

A Pilot Study of the Effect of Altering Airway Pressure on Systolic Pulse Pressure Variation in the Systemic and Pulmonary Arterial Circulations

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ABSTRACT

Objective: *Systolic pressure variation results from cyclical fluctuation in the intra-thoracic pressure associated with mechanical ventilation and has been used as a measure of relative hypovolemia in mechanically ventilated patients. The impact of the magnitude of the tidal volume and airway pressure on systolic pressure variation, however, has not been examined in mechanically ventilated patients.*

Methods: *Two patients underwent monitoring following elective cardiac surgery. Tidal volume was randomly varied between 3 and 11 mL/kg over a two minute interval, and the corresponding airway pressure was monitored, as were the effects on the systolic pressure variation of the systemic and pulmonary circulations.*

Results: *There was a strong correlation between increasing tidal volume and peak airway pressure ($p < 0.0001$). In addition, peak airway pressure strongly correlated with the systolic pressure variation of both the systemic and pulmonary circulations ($p < 0.0001$). The increase in diastolic pulmonary arterial pressure induced by insufflation correlated well with the associated increase in systolic blood pressure ($p < 0.0001$). Similarly, the increase in systolic pulmonary artery pressure (PAP) correlated with the associated decrease in systolic blood pressure induced by insufflation ($p < 0.0001$).*

Conclusions: *Systolic pressure variation in the systemic and pulmonary circulations is affected by tidal volume and peak airway pressure. This should be considered when using systolic pressure variation as a marker of intravascular volume status. Our findings regarding the correlations between changes in the pulmonary arterial pressure and the systemic arterial pressure induced by mechanical ventilation are consistent with the proposed physiological mechanisms of systolic pressure variation. (Critical Care and Resuscitation 2004; 6: 167-174)*

Key words: Systolic pressure variation, pulmonary artery pressure variation, plateau pressure, tidal volume, peak airway pressure

Systolic pressure variation (SPV) describes the fluctuations in arterial pressure which accompany mechanical ventilation, and results from changes in ventricular preload and afterload induced by alterations in intra-thoracic pressure.¹ The degree of systolic

pressure variation is increased in the presence of hypovolemia²⁻⁴ and has been used clinically as a predictor of responsiveness to a volume challenge.⁵⁻⁹ The effect of altering tidal volume on systolic pressure variation has been studied in ventilated dogs¹⁰ but has

only been studied in humans who are spontaneously breathing.¹¹ Left ventricular stroke volume variation, measured using pulse contour analysis, has been shown to be affected by tidal volume in patients who are mechanically ventilated.¹²

In an accompanying article, we described a novel technique which allows the simultaneous recording and archiving of airway pressure, as well as the systemic and pulmonary arterial pressure waveforms.¹³ We now describe the effects of altering tidal volume on these readings in two mechanically ventilated patients following elective cardiac surgery.

MATERIALS and METHODS

Approval for the study was obtained from the medical centre ethics committee, and signed consent was obtained from each patient studied. Both patients were intubated and mechanically ventilated following elective coronary artery bypass grafting for angina pectoris refractory to medical therapy and had radial arterial lines and right internal jugular pulmonary artery catheters inserted as part of routine care. Postoperative care, including inotropic and fluid therapy was dictated by the treating physician. Both patients were treated with milrinone and noradrenaline, and volume expansion was administered for systemic hypotension, oliguria, or reduced cardiac index in the context of low pulmonary arterial pressures. The ventilation mode was volume controlled ventilation and 5 cmH₂O of PEEP was applied. Patient 1 had mild renal dysfunction (plasma creatinine 0.125 mmol/L) and patient 2 had moderate LV dysfunction preoperatively.

Recordings of airway pressure, systemic arterial pressure and pulmonary arterial pressure were obtained as previously described.¹³ Following stabilisation in the intensive care unit, the tidal volume on the ventilator was altered over a one to two minute period, and continuous readings of pressure measurements were obtained. Two to four ventilator breaths with tidal volumes of 3, 5, 7, 9, and 11 mL/kg were delivered in a pre-allocated but random manner in an effort to eliminate any bias induced by a progressive increase in tidal volume over this range. In the case of patient 1 the order of the delivered breaths was 9, 3, 7, 11 and 5 mL/kg. Patient 2 received sequential tidal volumes of 7, 5, 3, 11, and 9 mL/kg.

