Original Article

Assessment of role of inferior vena cava collapsibility index and variations in carotid artery peak systolic velocity in prediction of post-spinal anaesthesia hypotension in spontaneously breathing patients: An observational study

Address for correspondence:
Dr. Rahul K. Anand,
Room No 5011, 5th Floor,
Teaching Block, All India
Institute of Medical Sciences,
New Delhi – 110 029, India.
E-mail: drrahulkanand@gmail.com

Submitted: 05-Sep-2021
Revised: 11-Dec-2021
Accepted: 06-Feb-2022
Published: 24-Feb-2022

Access this article online

Website: www.ijaweb.org

DOI: 10.4103/ija.ija_828_21

Quick response code

Sumit R. Chowdhury, Dalim K. Baidya, Souvik Maitra, Akhil K. Singh, Vimi Rewari, Rahul K. Anand

Department of Anaesthesiology, Pain Medicine and Critical Care, All India Institute of Medical Sciences, New Delhi, India

ABSTRACT

Background and Aims: Post-spinal anaesthesia hypotension (PSH) is common and can lead to significant morbidity and mortality. The inferior vena cava collapsibility index (IVCCI) and carotid artery peak systolic velocity variations (CAPVV) are two widely used parameters for assessing the volume status of critically ill patients which have also been investigated as predictors of PSH and hypotension after induction of general anaesthesia. In this study, we evaluated the diagnostic accuracy of IVCCI and CAPVV as predictors of PSH. **Methods:** A total of 50 patients aged between 18 and 65 years undergoing elective lower abdominal surgeries under spinal anaesthesia were included. The IVCCI and CAPVV were measured using ultrasound pre-operatively. After administering spinal anaesthesia, haemodynamic data were collected till 15 min. Our primary objective was to evaluate the role of IVCCI and CAPVV to predict PSH. The secondary objectives were to compare the predictive efficacy of these two parameters and to detect other parameters for predicting PSH. We constructed the receiver operator characteristic (ROC) curves for IVCCI and CAPVV and obtained the best cut-off values. **Results:** The PSH occurred in 34% of the patients. IVCCI >21.15 could predict PSH with 58.8% sensitivity and 69.7% specificity. CAPVV >18.33 predicted PSH with 70.6% sensitivity and 54.6% specificity and IVC max/IVCCI >60 could predict PSH with 58.8% sensitivity and 54.5% specificity. A composite model comprising IVCmax (maximum IVC diameter), CAPVV, and baseline mean blood pressure was able to predict PSH. **Conclusion:** Both IVCCI and CAPVV have poor diagnostic accuracy in predicting PSH in adult patients undergoing elective infra-umbilical surgery.

Key words: Arterial hypotension, cardiovascular monitoring, carotid ultrasound, corrected flow time, fluid responsiveness, haemodynamic monitoring, inferior vena cava, intrathecal anaesthesia, intravascular volume, spinal anaesthesia

INTRODUCTION

Post-spinal anaesthesia hypotension (PSH) is one of the most common side effects of spinal anaesthesia. Hypotension occurs due to decreased systemic vascular resistance (SVR) because of sympatholysis and decreased cardiac output (CO) because of reduced venous return. PSH may be associated with

How to cite this article: Chowdhury SR, Baidya DK, Maitra S, Singh AK, Rewari V, Anand RK. Assessment of role of inferior vena cava collapsibility index and variations in carotid artery peak systolic velocity in prediction of post-spinal anaesthesia hypotension in spontaneously breathing patients: An observational study. Indian J Anaesth 2022;66:100-6.

nausea, vomiting, syncope, dizziness, aspiration, cardiac arrhythmias, cardiac arrest, and even death. Intraoperative hypotension has been found to correlate with post-operative mortality.[1,2] Therefore, predicting PSH is particularly important so that preventive measures or timely interventions can be taken to avoid untoward events.

