HEMODYNAMIC PROFILE OF PATIENTS HAVING CESAREAN DELIVERY UNDER SPINAL ANESTHESIA OBTAINED BY CONTINUOUS MEASUREMENT OF CARDIAC OUTPUT

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Background

❖There are 1.5 million Cesarean deliveries (CD) annually in the United States. Most are performed under spinal or epidural anesthesia.

❖Dramatic hemodynamic instability occurs frequently with spinal anesthesia which may be associated with adverse effects on parturients and newborns.

❖Development of non-invasive CO monitors has made continuous measurement of CO available and provides clinicians ability to closely monitor hemodynamic changes in a timely manner.

❖ The aims of this study were to:

➢1) continuously measure CO and stroke volume (SV) using electric velocimetry and establish the hemodynamic profile of patients having CD under spinal anesthesia;

➢2) determine if real-time awareness of the CO and SV by the care-team translates into improved hemodynamic stability.

Methods

Forty-two healthy patients scheduled for elective CD under either spinal or combined spinal and epidural anesthesia (CSE) were recruited at the Massachusetts General Hospital.

Hemodynamic monitoring:

➢Continuous CO and SV monitoring: ICON (Cardiotronic®, La Jolla, California, USA), using electrical velocimetry technique.

➢Intermittent peripheral venous pressure (PVP): IV line in the upper extremity, measured at predefined time points

➢Systemic vascular resistance (SVR): calculated after completion of the study

$$SVR\ (dyn \cdot s \cdot cm^{-5}) = 80 * (MAP - PVP)/CO$$

Baseline value: hemodynamic variables prior to initiation of spinal anesthesia, in left tilt supine position.

Randomization:

➢Group A (n=21): the physicians did not know the CO/SV and managed the patients in a standard fashion;

➢Group B (n=21): the physicians were aware of the CO and SV measurements in real time.

Statistics:

➢Primary outcome: the maximum change in MAP and CO in response to the spinal anesthesia.

➢Secondary outcomes: vasopressor requirement, incidence of hypotension (MAP reduction ≥20% from baseline) and hypertension (MAP increase ≥20% from baseline).

➢Chi square, t-test or Mann-Whitney U-test was used to compare differences between two groups.

Results

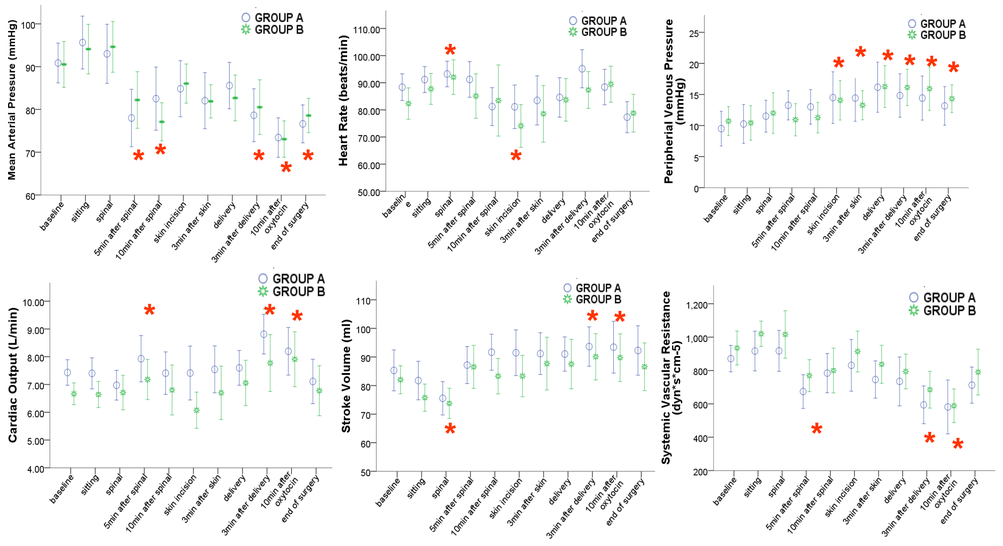

Figure 1. Hemodynamic changes at predefined time points during spinal anesthesia for cesarean delivery. * p<0.05 compared to the baseline value.

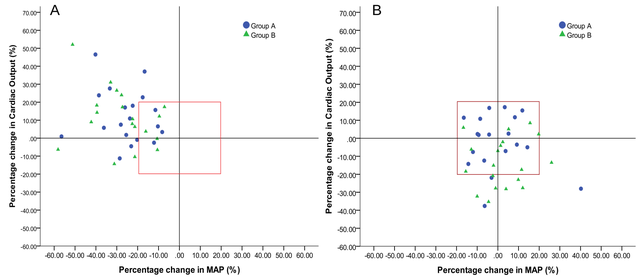

Figure 2. Cardiac output at the nadir and the peak of mean arterial blood pressure after spinal anesthesia. Data are presented as percentage changes from the baseline value. Percentage changes in cardiac output (CO) at the nadir (Panel A) and the peak (Panel B) of mean arterial pressure (MAP) after spinal anesthesia are plotted.

