

Ministry of Defence

Synopsis of Causation

Epilepsy

Author: Dr Tony Woolfson, Medical Author, Medical Text, Edinburgh
Validator: Dr Hadi Manji, The National Hospital for Neurology and Neurosurgery, London

September 2008

Disclaimer

This synopsis has been completed by medical practitioners. It is based on a literature search at the standard of a textbook of medicine and generalist review articles. It is not intended to be a meta-analysis of the literature on the condition specified.

Every effort has been taken to ensure that the information contained in the synopsis is accurate and consistent with current knowledge and practice and to do this the synopsis has been subject to an external validation process by consultants in a relevant specialty nominated by the Royal Society of Medicine.

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1. Definition

- 1.1. **An epileptic seizure** is the manifestation of an abnormal and excessive synchronised discharge of a set of cortical [neurones](#).¹
- 1.2. The neurones associated with epilepsy lie in the [cerebral cortex](#), particularly the [neocortex](#) and the [archicortex](#) and their connections with the [brainstem](#) and the [diencephalon](#).
- 1.3. **Epilepsy** is defined as a condition in which the sufferer is prone to recurrent epileptic seizures. This is an area of debate but in the practical situation epilepsy is said to be present when two or more attacks have occurred. There are other causes of seizures, however, such as anoxic seizures after a faint or vasovagal attack.
- 1.4. **Single seizures** in people without epilepsy can be caused by the same precipitating factors that may bring on seizures in those with the condition. The commonest causes in this group are metabolic disturbances such as hypoglycaemia, uraemia, electrolyte abnormalities, alcohol poisoning and withdrawal and drugs, both therapeutic and recreational.

2. Clinical Features

- 2.1. The prevalence of epilepsy is approximately 5-10 per 1000. The incidence is approximately 40–70 per 100,000. The risk of having epilepsy is approximately 1% at birth and 3% at 75 years of age.
- 2.2. Most, but not all seizures include involuntary movements of all or part of the body. In some there is disturbance of or loss of consciousness, but this is not always the case. Seizures can be divided into a number of different types depending on which, if any, part of the body is involved and whether or not consciousness is affected. A considerable number of different epilepsies and epileptic syndromes have been described, and in many of these, a number of different types of seizure may occur. Seizures² and the epileptic syndromes³ are usually classified according to the proposals of the WHO and the Commission on Classification and Terminology of the International League against Epilepsy.

2.3. Classification of epileptic seizures

2.3.1. Generalised seizures

Consciousness is almost always impaired from the onset. Motor manifestations and EEG changes are bilateral.

- ◆ **Tonic clonic seizures** (TCS). These are the most common type of seizures across all age ranges. There may be various prodromal symptoms including headache, mood change, abnormal appetite, light-headedness and sometimes an increase in myoclonic jerking for hours or even days before the convulsion. The tonic phase begins with flexion of the trunk, with upward deviation of the eyes, usually followed by extension and rigidity for some 10–30 seconds. This is followed by a series of clonic contractions and jerking. Respiration ceases and cyanosis may occur. Consciousness is usually lost during the first tonic phase. As consciousness returns, there may be confusion, headache, lethargy, drowsiness and focal weakness of a limb (Todd's paralysis). Most seizures of this type last less than 1 minute, but they may last longer. There are characteristic changes in the electroencephalogram (EEG), both during and (usually) between seizures. Status epilepticus is said to be present when epileptic seizures continue or are repeated without consciousness being regained for 30 minutes or more. It is a medical emergency
- ◆ **Tonic, atonic or clonic seizures**. Tonic seizures are characterised by short (10–45 seconds) sustained contractions of the [axial muscles](#), often with flexion of the upper limbs and flexion or extension of the lower limbs. There may be impairment of consciousness or alterations in [autonomic](#) control, but these are usually less severe than with tonic clonic convulsions. Return to normal is rapid. Tonic seizures may occur many times a day. Atonic seizures (drop attacks) involve an abrupt loss of postural tone of either the head and neck muscles or of all the postural musculature (like a rag doll). There may be disturbance of consciousness, but recovery is rapid, and [post-ictal](#) changes mild. Both tonic and atonic seizures are usually encountered in the context of diffuse cerebral damage and learning difficulties. Generalised clonic seizures are rare and are not normally seen in adults

