



Something to ponder

Still Crazy After All These Years

I am sure that, for some crazy reason, those of us who have focused our attention on the practice of obstetric anesthesia have sometime during the course of our careers come to realize that women who request or reconcile themselves to receive an epidural for their labor are, for lack of better words... weird. In fact, not only are they weird but their significant others (i.e. husbands, boyfriends, girlfriends, mothers, fathers) are also *at sea* in the same dingy. Consider the situation. Here is a woman who willingly exposes her backside to a stranger who is preparing to drive 3.5 inch spike into her back. The husband or significant other, in the mean time, stands idly back watching his loved one get impaled. This situation has to rate way up there on the, well...weird scale. I have given much thought to this phenomenon and have concluded that females of the child bearing age as well as their male counterparts are nuts. This revelation was attained scientifically during an evening of insobriety with a half dozen of my football buddies who managed to put away enough beer to fill up an Olympic-sized swimming pool. Somewhere around half time, the conversation segued from football to sex (for anyone who has imbibed enough beer, this is a definitely a natural flow of thought) with all of us eventually agreeing that having kids is a pretty stupid thing to do. If you really think about it, when was the last time you heard someone say they wanted to invest a million or so dollars in a product which continually breaks down, has an uncertain future, offers no guarantees what-so-ever, causes you perpetual sleep deprivation and as a side product, literally gives you shit? And here's the kicker: we convince ourselves that this thing we call a kid is cute. And for some of us there is no learning curve. You figure, OK, you made one mistake but to continue the process and have two or more of these kids. Hello!!! Is anybody home?!!! But I digress.

From the very early beginnings of my anesthesia career, which expands more than 40 years, the basic tenet I had been inculcated with and which is still being taught today is that pregnant females with hypertension of pregnancy (PIH) have a contracted plasma volume and thus are more sensitive to the vasodilating effects of spinal anesthesia and the drugs used to treat hypotension (6). Administering these patients regional anesthesia was deemed dangerous and should only be attempted with a full armamentarium of monitoring devices such as arterial lines and central venous catheters. And if regional anesthesia were selected then epidural anesthesia was the technique of choice since according to these "great illuminations" the level of anesthesia could gradually be advanced as intravenous fluids were "carefully" titrated to mitigate the fall in blood pressure. What a bunch of hogwash! I have never been able to titrate anything especially when it comes to raising the level of an epidural (Of course to be fair, I can't even boil water).

Furthermore, in my own practice, regardless of all the gyrations I put myself through, the hypotension ensuing from epidural anesthesia does not seem to be significantly different from that which is produced by spinal anesthesia and that reductions in mean arterial pressure following spinal anesthesia and before the delivery of the fetus are similar to the prophylactic reductions in blood pressure (fig 1-from Hood(3)) recommended prior to the induction of

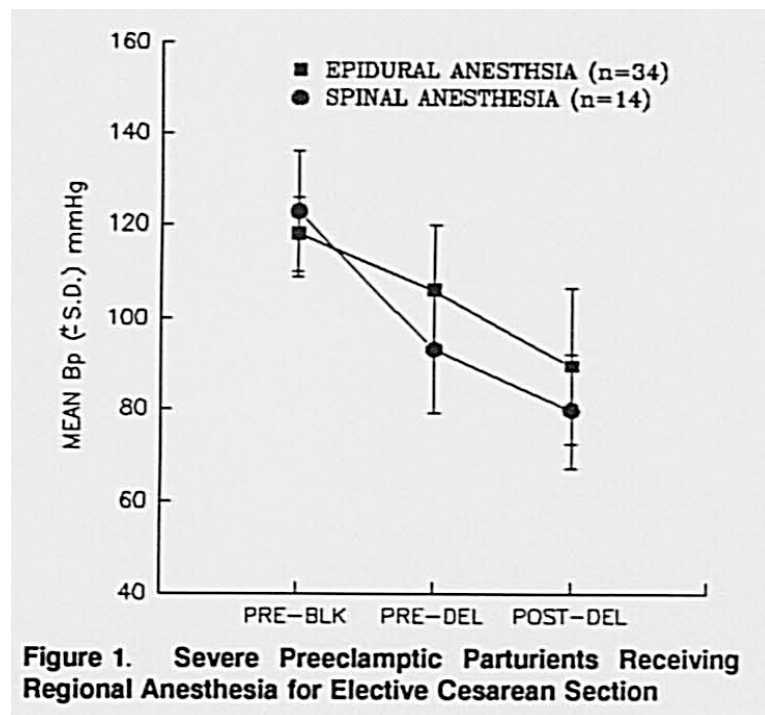


Figure 1. Severe Preeclamptic Parturients Receiving Regional Anesthesia for Elective Cesarean Section

