Prevention and treatment of cardiovascular instability during spinal anaesthesia for caesarean section

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Spinal anaesthesia is the method of choice for caesarean section. There is however a significant associated morbidity and mortality in South Africa, particularly in inexperienced hands. This review provides recommendations for safe practice for anaesthetists at all levels of expertise, with particular reference to the management of haemodynamic instability.

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There has been a recent major increase in the use of regional techniques in obstetric anaesthesia and a marked reduction in the number of anaesthesia-associated maternal deaths.[1] The most significant change in technique has been the adoption of spinal anaesthesia for caesarean section.[2] Since the first Confidential Enquiry into Maternal Deaths (CEMD) was performed (1952 - 1954), the decreased mortality associated with obstetric anaesthesia in the UK has been due to both the adoption of spinal anaesthesia as the preferred method, and to safer general anaesthesia. The development of obstetric anaesthesia as a subspecialty, and the introduction of strict supervision of junior anaesthetists, have also influenced obstetric anaesthesia safety.

In the USA, regional anaesthesia for caesarean section has been shown to be associated with a lower case fatality rate than general anaesthesia.[3] Nevertheless, spinal anaesthesia does have an associated morbidity and mortality, although mortality is rare.

In South Africa, the National Committee for the Confidential Enquiries into Maternal Deaths (1999 - 2001) reported that although more deaths were associated with general anaesthesia, there was a significant mortality associated with spinal anaesthesia for caesarean section. During this period 25 patients died under spinal anaesthesia, with little or no co-morbidity in most cases.[4]

Reasons for high morbidity and mortality associated with spinal anaesthesia in South Africa may include: *(i)* inadequate experience and training of the anaesthetist; *(ii)* inappropriate use of the technique in mothers with significant co-morbidity; *(iii)* the performance of both anaesthesia and surgery by the same practitioner; and *(iv)* neonatal resuscitation by the anaesthetist at the expense of dedicated care to the mother.

Many problems are avoidable or amenable to treatment by attention to details of safe practice, hence the provision of these detailed recommendations, intended to supplement the document on spinal anaesthesia issued by the Department of Health.[5] It is not implied that spinal anaesthesia is unsafe, or that general anaesthesia is preferable for caesarean section. The case fatality for general anaesthesia is probably even higher in relatively inexperienced hands, and spinal anaesthesia has other benefits, such as a lower morbidity, less blood loss and earlier bonding between mother and baby.

Prevention — pre-operative preparation

Safe practice of spinal anaesthesia includes both prevention of and timely intervention in the event of cardiovascular instability. Despite a prior obstetric or general medical work-up, the patient must be reassessed from an anaesthetic standpoint before intended surgery, especially in busy peripheral hospitals where the 'obstetrician' and 'anaesthetist' may have interchangeable roles.

Important in the anaesthetic assessment are a history of anaesthetic difficulties, co-morbid conditions, and documentation of medications and allergies. Progressive breathlessness on exertion could denote cardiac impairment. The patient's airway must be assessed, the chest auscultated, and the cardiovascular system examined, including recording of the pre-operative heart rate and blood pressure. Signs of volume depletion and specific contraindications to spinal anaesthesia must be sought (Table I).

The main advantages of spinal anaesthesia are the maintenance of normal protective airway reflexes, and avoiding tracheal intubation. However, airway reflexes may be lost in the event of a major complication, requiring emergency

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Table I. Absolute contraindications to spinal anaesthesia

Absent resuscitation drugs or equipment
Hypovolaemia
Cardiovascular co-morbidity, e.g. mitral stenosis or aortic stenosis
Coagulation abnormalities and/or thrombocytopenia ($< 75 \times 10^9$/l)
Depressed level of conciousness (Glasgow Coma Scale < 14)
Significant sepsis in the area of injection, or severe systemic sepsis with cardiovascular instability
Allergy to local anaesthetics

intubation. Even when spinal anaesthesia is planned, the airway must be carefully examined, oral intake restricted to clear fluids only during labour, appropriate pre-operative antacid prophylaxis provided, and the operating theatre equipment checked and prepared as for emergency general anaesthesia.

