EDITORIAL

Spinal anaesthesia in severe preeclampsia: time for reappraisal, or time for caution?

The choice of anaesthetic technique in severely preeclamptic women requiring caesarean section has been controversial for a number of years, but clinical experience has demonstrated the relative safety and value of well-managed incremental epidural anaesthesia. There is now general consensus amongst obstetric anaesthetists that this is the technique of choice for most patients, providing relatively smooth control of blood pressure, maintaining utero-placental perfusion, optimizing fetal outcome and eliminating the airway and haemodynamic problems associated with general anaesthesia. However, clinical urgency may occasionally preclude the use of epidural anaesthesia, whereupon the choice will lie between spinal and general anaesthesia.

The hazards of general anaesthesia in severe preeclampsia are well recognized, but the role of spinal anaesthesia is far from clear. Contemporary textbook advice is to avoid its use, fearing catastrophic hypotension. However, over the past few years a number of studies have been published or presented at international meetings which challenge traditional presumptions about the haemodynamic effects of spinal anaesthesia in severe preeclampsia. ¹⁻⁶ Despite reservations about individual aspects of these studies, collectively they provide some evidence to suggest that, in certain circumstances, spinal anaesthesia may indeed be an appropriate technique in this group of critically ill women.

The benefits of spinal anaesthesia include rapid onset of reliable, high quality surgical anaesthesia and avoidance of complications related to emergency general anaesthesia. However, side effects are common, even in non-complicated obstetric patients. Hypotension is reported in up to 80% of cases, and is sometimes remarkably resistant to treatment. Neither fluid preload nor ephedrine has been shown to prevent hypotension reliably, and the use of prophylactic ephedrine (particularly in severe preeclampsia) has not been evaluated. While the use of colloids or larger preload volumes appear to minimize hypotension, aggressive preloading before induction of spinal anaesthesia

is likely to be hazardous in severe preeclampsia due to the risk of iatrogenic pulmonary oedema.

Recently, the fetal effects of spinal anaesthesia have come under renewed scrutiny. Several studies, including some with large numbers of patients, suggest that fetal acidosis may be more common at elective caesarean section when spinal anaesthesia is used compared to epidural or general anaesthesia.7-10 Though probably not clinically significant to the healthy term fetus, in the presence of fetal compromise or impaired utero-placental perfusion, as in severe preeclampsia, this effect may be critical. While fetal acidosis is commonly attributed to inadequately treated hypotension, the cause is probably more complex, and the relative contributions of aortic and vena-caval compression following spinal anaesthesia are still unclear. Robson et al showed that a marked fall in cardiac output was associated with spinal but not with epidural anaesthesia, 10 nor with a slow incremental spinal technique.11 This suggests that the rapid onset of spinal blockade is the crucial, potentially harmful, factor. Interestingly, fetal pH correlated well with cardiac output, but not with blood pressure, reinforcing the clinical observation that upper limb blood pressure measurement is a poor indicator of organ perfusion.

There are numerous reasons to believe that spinal anaesthesia would not be a good choice in severe preeclampsia. Many of the known haemodynamic derangements contraindicate the sudden onset of extensive sympathetic blockade. Severely preeclamptic women have a markedly elevated systemic vascular resistance, and intravascular hypovolaemia. While most studies show that most patients have a normal or high cardiac index with a hyperdynamic left ventricle, a minority of patients have a low cardiac output due to a range of causes. Comparative data from Belfort and colleagues also suggest that tissue oxygen consumption/extraction is both significantly lower in severe preeclamptics than in normal pregnant women, and delivery-dependent.12 Therefore, any reduction in oxygen flux (such as a fall in cardiac output or

haemodilution following intravenous infusion) is likely to impair tissue (and fetal) oxygenation further.

Maternal mortality data from the UK show that pulmonary oedema is a major hazard in preeclampsia. Predisposing factors include increased capillary permeability and a low colloid oncotic pressure, which will be aggravated by crystalloid infusion. In addition, a minority of preeclamptics have disproportionately high left atrial pressures or respond to quite modest volume load with marked elevations in left-sided filling pressures, indicating impaired left ventricular function. The rapid fluid shifts associated with spinal anaesthesia, and consequent aggressive intravenous fluid therapy, and the inevitable autotransfusion from the contracted uterus following delivery, make iatrogenic pulmonary oedema a particular risk.

