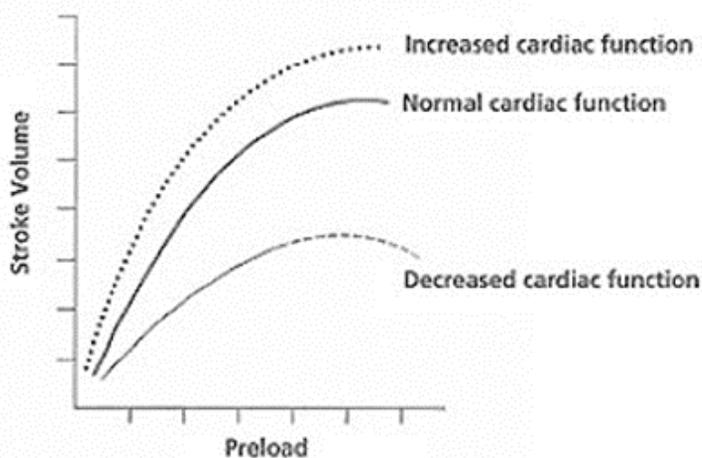


# Cardiac Output Monitoring

By David Cain, Senior ICM trainee, July 2016

- Prescription of fluid therapy should be guided by the patient's history, clinical examination and clinical measurements – such as cardiac output - which are carefully interpreted in the context of the clinician's previous experiences with similar cases.
- Cardiac output is an attractive marker to guide fluid therapy because oxygen delivery to the body is dependent upon cardiac output (as well as haemoglobin content and saturations). Therefore, by optimising cardiac output the clinician may optimise organ oxygen delivery.
- Standard measurements of cardiac function (heart rate, blood pressure) do not measure blood flow.
- Although blood pressure is not a marker of cardiac output, a sufficient blood pressure is required to push blood across tissue beds. In general, this mean blood pressure (MAP) is believed to be 60-65mmHg. If this blood pressure cannot be achieved with fluid loading then a vasopressor (noradrenaline, vasopressin) may be used.
- The heart is a demand pump. The body increases cardiac output through mobilisation of venous fluid reserves. Cardiac output subsequently increases through a combination of distension-driven contractility (Moving along the normal cardiac function line in Fig 1) and heart rate).



*Fig 1. Starling curve*

*The relationship between preload and cardiac output. Central venous pressure (CVP) is the most commonly used marker of cardiac preload.*

- A fluid challenge administered through a venous cannula has the same effect. Repetitive fluid challenges will ultimately lead to a state of maximal cardiac output, where cardiac muscle and heart rate cannot deliver any further increase in cardiac output. At this point an inotrope (dobutamine) may increase cardiac output further, meaning a greater amount of blood is pumped for a given preload (shift to the increased cardiac function curve in Fig 1)

## **General notes concerning cardiac output monitoring**

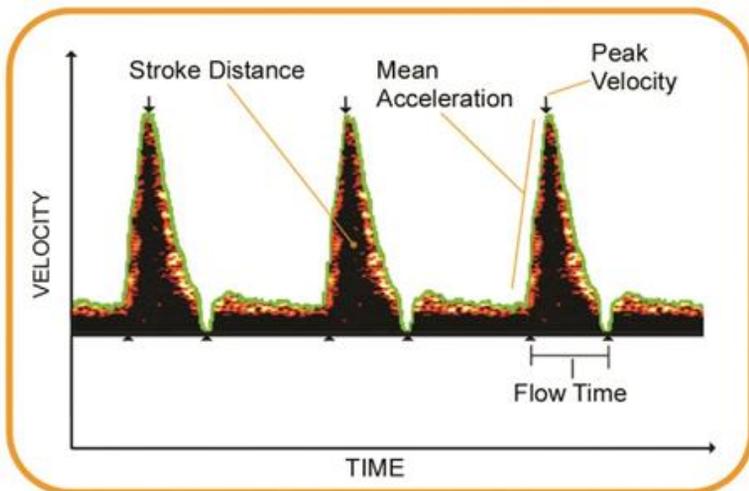
- If a fluid challenge were administered to you now, your cardiac output would increase. This does not mean that a fluid challenge was necessary. Therefore, the assessment of cardiac output should begin with an assessment of whether the body needs a greater cardiac output – i.e. Tissue perfusion or function!
- Measures of organ perfusion include capillary refill, warm/cold levels in limbs and urine output, underlining the importance of clinical assessment.
- Venous lactate and central venous oxygen saturations are markers of global oxygen delivery.
- While low cardiac output states may limit organ function and cause harm, excessive amounts of fluid will be similarly detrimental, e.g. leaking into the lungs and exacerbating lung injury.
- The timing of fluid therapy is important. It is generally agreed that patients need most fluid during the early stages of critical illness, and that some of this fluid may have to be removed as they recover.
- Fluid therapy should therefore be individualised on a daily – or even hourly – basis.

