Preload
The Starling Law

A fluid “responder” is defined as a patient who increases SV by 15%.

Miller et al, 2016
LV Preload (Venous Return, VR)

Effect of sympathetic nervous system on the splachnic venous district

The higher VR the higher EDV

VR = \frac{\text{MBP} - \text{RAP}}{\text{SVR}}

MBP = Mean Blood Pressure
RAP = Right atrial pressure
SVR = Systemic vascular resistance
Invasive static markers of Fluid Responsiveness

Static markers:

- Central Venous Pressure (CVP)
- Pulmonary capillary wedge pressure (PCWP), marker of LVFP
- Ventricular volumes (mainly EDV)
Echocardiographic static markers of Fluid Responsiveness

Static Parameters

Size of the left ventricle: predictive of FR only when the left ventricle is very small. (LV end-diastolic area in P-SAXS <10 cm²)

Significant narrowing of LVOT during systole in pts with HCM

LV restrictive filling pattern
Preload Dynamic Markers of Fluid Responsiveness

Dynamic markers (provoking the circulation by inducing changes of loading conditions):

- Heart-Lung Interactions
- Postural changes
1. Heart-Lung Interaction

This effect is exaggerated in states of low circulating volume and attenuated in the overloaded system or when either ventricle is failing.
Heart-Lung Interaction

**Delta Down**
- Increased Intrathoracic Pressure (ITP)
- Reduced Venous Return (VR) (hypovolemia)
- Reduced Right Ventricular Stroke Volume (RV SV)
- Reduced Left Ventricular Stroke Volume (LV SV) (in few heart beats later)

**Delta Up**
- Increased Intrathoracic Pressure (ITP)
- Increased Left Ventricular Afterload (PAH, RV HF)
- Reduced Left Ventricular Stroke Volume (LV SV)

- dUp is unrelated to fluid responsiveness

**Fluid**
- Presence of fluid

**No Fluid**
- Absence of fluid

ITP = Intrathoracic Pressure
TTP = Transpulmonary Pressure
Echocardiographic dynamic markers of FR

Dynamic Parameters: Stroke volume (SV) variation

SV measurement

SV can be calculated by measuring VTI and LVOT diameter at the same point
How to measure SV variation

Respiratory variation of Stroke Volume

SV variation >12% accurately predicts FR
SV variation >14% has a very high positive predictive value
SV variation <10% has a high negative predictive value

The simple peak velocity variation >12% predicts FR

Tracing the largest and the smallest VTI over a respiratory cycle allows the percentage change to be calculated

SV Variation = \[100 \times \frac{SV_{\text{max}} - SV_{\text{min}}}{0.5 \times (SV_{\text{max}} + SV_{\text{min}})}\]
Great Veins Variation (over the respiratory cycle)

Inferior vena cava (IVC) variation

Background:

IVC in spontaneously breathing patients correlates with right atrial pressure (RAP).
- Best cut-off value for RAP > or < 10 mmHg is 2 cm.
- A small (<10 mm) IVC suggests a good FR.

A RAP <10 mmHg can be assumed if IVC is <12 mm in ventilated patients.
**Great Veins Variation** (over the respiratory cycle)

**Inferior vena cava (IVC) variation**

In controlled ventilation, the IVC expands in inspiration and reduces in expiration.

This variation is abolished when RAP is high. The absence of IVC respiratory variation predicts Fluid Unresponsiveness. Large variations of IVC respiratory variation accurately predicts FR.
Great Veins Variation (over the respiratory cycle)

Inferior vena cava (IVC) variation (The collapsibility index)

First Formula

\[ DV_{IVC} = 100 \times \frac{(D_{\text{max}} - D_{\text{min}})}{D_{\text{mean}}} \]

A diameter variability cut-off value >12% identifies Fluid Responders
Great Veins Variation (over the respiratory cycle)

Inferior vena cava (IVC) variation (The collapsibility index)

**Second Formula**

IVC Diameter Variability (DV)

\[ DV_{IVC} = 100 \times \frac{D_{max} - D_{min}}{D_{min}} \]

A diameter variability cut-off value >18% identifies Fluid Responders
Great Veins Variation (over the respiratory cycle)

Superior vena cava (SVC) variation (The collapsibility index)

The SVC is difficult to see with TTE but can be easily visualized by TOE.

Diameter changes are opposite of the IVC in ventilated patients.

A variation in SVC >36% is predictive of FR.

$$DV_{SVC} = 100 \times \frac{(D_{max} - D_{min})}{D_{min}}$$
2. Postural Changes: Invasive Passive leg raising (PLR)

**Invasive Prediction of Fluid Responsiveness**

**Pulse pressure variation**
(Pulse pressure = Systolic BP – diastolic BP, Waveform analysis)  
**Threshold: 12% Increase**

**Stroke volume variation**
(Waveform analysis)  
**Threshold: 12% Increase**

Despite useful, these methods are limited by the requirement of an arterial cannula and a precise arterial trace.

Confounders limiting accuracy:
Movement artifacts, Catheter kinking, Over- or Under-damping of waveform
Echocardiographic postural changes: Passive leg raising (PLR)

Passive Leg Raising (PLR) variation of Stroke Volume

Tilting a patient from a 45° semi-recumbent head-up position to a 45° leg-up position (= transfer up to 300 mL of blood into the central circulation).

SV or simply VTI is measured before and after 1 min after PLR.

SV or VTI increment >10% suggests FR.

36 % VTI Increment
“Fluid Challenge” effect on SV

“Fluid challenge” definition:  
Rapid administration of 250-500 mL of intravenous fluid

Procedure:  
Assessment of the LVOT VTI response to fluid challenge  
(immediately before and after)

Pittance:  
Despite SV increase, hemoglobin and hematocrit are necessary diluted.  
Accordingly, the balance of these effects (SV increase vs Hb dilution)  
determines whether overall O₂ delivery has been augmented or not.
Fluid Tolerance

PREDICTION OF FLUID RESPONSIVENESS
In both spontaneously breathing and ventilated patients:
• A small IVC that varies in size with respiration
• Non dilated right heart chambers
• A non displaced interventricular septum
• An absence of RV and LV failure
• An absence of markers of elevated LVEDP

PREDICTION OF FLUID UNRESPONSIVENESS
• When VTI no longer significantly increase with a fluid bolus
• Transmitral E velocity and E/A ratio are increased
• RV size is increased
Conclusions

Small hyperdynamic ventricles with a small IVC suggest significant hypovolemia.

In a shocked patient without signs of overt hypovolemia, dynamic indices of FR should be sought.

Echocardiography can provide additional information on the validity of other clinical and monitoring markers.

Echocardiography informs about the dangers of delivering a fluid bolus in terms of adding extravascular fluid or worsening LV filling.

When interpreting echo findings, the intrinsic limitations of the technique in a given patient shall be taken into account.
The “grey zone” approach to flow variation assessment
SV variation: Conditions to be satisfied

1. Sinus Rhythm
2. Tidal volumes > 8 ml/Kg
3. Normal intra-abdominal pressure
4. Intact thorax
How to estimate fluid responsiveness

Whether the patient improves with fluid infusion, vasopressors or inotropes can be difficult to predict in the emergency room.

Measurements of flow need more equipment (e.g., $O_2$ saturation, Lactate), expertise and time than the simplest parameters such as blood pressure, and the values achieving an adequate tissue perfusion are not easily predictable.

Echocardiographic ultrasound exam is an evidence-based approach and ideally suited to address this issue.