EXPANDED APPLICATIONS
FOR HEMODYNAMIC
MONITORING 2009: APCO and
dynamic variables of Volume
status : Clinical Application

William T. McGee, M.D., M.H.A.
Director, ICU Performance Improvement
Baystate Medical Center, Springfield, MA
Associate Professor of Medicine and Surgery
Tufts University School of Medicine
Boston, MA
Objectives

- SVV during PPV predicts volume responsiveness
- Arterial waveform analysis; time responsive. beat to beat advantage over thermodilution; minutes CCO
- Var dynamic CVP, PCWP static

Simple resuscitation

- Volume until hemodynamic function optimized
- Individualized
- Cardiac function: dynamic change; ischemia, (tone)SVR
- Cardiac compliance: dynamic MAP, HR, ischemia
- R & L heart interactions
- Heart lung interactions
- PPventilation & PEEP, lung compliance
- Afterload
- Venous Capacitance: dynamic? What is it?
- Vascular tone: dynamic
- CONCEPT
- Multiple Starling curves for individuals
- Volume until optimized? Remains elusive. May not be simple!
Effects of various interventions on Frank-Starling Curves

1) Increasing Preload (volume)

Normal Contractility

2) Inotropy

3) Afterload Reduction

Failing Heart

LV Filling (LVEDV, LVEDP, PAOP, CVP)

Guyton; Starling curves; goal of resuscitation or fluid therapy is to optimize an individual patient on their curve

? Which curve?
Dynamic changes

See Answer below

Technology is now available: data exists dynamic measures of fluid responsiveness
Use of CVP in Critical Illness?

Predictor of volume responsiveness

1986

- Pulmonary HTN  acute PE
- Mechanical ventilation MAirwayP  PEEP
- Lung compliance: emphysema v ARDS
- Heart compliance  Tamponade
- Venous capacitance

- Heart and or Lung Disease

Cardiac filling pressures are poor predictors of fluid responsiveness SS: 500cc Hespan; >15% increase in CO

Beauty of Basic Physiology!! immutable

Osman, Crit Care Med, 2007
Response to Volume Challenge Based on Preload

<table>
<thead>
<tr>
<th>Reference</th>
<th>RAP (mmHg)</th>
<th>PAOP (mmHg)</th>
<th>RVEDVI (ml/m²)</th>
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<tbody>
<tr>
<td>[4]</td>
<td>6 ± 1</td>
<td>8 ± 1</td>
<td>143 ± 19</td>
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<tr>
<td>[5]</td>
<td>9 ± 4</td>
<td>10 ± 4</td>
<td>104 ± 27</td>
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<td>[6]</td>
<td>7 ± 2</td>
<td>10 ± 4</td>
<td>80 ± 28</td>
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<td>[7]</td>
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<td>12 ± 2</td>
<td>177 ± 31</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>105 ± 31</td>
</tr>
</tbody>
</table>

| P         | NS         | NS          | NS             |

Values are expressed as mean ± standard deviation. *Right ventricular end-diastolic volume (ml).

Michard, Teboul Crit Care 2000;4:282-289
Objective; today

- Dynamic changes in SV during the respiratory cycle (condition PPV) determines a unique point on the Starling Curve for individual patients and will predict volume responsiveness.

- SV BP (PP)

Simple to Conceptualize

Variation in arterial pressure waveform that we can see from the bedside or feel in the pulse during the respiratory cycle, predicts response to volume: we know this!

