Advanced cardiovascular monitoring

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Abstract

Advanced cardiovascular monitoring has a role in the assessment and management of both haemodynamically unstable critically ill and high-risk perioperative patients. Clinical indicators and standard monitoring may be unreliable or insufficient in these patients. This article reviews basic cardiovascular physiology, goal directed haemodynamic therapy and advanced cardiovascular monitoring with evidence and recommendations. Specific monitoring modalities will be described including invasive pressure monitoring, arterial waveform analysis and cardiac output estimation by dilutional, Doppler and echocardiography methods. Emerging technologies will also be discussed.

Keywords Arterial waveform analysis; cardiac output; echocardiography; enhanced recovery; goal directed haemodynamic therapy; invasive pressure monitoring; LiDCO; oesophageal Doppler; PiCCO

Introduction

Perioperative complications can lead to death, prolonged postoperative stay and long-term morbidity. They can arise from perioperative tissue hypoperfusion caused by hypovolaemia or myocardial insufficiency. There is increasing awareness of the need to manage this. Unfortunately this is not as straightforward as giving everyone generous amounts of intravenous fluids. A patient's cumulative fluid balance has been shown to affect their outcome. Whilst hypovolaemia can result in tissue hypoperfusion and shock, studies have shown that a positive fluid balance is associated with increased mortality in septic shock and increased morbidity following surgery.

Advanced cardiovascular monitoring provides more detailed information about circulatory status. This is often deranged in critically ill or high-risk surgical patients (usually defined as those with a >5% perioperative mortality rate). This information can help the clinician optimize the patient's intravascular volume and cardiac status by guiding the rational use of intravenous fluids and vasoactive drugs.

Cardiovascular physiology

In order to understand the role of advanced cardiovascular monitoring, we will first review some basic physiological principles (Box 1).

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Basic physiological relationships

Cardiac output (CO) in L/min equals stroke volume (SV) × heart rate (HR)

- $CO = SV \times HR$

Stroke volume (SV) is the product of the End Diastolic Volume (EDV) minus the End Systolic Volume (ESV). It depends on preload, contractility and afterload.

- $SV = EDV - ESV$

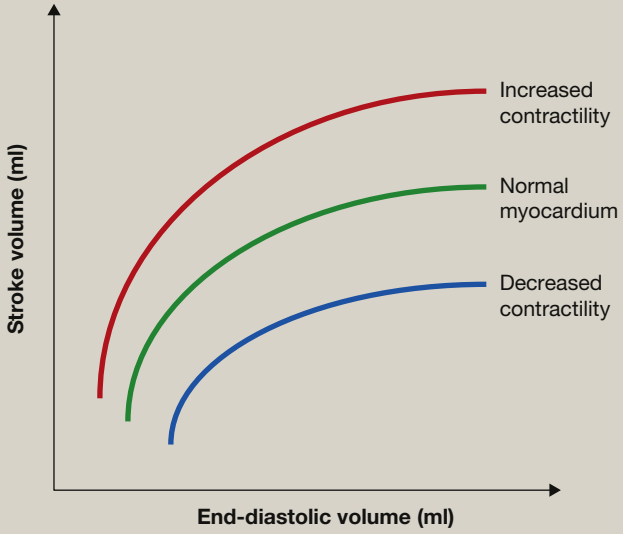
The Mean Arterial Pressure (MAP) equals CO × systemic vascular resistance (SVR)

- $MAP = CO \times SVR$

Box 1

The Frank–Starling law of the heart states that the stroke volume will increase proportionally with filling of the heart, or end-diastolic volume up to a maximum point. This relationship is shown in Figure 1. When the top of the curve is reached, further filling will not increase the stroke volume. In fact, further fluid could lead to reduced stroke volume in individuals with cardiac failure. When the plateau of the curve is reached, stroke volume may be increased further by increasing myocardial contractility with the use of inotropic drugs.

Frank–Starling curves



Franke–Starling curves, demonstrating the response of stroke volume to change in end-diastolic volume and differing states of cardiac contractility.

Figure 1

Fluid challenge

Haemodynamic instability can arise for many reasons. Poor cardiorespiratory reserve in the face of major surgery or in addition to factors such as hypovolaemia, sepsis or metabolic acidosis are among these. The expansion of intravascular volume with a so-called 'fluid challenge', is the appropriate first line intervention for most patients. Approximately 50% of haemodynamically unstable critically ill patients will respond to a fluid challenge. A baseline assessment of intravascular volume and the response to a fluid challenge can be made by examining skin turgor, capillary refill time, heart rate, blood pressure, jugular venous pressure, cognitive state and urine output. Unfortunately, these indicators can be unreliable and therefore advanced cardiovascular monitoring techniques have been developed to help assess circulatory status.

