EPILEPSY
TOWARDS TOMORROW

HIPPOCRATES MEDECIN

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Cover illustration of Hippocrates by Thevet (1584) courtesy of Mary Evans Picture Library.
Hippocrates (460-377 BC) was the first person to recognise epilepsy as an organic process of the brain.

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To collect data from other countries.

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Introduction

Since the publication of the Office of Health Economics' first booklet on epilepsy in 1971, advances in medical treatment, better seizure control and wider availability of appropriate information and counselling opportunities are making it somewhat easier for people with epilepsy to adjust to the condition and integrate fully into society. However, the quality of medical care varies, as does the availability of information and counselling support and thus for many people epilepsy continues to be a distressing condition to accept and live with.

Difficulties may arise from a variety of different factors. In a survey of nearly 2,000 people with epilepsy (Table 1) it was found that they were experiencing significant and wide ranging problems in their daily lives (British Epilepsy Association, 1990).

The unpredictable nature of seizures and the often erratic course of the condition from year to year can create stress for the individual

Table 1 **Frequency of problems reported by survey respondents: number of respondents: 1,958**

Aspect of life	*No problems* %	*Some problems* %	*Serious problems* %	*Problem index** %
Driving and transport	17.8	37.8	44.5	82.3
Social life/leisure	28.9	56.4	14.7	71.1
Employment	27.7	35.4	36.9	72.3
School	36.8	40.5	22.7	63.2
Further education	57.1	29.8	13.2	43.0
Vocational training	61.1	25.2	13.7	38.9
Self image and well-being	29.0	52.3	18.7	71.0
Legal matters	81.1	14.2	4.7	18.9
Medication	27.4	50.1	22.5	72.6
Personal relationships	47.5	40.9	11.6	52.5
Health care from doctors etc	58.4	31.7	10.0	41.7
Support from Social Services	70.0	18.1	12.0	30.1
Insurance and pensions	50.8	35.0	14.1	49.1
Claiming welfare benefits	70.4	19.5	10.1	29.6
Society's attitudes	31.8	49.5	18.6	60.1
Having a family	67.2	20.4	12.3	32.7
Other	29.4	28.7	41.9	70.6

* The 'problem index' column contains a measure derived from combining 'some' and 'serious' problems and reflects the overall degree to which an aspect of life is problematic.

Source: British Epilepsy Association, 1990

and their family. In the same survey, 71 per cent of respondents said that they had problems with their self image and well being. Self confidence and self esteem can be undermined particularly if there is a sudden return of seizures after a period of remission or if an attack occurs in public during which there is a loss of body control and incontinence. There may also be anxieties, sometimes exaggerated, about injury, brain damage or death resulting from seizures.

Family reactions may also create problems for the person with epilepsy. Sometimes there is a denial of the diagnosis, concealment of the condition and rejection of the person. Other families may be over protective tending to create dependency. Family relationships are considered in more detail later in the paper but it is important to note that excessive protection of children and young adults with epilepsy can intensify the tensions which occur naturally during adolescence and/or result in the long term immaturity of the individual.

Employment difficulties continue to be commonplace. In the survey by the British Epilepsy Association (1990) 72.3 per cent of respondents indicated that they had experienced problems with employment. Many occupations today have a car driving component, and a diagnosis of epilepsy may bring with it not only the immediate loss of driving licence but also the consequent loss of a job. Some restrictions on employment for people with epilepsy are legally based, for example driving public service vehicles is prohibited to anyone who has had a seizure since the age of five. But some discrimination in employment against people with epilepsy is based on false assumptions and generalisations about the condition. The extent to which people with epilepsy face problems in obtaining employment is discussed more fully in a later section of the paper.

The costs of epilepsy to society and to the individual are considerable. It is estimated that the cost of epilepsy to the United Kingdom National Health Service in 1988 was £109.27 million (see Table 13). This figure includes the cost of hospital in-patient stays, general practice consultations and pharmaceuticals. However this figure does not take account of the costs of underemployment, vocational rehabilitation and special education, residential care and excess mortality. A study conducted in the United States (Commission for the control of epilepsy, 1978) considered these areas in their calculation of the costs of epilepsy and arrived at a cost (inflated to 1989 prices and converted to sterling) of approximately £5,000 million. If it is taken that the population of the UK is approximately one-fifth of the USA and it is conservatively assumed that the cost to the UK is only a half of that in the USA it can be estimated that the cost of epilepsy in 1989 in the UK was £500 million. Even this calculation does not deal with the question of the cost to the individual with epilepsy of a restricted quality of life.

With a disorder as common as epilepsy and one which presents a major medical, social and economic problem it might be expected that reliable information on its frequency and distribution would be readily available. Unfortunately this is not the case. Several factors contribute to this lack of epidemiological data. Firstly, doctors are not obliged to report patients with epilepsy to local health authorities; consequently it is difficult to accurately assess the frequency of the disorder. Secondly, the results of epidemiological studies have shown some considerable discrepancy due to differences in epilepsy definition, measurement methodologies and analytical methods (Holmes, 1987). These epidemiological studies are examined later in the paper.

The third factor which contributes to the lack of information about the number of people with epilepsy is that not all people with epilepsy are evaluated by a doctor. This may be due to the very mild symptoms experienced by the person or a fear of prejudice in others.

The fear of prejudice and how people will react to them are perceived to be very real problems for the person with epilepsy. However, it is worth considering to what extent there is prejudice against people with epilepsy and to what extent it is simply a reaction to the sometimes defensive attitude of the person with epilepsy who expects to be treated with prejudice. Whilst it cannot be denied that people with epilepsy are discriminated against in all areas of life, from education and employment to the forming of personal relationships, discrimination is almost certainly less prevalent than some people with epilepsy believe. By expecting to be discriminated against and consequently hiding their condition, or being less than open about it, it could be argued that these people are helping to keep prejudice alive.

However, it is probably true to say that few conditions or diseases have the kind of stigma which is attached to epilepsy. The explanations which have existed over the centuries as to the causes of epilepsy, which are discussed in the following section, probably go some way to explaining the stigma. But perhaps a more important reason for the stigma is fear due to ignorance about the nature of epilepsy. Epilepsy is often confused in the mind of the public with mental handicap or mental illness.

For most of the population epilepsy is commonly associated with the most dramatic seizure type, the generalised convulsion or as they were formerly known the 'grand mal' seizure; it is sometimes believed that once a person has experienced this type of seizure, they will continue to have seizures for the rest of their lives. This indicates several misconceptions about epilepsy: in the first place not all epileptic seizures are generalised seizures, although they are the most common (the different types of epilepsy will be discussed later

in the paper); secondly, it is estimated that between 70 and 80 per cent (Goodridge and Shorvon, 1983, Duncan, 1991) of people with epilepsy will have their seizures controlled with adequate medication; and finally, although there are approximately 30,000 new cases of epilepsy diagnosed each year in Britain, Goodridge and Shorvon found that approximately 70 per cent of patients went on to long term remission.

Prejudice against epilepsy and the person with epilepsy, though it appears to be lessening, still exists. The only effective way of overcoming this prejudice and ignorance is through publicity and education. It is hoped that this paper will contribute to a greater understanding by both the public and health care professionals of the nature and causes of epilepsy and a greater awareness of the problems encountered by people with the condition. It is important for those who do not suffer from epilepsy to realise the extent of the problem, in order that those with epilepsy have an opportunity to lead a better and fuller life in the future.

Historical aspects

Until relatively recently epilepsy was considered to be a single disease rather than, as we know today, a sign of underlying brain dysfunction. As consequence, the term 'epilepsy' has been taken and applied to a variety of conditions. The only common factor among these conditions is the propensity for the occurrence of a 'paroxysmal (occasional) discharge of cerebral neurones sufficient to cause clinically detectable events that are either apparent to the subject or to an observer' (Hopkins, 1987), or in other words a seizure.

The words 'epilepsy' and 'epileptic' are of the same origin as the greek word 'epilambanein' meaning 'to seize' or 'to attack'. As far back as Babylonian times it was believed that all diseases were attacks or seizures by gods or demons. Over time this concept has been applied almost exclusively to epileptic seizures, during which the affected individual was unconscious but their body was agitated as if someone else was in control of its movements.

Hippocrates (460-377 BC) was the first person to recognise epilepsy as an organic process of the brain. In the book 'On the Sacred Disease' of which Hippocrates has been attributed as author, he condemns the supernatural theory for the cause of epilepsy as a 'shelter for ignorance and fraudulent practices' (Temkin, 1971). Hippocrates believed that epilepsy was hereditary and was caused by too much phlegm in the brain and in the arteries leading to the head, resulting in the air supply being cut off (it was believed at this time that blood

only circulated in the veins whilst arteries carried air). The sight of foam around the mouth of a person having a seizure was taken as proof of the accumulation of phlegm. Hippocrates argued that because (as he believed) the releasing factors for the attack were the cold, sun and wind, and since these environmental factors were clearly divine it therefore followed that because these factors affected all diseases, all diseases were divine. Epilepsy, said Hippocrates, should consequently not be treated by magic but by diet and drugs.

For thousands of years people continued to believe that the body contained four liquids, blood, black and red gall and phlegm. In 100 AD Galen suggested that epilepsy was caused by a build up of phlegm in an arm or a leg which explained why convulsions often started in these limbs and then spread to the rest of the body. At the time the recommended treatment was for a tourniquet to be applied to the affected limb or even for it to be amputated. If the seizures did not have a localized start even more drastic measures were taken to remove the phlegm, which by then was thought to have reached the head. The removal of the phlegm was achieved by boring a small triangular hole in the skull, usually at the rear, to create a drain.

During the Middle Ages magical practices, some merely superstitious (amulets of peony and stone), others bordering on the occult (frog's liver, dog's blood and crushed human bone) were popularly used in the treatment of seizures. It was not unknown for the village priest to assist in the performance of some of these rituals.

Throughout the centuries the strength of the belief that epilepsy was caused by demon possession owes much to the spread of Christianity. In the New Testament of the Bible, Jesus drives out a spirit which had possessed a young boy since he was a small child and frequently caused him to be thrown to the ground, foaming at the mouth and in convulsions. Although it is not stated to be epilepsy, early physicians recognised that the boy demonstrated the main characteristics. As a result of this early diagnosis the belief that epilepsy was due to demon possession was popularised; an idea which still prevails today in certain parts of the world. Less than a decade ago a girl died following an epileptic seizure in Germany because a priest persisted in trying to exorcise the devil instead of referring her to a hospital (Meinardi, 1989).

This belief, understandably, created certain problems of diagnosis and treatment. Religious therapies consisted of prayer and fasting based on Jesus' explanation to his disciples when expelling the spirit from the young boy (St Mark 9 v 29, St Matthew 17 v 21) that 'This kind come forth by nothing but by prayer and fasting' and fasting in fact formed the basis of early twentieth century treatment for epilepsy. The patient first fasted for a few days and was then introduced to a special diet. The ketogenic diet has a very high fat content

and in addition to being unpalatable is also expensive. With the development of a variety of anticonvulsant medications this therapy is rarely used today. However, according to the Greek translation of the biblical text it is clear that it is the healer rather than the patient who is supposed to pray and fast.

It was also thought for a time that epilepsy was an infectious disease caused by various toxins which could attack the body from the outside. Convulsions were the body's attempts to get rid of these toxins in the same way as it was believed that hiccups were the stomach's attempt to get rid of damaging food. This belief naturally gave rise to a marked social discrimination against sufferers and led to the establishment of epilepsy colonies along the same lines as those for leprosy, some were still in existence in the United Kingdom in the 1960's.

Advances in neurology in the eighteenth and nineteenth centuries re-established the view of Hippocrates that epilepsy was caused by diseases of the brain and this led to a better understanding of the nature of epilepsy. Hughlings Jackson (born 1835) advanced contemporary knowledge still further, his greatest achievement was the formation of the concept of epilepsy as a sudden discharge of cerebral neurones, a concept which has stood the test of time and been confirmed by electroencephalographic (EEG) studies. Jackson defined epilepsy as 'an occasional, sudden, massive, rapid and local discharge of the grey matter' (Scott, 1969).

This enlightenment did not however bring an end to the myths about epilepsy, which were generally believed to be facts. In the latter part of the nineteenth century sexual excess and masturbation were regarded as the commonest cause of seizures and castration was reported as a therapeutic procedure for men with epilepsy. These misconceptions led by chance to the first real therapeutic breakthrough. Bromide was known to cause temporary impotence in men and for this reason it was first tried as a treatment for epilepsy in the 1850's. It turned out to be the first effective anticonvulsant, although its importance was only gradually recognised (OHE, 1971). Today, there are a range of different therapies available for the treatment of different epileptic seizures and these are discussed in more detail later in the paper.

The types and causes of epilepsy

An epileptic seizure can be induced in anyone, given the right combination of circumstances. For example, it is possible to provoke a seizure by electrical stimulation, this is the basis of electro-convulsive therapy occasionally used in the treatment of mental illness. The common factor in seizures of all types is a sudden abnormal electrical discharge. Box 1 lists the different seizure types and their clinical manifestations.

The causes of epilepsy were in the past considered to be either: idiopathic (primary), that is a seizure disorder for which no evident cause can be found by history, physical examination or diagnostic testing; or symptomatic (secondarily), which are those seizures which appear to the consequence of underlying conditions such as trauma, brain tumour and vascular diseases. Today it is recognised that a variety of genetic, environmental and normal physiological factors can influence both the onset and the clinical appearance of epilepsy. There are a number of known causes and predisposing factors for epilepsy and these are different at different ages.

Inheritance

Until about 30 years ago many doctors suspected or believed that epilepsy was inherited and to some extent in the general population this is still a popular belief. This belief was so strong that in some states of America and in some Scandinavian countries people with epilepsy were forbidden to marry. Whilst it is certain that genetic factors contribute to epilepsy the part it plays should be put in perspective. For a large proportion of people with epilepsy their condition was not inherited and there is little likelihood of them passing it on to their children.

Inheritance of epilepsy can occur in a number of different ways. The first, the rarest and the most easily explained is that certain genetic diseases which are passed from parent to child cause epilepsy. In some diseases the gene may be recessive, that is the effects of the gene will only be experienced if the child receives the affected gene from both parents. The parents themselves, although carrying the gene, will be symptomless and will not show the abnormality since the other member of their pair of genes is normal. There are certain rare disorders of metabolism of the brain collectively known as lipidoses which are inherited by recessive genes. Such lipidoses causing epilepsy include Gaucher's disease and Tay-Sachs disease (Hopkins, 1988).

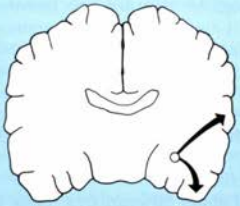
In other genetically determined diseases inheritance is through a dominant gene, that is one of the parents will not only carry the gene but will also show its effects. In such cases the parent will on average

BOX 1 Seizure classification

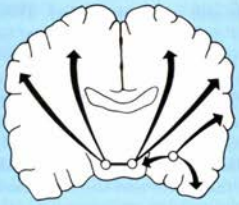
The different types of seizures seen in people with epilepsy can be classified in many different ways: by symptoms, by the cause of the epilepsy, by the area of the brain where the abnormal electrical discharge begins, or by the changes observed on an electroencephalographic (EEG) recording. It is essential that seizures are classified since treatment will vary according to seizure type, as will be discussed later in the paper. The International Classification of Seizures used today enables doctors both nationally and internationally to exchange the results of research and the experience of treatment of patients, though some non-specialist doctors find it unduly complicated to use. This classifica-

Figure 1 **Different types of epileptic seizure**

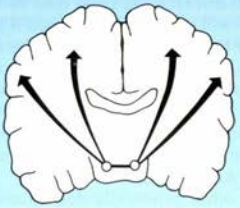
Partial seizure – the paroxysmal discharge spreads locally from a focus of abnormal cells.



Partial seizure with secondarily generalisation – the discharge spreads locally, and also to brainstem structures which spread the discharge widely through the brain.



Primary generalised seizure – the discharge spreads symmetrically throughout the brain from the beginning.



Source: Hopkins, 1988

tion takes account of the symptoms during a seizure and of the area of the brain where there is an abnormal electrical discharge. Partial seizures begin in one cerebral hemisphere; generalised seizures are bilateral from the start (see Figure 1). Seizures can be divided into the following groups:

Partial (focal, local) seizures

Partial seizures occur when a limited area of the cerebral hemisphere is affected by an abnormal electrical discharge. They may be simple partial seizures in which there is no loss of consciousness and it is thought that only a single area of the cerebral hemisphere is involved, or complex partial seizures in which the person is unaware of events. In this type it thought that areas of both cerebral hemispheres are affected. A simple partial seizure may develop into a complex partial seizure, and both may develop into a secondarily generalised seizure, in which the whole of both cerebral hemispheres are involved by seizure discharge.

