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Original Article

Maternal Heart Rate As A Predictor of Post-Spinal Hypotension in Parturients

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ABSTRACT

Background: Spinal Anaesthesia is the most preferred route of anaesthesia in parturients for caesarean section. Maternal hypotension after spinal induction is the most common complication which may result in adverse maternal and foetal outcomes. The decrease in systemic vascular resistance due to the blockade of pre-ganglionic sympathetic fibres causes maternal hypotension. Analysis of heart rate is the most economical, easiest non-invasive method of assessment of the autonomic nervous system.

Material and Methods: This is a prospective observational study carried out at NSCB medical college and hospital, Jabalpur from March 2019 to August 2020 on 225 pregnant women who underwent elective caesarean section under spinal anaesthesia. All patients were explained in detail about the anaesthetic procedure and written informed consent was obtained. All the patients were pre-loaded with lactated Ringer's solution (15ml/kg) 15 min prior to spinal anaesthesia. Basal HR was determined with pulse oximeter by taking average of five independent recordings, every minute in sequence. Immediately after subarachnoid block, blood pressure measurement was recorded and repeated every 3 min in first 30 min and cycled to 5 min till end of surgery. Patients developing more than 20% drop in themean arterial pressure (MAP) were treated with parenteral ephedrine 3 mg bolus. The foetuses were monitored immediately at 1 min and 5 mins immediately after birth and APGAR scores were calculated.

Results: Significant positive correlation of baseline maternal Heart Rate with post-spinal hypotension and vasopressor requirements was noted. Subjects with baseline HR ≥ 90 beats per minute had an 81% chance (Positive predictive value) of developing marked hypotension after SA. There was no significant difference in APGAR scores based on the degree of hypotension observed.

Conclusion: Baseline HR prior to hydration may be useful to predict post SA hypotension. Higher baseline HR, possibly reflecting a higher sympathetic tone, may be a useful parameter to predict postspinal hypotension.

Key Words: *Heart rate, Hypotension, Spinal anaesthesia, Parturients, Caesarean Section*

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INTRODUCTION

There has been drastic decline in mortality and morbidity related to anaesthesia for caesarean delivery in past decades to 1.7per million [1]. Maternal complication in general anaesthesia compared with neuraxial blockade has led to the increased employment of spinal and epidural anaesthesia for both elective and emergency caesarean section [2]. Spinal anaesthesia is the most preferred route of anaesthesia in parturients for caesarean section [3].

Maternal hypotension is the most common complication after the subarachnoid block resulting in adverse maternal and fetal outcomes [4, 5]. Hypotension during central neuraxial blockade is mainly a result of decreased systemic vascular resistance after blockade of preganglionic sympathetic fibres [6]. Sympathetic activity is exaggerated in pregnant women in comparision to non-pregnant women [7, 8]. Changes in the regulation of the Autonomic nervous system among the healthy pregnant patients explain the hemodynamic differences in response to subarachnoid block. Prospective assessment of the autonomic nervous system control might help in detecting patients at risk of severe hemodynamic impairment. Analysis of Heart Rate is among one of the non-invasive methods of assessing the activity of the Autonomic nervous system [9]. Anesthesia induced hypotension might possess severe adverse effects on both the mother and the child [10]. There has been no proven evidence for preventing the hypotension caused by regional anesthesia with pre-hydration with IV Crystalloids or IV Colloids [11] as well as prophylactic IM or IV vasopressors [12, 13, 14, 15 & 16].

The preoperative heart rate may differ between patients in relation to the severity of hypotension after subarachnoid block. Therefore preoperatively analysed heart rate may predict hypotension after spinal anesthesia [17]. This could

prove beneficial in the initiation of measures to prevent this drop in blood pressure. With this information in the backdrop, the study was conducted in the Department of Anaesthesiology and Critical care, Netaji Subash Chandra Bose Medical College, Jabalpur to determine the association of preoperative heart rate with post spinal hypotension in patients presenting for caesarean section.

