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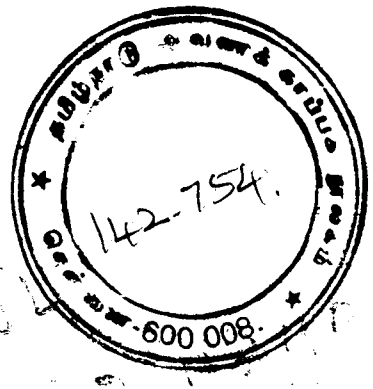
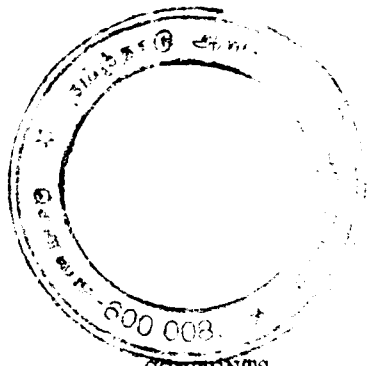


IMPORTANT NOTICE

It is imperative that these quarterly Medical Bulletins are published regularly as they are in great demand by the Medical Profession all round, even outside this State. For this purpose sufficient number of articles of scientific interest are absolutely necessary. You are requested to kindly co-operate by regularly contributing any articles of scientific interest. Such articles may be kindly sent in duplicate.



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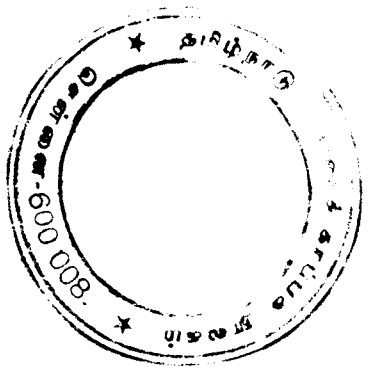

A. SYMPOSIUM ON EPILEPSY

INTRODUCTION

The study of epilepsy is of great value. It has helped to control a disease which affects a fairly good proportion of mankind. In addition a careful study of the various manifestations of epilepsy has contributed to an increased understanding of the physiology of brain function. It has given an insight into the functional localization of the cerebral cortex and the organisation of function at the various levels in the nervous system.

This symposium was held at the Madras Medical College.

CLINICAL ASPECTS OF EPILEPSY

Epilepsy, a dreaded disease, has been recorded since ancient times. Modern knowledge of epilepsy has led to a hopeful prospect for the sufferers.

Epilepsy is a symptom caused by a variety of pathological lesions. The lesion in epilepsy may be either in the cerebral cortex, the subcortical structures or in the brain stem. From the time of Hughlings Jackson, our understanding of the functions of the various levels of the nervous system has increased considerably. Penfield, after an extensive study of the human cerebral cortex and the manifestations of epilepsy had postulated that the integration of the cortical neuronal discharges takes place in the centrally situated structures of the brain. To these he has given the name 'THE CENTRENCEPHALIC SYSTEM'.

The brain is organised in such a way that all the incoming impulses are elaborated and analysed in the cerebral cortex. Similarly all the outgoing motor impulses are formulated, analysed and later projected by the brain.

When there is an irritative lesion in any of the areas of the brain, there is a sudden upset in the pattern of neuronal activity. This leads to a manifestation of abnormality namely

either cessation of function or an abnormal activity. Depending on the area affected, the manifestation of epilepsy differs. If the lesion is in the centrencephalic system, the attacks are likely to set in with loss of consciousness, whereas if the lesion is elsewhere in the cortex, then there may be an aura followed by march of symptoms. In the motor area, the initial symptoms may be the twitching of a muscle. In the sensory area, it may be loss of sensation or parasthesia in a portion of the body. In the occipital region it may be a visual phenomenon. In the temporal region it may be a disturbance of speech memory or a visceral sensation. Thus a careful study of the onset of the onset of the attack will provide a clue to the site of the lesion.

In addition to these attacks which are termed grandmal attacks, there may be smaller attacks known as petitmal. The petitmal attacks may be minimal with only a momentary lapse of consciousness or major accompanied by myoclonic jerks. Occasionally a petitmal attack may go on into a grandmal seizure.

It must also be remembered that diseases elsewhere in the body may induce convulsions or seizures. Hence a thorough investigation is necessary to decide the cause of epilepsy. Cardiovascular diseases, hypoglycaemia, metabolic diseases or electrolyte disturbances may cause generalised convulsions.

Clinically a thorough study of the onset of the disease and the pattern of its spread gives the physician a fair indication of the nature of the disease. From this it can be decided what further investigations are necessary before deciding on the line of treatment.

THE PATHOLOGY OF EPILEPSY

I. The Pathology of Epilepsy can best be discussed under three main headings, as shown below:—

(*a*) The alteration in structure as demonstrated by macroscopic, microscopic and histo-chemical methods.

(*b*) The alteration in the electrical action of the brain in general and certain areas in particular; and

(*c*) The alteration in the neuro-chemistry.

The altered structure and its significance.—The mere demonstration of a lesion (either macroscopically or microscopically) cannot be taken to mean that that particular defect is the cause of the seizure. This may seem to be simple but this

distinction is important. The Ammon's horn is the term applied to the hippocampus. This area contains plenty of grey cells. It has been divided into many divisions. Lesions confined to some of these areas have been thought to be responsible for epileptic seizures. But, by stimulation of the hippocampus, Penfield and others could not elicit any type of response similar to a seizure. It is thought that this sclerosis is the result (and not the cause) of the fit. Of course, this applies only to the Ammon's horn, but not with regard to the uncus or amygdaloid complex. Again in certain conditions like kernicterus, lesions of the Ammon's horn or of even various areas in the rest of the temporal lobe are seen. But no convulsive seizures have been seen in such cases.

The next thing to consider are the factors producing these lesions. These areas, particularly the amygdaloid, anterior temporal and uncus seem to be vulnerable. This vulnerability seems to consist in:—

(*a*) Low threshold for electrical stimulation; and

(*b*) Sensitivity to hypoxia.

This hypoxia may occur during birth or during a fit.

Again there seems to be a tendency for this area of the brain to be injured either during birth or in accidents—both by direct impact and by countercoup. Structures like the sphenoidal ridge, the sphenosellar recess and the tentorial edge may injure the temporal lobe.

Penfield and his co-workers have described uncal herniation during the moulding of the head leading to sclerosis of this area.

The factor of blood supply has also to be considered. These areas are mostly supplied by the anterior choroidal and the cephalic end of the posterior cerebral. Either compression or a spasm of the arteries may be responsible for ischaemic necrosis and subsequent formation of epileptogenic foci.

The exact site of lesion in an epileptic seizure cannot be determined on the aura alone. In some cases, the aura may be masked by amnesia.

What exactly happens in epilepsy? It has been thought that all the cells discharge simultaneously in generalised epilepsy. While this might occur after the attack is established, it is more probable that at first the discharge occurs on one side

(even in generalised seizure) deep down in the brain stem. From here, it spreads all over in a global manner or in any other manner.

The locus of the lesion alone does not determine the disease. Not only must there be an abnormal type of electrical discharge but there should be susceptible cells all around to help in the spread of the seizure.

When the electrical discharge starts in the uncal region and spreads on to the brain stem after the initial aura there is loss of consciousness.