- ◆ **Absence seizures (AS).** Absence seizures (petit mal) may be typical or atypical. Typical seizures occur with an abrupt loss of consciousness and cessation of all motor activity, possibly accompanied by clonic contractions of the eyelids or face, [automatisms](#) and autonomic, tonic or atonic features. These rarely last longer than 10 seconds and there is no [aura](#) or post-ictal state. They can be repeated many times in a day. Atypical absences are longer and milder, with tonic, atonic and myoclonic features
- ◆ **Myoclonic seizures** are single or multiple, often symmetrical rapid muscular jerks. The most common sites are the face and shoulder girdles, but the whole body may be involved. Consciousness is usually not affected. Myoclonus may be triggered by action, noise or startling

2.3.2. **Partial (localised) seizures**

- ◆ **Simple partial seizures.** These consist of motor, sensory, autonomic or psychic symptoms without disturbance of consciousness. They can occur in isolation, or they may precede complex partial seizures or tonic clonic seizures. Motor manifestations include focal (Jacksonian) tonic and clonic movements, speech changes and forced turning of the head. Abnormal sensations may be visual, auditory, [gustatory](#) or [olfactory](#). Autonomic disturbances may involve sweating, flushing, change of skin colour and hair erection. Psychic symptoms include illusions or hallucinations of mood, memory, and time (déjà vu). The nature of the symptoms depends on the part of the brain involved in the epileptic discharges ⁴
- ◆ **Complex partial seizures.** These are similar to simple partial seizures at onset ("auras"), with the addition of disturbances of consciousness and automatisms. Automatisms are involuntary actions such as lip smacking, chewing, fiddling movements of the hands, patting or rubbing. They are most common in seizures arising from the mesial temporal lobe of the brain.

Partial seizures may evolve into secondary generalised seizures. The partial seizure ("aura") precedes the generalised tonic clonic seizure.

2.4. **Epilepsies and epileptic syndromes**

Some epilepsies with clear aetiologies can be defined as specific diseases. Where a cluster of signs and symptoms is recognised but may have multiple causes, this is called an epileptic syndrome. The epilepsies and syndromes may be generalised or localisation-related (partial or focal).³ The former have no obvious cause and may be genetic in origin. Most patients with idiopathic epilepsies have normal intellectual function and normal investigations. Focal epilepsies with abnormalities on neuroimaging which are presumed to be the cause of the seizures are described as lesional.

2.4.1. **Generalised epilepsies**

- ◆ **Idiopathic epilepsy (Primary generalised epilepsy)**

These have no readily identifiable cause and are generalised seizures from the outset.

- **Absence epilepsy (AE).** This is described as Childhood-onset AE (CAE) or Juvenile-onset AE (JAE), depending on when the first episodes occur. There are typical and atypical forms. There may be many absences each day. Tonic clonic seizures occur in 30% of CAE patients and in 80% of those with JAE. Intelligence and neurological examination is usually normal
- **Myoclonic epilepsies of childhood.** In these, myoclonus is often only a part of a more severe neurological syndrome. A number of different types have been described⁵
- **Progressive Myoclonic Epilepsies (PMEs)** are a group of disorders which can cause any level of disability from mild neurological impairment to death in early childhood.⁶ They may be associated with well-defined biochemical disorders such as a glycogen storage disease, have biochemical or pathological markers but a poorly understood mechanism of damage (as in Huntington's Chorea), or may have no identifiable markers. They generally start in childhood, but can present at any age, and may be associated with ataxia, [spasticity](#), peripheral neuropathy and abnormal movements in addition to the myoclonic seizures
- **Juvenile Myoclonic Epilepsy (JME)** presents in adolescence with the appearance of myoclonic seizures.⁷ Most patients with JME also have tonic clonic seizures and 30% experience absences
- **Epilepsies with tonic clonic seizures.** Tonic clonic seizures occur in most of these syndromes. Idiopathic TCS are not preceded by an aura but there may be some warning symptoms. Onset of seizures tends to be in adolescence and intelligence and neurological examination are normal
- **Epilepsies with tonic or atonic seizures.** These are not fundamentally different in onset or prognosis from other primary generalised seizures
- **West's syndrome**⁸ consists of infantile spasms, psychomotor retardation and specific changes in the EEG. There are a variety of underlying causes including metabolic and degenerative disorders as well as anoxic birth injury. Half the patients develop Lennox-Gastaut syndrome (see below)
- **Lennox-Gastaut syndrome**⁹ is also caused by a variety of brain disorders and is characterised by multiple seizure types and cognitive disabilities. Status epilepticus is frequently observed, and mental retardation and behavioural abnormalities are found in about half of the patients