general anesthesia and tracheal intubation (3). And the literature is replete with studies that verify my contention(1,2,3,5,7,8,9,10). Take for example the study by Hood who retrospectively examined the records of 103 non-laboring, severely pre-eclamptic patients and found that the lowest mean blood pressure was similar after epidural or spinal anesthesia. Okay, it is true that there was greater crystalloid administration in the spinal group (1780+/-838ml) than the epidural group (1359+/-674ml) but the intraoperative use of ephedrine and neonatal Apgar scores were similar (1). Similarly a prospective randomized study(5) found no statistically significant difference in Apgar scores or in umbilical arterial blood gas analysis between patients receiving epidural and spinal anesthesia although the mean lowest levels of systolic, diastolic, and mean arterial pressure from induction to delivery were consistently lower in the spinal group but was easily treated with judicious use of

ephedrine; in fact, hypotension lasted no longer than 1 minute in either the spinal or epidural group. Despite the somewhat lower pressures in the spinal group the volume of intravenous fluid given to this group did not differ from that given to the patients in the epidural group. And for those naysayers who still believe that regional anesthesia for the severely pre-eclamptic patient is contraindicated consider that the reduction in blood pressure produced by spinal anesthesia has the added benefit of shortening the QTc interval, the prolongation of which frequently occurs in severe preeclamptic patients who have hypertension and hypocalcemia (4). Elizabeth Drake and her co-authors in their review of the anesthetic implications for patients with prolonged QTc point out that patients with prolonged QTc intervals are more prone to the development of torsades de pointes and ventricular fibrillation (11).

So the idea that parturients with PIH actually fair well with regional anesthesia despite their relative decrease in plasma volume has lead me to another bemusement. Perhaps the best way to treat hypotension from neuroaxial anesthesia is with vasopressors and not with the 20 ml per kg fluid pre-load we've been enjoined to practice since Marx and Wollman (12,13) introduced this strategy back in the 1960's‡.

After all, the infusion of crystalloid or colloid that was initially thought to be successful in preventing hypotension (13) has not been replicated in subsequent studies (17,18,19). And why is this, you may ask? Because in a *normal pregnant female* (that has to be an oxymoron) responses to endogenous pressors, particularly angiotensin II are reduced (20) possibly as a consequence to the increase synthesis and release of nitric oxide (21) as well as the fact that α -adrenoceptor mediated vasoconstriction is impaired more than β -adrenoceptor-mediated vasodilatation. And so the scale now tilts toward a vasodilated state, which tends to be aggravated by the sympathectomy produced by spinal anesthesia. Consequently, the use of sympathomimetic vasopressors to sustain arteriolar tone and thus arterial pressure has become the most important strategy for safe spinal anesthesia in contemporary practice (23). This precept, forty years ago (24) was considered noxious or at least gratuitous since the prevailing thought was that the reduction in venous return was due solely to vena caval compression which could only be mitigated through the judicious use of intravenous fluids.

‡ Their stratagems were the extensions of the hypothesis proposed by Holmes (14) who opined that compression of the inferior vena cava by the gravid uterus caused hypotension after spinal anesthesia because venous return was reduced and thus cardiac output decreased. Studies conducted during this time had indeed verified that the vena cava is affected by the gravid uterus. For example, Scott showed that femoral venous and distal inferior caval pressures were greater in the supine position and decreased when the patient assumed a lateral position (15). Others using angiography showed occlusion of the inferior vena cava and distension of the collateral azygos circulation in 12 supine patients having cesarean section under general anesthesia and even in the lateral position the abdominal vena cava remained partially occluded(16). However, none of these studies proved a direct link between changes in venous behavior and hypotension

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