TO COMPARÉ THE HEMODYNAMIC PARAMETERS SUCH AS BLOOD PRESSURE, HEART RATE AND SPO₂ DURING SPINAL ANAESTHESIA.

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Abstract
The study was undertaken in the department of Anaesthesiology, Index Medical College, Hospital & Research Centre, Indore. The study included 90 patients (age 20-35 years) undergoing elective caesarean section under spinal Anaesthesia. Vital parameters were monitored and blood pressure & heart rate reading were taken 3 times at 2 minutes interval and lowest MAP and heart rate were taken as baseline for each group respectively. All the patients were preloaded with 500 ml of ringer lactate solution. Test drug was injected IM just after the induction of spinal anaesthesia. All the patients were observed and hemodynamic data recorded for 60 minutes after spinal anaesthesia. The patients were monitored for hypotension (decrease in MAP>25% of baseline MAP), bradycardia (heart rate<50 beats/minute), nausea, vomiting.

Keywords: Hemodynamic, SpO₂, Heart & Spinal Anaesthesia.

Introduction:
Spinal anaesthesia was initially given inadvertently by Corning in 1885 and first planned anaesthesia for surgery in man was performed by August Bier on 16th August 1898. It provides satisfactory anaesthesia for lower abdominal, urological and lower limb surgeries and it is often associated with a marked fall in blood pressure during and after anaesthesia due to various factors like diminished cardiac output consequent upon decreased venous return due to blockade of sympathetic nerves arising from T₁-L₂ leading to dilatation of both resistance and capacitance vessels and lack of propulsive movement on veins. Secondly paralysis of sympathetic nerve supply of heart and adrenal gland leads to subsequent catecholamine depletion and thirdly ischemia and hypoxia of vital centers leading to depression of circulatory system. It is desirable to record the blood pressure every 5-10 minutes but there is considerable difference of opinion as to the extent to which it should be allowed to fall before corrective measure are to be taken.

Continuous spinal anaesthesia (CSA) provides extension of blockage during surgery and versatile pain management during the postoperative period via an indwelling catheter allowed intermittent injection of local anaesthetic into the subarachnoid space. Better cardiovascular stability with a smaller dose of local anaesthetic and shorter surgery onset time were reported in CSA.

Material & Method
The study was undertaken in the department of Anaesthesiology, Index Medical College, Hospital & Research Centre, Indore from Oct 2017 to Sep 2018. The study included 90 patients (age 20-35 years) undergoing elective caesarean section under spinal Anaesthesia. Pre-Anaesthetic check-up was done in all the patients which included:

1. Elucidating history of diabetes, hypertension, asthma, tuberculosis, previous cardiovascular or central nervous system abnormalities, drug allergy, previous surgery, or any other significant history.
2. Examination including pulse, blood pressure, cardiovascular examination, respiratory system examination, spinal abnormalities, other systems.
3. Investigations including haemoglobin, complete blood counts, serum electrolytes, INR, blood sugar, serum urea, serum creatinine, chest X-ray, ECG as and when applicable.

Informed consent was obtained from each patient.

INCLUSION CRITERIA
1. Woman of age between 20-35 years
2. ASA grade I or II
3. Undergoing elective caesarean section.

**EXCLUSION CRITERIA**

1. Known hypertensive or those with a resting arterial pressure more than 130/90 mmHg.
2. Patient with hypovolemia or hypotension
3. Patients with diabetes, respiratory disease, cardiac disease, epilepsy.
4. Height less than 150 cm
5. Allergic to any drug to be used
6. Any other contraindication for spinal anaesthesia

**Results**

**Baseline MAP and pulse observations**

MAP (mean arterial pressure) = \( \frac{\text{SBP}+2\text{DBP}}{3} = \text{DBP}+\frac{1}{3}\text{PP} \)

Mean of baseline MAP and Pulse in all the groups

<table>
<thead>
<tr>
<th></th>
<th>Group C</th>
<th>Group E</th>
<th>Group P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline MAP</td>
<td>89.73±6.19</td>
<td>89.9±4.97</td>
<td>88.1±4.88</td>
</tr>
<tr>
<td>Baseline Pulse</td>
<td>92.8±10.06</td>
<td>94.13±11.83</td>
<td>92.1±12.88</td>
</tr>
</tbody>
</table>

**Table 2: Comparison of various groups with respect to baseline MAP**

<table>
<thead>
<tr>
<th></th>
<th>Group C &amp; E</th>
<th>Group C &amp; P</th>
<th>Group E &amp; P</th>
</tr>
</thead>
<tbody>
<tr>
<td>P value</td>
<td>0.9070</td>
<td>0.0848</td>
<td>0.1623</td>
</tr>
<tr>
<td>Significance</td>
<td>Not significant</td>
<td>Not significant</td>
<td>Not significant</td>
</tr>
</tbody>
</table>

**Discussion**

Ephedrine due to its predominantly β-agonist activity is expected to cause an increase in heart rate. On the other hand, phenylephrine, with predominant α-agonist action, causes a rise in the arterial blood pressure without any direct effect on the heart rate. This leads to activation of baroreceptor reflex and subsequent decrease in the heart rate indirectly. Bradycardia could also be caused by cardiac sympathetic denervation associated with high spinal block.

In our study only 2 patients in the phenylephrine group developed bradycardia for which IV glycopyrolate was given, rest of the patients were stable except transient tachycardia in few patients.

In the study by Kohki Nishikawa and associates (2002), bradycardia (heart rate< 50bpm) after IM administration of phenylephrine was not observed in any of the groups. Also none of the patients in any group developed bradycardia in the study done by Ayorinde BT et al (2001). In both of these studies phenylephrine and ephedrine were administered IM prophylactically.

Bradycardia was observed in various studies in patients receiving IV bolus or infusion of phenylephrine either prophylactically or therapeutically. (Dinesh Sahu et al 2003, Ngan Kee WD et al 2004)

**Conclusion**

Vital parameters were monitored and blood pressure & heart rate reading were taken 3 times at 2 minutes interval and lowest MAP and heart rate were taken as baseline for each group respectively. All the patients were preloaded with 500 ml of ringer lactate solution. Test drug was injected IM just after the induction of spinal anaesthesia. All the patients were observed and hemodynamic data recorded for 60 minutes after spinal anaesthesia.

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**References**