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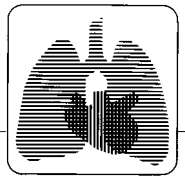
Chest 1999;116;1085-1091
DOI: 10.1378/chest.116.4.1085



opinions/hypotheses

Pulmonary Artery Catheterization and Esophageal Doppler Monitoring in the ICU*

Paul E. Marik, MD, FCCP

The clinical assessment of cardiac performance and ventricular preload is notoriously unreliable in critically ill patients. Consequently, a number of technologies have been developed to provide the clinician with indexes of cardiovascular function to assist in therapeutic decision making. Foremost among these is the pulmonary artery catheter (PAC). Indeed, the PAC has largely shaped the practice of modern critical care. Yet, the information provided by the PAC is largely misunderstood, and its efficacy is never proven. Recently, continuous esophageal Doppler monitoring has emerged as an alternative to pulmonary artery catheterization. This paper evaluates the clinical utility of the PAC and esophageal Doppler monitoring in assessing the hemodynamic status of ICU patients. ***(CHEST 1999; 116:1085–1091)***

Key words: cardiac function; esophageal Doppler; ICU; pulmonary artery catheter; pulmonary artery occlusion pressure; right ventricular end-diastolic volume index

Abbreviations: CI = cardiac index; CO = cardiac output; Ftc = corrected flow time; LVEDP = left ventricular end-diastolic pressure; LVEDV = left ventricular end-diastolic volume; PAC = pulmonary artery catheter; PAOP = pulmonary artery occlusion pressure; Pcap = pulmonary capillary pressure; PEEP = positive end-expiratory pressure; RVEDV = right ventricular end-diastolic volume; RVEDVI = right ventricular end-diastolic volume index; RVEF = right ventricular ejection fraction

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[1] and Fein and colleagues,[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.[3] This presystolic fiber stretch, or preload, is proportionate to end-diastolic volume.[4] Left ventricular end-diastolic volume (LVEDV; preload) is therefore a major factor determining cardiac output (CO).[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 (*ie*, recruitable CO). In addition, an estimation of CO is essential in patients with evidence of inadequate tissue perfusion.[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.

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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.[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.[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.[15–18] An analysis of this data reveals that the three methods are of equal merit and can be used as independent references.[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.[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.[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.[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 refection of the left ventricular end-diastolic pressure (LVEDP); and finally, and most importantly, (4) that there is a linear and predicable 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.[23]

Morris and colleagues[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.[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.[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.[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 juxtacardiac pressure. A PEEP >8 to 10 cm H_2O increases juxtacardiac pressure and, therefore, the pressure gradient between the left atrium and atmospheric pressure, but not the transmural distending pressure.[27] This artifactually increases the PAOP.[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.[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.[29–39] 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.[40] The increase in the LVEDV occurring in association with a decrease in the RVEDV is referred to as diastolic ventricular interaction.[41] 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[20] demonstrated a poor correlation between blood volume (as measured by an isotope technique) and the PAOP. Subsequently, Calvin and coworkers[21] demonstrated a poor relationship between the LVEDV (as measured by radionuclide angiography) and the PAOP. Thys et al[42] 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.[43–58] 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.[23] 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.[23] 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.[59–63]

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 RVEDVI and cardiac index (CI), and they have found the RVEDVI to be superior to the PAOP in determining the preload status of patients.[44,45,47,49–52,55,64] However, some authors have suggested that the correlation between the RVEDVI and CI is related to the fact that these two variables are mathematically coupled.[65] However, the correlation between the RVEDVI and CI remains when the variables are mathematically uncoupled or the CI is determined by the Fick method.[45,64] For any given patient, the relationship between the RVEDVI and CI will depend on right ventricular function. This was recently illustrated by the report of Cheatham and colleagues,[53] who demonstrated that the correlation coefficient between RVEDVI and CI improved when it was stratified by RVEF.

