ISSN 2474-8919

ANESTHESIOLOGY
Open Journal

Openventio
PUBLISHERS

Review

Perioperative Fluid Management In Obstetric Patients

Vinodh Natarajan, MBBS, MD[1]; Uma Hariharan, MBBS, DNB, PGDHM[2*]; Rakesh Garg, MD, DNB, FICA, PGCCHM, MNAMS, CCEPC, FIMSA[3]

[1]Department of Anaesthesiology Dr. Ram Manohar Lohia Hospital (Dr. RMLH) & Post-Graduate Institute of Medical Education and Research (PGIMER), Central Health Services, New Delhi, India
[2]Department of Anaesthesiology and Intensive Care, Dr. Ram Manohar Lohia Hospital (Dr. RMLH) & Post-Graduate Institute of Medical Education and Research (PGIMER), Central Health Services, New Delhi, India
[3]Department of Anaesthesiology, Pain and Palliative Medicine, Associate Professor, Dr BRAIRCH, All India Institute of Medical Sciences, New Delhi, India

Corresponding author
Uma Hariharan, MBBS, DNB, PGDHM
Fellowship Onco-Anesthesia, Advanced Regional Anesthesia & Palliative Care, Assistant Professor, Department of Anaesthesiology and Intensive Care, Dr. Ram Manohar Lohia Hospital (Dr. RMLH) & Post-Graduate Institute of Medical Education and Research (PGIMER), Central Health Services, New Delhi, India;
Tel. +91-9811271093; E-mail: uma1708@gmail.com

Article information
Received: January 21st, 2018; **Revised:** March 3rd, 2018; **Accepted:** April 9th, 2018; **Published:** April 20th, 2018

Cite this article
Natarajan V, Hariharan V, Garg R. Perioperative fluid management in obstetric patients. *Anesthesiol Open J.* 2018; SE(1): S1-S7. doi: 10.17140/AOJ-SE-1-101

ABSTRACT

Perioperative fluid management can be confusing due to numerous opinions, formulas and clinical inferences, which result in a picture that is often misleading. Errors in fluid therapy are the most common cause of perioperative morbidity and mortality. In pregnancy, a woman undergoes significant changes in several anatomic, physiologic and biochemical processes which result in a unique state of body fluid dynamics. Even a slight fluid overloading may result in potentially fatal complications like pulmonary oedema in pregnant individuals. Hence, it is mandatory for healthcare providers to understand the changes in fluid dynamics occurring during pregnancy assess the impact of various patient-related or pregnancy-induced comorbid conditions on fluid dynamics and also know the effect of anaesthetic interventions before starting any fluid therapy for them. This article analyses the basic concepts in perioperative fluid management along with a sound review of literature and highlights the importance of a meticulous fluid therapy in the obstetric population.

Keywords
Fluid management; Colloid osmotic pressure; Cesarean delivery; Preloading; Co-loading; Goal-directed fluid therapy; Vasopressor; Fetal acidosis.

Abbreviations
GDT: Goal-Directed Therapy; MRI: Magnetic Resonance Imaging; BJR: Bezold-Jarisch Reflex; ERAS: Enhanced Recovery After Surgery; SV: Stroke Volume; GDFT: Goal-Directed Fluid Therapy; HDP: Hypertensive Disorder of Pregnancy; CSEA: Combined Spinal Epidural Anesthesia; RL: Lactated Ringer; NS: Normal Saline.

INTRODUCTION

The concepts of perioperative fluid management are numerous and rapidly changing.[1] Pregnancy represents a unique state in body fluid dynamics where dramatic alterations begin shortly after conception and almost totally resolve following delivery. These alterations are considered abnormal for non-pregnant individuals but are well tolerated by pregnant women. Healthcare professionals should be aware of the impact of these changes and fluid management should be done accordingly, without affecting the physiological state.

PHYSIOLOGICAL CHANGES IN PREGNANCY

Pregnancy results in a number of physiological changes in fluid dynamics which have a significant impact on fluid management. Plasma volume increases by 15% during the first trimester, rises rapidly during the second trimester to 50-55% above the prepregnant level

