The bedside assessment of cardiac performance and ventricular preload is perhaps one of the most difficult and yet vitally important problems in critical care medicine. The traditional clinical signs of cardiac function such as BP, urine output, jugular venous distension, skin perfusion, and skin turgor are unreliable in the ICU setting. This is illustrated by the studies of Connors et al\textsuperscript{1} and Fein and colleagues,\textsuperscript{2} who demonstrated that in the majority of instances, critical care staff were unable to correctly predict a patient’s hemodynamic profile from clinical examination alone.

According to the Frank-Starling principle, the vigor of cardiac contraction relates directly to muscle fiber length at end-diastole.\textsuperscript{3} This presystolic fiber stretch, or preload, is proportionate to end-diastolic volume.\textsuperscript{4} Left ventricular end-diastolic volume (LVEDV; preload) is therefore a major factor determining cardiac output (CO).\textsuperscript{3} In order to make rational management decisions in terms of fluid and vasoactive drug therapy, the intensivist needs to correctly assess the patient’s preload. An accurate knowledge of preload is essential in determining the adequacy of fluid resuscitation. The clinician needs to be able to predict the change in CO in response to a fluid challenge (i.e., recruitable CO). In addition, an estimation of CO is essential in patients with evidence of inadequate tissue perfusion.\textsuperscript{5} Ideally, the technology that provides these hemodynamic parameters should be noninvasive, accurate, reliable, and continuous. Currently, no single monitoring tool meets all of these criteria. Thermodilution CO combined with radionuclide ejection fraction (and the calculation of LVEDV) is the most accurate method of determining cardiac performance in the ICU. However, radionuclide cardiac imaging in the ICU is essentially a research tool with many limitations.
Although the risk/benefit ratio of pulmonary artery catheterization has yet to be determined, this procedure has become one of the most common procedures performed in critically ill patients around the world.\textsuperscript{6–11} This paper will outline the utility and limitations of the pulmonary artery catheter (PAC) and the emerging role of esophageal Doppler monitoring in assessing cardiac performance in the ICU.

**The PAC**

When faced with a critically ill patient with hemodynamic instability, the question that is often posed is whether a PAC should be inserted. There is little scientific data to help answer this question. It is clear that the inappropriate use and poor understanding of the PAC leads to excessive mortality.\textsuperscript{9,12–14} It is also evident that the PAC is a very useful diagnostic tool that aids in the management of critically ill patients. PACs should therefore only be used by physicians who have extensive experience in their use. Furthermore, the data must be interpreted in the context of the clinical scenario. Too often, the attending physician reviews the patient’s “numbers” without ever setting eyes on the patient.

**Thermodilution CO**

There is no true reference technique for the clinical determination of CO. The reproducibility and accuracy of the thermodilution method of CO determination has been compared with both the Fick method and the dye-dilution method.\textsuperscript{15–18} An analysis of this data reveals that the three methods are of equal merit and can be used as independent references.\textsuperscript{19} However, an assessment of the reproducibility of the thermodilution technique demonstrates that there must be a difference of at least 15\% between the mean of three CO determinations to be clinically significant.\textsuperscript{19}

**The Pulmonary Artery Occlusion Pressure and Preload**

Since its introduction to clinical medicine almost 30 years ago, the PAC has been assumed to be a reliable and valid indicator of left ventricular preload. Indeed, perhaps the most common reason for inserting a PAC in the ICU is to measure the pulmonary artery occlusion pressure (PAOP) in order to assess a patient’s “volume status.” However, it was not long after the introduction of the PAC that studies began to appear demonstrating that the PAOP was a poor reflection of preload.\textsuperscript{20,21} Despite the fact that this observation has been confirmed in a multitude of studies, many physicians still erroneously believe that the PAOP is useful in assessing a patient’s intravascular volume. This factor together with the incorrect interpretation of the PAOP may largely explain the excess mortality associated with the use of the PAC.\textsuperscript{9,12–14,22}