The value of the RVEDVI is the ability to predict the change in CO in response to a fluid challenge. Diebel and colleagues[55] demonstrated the RVEDVI to be an excellent predictor of recruitable CO, whereas the PAOP performed poorly. The optimal RVEDVI 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 RVEDVI has been reported to range from 90 to 140 mL/m^2.[51,52,55] When a volumetric PAC is used, the optimal RVEDVI 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.[28,49,53,61]

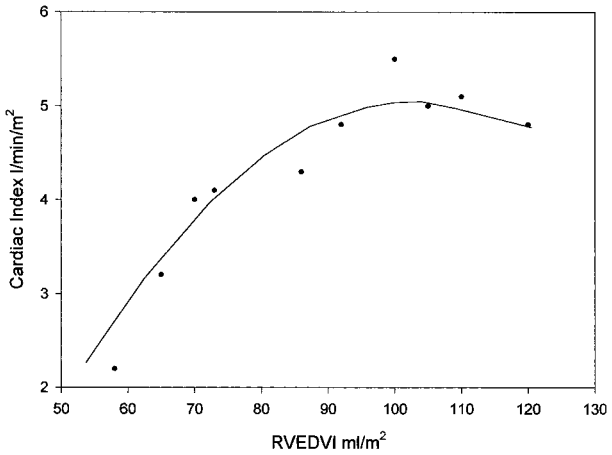

FIGURE 1. Ventricular function curve for a patient with pneumonia showing the relationship between the RVEDVI and CI. The optimal RVEDVI was estimated to be about 105 mL/m^2.

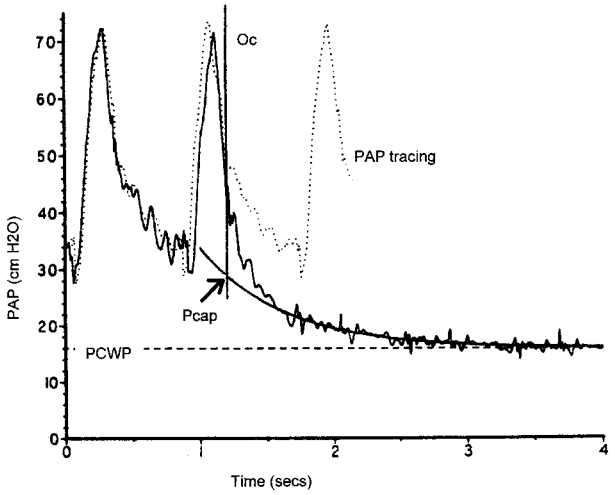

FIGURE 2. The phasic pulmonary artery pressure trace (dotted line) is superimposed on the pulmonary artery pressure trace during pulmonary artery occlusion (solid line). The time of pulmonary artery occlusion can then be identified (Oc) when the two traces sharply diverge. Pcap is estimated as the pressure at which the exponential approximation to the occluded trace (see text) intersects the vertical line drawn at the moment of occlusion. (Reproduced with permission from Collee et al.[67])

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 colleagues[67] 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 al[68] 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 transesophageal Doppler is currently the most promising noninvasive technique for monitoring cardiac function in ICU patients. The esophageal Doppler 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 transcutaneous approach. The close proximity of the descending aorta to the esophagus provides an excellent window for obtaining Doppler signals. Furthermore, once positioned, the transesophageal 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-

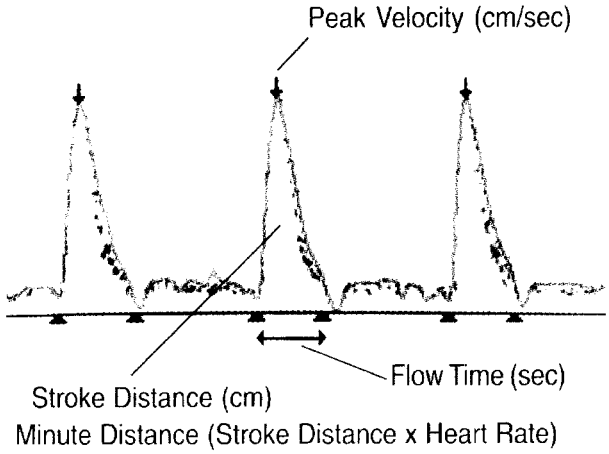

FIGURE 3. Esophageal Doppler flow-velocity waveform.

