Hemodynamic monitoring in intensive care unit: where we are today?

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ABSTRACT
A significant alteration of attitude towards hemodynamic monitoring in clinical setting happened during the last half of 1970. This occurred as a consequence towards the relative insensitivity and unresponsiveness of clinical methods to diagnose the rapid physiologic changes, especially in the critical care settings. During the 1980s the principles of hemodynamic monitoring allowed the basic physiological measurements to be applied in a consistent and meaningful manner for the management of a cardiovascular aspect of critical illness. The first paradigm shift in the hemodynamic monitoring can be traced back to the development of cardiac catheterization by Werner Forssmann in 1929. The idea of advanced hemodynamic monitoring was developed by HJC Swan, who developed balloon-tipped Swan-Ganz catheter from his observation of motion of sailboats on the Santa-Monica Bay in 1967. For more than 30 years, the pulmonary catheterization method has generally been accepted and is still the clinical standard to which the other methods are compared. Recent advances in technology have led to the development of minimally invasive (Flo-trac/Vigileo and transesophageal echocardiography) and noninvasive methods (impedance cardiography) for hemodynamic monitoring in critical care unit. The current narrative review is an attempt to highlight the present perspectives in hemodynamic monitoring in the Intensive Care Unit (ICU).

Key words: Hemodynamic monitoring, intensive care unit, indications

INTRODUCTION
Hemodynamics is defined as the study of the forces involved with blood circulation. Hemodynamic monitoring is started with the monitoring of heart rate using “simple skills of a finger on pulse” and then moved on to more and more sophisticated techniques like ECG, invasive blood pressure monitoring, etc. The hemodynamic status of critically ill patients can be assessed either by noninvasive single parameter or various invasive techniques that provide multiparameter hemodynamic measurements. As a result, comprehensive data can be provided for the clinician to a pro-actively advice hemodynamic crisis and safely manage the patient instead of reaching to the late indicators of hemodynamic instability.

HEMODYNAMIC AND HEMODYNAMIC MONITORING, WHY IT IS SO SPECIAL IN INTENSIVE CARE UNIT (ICU)?
Hemodynamic instability is defined as global or regional perfusion that is not adequate to support normal organ function. The primary goal of hemodynamic monitoring in ICU is to assess the adequacy of vital organ perfusion with regard to maintaining oxygen delivery. Adequate volume management of critically ill patients is crucial, as under or over resuscitation is associated with adverse clinical outcome. Ongoing advancement of monitoring techniques has shed new insight towards the pathophysiological changes associated with critical illness. The method of obtaining continuous and accurate measurements has evolved over the years from non-invasive to invasive and semi-invasive techniques. Each method has its merits and drawbacks. This article is not meant to review the...
technology of the various systems in any detail, nor to identify one monitoring aid that would suitable for all patients, rather focus on the utility advantages and limitations of each system with the aim to guide the choice of monitoring systems in critically ill patients.

**INDICATIONS FOR HEMODYNAMIC MONITORING IN ICU**

- Diagnosis and management of shock from various etiologies (Hemorrhage, hypovolemia pulmonary embolism, heart failure, sepsis etc.)
- Management of complicated myocardial infarction (MI)
  - Cardiogenic shock
  - Ventricular septal rupture following acute MI
  - Severe left ventricular failure
  - Refractory ventricular tachycardia
  - Right ventricular infarction
  - Severe unstable angina refractory to routine management
- To determine the cause of dyspnoea and hypoxia (e.g., pulmonary disease versus left ventricular failure)
- Assessment of possible cardiac tamponade
- Assessment of after load reduction therapy in patients with left ventricular failure
- Management of critically ill patients with associated cardiovascular problem
- Management of post-operative cardiac surgical patients
- Management of patients with cardiovascular disease undergoing noncardiac surgery

