NEUROLOGICAL SEQUELAE IN HYPERTENSIVE DISORDERS OF PREGNANCY

by

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Introduction

In hypertensive disorders the basic pathology is vasospasm. The involvement of central nervous system which is wide spread in eclampsia can lead to certain temporary or permanent neurological sequelae. Also the paucity of large scale work on electroencephalographic status in eclampsia and hypertensive disorders in pregnancy urges the need for research in this direction.

Review of Literature

Donaldson (1970) reviewed the literature on EEG in eclampsia and concluded that it may be normal within 6 months but some minor abnormalities may persist. Rosenbaum and Maltby (1943) have suggested EEG's use in prediction of eclampsia development in susceptible women based on finding of cerebral dysrhythmia. Whitacre *et al* (1947) reported tracings of EEG made on 6 cases and produced the sample of one made during convulsion and in recovery phase. The EEG abnormalities became completely normal in 12 days. Just (1948) performed serial EEG in 9 patients of eclampsia and found that when the BP did not change. EEG did not improve.

Material and Methods

Full neurological assessment of 39 patients, 26 eclamptics and 13 noneclamptic hypertensives were done. Mental status and fundus examination was done on admission and rest of the examination was performed 7 days after delivery. All cases had EEG done using 16 channel Grass Machine (model 8-16 BC, serial 682 Q22) with standard 10-20 system of electrode placement. Provocative tests of hyperventilation and photic stimulation were used. Throughout programme 2 & 3 (Vertical & transverse run) were used.

Observations

Out of total 39 patients, the 13 noneclamptic hypertensives had normal neurological examination. Hence the data of only 26 eclampsia patients are being presented.

Changes were classified according to American Ophthalmological Association.

Further neurologic assessment was done 7 days after delivery. Motor and Sensory

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Accepted for publication on 12-4-84.

TABLE I
Mental Status

Consciousness	No. of cases	Percentage	Dilated pupils	Planter reflexes
Fully conscious	13	50	None	Flexer
Semicomatose	13	50	None	Extension
Deeply comatose	—	—	—	—

TABLE II
Fundus Examination

Grade '0'	1	2	3	4
No. of cases 22	3	X	1	—
Percentage 84.72%	15.38	—	3.85	—

systems and perimetry were normal. Cranial nerves were intact in all cases. In the sensory system posterior column and spinothalamic tracts were intact in all. Cortical sensations were impaired in 8 (30.77%) women.

Soft Neurological Signs

These were looked for by testing for higher functions of various parts of cerebrum.

The abnormalities were in the form of high voltage slowing associated with focal sharp waves and other evidences of excessive neuronal discharge. The borderline abnormalities were in the form of excessive response to hyperventilation in 2 and in the form of occassional burst of delta

TABLE III
Frontal Lobe

	No. of cases	Percentage
Presence of Primitive reflexes	None	—
Impairment of memory:		
(i) Remote	None	—
(ii) Recent & immediate	18	69.23
Parietal Lobe:		
Abnormal optokinetic nystagmus	None	—
Constructional apraxia	6	23.07
Double simultaneous stimulation Test Negative	20	76.92
Poor spatial organisation and 2 point discrimination	8	30.77
Fronto Temporal Lobe:		
Abnormalities of calculation and fund of knowledge	None	—
Occipital:		
Visual Agnosia	3	11.54
Psychogenic Abnormalities:		
Depression	5	19.23
Irritability	4	15.38
Apathy and lassitude	8	30.77

TABLE IV
E.E.G. Observations

No. of EEG Tracing	Delivery to EEG interval in days	Eclampsia-EEG interval (a) plus time in hours	No. of Fits
Normal 18 (69.23%)	3-24	3-40-(a)	2-40
Borderline 4 (15.38%)	4-22	6-30-(a)	1- 8
Abnormal 4 (15.38%)	6-15	5-50-(a)	5-50

activity in another 2 women. BP at the time of E.E.G. recording was normal in all but one patient and she had abnormal tracings.

Discussion

Of 39 cases, the 12 noneclamptic hypertensives revealed no abnormality clinically or electrophysiologically in E.E.G. records. Seven of these 13 had past history of hypertension/eclampsia in the previous pregnancy. Three normotensive pregnancy cases with past history of eclampsia or pre-eclampsia also had normal E.E.G. These facts suggests that hypertension per se does not unusually affect the neurological system adversely if eclampsia development is prevented.