MATERIALS AND METHODS

This is a prospective observational study conducted at NSCB Medical college, Jabalpur from March 2019 to August 2020. All pregnant women of the ASA grade II scheduled for elective caesarean section under spinal anaesthesia, after the obtainment of informed consent were included in the study. Patients undergoing emergency caesarean section, with significant cardiovascular diseases, renal, hepatic, thyroid disorders, with Diabetes Mellitus, Pre-eclampsia, Eclampsia, Uteroplacental insufficiencies, raised body temperature, severe anemia (≤ 7 gm%), &short stature (< 140 cm) were excluded. Total 225 pregnant women of ASA2 undergoing elective caesarean section participated in the study.

All patients were explained in detail about the anaesthetic procedure and written informed consent obtained. Before spinal anaesthesia, all the patients were pre-loaded with lactated Ringer‘s solution (15ml/kg) 15 min prior to spinal anaesthesia. No anticholinergics were given as pre‐ medication. Basal HR was determined with pulse oximeter by taking average of five independent recordings, every minute in sequence. Baseline blood pressure was recorded by taking average of five independent recordings, every minute in sequence with the help of non‐ invasive blood pressure monitor. Patient‘s peripheral oxygen saturation, surface temperature, and electrocardiogram were monitored. Basal values were recorded.

The patients having a HR of 90 beats per minute (bpm) or less were included in group 1, while those having HR of 91 bpm or more were included in group 2(during statistical analysis). The patients were placed in sitting position and dural puncture performed at L3‐ L4 inter space with the use of 23 G spinal needle. Hyperbaric bupivacaine (0.5%) 12 mg(2.4ml) injected intrathecally and patient made to lie down with wedge under right buttock. Blood pressure measurement was recorded immediately after subarachnoid block and repeated every 3 min in first 30 min and cycled to 5 min till end of surgery. Patients developing more than 20% drop in theirmean arterial pressure (MAP) were noted and treated with parenteral ephedrine 3 mg bolus. Ephedrine treatment was repeated as indicated up to maximum of 30 mg throughout the surgery. The amount of ephedrine administered within 30 min after spinal anaesthesia was used to calculate ephedrine requirements. The fetus was monitored immediately at 1 min and 5 mins immediately after birth and APGAR scores were calculated.

RESULTS AND DISCUSSION

Table 1: Patient parameter (AGE) and incidence of post-spinal hypotension

Variables	Hypotension (%)	Normotension (%)
Age Group		
19-20 years	10 (7.90)	7 (7.10)
21-25 years	65 (51.60)	64 (64.60)
26-30 years	48 (38.10)	27 (27.30)
31-35 years	3 (2.40)	1 (1.0)
Chi square (P value)	4.24 (0.239)	
Mean(SD)	24 (3)	24 (3)
t test (P value)	0.95 (0.342	

Table 2: Patient parameter (Gravida and Parity) and incidence of post-spinal hypotension

Variables	Hypotension (%)	Normotension (%)
Gravida		
1	43 (34.10)	29 (29.30)
2	65 (51.60)	58 (58.60)
3	14 (11.10)	11 (11.10)
4	4 (3.20)	1 (1.00)
Chi square (P value)	2.07 (0.582)	
Primi	43 (34.10)	29 (29.30)
Secundi	65 (51.60	58 (58.60)
Multi	18 (14.30)	12 (12.10)
Chi square (P value)	1.10 (0.593)	

Table 3: Incidence of post-spinal hypotension in relation to baseline maternal heart rate

Variables	Hypotension (%)	Normotension (%)
Heart Rate (Pre Op)		
<80	12 (9.50)	53 (53.50)
80-90	52 (41.30)	44 (44.40)
>90	62 (49.20)	2 (2.10)
Chi square (P value)	80.70 (<0.0001)	
Mean(SD)	89 (7)	79 (7)
t test (P value)	11.38 (<0.0001)	

Table 4: Patient parameter (BMI) and incidence of post-spinal hypotension

Variables	Hypotension (%)	Normotension (%)
BMI		
Underweight (<18.5)	0	0
Normal (18.5 – 24.9)	9 (7.14)	10 (10.10)
Overweight (25-29.9)	74 (58.73)	54 (54.55)
Obese (30-39.9)	43 (34.13)	35 (35.35)
Chi square (P value)	0.769 (0.681)	
Mean(SD)	29.25 (2.68)	28.85 (2.93)
t test (P value)	1.06 (0.290)	