A total of 17 sets of readings and 353 individual breaths were obtained for the two patients over a two to four hour time frame. Over the course of the readings patients 1 and 2 received three and four discrete episodes of volume expansion, respectively. Peak airway pressure was measured at the plateau phase of the insufflation, just prior to the pressure decent (figure 1). Systolic pressure variation and pulse pressure variation

were calculated as previously described.¹⁴ The same formulae were employed to calculate these variables for the pulmonary circulation.¹³ In addition, the systolic and diastolic blood pressures of the systemic and pulmonary circulations of the beat prior to insufflation were recorded as a reference point for the changes in pressures induced by insufflation (figure 1).

Statistical analyses

Correlations between peak airway pressure and systolic pressure variation were analysed using the Spearman - rank linear regression analysis on MS Windows Stat-view (Abacus Concepts, Berkeley, CA).

RESULTS

There was significant correlation between tidal volume and peak airway pressure over the range of tidal volumes delivered ($r = .968$ for patient 1; $r = .869$ for patient 2, $p < 0.0001$ for both). Increasing airway pressure resulted in a progressive increase in the degree of systolic pressure variation in the pulmonary and systemic arterial circulations (figures 2, 3, and 4).

In the systemic arterial circulation there was a significant correlation between peak airway pressure and the measured values of SPV, % SPV, and ΔPp (Figure 3). In the pulmonary circulation, significant correlation was also seen with PA-SPV and %PA-SPV. However, there was only a weak correlation between peak airway pressure and ΔPp for the pulmonary circulation (figure 4).

The relationship between the change in pulmonary arterial pressure and systemic arterial pressure was analysed to determine whether the effects of changing tidal volume on systolic pressure variation were consistent with the proposed physiological basis for arterial systolic pressure variation. We found a significant correlation ($r = 0.722$ for patient 1, and $r = 0.794$ for patient 2) between the increase in diastolic PAP associated with insufflation, and the maximum increase in systolic blood pressure (figure 5). Similarly, there was a significant correlation ($r = 0.656$ for patient 1 and $r = 0.516$ for patient 2) between the increase in systolic PAP associated with insufflation, and the maximum decrease in systolic blood pressure (figure 5).

DISCUSSION

Previous studies have shown that systolic pressure variation is increased in the presence of absolute or relative hypovolemia²⁻⁴ and systolic pressure variation has been used clinically as a predictor of cardiac output responsiveness to a volume challenge.⁵⁻⁹

However, these studies did not considered the impact of tidal volume and airway pressure on systolic pressure variation. The effect of altering tidal volume on systolic

