Effects of Spinal Anaesthesia on Left Ventricular Function: An Observational Study using Two-Dimensional Strain Echocardiography

Fabrice Ferré1, Clément Delmas1,2, Didier Carrié2, Thomas Cognet2, Olivier Lairez2,3, Vincent Minville1,3

1Department of Anesthesiology and Critical Care Medicine, Toulouse University Hospital, Toulouse, France
2Department of Cardiology, Department of Cardiac Imaging Center; Department of Nuclear Medicine, Rangueil University Hospital, Toulouse, France
3INSERM U 1048, I2MC, BP 84225, 31432, Toulouse cedex, France

Abstract

Objective: Hypotension frequently occurs during spinal anaesthesia (SA), especially in the elderly. This side effect could have a cardiac component per se (myocardial contractility impairment). Two-dimensional (2D) strain and strain rate imaging are new echocardiographic methods allowing an accurate assessment of myocardial function by quantifying myocardial deformation. Allowing quantification of minor myocardial dysfunction not detectable by standard echocardiography, strain imaging could bring new perspective on the cardiac effect of SA. Our objective was to evaluate the effects of SA on left ventricular function assessed by 2D strain echocardiography.

Methods: In this prospective observational study, we enrolled 20 patients older than 60 years, who underwent elective lower-limb surgery under SA. Myocardial strain imaging were collected before and 20 minutes after SA (injection of 10 mg of isobaric bupivacaine with 5 μg of sufentanil).

Results: We observed an increase in global longitudinal reconnoitering (Δ−0.2±0.3% s⁻¹; p<0.005), whereas left ventricular ejection fraction was not modified by SA.

Conclusion: This slight increase in myocardial contractility could be an adaptive mechanism to compensate the preload decrease and limit the blood pressure drop.

Keywords: Strain, reconnoitering, echocardiography, spinal anaesthesia

Introduction

Two-dimensional (2D) strain and strain-rate imaging are new non-invasive methods that allow the assessment of myocardial function through myocardial deformation by transthoracic echocardiography. Due to its ability to differentiate between active and passive movements of myocardial segments, it allows a comprehensive assessment of myocardial function and early detection of myocardial dysfunction with accuracy and reliability (1). As the spectrum of its potential clinical applications is very broad, strain imaging has recently become a subject of great interest for clinicians.

We thought that this echocardiographic tool could bring a new perspective on the still controversial ‘cardiac effect theory’ of spinal anaesthesia (SA) (2-5). Arterial hypotension is frequently observed during SA, especially in the elderly (6). This side effect, mainly due to preload and afterload decrease secondary to the sympathetic block, could cause a cardiac component per se (7). Indeed, arterial pressure determinant analysis with the well-known equation Arterial Pressure=Cardiac Output × Systemic Vascular Resistance, shows that the arterial pressure decrease observed in this setting could be theoretically due to an impairment in cardiac contractility. As they are load dependent, usual left ventricular (LV) systolic function parameters, such as LV ejection fraction (LVEF), are inefficient for exploring myocardial effects of SA. Because strain imaging is less likely dependent to loading conditions, this could shed a new light on the cardiac effect of SA. In this observational study, we explored the myocardial effects of SA by 2D strain echocardiography in the elderly.
Methods

This prospective observational study was approved by the local research ethics board (Comité d’Éthique Recherche, CHU Toulouse, Chairperson Dr Jean-Marie Conil, registration number 37-0911) and carried out according to the declaration of Helsinki. All patients gave written informed consent.

Twenty consecutive patients over 60 years of age, referred for elective lower-limb surgery under SA, were enrolled. According to routine practice at our institution, anti-hypertensive medications, except β-blockers, were discontinued the day before surgery. The usual criteria of fasting (6 hours for solids and 2 hours for non-particulate liquids) were observed. Patients received no pre-medication prior to arrival to the operating room. All patients were monitored in accordance with current guidelines, including non-invasive blood pressure (NIBP), as well as continuous electrocardiogram (ECG) and pulse oximetry (SpO2). Before the placement of spinal anaesthesia and the baseline haemodynamic and cardiovascular measurements, all patients received an intravenous bolus of 8 mL kg⁻¹ of Lactated Ringers® (LR) for over 30 minutes. The baseline mean arterial blood pressure (MAP) was calculated as the average of three consecutive measurements prior to placement of the SA.

