◆ EDITORIAL VIEWS

Anesthesiology
1999; 91:1565–7

Spinal Hypotension Associated with Cesarean Section

Will Preload Ever Work?

Recent years have seen a move away from large-volume intravenous hydration as prophylaxis against post–spinal hypotension.[1,2] Although controversy still exists, there is accumulating evidence that crystalloid solutions are particularly ineffective in preventing hypotension after extensive sympathetic blockade associated with spinal anesthesia. One reason crystalloid solutions may not be effective is their short intravascular half-life. In this issue of ANESTHESIOLOGY, Ueyama *et al.*[3] provide data to support the limited and transient effect of 1,500 ml (more than 20 ml/kg) crystalloid on blood volume and cardiac output compared with hydroxyethyl starch 6% (HES). They suggest that to effectively prevent hypotension associated with obstetric spinal anesthesia by intravenous volume administration, augmentation of blood volume must be large enough to result in a significant increase in cardiac output.

The widespread prophylactic use of crystalloid solutions followed work in gravid ewes that showed the value of rapid intravenous fluid administration in partly restoring uterine blood flow after spinal hypotension.[4] Blood pressure usually is maintained in the face of vasodilation (other than that caused by central nerve blockade) by a reflexive increase in cardiac output. However, in the presence of spinal induced venodilation, venous return is reduced to an extent that cardiac output cannot increase and is often reduced.[5] The result is severe hypotension with reduced uteroplacental perfusion. Therefore, efforts were made to increase cardiac preload before instituting spinal blockade in the hope of preventing subsequent hypotension; the concept of intravenous "preload" was born.

Initial clinical studies of crystalloid prehydration in obstetric spinal anesthesia appeared to be promising, [6,7] but the results were clouded by issues such as the presence of labor (which decreases the incidence of hypotension), aortocaval compression, and study design. Although intravenous fluid preload became an established part of anesthetic practice, it soon became apparent that it was not completely effective in preventing hypotension.[8] However, it was assumed that, because the theory was sound, more preload was required in some patients. Hence, large volumes (up to 30 ml/kg) were increasingly used or studied.[9,10] Nevertheless, the problem remained.

A number of studies have shown either no[1] or a clinically insignificant[11] reduction in the incidence of spinal hypotension after crystalloid preload, leading some to abandon it altogether and others to remove it from routine mandatory practice. The use of colloid solutions for spinal preloading met with mixed results. Gelatin solutions may be ineffective and have not achieved the degree of success seen with modified starch solutions.[12,13] This may be a result of the difference in intravascular half-life between the two, supporting the argument used to explain crystalloid failure. However, does this necessarily relate to the concept of preload?

The indocyanine green dilution method used by Ueyama *et al.*[3] to estimate blood volume and cardiac output does not permit a simultaneous assessment of cardiac preload. Nevertheless, similar modest increases in blood volume produced by 1,500 ml lactated Ringer's and 500 ml HES (8 and 10% respectively) were associated with modest increases in cardiac output (11 and 14%), without a significant reduction in the incidence of subsequent hypotension. Doubling the increase in blood volume (to 19%) by use of 1 l HES more than tripled the associated increase in cardiac output (to 43%). Because heart rate did not change substantially, the change must have been a result of an increase in stroke volume. Because there was no change in arterial pressure associated with volume administration, this implies a decrease in vascular resistance. Increases in cardiac output occur with normovolemic hemodilution. An increase in cardiac output of 29% may be seen after isovolumic reduction of hematocrit to 33%.[14] It is likely that hypervolemic hemodilution would produce an even greater increase in cardiac output *via* a number of possible mechanisms,

◆ This Editorial View accompanies the following article: Ueyama H, Yan-Ling H, Hironobu T, Mashimo T, Yoshiya I: Effects of crystalloid and colloid preload on blood volume in the parturient undergoing spinal anesthesia for elective cesarean section. ANESTHESIOLOGY 1999; 91:1571–6.

Accepted for publication August 26, 1999.

Key words: Blood volume; obstetric anesthesia; spinal anesthesia and analgesia.

including increased cardiac preload, decreased blood viscosity, and peripheral vasodilation caused by reduced arterial blood oxygen content. Increased cardiac filling alone might reduce afterload *via* atrial stretch receptors and atrial natriuretic peptide. Which of these factors is responsible for the reduced incidence of hypotension? Parturients with an increased baseline systemic vascular resistance index or systolic blood pressure have been shown to be at increased risk of subsequent hypotension after regional anesthesia.[15] Is it possible that the key to prevention of post–spinal hypotension is a sustained increase in cardiac output and reduction in afterload before central sympathetic blockade, rather than a transient increase in cardiac preload?

