Epilepsy: pathophysiology, clinical manifestations and treatment options

Epilepsy: Pathophysiology, clinical manifestations and treatment options

Abstract – Epilepsy is a global health problem affecting approximately 50 million people worldwide. It is one of the most common chronic neurological diseases in the world with serious physical, economic and discriminatory consequences in some parts of the world. However, the healthcare burden and financial cost of treating epilepsy can be reduced with appropriate and prompt interventions. Although epilepsy and its treatment are complex, current evidence suggest insufficient knowledge of seizure classification and a gap in epilepsy diagnosis and care. This article provides an overview of the condition, incidence, aetiology, pathophysiology, current classification, clinical manifestations and symptom management. Thus, the reader will be able to improve their knowledge about care management of people with epilepsy and therefore help to reduce the risk of mortality associated with the medical condition.

Epilepsy is a medical condition that has been recognised as far back as 4000 BC (WHO 2017). It is a non-contagious neurological disorder resulting from outbursts of excessive electrical discharges in a group of brain cells. The World Health Organisation (WHO 2017) defines epilepsy as a chronic disorder of the brain characterised by recurrent seizures, which are a series of involuntary movements of a part of the body (partial) or the whole (generalised) body with or without loss of consciousness. According to Cooper and Gosnell (2015), epilepsy is characterised by recurrent episodes that may include seizure, sensory disturbance and abnormal behaviour. Thus, epilepsy can vary from a brief lapse of attention or muscle jerks to a severe and prolonged involuntary movements.

Incidence

The incidence of seizure varies with age and geographical location. Although anyone may experience seizure, the highest incidence is in people younger than 2 years and older than 65 years (Kerr, Huether and Narayanan 2017) and the onset occurs before age 20 in 75% of cases (Van Meter and Hubert 2014).There are an estimated 3 to 6 million people living with epilepsy in the United States

(Van Meter and Hubert 2014) and epilepsy affects 362,000 – 415,000 people in England and Wales annually (NICE 2016) with direct and indirect cost of £2 billion. Although epilepsy is a global disease, almost four-fifths of affected people live in low and middle income countries. The geographical variation of the prevalence of the disease is attributed to the endemic nature of malaria in some areas, high incidence of road traffic injuries, birth related injuries, lack of availability of preventive programmes and medical infrastructure (WHO 2017).

Aetiology

The causes of this chronic, non-communicable disease are as complex as its manifestations. Broadly, epilepsy is classified into two major categories: idiopathic and secondary epilepsy.

Primary (idiopathic) epilepsy

Idiopathic epilepsy has no obvious underlying cause and is the most common form of epilepsy which accounts for 60% of people with the medical condition. Although there is no identifiable cause for seizures in many people, an inherited predisposition to hypersensitivity of the neurons is considered to play a role (Van Meter and Hubert 2014, Bautista 2013).

Secondary (symptomatic) epilepsy

In this category, there is a known cause. The causes could be due to prenatal or perinatal injuries, congenital abnormalities, a severe head injury, stroke, infection, certain genetic syndromes or a brain tumour (WHO 2017). Symptomatic seizures may occur due to primary neurological disease or be precipitated by structural abnormalities of the brain such as hypoxia, intracranial haemorrhage, central nervous system infection, injury or electrolyte imbalance, metabolic disorder, congenital malfunctions, genetic predisposition or drug or alcohol abuse(Kerr, Huether and Narayanan 2017). Although, many people with developmental disabilities due to serious neurological problems also

have epilepsy, epilepsy is not associated with intellectual capabilities in the absence of other neurological disorder (Bautista 2013).

Pathophysiology

The brain is a sensitive organ protected by the rigid bone of the skull (figure 1). The brain is divided into four structural parts: cerebrum, diencephalon, brainstem and cerebellum. The cerebral hemisphere accounts for about 83% of the total brain mass and it has five lobes: frontal, parietal, occipital, temporal and insular lobes (Boor, Cook and Shepherd 2016). The brain coordinates the functions of the body by controlling the activities of the neurons which are specialised nerves that conduct electrical impulses throughout the central nervous and peripheral systems. To fulfil their functions of controlling cognitive activities, sensory perception and skeletal muscle contractions, the nerve cells (neurons) of the brain must work in harmony. The electrical activity of the brain can be measured by an electro-encephalogram (EEG) and the EEG recording is a useful diagnostic tool for epilepsy.

