Stroke Volume, Pulse Pressure, PVI and PI during Spontaneous Ventilation in a Model of Hypovolemia.

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Introduction

Dynamic variables predict fluid responsiveness during mechanical ventilation. Non-invasive detection of evolving hypovolemia during spontaneous ventilation would be of importance in initial assessment of trauma patients. The pleth variability index (PVI) has been shown to detect hemodynamic changes during passive leg raise in spontaneous ventilation [1]. Corresponding studies have not been reported in models of hypovolemia. The perfusion index (PI) is related to stroke volume (SV) as it represents the relation between the dynamic and static component in photoplethysmography. The first aim of this study performed on spontaneously breathing volunteers was to explore the relation between PVI and changes in SV in a model of central hypovolemia using lower body negative pressure (LBNP). An additional aim was to relate changes in PI to corresponding changes SV.

Methods

Six spontaneously breathing healthy volunteers were studied at baseline and LBNP levels of -20, -40, -60 and -80 mm Hg. At each level, mean arterial pressure (MAP), heart rate (HR), PP, SV, PVI and PI were recorded and averaged over 1 min after 1 min of stabilization. MAP and PP were measured by Finapres, SV by suprasternal Doppler and PVI and PI by Masimo Radical-7 photoplethysmograph. Relations were evaluated by Pearson correlation.

Results

Changes in SV were significantly correlated to changes in non-invasive PP ($r=0.69$, $p<0.001$), but not PI ($r=-0.28$, $p=0.19$) (fig 1). PVI at each LBNP level was not significantly correlated to changes in SV from baseline ($r=0.10$, $p=0.64$) (fig 2). A weak correlation was found between corresponding changes in SV and PVI ($r=-0.35$, $p=0.10$)

Conclusion

During central hypovolemia in spontaneously breathing volunteers, non-invasively measured changes in PP correlates with changes in SV. We found no significant relation between the level or changes of PVI and changes in SV. This preliminary study performed on a limited number of volunteers raises concern for using PVI to assess hypovolemia in spontaneously breathing patients.

1) Keller et al. Crit Care. 2008;12(2):R37.

Hemodynamic data					
LBNP-level (mmHg)	Baseline	-20	-40	-60	-80
HR (beats min-1)	57(51-67)	56(49-61)	61(52-70)*	66(57-80)*	80(61-102)*
SV (ml)	77(59-87)	64(53-84)*	56(44-71)*	46(33-68)*	30(20-68)*
MAP (mmHg)	68(62-91)	83(64-97)	89(53-95)	88(57-93)	88(48-91)
PP (mmHg)	54(38-58)	53(31-63)	49(28-63)	48(26-60)	41(24-55)*
PI (%)	2.5(1.3-8.5)	2.1(1.0-7.8)*	1.4(0.7-6.3)*	1.9(1.0-4.7)	2.3(1.1-3.7)
PVI (%)	18(10-27)	23(9-27)	19(14-30)	22(12-33)	19(12-33)

Data are median(range). *=Significantly different from baseline, Wilcoxon signed ranks test, $p<0.05$.

Figure 1