

Epidural compared with general anaesthesia for caesarean delivery in conscious women with eclampsia

J. Moodley^{a,*}, G. Jjuuko^a, C. Rout^b

Objective To compare retrospectively the outcome of caesarean section under epidural anaesthesia with that of general anaesthesia in “stable” women with eclampsia.

Design Retrospective review.

Method Over the five-year study period, there were 533 women with eclampsia and of these 66 women (12.4%), fulfilled the criteria of being ‘stable’. Of the 66 women, 37 received epidural, 27 general, and 2 spinal anaesthesia.

Results There were no major complications with either general or epidural anaesthesia. Epidural anaesthesia was associated with higher one-minute Apgar scores.

Conclusion This study indicates that both maternal and neonatal outcomes are not affected adversely by the use of epidural anaesthesia in selected cases of eclampsia.

INTRODUCTION

Hypertensive diseases of pregnancy constitute the most common cause of maternal mortality in South Africa, accounting for approximately 20% of maternal deaths¹. Inadequate antenatal care is responsible for much of the morbidity of pre-eclampsia and may contribute to mortality by failure to recognise early clinical signs, with delayed referral and consequent delayed appropriate obstetric intervention. Delayed referral is associated with progression of the disease. Women often present with severe disease, characterised by hypertensive crisis, generalised oedema and abnormal laboratory tests indicating multi-organ dysfunction, most significantly affecting the renal, haemostatic and hepatic systems. Such circumstances usually warrant termination of the pregnancy, which currently is the only cure for the condition. The need for expeditious delivery often requires operative intervention and caesarean delivery rates may be as high as 60% or more².

The woman with severe pre-eclampsia is a significant challenge to the anaesthetist. The combination of severe labile hypertension, intravascular volume depletion, and multiple organ dysfunction demands a haemodynamically stable technique. General anaesthesia may be asso-

ciated with a severe haemodynamic response to laryngoscopy and intubation that might increase the risk of intracranial haemorrhage and pulmonary oedema^{3,4}. This is in addition to the known risks associated with general anaesthesia in pregnancy (e.g. difficult intubation and pulmonary aspiration of gastric contents)⁴. Drug interactions may also be a problem, particularly between magnesium sulphate, neuromuscular blocking agents, calcium channel blockers and inhalational anaesthetics⁵. Regional anaesthesia on the other hand may be associated with severe hypotension, high motor neuronal blockade and the possibility of a convulsion occurring during the course of the procedure⁶. However, despite these potential problems, the relative haemodynamic stability conferred by epidural anaesthesia combined with decreased catecholamine concentrations and improved uteroplacental and peripheral perfusion make it the preferred choice for hypertension in pregnancy^{7–11}. Even spinal anaesthesia may be associated with less haemodynamic variability than general anaesthesia for caesarean section in pre-eclampsia, despite the more rapid onset of sympathetic blockade¹².

Anaesthesia in eclampsia, however, still provokes debate. Most anaesthetists consider regional anaesthesia inappropriate where there is an increased risk of loss of control of the airway, and where the risk of further convulsions following magnesium sulphate therapy is between 6% and 13%¹³. Raised intracranial pressure in eclampsia raises the possibility of cerebellar tonsillar herniation in association with dural puncture¹⁴. Nevertheless, the clinical condition of women with eclampsia varies considerably, from the haemodynamically stable, conscious and cooperative woman to the comatose woman with uncontrollable hypertension, anuria and coagulopathy. It would seem inappropriate to apply a general recommendation of anaesthetic care to such a

^aDepartment of Obstetrics and Gynaecology and MRC/UN Pregnancy Hypertension Research Unit, Nelson R Mandela School of Medicine, University of Natal, South Africa

^bDepartment of Anaesthetics, Nelson R Mandela School of Medicine, University of Natal, South Africa

* **Correspondence:** Professor J. Moodley, Department of Obstetrics and Gynaecology, University of Natal Medical School, Private Bag 7, Congella, 4013, South Africa.

wide clinical spectrum. The use of regional anaesthesia for the otherwise stable eclamptic may avoid the known hazards of general anaesthesia^{3,15,16}.