Pleural pressure change impact on VR (phasic)

Spontaneous Breathing
Pulsus Paradoxicus
Severe hypovolemia; Status asthmaticus; Tamponade
Effect of Volume Expansion on ΔPulsePressure in ARDS

SVV PPV SPV

- \( V \) = Variability very dependent on volume status of the patient and Delta Pleural Pressure 2 factors: (lung compliance; PEEP) & Vt Emphysema vs. ARDS

- And cycle length; regular NMB; cardiac compliance

- Paradoxical pulse in Severe Asthma & Tamponade ex. *Spontaneous breathing*

- Variability is increased in hypovolumia!!

\[ \Delta SV \text{ (SVV)} \] Starling relationship: respiratory variation in SV @ different preloads

\[ \Delta SV_1 \] small variation

\[ \Delta SV_2 \] large variation

\[ \Delta EDV_1 \] starting point is not

\[ \Delta EDV_2 \]

The increase of preload volume is equal: \( \Delta EDV_1 = \Delta EDV_2 \)

\( \Delta SV_1 \) \( >> \) \( \Delta SV_2 \)

\( \Delta EDV \) induced by ventilator
Arterial pulse pressure variations during mechanical ventilation to monitor preload-dependency and response to fluid therapy

- Monitor the change in cardiac output in response to PEEP in ARDS; PEEP = 0 or 10 cm H2O
- Compared the ability of arterial pressure variation during PPV to predict the subsequent increase in cardiac output with fluid loading in Sepsis

\[ \Delta PP \text{ Predicts the Decrease in Cardiac Index with the addition of 10 cm H}_2\text{O PEEP (ARDS)} \]
Does Pulse Pressure Variation Predict Preload Responsiveness?

Study Protocol
Measurements were performed in duplicate in septic hypotensive patients
- prior to volume expansion
- 30 minutes after volume expansion (500 mL 6% Hetastarch)


Baseline \( \Delta PP \) Predicts Volume Responsiveness (500cc Hespan) in Hypotensive Septic Patients

\( y = 1.01x - 1.46 \)
\( r^2 = 0.85 \)

Receiver-Operator Characteristic (ROC) Curve for Predicting >15% Increase in Cardiac Output

AUC=0.98
PPV(13%)
Sens 94%
Spec 96%

SVV is inversely related to SV.

2/2007 Mcgee, Hatib, SCCM
SV – SVV mirrors Frank-Starling EDV – SV Relationship
Preload increases from A to B

Based on the F-S relationship optimal preload was approximately 230 cc. This corresponded to a SVV of less than 15%. An arterial waveform can provide information about cardiac performance similar to a Frank – Starling curve.

2/2007 Mcgee, Hatib SCCM

Blood Volume and Renal Replacement Therapy
Chest 10/08 Philadelphia

The Purpose:
To determine if blood volume changes during RRT in the form of either continuous (CVVHD) or intermittent (IHD) dialysis correlate with changes in cardiac performance as measured by minimally invasive techniques

The Hypothesis:
As blood volume either increases or decreases during a dialysis session, the cardiac performance responds accordingly
Blood volume vs Indexed SV: volume removal CVVH

\[ y = -0.3663x - 0.1162 \]

\[ R^2 = 0.8676 \]

-18  -16  -14  -12  -10  -8  -6  -4  -2  0  2

-5

0

5

10

15

20

25

30

35

40

45

% Delta BV

Delta SVI
Optimal

• Fluid therapy
• Volume status
• Blood pressure
• CVP; PCWP
• CO
• SV
  – RVEDV
  – RVEF
• EF
• for a given condition have not been determined. Exception ALI/ARDS

But; most (all) intensivists agree!

• There are multiple clinical scenarios where optimization of cardiac function is a desirable goal
• Dynamic variables PPV SVV should help facilitate this
There are multiple clinical scenarios where optimization of cardiac function is a desirable goal.

SVV & SV analysis should help facilitate this.

If SVV is elevated and SV is low, SV likely can be improved with volume.

R R interval afib, dysrhythmia

Converse may not always be true: some pts with SVV<13% may also be volume responsive.

▲ pleural pressure, lowVt, stiff lungs etc: heart lung compliance issues

Challenges: 1)Vt 2)leg raising 3) Volume

Assess impact on SV
Challenges

- Bedside experiments
- Why we went into CCM!!!
  Immediate results.

