Guidelines for managing hypotension during spinal anesthesia for cesarean delivery

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Pregnancy produces significant hemodynamic changes allowing the cardiovascular system to meet the increased metabolic demands of pregnancy. The pregnant patient with normal cardiac function can accommodate significant alterations in the cardiovascular system without difficulty. However, these changes have major implications for anesthetic management, especially in high-risk patients.

Knowledge of physiologic cardiac changes occurring during pregnancy is essential to optimize obstetric anesthesia care.

Cardiovascular physiologic changes in pregnancy

Blood volume

Estrogen induced renine increase results in active sodium reabsorbtion in the renal tubules, causing an increase in total body water from 6.5 L to 8.5 L by the end of gestation (1). Increase in the blood volume occurs very early in the pregnancy, by the 6th week, and reaches a 50 % increase by the second trimester (2). The plasma volume increases by approximately 50%, while the red blood cell mass by only 33%. This disproportionate increase of the plasma volume accounts for hemodilution and decreased hemoglobin concentration and therefore diminished oxygen carrying capacity (3).

The increase in blood volume enables increased uterine blood flow to meet growing nutritional and oxygenation needs of the fetus. It also enables blood loss of average 500 ml at vaginal delivery and 1000 ml at cesarean delivery, without physiological decompensation.

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Cardiac output

Cardiac output is gradually increasing at 8 to 10 weeks gestation and reaching a 50% increase by term (1). This increase is primarily a result of an increase of 30 % in stroke volume as heart rate increases only about 10% (3). A further increase in cardiac output of 40% may occur in the second stage of labor. The increase in cardiac output allows increased perfusion of the uterus. The blood flow through the uterus is 450 to 650 mL/min at term representing approximately 25% of cardiac output.

Blood pressure

Systolic and diastolic blood pressures drop during pregnancy by 5-15 mm Hg with the nadir occurring at 28 weeks' gestation, as a result of decreased systemic vascular resistance.

The decrease in the systemic vascular resistance is a consequence of progesterone induced vasodilatation together with the decreased resistance of the placental bed.

Blood pressure returns to pre-pregnancy values during the third trimester.

Aortocaval compression

Supine position results in compression of the inferior caval vein by the enlarged uterus and consequent obstruction of venous return and decreased cardiac output, reduced placental perfusion and decreased fetal oxygenation (1).

Supine hypotensive syndrome occurs in about 10% of pregnant women at term (4) and has been described to occur as early as the 16th week of gestation. The nature and severity of symptoms range from unspecific complaints to severe maternal hypotension, loss of consciousness, cardiovascular collapse, and consecutive fetal depression.

Spinal anesthesia is widely used as the procedure of choice for cesarean delivery (5). In comparison to epidural anesthesia it is faster, easier to perform, patients are more comfortable, complication rates are lower, and it is more cost effective (6). Spinal block causes peripheral vasodilation and venous pooling, which may result in maternal hypotension. Maternal hypotension after spinal anesthesia for cesarean delivery, without prophylactic measures, has a very high incidence (80%-100%) (7-9). Even though highly investigated, spinal induced hypotension remains a major concern, and it has been referred to as the "Holy Grail" of obstetric anesthesia (10). The detrimental effects of the spinal induced hypotension are maternal and fetal. Maternal effects are nausea, vomiting and dizziness (8). Hypotension results

in reduced uterine and intervillous blood flow with potential fetal hypoxia and acidosis (5).

Treatment and prevention of hypotension has been the subject of much investigation and controversy.

Prophylactic measures include: 1) left lateral tilt, 2) fluid preload, 3) vasopressors, 4) low dose spinal anesthesia.

Lateral left tilt

A 15° left lateral tilt is used routinely during cesarean section, to prevent aorto-caval compression, however it is not sufficient as a sole method (8). Left uterine displacement is achieved by tilting the operating table or by placing a wedge under the woman's hip (4). Aorto-caval compression also may increase the spread of spinal anesthesia (11).

Fluid preloading

Preloading the circulation with crystalloids or colloids is aimed at the volume expansion that alleviates the vasodilation induced by spinal anesthesia. Ueyama et al. (12) demonstrated that the increase of blood volume with volume preload must be great enough to result in a significant increase in cardiac output.

Cristalloid solutions

The role of crystalloid preload in the prevention of hypotension has been questioned (13,14). Rout et al. (13) reported a 55% incidence of hypotension in elective cesarean delivery patients after preloading with 20 ml/kg cristalloids as compared with 71% in patients without hydration. The lack of efficacy of crystalloid solutions is explained by the rapid distribution into the interstitial space due to their short intravascular half-life (8,12).

Colloid solutions

Colloid solutions seem more effective in preventing spinal induced hypotension than crystalloid solutions, reflecting the greater and more prolonged augmentation of circulating volume. However they have several disadvantages: high cost and more side effects (anaphylactoid reactions, interference with blood coagulation and excessive volume expansion leading to pulmonary edema) (15). The use of colloid solutions proved to decrease but not abolish the incidence of hypotension after spinal anesthesia for elective cesarean delivery (16).