2.4.2. Partial (focal) epilepsies

- ◆ Partial seizures usually arise from focal structural abnormalities in the brain. They may be described as **simple**, where there is no impairment of consciousness or **complex** when there is impairment of consciousness. Partial seizures may become generalised (secondary generalised seizures). Where an anatomical focus can be clearly identified, partial seizures are described as **symptomatic**. Where it is not possible to distinguish whether seizures arise from a focus which can not be

identified or are primary generalised seizures, this is described as **cryptogenic** epilepsy

- ◆ **Benign focal epilepsies of childhood.** These are usually idiopathic, with simple or complex partial seizures, but may be followed by loss of awareness ("complex partial seizures") and progress to generalised tonic clonic seizures
- ◆ **Temporal lobe epilepsy (TLE).** This is by far the most common of the focal epilepsies which begin in adolescence and early adulthood. Auras of fear, sensory disturbances, psychic phenomena and auditory hallucinations are the usual initial manifestations, with staring, automatisms and sometimes limb movements occurring if the seizures are complex. Most seizures last between one and two minutes
- ◆ **Extra temporal (neocortical) epilepsy.** Seizures may arise from epileptiform activity in various parts of the neocortex. Frontal lobe disturbances are the most frequent after TLE, followed by the parietal and occipital lobes. Symptomatology depends on the part of the brain affected, for instance visual hallucinations or transitory blindness associated with a seizure are likely to arise from occipital lobe activity

2.5. Precipitating factors

In people with epilepsy, many factors can precipitate seizures. These include **electrolyte disturbances** (sodium, potassium, calcium, magnesium), **toxins** (particularly alcohol), and **drugs** (such as tricyclic antidepressants, antipsychotics, anticholinergics, antihistamines, methylxanthines, cocaine, some antibiotics and withdrawal from barbiturates, and benzodiazepines.) Seizures may also be induced by other metabolic disturbances such as **hypoglycaemia**, **hypoxia** and **ischaemia**. **Sleep deprivation** is a powerful precipitant in many patients. **Stress** and **high fever** can both bring on seizures in predisposed patients. In some women, there is a link with phases of the menstrual cycle (catamenial epilepsy). Approximately 3% of patients with epilepsy are photosensitive, and attacks may be triggered by flashing lights or flickering television screens.

2.6. Differential diagnosis

Differentiating epilepsy from other conditions may prove to be difficult. Since patients may lose consciousness for reasons other than epilepsy, it is vital to obtain accounts of the circumstances and the appearance of the patient from witnesses. Nowadays, it may be possible to ask relatives to make video recordings of attacks, and this can be very helpful.

2.6.1. **Non-epileptic seizures.**¹⁰ These were previously known as psychogenic or pseudoseizures. They are three times more common in women than in men, and usually start in adolescence or early adulthood. There are two main categories: attacks of motionless collapse and attacks with motor phenomena. Non-epileptic seizures are often more prolonged than those of true epilepsy. The motor manifestations consist of trembling, waxing and waning of movements, semi-purposeful thrashing of all four limbs, pelvic movements and back arching. During attacks the patient may resent eye

opening. Urinary incontinence and self injury may occur. They are triggered by emotional stress. 10 – 60% of patients with pseudoseizures also have epilepsy, making the diagnosis potentially very difficult.