The knowledge of the pathology of epilepsy is astonishingly limited. From a slide of brain tumour a pathologist can say what one is dealing with, e.g., a glioblastoma or a secondary deposit. But as far as epilepsy goes, from a given slide, the pathologist can only give a general opinion. That is to say, the changes that are seen (with the various techniques available to us) are essentially non specific. This consists of disorders of structure or various types of degeneration but there is no histopathological picture from which a pathologist can deduce the presence of an epileptogenic focus.

A variety of histopathological changes may be seen. These include patchy or focal areas of destruction of neurones with reactive proliferation of glia. There may be calcification too. Macroscopically these areas may be seen to be tough and leathery. Such and similar lesions have been described in the cortex, thalamus and even cerebellum.

Time or space does not permit a detailed clinicopathological correlation between the various types of seizures. These can also be dealt with on electro pathological lines also.

The recent understanding of the function of rhinencephalon has shed much light on the existing knowledge of epilepsy—particularly the so-called temporary lope epilepsy. These areas may show alteration like abnormalities of structure or only electrical abnormalities. Again the abnormalities may be purely chemical.

The three per second spike and wave pattern occurs in lesions which occupy the inferior or medial aspect of the frontal lobe.

Bilateral rhythm 4-6 may occur in uncus or amygdaloid lesions. The rapid spike pattern usually involves the brain

There are also some other interesting points in connection with these foci. One is, that there may be a mirror focus on the contralateral side. The other is that the removal of the focus and subsequent relief of epilepsy cannot at all mean that the lesion was at that spot. The relief might occur because the circuit had been broken.

The Electro Chemical Concept

It has been generally thought that the seizure genesis is the result of an elevation of excitability of the cells. This view is slowly giving way to the other that the seizure is the result of a reduction of inhibition. It is generally known that the reason why a particular impulse fails to evoke more than a single response is that the cell becomes refractory. This refractory state can be considered from two aspects. The first is that ordinarily some impulses are inhibited by a special type of cell called the Renshaw cell. This was first described in the spinal cord but now this type of cell has been found to be represented all over the C.N.S. The Renshaw cell type may be represented by a special area on the post-synaptic membrane. Of course even this mechanism of action has been explained on a neuro-chemical basis. The inhibition of this Renshaw cell tyre is supposed to be responsible for the widespread nature of the epileptic seizure. Actually certain substances like strychnine, described as convulsants are known to act on this Renshaw cell and hence some have called strychnine, the curare of the Renshaw cell.

The other aspect of altered electrical nature is yet to be confirmed. This postulates that there is a graded response type of excitation involving both dendrites and axons and the electrical impulse passes not by the pathway of the cell-axon-cell but more by continuity. This is contrary to the Jacksonian concept.

The widespread nature of a seizure naturally brings to the mind the autonomic nervous system. Here is a single stimulus evoking a universal response. Even here the cholinergic mechanism (because of the efficiency of the inhibitory enzyme) is not as universal in action as the adrenergic. Much work is being done in this field.

Neurochemical Basis

The knowledge of neurochemistry commenced only with Thudichum. Subsequently the rate of growth of this field has been phenomenal, and now we feel that we are almost on the brink of discovering the ultimate secret.

There are certain limitations in the histochemical study of epileptogenic foci. For example, the mere presence of increased acetylcholine (or for that matter any biochemical change) must be demonstrated not only in the ictal or postictal period but also in the preictal stage. Failure to understand this might lead to false conclusions. We saw a similar state of affairs with regard to other changes too.

On a purely hypothetical basis in 1940, Penfield and others introduced a concept that there is a substance which was responsible for the seizure. Possibly this substance is similar to, if not identical with acetylcholine. While a number of altered chemicals have been identified and discussed we will take only the more important one.

Acetylcholine

Acetylcholine has been demonstrated in the cerebral cortex and also in the deeper nuclei. It is known that acetylcholine is the mediator of neural impulse. Cholinesterase has also been demonstrated in the C.N.S. By means of various experiments the role of acetylcholine in seizures has been fairly well established. (*a*) There is a detectable amount of free acetylcholine in the C.S.F. of patients with frequent seizures. (*b*) In induced animal seizures, increase of acetylcholine is detectable even before the seizures commence. (*c*) There is also an accompanying increase in cholinesterace activity. (*d*) Acetylcholine directly applied to the neurons produces hyperactivity. (*e*) In post-traumatic cases where fits are seen, increase in acetylcholine is seen. This is perhaps due to the injury releasing acetylcholine. Recently we had a case where a few minutes after injury the child had fits. At the same time and also after this the child also exhibited marked gastric peristalsis (of an intensity which we see in cases of Duodenal obstruction). This goes to illustrate the acetylcholine production.

The acetylcholine of the brain is in two forms. One is the free acetylcholine and the other is the bound form. The latter is presumed to be held in insoluble form and possibly bound with proteins. . It is released only with suitable chemical treatment.

When the brain slides of epileptogenic foci are studied, it is found that the slices have the normal free acetylcholine. But they do not produce the normal bound acetylcholine. This is due to some defect which interferes with what we may call the binding capacity of the cells. The production of acetylcholine

is the same but more of it leaks out as it were. The increased leakage of acetylcholine results in a compensatory increased production of cholinestrase.

Glutamic Acid Metabolism

Incubation of epileptogenic samples of brain tissues showed also abnormalities of glutamic acid metabolism. During incubation of these slices it was found that there is a loss of glutamic acid in the epileptogenic samples.

Glutamic acid is an amino-acid with interesting metabolic status. It is a sort of connecting link between pyruvate metabolism and protein, ammonia and N^2metabolism. By taking part in transamination reaction it is necessary in aminoacid interactions. By deamination it can enter the Krebb's cycle. It can also be decarboxylated to r-aminobutyric acid (G.A.B.A.) and then enter the Krebb's cycle at the succinate stage.

The loss of glutamic acid is not due to its conversion into any of these ingredients. Actually the level of r-aminobutyric acid also falls. The role of G.A.B.A. is also supposed to be inhibitory.

Of course G.A.B.A. curves fall during seizures induced by Fluracetate and strychnine but these are not of any significance.

The consulvant action of methionine sulphoximine is supposed to be due to its action on acetylcholine and also glutamic acid production. Any substance which promotes glutamine metabolism must then prevent seizures. In this connection trials with L-asparagine are really convincing. The L-asparagine acts thus—

L—asparagine—aspartic acid and then enters the Krebb's cycle.

The convulsant effects of pyridoxine deficiency is supposed to be due to the deficiency interfering with amino acid metabolisms.

Potassium Metabolism

The third factor is the inability of the cells to hold the potassium. The reason for this is not known. In view of the role potassium in the synthesis of acetylcholine and neuronal transmission it is thought that this inability is of some importance.

These three defects may or may not co-exist in the same sample. They may in themselves indicate that these individual defects may be responsible for seizures or there may be various facets of a simple metabolic derangement.

Therapeutic proof with adequate substitution therapy is still lacking, except with regard to pyridoxine and aspartic acid. A.T.P. also corrects these metabolic derangement. All these indicate that these defects are essentially reversible.

By interference with supply of energy, hypoglycaemia and hopoxia can cause fits.

Better proof of these can come if we can understand the mode of action of anti-convulsants. Anti-convulsants can act in (1) on blood supply and prevent seizure; (2) by acting on diseasesd neurones and thus suppressing seizures; (3) action on surrounding neurones to prevent the spread. This last may be done by increasing their refractory state.

In this connection the dephenyl hydantoins are supposed to act by decreasing the total and intracellular potassium. Acetazolamide is supposed to act this way through the carbonic anhydrase system.

