

AnesthesiaDotCalm Newsletter



News You Can Use

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Spinal Anesthesia for the Severe Preeclamptic



For as long as I can remember, (I don't seem to have any problems with long term memory; it's the short term memory that has me scratching my head) the anesthetic management for severely preeclamptic patients requiring cesarean section has been controversial. In 1985, *Williams Obstetrics* recommended avoiding regional anesthesia at all costs. Based on survey and anecdotal reports, it was hypothesized that regional anesthesia in this population would pose too much of a risk to the mother and fetus. It's been argued that splanchnic blockade in the presence of the reduced plasma volume that often attends parturients with severe preeclampsia may cause dramatic drops in blood pressure with resulting deleterious effects to maternal and fetal well-being. (1) And the large volumes of aqueous fluid given either prophylactically or in an attempt to correct hypotension was also thought to place the severely preeclamptic patient at unusual risk for iatrogenic pulmonary edema. (2) Intravenous pressor agents such as ephedrine were also hypothesized to be hazardous to severely preeclamptic women because of their increased sensitivity to these agents resulting from their altered production and/or clearance of prolactin. (3-7)

Additionally, the concern of spinal cord injury resulting from hemorrhagic complications which may occur in the attempt to provide neuroaxial anesthesia to the parturient with severe preeclampsia has led the anesthetic community to shun regional techniques and opt for what I consider the more riskier alternative: general anesthesia

But as Paul Harvey would say, "Here's the rest of the story"

It is estimated that 2% of the entire population harbours an intracranial aneurysm, though most of these patients never become symptomatic(8) Women in their childbearing years are at greater risk for stroke than their male, age-matched counterparts. In fact, the incidence of stroke during pregnancy and the puerperium is higher than was previously thought (9) The American Maternal Mortality Collaborative reported cerebrovascular disease as the fifth cause of maternal deaths during 1980–1985 (10). What is most startling, by 1990 stroke was considered the second leading cause of death of women in Canada and the United States(11,12) A study published in the September 2005 issue of *Obstetrics & Gynecology* (13) looked at data on stroke and pregnancy discharges within the Agency for Healthcare Research and Quality's Nationwide Inpatient Sample from the Healthcare Cost & Utili-

zation Project for years 2000 to 2001. A total of 2850 discharges for pregnancy complicated by stroke were identified. This translated into a rate of 34.2 strokes for every 100,000 deliveries--a rate higher than was previously deduced in an earlier Canadian study, published in *Stroke* in 2000. That study used similar data and methodology and reported an incidence of 26 strokes for every 100,000 pregnancies(14). Women who had experienced stroke were 70% more likely to have had a complication of pregnancy than were controls. Moreover, only preeclampsia and gestational diabetes were associated with stroke(15). And although the majority of strokes associated with pregnancy result from arterial occlusions almost 40% result from subarachnoid hemorrhage. (34) and that the induction of general anesthesia in this population may play a significant role in these hemorrhagic complications. It has long been known that following intubation, CPP and ICP increase significantly after intubation and even esmolol or lidocaine as an iv bolus of 1.5 mg.kg-1 before laryngoscopy and intubation do not completely prevent the increase in MAP(29). Although sodium pentathal, the drug that is currently the mainstay for the induction of general anesthesia in the pregnant population produces very favorable decreases in cerebral blood flow (CBF) and cerebral metabolic rate for oxygen (CMRO2),(30) large doses (4-6 mg.kg-1 iv) are required to decrease ICP during induction and intubation. This "deep" level of barbiturate anesthesia often produces marked decreases in MAP and CPP in these patients who are usually hypovolemic as a result of their disease process. This decrease in MAP and CPP produced by sodium pentathal may result in compromised regional cerebral blood flow (rCBF)(31). Conversely, "lighter" anesthetic levels using smaller amounts (2-3 mg • kg-1) of sodium pentathal will usually not attenuate the increases in MAP and CPP produced during laryngoscopy with tracheal intubation(32). Consequently, large increases in CPP can either increase intracranial blood volume, thereby increasing ICP, or disrupt the blood-brain-barrier (BBB), causing cerebral edema(33).and/or intracranial hemorrhage especially if parturient has cerebral arterio-venous malformations(AVM). Besides with the advancing gestational age both aneurysm and AVM bleed more frequently(16). Bleeding disorders, uncontrolled hypertension and cocaine abuse are other precipitating factors(17).The incidence of maternal stroke resulting from bleeding cerebral aneurysm is 1 in 6000 to 1 in 30,000 pregnancies, with a 20% mortality and 50% incidence of permanent neurological sequelae(16)

