Eclampsia — a method of management

A preliminary report

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Summary

Sixty-seven cases of eclampsia treated at King Edward VIII Hospital, Durban, during a 1-year period are reviewed. A protocol for the management of eclampsia, based upon our experience, is presented.

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It is sometimes informative to review the management of a condition, particularly if this leads to improved understanding and hence better management. This is especially the case as regards a condition like eclampsia. The aetiology is unknown, and which organ is going to bear the brunt of the disorder in a particular patient is usually unpredictable. Management is therefore aimed at whichever symptoms or signs are most dangerous to the patient at that time. Even this can be hazardous; in our endeavour to rectify matters a multitude of drugs may be used and the problems of polypharmacy and drug interactions arise.

It is not valid to compare results of clinical studies on eclampsia carried out at different centres, as local factors other than the treatment regimen often influence the outcome (e.g. the incidence of underlying renal disease and unbooked, elderly, grand multiparas). With these thoughts in mind a prospective analysis of all patients with eclampsia presenting at King Edward VIII Hospital, Durban, between January and December 1980 was undertaken. The aims of management during the study were to: (*i*) control and prevent further convulsions; (*ii*) monitor all systems and treat abnormalities accordingly; (*iii*) achieve normotension; and (*iv*) resort to caesarean section if delivery was unlikely to occur within 6 - 8 hours.

Analysis of the patients

There was a total of 23 902 deliveries during the 12-month study period, 16 276 in the central hospital and 7 626 at the peripheral clinics. Sixty-seven eclamptic patients were seen during the same period, giving an 'eclampsia rate' of 2,3/1 000 deliveries. Eight patients died from eclampsia during the period, a mortality rate of 11,9%.

Parity (Table I). Like pre-eclampsia, eclampsia is commonest in primigravidas, and 44 (65%) of our patients were primigravid. Of relevance is the fact that 3 of 6 patients who had had 5 or more pregnancies died.

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TABLE I. PARITY OF ECLAMPTIC PATIENTS

Parity	No. of patients	Deaths No.	Deaths %
Primigravid (0)	**44**	**3**	**6,8**
1 - 4	**17**	**2**	**11,7**
5+	**6**	**3**	**50,0**
Total	**67**	**8**	

Age (Table II). The majority were aged between 16 and 20 years. Of those who died from eclampsia, 2 were 16 - 20 years old, 1 was 23 years old and 5 were older than 30. It would therefore appear that the older patient is more likely to die from eclampsia, as half of the 10 patients older than 30 years died.

TABLE II. AGE OF ECLAMPTIC PATIENTS

	Age (yrs) ≤15	16-20	21-25	26-30	31+	Total
Total No.	**2**	**39**	**12**	**4**	**10**	**67**
No. of deaths	**0**	**2**	**1**	**0**	**5**	**8**

Antenatal care. Forty-three eclamptic patients had received no antenatal care. Of those who died 7 had received no antenatal care, while the eighth had received inadequate antenatal care, having paid only one early antenatal visit. It should be noted that even among the booked patients antenatal care was inadequate; 13 patients had made only one visit for antenatal care.

Period of gestation. In 60 of the cases (90%), the duration of gestation at the onset of eclampsia was 30 weeks or more; 20 were in the 30th - 35th week, while 40 were more than 36 weeks' pregnant. Seven patients were less than 30 weeks' pregnant.

Number of convulsions experienced (Table III). Sixty-one patients experienced convulsions during the antepartum period, 3 during the intrapartum period and 3 after delivery. Only 3 patients experienced their initial convulsions in hospital, 2 in the intrapartum period and 1 after delivery.

TABLE III. NUMBER OF FITS IN EACH PATIENT

No. of fits	No. of patients	No. of deaths
1	**20**	**0**
2	**18**	**1**
3	**12**	**1**
4	**7**	**4**
5	**7**	**2**
≥6	**3**	**0**

Level of consciousness at admission (Table IV). Five of the patients who died were deeply unconscious at admission —unconsciousness is therefore a bad prognostic sign.

