Spinal-induced hypotension at caesarean section

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Abstract

Spinal anaesthesia is preferred for caesarean section, yet hypotension remains a significant problem and may adversely affect mother and baby. Understanding the physiological causes is essential to direct management. The international consensus of management includes vasopressors, intravenous fluids and avoidance of aorto-caval compression. Phenylephrine by infusion is now the vasopressor of choice. Low-dose spinal anaesthesia can reduce the incidence but risks inadequate anaesthesia. Novel means of predicting or more rapidly detecting spinal-induced hypotension include the use of continuous non-invasive blood pressure and cardiac output monitoring devices. Computer-aided closed-loop feedback systems with automated delivery of vasopressors permit timely treatment but remain outwith clinical practice.

Keywords Anaesthesia; caesarean; ephedrine; hypotension; noradrenaline; phenylephrine; spinal; vasopressors

Royal College of Anaesthetists CPD Matrix: 2B04

Learning objectives

After reading this article, you should be able to:

- define spinal-induced hypotension during caesarean section
- describe the mechanisms behind spinal-induced hypotension
- identify mothers and babies at particular risk of spinal-induced hypotension and its effects.
- outline pharmacological strategies for preventing and treating hypotension
- describe non-pharmacological methods to prevent hypotension

The problem

Spinal anaesthesia is the preferred choice for caesarean section but commonly associated with hypotension. This often results in maternal adverse effects, including nausea, vomiting, syncope and dyspnoea, which are unpleasant and detract from the birth experience. Understanding of the neonatal adverse effects secondary to spinal-induced hypotension is relatively limited but both duration and severity seem to be important. Maternal hypotension lasting more than 2 minutes is associated with fetal umbilical acidosis, and more than 4 minutes with neurobehavioural changes at 4–7 days of life. Some babies may be more vulnerable than others; a French study found association between spinal anaesthesia and increased neonatal mortality in early preterm babies delivered by caesarean section.[1] Also, in mothers suffering from chronic hypertension, spinal anaesthesia has been associated with a decreased uterine artery pulsatility index, indicating uterine blood flow vulnerability.[2] It seems that when there is fetal compromise, uteroplacental perfusion is less tolerant of maternal hypotension.

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Definition

The 2018 International consensus statement defines hypotension as $< 80\%$ of baseline systolic blood pressure (SBP). It recommends maintaining SBP at $\geq 90\%$ of an accurate baseline reading obtained before spinal anaesthesia.[3]

The mechanism of hypotension after spinal anaesthesia

Previously, caval compression and venodilation causing decreased venous return and cardiac output were thought to be the dominant mechanisms. Multiple studies have now demonstrated that the reduction in systemic vascular resistance (SVR) due to small artery vasodilation induced by sympathetic block is more significant.[4] As SVR decreases, the heart rate (HR) and stroke volume increase through baroreceptor-mediated response via the sympathetic cardiac accelerator fibres. Cardiac output (CO) will therefore increase, but this is usually insufficient to maintain SBP. Spinal block levels affecting T1–T4 cardiac accelerator fibres can impair the compensatory increase in the HR and CO, although HR does not always correlate with the block height as sometimes sudden bradycardia can occur secondary to vasovagal activation (Bezold–Jarisch reflex).[3]

Predicting occurrence of hypotension

Identifying mothers with higher risk of developing hypotension would allow more targeted monitoring and therapy. The known risk factors are listed in Box 1.

Supine hypotensive syndrome occurs in susceptible patients suffering from exaggerated caval compression. This is tested by observing changes in the HR and SBP measured in both supine and lateral position. It is defined as a decrease in the systolic blood pressure of at least 15–30 mmHg in the supine position. The severity of hypotension is greater in women with a positive test.[4]

Other predictive tools have been investigated. Zieleskiewicz et al. used cardiac ultrasound measured subaortic variation in the velocity time integral after passive leg raising. This predicted both severity of hypotension and fluid responsiveness.[5] Berlac and Rasmussen studied cerebral oxygenation (ScO_2) using cerebral near-infrared spectroscopy. They showed that $\geq 5\%$ decrease in ScO_2 preceded the occurrence of hypotension.[6] These

Risk factors for developing hypotension following spinal anaesthesia in the obstetric population[3,7]

- Absence of labour [a]
- High pre-operative anxiety
- High baseline heart rate (>80–90 beats per minute)
- High heart rate variability
- Recent history of supine intolerance/supine hypotensive syndrome
- Peak sensory block height > T4–T6

[a]During uterine contractions up to 300 ml are shifted into the central circulation. This auto-transfusion may protect mothers from the hypotensive effects of spinal anaesthesia.

