The Interface Between Monitoring and Physiology at the Bedside

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KEYWORDS

• Hemodynamic instability • Shock • Hemodynamic monitoring

KEY POINTS

- Bedside measures of hemodynamic instability include mean arterial pressure, hypotension, and mixed venous oxygen saturation.
- Causes of circulatory shock can be divided into hypovolemic, cardiogenic, obstructive, and distributive shock, and the hemodynamic patterns are characteristic for each cause.
- The different causes of circulatory shock usually require different types of treatment modalities, making the correct etiologic diagnosis important.
- Pharmacotherapies for hemodynamic instability include vasopressors, inotropes, and vasodilators.
- Technological advances to restore hemodynamic instability include the use of ventricular assist devices and continuous renal replacement therapies.

HEMODYNAMIC INSTABILITY

Hemodynamic instability as a clinical state represents either a perfusion failure with clinical manifestations of circulatory shock or heart failure or 1 or more out-of-threshold hemodynamic monitoring values, which may not necessarily be pathologic. Circulatory shock can be produced by decreases in cardiac output relative to metabolic demands, such as decreased intravascular volume (hypovolemic), impaired ventricular pump function (cardiogenic), or mechanical obstruction to blood flow

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(obstructive) or by misdistribution of blood flow independent of cardiac output (distributive). The prompt identification and diagnosis of the probable cause of hemodynamic instability, coupled with appropriate resuscitation and (when possible) specific treatments, are the cornerstones of intensive care medicine.[1] Hemodynamic monitoring plays a pivotal role in the diagnosis and management of circulatory shock.

The management of the critically ill patient often requires continual monitoring of hemodynamic variables and the functional hemodynamic status, because of the level of cardiovascular instability that the circulatory shock creates. Patterns of hemodynamic variables often suggest hypovolemic, cardiogenic, obstructive, or distributive shock processes as the primary causes of hemodynamic instability. These different types of causes of circulatory shock usually require different types of treatment modalities, making these differential distinctions important. Diagnostic approaches or therapies based on data derived from hemodynamic monitoring in the critically ill patient assume that specific patterns of derangement reflect specific disease processes, which respond to appropriate interventions.[2]

MEAN ARTERIAL PRESSURE AS A MEASURE OF HEMODYNAMIC INSTABILITY

Organ perfusion is dependent on input organ perfusion pressure and local vasomotor tone. Local vasomotor tone varies inversely with local tissue metabolic demand. For most organs except the kidneys and heart, independent changes in arterial pressure higher than some minimal value are associated with increased vasomotor tone to keep organ perfusion constant and are therefore not entirely dependent on cardiac function and cardiac output. In such situations, cardiac output is important only to allow parallel circuits to maintain flow without inducing hypotension, and cardiac function is important only in sustaining cardiac output and a given output pressure without causing too high a back pressure in the venous circuits. Hypotension, on the other hand, decreases blood flow to all organs. Operationally, mean arterial pressure (MAP) is the input pressure to all organs other than the heart. Diastolic aortic pressure is the input pressure for coronary blood flow. MAP is estimated to be equal to the diastolic pressure plus one-third the pulse pressure between diastole and systole. Over a wide range of MAP values, regional blood flow to the brain and other organs remains remarkably stable because of autoregulation of local vasomotor tone to keep that local blood flow constant despite changing MAP. However, in a previously normotensive patient, once MAP decreases lower than ~60 mm Hg, then, tissue perfusion may decrease independent of metabolic demand and local autoregulatory processes. As tissue blood flow decreases independent of metabolic demand, then, tissue O_2 extraction increases to keep local O_2 consumption and metabolic activity constant. This process occurs routinely in most individuals and, if transient, is not pathologic. However, if tissue blood flow decreases further than increased O_2 extraction can compensate for, then end-organ ischemic dysfunction follows. Despite the lack of sensitivity of a nonhypotensive MAP to reflect hemodynamic stability, measures of MAP to identify hypotension are essential in the assessment and management of hemodynamically unstable patients, because hypotension must decrease autoregulatory control, and increasing MAP in this setting also increases organ perfusion pressure and organ blood flow.

HYPOTENSION AS A MEASURE OF HEMODYNAMIC INSTABILITY

Hypotension directly reduces organ blood flow, is synonymous with hemodynamic instability, and is a key manifestation in most types of circulatory shock. It also causes coronary hypoperfusion, impairing cardiac function and cardiac output. However, the