A. Simple partial seizures

As the seizures start in a localized area of the brain, the patient will often experience an aura – a warning that a seizure has begun. This aura can differ widely from person to person but will generally be the same each time for the individual. The most common aura is an uncharacteristic sensation in the stomach rising to the head, but it can involve light, smell, taste or other sensations.

In some cases abnormalities occur in the area of the brain which controls the movements of the muscles or the senses of touch and pain. The symptoms will depend on whether the area affected controls the face, an arm or a leg or if it controls an influencing movement which will show itself in the form of convulsions. If the symptoms only affect the sense of touch and pain, they will consist of a feeling of numbness or tingling in the affected area.

During seizures changes can occur in a persons perceptions, for example: time may appear to pass either very slowly or quickly, there may be changes in light, sound and space perception, well known things or surroundings seem quite strange and vice versa (jamais vu or deja vu), and pronounced anxiety or exhilaration may be experienced.

B. Complex partial seizures

A complex partial seizure may evolve from a simple partial seizure or consciousness can be impaired from the beginning. In the latter case the onset of the seizure may not have been noted or the person may have no memory of it. In complex partial seizures the abnormal discharges are most often localized in the temporal lobes.

It is during these seizures that the automatisms, automatic movements, where the patient pulls at his clothes or things around him are witnessed. In addition, face pulling, chewing or lip smacking movements and aimless repeated movements often occur.

C. Secondarily generalised seizures

In both simple and complex partial seizures the electrical discharges can spread to the entire brain. This is called a secondarily generalisation. This leads to a seizure ending with generalised convulsions and unconsciousness possibly preceded by a slow spreading 'march' of the symptoms.

Generalised seizures

A. Absence seizures

Absences, sometimes known as petit mal or minor seizures, usually occur in children but in rare instances also occur in adults. The seizure takes place without warning and usually involves a short period of loss of consciousness during which the child temporarily 'blacks out'. In nearly all cases this period of unconsciousness is so short that the child's muscular tension is preserved and they therefore do not fall over. Occasionally, the muscles may relax in the hand and the child will drop what it was holding. Should a seizure last any longer the child may fall to the floor, but this is rare. During seizures it is possible to see eye blinking, the eyeballs rolling up, face pulling or twitching, however, these symptoms are not marked and frequently go unnoticed. The seizures stop as suddenly as they start and the child immediately regains normal consciousness. In contrast with other seizure types, absences happen with great frequency, in some children occurring as often as several hundred times a day. This obviously has important implications for educational development and some children may develop behaviourial problems (see page 38). Absences can be further complicated by convulsions.

B. Generalised convulsions

1 Myoclonic Seizures – these are often associated with absence seizures and are particularly frequent soon after waking. They are brief shock like contractions (jerks – some resemble normal startle reflex) of single muscles or muscle groups and may affect the limbs, trunk or face. There is usually no loss of consciousness. Myoclonic jerks are not always epileptic in origin; isolated myoclonic jerks are a normal phenomenon on relaxing or going to sleep and can arise at spinal cord level.

2 Tonic, Clonic and Tonic-Clonic (Grand Mal) Seizures – tonic-clonic seizures begin with loss of consciousness, generally with no warning that the individual can later recollect, although there may have been an increase in the number of absences or myoclonic jerks prior to the event. At this stage, known as the tonic (contraction) phase the person falls to the ground. This is because of the widespread contraction of the muscles, the body is rigid and incapable of maintaining a normal posture. The respiratory muscles also contract, forcing the air in the chest out through the larynx, so there may be an involuntary noise – a grunt or cry at the onset of the attack. During the tonic phase breathing ceases and the person may turn blue through a lack of oxygen in the blood (cyanosis). In the clonic phase that follows there is rhythmic jerking of the face and limbs and irregular breathing. This phase may last for several minutes and the jerking then slows and stops. Recovery of consciousness may then occur gradually or in a matter of minutes and confused behaviour and automatisms may be seen. After a seizure a person will often want to sleep for an hour or two and on awakening may have a headache, nausea and stiff, aching limbs. Incontinence, tongue biting and injuries sustained on falling to the ground can occur.

In tonic seizures, there is a loss of consciousness and rigid extension of the trunk and limbs so that the patient falls 'like a felled tree' either forwards or backwards, and may sustain severe injuries to the face and head. In a clonic seizure there is an abrupt onset of jerking movements of the limbs without a preceding tonic phase.

Unclassified epileptic seizures
Includes all seizures that cannot be classified because of inadequate or incomplete data and some that defy classification into the above described categories. These include some neonatal seizures, for example rhythmic eye movements, chewing, and swimming movements.

Status epilepticus
The term 'status epilepticus' is used whenever a seizure persists for a sufficient length of time (usually more than 30 minutes) or is repeated frequently enough that recovery between attacks does not occur. Status epilepticus can occur as repeated convulsions, partial or generalised, or as a constant remoteness as in the case of complex partial seizures or absences. Status epilepticus with generalised convulsions is a matter of life and death and immediate hospitalisation and treatment is necessary.

Sources: Engel, 1989, Duncan, 1985, Hopkins, 1988, Dam and Gram, 1985, Brodie, 1990.

pass on the affected gene to half of his or her children (the other children receiving the other member of the relevant gene pair). Tuberose sclerosis and neurofibromatosis are transmitted in this way. It should be pointed out that not all people with tuberose sclerosis or neurofibromatosis will experience epileptic seizures.

The second way in which inheritance plays a part in causing epilepsy is more common than the above but the risk should be kept in proportion. It has been found that approximately 40 per cent of the siblings of a child with idiopathic epilepsy (that is epilepsy for which the cause is unknown) will show the characteristic electroencephalographic (EEG) recording without any overt seizures (Hopkins, 1988). This would suggest that the biochemical abnormality which causes the abnormal EEG record is inherited, but that this abnormality is not necessarily expressed in clinically apparent seizures. A smaller proportion of parents of children with idiopathic epilepsy will also show the characteristic EEG changes. Since it is known that these EEG changes become less frequent with age it is quite possible that these parents had unrecorded discharges without overt seizures in childhood.

Another aspect of the area of inheritance and epilepsy is that of an inherited 'convulsive threshold'. As mentioned earlier anybody can be made to have a seizure given a strong enough stimulus. It has been suggested that some people have a lower threshold than others and that in some way this is inherited.

Neonatal or birth trauma

Complications during the birth used to be a frequent cause of seizures

in the newborn but these have been much reduced by better obstetric services. Anoxia, meaning a lack of sufficient oxygen, may occur at birth for a variety of reasons, a prolonged labour, the umbilical cord getting caught around the baby's neck or the baby failing to breathe immediately after birth are three examples. Lack of oxygen can result in brain damage and if severe may result in mental retardation, cerebral palsy or epilepsy. Julius Caesar is known to have suffered from epilepsy and it has been hypothesised that it may have been caused by birth trauma (the delivery being Caesarian).

Further causes of seizures in the newborn are hypoglycaemia and hypocalcaemia. Severe hypoglycaemia (low blood glucose level) resulting in seizures may be seen in newborn babies, particularly in those born prematurely or to diabetic mothers. Seizures due to a low serum calcium, hypocalcaemia, are also fairly frequently seen in the newborn. One cause is early feeding with cow's milk which is rich in phosphates and leads to increased renal excretion of calcium and then hypocalcaemia. However, in both these conditions following treatment the seizures will end. Congenital malformation of the brain and the blood vessels of the brain is a rare cause of epilepsy.

Accident and illness

In all age groups, head trauma can produce acute epileptic seizures occurring within the first few days after concussion. Jennett's (1962) analysis of 1,000 hospital admissions for head injury in Oxford showed that risk of subsequent epilepsy was about one per cent in uncomplicated injuries. The risks were found to be higher if the injury was complicated by early seizures or haematoma. These seizures do not necessarily predict the development of chronic epileptic disturbance and such acute events should generally be considered to be reactive seizures, the head injury being the precipitating factor.

Brain tumours can also cause epileptic seizures by interfering with the surrounding neurones. However, the incidence of primary brain tumour is very low, approximately 10 per 100,000 per annum and overall only 35 per cent of patients with brain tumours will experience one or more seizures (Hopkins, 1988).

The probability that a tumour is the cause of a first seizure in a patient varies according to age. Only a very small percentage of children presenting with a first seizure have a tumour. In adults over the age of 40 years, presenting with a first seizure, the probability of finding a tumour is between 11 and 20 per cent rising to between 30 and 60 per cent in middle aged and elderly adults (Feldman, 1983).

Bacterial meningitis can damage the brain at any age from the newborn to old age and thus cause epileptic seizures. But early treatment with antibiotics nearly always prevents damage to the brain cortex. However, if treatment is delayed or the organism is resistant to the

type of antibiotic chosen it is possible that the affected cortical cells will act as a focus for seizures in subsequent years. Meningitis due to tuberculosis is particularly likely to result in later epilepsy (Hopkins, 1988).

Haemorrhages in the brain and cerebrovascular disease can both cause the destruction of brain tissue and lead to the formation of a scar. This will aggravate the surrounding nerve cells. Approximately 10 per cent (Dam and Gram, 1985) of patients who have suffered a stroke will subsequently develop epilepsy, seizures will usually begin within a year of a stroke. Pre-senile dementia, Alzheimers' disease, in which the cerebral neurones gradually become fewer in number has also been found to be associated with seizures. Multiple sclerosis can also be complicated by both focal and generalised seizures.

Alcohol and drugs

Whilst it is commonly known that alcohol may precipitate seizures in those people who have previously had seizures it is less well known that there is an association between chronic alcohol abuse and the occurrence of seizures. Only in recent decades has it been demonstrated that most of these seizures are the result of withdrawal from alcohol (Mattson, 1983). Isbell (1967) was able to show that the complication associated with alcoholism including tremors and seizures were observed at the time of ceasing or decreasing alcohol consumption. Similarly, there is evidence to suggest that with barbiturates after a continued period of use for several weeks or months followed by rapid withdrawal seizures may be precipitated.

Antidepressant drugs of the tricyclic group have been found to lower the convulsive threshold and induce seizures in between 0.2 and 4 per cent of patients receiving the normal therapeutic dose (Feldman, 1983). Many of the patients in these studies however, had either a history of previously diagnosed epilepsy or predisposing risk factors, that is for example, family history, brain damage and electroconvulsive therapy.

Precipitating factors

Most people with epilepsy, whatever the cause, analyse their lives in order to detect factors which may or may not trigger seizures and there are clearly a number of factors which do appear to precipitate seizures. These include lack of sleep, menstruation and stress and worry. Some people with epilepsy are photosensitive and have a photoconvulsive response. That is seizures may be triggered by flickering lights, television pictures or the interruption of steady light filtered through trees observed from a moving car or train.

Due to the discrimination practised against people with epilepsy it is crucial that the diagnosis of epilepsy is only arrived at when the clinician is absolutely certain it is correct. In order to make an accurate diagnosis the doctor must compare the case history with the results of an examination of the patient.

As described earlier, not only does the diagnosis of epilepsy rest on repeated seizures, the causes are numerous. Many people will at some point in their life have a single generalised seizure due to physical or psychological stress. Equally, many of these people will never experience another attack and it is therefore important that they are not labelled as being 'epileptics' too hastily particularly in view of the impact that this may have on their lives both socially and economically (see section on Personal and social aspects of epilepsy, page 36).

Epidemiology

The epidemiology of epilepsy presents particular problems since 'accurate statistics are notoriously difficult to obtain, especially in a disorder such as epilepsy' (WHO, 1957). Although this statement was made over thirty years ago the situation remains very similar today, with only a limited amount of epidemiological data on epilepsy published in Great Britain.

Incidence[1]

The most comprehensive epidemiological study available in this area is by Hauser and Kurland (1975) obtained from the population of Rochester, Minnesota between 1935 and 1967. Comparisons between studies are difficult because of differences in definition, measurement methodologies, and analytical methods. This is particularly true for incidence rates which are subject to wide variations due to imprecise definitions and the inability to identify all persons with epilepsy within a specified population. This frequently results in the underestimation of incidence rates. Table 2 illustrates the variation in incidence rates when studies use different methodologies and definitions.

Although various methodologies and definitions were adopted, the incidence rates obtained from the majority of studies were between 20 and 50 per 100,000. The rather higher rates from the

1 There is some confusion over the terms incidence, cumulative incidence and prevalence: Incidence refers to the number of new cases of epilepsy within a given population in a given period. Cumulative incidence is calculated by adding incidence rates for different age groups. Prevalence is the number of cases present in a given population at a given time.

Table 2 **Studies on incidence of epilepsy**

Country author	*Case ascertainment method*	*Population studied*	*Annual incidence per 100,000*	*Definition, comment*
Denmark, Juul-Jenson & Ipsen, 1976	Medical records, 'Epilepsy Register'	Aarhus, Denmark	30	Including 'Observations for epilepsy'.
Great Britain, Crombie et al, 1960	Information from GP's	Population of 67 practices, England and Wales	63	Included single and febrile seizures.
Great Britain, Pond et al, 1960	Review of records of GP's	Population of 14 practices, SE England	70	First diagnoses of epilepsy.
Great Britain, Brewis et al, 1966	Review of medical records, central register of patients	Carlisle, England	30	Symptomatic epilepsy not included, recurrent seizures only.
Guam, Stanhope et al, 1972	Records of neurological clinic	Guam	23	More than one seizure.
Guam, Stanhope et al, 1972	Field survey	Guam, same area	46	Definition as above; age adjusted rate for US population = 35.
Iceland, Gudmundsson 1966	Review of medical records, re-examination	Iceland	26	Clinical diagnosis of epilepsy.
Japan, Sato 1964	Medical records	Niigata	17	Symptomatic seizures excluded.
Norway, Krohn 1961	Review of medical records	Northern Norway	11	Recurrent seizures only.
Norway, De Graaf 1974	Records of neurological and EEG services	Subarctic Norway	33	Patients of GP's not included, large area, difficult travelling.

Poland, Grudzinska 1974	Records of Epilepsy Clinic	Industrialized urban area, Zabrze	22	First attendance, clinical diagnosis.
Poland, Zielinski 1974a	Records of neurological and psychiatric services	Warsaw	20	First attendance, recurrent seizures, febrile seizures excluded.
Sweden, Blom et al 1978	Follow-up of children after first seizure	Swedish county children up to 16	82	Recurrent, afebrile seizures only.
USA, Kurland 1959	Review of the Mayo Clinic medical records	Rochester, Minnesota	30	More than one afebrile seizure.
USA, Hauser and Kurland 1975	Records of Mayo Clinic	Rochester, Minnesota 1955-1964 1965-1967 1935-1967	 54 46 49	More than one afebrile seizure.

Source: Zielinski, 1982

British authors (Crombie et al, 1960; Pond et al, 1960) are probably due to the inclusion of patients who have experienced only a single seizure and those with febrile convulsions. Incidence rates are also sensitive to the data base used, indeed when studies relied solely on medical records the rates were substantially lower, whilst when more complete methods were employed the rates were as much as twice as high.

A number of authors have found evidence of a higher incidence rate among the male population (Pond et al, 1960 I). One suggestion explaining this phenomenon is the greater number of head injuries in males. This is confirmed by a survey (Zielinski, 1974a) in Warsaw where it was noted that head injury prior to the onset of seizures occurred nearly twice as often in males (30 per cent as against 16 per cent in females). However, the Rochester study found the differences between the sexes not to be statistically significant.