- 2.6.2. **Syncope.** The characteristic of syncopal attacks is a failure of adequate cerebral perfusion secondary to a fall in blood pressure. The two main sets of causes are vasovagal and cardiac.
- 2.6.3. **Vasovagal syncope** is due to a fall in cardiac output from vagally mediated bradycardia and peripheral vasodilatation. Usually the patient is aware of an impending loss of consciousness, with the darkening of vision or dizziness and tinnitus. Self injury from a violent fall is uncommon. Sometimes, particularly if the fall in blood pressure is severe, there may be some clonic jerks of the limbs which may be confused with an epileptic seizure. However, it is most unusual to see a characteristic tonic clonic pattern.
- 2.6.4 **Cardiac syncope** is due to cardiac arrhythmia or arrest. This is usually sudden without a warning.
- 2.6.5 **Transient ischaemic attacks.** In some patients with transient ischaemic episodes secondary to microembolisation from the carotid arteries there may be brief periods of limb shaking, failure of speech or disturbances of consciousness. This may be associated with unilateral weakness of the limbs. The attacks tend to be rather different from epileptic seizures, and the clinical course will usually make the differentiation clear, since TIAs do not evolve (other than to resolve) over time.
- 2.6.6 **Migraine.** In basilar migraine seen in children and adolescents, there is occipital headache, visual disturbance, ataxia and dysarthria. Characteristically, these symptoms progress over minutes or hours. Rarely, they are accompanied by tonic clonic seizures (convulsive migraine).
- 2.6.7 **Hyperventilation.** Patients with hyperventilation syndrome may have a plethora of symptoms including dizziness, weakness, paraesthesiae, chest pain and altered consciousness. This should not normally be confused with epilepsy but if carpopedal spasm is present there may be some difficulty.
- 2.6.8 **Narcolepsy and cataplexy.** Narcolepsy is excessive sleepiness during the daytime. This may come on very suddenly and semiautomatic behaviour may occur. Cataplexy is precipitated by sudden arousal such as laughter or emotion. There is a sudden loss of tone leading to a drop attack. Other features are sleep paralysis and hypnagogic hallucination.
- 2.6.9 **Drop attacks.** Some of these attacks are probably due to vertebrobasilar insufficiency, but the pathogenesis is unknown. They occur most often in elderly women who suddenly drop to their knees. There is no impairment of consciousness and the attack is quickly over.
- 2.6.10 **Concussive convulsions.** These occur immediately after a head injury such as head clashing and some individuals may demonstrate transient convulsive movements. The outlook for the long-term is excellent.¹¹

2.7 Investigation

- 2.7.1 **Electroencephalography.** Although generally seen as the gold standard of diagnosis of epilepsy, only about one half of the patients with established epilepsy will show abnormalities on a routine electroencephalogram (EEG). Various precipitants, such as hyperventilation and photic stimulation may be used to bring out abnormal patterns. Repeat investigations and sleep deprived recordings are helpful. Epileptiform discharges are also seen in between 1 and 4% of individuals who never have a seizure. The EEG may have some value in predicting seizure recurrence in patients on treatment usually in children.
- 2.7.2 **Computerised tomography (CT).** Before Magnetic Resonance Imaging (MRI) was more widely available, CT scanning was used to detect structural abnormalities in the brain. It may still be useful in some circumstances to exclude the presence of large lesions when it is not possible to obtain an MRI scan immediately.
- 2.7.3 **Magnetic resonance imaging.** MRI scanning is more sensitive and specific than CT, and can reveal both smaller lesions and more types of lesions. It may be particularly useful in patients with partial seizures and those with poor seizure control. It may also detect the types of malformations of development of the cerebral cortex seen in migration disorders.
- 2.7.4 **Video-EEG-telemetry** is a useful investigation when the diagnosis of epilepsy is in doubt. This is the most sensitive and specific test for epilepsy.

