

Very few notes I'm afraid – during the opening morning session I was absorbed with presenting in the registrar's stream, and then spent the whole afternoon in various the airway workshops.

Between the two I attended these two sessions:

**Clinical haemodynamic monitoring – Ivan Joubert – Department of Critical Care, University of Cape Town & Groote Schuur Hospital**

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- The numbers themselves are not the target!
- Goal is perfusion – harmony at a cellular level
- What is shock? Physiologic -> inadequate tissue perfusion
- The tools we traditionally use:
  - ECG – rate and rhythm
  - BP – a surrogate for perfusion
  - Urine output
- These are blunt tools! More confounds the issue:
  - Relationships are almost never linear!
  - Resuscitating never gets us back to the starting point – to get the numbers the same we tend to over-resuscitate
- We need better end-points!
- FIRST trial used as a discussion point – what resus endpoints to use?
- HR, CVP and MAP did not correlate well with improvements in lactate.
- The problem with CVP...
  - Has been used as a “Gold Standard” for optimising fluid volume
  - CVP is a function of posture, ventricular compliance, systemic venous tone, intrathoracic pressure and fluid volume.
  - Many pitfalls in interpreting CVP
  - Data suggests it is not reliable in the critically ill NOR is it reliable in healthy patients!
  - PCWP suffers the same drawbacks
  - See Kumar et al Crit Care Med 2004;32:691-699 for study of CVP in healthy patients
  - LVEDV/RVEDV/stroke volume index – NONE of these had a correlation with CVP before and after fluid loading.
  - CVP in critically ill – see Lichtwarck-Aschoff et al, Intensive Care Med 1992
  - The use of monitoring changes in CVP in response to fluid challenges was proposed initially based on tenets of cardiac physiology, but has never been proven through study
  - See Chest 2008 – Does CVP predict fluid responsiveness – A Systematic Review and A Tale of Seven Mares
  - The receiver-operator curve for CVP/fluid responsiveness follows the line of equal probability.
- What are good goals?
  - Good perfusion is the ultimate goal
  - Warm peripheries (toes and nose)

- Clearance of lactate
- Improvement in the blood gas (pH and standardised base excess)
- Pulse oximeter
  - What is the plethysmograph? It's a volume/flow change detector!
  - Why look at the trace? It tells us about peripheral capillary perfusion!
  - Use of ventilation-induced plethysmographic variations to optimise patient fluid status (See Desebbe & Cannesson, Curr Op Anaes 2008 21:722-778)
  - Monnet & Teboul Crit Care 2005 – Do we have our finger on the solution?
- The big focus?
  - Dynamic indicators such as...
  - Systolic pressure variation
  - Pulse pressure variation
  - Etc.

### **Xavier Monnet – Paris Bisect Hospital**

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- Starling is a curve, not linear – we cannot expect a linear response. There are many Starling curves depending on the patient's age, condition and pathology.
- Giving too much fluid to patients increases mortality (see ARDSnet study!)
- How do we predict fluid responsiveness?
  - CVP has been disproven
  - Responders and no-responders cannot be separated by CVP value (See Osman 2007)
  - Repetitive fluid challenges can be used, but over time this results in fluid overload
  - Respiratory variation of pulse pressure does not require fluid administration, and has been shown by multiple studies to be an effective measure
  - PP variation does have important limitations:
    - Can't be used if the patient has arrhythmias
    - Can't be used if the patient has spontaneous breathing efforts
    - Can't be used in ARDS/low VT/poor compliance
  - Only a small portion of ICU patients are suitable for PPV testing
  - End-expiratory occlusion (EEO) allows increased venous return, and can thus predict fluid responsiveness
  - This can be demonstrated very easily if the patient is on a cardiac output monitor, and works even better if patients are on high PEEP (such as ARDS)
  - The passive leg raise (PLR) test also predicts fluid responsiveness well without risk of fluid overload. 10% increase in cardiac output is important; using arterial pressure results in false negatives.