Anaesthetic policy in our unit has been to judge each case on its own merits and choice of anaesthetic technique for the relatively stable conscious eclamptic is based on factors such as the perceived risk of a difficult intubation, platelet count, fetal compromise and the preference of the woman. This retrospective study compares over five years the outcome of caesarean section under epidural anaesthesia with that of general anaesthesia in women with stable eclampsia.

METHODS

The hospital records of all women with eclampsia between January 1995 and September 1999 were reviewed. Eclampsia was defined as a convulsion occurring in a pregnant woman in association with a blood pressure of $>140/90$ mmHg. "Stable" eclampsia was defined as: 1. Glasgow coma score ≥ 14 ; 2. rapidly acting anti-hypertensive agent not required; 3. platelet count $\geq 100,000/\text{mm}^3$; 4. cooperative; 5. central venous pressure >5 cm H_2O ; 6. normal fetal heart rate pattern on electronic monitoring; and 7. no additional maternal or fetal complications.

All maternal, neonatal and anaesthetic complications were recorded. All women with eclampsia were treated using standard methods, previously described². Briefly, this entailed magnesium sulphate administered by continuous intravenous infusion of 2–3 g/h following an initial loading dose of 6 g started before delivery and continued for 24 hours postpartum, monitoring of the central venous pressure and administration of intravenous fluid (usually modified Ringer's lactate), at an infusion rate of 100 mL/h or to maintain a central venous pressure of 4–6 cm H_2O , and expeditious delivery. Women deemed unlikely to deliver within 6–8 hours or who had an obstetric indication were delivered by caesarean section.

Epidural anaesthesia was induced with an 18 g catheter introduced between the sacrum and fourth lumbar vertebra in the sitting position, using the loss-of-resistance to air or saline technique via a 16g Tuohy needle. Intravenous fluid was administered if necessary beforehand to increase the central venous pressure to 4–6 cm H_2O , but a fixed volume "prehydration" was not used. Following a test dose of 4–5 mL, anaesthetic solution was administered in increments to achieve a sensory blockade to at least T4. The solution used was either 0.5% bupivacaine with fentanyl 5 $\mu\text{g}/\text{mL}$, or lignocaine 2% with fentanyl 5 $\mu\text{g}/\text{mL}$ and epinephrine 5 $\mu\text{g}/\text{mL}$. Hypotension (defined as a decrease in systolic blood pressure by 30% from baseline or < 100 mmHg) was treated by increasing the rate of intravenous infusion of crystalloid and intravenous ephedrine in 5 mg increments.

General anaesthesia was administered using a modified rapid sequence induction. Following 3–5 minutes of pre-oxygenation, alfentanil 10–15 $\mu\text{g}/\text{kg}$ was administered intravenously followed one minute later by etomidate 0.2–0.3 mg/kg intravenously, magnesium sulphate 20–30 mg/kg intravenously and suxamethonium 1 mg/kg intravenously in rapid sequence. Cricoid pressure was applied with administration of the etomidate. Following intubation with a cuffed endotracheal tube and confirmation of its position by capnography and auscultation, anaesthesia was maintained using isoflurane 0.5–1.5% in oxygen and nitrous oxide (50:50) or oxygen and air (50:50) administered by mechanical ventilation, to an end-tidal partial pressure of carbon dioxide of 4–4.3 kPa. Muscle relaxation was maintained using suxamethonium by repeated bolus injection or continuous intravenous infusion, and monitored using a nerve stimulator at the wrist placed over the median nerve. Glycopyrrolate 0.4 mg was administered intravenously in the event of significant bradycardia (< 60 bpm). Following delivery of the infant, omnopon 15–20 mg was administered intravenously. At the end of the procedure, extubation was performed with the women conscious in the left lateral position.