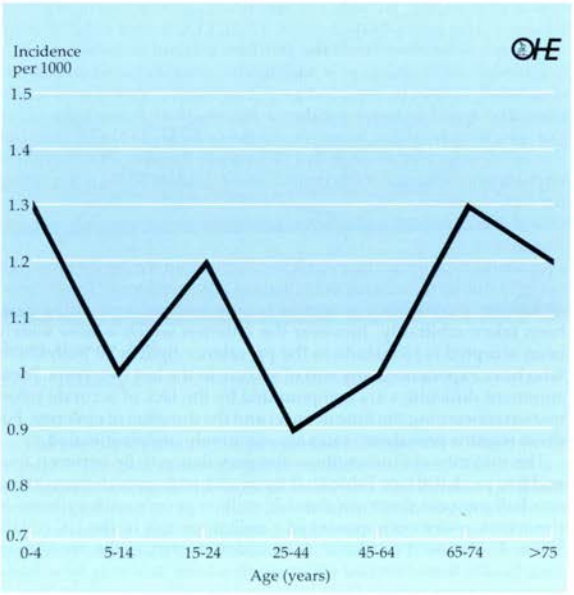
A general consensus exists among the studies concerning the age-specific incidence rates (see Table 3). The rates are high in the first years of life, fall somewhat in adolescence, and thereafter are at their lowest. However, there are notable differences in the figures for the

Table 3 **Proportion of age-specific incidence rates for epilepsy**

Age	*Rochester, USA 1945-1954*	*Carlisle, UK 1955-1961*	*Iceland 1959-1964*	*Rochester, USA 1935-1964*	*Warsaw, Poland 1970-1972*
All ages = 1.00	30	28	33	45	20
0-9	3.40	2.03	1.87	1.88	2.28
10-19	0.70	1.71	1.78	0.80	1.18
20-29	0.13	0.78	0.69	0.51	0.64
30-39	0.70	0.57	0.54	0.60	0.74
40-49	0.33	0.78	0.42	0.62	0.77
50-59	0.60	0.75	0.33	0.82	0.69
60 and above	0.70	0.42	0.18	1.33	0.93

Source: Zielinski, 1982

Figure 2 **Incidence rate per 1,000 1981/82 survey**



Source: Adapted from OPCS, 1986

age group 60 and over, but most commentators agree that the incidence rate rises in elderly patients. The diverse figures which were obtained are probably due to the differing ascertainment methods. More recent data from England and Wales (see Figure 2) illustrates the trend described, except a peak also occurs at age-group 15-24; this may be explained because the data were drawn from a small sample and could be unrepresentative.

An interesting and important statistic is the cumulative incidence which is calculated by adding the incidence rates for different age groups. Thus it gives the 'maximum' risk of developing epilepsy during an individuals lifetime (see Figure 3). The cumulative incidence indicates that by the age of 80 the risk of currently having or having had epilepsy is about 3.2 per cent.

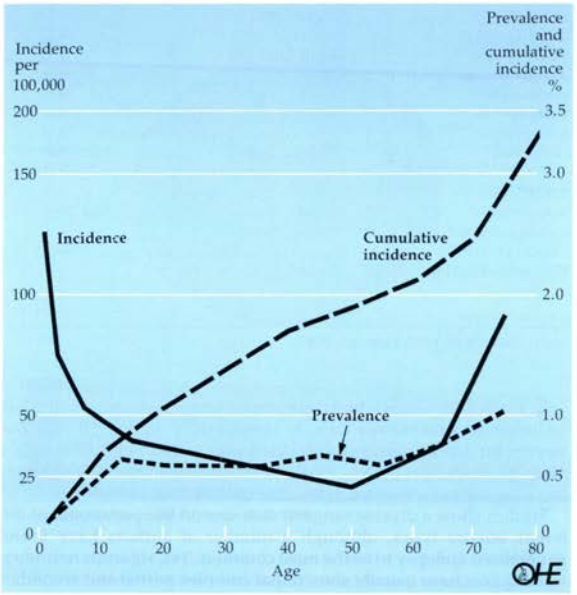
The concept of epilepsy in less developed countries (LDC's) is different from that in the developed world as priorities in health care in LDC's centre on basic life problems such as overpopulation, starvation and infection, leaving few resources for less essential health issues such as epilepsy (Robinson, 1982). LDC's tend only to recognise classical seizures, thus the statistics will not include any other manifestations of epilepsy, in addition the rates are based on hospital data which cannot be regarded as representative of the total population. The result is lower incidence figures than those reported in Europe (Walker, 1972), however, evidence by Dada (1970) indicates the incidence to be as high in LDC's as in Europe. Therefore it is important to be aware of the limitations of the data when interpreting the LDC statistics.

Prevalence

Prevalence figures can be even less reliable than incidence rates particularly due to the various definitions of active epilepsy. The decision no longer to consider a person as having epilepsy has, in the past, been taken arbitrarily, however the criterion which is now widely been accepted is to include in the prevalence figures all individuals who have experienced any sort of seizure in the last two years. Measurement difficulties are compounded by the lack of accurate information concerning the time of onset and the duration of epilepsy. For these reasons prevalence rates are commonly underestimated.

The majority of studies show the prevalence to lie between four and five per 1,000 (see Table 4). If the world-wide prevalence is about one half per cent there are about 25 million people with epilepsy in the world, and over a quarter of a million people in the UK (OHE, 1989). If lifetime prevalence is considered, that is the number of people who have ever had epilepsy, about one in 30 may have had a seizure at some time in their lives, or 2.85 million people in the UK (Shorvon, 1990).

Figure 3 **Age-specific rate, cumulative incidence rate and prevalence rate of epilepsy. Data from Rochester, Minnesota, 1935-1974.**



Source: Hopkins, 1988

The prevalence among the population of Rochester is illustrated in Figure 3. The prevalence of epilepsy at different ages is a reflection of the balance between the ages of onset and the duration of illness before remission (or in a few cases death). The divergence between the prevalence and cumulative incidence after the age of ten is primarily explained by a remission in seizures. A more or less constant prevalence is shown through the middle years of life, about 0.68 per cent, this figure is reduced in children while increasing with advancing age (Juul-Jensen et al, 1983).

Table 4 **Prevalence of epilepsy in various countries**

Survey	*Country*	*Prevalence/ 1000*
RCGP (1960)	England and Wales	4.19
RCGP (1960)	Scotland	4.03
RCGP (1960)	South Australia	1.77
Juul-Jenson and Ipsen (1973)	Denmark	6.9
Hadock (1973)	Ghana	4.0
Orley (1970)	Uganda	4.0
Levey (1964)	South Rhodesia (now Zimbabwe)	7.4
Giel (1968)	Ethiopia – urban	5.0
	– rural	6.0
Sato (1953)	Formosa (now Taiwan)	1.3
Sato (1964)	Niigata, Japan	1.5
Zielinski (1974a)	Warsaw, Poland	9.2
Gomaz et al (1978)	Bogota, Colombia	19.5
Hauser and Kurland (1975)	Rochester, Minnesota	5.3 (1950) 6.2 (1960) 5.7 (1965)

Source: Robinson, 1982; Zielinski, 1982

In studies in which febrile seizures or single isolated seizures are included the prevalence rate is considerably higher. Prevalence figures for febrile seizures vary from one to 150 per 1,000, with a mean of 53.4/1,000 (Tsuboi, 1984), illustrating again that methodology has a significant influence in the determination of statistics.

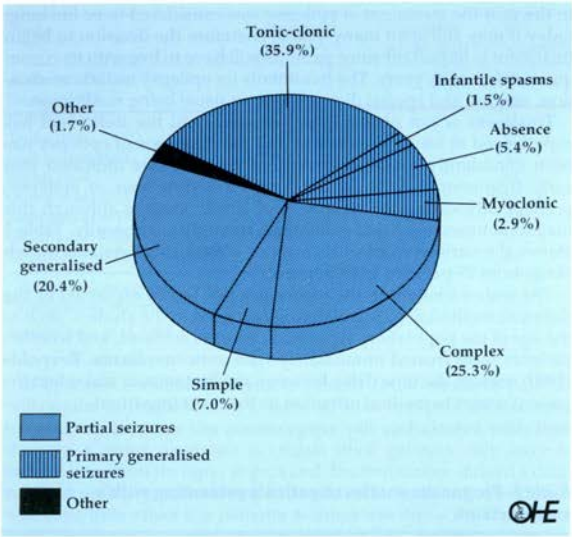
Studies show a diverse range of statistics on the prevalence of different seizure types, although a number of authors have found generalised epilepsy to be the most common. Yet, rigorous neurological inquires have usually shown that complex partial and secondarily generalised seizures account for over 50 per cent of prevalent cases, see Figure 4 (Hauser et al, 1975; Juul-Jensen et al, 1983).

Mortality statistics

Mortality rates are not a good indicator of the frequency of epilepsy since only a small number of those with the condition actually die as a result of their seizures, and even if the seizures have been a factor in their demise mortality statistics may not mention epilepsy. Indeed, in the Rochester study only 11 per cent of all deaths among those with epilepsy had convulsions or epilepsy mentioned on their death certificate. In fact the commonest cause of death recorded on death certificates of those with epilepsy is some form of respiratory obstruction, smothering or swallowing vomit.

Due to the problems associated with mortality data and epilepsy

Figure 4 **Frequency of seizure types**



Source: Adapted from Juul-Jensen et al, 1983

Hauser and Kurland (1975) preferred to use the survival rate of those with epilepsy compared to controls matched by age and sex. Their results indicated that five years after diagnosis the ratio of survivors with epilepsy to the control was 95 per cent, at ten years 90 per cent and at 15 years 89 per cent. This shows that excess mortality is most likely to be in the early years after diagnosis. A significant result is that survival was much less in those whose epilepsy was assigned a cause – such as neurological dysfunction.

Interpreting epidemiological data on epilepsy can be hazardous particularly since studies have used various methodologies and definitions. Unless all studies use a constant base the data cannot be compared.

Treatment and therapy

In the past the treatment of epilepsy was considered to be life long; today it may still span many years. Therefore the decision to begin treatment is important since patients will have to live with its consequences for many years. The treatments for epilepsy include medication, surgery and special diets – the most usual being medication.

Treatment is not normally undertaken until the individual has experienced at least two seizures and the diagnosis of epilepsy has been confidently established. Recent research has indicated that early treatment may affect the long term prognosis of epilepsy; perhaps advocating the treatment of single seizures although this may sometimes mean that patients are treated unnecessarily. Table 5 shows the various rates of recurrence after a single seizure which range from 27 per cent to 78 per cent.

The wide variations in the recurrence rate can be explained by the different methodologies and definitions used in the studies, such as the age of the population, types and causes of seizures, and whether patients were treated immediately with anticonvulsants. Reynolds (1990) regards the time delay between the first seizure and when the patient comes to medical attention as the most important.

Table 5 **Prognostic studies of patients presenting with a single seizure**

Author	*Number of patients*	*Length of follow up (months)*	*Rate of recurrence (%)*
Thomas (1959)	42	–	27
Johnson et al (1972)	77	36	64
Saunders and Marshall (1975)	33	26	64
Cleland et al (1981)	70	57	39
Hauser et al (1975)	244	12	16
(1975)		24	21
(1982)		36	27
Todt et al (1985)	179	12	59
Camfield et al (1985)	168	–	52
Elwes et al (1985)	139	36	71
Annegers et al (1986)	380	60	56
Hopkins et al (1988)	100	36	52
Hart et al (1990)	564	12	67
		36	78
Goodridge and Shorvon (1983)	122	–	81

Source: Reynolds, 1990; Hart et al, 1990; Hauser et al, 1975; Goodridge et al, 1983.II

Table 6 **Studies of the prognosis for seizure control in patients with epilepsy**

Study	*Number of patients*	*Seizure free period (year)*	*% seizure free*
Alstrom 1950	897	3	22
Strobos 1959	228	2	38
Kiorboe 1960	130	4	32
Trolle 1960	799	2	37
Juul-Jensen 1963	969	2	32
Rodin 1968	90	2	32
Currie et al 1971*	666	1	40
Annegers et al 1979**	457	5	70
Okuma and Kumashiro 1981	1868	3	58
Goodridge and Shorvon 1983**	122	4	69

* Temporal lobe epilepsy only
**Community surveys

Source: Elwes et al, 1984

If epilepsy is established and treatment recommended, patients will require full explanations in terms of what to expect, for example, interactions between the contraceptive pill and alcohol with their therapy. Correct diagnosis is critical since patients may have to endure long term therapy, stigma and discrimination, indeed a diagnosis of epilepsy and subsequent treatment with anticonvulsants will have little effect if a patients seizures are due a different diagnosis. Accurate diagnosis could be aided by EEG findings as a recent Dutch study (Donselaar et al, 1991) found that epileptic discharges were associated with a greater risk of seizure recurrence after a single seizure.

The possible importance of early treatment is highlighted in recent studies which suggest that the long term prognosis of epilepsy is influenced by the number of seizures which have occurred before treatment. Thus early treatment may prevent the development of chronic epilepsy. If this were the case it would have a profound effect on the management recommended for single seizures.

According to contemporary studies (see Table 6) the prognosis for the majority of people with epilepsy is good, this is contrary to the gloomy remarks of Rodin (1968) and Gowers (1881). Rodin concluded that '80 per cent of patients with epilepsy are likely to have a chronic seizure disorder. This does not rule out short term remissions or changes in seizure patterns, it merely reemphasises that epilepsy should be regarded as a chronic condition with remissions and exacerbations.' Rodin's pessimistic prognosis was influenced by selection bias which is inherent in cross sectional studies based on

chronic hospital populations. The methodology of other studies which reached similar conclusions is also questionable with different definitions of remission, durations of follow up and classifications of seizures. In addition extensive monitoring of serum levels was not employed to assess either optimal use of medication or poor compliance.

Two retrospective studies (Goodridge et al, 1983.II; Annegers et al, 1979) have verified that the prognosis, for most patients, is good. Annegers et al identified all newly diagnosed patients with epilepsy in Rochester (excluding febrile seizures, single seizures, convulsions associated with acute illness), of those followed up for twenty years as many as 65 per cent had been fully controlled without seizures for at least five years. Similar results were obtained in a study by Goodridge and Shorvon.

Further support comes from a number of prospective studies of newly diagnosed, previously untreated patients presenting themselves to hospitals (Reynolds et al, 1983; Turnbull et al, 1985; Elwes et al, 1984). About 75 per cent of such patients were found to achieve a two year remission from epilepsy when receiving treatment with a single anticonvulsant.

Several factors may be associated with a poor prognosis, the most frequently cited are; partial seizures, high frequency of tonic-clonic seizures before treatment, neurological, social or psychiatric handicap and a family history of epilepsy.

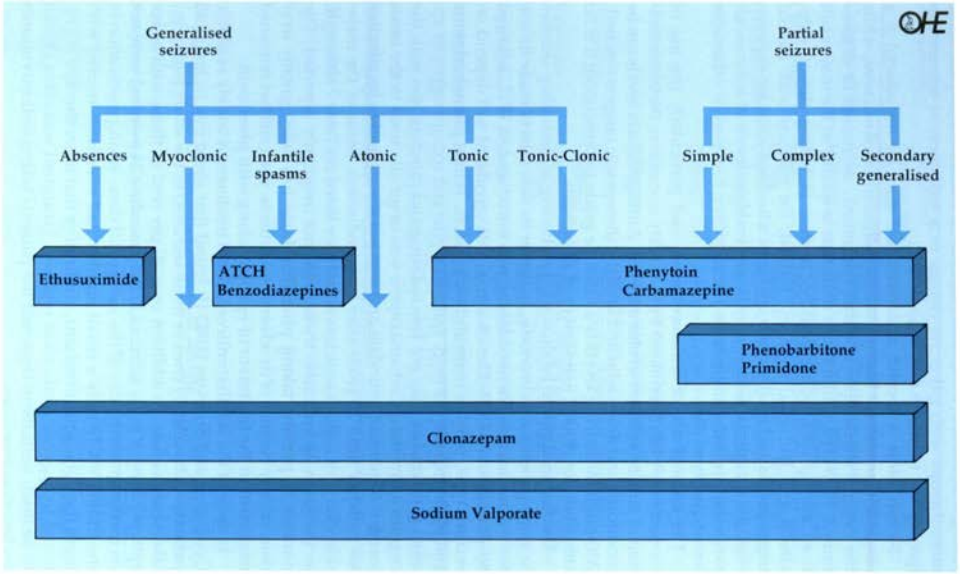
The most usual form of treatment is anticonvulsant therapy. In 1988 in Great Britain five million prescriptions for anticonvulsants were dispensed, that is one per cent of all prescriptions dispensed[2] (OHE, 1989). However, of an estimated 280,000 people with epilepsy in Great Britain, 70,000 are not helped by anticonvulsants (Dover, 1989).

Until the 1970's polytherapy (more than one anticonvulsant) was the most widely used and accepted practice, now monotherapy is generally recognised as being superior. The basis of monotherapy is accurate diagnosis and assessment of a patients seizure type(s) followed by the selection of a single appropriate anticonvulsant.

The anticonvulsant prescribed will depend on the particular type of epilepsy; anticonvulsants which are effective in controlling one person's seizures may be totally unsuitable in another case (see Figure 5). Shorvon and Reynolds (1979) actually found that monotherapy reduced seizure frequency by at least 50 per cent in 16 of the 29 patients (55 per cent) with previously 'intractable' seizures unresponsive to polytherapy. These findings were corroborated by

2 Total number of prescriptions dispensed for all therapeutic groups was 413 million in 1988.

Figure 5 **Seizure types and effective medicines**



Source: Adapted from Pellock, 1988

Albright and Bruni (1985) who successfully transferred 39 patients to monotherapy with either no change or a reduction in seizure frequency in a 16 month follow up period.