Intravenous (IV) fluid loading is a relatively older approach to prevent PSH. In approximately 50% of the haemodynamically unstable patients, fluid boluses fail to improve the cardiac volume satisfactorily.[3] Empirical fluid loading is of little or no value in a fluid non-responsive patient and may even worsen the patient outcome. Due to the lack of any concrete tool to predict PSH, differentiating the cohort of patients requiring fluid boluses versus vasopressors remains a dilemma. There are many static and dynamic indices of fluid responsiveness with variable performance in different settings.[4] Considerable controversy exists regarding the efficiency of traditional methods of predicting volume status as predictors of PSH. Still, there has been a continuous search for an easy, non-invasive, and reliable predictor of PSH. Various non-invasive modalities, that is, transthoracic echocardiography (TTE), transthoracic bioimpedance, passive leg raising test (PLRT) etc. have been used to predict PSH in different sub-groups including the obstetric patients, revealing varied results.[5] However, TTE is time-consuming and needs significant operator efficiency; bioimpedance derived CO measurement is costly and not readily available.

In contrast, ultrasonographic assessment of the inferior vena cava collapsibility index (IVCCI) and carotid artery peak systolic velocity variation (CAPVV) is easy to perform, non-invasive, time-efficient, and readily available as well. The ultrasonographic measurement of IVCCI and CAPVV as predictors of fluid responsiveness has been extensively studied in different patient populations, predominantly in the critically ill patients. Pre-operative IVCCI is a reliable predictor of hypotension after induction of general anaesthesia with high specificity.[6] In a randomised trial, fluid therapy guided by IVC parameters before spinal anaesthesia was found to have a significant reduction of PSH.[7] However, no study so far has evaluated the role of CAPVV in predicting PSH. We hypothesised that patients with increased respiratory variations in IVC diameter and carotid artery flow velocity would have more chances of developing PSH. Our primary objective was to evaluate the role of IVCCI and CAPVV in predicting PSH. The secondary objectives were to compare the predictive efficacy of these two parameters and to detect other parameters for predicting PSH.

METHODS

After obtaining the ethics committee approval (IECPG-477/29.11.2017) and registration from the Clinical Trials Registry-India (CTRI/2018/03/012485), 58 patients aged between 18 and 65 years, of American Society of Anesthesiologists (ASA) physical status I and II undergoing elective lower abdominal surgeries under spinal anaesthesia were recruited. This was a prospective study which took place over a duration of one year in a tertiary care hospital in India. We excluded the patients who were hypertensive or had any cardiac disease, in whom a good-quality ultrasound image could not be obtained and pregnant females. Informed and written consent was obtained from all patients before recruiting them in the study.

We ordered standard fasting (6 h for solids and 2 h for clear fluids) in all the patients. After shifting the patient into the operation theatre, the IVCCI and CAPVV were measured by a single investigator. For IVCCI measurement, the patients were laid supine on a flatbed and were asked to breathe without deep inhalation and at a rate of 12–15 breaths/min. Using a 2–5 MHz curvilinear ultrasound probe (device—S-Nerve US system; Fujifilm SonoSite, Inc, Bothell, WA), IVC was identified by tracing it upwards till the right atrium. The IVC diameter was seen 1 cm distal to the point where the hepatic veins join IVC.[6] By selecting the M-mode, the maximum and minimum diameters (IVC max and IVC min) of IVC were measured and the IVCCI was calculated using the following formula: IVCCI = ([IVC max-IVC min]/IVC max) × 100. The results were expressed in percentages.

For the CAPV measurement, standing at the head end of the patient and turning the patient's neck to the left, a 6–13 MHz linear probe of the same machine was used to identify the right common carotid artery (CCA). In the long-axis view, pulse wave Doppler was used after placing the sample volume at the centre of the arterial lumen, 2 cm proximal to the bifurcation of the CCA. Doppler beams were adjusted to maintain the angle <60° for obtaining the optimal signal. The maximum (Peak systolic velocity [PV]

max) and minimum (PV min) peak systolic velocities were measured from the spectral doppler tracing with a sweep speed of 50 mm/s. CAPVV was calculated as: CAPVV = ([PV max- PV min]/PV mean) × 100, where PV mean = (PV max + PV min)/2 and the results were expressed in percentages. For both IVCCI and CAPVV, three readings were obtained and their mean was taken as the final value for statistical analysis. The operator (SRC) who obtained the ultrasound images was competent in basic critical care ultrasonography. All the images were reviewed by another experienced researcher competent in advanced critical care ultrasonography and poor-quality images were excluded. The CAPVV and IVCCI were both calculated by an operator who was blinded to the intraoperative haemodynamics.