TABLE IV. LEVEL OF CONSCIOUSNESS AT ADMISSION

	No. of patients	No. of deaths
Fully conscious	5	
Responsive but not deeply unconscious	55	3
Deeply unconscious	7	5
Total	67	8

Drug therapy. All of the patients received magnesium sulphate ($MgSO_4$), diazepam and dihydrallazine. Three of the patients who died had also been given either reserpine, labetalol or diazoxide.

Blood pressure. Seven of the patients who died had had a diastolic blood pressure of > 120 mmHg at admission.

Proteinuria. Proteinuria of grade 2+ to 3+ was present in all patients.

Fetal condition and mode of delivery. On admission 8 patients presented with an intra-uterine fetal death; of these 3 were delivered by caesarean section. Among the infants of those presenting with live fetuses there were 3 neonatal deaths (2 in infants weighing more than 1 500 g). Thirty mothers delivered vaginally and 37 by caesarean section.

Complications. Two patients had oliguria, 1 requiring haemodialysis. Eclampsia was associated with abruptio placentae on 2 occasions, with puerperal psychosis in 2 patients, urinary tract infection in 3 and chest infection in 3.

Autopsies. Autopsies were performed in only 3 cases. In 1 there was an obvious cerebral haemorrhage, and in the other 2 there was evidence of cerebral oedema.

Discussion

The incidence of eclampsia of 2,3/1 000 deliveries at King Edward VIII Hospital, Durban, is high. This is probably due to the fact that King Edward Hospital is a large referral hospital for the whole Natal coast. Nevertheless, it should be pointed out that eclampsia is preventable, and the very fact that there are any eclamptics at all is an indictment of our inadequate antenatal services.

Perinatal mortality

Crichton *et al.*[1] demonstrated an improvement in perinatal mortality by early recourse to caesarean section. In 240 abdominal deliveries the perinatal mortality was 35%, compared with 47% in 83 vaginal deliveries. The improved prospects of babies weighing between 1 360 g and 2 708 g was particularly notable. In this series the overall perinatal mortality rate was 16,4%. There were 3 perinatal deaths among the live infants of 34 patients who underwent caesarean section, giving a corrected perinatal mortality rate of 8,8%. One of the infants who died weighed 1 200 g; the other 2 weighed over 1 500 g. Altogether, 37 caesarean sections were performed, 34 in patients early in labour with live babies. The other 3 caesarean sections were performed solely on behalf of the mother's health. Thus, at King Edward VIII Hospital the policy of early caesarean section if delivery is unlikely to occur within 8 hours seems justifiable.

Maternal mortality

The maternal mortality rate of 11,9% (8/67) is very high, but it should be realized that King Edward VIII Hospital is a referral centre and many patients arrive in a neglected condition because of the lack of immediate medical supervision and difficulties as regards transport. Seven patients were admitted in a deeply unconscious state, and of these 5 died.

Factors associated with increased maternal mortality

Lack of antenatal care. Forty-three of the 67 patients had received no antenatal care, and of the 24 who had booked 13 had made only a single 'booking' visit to the antenatal clinic. Seven of the 8 patients who died from eclampsia had received no antenatal care, while the eighth had made only one visit.

Age and parity. These are important risk factors; of the 8 patients who died, 5 were over the age of 30. Three of 6 patients who had had 5 or more pregnancies died.

Prolonged labour. In 2 cases of maternal death prolonged labour was an associated feature. A 33-year-old woman, para 4, had a 24-week pregnancy terminated by intra-amniotic administration of oxytocin and prostaglandins. This termination lasted 18 hours and resulted in a ruptured uterus. The patient had a cardiac arrest during resuscitation. In the other patient the induction-delivery interval was 14 hours. She was a primigravida who presented in early labour in a semiconscious state; delivery by forceps was followed by a fatal postpartum haemorrhage.

Polypharmacy. In 3 cases of maternal death a combination of $MgSO_4$, dihydrallazine and either reserpine, labetalol or diazoxide had been used; a number of reports have stated that combinations of diazoxide and other vasodilatory or catecholamine-depleting agents may result in severe hypotension.[2] Polypharmacy may lead to drug interactions with uncertain effects, and could possibly have played a part in these deaths. On the other hand, inability to control the hypertension with a single agent may indicate severe disease, thus resulting in higher mortality. Combinations of antihypertensive drugs should be used only where a single agent is ineffective. The possibility of synergism between drugs or the potentiation of the action of one drug by another must be borne in mind and dosages suitably adjusted.