Published under Special edition *"Fluid Management in Perioperative Period"*

Openventio PUBLISHERS

and changes little during the remainder of the pregnancy. Red blood cell volume falls during the first 8 weeks of pregnancy, increases to the prepregnant level by 16 weeks, and undergoes a further rise to 30% above the prepregnant volume at term. These changes result in 10%, 30%, and 45% increases in total blood volume by the end of the first, second, and third trimesters, respectively. By the term, total blood volume increases by 45% and red cell volume increases by 30% and this differential leads to a condition called "physiological anaemia of pregnancy" or "hemodilution of pregnancy". The hemodilution, from increased plasma volume, is due to activation of renin-angiotensin-aldosterone system by pregnancy hormones, oestrogen, and progesterone. Another important factor in maintaining fluid homeostasis is the colloid osmotic pressure which is due to plasma proteins in the blood (mainly albumin). During pregnancy, albumin level falls from 7.8 g/dL to 7.0 g/dL and resultant the colloid osmotic pressure also decreases approximately by 5 mmHg and comes to 22 mmHg (normal range 25-27). Increase in blood volume and fall in colloid osmotic pressure predisposes the pregnant patient to a higher risk of developing pulmonary oedema, especially during fluid loading.[2]

CAUSES FOR INTRAVASCULAR VOLUME DERANGEMENTS

Perioperative maintenance of adequate intravascular volume status in pregnant individuals is very important to achieve optimal outcomes after surgery. Pre-operative fasting, anaesthesia-related factors like neuraxial blocks and surgical site bleeding are the major causes for intravascular volume derangements in the obstetric population. Pre-operative fasting overnight for approximately 8 hours does not significantly reduce intravascular volume. Nevertheless, pre-operative dehydration should be avoided by limiting the period of fasting and encouraging patients to consume clear oral liquids up to four hours before surgery.[3] Usually, pregnant females are exposed to rapid intravascular fluid fluctuations during cesarean deliveries. Spinal anaesthesia is used frequently for cesarean deliveries because of its rapid onset, minimal risk of anaesthetic toxicity and negligible transfer of the drug to the foetus, as well as a mere risk of failure of the block. However, higher incidence of hypotension is a major disadvantage. Prevention of spinal anaesthesia-induced hypotension is of utmost importance as the life of the mother as well as foetus is at risk. Several methods and techniques like intravenous fluid boluses, left uterine displacement, prophylactic vasopressors and utilizing compression stockings onto the lower extremities have been tried and utilised in daily routine obstetric practice.[4] Traditionally, 15 degrees of left uterine displacement has been recommended for pregnant patients to reduce aortocaval compression ("supine hypotension syndrome"), which occurs in the supine position when the uterus is at or above the umbilicus. A foam or wood wedge, pillow, or rolled blanket may be used, or the table can be tilted, or the uterus can be manually displaced.[5] Uterine displacement at cesarean delivery improves neonatal acid-base status. A systematic review published in the year 2013 was not able to determine the optimum method or maternal position.[6] Magnetic resonance imaging (MRI) examination of aortic and inferior vena cava volumes in pregnant and nonpregnant peers have shown that inferior vena cava volume, but not aortic volume, is influenced by patient position; specifically, a left-lateral tilt of at least 30 degrees is needed to improve vena cava volume. A lateral tilt of 30 degrees may be impractical during cesarean delivery, and further study is required before changing practice.[7]

ASSESSMENT OF FLUID STATUS

The purpose of assessing the intravascular volume status is to guide the fluid administration in order to maintain adequate tissue perfusion. Traditionally, fluid status was assessed by using static parameters like heart rate, blood pressure, urine output, central venous pressure and pulmonary artery occlusion pressure. Nowadays, these parameters are found to be inferior and replaced by new modern techniques like pulse contour analysis, oesophageal Doppler, and echocardiography.[8] Oesophageal Doppler monitoring is a validated form of monitoring cardiac output and requires the insertion of a thin plastic tube into the oesophagus. Arterial pulse contour analysis measures the stroke volume on a beat-to-beat basis from an arterial pulse waveform, but the main drawback of it is that it requires arterial line placement which is not always needed in all patients.[9] Recently, Biais et al used the Infinity CNAP SmartPod™ which provides non-invasive continuous beat-to-beat measurements of arterial blood pressure and values of respiratory-induced variables in the pulse pressure non-invasively in the finger.[10] With transthoracic or transesophageal echocardiography, intravascular volume status can be readily assessed by measuring inferior vena-caval collapsibility index and by qualitative visual inspection of left ventricle cavity size. But, this technique requires an additional skill to the attending anesthesiologists.[11] In recent times, individualised goal-directed therapy has to become popular and more effective than traditional liberal or fixed-volume fluid therapy. The concept of goal-directed therapy (GDT) was first described by Shoemaker in 1988 and it means that achieving supranormal circulating functions (the target values for cardiac index, oxygen delivery, and oxygen consumption) is by appropriate use of fluids and inotropes in the perioperative period.[12] Perioperative GDT describes fluid administration, with the aim of optimising a patient's cardiac function and ultimately oxygen delivery. Several clinical reviews support the use of GDT in the perioperative settings. In a recent meta-analysis, fluid administration with the GDT approach was associated with improved clinical outcome when compared with liberal fluid management regimens.[13] However, GDT approach of fluid administration is not required when Enhanced Recovery After Surgery (ERAS) regimen protocols are strictly followed. ERAS protocols implement the multiple processes including the avoidance of preoperative overhydration, use of an intraoperative restricted fluid approach, emphasis on early postoperative alimentation and ambulation.[14] The concept of ERAS on caesarean deliveries is not new and is found to be effective in reducing prolonged hospital stay among the pregnant women.[15]

