Optimal intraoperative haemodynamic management

Major surgery in high-risk patients is associated with significant morbidity and mortality. This review focuses on how, with appropriate haemodynamic management, patient outcomes can be improved.

The aim of optimal intraoperative haemodynamic management during all types of surgery is to maintain stability, ensure adequate oxygen delivery and avoid inducing either myocardial ischaemia or pulmonary oedema.

The National Enquiry into Perioperative Deaths (Campling et al, 1993) has found that the estimated risk of death within 30 days of any surgical procedure is between 0.7 and 1.7%. Those patients with a predicted mortality in excessive of 5% are generally regarded as 'high risk'. It has been reported that only 7.5% of patients undergoing major surgery are considered high risk and yet this group account for more than 80% of all postoperative deaths.

Identifying which patients are high risk can be difficult, with both surgical and patient-based factors involved (*Table 1*) (Shoemaker et al, 1988). Surgical factors include those that are associated with major fluid shifts, significant blood loss, emergency abdominal surgery and those with a direct impact on the cardiovascular system (e.g. abdominal aortic aneurysm repair). The main patient factors are a poor functional capacity and/or an acute deterioration such as sepsis, massive blood loss or acute renal impairment. Given the increased risk of perioperative complications, those patients considered high risk require greater evaluation and a more focused perioperative plan.

Table 1. Clinical criteria for high-risk surgical patients

Previous severe cardiorespiratory illness – acute myocardial infarction, chronic obstructive pulmonary disease, or stroke
Late-stage vascular disease involving aorta
Age > 70 years with limited physiological reserve in one or more vital organs
Extensive surgery for carcinoma (e.g. oesophagectomy, gastrectomy, cystectomy)
Acute abdominal catastrophe with haemodynamic instability (e.g. peritonitis, perforated viscus, pancreatitis)
Acute massive blood loss > eight units
Septicaemia
Positive blood culture or septic focus
Respiratory failure: partial pressure of oxygen in arterial blood (PaO_2) < 8.0kPa on fractional inspired oxygen concentration (FiO_2) > 0.4 or mechanical ventilation > 48 hours
Acute renal failure: urea > 20 mmol/litre or creatinine > 260 mmol/litre

From Shoemaker et al (1988)

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Cellular hypoxia

Cellular hypoxia is important as it is a precursor to gross organ dysfunction. It may induce an inflammatory response, impair healing and increase the risk of infections. The initial stages of cellular hypoxia are often covert but are potentially reversible, but once overt organ dysfunction is evident irreversible damage may already have occurred. The aim of perioperative haemodynamic management is to prevent and reverse covert cellular hypoxia. As we can neither easily measure nor modify oxygen demand, our objective is to improve the circulatory function and thereby optimize global oxygen delivery to a level that should exceed demand.

Haemodynamic management

The haemodynamic management of a high-risk surgical patient begins preoperatively and extends well into the postoperative phase. Appropriate management at each stage is essential if serious complications are to be avoided.

Preoperative

The identification of high-risk patients should occur preoperatively. Chronic illnesses should be stabilized and any acute deterioration where possible reversed. Patients with a history of ischaemic heart disease need special attention as evidence now suggests that those with significant ischaemia on stress testing (exercise or dobutamine stress echocardiogram) benefit from perioperative beta-blockade (Mangano et al, 1996; Poldermans et al, 1999). The American College of Cardiology/American Heart Association Task Force have published excellent guidelines on the assessment and appropriate workup of patients with cardiovascular disease undergoing non-cardiac surgery (Eagle et al, 1996).

Intraoperative

High-risk patients undergoing major surgery require intensive monitoring that exceeds the minimal monitoring standards as published by the Anaesthetic Association of Great Britain and Ireland (2000). Urethral catheterization with the use of an urometer, core temperature monitoring, and in patients with a history of ischaemic heart disease five-lead electrocardiograph with ST analysis are all indicated. Invasive arterial pressure monitoring is a requirement as it allows beat to beat analysis, is more accurate over a larger range of pressures and also permits easy access for arterial blood–gas analysis. General standards of anaesthesia apply such that the patient should receive a balanced anaesthetic, it should be remembered that cardiovascular instability does not excuse an inadequate depth of anaesthesia. The patient should be kept warm and well oxygenated. Tachycardia, hypotension and hypertension should all be avoided. Biochemical markers of tissue hypoxia (lactate, base excess or mixed venous saturations) should be used to monitor and guide therapy.

Fluid therapy

Intraoperative fluid therapy is a central component of haemodynamic management. Tissue perfusion and oxygen delivery are reliant on an adequate cardiac output, which itself is dependent on an adequate left ventricular end diastolic volume or preload.