Despite its short intravascular half-life, intravenous crystalloid has been shown to significantly increase cardiac filling pressures before spinal anesthesia.[16,17] However, this increase is rapidly reversed after the onset of sympathetic blockade. In the absence of data showing a sustained increase in atrial pressure or left ventricular end-diastolic volume during spinal anesthesia without hypotension, the whole concept of fluid preload might be called into question. Rapid administration of crystalloid solutions can cause hemodilution and increase cardiac output,[18] but the volumes required to produce the same degree of change seen with 1 l HES would not be practicable, and even then would be relatively transient. The increase, albeit transient, of cardiac preload seen with crystalloid suggests the possible role of other mechanisms in the prevention of hypotension by colloids.

Whatever the mechanisms of hypotension and its prevention, there are some practical issues regarding the use of large volumes of intravenous colloids before spinal anesthesia for cesarean section. Even with the use of 1 l HES, the incidence of hypotension was 17% (upper 95%, confidence limit 48%). Would it be possible to reduce this even further by use of larger volumes, and, if so, by how much more? After delivery of a neonate, there is an autotransfusion of blood from the contracted uterus. Would this constitute a risk of pulmonary edema in the presence of an already expanded blood volume? There is a small risk of allergy to colloidal solutions, and they are expensive. Before the prophylactic use of colloids becomes part of routine practice, the potential problems and cost need to be balanced against the value of improved outcome and the cost and value of other methods of prevention, such as prophylactic administration of vasopressors. It is likely that the use of colloid alone will not eradicate post–spinal hypotension. Even if it could, is this sufficient reason to use it?

Is "prevention better than cure" applicable to spinal hypotension? Although hypotension can be catastrophic and pose a threat to both mother and infant, it is not always severe, is readily treated, and, if short-lived, does not significantly affect neonatal outcome. Studies showing significant differences in the incidence of hypotension may fail to show a significant difference in umbilical blood gas and acid–base status at delivery between infants born of hypotensive and nonhypotensive mothers.[10] Even studies that show significant biochemical differences may not show any clinical differences in Apgar or neurobehavioral scores.[19]

In summary, the study by Ueyama *et al.*,[3] although confirming the ineffectiveness of crystalloid, does not persuade us to immediately change to prophylactic colloid administration to prevent spinal hypotension associated with cesarean section. However, it does lead us to question the concept of preloading in this situation. As there is no method that is guaranteed to prevent spinal hypotension and improve neonatal outcome, we continue to advocate vigilant monitoring of maternal blood pressure at least every minute after spinal injection, with immediate treatment of hypotension by bolus intravenous ephedrine.

Chris Rout, F.F.A.R.C.S.
Research Professor
D. Anthony Rocke, F.R.C.P. (Edin), F.C.A.(SA)
Professor and Head
Department of Anaesthesia
Faculty of Medicine
University of Natal
Durban, South Africa
Rout@med.und.ac.za

References

1. Jackson R, Reid JA, Thorburn J: Volume preloading is not essential to prevent spinal-induced hypotension at caesarean section. Br J Anaesth 1995; 75:262–5

2. Critchley LAH: Hypotension, subarachnoid block and the elderly patient. Anaesthesia 1996; 51:1139–43

3. Ueyama H, Yan-Ling H, Tanigami H, Mashimo T, Yoshiya I: Effects of crystalloid and colloid preload on blood volume in the parturient undergoing spinal anesthesia for elective cesarean section. ANESTHESIOLOGY 1999; 91:1571–6

4. Greiss FC, Crandell DL: Therapy for hypotension induced by spinal anesthesia during pregnancy: Observations on gravid ewes. JAMA 1965; 191:793–6

5. Green NM, Brull SJ: The cardiovascular system, Physiology of Spinal Anesthesia. Edited by Greene NM, Brull SJ. Baltimore, Williams & Wilkins, 1993, pp 85–199

6. Wollman SB, Marx GF: Acute hydration for prevention of hypotension of spinal anesthesia in parturients. ANESTHESIOLOGY 1968; 29: 374–80

7. Marx GF, Cosmi EV, Wollman SB: Biochemical status and clinical condition of mother and infant at cesarean section. Anesth Analg 1969; 48:986–93

8. Clark RB, Thompson DS, Thompson CH: Prevention of spinal hypotension associated with cesarean section. ANESTHESIOLOGY 1976; 45:670–4

9. Russell IF: Effect of posture during the induction of subarachnoid analgesia for caesarean section. Right v left lateral. Br J Anaesth 1987; 59:342–6