PRELOADING *VS.* CO-LOADING

Another method to counteract the hypotension following spinal anaesthesia in caesarean deliveries is the fluid loading. The administration of fluid bolus can be done before and at the time of administration of spinal anaesthesia, the techniques named appropriately as preloading and co-loading respectively. Wollman and Marx first described the concept of preloading, by administering 10-20

Openventio PUBLISHERS

ml/kg of intravenous crystalloids in pregnant females around 15-20 minutes prior spinal anaesthesia. Initially, this concept was accepted and found to have beneficial effects.[16] But however, recent studies demonstrated that preloading may induce the release of atrial natriuretic peptide which in turn damages endothelial glycocalyx and leads to increased rate of excretion of preload fluid from the intravascular fluid.[17] Then comes the concept of preloading with colloids which are retained in the intravascular space for more duration than crystalloids. But, it also fails to be effective due to increased cost, damage to the endothelial glycocalyx, the possibility of derangement in coagulation and the risk of anaphylaxis.[18] Due to inconsistent results from preloading, the concept of co-loading has gained wide acceptance among anaesthesiologists. Co-loading seems to be appropriate physiologically as fluid administration coincides exactly with the time of maximal vasodilatory effect of spinal anaesthesia. Co-loading seems to be a safer technique except for the risk of decreasing oxygen carrying capacity and increasing risk of pulmonary oedema. Various studies had compared preloading with co-loading and concluded that the incidence of hypotension was similar as was the requirement of vasopressor boluses in both the methodologies. The results are almost similar when colloids have been replaced with crystalloids in studies of similar designs comparing the potential benefits of preloading and co-loading.[19] Banerjee A et al in his meta-analysis also stated similar results and none of the two techniques were found to be superior to one another in reducing the incidence of hypotension.[20] When deciding the choice of fluid therapy, recent Saline against Lactated Ringer's or Plasma-Lyte in the Emergency Department (SALT-ED) trial mentioned that balanced salt solutions like Plasmalyte A™ and Lactated ringer (RL) resulted in a lower incidence of major adverse kidney events and electrolyte imbalance when comparing with normal saline (NS).[21] As far as colloids are concerned, third generation colloid such as Tetrastarch (130/0.4) seems to be safer than other types of colloids, as it does not cause significant renal damage.[22] This needs to be investigated further in the pregnant population.