Parturients are about seven times more likely to suffer a failed intubation than general surgical patients (18), which means that for every 300 general anesthetics given to a parturient, one will fail. Now consider that the number one cause of death in the pregnant population is aspiration and/or difficulties in airway management during general anesthesia (19). In the United States between 1987 and 1990, 1453 deaths were judged to be pregnancy-related. This was calculated to mean that for every 100,000 live births approximately 9 women would die! (a pregnancy-related mortality ratio was 9.1 /100,000 live births). From 1979 through 1990, CDC records revealed that 4097 pregnancy-related deaths of which 129 cases were anesthesia-related. Most deaths (82%) occurred in women undergoing caesarean section, 5% were associated with vaginal delivery, and in 13% the type of delivery could not be determined. And here is the eye opener, which of course is not surprising: general anesthesia was involved in 52% of the cases and that these deaths were related to airway management problems. The death rate related to a regional anesthesia was about 25% and of these (here's another eye opener) 70% involved epidural anesthesia. It appears that local anesthetic toxicity or high blockade were the primary contributors leading to maternal death.

But despite the inherent risks associated with regional anesthesia it still is the safer approach. From 1979 through 1984, the rate of death from general anesthesia during caesarean section was 20 per

million, and from 1985 through 1990, the rate was 32.3 per million. In contrast during that same study period, the rate of death during caesarean section with regional anesthesia decreased from 8.6 to 1.9 per million (19).

I think the anesthesia community has finally begun to see the light. Obstetric anesthesiologists are cautiously approaching the issue of regional anesthesia in these patients, using primarily epidural anesthesia in preference to spinal anesthesia(20).

A common justification for preferring epidural to spinal anesthesia is the belief that incrementally dosing the epidural catheter increases the epidural sensory blockade in stages and minimizes the risks of hypotension. However, spinal anesthesia has the benefit of producing regional anesthesia more rapidly and, some anesthesiologists believe, more reliably compared with epidural anesthesia(21).

The rapid onset of spinal anesthesia may be particularly beneficial in severely preeclamptic patients who frequently require urgent cesarean section. One prospective study compared epidural and combined spinal-epidural (CSE) anesthesia for severely pre-eclamptic patients undergoing cesarean section and concluded that changes in blood pressure are similar after epidural or CSE anesthesia(22). Hood (23) retrospectively studied 138 severely preeclamptic patients receiving either epidural or spinal anesthesia for cesarean section. They found the average reductions in mean arterial pressure were mild (15-25%) for both epidural and spinal anesthesia. This finding was corroborated by prospective (24) and earlier uncontrolled studies of preeclamptic patients receiving high spinal analgesia (25-27).

Although we should be concerned about producing iatrogenic pulmonary edema resulting from excessive intravenous crystalloid administration the Hood study tends to give us some reassurance that this is a rare event. Pulmonary edema presented in the postoperative period only once and that was in a parturient that had underlying myocardial pathology.

And the amount of intravenous fluids he had given does not seem any greater than what would be normally given to the parturient with an uncomplicated pregnancy. He found patients having spinal anesthesia received approximately only 400 ml more intravenous fluid than patients receiving epidural anesthesia (means: spinal, 1780 ml; epidural, 1359 ml). Management of hypotension seemed to be relatively uncomplicated, with patients receiving modest doses of intravenous ephedrine and intravenous crystalloid. Total ephedrine use was minimal (median dose, 0; 75th percentile dose, 8.75-10 mg) and similar for epidural and spinal anesthesia.

Finally, although hemorrhagic complications can occur after neuraxial blockade, the actual incidence of spinal cord injury resulting from hemorrhagic complications is unknown; the reported incidence is estimated to be less than 1/150,000 for epidural anesthesia and 1/220,000 for spinal anesthesia(28). With such low incidences, it is difficult to rationalize withholding neuraxial anesthesia from this group of patients. In my way of thinking, neuraxial anesthesia is way safer even in the presence of low

platelets. Retrospective studies tend to reinforce this contention. For example, Frenk and his coworkers retrospectively reviewed the medical records of parturients from 1997 to 2002 who had platelet counts less than 100,000. Of the 177 patients reviewed, 90% had platelet counts slightly greater than 70,000 but less than 100,000 and received neuraxial anesthesia. No neurological complications were documented in any of the patients reviewed(35). In their retrospective review of 2929 parturients, Rasmus and colleagues concluded that platelet counts of 15,000 - 99,000/micrL did not increase the risk of neurological complications after regional anesthesia(36).