For the PAOP to be an accurate measure of LVEDV, all of the following criteria must be met: (1) a valid and accurate PAOP tracing is obtained; (2) the PAOP is correctly interpreted; (3) the PAOP is an accurate reflection of the left ventricular end-diastolic pressure (LVEDP); and finally, and most importantly, (4) there is a linear and predictable relationship between the LVEDP and the LVEDV. As will become evident, in most clinical situations it is rare for all of these criteria to be met, and the PAOP therefore becomes a very poor and misleading measure of left ventricular preload.\textsuperscript{23}

Morris and colleagues\textsuperscript{24} assessed the technical adequacy of 2,711 PAOP recordings. These authors reported that 31\% of these recordings were technically inadequate, resulting in unreliable readings. It should be appreciated that numerous factors, including improper positioning of the PAC, incorrect calibration and balancing of the transducer, and excessive damping may result in invalid PAOP values.\textsuperscript{23–25} Even if a valid PAOP waveform is obtained, it is likely that in as many as 50\% of cases, the PAOP will be incorrectly interpreted.\textsuperscript{12–14} Even among physicians with special qualifications in critical care medicine, there is often disagreement as to the correct interpretation of the PAOP tracing, resulting in large interobserver variability.\textsuperscript{22,26} These factors add to the unreliability of the PAOP reading.

The distending pressure resulting in left ventricular diastolic filling is the difference between the simultaneous intracavity pressure and the juxtacardiac pressure. A noncompliant ventricle or one surrounded by increased intrathoracic pressure requires a higher-than-normal intracavitary pressure to achieve any specified presystolic volume. Increased intrathoracic pressure associated with positive pressure ventilation and the positive end-expiratory pressure (PEEP) has a significant effect on juxta- cardiac pressure. A PEEP > 8 to 10 cm H\textsubscript{2}O increases juxta-cardiac pressure and, therefore, the pressure gradient between the left atrium and atmospheric pressure, but not the transmural distending pressure.\textsuperscript{27} This artifactually increases the PAOP.\textsuperscript{27} Formulas that subtract a percentage of the PEEP from the PAOP are of little practical value, because the fraction of the PEEP that is transmitted to the heart is difficult to estimate.\textsuperscript{27,28}

The use of the PAOP to measure left ventricular preload in absolute or relative terms assumes a direct relationship between the LVEDP and the LVEDV. This pressure-volume curve which describes left ventricular compliance is normally curvilinear. Fur-
thermore, alterations in left ventricular compliance shifts the pressure-volume curve. Factors that alter left ventricular compliance include left ventricular preload, left ventricular afterload, left ventricular mass, and ventricular fiber stiffness. Myocardial ischemia, sepsis, diabetes, obesity, aging, sustained tachycardia, dialysis, cardioplegia, as well as other factors alter myocardial fiber stiffness. In addition, the left ventricular pressure-volume curve is affected by the degree of right ventricular filling. Since the two ventricles are physically coupled by the interventricular septum and by the constraining effects of the pericardium, the end-diastolic pressure-volume curve of either ventricle is dependent on the diastolic volume of the other. In normal control subjects, volume unloading will result in a decrease in both the LVEDV and the right ventricular end-diastolic volume (RVEDV). However, in patients with pulmonary hypertension, a decrease in venous return with a reduction in the RVEDV may be associated with a paradoxical increase in the LVEDV. The increase in the LVEDV occurring in association with a decrease in the RVEDV is referred to as diastolic ventricular interaction. In critically ill patients, many of the factors that determine left ventricular compliance are in a state of dynamic flux, making it exceedingly difficult to estimate the LVEDV from the LVEDP. This alteration in the left ventricular pressure-volume curve is the major factor accounting for the poor relationship between the PAOP and the LVEDV. This observation was noted as early as 1975, when Baek and colleagues demonstrated a poor correlation between blood volume (as measured by an isotope technique) and the PAOP. Subsequently, Calvin and coworkers demonstrated a poor relationship between the LVEDV (as measured by radionuclide angiography) and the PAOP. Thys et al compared the PAOP to the LVEDV as determined by two-dimensional echocardiography. These authors found a poor correlation (r = 0.34) between these two variables. The value of preload determination in any individual patient, however, is being able to predict the change in CO in response to fluid loading (ie, recruitable CO). Numerous studies in diverse clinical settings have demonstrated that the PAOP is a very poor estimate of left ventricular preload and a poor predictor of the change in CO in response to a fluid challenge. From the available data, the PAOP must be regarded as an unreliable index of the LVEDV both in large patient groups as well as in individual patients assessed over time. The change in the PAOP in response to fluid loading reflects left ventricle compliance rather than providing an indication of the adequacy of left ventricular filling. The change in CO in response to fluid loading, however, provides an indication of the position of the ventricle on the Frank-Starling curve.