The heart rate, blood pressure and urine output, although often used, are very crude markers of fluid volume status that can be affected by many confounding factors. An appropriate mean arterial blood pressure (MAP) within autoregulation limits may be required to ensure adequate perfusion of vital organs, but in itself it is not a marker of volume status since in the presence of hypovolaemia the MAP may initially be maintained by an increasing systemic vascular resistance.

The best way to assess volume status remains a contentious issue. There are now a number of monitors available, all with varying complexity and degrees of invasiveness. They each use differing endpoints which may be categorized as either pressure or volumetric monitors.

Pressure monitoring

The two principal pressure monitors are the central venous catheter and the pulmonary artery catheter, which look at the central venous and pulmonary artery occlusion pressures respectively. These have historically been the mainstay of fluid management, with the pulmonary artery catheter viewed as the gold standard. Unfortunately these give an indication of filling pressures, whereas preload is volume dependent. This is made more complicated by the fact that the relationship between ventricular pressures and volumes depends on ventricular wall compliance which is not constant. There is also little clinical evidence to support that either central venous or pulmonary artery occlusion pressures confer any significant benefit in guiding fluid therapy. As a result there is now considerable debate as the role of such pressure monitors (Connors et al, 1996).

Interestingly, both these devices also allow the direct measurement of central and mixed venous saturation. In the presence of hypoxia, cells initially compensate by extracting more oxygen from the blood, leading to reduced central venous saturations. As reduced central venous saturations are a marker of impending or actual tissue hypoxia, they can thus be used to assess the adequacy of oxygen delivery and to guide fluid therapy. Rivers et al (2001) used central venous saturations to optimize the balance between oxygen delivery and consumption in early sepsis and this resulted in an absolute reduction in mortality of 16%. The target in the treatment group was to improve the central venous oxygen saturation to greater than 70%, achieved through fluid therapy, blood transfusions and where needed ionotrope therapy.

Volumetric monitors include pulse contour analysis, transoesophageal Doppler and transoesophageal echocardiography (TOE). These monitors do not assess filling volumes directly, but instead measure the stroke volume and also monitor the variation in systolic pressure, pulse pressure and stroke volume in response to mechanical ventilation. The variation in these haemodynamic parameters to positive pressure ventilation gives a dynamic assessment of fluid volume status, and this is now considered the most accurate method of predicting whether a patient will respond to a fluid challenge (Reuter et al, 2003).

Studies using these devices to guide intra- and postoperative fluid resuscitation have shown a significant reduction in complications and length of hospital stay over standard care. Sinclair et al (1997) and Venn et al (2002) used the oesophageal Doppler to guide fluid therapy in patients having surgery for proximal femoral fractures. Fluid therapy was given to an endpoint of maximal stroke volume, with the treatment group receiving significantly greater volumes of fluid (about 500 ml more). They demonstrated a reduction in time to medical recovery, assessed as being fit for discharge from hospital, from 20 days in the control group to 12 days in the treatment group. Gan et al (2002) compared standard fluid therapy to that guided by an oesophageal Doppler in general surgical patients. Fluid therapy was targeted to a maximal stroke volume, and this treatment led to a significant reduction in length of hospital stay and earlier return to enteral feeding.

TOE gives excellent information on ventricular filling, global and regional cardiac function and can also demonstrate myocardial ischaemia. Unfortunately the usefulness of the TOE as an intraoperative tool is limited by the fact that it requires specialist skills, is expensive and gives non-continuous information.

It must be remembered that the single isolated values measured or derived from any of the above monitors are all of limited use, instead it is the trend in the values obtained or response to an intervention (such as fluid challenge) that is of most importance.

Types of fluid therapy

Fluid resuscitation and maintenance may be with crystalloids, colloids or a combination of both. Crystalloids rapidly equilibrate throughout the extracellular fluid, whereas colloids are at least initially confined to the intravascular space. This means that to achieve the same volume of intravascular fluid expansion, three times as much crystalloid must be given as compared to colloid, which may be associated with tissue oedema. Owing to the high concentration of chloride in 0.9% 'normal' saline, large volumes of sodium chloride-based crystalloid solutions and colloid suspensions can lead to a hyperchloraemic metabolic acidosis. This can complicate arterial blood gas interpretation and has been shown to impair both renal function (Reid et al, 2003) and coagulation (Ekseth et al, 2002) – neither of these changes has been shown to affect patient outcome (Waters et al, 2001). Despite the differences between different crystalloid solutions and between crystalloids and colloids, no study has been able to show any outcome benefit of one type of fluid over another (Choi et al, 1999; SAFE Study Investigators, 2004).

Patients with evidence of tissue ischaemia despite adequate fluid resuscitation may benefit from ionotrope therapy. The goal of this is to improve the cardiac output and thereby improve oxygen delivery.

