Minimally invasive hemodynamic monitoring for the intensivist: Current and emerging technology

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Objective: To review minimally invasive cardiac output monitoring devices currently available for use in the intensive care unit.

Data Sources: Medline search from 1966 to present plus cited reference studies and abstracts from available product literature.

Study Selection: Selection criteria included published reports and abstracts comparing the accuracy of minimally invasive cardiac output monitors to a “gold standard.”

Data Synthesis: Many reports have been published on the accuracy of individual minimally invasive cardiac output monitors, but cumulative data reviewing each type of monitor have not been synthesized and made available to the clinician.

Conclusions: Emerging noninvasive or minimally invasive means of cardiac output monitoring are based on varied physiologic principles and can be used for following hemodynamic trends. Each of these methods has advantages and disadvantages; it is important for the clinician to understand the strengths and limitations of each device to effectively use the information derived. (Crit Care Med 2002; 30:2338–2345)

Monitoring of physiologic variables comprises an integral part of the care of the critically ill patient and assists the intensivist in both diagnostic and treatment strategies. There has been much debate in the literature regarding the usefulness and safety of invasive hemodynamic monitoring in the intensive care unit (ICU). Several studies have shown improved outcomes from hemodynamic monitoring in high-risk surgical patients, but there is conflicting evidence as to benefits in the critically ill medical patient (1, 2). A recent retrospective study suggested that the use of a pulmonary artery catheter was associated with increased mortality rate of 39% compared with patients who did not receive the catheter (3). In 1997, a consensus statement was published which concluded that there was no basis for a proposed Food and Drug Administration moratorium on pulmonary artery catheter use (4). Complications associated with invasive techniques have led to growing interest in the development of newer noninvasive or minimally invasive means of hemodynamic monitoring including applications of the Fick principle, Doppler technology, thoracic-electrical bioimpedance, and pulse contour devices. The purpose of this review is to summarize the current less invasive technologies available, the physiologic principles underlying their applications, and the relative advantages and limitations associated with these devices. We have focused on technologies that can be readily used by the intensivist without requiring additional specialized training such as echocardiography. An improved understanding of these emerging technologies will assist the intensivist in applying the appropriate device to his or her particular practice setting.

Indirect Fick Method. Before we describe the indirect Fick method for determining cardiac output, it will be useful to review the Fick principle. Adolf Fick first introduced this concept in 1870 when he said, “the total uptake or release of a substance by an organ is the product of the blood flow to the organ and the arteriovenous concentration of the substance.” This is simplistically translated into “rate of indicator out equals rate of indicator in plus rate of indicator added.” This is represented mathematically by

\[
CO = \frac{V_O_2}{C_aO_2 - C_vO_2} \quad [1]
\]

where CO is cardiac output, \(V_O_2\) is oxygen consumption, \(C_aO_2\) is arterial oxygen content, and \(C_vO_2\) is mixed venous oxygen content.

Since the Fick principle can be used with a multitude of indicators and can be used if the indicator is added or removed, another plausible indicator is \(C_vCO_2\). Substituting \(CO_2\) for oxygen in Equation 1 yields the indirect Fick equation

\[
V_{CO_2} \quad \text{the clearance of } CO_2, \quad C_vCO_2 \quad \text{the mixed venous content of } CO_2, \quad \text{and } C_aCO_2 \quad \text{is the arterial content of } CO_2.
\]

\[
V_{CO_2} \quad \text{can be calculated by the difference in } CO_2 \text{ content between expired and inspired gasses. } C_aCO_2 \text{ can be obtained from arterial blood gas or estimated from end-tidal } CO_2 \text{ (in healthy subjects with no diffusion abnormalities, alveolar } CO_2 \text{ approximates arterial } P_aCO_2). \quad C_vCO_2 \text{ is much more difficult to get noninvasively.}
\]

A partial rebreathing technique has been used to eliminate the need to directly measure \(C_vCO_2\). By taking advantage of a mathematical technique known as the law of ratios, the clinician can manipulate the indirect Fick equation such that a measurement of \(C_vCO_2\) is not necessary to calculate cardiac output (Fig. 1).

