

Michael R. Pinsky

## Functional hemodynamic monitoring

Received: 3 December 2001  
Accepted: 3 December 2001  
Published online: 20 March 2002  
© Springer-Verlag 2002

M.R. Pinsky (✉)  
University of Pittsburgh Medical Center, Pittsburgh, Pa., USA  
e-mail: pinskymr@anes.upmc.edu

The management of the critically ill patient often requires continual monitoring of cardiovascular status. Numerous invasive and noninvasive technologies have been used to monitor cardiovascular status. Presently, highly invasive hemodynamic monitoring appears to be needed to define and treat the specific causes of cardiovascular instability. However, these complex monitoring systems often require pulmonary arterial catheterization and have not been shown to provide critically ill patients with a survival advantage [1]. In fact, the utility of hemodynamic monitoring itself has never been documented to improve survival. In part, this lack of benefit from hemodynamic monitoring may reflect the use of poorly validated systems of hemodynamic profile analysis and unproven treatments.

In the assessment and management of critically ill patients, the actual hemodynamic monitoring questions are physiological in their language but need to be practical and concrete in their application. Perhaps the most pertinent function hemodynamic question is: Will cardiac output increase with volume loading? Documenting that an unstable patient is preload-responsive defines the initial treatment as fluid resuscitation. Since a majority of the patients presenting with cardiovascular insufficiency are preload-responsive and often show dramatic improvement in their cardiac output and organ perfusion with fluid resuscitation alone, this is an important initial therapeutic question. However, in those unstable patients who are not preload-responsive, fluid resuscitation may induce acute heart failure or pulmonary edema, if cor

pulmonale or acute left ventricular (LV) failure, respectively, are the causes of cardiovascular insufficiency. Thus, being able to identify preload responsiveness quickly, accurately, and with a minimum of invasive risk prior to actually performing a fluid challenge is highly desirable.

Data from numerous clinical trials have documented repeatedly that neither right atrial pressure ( $P_{ra}$ ) or pulmonary artery occlusion pressure ( $P_{pao}$ ) predict well the subsequent response of the subject to an intravascular fluid challenge [2]. Furthermore, measures of absolute LV volumes are only slightly better at predicting preload responsiveness. Clearly, subjects with small LV end-diastolic volumes can have a limited response to a volume challenge if their filling is limited either by tamponade, cor pulmonale, or diastolic stiffening. Furthermore, ventilation and ventilatory therapies, such as the use of positive end-expiratory pressure often complicates this analysis by dissociating filling pressures from measured intrathoracic vascular pressure because of both increasing intrathoracic pressure and cardiac compression by lung expansion [3]. However, ventilation, by phasically altering  $P_{ra}$  also serves as a sine wave forcing function on venous return and can be used to define cardiovascular performance. Several groups have applied this concept to assess preload responsiveness. The importance of these applications to bedside monitoring is finally being understood.

Since  $P_{ra}$  is the backpressure to venous return, if  $P_{ra}$  should decrease during spontaneous inspiration, venous return will transiently increase, increasing cardiac output. If, however, the right ventricle is unable to dilate further,  $P_{ra}$  will not decrease during inspiration even though intrathoracic pressure decreases. At the extreme, spontaneous inspiration-associated increases in  $P_{ra}$  reflect severe right ventricular failure and are referred to as Kussmaul's sign. Madger et al. [4] used the fall in  $P_{ra}$  to predict which patients would increase their cardiac outputs in response to a defined fluid challenge. They found

that if  $P_{ra}$  decreased by more than 2 mmHg during a spontaneous breath, cardiac output increased in 16 or 19 patients in response to 250–500 ml saline bolus infusion. If  $P_{ra}$  did not decrease, cardiac output increased in only one of 14 patients. These data are important because they focus on both right ventricular function and spontaneous ventilation, two areas of study with markedly few clinical trials.

