A STUDY OF PERIOPERATIVE HAEMODYNAMIC ASSESSMENT BY NICOM (NON INVASIVE CARDIAC OUTPUT MONITORING) IN PARTURIENT POSTED FOR CAESAREAN SECTION UNDER SUBARACHNOID BLOCK
Vaishali C. Shelgaonkar¹, Sandhya P. Manjrekar², Jaideep Sonowal³

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ABSTRACT: BACKGROUND: Pregnancy is a state of dramatic haemodynamic changes to accommodate the increasing needs of a pregnant body. These changes can be further complicated by preeclampsia which occurs in 6-12% of all pregnancies, showing varied haemodynamic profiles, making it difficult for the clinicians to plan a goal directed management or therapy plan. These patients posted for caesarean section under spinal anaesthesia management become more challenging. AIMS: To evaluate NICOM, based on bioreactance, as an aid to assess the haemodynamic changes in normal & pregnancy induced hypertensive (PIH) parturient during elective caesarean delivery under SAB. Also to assess the utility of NICOM, to determine haemodynamic variation, response to intravenous fluids or drugs & to compare fluctuations in cardiac output. SETTINGS & DESIGN: This prospective observational cohort study was carried out on 80 antenatal patients after dividing in Normotensive /group N or Hypertensive parturients/ group H undergoing elective caesarean section under spinal anaesthesia. METHODS: Perioperatively haemodynamic variables including systolic (SBP), diastolic (DPB), mean arterial (MAP) pressures, heart rate (HR), stroke volume (SV), total peripheral resistance (TPR) & cardiac output (CO) were monitored continuously by NICOM. STATISTICAL ANALYSIS: All data were analyzed by specific statistical methods (Chi Square, t-Test, Z-test, Fisher's exact test and Yates' correction) where ever applicable. RESULTS: There were significant fluctuations noted in SBP, HR and CO after SAB in spite of preloading and in post-delivery period in both the groups. Pre eclamptic women showed higher SBP, DBP, MAP, CO & TPR compared to healthy pregnant women. DISCUSSION: There were significant changes in SV, CO, TPR that occurred under SAB in both normal and PIH parturients, more in PIH group. These diverse haemodynamic profiles analyzed in details by NICOM. The clinical effect of fluid challenge & vasopressors for treatment of hypotension is evident. CONCLUSIONS: NICOM is a promising monitoring system for parturients undergoing operative deliveries, allowing precise haemodynamic assessment & provide goal-directed therapy in women with preeclampsia. KEYWORDS: NICOM, PIH, Bioreactance, Cardiac Output, TPR.

INTRODUCTION: Pregnancy is a state of dramatic haemodynamic changes such as SBP (systolic blood pressure), DBP (diastolic blood pressure), Heart rate (HR), Cardiac output (CO), Stroke volume (SV) and systemic vascular resistance (SVR), to accommodate the increasing needs of a pregnant body. The circulatory system works harder to provide blood flow to the
placenta and the growing foetus. These changes can be further complicated by Preeclampsia (pregnancy induced hypertension) which occurs 6-12% of all pregnancies.\textsuperscript{1,2} Preeclampsia has always been found to show varied haemodynamic profiles, making it difficult for the clinicians to plan a goal directed management or therapy plan.\textsuperscript{3} These patients posted for caesarean section under spinal anaesthesia management become more challenging due to sympathetic blockade and hypotension.\textsuperscript{4}

Cardiac output (CO) is a fundamental measure for the assessment of cardiac performance and is applied widely to detect the presence of cardiovascular disease and monitor its progression, as well as to monitor patients in challenging hemodynamic circumstances and to optimize therapy.\textsuperscript{5-7} Thermodilution technique though a gold standard due to its invasive, time-consuming, relatively expensive, requires the attention of a trained physician and associated with a degree of risk and complications.\textsuperscript{8} With such evident flaws, numerous less invasive methods have been proposed to measure CO but they are also beset with limitations, specifically regarding their reliability and reproducibility.\textsuperscript{9-13}

Thoracic bioimpedence technique was up till now considered as easier and simpler way of monitoring CO noninvasively, it has been used in many obstetric settings.\textsuperscript{10,11,14-20} But due to its limitations, most promising technology of bioreactance, which measures the phase shift in voltage across the thorax is being popular.\textsuperscript{21}

\textbf{Figure 1:} The NICOM system is equipped with 4 sensor pads. Each pad contains an outer, transmitting sensor and an inner one for receiving. The sensors are applied around the heart on the chest or back.

