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Long Case Report

Effect of Neuraxial Anaesthesia on Left Ventricular Diastolic Function Assessed by Transthoracic Echocardiography

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Abstract

Purpose: To evaluate the effect of neuraxial anaesthesia on left ventricular (LV) diastolic function in clinical setting using transthoracic echocardiography (TTE).

Methods: This prospective observational study was performed in 50 adult patients undergoing elective orthopaedic surgical procedures under neuraxial anaesthesia for lower limb surgery. TTE was performed before, 20, 40 and 60 minutes after neuraxial anaesthesia. Pulsed wave Doppler of the transmitral flow (TMF), pulmonary venous flow (PVF), deceleration time (DT) and propagation velocity (Vp) were measured. Septal and lateral wall mitral annular velocities (E', A') were assessed by tissue Doppler imaging (TDI). The maximum diameter of left atrium (LA) and right atrium (RA), LA volume index, left ventricular (LV) and right ventricular (RV) end-diastolic area (EDA), end-systolic area (ESA), fractional area change (FAC),LV end-diastolic volume (EDV), end-systolic volume (ESV), were measured from apical 4-chamber view (A4CV) view.

Results: There were 50 patients in the cohort of whom 48 had normal diastolic function preoperatively. Following neuraxial anaesthesia, mean arterial pressure decreased (96.6±1.52 to 83.7±0.3, $p < 0.001$) while heart rate remained unchanged (84.4±16.6 to 85.3±15.0, $p = 0.436$). The dimensions and volumes of cardiac chambers, LV FAC and RV FAC transmitral pulse wave Doppler, DT, Vp, PVF and mitral annular TDI did not vary after neuraxial anaesthesia ($p > 0.05$).

Conclusion: In patients with normal diastolic function, neuraxial anaesthesia does not alter diastolic function indices and grading. It is recommended that the study should be performed in patients with documented diastolic dysfunction to demonstrate beneficial or detrimental effects of central neuraxial blockade, if any.

Keywords: Spinal anaesthesia, Neuraxial anaesthesia, Transthoracic echocardiography, Diastolic function, Left ventricle

Introduction

Abnormal ventricular diastolic function may lead to clinical heart failure (HF) in 40 to 50% of patients suffering from HF despite their having normal systolic function. [1, 2] Left ventricular (LV) diastolic function plays a major role in determining the overall cardiovascular performance, and heart failure resulting from diastolic dysfunction may occur in the absence of/or precede the development of abnormalities in systolic function. Unrecognized and untreated diastolic dysfunction may increase perioperative mortality and morbidity [3-5]. The incidence of diastolic dysfunction is increasing alarmingly due to age and increase in comorbidities such as hypertension, diabetes mellitus, thyroid diseases, chronic kidney disease and others. [6, 7] Clinically, diastolic function may be measured reliably by Transthoracic Echocardiography (TTE). Conventionally, transmitral pulsed wave Doppler and pulmonary venous Doppler are used to identify diastolic dysfunction. However, the major disadvantages of these techniques are their dependence on left ventricular loading conditions and a display of biphasic response to increasing

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disease severity. [8, 9] These limitations have led to the development of newer modalities of assessment independent of preload conditions.

Neuraxial anaesthesia, which includes spinal and epidural anaesthesia, is one of the most commonly used anaesthetic techniques for surgery below the rib cage. Neuraxial anaesthesia can lead to alteration of preload and afterload due to sympathetic blockade. These altered loading conditions can affect left ventricular diastolic function in patients with normal as well as abnormal diastolic function. Moreover, unrecognized diastolic dysfunction in the presence of near normal systolic function can have adverse consequences like hemodynamic instability and acute pulmonary edema in the perioperative period. [10, 11] Consequently, anaesthesiologists will benefit from an understanding of the effect of neuraxial anaesthesia on ventricular diastolic function.