Number of fits. The greater the number of fits a patient has, the greater the risk of death (Table III). Six patients who died had had 4 or more fits.

Unconsciousness at admission. Only 2 of 7 patients who were unconscious at admission survived. These 2 patients were young primigravidas and both regained consciousness within 48 hours of delivery.

Summary of risk factors. It would seem that the unbooked, parous patient over the age of 30, if admitted when unconscious, is at high risk. If, in addition, polypharmacy is used and labour is prolonged with haemorrhage and/or operative shock, the chances of maternal death are great.

Type of anaesthesia

Of the 37 patients delivered by caesarean section, 35 received general anaesthesia and 2 received epidural anaesthesia. No patient delivering vaginally received anaesthesia. General anaesthesia was administered to all patients with impaired consciousness. In patients with diastolic blood pressure readings of about 110 mmHg a 0-1% solution of trimetaphan camsylate was used to blunt the hypertensive response to laryngoscopy and intubation. As all patients had received $MgSO_4$, smaller doses of muscle relaxants than usual were used. Whenever possible neuromuscular blockade was not reversed at the end of the operation and the patients were transferred to the intensive care unit. Epidural anaesthesia with bupivacaine 0,5% was used in conscious patients undergoing caesarean section. No marked fall in blood pressure occurred in either case. Morphine 2 mg in 10 ml 5% dextrose water administered via an indwelling epidural catheter

was used to provide postoperative analgesia. Patients operated on under epidural anaesthesia fared very well postoperatively. Four of the 8 patients who died had had a caesarean section performed under general anaesthesia. In none of these patients was death attributable to the anaesthetic itself.

Postmortem findings

Permission to perform autopsies was obtained on 3 occasions only; in 2 cases minimal lesions were found, except for cerebral oedema.

Raised intracranial pressure

We believe that a raised intracranial pressure (ICP) may be an important and lethal feature of eclampsia. Our management of eclampsia would be markedly modified if this was so. Marked depression in the level of consciousness in the absence of excessive sedation or a recent convulsion in a patient suffering from eclampsia suggests raised ICP, which may be due to cerebral oedema, cerebral hyperaemia or an intracerebral haemorrhage.

If ICP is raised in eclampsia it is important to have some idea of its significance and management. The intracranial contents (brain solids, brain water, blood and cerebrospinal fluid (CSF)) are neither compressible nor distensible. An increase in the volume of any of these without a corresponding decrease in the volume of another will increase ICP.

In a patient with normal CSF pathways and a space-occupying lesion the initial and most important compensation for increasing cerebral volume is made by displacement of CSF from the head into the compliant spinal sac.[3] There may also be some reduction in the rate of formation and reabsorption of CSF and a decrease in the water content of the normal brain. The patient at this stage is said to be at point A (Fig. 1), which shows the relationship between increasing intracranial volume and ICP. At this point any acute rise in intracranial volume would cause a shift of CSF into the spinal sac and a shift of blood out of the spinal epidural veins and only a small transitory rise in ICP. The 'brain' at this stage is said to be relatively 'compliant'.

As intracranial volume increases the compensatory mechanisms are exhausted. Eventually a point is reached (point B in Fig. 1) at which a further increase in the volume of any of the intracranial contents will be associated with a very steep rise in ICP.[3]