All the women were transferred to the operating room in the left lateral position and received 30 mL 0.3 molar sodium citrate orally before transfer to the operating table. Following delivery, oxytocin 20 iu/L (in modified Ringer's lactate) was commenced by intravenous infusion and cefoxitin 2 g administered intravenously following an initial test dose. The women were discharged from the anaesthetic recovery room to a high dependency area for 24–48 hours for continued haemodynamic monitoring. (Fisher's Exact test and test Kruskal Wallis non-parametric ANOVA) were used for comparisons between the two groups. The study was performed with institutional ethical approval.

RESULTS

Over the five years there were 533 women with eclampsia (12.1 per thousand deliveries) and of these, 66 women (12.4%) fulfilled the criteria for "stable" eclampsia, and required operative delivery (Table 1). Of the 66 women, 37 received epidural anaesthesia, 27

Table 1. Eclampsia rates over the 5-year period. Total number of eclamptics ≈ 533 .

Year	No. eclamptics	No. deliveries	Eclampsia rate/ 1000 deliveries
1995	119	9 266	12.8
1996	103	9 757	10.6
1997	101	9 318	10.8
1998	83	7 545	10.8
1999	127	8 105	15.6

general anaesthesia and two spinal anaesthesia. For the purpose of this report, the 2 women who received spinal anaesthesia were excluded.

Table 2 shows the clinical data of the two groups of women. All women had hypertension and proteinuria but none required a rapidly acting hypotensive agent. Table 3 shows the neonatal outcome. There were two stillbirths in the epidural group and four in the general anaesthetic group. More babies ($n = 25$) had Apgar scores of >7 at one minute in the epidural group, than in the general anaesthesia group ($P = 0.034$). Of the babies admitted to the special care nursery, four in the epidural group and three in the general anaesthesia group required ventilatory support.

There was one maternal death. A 30 year-old woman, who had eclampsia at 38 weeks of gestation, received epidural anaesthesia. Her post-operative course was uneventful until the fourth post-operative day when she developed labile hypertension, refractory to standard antihypertensive therapy. While being investigated for underlying hypertensive disorders, she collapsed and died on the 10th post-operative day. The death was unrelated to anaesthesia; post-mortem examination subsequently revealed a ruptured aortic aneurysm.

There were no serious maternal complications specifically related to anaesthesia in either group. One woman who received epidural anaesthesia developed urinary

retention on the third day following a caesarean section for poor progress in labour. Transient hypotensive episodes occurred in five women during caesarean delivery under epidural anaesthesia; three were primigravidae, and two multigravidae. These episodes occurred after delivery and responded promptly to intravenous ephedrine. All five babies born to these mothers had one minute Apgar scores >7 .

DISCUSSION

It has been reported that for pregnancy the fatality rate directly attributed to anaesthesia between 1985 and 1990 in the United States was 17 times greater with general anaesthesia compared with regional anaesthesia¹⁷. The fatality rates with general and spinal anaesthesia for the varying degrees of severity of pre-eclampsia or eclampsia are, however, unknown. The risks may be even greater because general anaesthesia in severe pre-eclampsia and eclampsia has the specific hazards of laryngeal oedema and hypertensive response to laryngoscopy and intubation⁴. In general, epidural anaesthesia is the preferred method for caesarean delivery in pre-eclampsia⁵. Wallace *et al.*¹⁸ performed a randomised trial which compared the effects of general with combined epidural and spinal anaesthesia in severe pre-eclampsia and concluded that

Table 2. Clinical data of all stable eclamptics. IOL = induction of Labour; CS = caesarean section; CVP = central venous pressure; BP = blood pressure. Values are given as mean (SD), mean [range] or n {%}.