A study by Couture et al. evaluated the effect of induction of general anaesthesia on diastolic function using echocardiography. The authors concluded that left ventricle (LV) diastolic function score improved after induction of anaesthesia, most likely, but inconclusively, related to the altered loading conditions. [12] Gare et al. noted that mild sedation with midazolam or propofol did not result in changes in left ventricle diastolic function indices. [13] However, no specific clinical study has been carried to our knowledge to analyse the effect of neuraxial anaesthesia on left ventricular diastolic function. Thus, we conducted a prospective study to evaluate the effect of neuraxial anaesthesia on LV diastolic function in surgical patients requiring spinal anaesthesia for lower extremity surgery. We hypothesized that neuraxial anaesthesia by altering loading conditions may alter left ventricular dimensions and diastolic function indices.

Materials and Methods

After obtaining approval from our ethics review board we conducted a prospective observational study in tertiary care centre in 60 consecutive consenting elective orthopaedic surgical patients ≥ 18 years age. The study was registered the in clinical trial registry-India. All patients were scheduled to receive a central neuraxial block. Patients with known valvular heart diseases, non-sinus rhythms, psychiatric diseases, emergency surgery, those with permanent pacemaker implantation and renal dysfunction were excluded from the study. Similarly, patients with contraindications for neuraxial anaesthesia were also excluded. Preoperative data included demographic information, presence of co-morbid conditions, concurrent medications, and the results of basic laboratory tests. All anti-hypertensive medications except angiotensin converting enzyme inhibitors /angiotensin receptor blockers were continued till the day of surgery. Once in the operating room (OR), monitoring was initiated following American Society of Anaesthesiologist (ASA) standards and included non-invasive blood pressure (NIBP), continuous electrocardiogram (ECG) and pulse oximetry (SpO_2). Baseline hemodynamic parameters such as oxygen saturation, heart rate, and blood pressure were noted. All study patients underwent a thorough TTE examination at baseline by an experienced cardiac anaesthesiologist using a GE healthcare echocardiographic machine (VENUE GO, General Electric Healthcare, Wauwatosa, WI, United States)) and a 3Sc, 1.6-4.5 MHz Probe. TTE measurements were done as per the ASE recommendations and included the following: Left ventricular end-diastolic area (LVEDA), left ventricular end-systolic area (LVESA), and fractional area change (LVFAC). The LV volumes were calculated during systole and diastole and the left ventricular ejection fraction (LVEF) was computed using the Modified Simpson's biplane method via the apical four chamber view.

Transmitral flow (TMF) velocity was assessed by aiming the Pulse wave doppler (PW) on mitral leaflet tip in the apical four chamber view. Peak early diastolic flow velocity (E), peak late diastolic flow velocity (A) and E/A were measured and the mitral valve deceleration time was obtained. Pulmonary venous flow (PVF) was evaluated using PW Doppler, and peak systolic (S), diastolic (D), atrial reversal (AR) flow velocities and S/D ratio were measured. Left atrium diameter and left atrium maximum volume index (expressed as ml/BSA) was calculated from the apical four chamber view before opening of the mitral valve as per ASE recommendations. Mitral valve annular velocity (S' and E') were obtained at the lateral annulus by using the tissue Doppler method. Later E/E' was calculated. Similarly, septal tissue Doppler velocity was also measured. The right ventricle end-diastolic area (RV EDA), RV end-systolic area (RV ESA), and RV fractional area change (RV FAC) was calculated from the apical four chamber view. All parameters were measured in sinus rhythm and during stable hemodynamics. An average of three consecutive readings of the above parameters were obtained and used for statistical analysis. All echocardiographic images and loops were digitally stored and analysed off-line by two independent cardiac physician echocardiographers blinded to the study. The grading of LV diastolic function was based on ASE recommendations. [14]