Methods: Passive leg raising and volume challenge NS:
Aortic blood flow measured at arrows (SV)

71 Mechanically ventilated, shock pts in the ICU
Max effect of PLR, 30s (SV) bolus given over 10 min

Monnet, Pinsky, et al. CCM 2006 vol 34#5 pp.1402-1407
The passive leg-raising test consists of measuring the hemodynamic effects of a leg elevation up to 45°.

A simple way to perform the postural maneuver is to transfer the patient from the semirecumbent posture to the passive leg-raising position by using the automatic motion of the bed.


Open circles = non responders; † and # p<.05 from baseline; Passive leg raising represents a rapidly reversible fluid challenge and is safe.
Kim HK, Pinsky MR.

*Effect of tidal volume, sampling duration, and cardiac contractility on pulse pressure and stroke volume variation during positive-pressure ventilation*


- 7 mongrel dogs
- Anesthetized Pentothal
- PAC - TD - CO
- PICCO device arterial waveform thoracic aorta
- Dogs were killed 100 mg. Pentothal followed by KCl
**PPV v cycle length**

![Graph showing PPV v cycle length](image)

Figure 1. The effect of varying the number of respiratory cycles used in the calculation of pulse pressure variation (PPV) for all conditions and all animals. Data are means ± sd; n = 6, *p < 0.05 vs. eight respiratory cycles.

**Impact of varying Vt on PPV/SVV**

![Graph showing impact of varying Vt on PPV/SVV](image)

Figure 2. The effect of varying tidal volume (Vt) in the calculation of pulse pressure variation (PPV) and stroke volume variation (SVV) for all conditions and all animals. Data are means ± sd; n = 7, *p < 0.05 vs. Vt of 10 mL/kg.
Preload: norepinephrine & B blocker v. PPV/SVV

**Impact of pleural pressure (compliance) on analysis of SVV**

- Recruitable SV
  - Vt: 400 A/C: 20 SVI: 35 SVV: 8-10
  - Vt: 700 A/C: 10 SVI: 33 SVV: 18-20
- Fluid bolus: 500ccNS over 15 minutes
  - Vt 400 A/C: 20 SVI: 44 SVV: 7-10

SVI increased > 30%
Influence of Tidal Volume on Stroke Volume Variation. Does it really matter?

Linearity of response; individual Starling curve

Fig. 1 Pre-infusion arterial pulse pressure variation and fluid-loading induced increase in cardiac output in the "low" and the "high" tidal volume (Vt) groups.

CONCLUSION : SVV

Arterial waveform analysis during Positive Pressure Ventilation predicts an individual’s position on their Starling Curve and **optimization of cardiac performance is good**!

![Graph showing SVV variability](image)

- **Less variability**
  - SVV is low $<$ 13%

- **More variability**
  - SVV is high $>$ 13%
“The greater the ignorance the greater the dogmatism”

Sir William Osler,
Montserrat Medical Journal (1902)

FINAL very SIMPLIFIED
HEMODYNAMIC ALGORITHM*
SVV & SI: BP and u/o

Volume Responsive: SVV > 13%

Yes

Fluid Infusion

No

SI Normal

SI Low

SI High

Pressor

Inotrope

Diuretic
PHYSIOLOGIC ALGORITHM USING SVV & SI

**Volume Responsive: SVV>13%**

- Yes
  - Fluid Infusion 1 liter NS

- No
  - 1. SI Normal
  - 2. SI Low
  - 3. SI High
    - Pressor
    - Inotrope/Vasodilator
    - Diuretic

**Non-volume responsive (SVV<13%)**

*Remember if pt is fine do nothing!! (can’t make them better)*

The clinical impression of non-volume responsive patients along with the stroke index directs therapy.

