Original Article

Relationship between pulse pressure variation and stroke volume variation with changes in cardiac index during hypotension in patients undergoing major spine surgeries in prone position - A prospective observational study

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Abstract

Background and Aims: Dynamic indices such as pulse pressure variation (PPV) and stroke volume variation (SVV) are better predictors of fluid responsiveness than static indices. There is a strong correlation between PPV and SVV in the prone position when assessed with the fluid challenge. However, this correlation has not been established during intraoperative hypotension. Our study aimed to assess the correlation between PPV and SVV during hypotension in the prone position and its relationship with cardiac index (CI).

Material and Methods: Thirty patients aged 18–70 years of ASA class I–III, undergoing spine procedures in the prone position were recruited for this prospective observational study. Hemodynamic variables such as heart rate (HR), mean arterial pressure (MAP), PPV, SVV, and CI were measured at baseline (after induction of anesthesia and positioning in the prone position). This set of variables were collected at the time of hypotension (T-before) and after correction (T-after) with either fluids or vasopressors. HR and MAP are presented as median with inter quartile range and compared by Mann-Whitney U test. Reliability was measured by intraclass correlation coefficients (ICC). Generalized estimating equations were performed to assess the change of CI with changes in PPV and SVV.

Results: A statistically significant linear relationship between PPV and SVV was observed. The ICC between change in PPV and SVV during hypotension was 0.9143, and after the intervention was 0.9091 ($P < 0.001$). Regression of changes in PPV and SVV on changes in CI depicted the reciprocal change in CI which was not statistically significant.

Conclusion: PPV is a reliable surrogate of SVV during intraoperative hypotension in the prone position.

Keywords: Cardiac index, FloTrac™, hypotension, prone, pulse pressure variation, stroke volume variation, vigileo

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Introduction

Hemodynamic fluctuations seen intraoperatively are multifactorial. Fluid therapy is a double-edged sword and poses a therapeutic dilemma for the anesthesiologist.[1] In the past, traditional estimates of blood volume such as central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) have been relied upon in the management of intraoperative hypotension. However, both the variables are unreliable both in estimating preload and in predicting the responsiveness to fluid therapy.

Dynamic indices, which are based on cardiopulmonary interactions such as pulse pressure variation (PPV), stroke volume variation (SVV), systolic pressure variation (SPV), Pleth variability index (PVI), and inferior vena cava diameter (IVCD) are reliable predictors of fluid responsiveness.[2-4]

Intraoperatively, derivatives of arterial pressure waveform analysis such as PPV and SVV have been demonstrated to be useful indicators in guiding intraoperative fluid therapy by various trials. Although PPV is easily measured (Intellivue MP50 monitor, Philips Medical Systems, Boeblingen, Germany), the measurement of SVV requires a specialized monitor (FloTrac™/Vigileo system, Edward LifeSciences, Irvine, California, USA), which is not easily available. Studies have established a good correlation between SVV and PPV to fluid challenge suggesting that PPV can be used as a surrogate to SVV with a correlation coefficient of 0.769.[3,5]

To our knowledge, no studies have compared the correlation between PPV and SVV during periods of intraoperative hypotension to assess fluid or vasopressor responsiveness. We hypothesized that the correlation between PPV and SVV will decrease during periods of intraoperative hypotension in the prone position, and that PPV may not be a reliable indicator of fluid responsiveness in the prone position as SVV.

The primary objective of our study was to compare the correlation between PPV and SVV during hypotension and after the restoration of blood pressure to baseline in patients undergoing spine surgery in the prone position. The secondary objective was to assess the change in CI with changes in PPV and SVV.

Material and Methods

After obtaining approval of the Institutional Review Board (IRB Min. No. 8562 [OBSERVE]) and Ethics Committee, 30 patients fulfilling the inclusion criteria and scheduled for spine procedures in the prone position were recruited for this prospective observational study in a tertiary hospital, from January 2014 to December 2014. The inclusion criteria were 18–70 years of age, American Society of Anesthesiologists (ASA) class I–III patients, elective major spine instrumentation procedures, surgery in the prone position. Excluded from the study were patients unwilling to participate, undergoing emergent surgery, with body mass index (BMI) >30 kg/m^2, valvular heart disease, left ventricular dysfunction (ejection fraction <50%), arrhythmias, conduction disturbances, patients with chronic obstructive lung disease and interstitial lung disease. A written informed consent was obtained by the primary investigator a day before surgery.

