Central Venous Pressure
What is it good for?

Levi Procter, MD
Acute Care Surgery, Trauma and Surgical Critical Care
NO UNAUTHORIZED DISCLOSURES
Objectives

- Define CVP
- How it has been used
- Does it predict fluid responsiveness?
- What do we do?
What is CVP?

- Intraluminal pressure in superior vena cava near/in right atrium
- Static measurement
- CVP = RAP = RVEDP
- RVEDP *assumed* to indirectly reflect right ventricular end-diastolic volume index (RVEDI)
- RVEDI is *assumed* to approximate LVEDV which correlated to LV stroke volume thus CO
- So: CVP = RAP = RVEDP = RVEDI = LVED = LVSV = CO
- This *assumes* no pathology between a complicated circuit
Total Venous Volume

- 70% of blood volume in venous system

\( V_s = \text{systemic veins (extremities/IVC, etc)} \)

\( V_u = \text{Splanchnic circulation and cutaneous veins} \)
Important Definitions/Concepts

• Splanchnic and cutaneous veins contain $\alpha_1$ and $\alpha_2$-adrenergic receptors and therefore are highly sensitive to adrenergic stimulation
• Venous Return (VR) = \(\text{[Mean Circulatory Filling Pressure – CVP]} / \text{Venous Resistance}\)
• CO directly related to VR
• VR can only be increased by increased MCFP and/or decrease CVP
• Main determinant of MCFP is Vs
• MCFP appx 7-12 in humans in steady state (extrapolated from dogs)
• Normal CVP is 2-3 mmHg
• So why does CVP increase CO during infusion of IVF?
• The increase in MCFP (increases Vs) is larger than the increase in CVP
Factors that decrease CVP

- Decreased Vs due to a overall decrease in Vu (ie. hemorrhage)
- Increased venodilation (i.e. cirrhosis, blood sequestered into splanchnic circulation)
- Increased venoconstriction (increased resistance in the liver and portal system)
Factors that increase CVP

• Decreased cardiac pump function:
  - dysrhythmias
  - decreased contractility
  - valvular disease
• Increased PEEP
• Hypoxia
• Pericardial effusion
• IAH
• Continued volume infusion
Normal CVP

• Does NOT mean normovolemic
• Body can have 10-12% blood loss and tolerate by shunting of blood out of Vu to Vs
• Can reflect:
  - normovolemic
  - hypovolemic (10-12%)
  - hypervolemic (increase Vu) – this is manifested by increased IAH b/c bowel swelling
So where did use of CVP in resuscitation come from?
• 25 consecutive thoracotomy patients
• Peripheral line via basilic or saphenous
• Guided into right atrium
• Connected to manometer after surgery
• 23/25 had CVPs of 2-8 mmHg
• None of these had any sig changes in vitals, UOP, HCT/HG
• 2/25 had sig fluctuation in CVP
Case 14

- No change in BP
- Minimal tachy
- No change in UOP
- They transfused 1500 ml blood over 5 minutes based on this drop in CVP alone
- Patient got 24 units blood in first 40 hours based on CVP alone
- Radionucleotide testing demonstrated no sig change in volumes with data presented (pre and post op)
Case 17

- CVP fell
- They claim it caused myocardial ischemia => AFIB w/RVR
- Hg/Hct – 10/43 at time of “ischemia”
- Given O2 and Dig
- Corrected CVP when heart out of afib
Therefore....

It has been determined that right atrial pressure is an accurate and sensitive recording of the effective circulating blood volume.

It is suggested that this method be adopted for any patient in whom suspected alteration of hemodynamics may be considered of significant proportion.

• Low CVP = volume deplete
• High CVP = volume overloaded
Pooled only studies that evaluated the following:
- the correlation coefficient between CVP and measured blood volume
- or the correlation coefficient or receiver operator characteristic (ROC) between CVP or change in CVP (CVP) and change in stroke index/cardiac output following a fluid challenge
Increase in the stroke index or cardiac index of 10 to 15% was used as an index of fluid responsiveness.

The pooled correlation coefficient between baseline CVP and change in stroke index/cardiac index (reported in 10 studies) was 0.18 (95% CI, 0.08 to 0.28).

The pooled area under the ROC curve (reported in 10 studies) was 0.56 (95% CI, 0.51 to 0.61).

The pooled correlation between CVP and change in stroke index/cardiac index (reported in seven studies) was 0.11 (95% CI, 0.01 to 0.21).

The baseline CVP (reported in 11 studies) was 8.7 ± 2.3 mm Hg in the responders, as compared to 9.7 ± 2.2 mm Hg in non-responders (not significant; p = 0.3).
• CVP has no correlation to circulating volume
• CVP does not predict responsiveness to fluid challenge = 50% chance of predicting responsiveness
Does the Central Venous Pressure Predict Fluid Responsiveness? An Updated Meta-Analysis and a Plea for Some Common Sense*

Paul E. Marik, MD, FCCM¹; Rodrigo Cavallazzi, MD²

• Re-reviewed all data (including newer studies)
• Only studies that reported the:
  - Correlation coefficient or AUC between the CVP and change in cardiac performance following a fluid challenge
  - PLR maneuver/postural change
  - positive end-expiratory pressure challenge were included in this analysis

(Crit Care Med 2013; 41:1774–1781)
Mean baseline CVP was 8.2 ± 2.3 mm Hg in the fluid responders and 9.5 ± 2.2 mm Hg in the non-responders.