The rebreathing values are obtained by introducing an additional 150 mL of dead space into the ventilator circuit and taking measurements once a new equilibrium has been established. Assuming that the mixed venous \(CO_2\) concentration...
does not change significantly throughout the rebreathing period, the terms associated with $CvCO_2$ cancel each other out of the equation and are not needed for the calculation.

There are several technical problems with the indirect Fick technique. First, the difference between $PvCO_2$ and $PaCO_2$ is usually only about 6 mm Hg; consequently, small errors in the measurement of either of these values results in a large change in calculated cardiac output. Second, the relationships assumed are only valid when the $PaCO_2$ is $\geq$ 30 torr when the $CO_2$-hemoglobin dissociation curve is linear (5). If the patient hyperventilates and the $PaCO_2$ is $<$ 30 torr, the relationship is no longer valid. Third, shunted blood is not measured. Fourth, changes in mechanical ventilator settings that alter dead space or ventilation/perfusion relationships may produce a calculated alteration in cardiac output when in fact none has occurred.

Table 1 summarizes the available experimental data for cardiac output determinations with this method (6–11). The partial rebreathing technique gives a better approximation of cardiac output in patients who are less critically ill and have normal alveolar gas exchange. In the study by Gama de Abreu et al. (6), sheep with severe lung injury were used, and the comparison to thermodilution cardiac output was relatively poor. Based on these results, this technique is probably best suited for monitoring trends in critically ill patients with stable lung function rather than diagnostic interpretation. Currently, one device is commercially available in the United States (NICO Sensor, Novametrix Medical Systems, Wallingford, CT).

### Esophageal Doppler Monitoring

Doppler techniques have been employed in the suprasternal, transgastric, and transesophageal locations to estimate cardiac output noninvasively. The transesophageal approach is readily available to the intensivist and offers a number of advantages including close proximity to the descending aorta and stability of the probe within the esophagus. The use of esophageal Doppler monitoring (EDM) to measure cardiac output noninvasively was first described in 1971 and later refined in 1989 (12, 13). The technical basis for this technique is the concept that flow in a cylinder is equal to the area of the cross-section of the cylinder times the velocity of fluid in the cylinder. The cross-sectional area of a cylinder is equal to the area of a circle or $\pi r^2$ (2). In the case of aortic blood flow, the movement of blood is pulsatile and the velocity changes with time. Thus, the velocity can be characterized by the area under the velocity-time curve between two points in time (Fig. 2).

The area under the curve can be computed mathematically as the integral of the derivative of volume over time (dV/dt) from T1 to T2, where T1 represents the onset of flow and T2 represents the end of flow. This value is termed the time-integrated velocity. Stroke volume (SV) is calculated by multiplying the cross-sectional area by the time-integrated velocity. Once SV is known, cardiac output can be easily determined by the relationship, $CO = heart rate (HR) \times SV$.

With Doppler technique, the velocity of blood flow across the aortic valve or in the descending aorta can be measured. In EDM, the Doppler probe measures flow in the descending aorta. The probe is approximately the size of a nasogastric tube and can be placed noninvasively with a similar technique to placing a nasogastric tube.

### Table 1. Partial rebreathing vs. thermodilution (comparative studies)

<table>
<thead>
<tr>
<th>Author</th>
<th>n</th>
<th>Patients</th>
<th>Comparison</th>
<th>Bias, L/min</th>
<th>Precision, L/min</th>
<th>r</th>
<th>r²</th>
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<td>Gama de Abreu et al.</td>
<td>20</td>
<td>Sheep ARDS</td>
<td>PATD</td>
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<td>Kuck et al.</td>
<td>36</td>
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<td>.51</td>
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<td>Kuck et al.</td>
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<td>Loeb et al.</td>
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<td>Watt et al.</td>
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<td>PATD</td>
<td>-0.19</td>
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<td>.51</td>
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</table>

ARDS, acute respiratory distress syndrome; PATD, pulmonary artery thermodilution; CABG, coronary artery bypass graft.