E.E.G. AND EPILEPSIES

Introduction of E.E.G. has been a turning point in the erstwhile silent fields of Neurophysiology. New frontiers and avenues of investigation have been opened up for study, to confirm or disprove many of the popular beliefs in clinical neurology and physiology. But, the greatest clinical advantage has been so far in the field of epilepsies; in their diagnosis, their management and their cure by neurosurgical methods.

In E.E.G. a discharge means a sudden, single or groups of abnormal waves supervening on or replacing the normal rhythm. This discharge may be in classical form, spikes (or sharp waves), i.e., waves of very short duration less than 15 millisecs., in case of spikes and even up to ½ a min in case of sharp waves. This is called an epileptic discharge. Similarly spike and wave complexes of 3 c-sec. or lesser frequency are classical examples of epileptic discharge. Other types of discharges which may be termed convulsive discharges are bursts of large amplitude grouped waves without any particular form. Large amplitude slow waves are usually not a convulsive discharge. They indicate neuronal disease and may occur in parenchymatous disease or be postictal at the site of focal discharge.

Initially, when E.E.G. was introduced, it was hailed as a supersorting apparatus to simplify classification of epilepsies. Certain patterns were equated to certain clinical entitles, e.g., colleagues at Harward. Paroxysmal fast rhythm or crescendo spikes were said to be diagnostic of grandmal; flat topped waves 4 c-sec. were said to denote psychomotor epilepsy and so on. They did a lot of their work with monopolar records and devoted their main attention to the morphology or the shape of the curve. Very soon they and others all over the world found that this correlation between clinical fit and E.E.G. broke down very often. Spike and wave complexes were recorded from patients having an obvious major seizure.

Meanwhile the Montreal school with Penfield and Jasper at its head, after considering the above difficulties settled down to discover a better method of study.

They devoted their attention to the neuroanatomical location of origin of the waves rather than their morphology. The classification that is discussed below is based on their studies and is in use in majority of the institutions. Before I proceed to detail this classification, I would like to describe a few neuroanatomical advances:—

1. Stimulation of interlaminar mass in Thalamus at a particular frequency gives rise to bilateral symmetrical synchronous spike and wave complexes.

2. If the upper part of midbrain is stimulated, at the same time these complexes are inhibited.

Penfield and his collaborators gave the name of centrencephalon to these structures; they consist of interlaminar mass connecting two thalamii, hypothalamus and upper part of midbrain. This is not an anatomically clear cut structure. It is a concept of a functional neuronal congregation. This concept has been brilliantly criticised by Sir Francis Walshe; yet it has a large number of supporters.

The few axioms enunciated by Penfield and Jasper are:—

(*a*) all idiopathic epileptic discharges originate from this centrencephalon. Since it is a central structure, discharges go out to both cortices at the same moment and hence we have a discharge which is bilateral—symmetrical congruous and synchronous. Similarly if this centrencephalon is fired off by any means outside its body there will be the classical bilateral

discharge recorded in E.E.G. In idiopathic epilepsies the discharge is supposed to originate from within this centrencephalon. When the discharge is 3 c-sec. spike and wave complexes the clinical picture is mostly of a Petitmal type and when of fast high voltage type, usually a grandmal seizure. This is what is described as Primary subcortical Epilepsy.

An important corollary follows, that if there is a unilateral area of nervous disease in the cortex and it is epileptogenic, then whenever this epileptic discharge is mild or moderate it will be recordable as a localised discharge from the relevant area without any such disturbance from other areas of the cortex. But if the discharge is powerful enough or the lesion is close enough to the centrencephalon it will activate the latter and through it produce bilateral discharges which may be almost symmetrical except that the abnormal discharge at this focus precedes the generalised bilateral discharge. Hence, by locating the site from which the discharge starts earliest, one can conclude, this is a focal epileptogenic lesion presenting as a centrencephalic discharge. This is known as the secondary subcortical epilepsy.

From this the following classification will become easily understandable. This classification is not perfect but answers most of the problems and for want of a better one—is the most widely used.

CLASSIFICATIGN.

1. *Primary facal cortical epilepsy* is one spot on the cortex that is the graph derived from one or two electrodes shows evidence of epileptic discharge while none are present in any other site.

2. *Primary subcortical epilepsy.*—This, as had been already discussed, is assumed to be due to initiation of discharge in the centrencephalon itself, the so called Idiopathic Epilepsies covering grandmal and petitmal groups.

3. *Secondary Subcortical Epilepsy.*—This also has been already explained. This is a symptomatic epilepsy which starts from a focus in one hemisphere, traverses to the centrencephalon and there fires the latter off, which in turn shows off as bilateral symmetrical synchronous discharges which may be confused with Primary Subcortical Epilepsy.

4. There is a group of cases where the several activities of the brain are very much disturbed with no particular pattern. There is evidence of multiple or changing foci. These are classified as *Diffuse Dysrythmias.* This includes all those that do not fall in the above three groups.

Few practical points of importance in differentiation between Primary and Secondary subcortical Epilepsies are as follows:—

1. The interseizure record in case of pure primary subcortical Eplipesy of idiopathic variety is, in majority of cases within normal limits while those in secondary subcortical epilepsy are usually abnormal. The abnormality may be very slight, e.g., disturbances of normal rhythm or gross as in cases of space occupying lesions where epilepsy is a symptom of the cerebral lesion.

2. The symmetry, synchrony and congruity of the waves on both sides in secondary sub-cortical epilepsy is not quite as exact as observed in primary variety.

Once these differences are suspected, careful measurement of the commencement of the dysrhythmia is conducted to find out if the abnormal discharge precedes in any one or more channels before others. Study of epilepsy is conducted not only for itself but for its significance as a diagnostic sign of an underlying lesion.

Study of epilepsies with repeated E.E.G. has shown that localized epileptogenic areas, which remain localised in E.E.G. and in clinical description have done well with surgical excision. Here I would like to mention that Temporal lobe disease is by far the commonest cause of the secondary subcortical epilepsy and masquerades as the so called idiopathic epilepsy. Local lesions in other areas like frontal lobes and medial surfaces of cerebral hemispheres, also tend to produce secondary subcortical epilepsies. As will be evidenced by E.E.G. analysis, many so-called idopathic epilepsies have been found to have a focus and hence have come to be classified as symptomatic epilepsy in clinical sense and secondary subcortical epilepsy in E.E.G. sense. Electrodes like sphenoidal, pharyngeal and tympanic electrodes are used along with activating procedures like hyperventilation. I.V. Metrazal or Megimide, I.V. pentothal or sleep records, light flicker, etc., to locate the primary source of origin of an apparently primary subcortical epilepsy so as to ultimately prove it to be of secondary type. These electrodes are an attempt to pick up electrical discharge from inferior and medial surfaces of the temporal lobe which is one of the most common sites of epileptic discharge.

Electrocorticogram is the term applied to the records obtained directly from the electrodes placed on the brain surface. Apart from a wide field of interesting physiological research, this method provides a means by which a suspected site of persistent focal lesion is recorded from the brain surface, stimulated to confirm whether it produces some type of fit and later excised with confidence, the only method of curing certain epilepsies.

To conclude, routine and when necessary, serial electro encephalograms are a great help under the following conditions:—

1. Diagnosis of a bizarre form of epilepsy or an epilepsy with changing pattern of onset. It may be pointed out that by careful history taking and recording of evidence of onlookers, one can reasonably diagnose most types of epilepsies clinically. Most of the time there is no need for an electroencephalogram in patients suffering from classical grandmal or petitmal.