**CLASSIFICATION OF METHODS AND AVAILABILITY FOR EQUIPMENTS HEMODYNAMIC MONITORING**

- Non-invasive
  - Clinical assessment
  - Non-invasive blood pressure (NIBP) measurement
  - Temperature
  - Pulse oximetry
  - Capnography
  - Bio impedance technology (transthoracic)
  - Bioimpedance peripheral wave form analysis (transcutaneous)
- Transthoracic echocardiography (TTE)
- Semi-invasive
  - Esophageal Doppler
  - Transesophageal echocardiography
- Invasive haemodynamic monitors
  - Invasive arterial pressure (IBP)
  - Central venous pressure (CVP)
  - Pulse contour analysis/arterial wave form analysis
  - Mixed venous oxygen saturation (SVO₂) and central venous oxygen saturation (ScVO₂)
  - Pulmonary artery catheter (PAC) for monitoring of cardiac output and derived parameters
    - Stroke volume (SV)
    - Stroke volume variation (SVV)
    - Systemic vascular resistance (SVR)
    - Pulmonary vascular resistance (PVR)
    - Oxygen delivery (DO₂)
    - Oxygen delivery index (DO₂I)
  - Monitoring of right ventricle
  - Monitoring of extravascular lung water and intrathoracic blood volume
  - Monitoring of organ perfusion and microcirculation
  - Blood lactate
  - Gastrointestinal tonometry
  - Near infrared spectroscopy
  - Tissue oxygen tension

**WHAT AN IDEAL HEMODYNAMIC MONITORING SYSTEM SHOULD?**

- Be easy to use
- Be readily available
- Have rapid response time
- Provide accurate and reproducible measurements that is able to provide therapy
- Be operator independent

**CLINICAL ASSESSMENT**

The parameters used for clinical assessment are highly subjective and suffer from inter-observer variability and does not give minute to minute recording for a longer time.
PULSE OXIMETRY

Continuous pulse oximetry monitoring not only measures oxyhemoglobin saturation but also gives an evidence of volume status of the critically ill patients and used to titrate supplemental oxygen. Patients with reduced peripheral blood flow from vascular disease, sepsis or pallor of extremities may not show a pulse oximetry reading.

CAPNOGRAPHY

It is a standard monitoring aid in intubated and nonintubated patients in the operating room as well as in intensive care unit (ICU). End-tidal CO₂ in the lung is directly proportional to the pulmonary blood flow. The progressive decline reflects an increasing dead space as experienced during cardiac arrest, massive pulmonary embolism or severe bronchospasm. EtCO₂ measurement during cardiopulmonary resuscitation (CPR) may serve as a surrogate to CO and used to assess the adequacy of chest compression. During chest compression, the EtCO₂ should be >10 mmHg and returned to baseline signifies the return of spontaneous circulation. Failure to obtain and maintain EtCO₂ >10 mmHg is considered to be associated with insufficient or failed resuscitation measures.

INVASIVE BLOOD PRESSURE MONITORING

The usual sites are radial, femoral, brachial and dorsalis pedis artery. The radial artery is the most common site of cannulation, due to easy accessibility and relatively low complication rates. The cannula is inserted either by modified Seldinger technique, direct cannulation or ultrasound guidance. Potential complications of arterial cannulation are an infection, thrombosis, distal embolization, arterial spasm, bleeding and accidental drug injection. Damping may occur and confused with low blood pressure.

CENTRAL VENOUS PRESSURE (CVP)

CVP has been traditionally used as a surrogate of right ventricular preload and overall intravascular volume. The normal CVP in a spontaneously breathing patient is 0-5 mmHg, and in mechanically ventilated patient it is up to 10 mmHg. The general consensus is that absence of respiratory variations in the CVP wave form predicts a lack of response to the CO to further fluid administration. CVP has been compared favorably to the PAC for the management of acute respiratory distress syndrome (ARDS) patients. During the initial hours of presentation of a septic shock patient, the protocol involves maximizing CVP to >8 mmHg and then using vasoactive medications to achieve a MAP>65 mmHg. Once the MAP is to maintain ScVO₂ >70% by transfusing red blood cells if the hematocrit is <30% and by using inotropes if the hematocrit is >30%. However, a single reading of CVP does not imply patient fluid status. An increase in CVP following a fluid bolus may represent the fluid status as well as ventricular function but is controversial.

