

# Haemodynamic effects of central neural blocks

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Haemodynamic effects are the most important physiological responses to central neural blocks. This article is focused on the etiology of hypotension, bradycardia and asystole understanding of which is essential for the anesthesiologist for successful management in the perioperative period. The main causes of cardiovascular changes are decreased cardiac output, systemic vascular resistance and reflexes related to baroreceptors. Hypotension and / or bradycardia are usually of short duration and easily treatable. However, haemodynamic changes in hypovolaemic, elderly patients with comorbidity, and patients with increased catecholamine production, due to excessive alcohol intake, emergency situation, can be significant with worse outcomes. Therefore it is essential to correct hypovolaemia before surgery. Timely notification, identification and appropriate treatment of haemodynamic changes caused by already performed central neural blockade remain important in the perioperative period as well. Vital functions must be monitored throughout surgery so that adverse cardiovascular reactions could be managed with timely and adequate treatment including elevation of the legs, oxygen and infusion therapy, vasoactive and anticholinergic drugs.

**Key words:** haemodynamic effects, central neural blocks, spinal anaesthesia, epidural anaesthesia

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## INTRODUCTION

The cardiovascular changes are the most important physiological responses to central neuraxial blockade: spinal, epidural and combined spinal epidural (CSE) anaesthesia (1, 2). These responses are related with the autonomic denervation and

higher levels of neural blockade, and vagal nerve innervations. Usually, these haemodynamic effects could be considered as normal reactions to central neural blockade (1). However, severe hypotension may have significant outcomes in elderly patients with limited reserve and atherosclerosis, coronary artery disease (3, 15). The incidence of hypotension is reported to occur from 5% to 81% depending on the definition (2, 9). The incidence of bradycardia is registered from 9% to 74%. Kopp and co-authors in 2005 have stated that the incidence of cardiac arrest during neuraxial blockade in twenty-year

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period at the Mayo Clinic was 1.8 per 10 000 patients. Cardiac arrest was defined as severe hypotension, bradycardia or both requiring resuscitation with defibrillation, chest compressions, and / or the use of vasopressors (4). There were more arrests in patients after spinal versus epidural anaesthesia (4). Generally, each anaesthesiologist must have a thorough understanding of the haemodynamic effects of central neural blocks for successful perioperative management of patients undergoing this widely used anaesthesia.

## CAUSE

Sympathetic denervation causes vasodilation of arteries and arterioles with distribution of the central blood volume to the splanchnic circulation and lower extremities (1, 5) and slight decrease in myocardial contractility.

Reduction of cardiac output (CO) and systemic vascular resistance (SVR) are the main causes of significant hypotension (2). In young healthy normovolaemic patients SVR decreases approximately by 15–18% even with significant sympathetic blockade, mean arterial pressure decreases only by 15–18% and CO is maintained normal (1, 2). Preload is an important determinant of CO. CO depends on the patient positioning and remains unaffected if legs are elevated above the level of the heart (1).

In elderly patients SVR decreases by 23–26%, central venous pressure by 2–3 mmHg, and left ventricular end diastolic volume decreases by 20% (2). Therefore, the haemodynamic effects are considerably notable and might be especially harmful to elderly patients with limited cardiac reserve compared with younger patients.

Sympathectomy reduces the venous return to the heart, paradoxically, the vagal tone increases leading to marked bradycardia and asystole (2, 6, 7). The significant decrease in preload may initiate the following three reflexes which cause cardiovascular collapse and syncope (2, 5–7). The first reflex involves direct stretching of the pacemaker cells in the sinoatrial node (2, 5, 7). The decrease in venous return statement causes less stretching and can lead to a drop of heart rate (2, 6, 7). The second reflex includes baroreceptors located in the right atrium and the vena-cava-atrial junction (2, 7).

The third reflex, called the Bezold-Jarisch reflex, is mediated by cardiac baroreceptors located in the inferoposterior wall of the left ventricle (2, 5, 7). Initially it is triggered by decrease of central blood volume, followed by ventricular volume decrease and ventricular contractility increase (2, 5, 7). The vasomotor centre is stimulated via afferents of the vagus nerve, and increased vagal efferent activity leads to bradycardia and vasodilation (2, 5, 6).

Haemodynamic effects during the central neural block depend on the patient's comorbidity, clinical settings, type of surgery, anaesthetic technique.