2. Studying the cortical patterns in epilepsies not responding to anticonvulsants or deteriorating in fits and mental function while under treatment.

3. Serial follow up of those who have been well controlled on treatment to aid in withdrawal of the drug if possible after two to three years of continuous treatment.

4. Investigation of all epilepsies suggestive of a focus of origin. Hence all focal epilepsies with a definite aura should be investigated. Here, there is the advantage of localising the focus of discharge electrically as well as discovering an underlying cause (e.g. tumour as a cause for the epileptogenic activity). It would be best to consider epilepsy as a symptom in these cases and to investigate the cause for the same. A meningioma may present as a case of focal fits with no other neurological symptoms or signs and may be located by electroencephalogram along with the focal epileptic discharge.

5. Electrocorticography in cases with a consistent localised focus in electroencephalogram to confirm the exact site of discharge for possible ablation of the epileptic zone.

Electroencephalography is a recently developed and highly complicated subject. Already the horizon of its indication is expanding. Greater knowledge of the brain mechanism will provide other avenues in the investigations, treatment and cure of epilepsies in the near future.

THE PLACE OF ROENTGENOLOGY IN THE STUDY OF CLINICAL EPILEPSY

Epilepsy by itself is not a disease but is merely the outward manifestation of an underlying disturbance in brain function. All cases of epilepsy fall into two large general groups, namely, those due to unknown aetiological factors, the so-called *Idiopathic or Essential variety*. This group naturally includes cases without demonstrable cerebral lesions, such as epilepsy that follows toxic and febrile reactions, the idopathic, familial type of epilepsy, the epilepsy that follows hypoglycemia and other less well understood lesions, for example angioneurotic states and circulatory arrest. The second group includes cases which are secondary to some specific causes such as birth injury, congenital brain defects, tumours, abscesses, inflammatory lesions, hæmorrhage, brain scars, arteriosclerosis are therefore classified under the heading of *Symptomatic or Secondary variety*. This group usually shows abnormality in X-ray study and the demonstrable cerebral lesions include expanding lesions, cerebral cicatrix, local cerebral atrophy, local Microgyria, brain cyst, diffuse cerebral disease, diffuse cerebral vascular disease and a miscellaneous group of diseases including congenital lesions.

The X-ray procedures consist of: (1) Routine plain films of the skull (2) Encephalography either Pneumoencephalography or ventriculography and (3) Cerebral arteriography.

Plain X-ray of the skull.—They may not reveal any abnormality in the majority of instances. However it is not possible to foresee when such examinations will be of value and hence skull films should be taken routinely as the first step in the roentgenographic work up of the epileptic. The routine views consist of posteroanterior, occipital and both laterals with stereoscopic view of one of the laterals.

The following conditions can be appreciated by the plain films of the skull, fractures, increased intracranial pressure, craniostenosis, micro and macro cranial hemiatrophy and intracranial calcification which may be either physiological or pathological.

Fractures of the skull.—Trauma to the head may damage the brain, the dura or the skull singly or in combination. Focal cerebral atrophy, meningocerebral scars or defective dura, being the sequalæ to such trauma may be potential factors in epilepsy of later origin.

The trauma may produce only a contusion or small subpial hæmorrhage of the cortex resulting in focal cerebral atrophy. A contracting meningocortical cicatrix forms if the dura and brain are damaged at the point.

Depressed fractures or penetrating injuries such as bullet wounds increase the likelihood of epilepsy, coming on many years after the trauma due to the formation of a meningo-cerebral cicatrix.

In cases where the head injury has been severe and a defect in the dura remains, one observes the presence of erosions or irregularities of the inner table of the skull and this type of irregularity of the skull may well indicate the site of the epileptogenic focus.

Microcrania or small skull is common in those patients who have sustained brain damage at birth or during the first year of life. It is usually associated with a imperfectly developed brain. These patients usually are mentally retarded and often suffer from convulsive seizures. If the brain damage has been severe, premature closure of the sutures occurs with varying degrees of microcephaly. The cranial vault is small and out of proportion to the normal facial bones. This is an under-development of the vault of the skull which is secondary to the under-development of the brain. A small head alone does not constitute microcephaly; the size of the cranium must be considered in relation to the size of the face and rest of the body.

In true microcephaly the forehead slopes backwards to a flattened vault; usually the occiput is nearly normal in size and shape. The bones of the face are average in size; the nose is long. The bones of the vault are often thickened. The condition may be familiar.

Craniostenosis.—This constitutes a group of conditions in which the most important feature is closure of the cranial sutures in infancy or even before birth. It is commonly associated with symptoms of increased intracranial pressure. Convulsive seizures may occur. Oxycephaly is the most common variety of craniostenosis and the coronal suture is closed and a 'steeple-shaped head' results. The patients with this condition show considerable proptosis. Scaphocephaly (boat-shaped head) is usually secondary to closure of the sagital suture so that growth takes place in the sagitital plane only. Plagiocephaly is least common and is caused by closure of the suture on one side of the skull retarding development on that side and producing asymmetrical or slanting skull.

Cranial hemiatrophy.—The presence or absence of cerebral hemiatrophy is probably related to the age in which the cause for the patient's epilepsy has its inception. In that the maximal growth of the brain occurs during the first two years of life one

would expect the greatest degree of hemiatrophy to be found in patients whose disease dates back to this early period in their lives. From the age of two years until puberty, slow but steady growth of the brain continues and here too one might expect hemiatrophy but it will be less marked. As a rule, there is so little development of the brain after puberty that hemiatrophy in an individual whose disease started later in life would be far less common.

The skull is usually asymmetrical and in the postero-anterior view the two halves of the skull differ in size. The left half is usually larger than the right. In cranial hemiatrophy when fully developed, postero-anterior view of the skull shows an enlargement of the frontal and ethmoid sinuses and mastoid cells together with elevation of the petrous bone on the side of the small cerebral hemisphere. The enlargement of these air containing structures is probably an attempt on the part of nature to bring the skull back to normal size. As already indicated such asymmetry of the skull will not occur following brain damage which originates later on in life after the brain has once matured.

Intracranial calcifications.—Calcifications within the cranial vault may be either physiological or pathological.

Physiological intracranial calcifications include calcium deposits in the choroid plexus, falx cerebri, pineal body and pacchionian granulations. The clinical significance of these calcifications is that in atrophic lesions of the brain the calcified pineal, falx or choroid plexux may be displaced towards the side of the lesion, if the degree of atrophy is of considerable size. Pathological calcification may be seen in neoplasms, craniopharyngiomas, vascular, inflammatory, parasitic, degenerative and other miscellaneous pathological disorders.

Sturge-Weber syndrome is a vascular disorder in the brain in which calcification is frequently seen. The syndrome consists of facial nævus, congenital glaucoma, epilepsy and intracranial calcifications. The intracranial calcifications present a highly characteristic roentgenographic appearance. They usually are massive and seem confined to the cerebral hemispheres. Their configuration suggests that the calcifications follow the gyri and with a significant clear space between the periphery of the cerebral convolutional calcification: often with double contours, with a significant clear space between the periphery of the calcified process and the inner table of the skull. The occipital lobe is the one which is involved frequently.

Vascular calcifications are also seen in cerebral aneurysm and in subdural hæmatomas.

Calcifications seen with inflammatory processes, include those in old pyogenic processes, healed intracranial tuberculomas, toxoplasmosis and ecchinococcus disease. Because of healing and calcification of these inflammary lesions, a contracting crebral sear may form with epilepsy later on.