More studies focus on the effects of positive-pressure ventilation on LV output. Positive-pressure ventilation induces phasic changes in LV stroke volume although similar cyclic changes in venous return. The magnitude of these changes in stroke volume are a function of the size of the tidal breath, the subsequent increase in intrathoracic pressure, and the extent that changes in LV output are determined by changes in LV filling pressure. Beat-to-beat changes in LV stroke volume can be easily monitored as beat-to-beat changes in arterial pulse pressure variation (PVV), since the only other determinants of pulse pressure, arterial resistance, and compliance cannot change enough to alter pulse pressure during a single breath. Based on this logic, Perel et al. [5] and Szold et al. [6] examined the systolic pressure variation (SPV) induced by a defined positive-pressure breath both in animals made hypovolemic and in humans with heart failure, demonstrating that the SPV, as specifically the decrease in systolic pressure from an apneic baseline, referred to a  $\Delta$ down, identified hemorrhage and was minimized by fluid resuscitation. Tavernier et al. [7] subsequently validated these findings. The concept of SPV assumes that all the changes in systolic pressure can be explained by parallel changes in LV stroke volume.

Unfortunately, Denault et al. [8] could not demonstrate any relationship between LV stroke volume, estimated by transesophageal echocardiographic analysis, and SPV, suggesting that factors other than LV stroke volume contribute to SPV. Michard et al. [9, 10] reasoned that arterial PVV rather than SPV would more accurately reflect changes in LV stroke volume because arterial PVV is not influenced by the intrathoracic pressure-induced changes in both systolic and diastolic arterial pressure. They compared SPV with PPV as predictors of the subsequent increase in cardiac output in response to fluid loading in septic ventilator-dependent patients. Their data convincingly demonstrated that both PPV and SPV greater than 15% were far superior to measures of either  $P_{ra}$  or  $P_{pao}$  in predicting an increase in cardiac output response to volume loading. Furthermore, the greater the PPV or SPV was, the greater the subsequent increase in cardiac output. However, PPV claimed a slight although significant advantage over SPV in terms of greater precision and less bias. Since PPV attempts to monitor LV stroke volume changes, it was not surprising that Feissel et al. [11] demonstrated that aortic flow variation, as measured by transesophageal two-dimensional echocardiography pulsed Doppler of the aor-

tic outflow tract, followed a similar response to PPV in response to fluid loading. The flow variation data are very important because flow is the primary variable from which SPV and APP derive their validity. Although less invasive than arterial pressure monitoring, echocardiographic analysis is far from ideal as a hemodynamic monitoring tool. It requires the continuous presence of an experienced operator. Echocardiography requires using expensive and often scarce equipment. Finally, measures of aortic root flow variation cannot be made online or continuously over prolonged periods of time.

Thus, Reuter et al. [12] attempted to apply the same stroke volume variation (SVV) logic using an alternative method that by-passes these limitations. They used a relatively old technology [13] referred to as the arterial pulse contour technique. The technique calculates LV stroke volume from the impedance characteristics of the pulse pressure waveform using a complex and proprietary algorithm [14]. The technique has enjoyed limited success in the critical care arena [15]. The measure of SVV then comes from the beat-to-beat analysis of these measures over a breath. Reuter et al. measured a vast variety of independent measures of cardiovascular status, namely echocardiographic measures of cross-sectional LV area, intrathoracic blood volume index, and both central venous pressure and  $P_{pao}$ . They demonstrated that their pulse contour SVV measures behaves in a fashion predicted from previous studies of SVP, arterial PPV, and aortic flow wherein actual measures were made. If these data are correct, this study marks a major step forward in the use of functional measures of hemodynamic status. The greater the SVV, the more cardiac output can be expected to increase in response to volume loading. Furthermore, if the SVV is less than 10%, cardiac output does not increase in response to volume loading and may be avoided as a therapeutic challenge.

The study by Reuter et al. [12] has some limitations, however, that make the application of these data in the management of patients today unadvisable. First, the arterial PPV data of Michard et al. [10] used a tidal volume of only 6 ml/kg, whereas the present study used a much larger tidal volume of 13–15 ml/kg. It is not clear whether similar SVV would be seen by this technique if the tidal volumes used were in the range of clinically relevant breaths. This is important because the measure of SVV by the pulse contour method has not been validated under positive-pressure ventilation conditions. The pulse contour method accurately tracks mean cardiac output [15]. However, it has never been validated for its accuracy to assess beat-to-beat variations in LV stroke volume. This is not a minor point. Denault et al. [8] demonstrated that much of the power signal of the arterial pulse is altered by ventilation, making the measure of SVV variation by the pulse contour technique questionable at best since it uses the power spectral analysis to estimate volume. That the authors saw a variation in their derived

“stroke volume” variable and that it changed as expected in response to volume loading is encouraging but not definitive in allowing us to embrace this monitoring technique as a simpler method of measuring preload responsiveness. Second, and perhaps more concerning, the study design did not test the hypothesis that the cardiac output response to volume loading in critically ill patients could be predicted by SVV. The authors merely showed that a post hoc analysis of changes in SVV were correlated with subsequent stroke index changes in hemodynamically stable patients. Clearly, over time in postoperative patients arterial tone and rheology change, and these variables can alter pulse contour characteristics in unforeseen ways. Thus, this technique needs to be validated.