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.[81–83] The probe has been reported to have been left *in situ* for > 2 two weeks without complications.[84] Lefrant and colleagues[77] 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 provides 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 colleagues[71,82] 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 in limited. Sinclair and colleagues[87] 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 noninvasive 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]

REFERENCES

1 Connors AF, MCCaffree DR, Gray BA. Evaluation of right-heart catheterization in the critically ill patient without acute myocardial infarction. N Engl J Med 1983; 308:263–267
2 Fein AM, Goldberg SK, Walkenstein MD, et al. Is pulmonary artery catheterization necessary for the diagnosis of pulmonary edema? Am Rev Respir Dis 1984; 129:1006–1009
3 Starling EH. The Linacre lecture on the law of the heart. London, UK: Longmans, Green and Co., 1918
4 O'Quin R, Marini JJ. Pulmonary artery occlusion pressure; clinical physiology, measurement and interpretation. Am Rev Respir Dis 1983; 128:319–326
5 Marik PE, Varon J. The hemodynamic derangements in sepsis: implications for treatment strategies. Chest 1998; 114:854–860
6 Robin ED. The cult of the Swan-Ganz catheter: overuse and abuse of the pulmonary flow catheters. Ann Intern Med 1985; 103:445–449

7 Shoemaker WC. Use and abuse of the balloon tipped pulmonary artery (Swan-Ganz) catheter: are patients getting their money's worth? Crit Care Med 1990; 18:1294–1296
8 Robin ED. Death by pulmonary artery flow directed catheter. Chest 1987; 92:727–731
9 Connors AF, Speroff T, Dawson NV, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients. JAMA 1996; 276:889–897
10 Zion MT, Balkin J, Rosenmann D, et al. Use of pulmonary artery catheters in patients with acute myocardial infarction: analysis of experience in 5,841 patients in the SPRINT registry. Chest 1990; 98:1331–1335
11 Swan HJC, Ganz W, Forrester JS, et al. Catheterization of the heart in man with the use of a flow-directed balloon-tipped catheter. N Engl J Med 1970; 283:477–451
12 Iberti TJ, Fischer EP, Leibowitz AB, et al. A multicenter study of physician's knowledge of the pulmonary artery catheter: Pulmonary Artery Study Group JAMA 1990; 264:2928–2932
13 Trottier SJ, Taylor RW. Physicians attitudes toward and knowledge of the pulmonary artery catheter: society of Critical Care Medicine membership survey. New Horizons 1997; 5:201–206
14 Gnaegi A, Feihl F, Perret C. Intensive care physician's insufficient knowledge of right-heart catheterization at the bedside: time to act? Crit Care Med 1997; 25:213–220
15 Branthwaite MA, Bradley RD. Measurement of cardiac output by thermal dilution in man. J Appl Physiol 1968; 24:434–438
16 Olsson B, Pool J, Vandermoten R, et al. Validity and reproducibility of determination of cardiac output by thermodilution in man. Cardiology 1970; 55:136–148
17 Ganz W, Donosco R, Marcus HS, et al. A new technique for measurement of cardiac output by thermodilution in man. Am J Cardiol 1971; 27:392–396
18 Hodges M, Downs JB, Mitchell LA. Thermodilution, and Fick cardiac index determinations following cardiac surgery. Crit Care Med 1975; 3:182–184
19 Stetz CW, Miller RG, Kelly GE, et al. Reliability of the thermodilution method in the determination of cardiac output in clinical practice. Am Rev Respir Dis 1982; 126:1001–1004
20 Baek SM, Makabaki GG, Bryan-Brown CW, et al. Plasma expansion in surgical patients with high central venous pressure (CVP): the relationship of blood volume to hematocrit, CVP, pulmonary wedge pressure, and cardiorespiratory changes. Surgery 1975; 78:304–315
21 Calvin JE, Driedger AA, Sibbald WJ. Does the pulmonary capillary wedge pressure predict left ventricular preload in critically ill patients. Crit Care Med 1981; 9:437–443
22 Marik PE, Varon J, Heard SO. Interpretation of the pulmonary artery occlusion (wedge) pressure: physicians' knowledge *versus* the experts' knowledge. Crit Care Med 1998; 26:1761–1763
23 Raper R, Sibbald WJ. Misled by the Wedge? The Swan-Ganz catheter and left ventricular preload. Chest 1986; 89:427–434