Box 1

are small single centre studies and these tools are currently neither widely available nor practical to use outside the research setting.

Management

Based on our understanding of the mechanism of spinal induced hypotension, maintaining SVR, venous tone/capacitance and venous return are the principle approaches.

Non-pharmacological

Prevention of vena caval compression and lower limb venous pooling: A 15° left lateral tilt or left manual displacement of uterus is associated with higher SBP, CO and reduced vasopressor requirements than supine positioning. In practice such tilt is rarely achieved as it can make operating difficult, but should be applied in the preparation phase.[3]

Leg compression is associated with less hypotension when compared with no compression, but no device is superior. Venous compression devices and leg elevation are not of proven benefit.[3]

IV fluid loading: No IV fluid regimens are effective alone in preventing spinal induced hypotension but are important adjunct to improve the haemodynamic stability provided by vasopressor therapy. The main findings from numerous fluid loading studies are that a 500 ml colloid pre-load reduces incidence and severity of hypotension.[8] A 1000 ml crystalloid co-load (rapid infusion after induction of spinal) is also as effective and avoids safety concerns associated with colloids.[3,8]

Pharmacological management

Phenylephrine versus ephedrine: Phenylephrine has a pure direct agonist effect on α 1 receptors with immediate onset and intermediate duration of action. Currently it is the vasopressor of choice for prevention and management of spinal mediated hypotension. Ephedrine is an agonist at alpha 1, beta 1, beta 2 receptors with both direct and indirect actions. It was traditionally used on the basis of animal studies that indicated more favourable uteroplacental flow. Ephedrine, however, is less efficacious, slower in onset, has greater placental transfer and is associated with a fivefold increased risk of fetal acidosis determined by umbilical artery gas analysis. This acidosis is due to beta agonist effects in the fetus including increased carbon dioxide and lactate production. This difference between the two vasopressors was evident during elective caesarean sections but not in the emergency ones.[3]

Continuous infusion of phenylephrine starting immediately after spinal anaesthesia induction is superior at preventing hypotension, nausea and vomiting when compared to intermittent rescue boluses treatment. It also reduces the workload of the attending anaesthetist.

Phenylephrine does cause a reduction in maternal heart rate and cardiac output although this without adverse effect on the fetus. Evidence suggests that the dose 25–50 mcg/min maintains maternal haemodynamic stability without compromizing cardiac output although additional boluses may be required.

Box 2 shows a suggested protocol for management of spinal hypotension during caesarean section using vasopressors.[3]

Noradrenaline: A strong α-1 and modest β-1 agonist with immediate onset and short duration of action, it is potentially the ideal vasopressor since the β-1 effect maintains maternal HR and CO stability without adverse fetal effects. The dose ratio of noradrenaline to phenylephrine is suggested to be 1:17.[9] Its use still mostly limited to clinical trials using dilute concentration (6 μg/ml) delivered with running IV fluid into a large peripheral vein. While a potential alternative to phenylephrine there are safety concerns about preparing and handling such a potent agent outside the critical care environment.

Metaraminol is a mixed α-1 and weak β-1 agonist. It has both direct and indirect effects and prolonged use shows tachyphylaxis. The dose ratio of metaraminol to phenylephrine is suggested to be 5:1. While not inferior to phenylephrine, obstetric use is limited and there is a tendency to more hypertensive episodes.[3]

Ondansetron: Prophylactic 5HT3 antagonist ondansetron is associated with less hypotension, bradycardia, nausea and

After spinal injection

- Set NIBP measurement to 1 minute cycles
- Start rapid infusion 1000 ml warmed crystalloid
- Start IV phenylephrine infusion at rate of 25–50 $\mu g.min^{1}$
- Ensure uterine displacement from vena cava
- Titrate infusion to SBP response adjusting the infusion rate by 5–10 $ml.hour^{-1}$.
- Add IV bolus of 50–100 μg if SBP <80% baseline
- Use boluses of ephedrine 3–6 mg to treat bradycardia.
- Use glycopronium 200 μg to treat bradycardia if SBP< 80% baseline
- Between NIBP measurements the heart rate can be used as a surrogate for cardiac output and infusion rate adjusted to keep it > 60 beats/min
- Wean phenylephrine infusion after delivery
- Beware hypotensive effects of oxytocic drugs and concealed haemorrhage