3. Aetiology

- 3.1. In two thirds of cases, no clear aetiology can be identified.
- 3.2. Epilepsy involves alterations in normal physiology. A seizure is produced by synchronous and sustained firing of groups of neurones in the brain. The clinical manifestations of the seizure depend on the site and size of the area involved. There may be disturbances of excitatory or inhibitory influences, or of both.
- 3.3. **Genetic factors** are known to be important.
 - 3.3.1. More than 150 **single-gene defects** are associated with epilepsy.¹² Seizures commonly form a part of autosomal recessive enzyme-deficient hereditary metabolic disorders which are apparent in early childhood, such as Tay-Sachs disease.
 - 3.3.2. Several **autosomal dominant types** have also been identified, such as autosomal dominant nocturnal frontal lobe epilepsy (ADNFLE), found on Chromosome 20 codes for the nicotinic Ach receptor subunit.
 - 3.3.3. Genetic factors are believed to play a part in the aetiology of many, if not all cases, but the exact mode of inheritance has not yet been determined in the majority of types.¹³
 - 3.3.4. **Childhood and Juvenile absence epilepsy** may be inherited as an autosomal dominant condition. Siblings and parents can often be shown to have the characteristic 3Hz spike wave EEG pattern, even though they may never have seizures.
 - 3.3.5. **Juvenile myoclonic epilepsy** is genetically determined, with the gene situated on chromosome 5. Children with other myoclonic epilepsies of infancy and childhood often have severe neurological damage, of which the epilepsy is a manifestation. Those who are neurologically normal and have the benign form may have a history of febrile seizures in infancy, and 30% have other people with epilepsy in the family.
 - 3.3.6. There is evidence that **post-traumatic epilepsy** is more likely to follow head injury in patients with a family history of seizures.
- 3.4. **Migration disorders** (cortical dysplasias) are developmental abnormalities of the cellular organisation of the neocortex. They cause some cases of childhood epilepsy. Some are genetically determined and are linked to mutations on the X chromosome.
- 3.5. **The idiopathic epilepsies** have no identifiable structural cause by definition.
- 3.6. There are many **organic causes** of seizures.
 - 3.6.1. **Cerebral tumours** may result in epilepsy in patients of all ages, but have a peak incidence in middle age. 40% of adults presenting with new onset focal epilepsy have an underlying brain tumour in the cerebral cortex. The tumours may be benign (e.g. meningioma) or malignant, either primary or secondary. Treatment of the tumour by surgery or radiotherapy may not stop the seizures occurring.

- 3.6.2. **Infections** such as **bacterial meningitis, viral meningitis, encephalitis** and **cerebral abscesses** may cause seizures. Rarer causes include tuberculosis, cysticercosis, toxoplasmosis, toxocariasis and HIV.
- 3.6.3. **Degenerative diseases** are becoming more common in an ageing population. Seizures may be seen in **Alzheimer's disease**.
- 3.6.4. **Vascular disease** involving the cerebral vessels can result in seizures in a variety of ways. Cerebral emboli probably do so, by causing an acute ischaemia. Seizures may follow strokes of thrombotic or haemorrhagic origin. Diffuse small vessel disease may also present as epilepsy, as may patients with large arterial venous malformations, cerebral aneurysms and cavernomas.
- 3.6.5. Patients with **multiple sclerosis** may have seizures. The prevalence is approximately 1-5% in this group.
- 3.6.6. **Temporal lobe epilepsy** may be associated with [mesial](#) (hippocampal) temporal sclerosis, in which there is significant loss of neurones in the CA1, CA3, CA4 and dentate granule cells. Loss of innervation of the granule cells leads to the production of more [synapses](#) and an excitatory process which may initiate and propagate seizures. In this condition, there is in many cases a history of prolonged febrile seizures, head injury or central nervous system infection. In some, it may result from cortical dysplasia.
- 3.6.7. **Post-traumatic epilepsy** may follow head injuries, and can be associated with focal or generalised seizures. It is more common in societies with a higher rate of violence and in military personnel who may be injured in action.¹⁴
- ◆ Some patients may have only 1 seizure, but in a group of patients who had a single late (> 1 week after injury) seizure, 86% had another within 4 years.¹⁵ After a severe head injury, the likelihood of beginning to have recurrent seizures is known to be increased for at least 10 years¹⁶
 - ◆ The risk is related to the severity of the injury. After mild blunt injuries with only a brief loss of consciousness, the risk is only slightly greater than in a control population¹⁷
 - ◆ With moderate injury (fractured skull and/or unconsciousness lasting between 30 minutes and 24 hours) the risk is approximately double that in a control group at 2% at 5 years, 2.5% at 10 years and 3% at 20 years
 - ◆ When the injury has been severe, with cerebral contusion, intracranial laceration and/or unconsciousness lasting more than 24 hours, the risk is much higher at some 6% at 1 year, 10% at 5 years and 16% at 10 years
 - ◆ With penetrating head injuries, 50% of patients have active epilepsy after 15 years
 - ◆ Overall, the relative risk of developing epilepsy after head injury falls with time, being 12.7 after 1 year, 4.5 up to 5 years and 1.4 after 5 seizure-free years
- 3.6.8. **Extra temporal (neocortical) epilepsies** have an organic identifiable cause in 75% of cases. Pathologies include neoplasms, head injury, central nervous system infections and