Nonetheless, most clinicians still recommend using caution when providing regional anesthesia to these patients. They feel that the use of neuraxial anesthesia should be made on a case to case basis and only after additional bleeding studies are obtained. In the presence of thrombocytopenia, platelet function screens and d-dimer levels may be beneficial in helping the anesthesia practitioner decide if spinal anesthesia is the most appropriate technique.

And now you know the rest of the story.

References:

1. Hypertensive disorders in pregnancy, Williams Obstetrics. Edited by Pritchard JA, MacDonald PC, Gant NF. Norwalk, Appleton-Century-Crofts, 1985, pp 353-65).
2. Sibai BM, Mabie BC, Harvey CJ, Gonzalez AR: Pulmonary edema in severe preeclampsia-eclampsia: Analysis of thirty-seven consecutive cases. *Am J Obstet Gynecol* 1987; 156:1174-9)
3. Talledo O, Chesley LC, Zuspan FP: Renin-angiotensin system in normal and toxemic pregnancies: 3. Differential sensitivity to angiotensin II and norepinephrine in toxemia of pregnancy. *Am J Obstet Gynecol* 1968; 100:218-21 T.
4. Öney, O. Bellmann, and H. Kaulhausen Relationship Between Serum Prolactin Concentration, Vascular Angiotensin Sensitivity and Arterial Blood Pressure During Third Trimester Pregnancy *Arch Gynecol Obstet* (1988) 243:83-90
5. B. Ho Yuen, Wendy Cannon, Suzanne Woolley, Ellen Charles (1978) _MATERNAL PLASMA AND AMNIOTIC FLUID PROLACTIN LEVELS IN NORMAL AND HYPERTENSIVE PREGNANCY_ *BJOG: An International Journal of Obstetrics and Gynaecology* 85 (4), 293-298.
6. MS Manku, DF Horrobin, M Karmazyn and SC Cunnane)
Prolactin and zinc effects on rat vascular reactivity: possible relationship to dihomo-gamma-linolenic acid and to prostaglandin synthesis *Endocrinology*, Vol 104, 774-779.
7. DE Mills and RP Ward Effect of prolactin on blood pressure and cardiovascular responsiveness in the rat. *Experimental Biology and Medicine*, Vol 181, 3-8.
8. Guglielmi G, Vinuela F. Intracranial aneurysms, Guglielmi electrothrombotic coils. *Neurosurg Clin North Am* 1994; 5(3):427-35.)
9. James AH, Bushnell CD, Jamison MG, Myers ER. Incidence and risk factors for stroke in pregnancy and the puerperium. *Obstet Gynecol.* 2005;106:509-516.
10. Rochat RW, Koonin LM, Atrash HK, Jewett JF. Maternal mortality in the United States: report from the Maternal Mortality Collaborative. *Obstet Gynecol.* 1988;72:91-97.[Abstract]
11. Barnett HJM. Stroke in women. *Can J Cardiol.* 1990;6(suppl B):11B-17B.