Figure 1. Partial CO₂ rebreathing differential Fick equation.

Figure 2. Determination of stroke volume in esophageal Doppler monitoring. $dV/dT$, derivative of volume over time; $T_1$, onset of flow; $T_2$, end of flow.

Some technical problems can limit the accuracy of cardiac output measurements by esophageal Doppler monitoring. The first thing to consider is that the descending aorta only receives a portion of the cardiac output, and the CO value derived from EDM is only an estimate of cardiac output based on descending aortic blood flow. Therefore, a correction factor must be added to account for this discrepancy. Another important consideration is the importance of positioning...
of the probe to get accurate measurements. To have a good approximation of velocity, the Doppler beam should be within 20° of axial flow. The accuracy of the cross-sectional area estimation is crucial to the calculation of cardiac output because any error in r is squared before it is used in the final equation. Also, the assumption that the aorta is cylindrical is not always valid. The cross-sectional area of the aorta is actually dynamic and is dependent on the pulse pressure and aortic compliance. Furthermore, flow in the aorta is not always laminar. Conditions such as tachycardia, anemia, and aortic valve disease can cause turbulent aortic blood flow and alter velocity measurements.

There have been limited studies concerning the accuracy and clinical benefits of EDM. In 1998, Cariou and colleagues (14) found that aortic blood flow is proportional to cardiac output over a wide range of cardiac output values (r = .80) and that aortic diameter can be reliably measured with m-mode ultrasound when compared with transesophageal echocardiography. Measurements of cardiac output by EDM have been correlated with both thermodilution and Fick methods (Table 2) (15–21). Of note, all of these studies used a nomogram based on the patient’s age, gender, and weight to estimate cross-sectional area of the descending aorta. Although initial results are promising, more studies are needed to make a decision regarding the accuracy of this technique in critically ill patients. EDM-derived cardiac output using m-mode measurement of aortic diameter may yield more closely correlated values to thermodilution, but this has not yet been confirmed in clinical trials.

EDM allows the measurement of corrected flow time (FTc) as a measure of cardiac preload and peak flow velocity as a measure of contractility. The FTc is the systolic flow time corrected for heart rate expressed in milliseconds. In a series of critically ill surgical patients, the FTc correlated more strongly with cardiac output than pulmonary artery occlusion pressure (16). Additionally, the longest FTc has been shown to correlate with the optimal level of left ventricular filling in mechanically ventilated patients (22). The FTc was recently evaluated by using the end-diastolic short axis (derived from transesophageal echocardiography) as the “gold standard.” In 34 patients undergoing coronary artery bypass grafting, the average correlation coefficient was .49 (23). Clinically, the EDM has proven beneficial for perioperative monitoring as a means to decrease morbidity in elective femur fracture fixation (24) and has been reported to yield beneficial information for treatment of sepsis in humans (25). EDM is well suited for the ICU and can be applied to a wide spectrum of patients with few contraindications (e.g., severe agitation, esophageal malignancy/perforation, severe bleeding diathesis, or aortic dissection). However, it is limited by the variability that can occur due to probe positioning, and additional training is required to ensure proficiency (26). Several commercially available devices estimate cardiac output by using Doppler esophageal monitoring technology: the CardioQ (DelteX Medical, Branford, CT), the Oesophageal Doppler Monitor II (Abbot Laboratories, North Chicago, IL), and the Dynemo 3000 (Sometec, Paris, France), which uses a nomogram to predict aortic diameter. The Hemosonic 100 (Arrow International, Reading, PA) uses m-mode ultrasound to measure aortic diameter.

