# Prevention of Hypotension following Spinal Anaesthesia in Caesarean Section - then and now

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## ABSTRACT

Hypotension during spinal anaesthesia for caesarean section remains a common scenario in our clinical practice. Certain risk factors play a role in altering the incidence of hypotension. Aortocaval compression counteraction does not help to prevent hypotension. Intravenous crystalloid prehydration has poor efficacy; thus, the focus has changed toward co-hydration and use of colloids. Phenylephrine is established as a first-line vasopressor, although there are limited data from high-risk patients. Ephedrine crosses the placenta more than phenylephrine and cause possible alterations in the foetal physiology.

## Key Words

*caesarean section, hypotension, spinal anaesthesia*

## INTRODUCTION

Spinal anaesthesia has become the method of choice for anesthesia for elective caesarean delivery.[1] It is frequently accompanied by hypotension, which may be defined in absolute terms as a systolic blood pressure of 90 or 100 mmHg or in relative terms as a percentage (20 percent from baseline). Hypotension caused by a reduction in systemic vascular resistance is normally compensated by an increase in cardiac output. This is attenuated under spinal anaesthesia by an increase in venous capacitance because of venodilatation in the lower part of the body. The situation is further compounded in pregnancy by aortocaval compression. Thus, instead of compensatory increase, cardiac output usually decreases.[2] This is the combined effect of reduced cardiac output and decreased systemic vascular resistance accounts for hypotension after spinal anaesthesia.

## ETIOLOGY

The incidence of hypotension can be as high as 80%[3]; the severity depends on the height of the block, the position of the parturient, and whether prophylactic measures were taken to prevent the hypotension.

Measures that decrease the risk of hypotension to varying degrees include intravenous administration of fluids, avoidance of aortocaval compression, and monitoring of blood pressure at frequent intervals after placement of regional anaesthetic. If recognized and treated promptly, transient maternal hypotension may not be associated with maternal or neonatal morbidity.[4]

The higher the segmental sympathetic blockade, the greater is the risk of hypotension and associated emetic symptoms.[5] The supine position significantly increases the incidence of hypotension. Ueland and colleagues observed an average reduction of blood pressure from 124/72 to 67/38 mmHg in mothers who were placed in the supine position following the induction of spinal anesthesia, whereas the blood pressure averaged 100/60 mmHg for mothers in the lateral position.[6]

Uterine blood flow is pressure dependent as there is no autoregulation on the placental bed. As a consequence of this, prolonged maternal hypotension is damaging to the fetus and it is also frequently associated with maternal nausea and vomiting. Brief episodes of maternal hypotension have lowered Apgar scores, prolonged time to sustained respiration and prolonged fetal acidosis.[7]

## AORTOCAVAL COMPRESSION

Aortocaval compression must be avoided before and during the performance of caesarean section. During supine position the gravid uterus of the pregnant woman compresses the aorta and the inferior vena cava against

the bodies of lumbar of vertebra. This results in decreased venous return which may decreases maternal cardiac output and blood pressure leading to compromised uteroplacental perfusion. Therefore, it is necessary to maintain left uterine displacement before and during caesarean section, regardless of the anaesthetic technique.[8] This may be accomplished by placing a wedge of 12 centimeter beneath the right buttock. Although widely used, this procedure is variably applied,[9] and does not prevent hypotension after spinal anesthesia.[10]

## INTRAVENOUS FLUID THERAPY

Fluid pre-loading was routinely used upto 87% of cases in spinal anesthesia for caesarian section.[11] Rout et al noted that the incidence of hypotension was reduced from 71% in patients without prehydration to 55% in patients who received crystalloid 20ml/kg.[12] However, some study showed that using 10ml-30ml/kg Ringer's lactate for acute volume expansion before induction of spinal anesthesia, no differences in the indices of maternal hypotension or dosage of ephedrine was observed.[13] Both the rate[14] and volume[15] of crystalloid preloading have also been shown to be unimportant. Studies of this kind have led to a reappraisal of the role of fluid preloading.[16,17] It is still reasonable to administer a modest amount of crystalloid preload before spinal injection, as patients for elective surgery are often relatively dehydrated. However, there is no need to delay emergency surgery in order to preload.