vascular malformations¹⁸

- 3.6.9. **West's syndrome** has an identifiable cause in some patients and not in others. The proportion of those with identifiable causes (symptomatic) has risen with the improvements in neuroimaging and neurogenetics. The commonest single cause, comprising 25% of symptomatic cases, is tuberous sclerosis. This is an autosomal dominant condition with mutations of tumour suppressor genes located on chromosomes 9 and 16. Congenital malformations make up some 30% of cases and the majority of the rest have their origins in perinatal complications such as haemorrhage or ischaemic injury. Rarer causes are tumours, CNS infections and some inherited metabolic diseases.
- 3.6.10. **Lennox-Gastaut syndrome** usually occurs in patients with congenital malformations, ischaemic brain injury or disorders such as tuberous sclerosis. About one third of patients have a history of West's syndrome, and there is sometimes a history of epilepsy in the family.
- 3.6.11. **Stress** is never the only cause of epilepsy, which is always associated with some physical disorder of the brain. However, the frequency of seizures in patients with epilepsy may be increased in stressful circumstances.

4. Prognosis

- 4.1. The prognosis of different types of epilepsy is very variable, both within and between the different types.
 - 4.1.1. In the **generalised adult epilepsies**, adverse prognostic factors include short duration between seizures, learning difficulties, abnormal physical signs and a combination of seizure types.
 - 4.1.2. Recurrence of seizures after a 5 year seizure-free period is relatively low in patients both on and off regular treatment.
 - 4.1.3. **Absence epilepsy** usually remits in the mid-teenage years, but about 25% of patients continue to have attacks and TCS. Adverse prognostic factors include intellectual impairment, poor response to medication and myoclonic seizures.
 - 4.1.4. Most **benign focal epilepsies of childhood** spontaneously remit by approximately 18 years of age. Some patients, particularly with benign childhood epilepsy with occipital paroxysms, continue to have seizures (usually TCS), and are unable to stop their medication.
 - 4.1.5. The prognosis in **post-traumatic epilepsy** depends on the severity and the site of the brain damage. Specific adverse risk factors include **subdural haematoma**, **surgery** to deal with bleeding or haematoma formation, **penetrating head injury**, the occurrence of **early (1 day – 1 week post injury) seizures**, **depressed skull fracture**, having a **Glasgow Coma Scale** score of 8 or less at any point and the presence of parietal brain injury. The Glasgow Coma Scale¹⁹ is a grading system which describes the level of consciousness. It uses a scale of 3 (No eye opening, no verbal response, no motor response) to 15 (Opens eyes, moves and speaks to command).
 - 4.1.6. Patients with **temporal lobe epilepsies** usually require lifelong treatment, although some with structural lesions in the temporal lobe may be treated successfully by surgery.
 - 4.1.7. The prognosis of the **extra temporal neocortical epilepsies** is difficult to determine because the description itself came from various series of patients who had had surgical treatment to relieve their symptoms. Surgery may be used very successfully in some of these patients, depending on the type and site of the lesion.
 - 4.1.8. **Juvenile myoclonic epilepsy** has an extremely good prognosis, and seizures are very well controlled on simple medication if precipitating factors are avoided. Patients should, however, continue medication indefinitely because seizures tend to recur despite long periods without them.
 - 4.1.9. In **West's syndrome**, infantile spasms generally cease completely by 5 years of age. However, more than half of children with West's syndrome go on to develop Lennox-Gastaut syndrome or some other form of epilepsy. Only 5% remit completely, and there is some degree of mental retardation in more than 50%. The condition leads to death in between 5 and 20% of patients. Poor prognostic factors are known aetiology, continuing EEG abnormalities and lesions demonstrable on imaging.