ROLE OF VASOPRESSORS

As it is evident from the above discussion, the incidence of hypotension is similar in both preloading and co-loading with either crystalloids or colloids. Whenever there is hypotension during cesarean section, perfusion to the foetus will get affected. This is evident from the change in umbilical cord pH and low Apgar scores. So, it is always appropriate to treat the hypotension with vasopressors. Prophylactic doses of vasopressors are found to be superior in preventing adverse neonatal outcomes when compared with reactive treatment.[23] Ephedrine in a dose of 0.25 mg/kg has been a drug of choice for more than 30 years to counteract the hypotension seen in cesarean deliveries. Ephedrine is a sympathomimetic that has both a direct (alpha and beta receptor agonist) and indirect (release of norepinephrine from presynaptic nerve terminals) mechanism of action with the favourable impact on uterine blood flow. Phenylephrine is a short-acting, potent vasoconstrictor that causes an increase in both systolic and diastolic blood pressure due to its alpha-2 agonist action. Traditionally, phenylephrine had been used as a second line vasoconstrictor in obstetrics because it was mistakenly thought to compromise uteroplacental circulation. In 1988, Ramanathan and Grant found that phenylephrine did not cause foetal acidosis when used to treat maternal hypotension. This may be because there is a significant placental reserve of oxygen, or that the relatively high doses of α agonists have little effect on placental blood flow because of differing placental anatomy and physiology.[24] A study by das et al found that continuous infusion of phenylephrine appeared superior at preventing hypotension, nausea, and vomiting when compared with a prophylactic bolus dose of 50 μg phenylephrine. Sen et al reported similar results when comparing patients having a phenylephrine infusion with those having an initial prophylactic dose of 50 μg phenylephrine, followed by intermittent 50 μg doses. From this evidence, it appears that a prophylactic phenylephrine infusion is superior to bolus administration alone and that delaying the start of the infusion could limit its efficacy in reducing the incidence of hypotension.[25] International consensus statement for management of hypotension during cesarean sections in 2018 also mentioned that administration of phenylephrine as a prophylactic infusion is the first line of management in reducing the incidence of hypotension and nausea-vomiting compared with bolus administration.[26] A recent study done by Dong L et al revealed that prophylactic norepinephrine infusion is also as effective as phenylephrine and provides a cost-effective alternative to phenylephrine.[27] Nevertheless, more research is needed to evaluate its use in caesarean deliveries. Mephentermine has also been shown to be equally effective when compared to phenylephrine in preventing post-spinal hypotension in cesarean section. The cost-effectiveness of mephentermine justifies continued use of mephentermine in some developing countries, despite the availability of other vasopressors. Mephentermine increases blood pressure mainly by augmenting cardiac output.[28] Although not used to treat hypotension, Ondansetron reduces the early incidences of hypotension and bradycardia through serotonin (5HT3) mediated Bezold-Jarisch reflex (BJR) attenuation.[29]

Hence, it is evident that judicial use of fluids by using appropriate monitors and the prophylactic use of vasopressor doses are the main principles for managing hypotension in obstetric individuals during the perioperative period.

NEONATAL EFFECTS

Maternal fluid management can have a secondary effect on the foetus. Investigations with pregnant ewes demonstrated that acute increase in maternal vascular volumes does not promote fluid transfer into foetus per se. Nevertheless, an acute decrease in maternal colloid oncotic pressure with the administration of hypotonic solutions can cause fluid shifts into foetus as evident by the decrease in foetal plasma osmolarity and increase in foetal urine flow. This appears to demonstrate that maternal oncotic pressure is more responsible rather than vascular hydrostatic pressure changes for determining the fluid shifts into the foetus. When comparing colloid and crystalloid loading in pregnant individuals, no significant changes in foetal myocardial contraction and left ventricular (LV) function were reported so far.[30] In a randomised controlled trial, Tawfik et al found that neither crystalloid co-load nor colloid preload can totally prevent hypotension and should be combined with vasopressor use for an optimal neonatal outcome.[31] Tercanli et al also assessed the effect of crystalloid preloading and co-loading in

Openventio PUBLISHERS

parturients and found that there are no significant differences in neonatal outcomes by APGAR scores and cord blood pH.[32] Acute fetal stress is a sensitive marker of neonatal outcome and is represented by umbilical cord pH and $PaCO_2$ values. Jain K et al in their randomised study emphasised the importance of fetal blood gas measurements and effect of vasopressors on it. The author suggested that the use of a higher dose of phenylephrine (>35 mcg) is associated with umbilical cord pH>7.2 following elective caesarean deliveries.[33] A meta-analysis of twenty trials to investigate fetal acidosis with the use of ephedrine and phenylephrine, a better control on hypotension with both the drugs were found, but with decreased the risk of fetal acidosis under phenylephrine prophylaxis.[34] Recent studies are showing promising results for norepinephrine as an ideal vasopressor agent during caesarean deliveries. Mild beta agonist action of norepinephrine favours its use as a more suitable vasopressor than phenylephrine which is associated with bradycardia in some situations. There is no significant difference in neonatal outcome when comparing phenylephrine and norepinephrine. More well-constructed randomised trials are needed to enlighten norepinephrine in the treatment of post-spinal hypotension in caesarean deliveries.[35] Although not uniformly observed to be different than the infants of parturients undergoing vaginal deliveries, infants born to mothers undergoing cesarean deliveries were found to have low colloid osmotic pressure. Among the other variable causes, intraoperative fluid therapy is also believed to have a major effect on foetal colloid osmotic pressure.[29] Watson J et al suggested that the restricted IV fluids policy did not affect new-born weight loss during the delivery period. His exploratory analyses showed that breastfed new-born weight loss increases when intrapartum volumes infused are >2500 mL. It may be prudent to consider volumes of IV fluid infused intrapartum or during surgery as a factor that may have contributed to early new-born weight loss in the first 48 h of life.[36]

SPECIAL OBSTETRIC SITUATIONS

Hypertensive disorder of pregnancy (HDP): Elevated blood pressure (BP) in pregnancy is considered to compensate the reduced transplacental blood flow due to systematic arterioles spasm. Major maternal complications associated with HDP are placental abruption, haemolysis, elevated liver enzymes, low platelets syndrome, disseminated intravascular coagulation, neurologic deficits, pulmonary edema and acute renal failure. Widespread endothelial dysfunction can also occur in the placenta that ultimately leads to placental ischemic injury or even infarction.

