

Pulse oximetry plethysmographic waveform during changes in blood volume

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Systolic pressure variation (SPV) and its dDown component have been shown to be sensitive factors in estimating intravascular volume in patients undergoing mechanical ventilation. In this study, ventilation-induced changes in pulse oximeter plethysmographic waveform were evaluated after removal and after reinfusion of 10% estimated blood volume. The plethysmographic waveform variation (SPV_{plet}) was measured as the difference between maximal and minimal peaks of waveform during the ventilatory cycle, and expressed as a percentage of the signal amplitude during apnoea. dUp_{plet} and dDown_{plet} were measured as the distance between the apnoeic plateau and the maximal or minimal plethysmographic systolic waveform, respectively. Intravascular volume was changed by removal of 10% of estimated blood volume and followed by equal volume replacement with Haemaccel. A 10% decrease in blood volume increased SPV_{plet} from mean 17.0 (SD 11.8)% to 31.6 (28.0)% ($P=0.005$) and dDown_{plet} from 8.7 (5.1)% to 20.5 (12.9)% ($P=0.0005$) compared with baseline. Changes in plethysmographic waveform correlated with changes in arterial SPV and dDown ($r=0.85$; $P=0.0009$). In the absence of invasive arterial pressure monitoring, ventilation-induced waveform variability of the plethysmographic signal measured from pulse oximetry is a useful tool in the detection of mild hypovolaemia.

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Clinical estimation of intravascular volume is a crucial and common issue in the management of patients during anaesthesia and intensive care. The waveform variability of systolic arterial pressure during positive pressure ventilation, termed systolic pressure variation (SPV), has been shown to be useful in estimating different haemodynamic conditions.^{1–4} SPV and its dDown component have been found to correlate well with variables that estimate heart preload (pulmonary artery occlusion pressure (PAOP) and left ventricular end-diastolic volume).^{3,5} In addition, SPV and dDown changes correlated with changes in cardiac output in patients undergoing spinal surgery.⁶ The main limitation of this method is that it requires the use of an intra-arterial cannula, an invasive procedure associated with multiple complications.^{7–9}

In addition to oxygen saturation values, the pulse oximeter displays the plethysmographic waveform of the pulse signal. The plethysmographic waveform represents changes in blood volume. This waveform has no absolute value for

assessment, and therefore it is not quantitative.¹⁰ Not unlike the arterial signal, the plethysmographic signal is characterized by two waveforms: a fast one, which results from heart stroke volume, and a second, slow frequency waveform, which is synchronous with respiration. Estimation of intravascular blood volume using the plethysmographic signal has been suggested in previous studies; however, these studies were not conducted under controlled haemodynamic conditions.^{11,12}

We have assessed ventilation-induced variations in the pulse oximetric plethysmographic waveform signal in patients undergoing controlled intravascular volume changes, and compared these findings with those obtained using arterial waveform analysis.

Patients and methods

We studied 12 anaesthetized patients (eight females, four males, median age 16.6 (range 13–31) yr, median weight

53.2 (SD 9.1) kg) with progressive adolescent idiopathic scoliosis (ASA I–II) undergoing instrumental posterior spine fusion involving haemodilution. The study was approved by the Institutional Human Investigation Committee, and verbal informed consent was obtained from all patients and their parents. Patients were premedicated with morphine 0.1 mg kg^{-1} i.m. and diazepam 0.2 mg kg^{-1} orally. Anaesthesia was induced with penthotal 5 mg kg^{-1} and vecuronium 0.1 mg kg^{-1} and maintained with $0.5 \pm 0.1\%$ isoflurane and 70% nitrous oxide in oxygen. Patients' lungs were ventilated with a tidal volume of 10 ml kg^{-1} at a frequency of 8–10 bpm adjusted to maintain end-tidal carbon dioxide partial pressure at 4.0–4.7 kPa. In addition to standard monitoring, patients were monitored with a radial artery catheter (Venflon 20G, Ohmeda AB, Heisingborg, Sweden) and a pulmonary artery catheter (Arrow 7F, Pennsylvania, USA). All haemodynamic variables and waveforms were obtained from a Datex AS-3 monitor and printer (Datex, Helsinki, Finland). Cardiac output was measured by thermodilution in triplicate, and all thermodilution curves were checked before the cardiac output value was obtained. The mean value of three measurements with a variability of less than 15% was accepted.