### Table 2. Esophageal Doppler monitor vs. “gold standard”

<table>
<thead>
<tr>
<th>Author</th>
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<th>Patients</th>
<th>Comparison</th>
<th>Bias, L/min</th>
<th>r</th>
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<td>CABG</td>
<td>EM</td>
<td>.765</td>
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<td>Madan et al. (16)</td>
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<td>SICU</td>
<td>TD</td>
<td>.6</td>
<td></td>
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<tr>
<td>Valiente et al. (17)</td>
<td>46</td>
<td>MV</td>
<td>TD</td>
<td>.24</td>
<td>.95</td>
</tr>
<tr>
<td>Ballard et al. (18)</td>
<td>10</td>
<td>ICU</td>
<td>CCO</td>
<td>-.01</td>
<td></td>
</tr>
<tr>
<td>Leather and Wouters et al. (19)</td>
<td>14</td>
<td>OR</td>
<td>TD</td>
<td>-.89</td>
<td></td>
</tr>
<tr>
<td>Bernardi et al. (20)</td>
<td>22</td>
<td>MICU</td>
<td>TD</td>
<td>.92</td>
<td></td>
</tr>
<tr>
<td>Cuschieri et al. (21)</td>
<td>10</td>
<td>SICU</td>
<td>Fick</td>
<td>.846</td>
<td>.811</td>
</tr>
</tbody>
</table>

CABG, coronary artery bypass graft; EM, electromagnetic flowmetry; SICU, surgical intensive care unit; TD, pulmonary artery thermodilution; MV, mechanically ventilated patients; ICU, intensive care unit; CCO, continuous pulmonary artery thermodilution; OR, operating room; MICU, medical intensive care unit; Fick, calculated by direct Fick.

**Thoracic Electrical Bioimpedance**

Thoracic electrical bioimpedance (TEB) is another noninvasive means by which cardiac output can be estimated. The thoracic bioimpedance is the electrical resistance of the thorax to a high-frequency, very low-magnitude current. This measure is indirectly proportional to the content of thoracic fluids such that as the amount of thoracic fluid increases, the TEB decreases. Therefore, total fluid conductivity (TFC) is equal to the inverse value of TEB (1/TEB). Changes in cardiac output may be reflected as a change in overall bioimpedance or TFC. In this technique, six electrodes are placed on the patient: two in the upper thorax/neck area and four in the lower thorax. The electrodes not only detect changes in bioimpedance; they also monitor electrical signals from the heart. The measurement of changes in TEB to estimate SV was originally described by Kubicek in the 1960s

\[ SV = \frac{L}{Z_p} \times \left( \frac{Z}{VT \times (dZ/dt_{max})} \right) \]

where p is the resistivity of blood (ohm-cm), L is the distance between the two inner voltage-sensing electrodes (cm), Zp is the mean thoracic impedance between signals from the heart. The measurement of changes in TEB to estimate SV was originally described by Kubicek in the 1960s.

This equation was later modified by Bernstein (28) to account for the noncylindrical shape of the thorax. Bernstein (28) also introduced a calibration factor that compensated for variation in gender and degree of obesity. Currently available bioimpedance hemodynamic monitors use some derivation of this calculation such that SV is derived according to the following logic:

1. The rate of TEB change over time (dZ/dt) corresponds to change in aortic blood flow (assuming other factors affecting impedance do not change between times the measurements were made).
2. (dZ/dt)max corresponds to peak aortic blood flow.
3. Ejection phase contractility index (EPCI) = (dZ/dt)max × TFC.
4. Ventricular ejection time (VET) can be...
measured from the distance between the QRS intervals on the electrocardiogram sensing electrodes.