The two main life-threatening complications of spinal anaesthesia are severe cardiovascular instability in the form of catastrophic hypotension, and high motor neuronal blockade. As spinal anaesthesia is associated with cardiovascular instability, any pre-existing instability makes the procedure particularly hazardous. Pre-existing cardiovascular instability commonly results from haemorrhage or maternal cardiac disease.

Ongoing significant obstetric haemorrhage contraindicates regional anaesthesia, even if the patient is well resuscitated. However, if haemorrhage has been minimal, or was significant but has ceased and is corrected, spinal anaesthesia may be used. Attention must be paid to the probability of severe operative blood loss. Regional anaesthesia for caesarean section is employed in a significant percentage of patients with placenta praevia. However, caution should be exercised in the presence of grade 3 or 4 placenta praevia. Furthermore, placenta praevia in patients who have had a previous caesarean section is often associated with placenta accreta. The associated significant operative haemorrhage usually contraindicates the use of spinal anaesthesia, particularly if the placenta is anterior. In prolonged obstructed labour, ruptured uterus is a possibility and can be associated with torrential bleeding. Pre-existing maternal tachycardia and acute onset of fetal heart rate decelerations are ominous signs of uterine rupture.

The possibility of occult haemorrhage must be recognised, for example in cases of abruptio placentae. In the setting of prolonged neglected labour, signs of dehydration should be elicited.

Tables II and III list essential drugs, equipment and monitors required before the induction of spinal anaesthesia. Capnography is the best monitor of correct endotracheal tube placement, should this be necessary, and is a useful indicator of cardiac output.

Table II. Resuscitation drugs

Immediately available (drawn up)
Ephedrine 5 mg/ml and phenylephrine 50 µg/ml
Atropine 0.5 mg
Readily available (on the anaesthetic cart)
Adrenaline
Suxamethonium

Table III. Essential equipment and monitoring

The following should be immediately available in theatre before spinal anaesthesia is commenced:
An obstetric wedge to tilt the patient's pelvis 15° - 30° from horizontal
Anaesthetic machine with breathing circuit attached and checked for leaks
An appropriately sized anaesthetic face mask
A selection of oropharyngeal airways
A nasopharyngeal airway
Two working laryngoscopes with choice of blades
Cuffed oral tracheal tubes, sizes 5 - 7 mm
An introducer more than twice the length of the tracheal tube
Separate oxygen supply for patient mask or nasal cannulae
Self-inflating bag for assisted ventilation
Laryngeal masks (sizes 3 and 4)
Suction apparatus with both Yankauer and flexible suction catheters
A defibrillator should be available within the operating suite and checked daily for adequate charge and availability of electrode gel
Essential monitoring:
ECG
Pulse oximetry
Capnography
Non-invasive blood pressure

Pathophysiology of haemodynamic instability

Spinal anaesthesia for caesarean section is associated with extensive sympathetic blockade, since sensory block to a minimum level of T4 is required for adequate surgical anaesthesia, and the associated sympathetic block is usually at least two segments higher. Efferent activity in cardio-accelerator fibres (T1 - T4) may therefore also be inhibited. There is also an increased risk due to compression of the aorta and inferior vena cava by the gravid uterus. Aortocaval compression, although only causing symptoms in 3% of pregnant women, often leads to decreased blood supply to the uterus and reduced maternal venous return and cardiac output. Under spinal anaesthesia this may precipitate catastrophic hypotension. The normal physiological responses to hypotension are a baroreceptor-mediated increase in heart rate, and venous and arterial constriction, which produce a compensatory increase in cardiac output and a return of blood

pressure towards normal. All these mechanisms are impaired by spinal anaesthesia. Occasionally precipitous bradycardia may arise if there is sudden underfilling of the left ventricle. An explanation may be the activation of the Bezold-Jarisch reflex. This involves activation of C-fibres in the wall of the left ventricle, reflex activation of the vagus nerve and activation of sympathetic vasodilator efferent fibres to the muscle beds.[6] Such acute underfilling of the heart may occur during spinal anaesthesia for caesarean section, when rapid onset of sympathetic blockade is additive with aortocaval compression. As vagal reflexes remain intact the resulting bradycardia may be profound and cause a marked decrease in cardiac output. Without prompt appropriate intervention, cardiac arrest may follow.