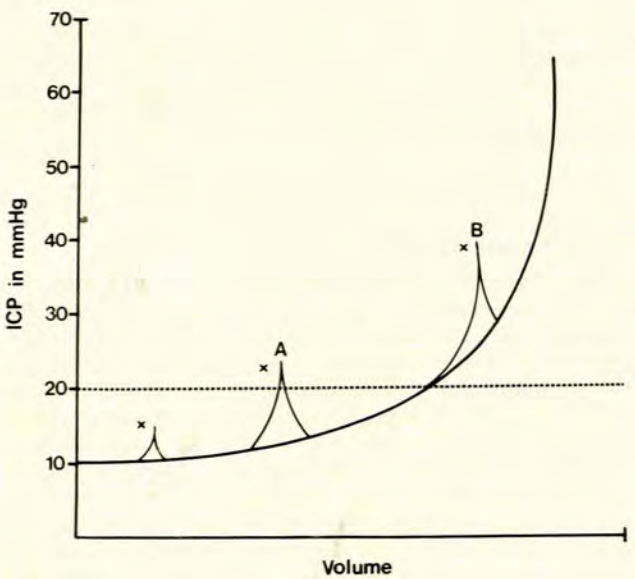

Fig. 1. Brain 'compliance' lessens with increasing intracranial volume (x depicts rises in ICP with minimal increases in intracranial volume).[3]

In the normal, healthy, conscious subject ICP will change with position, falling when the head is raised and rising when it is lowered. It will rise sharply and transiently during coughing or straining, and it may rise a little during sleep. The cerebral arterioles are very sensitive to changes in the partial serum carbon dioxide pressure (P_{CO_2}), and a small rise in P_{CO_2} results in significant cerebral arteriolar dilation and a consequent increase in cerebral blood flow (CBF). This increase in CBF tends to raise the ICP by two mechanisms: it causes an immediate increase in the intracranial blood volume and, secondarily, an increase in brain water. This latter follows from the increase in filtration pressure in the cerebral capillary which, in accordance with Starling's hypothesis (Fig. 2), produces an increase in tissue fluid formation and a reduction in its reabsorption.

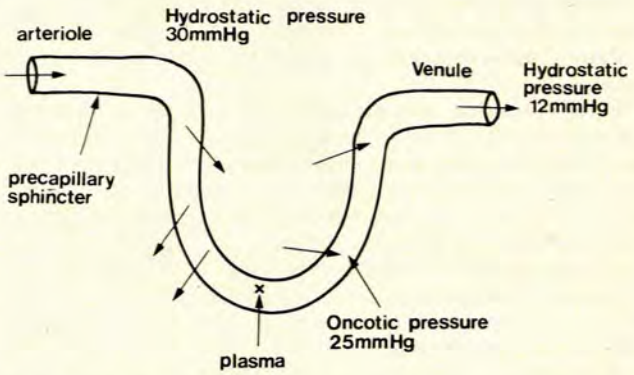

Fig. 2. Starling's hypothesis: the formation and reabsorption of tissue fluid. At the anterior end of the capillary the hydrostatic pressure is 30 mmHg. The oncotic pressure is 25 mmHg, there is a filtration pressure of 5 mmHg and fluid leaves the capillary. Arteriolar dilation increases the filtration pressure and tissue fluid formation. A rise in venular pressure results in a decrease in reabsorption.

The CBF remains remarkably constant despite considerable variations in systemic blood pressure. This is because the cerebral arterioles constrict when the systemic blood pressure rises and dilate when it falls. The underlying mechanism is probably independent of nervous control. Fig. 3 shows the normal relationship between CBF and arterial pressure, the CBF remaining constant between mean arterial pressures of 60 and 160 mmHg. Autoregulation of the CBF is impaired in patients with hypertension, the curve being shifted to the right. CBF is reduced by severe falls in blood pressure, overbreathing (which lowers P_{CO_2} and causes cerebral arteriolar constriction) and increased ICP.

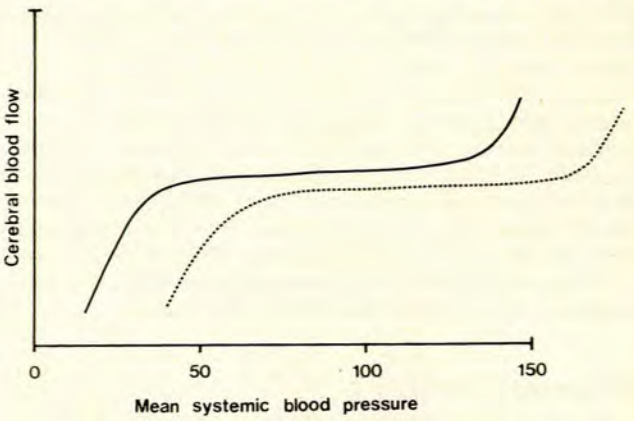

Fig. 3. The autoregulation of CBF. — = normotensive patient, ---- = hypertensive patient (curve shifted to right).[3]

