# A CLINICAL APPRAISAL OF THE EPILEPSIES IN GENERAL PRACTICE

A Report by the Research Committee of the College of General Practitioners\*

In our first paper<sup>1</sup> we tried to show the incidence of epilepsy in the community. In this article we are drawing on the mass of clinical material collected during the survey in an attempt to outline and illustrate the many forms of epilepsy seen in general practice. In all, some 1,500 cases were collected and described on the *proforma* cards, some of them in great detail, and they covered a wide range of epileptic attacks.

#### **Definitions**

Major epilepsy is a very dramatic type of seizure, and the syndrome has been recognized all down the ages. It has had many and varied names such as the sacred disease, possession by spirits, falling sickness, and so on. There was no clear understanding of the etiology of the disease, or indeed its many ramifications, until the latter half of the last century, when the brilliant pioneer work of Hughlings Jackson laid the foundations of the modern concepts of the attacks. His definition given to the phenomena in 1879 was, "Epilepsy is the name for occasional, sudden, excessive, rapid and local discharge of grey matter". This accurate and almost prophetic description of epilepsy has been amply confirmed by electroencephalographic studies which were initiated by Berger in 1929. In more recent times Penfield and Jasper<sup>2</sup> have done much to clarify the mechanism by reproducing epileptic phenomena in conscious patients by direct stimulation of the cortex.

Every doctor knows what he means by epilepsy, but to find an all embracing description to cover the many and varied forms of the condition was not easy. Dr Denis Williams advised us to use the following definition: "Epilepsy for our purpose will include all attacks primarily cerebral in origin, in which there is a disturbance of movement, feeling, or consciousness. It will therefore obviously

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not include fainting, aural vertigo, or psychologically determined attacks."

# The Mechanism of Epileptic Phemonena

An epileptic attack occurs when some of the brain cells react independently. This reaction may remain localized, or it may spread slowly or rapidly to other parts of the cerebrum. It can originate in two very different regions of the brain, the superficial cortical areas. or in the deeper brain stem regions. Cortical seizures tend to produce the first two types of disturbance suggested by Williams; that is, upsets of movement or feeling. Unconsciousness or disorders of behaviour are produced by seizures affecting the central integrating system of the brain stem, which extends from the thalamus down to the medulla oblongata. If the attack starts in this centrencephalon. the patient will as a rule lose consciousness at once, either in grand mal or petit mal, without any warning or aura. If however the reaction starts in a cortical focus it may spread by a kind of chain reaction, and when it reaches the centrencephalon it will cause a grand mal type of seizure. The cortical phase of the attack will produce an aura, but the major fit is the result of the brain stem reaction. If the attack moves slowly across the cortex, a typical Jacksonian fit will be produced. Hughlings Jackson's wife had such attacks, so that he had plenty of opportunities to observe them closely. Typically, twitching may begin in a single digit, and then "marches" up the hand and arm, leading perhaps to generalized convulsions and unconsciousness, but, of course, a Jacksonian fit can march across sensory areas, or indeed any part of the cortex, and it does not always extend to the centrencephalon and unconsciousness. For example, a woman of 45 started an attack by seeing flashes of light, and her vision failed so that she could hardly see across the room. Her left index finger then went numb, and this sensation passed up her arm to the elbow. Her left cheek then became affected, and the feeling extended round her mouth. She then felt just as if she had been to the dentist. These feelings all passed off in a few minutes, and the seizure was limited to cortical areas. Aura may start in any part of the cortex. For example one man smelt bacon frying before he had a "blackout". The ictal or epileptic focus here lay in the olfactory area of the cortex. According to Jasper and Penfield the epileptic cry is sometimes due to the stimulation of the phonation areas, and the cry is in fact an aura. With grand mal epilepsy, there is some degree of retrograde amnesia, so that there is always a chance that a brief cortical aura may have been forgotten by the patient. The focus of origin could be located by electroencephalography in many of these cases.