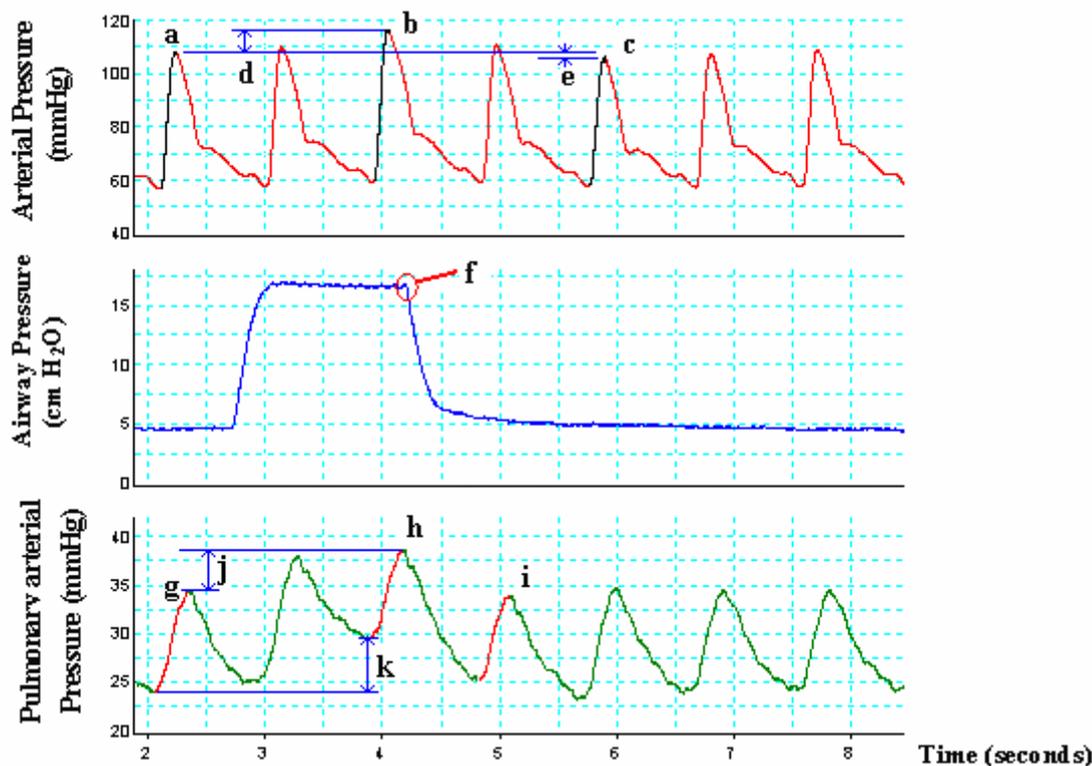


Figure 1. Simultaneous recordings of systemic arterial, airway, and pulmonary arterial pressure. **a** pre-insufflation beat for systemic arterial waveform **b** beat for maximum pressure for systemic arterial waveform **c** beat for minimum pressure for systemic arterial waveform **d** increase in systolic pressure associated with insufflation **e** decrease in systolic pressure associated with insufflation **f** peak airway pressure **g** pre-insufflation beat for pulmonary arterial waveform **h** beat for maximum pressure for pulmonary arterial waveform **i** beat for minimum pressure for pulmonary arterial waveform **j** increase in systolic PAP associated with insufflation **k** increase in diastolic PAP associated with insufflation.

pressure variation has been studied in mechanically ventilated dogs undergoing hypovolemia,¹⁰ but to our knowledge, this relationship has not been investigated in humans on mechanical ventilation.

In the present study, we demonstrated a strong correlation between the increase in peak airway pressure induced by increasing tidal volume, and the corresponding increase in systolic pressure variation in 2 patients who were mechanically ventilated following elective CABGS (figures 2 and 3). A similar effect was observed for the systolic pressure variation of the pulmonary arterial waveform (figures 2 and 4).

In patients with acute circulatory failure a Δ pulse pressure (Δ Pp) value of more than 13% predicts fluid responsiveness of cardiac output with a sensitivity of 94% and a specificity of 96%.⁵ However, this study did not take into account the effects of tidal volume on Δ Pp. In the present study, we were able to increase Δ Pp from 10% to 25% simply by increasing the peak airway pressure from 10 to 30 cmH₂O. This observation highlights the importance of taking into account the value of peak airway pressure when using systolic pressure variation as a marker of intravascular volume

status.

The physiological basis of systolic pressure variation is thought to relate to cyclical variations in aortic and pulmonary blood flow secondary to the transmitted intrathoracic pressures imposed on the great vessels.¹⁵ The initial increase in systolic blood pressure which is associated with insufflation, is thought to result from the increase in LV preload which is associated with emptying of the pulmonary capillaries.^{16,17} Concurrently, insufflation results in an increase in RV afterload, a reduction in RVEDV and the fall in systolic blood pressure over the subsequent cardiac cycles.^{17,18}

Our findings regarding the relationship between the changes in pulmonary arterial and systemic arterial pressure are consistent with the proposed physiological basis of systolic pressure variation. We analysed the effects of insufflation on the changes in pulmonary and systemic arterial pressure in comparison to the heart beat immediately pre-insufflation. There was a good correlation between the increase in diastolic PAP produced by insufflation and the corresponding increase in systolic blood pressure. Similarly, the increase in systolic PAP induced by insufflation, correlated well