Despite the dramatic anticipated consequences of spinal anaesthesia, however, it is unclear whether severe preeclamptics actually behave as expected. Indeed, it appears their behaviour may even be contrary to prediction. Almost 50 years ago, Assali and Prystowsky suggested that hypotension following sympathetic blockade was less common in preeclamptics than in normal pregnant women.14 In addition, a small study from Finland reported that preeclamptics (n=6) may have an exaggerated release of atrial natriuretic peptide following volume loading which could ameliorate the risks of acute fluid overload. 15 Of particular interest are several recent studies which report the use of spinal anaesthesia for caesarean section in a total of 287 preeclamptic women, and suggest that the technique is associated with relative haemodynamic stability and overall safety.1-6

However, the enthusiasm of some of the authors for spinal anaesthesia cannot always be fully supported by their data. The principal concerns are that the number of patients reported is small, not all had severe preeclampsia, the majority are from retrospective analyses of clinical practice and several of the studies have yet to be published in full, preventing critical evaluation. When assessing these studies, it is also important to look beyond the group mean response and note what happens to individual patients who have received spinal anaesthesia. One can deduce that a minority of patients did become significantly hypotensive, although there is no evidence to suggest it is any more common, or more dramatic, than with epidural anaesthesia.

The greatest body of support for the safety of spinal anaesthesia in preeclampsia comes from David Hood's group in the USA, who have used the technique successfully in over 130 patients and demonstrated remarkably similar (and safe) haemodynamic responses following both spinal and epidural techniques. In a 7-year retrospective analysis of over 200

preeclamptics, they found no difference in lowest blood pressure or ephedrine requirement between patients receiving either spinal or epidural anaesthesia for caesarean section. One slightly worrying difference was the higher intravenous fluid administration to patients receiving spinal anaesthesia, although whether this was due to increased requirement or anesthesiologist anxiety, is unclear. The publication of this study is eagerly awaited.

Despite certain caveats therefore, there is evidence that, in the right hands, spinal anaesthesia does not necessarily lead to catastrophic hypotension or major morbidity in severe preeclampsia. Epidural anaesthesia remains the technique of choice for caesarean section, and good inter-disciplinary communication between attentive anaesthetic and obstetric staff should usually enable an epidural catheter to be inserted where appropriate. However, the time is ripe for a cautious reassessment of the value of spinal anaesthesia for the relatively rare clinical situation where urgency does not allow for an epidural technique, when the choice lies between spinal and general anaesthesia.

Larger, prospective, preferably randomized, studies of spinal anaesthesia in both mild and severe preeclampsia are urgently needed. The haemodynamic effects and feto-maternal consequences of spinal anaesthesia need to be compared with the current benchmark technique, epidural anaesthesia. Since the perception of severe preeclampsia varies widely, and may include women with proteinuric hypertension and a range of other significant problems such as renal. hepatic or haemostatic dysfunction and HELLP or even eclampsia, future studies should include detailed evaluation of disease severity. Careful study design in terms of spinal technique, fluid management and haemodynamic monitoring will be important, as will early reporting of spinal-related maternal morbidity. and fetal outcome. In addition, experience with the use of combined spinal-epidural techniques may demonstrate useful compromises with respect to dosage, timing and haemodynamic effects.

Despite the recent encouraging data, the fact that there have been very few reports of spinal anaesthesia in preeclampsia in the literature should not be overlooked. Moreover, absence of published evidence of morbidity from spinal anaesthesia does not imply absence of risk. As always in this complex, bewildering and dangerous disease, the choice of anaesthetic technique must be based on assessment of the individual patient by a practitioner experienced in the pitfalls of preeclampsia. While there may well be occasions of urgency in some preeclamptic patients for whom the choice of spinal anaesthesia is considered safer than general anaesthesia, there is currently insufficient

evidence to support the widespread use of spinal anaesthesia in preeclampsia.

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