TTE was performed over four time points throughout the study. Baseline TTE was carried out in the operation theatre prior to institution of the lumbar neuraxial block. Similarly, TTE was performed after 20 minutes, 40 minutes and 60 minutes after the block. Neuraxial anaesthesia was performed aseptically, in the lateral decubitus/sitting position by an experienced anaesthesiologist; the interspace between L3-L4 or L4-L5 space was chosen for administering the block. Spinal anaesthesia was given using a 26 Gauge Quinke's needle injecting 10 mg of isobaric 0.5% bupivacaine (2 ml) with 25-50 µg of fentanyl (0.25-0.5 ml). Epidural anaesthesia was given using an 18G Tuohy needle and injecting 0.5% plain bupivacaine 12-15 ml given over 10 minutes into the entered epidural space. Patients were allowed to spontaneously breathe with supplemental oxygen at 4 L/min via nasal prongs. The dermatome level of the sensory block was assessed every 5 minutes for 20 minutes using an alcohol-soaked gauze pad bilaterally. The modified Bromage scale (0= no motor block; 1= straight leg hip flexion blocked; 2= knee flexion blocked; 3= complete motor block) was used to quantify the degree of motor block at 20 minutes after the placement of neuraxial anaesthesia. The patient's heart rate and blood pressure were continually measured at 5-minute intervals until the end of surgery. Hypotension was defined as a decrease in mean arterial pressure (MAP) by ≥ 20% from baseline and was treated with an intravenous bolus of 100 µg phenylephrine and repeated as necessary. Bradycardia was defined as heart rate ≤ 50 beats per minute (BPM) and was treated with 1 mg of atropine IV, if associated with hypotension. Co-loading of intravenous fluid was given per the discretion of the attending anaesthesiologist.

Statistics

Based on a pilot study, the sample size required was calculated to be 40 patients. For distribution of continuous data, we used the Kolmogorov– Smirnov one-sample and the Shapiro-wilk tests. Continuous variables with a normal distribution are expressed as mean + standard deviation (SD). Dichotomous data are expressed as numbers and percentages. The Wilcoxon rank-sum test was used to compare continuous variables (hemodynamic and echocardiographic data) obtained before and after neuraxial anesthesia. The Chi-square test or Anova test were used to evaluate for statistical significance ($p \leq 0.05$). All the statistical tests were two-sided and were conducted at a significance level of $\alpha = 0.05$. Data was analysed by using the statistical package for social sciences (SPSS Inc., Chicago, IL version 22.0).

Table 1: Study patients demographics and clinical data

Variable	Descriptive statistics
Age in years (Mean ± SD)	41.30 ± 11.76
Gender Male/Female (%)	37/13 (74/26)
Weight in kilogram (Mean ± SD)	68.16 ± 13.31
Height in centimeters (Mean ± SD)	166.32 ± 7.06
Body Surface Area (Mean ± SD)	1.71 ± 0.23
American Society Anaesthesiologist 1/3	36/12/2
Coronary artery disease: Yes/No (%)	1/49 (2/98)
Hypertension: Yes/No (%)	6/44 (12/88)
Diabetes mellitus: Yes/No (%)	9/41 (18/82)
Hypothyroidism Yes/No (%)	1/49 (2/98)
Hyperthyroidism Yes /No (%)	1/49 (2/98)
Hemoglobin in g/L (Mean ± SD)	11.78 ± 1.76
Serum creatinine in mg/dl (Mean ± SD)	0.91 ± 0.23
Spinal/Epidural	45/5
Postoperative Intensive Care Unit stay in days Yes/No (%)	1/49 (2/98)
leg surgery/hip bones surgery/knee joint surgery/ankle surgery	31/11/4/4

Table 2: Hemodynamic changes after neuraxial anaesthesia

Parameters	Baseline	20 minutes	40 minutes	60 minutes	p
HR (/min)	84.4±16.6	85.3±15.0	83 ± 12.3	81±11.1	0.436
SBP (mm Hg)	144.6±16.74	122.6±17.9	125.3±11.2	130.1±12.4	<0.001
DBP (mm Hg)	81.6±12.6	71.6±71.6	75.04± 9.7	76.7±10.34	<0.001
MAP (mm Hg)	96.6±1.52	83.7±0.3	87.6± 0.2	88.9± 0.14	<0.001