	Epidural anaesthesia ($n = 37$) Mean (Range or SD)	General anaesthesia ($n = 27$) Mean (Range or SD)	<i>P</i>
Age (years)	20.9 [15–34]	23 [16–35]	0.52
Parity			
0	29	14	0.06
1+	8	13	
Gestation (weeks)	32 [27–40]	33 [26–39]	0.97
No. of convulsions	2 [1–7]	3 [1–6]	0.85
Admission BP (mmHg)			
Systolic	168 (14)	174 (13)	
Diastolic	100 (10)	98 (12)	0.98
Peri-operative BP (mmHg)			
Highest			
Systolic	152 (12)	155 (14)	
Diastolic	97 (8)	100 (10)	0.09
Lowest			
Systolic	102 (11)	109 (8)	0.07
Diastolic	64 (12)	70 (13)	
Ephedrine for hypotension	5	0	0.07
CVP			
Highest	6 (2)	6 (3)	
Lowest	2 (2)	3 (2)	
Glasgow Coma Scale	14 [14–15]	14 [14–15]	
Platelet count			
Pre-operative	194 [119–269]	170 [112–350]	0.76
Post-operative	160 [105–370]	164 [101–420]	0.97
Indication for C/S			
Cervix unfavourable for IOL	30 {81}	20 {74}	
Poor progress in labour	7 {9}	7 {26}	0.83

Table 3. Obstetric and neonatal outcome. Values are given as *n*, *n* (%) or mean [range].

	Epidural anaesthesia <i>n</i> = 37	General anaesthesia <i>n</i> = 27	<i>P</i>
Fetal outcome			
Live birth	35 (95)	23 (85)	0.757
Stillbirth	2	4	
Apgar scores ≥ 7			
1 min	25 (71.4) ^a	10 (43.5) ^a	0.03
5 min	31 (88.6)	16 (69.3)	0.07
Birthweight (g)			
Live birth	2 329 [1260–2970]	2 053 [1050–3200]	0.826
Stillbirth	1 180 [930–1890]	1 240 [850–1900]	0.748
Neonatal outcome			
Special care nursery	12 (34)	7 (34.7)	0.987
Live (discharged home)	29 (82)	20 (87)	0.868
Neonatal death	6 (8)	3 (13)	
Maternal outcome			
Live	36	27	0.716
Dead	1	0	

^a *P* = 0.034.

regional anaesthesia is as safe as general anaesthesia. Similar findings have been reported by Hood *et al.*¹⁹ in a retrospective evaluation of epidural and spinal anaesthesia in women with severe pre-eclampsia who had had a caesarean section. Without entering the debate as to whether spinal or epidural anaesthesia should be used as the regional technique, our study demonstrates that epidural anaesthesia was not associated with major adverse events and was as acceptable as general anaesthesia for caesarean delivery in selected women with eclampsia. This, however was a retrospective study. We could not ensure that the women receiving epidural anaesthesia were similar in all respects to those receiving general anaesthesia. The decision to administer epidural anaesthesia was made by the individual anaesthetist caring for the woman. Nevertheless, both groups were fully conscious, cooperative and had platelet counts of $> 100,000/\text{mL}$, similar systemic and central venous pressures and neurological assessment scores at the onset of the operation.

In our study five women became hypotensive, all in the epidural group. None had required a rapidly acting anti-hypertensive agent before anaesthesia and all initially had a central venous pressure $> 5\text{cmH}_2\text{O}$. Treatment of the hypotension was relatively uncomplicated and all five women responded promptly to ephedrine. These findings are similar to those of Wallace *et al.*¹⁸ and Hood *et al.*¹⁹, who used ephedrine in 22%–30% and 23%–26% of women, respectively. In both these reports, ephedrine was administered before the infant was born, while in our study all five events occurred during the caesarean section following delivery of the baby. Concerns have been expressed about the use of ephedrine in severe pre-eclampsia because these women may be very sensitive to pressor agents. Several authors^{20,21} have found that increments of ephedrine given by bolus intravenous injections are a simple therapeutic measure against hypo-

tension in women with pre-eclampsia receiving epidural anaesthesia. Ephedrine gives better control of blood pressure with a significant reduction in the risk of nausea and vomiting, and in these studies was not associated with uterine hypertonicity or constriction of the uteroplacental blood supply.