A recent systematic review found that crystalloid was inconsistent in preventing hypotension and that colloid was significantly better.[18] Dahlgren et al[19] studied crystalloid compared with colloid for preloading. Hypotension was significantly reduced after larger volumes of colloid. It is postulated that parturient preoperatively susceptible to the supine position would benefit the most from colloid preloading. In another study of preloading comparing pentastarch with crystalloid, French et al[20] demonstrated a reduction in the incidence of hypotension in the colloid group (12.5% versus 47.5%). In contrast to these studies which all found colloid preload of benefit, Karinen et al failed to find any reduction in the incidence of hypotension when colloid was used.[21] Moreover, disadvantages of colloid include the additional cost, possibility of anaphylactoid reactions and excessive volume expansion, which might lead to pulmonary oedema.[22]

Several recent studies have compared prehydration versus cohydration both with crystalloids and colloids and shown that haemodynamic changes and vasopressor requirement are similar. Banerjee et al performed a meta-analysis (eight studies, 518 partuients) of studies that compared prehydration with cohydration. They found that the incidence of hypotension to be similar for (odds ratio 0.93, 95% confidence interval [CI] 0.54-1.6) cohydration to that for prehydration.[23]

To sum up, firstly, colloid is superior to crystalloid for fluid management with some recognizable adverse effects; secondly, one should consider the role of vasopressor along with the fluid used in management of hypotension[24]; and thirdly, prehydration is not superior to cohydration, implying that any urgent case should not be delayed on the pretext of prehydration.

## VASOPRESSORS

Ephedrine has been the drug of choice for more than 30 years in the treatment of maternal hypotension in obstetric spinal anesthesia when conservative measures fail. It has a good safety record, ready availability, and familiarity to most anesthesiologists. Ephedrine is a sympathomimetic that has both a direct (alpha and beta receptor agonist) and an indirect (release of norepinephrine from presynaptic nerve terminals) mechanism of action. Uterine blood flow, in particular was maintained more favorably with beta-agonists than with alpha-agonists. Ephedrine thus became the gold standard for this application and, in 2001, a survey of obstetric anesthetists in the United Kingdom found that more than 95% used ephedrine as the sole vasopressor, with only 0.4% choosing phenylephrine.[11] Ephedrine has a slow onset of action making it difficult to titrate and use it with an appropriate bolus dose. Regarding ephedrine prophylaxis, studies have looked at the effectiveness to prevent maternal hypotension. Ngan Kee and colleagues found that a 30- mg bolus of ephedrine administered over 30 seconds following intrathecal injection did not completely eliminate maternal hypotension, nausea, vomiting and fetal acidosis.[25] Shearer and colleagues also have found similar result. Thus, a single prophylactic dose is ineffective and the effectiveness depends on variable doses and the rate of administration.[26] The reason why ephedrine depresses fetal acid-base status more than phenylephrine is controversial. Older studies focused on differential effects of vasopressors on uteroplacental circulation. However, Ngan Knee et al[27] showed that ephedrine crosses the placenta more readily than phenylephrine. This was associated with greater fetal concentrations of lactate, glucose and catecholamine, and thus supports the hypothesis that depression of fetal pH and metabolic effects secondary to stimulation of fetal beta-adrenergic receptors cause base excess with ephedrine. Ephedrine, with its long duration of action still has a role in obstetric anesthesia to prevent or treat spinal induced hypotension when given in an appropriate dose. The optimal method to administer ephedrine, whether combined with other

vasopressor therapy or non medication therapy, awaits future study.

Phenylephrine is a short-acting, potent, vasoconstrictor that causes an increase in both systolic and diastolic blood pressure. It counteracts the vasodilatation due to neuraxial anaesthesia directly, restoring baseline blood pressure. Traditionally, it was used as a second line vasoconstrictor in obstetrics because of the concerns that it caused vasoconstriction in the uteroplacental circulation. Interest in phenylephrine was rekindled in 1988 by Ramanathan and Grant,[28] who found that it did not cause fetal acidosis when treating maternal hypotension. Numerous studies have confirmed these findings and almost all have reported higher umbilical artery (UA) pH values in neonates born to phenylephrine treated mothers.[29] A systematic review in 2002 summarized findings from seven RCTs comparing ephedrine with phenylephrine.[30] In this review phenylephrine was associated with higher UA pH values than ephedrine although there was no difference in the incidence of fetal acidosis (UA pH <7.2) or in the Apgar scores <7 at 1 and 5 minutes. When there is hypotension and bradycardia ephedrine continues to be the drug of choice[31]. Otherwise, phenylephrine, which has not been shown to be deleterious to the fetus, may well be the better agent. There are limited data comparing ephedrine and phenylephrine with regard to other maternal outcomes of interest including nausea and vomiting. One study found that the incidence of nausea was 66%