The Volumetric PAC

The use of a PAC with a rapid response thermistor and an ECG electrode allows recognition along the rewarming phase of the thermodilution curve of a series of plateaus that are produced by the pulsatile ejection of blood from the right ventricle. The temperature drop between two successive beats allows computation of the right ventricular ejection fraction (RVEF). Once the RVEF is known, the right ventricular end-systolic and end-diastolic volumes can be calculated from the stroke volume. Several groups of investigators have validated the RVEF measurements obtained by the thermodilution technique by comparing them with radionuclide imaging, echocardiography, and biplane angiography.

It has been suggested that the RVEDV index (RVEDVI) is a better indicator of preload in critically ill patients than the PAOP. Several groups of investigators have reported an excellent correlation between the RVEDV and cardiac index (CI), and they have found the RVEDV to be superior to the PAOP in determining the preload status of patients. However, some authors have suggested that the correlation between the RVEDV and CI remains when the variables are mathematically uncoupled or the CI is determined by the Fick method. For any given patient, the relationship between the RVEDV and CI will depend on right ventricular function. This was recently illustrated by the report of Cheatham and colleagues, who demonstrated that the correlation coefficient between RVEDVI and CI improved when it was stratified by RVEF.

The value of the RVEDV is the ability to predict the change in CO in response to a fluid challenge. Diebel and colleagues demonstrated the RVEDV to be an excellent predictor of recruitable CO, whereas the PAOP performed poorly. The optimal RVEDV will depend on right ventricular function (ie, RVEF), and it is likely that this value will change during the course of a patient’s illness. The optimal RVEDV has been reported to range from 90 to 140 mL/m². When a volumetric PAC is used, the optimal RVEDV should be determined by plotting RVEDVI against CI (see Figure 1). The volumetric PAC is particularly useful in determining the preload in patients who are being ventilated with PEEP, a setting in which the PAOP reading becomes uninterpretable.
Determination of Pulmonary Capillary Pressure

It has been assumed that the PAOP reflects the pulmonary capillary pressure (Pcap). This is based on the assumption that there is minimal resistance through the pulmonary veins, because, as the balloon is inflated and flow stops, resistance is no longer taken into account in the measurement of PAOP. In normal lungs, with minimal resistance in the pulmonary veins, this assumption may be correct and the PAOP may reflect the Pcap. However, various stimuli such as hypoxia and inflammatory mediators affect pulmonary arterial and venous resistance to varying degrees. Therefore, in the presence of increased pulmonary arterial and venous resistance, as in disorders such as ARDS and sepsis, there is no longer a consistent relationship between Pcap and PAOP.66 Pcap, not PAOP, is the driving pressure forcing fluid from the pulmonary microvasculature. Collee and colleagues67 reported a method of estimating the Pcap from the pulmonary artery pressure tracing with balloon occlusion. These authors identified two exponential pressure decay components, the slower one representing the discharge of the Pcap through the pulmonary venous resistance. By extrapolating this exponential to its origin at the moment of pulmonary artery occlusion, a pressure within the pulmonary vascular bed that approximates Pcap was identified (see Figure 2). Holloway et al68 validated this method in an animal model, where they demonstrated a close relationship between the Pcap estimated with this method and both the isogravimetric measurement and the Gaars mathematical estimate of microvascular pressure.69