Tuberculoma of the brain has a special interest for us since it is a common lesion of the brain in India. In a large series of verified tumours of the brain seen in the Neuro-surgical Department of the Government General Hospital, Madras, tuberculomas formed 20 per cent. This high incidence is due to tuberculosis being prevalent amongst us in various forms. Tuberculous infection of the nervous system is no exception, since it is hæmatogenous in origin from some primary source as the lungs. The cortex of the cerebrum or cerebellum is more frequently involved possibly because of more abundant blood supply. Tuberculoma tends to become calcified in its chronic stage and calcified tuberculomas of the brain may vary in size and number. The deposits of calcium in these lesions measure from a few millimeters to two to three centimeters in diameter. The individual calcifications have rather a dense homogenous centre, but their margin is serrated, irregular and lacelike. The entire lesion may be fragmented with small calcium spicules and plaques separated from the main centre of calcification. In a series of two hundred cases of tuberculomas of brain analysed, only eleven cases showed calcification in the skull. But the x-rays of the removed specimen show much more obvious calcification.

Cysticercosis.—It is caused by cysticercosis cellulosae and is acquired from eating measly pork. Larvae may occur in the brain and in the eye. The calcified nodules are usually small, approximating one, two or three millimeters in diameter.

Tuberose sclerosis syndrome.—In its full-blown form it presents the following symptomatology:—

(1) Epileptic seizures.

(2) Mental deficiency.

(3) Acneform butterfly facial rash (adenoma sebaceum Pringle).

(4) Periungual fibromas.

(5) Congenital tumours of the eye and of many other organs, including rhabdomyomas of the heart and hypernephromas of the kidney.

In this condition the roentegen examination of the head shows multiple small discrete areas of the calcification throughout the brain substance. These areas of calcification are often in the region of the sella, the basal ganglia and the choroid, in cerebellum, in the occipital poles and in the frontal lobes. Other miscellaneous calcifications include those in the basal ganglia, sometimes due to hyperparathyrodidism and calcifications due to healed pyogenic abscesses and toxoplasmosis.

Contrast studies of the brain, using air (Cerebral Pneumography).

In cranial roentgenography it is necessary to utilize some form of contrast to delineate properly the intracranial contents. The most common contrast used in studies of the brain is air or oxygen. Such procedures are referred to as encephalography and may be either (*a*) pneumoencephalography or (*b*) ventriculography. In this context, it is important to remember that no patient with focal epilepsy has been studied completely until pneumoencephalogram or ventriculogram or cerebral arteriography has been performed.

Pneumoencephalography.—Consists of replacing part of spinal fluid with oxygen or air and taking films of the head to show the size, shape and position of the ventricles, the size and shape of the basal cisterns and the patterns of the cerebral convolutions and sulci as outlined by air in the subarachnoid pathways. This is the most common method which gives the most accurate estimation of brain atrophy or hypoplasia in the epileptic cases.

Ventriculography.—Is the method of choice when there is increased intracranial pressure or the patency of the basilar foramina is the questionable and obviously is more effective under such conditions.

Encephalography, therefore, may demonstrate a normal ventricular system or there may be a demonstrable localised dilatation of one of the lateral ventricles with associated small subarachnoid collections of air suggesting the focus for a localised area of brain atrophy or meningocerebral scar. A localised enlargement of a portion of the lateral ventricles then is of particular significance. Such a focal enlargement may be found in an individual who does not suffer from epilepsy but if he does have convulsive seizures the lesion which is responsible will often lie somewhere in the neighbourhood of that portion of the lateral ventricle, which is most markedly dilated as shown by the encephalogram. It has been observed that lesions in the frontal and parietal lobe areas are more prone to produce seizures than lesions in the occipital lobe.

Large atrophic lesions are associated with marked compensatory enlargement of the ventricle adjacent to the zone of brain disturbance and in a general way the degree of ventricular enlargement together with the degree of enlargement of the subarachnoid spaces may be taken as a measure of the degree of destruction of brain tissue. It is possible to lateralise the focus in which brain atrophy is marked in such instances, but of course it will not be possible to diagnose the exact site of the discharging focus. The focus is more accurately localised by clinical means and E.E.G. or possibly craniotomy.

In expanding lesions of any great size, the ventricular system is compressed and together with the septum pellicudum will be displaced from the lesion. Arteriovenous aneurysms within the brain are apt to give simultaneous atrophy and expansion.

Cerebral arteriography.—By this method aneurysms (arteriovenous or saccular) vascular anomalies, neoplasms or cerebral arterial occlusions are demonstrated. Meningiomas which arise from the meningeal envelopes of the brain have certain favoured sites along the sinuses over the sylvian fissure at the lesser wing of the sphenoid in the olfactory groove and on the cribriform plate. They are tumours of slow growth and are usually benign. The menigiomas along the sagittal sinus in the region of the Rolandic fissure commonly produce attacks of focal epilepsy of a motor or sensory character in the contralateral limbs.

In cerebral angiograms, meningiomas can give a distinctive picture. The tumour circulation may cast a diffuse but faint shadow but in some cases actual vessels forming a close mesh work in the tumour itself and in its bed may be seen. The outline of the tumour vessels are often clean cut and regular in size. The arteries which feed the meningioma may be dilated. The collecting veins are distorted and in the phlebograms may be seen outlining part of the margin of the tumour.

Tuberculoma usually behaves like an intracranial tumour and the presenting symptoms may be one of epilepsy unattended by any signs of raised intracranial pressure or other symptoms of a progressive lesion.

We are able to recognize two groups of tuberculoma from angiographic appearances.

1. The vascular type, which is often deep and acts like a space occupying lesion. Surgery is often necessary.

2. The other group, smaller in number and shows mild vascularity. These are more superficial in location and require surgery only in selected cases.

Angriograms are also useful in determining the line of treatment to be adopted. Arterial shifting may occur without increase of intracranial pressure. In such cases surgery is not indicated.

The other features that have been noted are common to other space occupying lesions.

Arterio-Venous Malformation.—Although congenital in origin these lesions do not usually give rise to symptoms until early adult life. Sensory or motor focal epileptic attacks are common. The lesion consists of a coiled mass of vessels, some of them arteries, some of them veins, on the surface of the brain and extending into its substance. The essential feature of the lesion is that there is direct communication between the abnormal arteries and veins, as a result of an error of development of the vessels.

Cerebral angiography is essential both for diagnosis and for deciding the time of treatment. The arteriograms vary considerably from one case to another. The feeding arteries are often dilated to a great extent proximally.

Case Report.—Mrs. V., aged 40, admitted on 21st December 1959, into the Neurosurgical ward for focal fits starting in the left middle finger and spreading on to the left arm and forearm of duration of one year.

Clinical examination revealed normal fundus and no neurological deficit. Plain X-ray of the skull showed no abnormality.

A right cerebral angiogram was done and this revealed an arteriovenous malformation over the right parietal region. There was no shift of the anterior cerebral to the opposite side.

Case report 2.—Mr. S., aged 32, admitted for fits of three years duration. Neurological examination did not reveal any abnormality and fundus was normal.

Plain X-ray studies of the skull and chest did not reveal any positive findings.

Right cerebral angiogram showed a shift of the anterior cerebral to the opposite side in the antero-posterior view (Arterial Phase). In the lateral view (arterial phase) looping of the frontopolar artery was seen.

