Heart–lung interactions

Michael R. Pinsky

Purpose of review

Assessment of cardiovascular stability using ventilation-induced changes in measured physiological variables, referred to as functional hemodynamic monitoring, usually requires measurement of ventilation-induced changes in venous return. Thus, it is important to understand the determinants of these complex heart–lung interactions.

Recent findings

Several animal and human studies have recently documented that ventricular interdependence plays an important role during positive-pressure breathing, causing acute cor pulmonale. With the use of lower tidal volume ventilation in patients with acute respiratory failure, the incidence of acute cor pulmonale is decreasing proportionally. When present, however, it induces a stroke volume variation that is 180° out of phase with that seen in hypovolemic states, such that left ventricular stroke volume increases during inspiration rather than decreasing as seen in hypovolemia. Further, when either tidal volume or positive end-expiratory pressure levels are varied, both stroke volume variation and pulse pressure variation are affected in a predictable manner. The greater the swing in intrathoracic pressure, the greater the change in venous return.

Summary

Functional hemodynamic monitoring is becoming more prevalent. For it to be used effectively, the operator needs to have a solid understanding of how ventilation induces both pulse pressure variation and stroke volume variation in that specific patient.

Keywords

cardiovascular insufficiency, intrathoracic pressure, ventilation, volume responsiveness

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Department of Critical Care Medicine, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, USA

Correspondence to Michael R. Pinsky, MD, 606 Scaife Hall, 3550 Terrace Street, Pittsburgh, PA 15213, USA
Tel: +1 412 647 5387; fax: +1 412 647 8060; e-mail: pinskymr@upmc.edu

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Abbreviations

ITP	intrathoracic pressure
PEEP	positive end-expiratory pressure
PPV	pulse pressure variation
SVV	stroke volume variation

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Introduction

Cardiopulmonary interactions are central to cardiopulmonary homeostasis and are also a major determinant of the accuracy of hemodynamic monitoring. This topic was recently reviewed in this journal by Monnet *et al.* [1], who focused on the impact of breathing during heart failure. In this survey of recent publications I will focus on nonheart failure studies. This group of patients is important because new applications of heart–lung interactions to assess preload responsiveness focus on left ventricular pulse pressure variation (PPV) and stroke volume variation (SVV) during positive-pressure ventilation.

Physiological basis for heart–lung interactions

Clearly, any discussion of clinical implications of heart–lung interactions requires the reader to be cognizant of the fundamental physiological underpinnings of the observed physiological responses. I recently wrote a review article on this topic which covers these points in detail [2]. Briefly, heart–lung interactions can be understood based on the effects of changes in intrathoracic pressure (ITP) and lung volume on venous return and left ventricular ejection, and the energy needed to create these changes. During spontaneous ventilation, venous return increases with negative swings in ITP, subsequently increasing right ventricular volume and causing the intraventricular septum to move into the left ventricle. This is manifested by a spontaneous inspiration-associated decrease in left ventricular end-diastolic volume and decreased left ventricular diastolic compliance. This decreased left ventricular preload causes an immediate decrease in left ventricular stroke volume and pulse pressure which is referred to as pulsus paradoxus. The more vigorous the ventilatory efforts, the greater the ITP swings and the more pulsus paradoxus occurs.

Ventricular interdependence

Ventricular interdependence can be induced by changing pulmonary vascular resistance, the output to right

ventricular flow. Hyperinflation and thromboembolism both can increase pulmonary vascular resistance and cause the right ventricle to dilate, decreasing left ventricular preload. Importantly, as the practice of mechanical ventilation has changed in recent years, with smaller tidal volumes now being delivered (current initial volumes 5–7 now vs. 12–15 ml/kg in the 1980s), we are also seeing less cor pulmonale in our acute respiratory distress syndrome patients [3]. These points were recently discussed in an excellent clinical review by Jardin and Vieillard-Baron in this journal [4]. Clearly, lung over-distention by the excessive use of positive end-expiratory pressure (PEEP) may place undue stress on right ventricular function, producing cor pulmonale. Another form of lung over-distention is the transient over-distention observed with lung recruitment maneuvers. Although numerous studies have reported that lung recruitment maneuvers do not induce persistent cardiovascular insufficiency, there is the potential for right ventricular pressure overload during the recruitment maneuver itself. Indeed, Nielson *et al.* [5] documented that 40 cmH_2O resulted in transient right ventricular dilation and left ventricular collapse during the inflation hold maneuver. Although these effects are transient, the data suggest that if recruitment maneuvers are used at all in patients with borderline right ventricular failure, they should be used with caution and for 10 s or less, as outlined by an excellent perspective editorial by one of the thought leaders in the field of right ventricular dysfunction in critical illness [6].