4.1.10. Patients with **Lennox-Gastaut syndrome** have a relatively poor prognosis. Many have daily seizures continuing into adulthood despite medication, and mental development tends to be retarded. Factors associated with a worse prognosis are early onset, known aetiology and having West's syndrome previously.

4.2. **Driving.** There are strict and detailed regulations issued by the Driver and Vehicle Licensing Authority (www.dvla.gov.uk). People with epilepsy may drive a car or motor cycle if they have been seizure free for a year. A 3 year license is normally issued after this time. After 7 years freedom from seizures, the license is normally restored up till the age of 70. The regulations are considerably more stringent for heavy and public service vehicles. Specific guidance is given for all conditions in which consciousness or judgement may be impaired.

5. Summary

- 5.1. **Epilepsy is not a single disease.** It is a chronic disorder which is characterised by a tendency to have recurrent seizures, and it has many causes.
- 5.2. The **seizures** are caused by the co-ordinated **discharge of unusually excitable neurones** in the cerebral cortex.
- 5.3. **Seizures may be generalised or focal**, and may be precipitated by biochemical and metabolic disturbances.
- 5.4. Patients may have **anatomical abnormalities** identifiable by sensitive imaging techniques such as MRI scanning, but many do not.
- 5.5. **Some types of epilepsy are inherited.** Some, such as tuberous sclerosis, are inherited multisystem disorders which have epilepsy as a feature. In most cases a genetic component has yet to be defined, but may well be present.
- 5.6. **The prognosis is very variable**, and depends on the type of epilepsy and often on individual factors which are poorly understood.
- 5.7. **Injuries to the head** are associated with subsequent recurrent seizures in some cases, which may not commence until many years after the injury. The prognosis in post-traumatic epilepsy is closely associated with the extent and the site of the brain injury.

6. Related synopses

Stroke

Brain Tumour

7. Glossary

archicortex	The hippocampus and dentate gyrus.
atonic	Lack of normal tone.
aura	Subjective symptoms of epileptic origin without objective signs.
automatisms	Involuntarily motor or verbal acts, often purposeless, foolish or harmful.
ataxia	Failure of co-ordinated muscle movements.
autonomic	Pertaining to the autonomic nervous system (the sympathetic and parasympathetic systems – the part of the nervous system not under conscious control).
axial muscles	The symmetrical muscles of the trunk.
brainstem	The medulla oblongata, midbrain and pons.
cerebral	Pertaining to the brain.
clonic	Contractions and relaxations in rapid succession.
cortex	The part of the brain, mainly on the surface, that contains the bodies of most brain neurones; the grey matter of the brain.
diencephalon	The thalamus, hypothalamus, subthalamus and epithalamus.
gustatory	Pertaining to the sense of taste.
haematoma	A collection of blood in the tissues following damage to blood vessels (a bruise).
hypoglycaemia	Low blood glucose concentration.
hypoxia	Lack of oxygen.
infarct	Area of tissue which dies following interruption of blood supply.
ischaemia	Reduced blood supply (leading to hypoxia).
mesial	Arising from the hippocampus or amygdala.
myoclonus	Muscle jerk activity.
neocortex	All of the brain except the paleocortex and the archicortex .
neoplasm	New growth, usually cancerous.
neurone	Nerve cell.
olfactory	Pertaining to the sense of smell.
paleocortex	The pyriform and olfactory cortices.

post-ictal	Following a seizure.
sclerosis	Hardening of tissue due to excess growth of the connective tissue element.
spasticity	Increase in the normal tone of a muscle, with heightened deep tendon reflexes.
synapses	Structures across which nerve impulses are conducted between neurones.
tonic	Continuous contraction of a muscle.