## **The evidence**

- When interpreting the evidence surrounding cardiac output monitors it is important to understand that these studies do not simply test a monitor in isolation, rather they test a specific protocol within a specific institution. Many factors beyond the monitor may be responsible for the study findings.
- As with many areas of ICU research, early studies suggested promise which larger better conducted studies have failed to replicate.
- It is reasonable to say that non-specific use of supramaximal cardiac output (with frequent, high doses of inotropes) within the ICU kills patients. The patient group which probably benefits the most are those with early sepsis.

## The Oesophageal Doppler Monitor

- A doppler probe is passed through the mouth or nose, until the tip lies in the mid oesophagus. The probe is manipulated until an optimal signal is obtained from the adjacent descending aorta (Fig 2)
- The probe measures aortic red cell velocity and the period of flow (flow time). The device calculates continuous stroke volume by multiplying the mean blood speed with an estimate of aortic cross sectional area (derived from patients age and sex), plus a correction factor for unmeasured ascending aortic blood flow.
- Corrected flow time (FTc) is the time spent in systole for a normalised heart rate (60bpm). Values less than 320ms typically indicate hypovolaemia or high systemic vascular resistance, while values over 360ms suggest the opposite (Table 1)
- Users may enter values for CVP and MAP to estimate systemic vascular resistance (SVR), thereby differentiating causes of low FTc. Peak velocity provides an index of contractility/inotropy.



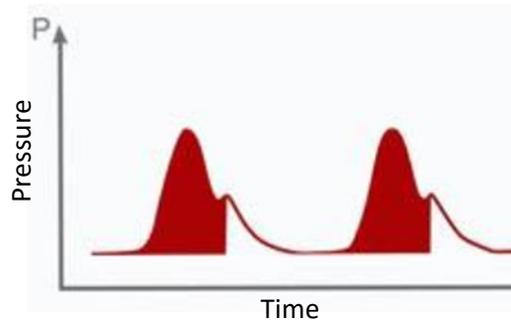
*Fig 2. The Oesophageal Doppler Monitor (Cain D, Edwards M, Hamilton M. (2014) Intraoperative fluid balance: what to measure. In: Challenging Concepts in Anaesthesia. Oxford pp29-43)*

State	Peak Velocity	Flow Time
Preload Reduction	-	↓
Preload Increase	-	↑
Afterload Increase	↓	↓
Afterload Decrease	↑	↑
Positive Inotropy	↑	-
Myocardial Depression	↓	-