This study assesses the haemodynamic profiles of normal and pre-eclamptic parturients during caesarean section under regional anaesthesia using NICOM.\textsuperscript{22,23}
MATERIAL AND METHODS: This prospective observational cohort study was carried out on 80 antenatal patients having ASA grade I or II, age in the range of 18 to 40 years, Height between 150-170 cm undergoing elective caesarean section. Patients were divided either in Normotensive/ group N (Parturients in 3rd trimester without any history of hypertension) or Hypertensive parturients/ group H (PIH diagnosed in 3rd trimester or earlier, not yet treated, or treated) undergoing elective caesarean section under spinal anaesthesia during a period of November 2012 to October 2013.

Any parturients having contraindication to spinal / epidural anaesthesia, ASA grade III or IV, cardiovascular diseases, Liver, Respiratory, Kidney, Endocrine diseases complicating pregnancy were excluded from the study. Preoperatively a detailed history of patients taken, informed consent and minimum necessary investigations obtained. Demographic data and baseline haemodynamic parameters were noted.

In operating room, the patient was placed supine and slightly tilted to the left to avoid aortocaval compression. Haemodynamic information gathered by NICOM. Other multipara monitoring devices attached. Wide bored IV access was obtained & premedication given in the form of Inj. Ranitidine, Inj. Ondansetron and Inj. Metoclopramide as per weight. All Patients were preloaded with Lactated Ringer's solution 10ml/kg over 10 min, before subarachnoid block and haemodynamic parameter are recorded.

SAB achieved using 0.5% hyperbaric bupivacaine as per patient’s height (2/2.2ml). Haemodynamic Parameters Collected Were i) By NICOM SV, CO, HR, Stroke Volume Variation (SVV) Non-invasive Blood Pressure (NIBP), TPR, Stroke Volume Index (SVI), Cardiac Index (CI) and Total Peripheral Resistance Index (TPRI) ii) By Multipara monitor O2 Saturation (SPO2), ECG, Pulse rate, Non-invasive Blood Pressure (NIBP). All these were recorded preoperatively and intraoperatively at various time intervals.

Hypotension if occurred intra-operatively was treated with a rapid infusion of crystalloids and if persisting a bolus of inj. Ephedrine 6mg IV and if required repeated. Bradycardia was treated with Inj. Atropine sulphate 0.6 mg i.v bolus. Bradypnoea (respiratory rate less than 10/min) was treated with oxygen supplementation and assisted ventilation.

ETHICS: This prospective observational cohort study was conducted in Department of Anaesthesiology at tertiary hospital in Maharashtra, after approval from the hospital ethics committee.

STATISTICS: All data were analyzed by specific statistical methods (Chi Square, t-Test, Z-test, Fisher's exact test and Yates' correction) applicable to the various sets of data. Power analysis was also done for the exact differences between the groups for all variables. P value: NS, p>0.05 = not significant, *p<0.05= significant, **p <0.01= highly significant, ***p<0.001= very highly significant, ****p<0.0001= extremely significant

OBSERVATIONS AND RESULTS: The demographic characters of the patients in the study were comparable except in age and weight.
<table>
<thead>
<tr>
<th>GROUP</th>
<th>Mean Age (Years)</th>
<th>Mean Weight (Kgs)</th>
<th>Mean Height (Cms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (n=40)</td>
<td>25.32 ± 3.68*</td>
<td>58.17 ± 9.70*</td>
<td>155.175 ± 5.68 NS</td>
</tr>
<tr>
<td>H (n=40)</td>
<td>27.25 ± 3.92*</td>
<td>69.2 ± 11.16*</td>
<td>156.725 ± 4.23 NS</td>
</tr>
</tbody>
</table>

Table 1: Demographic Data

Graph 1: Mean Heart Rate (beats/ min) at various time intervals

Graph 2: Variation in Mean SBP (mmHg) at various
Graph 3: variation in Mean DBP (mmHg) at various time intervals