A standard anesthesia protocol was followed for all patients who were premedicated with diazepam 0.1 mg/kg and omeprazole 20 mg orally in the morning of the surgery. In the operating room, standard monitoring, such as a 5-lead electrocardiogram, pulse oximeter (SpO_2), non-invasive arterial blood pressure, was established. An 8-cm, 3 French catheter (Leadercath Arterial; Vygon, United Kingdom) was inserted into the right or left radial artery after infiltrating skin with 2% lignocaine. The pressure transducer was zeroed at the phlebostatic axis and coupled to both Philips Intellivue MP50 monitor (Philips Medical Systems, Boeblingen, Germany) and the FloTrac™/Vigileo system (Edward LifeSciences, Irvine, California, USA).

Anesthesia was induced with fentanyl (1–2 μg/kg), propofol (1.5–2 mg/kg), and tracheal intubation facilitated with vecuronium (0.1 mg/kg). After confirmation of the endotracheal tube position, ventilation was controlled to maintain the end-tidal carbon dioxide ($EtCO_2$) of 35–40 mm Hg, with a tidal volume of 8 mL/kg, respiratory rate of 12–16/min, inspiratory: expiratory ratio (I: E) of 1:2, positive end-expiratory pressure (PEEP) of 5 cm water, fractional inspired oxygen (FiO_2) of 40%, and peak airway pressure maintained to <25 cm of water. Minimum alveolar concentration (MAC) of isoflurane was maintained at 0.8–1.0, and neuromuscular blockade was maintained with vecuronium infusion (0.001 mg/kg/min), and additional analgesia was provided with titrated doses of morphine (up to 0.1 mg/kg), fentanyl (up to 5 μg/kg), and intravenous paracetamol (1 gm). Intravenous crystalloids (10 mL/kg) were administered to all subjects before positioning in the prone position to counteract the relative hypovolemia secondary to the redistribution of central intravascular volume to the peripheries.

The patients were carefully positioned on the Relton Hall Operating Frame with chest and pelvic supports, with special attention to avoid abdominal compression and hemodynamic instability. The respiratory parameters such as peak and plateau airway pressures, tidal volume, and static and dynamic

compliance were monitored and standardized as per the protocol.

Hemodynamic variables such as heart rate (HR), mean arterial pressure (MAP), PPV, SVV, and cardiac index (CI) measured 5 min after achieving hemodynamic stability in the prone position were considered as the baseline.

Thereafter, all incidents of significant hypotension (20% decrease in MAP from baseline) intraoperatively, and the concomitant changes in PPV, SVV, and CI were recorded (T- before). These events were treated with either volume expansion (crystalloids or colloids) or vasopressors (noradrenaline and phenylephrine), depending on the clinical discretion of the attending anesthesiologists. Once the MAP was restored to the baseline value, the changes in PPV, SVV, and CI were recorded again (T-after). Depending on the occurrence of such events, a minimum of a single pair and a maximum of five pairs (T1-T5) of recordings in series were obtained from each patient. The data collection was done by the primary investigator and stored in a password-protected computer. The Philips Intellivue MP50 monitor displays real-time arterial pressure waveforms and PPV value as a percentage, using proprietary detection algorithms.

PPV (%) = (Pulse pressure$_{maximum}$ - Pulse Pressure$_{minimum}$)/ Pulse Pressure$_{mean}$ and averaged over four cycles of 8 s. The FloTrac™/Vigileo system analyzes the arterial pressure signal and computes the SV, SVV, and CI without external calibration.

SVV (%) = (Stroke Volume$_{maximum}$ – Stroke Volume$_{minimum}$)/ Stroke Volume$_{mean}$).

The mean, minimum, and maximum SV over a 20 s period were determined, and parameters were displayed continuously.