Hemodynamic monitoring has many roles in medicine. One is to diagnose disease and define cardiac performance. In the cardiac catheterization laboratory where all these techniques had their birth, that goal was clear. However, as we evolved into a treatment-oriented clinical practice, the paradigm of physiological status evolved as well into functional performance, a much different measure. The trend in hemodynamic monitoring toward more performance-based measures, such as preload responsiveness, is encouraging and marks the pathway toward more effective patient management, which at the end of the day is the reason why we use invasive monitoring at all.

## References

1. Connors AF Jr, Speroff T, Dawson NV, Thomas C, Harrell FE Jr, Wagner D, Desbiens N, Goldman L, Wu AW, Califf RM, Fulkerson WJ Jr, Vidaillet H, Broste S, Bellamy P, Lynn J, Knaus WA (1996) The effectiveness of right heart catheterization in the initial care of critically ill patients. SUPPORT Investigators. *JAMA* 276:889–8897
2. Lichtwarck-Aschoff M, Zeravik J, Pfeiffer UJ (1992) Intrathoracic blood volume accurately reflects circulatory volume status in critically ill patients with mechanical ventilation. *Intensive Care Med* 18:137–138
3. Pinsky MR, Vincent JL, DeSmet JM (1991) Estimating left ventricular filling pressure during positive end-expiratory pressure in humans. *Am Rev Respir Dis* 143:25–31
4. Magder S, Georgiadis G, Cheong T (1992) Respiratory variations in right atrial pressure predict the response to fluid challenge. *J Crit Care* 7:76–85
5. Perel A, Pizov R, Cotev S (1987) Systolic blood pressure variation is a sensitive indicator of hypovolemia in ventilated dogs subjected to graded hemorrhage. *Anesthesiology* 67:498–502
6. Szold A, Pizov R, Segal E, Perel A (1989) The effect of tidal volume and intravascular volume state on systolic pressure variation in ventilated dogs. *Intensive Care Med* 15:368–371
7. Tavernier B, Makhotine O, Lebuffe G, Dupont J, Scherpereel P (1998) Systolic pressure variation as a guide to fluid therapy in patients with sepsis-induced hypotension. *Anesthesiology* 89:1313–1321
8. Denaault AY, Gasior TA, Gorcsan J, Mandarino WA, Deneault LG, Pinsky MR (2000) Determinants of aortic pressure variation during positive-pressure ventilation in man. *Chest* 116:176–86
9. Michard F, Chemla D, Richard C, Wysocki M, Pinsky MR, Lecarpentier Y, Teboul JL (1999) Clinical use of respiratory changes in arterial pulse pressure to monitor the hemodynamic effects of PEEP. *Am J Respir Crit Care Med* 159:935–939
10. Michard F, Boussat S, Chemla D, Anguel N, Mercat A, Lecarpentier Y, Richard C, Pinsky MR, Teboul JL (2000) Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. *Am J Respir Crit Care Med* 162:134–138
11. Feissel M, Michard F, Mangin I, Ruyer O, Faller JP, Teboul JL (2001) Respiratory changes in aortic blood velocity as an indicator of fluid responsiveness in ventilated patients with septic shock. *Chest* 119:867–873
12. Reuter DA, Felbinger TW, Schmidt C, Kilger E, Goedje O, Lamm P, Goetz AE (2002) Stroke volume variations for assessment of cardiac responsiveness to volume loading in mechanically ventilated patients after cardiac surgery. *Intensive Care Med* <http://dx.doi.org/10.1007/s00134-002-1211-z>
13. Frank O (1899) Die Grundform des arteriellen Pulses. *Z Biol* 37:483–526
14. Wesseling K, Wit BD, Weber J, Smith NT (1983) A simple device for the continuous measurement of cardiac output. *Adv Cardiovasc Physiol* 5:16–52
15. Bock J, Barker B, Mackersie R, Tranbaugh R, Lewis F (1989) Cardiac output measurement using femoral artery dilution in patients. *J Crit Care* 4:106–111