Graph 4: variation in Mean MAP at various time intervals
Graph 5: variation In Mean CO (l/min) at various time

Graph 6: Variation in Mean SV (ml/beat) at various time
Table 2: Percentage of variation in cardiac output in group N and group H

<table>
<thead>
<tr>
<th>% of Decrease in CO</th>
<th>Events</th>
<th>No. of Patients in GROUP N</th>
<th>No. of Patients in GROUP H</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 10%</td>
<td>After Preloading</td>
<td>18</td>
<td>3</td>
<td>***</td>
</tr>
<tr>
<td></td>
<td>After SAB</td>
<td>13</td>
<td>14</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>At 10 Min</td>
<td>20</td>
<td>4</td>
<td>***</td>
</tr>
<tr>
<td>&lt;20%</td>
<td>After Preloading</td>
<td>12</td>
<td>3</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>After SAB</td>
<td>6</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>At 10 Min</td>
<td>11</td>
<td>2</td>
<td>*</td>
</tr>
<tr>
<td>&lt;30%</td>
<td>After Preloading</td>
<td>7</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>After SAB</td>
<td>2</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>At 10 Min</td>
<td>9</td>
<td>1</td>
<td>*</td>
</tr>
<tr>
<td>≥10%</td>
<td>After Preloading</td>
<td>22</td>
<td>37</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>After SAB</td>
<td>27</td>
<td>26</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>At 10 Min</td>
<td>20</td>
<td>36</td>
<td>*</td>
</tr>
</tbody>
</table>

Graph 7: Variation in Mean TPR (dynes. sec/cm$^5$) at various time
<table>
<thead>
<tr>
<th>Decrease in MAP</th>
<th>GROUP N n= 40</th>
<th>GROUP H n=40</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;10 %</td>
<td>39</td>
<td>39</td>
</tr>
<tr>
<td>&lt;20%</td>
<td>25</td>
<td>17</td>
</tr>
<tr>
<td>&lt;30%</td>
<td>7</td>
<td>3</td>
</tr>
</tbody>
</table>

Ephedrine use (no. of patients)  
20/40  
8/40

Mean number of Ephedrine bolus dose, Each bolus = 6 mg  
1.3 ±.47  
1 ± 0

Table 3: patients having hypotension during perioperative period with mean numbers of bolus doses of ephedrine used in both the groups

<table>
<thead>
<tr>
<th>TPR (dynes. sec/cm$^5$)</th>
<th>Events</th>
<th>Number of patients in Group N</th>
<th>Group H</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;800</td>
<td>After preloading</td>
<td>4</td>
<td>0</td>
<td>***</td>
</tr>
<tr>
<td></td>
<td>After SAB</td>
<td>7</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>At 10 min</td>
<td>5</td>
<td>5</td>
<td>***</td>
</tr>
<tr>
<td>800-1200</td>
<td>After preloading</td>
<td>26</td>
<td>11</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>After SAB</td>
<td>25</td>
<td>21</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>At 10 min</td>
<td>18</td>
<td>22</td>
<td>*</td>
</tr>
<tr>
<td>&gt;1200</td>
<td>After preloading</td>
<td>20</td>
<td>29</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>After SAB</td>
<td>8</td>
<td>19</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>At 10 min</td>
<td>17</td>
<td>13</td>
<td>*</td>
</tr>
</tbody>
</table>