## DIAGNOSIS AND MONITORING

The clinical symptoms are the most important diagnostic factors. Nausea, vomiting and syncope could be early signs of cardiovascular side effects. The incidence of these symptoms ranges from 9.2 to 2.3% (2). Hypotension is defined as the systolic blood pressure below 85–90 mmHg or a decrease from the baseline level by more than 30% (2, 7, 13). The onset of hypotension is slower in epidural and CSE anaesthesia with more time for compensatory mechanism (6). Bradycardia is regarded as the heart rate below 50 beats per minute (1, 2, 7, 13). A higher incidence of hypotension and bradycardia is associated with the level of the block higher than T5 (2, 6, 13). The electrocardiography, blood pressure, heart rate, oxygen saturation must be monitored during the central neuraxial block.

## MANAGEMENT

The use of 5–10° head down tilt, Trendelenburg position and leg elevation are the first steps in case of low blood pressure. Oxygen is necessary for the essential vital organs: the brain and myocardium. Accordingly, oxygen therapy must be supplied as fast as possible if hypotension develops.

Intravenous fluids prior induction of spinal anaesthesia are mandatory if the patient is hypovolemic. Crystalloids or colloids are effective in attenuation and treatment of hypotension. The preload with 1–2 l of crystalloids is effective, but it should be balanced against the disadvantages and dangers in the individual patient (7). Vasopressors (Ephedrine, Phenylephrine) are used for treatment of hypotension and bradycardia. The bolus (5–10 mg) of ephedrine which acts indi-

rectly and stimulates  $\alpha$ ,  $\beta_1$  and  $\beta_2$  receptors, or as a continuous infusion (50 mg/1000 ml of crystalloid) may be titrated to the desired effect (1, 2). Phenylephrine produces direct stimulation of only  $\alpha$  receptors. The intravenous dose is 50–100  $\mu\text{g}$  (1). Epinephrine produces direct stimulation of more  $\beta$  than  $\alpha$  receptors (1). Epinephrine should be used in case of severe bradycardia, resistant to ephedrine or phenylephrine. Intravenous atropine (0.4–1.0 mg) or glycopyrolate are the first choice in case of bradycardia (1, 2, 7).

## DISCUSSION

Haemodynamic changes are one of the most frequent effects of central neural blocks. Hartman and co-authors reported that the risk factors of cardiovascular effects are obesity, hypertension, and factors related to an increased catecholamine output: alcohol consumption, hypovolaemia, emergency surgery (7). Predictive factors of bradycardia are the use of betablockers, prolonged P-R interval, baseline heart rate below 60 beats per minute (6). Hanss and co-authors reported that a high sympathetic tone was associated with a decrease in blood pressure, and analysis of heart rate variability before spinal anaesthesia can be recommended as a potential noninvasive method for risk stratification of hypotension (10). The degree of the effect depends on the position of the patient's body, the type of surgery, the technique of anaesthesia. Severe hypotension is associated with high risk in case of advanced age, therefore the continuous spinal anaesthesia (CSA) is a well-established technique that has been used successfully in this group of patients. CSA is an effective technique in elderly patients undergoing lower extremity surgery (3, 14). Imbelloni and co-workers found that the CSA technique compared with the CSE block is safer for high-risk patients with unstable haemodynamics because the injected dose of a local anaesthetic is lower, it gives better control of the level of blockade and shorter duration of arterial hypotension (14).

## CONCLUSIONS

Haemodynamic effects are frequent physiological responses to central neural blockade caused by various mechanisms, but the most important reason is decreased venous return, and both prevention

and treatment are aimed at preserving or restoring adequate venous return. Consequently, correction of preoperative hypovolaemia, elevation of the legs, intravenous fluid infusion are the first actions which improve venous return and help to manage hypotension and/or bradycardia.

