

Clinical Paper

Hemodynamic instability

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Scope of the problem: Why is hemodynamic instability important?

The term "hemodynamic instability" is most commonly associated with an abnormal or unstable blood pressure, especially hypotension. Here, hemodynamic instability will be defined more broadly as global or regional perfusion that is not adequate to support normal organ function. This definition recognizes the obligation to insure adequate organ perfusion during the perioperative period.

Numerous hemodynamic parameters (heart rate, blood pressure, central venous pressure, pulmonary artery pressure, pulmonary artery occlusion pressure, and cardiac output) can be monitored and controlled. Abnormalities of any of these parameters may indicate hemodynamic instability, and control of any of these variables may restore adequate organ perfusion. However, the two variables that most directly reflect organ perfusion are blood pressure and indices of global perfusion, such as cardiac output or mixed-venous hemoglobin oxygen saturation (SvO₂).

Due to limits on coronary and cerebral autoregulation, hypotension may compromise adequate perfusion of the brain and heart. Animal studies have demonstrated that blood pressure is the primary determinant of cerebral and coronary blood flow when the limits of autoregulation are exceeded. However, studies relating hypotension to outcome have shown mixed results (1-6). Therefore, the clinician can only conclude that there are blood pressure thresholds below which coronary and cerebral ischemia may occur, that these are probably more stringent in individuals with fixed vascular stenoses, and that they may vary greatly among individuals.

For the same reasons that there are mixed data relating hypotension to outcome, it has been difficult to design studies relating outcome to decreased cardiac output or SvO₂ (7-17). Overall the evidence indicates that maintaining adequate global perfusion contributes to improved outcome. The difficulty is that the definition of "good" is unclear. Supranormal oxygen delivery (a cardiac index roughly greater than 4.5 L.min/m²) has not been widely accepted. A reasonable goal would be a cardiac index of 2.5-3 L/min/m² or greater in conjunction with an SvO₂ greater than 65-70% and a lactate level lower than 2-3 mM/l.



Who is at risk?

It is important for the anesthesiologist to understand the risk to the patient from pre-existing conditions or the surgical procedure to plan perioperative patient management. However, even healthy patients undergoing minor procedures may manifest hemodynamic instability due to anesthesia itself. Often, anesthesiologists are better prepared to manage the anticipated hemodynamic instability in a patient with significant cardiovascular disease undergoing major surgery than they are in a relatively healthy patient undergoing outpatient surgery.

Recognizing hemodynamic instability

A key concept necessary for understanding hemodynamic instability is the simple equation relating blood pressure and cardiac output:

$$\text{MAP} = \text{SVR} \times \text{CO} + \text{CVP}$$

(where MAP is mean arterial systemic blood pressure, SVR is systemic vascular resistance, CO is cardiac output, and CVP is central venous pressure). This equation warns us that a normal blood pressure does not guarantee an adequate cardiac output. Recognition of hemodynamic instability requires monitoring of both blood pressure and global perfusion.

While hypotension is probably the most commonly recognized manifestation of hemodynamic instability it is not the most common problem. An adequate blood pressure may insure coronary and cerebral perfusion (18,19); however, it does not insure renal and mesenteric perfusion, which cannot be measured directly. It is best to assume that appropriate "global" perfusion (i.e., cardiac output or SvO₂) will increase the chance that the kidneys and mesentery are also adequately perfused. There is ample evidence that maintaining blood pressure in the presence of inadequate cardiac output by excessive vasoconstriction of efferent glomerular arterioles is deleterious to renal function (20). Mesenteric circulation is even more sensitive to excessive vasoconstriction (21) and the global cardiac output needed to insure mesenteric and renal perfusion is unclear. However, cardiac indices lower than normal values must be viewed as suspect. In addition, it is most prudent to look at multiple indicators of adequate perfusion, such as cardiac output, SvO₂, and lactate concentrations.

Monitoring

Blood pressure

While noninvasive blood pressure monitoring may be quite accurate, if the patient is hemodynamically unstable it is important to monitor blood pressure invasively on a beat-to-beat basis. The most common catheterization site is the radial artery, although femoral, brachial and axillary arteries have also been used. The monitoring system must be appropriately zeroed and calibrated. It is often helpful to utilize both invasive and noninvasive monitoring in order to have both beat-to-beat monitoring and an independent measure of the accuracy of the invasive system. There may be discrepancies between peripherally measured blood pressure and central aortic pressures (aortic systolic lower than radial). However, in two important situations — immediately after cardiopulmonary bypass and in septic patients — the systolic blood pressure measured from the radial arterial catheter may be misleadingly low (22).