Table 4: Variation of TPR (dynes. sec/cm$^5$) in group N and group H

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Preop (Mean ±SD) n=40</th>
<th>After Preload (Mean ±SD) n=40</th>
<th>After SAB (Mean ±SD) n=40</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group N</td>
<td>91±11.04</td>
<td>88.47 ± 12.95</td>
<td>93.92 ± 10.84</td>
</tr>
<tr>
<td>Group H</td>
<td>97.5 ± 12.66</td>
<td>103.15 ± 18.32</td>
<td>106.25 ± 16.34</td>
</tr>
<tr>
<td></td>
<td>(*,p&lt;0.05)</td>
<td>(****,p&lt;0.0001)</td>
<td>(****,p&lt;0.0001)</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group N</td>
<td>6.38 ± 1.10</td>
<td>5.92 ± 1.11</td>
<td>6.27 ± 1.63</td>
</tr>
<tr>
<td>Group H</td>
<td>6.12 ± 0.79</td>
<td>6.29 ± 0.94</td>
<td>6.13 ± 1.11</td>
</tr>
<tr>
<td></td>
<td>(NS,p&gt;0.05)</td>
<td>(NS,p&gt;0.05)</td>
<td>(NS,p&gt;0.05)</td>
</tr>
<tr>
<td>MAP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group N</td>
<td>91.62 ± 6.33</td>
<td>87.67 ± 10.99</td>
<td>76.2 ± 9.90</td>
</tr>
<tr>
<td>Group H</td>
<td>111.32 ± 9.18</td>
<td>103.45 ± 7.67</td>
<td>98.15 ± 9.77</td>
</tr>
<tr>
<td></td>
<td>(****,p&lt;0.0001)</td>
<td>(****,p&lt;0.0001)</td>
<td>(****,p&lt;0.0001)</td>
</tr>
</tbody>
</table>
**DISCUSSION:** Despite a plethora of developments over recent decades, there still exists a clear need for highly accurate measures of CO that can be applied in various clinical settings in a cost-effective and scalable fashion. There is limited knowledge with respect to parameters of cardiac function in pregnancy and even less in the presence of pregnancy complications such as preeclampsia. Preeclampsia is a multisystem disease unique to human pregnancy characterized by hypertension and organ system derangement.

The disease is responsible for considerable morbidity and mortality complicating 5 - 8% of pregnancies and remains in the top three causes of maternal morbidity and mortality globally. These morbidity and mortality implications are most marked in poor, underprivileged, remote and rural communities and indigenous populations. Introduction of NICOM devices has enabled a better understanding of haemodynamics changes occurring during caesarean delivery. Rapid changes in CO and TPR have been demonstrated immediately after SAB and post-delivery.

Anne Doherty et al (2011) concluded that noninvasive monitoring based on bioreactance is able to give continuous haemodynamic data with clear and consistent signals and identified diverse haemodynamic profile in pregnant women at term. Nirav Y. Raval et al in their multicenter study that NICOM system for noninvasive CO measurement has acceptable accuracy in challenging clinical environments when compared to thermodilution TD thermodilution. Pierre Squara et al commented that the ability to detect significant CO changes were equivalent and acceptable for both technologies of NICOM & PiCCO (TD & PC).

Natesan Manimekalai et al mentioned NICOM was shown to be an easy to use monitor during caesarean sections provided valuable additional haemodynamic information during surgery that could be used in clinical management decisions and advocated routine use of NICOM in all caesarean section patients, especially in high risk obstetric patients where early intervention for developing hypotension is critical. Mrinalini Balki et al used in parturient with severe Aortic stenosis. They concluded NICOM may improve understanding of the peripartum changes in women with heart disease.

Incidence of Preeclampsia has increased in both the youngest and the oldest women of reproductive age which was evident from our study (ref table no. 1). Hansen et al (1986) reviewed in 18 studies and concluded that the risk of PIH increased with maternal age. However, studies separating severe preeclampsia from gestational hypertension still suggest that advancing maternal age has an independent effect thus higher age in PIH group in our study correlates with findings of other studies.
The difference of weight amongst two groups is statistically highly significant (p<0.001). Ferha Saeed et al (2011) mentioned that High BMI in pregnant women serves as a significant risk factor for developing hypertension in pregnancy. O'Brien et al found that the risk of preeclampsia doubled for each 5-7 unit increase in pre pregnancy body mass index (BMI). Getahun et al, 2007 stressed that there is strong relationship observed, the association between increasing changes in BMI and risk of PIH may support the theory that obesity-mediated inflammatory changes may play a role in the pathogenesis of PIH. Height was comparable in both the groups.

**Figure 2**: Pie diagram showing number of preeclamptic patients on treatment

Keerath K et al (2012) supported for spinal anaesthesia as the anaesthetic of choice in patients with severe preeclampsia, provided there is no contraindication. J. Karinen et al found reasonably stable maternal and utero-placental haemodynamic values in preeclamtic parturients undergoing caesarean section during spinal anaesthesia after prophylactic preloading with crystalloid. In this study higher heart rate was evident in group H (Mean ± SD=100.23 ± 3.19) as compared to group N (Mean ± SD=88.37 ± 6.03). Heart rate change was highly significant after preloading and SAB when compared with hypertensive parturients (p<0.001).