5. The volume of electrically participating tissues (VEPT) is estimated from tissue water volume, volumetric changes in pulmonary and venous blood induced by respiration, and volumetric changes in aortic blood flow produced by myocardial contractility. Accurate measurements of cardiac output in aortic blood flow depend on the ability to accurately measure the third determinant while filtering out the “noise” produced by the first two determinants. This technique is very sensitive to any alteration in position or contact of the electrodes to the patient. Thus, the clinician must preferably use the same electrode and electrode position between measurements and attempt to minimize conditions that may interfere with electrode contact such as perspiration. Also, the accurate measurement of VEPT relies on a constant R-R interval. In patients with atrial arrhythmias such as frequent premature atrial contractions or atrial fibrillation/flutter, errors in measurement of VEPT can lead to significant errors in measurements of cardiac output. Importantly, any acute change in tissue water content, such as pulmonary edema, pleural effusions, or chest wall edema, can alter bioimpedance readings irrespective of any changes in cardiac output.

Many studies have examined the accuracy of thoracic electrical bioimpedance, including a recent meta-analysis in 1999 (29). This analysis reviewed 154 studies of thoracic bioimpedance versus a gold standard. The results related to accuracy are summarized in Table 3. The authors’ conclusions were that TEB was probably useful for trend analysis but not accurate enough for diagnostic interpretation and that caution should be taken when the bioimpedance method is used with cardiac patients. Another study examined a large cohort of trauma patients, medical intensive care patients, and surgical intensive care patients \( n = 2,192 \) and found an overall correlation of \( r = .85 \) (30). Interestingly, when patients with severe pulmonary edema, pleural effusions, or excessively high TFC were excluded, the correlation was \( r = .93 \). This sub-group comprised 8% of the total cohort.

TEB is the most noninvasive method of estimating cardiac output. This property can be particularly useful in the ICU setting when caring for patients with relative or absolute contraindications to more invasive methods or when arterial venous access is problematic. However, TEB provides a less accurate estimate of cardiac output in patients with significant thoracic fluid overload such as pulmonary edema, pleural effusions, or massive peripheral edema (30). The prevalence of these conditions in the ICU will limit TEB use in that setting. When TEB is used in the ICU, the clinician should be mindful of the presence of these conditions and consider using an alternative method of hemodynamic monitoring. Many commercially available products incorporate bioimpedance technology. Products currently marketed for use in the ICU include BioZ (Cardiodynamics, San Diego, CA), IQ System (Reenaissance Technologies, Newton, PA), and CircMon (JR Medical, Estonia).

### Transpulmonary Cardiac Output

Cardiac output can also be measured noninvasively by using the transpulmonary thermodilution technique. Like pulmonary artery (PA) thermodilution, this method uses the Stewart-Hamilton equation to estimate cardiac output (see Equation 5).

\[
CO = \frac{(Ta - Tb) \times Vi \times K}{dT/dt} \quad [5]
\]

where \( CO \) is cardiac output, \( Ta \) is temperature before injection, \( Tb \) is temperature after injection, \( Vi \) is volume of injectate, \( K \) is a constant, and \( dT/dt \) is change in temperature per change in time.

Cold injectate is administered intravenously (usually in the central circulation), and the change in temperature is detected in the arterial system. There are several key differences between the transpulmonary technique and the pulmonary artery technique. First, the transpulmonary method does not require the insertion of a pulmonary artery catheter and is therefore less invasive. This may equate to a lower complication rate. Second, transpulmonary thermodilution measures left-sided cardiac output, and PA thermodilution measures right-sided cardiac output. In most circumstances, the left-sided CO approximates the right-sided CO; however, there are times when these values theoretically can diverge, for example, during positive pressure ventilation. Finally, transpulmonary thermodilution may be less dependent on respiratory variation given the difference in proximity to the thorax.