Continuous Esophageal Doppler

The transthoracic Doppler is currently the most promising noninvasive technique for monitoring cardiac function in ICU patients. The esophageal Doppler was first described in 1971 and subsequently refined by Singer provides a minimally invasive means of continuously monitoring cardiac function in the ICU.70,71 When an ultrasound beam is directed at a column of flowing blood, the reflected sound wave will shift in frequency. The magnitude of this Doppler shift is directly proportional to the velocity of blood flow. Stroke volume can be calculated by multiplying this average blood velocity during a systolic cycle by the ejection time (stroke distance) and by the cross-sectional area through which the blood flows (see Figure 3).71 Doppler signals can be obtained with an ultrasound probe placed externally at the suprasternal notch and directed at the ascending aorta. However, esophageal Doppler monitoring has a number of advantages over the transcatheter approach. The close proximity of the descending aorta to the esophagus provides an excellent window for obtaining Doppler signals. Furthermore, once positioned, the transthoracic probe is stabilized by the esophagus, thereby permitting continuous monitoring. The cross-sectional area of the descending aorta can be estimated by nomograms based on the patient’s age, weight, and height. A correction factor is required to transform the blood flow measured in the descend-
ing thoracic aorta into a global CO. Despite these assumptions and potential sources of error, a good correlation has been demonstrated between the CO measured by esophageal Doppler and simultaneously by thermodilution and Fick methods.71–79 Recently, a transesophageal Doppler probe with an ultrasonic probe that allows the near simultaneous measurement of both the velocity of the descending aortic blood flow and the descending aortic diameter has been described.80 The aortic cross-sectional diameter as measured with this device has been reported to correlate closely with that determined by transesophageal echocardiography, and the descending aortic blood flow showed good agreement with the CO as measured by thermodilution.80

In contrast to the PAC, the probe of the esophageal Doppler monitor can be inserted within minutes, it requires minimal technical skill, and it is not associated with major complications.81–85 The probe has been reported to have been left in situ for > 2 weeks without complications.84 Lefrant and colleagues77 have demonstrated that a period of training involving no more than 12 patients is required to ensure reliability of CO measurements with esophageal Doppler. A major advantage of transesophageal Doppler is the ability to provide continuous real-time monitoring.

Esophageal Doppler monitoring would be of limited clinical utility if it only provided an estimate of CO. However, the characteristics of the Doppler flow-velocity waveform provide information on both cardiac preload and contractility (see Figure 3). The peak flow-velocity that is readily identified as the apex of the waveform is a good indicator of myocardial contractility. Furthermore, the left ventricular ejection time (or flow-time) corrected for heart rate provides an index of preload. Laboratory studies have demonstrated a good correlation between Doppler peak velocity and electromagnetic catheter-measured flow as well as measured contractility.85 In addition, an infusion of dobutamine in normal subjects has been shown to produce a dose-dependent increase in the peak velocity.86 Esmolol was shown to have the opposite effect. Singer and colleagues71,82 demonstrated a good correlation between the corrected flow time (Ftc) and changes in preload: when preload was increased from hypovolemic states, the Ftc increased, and when preload was decreased from normovolemic states, the Ftc decreased. Despite the potential advantages of continuous esophageal Doppler monitoring, the clinical experience with this technology is limited. Sinclair and colleagues87 recently demonstrated the clinical utility of continuous esophageal Doppler monitoring in patients undergoing proximal femoral fracture repair. In this prospective, randomized, and blinded study, those patients whose intraoperative volume status was optimized by the use of esophageal Doppler had a higher CI at the end of surgery and a significantly shorter hospital stay.

**Conclusion**

Critically ill patients whose cardiac status remains difficult to determine clinically may benefit from invasive monitoring using a volumetric PAC or non-invasive monitoring with a continuous esophageal Doppler. Because the PAOP frequently provides misleading information, we believe that a nonvolumetric PAC has an unfavorable risk/benefit ratio. However, it is vital to emphasize that the information obtained from both the PAC and esophageal Doppler should never be interpreted in isolation. The change in heart rate, CO, PAOP, RVEDVI, Ftc, oxygenation, BP, and urine output in response to a therapeutic intervention needs to be evaluated and interpreted to guide further therapeutic decisions. Furthermore, these variables must be assessed in the context of the patient’s underlying disease process and the presence or absence of tissue hypoxia.5

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