The following co-morbid conditions may cause haemodynamic instability:

Morbid obesity. Despite difficulty in anaesthetic monitoring, and a degree of unpredictability of the level of spinal block, it remains safer to use spinal than general anaesthesia in these patients.

Valvular heart disease. Spinal anaesthesia is contraindicated in conditions that impair the cardiovascular response to sympathetic blockade, such as mitral or aortic stenosis, since cardiovascular collapse may occur.

Pre-eclampsia. Spinal anaesthesia is safe provided that the patient is optimally hydrated and has no coagulopathy.

HIV/AIDS. Patients with severe sepsis may have cardiovascular instability, but in those with respiratory disease, spinal anaesthesia is often safer. Spinal anaesthesia does however reduce FEV_1 (forced expiratory volume in 1 second) and vital capacity by approximately 25% in healthy parturients, and may therefore precipitate respiratory failure in patients with severe pre-operative respiratory compromise.[7]

Acute fetal compromise. This often requires the rapid administration of spinal anaesthesia, so that maternal conditions contraindicating the use of spinal anaesthesia may be missed.

Diagnoses may be missed or may be incorrect in busy units with a high incidence of maternal co-morbidity. Anaesthetists must rely on their own clinical skills and judgement but if in difficulty or doubt, telephonic advice from regional academic centres should be sought.

Management of haemodynamic instability

Intravenous access should be established using at least an 18G cannula, but at least one 14 or 16G cannula is essential if major blood loss is anticipated. The following aspects of technique in the performance of spinal anaesthesia contribute to the prevention or treatment of haemodynamic instability.

Subarachnoid bupivacaine

The correct dose and volume of subarachnoid bupivacaine should be injected at the appropriate level. Most units administer 9 - 12 mg of hyperbaric 0.5% bupivacaine (1.8 - 2.4 ml) with 10 - 20 µg of fentanyl at the L3/4 interspace, using a 25G pencil point spinal needle. Injection at or above L2/3 may rarely result in damage to the conus medullaris of the spinal cord, with the development of a syrinx and permanent neurological injury.[8]

Oxygen supplementation

Maternal oxygen supplementation using a 40% mask is routine practice in some units although it is probably unnecessary in the healthy parturient delivering a healthy term infant. Markers of oxygen free radicals may be raised in neonates whose mothers had received 60% oxygen,[9] but there is no hard evidence that 40% oxygen supplementation is harmful. It might be beneficial in conditions associated with placental insufficiency (for example pre-eclampsia), in parturients who are morbidly obese, in those with cardiac or respiratory co-morbidity, or in the presence of a non-reassuring fetal heart trace. If there is concern about maternal or neonatal wellbeing, as in maternal hypoxia, severe pre-eclampsia or a fetal heart trace suggestive of severe asphyxia, administration of 100% oxygen via the anaesthesia breathing circuit is advisable, at least before delivery. This would ensure adequate maternal pre-oxygenation should general anaesthesia be required, and could improve umbilical venous oxygen content. Umbilical venous content is significantly higher during general anaesthesia for caesarean section when 100% oxygen is administered than with 50% oxygen.[10]

Intravenous fluids

Delays for the administration of crystalloid preload are not justified in the normovolaemic patient, since the incidence of hypotension remains high despite the administration of large volumes of crystalloid.[11] Since crystalloid is rapidly redistributed into the extravascular space, colloidal solutions have been investigated as an alternative. However, large volumes of colloid are required to produce a sustained rise in blood volume and hence cardiac output.[12] Also, colloids may rarely be associated with allergy (responsible for at least one maternal death in South Africa), and the solutions are expensive. Therefore colloids are not widely recommended for this purpose.