*Table 1. Interpretation of Oesophageal Doppler Measurements*

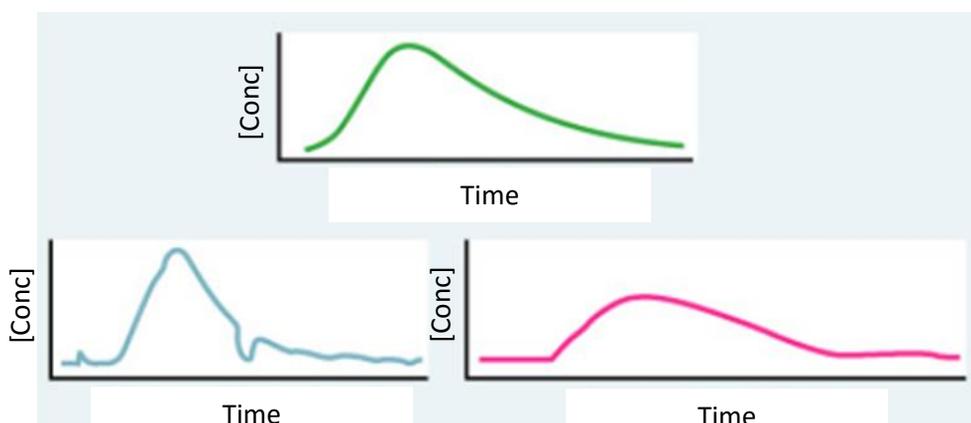
## Pulse contour analysis (LIDCO).

- Pulse contour analysis devices such as LIDCO analyse the arterial line waveform to estimate stroke volume. (Fig 3).
- The arterial line may be sited in radial artery.
- Again, CVP and MAP values may be used to calculate SVR



*Fig 3. Calculation of stroke volume from arterial line waveform*

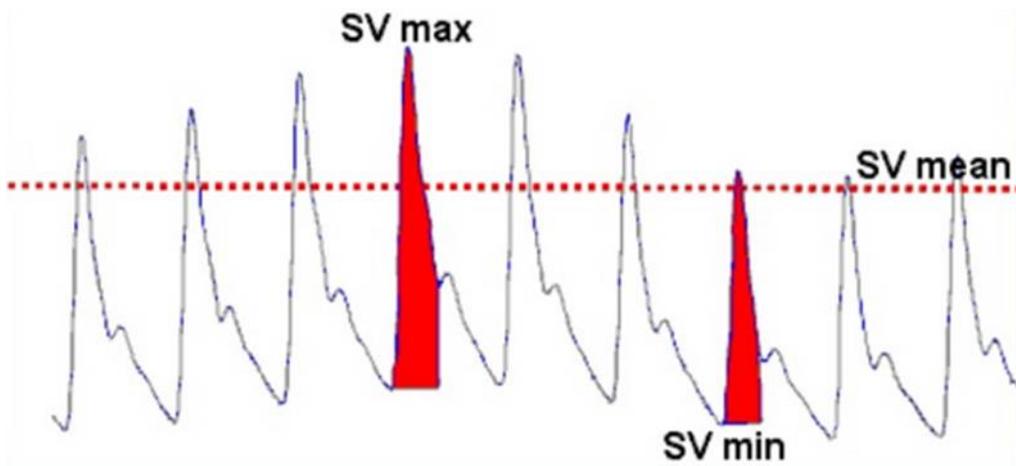
- A daily calibration step must be performed. Without the calibration step the trends in stroke volume may be used, but the absolute values will be more unreliable. The Lidco Rapid does not use a dilution calibration step.
- LIDCO uses the injection of a known amount of lithium through a peripheral venous cannula. A lithium detector then records the concentration of lithium arriving at the arterial line (Fig 4)



*Fig 4. Indicator dilution calculation of cardiac output  
Green = normal, Blue = high, Pink = low.*

## Respiratory variation / swing

- Recent versions of the LIDCO and ODM have additional software to estimate the effect of respiratory variation upon cardiac output or swing (Fig 5)
- As thoracic pressure changes with respiration, so will the volume of blood entering the left atrium, analogous to mini fluid challenges.
- Formal interpretation of swing requires pressure/volume controlled ventilation, so that the fluid challenge is consistent.
- Typically, a variation of <13% is considered to equate to loss of fluid responsiveness.



*Figure 5. Arterial line trace demonstrating respiratory swing*

## **Trend analysis versus absolute numbers**

- The underlying principles of each cardiac output monitor must be understood, otherwise the data they provide may not be interpreted correctly.
- For example, aortic regurgitation and arterial wall diseases such as aneurysms will clearly affect the readings.
- For these reasons the trends in response to challenges (dynamic measurements) are often considered to be more important than the static values which the machines record.