Table 5: Mean and P value of various parameters in regard to post-spinal hypotension

Variables	Hypotension	Normotension	Statistics	P value
APGAR1 [Median (IQR); (Range)]	7 [7-7 (6-8)]	7 [6-7 (5-8)]	0.68	0.497
APGAR5 [Median (IQR); (Range)]	9 [9-9 (8-10)]	9 [8-9 (8-10)]	0.90	0.367
Heart Rate [Mean (SD)]	89 (7)	79 (7)	11.38	<0.0001
SBP [Mean (SD)]	123 (9)	121 (7)	1.27	0.207
DBP [Mean (SD)]	79 (6)	78 (6)	0.89	0.372
MAP [Mean (SD)]	94 (6)	93 (5)	1.55	0.121
AGE [Mean (SD)]	24 (3)	24 (3)	0.95	0.342
WEIGHT [Mean (SD)]	69 (6)	67 (6)	1.80	0.074
HEIGHT [Mean (SD)]	153.29 (2.67)	152.76 (3.10)	1.37	0.172
Gestational Age [Mean (SD)]	39.33 (0.59)	39.31 (0.49)	1.06	0.292
BMI [Mean (SD)]	29.25 (2.68)	28.85 (2.93)	1.06	0.290

Table 6: Logistic regression analysis to predict maternal hypotension

Variables	Odds Ratio (95% CI)	P value
Age	0.96 (0.87-1.05)	0.341
Weight	0.96 (0.92-1.00)	0.075
Height	1.07 (0.97-1.17)	0.172
BMI	1.05 (0.96-1.16)	0.289
Gestational Age	1.30 (0.80-2.13)	0.292
Heart Rate	0.80 (0.76-0.85)	<0.0001
SBP	0.98 (0.95-1.01)	0.207
DBP	0.98 (0.94-1.02)	0.371
APGAR1	0.85 (0.55-1.32)	0.470
APGAR5	0.80 (0.49-1.33)	0.401
MAP	0.96 (0.92-1.01)	0.123

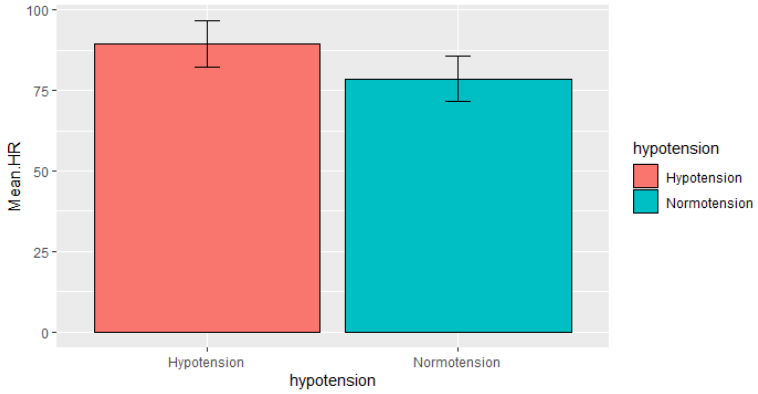

Figure 1: Incidence of post-spinal hypotension in regard to maternal basal heart rate (Box & Whisker plot)

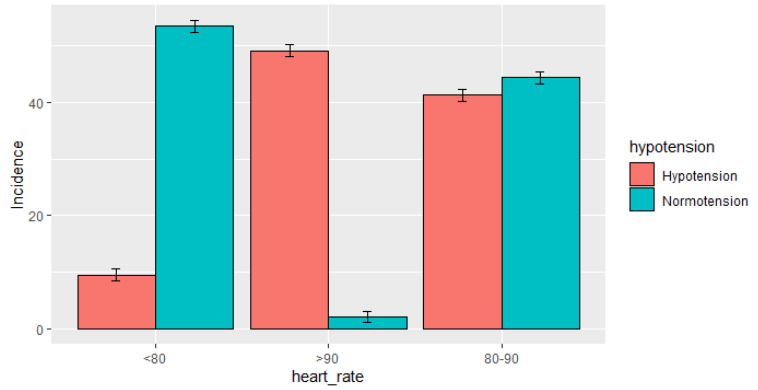

Figure 2: Incidence of post spinal hypotension is noticed higher in parturients with baseline heart rate 90/min

