Critical assessment of haemodynamic data

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It is important to recognize that established forms of cardiovascular monitoring such as invasive pressures, thermodilution for cardiac output and cardiac ultrasound techniques suffer from measurement error, which is often unquantifiable at the bedside. These errors may be systematic (such as a fixed offset) or random; they have the potential to mislead the clinician particularly under extreme physiological conditions.

Loading conditions of the heart

The traditional concepts of preload and afterload are useful but the terms are often loosely applied. Suitable definitions would be:

Preload = the force applied to the myocyte before it contracts.

Afterload = the load the myocyte has to move during muscle shortening.

Loading conditions for individual myocytes vary with their position in the myocardium. Unfortunately, the loading conditions of the intact ventricle are difficult to determine. Therefore, assessment of ventricular load involves compromise. The best approximation for ventricular wall stress uses the Law of Laplace, modified for thick walled structures. This is expressed as Lamé’s equation:

\[ \sigma \propto (P_1 - P_2) \times R/w \]  

\( \sigma \) = wall stress; \( P_1 \) = intraventricular pressure; \( P_2 \) = extraventricular pressure; \( R \) = ventricular radius; \( w \) = wall thickness.

\( P_1 \) for each ventricle can be estimated from central venous pressure (CVP) or pulmonary artery occlusion pressure (PAOP). \( R \) and \( w \) can be estimated from M-mode echocardiography.

Using this equation, preload and afterload can be predicted from measures of circumferential wall stress using data from M-mode echocardiography and invasive pressure monitoring. The calculation is impractical as a clinical tool but demonstrates the importance of positive end-expiratory pressure (PEEP), which influences, but is not equal to, \( P_2 \).

Arterial blood pressure

Oscillometry is the principle behind the non-invasive blood pressure (NIBP) systems used routinely during anaesthesia. It is well known that many factors affect accuracy. The most accurate value measured by NIBP is the mean arterial pressure (MAP); the diastolic pressure is the least accurate.

Invasive blood pressure (IBP) monitoring using an indwelling arterial catheter is a true reflection of systolic and diastolic pressure, assuming optimal damping. The MAP is calculated from the area under the curve. In health, there are only small differences in MAP measured at various cannulation sites. In contrast, during vasopressor infusion MAP is significantly lower in the radial artery when compared with large central arteries. By changing cannulation site to the femoral artery, Dorman and colleagues were able to reduce norepinephrine infusion rate by 30%.

Central venous pressure

In the absence of tricuspid valve disease, CVP correlates well with right ventricular end-diastolic pressure (RVEDP) but poorly with right ventricular end-diastolic volume (RVEDV). It does not correlate with left ventricular end-diastolic volume or left ventricular preload.

CVP is the best predictor of cardiac output deterioration resulting from increased intrathoracic pressure during mechanical ventilation. A high CVP is protective.

The mean CVP approximates to end-expiratory RVEDP. Conventionally, a patient monitor displays the mean estimated from the area under the curve of the CVP trace. This has the effect of compensating for minor fluctuations caused by respiratory artefact. There is no compensation for larger respiratory swings that result in inaccuracies in the digital display. Tricuspid regurgitation, which is common in the ventilated patient, also generates a discrepancy between the digital display and the end-diastolic, end-expiratory pressure.
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Pulmonary artery catheter data

Pulmonary artery occlusion pressure

PAOP is considered to be an indirect measure of left atrial pressure providing there is an uninterrupted measurement path between the catheter tip and the left atrium. Lack of valves in the pulmonary veins means this is a valid assumption as long as vessel collapse is prevented.

Venous collapse occurs causing artefact if alveolar pressure exceeds venous pressure (West zones I and II). A lateral chest x-ray may confirm that the catheter tip is below the left atrium but cannot identify a physiological region in which venous collapse does not occur (West zone III). Correct position is indicated if the maximal change in PAOP during the respiratory cycle is less than half the airway pressure change (peak airway pressure—PEEP) and any increment in PAOP associated with a rise in PEEP is less than half the increase. West zones are not static; zones I and II are increased by rises in airway pressures and hypovolaemia.

Lamé’s equation shows that preload is dependant upon transmural pressure gradient. Transmission of PEEP through the pericardium will vary between individuals and with lung parenchymal disease. Clinical studies have shown that, as PEEP rises, PAOP and LVEDV diverge, resulting in the recognition that under these circumstances PAOP is a poor measure of preload. P2 is not usually measured, but in normal subjects can be assumed to be insignificant. When it is appreciably greater than zero, the isolated measurement of chamber pressure (P1) does not yield useful information. When P2 is assessed experimentally using surgically placed transducers, the transmural gradient is found to closely correlate to LVEDV. The problem interpreting PAOP data is not because they are a bad index of LVEDP but because they are a bad index of transmural pressure. This makes PAOP data a questionable measure of preload with high levels of PEEP.