12. Kneppner LE, Giuliani MJ. Cerebrovascular disease in women. *Cardiology*.1995;86:339–348.
13. James AH, Bushnell CD, Jamison MG, Myers ER. Incidence and risk factors for stroke in pregnancy and the puerperium. *Obstet Gynecol*. 2005;106:509-516.
14. Jaigobin C, Silver FL. Stroke and pregnancy. *Stroke*. 2000;31:2948-2951.
15. Chireau MV, Bushnell CD, Brown H, et al. Pregnancy complications are associated with stroke risk later in life. Presented at: the 130th Annual Meeting of the American Neurological Association; September 25-28, 2005; San Diego.
16. Dias MS, Sekhar LN. Intracranial haemorrhage from aneurysm and arteriovenous malformations during pregnancy and puerperium. *Neurosurgery* 1990; 27: 855.
17. Lichtenfeld PJ, Fubin DB, Feldman RS. Subarachnoid haemorrhage precipitated by cocaine abuse. *Arch Neurol* 1984; 41: 223.
18. Lyons G. 2: Failed intubation. Six years' experience in a teaching maternity unit. *Anaesthesia* 1985; 40:759-62.
19. Hawkins JL, Koonin LM, Palmer SK, Gibbs CP. Anesthesia-related deaths during obstetric delivery in the United States, 1979 1990. *Anesthesiology*. 1997 Feb;86(2):277-84.
20. Cheek TG, Samuels P: Pregnancy-induced hypertension, *Anesthetic and Obstetric Management of High-risk Pregnancy*, Edited by Datta S. St. Louis, Mosby-Year Book, 1991, p 441.
21. Riley ET, Cohen SE, Macario A, Desai JB, Ratner EF: Spinal versus epidural anesthesia for cesarean section: A comparison of time efficiency, costs, charges, and complications. *Anesth Analg* 1995; 80:709-12.
22. Wallace DH, Leveno KJ, Cunningham FG, Giesecke AH, Shearer VE, Sidawi, JE: Randomized comparison of general and regional anesthesia for cesarean delivery in pregnancies complicated by severe preeclampsia. *Obstet Gynecol* 1995; 86:193-9.
23. Hood, D and Curry, R Spinal versus Epidural Anesthesia for Cesarean Section in Severely Preeclamptic Patients: A Retrospective Survey *Anesthesiology*: Volume 90(5) May 1999 pp 1276-1282.
24. Assali NS: Hypertensive complications of pregnancy: Experimental use of various drugs. *J Miss Med Assoc* 1952; 51:586-91.
25. Assali NS, Prystowski H: Studies on autonomic blockade: I. Comparison between the effects of tetraethylammonium chloride (TEAC) and high selective spinal anesthesia on blood pressure of normal and toxemic pregnancy. *J Clin Invest* 1950; 29:1354-66.
26. Karinen J, Rasanen J, Alahuhta S, Jouppila R, Jouppila P: Maternal and uteroplacental haemodynamic state in pre-eclamptic patients during spinal anaesthesia for Caesarean section. *Br J Anaesth* 1996; 76:616-20.
27. Pouta AM, Karinen J, Vuolteenaho OJ, Laatikainen TJ: Effect of intravenous fluid preload on vasoactive peptide secretion during Caesarean section under spinal anaesthesia. *Anaesthesia* 1996; 51:128-32.
28. Horlocker TT, Wedel DJ: Anticoagulation and neuraxial block: historical perspective, anesthetic implications, and risk management. *Reg Anesth Pain Med* 23:129, 1998

29. Samaha T, Ravussin P, Claquin C, Ecoffey C. Prevention of increase of blood pressure and intracranial pressure during endotracheal intubation in neurosurgery: esmolol versus lidocaine.: *Ann Fr Anesth Reanim.* 1996;15(1):36-40.
30. Stullken EH Jr, Milde JH, Michenfelder JD, Tinker JH. The nonlinear responses of cerebral metabolism to low concentrations of halothane, enflurane, isoflurane, and thiopental. *Anesthesiology* 1977; 46: 28-34.
31. Moss E, Powell D, Gibson RM, McDowall DG. Effects of tracheal intubation on intracranial pressure following induction of anaesthesia with thiopentone or althesin in patients undergoing neurosurgery. *Br J Anaesth* 1978; 50:353-60.
32. Unni VKN, Johnston RA, Young HSA, McBride RJ. Prevention of intracranial hypertension during laryngoscopy and endotracheal intubation. Use of a second dose of thiopentone. *Br J Anaesth* 1984; 56: 1219-23.
33. Messick JM Jr, Newberg LA, Nugent M, Faust RJ. Principles of neuroanesthesia for the nonneurosurgical patient with CNS pathophysiology. *Anesth Analg* 1985; 64: 143-74.)
34. Cheryl Jaigobin, MD, FRCPC; Frank L. Silver, MD, FRCPC *Stroke and Pregnancy Stroke.* 2000;31:2948-2951.
35. Frenk V, Camann W, and Shankar KB, Regional anesthesia in parturients with low platelet counts *Canadian Journal of Anesthesia* 52:114 (2005)
36. Rasmus KT, Rottman RL, Kotelko DM, Wright WC, Stone JJ, and Rosenblatt RM. Unrecognized thrombocytopenia and regional anesthesia in parturients: a retrospective review *Obstetrics & Gynecology* 1989;73:943-946 14. Johnson ME, Uhl CB. Toxic elevation of cytoplasmic calcium by high dose lidocaine in a neuronal cell line [abstract]. *Reg Anesth* 1997;22:A68.