Several clinical validation studies have compared transpulmonary thermodilution to a gold standard (Table 4) (31–34). These data suggest a good correlation between transpulmonary thermodilution and PA thermodilution. As one can see in Table 3, transpulmonary \( CO \) values are often greater than corresponding PA thermodilution values. The reason for this is not entirely clear. Proposed mechanisms include loss of indicator (cold) with transpulmonary and/or cold-in-
duced reduction in HR leading to decreased CO in the right heart compared with the left heart (33). Differences may also be partially explained by differences in sensitivity to respiratory variation, which would affect the PA thermodilution method to a greater extent. It appears that the correlation holds true for critically ill patients (33); however, confirmation studies are needed in an extended spectrum of clinical conditions.

**Pulse Contour Analysis**

In 1983, Wesseling and colleagues (35) developed an algorithm based on arterial pulse contour analysis to continually monitor CO. It is based on the concept that the contour of the arterial pressure waveform is proportional to SV, which can be estimated by the integral of the change in pressure from end diastole (t0) to end systole (t1) over time. The estimate of SV is also influenced by the impedance of the aorta (Z)

$$SV = \frac{\int_{t0}^{t1} \frac{dP}{dt} \, dt}{Z} \quad [6]$$

Z is dependent on the CO and the individual elastic/mechanical properties of the aorta at that particular time (bolus effect). To determine what the individual impedance is at any one point, CO must be determined by another method and used to calibrate the pulse contour device. This can be accomplished by a transpulmonary thermodilution method.

Pulse contour analysis has been extensively studied, and there are many available validation studies (Table 5) (36–39). In the study by Buhre et al. (36), the close correlation between pulse contour CO and PA thermodilution remained despite significant changes in CO induced by esmolol. In another study, pulse contour was compared with PA thermodilution and continuous CO by heater probe (40).

The results were documented before and after administration of phenylephrine. The close correlation between values obtained by continuous CO and PA thermodilution persisted after phenylephrine administration, but the values obtained by pulse contour and PA thermodilution diverged after the administration of vasodilator. This discrepancy suggests loss of correlation with significant changes in hemodynamics.

Most studies of the pulse contour method show excellent correlation with PA thermodilution, including patients with acute respiratory distress syndrome (37). There are conflicting studies as to the influence of significant changes in SVR that may occur after calibration (36, 40). Frequent recalibration during times of hemodynamic instability will minimize such errors. Currently available commercial devices require manual input of a value for central venous pressure. The location of the arterial sensing catheter in pulse contour analysis is an important consideration. Although the manufacturer of PICCO (Pulsion Medical, Munich, Germany) recommends placement in either the axillary or femoral position, the clinical validation studies for pulse contour were done with the arterial catheter in the femoral position. The accuracy of pulse contour seems to lessen when the arterial waveform analysis is obtained from a peripheral location such as the finger (41, 42). Use of the arterial sensing catheter in the radial position has not been clinically validated. The requirement of a proximal arterial catheter causes the pulse contour device to be more invasive than the other techniques described and may limit its usefulness.

Pulse contour devices also allow the measurement of global end diastolic volume (GEDV) to approximate intrathoracic blood volume (ITBV) and extravascular lung water (EVLW) as a surrogate for cardiac preload. ITBV and EVLW have traditionally been measured by the double indicator technique (thermodilution and indocyanine green) via a pulmonary artery catheter. ITBV is calculated by the product of CO and the mean transit time (MTT) of a plasma-bound indicator (ITBV = CO × MTT). With the less invasive transpulmonary technique, intrathoracic thermal volume (ITTV) is calculated by the product of CO and MTT of cold injectate. Also, the pulmonary thermal volume (PTV) is derived from the relationship PTV = CO × t, where t is the exponential decay time. This relationship assumes that the majority of temperature decay occurs in the largest mixing chamber (PTV; Fig. 3).

Once values for ITTV and PTV are obtained, they can be used to calculate GEDV from the equation GEDV = ITTV − PTV. A linear relationship has been found to exist between ITBV and GEDV (43–45) whereby ITBV = 1.25 × GEDV (this is an approximation based on the average of several trials). Also, EVLW can be calculated by subtracting ITBV from ITTV (EVLW = ITTV − ITBV).