No cases of pulmonary oedema have been reported after the rapid administration of 10 - 20 ml/kg of a crystalloid solution in healthy parturients before spinal anaesthesia for caesarean section.[13] Therefore, the best clinical approach, when intravenous fluids are used for this purpose, is to administer 20 ml/kg of crystalloid (glucose-free saline or an isotonic balanced salt solution) rapidly in the case of the healthy

parturient, at the time of induction of spinal anaesthesia. This maximises intravascular volume expansion during vasodilatation from the sympathetic blockade, and limits the effect of fluid redistribution. This practice is supported by studies in non-obstetric[14] and obstetric patients.[15]

Position

To eliminate the dramatic hypotensive effect of aortocaval compression under spinal anaesthesia, as much as 34° of lateral tilt may be required.[16] In practice, 15° of lateral tilt, or an obstetric wedge placed under the right buttock is used, but if this is insufficient and severe hypotension develops after the induction of spinal anaesthesia, the uterus can be displaced manually or the patient placed in the lateral position. If hypotension is intractable, the rapid delivery of the fetus will aid resuscitative efforts and may be life-saving for the mother.

Spinal anaesthesia may be performed in the sitting or the lateral position. The sitting position is technically easier, while the lateral position has the advantage of being able to restrict the upper level of the block. The thoracic kyphosis which usually restricts cephalad spread of local anaesthetic, and which is eliminated in the lateral position, may be recreated by the use of a support under the shoulder, neck and head. Vasopressor requirements are reduced using this technique, but it is more time-consuming. Patients with a history suggestive of severe aortocaval compression when supine benefit from using the lateral position, as the onset of the block is slower. The block is best performed in the right lateral position; the patient is then positioned supine with left lateral tilt, which prevents the development of a unilateral block.

Monitoring

Despite the above manoeuvres, many patients will develop significant hypotension, which may be extremely rapid, after induction of spinal anaesthesia, and it may take a minute or so before any administered vasopressor can take effect. Measuring the blood pressure every minute for at least 10 minutes or until delivery is therefore mandatory. In obese patients, where rapid blood pressure measurement by automated devices is often difficult, a quick estimation of the blood pressure can be obtained by palpation of the radial artery or noting the disappearance of the pulse oximetry wave form while observing the cuff pressure during the inflation phase. Thereafter, blood pressure measurement intervals should be maintained at 2 - 3 minutes. An automated non-invasive blood pressure monitor is an obligatory requirement in obstetric theatres. Other minimal essential monitoring is listed in Table III.

Clinical signs such as a palpable decrease in pulse volume, nausea and vomiting, and a sudden decrease in level of consciousness, are invaluable early warning signs of a marked decrease in cardiac output. Direct contact and communication with the patient throughout the procedure is of paramount importance, since premonitory symptoms may appear before electronic devices detect a change.

Vasopressors

Vasopressors should be available and drawn up ready for use. It is important that vasopressor use should be standardised throughout the country, and that all practitioners of obstetric anaesthesia be familiar with these agents. Ephedrine, phenylephrine and epinephrine (adrenaline) should be available in all units performing caesarean section.

The standard first line and very safe vasopressor is ephedrine, a mixed α-β adrenergic agonist with direct and indirect actions. Its prophylactic use may be advantageous in certain circumstances, but can cause unnecessary tachycardia and hypertension if routinely administered. However, normotensive patients at high risk for hypotension, such as the morbidly obese, diabetics with a high estimated fetal weight, or patients with a twin pregnancy, justify the administration of 5 - 10 mg of ephedrine immediately after induction of spinal anaesthesia. Ephedrine is available in 50 mg ampoules (1 ml) and should be diluted to 10 ml (5 mg/ml) before use. A reasonable approach in parturients without cardiovascular co-morbidity, is to administer ephedrine in 5 mg intravenous (IV) boluses if the systolic blood pressure decreases by 20%, or if the absolute value of the systolic pressure decreases to less than 100 mmHg, and to continue such boluses every minute until the blood pressure recovers to within 20% of the starting pressure, or to 100 mmHg. Boluses of 10 mg may be administered if the systolic pressure decreases by 30%. In cases where the heart rate decreases to less than 50 beats per minute in association with hypotension, atropine 0.5 - 1.0 mg also may be given IV immediately following the ephedrine (glycopyrrolate is much less reliable in effectively restoring heart rate in the setting of precipitous bradycardia). In the case of marked vagotonia, ephedrine is a more logical choice than the pure α-agonist phenylephrine, which may be associated with baroreceptor-mediated slowing of the heart rate.[17]

Phenylephrine is available in 10 mg ampoules (1 ml). This would constitute a potentially fatal overdose, and it should be diluted to 50 micrograms (μg) per millilitre before use. The safest method is to dilute 10 mg in 200 ml normal saline. The resulting solution can be withdrawn into a 10 ml syringe, available for immediate use.