The cerebral perfusion pressure (CPP) is the pressure available to push blood through the cerebral vascular tree: it is equal to the mean arterial pressure minus the ICP. A CPP of at least 30 mmHg is probably essential for the maintenance of cerebral viability.[3] As the ICP increases the mean arterial pressure must rise to maintain the CBF.

Starling's hypothesis (Fig. 2) provides the most useful model in a consideration of the pathogenesis of cerebral oedema. Cerebral oedema is most commonly caused by arteriolar dilation and increased capillary permeability. Potent causes of arteriolar dilation include a rise in P_{CO_2}, a fall in partial arterial oxygen pressure, a fall in pH, an accumulation of vaso-active metabolites and products of inflammation. A rise in ICP may also lead to cerebral vasodilation. This may be an intrinsic response of arteriolar muscle to a reduction in transmural pressure, or result from local accumulation of carbon dioxide and other metabolites due to impaired tissue perfusion. A rise in venous pressure may also initiate cerebral oedema.

From the above it can be seen that excessive lowering of the blood pressure may result in cerebral ischaemia in the eclamptic patient as a result of two mechanisms: (*i*) shift of the CBF autoregulatory curve to the right in hypertension (Fig. 3); and (*ii*) a rise in mean arterial pressure in order to maintain adequate cerebral perfusion in the presence of raised ICP.

In addition, the following factors should be avoided: (*i*) a mean arterial pressure in excess of the upper limit of autoregulation (this would lead to increased CBF and cerebral oedema, both of which would further increase ICP); and (*ii*) other factors raising ICP, including raised P_{CO_2}, coughing and straining, and the 'head-down' position.

Management of eclampsia (Table V)

In the absence of a clear understanding of the aetiology and pathophysiology of eclampsia, treatment must be symptomatic. The following protocol is based upon our experience and observations. The principles of management are: (*i*) provision of intensive care; (*ii*) control of fits; (*iii*) judicious lowering of the blood pressure; (*iv*) early delivery; and (*v*) continuation of sedation and blood pressure control for at least 48 hours after delivery.

Ideally this condition should be managed by a team familiar with hypertension and its complications in pregnancy. The team should consist of: (*i*) an obstetrician; (*ii*) an anaesthetist who has had training in intensive care and is conversant with the particular problems of pregnancy and parturition; (*iii*) 2 nurses, 1 trained in the intensive monitoring of patients, and a midwife; (*iv*) a neurosurgeon, should ICP monitoring be required; and (*v*) a paediatrician.

Management can be divided into: immediate management, management of labour/delivery and postpartum care. In addition, patients can be classified into two major categories: those with minimal or no impairment of consciousness, and those with marked impairment of consciousness.

Immediate management — minimal or no impairment of consciousness

Patients with minimal or no impairment of consciousness require control of convulsions, lowering of blood pressure and cardiovascular monitoring.

For control of convulsions and prevention of further convulsions, $MgSO_4$ is used in the following manner:

Give $MgSO_4$ 4 g slowly, intravenously, in not less than 4 minutes (4 g = 8 ml of a 50% solution diluted with 12 ml of distilled water). Follow immediately with 10 g by deep intramuscular injection, 5 g into each buttock (10 g = 20 ml of a 50% solution). Then give 5 g (10 ml of 50% solution) intramuscularly

TABLE V. FLOW SHEET: MANAGEMENT OF ECLAMPSIA

Eclampsia*

The conscious patient (minimal or no impairment of consciousness)