Finally, one should note that any system of monitoring actually gives the clinicians three blood pressures — diastolic, mean and systolic. Of these, the mean is least likely to be affected by monitoring artifacts and (with the exception of diastolic pressure and coronary perfusion) is the measure most directly correlated with organ perfusion. Systolic pressure often gets the most attention, but is often the least informative.

Cardiac output

While invasive monitoring of blood pressure is relatively common in surgical patients who are at higher risk for hemodynamic instability, cardiac output monitoring is less common. Clinical recognition of low cardiac output syndrome in the absence of hypotension can be difficult (23, 24). The mainstay for cardiac output measurement has been the pulmonary artery catheter, which allows measurement of cardiac output using the thermodilution technique, and which simultaneously allows measurement of central venous pressure, pulmonary artery pressure, and pulmonary artery occlusion pressure. Recent advances in applications of the thermodilution principle now allow for continuous cardiac output measurements. Furthermore, mixed venous blood samples can be drawn and mixed venous hemoglobin oxygen saturation can be monitored, either intermittently or continuously, using fiberoptic technology.

Given these capabilities, it is no surprise that the pulmonary artery catheter has been a mainstay of hemodynamic monitoring. However, its use is not without controversy. No definitive study has demonstrated that use of the pulmonary artery catheter improves outcome, rather two retrospective studies have suggested that mortality is increased by use of pulmonary artery catheters (25,26). The conclusion, if not readily accepted, must be taken seriously and has promoted interest in alternative methods of measuring cardiac output.

Promising technology has been introduced, including Fick measurement of cardiac output using CO₂ rebreathing, Doppler echocardiographic measurement of descending aortic flow, pulse contour analysis and thermodilution using femoral arterial temperature sensing and lithium dilution (27-29). While these new technologies have not yet been extensively validated, early studies indicate that each may provide accurate estimations of cardiac output, although there are caveats in their use (30).

Mixed-venous hemoglobin oxygen saturation (SvO₂)

One of the pitfalls of monitoring cardiac output is that the definition of an “adequate” cardiac index is unclear. While normal cardiac index is, in general, greater than 2.5 L/min/m², this is only a very rough guide to appropriate values of cardiac output. Consideration of venous hemoglobin oxygen saturation is a logical means of assessing the adequacy of cardiac output. From the Fick principle, we know that if delivery of oxygen is inadequate SvO₂ will decrease unless there is a concomitant decrease in oxygen consumption. Oxygen delivery depends on hemoglobin concentration, arterial oxygenation, and cardiac output. If hemoglobin saturation, and arterial oxygenation are essentially normal (or remain unchanged) then a low (or decreasing) SvO₂ (< 65-70%) indicates that cardiac output is inadequate for oxygen consumption.

In general, measurement of true mixed-venous hemoglobin oxygen saturation requires a sample from the pulmonary artery, and hence a pulmonary artery catheter. However, a simple central venous hemoglobin oxygen saturation may track the true SVO₂ and direct therapy for septic patients (17). It appears that this approach to monitoring the adequacy of cardiac output merits consideration in other clinical scenarios, especially as it avoids the necessity of a pulmonary artery catheter.

Echocardiography

When hemodynamic instability has been recognized — as either hypotension or low cardiac output syndrome — treatment may be greatly facilitated by echocardiography providing a quick and accurate assessment of preload and contractility (31). Preload refers to the stretch of the myocardial fiber and is best estimated by left-ventricular end-diastolic volume (LVEDV). Echocardiography provides cardiac dimensions, not pressures, and, unlike those obtained using the pulmonary artery catheter, echocardiographic measures of preload correlate well with LVEDV (32-34). In addition, echocardiography can estimate ejection fraction, a useful measure of contractility (33, 34).

Also, besides providing good estimates of the two most important determinants of cardiac output (preload and contractility), echocardiography assesses valve function, regional wall motion abnormalities, sources of emboli, pericardial constraints to normal hemodynamics (e.g., tamponade, constrictive pericarditis), as well as allowing the clinician to distinguish right from left heart performance. For these reasons, echocardiography is highly recommended whenever there is hemodynamic instability of unknown etiology or that is not readily corrected by standard therapy.

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