1. **SI Normal**: Pressor
   - Vasodilation, severe sepsis or septic shock

2. **SI Low**: Inotrope/Vasodilator
   - Low output state

3. **SI High**: Diuretic
   - Acute lung injury, ARDS, or previous massive resuscitation (wet lungs)
2 SVI Low
Inotrope/vasodilator

No further response to volume; Clear lungs;

or Leg Raising; wet lungs AKI (i.e., SVI does not improve) clinical pulmonary congestion and/or hypotension

Conclusions: FACTT: ALI/ARDS
nejm may and June 2006

• Optimal fluid balance is good; dry is better than wet, CVP <= 9; PCWP <= 13 CVP or PAC are equivalent. Unstated Goal of study. (eliminate PAC)

• Neither CVP or PCWP is good @ predicting optimal fluid balance: los not mortality is impacted; mortality not related to fluid therapy!!

• Diuretics (lasix) are useful in ALI/ARDS; 6-7l/wk difference (no increase in renal failure)
### SIMPLE HEMODYNAMIC ALGORITHM

**SVV & SI Hypotensive/Oliguric ALI/ARDS**

<table>
<thead>
<tr>
<th>Volume Responsive SVV&gt;13%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Yes</strong></td>
</tr>
</tbody>
</table>

#### ALI/ARDS

- 5-10% Ardsnet CVP/PCWP

1. **Volume Challenge**
   - **SI Normal** (40-50)
   - **SI Low (<40)**
   - **SI High (>50)**
   - **Pressor**
   - **Inotrope**
   - **Diuretic**

---

**FACTT Algorithm: Composite Protocol-Version 2**

**Appendix I**

<table>
<thead>
<tr>
<th>Intravascular Pressure</th>
<th>MAP &lt; 60 mm Hg or CVP &gt; 15 cm H2O</th>
<th>MAP ≥ 60 mm Hg AND off vasopressors (Dopamine &lt; 5 mcg/kg/min is not a vasopressor)</th>
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<tbody>
<tr>
<td></td>
<td>CVP</td>
<td>Average UDP &lt; 0.5 ml/kg/hr</td>
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<tr>
<td></td>
<td>PACO2</td>
<td>Average UDP ≥ 0.5 ml/kg/hr</td>
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<td>Conservative</td>
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<td>15-18</td>
<td>20</td>
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</tbody>
</table>

**NOTE:** numbers in upper right hand corner of each cell = cell numbers; superscripts refer to footnotes that may modify instructions.

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**Algorithm Version 1, v. 10/12/2006**

**FACTT Study Version 8**

**ARDSnet Study**

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Clinical Application

- 64 y.o. male DM, HTN, mild COPD; at rehab, post amputation for limb ischemia, SOB and is febrile. EW 102°; 80/50; 120; 33
  - Crackles diffusely bilateral
  - Abdomen – soft
  - Extremities – well healed wound; erythema and cellulitis on non-amputated side.
Data

• Chest x-ray – patchy infiltrates - consolidation, bilat.
• WBC 17; 29% bands; AG = 19; Cr 2.4
• 7.22/48/53/19
• EKG - LBBB

Impression

• Saturday @ midnight
  – Severe sepsis/septic shock
  – Complicated by:
    • ARF
    • ?ACS with pulmonary edema
    • Complex physiology
Clinical Picture

- BP 100; Levophed (olig/anuric) 0 – 5cc/hr x 8 hr
- 8L crystalloid by 8am
- Increased Cr and increased A-a gradient – abnormal chest x-ray

Serial Data

<table>
<thead>
<tr>
<th>SVV%</th>
<th>SI nl 40-50 cc/bt/m²</th>
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<tbody>
<tr>
<td>24</td>
<td>22</td>
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<tr>
<td>Tx</td>
<td>1L + 1L</td>
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SIMPLE HEMODYNAMIC ALGORITHM
SVV & SI Hypotensive

Data

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<th>SVV</th>
<th>SI</th>
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<tr>
<td>1L</td>
<td>1L</td>
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<td>38</td>
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<tr>
<td>u/o 20-25cc/hr</td>
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</table>
### SIMPLE HEMODYNAMIC ALGORITHM

**SVV & SI Hypotensive**

- **Volume Responsive SVV > 13%**
  - Yes
  - SI Normal (40-50)
  - SI Low (<40)
  - SI High (>50)
  - Pressor
  - Inotrope
  - Diuretic (?)