Vasopressors

Hypotension after spinal anesthesia for cesarean section can be minimized by appropriate vasopressor control (9,15,16). Historically, ephedrine was considered the vasopressor of choice in obstetrics, based on animal studies that showed more effectiveness in restoring maternal arterial pressure after hypotension with better preservation of uteroplacental blood flow compared with other vasopressors (17,18). However, recently this has been questioned due to ephedrine's limited efficacy and the large doses needed to maintain blood pressure. Furthermore, it has been demonstrated that there is an increased incidence of fetal acidosis in association with ephedrine as compared to phenylephrine (19,20). Possible explanations for increased fetal acidosis associated with ephedrine administration include increased fetal heart rate and metabolism as well as stimulation of endogenous release of fetal catecholamines.

Recent studies have demonstrated the efficacy of high dose of phenylephrine infusions for maintaining maternal blood pressure during spinal anesthesia for cesarean delivery (20,21).

In these studies despite the high-dose phenylephrine used (1.5 mg) the authors did not observe adverse neonatal effects. Although phenylephrine appeared safe from a fetal perspective, it may cause maternal bradycardia. The incidence of maternal bradycardia was as high as 25% when phenylephrine was used without cohydration and 17% when used with cohydration with crystalloids (22).

According to the Practice Guidelines for Obstetric Anesthesia "Intravenous ephedrine and phenylephrine are both acceptable drugs for treating hypotension during neuraxial anesthesia, however, "in the absence of maternal bradycardia, phenylephrine may be preferable because of improved fetal acid – base status in uncomplicated pregnancies"(23).

The usual approach is to allow post spinal hypotension to occur and then treat it accordingly. Because of the high incidence of the post spinal hypotension, it seems logical to preempt it. It was shown that preventing, rather than treating hypotension after it occurs, improves the neonatal acid-base status and reduces the incidence of nausea and vomiting in the mother (24). Ngan Kee et al. (21) reported a very low incidence (2%) of hypotension (defined as systolic blood pressure < 80% of baseline), using a combination of a high-dose phenylephrine infusion and rapid crystalloid cohydration, but had 47% reactive hypertension (defined as systolic blood pressure > 120% of baseline). The phenylehprine infusion regimen used was 100 µg/min started immediately after spinal injection and titrated to maintain systolic blood pressure near baseline values until uterine incision. Despite the controversy

regarding the choice of vasopressor and the optimal method of administration, it seems that phenylephrine offers significant advantages over ephedrine in treating and preempting spinal induced hypotension.

Low dose spinal

The risk of hypotension is related to the height and speed of onset of the spinal anesthesia. Using lower spinal dose as part of a combined spinal-epidural technique has been associated with improved maternal hemodynamic stability (25-27).

Doses of spinal bupivacaine between 5 and 7 mg proved to be sufficient to provide effective anesthesia. Van de Velde M et al. (27) reported a significant reduction in blood pressure rate using 6.5 mg hyperbaric bupivacaine as compared with 9.5 mg bupivacaine. In both groups 2.5 µg sufentanil was added. Langesæter et al. (28) recently showed that decreasing the spinal dose bupivacaine from 10 mg to 7 mg, significantly improves hemodynamic stability, when used with a low-dose phenylephrine continuous infusion of 0.25 µg/kg/min.

Epidural supplementation can be used for patients who do not achieve an adequate block for surgery.

Spinal anesthesia for preeclamptic patients

Epidural anesthesia was considered the optimal anesthetic technique for cesarean delivery in preeclamptic women (29). Spinal anesthesia in preeclamptic patients was considered unsafe, because of the concern that a sudden sympathectomy would result in profound hypotension due intravascular volume contraction. However, recent studies proved that spinal anesthesia is a safe technique for cesarean delivery in preeclamptic and severely preeclamptic women, in the absence of contraindications to regional anesthesia (30-33).

The incidence and severity of hypotension is similar in women with severe preeclampsia having a cesarean delivery with spinal or epidural anesthesia (32).

Furthermore, spinal anesthesia was associated with less hypotension in the preeclamptic as compared with normotensive women having cesarean delivery (31).

Possible reasons for reduced incidence of hypotension can be the smaller gestational weight and smaller uterine mass (less aortocaval compression) in the preeclamptic women.

Conclusions

There is no single method that prevents spinal induced hypotension. Avoid aorto-caval compression by left uterine displacement. Use low dose spinal as part of combined spinal epidural anesthesia. Phenylephrine seems to be the vasopressor of choice in the treatment of hypotension following spinal anesthesia. Preemptive phenylephrine infusion has encouraging results. Spinal anesthesia is safe in preeclamptic patients.

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