An adequate haemoglobin concentration is an important component of oxygen delivery. However, the transfusion of allogenic blood is not without well-documented risks (Williamson et al, 1999). Transfused blood has depleted levels of 2,3 diphosphoglycerate which initially impairs oxygen delivery, and transfusion of old blood has been shown not to improve regional tissue oxygenation. In patients without cardiovascular compromise two large studies have shown that a restrictive transfusion policy is well tolerated (haemoglobin >7 g/dl) (Hebert et al, 1999; Corwin et al, 2004). The ideal haemoglobin level in acute cardiovascular compromise and sepsis is less clear. Data from the resuscitation of patients with severe sepsis (Rivers et al, 2001) and targeted resuscitation of perioperative patients (Boyd et al, 1993) suggest keeping a haemoglobin level around 10 g/dl is associated with improved survival.

Vasoactive agents

In general, vasoactive drugs may be used intraoperatively to maintain an adequate MAP for organ perfusion, although they should only be used once the patient has been adequately fluid resuscitated. Using these agents to increase the MAP to the high end of the normal range has not been shown to confer benefit (Bourgoin et al, 2005). There is no evidence to support the use of low-dose dopamine as a reno-protective agent. The use of intravenous nitrates to prevent myocardial ischaemia has not been shown to be effective.

Patients with evidence of tissue ischaemia despite adequate fluid resuscitation may benefit from ionotrope therapy. The goal of this is to improve the cardiac output and thereby improve oxygen delivery. Ionotropes aim to improve cardiac contractility, but by increasing myocardial work they risk inducing arrhythmias and myocardial ischaemia. In selected high-risk patients (*Table I*) there is now evidence of improved outcomes by using fluid resuscitation, blood transfusions and vasoactive agents to improve global oxygen delivery to predefined levels or goals. This targeted resuscitation has become synonymous with goal-directed therapy (GDT). As discussed earlier, those patients with significant cardiovascular disease may instead benefit from perioperative beta-blockade, but this still needs to be combined with optimal fluid therapy.

Goal-directed therapy

Shoemaker (1972) showed improved survival in surgical patients who were able to achieve increased cardiac index (CI) (>4.5 litre/min/m^2) and oxygen delivery index (DO_2I) (>600 litre/min/m^2) in the postoperative period. Since this initial study there has been much interest and research into the benefit of GDT. The assumption behind GDT is that improving oxygen delivery to these levels pre-emptively may reverse or prevent tissue ischaemia before irreversible tissue damage has occurred. The physiological parameters or 'goals' used are usually those which were initially described by Shoemaker (CI >4.5 litre/min/m^2, DO_2I >600 litre/min/m^2).

GDT requires continuous cardiac output monitoring to guide both fluid and ionotopic therapy. Typically ionodilators, such as dopexamine, are used to achieve the target CI. There have been a number of studies to date that have shown that perioperative optimization improves outcome in terms of mortality, complications and length of hospital stay. Shoemaker et al (1988) did the first large outcome trial in GDT, and found that GDT significantly reduced mortality from 28% to 4% when compared to standard therapy. Boyd et al (1993) also demonstrated that GDT (CI >4.5 litre/min/m^2 and DO2I >600 litre/min/m^2) significantly improved mortality (22.2% *vs* 5.7%). The treatment group also had half the number of postoperative complications.

Postoperative

Optimal haemodynamic management does not finish at the end of surgery; close monitoring and control should extend well into the postoperative period. The cardiovascular consequences of surgery impact many days after surgery has finished. For example fluid shifts may develop owing to an ileus and there may be ongoing blood loss. The peak incidence of perioperative myocardial infarction occurs between 48 and 72 hours after surgery. It is therefore not surprising that optimization of oxygen delivery even in the postoperative period has been shown to confer significant benefits. Pearse et al (2005) have shown that early GDT in the postoperative period halved postoperative complications (0.7 *vs* 1.5 per patient) and reduced the length of hospital stay by 40%.

Conclusions

There is now good evidence to suggest that, in high-risk surgical patients, by optimizing oxygen delivery we can reduce the risk of perioperative complications initiated by tissue hypoxia. This involves identification of high-risk patients, beta-blockade where indicated, appropriate cardiovascular monitoring, well-guided fluid therapy and in selected patients ionotropic support. This process should begin in the preoperative phase and continue throughout the perioperative period. **BJHM**

Conflict of interest: St George's Intensive Care Unit performs research for LiDCO and Edwards Life Sciences.

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KEY POINTS

- Optimal haemodynamic management needs to be practised throughout the perioperative period.
- The aim is to avoid and/or reverse tissue hypoxia before overt organ dysfunction occurs.
- A combination of fluids, blood, oxygen and ionotropes may be required to improve oxygen delivery.
- Fluid therapy should be given in appropriate volumes and the effects monitored against defined endpoints.
- In high-risk surgical patients goal-directed therapy significantly reduces complications, mortality and length of hospital stay.