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## References

1. Cousins MJ, Carr DB, Horlocker TT, Bridenbaugh PO. Cousins and Bridenbaugh's Neural Blockade in Clinical Anesthesia and Pain Medicine. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2009.
2. Salinas FV, Sueda LA, Liu SS. Physiology of spinal anaesthesia and practical suggestions for successful spinal anaesthesia. *Best Pract Res Clin Anaesthesiol.* 2003; 17(3): 289–303.
3. Veering BT. Haemodynamic effects of central neural blockade in elderly patients. *Can J Anaesth.* 2006, 53(2): 117–21.
4. Kopp SL, Horlocker TT, Warner ME, Hebl JR, Vachon CA, Schroeder DR, et al. Cardiac arrest during neuroaxial anesthesia: Frequency and predisposing factors associated with survival. *Anesth Analg.* 2005; 100: 855–65.
5. Campagna JP, Carter C. Clinical relevance of the Bezold-Jarisch reflex. *Anesthesiology.* 2003; 98: 1250.
6. Stienstra R. Mechanisms behind and treatment of sudden, unexpected circulatory collapse during central neuraxis blockade. *Acta Anaesthesiol Scand.* 2000; 44: 965–71.
7. Hartmann B, Junger A, Klasen J, Benson M, Jost A, Banzhaf A, Hempelmann G. The incidence and risk factors for hypotension after spinal anesthesia induction: an analysis with automated data collection. *Anesth Analg.* 2002; 94: 1521–9.
8. Schmidt C, Hinder F, Van Aken H, Theilmeyer G, Bruch C, Wirtz SP, et al. The effect of high thoracic epidural anesthesia on systolic and diastolic left ventricular function in patients with coronary artery disease. *Anesth Analg.* 2005; 100: 1561–9.
9. Klasen J, Junger A, Hartmann B, Benson M, Jost A, Banzhaf A, et al. Differing incidences of relevant

- hypotension with combined spinal-epidural anesthesia and spinal anesthesia. *Anesth Analg.* 2003; 96: 1491–5.
10. Hanss R, Bein B, Weselch H, Bauer M, Cavus E, Steinfath M, et al. Heart rate variability predicts severe hypotension after spinal anesthesia. *Anaesthesiology.* 2006; 104: 537–45.
  11. Donati A, Mercury G, Iuorio S, Sinkovetz L. Haemodynamic modifications after unilateral subarachnoid anaesthesia evaluated with transthoracic echocardiography. *Minerva Anesthesiol.* 2005; 71: 75–81.
  12. Casati A, Fanelli G, Berti M, Beccaria P, Agostoni M, Aldegheri A, Torri G. Cardiac performance during unilateral lumbar spinal block after crystalloid preload. *Can J Anaesth.* 1997; 44(6): 623–8.
  13. Gudaityte J, Marchertiene I, Karbonskiene A, Saladzinskas Z. Low-dose spinal hyperbaric bupivacaine for adult anorectal surgery: a double-blinded, randomized, controlled study. *J Clin Anesth.* 2009; 21: 474–81.
  14. Imbelloni LE, Gouveia MA, Cordeiro JA. Continuous spinal anesthesia versus combined spinal epidural block for major orthopedic surgery: prospective randomized study. *Sao Paulo Med J.* 2009; 127(1): 7–11.
  15. Ledowski T, Preus J, Kapila R, Ford A. Skin conductance as a means to predict hypotension following spinal anaesthesia. *Acta Anaesthesiol Scand.* 2008; 52: 1342–7.

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## HEMODYNAMIKOS POKYČIAI CENTRINĖS NERVŲ BLOKADOS METU

### *Santrauka*

Hemodinamikos pokyčiai centrinės nervų blokados metu yra svarbiausias fiziologinis atsakas. Šis straipsnis sukoncentruotas į hipotenzijos, bradikardijos ir asistolijos etiologiją, kurią anesteziologas privalo suprasti nepriekaištingai, kad galėtų sėkmingai valdyti situaciją perioperaciniu periodu. Pagrindinės širdies ir kraujagyslių pokyčių priežastys yra širdies minutinio tūrio ir sisteminio kraujagyslių pasipriešinimo sumažėjimas, taip pat su baroreceptoriais susiję refleksai. Hipotenzija ir/ar bradikardija dažniausiai būna trumpalaikė ir lengvai koreguojama. Esant vyresnio amžiaus pacientų, sergančių ir kitomis ligomis, hipovolemijai, padidėjus katecholaminų gamybai dėl gausaus alkoholio vartojimo, hemodinamikos pokyčiai ūminių būklių metu yra dažnesni ir ženklesni, ir tai blogina išėitis, todėl hipovolemijos korekcija prieš operaciją yra svarbi. Perioperaciniu periodu taip pat svarbu laiku pastebėti, nustatyti bei atitinkamai koreguoti pokyčius, atsiradusius atlikus centrinę nervų blokadą. Operacijos metu turi būti stebimos gyvybinės funkcijos, o atsiradus sutrikimams būtina laiku atlikti adekvatų gydymą: pakelti apatines galūnes, taikyti deguonies terapiją, infuzinę terapiją, skiriant vazoaktyvius ir anticholonerinius medikamentus.

**Raktažodžiai:** hemodinamikos pokyčiai, centrinė nervų blokada, spinalinė anestezija, epidūrinė anestezija