At operation these changes were found to be due to a tumour in the left frontal lobe. On histopathological examination, this proved to be " ASTROCYTOMA ".

CONVULSIONS IN CHILDREN

Convulsions in children pose an important problem for the medical practitioner. This is a fairly common condition and causes a lot of worry and anxiety to the parents, the grand parents and the relatives. As the onset is dramatic and the features frightening, the role of the practitioner becomes important.

Febrile convulsions are common in children after the age of six months. It is not definitely known why some children are prone to get convulsions during the rise of temperature and why some others do not. The temperature that produces a headache or a rigor in an adult may cause a convulsion in a child, as the cerebral neurons are more irritable in childhood.

Certain fevers more than others are prone to cause convulsions. In the Western countries scarlet fever is a common cause of febrile fits. The upper respiratory tract infections, pyelitis, otitis media, pneumonia and exanthemata are likely to cause convulsions in children. Hence it is necessary in febrile convulsions to examine the child for evidence of infection in these respects.

In exanthemata fits are not uncommon. Fits occurring at the onset of the exanthemata are of less serious significance than fits occurring after 10 days. Occasionally it may be of more serious import indicating the onset of an encephalopathy. These encephalopathies may leave few residual signs or may lead to severe neurological sequelae. Post vaccinal convulsions occurring after 10 days may carry a serious prognosis, if the fits persist for a long time.

In whooping cough, fits may occur and may prove to be quite severe. When the convulsions begin the whoop may be suppressed and hence the diagnosis may be missed. It is wiser to enquire about the presence of a whoop, when a child gets convulsions. Encephalitis following whooping cough is relatively more benign.

Vigorous treatment is necessary for febrile convulsions as the convulsions may damage the brain if they are prolonged or repeat themselves too quickly. Hence drugs are necessary as well as measures to control the fever. In prolonged fits it may be necessary to use ice bags to bring the temperature down. In some centres like Melbourne controlled hypothermia is used in children with prolonged convulsions.

Acute nephritis may also cause convulsions in children. In nephritis in infants edema is not often prominent. Hence blood pressure and urine examination becomes essential.

Drugs containing ingredients which are convulsants may cause fits in children. It is worthwhile enquiring whether the child is taking any such tonic and stop it.

Drugs are also necessary because the fits may be the harbinger of regular epilepsy. Paraldehyde by injection, chloral hydras and potassium bromide by mouth are useful drugs. Injectable phenobarbitone is also of great value. The injections may have to be repeated every four hours till the fits are controlled. The hydantoin groups of drugs may also be given simultaneously but they are not of immediate value as it takes two or three days for their full effect to be felt. Along with the drugs, enema to clear the lower bowels is often indicated. Measures to control the temperature and the infection have to be undertaken. Antibiotics are indicated as septicemia in a child may mimic encephalitis. If fits are not still controlled it may be necessary to put the child under continuous anaesthesia and relaxants. For this the help of a trained anaesthetist is invaluable.

Meningitic and encephalitic infections.—Causing convulsions are much more serious than febrile convulsions. Hence the practitioner has to be on guard to exclude these conditions. This is specially so because nuchal rigidity and Kernigs sign in infants may not be seen at all. *Hence L.P. becomes very important* in such cases. Examination of the cerebrospinal fluid, the cell count, its biochemistry and bacteriology are of great help. Acute meningeal infections are not uncommon in children.

Occasionally tuberculous meningitis may have an acute onset and has to be borne in mind. Usually in tuberculous meningitis fits come 10 or 15 days after the onset. The cerebrospinal fluid examination is invaluable in these cases. Subdural effusions, cerebral abscesses and venous thrombosis may also cause convulsions.

One-third of all the fits in children are due to extracranial causes. Tetany is not uncommon though it is not recognized as often as it should be. This is caused by hypocalcaemia. Rickets is common in our country. In such cases tetany may be precipitated during fever and may be confused with febrile convulsions.

Lead encephalopathy is occasionally seen and should be recognized by its clinical signs.

Severe acute diarrhoea may occasionally cause convulsions. In children on tinned foods specific pyridoxine deficiency may induce fits.

Teething is often blamed as a cause of convulsions in children. There is no obvious direct connection between teething and convulsions. They occur at the same age group. Round worms are said to cause convulsions but neurotoxic manifestations of round worm infection are rare.

Convulsions in the new born.—Some causes of convulsions in the new born are indicated below and require appropriate treatment :—

(*a*) Intracranial birth injury.
(*b*) Kernicterus.
(*c*) Excess coramine at birth.
(*d*) Tetany.
(*e*) Tetanus.
(*f*) Anoxia, hypoglycaemia.

Lastly there are some children in whom there is no discoverable cause. The treatment given is symptomatic.

Studies have been made to find out the percentage of children with febrile convulsions who turn out to be epileptics in later life. This is not as high a percentage as one may be led to believe. About 10 to 15 per cent only go on for regular idiopathic epilepsy.

SURGERY IN EPILEPSY

Introduction

The treatment of Epilepsy has been related to the changing theoretical conceptions of the etiology of the disease, since the beginning of history. On seeing the handwork of pre-historic surgeons in the skulls of the neolithic period of man in Europe and of the early Incas of Peru, one may surmise that the operation of trephining constituted a semi-religious procedure designed to provide a portal of exit for evil spirits, the accepted cause of epilepsy and insanity then. Hippocrates denied that the convulsions were of divine origin and had given directions for trephining on the side of the head opposite to a local convulsion. After the dark ages, Barron Larry, a great Surgeon during Napoleonic war, related the case of focal epilepsy in

case of caries skull, where the attack was produced by pressure with finger. After the introduction of asepsis and antisepsis the flood gates of surgery were opened and Neurosurgery started making great advances. Horsley did the first craniotomy for post-traumatic epilepsy by excising the scar with the surrounding brain tissue. The patient was cured of his fits. Krause carried out the focal cortical excision in 1893. Rowe and Watts in 1936 and Tonnis in 1939 reported good results by excising the cerebral scar of post-traumatic epilepsy. After Penfield's work in Montreal interest in cortical excision has been aroused.

Varied procedures were adopted in earlier years for the cure of epilepsy. Bleeding from the arm or thigh, cruciate incisions in the occiput, cauterisation of the scalp and brain to thin out viscid humour or movable valve operation to prevent excessive increase in cerebro spinal fluid pressure, cervical sympathectomy and encexphalomyopexy to promote blood supply to the brain, castration, repairing acute angulation and flexure of the sigmoid colon and radical colectomy to cure constipation have all been done.

In treating a case of epilepsy, it must be remembered that there are many different causes for fits and that each type requires appropriate therapy. The physician who uniformly administers anticonvulsants is open to equal criticism as the surgeon who carries out a cranitomy without a reasoned and therapeutic plan. Effective surgical therapy cannot be described without a brief consideration of the pathology of the forms of epilepsy which are susceptible to such treatment. To discover the epileptogenic focus careful survey of the attack pattern by a study of the history of the disease and by a study after induction of an attack is essential. Neurological examination, E.E.G. and radiology are also essential.

The surgery of epilepsy is mainly concerned with symptomatic epilepsy. The essential pathology in symptomatic epilepsy is an epileptogenic lesion. The epileptogenic area is a lesion in central nervous system which by virtue of its presence induces in the ganglion cells under its influence a state of hyperactivity that results in the periodic neuronal discharge of an epileptic seizure. The ganglionic portion of the lesion is not a focus of epileptic discharge. The neurons in or near the pathological lesion are subjected to irritative influence and thus form a part of the true epileptogenic lesion. At the periphery of the minute areas periodical impairment of blood flow through one or another of small local blood vessels occurs. This relative

ischæmia produces hyper irritability of the nerve cell and results in fluctuating increases of electrical potential; this is seen in the E.E.G. as the brief high amptitude waves or spikes. If this periodical irritation and stimulation continues atrophy advances gradually and continuously.