Keeping in view the diffuse involvement of central nervous system in eclampsia, the neurological abnormalities are more expected in these patients. Some of our eclampsia cases did have some of these sequelae which are being discussed under clinical and electrophysiological (EEG) groups.

A. *Clinical neurological sequelae*

At the time of admission 50% of the 26 eclampsia cases were semicomatosed though none was deeply comatosed. Pupils were not dilated in any case as they all needed artificial dilatations for fundoscopy. The planter reflexes were extensor in semicomatosed patients and immediately after a fit in all.

Grade I Fundus changes were present in 3 (11.54%) and Grade III changes in 1 (3.85%) and were reversible in all. Rest of the neurological assessment was performed after 7 days of delivery. Perimetry, cranial nerves, motor, extrapyramidal and sensory system (posterior column and spinothalamic tract) were normal in all. Cortical sensations were impaired in 8 (30.77%).

Soft neurological signs were positive in some. Remote memory was intact in all but recent and immediate memory was impaired in most women (69.23%) on 7th postpartum day. Many of them demonstrated constructional aparxia (23.07%) and visual agnosia (11.54%). Double simultaneous stimulation test was negative in 76.92%. 30.77% patients exhibited poor spatial organisation and 2 point discrimination. The primitive reflexes (Grasp, snout, palmomental, sucking) were not elicitable in any.

Optokinetic nystagmus, calculation, and fund of knowledge appeared to be normal in all. Psychogenic abnormalities in the form of depression (19.23%), irritability (15.38%) and apathy and lassitude (30.77%) were quite common which could be primary or secondary to the use

of psychotropic drugs. All the soft neurological signs appeared to improve with time.

B. *Electrophysiological (EEG) Examination*

Out of 26 cases, 4 (15.38%) showed abnormal and border-line response each and 18 (69.23%) had E.E.G. within normal limits.

In all 4 abnormal EEGs convulsions started before delivery and continued in post partum period. Whitacre *et al* (1947) found completely normal EEG in 12 days. In our cases, normal EEG was obtained as early as 3 days and definite abnormalities were observed as late as 15 days after lelivery showing a wide range of individual variation regarding recovery from neuronal dysfunction secondary to vasospasm. The borderline cases showing excessive response to hyper-ventilation might point towards their susceptibility to develop seizure in predisposing conditions and thus may support the view of Rosenbaum and Maltby (1943) that cerebral dysrrhythmia could be a precipitating factor in eclampsia in a genetically, predisposed women. Jost (1948) found that cerebral dysrrhythmia patients became normal as their hypertension decreased. However, in the present study 25 of 26 patients were normotesive at the time of EEG. Only 1 patient had 150/90 mm Hg BP at that time (15 days postpartum). Her EEG was abnormal in the aspect that it exhibited delta activity throughout the record, predominantly bifrontally denoted diffused generalised neuronal dysfunction or metabolic encephalopathy. The number of fits did not seem to bear any relation with the EEG pattern. Our observations also suggest that delivery to EEG interval is more important in determining the occurrence of abnormalities in EEG than the first fit to EEG interval as the later was not found to vary significantly in the three groups of normal, abnormal and borderline EEGs.

Conclusion

1. Though there is no gross neurological defecit, soft neuropsychiatric signs are quite common after eclampsia in recovery phase indicating subtle neuronal dysfunction in the cerebral cortex. It is suggested that patients showing poor or slow recovery from these signs should be carefully followed for the development of future psychiatric or neurological illness.

2. Post delivery EEG abnormalities are not commonly seen in non-eclamptic hypertensive disorders of pregnancy.

3. In eclampsia, normal EEG records can be obtained as early as 3 days postpartum and abnormalities can be seen as late as 15 days postpartum.

4. The abnormalities are usually in the form of high voltage slowing, occasional focal sharp-waves, and abnormal delta activity denoting cortical neuronal dysfunction. These are more likely in patients in whom convulsions began before delivery and continued in the postpartum phase.

5. Borderline abnormalities in the form of excessive response to hyperventilation and nonspecific asymmetry and intermittent slowing may suggest that cerebral dysrrhythmia pattern indicate a potential candidate to develop eclampsia in provocative circumstances.

6. Blood pressure and number of fits did not seem to have any relation with the EEG patterns.

7. It is the termination of pregnancy which appears to be more important in determining EEG sequelae.

8. It is suggested from the results of this study that patients showing EEG abnormalities should be carefully followed-up for (a) development of essential hypertension (b) persistence or development of abnormal behavioural patterns (c) recurrence of seizure activity in future pregnancies (d) development of cortical epileptic disorder.

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