Spinal anaesthesia (midline puncture) was performed in the lateral decubitus position by a staff anaesthetist at L3-L4 or L4-L5 with a 25 Gauge Withacre needle (BD, Franklin Lakes, NJ, USA) injecting 10 mg of isobaric 0.5% bupivacaine (2 mL) with 5 µg of sufentanil (1 mL for a total volume of 3 mL). After injection the patients remained in the lateral decubitus position for 5 min and were then turned supine. The dermatome level of the sensory block (assessed by the loss of cold sensation using an alcohol soaked gauze pad bilaterally) was checked every 5 minutes for 20 minutes. 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. Immediately after the placement of the SA, the patient’s heart rate and NIBP were continually measured at 5-minute intervals until the end of surgery. Hypotension was defined as a drop of 20% or more from baseline MAP and was treated with an intravenous bolus of 100 µg phenylephrine and repeated as necessary every 5 min. Bradycardia was defined as heart rate of less than 50 beats per minute (BPM) and treated by injection of 1 mg of atropine, if associated with hypotension. Intra-operative hypotension was managed using intravenous bolus of 100 µg phenylephrine, and bradycardia without hypotension was treated with intravenous bolus of 10 µg kg⁻¹ atropine.

Transthoracic echocardiography (Imagic KM 60, Kontron Medical, Saint-Germain en Laye, France) was performed using a 2.5 MHz transducer with patients in the supine position at two time points: first just before the SA (baseline, once the infusion of 500 mL of Ringer’s Lacate had been completed), and second 20 minutes after the SA (once the anaesthetic block was found to be sufficient). A complete 2D gray scale echocardiography including the three standard apical views (four-, three- and two-chambers) with a frame rate >75 frames was performed for each patient. Digital data of 3 consecutive heart cycles were recorded and transferred to a personal computer with My Lab Desk workstation (Kontron Medical, Saint-Germain en Laye, France) for offline analysis. Endocardial border was defined manually in end-systole and automatically tracked frame by frame. Operator assessed optimal evaluation of both quality of tracking and region of interest. Global longitudinal strain (GLS) and reconnoitering were obtained by averaging all segmental longitudinal strain curves computed from the conventional apical two-, three- and four-chambers views (Figure 1). Negative longitudinal strain values described shortening of a given myocardial segment related to its original length. Left ventricular ejection fraction (LVEF) was assessed using the conventional apical two- and four-chamber views and the modified Simpson’s method.

Statistical analyses were performed using the SPSS 19® software. Data are presented as mean±SD or n (%) or median [min-max] as appropriate. The Wilcoxon rank-sum test was used to compare continuous variables (hemodynamic and echocardiographic data) obtained before and after SA. P≤0.05 was considered statistically significant. A search in

Figure 1. Segmental longitudinal strain and reconnoitering curves (distinctively coloured curves for left ventricular wall segments) obtained from apical 4-chamber views by speckle-tracking 2D-strain imaging. Global longitudinal strain and reconnoitering were obtained by averaging all segmental longitudinal strain curves computed from the conventional apical two-, three- and four-chambers views. LONG: longitudinal; ENDO: endocardial border.
the literature did not reveal a previous study evaluating the myocardial contractility effects of SA using 2D strain echocardiography, and therefore, we were not able to calculate a sample size \textit{a priori}. Thus, the sample size analysis was made according to a pilot study in our unit.

**Results**

Demographic characteristics are shown in Table 1. Ten patients (50%) experienced at least one episode of hypotension defined by a 20% decrease in the mean arterial pressure. The median [min-max] use of fluid expansion between the two echocardiographic measurements was 500 mL [250-1000] (crystalloids only). In all hypotensive cases, phenylephrine was used only to restore hemodynamics. The total dose of phenylephrine was [200 µg [0-300]. No bradycardia was observed during the study. Strain and echocardiographic data are shown in Table 2. SA induced an increase of global longitudinal strain (GLS) rate (Δ−0.2±0.3% s\(^{-1}\), p<0.005). GLS was not modified by SA (Δ−1.6±4.2%, p=0.13). LVEF was not modified by SA (Δ+4.8±10.5%, p=0.29), whereas systolic peak velocities at the mitral annulus by tissue Doppler imaging increased (Δ+1.2±1.4 cm/s\(^{-1}\), p=0.026). No modification of the LV stroke volume induced by SA was observed (−0.4±3.8, p=0.8). LV diastolic function parameters were not modified by SA (E/A ratio and E/Ea ratio). Concerning preload variables, LV tele-diastolic volume and the diameter of the inferior vena cava tend to decrease after SA (Δ−11.3±23.2 mL, p=0.1 and Δ−7.2±8.6 mm, p=0.09, respectively).

**Discussion**

Hypotension is frequently observed after SA, especially in the elderly (6). This side effect is an independent risk factor of mortality because of regional hypoperfusion (8). Thus, understanding underlying mechanisms is of particular importance. Echocardiographic strain and strain-rate imaging are promising tools for the evaluation of myocardial function in this setting (1). We identified, for the first time, a slight improvement of LV systolic function assessed by strain imaging during SA. This could be considered as a cardiac adaptive mechanism. Indeed, compensating the preload decrease induced by SA limits the drop in blood pressure. Our results are consistent with a previous study showing biphasic cardiac output changes during onset of SA (4).