In the survey, examples of many kinds of fits were described. These

will be used to illustrate attacks arising in different regions of the brain.

## Epilepsy with loss of Consciousness.

The tonic clonic grand mal type of seizure is well known to all family doctors, and needs no detailed description here. If clonic twitching is localized to one side of the body, the attack is focal in origin. There may be no tonic or clonic movements at all, and the patient then falls suddenly without any warning. These akinetic attacks have to be differentiated from syncope. With an ordinary faint there is usually a causal factor and some warning to the patient. The soldier standing to attention in the sun, the girl fainting in church, the woman passing out on the receipt of bad news, are all examples of syncope. With akinetic epilepsy there is no apparent cause and no warning. The attacks recur from time to time until a suitable anticonvulsant is found. The child can fall suddenly while dressing. The mother downstairs hears the thump, and from past experience she knows at once what has happened.

B.B., a girl of 9, was given to fainting attacks. These were sudden and without any warning. She would just collapse unconscious in a heap. There was never any convulsive movement. She was given phenobarbitone and has had no further attacks.

These akinetic attacks are not uncommon and appear to be most frequent in the young 10—15 age group and among old people.

Petit mal, which always originates from the centrencephalon, is much less common than major epilepsy. There is usually a brief or partial break in consciousness. Abnormal muscular movements are rare except round the eyes, and are always bilateral. There may be some loss of muscle tone, and the patient if standing tends to stagger, but never falls. The patient is usually unaware of the attack, and will resume a conversation just where it was broken off.

Miss J. R., aged 37, had up to ten or twelve attacks a day. These consisted of a transient loss of consciousness, with a fluttering of the eyelids, the eyes becoming vacant and glassy. She did not fall over, but tended to sway a little. She could have as many as four or five such fits during a single interview, especially if she was excited or upset. Occasionally she would have a grand mal attack, after which she would be unconscious for from 20 minutes to 2 hours.

Consciousness may not be completely lost in such attacks.

M.G., a woman of 30, had attacks which lasted for not more than half a minute. She had no warning of their onset, and she never fell, but would often sit down and she would then ask the time or the date. She usually knew that she had had an attack which was recognized as such by the family; but those who did not know her might well be unaware that anything unusual had happened.

M.H., a girl of 18, was treated for *petit mal* with various anti-convulsants, and in the end she took primidone. These tablets had the effect of cutting out completely the attacks of which she herself had been unaware, but she continued to have occasional "queer feelings", which only she knew about. These subjective attacks were a more socially acceptable form of epilepsy than her old *petit mal* seizure.

Muscular movements\* in petit mal are rare, but when they do occur they are bilateral.

M.C., a girl of 2, had several "bouts" a week. They could occur by day or by night. The picture was always the same. The child would start making queer noises and she would stare into space. At the same time her arms would both be raised about her head. She was obviously far away. The attacks lasted only a matter of seconds, and shaking her seemed to curtail them.

Occasionally, a child will have scores of short petit mal attacks in a day. These bouts are to some extent psychologically determined as the patient tends to have more when crossed, and they are sometimes used by the patient to get her own way. The child will have half a dozen attacks during an interview at the surgery. In spite of the frequency of these attacks which used to be called pyknolepsy, the ultimate prognosis is good, and the child is usually free from them by the age of 12 years.

J.D., a small girl of 4, began to have *petit mal* attacks. When she started to go to school they increased in number and at times were almost uncountable. She would have several in the doctor's surgery within a matter of minutes. For a few seconds her eyes would go vacant and her head would nod. There was considerable improvement on diamox and by the time she was 11 her attacks were very rare.

Myoclonic epilepsy. Myoclonic attacks are centrencephalic in origin. We all of us experience this sort of violent muscular jerking on occasions as we fall off to sleep. Such an attack is physiological and a hypnogogic phenomenon. Myoclonic epilepsy is similar in form, but not in origin and it can occur either by day or by night. According to Lennox<sup>3</sup> it belongs to the petit mal group of seizures.