- **No**

---

**CI • 2.7**

SVV decreasing as SV increases with volume resuscitation 1000 cc NS over 7 minutes

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11/27/2005
1:29:54 PM
Data

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<tr>
<td>7</td>
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<td>43</td>
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</tbody>
</table>

u/o 15cc/hr @ 11am
Remains on Levophed 14 L in 11 hrs: SI nl
SI high max@ 58cc by 3PM Lasix
Timely resuscitation; high A-a no PAC

SIMPLE HEMODYNAMIC ALGORITHM
SVV & SI Hypotensive/Oliguric

- Volume Responsive SVV > 13%
  - Yes
  - SI Normal (40-50)
    - Pressor
  - SI Low (<40)
    - Inotrope
  - SI High (>50)
    - Diuretic (?)
- No
  - 7%
  - Wet cxr
Outcome

• Discharged on day 8
• 7 days on ventilator
• Peak creatinine 3.3 → 1.6
• No other organ failures

Case 2

• 54 y.o. female; PVD
• Claudication
• Elective AAA repair
• Redo operation
Case in OR

- 8 u pRBCs
- 12L crystalloid
- Some ooze
- On Levophed, Vasopressin
- Dorsalis Pedis, Doppled on both sides
- To ICU Stable!

ICU Arrival

- Anuric
- On pressors
- Awake
- Clear lungs
- Edematous anasarca
- HR 150; BP 110/70; vent
- SVV 17 SI 20
SIMPLE HEMODYNAMIC ALGORITHM
SVV & SI Hypotensive

17% Volume Responsive SVV>13% ??

Yes

Volume Challenge

No

SI Normal (40-50)

SI Low (<40)

SI High (>50)

Pressor

Inotrope

Diuretic (?)

Data and Treatment

- Hct = 33; coagulopathic
- 2L + 4 FFP+10 platelets in 30 minutes
- SVV 7 SI 29
- HR 140
- Lopressor 2.5mg
- SVV 6 SI 36
- HR 115
ICU Treatment

- 30 min. after bolus IV @ 1L/hr
- SVV 16 SI 19
- HR 145
- Rpt bolus 2L x 3
- Transient improvement in
  - SVV <9 SI 28-36
- 9L in 3 hours after 12 L in OR
- Cyclical response in
  - SVV SI
- High variability SVV---Low stroke index
- HR ~ 135-150 BP 95-110 on pressors
- Class III hemorrhage
- Call surgeon → OR
- Hct >30
In OR

• Large bowel dead
• Patchy necrosis small bowel
• Open and shut
• Patient expires

• Clinical assessment adequate!

DERESUSCITATION

• 51 year old male admitted to ICU
  – Septic shock due to bacterial pneumonia
  – Aggressive volume resuscitation early in course
  – Required pressor support
  – Mechanical ventilation with high FiO₂ requirement
  – Developed acute kidney injury with failure
    • Placed on continuous venovenous hemodialysis (CVVH)
• Clinical course
  – Shock improved, pressor requirements markedly decreased by day 3 of ICU stay
  – Lung function remained poor, unable to wean ventilator
    • Chest x-ray consistent with pulmonary edema and pleural effusions
Data

- CI = 5.6
- SVI = 48
- SVV = 7%
- CO = 15.5 L/min
- SV = 132 mL
- CVP = 16 mmHg

DEresuscitating the VOlume
OVERloaded Patient

- This patient is ready to DEVOLVE
  - Applying algorithm is clearly safe to remove volume
- Patient tolerated negative fluid balance via CVVH well
  - Ventilator requirements improved on a daily basis
**Take Home Message**