↓

$MgSO_4$ regimen

↓

Control BP with hydrallazine to 100-105 mmHg diastolic

↓

Termination of pregnancy

↙ ↘

Viable fetus

↓

If delivery unlikely in 6 hours

↓

C/S, otherwise induce labour

Non-viable fetus/intra-uterine death

↓

If delivery unlikely in 12 hours; if fits recur; if BP problematic

↓

C/S, otherwise induce labour

The unconscious patient

1. **$MgSO_4$ regimen**
2. **Direct monitoring of arterial BP, central venous pressure**
3. **Unless delivery imminent, terminate the pregnancy by C/S under general anaesthesia. Lower BP judiciously before induction of anaesthesia.**
4. **After delivery: continue $MgSO_4$ therapy and manage patient in an intensive care unit.**

* Investigations to be performed on admission: full blood count, measurement of urea and electrolyte values, estimation of blood gases, clotting profile — platelets, fibrinogen value, crude clotting time — chest radiography.
C/S = caesarean section; BP = blood pressure.

every 4 hours. Before each dose, ensure: (*i*) that the urine output is greater than 30 ml/h; (*ii*) that the patellar reflexes are present; and (*iii*) that respiration is adequate. If any one of these parameters is not within the normal range, withhold that dose of $MgSO_4$. Continue $MgSO_4$ for 24 hours post partum.

Note that the antidote is intravenous calcium gluconate 10 ml administered slowly, and that intravenous diazepam 10 mg in a bolus injection can be given if the patient is having convulsions at admission or is extremely restless, while waiting for the $MgSO_4$ to take effect.

Fits occurring within 20 minutes of the administration of $MgSO_4$ are usually brief and do not recur. Further drug therapy is therefore not indicated. However, if the patient has a convulsion 20 minutes after the administration of $MgSO_4$ or a third fit, give another 2 g intravenous $MgSO_4$ slowly. If the patient fails to respond to this therapy a short-acting intravenous hypnotic is called for. Our preference here is for alphathesin.

$MgSO_4$ was used in the study because of the dangers of diazepam to the fetus and mother. Our past experience with the use of diazepam in eclampsia has shown that infants born to mothers given large doses of diazepam are usually flaccid, suffer from respiratory depression and are hypothermic. Mothers who received large doses of diazepam were deeply sedated and took a number of days to recover. During this period they were prone to aspiration of stomach contents and other respiratory problems. No mother or infant suffered from magnesium toxicity at the dosage range of $MgSO_4$ used. We have found that $MgSO_4$ is highly effective in reducing restlessness in the patient with impaired consciousness. In the conscious patient its use is not associated with deep or prolonged sedation.

$MgSO_4$ is generally thought to have both a peripheral and a central action. Peripherally it results in a neuromuscular blockade, while centrally it acts by cortical suppression of neuronal activity.

The $MgSO_4$ administration technique described above is that recommended by Pritchard.[4] $MgSO_4$ can also be given by continuous infusion of from 1 g to 1,5 g/h after a primary intravenous dose of 2-4 g.[5] Continuous infusion is advocated because the intramuscular injections are painful, even with an admixture of procaine. Repeated intramuscular injections are also said to lead to abscesses, but none have been seen at King Edward VIII Hospital. The disadvantage of continuous infusion is the absolute necessity for close monitoring because of the risk of respiratory arrest. Lipsitz[6] compared the infants of women treated according to our intramuscular regimen with infants whose mothers had been treated by continuous intravenous infusion of $MgSO_4$ 1,5 g/h. He concluded that after maternal intramuscular treatment the newborn infant is not likely to be compromised, whereas signs of hypermagnesaemia may be expected if continuous intravenous infusion is used, especially if for more than 24 hours. It is best to use a regimen that one is familiar with and that suits a particular hospital.