E. W., a man of 50, gave a history of attacks which had plagued him for years. Sometimes they occurred by day but more often they would awaken him at night. He had sudden attacks of cramp in both legs. The pain would make him get out of bed. He would go deadly pale and then pass unconscious for about 5 minutes. There were never any clonic movements, but there was occasionally a catch in his throat as he fell unconscious. He himself would go straight off to sleep after such an attack, but his wife who had seen it all, was always too afraid to drop off again. Weakness of the limbs affected by a fit lasting for a day or two after an attack is known as Todd's paralysis. E. W. had just such feeling in his legs, which he said felt tired and strained for the next 24 hours.

Drop attacks. The brain stem controls muscle tone as well as being the seat of consciousness, and an epileptic discharge in the appropriate area can cause a sudden loss of muscle tone so that the patient falls in a heap on the floor without any loss of consciousness. If this is brought on by emotion such as a "fit" of laughter it is called catalepsy. Usually there is no obvious cause and the patient is at a loss as to why she fell.

One old woman of 76 had had twelve such falls before she reported them to the doctor. For the past 4 years she has been on phenobarbitone gr. 1/4 three times a day, and she has had no further attacks.

B.S., a man of 63, with a brain stem tumour, first showed evidence of this

<sup>\*</sup>Some authorities hold that muscular movements never occur in petit mal,

lesion by his unexplained falls. The first happened as he reached up to fasten the top bolt of the door, and at once he found himself in a heap on the floor. Later, when on holiday, he went into a public convenience. As he came out his wife saw that he was sagging at the knees; he collapsed before he could reach the car. There was never at any time a loss of consciousness.

This "drop syndrome" is not at all uncommon, especially in old people, and in certain cases anti-convulsants can prevent further attacks. If the attacks are caused by ischaemia of the upper brain stem due to kinking of the vertebral arteries, anti-convulsant drugs will be of no benefit. Drop attacks with a loss of consciousness would amount to akinetic attacks.

# Focal attacks arising in the Cortex

Rolandic area. Movements initiated in this area are unilateral, and effect the contralateral side of the body. Only the mouth and lips have a bilateral representation.

R.P., a farmer of 74, used to have a series of attacks. These used to start with a twitching of the left face. It spread to his left arm and sometimes he became unconscious and incontinent. He could have as many as eight to ten attacks in a single evening if he forgot to take his primidone.

J.H., a girl of 6, awakened her parents because she was unable to control the twitching of her right arm. She passed urine under her and then became unconscious.

C.A., a man of 62, used to have attacks in which he used to lick his lips for about five minutes. He would then ask the time, and go to sleep for a while. His wife always knew the attack was nearly over when he asked the time.

Pre-Rolandic gyrus. There is a secondary motor area in this region on the mesial surface of the brain. Stimulation here is said to produce movement without any twitching, on the other side.

J.B., a boy of 12, used to have periodic attacks in which his left arm would go above his head, and at the same time his head would turn to the left. He would be embarrassed by the movement and he would try to pull the arm down with his right arm. His mother had noticed that often on awakening him, his head would turn to the left and his arm would go up. There was never any lapse of consciousness in this case.

Sensory attacks. These arise in the post central gyrus. An example has been given where a woman had attacks of numbness passing from the left index finger, up the arm to end in the face. Sensory and motor attacks can occur together.

P.B., a boy of 14, used to get a numb feeling on the right side of his neck and arm. Then the left side of his face would start to twitch. This was sometimes but not always the prelude to a *grand mal* attack.

Temporal lobe attacks. It is quite impossible to cover the subject of temporal lobe epilepsy in an article of this nature, so varied are the manifestations.

"The physical phenomena which are produced by epileptic discharge in the temporal lobe or by cortical stimulation are many, and include hallucinations of vision, hearing, smell, taste, touch: illusions of distance, size form, colour, motion, speed of sound and time: disturbances in equilibrium and body image; alteration of thought and affect, disorientation for time place and person; disturbances in speech and automatism, the latter with its accompanying memory defects."