Nevertheless a second anticonvulsant may be introduced if a single anticonvulsant has not successfully controlled seizures, while the original medication is either continued, decreased or discontinued. However, a significant reduction in quality of life may result from attempts to reduce seizures to zero including clinical side-effects and biological changes. Indeed, for patients with mental impairments and multiple seizure types who need constant supervision, the occasional seizure may be preferable to levels of anticonvulsants that further compromise the cognitive function or otherwise lower quality of life.

The rational use of anticonvulsants and particularly the use of monotherapy has been aided by the ability to measure the concentration of anticonvulsants in blood serum. Anticonvulsant levels can be monitored and doses adjusted until the therapeutic range is reached. Although measuring levels is a valuable guide to prescribing it is no substitute for clinical assessment of all patients.

The first medicine to be shown to be effective in controlling seizures in some patients was bromide, however its side effects made its use somewhat limited. Phenobarbitone was introduced in 1912, followed by phenytoin in 1939. The choice of anticonvulsant will depend on seizure type, plus its costs and side effects. Whilst phenobarbitone and phenytoin are still used, carbamazepine and sodium valporate are often preferred as they are less sedating and interfere less with the cognitive function. Carbamazepine was originally used in the treatment of trigeminal neuralgia and was not discovered to be effective for epilepsy until 20 years after its introduction. Some of the major side effects of various anticonvulsants are listed on Table 7 although, as the footnote indicates, these were not frequently reported. The number of patients taking phenobarbitone has fallen greatly over time, mainly because of its adverse side effects, yet it still has remained important in the treatment of epilepsy along with phenytoin.

In spite of the fact that anticonvulsant treatment is widely available Goodridge and Shorvon (1983 II) found that about one in ten of all patients with epilepsy (two or more seizures) in the study had never taken medication. In addition over 40 per cent of those with active epilepsy (one or more seizures in the preceding 24 months) were not on treatment on the day of the survey. Of those taking medication in the former study only a small number had ever been treated with more than one anticonvulsant, confirming that monotherapy is preferred to multiple medication regimes. Treatment was often shown to be brief, with more than half the patients taking anticonvulsants for less than five years.

Table 7 **Comparison of oral anticonvulsants**

Anticonvulsant	*Annual cost* (common daily dose used)*	*Important adverse effects*[3]
Carbamazepine	£65 (600mg)	Drowsiness, dizziness, visual disturbances, rashes, leucopenia, hair loss, acute renal failure, diplopia
Clonazepam	£101 (6mg)	Drowsiness, unsteadiness, behaviour disorders, bronchial hypersecretion
Ethosuximide	£94 (1000mg)	Nausea, vomiting, tiredness, dizziness, mood disturbances, leucopenia, rashes
Phenytoin	£28 (300mg)	Drowsiness, ataxia, gum hypertrophy, hirsutism, acne, facial coarsening, gastric distress
Phenobarbitone	£13 (180mg)	Drowsiness, lethargy, mental depression, ataxia, behaviour disturbances
Primidone	£27 (1000mg)	Drowsiness, behaviour disorders
Valporate	£96 (800mg)	Nausea, weight gain, tremor, alopecia, bleeding tendency, hepatotoxicity, hair loss
Vigabatrin	£168 (3000mg)	Aggression, drowsiness, fatigue, dizziness, irritability, memory and visual disturbances

*Approximate cost for adults based on NHS price (cost price ex. VAT) for December 1990 except for phenobarbitone which is based on proprietary preparation in 1983

Source: Mimms, December 1990; Davidson, 1983

3 Between 1964-1984 the actual number of adverse reactions reported was extremely low:

Adverse reaction	*No. of reports*	*Anticonvulsant*
Ataxia	14	phenytoin
	25	carbamazepine
Confusion	12	carbamazepine
Convulsions	2	phenytoin
Diplopia	12	carbamazepine
Dizziness	16	carbamazepine
Tremor	20	sodium valporate
Vertigo	10	carbamazepine

Recently, there has been a large amount of activity in the development of new anticonvulsants, including vigabatrin and lamotrigine. In December 1989 vigabatrin came on the market, at present it is only used for those seizures not controlled by other medication since clinical experience is limited and the possibility of rare side effects exists.

The difficulty in developing anticonvulsants is clinical testing. Since there is no justification for withholding treatment, it would be unethical to hold placebo controlled trials. Testing, currently, involves patients with resistant seizures continuing to receive the treatment with their existing therapy; this conflicts with the evidence of monotherapy and means that the medication is evaluated under relatively unfavourable circumstances. Nevertheless there are a number of medicaments presently undergoing clinical trials.

The annual costs for a particular course of anticonvulsants are indicated in Table 7. The cost is obviously greater for those individuals on more than one anticonvulsant. It should be noted that a cheap treatment is not necessarily cost effective, particularly if poor compliance leads to a patient being admitted to hospital. Monotherapy offers improved compliance and cost effectiveness, in terms of patient functioning and productivity, with increased alertness and mental function probably improving effectiveness and performance at work or school. The total cost of anticonvulsants to the health service was £21.4 million in 1988 (see Table 13).

For some patients the duration of epilepsy is short, for others it is a chronic problem requiring long term treatment. This variable prognosis leads to difficulties in deciding when to begin and end treatment. There is no real consensus on the length of remission before discontinuation of therapy is attempted, although more recent studies suggest at least two years.

The decision to discontinue therapy is an important one which should be discussed fully with the patient, the arguments for withdrawal include dose related adverse reactions, chronic toxicity and effects on behaviour and cognitive function. Conversely patients will be concerned about the recurrence of seizures which may have important effects on employment, self esteem and driving. Nevertheless, patients may experience a relapse even after many years on medication, possibly because of a failure in compliance.

When deciding whether to withdraw anticonvulsants from a patient it is important to be aware of patients who have a lower risk of relapse. At present few studies have undertaken to determine the success of withdrawing of anticonvulsants. Oller-Daurella et al (1976) found that withdrawing very slowly resulted in a much lower risk of relapse, although a recent multicentre study (Medical Research Council Antiepileptic Drug Withdrawal Study Group, unpublished) shows that a slow withdrawal over six months had a

similar recurrence rate as more rapid withdrawals (two-three months).

Most studies of withdrawal indicate a low risk of relapse in children with epilepsy. Withdrawal is particularly important for children because of the effects that some anticonvulsants have on the cognitive function which can cause significant learning and development difficulties. In a study of 88 children with epilepsy by Shinnar et al (1985) 75 per cent remained seizure free for up to five years after medication was discontinued. Thurston et al (1982) found a similar success rate with 72 per cent of children incurring no relapses.

In general, studies in adults have shown a somewhat higher rate of relapse. In a good study by Juul-Jensen (1964) it was found that five years after anticonvulsant withdrawal 40 per cent of patients had relapsed, of these half had a recurrence of seizures during the withdrawal period itself. Conversely, Callaghan et al (1988) found the relapse rate to be no different among adults (35 per cent) than among children (31 per cent) in a study with a mean follow-up of 26 months.

Attempts have been made to establish which factors influence the risk of recurrence. Those suggested include EEG findings, seizure type, duration of epilepsy, age of onset of seizures. Callaghan et al proposes that the withdrawal from certain anticonvulsants heightens the risk of relapsing. However, 'clearer evidence to identify those least at risk is required before widespread withdrawal of long-term medication can be recommended' (Patten, 1989).

A large multicentre study (Medical Research Council Antiepileptic Drug Withdrawal Study Group, unpublished) has attempted to improve understanding about withdrawal of anticonvulsants. In fact it is the only study to include a comparative group of patients randomised to continue therapy. The study investigated a risk which has largely been ignored by other studies, that is the risk of seizure recurrence in patients on prescribed therapy, which was found to be ten per cent.

The lack of information on the natural course of untreated epilepsy makes it impossible to evaluate the impact of anticonvulsants. Individuals whose seizures are not controlled by mainstream medication may require new anticonvulsants or alternative treatments such as surgery or special diets. One such diet is the ketogenic diet[4].

Surgery offers a useful alternative for patients with resistant seizures, particularly since many individuals are rendered seizure free after surgery. Results from the Mayo clinic (Meyer et al, 1986) show

4 The ketogenic diet requires one gram of protein per kilogram of body weight per day, plus fat to make up the additional calories with only a minimal amount of carbohydrate. The high fat and minimal carbohydrate makes the diet extremely unpalatable.

that 78 per cent of the sample experienced no seizures following temporal lobotomy. In addition Van Buren (1987) found a low morbidity and no mortality in a series of 560 craniotomies for surgery of epilepsy. In spite of its benefits, the current uptake of surgical treatment in most western countries, according to Polkey (1989), is probably too low. In the United Kingdom only about 100 operations for epilepsy are carried out per annum even though it is claimed that 2000 people per year could benefit from this treatment (Anonymous, 1990).

However, surgery is not cheap. The cost of epilepsy surgery can be up to £40,000 per patient (Anonymous, 1990). Yet, the patient may gain substantial benefits from a reduction in seizures and in terms of employability and the ability to live independently. Indeed, Augustine et al (1984) reports that of 32 surgical patients in 1984, those employed on a full time basis increased from 14 to 23 while the number who were underemployed decreased from eight to zero. Thus indirect costs could fall due to a reduction in the number underemployed.

The central message for individuals whose seizures cannot be controlled by anticonvulsants is that there are alternatives, and the prognosis for the majority of people with epilepsy is good. Nevertheless, the overall management of epilepsy may be improved by increasing the number of epilepsy centres and specialist clinics since, as Oxley et al (1987) explains, 'a parallel can be drawn with diabetes clinics which have been set up throughout the country and have greatly improved the care of diabetes generally'. The clinics could be organised along the lines of clinics for diabetes (Goodridge et al, 1983 I), providing facilities for initial diagnosis and assessment, plus planning the long term management of patients with epilepsy. The long term care could include regular consultations for patients with epilepsy to assess seizure control, any side effects and possible withdrawal so that treatment can be altered accordingly. In addition serum levels could monitored to judge the level of compliance. In other countries epilepsy clinics are now widespread, while in the United Kingdom few have been established even though the Reid Report (1969) recommended their expansion in 1969.

Problem of compliance

Compliance is simply the extent an individual's behaviour coincides with medical or health advice (Haynes et al, 1979). Therefore non-compliance ranges from those individuals who fail to have their prescriptions dispensed to others who stop their course of treatment before completion.

The problem of non-compliance is a serious one, particularly among individuals with epilepsy – taking medication irregularly is

one of the commonest reasons why seizures fail to be controlled. A study by Cramer (1989) found that missed doses and lack of sleep accounted for about 79 per cent of seizures.

Missed doses may be a particular problem in children whose treatment has to be administered at school and, unless supervised, can easily be forgotten. Thus medication which only needs to be taken once a day may be advantageous. However, Haynes et al (1979) cites three studies where there was no significant relationship between the number of daily doses and the extent to which patients conformed to their treatment. Therefore once-a-day preparations may be more for convenience rather than compliance.

With some preparations, such as phenytoin, phenobarbitone and sodium valporate a forgotten dosage can be taken the following day. However this is not possible in treatments such as carbamazepine which may cause adverse side effects.

Non-compliance is widespread among people with epilepsy. The number of non-compliars ranges from 42 per cent (Gibberd et al, 1970) to 60 per cent (Dawson et al, 1971). A survey by McCluggage et al (1984) of 75,000 general practitioner patients in Northern Ireland found that 23 per cent had deliberately stopped taking their medicine. Other studies (Evans et al, 1983; O'Hanrahaim, 1981) indicate that, on average, about 50 per cent of patients are compliant.

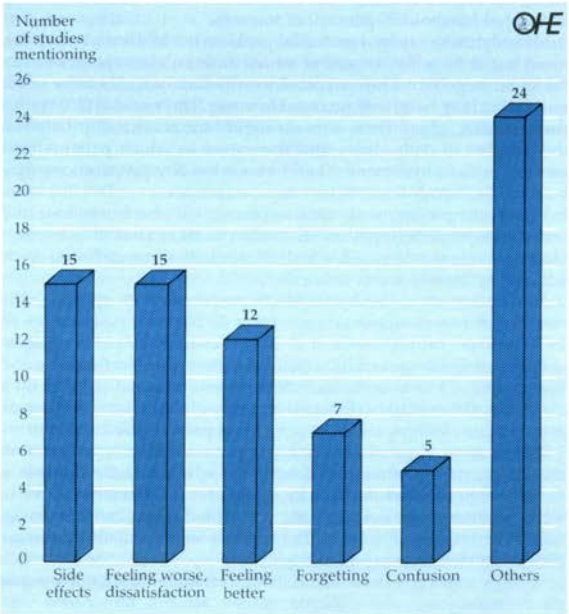
The reasons suggested for non-compliance are varied. One common proposition is that non-compliance is linked to the number of medicines prescribed. Most studies have found that when three or more medicines are concurrently prescribed, compliance falls significantly (Haynes et al, 1979). Therefore, it seems probable that compliance will be lower in patients whose epilepsy cannot be controlled by a single anticonvulsant, or, are taking other medication, for example the elderly.

Several studies have attempted to characterise non-compliant patients with epilepsy. One study by Eisler and Mattson (1975) on an unspecified number of outpatients reached conclusions similar to Bryant and Ereshefsky (1981). The former identified non-compliant patients as possessing:

1) inadequate insight in to the need of convulsants;
2) a poor understanding of directions for their use;
3) an inappropriate fear of addiction to their medication.

One surprise is the significant difference between the reasons the studies suggested for non-compliance of anticonvulsants and those given by the patients for all treatments. The most common reason for non-compliance given by all patients receiving treatment was dissatisfaction with prescribed medication either due to the side effects or because they felt worse after taking one medicine (see Figure 6). However, compliance is essential in the successful long term treat-

Figure 6 **Patients reasons for non-compliance for all treatments**



Source: Adapted from Hayes et al, 1977

ment of epilepsy, therefore, perhaps if doctors discussed the relative benefits of therapy and possible disadvantages of not taking medication, non-compliance might be prevented or at least reduced.

Compliance may therefore increase if better communication existed between doctor and patient, or pharmacist and patient, to explain the treatment and dispel any unnecessary fears.

Original pack dispensing, labelling and patient package inserts could all contribute and compliance aids such as dosage boxes could be used to help those patients who are prescribed more than one medicine. These measures are designed to increase understanding, although the effect on compliance is debatable. Pryse-Phillips et al (1982) found that patients remembered written information better

than verbal information, however this additional knowledge had little or no effect on compliance.

It is difficult to determine which individuals with epilepsy are non-compliant. Studies (Gallagher et al, 1971; Travers et al, 1972) have indicated that non-compliars are predominately women; conversely Bryant and Ereshefsky (1981) found both non-compliars and compliars to be similar in both age and sex. However, the latter observed that education correlated with compliance, that is, the more education an individual receives, the greater the compliance. By contrast Bryan (1976) believed it could not necessarily be assumed that educated patients are more compliant.

At present anticonvulsant serum levels (ACLs)[5] are the most reliable method of discovering non-compliant individuals as they indicate how well patients adhere to prescribed medicines. Wannamaker et al (1980) found that a reduction in the interval between visits to clinics by patients on stable anticonvulsant treatment resulted in an increase in the ACLs. From 30 patients on stable regimes, ACLs increased in 33 per cent of cases by decreasing intervals between clinic visits from a mean of 3.4 months to 1.1 months. When this solution was applied to patients who had ACLs below the 'therapeutic range', 73 per cent of patients showed improvements in ACLs.

Other studies (Gibberd et al, 1970; Lund et al, 1964) have confirmed that an increased frequency of clinic visits are associated with higher ACLs. Lund et al (1974) inferred that the maintenance of good ACLs could be achieved by seeing patients at two month intervals. Hence more frequent supervision offers an effective measure against non-compliance.

Improvement in compliance can be achieved by a variety of measures, for example, better advice, more frequent monitoring or original pack dispensing and patient package inserts. Although most of these measures increase cost and use scarce health resources 'effective prescribing is recognised as a cost-effective aspect of medicine' (OHE Briefing, 1983). Non-compliance is a considerable waste of resources. The probable result of non-compliance in individuals with epilepsy is more frequent seizures which can reduce confidence, create problems at work – particularly if the condition had not already been disclosed – disqualification from driving (for those who previously held a valid licence) and cause numerous other complica-

5 ACL refers to the amount of anticonvulsants (by weight) dissolved in a unit volume of serum. The therapeutic range are those ACL's that effectively control seizures, by regularly measuring serum drug concentrations an optimal drug therapy regime can be established for an individual patient. Different anticonvulsants have different therapeutic ranges. A frequent cause of lower than expected anticonvulsant serum concentration is non-compliance.

tions. Patients should be vigorously encouraged to adhere to their therapeutic regime since compliance is not only an efficient use of health care resources, but will also be of benefit to themselves by improving their quality of life.