Spontaneous ventilation and ventricular interdependence

Until recently it was felt that ventricular interdependence was minimal during normal tidal volume positive-pressure ventilation because the changes in ITP are small, making both the lung inflation-induced pulmonary vascular resistance and venous return changes small. Mitchell *et al.* [7], however, showed in dogs that positive-pressure ventilation also altered left ventricular output in a fashion explained by ventricular interdependence. They saw that with positive-pressure inspiration, as right ventricular dimensions decreased, the left ventricular dimensions increased and left ventricular stroke volume increased slightly (around 3–5%). These findings agree with previous work using echocardiographic estimates of right ventricular and left ventricular flows [8]. Importantly, the changes in right ventricular output were much greater than the changes in left ventricular output, again as previously described [9]. Thus, the usually observed small changes in arterial pulse pressure during positive-pressure ventilation may reflect ventricular interdependence and not preload responsiveness. As shown by Mitchell *et al.* [7], however, if ventricular interdependence is the primary process, positive-pressure inspiration is usually associated with an increase in left ventricular stroke volume, whereas, as we and others have previously shown, if the process is primarily one of decreasing venous return, left ventricular stroke volume increases during expiration. These concepts are highlighted in Fig. 1 [10], which shows the dynamic phase differences in stroke volume changes that are inspiration dependent. Thus, one needs to measure not only the magnitude of PPV and SVV, but its phase relationship to inspiration. If pulse pressure decreases with inspiration, then this is a preload-responsive pattern, whereas if it increases with inspiration, this reflects interdependence and may be a marker of either cor pulmonale or heart failure. Still, the interdependence-induced PPV and SVV are much smaller than the changes reported in preload-responsive patients.

Ventilation-induced pulse pressure variation and stroke volume variation

Since both PPV and SVV require a phasic change in venous return as their forcing function, ITP changes may be decreased if the chest is opened and the pleural cavities are violated. In fact, that was what DeBlasi *et al.* [11•] observed in their study of 25 patients during cardiac surgery. In open chest conditions the effect of positive-pressure breathing on heart–lung interactions is markedly reduced. Again, these findings agree with prior animal studies [10].

Effect of tidal volume on pulse pressure variation and stroke volume variation

Changes in tidal volume should also alter the phasic swings in ITP and thus the dynamic changes in venous return. The greater the tidal volume, the greater the cycle-specific changes in venous return augmenting PPV and SVV of the same volume status. These findings were reported by two separate groups of investigators: DeBacker *et al.* [12], in a simple and elegant clinical description of the impact of changing tidal volume on the associated arterial PPV, showed that PPV varied directly with tidal volume and Renner *et al.* [13•] showed that SVV also varied directly with tidal volume.