This full and concise summary of the types of fit in a paper<sup>4</sup> by

Karagulla and Robertson give some idea of the wide range of these attacks. They are much more common than is generally appreciated and the diagnosis may be difficult. Some of the symptoms are very difficult for the patient to describe, and as a result she tends to be labelled as neurotic by her doctor and relatives alike. No one will take her symptoms seriously, and as a result she develops secondary anxiety to add to her troubles. It is possible that some of the reputation bromides and phenobarbitone have earned in the therapy of psychoneurosis, may be derived by the occasional case of temporal lobe epilepsy which had been relieved by such treatment.

Mrs S.R. was a woman of 50. She had had queer attacks from the age of 13. She could remember the first one vividly. She was playing marbles at the time when she suddenly became frightened. She ran home to her mother, but the attack had gone off before she got home. After that she had them at regular intervals, and they caused her great distress. She used to get a feeling of intense and "unnatural" fear, with a queer feeling in the epigastrium. She hated people to see her distress, and would wander away until it passed over. It only lasted two minutes or so, but it always seemed like half an hour. This alteration in the speed of time during an attack always surprised her. Nobody would take her attacks seriously. She was said to be neurotic and, indeed, she had long periods of depression. In middle age the attacks are not so frequent and they no longer distress as they did.

Mrs I.T., a woman of 41, came along with queer symptoms which she found very difficult to describe. The picture was not one of an anxiety state, and there was no response to psychotherapy. She became tearful and depressed, and rather desperate about herself. Amphetamine did not help her, but she improved at once on sodium phenytoin. Her attacks ceased, and she comes along regularly for her prescriptions. There was no history of a major attack, but her husband had a full blown grand mal in the garden. "He must be a bit like me" was her apt observation some time later.

Mrs I.B., a woman of 48, had a reputation for being a troublesome neurotic, an opinion which was shared by her doctors and the family. From time to time the general practitioner was called out at all kinds of odd hours to see her, and there was never any evidence of organic disease. When the possibility of temporal lobe attacks were mooted, a more accurate history was taken. She said that the attack started with a sudden feeling that everything inside her had stopped working. She felt that she was floating and no longer in herself. She had to keep sitting up to prove that she was still there, and all the time she felt indescribably frightened. She was referred to a neurologist and an E.E.G. confirmed temporal lobe attacks, which have ceased since she was given an anticonvulsant. Her relief to find that at last she has a "respectable" illness which can be treated was considerable.

Miss I.F., aged 43, had had queer attacks for some ten years. She felt faint and everything seemed to stop. She had a pain behind her eyes and a sick feeling in her stomach. The attacks often awakened her at night and she had 3 or 4 every week. They seemed to be getting more frequent and more severe. She was given sodium phenytoin and she had not had an attack for the past seven years, except for one short period when she lowered the dose of the drug.

All temporal lobe attacks are not as vague and difficult to describe as these cases, and many interesting focal fits in this area were described by participating doctors; some of which were as follows.

Auditory areas.

M.R., a woman of 37, had attacks which affected both the hearing and the

vesibular cortical areas. The incident would start with a feeling of fullness in her ears. There was no deafness, but she felt apprehensive, and would sit down. Everything would then begin to spin, and sometimes she was sick. Finally a "ping ping" noise would be audible to her, and this signalled the end of the attack. They usually lasted for from 3 to 5 minutes and she never passed out completely.

Olfactory areas. Attacks starting in this area are heralded by the aura of a bad smell or a queer taste.

E.R., a woman of 73, had the sensation of a peculiar, foul smell which was followed by intense giddiness and confusion.

M.C., an old woman of 91, used to imagine that the house was on fire because she could smell burning. The attack would pass off in a few minutes, leaving her agitated and distressed.