in ephedrine treated mothers compared with 17% in the phenylephrine group.[32] A recent randomized clinical trial examined the maternal and neonatal effects of maintaining maternal blood pressure within 80%, 90%, or 100% of baseline levels using a phenylephrine infusion.[33] Using phenylephrine 100 mcg/ml infused at initial rates of 100 mcg/min, the investigators adjusted the dose depending upon whether blood pressure was kept within the assigned group's range. Woman in the 100% baseline group had fewer episodes of nausea and vomiting and their neonatal mean umbilical arterial pH was higher. Hypotension was better controlled with tight control of blood pressure using aggressive vasopressor administration. Phenylephrine appears to have survived the period of intense suspicion and concern over its use in obstetric anesthesia. It is reliable in its effect, although short acting, and its effect on the fetus appears to be even less than that of ephedrine.

Combinations of phenylephrine and ephedrine given together in the same syringe have previously been advocated, although the optimal regimen has not been determined. Mercier and colleagues compared an ephedrine/phenylephrine infusion with an ephedrine infusion alone and found that the incidence of hypotension in the combination group was half that in the ephedrine-alone group with a beneficial effect on umbilical artery pH.[34] However, when Cooper and colleagues performed a randomized, double blind trial comparing ephedrine, phenylephrine and ephedrine/phenylepherine infusions, there was no decrease in the incidence of maternal nausea and vomiting or neonatal acidosis when the combination was used compared with phenylephrine alone.[32] Reflecting upon these studies, the administration of vasopressor drugs by infusion as close to the time of the spinal anaesthesia administration as possible appears to be helpful in reducing the incidence of hypotension.

Metaraminol, a mixed alpha and beta agonist can be used for spinal induced hypotension. Ngan Kee and colleagues demonstrated that metaraminol was superior to ephedrine at maintaining both maternal blood pressure and fetal pH during spinal anesthesia for caesarean section. The doses of vasoconstrictors in this study were large and the benefits may have been exaggerated.[35] Angiotensin II is a potent vasoconstrictor with a short half life, which affects the uterine vasculature less than the other vasoconstrictors. Ramin and colleagues demonstrated a benefit to using angiotensin II over ephedrine when comparing fetal pH after prophylactic infusions of two drugs at caesarean section.[36] Angotensin II had to be used in infusion more over other limitations includes vailabilyt and cost. There are only few studies comparing angiotension infusion there is no meta analysis as such.

## OTHER METHODS

Low dose spinal anaesthesia for caesarean delivery combines a small dose of intrathecal local anesthetic with an opiod to reduce the incidence of hypotension. Tsen et al showed that with 12 mg bupivacaine along with 1000 ml of lactated Ringer's solution preloading and 10 mg ephedrine, the incidence of hypotension was 70%[37]; which further lowered to 58% when 9 mg bupivacaine was used along with 1000 ml lactated Ringer's solution preload with 15 mg ephedrine.[38] The incidence was further reduced to 31% when 25 µg of fentanyl and 5 mg bupivacaine was used.[39] Not a single patient in the low dose group achieved a complete motor block, whereas most of the patients in the plain bupivacaine group did. Despite the differences in motor block, the sensory block was sufficiently intense in both groups to provide surgical anaesthesia for all patients. Although the technique is promising, and one might intuitively expect a reduction in the incidence of hypotension and nausea with such low doses, there are insufficient data to support this conclusion.

## CONCLUSION

Management of hypotension during spinal anaesthesia in obstetrics continues to be controversial. Although most clinicians will continue to rely on non-invasive BP and cardiac output monitoring may prove useful in future. While fluid preload and left uterine displacement are often employed in an attempt to prevent this complication, a vasopressor is often required. Crystalloid prehydration should no longer be considered mandatory and the current focus is on timing of fluids and use of colloids. Apart from this, one may choose ephedrine or phenylephrine as a vasopressor. Ephedrine causes more depression of fetal acid–base status than phenylephrine, probably because ephedrine crosses the placenta more readily and has direct metabolic effects on the fetus. There is an abundance of evidence to suggest that phenylephrine is at least as good as ephedrine and a more liberal use of this drug is probably justified. Further work is required to determine the optimal therapy for hypotension in high-risk patients.

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