Personal and social aspects of epilepsy

Personal perceptions

Recent work has focused on the fact that perceptions of stigma by people with epilepsy may be more disabling than the seizures. Moreover, Ryan et al (1980) has demonstrated that these perceptions are more strongly influenced by self perceptions than by the objective facts of epilepsy. An individual's perception of epilepsy may be passed down from parent to child, so that 'the more parents convey a definition of epilepsy as something 'bad', and the less willing they are to talk about it with their children, the more likely the child is to see it as something to be concealed' (Schneider et al, 1980), which could cause a child to grow up feeling ashamed of their condition. This shame and the fear of encountering enacted stigma[6] is referred to as 'felt' stigma.

Felt stigma and the fear of meeting with rejection (both in social and employment terms) frequently lead people to conceal their epilepsy. In a British study by Scambler and Hopkins (1990) only 33 per cent of marriages which occurred after the onset of epilepsy were preceded by a full disclosure including the word epilepsy. A further 36 per cent partly disclosed their condition using words like 'dizzy spells', 'attacks', 'seizures' and so on, therefore some 31 per cent made no disclosure. The workplace was no different with only five per cent of those interviewed, who were in full time work, voluntarily disclosing their condition to their employer before starting work. All of these had seizures daily, thus had little option but to disclose. Although these individuals suffered felt stigma and concealed their condition, only a few could recall incidences of enacted stigma (see Employment & social class).

Similar results were found in a study of 392 people with epilepsy by Collings (1990) in Great Britain. Collings considered the association between social, psychological and physical well-being and the range of epilepsy-related and socio-demographic variables. A small group of factors were identified as being significant. The factor most

6 Enacted stigma refers to the actual discrimination of individuals with epilepsy solely due to their social unacceptibility or inferiority. It excludes legitimate discrimination, for example, those who are prohibited from driving due to their epilepsy.

strongly related to well-being was an individual's perception of themselves and of their epilepsy, other important influences were seizure frequency, time since diagnosis, a diagnosis of 'absence' seizures and being employed full time.

This study confirms previous research which has revealed that personal perceptions influence the well-being of an individual with epilepsy. Indeed, the perception of felt stigma can cause more disruption to the lives of people with seizures disorders than enacted stigma.

Dependence v independence

As recently as one hundred years ago epilepsy was regarded as a form of insanity for which the only help available was confinement in an asylum. In the community, people had little opportunities to work and had to resort to begging in order to survive. Today, the majority of people with epilepsy are able to live complete lives with only the occasional seizure and fortunately for those who are unable to live independently the situation has greatly improved.

It should be stressed that a considerable number of individuals with epilepsy can cope successfully in an ordinary environment, particularly if their epilepsy is not associated with any other disorders or disabilities. Clearly, such individuals should be helped and encouraged to take an active part in society. However, for some people with epilepsy, usually those with a disability such as mental handicap, living independently is not always possible or appropriate and small units for disabled people in their home area may be more suitable. Alternatively they could attend epilepsy centres. According to Laidlaw and Laidlaw (1982), people should only attend epilepsy centres if:

1) Patients who have disabilities that require hospital attention, but whose seizures can not be coped with easily in hospital.
2) Small groups who continue to have frequent or severe seizures despite the best medical treatment.
3) Able and intelligent individuals who should be able to earn an ordinary wage and live in an open community with the minimum support in order to live ordinary lives.

This type of environment may stimulate people and enable them to enjoy fulfilling and rewarding lives. For some individuals their stay may only be a transitory step towards independence, for others it offers a sheltered home for life.

The home is an ideal environment for encouraging independence, however, it can instill reliance on the family. Anxiety about a child and the nature of epilepsy (often misunderstood) may make parents overprotective; for example, staying in the child's bedroom throughout the night in case of a prolonged seizure which may worsen their

condition. This special treatment may mean that the child not only becomes more dependent on their parents and less self reliant but also spoilt and demanding, while other siblings may be resentful of the attention their brother/sister is receiving, thus creating family tensions. Conversely, parents may reject their child. For a child who already suspects that they are 'different' from their peers this rejection could be extremely damaging to their emotional and social growth.

If parents adjust and cope with having a child with epilepsy as a member of the family, it is probable that the child will cope too. However, for adjustment to occur the parents may have to go through a variety of emotions such as shock, fear, denial, anger, guilt, and depression before acceptance. Once accepted the parents have to let their child grow up in a 'normal' environment, allowing them to encounter adolescence and learn by their own mistakes. Leisure activities can offer an excellent opportunity for children with epilepsy to integrate with their peers and grow in confidence. Most activities, including active sports, are acceptable although certain activities such as swimming or riding may require supervision: what is required is a degree of common sense.

Intelligence and education

The 1981 Education Act stressed that children with special needs should be educated, whenever practical, in mainstream schools. This is true for some 60,000 children with epilepsy, with only a small number attending special schools (Radley, 1987). Those children placed in special schools are not different in terms of seizure frequency, but have additional handicaps (Kangesu et al, 1984). Whilst the vast majority attend ordinary schools it has been suggested that their intelligence is lower than their peers and they underachieve.

Rutter et al (1970) found the intelligence of children with epilepsy to be well below average. However, this study included children who derived their epilepsy from brain defects and disorders. Clearly, when 28 per cent of the survey sample have brain disorders the IQ level of the entire population will be considerably reduced. When the remaining 72 per cent were considered they had the same IQ distribution as the general population.

Yet, the same study found that children with 'uncomplicated' epilepsy (no brain defects) and average IQ had reading levels, in general, 12 months behind their chronological age. Perhaps a better guide would be reading retardation which includes chronological age and intellectual ability. Nevertheless, Rutter's results still indicated that a disproportionate number of children with 'uncomplicated' epilepsy underachieved, with 18 per cent of children retarded by two years or more compared to 6.8 per cent of the general population.

Conversely, Bagely (1971) found that reading retardation occurred in behaviorally disturbed individuals irrespective of their epilepsy. There has been criticism of the techniques used by Bagely; firstly recognition of single words only was required, and, secondly underachievement in older children of above average intelligence may have been missed as an upper limit of 15 years was adopted. Nevertheless, more recent surveys are in harmony with Bagely's conclusions (Camfield et al, 1984).

Few studies in other school subjects have been completed. Nevertheless, if children with epilepsy underachieve one would expect their examination success to be limited. Indeed, less than five per cent of young adults with continuing epilepsy (one seizure in the past two years or continuing with anticonvulsant medication) obtained 'O' levels and only one per cent 'A' levels. However, after leaving school a further 5.3 per cent and 2.3 per cent obtained 'O' and 'A' levels respectively (Prior et al, 1981). These results show that children with continuing epilepsy are not underachievers rather late educational developers. These results correspond to a study by Britton et al (1986) where educational and vocational qualifications of individuals with 'uncomplicated' epilepsy were identical to matched controls.

Moreover, a survey by Harding et al (1986) at an epilepsy clinic indicates that individuals with epilepsy may be overachievers since almost 15 per cent of the sample population went to university compared to eight per cent of the population norm; although extrapolating from such a small sample (n=22) may be hazardous.

Attacks and their aftermath may cause late educational development. Childhood 'absences', which can occur several hundred times a day, cause a sudden and momentary loss of contact with the surroundings, while other types of seizure may require recovery time causing the child to miss valuable hours of school time. It may only be when the attacks have been brought under control by anticonvulsants or childhood 'absences' stop that an individual can concentrate on school work. Intellectual impairment will normally only occur if the child has frequent clusters of tonic clonic attacks, or if anticonvulsant therapy adversely affects the cognitive function.

The education of children with epilepsy in mainstream schools is important since it provides an invaluable opportunity for these individuals to form friendships and reduce prejudice. Nevertheless, special schools also remain an essential provision for those with epilepsy particularly for individuals with multiple disabilities.

Relationships, marriage and children

Friends and relationships form an integral part of all our lives; unfortunately people with epilepsy find it difficult to form such bonds. In

1949 results of a survey indicated that parents may have prevented childhood friendships as 24 per cent objected to their child associating with a person who had seizures. Although the situation had improved by 1974 when a follow up survey showed that the figure had dropped to five per cent (Seligsohn, 1988). Nevertheless, low self esteem, fear of a seizure in public and people's reactions all contribute to inhibiting people with epilepsy from integrating fully in society.

The difficulty in forming relationships continues into adulthood with a lower frequency of marriages among people with epilepsy (Pond et al, 1960 II; Lindsay et al, 1979). Marriage rates of patients at a Montreal clinic were compared in 1941 and 1971 (Dansky et al, 1980). In 1941 both male and female patients had a low marriage rate, although the situation had not altered for men in 1971, for women the marriage rates now matched the remainder of the population. The study also found that if the onset of epilepsy was before the age of 10 years fewer patients married, in males this effect also extended into the 10-19 age group.

Fertility is also likely to be reduced in individuals with epilepsy. Webber et al (1986) found the fertility of women with epilepsy to be 85 per cent of the expected female rate, while men's fertility was reduced to 80 per cent of the expected male rate.

People with epilepsy are not only less liable to marry and less fertile, Fenwick et al (1985) also found high levels of sexual dysfunction. A possible explanation for the low sex drive among men with epilepsy is low levels of testosterone caused by anticonvulsants, particularly carbamazepine, phenytoin, phenobarbitone, and primidone, but not sodium valproate (Webber et al, 1986). These results have been verified in a large study (Mattson, 1982) of adult males receiving anticonvulsants for the first time; over 15 per cent of the patients complained of decreased libido and/or impotence.

For women with epilepsy the oral contraceptive remains an effective form of contraception, although certain anticonvulsants (phenytoin, carbamazepine and phenobarbitone) increase the metabolism inactivating oestrogen hormones more rapidly. Alternatively, if a couple plan to have a baby they may be concerned in the likelihood of their child having epilepsy (see Table 8).

Some individuals may consider the risk too high, while others judge it to be worthwhile in order to experience parenthood. Counselling before and during pregnancy can dispel any undue fears and provide invaluable advice and reassurance for both parents – epilepsy should not prevent child bearing.

Before or during pregnancy the doctor may amend the anticonvulsants prescribed to the women. In fact, treatment is normally withdrawn if the patient has not suffered an attack for two years, and for those whose attacks are infrequent the number of anticonvulsants will be reduced.

Table 8 **Probability of epilepsy in newborn**

Epilepsy in parents	*Probability of child having epilepsy*
None	1 in 200
One parent	1 in 40
Both	1 in 5 – 1 in 10

Source: Chadwick et al, 1987; Shorvon, 1990.

Table 9 **Common foetal malformations**

Anticonvulsant	*Malformation*
Phenytoin	Hare lip, cleft palate, malformations of the heart
Phenobarbitone	Hare lip, cleft palate, malformations of the heart
Sodium valproate	Neural defects
Vigabatrin	Inadequate evidence of safety during pregnancy; cleft palate

Source: Hopkins, 1988; Chaplin, 1989; Anonymous, 1989.

Anticonvulsants can cause foetal malformations, thus by reducing their number the risk is decreased. The incidence of malformations with monotherapy is 7.5 per cent against 15.6 per cent with combination therapy (Chaplin, 1989). Indeed, a study by Hill et al (1988) indicated a significant increase of cleft palate among children of mothers on anticonvulsants (20 per 1,000) as compared with 'control' mothers (four per 1,000). Certain anticonvulsants are associated with specific malformations (see Table 9).

Carbamazepine is considered to be the safest anticonvulsant during pregnancy (Chadwick, 1989), however, recent research in the USA reveals that it is not riskless. Infants exposed to carbamazepine *in utero* developed minor malformations and had one in five chance of severe developmental delay (Jones et al, 1989). Therefore doctors have the difficult task of trying to balance the therapeutic benefits of treatment against the potential risk to the baby.

During pregnancy the severity of seizures may alter, indeed in 20 per cent of women seizures improve or even go in to remission, while the majority (60 per cent) find their seizures remain unchanged. Unfortunately, seizure frequency increases in the remaining 20 per cent (Feely, 1990) mainly due to noncompliance or an alteration of anticonvulsants prescribed, although hormonal, metabolic, respiratory and psychological factors may also have some influence (Janz et al, 1982).

Some dispute remains whether a baby is adversely affected by breast feeding. Only minute amounts of anticonvulsants are found to be present in a mothers milk, however the contention is that even these small quantities interfere with the brain's maturation (Proceedings: Workshop on Epilepsy, Pregnancy, and the Child, 1982). Nevertheless, there is consensus that breast feeding should only be avoided if the medication is ethosuximide or primidone.

There is no reason why a child whose parent(s) have epilepsy should not have a normal childhood. If a parent does have a seizure at home sensible precautions will ensure that the child is not put at risk. If epilepsy is explained to the child and adults do not panic when the child first witnesses a seizure, no doubt they will be able to cope and deal with the situation.

Employment and social class

Employment is an integral part of most people's lives, it not only helps pay the bills and feed the family it can also increase self esteem and create the feeling of being a valued member of society. Yet, finding employment remains difficult for 50,000-100,000 of the 200,000 potential employees with epilepsy, if research findings are related to the prevalence of epilepsy in the adult population (Floyd, 1986). It is not surprising therefore, that many people with epilepsy do not inform their employers of their condition, with only one in ten always disclosing their disability (Scambler et al, 1980).

Nondisclosure disguises the true number of people with epilepsy in work. The prevalence of epilepsy among NHS employees is 1.35/1,000 (Lisle et al, 1986) which does not reflect the prevalence of active epilepsy in the general adult population – estimates vary from between four and five per 1,000 – suggesting that individuals have not disclosed their condition. MacIntyre's (1976) results are similar with one half of all cases undisclosed. Individuals with more frequent seizures are more likely to disclose their condition, however they are also liable to have greater difficulty in obtaining employment (Scambler et al, 1980).

A common reason for nondisclosure is the misconceptions held by employers about epilepsy. One frequent misconception is that people with epilepsy cause more accidents at work, yet empirical evidence is to the contrary. An analysis of industrial accidents in 29 large British organisations found only 18 accidents caused by seizures over a period of ten years – hence epilepsy does not contribute to a large number of industrial accidents (MacIntyre, 1976).

As people are not willing to risk encountering prejudice they conceal their condition from their employers and workmates. However, whilst nearly 90 per cent of adults with epilepsy perceived their epilepsy to be stigmatising only one third could give details of even

one incident when they suspected an individual of prejudiced behaviour (Scambler et al, 1980).

Concealment is not an ideal solution as dismissal can follow the discovery of the condition without the protection of the Employment Act. Moreover, fellow workers can be put at risk since some jobs may be unsuitable to those with epilepsy. Some examples of unsuitable jobs are given below (Espir et al, 1985):

Airline pilots, seamen, coastguards.

Drivers of trains, cranes.

Vocational drivers: of heavy goods, taxis, public service vehicles.

Work at unprotected heights, e.g. scaffolders, firemen.

Work with dangerous unguarded machinery, e.g. chainsaws.

If employers could learn that 'the capacities of each individual should be assessed and the placement should first be based on the individual's ability rather than on his disability' (Porter, 1984) then concealment would be unnecessary. Unless this occurs capable individuals will continue to be underemployed or unemployed, both reducing an individual's self worth and self esteem and also costing the country in lost Gross National Product (GNP) and welfare benefit payments.

Various studies have shown that individuals with epilepsy tend to be employed in unskilled and poorly paid jobs (Logan et al, 1958; Pond et al, 1960 II; Scambler et al, 1980), although these results may simply be due to the imbalance in general practice populations on which the studies are based. This imbalance could be explained by a higher prevalence of epilepsy in the social class IV and V. However, a Royal College of General Practitioners (RCGP) (1979) study found that although social classes IV and V consulted with their GP more frequently, rates of epilepsy were also high among class one (professional persons). The remaining three classes showed an inverse relationship with social class. The reason for the high figure in class one is unclear particularly as directors and administrators together have the lowest figures.

Extensive work by MacIntyre (1976) of numerous companies corresponds to the RCGP study with 55 per cent of employees in social class three or above (see Table 10). Although it is impossible to draw any conclusions from this data without a breakdown of the social class among the labour force in the companies surveyed, it does illustrate that people with epilepsy are not confined to the unskilled occupations, but can and do work in skilled professions.