All analyses were done by Statistical package for the social sciences (SPSS IBM SPSS Statistics for Windows, Version 21.0. Armonk, NY: IBM Corp.). Descriptive statistics were performed by mean with standard deviation for continuous variables and frequency with percentage for categorical variables. The hemodynamic variables monitored during hypotension and after intervention are presented as median with inter quartile range (IQR) and the comparison made by the Mann-Whitney U test. A P value < 0.05 was considered significant. Scatter plots between PPV and SVV were done separately during hypotension and after the intervention. Reliability was measured by intraclass correlation coefficients (ICC) during hypotension and after the intervention. Bland Altman plot done to SVV and PPV measured fluid responsiveness on the same scale. Generalized estimating equations (GEE) were performed to find the change of CI with changes in PPV and SVV at T1-T5 with an exchangeable correlation structure.

Biais *et al.*[3] reported a significant correlation (0.79) between PPV and SVV in response to fluid loading in the prone position. In comparison to the results from the study by Bias *et al.*,[3] we hypothesized a 20% reduction in correlation between PPV and SVV values during intraoperative hypotension, and accordingly, a sample size of 30 subjects was calculated by considering alpha and beta errors as 5% and 20%, respectively.

Results

We screened 49 patients for eligibility and enrolled 30 consecutive eligible consenting adults scheduled for elective spine surgery in the prone position. One subject was excluded from analysis as complete data could not be acquired due to technical error. The flow of participants is presented in Figure 1. The demographic characteristics and accompanying co-morbid illness of the remaining 29 subjects are presented in Table 1.

Descriptive paired test analysis of hemodynamic variables during and after treatment of hypotension at five different time points (T1 to T5, Before and After) are presented in Table 2. Of the 110 episodes of hypotension that were studied, fluid challenge alone was administered in 16 (14.5%), vasopressors alone were used in 60 (54.54%), and a combination of both was used in 34 patients (30.9%). For every significant change in the MAP, there was a concomitant significant change in both PPV and SVV ($P < 0.05$) at all time points. The change in CI was statistically significant ($P < 0.05$); however, no statistically significant change was observed with the heart rate.

Furthermore, the mean changes of PPV and SVV show a statistically significant linear relationship ($P < 0.05$). The CI shows a tendency towards a reciprocal relationship with both PPV and SVV, however, this is not statistically significant and will require larger numbers to demonstrate significance.

The ICC between the change in PPV with the change in SVV during periods of hypotension and after correction of the same is illustrated in Table 3. There is a strong positive correlation between PPV and SVV at all time points, the ICC is 0.9143 during the hypotension and 0.9091 after the intervention, and P value < 0.001, this indicates a substantial agreement between these methods. Figure 2a shows the Bland Altman Plot used to compare SVV and PPV, and the

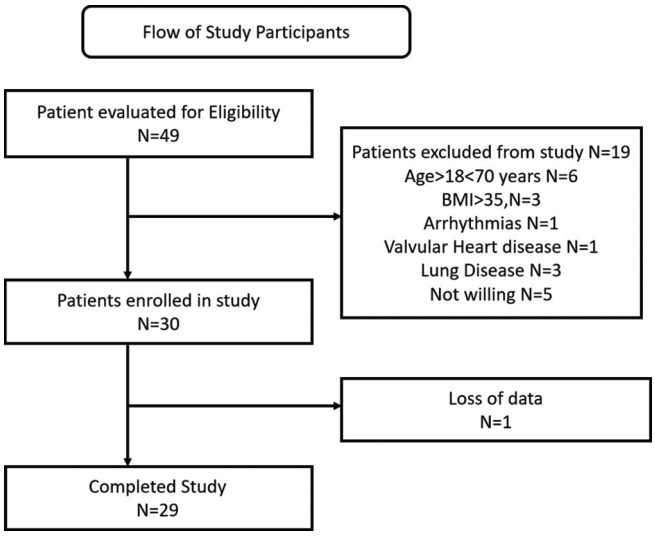

Figure 1: Study flow chart

scatter plot 2b represents the strength and magnitude of the correlation between PPV and SVV during the hypotension. A similar analysis to assess the correlation between PPV and SVV after the intervention is shown in Figures 2c and 2d, and both demonstrate a high degree of correlation between PPV and SVV. Table 4 shows the regression of changes in PPV and SVV, on changes in CI, as assessed using GEE, an exchangeable correlation structure was used. The mean change in PPV and SVV by one unit is accompanied by a reciprocal change in the CI, which is not statistically significant ($P > 0.05$).