HDP is prone to peripheral edema while the intravascular volume is paradoxically insufficient due to increased capillary permeability and decreased oncotic pressure. Severe pre-eclampsia may be accompanied with cardiac dysfunction, reduced oncotic pressure, elevated hydrostatic pressure and pulmonary capillary leak, all of which lead to pulmonary edema. Parturient with severe pre-eclampsia is poorly tolerant to overhydration if the ventricular dysfunction is present and is also sensitive to the sympathetic blockade. Three possible aetiologies of pulmonary oedema have been suggested in pre-eclampsia: (I) left ventricular failure (II) pulmonary capillary leak and (III) reduced colloid oncotic pressure. Colloid oncotic pressure (COP) is normally in the range of 25-28 mmHg. It is less in pregnancy, 22 mmHg at 34-36 weeks, about 18 mm Hg after delivery and may fall as low as 14 mmHg in pre-eclampsia.[37] Cesarean delivery, which is usually instituted to terminate a pregnancy in such parturient, is currently popular to be performed under combined spinal epidural anesthesia (CSEA). Singh et al suggested that spinal anaesthesia could be safely used for lower segment cesarean delivery in stable eclamptic patients to avoid risks of general anaesthesia.[38] There is also another school of thought that patients with severe pre-eclampsia experience less hypotension and have lower vasopressor requirements during spinal anaesthesia, compared with healthy women undergoing a caesarean section. These findings suggest that women with pre-eclampsia either have greater endogenous vasoactive mediators, or are more sensitive to exogenous vasopressors, compared with healthy pregnant women. Hence, co-loading and preloading may be confounding in the management of pre-eclampsia patient. Similarly, the requirements of vasopressors are also limited. International consensus for managing hypotension in caesarean section committee recommends that phenylephrine is the optimal first-line vasopressor to reverse the maternal haemodynamic changes induced by spinal anaesthesia in women with severe pre-eclampsia. The dose of phenylephrine required may be lower than in healthy women; hence, a prophylactic vasopressor infusion may not be required and, if used, should be started at a low dose with the effect on blood pressure monitored carefully.[25] The use of goal-directed fluid therapy (GDFT) with the LiDCO™ system targeted at optimizing maternal stroke volume (SV) may be beneficial in hypertensive disorders of pregnancy.[39]

Cardiac diseases: Neuraxial techniques are frequently used in women with cardiac disease undergoing cesarean section in contemporary clinical practice. In women with pulmonary hypertension, there is a trend towards lower mortality during cesarean section with neuraxial compared with general anaesthesia.[40] Titratable, catheter-based neuraxial techniques rather than single-shot spinal anaesthesia is advised in women with significant cardiac disease; the rapid-onset sympathectomy and hemodynamic changes associated with spinal anaesthesia are often poorly tolerated, especially with pre-load-dependent physiology (e.g., Fontan circulation) or fixed cardiac output states (e.g., severe aortic or mitral stenosis), so that the incidence of hypotension is best avoided and also there is no need for giving aggressive volume replacement. Phenylephrine is the suitable choice of vasopressor in stenotic lesions and ischemic heart disease patient, whereas, ephedrine is the choice of agent in regurgitant lesions.[41]

Peripartum haemorrhage: Peripartum haemorrhage is the most common cause of maternal mortality in the world. Adequate replacement of intravascular volume and good surgical haemostasis are needed to control and reduce the consequences of bleeding. Increase in maternal blood volume and coagulation proteins level can make pregnant patients tolerate up to 1000 and 1500 ml blood loss without significant hemodynamic changes. With the onset of symptoms and signs of volume loss, aggressive volume replacement is needed to maintain tissue perfusion and oxygenation and it should be done effectively with blood and colloids. In addition, with the mixing of foetal, maternal and other cellular substances, disseminated intravascular coagulation may occur with little or no