Using EVLW to guide fluid management in medical intensive care patients has been suggested to reduce the duration of mechanical ventilation and length of stay in the ICU (44). The ITBV has been suggested to be a better indicator of cardiac preload than pulmonary artery occlusion pressure (PAOP) and central venous pressure (44–46). In mechanically ventilated patients, this may be due to an artificial elevation of PAOP and central venous pressure from increased airway pressure. Lichtwarck-Aschoff et al. (46) demonstrated a correlation between change in ITBV and change in cardiac pressure.

![Figure 3. Blood volumes in measurement of global end diastolic volume](image)

### Table 5. Pulse contour vs. pulmonary artery thermodilibration

<table>
<thead>
<tr>
<th>Author</th>
<th>n</th>
<th>Patients</th>
<th>Comparison</th>
<th>r</th>
<th>Bias, L/min</th>
<th>Precision, L/min</th>
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</thead>
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<tr>
<td>Buhre et al. (36)</td>
<td>36</td>
<td>MIDCAB</td>
<td>PATD</td>
<td>.94</td>
<td>0.003</td>
<td>1.26</td>
</tr>
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<td>Zoliner et al. (37)</td>
<td>160</td>
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<td>PATD</td>
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<td>0.03</td>
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<td>.88</td>
<td>0.31</td>
<td>1.25</td>
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</table>

MIDCAB, minimally invasive coronary artery bypass; PATD, pulmonary artery thermodilibration; ARDS, acute respiratory distress syndrome.
Lithium Dilution Cardiac Output

As discussed earlier, pulse contour devices must be calibrated with a CO derived from another source (e.g., transpulmonary thermodilution). They also can be calibrated by using lithium dilution. In this technique, lithium chloride is injected into a central or peripheral venous catheter, and lithium is measured with a lithium-sensitive electrode in the peripheral arterial system. The electrode is outside the artery and thus requires withdrawal of a small (3-mL) sample of blood with each measurement. The change in voltage across a semipermeable membrane is related to the change in lithium concentration. A correction for serum sodium concentration is needed due to low selectivity of the membrane for lithium over sodium. A dilution curve for lithium is constructed, and cardiac output is calculated according to

$$\text{CO (L/min)} = \frac{\text{LiCl} \times 60}{\text{Area} \times (1 - \text{PCV})}$$

where LiCl is the dose of lithium chloride in mmol, Area is the area under the lithium-time dilution curve, and PCV is the packed cell volume, which is derived from the hemoglobin concentration.

Once the CO measurement is obtained, that value can be used to calibrate a pulse contour device.

Multiple trials have been done to study the accuracy of lithium dilution CO compared with various reference gold standards (Table 6) (50–52). Kurita et al. (53) found good correlation between lithium dilution and electromagnetic flowmeter-derived CO in the setting of hemodynamic changes induced by dobutamine and propanolol in an animal model. Data presented by Garcia-Rodriguez et al. (54) suggest that no accuracy is lost when the lithium is injected via a peripheral catheter.

Cardiac output values derived from lithium dilution correlate well with pulmonary artery thermodilution and transpulmonary thermodilution measurements (50–52). Compared with the thermodilution-derived pulse contour technique, lithium dilution cardiac output is less invasive because it does not require central circulation catheterization; however, it offers the clinician no direct assessment for cardiac preload. Also, the 3-mL blood draw required for each calibration may contribute to anemia and increase blood product transfusions. Although the lithium dilution method seems to perform well with only peripheral access, no accurate measurement of central venous pressure and thus SVR can be made without central venous catheterization. The lithium dilution method may be useful for the patient in whom cardiac output monitoring is required without a need for directly measured cardiac preload. For example, it could be useful in distinguishing high cardiac output versus low cardiac output in a patient with unexplained hypotension. Currently, there is one commercially available lithium dilution cardiac output monitor (LiDCO; LiDCO Ltd, London, UK).