Spinal anaesthesia (SA) was administered in all the patients in the sitting position at L3-L4 or L4-L5 vertebral level through median approach after local anaesthetic infiltration with 2.5 mL of 0.5% heavy bupivacaine with 25 μg of fentanyl (time 0 min). Co-loading was done with 15 mL kg^{-1} of Ringer lactate over 20 min. The heart rate (HR) and non-invasive blood pressure (NIBP) were recorded at 0 min, and then, every 2.5 min till 15 min after spinal anaesthesia. The block height was defined as the highest level of sensory blockade to cold, which was T6-T8 for all the patients. The positioning for surgery was done after 15 min of injection. PSH was defined as a reduction of the mean blood pressure (MBP) more than or equal to 20% of the baseline in 15 min after the injection of the drug in the subarachnoid space. The PSH was treated with IV boluses of ephedrine 6 mg every 5 min to a maximum of two doses. After that, a fluid bolus of 500 mL Ringer lactate was given if the patient was not responding to the vasopressor.

The sample size was estimated with *easyROC v. 1.3* web-based tool, which works on the R- platform.[8] At the time of designing this study, no previous clinical study evaluated the role of IVCCI in predicting PSH. Zhang *et al*.[6] reported that the area (95% confidence interval) under receiver operating characteristic (AUROC)curve was 0.9 for IVCCI to predict hypotension after the induction of general anaesthesia. From this study, we conservatively assumed that the IVCCI might have lower predictive validity of 0.7 for post-spinal induction hypotension. Based on this result, a sample of 48 patients (considering one-half of all the patients will develop hypotension) achieved 95% power and a probability of 0.05 for rejecting the null hypothesis. So, $n = 50$ patients were included in this study.

The lowest MBP recorded after SA was used to calculate the percentage of change from the baseline. All data were presented as median and inter-quartile range (IQR). The non-parametric and categorical variables were compared by the Mann–Whitney U test and the binary variables were compared by the Chi-square test. Shapiro-Wilk test was used to check the normality of IVCmax, IVCmin, IVCCI, and CAPVV receiver operator characteristic (ROC) curves were constructed for IVCCI and CAPVV as predictors of PSH and best cut-off values were obtained from Youden's index.[9] The AUROC of IVCCI and CAPVV were compared using DeLong's method.[10] The clinical predictors of PSH were found after performing the multivariable logistic regression analysis. Clinical variables, which were significantly different between the hypotensive and non-hypotensive patients at $P = 0.1$ or less, were included in the multivariable model. Some researchers recently analysed the utility of dIVCmax/IVCCI (dIVCmax is the maximum diameter of IVC).[11] This study was not available at the time of submitting the protocol for our study. We analysed IVCmax/IVCCI, IVCmax and IVCmin as *post hoc* analyses. All statistical analyses were conducted in STATA 13 software for Mac OS. (StataCorp. 2011. Stata Statistical Software: Release 13. College Station, TX: StataCorp LP.). While expressing BP and HR, we approximated the median value to the nearest integer.

RESULT

A total of 79 patients were assessed for eligibility initially. On meeting the exclusion criteria, 58 patients were recruited. We excluded eight more patients for different reasons like inability to obtain a satisfactory ultrasound image, conversion to general anaesthesia, etc. Finally, 50 patients were included for statistical analysis [Figure 1]. Out of the 50 patients, 17 (34%) patients developed PSH. We divided the patients into two groups as PSHY (developed PSH) and PSHN (not developed PSH). All demographic and haemodynamic data were comparable between the two groups, except baseline MBP (MBP0) which was significantly lower in the PSHY group. The HR was higher in the PSHY group than the PSHN in all time points, but it was not statistically significant [Table 1].

Ultrasonographic data were recorded [Table 2]. IVCCI >21.15 could predict PSH with 58.8% sensitivity,

69.7% specificity, 50% positive predictive value (PPV), and 74% negative predictive value (NPV), and AUROC 0.600 [Figure 2a]. CAPVV >18.33 predicted PSH with 70.6% sensitivity, 54.6% specificity, 44% PPV, 78.3% NPV, and AUROC 0.639 [Figure 2b]. IVCmax/IVCCI >60 could predict PSH with 58.8% sensitivity, 54.5% specificity, 40% PPV, 72% NPV, and AUROC 0.5267 [Figure 2c]. A logistic regression model combining IVCmax, CAPVV, MBP0 could predict PSH with AUROC 0.743 [Table 3]. When the diagnostic accuracy of IVCCI and CAPVV was compared using DeLong's method, none was found to be superior [Table 4].