warning and intensify blood loss. So, rapid volume replacement with blood and its component and invasive lines have become mandatory in those situations.[42] The WOMAN trial (World Maternal Antifibrinolytics trial) and a recent Cochrane database recommended the routine use of antifibrinolytics like tranexamic acid in a dose of 1 g/kg to reduce the blood loss during surgery.[43,44] The real-time coagulation monitors may assist in guiding fibrinogen therapy in the management of postpartum haemorrhage, but easy availability of such tests in most of the hospitals are questionable.[45] The task force on blood component therapy by anesthesiologists (ASA) have recommended that the transfusion of packed red blood cells, platelets and fibrinogen component therapy might be rarely indicated unless the haemoglobin is less than 7 g/dL, the platelet count is less than 50 x 10^9/L (provided there is no platelet dysfunction or microvascular bleeding), the fibrinogen concentration is less than 80-100 mg/dL in presence of microvascular bleeding.[46] Attention has been given recently to autologous donation, intraoperative cell salvage, and acute normovolemic hemodilution in the parturient population at high risk of maternal haemorrhage. Additional investigation will be required to validate the safety and utility of these approaches.

CONCLUSIONS

The physiological, mechanical and hormonal changes of pregnancy represent the adaptations that have a significant impact on fluid management. A better understanding of these alterations is a must for the anaesthesiologists before handling pregnant patients. The use of left uterine displacement, vigilant monitoring, judicious use of fluids and prophylactic vasopressors remain the most effective means of ensuring favourable maternal and foetal outcomes, including special measures and monitors for co-existing maternal diseases.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

REFERENCES

1. Bennett VA, Cecconi M. Perioperative fluid management: From physiology to improving clinical outcomes. *Indian J Anaesth*. 2017; 61: 614-621. doi: 10.4103/ija.IJA_456_17

2. Lund CJ, Donovan JC. Blood volume during pregnancy: Significance of plasma and red cell volumes. *Am J Obstet Gynecol*. 1967; 98: 393-403. doi: 10.1016/0002-9378(67)90160-3

3. Practice Guidelines for Obstetric Anesthesia: An Updated Report by the American Society of Anesthesiologists Task Force on Obstetric Anesthesia and the Society for Obstetric Anesthesia and Perinatology.Anesthesiology. 2016; 124:270-300.

4. Jacob JJ, Williams A, Verghese M, et al. Crystalloid preload versus crystalloid coload for parturients undergoing cesarean section under spinal anesthesia. *J Obstet Anaesth Crit Care*. 2012; 2: 10-15. doi: 10.4103/2249-4472.99309

5. Kundra P, Velraj J, Amirthalingam U, et al. Effect of positioning from supine and left lateral positions to left lateral tilt on maternal blood flow velocities and waveforms in full-term parturients. *Anaesthesia*. 2012; 67: 889-893. doi: 10.1111/j.1365-2044.2012.07164.x

6. Cluver C, Novikova N, Hofmeyr GJ, et al. Maternal position during caesarean section for preventing maternal and neonatal complications. *Cochrane Database Syst Rev*. 2013; 3: CD007623. doi: 10.1002/14651858.CD007623.pub3

7. Higuchi H, Takagi S, Zhang K, et al. Effect of lateral tilt angle on the volume of the abdominal aorta and inferior vena cava in pregnant and nonpregnant women determined by magnetic resonance imaging. *Anesthesiology*. 2015; 122: 286-293. doi: 10.1097/ALN.0000000000000553

8. Cove ME, Pinsky MR. Perioperative hemodynamic monitoring. *Best Pract Res Clin Anaesthesiol*. 2012; 26: 453-462. doi: 10.1016/j.bpa.2012.10.003

9. Gurudatt CL. Perioperative fluid therapy: How much is not too much? *Indian J Anaesth*. 2012; 56: 323-325. doi: 10.4103/0019-5049.100810

10. Biais M, Stecken L, Ottolenghi L, et al. The ability of pulse pressure variations obtained with CNAP device to predict fluid responsiveness in the operating room. *Anesth Analg*. 2011; 113: 523-528. doi: 10.1213/ANE.0b013e3182240054

11. Reeves ST, Finley AC, Skubas NJ, et al. Special article: Basic perioperative transesophageal echocardiography examination: A consensus statement of the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. *Anesth Analg*. 2013; 117: 543-558. doi: 10.1213/ANE.0b013e3182a00616

12. Shoemaker WC, Appel PL, Kram HB, et al. Prospective trial of supranormal values of survivors as therapeutic goals in high-risk surgical patients. *Chest*. 1988; 94: 1176-1186. doi: 10.1378/chest.94.6.1176