There was no significant change of heart rate in healthy parturients during perioperative period, significant fluctuation seen only after 10 min and 60min after SAB. In hypertensive parturients significant heart rate variation seen after preloading, at 10 min after SAB when compared with preoperative value (ref. graph no.1). Tihtonen K etal (2005) in his study mentioned that, sudden and significant haemodynamic changes take place at the moment of delivery. Intact physiological cardiovascular compensation mechanisms are needed to adapt to these challenges.

The difference between SBP of two groups was statistically highly significant. Also we have observed recovery in mean DBP to baseline value was delayed in group N while it was earlier in group H. The overall difference between two groups being statistically highly significant at various time intervals. It is observed from (ref. table no.3) that peri operatively <10% decrease in MAP equal in both the groups, but <20% and <30% decrease in MAP was more in
group N as compared to group H, and also frequency of use bolus doses in group N (1.3 ± 0.47) is more as compared to the group H (1 ± 0), so also total number of patients requiring Inj. Ephedrine was higher in Group N (20/40) as compared to in Group H (8/40).

In our study Systolic Blood Pressure is significantly higher in group H as compared to group N (ref. Graph no.2 & 3). Perioperative variation in group N noted after preloading, after SAB, at 5min, 10 min, 15 min, and 20 min when compared with preoperative value. Highly significant changes observed after SAB. In hypertensive group there was decrease in SBP after preloading, after SAB, and use of inj. ephedrine was less in group H then group N during post spinal period. The initial fall in SBP can be explained by the fact that vena cava is affected by the gravid uterus. The fall in SBP depends upon how effective is left uterine shift.

Aortocaval compression can reduce cardiac output and impair placental blood flow, so it remains rational to use lateral tilt during anaesthesia. In preeclampsia vascular epithelium is damaged by a process involving placental-derived proteins, leading to an imbalance between pro- and anti-angiogenic growth factors, which results in persistent vasoconstriction. In contrast, the normal pregnant patient is very sensitive to spinal anaesthesia, because of an altered balance of vascular tone. Responses to endogenous pressor, particularly angiotensin II, are reduced.

This is caused by an endothelium dependent alteration of vascular smooth muscle function. Additionally, there is increased synthesis of vasodilator prostaglandins and nitric oxide. These effects increase dependence on sympathetic vascular tone in normal pregnancy. The caval compression induced hypotension can be prevented to some extent after spinal block by preloading and was proposed to compensate for the venous blood said to be trapped in the legs, secondly, the tilt manoeuvre was advocated to reduce caval occlusion. Both these manoeuvres done in our study.

There is a wide variation in CO alterations when the spinal block is being established. Using a dye dilution technique, Ueland et al (1968) demonstrated a 35% decrease in CO when a spinal anaesthetic was performed (Tetracaine 7–10 mg with epinephrine 200 mcg) without prehydration, and the pregnant patient placed in the supine position. By contrast, utilizing the beat-to-beat CO measurements provided by bioimpedance, Tihonen et al, (2005) observed an immediate 11% increase in CO when 10 ml/kg colloid prehydration was given, hyperbaric bupivacaine 12–13.5 mg was administered, the patient was positioned in left uterine displacement, and a mean ephedrine dose of 0.53 mg/kg was provided.

Other factors that affect CO include, but are not limited to, prehydration, maternal positioning during and after the block, height and density of the block, presence of hypertensive disorders of pregnancy, gestational age, the number of gestations, the presence of labour, the prophylactic or therapeutic use of vasoactive substances, and the complex system of vascular tone and reactivity unique to each individual woman. One study (Robson S.C. et al,1992) demonstrated that the administration of 1 litre of lactated Ringers (LR) solution 15 min prior to neuroaxial anesthesia increased CO by 20%. In another study (Robson S. et al,1989) using LR solution in a mean volume of 805 ml as prehydration, observed a 10% increase in CO (from 7.01 to 7.70 l/min) with a relatively stable HR (83–82 bpm) but a significant increase in SV from 84 to 95 ml. Summarizing these studies, an increase in CO is observed with prehydration; however, the relative contributions of HR and SV
Cardiac output itself is controlled by heart rate and stroke volume changes (CO=HR x SV). In the present study, there was a significant decrease of SV after spinal anesthesia in group H (ref graph no.5 & 6).