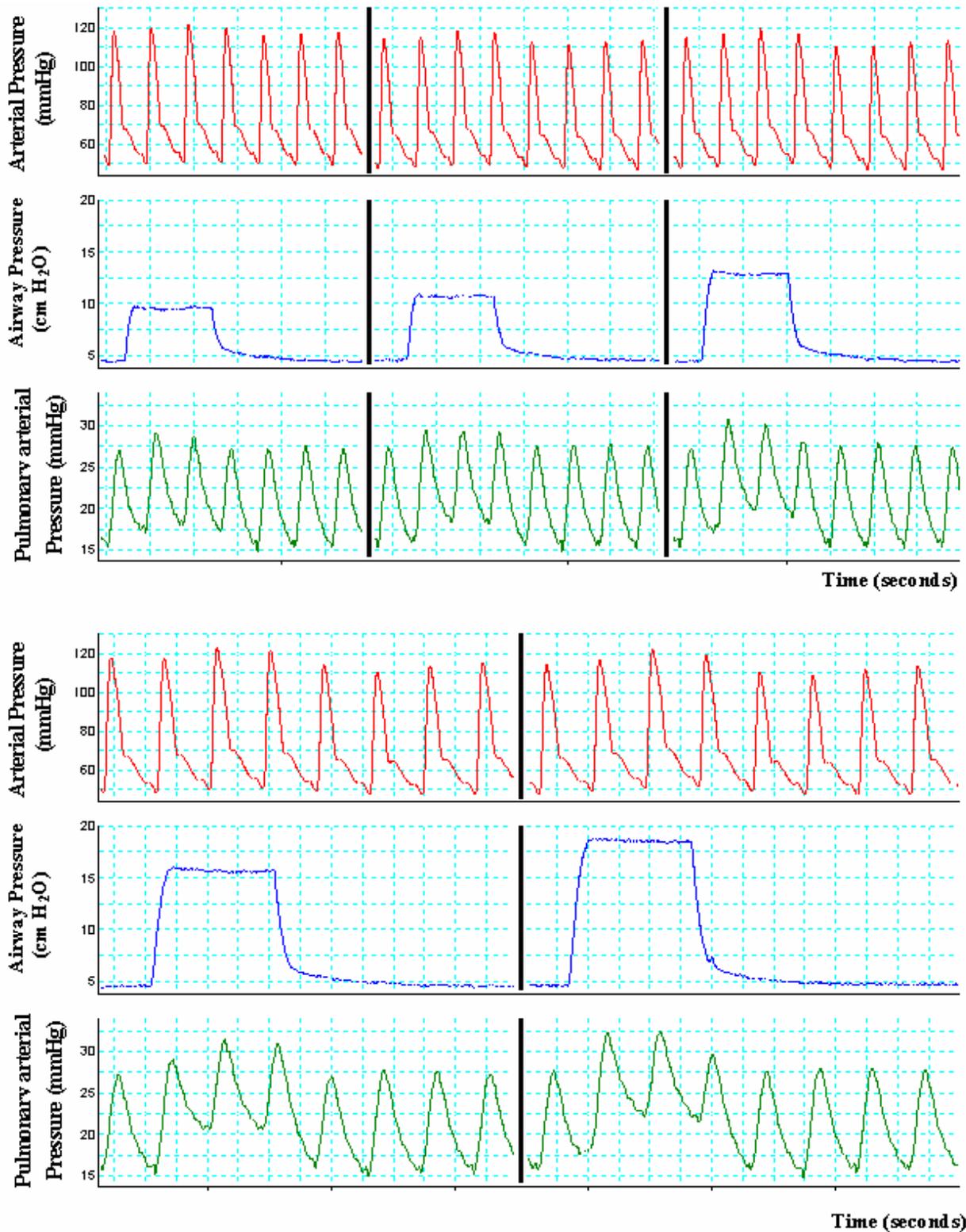


Figure 2. Graphic representation of the effects of increasing airway pressure on the systemic and pulmonary arterial waveforms. Tidal volume 3,5,7,9, and 11ml/kg were delivered over a 2 to 3 minute period in a random order.

