Thus, even if all those patients who failed to reply are taken as failures, as many as half of our cases claimed improvement. As these patients are acting as their own controls, the trial cannot be regarded as scientifically satisfactory; a prospective survey is important and this is under way.

CONCLUSION

Women with simple recurring cystitis may obtain worthwhile benefit from a regimen of perineal hygiene. When this fails, the husband should be considered as a possible source of contamination and brought into the scheme. For particularly stubborn cases, the choice may be made between a regular application of a non-irritant and non-specific antibacterial to the perineum and the adoption of a low-protein diet with or without the daily administration of a culture of the *Lactobacillus acidophilus*.

I should like to thank Mr J. G. Temple, F.R.C.S., for his assistance in the follow-up studies.

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**Eclampsia and the Anaesthetist**

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**SUMMARY**

The anaesthetists' role in the management of eclampsia is increasing. In part this increase is explained by the freer use of caesarean section as a form of delivery. In addition, in certain cases conduction anaesthesia is the treatment of choice in eclampsia.

The anaesthetist in modern practice is an expert in intensive care and as such can offer much to the obstetrician in the management of a condition where cardiac failure is imminent, electrolyte and metabolic balances are disturbed and the cornerstone of treatment is a form of controlled hypotension.


The role of the anaesthetist in eclampsia can be relatively minor or very major depending upon the approach of the obstetrician, the knowledge of eclampsia which the anaesthetist has and the anaesthetist's availability for prolonged periods at any time of the day or night. Above all, the treatment of eclampsia requires a clear-cut policy which is strictly adhered to by all who play any part in the management of such cases. A variety of therapeutic schemes has never produced, from both the maternal and foetal point of view, as good results as has a single scheme. In other words, schemes may be changed for series of cases but except for minor variations should not be altered for individual patients. It is into such a system that the anaesthetist will be incorporated as a team member.

**WHAT IS ECLAMPSIA?**

Eclampsia is a disease confined to pregnancy, labour or the early puerperium in which grand-mal type convulsions occur in a patient with a raised blood pressure, proteinuria and clinically obvious oedema. These latter characteristics are almost invariably present and exceptions are very rare. Craig in a series of 49 consecutive cases of eclampsia, found evidence of the impending fit in the known preconvulsive history and findings in all cases.
During an eclamptic fit a patient may die from asphyxia following the inhalation of vomitus or blood, cerebral haemorrhage or pulmonary oedema associated with cardiac failure, this third cause probably being the commonest. Many and frequent fits increase the likelihood of death for both mother and foetus.

However, eclampsia and the resultant foetal and maternal death are preventable by good antenatal care and by recognizing and treating the state of imminent eclampsia. Craig's showed that the majority of eclamptic fits occurred in hospital and were directly related to inadequate management. He defined imminent eclampsia as 'a condition seen in pregnant women in which symptoms of severe headache, visual disturbance, vomiting or epigastric pain are associated with a blood pressure of 160/110 mmHg or more and a proteinuria of 2 plus or more. Any two of these three features are sufficient for the diagnosis of imminent eclampsia to be made.' Imminent eclampsia has the same pathology, mortality rate and response to treatment as eclampsia. Therefore anaesthetists who will be involved in the management of eclampsia must understand fully that imminent eclampsia, apart from the convulsions, is an identical condition and requires the same management and care. For the rest of this article, therefore, eclampsia and imminent eclampsia are considered as one and the same condition.

With the falling incidence of eclampsia more cases of imminent eclampsia than eclampsia will be treated. The lowered incidence is due to wider and better antenatal services and to the better management of imminent eclampsia. The incidence in the hospitals of the University of Cape Town Obstetric Service has fallen from 6.0/1000 deliveries in 1958-59 to 2.1/1000 deliveries in 1969.