Goal directed haemodynamic therapy

Oxygen delivery (DO_2) to the tissues depends on cardiac output (CO) and arterial oxygen content (CaO_2) (Box 2). When DO_2 falls below a critical point, tissue hypoxia occurs. This is a key development preceding multi-organ failure and death.

Shoemaker and colleagues[1] demonstrated in 1988 that the perioperative use of a pulmonary artery catheter (PAC) to monitor and guide therapies aimed at increasing cardiac output and oxygen delivery in high-risk surgical patients reduced postoperative complications and length of hospital stay. The technique is termed goal directed haemodynamic therapy. Intravenous fluid and vasoactive drugs are given to achieve targeted variables on the cardiac output monitor that correlate with optimal preload, afterload and contractility. Since 1988, a number of safer and less invasive monitoring methods have largely replaced the PAC. The concept of goal directed therapy has been, and remains controversial. Goal directed *optimization* rather than goal directed *maximization* of cardiac output and oxygen delivery is more generally accepted at present.

The United Kingdom National Institute for Health and Care Excellence (NICE) estimates that 10% of all surgical procedures (more than 800,000 patients per year in the UK) are carried out in high-risk patients who they feel could benefit from goal-directed haemodynamic therapy.[2] Work from the National Confidential Enquiry into Patient Outcome and Death[3] suggests around 20,000 −25,000 deaths annually in the UK occur in hospital after a surgical procedure. Most of these occur in high-risk patients. Recommendations from this report include identification of the high-risk patient and subsequent perioperative optimization using cardiac output monitoring to guide fluid optimization. These topics are addressed in more detail elsewhere in this journal. The use of perioperative cardiac output monitoring in high-risk patients is increasing in the UK but is far from universal.

Oxygen delivery

$$DO_2 = CO \times C_aO_2 \times 10$$
$$C_aO_2 = (Hb \times S_aO_2 \times 1.34) + (P_aO_2 \times 0.0225)$$
$$DO_2 = HR \times SV \times [(Hb \times S_aO_2 \times 1.34) + (P_aO_2 \times 0.0225)] \times 10$$

Box 2

Enhanced recovery programmes

Enhanced recovery after surgery (ERAS) pathways are evidence based perioperative strategies aimed at improving recovery and shortening postoperative hospital stay. A priority is avoiding hypoperfusion. Intraoperative splanchnic hypoperfusion can delay the postoperative return of gut function and permit bacterial translocation across the gut wall causing sepsis. Unfortunately, seeking to avoid hypoperfusion by giving high total volumes of intravenous fluids or achieving a fluid balance more than 2 litres positive on the day of surgery, has been shown to predict poorer outcome. Because perfusion is difficult to assess with routine monitoring, the enhanced recovery partnership recommends advanced cardiovascular monitoring to guide intraoperative fluid therapy.[4] Aims for the clinical status of the patient by the end of surgery are shown in Box 3.

Invasive pressure monitoring

Arterial pressure monitoring

An arterial cannula provides continuous beat-to-beat measurement of arterial blood pressure and permits frequent sampling of arterial blood gases. This is indicated in the majority of critically ill and high-risk surgical patients. A plastic cannula is inserted into the artery. This is connected to a transducer via saline filled tubing which is continually flushed to prevent blockage. Arterial pressure is transmitted through the saline column and moves a diaphragm at the pressure transducer. This alters the resistance of a strain gauge, changing the current flow through a connected resistor. This change is then amplified and displayed as a measure of pressure (Figure 2a).

Interpretation of an arterial trace requires an understanding of sources of error in the waveform. A useful arterial trace must have an appropriate degree of damping to achieve a high degree of accuracy with minimal time lag in response to change. Excess damping will result in reduced size of the arterial waveform and altered values of systolic and diastolic pressure (Figure 2b). Common causes are kinking of the cannula or tubing, or air bubbles and blood clots within the fluid column. Pressure transducers can be affected by drift, where the displayed value changes over time despite the true value remaining constant. Drift is reset by calibration. This involves 'zeroing' the pressure transducer to atmospheric pressure at regular intervals.

Postoperative goals

Warm and well-perfused patient
No hypovolaemia
No coagulopathy
Hb >7 g/dl
'Zero fluid balance'
Minimum use of vasopressors

Box 3