Box 2

vomiting after spinal anaesthesia. The mechanism is not fully understood but might relate to inhibition of the vasovagal Bezold–Jarisch reflex thus maintaining stable CO.[10]

Others

Anaesthetic technique: The degree of spinal induced hypotension is dependent on the intrathecal local anaesthetic dose. Low dose spinal (<8 mg bupivacaine) is associated with a lower incidence of hypotension and maternal symptoms but reducing the dose is associated with a higher incidence analgesic supplementation during surgery, longer onset time, shorter duration and greater postoperative analgesia requirements. Combination of low doses with opioids and use of CSE techniques can offer a better balance of analgesia and haemodynamic stability.[11]

Pre-eclampsia: Sympathetic blockade produces a modest fall in SVR and blood pressure in women with pre-eclampsia, but only minimal reduction in CO. The requirement for blood pressure support in this group is usually low. Vasopressor infusion should be commenced at reduced rates and fluid loading cautious.

Future directions: Computer-controlled closed-loop feedback vasopressor infusion systems, using non-invasive or minimally invasive continuous blood pressure and CO monitoring to maintain a target have been developed in clinical trials. These may become cost effective and practical for clinical use. Also, they may involve the use of combined drug infusion systems, e.g. phenylephrine and ephedrine. The ability to reliably predict the onset of hypotension using readily measurable parameters would be welcomed.[3] ◆

REFERENCES

1 Laudenbach V, Mercier F, Roze J-C, et al. Anaesthesia mode for caesarean section and mortality in very preterm infants: an epidemiologic study in the EPIPAGE cohort. *Int J Obstet Anesth* 2009; **18:** 142–9.

2 Guedes-Martins L, Graç a H, Saraiva JP, et al. The effects of spinal anaesthesia for elective caesarean section on uterine and umbilical arterial pulsatility indexes in normotensive and chronic hypertensive pregnant women: a prospective, longitudinal study. *BMC Pregnancy Childbirth* 2014; **14:** 291.

3 Kinsella SM, Carvalho B, Dyer RA, et al. International consensus statement on the management of hypotension with vasopressors during caesarean section under spinal anaesthesia. *Anaesthesia* 2018; **73:** 71–92.

4 Bishop DG, Cairns C, Grobbelaar M, Rodseth RN. Heart rate variability as a predictor of hypotension following spinal for elective caesarean section: a prospective observational study. *Anaesthesia* 2017; **72:** 603–8.

5 Zieleskiewicz L, Noel A, Duclos G, et al. Can point-of-care ultrasound predict spinal hypotension during caesarean section? A prospective observational study. *Anaesthesia* 2018; **73:** 15–22.

6 Berlac PA, Rasmussen YH. Per-operative cerebral near-infrared spectroscopy (NIRS) predicts maternal hypotension during elective caesarean delivery in spinal anaesthesia. *Int J Obstet Anesth* 2005; **14:** 26–31.

7 Bishop DG. Predicting spinal hypotension during Caesarean section. *South Afr J Anaesth Analg* 2014; **20:** 170–3.

8 Middleton P, Chooi C, Cyna AM, et al. Techniques for preventing hypotension during spinal anaesthesia for caesarean section. *Cochrane Database Syst Rev* 2017; **8**.

9 Ngan-Kee WD. The use of vasopressors during spinal anaesthesia for caesarean section. *Curr Opin Anaesthesiol* 2017; **30:** 319–25.

10 Liu Q, Wang X, Zhu Y, et al. Efficacy of ondansetron for spinal anesthesia during cesarean section: a meta-analysis of randomized trials. *J Int Med Res* 2017; **46:** 654–62.

11 Arzola C, Wieczorek PM. Efficacy of low-dose bupivacaine in spinal anaesthesia for Caesarean delivery: systematic review and meta-analysis. *Br J Anaesth* 2011; **107:** 308–18.