Lowering of blood pressure. As regards blood pressure, rapid and excessive lowering may be disadvantageous to both mother and fetus, while polypharmacy is to be avoided wherever possible. The blood pressure should be lowered to between 100 and 105 mmHg diastolic. If the pulse rate is less than 120/min, dihydrallazine 12,5 mg diluted in 10 ml of water is slowly administered intravenously over 10 minutes. Repeated doses of dihydrallazine 6,25 - 12,5 mg can be given until the desired effect is achieved. If tachycardia develops and the patient requires further antihypertensive therapy, a cardioselective ß-adrenergic blocker should be used in combination with dihydrallazine in order to decrease the pulse rate. In this rare situation propranolol 1 mg intravenously is used in combination with dihydrallazine. If the patient is admitted with a pulse rate above 120/min (this is uncommon) diazoxide should be used at a dosage of 60-100 mg by rapid bolus injection every 5 minutes until the desired effect is achieved. Because interaction between diazoxide and dihydrallazine may lead to an extreme lowering of blood pressure and collapse, this drug combination should not be used.[7]

Monitoring of the cardiovascular system. The cardiovascular system needs intensive monitoring because the eclamptic patient is haemodynamically unstable. The heart rate is measured continuously using an ECG monitor. The blood pressure should be monitored continuously as marked changes may occur from minute to minute in hypertension in pregnancy. This can be done most conveniently by the use of either an intra-arterial line or an automatic non-invasive technique. Direct intra-arterial measurement is recommended for the restless and deeply unconscious patient.

Central venous pressure should be monitored in all patients. We use a 'drum cartridge catheter' inserted via an antecubital vein. A bedside chest radiograph is used to check the position of the catheter tip. Hypovolaemia is a well-known feature of pre-eclampsia and especially of eclampsia, and the temptation persists to overcome the hypovolaemia by infusing iso-osmotic or hyperosmotic fluids. Unfortunately this may cause circulatory overload unless there is concomitant relief of the vasospasm responsible for the decreased capacity of the intravascular compartment. Central venous pressure monitoring is therefore essential to prevent dangerous underfilling or overfilling. A balanced salt solution such as Ringer's lactate solution is used at King Edward VIII Hospital. The rate of infusion is such that the urine output is at least 0,5 ml/kg/h. Central venous pressure monitoring is adequate in most instances, but Swan-Ganz catheterization may be required to distinguish between cardiogenic and non-cardiogenic pulmonary oedema and in the management of left ventricular failure. Where facilities for continuous monitoring do not exist, the pulse rate, blood pressure and central venous pressure should be measured every 15 minutes.

Immediate management — marked impairment of consciousness

After administration of the 'loading dose' of $MgSO_4$ these patients should have their diastolic blood pressure lowered to 100-110 mmHg. It is postulated that the ICP is raised and that an excessive lowering of the blood pressure may reduce the CBF. This will in turn lead to a retention of metabolites and a further rise in ICP. The blood pressure should be lowered by giving intravenous dihydrallazine 12,5 mg slowly. If it is difficult to maintain an airway or if ventilation is inadequate the patient should be intubated and ventilated if necessary. Respiratory obstruction and hypoventilation lead to hypoxia and carbon dioxide retention. In addition to the obvious detrimental effects of these respiratory changes on both mother and fetus, a raised ICP will be further elevated. Arrangements should be made for urgent caesarean section. After delivery the patient should be managed in an intensive care unit. In order to determine whether or not these patients have a raised ICP, we intend transferring them to a neurosurgical unit for computed tomography. If this demonstrates cerebral oedema the ICP will be monitored with an extradural pressure transducer. Raised ICP, if present, will also be treated with steroids and/or diuretics.[3]

Further steps in management

The following are additional procedures and investigations: an indwelling Foley catheter, size 12/14F, is inserted into the bladder. Urine output should be measured hourly. The use of diuretics to clear peripheral oedema has not proved beneficial they may reduce blood volume and further impair uteroplacental and renal perfusion.[8] In addition, the effect of diuretics on electrolyte homeostasis, particularly of potassium, should discourage their routine use. The use of potent diuretics at King Edward VIII Hospital has as yet been limited to the treatment o

pulmonary congestion and incipient renal failure.[9] Fluids should be administered in sufficient quantity to produce a urine output of 0,5 ml/kg/h with the proviso that circulatory overload is avoided.

Investigations to be performed on admission include full blood count, measurement of urea and electrolyte values, the fibrinogen value, crude clotting time and arterial blood gases and chest (bedside unit) radiography. Disseminated intravascular coagulation does occur in pre-eclampsia/eclampsia, although no patient in this series had an obvious defect. If the clotting profile is initially normal the crude clotting time should be monitored every 2-4 hours. The level of consciousness should be monitored at 15-minute intervals.