Pulmonary capillary pressure

Measurement of pulmonary capillary pressure (Ppc) is difficult. It can be estimated using a PAC by identification of the instant of balloon occlusion using a high fidelity transducer. Occlusion must occur during diastole and be associated with minimal artefact during the first 200 ms. Given these limitations, accuracy has never been proven with a standard fluid-filled catheter. Ppc will give an estimate of LVEDP and propensity to cardiogenic pulmonary oedema formation. It is probably more accurate than PAOP in states of high pulmonary vascular resistance. Unfortunately, there are methodological problems associated with measurement of Ppc.

Cardiac output

The thermodilution technique has been a standard way of assessing cardiac output. There is no agreement between studies that have compared thermodilution with methods using the Fick principle or ultrasound indices.

Data derived from the thermodilution method vary depending upon the phase of the respiratory cycle in which they are measured. Estimations made at random during the respiratory cycle result in a coefficient of variation 2.5-times greater than if all estimations were made by bolus injections during the same phase of respiration. Reproducibility is improved further if bolus injections are made during a 15 s apnoea. This error is additional to error intrinsic to the method. Derived data such as systemic vascular resistance are particularly vulnerable to this phenomenon.

Some degree of tricuspid regurgitation is seen in nearly all critically ill, ventilated patients and significant in approximately half. Data from thermodilution are invalidated by significant tricuspid regurgitation.

Stroke work index

In this context, an indexed value is one that is related to patient size. It is determined by dividing the value by the body surface area. Body surface area is calculated using body weight and height; nomograms are available to simplify this.

The stroke work index (SWI) is used as a measure of myocardial oxygen consumption, myocardial contractility or work done by the ventricle. It is worth considering in detail as it is a difficult derivative to apply accurately. It is used widely in the literature.

SWI, for each ventricle, is calculated as the product of stroke volume index (SVI) and MAP (or mean pulmonary artery pressure for the right heart). Work done by the ventricle may be considered as external (work done to eject blood) and work done against the viscoelastic properties of the myocardium, considered internal. External work is further divided into stroke work (work done ejecting blood against aortic pressure) and work done imparting kinetic energy to ejected blood.

Kinetic energy normally accounts for <5% of external work, increasing to 10% with exercise, but may become significant in high output states with low MAP such as sepsis. It is usually ignored in the assessment of external work but can be calculated using Doppler echocardiography to measure velocity in the aortic root (see Appendix).

These values can be assessed using pressure–volume loops (Fig. 1). The sum of external work (area A) and internal work (area B) is the pressure–volume area (PVA). The relevance of PVA is that it is proportional to myocardial oxygen consumption (MVO2). Changes to inotropic and metabolic states alter the PVA–MVO2 relationship. This is clinically important. Increased cardiac work resulting from an increased inotropic state is associated with a disproportionate rise in MVO2.

Figure 2 depicts two single loops from different hearts (Fig. 1). The sum of external work (area A) and internal work (area B) is the pressure–volume area (PVA). The relevance of PVA is that it is proportional to myocardial oxygen consumption (MVO2). Changes to inotropic and metabolic states alter the PVA–MVO2 relationship. This is clinically important. Increased cardiac work resulting from an increased inotropic state is associated with a disproportionate rise in MVO2.

These examples demonstrate that in a poorly compliant ventricle with a low inotropic state, SWI does not represent external...
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Systemic vascular resistance/aortic impedance

Systemic vascular resistance (SVR) is calculated from three measured variables, each with its own error. This results in a cumulative error that is difficult to estimate. Resistance is an impediment to constant flow; impedance is impediment to changing flow.

In the cardiovascular system, oscillatory flow is superimposed on a non-pulsatile component. Using resistance as a measure of impediment to flow across the entire cardiovascular system is mathematically incorrect. Impedance is directly proportional to blood density and the elastic properties of the arterial tree and inversely proportional to the second power of the radius. In comparison, resistance is proportional to blood viscosity and length of the arterial tree and inversely proportional to the fourth power of the radius. Though the dominant factor in both relationships, radius may not be as significant as suggested by the Hagen–Poiseuille equation alone. Practically, impedance is so difficult to measure that, despite inaccuracies, SVR remains the most usable approximation of the total opposition to flow.