HR- Heart rate, SBP- Systolic blood pressure, DBP- Diastolic blood pressure, MAP- Mean arterial pressure

Table 3: Chamber dimensions, volumes and functions

Parameters	Baseline	20 minutes	40 minutes	60 minutes	p
LA Maximum Diameter (cm)	3.03±0.39	3.11±0.45	3.03±0.37	3.04±0.38	0.7
LVEDA (cm^2)	19.46±4.06	19.53±3.61	19.49±3.49	19.95±3.88	0.9
LVESA (cm^2)	10.68±3.44	10.43±2.92	10.15±2.76	10.33±2.77	0.8
LVFAC (%)	45.14±6.91	49±7.54	46.30±7.46	45.92±7.67	0.6
LVEDV (ml)	98.46±19.66	98.04±15.71	96.34±15.90	97.38±15.46	0.9
LVESV (ml)	42.30±9.23	43.3±7.65	42.37±7.69	42.10±6.96	0.8
LVEF (%)	56.76±6.74	54.90±6.56	56.04±6.30	55.84±5.84	0.5
RA Maximum Diameter	3.28±0.5	3.26±0.47	3.22±0.39	3.25±0.4	0.9
RVEDA (cm^2)	21.42±2.44	21.83±2.31	22.04±1.99	21.71±2.16	0.9
RVESA (cm^2)	11.70±1.68	11.48±1.66	11.30±1.59	11.25±1.48	0.4
RVFAC (%)	47.32±5.77	47.15±8.96	48.96±5.99	49.00±5.87	0.3

LA- left atrium, LVEDA- Left ventricular end-diastolic area, LVESA- Left ventricular end-systolic area, LVFAC- left ventricular fractional area change, LVEDV- left ventricular end-diastolic volume, LVESV- Left ventricular end-systolic volume, LVEF- Left ventricular ejection fraction, RA- right atrium, RVEDA- Right ventricular end-diastolic area, RVESA- Right ventricular end-systolic area, RVFAC- right ventricular fractional area change

Table 4: Left ventricular diastolic function parameters

Parameters	Baseline	20 minutes after spinal	40 minutes after spinal	60 minutes after spinal	p
E (m/s)	0.77±0.14	0.77±0.13	0.77±0.13	0.78±0.13	0.989
A (m/s)	0.60±0.13	0.59±0.13	0.59±0.11	0.58±0.11	0.922
E/A ratio	1.29±0.34	1.29±0.36	1.26±0.28	1.29±0.31	0.953
PVF S (m/s)	0.48±0.08	0.50±0.10	0.49±0.10	0.48±0.09	0.9
PVF D (m/s)	0.34±0.09	0.35±0.09	0.34±0.08	0.34±0.09	0.9
PVF S/D ratio	1.51±0.44	1.49±0.45	1.49±0.46	1.50±0.45	0.9
Septal wall e' (m/s)	0.10±0.03	0.10±0.03	0.10±0.03	0.09±0.02	0.707
Septal wall a' (m/s)	0.06±0.02	0.06±0.02	0.06±0.02	0.06±0.02	0.9
Septal wall E/e' ratio	7.95±2.92	7.73±2.65	8.19±3.64	8.06±3.09	0.8
Lateral wall e' (m/s)	0.12±0.03	0.11±0.03	0.11±0.02	0.11±0.03	0.1
Lateral wall a' (m/s)	0.07±0.02	0.07±0.02	0.07±0.02	0.07±0.02	0.75
Lateral wall E/e' ratio	6.69±2.59	6.72±2.40	6.76±2.42	6.61±2.30	0.99
DT (msec)	144.88±30.73	151.48±29.33	150.14±27.93	151.68±29.08	0.6
Vp(cm/sec)	60.20±6.80	60.60±5.19	59.12±5.66	59.86±5.11	0.6
LA volume index (ml/m^2)	22.98±6.32	23.43±6.63	23.88±6.74	24.05±6.89	0.8