THE PATHOLOGY OF ECLAMPSIA

The pathology or altered physiology of imminent eclampsia and eclampsia must be clearly understood by all who care for such patients. Zuspans' sums up the changes as follows: (i) arteriolar vasospasm—increased sensitivity to vasocostritcirs; (ii) compromised metabolic function with sodium retention; (iii) increased central nervous system irritability; (iv) compromised renal function; (v) alterations in the vascular compartment leading to haemoconcentration of ± 1000 ml volume deficit; and (vi) possibly a catabolic disease with a lack of protein.

Some elaboration of these points is necessary. The arteriolar vasocostriction is present throughout the body but not equally so in different organs. It is likely that the renal and uterine arteriolar spasm are more or less equal and therefore changes in renal function serve as a guide as to what is happening to the foetus in utero. Afferent glomerular arteriolar vasocostriction is the cause of the proteinuria and together with the sodium retention accounts for the decreased urinary output and the oedema. The increased central nervous system irritability has led to two schools of thought regarding the value of the electro-encephalogram in detecting those patients most likely to have fits. Maltby and Rosenbaum, Gibbs and Reid and McIntosh all conclude that eclampsia occurs on a predisposition of an inherited cerebral dysrhythmia. Jost, James, Kolstad and Poidevin have shown that the electro-encephalogram has no practical value in selecting patients likely to develop eclampsia, because the dysrhythmias found at the time of the convulsions may disappear with a lowering of the blood pressure or spontaneously up to 6 weeks after delivery. This latter point is important therapeutically because statements such as 'once the blood pressure has been controlled sedation is no longer necessary to prevent convulsions' are not entirely true.

THE TREATMENT OF ECLAMPSIA

Prevention is better than cure and adequate well-run antenatal services are most important. In cases of imminent eclampsia and eclampsia, the earlier treatment is commenced the better. Therefore the use of the 'Obstetric Flying Squad' is essential and treatment schemes which can safely be commenced in the patient's own home are needed.

The principles of treatment are to reduce arteriolar spasm so that the blood pressure falls and renal (and probably uteroplacental) function improves; to maintain some form of sedative regimen for 24 hours after the last convolution; and to effect delivery of the infant by whatever method carries the best prognosis for both mother and child. There are a wide variety of treatments in current vogue and to discuss each in detail is beyond the scope of this article. Changing trends in management do, however, warrant fuller assessment.

Until about 1955 regimens employing very heavy sedation were popular. They were invariably combined with Stroganoff's principles of nursing the patient in a dark, sound-reduced room with the minimum of tactile disturbance. Once convulsions were controlled a period of 24-48 hours was allowed before delivery was planned. Such sedative schemes included the use of barbiturates, opiates, paraldehyde, thiopentone and magnesium sulphate. Disadvantages were the relatively high maternal and foetal mortality and morbidity, a failure to control the blood pressure and the intensive, prolonged nursing care that was required. Subsequent sedative regimens using bromoethol (Avertin) and magnesium sulphate in a less empirical manner produced far better results because they not only sedated the patient but caused a marked lowering of the blood pressure. These methods are still fairly commonly used.

From the early 1950s there has been a swing towards using an entirely hypotensive drug approach in the management of eclampsia. Such schemes have employed almost all the hypotensive drugs available that can be administered intravenously and, less often, intramuscularly. The results from both the maternal and foetal angle are much better than those from a sedative regimen alone. The patient may also be nursed in less restrictive surroundings. Disadvantages are that these methods are not safe for initial domiciliary use and require extremely careful control to ensure that the blood pressure remains lowered but not dangerously low. Bryce-Smith concluded that conduction anaesthesia (continuous epidural) would effectively lower the blood pressure and improve tissue oxy-
genation. He carried out such a plan on 10 severe eclamptic patients with good results. Disadvantages were the time required of the anaesthetist, the risk of infection and the failure to obtain an immediate increase in urinary output. No evidence was found to suggest that conduction anaesthesia delayed or impaired uterine contractility, a fact since substantiated by others. There is a definite place for conduction anaesthesia in eclampsia. In certain selected units, as Bryce-Smith showed, it could be the treatment of choice and it could also be used in areas where eclampsia is rarely seen and where a competent anaesthetic service is available. Epidural anaesthesia for eclampsia is the best example of the anaesthetist playing a major part in the management from the outset. Willcocks and Moir have used epidural anaesthesia effectively in severe pre-eclampsia.