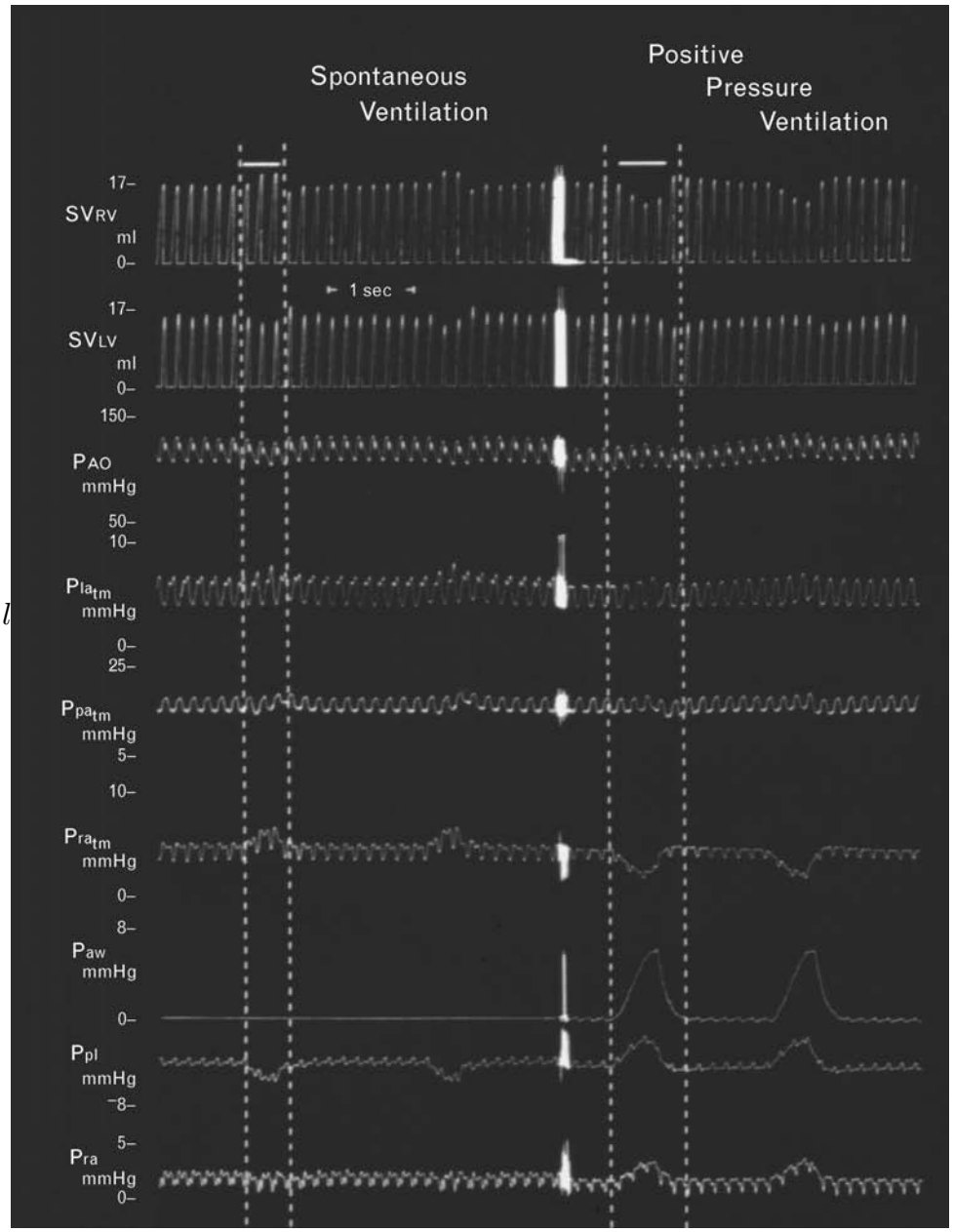
Effect of positive end-expiratory pressure on pulse pressure variation and stroke volume variation

Since the primary effect of PEEP is to distend the lungs and increase ITP, it normally reduces venous return and creates a functional hypovolemic state. Accordingly, Kubitz *et al.* [14•] showed in a porcine model that increasing PEEP levels increased both PPV and SVV. Interestingly, they also saw that this effect persisted in an open chest condition, albeit to a lesser degree.

Limits on the ability of arterial pulse contour to assess stroke volume variation

DeCastro *et al.* [15•] validated the finding that stroke volume calculated by the PiCCO arterial pulse

Figure 1 Simultaneous physiological variable display from an intact anesthetized dog during spontaneous breathing (left) and similar tidal volume positive-pressure breathing (right) [10]



SV_{RV}, right ventricular stroke volume; SV_{LV}, left ventricular stroke volume; P_{Ao}, aortic pressure; P_{latm}, left atrial transmural pressure; P_{patm}, pulmonary artery transmural pressure; P_{ratm}, transmural right atrial pressure; P_{aw}, airway pressure; P_{pl}, pleural pressure; P_{ra}, right atrial pressure. Dotted lines identify start and stop of breath. Note that SV_{LV} decreases in phase with the increase in SV_{RV} during spontaneous inspiration, consistent with ventricular interdependence, whereas SV_{LV} decreases after the SV_{RV} changes have stopped during positive-pressure ventilation, consistent with a transient decrease in pulmonary venous inflow.

contour technique closely tracks steady state arterial pressure, but does not also track dynamic SVV. These data agree with the recent canine study by Gunn *et al.* [16••] that examined how accurately this same device tracked SVV as vasomotor tone was pharmacologically varied.