Whether women receiving epidural anaesthesia required extra amounts of intravenous fluids cannot be determined from this review as the records were incomplete. Our protocol in eclampsia is to maintain central venous pressure, at 4–6 cm H₂O. None of the women in the study developed pulmonary oedema. The maximum central venous pressure recorded was no different between the groups. Protocols which demand fixed volumes of intravenous fluids to ensure adequate prehydration before regional anaesthesia may be associated with higher cardiac filling pressures and an increased risk of pulmonary oedema^{22,23}. In pre-eclampsia and eclampsia, the decreased plasma oncotic pressure and increased pulmonary permeability may further increase the risk of pulmonary oedema²³.

No woman in the epidural group had a further convulsion while on the magnesium sulphate regimen (upper 95% confidence limit for the risk of convulsions, 9.5%). The reported incidence of further convulsions in women receiving magnesium sulphate in the two arms of the Collaborative Eclampsia Trial was 13.2% and 5.7%¹³. However, in that trial 54% of women had two or more convulsions prior to entry. Also, an intravenous infusion regimen which may be associated with sub-therapeutic plasma magnesium concentrations was used in some women²⁴. The risk of further convulsions may be lower in fully conscious women whose cardiovascular systems are stable, who have not had repeated convulsions and who are receiving either intramuscular magnesium sulphate or an intravenous infusion of 2–3g/hour.

Although the risk of convulsion during the surgical procedure is low, continuous vigilance is necessary and the drugs and equipment must be made available for general anaesthesia.

In our study a number of the women were in labour prior to caesarean section but did not demonstrate evidence of acute fetal compromise. However, there were a number of low birthweight and preterm infants delivered in each group. These infants are at increased risk and may require considerable intervention in the neonatal special care unit. Requirement for immediate resuscitation, indicated by a low Apgar score at one minute, more commonly occurred in the infants of mothers receiving general anaesthesia. While there was no significant difference in the incidence of low Apgar scores at five minutes, this is largely due to the small numbers in each group. It is possible that the number of infants requiring further resuscitation may be as much as two to three times higher following delivery under general anaesthesia compared with epidural anaesthesia. However, their subsequent clinical course in the neonatal intensive care unit showed no continued advantage in the babies of mothers who received epidural anaesthesia. It is likely that the effects of prematurity, low birthweight and placental insufficiency outweigh any potential advantage conferred by choice of anaesthesia.

In conclusion, this retrospective comparison of epidural and general anaesthesia in a selected group of women with eclampsia indicates that both maternal and neonatal outcomes are not adversely affected by the use of epidural anaesthesia. The data suggest that epidural anaesthesia may be of advantage to the neonate but only in the immediate post-delivery period. The use of epidural anaesthesia avoided the known risks of general anaesthesia and was associated with a low incidence of relatively mild hypotension and no major complications. No major complications were observed in general anaesthesia also. However, the small number of women studied could not exclude an incidence of complications less than 12.8% with 95% confidence. Nonetheless, this study shows that up to 12% of women with eclampsia in an under-resourced setting may present in a haemodynamically stable state, and behave clinically and biochemically as most women with severe pre-eclampsia. In such circumstances, epidural anaesthesia for caesarean section is justified.

References

1. Moodley J, (for the National Committee on Confidential Enquiries into Maternal Deaths). Saving mothers: Report on Confidential Enquiries into Maternal Death in South Africa. *S Afr Med J* 2000;**90**:362–363.
2. Moodley J, Daya P. Eclampsia—a continuing problems in the developing world. *Int J Gynecol Obstet* 1993;**44**:9–14.
3. Lavies NG, Meiklejohn BH, May AE, Achola KJ, Feu D. Hypertensive