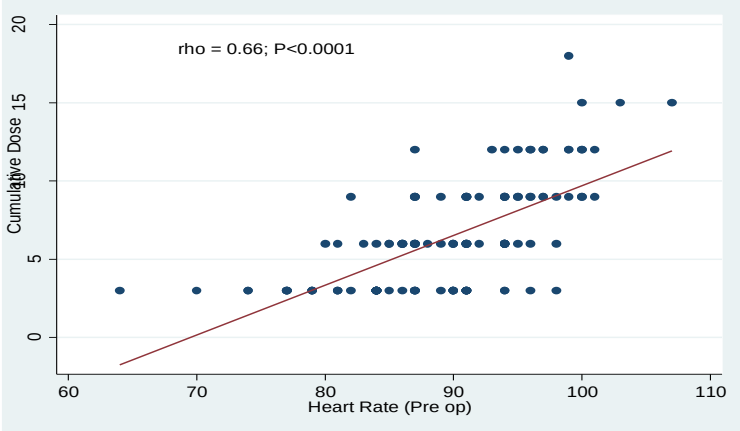

Figure 3: Spearman rank order correlation of maternal baseline heart rate and cumulative dose of vasopressors

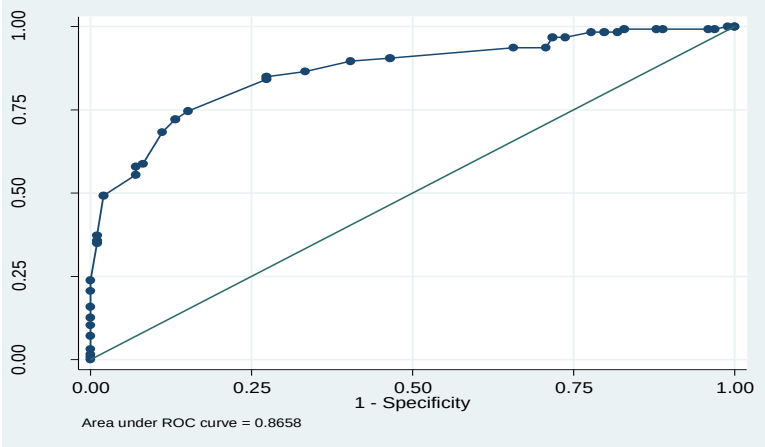

Figure 4: ROC CURVE

Spinal anaesthesia is produced by the injection of local anaesthetic, often together with an opioid adjunct, into the subarachnoid space, with the objective of blocking conduction in afferent sensory fibres that transmit pain impulses to the brain. However, conduction block from local anaesthetics is non-specific and preganglionic fibres to the sympathetic chain are also affected, resulting in sympathetic block and hypotension which can cause hypo-perfusion of the uterus and placenta. The extent to which the sympathetic chain is blocked is related to the degree of cephalad spread of local anaesthetic in the subarachnoid space [18]. In pregnant women, greater sensitivity to local anaesthetics results in higher blocks, and compounded by the effects of aortocaval compression, hypotension occurs with greater frequency and severity. There is also an increase in sympathetic versus parasympathetic activity [19] which predisposes to a greater degree of peripheral vasodilation. Epidural blockade produces a similar extent of sympathectomy, but there is a lower incidence and severity of hypotension since the rate of onset of sympathectomy is slower, allowing more time for cardiovascular compensation.

To understand how spinal anaesthesia affects the cardiovascular system, it is important to understand the basic principles of cardiovascular physiology.