Critical assessment of the calculated value for SVR is not the only problem. Consideration must also be given to the implication of the vascular resistance measurement. SVR is often used clinically as a measure of afterload. Under experimental conditions, changes in SVR significantly underestimate changes in afterload, as assessed by wall stress analysis (calculated from Lamé’s
Oesophageal Doppler monitoring

Oesophageal Doppler monitoring (ODM) uses Doppler ultrasound to measure blood velocity based on the Doppler shift of blood cells moving away from the transducer in the descending aorta. Haemodynamic variables can be derived from the shape of the resulting velocity curve, a plot of red cell velocities against time. It does not measure flow. Estimates of flow can be derived from velocity waveforms if the cross-sectional area of the aorta is known. Most commercially available monitors use nomograms to calculate the aortic cross-sectional area based on the patients height and weight. Newer models include the capability to measure aortic diameter directly.

The reliability of ODM is probably similar to alternative forms of cardiac output monitoring. Accuracy is dependent on a number of assumptions that are potential sources of error.

The oesophagus and aorta are assumed to be parallel. From the Doppler equation (Appendix), the calculated velocity depends on the inverse of the cosine of the angle between the ultrasound beam and direction of blood flow. Measurements are most accurate if the beam is directed along the path of flow \([\cos(0^\circ) = 1]\). If the beam is at right angles to flow no useful information is acquired \([\cos(90^\circ) = 0]\). Most probes use a fixed angle of 45°. If the aorta is unfolded with a 15° angle between the oesophagus and aortic flow, the velocity may be underestimated by ~20% or overestimated by ~40% depending on the direction of the aorta. Angulation of the aorta is common and occasionally extreme. The incidence and variance are unknown.

The aortic cross-sectional area (CSA) is assumed to be circular, accurately described by a nomogram for height and weight, and fixed. Although the diameter of the aortic root remains constant during extremes of blood pressure, this is not true of the descending aorta. CSA may decrease up to 31% of normotensive values during extreme hypotension in patients with known atherosclerotic disease (those undergoing coronary artery bypass grafting). In healthy sheep this relationship continues with hypertension causing an increase in CSA.

Flow is assumed to be laminar. If this is true the Doppler waveform shows a clear envelope because most red cells are moving at a similar velocity with few points appearing at lower velocities. Elderly patients and those with atherosclerotic disease frequently demonstrate turbulent aortic flow. In these cases cells are moving at different velocities and the centre of the velocity profile would be full. Turbulent flow renders the algorithms used to generate haemodynamic data inaccurate. Under these circumstances, even trends to the ODM derived data may be incorrect.

Because of these limitations, absolute values and trends in ODM data must be interpreted cautiously.

Conclusion

It is important to be aware of the principles used when monitoring haemodynamic parameters. Many of the techniques are methodologically and technically imperfect. There are little in the way of reference standards suitable for use in unstable patients. Validation of monitoring techniques is difficult and often performed in animal models or patients with incomparable pathophysiology. Consequently, accuracy at physiological extremes is poorly assessed. The degree of inaccuracy in an individual cannot be quantified. Confidence intervals have never been calculated for any cardiac output monitor. It is not possible to say how much a particular parameter must change before a real physiological change has occurred. Failure to review data critically increases the possibility of instituting the wrong haemodynamic intervention.

Despite all these imperfections, haemodynamic monitors remain valuable tools as long as the data they provide are interpreted critically.

Appendix

1. Kinetic energy:
   \[ E_k = \frac{m \cdot v^2}{2} \]
   \( m = \) mass, estimated from stroke volume; \( v = \) mean velocity in aortic root.

2. Hagen–Poiseuille equation:
   \[ R = 8 \cdot \eta \cdot L/\pi \cdot r^4 \]
   \( R = \) resistance; \( \eta \) (eta) = viscosity; \( L = \) length; \( r = \) radius.

3. Vascular impedance:
   \[ Z = \rho \cdot C/\pi \cdot r^2 \]
   \( Z = \) impedance; \( \rho \) (rho) = density; \( C = \) wave velocity (related to arterial elastance); \( r = \) radius.
4. Doppler equation:

\[ v = f_d \cdot c / 2f_t \cdot \cos(\theta) \]

\( v \) = velocity; \( c \) = speed of sound; \( f_t \) = transmitted ultrasound frequency; \( f_d \) = Doppler shift; \( \theta \) = angle between ultrasound beam and direction of flow.

References


10. Hillel Z, Zane E, Thys D. Systemic arterial blood pressure influences the cross-sectional area of the descending thoracic aorta. Anesthesiology 1988; 69: 11A

See multiple choice questions 66–69.