Hypotension in the elderly is common even after single injection low dose SA due to a decrease in cardiac output, left ventricular stroke volume and systemic vascular resistance (SVR) (9). Only a few studies have investigated the systolic function of the left ventricle during SA, and no significant changes have been reported in subjects with normal as well as moderate LV dysfunction. Our results in patients with low dose SA are consistent with these previous findings (5, 9, 10).

One limit of our study is that it is not a comparative study. However, we have demonstrated that even a low dose SA induced a decrease in cardiac output and SVR in all patients and is associated with hypotension (9).

**Conclusion**

Episodes of hypotension induced by SA and usually observed in the elderly are definitely not caused by a decrease in myocardial performance. Future investigations including a large number of patients with moderate or severe systolic dysfunction could be of major interest.

**Ethics Committee Approval:** Ethics committee approval was received for this study from the ethics committee of Comité d’Ethique Recherche, CHU Toulouse, Chairperson Dr Jean-Marie Conil, registration number 37-0911, Toulouse, France.

**Informed Consent:** Oral informed consent was obtained from all patients who participated in this study.
**Peer-review:** Externally peer-reviewed.

**Author Contributions:** Concept - V.M., O.L.; Design - V.M., O.L.; Supervision - V.M., O.L., D.C.; Data Collection and/or Processing - F.F., C.D., T.C.; Analysis and/or Interpretation - V.M., O.L., F.F., C.D., D.C.; Writing Manuscript - V.M., O.L., F.F.; Critical Review - D.C.

**Acknowledgements:** Dr Benoit Bataille for the statistical analysis and the editing of the manuscript.

**Conflict of Interest:** No conflict of interest was declared by the authors.

**Financial Disclosure:** This work should be attributed to the Department of Anesthesiology and Intensive Care, Toulouse University Hospital, Toulouse, France. Support was provided solely from institutional and department sources.

**References**

1. Dandel M, Lehmkuhl H, Knosalla C, Hetzer R. Left ventricular wall motion abnormality and myocardial dysfunction in stress cardiomyopathy: new pathophysiological aspects suggested by echocardiography. Int J Cardiol 2009; 135: e40-3.
2. Asehnoune K, Larousse E, Tadié JM, Minville V, Droupy S, Benhamou D. Small-dose bupivacaine-sufentanil prevents cardiac output modifications after spinal anesthesia. Anesth Analg 2005; 101: 1512-5. [CrossRef]
3. Mark JB, Steele SM. Cardiovascular effects of spinal anesthesia. Int Anesthesiol Clin 1989; 27: 31-9. [CrossRef]
4. Meyhoff CS, Hesselbjerg L, Køsielniak-Nielsen Z, Rasmussen LS. Biphasic cardiac output changes during onset of spinal anaesthesia in elderly patients. Eur J Anaesthesiol 2007; 24: 770-5. [CrossRef]
5. Rooke GA, Freund PR, Jacobson AF. Hemodynamic response and change in organ blood volume during spinal anesthesia in elderly men with cardiac disease. Anesth Analg 1997; 85: 99-105. [CrossRef]
6. Minville V, Fourcade O, Grousset D, Chassery C, Nguyen L, Asehnoune K, et al. Spinal anesthesia using single injection small-dose bupivacaine versus continuous catheter injection techniques for surgical repair of hip fracture in elderly patients. Anesth Analg 2006; 102: 1559-63. [CrossRef]
7. Butterworth J. Physiology of spinal anesthesia: what are the implications for management? Reg Anesth Pain Med 1998; 23: 370-3. Discussion 384-7.
8. Bijker JB, van Klei WA, Vergouwe Y, Eleveld DJ, van Wolfswinkel L, Moons KG, et al. Intraoperative hypotension and 1-year mortality after noncardiac surgery. Anesthesiology 2009; 111: 1217-26. [CrossRef]
9. Lairez O, Ferre F, Portet N, Marty P, Delmas C, Cognet T, et al. Cardiovascular effects of low-dose spinal anaesthesia as a function of age: An observational study using echocardiography. Anaesth Crit Care Pain Med 2015; 34: 271-6. [CrossRef]
10. Cabrera Schulmeyer MC, Vargas J, la Maza De J, Labbé M. Spinal anesthesia may diminish left ventricular function: a study by means of intraoperative transthoracic echocardiography. Rev Esp Anestesiol Reanim 2010; 57: 136-40. [CrossRef]