The summary AUC was 0.56 (95% CI, 0.54–0.58), with no heterogeneity between studies (Q statistic p = 0.9, I² = 0%).

The summary AUC was 0.56 (95% CI, 0.52–0.60) for those studies done in the ICU and 0.56 (95% CI, 0.54–0.58) for those done in the operating room.

Summary AUC was 0.56 (95% CI, 0.51–0.61) for the cardiac surgery patients and 0.56 (95% CI, 0.54–0.58) for the non-cardiac surgery patients.

Summary correlation coefficient between the baseline CVP and the delta SVI/CI was 0.18 (95% CI, 0.1–0.25), being 0.28 (95% CI, 0.16–0.40) in the ICU patients, and 0.11 (95% CI, 0.02–0.21) in the operating room patients.
ΔSPV (ΔPP) vs ΔCVP/ΔPWP (PAOP) and likelihood of fluid responsiveness

Michard F. AJRCCM. 2000

Kumar. Crit Care. 2004
To date...

- Over 100 studies have been published that show no correlation of CVP to predictability of fluid responsiveness\[1\]
- 2 have shown “some” correlation…in standing horses\[2-3\]

Why talk about this?

• Upwards of 90% of intensivists use CVP to guide resuscitation/fluid management [1-2]

• Surviving Sepsis Guidelines recommends CVP target of 8-12 mmHg prior to vasopressor/inotrope. [3]

Why is CVP difficult to interpret?

- Dependent upon:
  - position of patient
  - pulmonary vascular resistance
  - right ventricular compliance
  - valvular disease
  - left ventricular compliance
  - abdominal pressure
  - transpulmonary pressure
  - PEEP
  - etc...
Give them more fluid, who cares?

- Up to 50% of critically ill patients are NOT fluid responsive[1]
- More mortality in ARDS[2]
- More ventilator days[2]
- More ICU days and longer LOS[2-3]
- More renal failure[3]
- More intra-abdominal hypertension => more chances for ACS[4]
- More open abdomens -> more ECF/EAF-> more incisional hernias[5]
- Greater mortality in trauma[6]
- Costs more money to give more IV fluid
- Costs more money to care for the water logged patient complications

2. http://jic.sagepub.com/content/24/1/35.short
4. Abdominal compartment syndrome is common in medical intensive care unit patients receiving large-volume resuscitation.
What endpoints should we follow?

- Good question, data overall for single endpoint is pretty poor
- Lactate is probably best and should be measured frequently, ie q2-4 hours
- Lactate level and its clearance rate - predictors of mortality [1-2]
- Quicker clearance improves survival in hemorrhagic and septic shock [3-4]
- $S_c\text{VO}_2/S\text{VO}_2$ - however doesn’t always correlate with lactate clearance
- Base deficit – only useful if followed in conjunction with lactate, alone is not reliable
- Serum bicarbonate can be followed in conjunction with lactate
- $\text{DO}_2$ - supra-normal oxygen delivery => more death and/or no improvement
- Urine output – alone, not reliable
- Blood pressure – alone, not reliable
- MAP – alone, not reliable
- Heart rate – alone, not reliable
- Shock is a CELLULAR phenomenon

What do we use to determine need for fluid?

• For the residents and fellows:
  - Please stop telling me what their CVP is
  - Stop measuring it

• It only allows for decisions to be made on inaccurate data

• Educate nurses and other physicians to refrain from using CVP in management decisions in resuscitation
What can we use?

<table>
<thead>
<tr>
<th>Method</th>
<th>Technology</th>
<th>AUC*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulse pressure variation (PPV)</td>
<td>Arterial waveform</td>
<td>0.94 (0.93-0.95)</td>
</tr>
<tr>
<td>Systolic pressure variation (SPV)</td>
<td>Arterial waveform</td>
<td>0.86 (0.82-0.90)</td>
</tr>
<tr>
<td>Stroke volume variation (SVV)</td>
<td>Pulse contour analysis</td>
<td>0.84 (0.78-0.88)</td>
</tr>
<tr>
<td>Left ventricular end-diastolic area (LVEDA)</td>
<td>Echocardiography</td>
<td>0.64 (0.53-0.74)</td>
</tr>
<tr>
<td>Global end-diastolic volume (GEDV)</td>
<td>Transpulmonary thermodilution</td>
<td>0.56 (0.37-0.67)</td>
</tr>
<tr>
<td>Central venous pressure (CVP)</td>
<td>Central venous catheter</td>
<td>0.55 (0.48-0.62)</td>
</tr>
</tbody>
</table>

*AUC = area under the curve with 95% confidence intervals.
Central Venous Pressure, what is it good for?
Summary

• CVP not helpful (unless REALLY low or REALLY high)
• Fluid resuscitation should be based on accurate predictors of fluid responsiveness (ie. SVV, PPV)
Central Venous Pressure
What is it good for?

levi.procter@uky.edu