In the last 10 years tranquillizers and related drugs have been used in the management of eclampsia. Such drugs, particularly diazepam (Valium), have been effective in the control of status epilepticus and in small series have proved effective in controlling eclamptic convulsions. A loading dose of the drug is given followed by continuous intravenous therapy. In addition, hypotensive drugs are given intravenously as required to lower the blood pressure—smaller amounts are usually needed. Such a scheme has been in use in Cape Town for the past 3 years and although the results have not been critically analysed the method has been found both effective and simple.

From the purely obstetric point of view the most significant change in management has been the early delivery of the foetus. Once the maternal condition has been stabilized, i.e. in a matter of hours or occasionally immediately, delivery is planned. If the prospects for labour and delivery are very favourable, induction of labour is performed, otherwise caesarean section is done. Early caesarean section was pioneered by Krishna Menon in South Africa by De Villiers and Slabbert and Crichton et al. Thus, from caesarean section playing a small place in the management of eclampsia it is now not uncommon to find more than 50% of patients delivered by this method. As a result the anaesthetist is brought right into the forefront of the management.

POINTs TO CONSIDER WHEN ANAESTHETIZING AN ECLAMPTIC PATIENT

It is not my intention as an obstetrician to suggest what anaesthetic technique is best, but certain points should be considered: The anaesthetist must be fully aware of the effects on the mother and the foetus of the drugs used by the obstetrician. Hypotensive agents in particular may be short or long acting and can be potentiated by anaesthesia and the supine hypotensive syndrome—the latter often occurring when the patient is flat on her back on the operating table at the time of induction of anaesthesia.

Because of the pre-existing hypovolaemia giving haemocencentration, blood loss is poorly tolerated and eclamptic patients undergoing caesarean section should always have an intravenous drip running which is also maintained for 24 hours postoperatively. Blood for transfusion should be available and a loss of 300 ml or more justifies replacement despite the pre-operative haemoglobin level which is invariably falsely high due to haemoconcentration.

In these ways the troublesome operative and postoperative hypotension which may occur can be avoided and it will be unnecessary to use saline and corticosteroids intravenously, a method advocated by Du Toit.

The liver in eclampsia is always suspect and as a result many obstetricians and anaesthetists will not use even remote hepatotoxic drugs. Goldschmidt et al. investigated the causes of liver damage by potentially hepatotoxic anaesthetic agents and found that the liver damage occurred most frequently in dogs who had been on poor carbohydrate diets and who suffered a relative anoxia during anaesthesia. Clinical experience with Avertin, a hepatotoxic agent, in eclampsia has shown that provided there is no overdosage and no hypoxia liver damage is not found. Presumably better diets have provided the necessary carbohydrate protection.

In eclampsia the foetus is suffering from relative anoxia while the arteriolar spasm remains untreated. The latter, if possible, should be corrected before anaesthesia and any form of inhalation anaesthesia should contain not less than 50% oxygen. The foetus is also at high risk and tolerates poorly a thiopentone induction of anaesthesia. At birth an immediate failure to establish respiration requires intubation and administration of oxygen by intermittent positive pressure respiration. If a trained neonatal paediatrician is not available this usually becomes an additional duty for the anaesthetist.

As eclampsia becomes less frequent the experience of any one person in the management will also decrease. The obstetrician will have to consult with the anaesthetist before starting any new treatment regimen, and vice versa, so that untoward drug interaction in a very ill patient and the foetus may be avoided.

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