8. References

- ¹ Foldvary-Schaeffer N, Wyllie E. In: Goetz textbook of clinical neurology. 2nd.ed. Philadelphia: Saunders; 2003. p 1155-86.
- ² Commission on Classification and Terminology of the International League Against Epilepsy: Proposal for revised clinical and electrographic classification of epileptic seizures. *Epilepsia* 1981;22:489-501.
- ³ Commission on Classification and Terminology of the International League Against Epilepsy: Proposal for revised classification of epilepsies and epileptic syndromes. *Epilepsia* 1989;30:389-99.
- ⁴ Palmieri A, Gloor P. The localizing value of auras in partial seizures: a prospective and retrospective study. *Neurology* 1992;42:801-8.
- ⁵ Lombroso CT: Early myoclonic encephalopathy, early infantile epileptic encephalopathy, and benign and severe infantile myoclonic epilepsies: A critical review and personal contributions. *J Clin Neurophysiol* 1990;7(3):380-408.
- ⁶ Berkovic SF, Cochius J, Andermann E, Andermann F. Progressive myoclonus epilepsies: clinical and genetic aspects. *Epilepsia* 1993;34(Suppl 3):S19-S30.
- ⁷ Serratosa JM, Delgado-Escueta AV. Juvenile myoclonic epilepsy. In: Wyllie E, editor. *The treatment of epilepsy: principles and practice*. Philadelphia: Lea & Febiger; 1993. p. 552–70.
- ⁸ Jeavons PM, Livet MO. West syndrome: infantile spasms. In: Roger J, Bureau M, Dravet C, et al, editors. *Epileptic syndromes in infancy, childhood, and adolescence*. 2nd ed. London: John Libbey & Company; 1992. p. 53-65.
- ⁹ Beaumanoir A, Dravet C. The Lennox-Gastaut syndrome. In: Roger J, Bureau M, Dravet C et al, editors. *Epileptic syndromes in infancy, childhood, and adolescence*. 2nd ed. London: John Libbey & Company; 1992. p. 115-32.
- ¹⁰ Lesser R. Psychogenic seizures. *Neurology* 1996;46:1499-507.
- ¹¹ McCorry PR, Bladin PF, Berkovic SF. Retrospective study of concussive convulsions in elite Australian rules and rugby league footballers: phenomenology, aetiology, and outcome. *Brit Med J*. 1997;314:171-4.
- ¹² Berkovic SF, Scheffer IE. Genetics of the epilepsies. *Current Opin Neurol* 1999;12:177-82
- ¹³ Perkin GD. Epilepsy in later childhood and adults. In: Warrell D, Cox TM, Firth JD, Benz EJ, editors. *Oxford Textbook of Medicine*. 4th ed. Oxford: Oxford University Press; 2004. Chap 24.17.
- ¹⁴ Bruns J, Hauser WA. The epidemiology of traumatic brain injury; a review. *Epilepsia* 2003;44(10):2-10.
- ¹⁵ Frey LC. Epidemiology of posttraumatic epilepsy: a critical review. *Epilepsia* 2003;44(10):11-17.
- ¹⁶ Annegers JF, Hauser WA Coan FP et al. Posttraumatic epilepsy risk factors: a one year prospective study after head injury. *New Engl J Med* 1999;38:20-4.
- ¹⁷ Temkin NR. Risk factors for posttraumatic seizures in adults. *Epilepsia* 2003;44(10):18-20.
- ¹⁸ Sveinbjornsdottir S, Duncan JS. Parietal and occipital lobe epilepsy: a review. *Epilepsia* 1993;34(3):93–521.
- ¹⁹ Teasdale G, Jennett, B. Assessment of coma and impaired consciousness: a practical scale. *Lancet* 1974;ii:81-4