NON-ECLAMPTIC CAUSES OF CONVULSIONS IN THE THIRD TRIMESTER OF PREGNANCY*

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No doubt traditional textbook teaching is responsible for the fact that the diagnosis of eclampsia is made with unpardonable finality whenever convulsions occur in the last trimester. Occasionally the alternative possibility is entertained of idiopathic epilepsy, but seldom indeed is further diagnostic search considered.

The admission to our Unit in rapid succession of 2 patients suffering from convulsions—owing to neither eclampsia nor idiopathic epilepsy—stimulated a retrospective study of all patients admitted with the presenting feature of convulsions over the past 4 years. Although cognizant of the limitations of retrospective studies, I decided that the subject was of sufficient importance to merit review.

It would appear from the literature that convulsions due to other causes have assumed progressively increasing importance especially in centres where the incidence of eclampsia has fallen. For instance, Soifer[1] presented a series of 31 cases of convulsions and coma in pregnancy; of these only 6 were eclamptic, and he stated that in the north-eastern part of the USA convulsions in pregnancy are more often due to independent intracranial or metabolic complications.

CLINICAL MATERIAL

This study comprised all patients who presented with convulsions among admissions to the Obstetrical Unit of King Edward VIII Hospital, and McCord's Hospital, Durban, during the period 1960 - 1963. Thus among 64,300 deliveries, there were 119 patients with eclampsia and 35 patients with non-eclamptic convulsions.

TABLE I. CAUSES OF CONVULSIONS IN LATE PREGNANCY AMONG 154 PATIENTS

Cause of Convulsions			*No. of cases*
A.	Eclampsia		119
B.	Idiopathic epilepsy		12
C.	Hypertensive vascular disease		5
D.	Other vascular disorders		4
	(i) Subarachnoid haemorrhage	3	
	(ii) Amniotic fluid embolus	1	
E.	Metabolic		7
	(i) Hypoglycaemia	6	
	(*a*) Alcohol	2	
	(*b*) Drugs	4	
	(ii) Hepatic-acute yellow atrophy	1	
F.	Brain tumours		3
G.	Infections		4
	(i) Meningitis	3	
	(ii) Parasites (cysticercosis)	1	
			154

Idiopathic Epilepsy—12 Cases

Findings. Pregnancy would appear to have no typical effect upon epilepsy, because among the 8 epileptic patients in whom observations had been made on this aspect, 3 had experienced more fits, 2 had less and a third had noticed no change in the frequency of the fits.

Their past obstetrical history showed no greater tendency to abortion, and in each of their current pregnancies live full-term infants were delivered spontaneously with the exception of 2 complicated cases.

The apparently high incidence of pre-eclamptic toxaemia would appear to be significant among these patients. Four showed at least 2 of the signs of pre-eclamptic toxaemia and 1 patient had definite eclampsia. Thus the difficulty of differentiating between eclampsia in an epileptic and an epileptic fit in the presence of mild toxaemia will be appreciated.

Diagnosis and treatment. The diagnosis is mainly dependent upon the past history, and additional evidence is often gained from the finding of scars of old burns or injuries.

The fact that the blood pressure can be temporarily raised immediately after an epileptic fit, and that a minor degree of proteinuria can occur adds to the difficulty, for the picture in eclampsia is often similar. A careful analysis of the urine, however, will always reveal an increasing degree of proteinuria over the next few hours in eclampsia.

The relationship of epilepsy to pre-eclamptic toxaemia and eclampsia is of special interest, in that Rosenbaum and Maltby[2] showed that cerebral dysrhythmia on electroencephalogram tracings (which is seen in 90% of epileptics) is also seen in a high proportion (65%) of eclamptics, as against only 10% in non-convulsive pre-eclamptic toxaemic controls. Burnett[3] also regards dysrhythmia as a connecting link between the two conditions, and suggests that the dysrhythmia is activated by an appropriate trigger mechanism in each instance to produce the convulsion. In eclampsia the trigger is cerebral vasoconstriction with or without cerebral oedema. In epilepsy the exciting mechanism is associated with cerebral oedema, hypocalcaemia, CO_2 deficiency or alkalosis. The practical conclusion to be drawn from these facts would appear to be that pre-eclampsia occurring in an epileptic subject demands strict and early treatment, and measures must be directed particularly towards preventing or correcting oedema.

Status epilepticus is a serious complication which many authors regard as being inevitably fatal when seen in pregnancy (our only death in this series resulted from this complication). If convulsions cannot be controlled with paraldehyde in frequent large doses, then the use of curare and controlled respiration by an anaesthetist and early termination of the pregnancy would appear to be the best form of management. Intravenous Epanutin is also of value.

Hypertensive Vascular Disease—5 Cases

Hypertensive vascular disease occurred exclusively in multigravidae above the age of 30; 3 were over 35 years

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and had had more than 6 previous pregnancies. Where a previous history was available there had been severe headaches accompanied by vomiting and sometimes mental confusion, followed by convulsions. In one case the fits were Jacksonian in type, whereas the others were generalized. In 3 cases the convulsions were followed by local palsies. The blood pressure was very high in most cases, being more than 200/120 mm.Hg in 4 of the 5 cases. There was minimal or no proteinuria and oedema.

Three of the 5 patients who died within 48 hours of admission showed ophthalmoscopic features of long-standing hypertension and 2 had papilloedema. Each had evidence of cardiac enlargement and one was in cardiac failure. Lumbar puncture done in 2 of the patients (who later died) revealed an increase in cerebrospinal fluid pressure. The 2 patients who survived gradually improved over the next 2 - 3 days on a regime of sedation and hypotensives. One delivered a macerated foetus at the 35th week of pregnancy, and the other had a caesarean section 5 days after admission with the delivery of a live premature baby.