CVP wave form analysis is useful in the diagnosis of several conditions. A loss of “a” wave happens in atrial fibrillation. Cannon “a” waves (fusion of a and c waves) are generated in ventricular tachycardia, junctional rhythm, and complete heart block. Prominent “a” waves occur in tricuspid stenosis or reduced right ventricular compliance, pulmonary hypertension and pulmonary Stenosis.) Formation of “cv” waves (fusion of c and v waves) seen in tricuspid regurgitation. Bifid CVP waveform occurs in constrictive pericarditis (“x” and “y” descent is stiff and abrupt) and cardiac tamponade (prolonged or dampened “y” descent).

Because of a wide array of diagnostic potential of CVP monitoring, it has a high volume use. The monitoring method is however not free from some complications (bleeding from an arterial puncture, thrombosis, pneumothorax, infection, air embolism) and contraindications (patient refusal, SVC clot, the presence of pacemaker/defibrillator on the same side, mass in RA).

MIXED VENOUS OXYGEN SATURATION (SVO₂) / CENTRAL VENOUS OXYGEN SATURATION (ScVO₂) MONITORING

Mixed venous oxygen (SVO₂) is obtained by sampling from the pulmonary artery and provides an excellent measure of the balance between O₂ delivery and its utilization in the tissues. Mixed venous blood, when sampled from a central venous line (from SVC/RA and occurs prior to the return of the blood from the coronary sinus) is called central venous oxygen saturation (ScVO₂). There is still a debate whether ScVO₂ can serve as a surrogate to SVO₂ as the former analyzed only the blood returned from the upper part of the body. Trending ScVO₂ in critically ill patients may provide an acceptable surrogate for SVO₂ according to some authors.

\[
SVO₂ = \frac{SaO₂ × VO₂ × CO × 1.39}{Hb}
\]

Normal value: 65-75%. The value of ScVO₂ is 5-10% higher than SVO₂.
SVO$_2$ has been identified as a part of early goal directed therapy protocol to guide treatment according to the surviving sepsis campaign$^{14}$. During CPR, ScVO$_2$, greater than 72% was 100% predictive of return of spontaneous circulation while patients in which ScVO$_2$ did not raise above 30% during CPR did not have successful resuscitation$^{15}$. Table 1 depicts the changes in SVO$_2$ in different clinical situations.

**CARDIAC OUTPUT (CO)**

Accurate measurement of CO is essential in the management of critically ill patients. The available monitors not only provides the CO but also the other derived parameters e.g., stroke volume (SV), stroke volume variation (SVV), cardiac index (CI) DO$_2$, DO$_2$I, systemic vascular resistance (SVR), pulmonary vascular resistance (PVR) which on other hand estimate the volume assessment and oxygen delivery as well as tissue oxygen extraction status of the patients. The various available monitoring aids, their advantages, and disadvantages are described in Table 2. The accuracy of all the methods has been compared to that of PAC.

### Table 1: Causes of changes in SVO$_2$

<table>
<thead>
<tr>
<th>Change in SVO$_2$</th>
<th>Increased O$_2$ delivery</th>
<th>Decreased O$_2$ consumption</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased SVO$_2$</td>
<td>Supplemental O$_2$, Increased CO states Inotropes</td>
<td>General anaesthesia, Deep sedation, Hypothermia, Mechanical ventilation, Intracardiac/Intrapulmonary Shunt, Sepsis</td>
</tr>
<tr>
<td>Decreased SVO$_2$</td>
<td>Decreased O$_2$ delivery</td>
<td>Increased O$_2$ consumption</td>
</tr>
<tr>
<td>High SVO$_2$ despite evidence of end-organ Hypoxia</td>
<td>Micro vascular shunting Histotoxic hypoxia Abnormalities in distribution of blood flow</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2: Monitoring aids for CO measurement