24 Morris AH, Chapman RH, Gardner RM. Frequency of technical problems encountered in the measurement of pulmonary artery wedge pressure. Crit Care Med 1984; 12:164–170
25 Lipman J. Pitfalls in the interpretation of the pulmonary capillary wedge pressure. S Afr Med J 1985; 67:174–177
26 Komadina KH, Schenk DA, La Veau P. Interobserver variability in the interpretation of pulmonary artery catheter pressure tracings. Chest 1991; 100:1647–1654
27 Pinsky M, Vincent JL, De Smet JM. Estimating left ventricular filling pressure during positive end-expiratory pressure in humans. Am Rev Respir Dis 1991; 143:25–31
28 Qvist J, Pontoppidan H, Wilson RS. Hemodynamic responses to mechanical ventilation with PEEP. Anesthesiology 1975; 42:45–55
29 Pagani FD, Baker LS, Hsi C, et al. Left ventricular systolic and diastolic dysfunction after infusion of tumor necrosis factor-alpha in conscious dogs. J Clin Invest 1992; 90:389–398
30 Natanson C, Fink MP, Ballantyne HK, et al. Gram-negative bacteremia produces both severe systolic and diastolic cardiac dysfunction in a canine model that simulates human septic shock. J Clin Invest 1986; 78:259–270
31 Marsch SC, Dalmas S, Philbin DM, et al. Myocardial ischemia and reperfusion are associated with an increased stiffness of remote nonischemic myocardium. Anesth Analg 1996; 82:695–701
32 Yu P, Boughner DR, Sibbald WJ, et al. Myocardial collagen changes and edema in rats with hyperdynamic sepsis. Crit Care Med 1997; 25:657–662
33 Amirhamzeh MM, Dean DA, Jia CX, et al. Iatrogenic myocardial edema: increased diastolic compliance and time course of resolution in vivo. Ann Thorac Surg 1996; 62:737–743
34 Mahgoub MA, Abd-Elfattah AS. Diabetes mellitus and cardiac function. Mol Cell Biochem 1998; 180:59–64
35 Marik P, Varon J. The obese patient in the ICU. Chest 1998; 113:492–498
36 Vasan RS, Benjamin EJ, Levy D. Congestive heart failure with normal left ventricular systolic function: clinical approaches to the diagnosis and treatment of diastolic heart failure. Arch Intern Med 1996; 156:146–157
37 Lakatta EG, Mitchell JH, Pomerance A, et al. Human aging: changes in structure and function. J Am Coll Cardiol 1987; 10(suppl):42A–47A
38 Rozich JD, Smith B, Thomas JD, et al. Dialysis-induced alterations in left ventricular filling: mechanisms and clinical significance. Am J Kidney Dis 1991; 17:277–285
39 Asanoi H, Ishizaka S, Joho S, et al. Altered inotropic and lusitropic responses to heart rate in conscious dogs with tachycardia-induced heart failure. J Am Coll Cardiol 1996; 27:728–735
40 Atherton JJ, Moore TD, Lele SS, et al. Diastolic ventricular interaction in chronic heart failure. Lancet 1997; 349:1720–1724
41 Belenkie I, Dani R, Smith ER, et al. Effect of volume loading during experimental acute pulmonary embolism. Circulation 1989; 80:178–188
42 Thys DM, Hillel Z, Goldman ME, et al. A comparison of hemodynamic indices derived by invasive monitoring and two-dimensional echocardiography. Anesthesiology 1987; 67:630–634
43 Shippy CR, Appel PL, Shoemaker WC. Reliability of clinical monitoring to assess blood volume in critically ill patients. Crit Care Med 1984; 12:107–112
44 Reuse C, Vincent JL, Pinsky MR. Measurements of right ventricular volumes during fluid challenge. Chest 1990; 98:1450–1454
45 Durham R, Neunaber K, Vogler G, et al. Right ventricular end-diastolic volume as a measure of preload. J Trauma 1995; 39:218–223
46 Dennis JW, Menawat SS, Sobowale OO, et al. Superiority of end-diastolic volume and ejection fraction measurements over wedge pressures in evaluating cardiac function during aortic reconstruction. J Vasc Surg 1992; 16:372–377
47 Martyn JA, Snider MT, Farago LF, et al. Thermodilution right ventricular volume: a novel and better predictor of volume replacement in acute thermal injury. J Trauma 1981; 21:619–626
48 Godje O, Peyerl M, Seebauer T, et al. Central venous pressure, pulmonary capillary wedge pressure and intrathoracic blood volumes as preload indicators in cardiac surgery patients. Eur J Cardiothorac Surg 1998; 13:533–539
49 Diebel LN, Myers T, Dulchavsky S. Effects of increasing