Conclusion

The clinical use of heart–lung interactions is expanding. Interest in clinically relevant applications of PPV and SVV is growing as these parameters demonstrate their influence on patient outcomes. Great care should be taken, however, to ensure that the limitations of these

parameters are understood, because all measures need to be considered within the context of their physiological limitations.

Acknowledgement

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References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 609–610).

1 Monnet X, Teboul JL, Richard C. Cardiopulmonary interactions in patients with heart failure. Curr Opin Crit Care 2007; 13:6–11.

2 Pinsky MR. Cardiovascular issues in respiratory care. Chest 2005; 128: 592S–597S.

3 Viellard-Baron A, Schmitt JM, Augarde R, *et al.* Acute cor pulmonale in acute respiratory distress syndrome submitted to protective ventilation: Incidence, clinical implications, and prognosis. Intensive Care Med 2001; 29:1551–1558.

4 Jardin F, Vieillard-Baron A. Monitoring of right-sided heart function. Curr Opin Crit Care 2005; 11:271–279.

5 Nielson J, Ostergaard M, Kjaegaad J, *et al.* Lung recruitment maneuver decreases central haemodynamics in patients after cardiac surgery. Intensive Care Med 2005; 31:1189–1194.

6 Jardin F. Acute leftward septal shift by lung recruitment maneuver. Intensive Care Med 2005; 31:1148–1149.

7 Mitchell JR, Whitelaw WA, Sas R, *et al.* RV filling modulates LV function by direct ventricular interaction during mechanical ventilation. Am J Physiol 2005; 289:H549–H557.

8 Jardin F, Delorme G, Hardy A, *et al.* Reevaluation of hemodynamic consequences of positive pressure ventilation: emphasis on cyclic right ventricular afterloading by mechanical lung inflation. Anesthesiology 1990; 72:966–970.

9 Pinsky MR. Determinants of pulmonary artery flow variation during respiration. J Appl Physiol 1984; 56:1237–1245.

10 Pinsky MR, Matuschak GM, Klain M. Determinants of cardiac augmentation by increases in intrathoracic pressure. J Appl Physiol 1985; 58:1189–1198.

11 • DeBlasi RA, Palmisani S, Cigognetti L, *et al.* Effects of sternotomy on heart–lung interactions in patients undergoing cardiac surgery receiving pressure-controlled mechanical ventilation. Acta Anesthesiol Scand 2007; 51:441–446.

Nice clinical study documenting that when intrathoracic pressure changes are minimized by sternotomy the effect of ventilation on the circulation is also minimized. This is not a new finding in cardiopulmonary physiology, but has not been so well documented in humans until now.

12 DeBacker D, Heenen S, Piagnerelli M, *et al.* Pulse pressure variations to predict fluid responsiveness: influence of tidal volume. Intensive Care Med 2005; 31:517–523.

13 • Renner J, Cavus E, Meybohm P, *et al.* Stroke volume variation during hemorrhage and after volume loading: impact of different tidal volumes. Acta Anesthesiol Scand 2007; 51:538–544.

Nice animal study underscoring the interactions between tidal volume and stroke volume variation as intravascular volume is altered.

14 • Kubitz JC, Annecke T, Kemming GL, *et al.* The influence of positive end-expiratory pressure on stroke volume variation and central blood volume during open and closed chest conditions. Eur J Cardiothorac Surg 2006; 30:90–95.

This clinical documentation of the impact of PEEP on preload responsiveness supports the findings previously reported by others.

15 • DeCastro V, Goarin JP, Lhotel L, *et al.* Comparison of stroke volume and stroke volume respiratory variation measured by axillary arterial pulse-contour and by aortic Doppler echocardiography in patients undergoing aortic surgery. Br J Anaesth 2006; 5:605–610.

Good clinical study showing the strengths and limitations of arterial pulse contour methods to track stroke volume and stroke volume variation in humans. Supports the findings of [16••] below.

16 •• Gunn S, Kim HK, Harrigan P, Pinsky MR. Ability of aortic pulse contour and esophageal pulsed Doppler measures to estimate changes in left ventricular output. Intensive Care Med 2006; 32:1537–1546.

First study to actually compare arterial pulse contour analysis and esophageal Doppler estimates of stroke volume with directly measured stroke volume from an aortic flow probe as cardiac output was transiently varied by vascular occlusion and as vasomotor tone was pharmacologically altered.