“Surviving the CCU”

CHIA YEW WOON
MBBS, MMed (Int Med), MRCP(UK), EDIC, DDU(Critical Care), FAMS (Cardiology), FRCP Edin,
Diplomate, National Board of Echocardiography
Cardiologist & Intensivist
• Contemporary Cardiac Intensive Care Unit (CICU)
  – No longer just a coronary monitoring unit post MI
  – Complex cardiovascular patients
  – Who are critically ill
  – Often have non-cardiovascular critical illnesses accompanying or complicating primary cardiac presentation
    • Respiratory failure after APO requiring intubation and invasive mechanical ventilation
    • Acute kidney injury after cardiogenic shock requiring RRT
    • Hypoxic brain damage after cardiac arrest requiring TTM
  – Complications seen in ‘typical’ ICU patients e.g. VAP and CRBSI
Background

• Critically ill patients in other ICUs
  – Underlying chronic IHD and LV dysfunction
  – May develop cardiac complications
    • New onset tachyarrhythmias e.g. AF
    • New onset LV dysfunction e.g. septic cardiomyopathy
    • Type 2 MI

• Critically ill patients share great similarities
  – Critically ill cardiac patients in the CICU or
  – Critically ill patients in other ICUs with cardiac complications
“Surviving the CCU” – using “CCUS”
Critical Care Ultrasonography

- Can be done at bedside (*no need to transport a critically ill patient to radiology suite*)
- Rapid interpretation (*can be interpreted by the bedside clinician, do not need to wait for ‘radiology’ reports*)
- No ionising radiation (*unlike x-rays*), no iodinated contrast (*unlike CT scans*)
- Can be repeated serially as condition evolves
- Helps in procedures – higher first pass success and shorter time needed
Level 1 Critical Care Echocardiography

- Using 5 views, 2 modes to answer 4 questions
  - 5 views
  - Parasternal window: long axis / short axis
  - Apical window: 4 chamber / 2 chamber
  - Subcostal window: 4 chamber
Level 1 Critical Care Echocardiography

- Using 5 views, 2 modes to answer 4 questions
  - 2 modes
    - 2-dimensional
    - M-mode
  - 4 questions
    - What is the LV doing?
    - What is the RV doing?
    - What is the fluid status?
    - Is there pericardial effusion?
Level 1 Critical Care Echocardiography

• Use above skills to assess a hypotensive patient in the ICU
  – Hypovolaemia
  – Septic
  – Cardiogenic
  – Obstructive
2-Dimensional Echocardiography

Parasternal long axis view
2-Dimensional Echocardiography

Parasternal short axis view
2-Dimensional Echocardiography

Apical 4-chamber view
2-Dimensional Echocardiography

Apical 2-chamber view
1. What is the LV doing?
1. What is the LV doing?
1. What is the LV doing?
1. What is the LV doing?
1. What is the LV doing?
1. What is the LV doing?

- Hypotension with a dilated LV with severe LV systolic dysfunction
  - **Preload**: careful in fluid resuscitation – check for preload responsiveness and evidence of pulmonary edema first
  - **Contractility**: consider adding low dose inotropes to augment contractility
  - **Afterload**: careful in using high dose vasopressors which may be causing high LV afterload and impeding stroke volume in a poorly contractile LV
2. What is the RV doing?
2. What is the RV doing?
2. What is the RV doing?
2. What is the RV doing?
2. What is the RV doing?

- Hypotension with a dilated RV with severe RV systolic dysfunction and evidence of RV volume and pressure overload
  - **Preload**: stop fluid resuscitation – flattening of interventricular septum impeding LV diastolic filling
  - **Contractility**: consider adding low dose inotropes to augment RV contractility
  - **Afterload**: Careful in using vasopressors which may contribute to high RV afterload. Lower the PEEP and inflation pressure. Consider adding pulmonary vasodilators.
3. What is the fluid status?
3. What is the fluid status?

IVC distensibility index = \frac{(IVC_{\text{max}} - IVC_{\text{min}})}{IVC_{\text{min}}}

≥ 18% has 90% sensitivity, 90% specificity for predicting a fluid responder
4. Is there pericardial effusion?
4. Is there pericardial effusion?
4. Is there pericardial effusion?
4. Is there pericardial effusion?
4. Is there pericardial effusion?
Assessing Hypotension in ICU
Assessing Hypotension in ICU

- Using 5 views, 2 modes to answer 4 questions to assess a hypotensive patient in the ICU
  - **Hypovolaemia** → small LV cavity, usually hyperdynamic LV systolic function and IVC distensibility index > 18%
  - **Septic** → small LV cavity, usually hyperdynamic LV systolic function and IVC distensibility index > 18%
  - **Cardiogenic** → poor LV systolic function
  - **Obstructive**
    - pericardial tamponade: pericardial effusion (may not be big) with RA/RV collapse
    - pulmonary embolism: dilated RV and RV systolic dysfunction (volume +/- pressure overloaded RV shifting interventricular septum towards LV, impeding LV filling)
Ultrasound Guided Haemodynamics

• For patients who are in shock, use ‘ultrasound guided haemodynamics’
  – Using echo techniques to assess and optimise preload, contractility and afterload
Ultrasound Guided Haemodynamics

• Preload vs Preload Responsiveness
  – Important to predict preload responsiveness BEFORE giving fluid boluses as giving fluids to a patient who is not preload responsive is unlikely to improve the stroke volume/cardiac output and can worsen gas exchange
  – Static markers of preload such as CVP and PCWP SHOULD NOT be used to predict preload responsiveness
Does the Central Venous Pressure Predict Fluid Responsiveness? An Updated Meta-Analysis and a Plea for Some Common Sense

Paul E. Marik, MD, FCCM; Rodrigo Cavallazzi, MD

Background: Despite a previous meta-analysis that concluded that central venous pressure should not be used to make clinical decisions regarding fluid management, central venous pressure continues to be recommended for this purpose.