Management of labour

All deeply unconscious patients should be delivered by caesarean section, irrespective of fetal viability, unless delivery is imminent. A caesarean section should be performed in all other eclamptic patients only if the fetus is viable and unlikely to be delivered within 4-6 hours, or if: (*i*) fetal distress is present; (*ii*) convulsions persist; (*iii*) hypertension is refractory to treatment; or (*iv*) oliguria supervenes.

At King Edward VIII Hospital a viable fetus is regarded as one which weighs more than 1 500 g. In the patient who is fully conscious or who has minimal impairment of consciousness and who is carrying a dead or non-viable fetus, vaginal delivery should be attempted. The attempt at vaginal delivery should be abandoned if: (*i*) delivery is unlikely to occur within 12-14 hours; (*ii*) convulsions persist; (*iii*) hypertension is resistant to therapy; or (*iv*) oliguria supervenes.

Ergometrine should not be used in the third stage of labour as it may elevate the blood pressure further. Instead, oxytocin 20 IU in a litre of Ringer's lactate solution should be given intravenously in a titrated form (10 drops/min). The second stage should be assisted by forceps in all eclamptic patients having a vaginal delivery, in order to minimize maternal efforts at bearing down and prevent further increases in blood pressure.

Analgesia/anaesthesia. Continuous segmental lumbar epidural blockade is the method of choice for pain relief during labour in patients able to co-operate, provided there is no underlying coagulation defect. This form of analgesia is not suitable for the restless, unco-operative patient, and here systemic analgesia is preferred if labour is to be allowed to continue. As regards the patient who is fully conscious, co-operative and requires a caesarean section, regional anaesthesia in the form of an epidural is practical and avoids many of the cardiovascular and pulmonary complications of general anaesthesia. The technique of epidural anaesthesia should avoid hypotension and should be performed with central venous pressure monitoring. All other patients should receive a general anaesthetic. The anaesthetist should be informed of all the drugs used, so that the anaesthetic technique can be modified accordingly.

After operation/delivery

Continue administration of $MgSO_4$ for 24 hours. To prevent postpartum convulsions, anticonvulsant and, if necessary, hypotensive therapy must be continued for 48 hours. A 'diazepam drip' (80 mg in 1 000 ml Ringer's lactate) is used in patients in whom $MgSO_4$ is contraindicated (e.g. because of oliguria).

Future pregnancies. Postnatal examination and discussion are a vital part of management, especially if the uncomplicated pregnancy has been unsuccessful. If the blood pressure has returned to normal and there is no proteinuria at the postnatal examination, there is no reason why the patient should not start another pregnancy. She should be encouraged to report early in her next pregnancy and warned that intensive antenatal care will be instituted to pick up the earliest evidence of pre-eclampsia. About a third of patients who have had pre-eclampsia in their first pregnancy develop it in a later pregnancy, although it is usually mild,[10] and chronic hypertension is very common in these patients. If the patient is still hypertensive and proteinuric a nephrologist should also examine her. The patient should be advised against pregnancy until she is normotensive and her renal function has been assessed as being satisfactory.[11]

Conclusions

Unless there are drastic changes in the socio-economic status of and health services available to the Black population of the Natal coast, eclampsia with its associated high mortality is likely to be with us for many years.

In the meantime, mortality and morbidity can be reduced by: (*i*) the formation of an eclampsia task force in all large hospitals with a high incidence of the disease; (*ii*) selective booking of primigravidas in larger units; (*iii*) early referral of primigravidas with even the mildest hypertension to larger units; (*iv*) aggressive management of pre-eclampsia in labour; (*v*) more elaborate and intensive care of the eclamptic in certain designated areas of the labour unit; and finally (*vi*) excellent nursing care.[12]

An eclamptic task force has been formed at King Edward VIII Hospital to prospectively study every patient seen in 1982. The management of eclampsia to be followed has been outlined in Table V. The results of this prospective study will be reported in the future.

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