DISCUSSION

The principal finding of our study was that neither IVCCI nor CAPVV were good predictors of PSH in adult patients undergoing infra-umbilical surgery under spinal anaesthesia, although a composite model including the ultrasound parameters and baseline MBP can predict PSH efficiently.

We also found that the age, height, and weight of patients could not predict PSH. This is opposite to the finding of Hartmann *et al.*,[12] who found these parameters to be associated with PSH. The small sample size of our study could have contributed to this finding. Moreover, they defined PSH as a 30% drop in MAP within 10 min of the administration of spinal anaesthesia, unlike our study. We found a good correlation of baseline MBP with PSH ($P < 0.001$). This is in agreement with the results of Carpenter *et al.*,[13] who found the baseline systolic blood pressure (SBP) <120 mmHg to be a predictor of PSH, where PSH was defined as SBP <90 mmHg.

The exact definition of PSH is very controversial. One extensive literature search revealed 15 different definitions of PSH. Decrease to 80% in systolic blood pressure (SBP) from the baseline, and combined criteria as 80% decrease in SBP from the baseline or SBP <100 mmHg were the commonest definitions.[14] Not surprisingly, the incidence of PSH has varied widely in different studies. The incidence of PSH in the current study (34%) is like in various other studies in the literature.[13]

In a study recruiting 60 patients undergoing elective knee joint replacement surgery, the investigators measured the inspiratory and expiratory IVC diameters before and after SA. There were no significant changes from the baseline measurements in the IVC diameters in the patients with or without PSH or bradycardia.[15] Similar results were obtained by Jaremko *et al.*[16] who found no significant difference between baseline

Table 1: Demographic and haemodynamic variables

Parameters	All Patients (*n*=50)	PSHY (*n*=17)	PSHN (*n*=33)
Age (years)	46.5 (35-61)	49 (42-60)	45 (35-61)
Body weight (kg)	60 (54-65)	60 (56-65)	60 (54-65)
Height (cm)	164 (160-165)	162 (158-165)	165 (162-165)
Sex (male/female)	38/12	12/5	26/7
MBP0 (mmHg)	99 (86-108)	109 (105-115)	96 (84-106)
HR0 (bpm)	88 (76-100)	87 (76-107)	88 (78-98)

n=number of patients; MBP0-mean blood pressure at time=0 min (when spinal anaesthesia was administered); HR0-heart rate at time=0 min; PSHY- patients who developed post-spinal hypotension; PSHN- patients who did not develop post-spinal hypotension

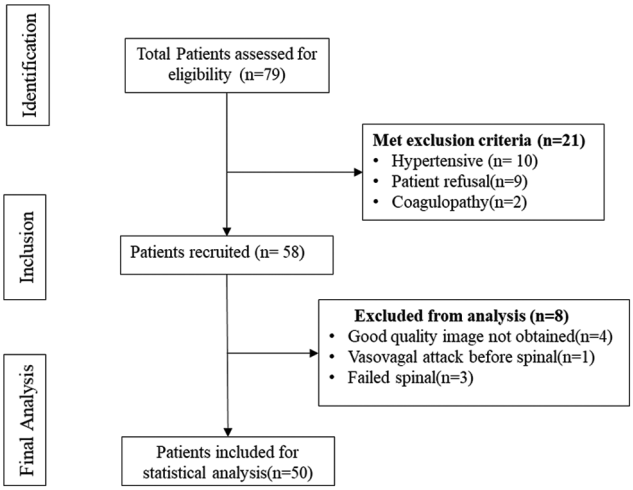

Figure 1: Flow diagram showing patient recruitment process

Table 2: Ultrasound measurements and parameters

Parameters	All Patients (*n*=50)	PSHY (*n*=17)	PSHN (*n*=33)	Significance (*P*-value)
IVC max (cm)	1.10 (0.9-1.35)	1.30 (0.93-1.45)	1.1 (0.85-1.23)	0.06
IVC min (cm)	0.70 (0.49-0.80)	0.74 (0.47-1.09)	0.70 (0.51-0.78)	0.33
IVCCI	16.14 (9.79-23.18)	21.2 (11.68-26.87)	15.16 (9.79-21.31)	0.24
CAPVV	21.06 (12.53-29.39)	26.45 (14.48-32.40)	17.43 (12.53-27.49)	0.10
IVCmax/IVCCI	60 (50-100)	70 (40-120)	60 (50-100)	0.75