13. Corcoran T, Rhodes JE, Clarke S, et al. Perioperative fluid management strategies in major surgery: A stratified meta-analysis. *Anesth Analg*. 2012; 114: 640-651. doi: 10.1213/ANE.0b013e318240d6eb

14. Joshi GP, Kehlet H. CON: Perioperative goal-directed fluid therapy is an essential element of an enhanced recovery protocol? *Anesth Analg*. 2016; 122: 1261-1263. doi: 10.1213/ANE.0000000000001233

15. Corso E, Hind D, Beever D, et al. Enhanced recovery after elective caesarean: A rapid review of clinical protocols, and an umbrella review of systematic reviews. *BMC Pregnancy Childbirth*. 2017; 17: 91-93. doi: 10.1186/s12884-017-1265-0

16. Wollman S, Marx C. Acute hydration for prevention of hypotension of spinal anesthesia in parturients. *Anesthesiology*. 1968; 29:

Published under Special edition *"Fluid Management in Perioperative Period"*

374-380.

17. Pouta AM, Karinen J, Vuolteenaho OJ, et al. Effect of intravenous fluid preload on vasoactive peptide secretion during Caesarean section under spinal anesthesia. *Anaesthesia*. 1996; 51: 128-132. doi: 10.1111/j.1365-2044.1996.tb07698.x

18. Cyna AM, Andrew M, Emmett RS, et al. Techniques for preventing hypotension during spinal anesthesia for cesarean section. *Cochrane Database Syst Rev*. 2006; 4: CD002251. doi: 10.1002/14651858.CD002251.pub2

19. Bajwa SJ, Kulshrestha A, Jindal R. Co-loading or pre-loading for prevention of hypotension after spinal anesthesia! A therapeutic dilemma. *Anesth Essays Res*. 2013; 7: 155-159. doi: 10.4103/0259-1162.118943

20. Banerjee A, Stocche RM, Angle P, et al. Preload or coload for spinal anesthesia for elective cesarean delivery: A meta-analysis. *Can J Anaesth*. 2010; 57: 24-31. doi: 10.1007/s12630-009-9206-7

21. Self WH, Semler MW, Wanderer JP, et al. Balanced crystalloids versus saline in noncritically Ill adults. *N Engl J Med*. 2018; 378: 819-828. doi: 10.1056/NEJMoa1711586

22. Van D, Linden P, James M, et al. Safety of modern starches used during surgery. *Anesth Analg*. 2013; 116: 35-48. doi: 10.1213/ANE.0b013e31827175da

23. Mercier FJ. Cesarean delivery fluid management. *Curr Opin Anaesthesiol*. 2012; 25: 286-291. doi: 10.1097/ACO.0b013e3283530dab

24. Mitra J K, Roy J, Bhattacharyya P, et al. Changing trends in the management of hypotension following spinal anesthesia in cesarean section. *J Postgrad Med*. 2013; 59: 121-126. doi: 10.4103/0022-3859.113840

25. Nag DS, Samaddar DP, Chatterjee A, et al. Vasopressors in obstetric anesthesia: A current perspective. *World J Clin Case*s. 2015; 3: 58-64. doi: 10.12998/wjcc.v3.i1.58

26. Kinsella SM, Carvalho B, Dyer RA, et al. International consensus statement on the management of hypotension with vasopressors during cesarean section under spinal anesthesia. *Anaesthesia*. 2018; 73: 71-92. doi: 10.1111/anae.14080

27. Dong L, Dong Q, Song X, et al. Comparison of prophylactic bolus norepinephrine and phenylephrine on hypotension during spinal anesthesia for cesarean section. *Int J Clin Exp Med*. 2017; 10: 12315-12321. doi: 10.1097/ALN.0000000000000602

28. Mohta M, Janani SS, Sethi AK, et al. Comparison of phenylephrine hydrochloride and mephentermine sulfate for prevention of post-spinal hypotension. *Anaesthesia*. 2010; 65: 1200-1205. doi: 10.1111/j.1365-2044.2010.06559.x

29. Potdar MP, Kamat LL, Jha TR, et al. Effect of ondansetron in attenuation of post-spinal hypotension in caesarean section: A comparison of two different doses with placebo. *J Obstet Anaesth Crit Care*. 2017; 7: 69-74. doi: 10.4103/joacc.JOACC_7_16

30. Hepner D, Tsen LC. Fluid management in obstetrics. In: Robert GH, Prough DS, Svensen CH, eds. *Perioperative Fluid Therapy*. 2nd ed. Boca Rat, FL, USA: CRC Press; 2016; 405-418.