Blood pressure (BP) is determined by the following equation:
Mean arterial pressure (MAP) = Cardiac output (CO) × Systemic vascular resistance (SVR)

Cardiac output CO is the volume of blood pumped by the heart per minute, and is equal to the product of the heart rate (HR) and stroke volume (SV), the latter of which is determined by preload, afterload and contractility. During spinal anaesthesia, hypotension occurs as a result of a decrease in SVR and/or CO. The overall haemodynamic effects can be divided into three main categories: Hypotension during spinal anaesthesia for caesarean section

The effects on preload

Starling's law of the heart states that the force of contraction of the cardiac muscle fibres is directly proportional to their initial resting length, or preload. Stretching sensitizes the myofibrils to calcium and increases the force of cardiac contraction. In the intact heart, preload is determined by the end-diastolic volume which is dependent on venous return. The sympathectomy that accompanies spinal anaesthesia results in venodilatation which causes pooling of blood peripherally and reduces venous return and preload. The decrease in preload reduces CO and thus contributes to hypotension. Clinically, left ventricular end diastolic volume cannot easily be measured, and preload is assessed by measuring central venous pressure (CVP) or the pulmonary artery wedge pressure.

The effects on afterload

Afterload is the resistance against which the left ventricle must contract and is determined mainly by the systemic vascular resistance (SVR). SVR is dependent largely on arteriolar vasomotor tone which is decreased by the sympathectomy caused by spinal anaesthesia. Vasodilatation and a decrease in SVR occurs which contributes to hypotension. Although vasodilatation may improve peripheral blood flow, it may also cause shunting which can result in regional tissue hypoxia. Initially, vasodilatation may lead to an increase in CO due to improvement in cardiac performance but excessive vasodilatation invariably leads to hypotension.

The effects on the heart

Direct effects on the heart include effects on HR and contractility, the pumping ability of the heart. The effect of the sympathetic nervous system on the heart is to increase both HR and contractility. These actions are opposed by parasympathetic innervation via the vagus nerve. Sympathetic block from high spinal anaesthesia can affect cardiac function and reflexes in several ways. An increase in baroreceptor sensitivity has been shown to occur in pregnancy, and HR can increase secondary to a reflex baroreceptor-mediated response to hypotension [20]. Conversely, HR may also decrease either directly from a decrease in cardiac sympathetic stimulation resulting from block of sympathetic outflow from the upper thoracic segments, or indirectly via the Bainbridge reflex.

CONCLUSION

In the present study entitled "Maternal heart rate as a predictor of post-spinal hypotension in parturients" there was a high statistically significant correlation ($p < 0.0001$) between preoperative baseline heart rate and hypotensive episodes in patients undergoing elective caesarean section after spinal anaesthesia.

There was no statistically significant difference in the APGAR scores of the babies born to mothers irrespective of the number of hypotensive episodes after induction of spinal anaesthesia (APGAR 1: $p = 0.497$; APGAR 5 : $p = 0.367$) and the preoperative heart rate.

Higher the preoperative baseline heart rate , the required dose of vasopressor bolus dose for hypotension increased. Other variables such as age, weight, height, gestational age, parity, BMI had no significant impact on the occurrence of post spinal hypotension.

Most women who undergo Caesarean section will receive spinal anaesthesia. Hypotension is the most common problem, and requires prompt treatment to avoid maternal symptoms and fetal acidosis. Rapid detection of hypotension will minimize the period of untreated hypotension, and non-invasive BP measurements cycled at 1- min intervals is recommended. Assessment of baseline heart rate is easy, non-invasive, less time consuming and can be the most economical way in identifying the patient groups at risk for post-spinal hypotension.

Ethics approval and consent to participate

The protocol of the study was approved by the ethical committee of NSCB Medical College, Jabalpur. The participants were included in the study only after informed written consent was obtained.

List of abbreviations

SV – stroke volume ; CO – cardiac output; SVR – systemic Vascular resistance; SBP – systolic Blood Pressure; DBP – Diastolic Blood Pressure ; HR – Heart rate ; APGAR –Appearance, Pulse, Grimace, Activity & Respiration; BMI –Body Mass Index; ASA – American Society of Anaesthesiologists.

Data Availability

Not available

Conflicts of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

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Authors' contributions

KP analyzed and interpreted the patient data. AT collected the data and was a major contributor in writing the manuscript. All authors read and approved the final manuscript.

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Not available.

Supplementary Materials

Not available

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