- and catecholamine response to tracheal intubation in patients with pregnancy induced hypertension. *Br J Anaesth* 1989;**63**:429–434.
4. Mroz LA. Hypertensive disorders of pregnancy. *Anes Clin N Am* 1999;**17**:679–691.
 5. Ramanathan J, Coleman P, Sibai B. Anaesthetic modification for haemodynamic and neuroendocrine response to caesarean section delivery in severe pre-eclampsia. *Anes Anal* 1991;**73**:772–779.
 6. Reynolds F. Epidural analgesia in obstetrics. Pros and cons for mother and baby. *BMJ* 1989;**23**:299.
 7. Hogg B, Hauth JC, Caritis SN, et al. Safety of labour epidural for women with severe hypertensive disease. *Am J Obstet Gynecol* 1999;**181**:1096–1101.
 8. Jouppila P, Jouppila R, Holleman A, et al. Lumbar epidural to improve intervillous blood flow during labour in severe pre-eclampsia. *Obstet Gynecol* 1982;**59**:158–161.
 9. Ramos-Santos E, Devoe LD, Wakefield ML, et al. The effects of epidural anaesthesia on Doppler velocimetry of umbilical and uterine artery in normal and hypertensive patients during active labour. *Obstet Gynecol* 1991;**77**:200–206.
 10. Moore TR, Key TC, Risner LS, Resnik R. Evaluation of use of continuous lumbar epidural anaesthesia for hypertensive pregnant women in labour. *Am J Obstet Gynecol* 1985;**152**:404–412.
 11. Abboud T, Artal R, Sarkis F, et al. Sympathoadrenal activity, maternal, fetal and neonatal responses after epidural anaesthesia in preterm patients. *Am J Obstet Gynecol* 1982;**144**:905–912.
 12. Rout CC, Ward S, Roche DA. Haemodynamic variability at emergency caesarean section in hypertensive patients – spinal versus general anaesthesia. *Anesthesiology* 2000;**92**:A50.
 13. The Eclampsia Trial Collaborative Group. Which anticonvulsant for women with eclampsia? Evidence from the Collaborative Eclampsia Trial. *Lancet* 1995;**345**:1455–1463.
 14. Richards AM, Moodley J, Graham DI, Bullock MRR. Active management of the unconscious eclamptic patient. *Br J Obstet Gynaecol* 1986;**93**:554–562.
 15. Brimacombe J. Acute pharyngolaryngeal oedema and pre-eclampsia-eclamptic toxemia. *Anaes Intensive Care* 1992;**20**:97–98.
 16. Jouppila P, Kuikka J, Jouppila R, Hollman A. Effect of induction of general anaesthesia for caesarean section on intervillous blood flow. *Acta Obstet Gynaecol Scand* 1979;**58**:249–253.
 17. Hawkins JL, Koonin LM, Palmer SK, Gibbs CP. Anaesthesia related deaths during obstetric delivery in the United States, 1979–1990. *Anesthesiology* 1997;**86**:277–284.
 18. Wallace DH, Leveno KJ, Cunningham FG, et al. Randomised comparison of general and regional anaesthesia for caesarean delivery in pregnancies complicated by severe pre-eclampsia. *Obstet Gynecol* 1995;**86**:193–199.
 19. Hood DD, Curry R. Spinal versus epidural anaesthesia for caesarean section in severely pre-eclampsia-eclamptic patients: a retrospective survey. *Anesthesiology* 1999;**90**:1276–1283.
 20. Ramanathan S, Grant GJ. Vasopressor therapy for hypotension due to epidural anaesthesia for caesarean section. *Acta Anaesthio Scand* 1988;**32**:559–565.
 21. Sipes SL, Chestnut DH, Vincent RD, et al. Which vasopressor should be used to treat hypotension during magnesium sulphate infusion and epidural anaesthesia? *Anesthesiology* 1992;**77**:101–108.
 22. MacLennan FM, MacDonald AF, Campbell DM. Lung water during puerperium. *Anaesthesia* 1987;**42**:141–147.
 23. Benedetti TJ, Kates R, Williams V. Haemodynamic observations in severe pre-eclampsia complicated by pulmonary oedema. *Am J Obstet Gynecol* 1985;**152**:330–334.
 24. Sibai BM, Graham JM, McCubbin JH. A comparison of intravenous and intramuscular magnesium sulfate regimens in pre-eclampsia. *Am J Obstet Gynecol* 1984;**150**:728–733.

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