E- transmitral E wave, A- transmitral A wave, E/A- transmitral E/A ratio, PVF S- pulmonary vein flow systolic wave, PVF D- pulmonary vein flow diastolic wave, PVF S/D ratio- pulmonary vein flow systolic/diastolic wave ratio, Sw e'- Septal wall tissue Doppler imaging measurement of the mitral annulus velocity E wave, Sw a'- Septal wall tissue Doppler imaging measurement of the mitral annulus velocity A wave. Sw E/e'- Septal wall transmitral pulse doppler E wave velocity/tissue Doppler imaging measurement of the mitral annulus velocity E wave, Lw e'- lateral wall tissue Doppler imaging measurement of the mitral annulus velocity E wave, Lw a'- lateral wall tissue Doppler imaging measurement of the mitral annulus velocity A wave, Lw E/e' – Lateral wall transmitral pulse doppler E wave velocity/tissue Doppler imaging measurement of the mitral annulus velocity E wave, DT- deceleration time, Vp- Propagation velocity, LA- left atrium

Table 5: ASE gradings before and after neuraxial anaesthesia

ASE grading of DD	Baseline n (%)	20 minutes n (%)	40 minutes n (%)	60 minutes n (%)	p
No DD	48 (96%)	48 (96%)	48 (96%)	48 (96%)	0.238
Yes DD	0	0	0	0	
Indeterminate	2 (4%)	2 (4%)	2 (4%)	2 (4%)	

ASE -American society of Echocardiography, DD- diastolic dysfunction

Results

Of the 60 consecutive patients screened, 3 patients with mitral regurgitation, 1 patient with aortic regurgitation, 5 patients with poor echocardiographic windows and 2 patients with failed neuraxial block were excluded from the study. The remaining 50 patients were included in the final analysis. All patients in this cohort underwent their orthopedic surgical procedure without the need for supplemental general anesthesia. The demographic data are summarized in Table 1.

After spinal anaesthesia, there was no significant change in heart rate (p=0.46), however there was significant decrease in mean arterial pressure (MAP)(p <0.001) as shown in Table 2. Phenylephrine was utilised in 12 cases to maintain MAP. TTE revealed that 6 patients had LV hypertrophy. The average baseline LVEF and RV FAC of our patients were 56.8±6.8 % and 47.3±5.8, respectively. Following neuraxial anaesthesia, the various volumes and dimensions of LV and RV did not change significantly from baseline (Table 3). No significant change was seen in the peak transmitral E to A wave velocity after neuraxial anaesthesia. Similarly, there were no changes in PVF, mitral annular velocities, DT, Vp and LA volume indices after neuraxial anaesthesia (Table 4). There were no significant changes in diastolic function grading after neuraxial anaesthesia as shown in Table 5. In our study cohort, only one patient required ICU admission. All the other patients were transferred to their regular medical units without postoperative complications.