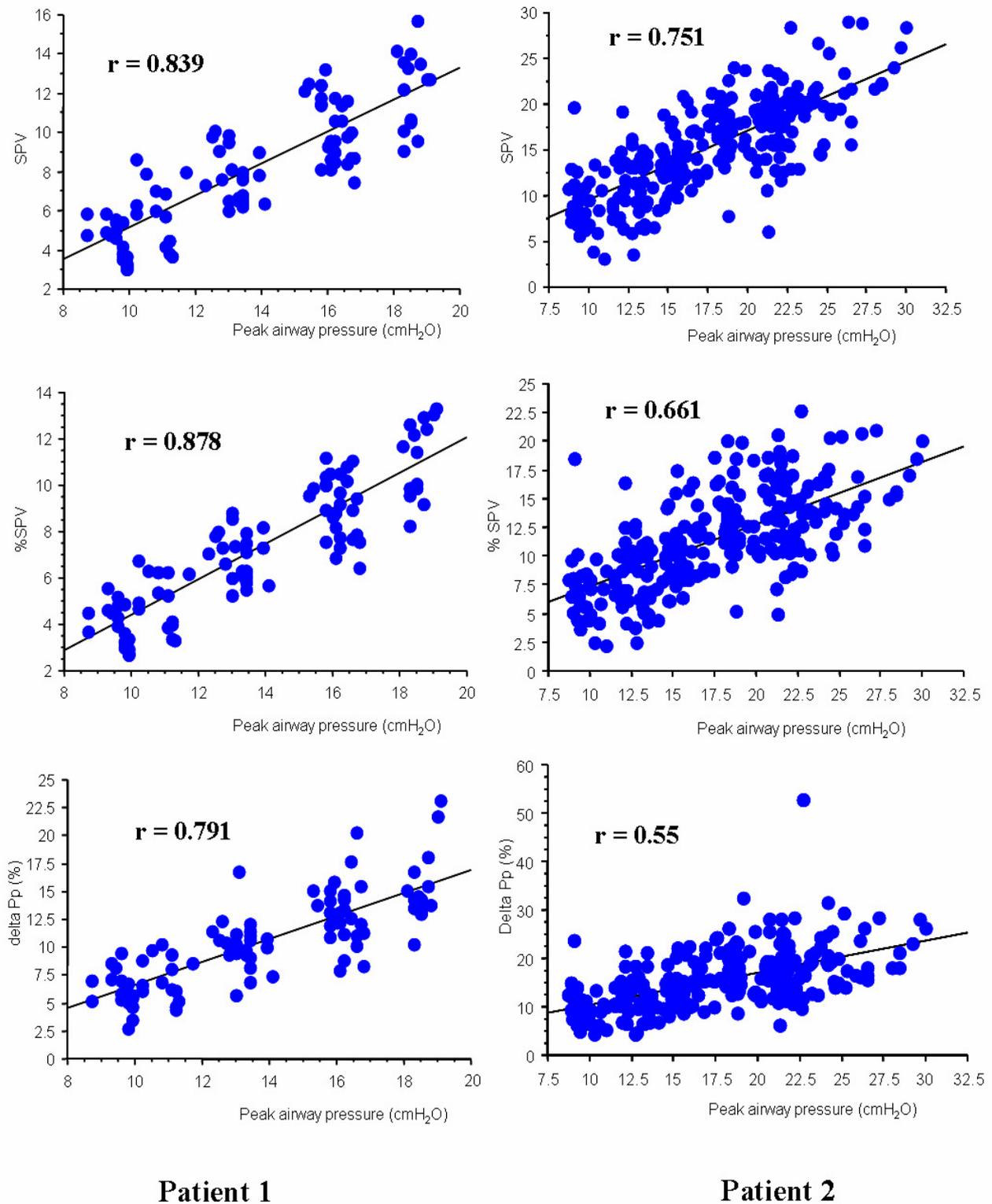
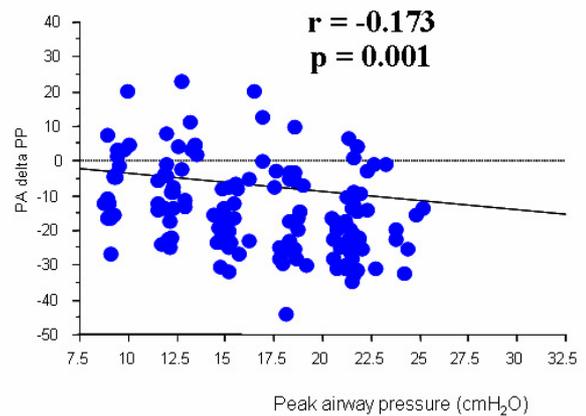
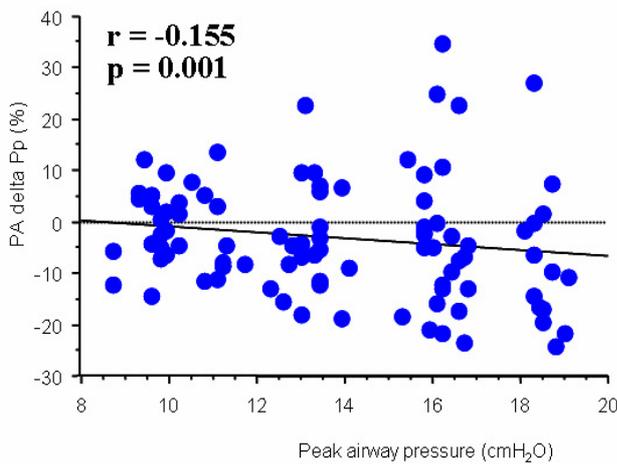
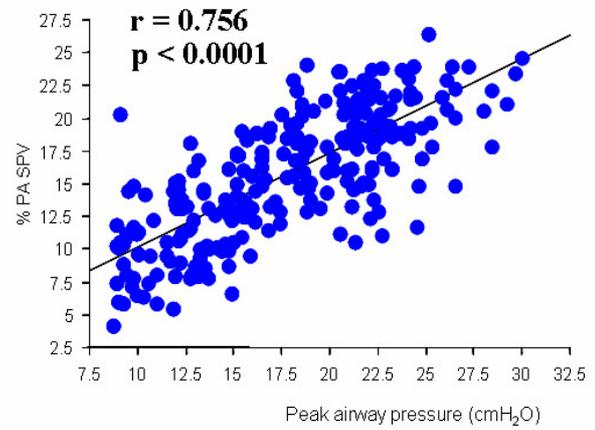