However, this effect was likely physiologically compensated by an increase in HR. As from our previous observation we noted that in group H there was higher heart rate as compared to group N. Also we noted in hypertensive group preoperative CO lower and higher TPR as compared to the normotensive group. Thus there was low SV in hypertensive as compared to normal. But After SAB due to sympathetic blockade there is decreasing TPR for which gradual increase in CO and SV observed. Because CO is the product of HR and SV, it is important to note that as HR decreases, CO may also decrease, depending on whether alterations in SV can compensate for the change. This may be especially relevant during the onset of neuroaxial anesthesia because venodilation results in decreased preload and an inability to increase SV to compensate for a decreased HR.

Peri-operatively Mean TPR higher in group H then group N and which is statistically significant (ref. graph no.7). In group H Mean TPR was maintained after preloading and in post spinal period but significant decrease noted at 10 and 15 min after SAB when compared with preoperative value but values are maintained in normal range. This can be explained with that in preeclamptic parturients due to release vasoactive substances there is increase peripheral resistance. In group N there was significant decrease of TPR noted 20 min after SAB. Overall TPR is maintained in both the groups. In fact there was significant rise (1200-1400ml) and even >1400 ml after preloading in Group H, suggesting overall vasoconstriction state in PIH patients (ref. table no.4).

Judith U. et al in 1997 in their serial study on 14 normal pregnant showed that arterial compliance increases approximately 30% in the first trimester, and remains increased thereafter. This increase is accompanied temporally by the expected decrease in SVR. Such an increase in vessel distensibility might be secondary to vascular remodelling changes or reduced smooth muscle tone, documented to occur very early in pregnancy. It is plausible that increased smooth muscle tone in preeclampsia mainly is responsible for our results, which are comparable those of Elvan-Taspınar et al.

However, data from parturients undergoing spinal anesthesia for caesarean Delivery have demonstrated a decrease (from baseline) in SVR in the range of 26–31%, begging the question of whether arterial vasodilation plays a greater role than previously anticipated. Finally, more recent studies specifically looking at the arterial circulation have led to believe that "in the fluid replete parturient undergoing elective caesarean delivery, moderate spinal hypotension (20% decreases from baseline) primarily reflects decreased systemic vascular resistance".

To present in nutshell from five important haemodynamic parameters observed in two study groups of normotensive and hypertensive parturients at three important variables times are concise (ref. table no. 5). Heart rate is an only parameters in which fluctuation are seen. MAP also showed a higher preoperative value in group H. TPR in hypertensive parturients is well maintained, and higher than normotensive one, proving the pathophysiological change of generalized vasoconstriction in them. CO and SV were maintained in physiological limits in both the groups.
This being an observational study, without any intervention or comparison with other invasive or noninvasive technique of haemodynamic monitoring, we have just co-related the observed findings in normotensive and hypertensive parturients. Transthoracic bioreactance technique is newer, simple to use and provided a very clear and consistent monitoring. It helped us to identify distinct haemodynamic profiles which are considered or compared to previous studies. It also helped to demonstrate realistically the option of stabilizing intraoperative haemodynamics in caesarean deliveries by addressing the exact mechanism of decompensation.

CONCLUSION: Non-invasive cardiac output monitor (NICOM) based on bioreactance principle is a comprehensive operator independent, continuous bedside monitoring device. It was possible to obtain continuous haemodynamic data with clear and consistent signals in our study. Though the study has certain limitations like the non-invasive technique is not been compared with standard, thermo dilution, or invasive techniques, but on the basis of supportive studies proving their comparable efficiency, our findings were in accordance to them.

NICOM may offer a valuable opportunity to make early diagnoses and provide goal directed therapy in women at term, with preeclampsia and other medical conditions affecting pregnancy, where invasive monitoring may not be feasible.

BIBLIOGRAPHY:


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