Discussion

This echocardiographic study found that the administration of neuraxial anaesthesia did not alter the TTE obtained parameters of diastolic function in patients undergoing orthopaedic surgery. Diastolic dysfunction is being increasingly recognised during non-cardiac surgery, and has been shown to affect perioperative outcomes. [15] The reported prevalence of the diastolic dysfunction in the general population, however, varies from 11.1 to 34.7%. [16-18]. The impaired relaxation and increased stiffness observed in a dysfunctional left ventricle results in an increase in the LV filling pressure and pulmonary venous pressure, thereby inducing left atrial enlargement. [19, 20] Patients with LV diastolic dysfunction are susceptible to developing pulmonary edema and atrial arrhythmias. [21] Matyal et al. evaluated 313 vascular surgery patients and demonstrated that LV diastolic dysfunction is a predictor of adverse cardiovascular outcomes. [22] Similarly, Flu et al. demonstrated that asymptomatic LV diastolic dysfunction is a predictive factor of 30-day and long-term cardiovascular adverse outcomes in open vascular surgery patients. [23] LV diastolic function can be assessed by TTE. Although various parameters are available, E/E' ratio is a relatively load independent characteristic that correlates with LV filling pressure and predicts certain cardiovascular outcomes, such as cardiomyopathy, acute myocardial infarction, and atrial fibrillation. [24-27] Cho et al. found that E/E′ > 15, pulmonary artery systolic pressure > 35 mm hg , and LVH in preoperative TTE predicted postoperative pulmonary edema, and E/E′ > 15 predicted major adverse cardiac events (MACE) in the patients who underwent low- or intermediate-risk noncardiac surgery. [26] We did not observe any significant changes in diastolic function parameters in this study cohort of orthopedic patients. Similarly, dimensions of RA, RV, LA and LV did not vary statistically after administration of neuraxial anaesthesia. Diastolic parameters may be affected by changes in preload, afterload, respiration, heart rate and left atrial pressure. LV EDV did not change after neuraxial anaesthesia suggesting that preload variation was not statistically significant in our study patients. In addition, the presence of spontaneous ventilation that results in a negative pleural pressure during inspiration and the concomitant reduction in intrathoracic pressure is known to augment preload (not seen during positive pressure ventilation). Furthermore, in our study, we observed that there was no significant change in heart rate. Previously, Hung et al. showed that volume reduction using hemodialysis is associated with a change in TDI and thus is not load independent. [28] Similarly, Abali et al showed that preload reduction of 500 ml by blood donation does not affect TMF velocities, Vp, and mitral annular tissue doppler velocities. [29] These studies suggest that preload variation alone might not significantly change diastolic parameters of LV. Relative maintenance of adequate preload and heart rate after neuraxial anaesthesia could be the reason for lack of variation in LV diastolic performance. Similar to this study, Ferre et al. in their prospective observational study in 20 elderly patients found that E and E/E' did not vary after 20 minutes of spinal anaesthesia. [30] However, they observed a slight increase in myocardial contractility that could be an adaptive mechanism to compensate the preload decrease and limit the fall in blood pressure. In contrary to ours results, Schulmeyer et al. observed significant changes in LV systolic and diastolic function parameters after spinal anaesthesia, however, they did not find any significant change in LV volumes. [31] In addition, the author mentioned that there was no correlation between the level of spinal block and the magnitude of changes. Couture et al. studied diastolic function after general anaesthesia induction in which they found a significant reduction in the atrial components of the TMF velocities and mitral annular tissue doppler velocities. They also observed increase in LA, RA and RV chamber dimensions and decrease in LV dimensions. They concluded that general anaesthesia improves the LV diastolic function score in patients with LV diastolic dysfunction. They indicated that the reduction in venous return, negative inotropism of general anesthesia, and the effects of positive pressure ventilation were the reason for alterations in LV diastolic parameters. In contrast to general anaesthesia, presence of spontaneous ventilation, minimal change in preload as evidenced by non-significant changes in LVEDV and lack of change in LV systolic function were the reasons for relatively stable LV diastolic parameters after neuraxial anaesthesia in our study. One of the strengths of our study is that we observed changes in both load dependent and load independent parameters of diastolic function, suggesting that none of the parameters varied with onset of neuraxial anaesthesia.

Limitations

One limitation in this study is that none of the patients had preoperative diastolic dysfunction indicating the need for evaluation of the effect of neuraxial anaesthesia in patients with severe diastolic dysfunction. Next, our study is a single centre study with a limited sample size. A multi-centre study with a larger sample size is likely to provide us more detailed information regarding the effect of neuraxial anaesthesia on LV diastology. Third, we included orthopedic patients with no significant change in patient positioning. Future studies should be conducted in obstetrics and gynaecology surgical patients, urology patients, and elderly patients which would help in better understanding of the effect of neuraxial anaesthesia on LV diastolic function. Finally, the impact of neuraxial anaesthesia on diastolic performance in patients with reduced LV systolic function will need to be explored.

Conclusion

In summary, the present study indicates that neuraxial anaesthesia does not affect indices of LV diastolic performance in patients with normal diastolic function and preserved LV systolic function as evaluated by TTE in orthopedic surgical patients.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the Journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

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