31. Tawfik MM, Hayes SM, Jacoub FY et al. Comparison between colloid preload and crystalloid co-load in cesarean section under spinal anesthesia: A randomized controlled trial. *Int J Obstet Anesth*. 2014; 23: 317-323. doi: 10.1016/j.ijoa.2014.06.006

32. Tercanli S, Schneider M, Visca E, et al. Influence of volume preloading on uteroplacental and fetal circulation during spinal anaesthesia for caesarean section in uncomplicated singleton pregnancies. *Fetal Diagn Ther*. 2002; 17: 142-146. doi: 10.1159/000048027

33. Jain K, Makkar JK, Subramani VS, et al. A randomized trial comparing prophylactic phenylephrine and ephedrine infusion during spinal anesthesia for emergency cesarean delivery in cases of acute fetal compromise. *J Clin Anesth*. 2016; 34: 208-215. doi: 10.1016/j.jclinane.2016.03.015

34. Veeser M, Hofmann T, Roth R, et al. Vasopressors for the management of hypotension after spinal anesthesia for elective caesarean section. Systematic review and cumulative meta-analysis. *Acta Anaesthesiol Scand*. 2012; 56: 810-816. doi: 10.1111/j.1399-6576.2011.02646.x

35. Ngan Kee WD, Lee SWY, Ng FF, et al. Prophylactic norepinephrine infusion for preventing hypotension during spinal anesthesia for cesarean delivery. *Anesth Analg*. 2017; 7:11-14.

36. Watson J, Hodnett E, Armson BA, et al. A randomized controlled trial of the effect of intrapartum intravenous fluid management on breastfed newborn weight loss. *J Obstet Gynecol Neonatal Nurs*. 2012; 41: 24-32. doi: 10.1111/j.1552-6909.2011.01321.x

37. Engelhardt T, MacLennan FM. Fluid management in pre-eclampsia. *Int J Obstet Anesth*. 1999; 8: 253-259. doi: 10.1177/1753495X13486896

38. Singh R, Kumar N, Jain A, et al. Spinal anesthesia for lower segment Cesarean section in patients with stable eclampsia. *J Clin Anesth*. 2011; 23: 202-206. doi: 10.1016/j.jclinane.2010.08.011

39. Xiao W, Duan QF, Fu WY, et al. Goal-directed fluid therapy may improve hemodynamic stability of parturient with hypertensive disorders of pregnancy under combined spinal epidural anesthesia for cesarean delivery and the well-being of newborns. *Chin Med J (Engl)*. 2015; 128: 1922-1931. doi: 10.4103/0366-6999.160546

40. Lane C, Trow T. Pregnancy and pulmonay hypertension. *Clin Chest Med*. 2011; 32: 165-174. doi: 10.1007/s12471-011-0219-9

41. Langesaeter E, Dragsund M, Rosseland LA. Regional anesthesia for a cesarean section in women with cardiac disease: A pro-

spective study. *Acta Anaesthesiologica Scandinavica*. 2010; 54: 46-54. doi: 10.1111/j.1399-6576.2009.02080.x

42. Snegovskikh D, Clebone A, Norwitz E, et al. Anesthetic management of patients with placenta accreta and resuscitation strategies for associated massive hemorrhage. *Curr Opin Anaesthesiol*. 2011; 24: 274-281. doi: 10.1097/ACO.0b013e328345d8b7

43. Shakur H, Elbourne D, Gülmezoglu M, et al. The WOMAN Trial (World Maternal Antifibrinolytic Trial): Tranexamic acid for the treatment of postpartum haemorrhage: An international randomised, double blind placebo-controlled trial. *Trials*. 2010; 16; 11: 40. doi: 10.1186/1745-6215-11-40

44. Shakur H, Beaumont D, Pavord S, et al. Antifibrinolytic drugs for treating primary postpartum haemorrhage. *Cochrane Database Syst Rev*. 2018; 2: CD012964. doi: 10.1002/14651858.CD012964

45. Solomon C, Collis RE, Collins PW. Haemostatic monitoring during postpartum haemorrhage and implications for management. *Br J Anaesth*. 2012; 109: 851-863. doi: 10.1093/bja/aes361

46. Apfelbaum M. Gregory A. Nuttall D, et al. Practice guidelines for perioperative blood management: An updated report by the American Society of Anesthesiologists Task Force on Perioperative Blood Management. American Society of Anesthesiologists Task Force on Perioperative Blood Management. *Anesthesiology*. 2015; 122: 241-275. doi: 10.1097/ALN.0000000000000463