n=number of patients; IVCmax- maximum diameter of inferior vena cava; IVCmin- minimum diameter of inferior vena cava; IVCCI-inferior vena cava collapsibility index; CAPVV- carotid artery peak systolic velocity variation; PSHY- patients who developed post-spinal hypotension; PSHN- patients who did not develop post-spinal hypotension

and post-intervention IVC parameters among the patients who were hypotensive after SA and those who were not.

In an observational study among 45 pregnant females undergoing caesarean section, the IVCCI was obtained with a wedge under the right hip and without it. The IVCCI predicted PSH with poor sensitivity and specificity in both groups.[17]

A few other sonographic parameters like caval aorta index (IVC diameter: aortic diameter) and dIVCmax (maximum diameter of IVC at expiration): IVCCI ratios have also been evaluated as predictors of PSH. The pre-operative caval aorta index was found to be a better predictor of PSH (sensitivity 96%) than IVCCI (sensitivity 84%) in elderly patients.[18] Saranteas *et al*.[11] found that dIVCmax: IVCCI predicted PSH better than IVCCI >0.3 ($P = 0.032$). This measurement takes both static (dIVCmax) and dynamic (IVCCI) components into account. However, in the current study, the caval aorta index was not measured and IVCmax/IVCCI was not found to be a good predictor of PSH.

Table 3: Multivariable logistic regression to predict PSH

Variable	Odds ratio	95% CI	Significance (*P*-value)
IVCmax	1.69	0.34-8.43	0.52
CAPVV	1.00	0.96-1.04	0.93
MBP0	1.06	1.00-1.12	0.04*

IVCmax- maximum diameter of inferior vena cava; CAPVV-carotid artery peak systolic velocity variation; MBP0-mean blood pressure at time=0 min (when spinal anaesthesia was administered); CI- confidence interval *P<0.05 (statistically significant)

The reasons behind our findings are probably multifactorial. First, some researchers used the IVC ultrasound-guided fluid therapy while we co-loaded the patients with 15 mL kg^{-1} of Ringer lactate without any specific USG-guided endpoint of fluid therapy.[6,7] The co-loading might have prevented PSH in hypovolaemic patients. Second, the IVCCI is also determined by the patient's respiratory effort and pattern.[19,20] The diaphragmatic descent can compress the IVC and can cause more reduction of the IVC diameter during inspiration in abdominal breathing as compared to thoracic breathing and falsely elevate the IVCCI. This can happen in anxious pre-operative patients. However, our patients were premedicated with anxiolytics and we asked them to breathe normally and to avoid deep breathing during sonography. Third, the diameter of IVC decreases and its respiratory variations increase significantly with advancing age.[21] About 30% of the patients ($n = 15$) in our study were aged ≥60 years. The age-related change in the IVC diameter might have impacted our outcomes.

We have found that CAPVV is also a poor predictor of PSH. Our result agrees with the results of Maitra *et al*.,[22] who reported CAPVV to be a poor predictor of post-induction hypotension in patients receiving general anaesthesia for elective surgeries. CAPVV >18.8% could predict hypotension with only 61.9% sensitivity and 67.4% specificity in their study. The different sensitivity of IVCCI and CAPVV in our study is probably because the peak velocity

Table 4: Comparison of diagnostic accuracy of IVCCI and CAPVV

Parameter	AUROC (*n*=50)	Standard error (95% CI)	Significance (*P*-value)
IVCCI	0.408	0.086 (0.238-0.577)	0.13
CAPVV	0.568	0.083 (0.405-0.731)	

n=number of patients; IVCCI- inferior vena cava collapsibility index; CAPVV- carotid artery peak systolic velocity variation; AUROC-area under receiver operating characteristic curve, CI- confidence interval