C.D., a woman of 53, used to have noctural attacks. She would awaken with a taste of liquorice in her mouth. This was sometimes accompanied by eneuresis, and the next day she would have a severe headache and anorexia.

Visual areas. These attacks are diverse in character, and can vary from simple flashes of light, or complicated hallucinosis.

S.C. was a man of 20 who was liable to attacks which usually came on at night. He saw a star which gradually became bigger until, after about half a minute, he passed out altogether.

E.D., a man of 38, used to see a candle fluttering before his eyes, and he had a feeling that he was in somebody's power\*. This was followed by unconsciousness.

Z.B., was a woman of 57 who used to have visions of scenes. Some were familiar, and some were of places she had never seen before. Things flashed through her mind for a second or two and then she would vomit and become confused.

Mrs S.Y., an old woman of 72, had the strangest attacks in the series. In her first fit she fell and broke both her wrists. A month later she had her first visual episode, in which she saw a little man in dark clothing with unrecognizable features come to her from the right side. He would stand laughing at her for about 10 seconds. She would then begin to make smacking noises with her mouth and the little man would disappear. There were many such visitations and on some of these occasions she would pass out altogether.

## Speech areas.

I.G., a woman of 55, has had attacks on and off for 30 years. She gets a sickly feeling and finds herself unable to speak for about ten nimutes. She does not lose consciousness, but is occasionally incontinent. She can carry on pouring out cups of tea and so on.

H.W. was a man of 37 who used to have several attacks a day and then he would be free from trouble for a month or more. He would find it impossible to get words out and he was unable to write during an attack. He felt that his consciousness was in no way impaired. He passed out completely on one occasion.

Déjà vu phenomenon. This is a borderline epileptic attack which is experienced by many normal people. It is attributed to the temporal lobes and is a condition of increased awareness.

M.O., a woman of 25, had had attacks on and off for 3 or 4 years. She used to see something like the clothes hanging on the line and it would come over her

<sup>\*</sup>Note the transient schizophrenic symptom.

that she had seen it all before. She then went cold and felt sick. She trembled and was very frightened as she thought she might pass out altogether. The attacks would last for 3 or 4 minutes and leave her with a headache.

R.C. was a man of 42 who had attacks which lasted for a matter of seconds only: He felt odd and any object that he was looking at seemed to assume excessive importance. He felt weak and had to sit down. Thoughts rushed madly through his head in a rapid series of images and memories. After 2 or 3 minutes he was fit to carry on with his work.

W.G. was a man of 20. He had sudden attacks of giddiness and strange feelings of familiarity, followed by a period of unconsciousness which lasted for several minutes.

Feelings of unreality. These attacks are in a sense the converse of excessive awareness. Things tend to feel strange and unreal.

G.K. was a woman of 47 who was liable to several attacks in the course of a week. Soon after getting up she would have a strange feeling of unreality, accompanied by a tingling and numbness of the left arm and leg.

G.P., a man of 38, used to get attacks in which he felt he was no longer inside himself. His body would go on working, but he was no longer inside it. The episode would last for from 5 to 10 minutes. It was in no way a fearful experience as he had no feelings, either good or bad.

Psychomotor automatism. The site of this lesion is by no means certain. Penfield and Jasper ascribe it to the centrencaphalon, but others suggest that they originate in the temporal lobes.

Miss G.H. was a woman of 65 when she started to have her attacks. They were initiated by fumbling, sometimes with her clothing and sometimes with her hand bag. She was mentally far away, and not interested in her environment. She would sometimes empty her food into her lap, or lift a bowl of soup above her head. Once in the theatre much to the embarrassment of her friends she stood up in the middle of an act, worked her way to the end of the row and tried to open the emergency doors. She never had any knowledge of what she had done after such attacks, which were abolished once she was put on to primidone.

S.V., a man of 64, used to have attacks in which he champed with his lips, while he looked pale and dazed for a few minutes. He was bewildered for some time after the attack. This kind of seizure has been aptly called by Penfield a masticatory pantomime.