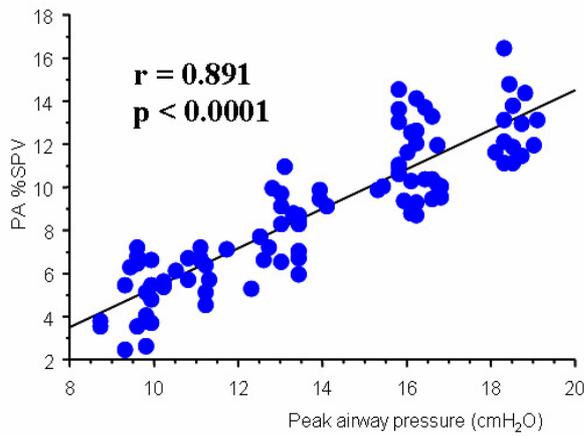
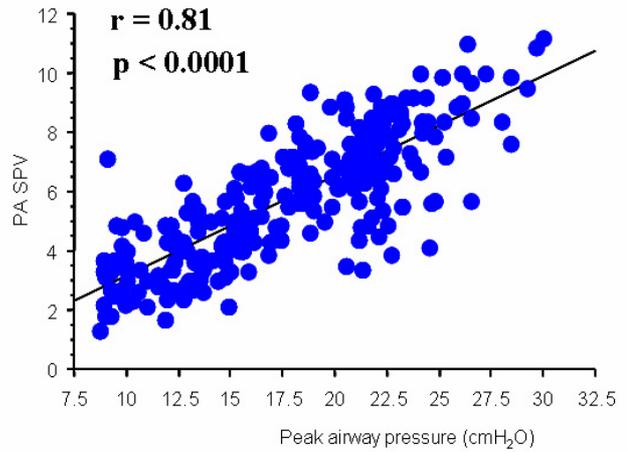
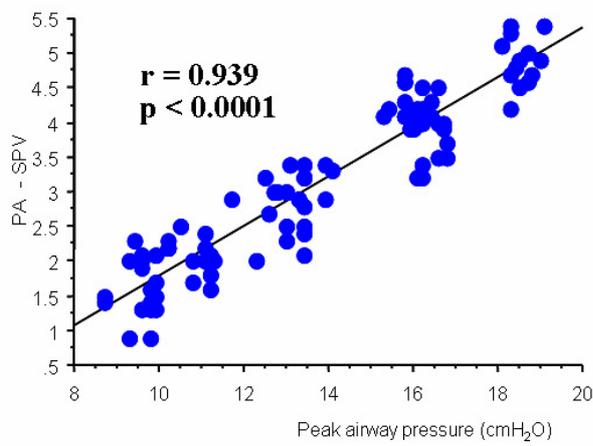