Discussion

The results of our study show that during intraoperative hypotension and after the restoration of the MAP to baseline, either with volume/vasopressors or both, there is a significant change of PPV and SVV at each time point with a corresponding change in CI. Our data also suggest that there is a strong positive correlation between changes in PPV and SVV. Although there was a reciprocal change in CI with changes in PPV and SVV, it did not assume statistical significance.

Precise estimation of tissue perfusion is critical to guarantee adequate oxygen delivery as overzealous fluid administration predisposes to perioperative complications and delays discharge.[6] Determination of volume status by static measures is imprecise and has been outdated by dynamic indices of fluid responsiveness such as PPV, SVV, systolic pressure variation (SPV), pleth variability index (PVI), inferior venacaval diameter, and aortic doppler flow that determine preload based on cardiopulmonary interactions.[7-10] Transthoracic echocardiographic and

Table 1: Patient characteristics

Characteristics	Mean (Range)
Age	56.4±7.65
Sex (male/female) %	11/18 (37.9/62.1)
ASA classification (I/II/III) (%)	0/26/3 (89.66/10.34)
Anesthesia duration (min)	226±57.3 (120-330)
Blood loss (mL)	667±576.03
Levels operated (number)	2.5 (2-6)
Hypertension (%)	24 (82.76)
Diabetes Mellitus (%)	15 (51.72)
Ischemic Heart Disease (%)	3 (10.35)
Both Diabetes and Hypertension (%)	13 (44.83)

All data are expressed as mean (SD) or number of patients (percentages). Levels operated is expressed as mean (range)

esophageal doppler assessment of cardiac output (CO) are often challenging to use in the prone position. The alteration in the left ventricular output is the primary contributing factor for the change in pulse pressure and when ventricular preload and stroke volume (SV) are modified by positive pressure ventilation, which is reflected as PPV.[11]

The study by Biais *et al.*[3] has shown that there is an absolute increase in the PPV and SVV values and both retain their ability to predict fluid responsiveness. The patients were subjected to volume expansion (VE) with 500 mL of hetastarch 6%, in the supine and prone position, and volume responders exhibited a percentage increase in CI of at least 15%. Both PPV and SVV correlated with VE-induced changes in CO but defined a greater threshold in the prone (15%) as compared to the supine position (11%).

Comparison of PPV with corrected flow time (FTc) measured using the esophageal doppler showed that the predictability of PPV was significantly higher than that of FTc in the prone position, hence a useful index for guiding fluid therapy with minimal alterations in their optimal cutoff values.[4] A more recent study demonstrated that PPV in the prone position can predict fluid responsiveness as good as PPV in the supine, only if BMI is <30 kg/m^2, static respiratory compliance value in prone is >31 mL/cmH_2O, and a tidal volume of at least 8 mL/kg is delivered.[12,13] PPV is not a reliable monitor in the intensive care unit (ICU), given the irregular rhythms and during spontaneous respiratory efforts.[14]

Unlike our study, the above-mentioned studies have demonstrated fluid responsiveness by a prescribed dose of crystalloid or colloid loading and the assessment of PPV and SVV thereafter. None of the studies to date have assessed the correlation between PPV, SVV, and CI in the clinical setting of hypotension. Our emphasis is on the ability of these dynamic indices to provide reliable therapeutic guidance during sudden hemodynamic fluctuations encountered during

Table 2: Descriptive paired test analysis of hemodynamic parameters during hypotension and following intervention