**Hypotension, Oliguria; ALI/ARDS**

**Volume overloaded**

**Volume Responsive Algorithm**

© WT McGee MD 2005

- **Volume Responsive SVV>13%**
  - **YES**
    - **Volume Challenge**
      - **SVI Normal (40-50)**
        - **Pressor**
      - **SVI Low (<40)**
        - **Inotrope**
      - **SVI High (>50)**
        - **Diuretic**
  - **NO**
    - **SEPSIS**
    - **CHF**
    - **ALI/ARDS**

**SIMPLE HEMODYNAMIC ALGORITHM ALI/ARDS DEVOLVE**

- **Volume Responsive SVV>13%**
  - **Yes**
    - **Volume Challenge**
      - **SI Normal (40-50)**
      - **SI Low (<40)**
      - **SI High (>50)**
  - **No**
    - 5-10% Ardsnet CVP/PCWP
      - **Pressor**
      - **Inotrope**
      - **Diuretic**

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CONCLUSION:
Arterial waveform analysis predicts an individual’s position on their Starling Curve

Volume Responsiveness then reflects basic physiology

The purpose of hemodynamic monitoring is to assess circulatory performance: Pinsky

• To determine if cardiac output is keeping up with tissue O₂ demand, and if not...

• To determine what components of the hemodynamic profile need to be adjusted to re-establish consumption-demand balance and achieve the optimal cardiac and mixed venous O₂ reserve
Final Simplified Physiologic Algorithm
SVV & SV; ScVO2 BP u/o ALI: ICU Version

Volume Responsive: SVV>13%

Yes

Fluid Infusion

No

SV Normal

SV Low

SV High

ScVO2 evaluate O2 extraction

?Pressor/Sepsis

?Inotrope/CHF

Re-evaluate O2 extraction, SVV & SV*

Diuretic ALI/ARDS

* Quickly decide if treatment is optimal

Physiologic Algorithm Utilizing SVV, SI, & ScVO2

Volume Responsive: SVV>13%

No

SI Normal

SI Low

ScVO2 evaluate O2 extraction*

?Pressor

?Inotrope/ Vasodilator

Re-evaluate DO2 O2 extraction, SVV, & SI

* is DO2 adequate?
**Complex $O_2$ Extraction PAC**

- $DO_2 = CO(CaO_2)$
- $VO_2 = CO(CaO_2 - CVRO_2)$
- $\frac{VO_2}{DO_2} = \frac{DO_2 - VRO_2}{DO_2}$
- $\frac{CO (CaO_2) - VR (CvO_2)}{CO (CaO_2)}$
- $\frac{CO (Hb SaO_2 1.36) - VR (Hb SvO_2 1.36)}{CO (Hb SaO2 1.36)}$
- $1 - \frac{SvO_2}{SaO_2}$

**Simple $O_2$ Extraction**

**CVC not PAC**

- $SaO_2 - ScvO_2$
- $100% - 70% = 30%$ cellular uptake
How to use O2 extraction: clinical problem exists

- No evidence or known benefit to increasing DO2 (may be harm) when extraction is normal or low. 33% or less; Organ failure and mortality may occur but unrelated to DO2 at this time (may have been prior deficit)
- When extraction is increased > 40% vigorous efforts to increase D02 should be employed and may improve outcome. Resuscitation paradigm: Intensivists, anesthesia, surgeons are expert!

Septic Shock, ATN, ALI: Resuscitation 5.5 hours – Volume, Pressors, and Antibiotics

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Measures of Flow and Perfusion

CO/SV and O2 extraction

ScvO2/Svo2

- Basic resuscitation is accomplished
- Hemodynamic therapy is optimized
- Status is continuously monitored
- Assures timely intervention
- Rare patient dies from a correctable DO2 problem

Move beyond questions of hemodynamics
CONCLUSIONS
Variability in SV during PPV is a better predictor than traditional measures of volume status that the patient will respond to a volume challenge.

SIMPLY
Greater variability: greater predicted response to volume challenge

Interestingly response is linear!