<table>
<thead>
<tr>
<th>Sl. no.</th>
<th>Method and Principle</th>
<th>System</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Thermodilution</td>
<td>Pulmonary artery catheter (PAC)$^{16}$</td>
<td>Standard method, Widely used, Time tested, Provide simultaneous measurements of other haemodynamic parameters in addition to CO</td>
<td>Invasive, Requirement of training, Complications related to invasiveness, Cannot be used in right sided cardiac lesions.</td>
</tr>
<tr>
<td>2.</td>
<td>Transpulmonary indicator dilution</td>
<td>a. Picco (Pulsion Medical System, Munich, Germany)$^{17}$</td>
<td>1. Need dedicated arterial waveform, 2. Less accurate.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Use lithium as an indicator</td>
<td>b. LiDCO (Ltd. London, UK)$^{18}$</td>
<td>1. Inaccurate in case of hyponatremia, 2. Interference by non-depolarizing muscle relaxant.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Calculate CO by using ultrasound technology to measure changes in blood ultrasound velocity and blood flow following an injection of warm saline solution.</td>
<td>c. CO status$^{19}$</td>
<td>Less invasive (it uses an arterial catheter and central venous catheter).</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Same as Picco system</td>
<td>d. Volume view$^{20}$</td>
<td>1. Less invasive, 2. Measure global end-diastolic volume and extravascular lung water.</td>
</tr>
<tr>
<td>3.</td>
<td>Arterial Pressure wave form derived</td>
<td>Flo-trac Vigileo (Edward Life Sciences)$^{21}$</td>
<td>1. Less invasive (use radial artery catheter), 2. Rapid measurement</td>
<td>1. Need an optimal arterial pressure tracing, 2. A drift in values occurs whenever there is a major change in vascular compliance, e.g. vascular leak syndrome and aortic regurgitation.</td>
</tr>
</tbody>
</table>
Several interpretations can be made by the above equipments in critically ill patients which can guide the management (Table 3 and Table 4).

**MONITORING OF RIGHT VENTRICLE**

Maintenance of circulatory homeostasis depends on an adequate function of both the ventricles. There is growing interest in the importance of the neglected right side of the heart, particularly in patients suffering from sepsis\(^2^0\). CVP, RAP or RVP have been demonstrated to be invalid for judging right ventricular function or right ventricular loading condition\(^2^1\). Measurement of right ventricular ejection fraction (RVEF) has a tremendous role in the clinical outcome of cardiac surgical patients. However, no clinical trials are available showing a beneficial impact of RVEF monitoring in clinical outcome.

<table>
<thead>
<tr>
<th>4.</th>
<th><strong>Esophageal Doppler</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>An ultrasound beam is directed along the aorta using a probe (passed via oral or nasal route). A part of the beam is reflected back by the moving red blood cells at different frequency. The resultant Doppler shift can be used to calculate the flow velocity and volume, hence CO.</td>
<td>Cardio Q(^2^0)</td>
</tr>
</tbody>
</table>
| 1. Useful in optimization of fluid management in high risk patients  
2. Easy to use | 1. Special skill required  
2. Measurement is intermittent  
3. Uncomfortable in a non-intubated patient.  
4. Invalid in the presence of intraortic balloon pump.  
5. Need further validation in critically ill patients. |

<table>
<thead>
<tr>
<th>5.</th>
<th><strong>CO(_2) rebreathing method</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>It is based on Fick’s principle. It uses a CO(_2) sensor, a disposable airflow sensor and a disposable rebreathing loop. CO(_2) production is calculated from minute ventilation, CO(_2) content, and the arterial CO(_2) content is estimated from end tidal CO(_2).</td>
<td>NICO(^2^0)</td>
</tr>
</tbody>
</table>
| 1. Easy to use | 1. Less reliable in respiratory failure and rapid hemodynamic changes.  
2. Controversial in critical care setting. |

<table>
<thead>
<tr>
<th>6.</th>
<th><strong>Suprasternal Doppler</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>works in the same principle as that of esophageal Doppler</td>
<td>USCOM(^2^0)</td>
</tr>
</tbody>
</table>
| 1. Easy  
2. Reliable | 1. Special Skill required.  
2. Difficult to get the window in some patients. |