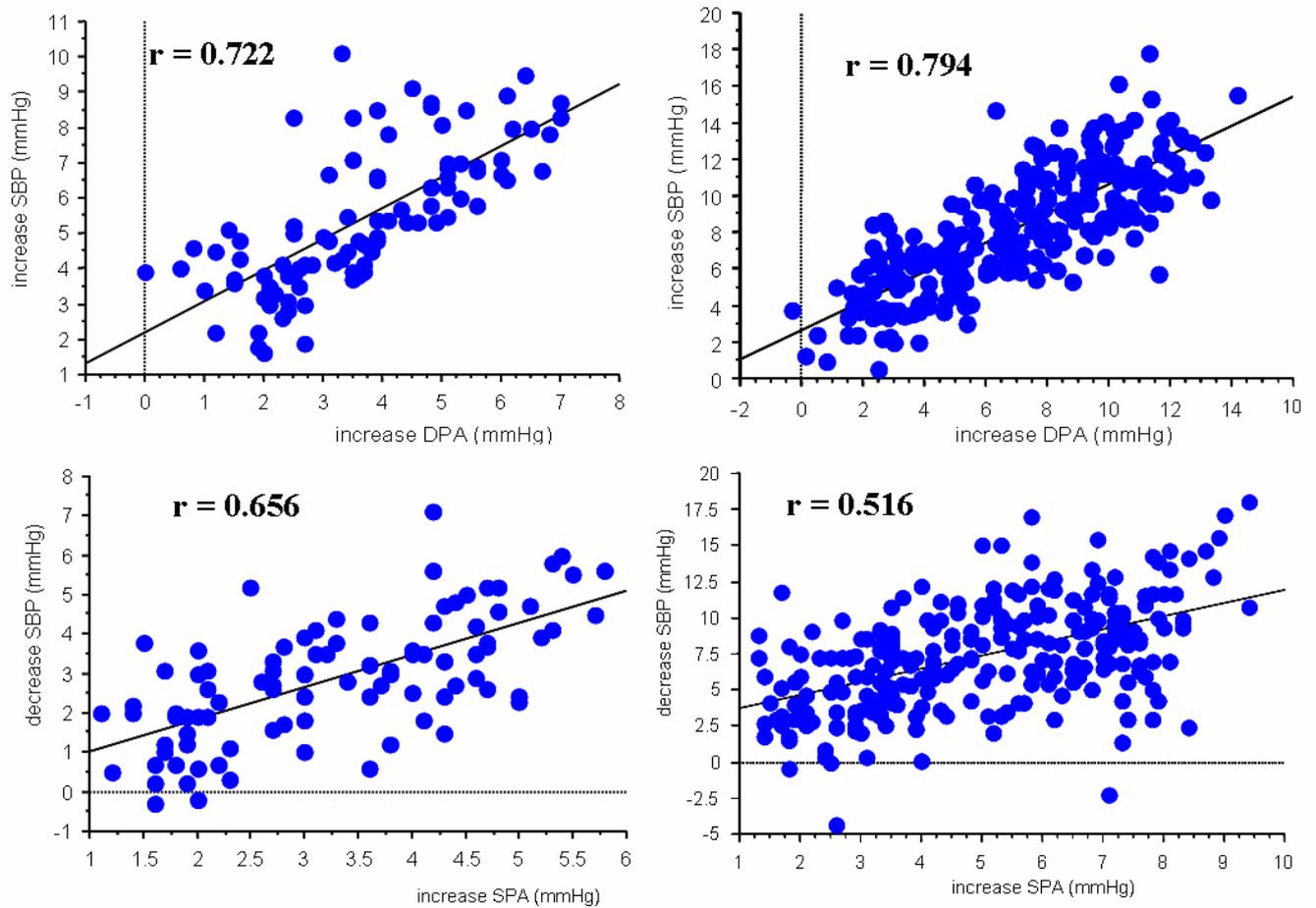
Figure 3. Scatter plots showing correlation between increasing peak airway pressure and SPV, %SPV, and ΔPp in the systemic circulation for 2 patients. $p < 0.0001$ for all data shown. SPV = systolic pressure variation, $\Delta Pp = \Delta$ pulse pressure