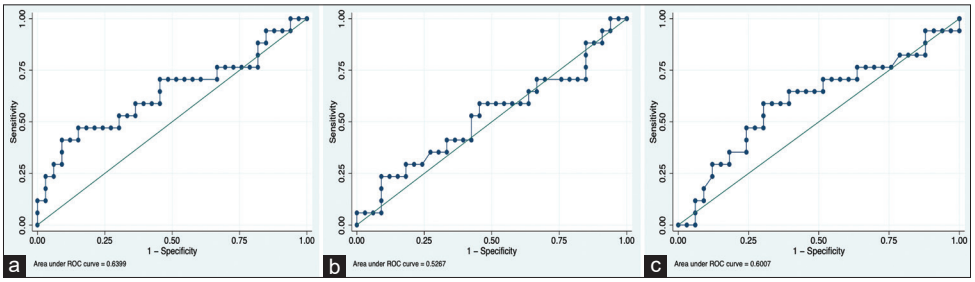

Figure 2: (a) Receiver operating characteristic curve showing the ability of pre-operative inferior vena cava collapsibility index to predict post-spinal hypotension. Area under the curve is 0.600; (b) Receiver operating characteristic curve showing the ability of pre-operative carotid artery peak systolic velocity variation to predict post-spinal hypotension. Area under the curve is 0.639; (c) Receiver operating characteristic curve showing the ability of the ratio of maximum inferior vena cava diameter and inferior vena cava collapsibility index to predict post-spinal hypotension. Area under the curve is 0.5267

in the carotid artery and IVC diameter is affected differently by the respiratory efforts and patterns in spontaneously breathing patients. Although the basic mechanism of PSH is a pre-ganglionic sympathetic block produced by the local anaesthetics, it is not explained by decreased pre-load or after-load. The pathophysiology of PSH is multifactorial which includes circulatory effects of local anaesthetics, relative adrenal insufficiency, skeletal muscle paralysis, ascending medullary vasomotor block, and associated respiratory insufficiency. That is probably the reason the tools successfully used for predicting fluid responsiveness in different settings are not efficient in predicting PSH.

Our study was underpowered with small sample size. As the possibility of a type II error cannot be ruled out, the result of the binary regression model should be considered as 'exploratory' rather than 'confirmatory'. We did not obtain the aortic diameter, aortocaval index, carotid corrected flow time (cFT), or other parameters, which are less affected by respiration. Invasive BP monitoring was avoided just for the research purpose when clinically not indicated. However, theoretically, invasive monitoring would have given us more precise data. We have not assessed the correlation of PSH with the type of surgery, which may be a confounding factor.