<table>
<thead>
<tr>
<th>7.</th>
<th><strong>Echocardiography(^2^0)</strong></th>
</tr>
</thead>
</table>
| Transthoracic echocardiography | 1. Non invasive  
2. Give additional information about cardiac chambers apart from CO.  
Kissing papillary muscles may incite fluid administration where as poorly contractile myocardium may suggest the need for an inotrope like dobutamine | 1. Need special skill  
2. Long learning curve  
3. Expensive instruments |

| Transthoracic echocardiography | 1. Easy  
2. Reliable | 1. Semi invasive  
2. Need special skill  
3. Long learning curve  
4. Expensive instruments |

<table>
<thead>
<tr>
<th>8.</th>
<th><strong>Thoracic bioimpedance(^2^0)</strong></th>
</tr>
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<tbody>
<tr>
<td>Conductivity of a high frequency, low amplitude alternating current passed across the thorax changes as the blood flow varies with each cardiac cycle. These changes can be measured using electrodes placed on a patient’s chest and used to generate wave been form, from which CO and other parameters can be measured.</td>
<td>ICON</td>
</tr>
<tr>
<td>Can measure thoracic fluid content</td>
<td>Not reliable in acutely ill patients</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>9.</th>
<th><strong>Thoracic bioimpedance(^2^1)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Measures changes in the frequency of the electrical currents traversing the chest</td>
<td>NiCOM</td>
</tr>
<tr>
<td>Validated only in one study</td>
<td></td>
</tr>
</tbody>
</table>

PA, pulmonary artery; CO, cardiac output
MONITORING OF EXTRAVASCULAR LUNG WATER (EVLW) AND INTRATHORACIC BLOOD VOLUME (IBV)

Intrathoracic blood volume appears to be a more reliable indicator of preload than the cardiac filling pressures\(^\text{21}\). Depressed left ventricular performance increases hydrostatic pressure in the pulmonary circulation, synergistically influencing fluid flux across a damaged pulmonary microvascular membrane and increased EVLW. Using EVLW and IBV monitoring, a reduction in ICU stay and hospital stay has been shown by some authors\(^\text{21}\).

MONITORING OF ORGAN PERFUSION AND MICROCIRCULATION

Organ failure in critically ill patients is subsequent to inadequate tissue perfusion and oxygenation. Since tissue hypoxia can persist despite the presence of an apparently adequate systemic blood flow, blood pressure and arterial oxygen content, monitoring of specific indices of oxygenation at tissue level is essential.

Blood lactate: Blood lactate concentration above 2 mmol/L is generally considered as a biochemical indicator of inadequate oxygenation in critically ill patients\(^\text{22}\).

Understanding the complex process of tissue lactate production and utilization is important to understand the usefulness and potential limitations of monitoring blood lactate levels.

Gastrointestinal tonometry: Measurement of gastric mucosal pH has emerged as an attractive option for diagnosing and monitoring of splanchnic hypoperfusion and relevance for predicting the morbidity in critically ill patients. Gastrointestinal tonometry measures gut mucosal PaCO\(_2\), a clinically useful variable that is sensitive to alterations in splanchic perfusion and oxygenation\(^\text{23}\). This measurement might provide an insight that is among the first to develop an inadequacy of tissue oxygenation in circulatory shock and is last to be restored by resuscitation.

Near infrared spectroscopy: This is a continuous noninvasive monitoring aid works on the principles of light emission and absorption to determine tissue oxygenation\(^\text{24}\). Its’ use in a critical care setting is still under trial.