Patients were in the supine position with the arms at axillary level. Baseline variables were recorded 30–60 min after induction of anaesthesia and then 10% of the patient's estimated blood volume was drained passively into a blood donation bag over 10 min. After 5 min, a further set of recordings were made. Haemaccel (3.5% gelatin solution, Behringwerke AG, Marburg, Germany) was then given in a volume equal to shed blood over 10 min. After an additional 5 min, a third set of measurements and waveforms were recorded. No vasoactive drugs were used during the entire study. Autologous blood was re-transfused later during surgery.

Waveform analysis

All waveforms and variables were calculated using paper records obtained from the Datex AS-3 recorder. Arterial SPV was measured as the difference between maximal and minimal systolic arterial pressure. dUp and dDown were measured as the difference between maximal inspiratory and minimal expiratory systolic arterial pressures relative to systolic arterial pressure during short apnoea (Fig. 1).

The plethysmographic SPV (SPV_{plet}) was measured as the vertical distance between maximal and minimal peaks of waveforms during the ventilatory cycle and expressed as a percentage of the amplitude of the plethysmographic signal during apnoea (Fig. 2). The plethysmographic dUp (dUp_{plet}) and plethysmographic dDown ($\text{dDown}_{\text{plet}}$) were measured as the vertical distance between the apnoeic plateau and the maximal or minimal plethysmographic systolic peaks, respectively. Because the plethysmographic waveform has no units, we calculated the plethysmographic waveform variables (dUp_{plet} and $\text{dDown}_{\text{plet}}$) as a percentage of apnoeic amplitude height. SPV and SPV_{plet} were meas-

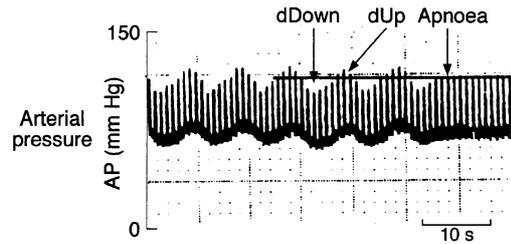


Fig 1 Arterial pressure (AP) waveform analysis. The horizontal line indicates systolic arterial pressure during apnoea. dDown is the difference between apnoeic and minimal systolic arterial pressure. dUp is the difference between maximal and apnoeic systolic arterial pressure. Systolic pressure variation (SPV) is the difference between maximal and minimal systolic arterial pressure.

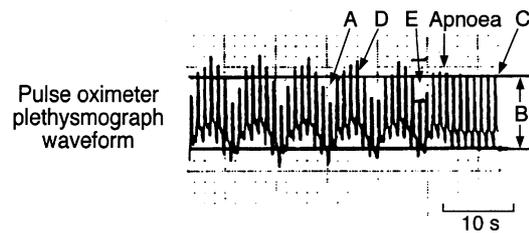


Fig 2 Plethysmographic waveform analysis. A: $\text{dDown}_{\text{plet}}$ is the distance between the minimal peak to the apnoeic plateau. B: Signal amplitude during apnoea. C: Apnoeic plateau. D: dUp_{plet} is the distance between the maximal peak to the apnoeic plateau. E: SPV_{plet} is the distance between maximal and minimal waveform peaks.

ured during five consecutive mechanical breaths before apnoea, and the mean value was obtained for analysis. All other variables were measured in the last ventilatory cycle before apnoea (so that SPV is not the sum of dUp and dDown). During the study, the gain of the plethysmographic waveform signal remained unchanged, as the Datex AS-3 monitor enables a choice between manual and automatic gain control.

Statistical analysis

Values after blood donation were compared with baseline values, and the variables after infusion of Haemaccel were compared with values obtained after blood donation. Comparison of values was performed using the Wilcoxon signed rank test. The plethysmographic waveform changes were compared with corresponding changes in the arterial waveform using the Spearman correlation test. Statistical significance was determined as $P < 0.025$. Data are presented as mean (SD).

Results

Blood volume reduction

Ten percent loss of estimated blood volume resulted in increased heart rate without changes in mean arterial pressure (Table 1). Central venous pressure and PAOP decreased after blood donation. Although cardiac output did not decrease, stroke volume decreased significantly (Table 1). Values of SPV and dDown, in both arterial and plethysmo-