If the patient has a marked tachycardia at the outset, or following the use of a cumulative dose of 25 mg of ephedrine, phenylephrine in boluses of 50 μg IV is very effective. In preserving uterine blood flow, α-agonists are as effective as ephedrine.[18] The venous capacitance vessels are more susceptible to low concentrations of α-agonists than the arterioles; hence small doses of phenylephrine may improve preload and hence cardiac output, by reversing spinal

anaesthesia-induced venodilatation and mesenteric vasodilatation. Phenylephrine is also very effective by continuous infusion (0.05 - 1 µg/kg/min), since the elimination half-life is only a few minutes. Ephedrine is associated with a greater umbilical arterial base deficit than phenylephrine,[19] which may be due to a β-adrenergically mediated increase in neonatal metabolic rate secondary to ephedrine, which crosses the placenta. It is therefore reasonable to restrict the total ephedrine dose to about 25 mg unless there is a precipitous bradycardia, and to continue with phenylephrine as described. Since the clinical neonatal outcome is not worse after the use of ephedrine, it currently remains the first-line agent in most units.

Adrenaline is not routinely used as an agent to prevent or treat spinal hypotension. Following a bolus dose its effect is brief and it is associated with cardiac arrhythmias. Because its use may be life-saving for cases of refractory hypotension, it should be rapidly available in theatre if not drawn up beforehand.

Oxytocin

Oxytocin administered as a bolus causes large increases in heart rate and cardiac output 1 minute after a 5 or 10 IU bolus, and significant hypotension follows a 10 IU bolus.[20] Some women with hypovolaemia or cardiac disease may be unable to mount compensatory responses, and are at risk of haemodynamic collapse, as illustrated by a reported maternal death.[1] Therefore, the initial oxytocin dose (5 IU) should be administered by slow injection in the healthy parturient, or by continuous infusion only in the unstable patient. This may be followed by a slow infusion (60 - 100 ml/hour) of oxytocin 20 IU/l. In cases of persistent uterine hypotonia, a further 5 IU bolus may be given slowly over at least 1 minute.

Pre-eclampsia and spinal anaesthesia

In the pre-eclamptic patient with acceptable coagulation and platelet count > 75×10^9/l, spinal anaesthesia is safe, provided that fluid balance has been adjusted and effective vasodilator therapy commenced before caesarean section. Perioperative intravenous fluids should be restricted to 10 ml/kg in the absence of haemorrhage, since there is a risk of pulmonary oedema in patients given excessive fluids, after regression of the block. Pre-eclamptic patients do not exhibit more marked cardiovascular responses to conventional doses of vasopressors than normal parturients, under spinal anaesthesia.[21,22] Severe pre-eclamptics may exhibit less hypotension during spinal anaesthesia than healthy parturients.[23] If hypotension does occur, it has a delayed onset. Hypotension should be treated according to similar guidelines as in normal parturients, with the additional recommendation that vasopressors should not be given while the mean arterial pressure remains above 100 mmHg. Ergometrine is best avoided in pre-eclampsia, since it may cause severe hypertension.

In pre-eclamptics with a non-reassuring fetal heart trace, spinal anaesthesia has been found to be associated with acceptable pulse and blood pressure changes, although it may be associated with a greater neonatal umbilical arterial base deficit and lower pH than general anaesthesia.[24]

Pathophysiology of high motor block

The requirement for caesarean section is for a sensory level of T4, but the use of a single-shot spinal technique is unpredictable, and some patients will have a higher sensory block. If an accompanying motor blockade extends above the 6th cervical level, diaphragmatic weakness can occur and respiratory arrest may follow, with subsequent hypoxic cardiac arrest if prompt action is not taken.