These patients are all typical of advanced hypertensive disease with encephalopathy, and the features described above serve to differentiate them from eclampsia without further elaboration. The importance of differentiating is naturally vital since the prognosis and further management of the 2 conditions differ so greatly.

Subarachnoid Haemorrhage—3 Cases

Apart from the 3 convulsive cases included in this series, 3 further cases of subarachnoid haemorrhage occurred without convulsions during the period under consideration. Pedowitz and Perrell,[4] reviewing 79 cases of subarachnoid haemorrhage in pregnancy, found the incidence of convulsions to be 33% and suggested that the true incidence of subarachnoid haemorrhage in pregnancy was probably higher than is generally realized, for many pass unrecognized and are treated as pre-eclamptic toxaemia.

Our patients, who were between 30 and 38 weeks pregnant at the time of their haemorrhage, were typical in their clinical presentation and course. It is worth mentioning, however, that Eastman pointed out that subarachnoid haemorrhage causes almost as many deaths in pregnancy as does heart disease. The maternal mortality rate according to Pedowitz and Perrell[4] is 40%, which is the same as for the non-pregnant patient.

At this juncture it is opportune to stress the importance of lumbar puncture in any convulsive pregnant patient who does not present a straightforward uncomplicated picture of eclampsia.

The presence of blood in the cerebrospinal fluid raises the question of angiography, and, following a careful neurological examination, checked by a physician if doubt exists, an early consultation with a neurosurgeon is of paramount importance. Bilateral carotid angiography should be done once there is clinical evidence that bleeding has stopped, and should this fail to reveal the site of haemorrhage a vertebral angiogram is required. A ruptured aneurysm requires neurosurgery and the type of operation depends on the type and site of the lesion. If no aneurysm is demonstrated then conservative management is carried out.

Feldman *et al.*[5] pointed out that hypertension or toxaemia in no way precludes the possibility of a concomitant aneurysm being the cause of intracranial bleeding. Thus the importance of a more thorough investigation of such cases with the possibility of neurosurgical intervention in mind is self-evident.

The behaviour of our 3 cases, in keeping with that of larger series, bears out the fact that the *incidence of subarachnoid haemorrhage parallels the haemodynamic changes in pregnancy* and is not related to the stress of physical exertion in labour. Thus we find the greatest incidence in the third trimester at the time when the cardiac output and blood volume reach their peak.

This observation should guide the obstetrical management of these cases, and Barnes[6] states that when the lesion has been treated surgically, or managed conservatively by 6 weeks of bed rest, normal vaginal delivery at term should be allowed. When no lesion has been found, or when, for any other reason, radical treatment is not attempted and labour is expected within 4 - 6 weeks of the haemorrhage, the lesion will not have had time to heal firmly, and it is possibly safest to deliver by caesarean section.

Hypoglycaemia—6 Cases

Hypoglycaemia (apart from diabetes) has not received recognition in the literature as a cause of convulsions in pregnancy. In Durban, however, we are familiar with this problem, for patients with hypoglycaemic convulsions and coma frequently present themselves in the medical wards of King Edward VIII Hospital, and we have treated 6 such pregnant cases in our Unit.

Of course the primary cause for hypoglycaemia must be sought. We recognize special varieties of hypoglycaemia in our area:

(a) *Post-alcoholic hypoglycaemia* which occurs in a person whose liver has been rendered vulnerable to toxins by a long period of poor dietary intake. Two of our cases were of this variety.

Another point that must be emphasized is that vomiting and headache, which can be early symptoms of brain tumours, are not usually considered seriously in the pregnant woman. For this reason neglect in early recognition, and therefore treatment, sometimes results. Likewise, the more advanced evidence of a brain tumour can all too easily be explained away as pre-eclamptic toxaemia.

In their study of this type of hypoglycaemia, Neame and Joubert[7] found evidence of severe liver dysfunction in all their cases. Clinically they are typical in their physical findings and are discovered with the aid of a high index of clinical suspicion. A stale smell of liquor usually haunts their breath.

Their response to the injection of 50 ml. of 50% dextrose solution is immediate and dramatic.

(b) *Hypoglycaemia due to herbal medicines.* The second 'local' variety of hypoglycaemia, of which we had 4 convulsive cases, appears to result from Zulu herbal medicines as is described by Neame and Pillay.[8] The patients present with a hypoglycaemia that does not respond, or responds poorly, to intravenous dextrose solution. Many patients die and at necropsy there is a diffuse centrilobular zonal necrosis of the liver. In many cases there is also bilateral acute tubular necrosis.

Thus when the clinical picture is at all suggestive of hypoglycaemia, blood should be taken for sugar estimation, and intravenous glucose administered on admission of the patient, without awaiting the laboratory results.

Brain Tumours—3 Cases

The diagnosis of a brain tumour as the cause of the convulsions was first suspected in 3 patients upon the discovery of severe papilloedema and raised cerebrospinal fluid pressure. The importance of a careful ophthalmoscopic examination in all patients presenting with convulsions is illustrated by these cases.

The pregnant state causes a deterioration of the symptoms caused by angiomas and meningiomas, but there is regression between pregnancies. The more malignant gliomas seem to grow with special rapidity in pregnancy, but this is merely an impression for their behaviour is so variable at all times. Increased cerebral oedema in pregnancy appears to bring on the symptoms of a brain tumour at an earlier date than would otherwise have occurred.

The management of a patient with a brain tumour in pregnancy is usually carried out without regard for the pregnant state, and neurosurgery is performed where indicated.

CONCLUSION

The importance of bringing to the pregnant woman with convulsions the same degree of clinical acumen and meticulous investigation as to the non-pregnant patient, is obvious from this study. The hurried assumption that all are eclamptic can easily be responsible for preventable maternal deaths.

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