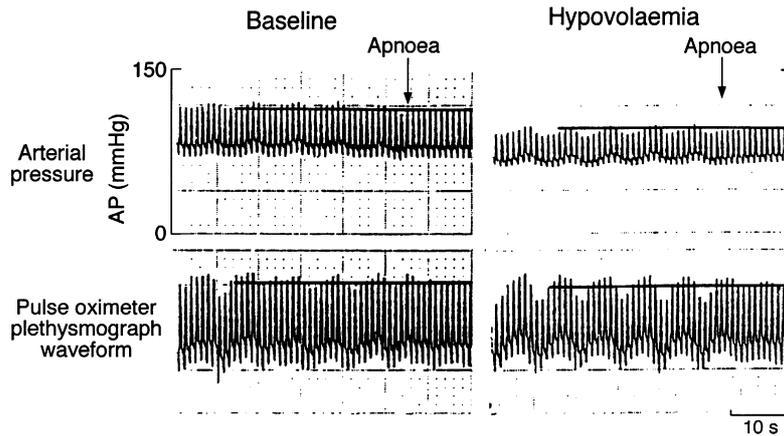


Fig 3 Arterial and plethysmographic waveform change. An example of arterial and plethysmographic waveform changes secondary to hypovolaemia. The horizontal line indicates the plethysmographic systolic signal during apnoea and apnoeic arterial pressure. Baseline: SPV 5.9 mm Hg, dDown_{plet} 15.8%, dDown_{plet} 5.3%. Hypovolaemia (after removal of 10% of estimated blood volume): SPV 8.26 mm Hg, dDown_{plet} 5.3 mm Hg, SPV_{plet} 33%, dDown_{plet} 22%.

Table 1 Haemodynamic variables during haemodilution (mean (SD)). Hypovolaemia=After removal of 10% of estimated blood volume; Replacement=after Haemaccel infusion; CVP=central venous pressure; PAOP=pulmonary artery occlusion pressure; PCV=packed cell volume; SPV=systolic pressure variation; SPV_{plet}=plethysmographic systolic pressure variation; dDown_{plet}=plethysmographic dDown; dUp_{plet}=plethysmographic dUp. **P*<0.025, hypovolaemia compared with baseline, and replacement compared with hypovolaemia

Variable	Baseline	Hypovolaemia	Replacement
Heart rate (beat min ⁻¹)	67.0 (8.7)	74.0 (8.6)*	65.0 (7.4)*
Mean arterial pressure (mm Hg)	62.0 (9.0)	62.8 (13.0)	66.7 (14.0)
CVP (mm Hg)	7.0 (3.9)	5.7 (4.0)*	9.0 (5.0)*
PAOP (mm Hg)	8.8 (4.0)	6.1 (4.0)*	10.8 (5.1)*
Cardiac output (litre min ⁻¹)	3.8 (0.9)	3.7 (0.6)	5.0 (1.4)*
Stroke volume (ml)	61.2 (14.9)	54.5 (14.0)*	87.9 (39.6)*
PCV (%)	34.3 (4.3)	34.7 (3.7)	30.7 (4.0)*
SPV (mm Hg)	6.7 (2.7)	9.6 (3.2)*	6.0 (2.2)*
dDown (mm Hg)	1.6 (1.3)	6.3 (3.1)*	1.7 (1.8)*
dUp (mm Hg)	4.8 (2.0)	3.2 (2.5)	4.5 (3.2)
SPV _{plet} (%)	17.0 (11.8)	31.6 (28.0)*	12.7 (7.9)*
dDown _{plet} (%)	8.7 (5.1)	20.5 (12.9)*	7.3 (6.3)*
dUp _{plet} (%)	9.9 (10.1)	6.8 (9.4)	6.9 (6.4)

Table 2 Spearman correlation coefficient between the change in arterial and plethysmographic waveforms. Hypovolaemia=Changes after removal of 10% of estimated blood volume compared with baseline; Replacement=changes after Haemaccel infusion compared with baseline; SPV_{plet}=plethysmographic systolic pressure variation; dDown_{plet}=plethysmographic dDown; dUp_{plet}=plethysmographic dUp; SPV=systolic pressure variation

Variable	Hypovolaemia		Replacement		
	<i>r</i>	<i>P</i>	<i>r</i>	<i>P</i>	
SPV	SPV _{plet}	0.85	0.0009	0.36	0.27
dDown	dDown _{plet}	0.72	0.013	0.43	0.19
dUp	dUp _{plet}	-0.21	0.52	-0.49	0.12

graphic waveforms, increased significantly after blood withdrawal (Table 1, Fig. 3). Changes in the plethysmographic waveform after blood withdrawal correlated well with corresponding changes in the arterial waveform. The correlation was significant for both SPV_{plet} and its dDown_{plet} component (Table 2). The dUp component of both curves

did not change significantly. Packed cell volume remained unchanged after blood donation.