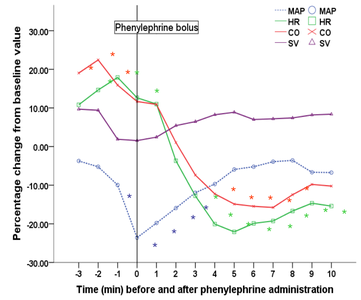

Figure 3. Hemodynamic changes before and after the administration of phenylephrine bolus. Percentage changes (%) in mean arterial pressure (MAP), cardiac output (CO), heart rate (HR) and stroke volume (SV) are shown in this figure with pooled data from group A and group B (n=15). * p<0.05 compared to baseline.

Table 1. Vasopressor requirement and maximum changes in cardiac output and mean arterial pressure before delivery

	Group A		Group B		Mean	95% CI		
	Mean	Std	Mean	Std	Difference	Lower	Upper	*p* value
Phenylephrine in total, mg	0.63 ± 0.33		0.55 ± 0.33		0.08	-0.13	0.30	0.44
Phenylephrine infused, mg	0.58 ± 0.3		0.44 ± 0.28		0.14	-0.05	0.33	0.15
Rescue phenylephrine, mg	0[0-0.08]		0.08[0-0.16]					0.06
Rescue bolus times, times	0[0-1]		1[0-2]					0.03*
Ephedrine, mg	0[0-0]		0[0-10]					0.30
Incidence of hypotension, n (%)	14(73.7%)		13 (68.4%)					0.72
Baseline MAP, mmHg	90.66±8.46		90.11±10.05		0.55	-5.56	6.66	0.86
Minimum MAP, mmHg	68.16±12.64		66.26±11.39		1.89	-6.02	9.81	0.63
Percentage change, %	-24.92±11.35		-26.13±11.88		1.21	-6.44	8.86	0.75
Time to the minimum MAP, min	8.68±4.23		9.53±3.55		-0.84	-3.41	1.73	0.51
Maximum MAP, mmHg	92.11±15.20		90.05±8.26		2.05	-6.00	10.10	0.61
Percentage change, %	1.61±13.77		0.68±11.17		0.94	-7.32	9.19	0.82
Time to the maximum MAP, min	12.11±3.74		15.21±4.12		-3.11	-5.69	-0.52	0.02*
Baseline CO, L/min	7.32±1.11		6.84±0.87		0.48	-0.17	1.14	0.14
Minimum CO, L/min	6.18±0.97		5.48±1.26		0.70	-0.04	1.44	0.06
Percentage change, %	-15.07±10.30		-20.03±12.91		4.96	-2.73	12.64	0.20
Time to the minimum CO, min	11.97±3.91		12.92±4.32		-0.95	-3.71	1.81	0.49
Maximum CO, L/min	9.22±1.74		8.67±1.78		0.54	-0.61	1.70	0.35
Percentage change, %	25.77±12.97		26.37±16.44		-0.60	-10.34	9.14	0.90
Time to the maximum CO, min	8.03±4.56		9.00±4.24		-0.97	-3.91	1.97	0.51

❖Thirty-eight patients were included in the final analysis. Four patients were excluded because of (1)extensive trembling interfering with data quality or (2)failed spinal anesthesia.

❖No difference in patients' demographics, baseline hemodynamic variables and anesthesia method.

❖Percentage changes in hemodynamic variables at the predefined time points were not different between the two groups.

➢Significant decrease in MAP and SVR and increase in CO occurred both immediately after spinal anesthesia and delivery.

❖There were no differences in the vasopressor dosage and maximum changes in MAP and CO in response to spinal anesthesia.

➢At the nadir of MAP after spinal anesthesia, MAP was decreased ≥20% from baseline in 71.1% of patients, while the corresponding CO was increased in 76.3% of patients and in 23.6% of the patients the increment of CO was ≥20% from baseline.

➢At the peak of MAP after spinal anesthesia, MAP was within ±20% from baseline in 94.7% of the patients, however, 26.3% of the patients showed reduction in corresponding CO of ≥20% from baseline.

❖ Effect of phenylephrine bolus on hemodynamics:

➢Mean percentage change in MAP was -23.6 ± 13.8 % from baseline at the time of phenylephrine first bolus.

➢Significant increase in HR and CO took place 2-3 min before the occurrence of hypotension.

➢CO and HR decreased significantly after phenylephrine bolus.

Discussion

❖ Using continuous CO monitoring, we were able to determine the CO, SV and SVR in a timely manner and obtain an instantaneous hemodynamic profile.

➢CO was increased immediately after spinal anesthesia initiation, which corresponded well with the reduction in SVR.

➢In the majority of cases, phenylephrine corrected hypotension, increased SVR, but caused significant CO reduction (-16%). Therefore, maintaining BP close to or in the normal range using phenylephrine but without knowing CO may result in significant reduction in O2 delivery during CD under spinal anesthesia. Since we often do not continuously monitor fetal heart rate during CD, fetal heart rate decelerations might occur more frequently than expected due to a reduction in O2 delivery. This observation implies continuous measurement of CO together with BP may provide a better, instantaneous hemodynamic profile to guide management of these patients.

❖In this study we also intended to determine if awareness of the instantaneous hemodynamic profile translates into better hemodynamic stability in term of CO and MAP. However, our results did not show a significant difference in patients' hemodynamic stability or vasopressor requirements between the two groups.

➢Since behavior change often requires constant and persistent re-enforcement, we believe that failure to improve patients' hemodynamic stability was probably due to incomplete incorporation of the real-time data and a change in practice. Further study is needed to test this notion.

References:
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