A more definite association appears to exist between seizure frequency and employment status. Numerous studies (Aston, 1974; MacIntyre, 1976; Scambler et al, 1980) indicate that frequent seizures are detrimental to employment. MacIntyre found that from a total of 94,700 employees only one individual suffered more than one attack

Table 10 **Social class and epilepsy (Total employees 94,700)**

Social class	*Number known to have epilepsy*	*Pecentage*
I	3	3
II	12	10
III	50	42
IV	34	28
V	20	17
Totals	119	100

Source: MacIntyre, 1976.

a week, while 96 per cent had less than one attack per year or were apparently symptom-free. These results are similar to a smaller survey by Scambler and Hopkins (1980), who concluded that those individuals who experienced either a generalised or partial seizure more frequently than once a month and who also came from a working class background had little chance of full-time work.

To reduce discrimination it has been suggested that medical conditions should be given on a separate health declaration form, only to be scrutinized by qualified personnel, ensuring that the person who is the most suitable is employed irrespective of their epilepsy.

Employment offers an excellent opportunity for people with epilepsy to improve their lives;
'to deny work is not only offensive to civilized standards, but presents an almost insurmountable obstacle to (their) full integration within society' (National Association of Mental Health, 1972).

Elderly

The diagnosis of epilepsy in the elderly (men aged 65 years and over, women 60 years and over) poses a problem for doctors since patients, who frequently live alone, often give confused accounts of their 'funny turns'. The difficulty in making a diagnosis may explain why, in the UK, only 28 per cent of patients over 60 years who have experienced a seizure are referred for specialist investigation (Placito, 1989).

An American study shows the possibility of developing epilepsy significantly increases with age. Hauser and Kurland (1975) found that the annual incidence doubles from 40/100,000 at 60 years to 80/100,000 at 75 years of age. Studies in the UK confirm that first seizures are twice as common over the age of 65 than between the ages of 25 and 64 (RCGP, 1960), although data indicate that once 65 years is reached the rate remains constant. Certain diseases and disorders in the elderly heighten the risk of developing epilepsy (see Table 11).

Table 11 **Causes of epilepsy in the elderly**

	Hildick-Smith (1974) (%)	*Roberts (1982) (%)*	*Luhdorf (1986) (%)*
Cerebrovascular disease	42	44	32
Cerebral tumour	10	12	14
Senile dementia/cerebral atrophy	14	7	2
Toxic/metabolic causes	12	6	12
Other/unclassified	22	31	40

Source: Adapted from Hildick-Smith, 1988.

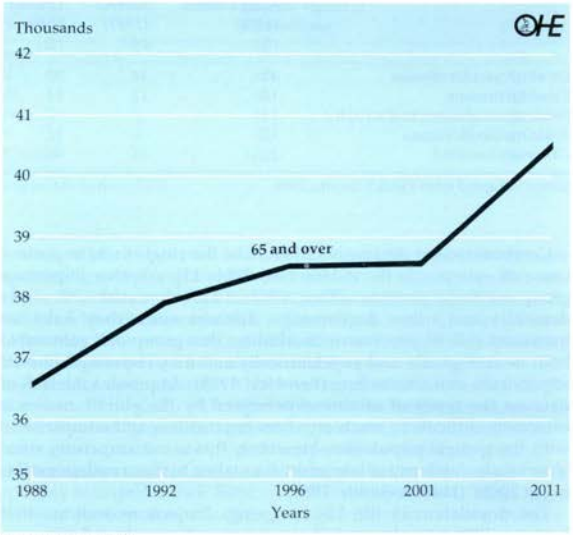
Cerebrovascular disease is shown to be the single most important cause of epilepsy in the elderly (see Table 11). Another important group are those patients whose seizures are associated with senile dementia and other degenerative diseases since they have an increased risk of psychiatric morbidity; this group are vulnerable both neurologically and psychiatrically and may require additional supervision and counselling (Fenwick, 1988). At present the lack of data on the types of seizure experienced by the elderly makes it extremely difficult to reach any firm conclusions and comparisons with the general population. However, this is not surprising since 'previously, 'epilepsy of late onset' was taken to mean epilepsy starting at 20-35' (Hildick-Smith, 1988).

The population in the UK is ageing. Projections indicate that between 1988-1996 the over 65 age group will grow by 2.5 per cent, while the over 75's will rise by 37.5 per cent (OHE, 1989). As the incidence of epilepsy rises with age – at least up to the age of 65 years (see earlier) – it can be anticipated that there will be an increasing number of aged and very aged patients with epilepsy (see Figure 7).

This will put health care resources under pressure particularly in terms of GP and neurologists time, and prescriptions dispensed. Costs to the health service will also rise since the over 75's receive on average 24 prescription items per annum compared to 12 items for the 65-74 age group and only 5.3 items per year for those of working age (Griffin et al, 1990).

Nevertheless, some debate exists as to whether elderly patients with epilepsy should be treated with anticonvulsants as they are more vulnerable to the minor adverse effects due to age related changes in the brain and cerebral conditions. Thompson (1981) observed that phenytoin had adverse effects on memory, concentration, mental and motor speed, while sodium valporate impaired decision making. Conversely, Thompson and Trimble (1981) found the effects of valporate against a placebo to be minimal. A number of

Figure 7 **Epilepsy in the elderly**



Source: OHE, 1989

studies, for example Andrews et al (1986) showed that carbamazepine had the least impact on the cognitive function – this may be due to its early use in trigeminal neuralgia, a condition of the elderly, so that doctors have simply learnt how best to use it in the elderly.

In addition, anticonvulsants may interact with other medication, although the effects have mainly been of minor clinical significance. However, research in this area is limited and more is required to ensure doctors make an informed decision whether to prescribe anticonvulsants to the elderly.

Driving and Insurance

Driving has become a feature of most people's lives whether it is purely for leisure, a means of getting to work or part of their job. Therefore any restrictions on this liberty can be a severe disadvantage. People with epilepsy are only permitted to hold an ordinary driving licence in the United Kingdom if they have been free from

seizures for two years, or have only had attacks whilst asleep for at least three years. The rules regarding heavy goods and public service vehicle licences are more strict, debarring any individual who has suffered a seizure since the age of five years.

Doctors should inform their patients that they are required by law to notify the Driver and Vehicle Licensing Centre (DVLC) of any condition which may affect their fitness to drive unless it is not expected to last for more than three months. In turn, the DVLC will contact the GP and on his information decide on a persons eligibility to drive. However, a study by Harvey and Hopkins (1983) found that neurologists had a poor understanding of the Driving Licence Regulations, thus inaccurate advice may be given to patients concerning their eligibility to drive which can later cause disappointment. The lack of understanding is not surprising since the DVLC do not publish any information which could help doctors and/or patients to comprehend the regulations.

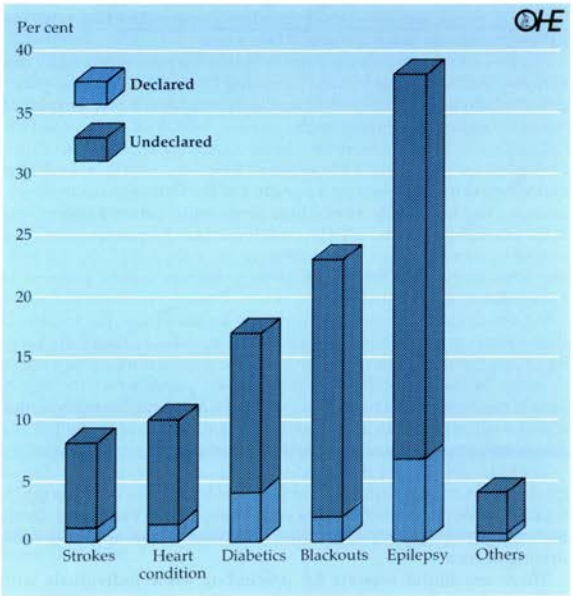
If a driver fails to inform the licensing authorities or the insurer of their epilepsy, the motor insurer will usually refuse to meet the liability of any comprehensive policy claim for personal injury or vehicle damage. In the case of third party insurance, payment is compulsory only if negligence can be proved, as a seizure is not normally considered negligent the insurer has no obligation to meet the bill if a driver collapses at the wheel due to a seizure.

There is little doubt that many individuals with epilepsy do not inform the licensing authorities of their condition. Van der Lugt (1975) found that 96 per cent of drivers with epilepsy involved in accidents had not previously reported their condition when applying for a driving licence.

There are sound reasons for precluding some individuals with epilepsy from driving. Indeed, the accident rate among licensed drivers with epilepsy is 1.3-2.0 times the age matched controls without epilepsy (Harvey et al 1983). Although, accident rates for individuals with epilepsy are similar to rates for other, often less strictly regulated, medical conditions such as diabetes, mental illness and drug use (Krumholz et al, 1991).

Nevertheless, data from the Department of Transport (Taylor, 1983) shows that seizures are largely responsible for accidents where drivers have collapsed at the wheel but survived and could resume driving later. From 1605 police reported accidents where drivers have collapsed at the wheel 38 per cent (610 accidents) were caused by witnessed generalised seizures, while strokes accounted for only eight per cent (128 accidents), other causes are illustrated on the Figure 8. Figure 8 also shows the percentage of people who had not declared their condition, some 82 per cent of those with epilepsy, although 12 per cent were experiencing their first seizure.

Figure 8 **Causes of 1,605 road accidents involving collapse at wheel**



Source: Taylor, 1983

Studies by Norgaard (1961) and Beaussart et al (1979) found that between 8.5 per cent and 13.5 per cent of drivers with epilepsy have had one or several accidents, however these accidents are often less serious, both with regard to physical injury and material damage, than those caused by 'normal' drivers. Of these accidents 75-83 per cent occurred in drivers with complex partial seizures (Van Lugt 1975). Other studies confirm that people with epilepsy are involved in less serious accidents. An investigation of 200 fatal accidents, has shown that seven per cent were caused by alcohol intoxication compared with none as a result of an epileptic seizure (Petitnicolas, 1987). Therefore, whilst individuals with epilepsy may be more likely to be involved in an accident it is probable they will not be seriously injured.

An explanation given for the increased likelihood of road accidents in drivers with epilepsy is the toxic effects of some anticonvulsant treatments. The studies carried out are so far are inadequate, resulting in inconsistent and incomplete results. Nonetheless, a common finding (Trimble, 1981) is that phenytoin has a marked effect on the cognitive function; this effect is not apparent with the use of sodium valporate and carbamazepine. Caution is necessary when extrapolating from these results since the studies have only occurred under laboratory conditions.

As driving allows individuals a certain amount of independence it is not surprising that many individuals with epilepsy, and other disorders (see Figure 8), fail to inform the licensing authorities of their condition. Indeed, the stringent driving restrictions placed on individuals with epilepsy may be counterproductive since they may discourage the disclosure of an individual's condition.

Socio-economic costs of epilepsy

Suicide

Exact figures on the incidence of suicide and self-harm among individuals with epilepsy are difficult to acquire, although the majority of studies conclude that people with epilepsy are more liable to commit suicide and cause self-harm than the remainder of the population.

Studies by Mackay (1979) in Glasgow and Hawton et al (1980) in Oxford found the overall incidence of self-harm to be seven and five times higher, respectively, than in the rest of the population. In the Oxford survey men with epilepsy made twice as many attempts as women; a finding which is atypical of the population in general. An outcome common to both studies was the tendency for people with epilepsy to attempt suicide repeatedly, although neither study offered any explanation.

Studies have indicated that the rate of suicide is somewhat less than the incidence of self-harm among individuals with epilepsy. A study by Henriksen et al (1970) of the 2,763 adults discharged from four neurological clinics in Denmark indicated that approximately 20 per cent of the 164 deaths were suicide; about three times the predicted rate whereas self harm is about five times the predicted rate (Hawton et al 1980).

A review of eleven diverse studies on mortality and suicide in epilepsy by Barraclough (1980) confirmed that there was an increased risk of suicide in individuals with epilepsy. Barraclough found the rate among this group to be about four times the expected, and, was highest (twenty five times greater than expected) in those with temporal lobe epilepsy.

There have been a number of suggestions in an attempt to explain the reasons why people with epilepsy resort to suicide and self-harm[7]. Matthews et al (1982) deemed feelings of helplessness, lack of control, low self esteem and anxiety to be important risk factors. These factors concur with Fisher's (1986) view, who suggested that stress is most likely to be experienced when a patient perceives that they have little control over their condition, or, when epilepsy occurs later in life. Yet, if seizures can be controlled by medication, and, if patients are provided with adequate information about the extent to which their lives may or may not be restricted, then they may be capable of developing effective coping strategies and avoid such an extreme measure as suicide.

Betts et al (1976) drew attention to the burdens that patients with epilepsy have to bear, and suggested that these lead to depression. A particular burden for people with epilepsy is employment (see Employment and social class pp 42), especially if they have a family to support. This may, in part, explain the high incidence of self-harm in a survey by Hawton et al (1980) where over the half the men were unemployed.

A worrying factor is that a child with epilepsy may be more prone to developing emotional problems than a child who does not have epilepsy (Livingstone, 1972). These difficulties often include severe anxiety and depression as well as fears and feelings of insecurity which could lead to suicide.

The anticonvulsants prescribed for patients can cause depression; furthermore, they are also the most frequently used preparations in suicides by individuals with epilepsy (Hawton et al, 1980).

There is a considerable amount of literature concerning the relationship between epilepsy and psychiatric disorders, especially with reference to temporal lobe epilepsy. A general practice survey of patients with epilepsy by Pond and Bidwell (1960 II) established that 29 per cent of the sample had 'psychological difficulties'. Individuals with temporal lobe epilepsy had a higher rate of hospitalisation, and higher rates of severe personality change and psychosis. A study in Poland by Zielinski (1974) from non selected patients with epilepsy indicated that some 58 per cent showed 'mental abnormality' while around three per cent had psychotic symptoms. However, much of the evidence is inconclusive and contradictory on the subject of temporal lobe and generalised seizures causing personality or psychiatric disorders. In order to discover whether such links exist more research is required and the pitfalls of previous studies must be

7 Self-harm is a deliberate non-fatal act committed in the knowledge that it was potentially harmful.

avoided ensuring a precise definition of terms, no sampling bias and proper diagnosis of the type of epilepsy.

It is clear that psychiatric disorders, suicide and self-harm are more prevalent among people with epilepsy. Routine mental status examinations may enable doctors to recognise those patients with epilepsy suffering from depression who are potentially suicidal. Alternatively, physicians may become aware of patients at risk by examining factors such as control, esteem and anxiety. Once the doctors have recognised those at risk, counselling can be arranged and implemented.

It is also important for clinicians to ensure that medication is carefully monitored so that the level of anticonvulsants are at their smallest effective dose and monotherapy is used wherever possible to avoid the depressant effects of polypharmacy (Brit Med J, Editorial 1980). Health professionals should also be made aware of the effects a child's condition may have on psychological health of other members of the family, as it has been suggested that there is 'an association between psychiatric disturbance in the chronically epileptic children and increased psychiatric morbidity among their mothers' (Hoare, 1984), indeed, guilt is common among mothers of children with epilepsy. Thus they would be in a better position to help the child and the family cope with the illness.

Suicide and self-harm can give rise to significant economic costs, particularly the consumption of scarce hospital resources. This includes direct hospital costs such as the use of ambulance services and beds in the emergency department. Whilst individuals who repeatedly harm themselves – which is common in patients with epilepsy – and serious cases requiring prolonged treatment in intensive care units, use considerably more resources.

The indirect costs include specialist counselling for the patients and in some cases family and friends. Indeed, suicide can have significant impact on family and friends, in particular it has been found to have an emotional disturbance on a child whose parent has committed suicide (Shepherd et al, 1976). In addition suicide and self-harm causes both a permanent and temporary reduction in the labour force; although this is likely to only have a negligible economic effect.

The rate of suicide and deliberate self-harm is greater among those with epilepsy than the remainder of the population, by closer supervision a large number of these depressed and potentially suicidal individuals can be observed and therefore be helped. This will avoid the pointless waste of an individuals life and the consumption of scarce health resources which could be employed elsewhere.

Long-stay institutions

Life was grim for individuals with epilepsy at the end of the nineteenth century, particularly if their seizures seriously interfered with employment prospects. Unemployed individuals would have to rely on the generosity of friends and relatives in order to survive. In the absence of such support, and if charitable help was unavailable, the only long term alternatives were the poor law workhouse, the workhouse infirmary, or the county lunatic asylum.

The situation had improved by the turn of the century when a number of residential centres, referred to as colonies, were established. The founders of the colonies aimed to provide a sheltered working and living environment, but precluded those adults or children with epilepsy who were also severely physically disabled, mentally handicapped or mentally ill.