Blood volume replacement

After volume replacement with Haemaccel, heart rate decreased while arterial pressure remained unchanged (Table 1). Although volume replacement with Haemaccel was equal in volume to donated blood, central venous pressure, PAOP, stroke volume and cardiac output increased above baseline values (Table 1). SPV_{plet}, dDown_{plet}, SPV and dDown decreased (Table 1), although the changes in these two waveforms did not correlate well (Table 2). There were no significant changes in dUp and dUp_{plet} as a result of volume replacement. Packed cell volume decreased significantly after volume replacement with Haemaccel from 34.3 (4.3)% to 30.7 (4.0)% (*P*=0.0034).

Discussion

The pulse oximeter is a standard monitor in anaesthesia and intensive care. In addition to monitoring arterial oxygen saturation, the pulse oximeter displays the plethysmographic waveform of the signal. In this group of patients, mild deliberate hypovolaemia caused a significant increase in SPV_{plet} and its dDown_{plet} component. Although the mechanism of the ventilation-induced variability in plethysmographic signal is unclear, changes after blood loss were seen similarly in both plethysmographic and arterial waveforms. The increase in both SPV and SPV_{plet} was mostly a result of the increase in the dDown components of the curves. This supports previous experimental and clinical data that showed that the SPV increase during hypovolaemia was caused only by the dDown component.¹⁻³ SPV and its dDown component of the arterial waveform give an accurate estimation of cardiac preload.¹⁻⁶ Both arterial and plethysmographic waveforms express stroke volume, and their visual similarity is so obvious that there have been several

attempts to estimate haemodynamic changes by analysing changes in plethysmographic waveform.^{11 12} The main problem in the analysis of the plethysmographic waveform is quantification of the signal. There are no units by which to measure the plethysmographic signal.¹⁰ Partridge measured SPV_{plet} (in mm) from the printed output, but the scale of the signal was adapted automatically which complicated signal analysis.¹¹ We used the amplitude of the plethysmographic signal during apnoea as a standard for calculating waveform variability. All plethysmographic variables were expressed as a percentage of this value (Fig. 2). This allowed us to measure ventilation-induced variability relative to the most stable signal amplitude (one recorded during a short apnoeic period) and thus to compare values between patients despite the lack of units.

The mechanism of the increased dDown during hypovolaemia has been explained by increased sensitivity of heart stroke volume to respiratory-induced fluctuations in preload.^{1 13} Whether the same explanation can be applied to the plethysmographic curve is unclear. The plethysmographic signal was found to be very sensitive to vasoconstriction, which makes it a less accurate expression of changes in stroke volume than the arterial waveform.¹² A more accurate analysis of the changes in plethysmographic signal was beyond the scope of our study.

The degree of hypovolaemia induced in our study can be defined as mild.¹⁴ Although our patients were anaesthetized, this mild hypovolaemia caused an increase in heart rate that kept cardiac output at the same level, although stroke volume and PAOP decreased significantly. Statistically there was a change in central venous pressure after blood donation, but the change was not clinically important, and this haemodynamic variable is still considered less sensitive than arterial waveform variability in diagnosing mild hypovolaemia.^{1 6}

Our study supports previous observations which found that volume replacement with Haemaccel resulted in a haemodynamic condition very different from baseline conditions, a phenomenon known as acute isovolaemic haemodilution.^{15 16} Cardiac output, central venous pressure and PAOP increased to values greater than baseline. The arterial waveform recovered to baseline values as a result of infusion of Haemaccel. Although volume replacement returned SPV_{plet} and $dDown_{plet}$ to values similar to baseline, there was no correlation between the magnitude of changes in the arterial and plethysmographic waveforms after volume replacement with Haemaccel (Table 2). This may be explained by the high sensitivity and variability of the plethysmographic signal to humoral and neurogenic factors.¹²

Although SPV_{plet} and $dDown_{plet}$ are defined in a fashion comparable with the definition of arterial SPV, the two SPV components are not truly comparable. The difference in their calculation could influence the ability of SPV_{plet} to predict arterial SPV or the presence of hypovolaemia. In

addition, we have studied only young healthy individuals and our results refer to this population only.

In summary, we have demonstrated that decreased preload resulted in waveform variation of the plethysmographic signal similar to the variation observed in the arterial waveform. Studies to assess ventilation-induced plethysmographic waveforms during volume replacement, and in different haemodynamic conditions, are needed. Respiratory-induced waveform variability of the pulse oximetric plethysmographic signal is a useful measurement in the detection of mild hypovolaemia.

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