**CONCLUSIONS**

Many methods are available to noninvasively measure cardiac output. The characteristics of each technique re-

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**Table 6. Lithium dilution cardiac output vs. pulmonary artery thermodilution**

<table>
<thead>
<tr>
<th>Author</th>
<th>n</th>
<th>Patients</th>
<th>Comparison</th>
<th>r</th>
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<td>Age 5days-9yrs</td>
<td>TPTD</td>
<td>.98</td>
<td>−0.1</td>
<td>0.3</td>
</tr>
</tbody>
</table>

PATD, pulmonary artery thermodilution; TPTD, transpulmonary thermodilution.

**Table 7. General features of available minimally invasive cardiac output technologies**

<table>
<thead>
<tr>
<th>Method</th>
<th>Accuracy</th>
<th>Estimate of Cardiac Preload</th>
<th>Special Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indirect Fick</td>
<td>+ + + +</td>
<td>No</td>
<td>Intubated, accuracy limited by cardiopulmonary disease</td>
</tr>
<tr>
<td>EDM</td>
<td>+ + +</td>
<td>Yes (FTc)</td>
<td>Patient movement, specialized training</td>
</tr>
<tr>
<td>TEB</td>
<td>+ + +</td>
<td>No</td>
<td>Decreased accuracy with abnormal cardiac rhythm, severe peripheral edema</td>
</tr>
<tr>
<td>Transpulmonary/pulse contour</td>
<td>+ + + +</td>
<td>Yes (ITBV)</td>
<td>Requires proximal arterial access</td>
</tr>
<tr>
<td>Lithium dilution</td>
<td>+ + + +</td>
<td>No</td>
<td>Does not require central circulation catheterization</td>
</tr>
</tbody>
</table>

*Compared with pulmonary artery thermodilution (highest accuracy corresponds to + + + + and lowest accuracy corresponds to + ).

EDM, esophageal Doppler monitor; FTc, corrected flow time; TEB, thoracic electrical bioimpedance; ITBV, intrathoracic blood volume.
It is important for the clinician to understand the strengths and limitations of each device to effectively use the information derived.

viewed are summarized in Table 7. In an era of questionable utility and safety of the invasive pulmonary artery catheter, a safe and reliable means by which to measure cardiac output more noninvasively in critically ill patients is welcomed. In general, most methods are based on sound physiologic principle and can be used for following hemodynamic trends. The indirect Fick methods are convenient and relatively easy to apply to mechanically ventilated patients but may not be accurate enough for initial diagnostic information in a patient with significant lung disease or multiorgan failure. The esophageal Doppler monitor, although slightly more invasive and operator dependent than others, is associated with low risk and may be a better alternative for the critically ill patient. More studies are needed to document accuracy of this method by using m-mode ultrasound for measurement of aortic diameter. The bioimpedance methods tend to lose accuracy in the setting of intrathoracic fluid shifts such as occur in acute respiratory distress syndrome, congestive heart failure, peripheral edema, or pleural effusions, which may limit its use in the intensive care setting. The pulse contour devices offer a heat-to-beat measurement of cardiac output and have shown good correlation with pulmonary artery thermodilution during times of stable hemodynamics. These devices should be recalibrated frequently in patients with unstable hemodynamics. Pulse contour devices also allow estimation of ITBV to assess cardiac preload. In some cases, ITBV may be more useful for volume resuscitation than the traditional pulmonary artery catheter derived PAOP (55). Each of these methods has advantages and disadvantages; it is important for the clinician to understand the strengths and limitations of each device to effectively use the information derived. Intensivists work in a rapidly changing environment and must be able to critically evaluate emerging technologies before widespread application in patients. In a time when the pulmonary artery catheter is coming under scrutiny due to safety and accuracy concerns, less invasive devices may be an appropriate alternative.

REFERENCES