D.B., a man of 31, was seen in an attack of confusion. He was not unconscious, but he sat gazing into space. If questioned he looked worried but could not reply. He resented any attempt to do anything for him. The attacks would last for from 10 to 20 minutes, and he had no recollection of them afterwards. On one occasion he must have passed out altogether as he was surprised to find himself lying in a ditch.

#### Discussion

A large number of cases have been described to illustrate the wide variety of epileptic phenomena. Many of these attacks merge with the normal experiences of every day life. For instance narcolepsy is an exaggeration of the postprandial nap enjoyed by many men on a Sunday afternoon. Myoclonus as one drops off to sleep is a physiological experience. Most people at some time in life have déjà vu phenomena. As Penfield is reputed to have said, we are all epileptic, but some of us are more epileptic than others.

The first paper on this survey suggested that about five per cent

of the population had an epileptic attack of some sort during life. If one included all the minor epileptic phenomena it is quite obvious that this figure must be very much higher. Many of these minor forms are accepted without complaint by the patients: and many more people who go to their doctors because of their symptoms fail to have them accurately diagnosed. In this paper we have tried to illustrate some of the more unusual types. To be aware that they exist is the first step towards better diagnosis. Lennox<sup>3</sup> gives four criteria of diagnosis, to which we have added three more. Six out of these seven diagnostic points can be sorted out and clarified in the consulting room of the general practitioner.

# Diagnostic criteria.

- \*1. An accurate history of the attacks both from the patient and relatives who have seen the attacks is fundamental. Epileptic phenomena which do not produce unconsciousness usually only last for a few seconds or at the most a few minutes.
- 2. If the patient has an ictogenic focus, this will always produce the same kind of attack. This sameness of the episodes is quite typical. Penfield and Jasper were often able to locate these foci in the conscious patient, as the patient was able to recognize when the electrode was reproducing the symptoms of his attacks.
- 3. If the attack awakens the patient in the middle of the night, this is almost pathognomonic of epilepsy.
- 4. The patient who presents with one form of epilepsy often has other manifestations, such as déjà vu phenomena, ictal fear, uncinate smell fits, feelings of unreality, and so on.
  - \*5. There is often a family history of epilepsy or migraine.
- \*6. The attacks are often dramatically obliterated by an anticonvulsant drug or a combination of such drugs.
- \*7. Electroencephalographic records are a most useful objective diagnostic weapon. It is in fact the only major diagnostic instrument not directly available to the family doctor. It is beyond the province of this paper to discuss electroencephalography. Suffice it to say that not only is the investigation most useful as a confirmatory test, but the type of wave can often dictate the form of treatment to be prescribed.

The primary function of the family doctor in a study of the epilepsies is that of diagnosis. *Grand mal* attacks are self evident, but many of the minor forms pose a diagnostic problem. If we in general practice are to be encouraged to spot these cases more frequently, our neurological and psychiatric colleagues must be

<sup>\*</sup>The criteria of Lennox.

prepared to help us, both in confirming the diagnosis, and advising us on treatment, or bearing with us if the case is not what we thought.

## Summary

The mechanism of epileptic phenomena is discussed.

Various forms with loss of consciousness are described.

Examples of focal epilepsy are given.

The difficulty in diagnosing temporal lobe types is stressed.

Diagnostic criteria are listed. The family doctor is in a very good position to spot these cases, once he is aware of the many and varied forms.

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#### REFERENCES

- (1) College of General Practitioners. Brit. med. J. 1960. 2, 416.
- (2) Penfield, W. Jasper, H. (1950). Epilepsy and the Functional Anatomy of the Human Brain. London.
- (3) Lennox, W.G., (1960). Epilepsy and Related Disorders. Vol. II. London.
- (4) Karagulla, S., and Robertson, E. E. Brit. med. J. 1955. 1, 748.