	Median (25th Percentile and 75th Percentile)				
Time	**HR**	**MAP**	**PPV**	**SVV**	**CI**
T1: Before	81 (69, 89.5)	58 (52, 64)	14.0 (10.5, 24)	15.5 (12, 20.5)	2.2 (1.7, 2.7)
After	76 (66.5, 94)	78 (69.5, 92)	13.5 (10.5, 20)	14.0 (10, 18)	3.0 (2.5, 3.3)
P	0.801	<0.001	0.014	0.047	<0.001
T2: Before	79.5 (59.5, 92)	59 (52.5, 65)	14.0 (10, 22)	16.5 (13.5, 20)	1.9 (1.5, 2.6)
After	70.5 (62, 83)	75.5 (72, 81.5)	12.0 (11, 17.5)	14.5 (12, 18)	2.8 (2.3, 3.4)
P	0.141	0.047	0.062	0.065	0.003
T3: Before	68 (59, 84)	55 (51, 59)	15.0 (12, 21)	15.0 (12, 19)	2.0 (1.7, 2.7)
After	67 (60, 87)	73 (68, 82)	15.0 (8, 19)	13.0 (11, 17)	2.9 (2.2, 3.2)
P	0.140	0.007	0.012	0.092	0.019
T4: Before	66.5 (58.5, 85)	55 (50.5, 59)	17.5 (13.5, 27)	17.5 (12.5, 22)	1.9 (1.6, 2.4)
After	73 (61, 86)	87 (77.5, 94)	13.0 (9.5, 15)	14.0 (12, 15)	3.0 (2.7, 3.9)
P	0.393	<0.001	0.001	0.010	<0.001
T5: Before	61 (57, 95)	53 (47, 57)	18.0 (16, 25)	18.0 (17, 22)	1.7 (1.5, 2)
After	79 (57, 82)	76 (72, 78)	12.0 (10, 16)	15.0 (11, 16)	2.8 (2.4, 3.2)
P	0.799	0.008	0.033	0.049	0.008

HR - heart rate, MAP - mean arterial pressure, PPV - pulse pressure variation, SVV - stroke volume variation, CI - cardiac index. T1, T2, T3, T4, and T5 - First, second, third, fourth, and fifth incidents of hypotension, respectively. Before - During Hypotension, After - After correction

Table 3: Intra-class correlation coefficient between change in PPV and change in SVV

	Intraclass Correlation Coefficient Before	Intraclass Correlation Coefficient After	Intraclass Correlation Coefficient Change
T1	0.92 (0.83-0.96)	0.94 (0.88-0.97)	0.89* (0.75-0.95)
T2	0.91 (0.79-0.96)	0.88 (0.73-0.95)	0.91* (0.79-0.96)
T3	0.88 (0.70-0.95)	0.91 (0.78-0.96)	0.78 *(0.48-0.91)
T4	0.90 (0.73-0.97)	0.84 (0.54-0.94)	0.90* (0.69-0.97)
T5	0.97 (0.86-0.99)	0.94 (0.75-0.99)	0.99* (0.93-0.99)
Overall	0.91 (0.87-0.94)	0.91 (0.86-0.94)	0.91* (0.86-0.94)

**All are highly significant with P<0.001*

major surgeries in the prone position rather than the assessment of fluid responsiveness to a fixed dose of volume expansion.

Intraoperative hypotension may be because of a variable combination of absolute hypovolemia following blood loss and/or relative hypovolemia secondary to anesthesia-induced vasodilatation. Prone position induces physiological changes such as an increase in intra-abdominal pressure (IAP), a change in systemic vascular resistance (SVR) with a complex effect on ventriculoarterial coupling, contributing to relative hypovolemia.[12]

An elegant experiment on anesthetized, mechanically ventilated rabbits that underwent progressive hypotension by either controlled hemorrhage or intravenous sodium nitroprusside infusionshowed that both graded hemorrhage and pharmacologic vasodilation induced a similar amplification in PPV. This reinforces the idea that increases in PPV does not necessarily represent hypovolemic status but rather indicate cardiovascular responsiveness to fluid infusion irrespective of the cause of hypovolemia, be it absolute or relative.[15] In a study of changes in SPV and PPV in 12 pigs, where CO