Symptomatic Epilepsy

Symptomatic epilepsy can be caused by expanding lesions or atrophic lesions. The most important expanding lesion is an *intracranial tumour.* 30.9 per cent of tumour cases have epilepsy as a symptom (51 per cent of these were intracerebral and 67 per cent were extracerebral). The nearer the tumour is to the central sulcus the more is the likelihood of epilepsy. Epilepsy as a symptom is more frequent in the temporal, the frontal and the parietal regions in the same order. Incidence of epilepsy in slow growing tumours is high as shown by the following figures :—

	PER CENT.
Gliblastoma	44
Oligodendroglioma	86
Astrocytoma	84
Meningioma	71

It is the abnormal area between the tumour and the normal cortex which causes the abnormal discharge. The tumour by itself is inert. The onset of epilepsy in an adult for the first time should be investigated with all the available methods to exclude an expanding lesion.

Case Report No. 1.—Patient. . . S. . . (N.S. No. 574|58), Age: 38; was admitted on 1st February 1960 for focal fits of the left upper and lower limbs on and off for the past three years. The attacks started with unconsciousness. There was no aura. Neurological examination did not reveal any abnormality. Plan X-ray of the skull was not contributory. Right

Cerebral angiogram revealed a tumour in the right frontal region. On craniotomy a tumour was found near the falx infiltrating the cortex. The cortex was firm and vascular in the right frontal lobe. The tumour was removed and the pathology report was an astrocytoma.

It is our practice to give patients who come with fits, anticonvulsants for three years post operatively; to those patients without fits no anticonvulsants is given unless they develop fits.

Subdural hoematomas.—Do not cause fits unless associated with brain injury.

Brain abscess is apt to be associated with focal epileptic seizure during the early stage. Recurring seizures are rare. They may occur months and years after the initial infection as the result of a scar.

Atrophic Lesions

During birth, hæmorrhage, contusion, laceration or vascular occlusion may occur. If occlusion of a cerebral artery occurs the part of the hemisphere supplied by it is destroyed and the necrosed brain may disappear quickly leading to ventricular dilation on that side. If two or three gyri are affected by laceration, focal compression or focal ischæmia, they become atrophic leading to focal microgyria. These lesions can cause seizures after five or ten years and even after twenty or thirty years especially in birth compression of the temporal lobe. The healthy gyrus overgrows the atrophic gyrus. The total volume of the hemisphere is less than the opposite side. This leads to asymmetry of the skull well shown in an antero posterior X-ray of the skull. The growth of the cranium in infancy depends on the outward thrust of the brain. It expands rapidly during the first year or two. The growth of each hemisphere depends on the individual enlargement of each lobe and that of each lobe on the gyrus. Brain cysts may be present and may cause fits. They are fluid collections within the hemisphere usually connecting with the ventricle or the subarachnoid space. These occur as a result of occlusion of a large artery probably due to meningitis and encephalitis in childhood. Meningitis may cause meningo-cerebral ciatrix due to inflammatory closure of pial vessels and they are usually not amenable to surgery.

Seizures will be considered for operation when there is a focus and when medical treatment fails. Two cases are reported here.

Case Report No. 2.—Patient C. (N. S. No. 489 of 1960) age 8 years was admitted for focal fits of the left side of the body. At the age of 1½ years he had an attack of high fever which lasted for about seven days during which time the child was unconscious. After the fever it was found that the left side was paralysed. Later the child was found to be restless and the mental development was poor. Fits developed involving the left upper limb. The frequency of fits increased and could not be controlled with medication. On clinical examination the patient was restless. Memory and intelligence were poor but eranial nerves were normal. There was a left sided hemiplegia.

Plain X-ray was normal.—E.E.G. showed a big area of cortical atrophy on the left side. So it was decided to remove the focus of atrophic brain with the help of electro-corticography. Cortical excision of the atrophic area was done.

After surgery the patient improved and the fits were controlled with anticonvulsants. His mental condition also showed definite improvement.

Case Report No. 3.—Patient S.N. was admitted for fits of four years' duration. Four years ago she had high fever for two days during which she was unconscious. From that time onwards she developed regular attacks of fits.

The fits started in the right half of the face and spread to the right upper and lower limbs. The fits increased to as many as 5 or 6 in a day. Neurological examination revealed a right sided hemiplegia with upper motor neurone type of facial palsy on the right side coupled with mental retardation.

The left cerebral angiogram showed a shift of the anterior cerebral to the same side. Since the patient had fits with mental retardation craniotomy was done on the left frontal region. The dura was found thickened. The cortex was found leathery and yellowish and therefore removed.

The fits were controlled by anticonvulsant post-operatively. The mental calibre of the patient is now improved. Removal of an abnormal irritable focus improves function in the rest of the brain.

Inflammatory and parasitic diseases.—Pyogentic meningitis and cerebral abscess can cause fits and suitable treatment for the original cause will control the fits.

Syphilis is not a common cause of epilepsy. Gumma of brain is rare and may be the cause of focal epilepsy.

Case Report No. 4.—Patient P. (23 years) N.S. No. 1118 of 1952 was admitted for fits of three months' duration. Three months earlier the patient developed twitchings of tne muscles of the right hand and then the facial muscles. Later on, the fits spread on to the right upper and lower limbs. The patient was unconscious for half an hour after the fits. During the period of three months he has had about 15 attacks. Neurological examination was normal, except for slight weakness of the right upper limb.

X-rays of the skull were normal. B.P. 140|90 Lumbar puncture showed cerebrospinal fluid to be clear. Proteins 70 m.g. per cent. Globulin: Nil. Chlorides: 759 mg. Sugar: 57 mg. per cent. Cells: 5 per cmm. Cerebrospinal fluid for Khan positive. The patient was given a full course of anti-syphilitic treatment. The fits did not subside despite this. The E.E.G. revealed a focus in the left motor area. Since the fits were not controlled with anticonvulsants and antibiotics, craniotomy was done on 13th October 1952. This revealed a well localised thickened area of dura at the expected site of irritation. This was removed. The patient was cured of the fits. The pathology report showed 'gumma' of the dura.

Tuberculoma is not an uncommon cause of fits.

Case report No. 5.—Pationt . . T . . aged 3 years was admitted for left focal fits and left hemiplegia of six months duration. The complaint started as fever and pain all over the body three months earlier.

Neurological examination revealed papilloedema with a left-sided hemiplegia.

X-ray skull showed evidence of increased intracranial tension. X-ray chest: N.A.D. Ventriculogram was done and it showed a tumour in the right frontal region. On craniotomy a firm yellowish tumour was removed from the right frontal region.

The pathology report was Tuberculoma. Patient was on antituberculous treatment and anticonvulsants. This patient did well and was completely cured of the fits. Tuberculomatous processes that are subcortical or infiltrate the cortex are more prone to cause epilepsy.

Cysticercosis is not an uncommon cause of fits in our country. The cysts lie in the cortex and subcortical tissue and induce an irritative focus. If the fit is focal, removal of cysts from the particular area may be of benefit.