Today, the majority of people with epilepsy are able to live in their own homes, however there are some occasions when long term care has to be considered due to the degree of a persons disability and/or epilepsy. It is difficult to establish the exact number of people with epilepsy in residential care, or the degree of their handicap due to their epilepsy compared to other problems. Morgan and Kurtz (1987) have estimated that in the mid-1970's between 10,000 and 15,000 people with epilepsy were in residential or long term hospital care of some type – that is between five per cent and eight per cent of people with epilepsy in England and Wales.

The Reid Committee Report (1969) anticipated that the demand for long term residential care for people with epilepsy would decline. The Committee suggested three reasons for this view:

1) Improved diagnosis, treatment and assessment, plus developments in rehabilitation would reduce the problem of epilepsy for the patients.

2) Social problems accounted for at least some of the admissions. The Committee believed that:

'in the climate of opinion today, the social services ought to be able to find solutions to such problems within the community for most people with epilepsy'.

3) There will always be a small number of people with epilepsy who require long term residential care. Local authorities were planning to provide homes and hostels for those with handicaps, the Committee believed that those people with epilepsy requiring permanent care should be cared for in this sort of accommodation rather than epilepsy centres.

The statistics seem to show the fulfilment of the Reid Committee's prophesy, with the numbers resident in epilepsy centres falling by 17 per cent (1967-1976). Indeed, the numbers resident in the state sector fell by 30 per cent, while a decline of only eight per cent occurred in

the independent sector (Morgan et al, 1987). However, this was not altogether the realisation of the Reid Commission view. The fall of numbers in the state sector was probably the result of closure policies and policies to reduce the amount of accommodation provided exclusively for people with epilepsy. In contrast, the reduction in the independent sector was caused by the contraction in the number of places available due the modernisation of accommodation.

Admissions to epilepsy centres and other forms of long-term care could possibly be reduced by expenditure on special centres in the community for epilepsy. The aims of a special centre are to diagnose accurately, prescribe anticonvulsants appropriately and to restore or teach abilities so that the patient can maximise his social independence. This may improve some patients employability, lower health service utilisation, and reduce the level of dependency of those still needing continuing care. Therefore, this form of expenditure reduces the burden on health service resources and can, potentially, increase self esteem and an individual's quality of life.

In spite of this only a few specialist centres for epilepsy exist. Indeed, there are about the same number of people with epilepsy as with insulin dependant diabetes, yet only ten specialist clinics for epilepsy exist while every district general hospital and many general practices run special diabetic clinics.

The financial costs

The direct financial cost of the majority of long stay institutions is the responsibility of local authorities. The exact number of individuals with epilepsy resident at these establishments in Great Britain is difficult to establish and therefore a costing cannot be calculated. Nevertheless, costing has been attempted in the United States by the Department of Health, Education and Welfare (US Commission 1978). Although these figures are not comparable to the United Kingdom they serve to illustrate that patients with epilepsy use valuable resources particularly in the health care sector.

Since not all patients with epilepsy have the same economic load the patients were divided in to five groups:

1. Institutionalised patients (including those in nursing homes, institutions for the mentally retarded, state mental homes, etc.).

2. The residential equivalent (patients in the community requiring or waiting for institutional care).

3. Patients in the community requiring regular medical attention for their epilepsy.

4. Patients in the community requiring only occasional medical care for their epilepsy.

5. The unidentified patient in the community whose epilepsy is unreported.

In the calculation of the cost of epilepsy, the Commission ignored group five, while patients in group four were only included in the cost of medication. The contribution of other groups are indicated on Table 12. The Commissions estimates are based on a prevalence of epilepsy of 6.6/1,000 (Hauser et al, 1975).

The estimated total cost of epilepsy in the United States in 1975 was $3.621 million (about 20 per cent of the total cost for diseases of the nervous system), or about £5,000 million at 1989 prices. Other estimates of the total annual cost of epilepsy in the USA have been calculated and are somewhat larger than the Commission's figures. If it is assumed that the United Kingdom has one-fifth the population of the USA and the cost of epilepsy is only a half, it can be estimated that epilepsy in the UK cost £500 million in 1989.

There are a number of costs for epilepsy which can be calculated, including hospital and GP costs (see Table 13). In 1988 the hospital sector absorbed £84.22 million on hospital services for individuals with epilepsy, about 0.6 per cent of all hospital costs. In general practice in 1969, 0.5 per cent (OHE, 1971) of consultations were specifically for epilepsy, by 1988 epilepsy accounted for only 0.2 per cent of consultations. In 1988 the cost of epilepsy to general practice was £3.65 million if this proportion is used as the basis for calculation. In the pharmaceutical service, epilepsy cost £21.4 million, or 0.9 per cent of the total. Excluding the other parts of the service for which estimates are not available this gives a total cost of epilepsy to the National Health Service of £109.27 million. Other costs are difficult to quantify, particularly indirect costs such as lost productivity as well as the high personal costs of epilepsy (see relevant chapters).

Returning to the United States figures on Table 12, individuals with epilepsy in group one or two cost large sums of money. It would therefore be prudent for the government to invest in special epilepsy centres to reduce the burden on health service resources, and, potentially increase earnings and output if individuals become employed. Nevertheless, there remains a small group of patients whose epilepsy is so severe and who may in addition have emotional and physical disorders, that life in the community is not feasible and for whom residential care will continue to be needed. These individuals still need their personal dignity thus the emphasis should be placed on creating a home not an institution.

Table 12 **The overall annual cost of epilepsy in the United States**[8]

	Cost in US at 1989 prices in US dollars	*Cost in US at 1989 prices in £ sterling*
Unemployment (groups 2, 3)	1,140,360,000	691,127,270
Unemployment (group 1)	700,570,000	424,587,880
Underemployment (group 3)	1,142,570,000	692,466,670
Excess mortality (groups 1, 2, 3)	961,350,000	582,636,360
Treatment costs (groups 2, 3)	735,930,000	446,018,180
Costs of institutionalization (group 1)	2,006,680,000	1,216,169,700
Costs of residential care (group 2)	817,700,000	495,575,760
Costs of drugs (groups 1, 2, 3, 4)	243,100,000	147,333,330
Vocational rehabilitation and special education (groups 1, 2, 3)	170,170,000	103,133,330
Cost of research	83,980,000	50,896,970
Total Cost	8,002,410,000	4,849,945,500

Source: Recalculated from the Commission for the control of epilepsy and its consequences, 1978.

8 The costs are based on the Commissions estimates, inflated from 1975 prices to 1989 prices and converted to sterling using the mean exchange rate for 1989 of $1.65:£1. For a full explanation of the original calculations see Shorvon, 1989.

Table 13 **The cost of epilepsy to the National Health Service, UK. 1988**[9]

Health service sector	*Cost attributed to epilepsy £ million*	*Total cost £ million*	*Per cent attributed to epilepsy*
Hospital services	84.22	13,704	0.6
General practice	3.65	1,823	0.2
Pharmaceutical services	21.4	2,528	0.9
Others	Not available	5,572	Not available
Total	109.27	23,627	0.5

Source: OHE

9 The estimates are calculated as follows:
Pharmaceutical Services – Number of anticonvulsant prescriptions multiplied by the net ingredient cost for 1988 (OHE, 1989). The total cost of pharmaceutical services includes charge paid by patient.
Hospital Services – Per cent attributed to epilepsy is calculated on the basis of in-patient costs and is assumed to be the same for all hospital services.
GP consultations – The proportion of consultations specifically for epilepsy (OPCS, 1986) was applied to the total expenditure on the General Medical Services in the UK in 1988 (OHE, 1989).

Looking ahead

Recently, there has been a revival of interest in the condition of epilepsy, yet it receives far less attention than areas such as diabetes and hypertension despite the fact that epilepsy is a common and chronic problem. .

The treatment of the condition is normally supervised by the GP, following referral to a consultant for diagnosis, initial assessment and recommendation for management. Whilst the GP has clinical responsibility for the patient, since he would normally be prescribing in accordance with the treatment recommended by the consultant, the GP does not have full clinical control.

The introduction of widespread epilepsy clinics could extend a GP's clinical control by increasing the management of the condition, including reviewing the need for medication, checking anticonvulsant serum levels and counselling. This would improve seizure control and reduce the need for polytherapy (Placito, 1989). Seizures may become better controlled by increased compliance – compliance is essential in the effective treatment of epilepsy.

The introduction of indicative prescribing amounts (formerly indicative drug budgets) may widen the division between a GP's clinical responsibility and clinical control, since the GP may be reluctant to prescribe an expensive new treatment recommended by the consultant or even take on new patients with chronic epilepsy. In addition, newer treatments tend to be expensive due to the extremely high development cost which are inevitably passed on to the consumer and consequently older drugs may be favoured.

However, overall costs have to be taken in to consideration. Newer medications (such as sodium valporate) may be safer, causing fewer side effects, and be more effective in terms of fewer seizures, perhaps preventing the need for hospital treatment which is advantageous both economically and socially. The economic benefits accrue by reducing both the number of admissions and the duration of in-patient stay. Indeed, the volume of in-patient treatment for epilepsy in England was reduced from 500,053 bed days in 1957 (Wells, 1987) to 311,920 in 1985 (HIPE, 1985); an estimated hospital saving of £21 million[10] at 1988 prices from the development of effective medicines if it is assumed that bed occupancy has remained constant during the period 1985-1988. However the full benefits of effective prescribing will not occur if doctors are over prescribing or prescribing inappropriate or old fashioned medicines – implying a need for additional knowledge.

10 Calculated on the basis of an average daily in-patient cost of £110.47 of all hospitals in England in 1988 (OHE estimate).

It is not only doctors who need re-educating, but society in general, as 'to a great extent the social problems of people with epilepsy are created by the attitude of society towards them'. (Reid report para 40). Attitudes are more enlightened than in the past, nevertheless ignorance and fear still adversely effect employment prospects and social life. Due to the stigma and discrimination associated with epilepsy, most people conceal their condition. The stigma may be reduced if the term 'pre-epilepsy' was used for patients who have had a set number of seizures within a given period. The education system could also perform a vital role in the provision of basic information about epilepsy to children and young people who do not have the condition.

The voluntary sector also plays an important role in the education of the general public, as well as providing information, advice and counselling for many people with epilepsy and those caring for them. Voluntary organisations are also responsible for running many of the residential epilepsy centres and providing other forms of accommodation. Their aim is to promote independence, thus avoiding the need for institutionalisation. Individuals who are at present in long stay institutions, particularly those with additional conditions to their epilepsy, such as mental illness, could soon be returning to the community due to the NHS and Community Care Act. Many may turn to voluntary organisations for help which will further strain their financial resources.

The law also appears to be unreasonable, since it classifies epilepsy as a disease of the mind. Therefore, acts committed during or in the aftermath of a seizure have be held to have happened as a result of insane automatism; unless the defence can show that the seizure was triggered by an external factor. The result of this plea is that the individual has to be found not guilty by reason of insanity, consequently the judge must make the order for admission to a mental institution for at least six months. Thus many individuals plead guilty to avoid committal; a situation which is, at the very least, morally unfair. This was highlighted recently when a young man pleaded guilty to a criminal damage charge, which occurred during a seizure, rather than be committed to a mental hospital. He received a conditional discharge with £100 costs and lost his job (Anonymous, 1991). The success of a Private Members Bill (John Greenway, Conservative, Ryedale) which had its first reading in December 1990 may improve the situation for these individuals by increasing a judge's sentencing options in such cases.

In the future new anticonvulsants may be available to help further control seizures, undoubtably improving an individuals quality of life. Nevertheless, a significant improvement will only occur if epilepsy, and those suffering from the condition, are treated with

understanding by both the medical profession and the public so that they no longer feel they have to conceal their condition. This will depend on the success of public education programmes in aiding awareness and acceptance.

Perhaps if interest and understanding increases a corresponding growth in research funding may occur since, at present, clinical research for epilepsy is seriously underfunded. Diabetes and epilepsy are similar, in that both are chronic conditions affecting comparable numbers of people yet the research quotient[11] from all sources is over £13 per person with diabetes, while only £0.90-£1.20 per individual with epilepsy per year (Shorvon, 1991). This is not only low for a disease, it is also low internationally. If this situation continues fresh advances in treatment and improvements in quality of life for individuals with epilepsy seem unlikely.

11 Calculated by dividing the annual research expenditure from all sources by the prevalence of the disease in the UK population.

References

Albright P, Bruni J (1985). Reduction of polypharmacy in epileptic patients. Arch Neurol; 42: 797-799.

Andrews D G, Bullen J G, Tomlinson L et al (1986). A comparative study of the cognitive effects of phenytoin and carbamazepine in new referrals with epilepsy. Epilepsia; 27: 128-134.

Annegers J F, Hauser W A, Elvebach L R (1979). Remission of seizures of relapse in patients with epilepsy. Epilepsia; 20: 729-737.

Anonymous (1989). Chemist & Druggist. New treatment for epilepsy. Nov 25.

Anonymous (1990). Daily Telegraph. Rabbi Blues secret illness. May 22.

Anonymous (1991). General Practitioner. Legal 'Catch 22' faces people with epilepsy. Feb 1.

Aston R H R (1974). Personal Communication. In: MacIntyre I (1976). Epilepsy and employment. Community Health; 7: 195-204.

Augustine E A, Novelly R A, Mattson R H et al (1984). Occupational adjustment following neurosurgical treatment of epilepsy. Ann Neurol; 15/1: 68-72.

Bagley C (1971). The social psychology of the child with epilepsy. Rutledge & Kegan Paul: London.

Barraclough B (1980). Suicide and epilepsy. In: Reynolds E H, Trimble M (eds). Symposium on psychiatric aspects of epilepsy, Institute of Psychiatry. London, June 1980. London: Churchill Livingstone.

Beaussart M, Faou R, Faou-Pellerey C (1979). Crises et accidents chez les conducters epileptics. Lille Medical; 24: 746-751.

Betts T A, Merskey H, Pond D A (1976). Psychiatry. In: Laidlaw J, Richens A (Eds). A Textbook of Epilepsy. Edinburgh: Churchill Livingstone.

Betts T, Harding G (1986). Epilepsy in tertiary education. In: Epilepsy and Education (1986). Oxley J, Stores G (Eds). Education in Practice for Labaz Sanofi UK Ltd.

British Epilepsy Association (1990). Towards A New Understanding. British Epilepsy Association.

Brit Med J (1980). Editorial. Suicide and Epilepsy. 281: 530.

Britton N, Morgan K, Fenwick P B C et al (1986). Epilepsy and handicap from birth to age 36. Dev Med Child Neurol; 28: 719-728.

Brodie M J (1990). Status epilepticus in adults. Lancet; 336: 551-552.

Bryan C K (1976). Patient information versus patient education. Drug Intelligence and Clinical Pharmacy; 10: 314-318.

Bryant S G, Ereshfsky L (1981). Determinants of Compliance in Epileptic Drug Intelligence and Outpatients. Drug Intelligence and Clinical; 15: 572-577.

Callaghan N, Garrett A, Goggin T (1988). Withdrawal of anticonvulsant drugs in patients free of seizures for 2 years: a prospective study. New England Journal of Medicine; 318: 942-346.

Camfield P R, Gates R, Ronen G et al (1984). Comparison of cognitive ability, personality profile and school success in epileptic children with pure right versus left temporal foci. Ann Neuol; 15: 122-126.

Chadwick D, Usiskin S (1987). Living with epilepsy. Positive Health Guide.

Chadwick D (1989). Epilepsy in women of childbearing age. Brit Med J; 299: 1163-1164.

Chaplin S (1989). How safe are antiepileptic drugs in pregnancy? Mimms Magazine: 25-26.

Collings J A (1990). Epilepsy and Well-Being. Soc. Sci. Med; 31: 2: 165-170.

Cramer J (1989). Better compliance and bid dosing. Scrip No 1465.

Dada T O (1970). Epilepsy in Lagos. Nigera. Afr J Med Sci; 1: 161.

Dam M, Gram L (1985). Epilepsy – Prejudice and fact. Munksgaard.

Dansky L, Andermann E, Andermann F (1980). Marriage and fertility in epileptic patients. Epilepsia; 21: 261-271.

Davidson D L W (1983). Anticonvulsant drugs. Brit Med J; 286: 2043-2045.

Dawson K P, Jamieson A (1971). Value of phenytoin estimation in management of childhood epilepsy. Arch Dis Child; 46: 386-388.

Donselaar van C A, Geerts A T, Schimsheimer R-J (1991). Idiopathic first seizure in adult life: who should be treated. Brit Med J; 302: 620-623.

Dover C (1989). Drug hope for victims of epilepsy. Daily Express. 29 Nov.

Duncan J (1985). Understanding Epilepsy. The National Society for Epilepsy Handbook. NSE

Duncan J (1991). Modern treatment strategies for patients with epilepsy: a review. Journal of the Royal Society of Medicine; 84: 159-162.