Management of high motor block

High spinal (motor) block is a rare complication of spinal anaesthesia, with a reported incidence of 1 in 3 000 procedures.[25] Cardiovascular instability during spinal anaesthesia is far more common than diaphragmatic paralysis, and the loss of consciousness associated with precipitous hypotension may masquerade as high motor block. Hence, rapid intervention is paramount to treat cardiovascular instability. In unresponsive patients, airway safety must also be addressed.

High motor block ascending to the cervical segments can be identified by close observation of the patient and testing motor function at the appropriate segmental level. Early signs of high block may include 'pill-rolling' movement of the patient's hands followed by repeated lifting of her hands from the arm rests, as she experiences a subjective feeling of weakness in the arms. A motor block at the C8 - T1 level should be tested by establishing whether the patient has an adequate hand grip. A more critical level is C5, above which diaphragmatic paralysis will occur. A good test for C5 - C6 motor weakness is to ask the patient to flex her elbow repeatedly by touching her own shoulder. The inability to cough and, as the block advances to a high cervical level, to phonate, are diagnostic of high motor block. Occasionally the onset of high block is so rapid that the first sign is an unresponsive, apnoeic patient. Hypotension is common and may be confused with profound hypotension accompanied by brainstem ischaemia. After immediate treatment of the hypotension, as described above, the patient should in either circumstance receive 100% oxygen using an anaesthetic mask and circuit. Adequacy of ventilation can be assessed by observing bag movement during inspiration. The patient may be conscious, and should be warned that general

anaesthesia might be necessary. If ventilation is inadequate, the assistant should apply cricoid pressure, preparatory to a rapid sequence induction of general anaesthesia. A small dose of either etomidate or ketamine is advisable, followed by suxamethonium to facilitate tracheal intubation.

Summary of management of spinal anaesthesia

- Exclude contraindications to spinal anaesthesia.
- Perform thorough assessment of airway and co-morbidity, and prepare the mother accordingly.
- Prepare and check equipment, including requirements for general anaesthesia.
- Establish adequate venous access.
- Draw up vasopressors.
- Measure blood pressure, establish ECG and pulse oximetry.
- Perform spinal anaesthesia in sitting or right lateral position.
- Rapidly co-administer 20 ml/kg of crystalloid solution IV during the procedure and commencement of surgery (restrict to 10 ml/kg in the pre-eclamptic patient).
- Place the mother supine with left lateral uterine displacement using an obstetric wedge and/or table tilt.
- Administer face-mask oxygen as appropriate.
- Commence blood pressure readings every minute for the first 10 minutes or until delivery, and thereafter every 2 - 3 minutes if stable.
- Manually displace the uterus or turn patient into the left lateral position in the event of sudden cardiovascular collapse.
- Administer ephedrine 5 mg IV if systolic blood pressure decreases by 20% (10 mg if decrease > 30%), or to less than 100 mmHg, and repeat the dose every minute until systolic blood pressure recovers. Consider prophylactic ephedrine in the morbidly obese patient, or if the estimated fetal weight is > 4 kg, or in the case of twin pregnancy.
- Proceed to phenylephrine 50 µg bolus IV doses after a total of 25 mg ephedrine has been administered, or if marked tachycardia occurs; consider phenylephrine from the outset if the starting heart rate is above 120/minute.
- Give atropine 0.5 - 1 mg IV if sudden bradycardia occurs with associated hypotension (not following baroreceptor-mediated bradycardia after phenylephrine administration).
- Adrenaline should be available (seldom necessary).
- Intubate and ventilate the patient in the rare event of high motor block, having first treated haemodynamic instability.
- Administer 5 IU oxytocin at delivery, over at least 1 minute in the healthy parturient, followed by a slow infusion of 20 IU/l.
- Avoid bolus oxytocin in the unstable patient; then administer by infusion only.

The above recommendations should assist aneathetists in improving the safety of their practice, ultimately reducing the considerable morbidity and mortality associated with spinal anaesthesia for caesarean section in South Africa.

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