Case Report No. 6.—Patient . .R . . (9 years) N.S. No. 1345|52 was admitted on 2nd October 1952 from the Ophthalmic Hospital for a complaint of blindness, headache and fits. These fits usually started in the left thumb and later spread to the rest of the left limb and the body.

Neurological examination revealed bilateral post-papillœdemic optic atrophy.

Ventriculography showed that the right ventricle did not fill up properly. The other ventricle was normal. A right lateral osteoplastic flap was turned and a big subdural hæmatoma was found. On excising the membranes of the heamatoma it was found that the cortex was studded with cysticercosis. They were removed. Post-operatively the patient was free from fits.

Focal epilepsy may be caused by *vascular lesions.* Arteriovenous aneurysms cause fits by causing ischæmia leading on to focal cortical atrophy.

Case Report No. 7.—Patient . . . R. . N.S. No. 2436|59, age 40 years complained of left focal fits.

Neurological examination showed no abnormality.

X-ray skull was normal. Right cerebral angiogram revealed an extensive arteriovenous malformation over the right parietal region. This was removed under hypothemia. The patient improved.

Focal epilepsy may occur *after trauma.* The incidence of post-traumatic epilepsy is ten times more common with penetration of the dura than without. After injury the damaged cortex is absorbed, neurones disappear and over-growth of astrocytes, connective tissue and fibroblasts occur. The scar attached to the dura is gelatinous in appearance and is tougher than the cerebral tissue. The collagen and glial fibres exert traction on the vaso astral framework of the brain, causing wandering of the ventricle, which can be demonstrated in pneumograms.

The cerebral cicatrix itself can cause epilepsy rarely. Cerebral cicatrix may occur due to cerebral contusion or laceration.

Case Report No. 8.—Patient . . N . . 18 years N.S. No. 38|60 was admitted for fits on the left side of the body of a year's duration. He gave a history of head injury five years ago.

Neurological examination revealed no abnormality.

Plain X-ray skull showed a depressed fracture in the right frontal region. Craniotomy was done since the fits were not controlled by anticonvulsants. It revealed the dura adhering to the pericranium. There were firm adhesions between the cortex and the dura as revealed on separating the adhensions and opening the dura. There was a cystic cavity of 2 cm. diameter in the cortex as deep. The meningocerebral adhesions and the cyst were completely excised. Post-operatively the patient was free from fits.

Temporal Lobe Epilepsy

This requires special consideration as it is fairly common. Surgery is now being practised increasingly with success in this condition. Temporal lobe epilepsy includes epileptic seizures originating not only in the temporal lobe but also the adjacent region of insular and uncinate regions. Views differ regarding the pathology of non-neoplastic lesions causing temporal lobe epilepsy. Penfield, Baldwin and Earl consider all the atrophic lesions in temporal lobe as due to birth injury. During birth compression, the medial and the inferior portion of the temporal lobe, viz., the Uncus and the Hippocampus herinate through the tentorial opening. The arteries, viz., the anterior choroidal artery and some branches of the middle cerebral and the posterior cerebral arteries may be compressed resulting in temporary ischæmia of the regions supplied by these vessels. This lead to atrophy of the anterior part of temporal convolution, deep portions of the Sylvian fissure, the Uncus and the Hippocampus. This lesion which occurs as a result of compression at the incisura of the tentorium is called incisural sclerosis. Others consider the same mechanism will operate in cerebral oedema resulting from encephalitis of childhood and also in head injuries in the adult.

recent drugs bromides have largely been given up in the treatment of epilepsy but they find a place even now in the management of some intractable cases.

Phenobarbitone.—The barbiturate group of drugs came into use at the beginning of this century and have proved their value in controlling epilepsy. Phenobarbitone is the chief drug in this group. It has been found to be of immense value in controlling most convulsions. It is also of value in treating acute cases as injectable barbiturates are available. The chief drawback of the barbiturate group of drugs is the tendency to induce sleep in many patients. This led to further researches on anticonvulsants and in 1934 Merrit and Putnam discovered the hydantoinates after chemotherapeutic trials of a large number of drugs.

Hydantoinates.—The hydantoinate group of drugs form the chief basis of the present day treatment of epilepsy. Dyphenyl hydantoin is marketed under various names like Dilantin, Phenytoin, Eptoin, etc. A dose of 0.1 gram twice or thrice a day is found sufficient in most cases. The value of hydantoin is that in the majority of patients, it does not induce sleep or drowsiness. The best combination has been found to be hydantoinates with phenobarbitone 0.1 gram or 1½ grains of diphenyl hydantoin with 30 to 50 mgs. (½ grain) of phenobarbitone at bed time forms an ideal combination.

In cases which do not respond to the above, the dosage may be increased up to about 4 to 6 tablets of dyphenyl hydantoin (0.1 gram) and to about 100 or 150 mgs. of phenobarbitone, provided this does not make the patient drowsy.

The drugs have to be taken continuously, every day without a break for a minimum period of 3 to 4 years. This is the only way in which cure may be effected.

Mesantoin (methyl hydantin) is also of volue in conbating epilepsy.

Mysoline or primidone has been discovered recently as an anticonvulsant drug. Our experience has been that Indian patients tolerate mysoline less well than other group of patients reported in the literature. Giddiness and drowsiness may come on even with a small dose of primidone but in certain cases it has been found to be an useful adjuvant to other drugs. Certain other drugs like phenurone, etc., are also useful in combating epilepsy but require careful supervision.

The diones.—All the above drugs refer to the control of grandmal type of epilepsy. In petitmal epilepsy the specific drug is trimetha-dione and paramethadione known respectively as Tridione and Paradione. The usual dosage is 0.3 gram 2 or 3 times a day. In typical petitmal the results are dramatic.

Toxic Symptoms.—When these above drugs are being administered it is very necessary for the patient and the physician to be vigilant for the production of toxic symptoms. This is specially important with regard to the diones. Toxic manifestations may come either after prolonged administration of the drugs or after the first few doses.

Gastritis, nausea, vomiting, diarrhoea, giddiness and xtaxia may be the manifestations of hydantoin toxicity. Occasionally hydantoinates may depress the bone marrow and cause agranulocytosis. This is specially to be watched when tridione is being administered. Hypetrophy of the gums is seen in many patients receiving hydantoinates. This is no contra indication for the treatment. Daily hygenic care of the teeth the gums and mouth with regular massage of the gums often prevents the hyperplasia.

In addition to the medical treatment of epilepsy it is very essential to look after the social and the educational aspects of the life of an epileptic. He must be given all opportunities for improving his education. The co-operation of the educationist is very essential. Later when he seeks employment every opportunity must be given for the epileptic to get a useful employment. Education and useful employment prevent mental strain and inferiority complex in the epileptic. Such strain itself may induce an epileptic attack. The society owes to its disabled to encourage them to attain a state as near to normallity as possible and the epileptics deserve all our consideration and help.

CONCLUDING REMARKS.

Epilepsy is a subject that has been interesting physicians from time immemorial. In the foregoing symposium we have listened with interest to the various aspects of epilepsy its diagnosis and treatment. As has been emphasised, with proper diagnosis and treatment, it is possible for the majority of epileptics to have relief and to live a fairly normal and useful life. With the modern advancement in the investigation of the nervous system and in neuro physiology it has been possible to understand more about this disease. Incidentally the study of epilepsy has helped in unravelling the mysteries of the functioning of the brain also.

I am sure that in the years to come more useful drugs will be discovered that will act on intractable cases and we of the medical faculty shall be able to give benefit to almost all the patients, suffering from epilepsy.