Patient 1

Patient 2

Figure 4. Scatter plots showing correlation between increasing peak airway pressure and SPV, %SPV and ΔPp in Pulmonary arterial pressure for 2 patients.



Patient 1

Patient 2

Figure 5. Scatter plots showing the correlation between the changes in pulmonary arterial pressure associated with insufflation and the corresponding effects on components of the systolic pressure variation. The maximum increase in systolic and diastolic PAP induced by insufflation were compared with the values seen in the beat prior to insufflation. $p < 0.0001$ for all data.

with fall in systolic blood pressure seen at the nadir of the systolic pressure variation.

We conclude that the magnitude of the prescribed tidal volume and corresponding airway pressure have a significant impact of the magnitude of systolic pressure variation of both the systemic and pulmonary arterial circulations. Thus, either should be taken into account when using systolic pressure variation as a marker of intravascular volumes status.

Our findings regarding the correlations between changes in the pulmonary arterial pressure and the systemic arterial pressure are consistent with the proposed physiological mechanism of systolic pressure variation. Further studies need to be conducted to determine the relative contribution of volume challenge and airway pressure on SPV and to correlate the ventilator induced changes in systolic pressure variation

with echocardiographic studies of trans-valvular Doppler flow and ventricular end-diastolic dimensions.

REFERENCES

1. Jardin F. Cyclic changes in arterial pressure during mechanical ventilation. *Intensive Care Med* 2004;30:1047-1050.
2. Coyle JP, Teplick RS, Long MC, Davison K. Respiratory variations in systemic arterial pressure as an indicator of volume status. *Anesthesiology* 1983;58:A53.
3. Perel A, Pziov R, Cotev S. Systolic blood pressure variation is a sensitive indicator of hypovolemia in ventilated dogs subjected to graded hemorrhage. *Anesthesiology* 1987;67:498-502.
4. Coriat P, Vrillon M, Perel A, et al. A comparison of systolic blood pressure variation and echocardiographic estimates of end-diastolic left ventricular size in patients after aortic surgery. *Anesth Analg* 1994;78:46-53.

5. Michard F, Boussat S, Chemla D, et al. Relationship between respiratory changes in arterial pressure and fluid responsiveness in septic patients with acute circulatory failure. *Am J Respir Crit Care Med* 2000;162:134-138
6. Bennett-Guerrero E, Kahn RA, Moskowitz DM, Falcucci O, Bodian CA. Comparison of arterial systolic pressure variation with other clinical parameters to predict the response to fluid challenges during cardiac surgery. *Mt Sinai J Med* 2002;69:96-100.
7. Travernier B, Makhotine O, Lebuffe G, Dupont J, Scherpereel P. Systolic pressure variation as a guide to fluid therapy in patients with sepsis-induced hypotension. *Anesthesiology* 1998;89:1313-1321.
8. Reuter DA, Felbinger TW, Kilger E, Schmidt C, Lamm P, Goetz AE. Optimizing fluid therapy in mechanically ventilated patients after cardiac surgery by on-line monitoring of left stroke volume variations. Comparison with aortic systolic pressure variation. *Br J Anaesth* 2002;88:124-126.
9. Ornstein E, Eidelman LA, Drenger B, Elami A, Pizov R. Systolic pressure variation predicts the response to acute blood loss. *J Clinical Anesthesia* 1997;10:21-25.
10. Szold A, Pziov R, Perel A. The effect of tidal volume and intravascular volume state on systolic pressure variation in ventilated dogs. *Intensive Care Med* 1989;15:368-371.
11. Rooke GA, Schwid HA, Shapira Y. The effect of graded hemorrhage and intravascular volume replacement on systolic pressure variation in humans during mechanical and spontaneous ventilation. *Anesth Analg* 1995;80:925-932.
12. Reuter DA, Bayerlein J, Goepfert MSG, et al. Influence of tidal volume on left ventricular stroke volume variation measured by pulse contour analysis in mechanically ventilated patients. *Intensive Care Med* 2003;29:476-480.
13. Jones DA, Baldwin I, Bellomo R. A technique for the determination of systolic pressure variation in the systemic and pulmonary arterial circulations. *Critical Care and Resuscitation* 2004;6:204-208.
14. Michard F, Chelma D, Richard C, et al. Clinical use of respiratory changes in arterial pulse pressure to monitor the hemodynamic effects of PEEP. *Am J Respir Crit Care Med* 1999;159:935-939.
15. Morgan BC, Martin WE, Horbein TF, Crawford E, Gunteroth WG. Hemodynamic effects of intermittent positive pressure respiration. *Anesthesiology* 1966;27:584-590.
16. Jardin F, Farcot JC, Gueret P, Post JF, Ozier Y, Broudarias JP. Cyclic changes in arterial pressure during respiratory support. *Circulation* 1983;68:266-274.
17. Viellard-Baron A, Chergui K, Augarde R, et al. Cyclic changes in arterial pulse during respiratory support revisited by Doppler echocardiography. *Am J Respir Crit Care Med*. 2003;168:671-676.
18. Jardin F, Delorme G, Hardy A, Auvert B, Beauchet A, Bourdarias J-P. Reevaluation of hemodynamic consequences of positive pressure ventilation: emphasis on cyclic right ventricular afterloading by mechanical lung inflation. *Anesthesiology* 1990;72:966-970.