airway pressure and PEEP on the assessment of cardiac preload. J Trauma 1997; 42:585–590
50 Chang MC, Meredith JW. Cardiac preload, splanchnic perfusion, and their relationship during resuscitation in trauma patients. J Trauma 1997; 42:577–582
51 Chang MC, Blinman TA, Rutherford EJ, et al. Preload assessment in trauma patients during large-volume shock resuscitation. Arch Surg 1996; 131:728–731
52 Diebel L, Wilson R, Tagett MG, et al. End diastolic volume: a better indicator of preload in the critically ill. Arch Surg 1992; 127:817–822
53 Cheatham ML, Nelson LD, Chang MC, et al. Right ventricular end-diastolic volume index as a predictor of preload status in patients on positive end-expiratory pressure. Crit Care Med 1998; 26:1801–1806
54 Krahmer RL, Fang HK, Vitello J, et al. Pulmonary capillary wedge pressure estimates of left ventricular preload are inaccurate in endotoxin shock: contribution of Starling resistor forces to septic pulmonary hypertension. Shock 1994; 2:344–350
55 Diebel L, Wilson RF, Heins J, et al. End-diastolic volume versus pulmonary artery wedge pressure in evaluating cardiac preload in trauma patients. J Trauma 1994; 37:950–955
56 Beaupre PN, Cahalan MK, Kremer PF, et al. Does pulmonary artery occlusion pressure adequately reflect left ventricular filling during anesthesia and surgery [abstract]? Anesthesiology 1998; 59:A3
57 Hansen RM, Viquerat CE, Matthay MA, et al. Poor correlation between pulmonary artery wedge pressure and left ventricular end-diastolic volume after coronary artery bypass graft surgery. Anesthesiology 1986; 64:764–770
58 Hoyt JD, Leatherman JW. Interpretation of the pulmonary artery occlusion pressure in mechanically ventilated patients with large respiratory excursions in intrathoracic pressure. Intensive Care Med 1997; 23:1125–1131
59 Vincent JL, Thirion M, Brimioulle S, et al. Thermodilution measurement of right ventricular ejection fraction with a modified pulmonary artery catheter. Intensive Care Med 1986; 12:33–38
60 Dhainaut JF, Brunet F, Monsallier JF, et al. Bedside evaluation of right ventricular performance using a rapid computerized thermodilution method. Crit Care Med 1987; 15:148–152
61 Jardin F, Brun-Ney D, Hardy A, et al. Combined thermodilution and two-dimensional echocardiographic evaluation of right ventricular function during respiratory support with PEEP. Chest 1991; 99:162–168
62 Urban P, Scheidegger D, Gabathuler J, et al. Thermodilution determination of right ventricular volume and ejection fraction: a comparison with biplane angiography. Crit Care Med 1987; 15:652–655
63 Voelker W, Gruber HP, Ickrath O, et al. Determination of right ventricular ejection fraction by thermodilution technique: a comparison to biplane cineventriculography. Intensive Care Med 1988; 14(suppl 2):461–466
64 Chang MC, Black CS, Meredith JW. Volumetric assessment of preload in trauma patients: addressing the problem of mathematical coupling. Shock 1996; 6:326–329
65 Civetta JM, Kirton O, Civetta-Hudson J. Mathematical coupling: why correlations of right ventricular function ae so good? Crit Care Med 1995; 23(suppl):A126
66 Levy MM, Sakai D, Klinger JR. Pulmonary capillary pressure in sepsis and cardiogenic shock [abstract]. Am J Respir Crit Care Med 1996; 153:A6001
67 Collee GG, Lynch KE, Hill RD, et al. Bedside measurement of pulmonary capillary pressure in patients with acute respiratory failure. Anesthesiology 1987; 66:614–620