Aim: To perform an updated meta-analysis incorporating recent studies that investigated indices predictive of fluid responsiveness. A priori subgroup analysis was planned according to the location where the study was performed (ICU or operating room).

Data Sources: MEDLINE, EMBASE, Cochrane Register of Controlled Trials, and citation review of relevant primary and review articles.

Study Selection: Clinical trials that reported the correlation coefficient or area under the receiver operating characteristic curve (AUC) between the central venous pressure and change 0.56 (95% CI, 0.54–0.58) for those done in the operating room. The summary correlation coefficient between the baseline central venous pressure and change in stroke volume index/cardiac index 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.

Conclusions: There are no data to support the widespread practice of using central venous pressure to guide fluid therapy. This approach to fluid resuscitation should be abandoned. (Crit Care Med 2013; 41:1774–1781)

Key Words: central venous pressure; fluid challenge; hemodynamic monitoring; meta-analysis; volume responsive
A fluid responder is someone who will increase stroke volume/cardiac output by $\geq 15\%$ after a 500 mls fluid bolus.
Ultrasound Guided Haemodynamics

• Preload vs Preload Responsiveness
  – Use *dynamic markers* such as those based
    • *Respirophasic variations* (*need to fulfill certain pre-conditions*) or
    • *Passive leg raising*
Ultrasound Guided Haemodynamics

• Using respirophasic variations
  – Examples: IVC distensibility/variability index, stroke volume variation, pulse pressure variation
  – Preconditions: passive on MV, $V_T$ 8 mls/kg PBW, no significant arrhythmias or RV dysfunction
Ultrasound Guided Haemodynamics

• Calculating stroke volume using echo
  – Stroke volume = LVOT area x LVOT stroke distance
Ultrasound Guided Haemodynamics

- Using **passive leg raising**
  - Note the change in *stroke volume* or *LVOT VTI*
  - Not change in *MAP!*

\[ \uparrow LVOT \text{ VTI} \geq 12.5\% \text{ has 77\% sensitivity, 100\% specificity for predicting a fluid responder} \]
Ultrasound Guided Haemodynamics

- Even after you have determined that patient is likely fluid responsive, you have to check whether there are contraindications to fluid boluses e.g. in acute pulmonary edema, before administering fluids.
Patient in Shock e.g. ↓mentation / ↓urine output / ↑lactate / ↓ScvO₂

- Passive on mechanical ventilation
  - Tidal volume ≥ 8 mls/kg PBW
  - No significant arrhythmias
  - No significant RV dysfunction

- Measure IVC Distensibility Index /
  Stroke Volume Variation /
  Pulse Pressure Variation

 Fluid Responsive?

- Fluid Responsive?
  - yes
  - yes
  - yes
  - no

- no
  - Isotonic fluids 250 - 500 mls over 15 - 30 mins and reassess

- no
  - Any evidence of pulmonary edema?
    - e.g. ≥ 3 anterior B lines bilaterally on Lung US

- yes

Start/Increase Inotropes/Vasopressors

CYW 2015
A Patient Yesterday

- MAP 72 mmHg, HR 100/min
  - on Dobutamine 3 mcg/kg/min and NorA 0.4 mcg/kg/min

**IVC distensibility index** = \( \frac{(1.83 - 1.58)}{1.58} \times 100 = 16\% \)
LVOT diameter = 1.86 cm
LVOT area = 2.72 cm$^2$

LVOT VTI = 7.47 cm

Stroke Volume = LVOT area × LVOT VTI = 20.3 cm$^3$ = 20.3 mls

Cardiac Output = Stroke Volume × Heart Rate = 2.0 L/min

Cardiac Index = 1.48 L/min/m$^2$
A Patient Yesterday

**Review - ICU Team**

- **Echo Haemodynamics**
  - MAP 72 mmHg
  - HR 100/min on Dobutamine 3 mcg/kg/min and Noradrenaline 0.4 mcg/kg/min
  - Preload
    - CVP 12 mmHg
    - Preload responsiveness: 
      \[
      \frac{1.83 - 1.58}{1.58} = 16\% 
      \]
      suggesting that patient is not likely to be a fluid responder

- **Contractility**
  - LVOT 1.86 cm, so LVOT area = 2.71 cm$^2$
  - LVOT VTI 7.47 cm, so Stroke Volume = 20.2 cm$^3$ = 20.2 mls
  - HR 100/min, so Cardiac Output = 2024 mls/min = 2.0 L/min
  - BSA 1.37 m$^2$, Cardiac Index 1.48 L/min/m$^2$

- **Afterload**
  - SVRI = (72-12)/1.48 x 80 = 3234

**Plan**

1. Decrease Noradrenaline in 0.02 mcg decrements
2. Keep Dobutamine at 3 mcg/kg/min

Dr Chia Yew Woon
Cardiologist & Intensivist

CIS-ICU-34-00
Summary

- Critically ill patients share great similarities regardless of which unit they are managed in.
- The use of critical care ultrasound can help to assess and optimise the haemodynamics of a critically ill patient.
- It is easy to learn and a skill that all physicians working in a critical care environment should attempt to acquire.