Eisler J, Mattson R H (1975). Compliance in anticonvulsant drug therapy. Epilepsia; 16: 203.

Elwes R D C, Johnson A L, Shorvon S D, Reynolds E H (1984). The prognosis for seizure control in newly diagnosed epilepsy. New England Journal of Medicine; 311: 944-947.

Engel J (1989). Seizures and Epilepsy (Contemporary Neurology). Philadelphia USA: F A Davis.

Espir M, Floyd M (1986). Epilepsy and recruitment. In: Epilepsy and Employment. Edwards F, Espir M, Oxley J (Eds). Royal Society of Medicine Services International Congress and Symposium Series No.86. Royal Society of Medicine Services Ltd.

Evans L, Spelman M (1983). The problem of non-compliance with drug therapy. Drugs; 25: 63-76.

Feely M (1990). Epilepsy holds no bar to motherhood. Doctor. 2 Aug.

Feldman R G (1983). Management of underlying causes precipitating factors of epilepsy. In: Epilepsy: diagnosis and management. Browne T R, Feldman R G (Ed). Little, Brown & Co: Boston.

Fenwick P, Toone B, Whetter M et al (1985). Sexual behaviour in a centre for epilepsy. Acta Neurologica Scandinavica; 71: 428-435.

Fenwick P (1988). Psychiatric disorders of epilepsy in the elderly. In: Epilepsy and the Elderly. Ed: Tallis R. Round Table No. 9. Royal Society of Medicine Services.

Fisher S (1986). Stress and Strategy. Lawrence Erlbaum: London.

Floyd M (1986). A review of published studies on epilepsy and employment. In: Epilepsy and Employment. Edwards F, Espir M, Oxley J (Ed). Royal Society of Medicine Services International Congress and Symposium Series No.86. Royal Society of Medicine Services Ltd.

Gallagher B B, Baumel I P (1971). Diphenylhydantoin, phenobarbital and primidone serum concentrations, distribution, and toxicity in a large population of epileptic patients. Neurology; 21: 394-396.

Gibberd F B, Dunne J F, Handley A J, Hazleman B L (1970). Supervision of epileptic patients taking phenytoin. Brit Med J; 1: 147-149.

Goodridge D M G, Shorvon S D (1983). Epileptic seizures in a population of 6,000. I: Demography, diagnosis and classification, and role of the hospital services. Brit Med J; 287: 641-644.

Goodridge D M G, Shorvon S D (1983). Epileptic seizures in a population of 6,000. II: Treatment and prognosis. Brit Med J; 287: 645-7

Gowers W R (1881). Epilepsy and other chronic convulsive diseases. London: Churchill Livingstone.

Griffin J, Chew R (1990). Trends in usage of prescription medicines by the elderly and very elderly between 1977 and 1988. ABPI.

Hart Y M, Sander J W A S, Johnson A L, Shorvon S D (1990). National General Practice Study of Epilepsy: recurrence after a first seizure. Lancet; 336: 1271-1274.

Harvey P, Hopkins A (1983). Neurologists, epilepsy and driving. In: Driving and Epilepsy. Ed: Goodwin R B, Epsir L M E. Royal Society of Medicine International Congress and Symposium Series No.60. Academic Press Inc. (London) Ltd, and Royal Society of Medicine.

Hauser W A, Kurland L T (1975). The epidemiology of epilepsy in Rochester, Minnesota, 1935 through 1967. Epilepsia 1975; 16: 1-66.

Hawton K, Fagg J, Marsack P (1980). Association between epilepsy and attempted suicide. J Neurosurgery Psychiatry; 43: 168-170.

Hayes et al (1977). In: OHE (1983). Keep on taking the tablets?

Haynes R B, Taylor D W, Sackett D L (1979). Compliance in Health Care. John Hopkins Press.

Henriksen B, Juul-Jensen P, Lund M (1970). The mortality of epileptics. In: Life Assurance Medicine. Proceedings of the 10th International Conference of Life Assurance Medicine. (Ed: Brackenridge). Pitman: London.

Hildick-Smith M (1988). Some aspects of epilepsy: a geriatricians view. In: Epilepsy and the Elderly. Ed. Tallis R. Round Table Series Number 9. Royal Society of Medicine.

Hill L, Murphy M, McDowall M, Paul A H (1988). Maternal drug histories and congenital malformations: limb reduction defects and oral clefts. J Epid. & Community Health; 42: 1: 1-7.

Hospital In-patient Enquiry (1985). London: HMSO.

Hoare P (1984). Psychiatric disturbance in the families of epileptic children. Developmental Medicine and Child Neurology; 26: 14-19.

Holmes G L (1987). Diagnosis and management of seizures in children. W B Saunders: Philadelphia.

Hopkins A (1987). Definitions and Epidemiology of Epilepsy. In: Epilepsy (Ed: Hopkins A). Chapman & Hall: London.

Hopkins A (1988). Epilepsy – The Facts. Oxford University Press.

Isbell H et al (1967). An experimental study of the etiology of 'run fits' and delirium tremors. Q J Study Alcohol 1967; 16: 1.

Janz D et al (Eds) (1982). Epilepsy, Pregnancy and Child. New York: Rover Press.

Jennett W B (1962). Epilepsy after blunt head injuries. Heinmann Medical Books Ltd. London.

Jones K J, Lacro R V, Johnson K A, Adam J (1989). Pattern of malformation in the children of women treated with carbamazepine during pregnancy. New England Journal of Medicine; 320: 1661-1666.

Juul-Jenson P (1964). Frequency of recurrence after discontinuance of anticonvulsant therapy in patients with epileptic seizures. Epilepsia; 5: 352-363.

Juul-Jenson P, Foldspang A (1983). Natural history of epileptic seizures. Epilepsia; 24: 297-312.

Kangesu E, McGowan M E L, Edel J (1984). Management of epilepsy in schools. Archives of Disease in Childhood; 59: 45-47.

Krumholz A, Fisher R S, Lesser R P, Hauser W A (1991). Driving and Epilepsy. JAMA; 265: 5: 622-626.

Laidlaw J, Laidlaw M W (1982). Chapter 15. In: Laidlaw J, Richens A, (1982) Textbook of Epilepsy 2nd Ed. Churchill Livingstone.

Lindsay J, Ounsted C, Richards P (1979). Long term outcome in children with temporal lobe seizures. II. Marriage, parenthood and sexual indifference. Developmental Medicine and Child Neurology; 21: 433-440.

Lisle J R, Waldron H A (1986). Employees with epilepsy in the National Health Service. Brit Med J; 292: 305-306.

Livingstone S (1972). Comprehensive management of epilepsy in infancy, childhood and adolescence. Springfield: Thomas.

Logan W P D, Cushion A A (1958) Morbidity Statistics from General Practice. London: HMSO.

Lund L, Jorgensen R S, Kuhl V (1964). Serum diphenylhydantoin (phenytoin) in ambulant patients with epilepsy. Epilepsia; 5: 51-58.

Lund L (1974). Anticonvulsant effect of diphenylhydantoin relative to plasma levels. Arch Neurol; 31: 289-294.

MacIntyre I (1976). Epilepsy and employment. Community Health; 7: 195-204.

Mackay A (1979). Self-poisoning – a complication of epilepsy. Brit J Psychiatry; 137: 277-182.

Matthews W S, Barabas G, Ferrari M (1982). Emotional Concomitants of Childhood Epilepsy. Epilepsia; 23: 671-681.

Mattson R H (1982). Unpublished. In: Browne T R, Feldman R G (Ed). Little, Brown.

Mattson R G (1983). Seizures associated with alcohol use and withdrawal. In: Epilepsy: diagnosis and management. Browne T R, Feldman R G (Ed). Little, Brown & Co: Boston.

McCluggage J R, Ramsey H C, Irwin W G, Dowds M T (1984). Anticonvulsant therapy in general practice in Northern Ireland. Journal of the Royal College of General Practitioners; 34: 24-31.

Medical Research Council Antiepileptic Drug Withdrawal Study Group (unpublished). A Randomized Study Of Antiepileptic Drug Withdrawal In Remission Of Epilepsy.

Meinardi H (1989). Some Historical Considerations. Conference of International League Against Epilepsy.

Meyer F B, Marsh W R, Laws E R, Sharbrough F W (1986). Temporal lobectomy in children with epilepsy. J Neurosurg; 64: 371-377.

Mimms Monthly Index of Medical Specialities. Dec 1990. P92-94.

Morgon J, Kurtz K (1987). Department of Health and Social Security – Special services for people with epilepsy in the 1970's. HMSO.

National Association of Mental Health (1972). Jobs – but not for the disabled. MIND Report No 8. London.

Norgaard A (1961). Sygdomne og andre arsager til trafikulykkei. Ugeser Laeg; 123: 947-952.

Office of Population Censuses and Surveys, Royal College of General Practitioners, Department of Health and Social Security (1986). Morbidity Statistics from General Practice. Third National Study. 1981-82. London: HMSO.

Office of Population Censuses and Surveys (1988). Mortality Statistics. England and Wales. London: HMSO.

OHE (1971). Epilepsy in Society.

OHE Briefing (1983). Keep on taking the tablets? A review of the problems of patient non-compliance.

OHE Compendium of Health Statistics (1989).

O'Hanrahan M, O'Malley K (1981). Compliance with drug treatment. Brit Med J; 283: 298-300.

Oller-Durella L, Pamies R, Oller L (1976). Reduction or discontinuance of antiepileptic drugs in patients seizure free for more than 5 years. In: Janz D (Ed). Epileptology. Stuttgart: Thieme-Verlog: 218-227.

Oxley J, Espir M, Shorvon S et al (1987). The framework of medical care for epilepsy. Health Trends; 19: 13-17.

Patten J P (1989). Anticonvulsants: care is needed when stopping treatment. Modern Medicine: Dec 1989: 879.

Pellock J M (1988). Treatment of seizures in childhood. Virginia Medical; 115: 223-225.

Petitnicolas C (1987). The risk of epilepsy in the driver's seat. Le Figaro.

Placito M (1989). GP's slated on epilepsy care of the over 60's. Doctor. 2 Aug.

Placito M (1989). Specialist epilepsy clinics win backing. Hospital Doctor. 2 Nov. P28.

Polkey C E (1989). Surgery for epilepsy. Archives of Disease in Childhood; 64: 185-187.

Pond D A, Bidwell B H (1960). A survey of epilepsy in 14 general practices. II. Social and psychological aspects. Epilepsia; 1: 285-299.

Pond D A, Bidwell B H, Stein L A (1960). A survey of epilepsy in 14 general practices. 1. Demographic and medical data. Psychiatr Neurol Neurochir; 63: 217-236.

Porter R J (1984). Epilepsy: 100 Elementary Principles. London: Saunders.

Prior D, Linford M (1981). The young physically handicapped in Hounslow: a study of adolescents and young adults with severe locomotor handicaps. Research and Planning Section, Social Services Department. London Borough of Hounslow.

Proceedings: Workshop on Epilepsy, Pregnancy and the Child (1982). In: Janz D et al (Eds) (1982). Epilepsy, Pregnancy, and Child. New York: Rover Press.

Pryse-Phillips W, Jardine F, Bursey F (1982). Compliance with drug therapy by epileptic patients. Epilepsia; 23: 269-274.

Radley R (1987). The educational needs of children with epilepsy. In: Epilepsy and Education. Ed: Oxley J, Stores G. Labaz Sanofi UK.

Reid Report (1969). People with Epilepsy. Report of a Joint Sub-Committee of the Standing Medical Advisory Committee on Health and Welfare and Handicapped Persons. London: HMSO.

Reynolds E H, Elwes R D C, Shorvon S D (1983). Why does epilepsy become intractable? Prevention of chronic epilepsy. Lancet; ii: 952-954.

Reynolds E H (1990). Changing view of prognosis of epilepsy. Brit Med J; 301: 1112-1114.

Robinson N (1982). The epidemiology of epilepsy. Nursing Times. Oct. 13.

Rodin E A (1968). The prognosis of patients with epilepsy. Springfield, Illinois: Thomas.

Royal College of General Practitioners (1960). A survey of the epilepsies in general practice. Brit Med J; 2: 416.

Royal College of General Practitioners, Office of Population Censuses and Surveys, Department of Health and Social Security (1979). Morbidity Statistics from general practice. 1971-72. 2nd National Study. HMSO: London.

Rutter M, Tizard A, Whitmore K (1970). Education, health and behaviour. London: Longmans.

Ryan R, Kempner K, Emleu A C (1980). The stigma of epilepsy as a concept. Epilepsia; 21: 433-444.

Scambler G, Hopkins A (1980). Social class, epileptic activity and disadvantage at work. J Epidemiol Community Health; 34: 129-133.

Scambler G, Hopkins A (1990). Generating a model of epileptic stigma: The Role of Qualitative Analysis. Soc. Sci. Med; 30: 11: 1187-1194.

Schneider J, Conrad P (1980). In the closet with illness: epilepsy, stigma potential and information control. Soc. Problems; 28: 32-44.

Scott D (1969). About Epilepsy. British Epilepsy Association.

Seligsohn R E (1988). The personal and emotional aspects of epilepsy. International Social Work; 131: 165-172.

Shepherd D M, Barraclough B M (1976). Brit J Psychiat; 129: 267-276.

Shinnar S, Viking E P G, Mellits E D et al (1985). Discontinuing antiepileptic medication in children with epilepsy after 2 years without seizures: a prospective study. New England Journal of Medicine; 313: 976-980.

Shorvon S D, Reynolds E H (1979). Reduction of polypharmacy for epilepsy. Brit Med J; 2: 1023-1025.

Shorvon S D (1983). Specialist services for the non-institutionalised patient with epilepsy. Health Trends; 15: 40-45.

Shorvon S D (1990). Epidemiology, classification, natural history, and genetics of epilepsy. Lancet; 336: 93-96.

Shorvon S D (1991). The lack of funds for clinical research in the UK. Journal of the Royal College of Physicians of London; 25: 1: 31-32.

Taylor J F (1983). Epilepsy and other causes of collapse at the wheel. In: Driving and Epilepsy. Ed: Goodwin R B, Epsir L M E. Royal Society of Medicine International Congress and Symposium Series No.60. Academic Press Inc. (London) Ltd, and Royal Society of Medicine.

Temkin O (1971). The Falling Sickness. John Hopkins Press, Baltimore.

Thompson P J (1981). Anticonvulsant drugs, cognitive function and behaviour. London: University of London. PHD. Thesis.

Thompson P J, Trimble M (1981). Sodium valporate and cognitive function in normal adults. Br J Clin Pharmacol; 12: 179-182.

Thurston J H, Thurston D L, Hixon B B, Keller A J (1982). Prognosis in childhood epilepsy: additional follow-up of 148 children. New England Journal of Medicine; 306: 831-836.

Travers R D, Reynolds E H, Gallagher B B (1972). Variation in response to anticonvulsants in a group of epileptic patients. Arch Neurol; 27: 29-33.

Trimble M R (1981). In: Current developments in Psychopharmacy. Eds Valzelli L, Essman W. Vol 6; 65-91. Spectrum: New York.

Tsuboi T (1984). Epidemiology of febrile and afebrile convulsions in children in Japan. Neurology; 341: 175-181.

Turnbull D M, Howell D, Rawkins M D et al (1985). Which drug for the adult epileptic patient: phenytoin or valporate? Brit Med J; 290: 815-819.

US Commission for the Control of Epilepsy and its consequences (1978). Plan for nationwide action on epilepsy. DHEW Publication no (NIH) 78-279.

Van Buren J M (1987). Complications of surgical procedure in the diagnosis and treatment of epilepsy. In: Engel J (Ed). Surgical treatment of the epilepsies. New York, Raven Press; 1987: 465-475.

Van der Lugt P J M (1975). Traffic accidents caused by epilepsy. Epilepsia; 16: 747-751.

Walker A E (1972). The current state of epilepsy in some developing countries. Epilepsia; 15: 99.

Wannamaker B, Morton W, Gross A, Saunders S (1980). Improvement in Anti-epileptic Drug Levels Following Reduction of Intervals Between Clinic Visits. Epilepsia; 21: 155-162.

Webber M, Hauser W, Ottman R, Annegers J (1986). Fertility in persons with epilepsy: 1935-1947. Epilepsia; 27: 746-752.

Wells N (1987). Costs and benefits of pharmaceutical research. OHE.

WHO (1957). Wld Hlth Org. techn. Rep. Ser., No.130.

Zielinski J J (1974a). Epilepsy and mortality rate and cause of death. Epilepsia; 15: 191-201.

Zielinski J J (1982). Epidemiology. In: Laidlaw J, Richens A (1982) A Textbook of Epilepsy. 2nd Edition. Churchill Livingstone.

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