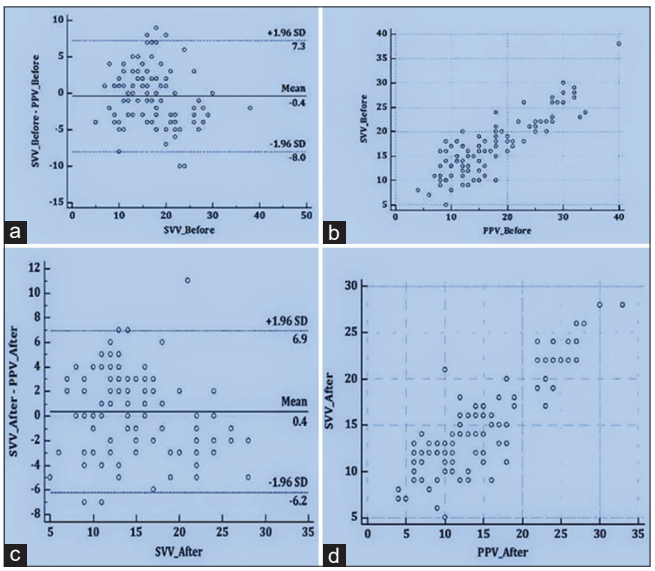

Figure 2: (a) Bland Altman Plot comparing SVV and PPV during hypotension (b) Scatter plot of the strength of correlation of SVV and PPV during hypotension (c) Bland Altman Plot comparing SVV and PPV after intervention (d) Scatter plot of the strength of correlation of SVV and PPV after intervention

was maintained primarily with vasopressors during a reduction in hemodynamics, they found that both SPV and PPV

Table 4: Regression of change in PPV, SVV on change in CI (Generalized estimating equations)

	Coefficient	95% CI	*P*
PPV vs CI Before			
Intercept	3.05	1.76-4.34	<0.001
PPV	-0.04	-0.09-0.02	0.195
PPV vs CI After			
Intercept	3.46	2.61-4.31	<0.001
PPV	-0.01	-0.05-0.03	0.579
SVV vs CI Before			
Intercept	3.55	1.97, 5.13	<0.001
PPV	-0.07	-0.15, 0.01	0.077
SVV vs CI After			
Intercept	4.05	2.84, 5.26	<0.001
SVV	-0.05	-0.11, 0.01	0.107

correlated with changes in SVV.[16] Therefore, in the absence of volume depletion, PPV is reliable when blood pressure is augmented by vasoconstrictors in the prone position.

Our study demonstrates the correlation between SVV and PPV in the setting of intraoperative hypotension irrespective of the etiology and the intervention. This is further reinforced by the fact that changes in PPV and SVV are not significantly related to changes in CI. In a similar study of patients with cervical myelopathy in the prone position, hypotension was not associated with a decrease in SV, CO, and CI as measured using the bioimpedance technique.[17] Measurement of the SVR would have provided an individualistic approach addressing the underlying cause for pulse and stroke volume variations.

In patients with comorbidities such as longstanding diabetes or hypertension, there is a theoretical possibility that PPV is unreliable as it is influenced by arterial compliance.[18] In our study, we found that PPV is as reliable as SVV even in diabetics and hypertensives. A very recent study has shown a similar positive correlation between SVV and PPV (0.732) using Captesia™ (GalenicApp, Vitoria, Spain). The CI measured using Captesia had a significant positive correlation with CI measured by Vigileo, although in the supine position.[19]

When compared to other pulse contour analyzing modalities like pulse contour cardiac output (PiCCO) and lithium dilution cardiac output (LiDCO), the FloTrac™ has limited reliability.[20]

Limitations

Measurement of SVR requires placement of a central venous catheter which is not often required for spine procedures. Hence, we could not quantify SVR, and the reduction in the CI was treated appropriately either with volume expansion with or without administration of vasopressors based on clinical assessment. Assessment of the changes in PPV and SVV to hypovolemia and systemic vasodilatation and its correlation with CO would have thrown more light into the reason for the reduction in cardiac index. Estimation of the systemic vascular resistance index (SVRI) would be highly informative in analyzing the reduction in CI and its effect on the dynamic indices. Moreover, the influence of intra-abdominal pressure on the dynamic indices of fluid responsiveness was not assessed. It also would have been interesting to know the changes from normotensive to hypotensive state. Larger sample sizes would have demonstrated a significant reciprocal relationship of CI with PPV and SVV.

Conclusion

Our findings suggest that in the setting of intraoperative hypotension in the prone position, both PPV and SVV are reliable predictors of fluid responsiveness with a strong correlation between them. Hence, PPV which could be derived from standard arterial line monitor could be utilized intraoperatively to reliably guide fluid therapy in the prone position when expensive cardiac output monitors are unavailable for clinical use. There is, however, no consistent significant relationship between changes in PPV and SVV with that of CI in the prone position. More studies with larger sample sizes are required in clinical settings to understand the complex interaction between the various hemodynamic factors responsible for fluid responsiveness in the prone position.

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Conflicts of interest

There are no conflicts of interest.

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