10. Norris MC: Hypotension during spinal anesthesia for cesarean section: Does it affect neonatal outcome? Reg Anesth 1987; 12:191–3

11. Rout CC, Rocke DA, Levin J, Gouws E, Reddy D: A re-evaluation of the role of crystalloid preload in the prevention of hypotension associated with spinal anesthesia for elective cesarean section. ANESTHESIOLOGY 1993; 79:262–9

12. Vercauteren HP, Hoffman V, Coppejans HC: Hydroxyethyl starch compared with modified gelatin as volume preload before spinal anaesthesia for caesarean section. Br J Anaesth 1996; 76:731–3

13. Riley ET, Cohen SE, Rubenstein AJ, Flanagan B: Prevention of hypotension after spinal anesthesia for cesarean section: Six percent hetastarch versus lactated Ringer's solution. Anesth Analg 1995; 81: 838–42

14. Korosue K, Ishida K, Matsuoka H, Nagao T, Tamaki N, Matsumoto S: Clinical, hemodynamic and hemorheological effects of isovolemic hemodilution in acute cerebral infarction. Neurosurgery 1988; 23:148–53

15. Ouzounian JG, Masaki DI, Abboud TK, Greenspoon JS: Systemic vascular resistance index determined by thoracic electrical bioimpedance predicts the risk for maternal hypotension during regional anesthesia for cesarean delivery. Am J Obstet Gynecol 1996; 174:1019–25

16. Rout CC, Akoojee SS, Rocke DA, Gouws E: Rapid administration of crystalloid preload does not decrease the incidence of hypotension after spinal anaesthesia for elective Caesarean section. Br J Anaesth 1992; 68:394–7

17. Karinen J, Rasanen J, Alahuhta S, Jouppila R, Jouppila P: Effects of crystalloid and colloid preloading on uteroplacental and maternal haemodynamic state during spinal anaesthesia for caesarean section. Br J Anaesth 1995; 75:531–5

18. Wennberg E, Frid I, Haljamäe H, Wennergren M, Kjellmer I: Comparison of Ringer's acetate with 3% dextrose 70 for volume loading before extradural caesarean section. Br J Anaesth 1990; 65:654–60

19. Corke BC, Datta S, Ostheimer GW, Weiss JB, Alper MH: Spinal anaesthesia for caesarean section. The influence of hypotension on neonatal outcome. Anaesthesia 1982; 37:658–62

Anesthesiology
1999; 91:1567–70

Ventilatory Management of Severe Acute Respiratory Failure for Y2K

FOR more than 30 years, mechanical ventilation has been a cornerstone of the treatment of patients with acute respiratory distress syndrome (ARDS). Experience has led to value its obvious benefit of supporting gas exchange, but many clinicians have accepted its harmful side effects on the lung as inevitable. Recently, however, mechanical ventilation has undergone an important re-evaluation as an intervention that can support, damage, or even protect the acutely injured lung. Our simplistic practices have been called into question. It is no longer sufficient to merely set a tidal volume (V_T) of 10–15 ml/kg, add a bit of positive end-expiratory pressure (PEEP), and control the respiratory rate to achieve a Pa_{CO_2} of 40 mmHg. The article by Ullrich *et al.*[1] in this issue of ANESTHESIOLOGY describes one approach to ARDS that resulted in a high survival rate. Beyond admiring their superb clinical results, we need to answer a fundamental question: What ventilatory strategy, based on sound physiology and clinical data, can we now recommend to treat severe acute respiratory failure?

Mechanical ventilation with large V_T or high pressure can injure the lung.[2] Although a V_T of 12 ml/kg and an associated alveolar pressure of 30 cm H_2O, commonly used during general anesthesia and surgery, are unlikely to damage normal lungs, similar settings may aggravate lung injury during prolonged mechanical ventilation for acute respiratory failure. This concept is not new, and more than 20 years ago led to the idea of "resting the lung" and supporting respiration by extracorporeal membrane oxygenation.[3] Although extracorporeal membrane oxygenation (ECMO) and its subsequent modifications have not reached widespread use, new physiologic

This Editorial View accompanies the following article: Ullrich R, Lorber C, Röder G, Urak G, Faryniak B, Sladen RN, Germann P: Controlled airway pressure therapy, nitric oxide inhalation, prone position, and extracorporeal membrane oxygenation (ECMO) as components of an integrated approach to ARDS. ANESTHESIOLOGY 1999; 91:1577–86.

Accepted for publication September 13, 1999.

Key words: ARDS; barotrauma; mechanical ventilation.