CONCLUSION

Neither IVCCI nor CAPVV was able to predict PSH in adult patients undergoing elective infra-umbilical surgery. A model consisting of IVCmax, CAPVV, and baseline MBP could predict PSH. However, the results of our study should be interpreted with caution and further studies with larger sample sizes are suggested particularly in elderly patients and in patients with pre-existing cardiovascular diseases.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Alghanem SM, Massad IM, Almustafa MM, Al-Shwiat LH, El-Masri MK, Samarah OQ, *et al*. Relationship between intraoperative hypotension and post-operative complications in traumatic hip surgery. Indian J Anaesth 2020;64:18-23.
2. Sanborn KV, Castro J, Kuroda M, Thys DM. Detection of intraoperative incidents by electronic scanning of computerised anaesthesia records. Comparison with voluntary reporting. Anesthesiology 1996;85:977-87.
3. Mackenzie DC, Noble VE. Assessing volume status and fluid responsiveness in the emergency department. Clin Exp Emerg Med 2014;1:67-77.
4. Osman D, Ridel C, Ray P, Monnet X, Anguel N, Richard C, *et al*. Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge. Crit Care Med 2007;35:64-8.
5. Donati A, Mercuri G, Iuorio S, Sinkovetz L, Scarcella M, Trabucchi C, *et al*. Haemodynamic modifications after unilateral subarachnoid anaesthesia evaluated with transthoracic echocardiography. Minerva Anestesiol 2005;71:75-81.
6. Zhang J, Critchley LA. Inferior vena cava ultrasonography before general anaesthesia can predict hypotension after induction. Anesthesiology 2016;124:580-9.
7. Ayyanagouda B, Ajay BC, Joshi C, Hulakund SY, Ganeshnavar A, Archana E. Role of ultrasonographic inferior venacaval assessment in averting spinal anaesthesia-induced hypotension for hernia and hydrocele surgeries—A prospective randomised controlled study. Indian J Anaesth 2020;64:849-54.
8. Goksuluk D, Korkmaz S, Zararsiz G, Karaagaoglu A Ergun. easyROC: An interactive web-tool for ROC curve analysis using R language environment. R J 2016;8:213.
9. Youden WJ. Index for rating diagnostic tests. Cancer 1950;3:32-5.
10. DeLong ER, DeLong DM, Clarke-Pearson DL. Comparing the areas under two or more correlated receiver operating characteristic curves: A nonparametric approach. Biometrics 1988;44:837-45.
11. Saranteas T, Spiliotaki H, Koliantzaki I, Koutsomanolis D, Kopanaki E, Papadimos T, *et al*. The utility of echocardiography for the prediction of spinal-induced hypotension in elderly patients: Inferior vena cava assessment is a key player. J Cardiothorac Vasc Anesth 2019;33:2421-7.
12. Hartmann B, Junger A, Klasen J, Benson M, Jost A, Banzhaf A, *et al*. The incidence and risk factors for hypotension after spinal anaesthesia induction: An analysis with automated data collection. Anesth Analg 2002;94:1521-9.
13. Carpenter RL, Caplan RA, Brown DL, Stephenson C, Wu R. Incidence and risk factors for side effects of spinal anaesthesia. Anesthesiology 1992;76:906-16.
14. Klöhr S, Roth R, Hofmann T, Rossaint R, Heesen M. Definitions of hypotension after spinal anaesthesia for caesarean section: Literature search and application to parturients. Acta Anaesthesiol Scand 2010;54:909-21.
15. Mačiulienė A, Gelmanas A, Jaremko I, Tamošiūnas R, Smailys A, Macas A. Measurements of inferior vena cava diameter for prediction of hypotension and bradycardia during spinal anaesthesia in spontaneously breathing patients during elective knee joint replacement surgery. Medicina (Kaunas) 2018;54:49.
16. Jaremko I, Mačiulienė A, Gelmanas A, Baranauskas T, Tamošiūnas R, Smailys A, *et al*. Can the inferior vena cava collapsibility index be useful in predicting hypotension during spinal anaesthesia in a spontaneously breathing patient? A mini fluid challenge. Acta Med Litu 2019;26:1-7.

17. Singh Y, Anand RK, Gupta S, Chowdhury SR, Maitra S, Baidya DK, *et al*. Role of IVC collapsibility index to predict post spinal hypotension in pregnant women undergoing caesarean section. An observational trial. Saudi J Anaesth 2019;13:312-7.
18. Salama ER, Elkashlan M. Pre-operative ultrasonographic evaluation of inferior vena cava collapsibility index and caval aorta index as new predictors for hypotension after induction of spinal anaesthesia: A prospective observational study. Eur J Anaesthesiol 2019;36:297-302.
19. Laborda A, Sierre S, Malvè M, De Blas I, Ioakeim I, Kuo WT, *et al*. Influence of breathing movements and Valsalva manoeuver on vena caval dynamics. World J Radiol 2014;6:833-9.
20. Kimura BJ, Dalugdugan R, Gilcrease GW 3rd, Phan JN, Showalter BK, Wolfson T. The effect of breathing manner on inferior vena caval diameter. Eur J Echocardiogr 2011;12:120-3.
21. Masugata H, Senda S, Okuyama H, Murao K, Inukai M, Hosomi N, *et al*. Age-related decrease in inferior vena cava diameter measured with echocardiography. Tohoku J Exp Med 2010;222:141-7.
22. Maitra S, Baidya DK, Anand RK, Subramanium R, Bhattacharjee S. Carotid artery corrected flow time and respiratory variations of peak blood flow velocity for prediction of hypotension after induction of general anaesthesia in adult patients undergoing elective surgery: A prospective observational study. J Ultrasound Med 2020;39:721-30.