68 Holloway HM, Perry J, Downey J, et al. Estimation of effective pulmonary capillary pressure in intact lungs. J Appl Physiol 1983; 54:846–851
69 Sageman WS, Amundson DE. Thoracic electrical bioimpedance measurement of cardiac output in postaortocoronary bypass patients. Crit Care Med 1993; 21:1139–1142
70 Side CD, Gosling RJ. Non-surgical assessment of cardiac function. Nature 1971; 232:335–336
71 Singer M, Clarke J, Bennett ED. Continuous hemodynamic monitoring by esophageal Doppler. Crit Care Med 1989; 17:447–452
72 Huntsman LL, Stewart DK, Barnes SR, et al. Noninvasive Doppler determination of cardiac output in man: clinical validation. Circulation 1983; 67:593–602
73 Freund PR. Transesophageal Doppler scanning versus thermodilution during general anesthesia: an initial comparison of cardiac output techniques. Am J Surg 1987; 153:490–494
74 Mark JB, Steinbrook RA, Gugino LD, et al. Continuous noninvasive monitoring of cardiac output with esophageal Doppler ultrasound during cardiac surgery. Anesth Analg 1986; 65:1013–1020
75 Perrino AC, Fleming J, LaMantia KR. Transesophageal Doppler Ultrasonography: evidence for improved cardiac output monitoring. Anesth Analg 1990; 71:651–657
76 Davies JN, Allen DR, Chant AD. Non-invasive Doppler-derived cardiac output: a validation study comparing this technique with thermodilution and Fick methods. Eur J Vasc Surg 1991; 5:497–500
77 Lefrant JY, Bruelle P, Aya AG, et al. Training is required to improve the reliability of esophageal Doppler to measure cardiac output in critically ill patients. Intensive Care Med 1998; 24:347–352
78 Feinberg MS, Hopkins WE, Davila-Roman VG, et al. Multiplane transesophageal echocardiographic Doppler imaging accurately determines cardiac output measurements in critically ill patients. Chest 1995; 107:769–773
79 Cuschien J, Rivers E, Caruso J, et al. a comparison of transesophageal Doppler, thermodilution and Fick cardiac output measurements in critically ill patients [abstract]. Crit Care Med 1998; 26(suppl):A62
80 Cariou A, Monchi M, Joly LM, et al. Noninvasive cardiac output monitoring by aortic blood flow determination: evaluation of the Sometec Dynemo-3000 system. Crit Care Med 1998; 26:2066–2072
81 Singer M. Esophageal Doppler monitoring of aortic blood flow: beat-by-beat cardiac output monitoring. Int Anesth Clin 1993; 31:99–125
82 Singer M, Bennett ED. Non-invasive optimization of left ventricular filling by esophageal Doppler. Crit Care Med 1991; 19:1132–1137
83 Valtier B, Cholley BP, Belot JP, et al. Noninvasive monitoring of cardiac output in critically ill patients using transesophageal Doppler. Am J Respir Crit Care Med 1998; 158:77–83
84 Gan TJ, Arrowsmith JE. The oesophageal Doppler monitor. BMJ 1997; 315:893–894
85 Wallmeyer K, Wann LS, Sagar KB, et al. The influence of preload and heart rate on Doppler echocardiographic indexes of left ventricular performance: comparison with invasive indexes in an experimental preparation. Circulation 1986; 74:181–186
86 Singer M, Allen MJ, Webb AR, et al. Effects of alterations in left ventricular filling, contractility, and systemic vascular resistance on the ascending aortic blood velocity waveform of normal subjects. Crit Care Med 1991; 19:1138–1145
87 Sinclair S, James S, Singer M. Intraoperative intravascular volume optimisation and length of hospital stay after repair of proximal femoral fracture: randomised controlled trial. BMJ 1997; 315:909–912