Table 3: Changes in hemodynamic variables in different critical care settings

<table>
<thead>
<tr>
<th>Sl. No.</th>
<th>Diagnosis</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Constrictive pericarditis</td>
<td>RAP = PAOP</td>
</tr>
<tr>
<td>2</td>
<td>RV infarction following Acute M1</td>
<td>Increased RAP with Normal PAOP</td>
</tr>
<tr>
<td>3</td>
<td>Post M1 VSD</td>
<td>&gt;7% difference in O(_2) saturation between RA and PA</td>
</tr>
<tr>
<td>4</td>
<td>Acute MR</td>
<td>Giant V wave in PA Wedge tracing</td>
</tr>
<tr>
<td>5</td>
<td>Pericardial tamponade</td>
<td>RAP = PAOP with blunted Y descent.</td>
</tr>
<tr>
<td>6</td>
<td>Chronic heart failure</td>
<td>Low SVR despite Low CO and hypotension</td>
</tr>
</tbody>
</table>

RAP, right atrial pressure; RV, right ventricle; PAOP, pulmonary artery wedge pressure; MI, myocardial infarction; VSD, ventricular septal defect; RA, right atrium; SVR, systemic vascular resistance; CO, cardiac output

Table 4: Expected changes in hemodynamic parameter in different shock states

<table>
<thead>
<tr>
<th>Shock states</th>
<th>CO/SV</th>
<th>SVR</th>
<th>PVR</th>
<th>CI</th>
<th>SVO(_2)</th>
<th>RAP</th>
<th>RVP</th>
<th>PAP</th>
<th>PAOP</th>
<th>Preload</th>
<th>SVV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic</td>
<td>↓</td>
<td>↑</td>
<td>N</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Septic</td>
<td>Variable</td>
<td>↓</td>
<td>N/↑</td>
<td>N/↑</td>
<td>N/↑</td>
<td>N/↑</td>
<td>N/↑</td>
<td>N/↑</td>
<td>Variable</td>
<td>N/↑</td>
<td></td>
</tr>
<tr>
<td>Hypovolemic</td>
<td>↓</td>
<td>↑</td>
<td>N</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
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<td></td>
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<tr>
<td>Obstructive</td>
<td>↓</td>
<td>↑</td>
<td>N</td>
<td>↑</td>
<td>↓</td>
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</tr>
</tbody>
</table>

CO, cardiac output; SVR, systemic vascular resistance; SV, stroke volume; PVR, pulmonary vascular resistance; CI, cardiac index; SVO\(_2\), mixed venous oxygen saturation; RAP, right atrial pressure; RVP, right ventricular pressure; PAP, pulmonary artery pressure; PAOP, pulmonary artery wedge pressure; SVV, stroke volume variation
NEVER FORGET THE FOLLOWING PRINCIPLES OF HEMODYNAMIC MONITORING IN ICU

- Hemodynamic monitoring can only improve if monitoring device is sufficiently accurate to able to influence therapeutic decision making, produce relevant data pertinent to the patient concerned.

- The optimal monitoring system is to be individualized. Even CVP and arterial waveform analysis are sufficient enough to manage volume in the absence of arrhythmia.

- PAC is not a good option during initial resuscitation but can be reserved for complex cases; where monitoring of SVO₂, CI and CO plays a key role in management.

- As the optimal hemodynamic values vary according to the age and pre-existing cardio- respiratory problem, keeping the targets like MAP above 65 mmHg, CVP>8 mmHg, and DO₂>600ml/min/m² can be potentially dangerous.

- Multiple hemodynamic variables to be integrated while managing a complex patient

- One should remember that increasing the CO and SVO₂ by increasing fluid administration is not always good. It may lead to massive edema and worsen outcome.

- Continuous measurement and measurement of a trend over time is more important than a single reading.

SUMMARY

Hemodynamic monitoring is accepted as standard practice in critical care settings throughout the world. Because of risks and costs associated with invasive techniques, a large fraction of high risk patients are left untreated. Advances in medical microelectronics and the advent of device-based diagnostic have enabled long term ambulatory monitoring of critically ill patients. As technology evolves, devices will likely to continue to miniaturize while their capabilities grow. Future directions for implantable hemodynamic monitors may come up for better management of critically ill patients. Implantable hemodynamic monitors can detect other clinically significant events such as poorly tolerated arrhythmias or hemodynamic shifts that may be affecting patients and were previously unappreciated.

REFERENCES