A seizure is due to sudden, uncontrolled depolarisation of neurons resulting in abnormal motor or sensory activity with or without loss of consciousness (Van Meter and Hubert 2014).The exact cellular activities that initiate seizure are not completely clear, however theories of specific mechanisms for seizure activity include altered permeability of the neuronal membrane, reduced inhibitory neuronal control or imbalance of neurotransmitters (Van Meter and Hubert 2014). According to Spray (2013), seizure results from imbalance between neuronal excitation and inhibition which arise from hyperexcitation and hypersynchronisation of a neuronal network. The alterations in ions and neurotransmitters result in hyperexcitation and loss of inhibitory neurons give rise to hypersynchronicity which can result in partial or generalised seizure depending on its spread of activity (Boss and Huether 2017). Neuronal membrane property alteration may be a result of hypoxia, alkalosis, hypoglycaemia and abnormal neurotransmitter properties, which may cause the

release of large amounts of neurotransmitters at the synapse and consequently promote seizure (Farine 2003).Theabnormalneuronal discharge results in clinical manifestations (Table 1) with alteration in motor function, sensation, autonomic function, consciousness and behaviour (Kerr, Huether and Narayanan 2017).

The neurons in the epileptogenic focus have a lower threshold for stimulation and therefore are hyperexcitable. The irritable neurons are easily activated by any physiological changes and certain conditions such as fatigue or lack of sleep, stress, fever and constipation may lower the threshold for seizures. The focal cells stimulate the nearby normal cells leading to the spread of the activity (Van Meter and Hubert 2014). Factors that may trigger epilepsy in some people include physical stimuli e.g. loud noise, bright light or biochemical stimuli such as excessive fluid retention,hypoglycaemia, change in medication, alkalosis, sudden withdrawal from sedatives and alcohol (Van Meter and Hubert 2014).The immediate period following an epileptic seizure is regarded as a postictal state and this can include signs of headache, confusion, dysphasia, memory loss, short term paralysis or deep sleep (Kerr, Huether and Narayanan 2017).

Diagnosis

Although all seizures are characterised by abnormal and excessive discharge of impulses originating from brain cells, its diagnosis requires a careful assessment. Whilst a diagnosis of epilepsy may be suspected in a person who has experienced a seizure, it is important to determine whether the signs and symptoms are epileptic seizures or a condition that mimics epilepsy.In addition, there is a condition known as Non Epileptic Attack Disorder (NEAD), which is an episode of behavioural, and movement changes that are not caused by alteration of electrical activities in the brain but can be difficult to distinguish from epileptic seizures (NICE 2016).According to Epilepsy Research UK (2017), there is an unacceptably high rate of misdiagnosis of epilepsy in the UK, leading to unnecessary

prescription of anti-epileptic drugs for people who do not have epilepsy while people with epilepsy sometimes wait for a long time to begin treatment.

The following diagnostic techniques can be useful to diagnose epilepsy (Table 2). A detailed medical history obtained from the patient and family will describe the type of seizure, manifestations, cause, duration and previous and current treatments. Information about the use of drug, previous brain injury or infection are useful to aid diagnosis and exclude other causes.In addition to eliciting a thorough history, Kerr, Huether and Narayanan (2017) state that physical examination and laboratory tests such as electrolytes and urea can identify systemic conditions that are known to promote neurologic conditions associated with seizures. Other diagnostic investigations may be indicated and this include:

- EEG to determine the location and type of seizure.
- Magnetic Resonance Imaging (MRI) scan can detect any structural changes in the brain
- Computerised axial tomography (CT or CAT scan) – Although it produce a less detail in comparison to MRI scan, it is useful if MRI scan is contra-indicated, e.g. for a patient that requires anaesthesia for MRI scan or patient with a heart pacemaker. However, CT scan is not suitable for those who are pregnant because it uses x-ray to take images of the brain and this could affect the unborn baby.
- Video telemetry - Video-EEG recording can be used to distinguish between different types of seizure.

  During seizures, there is increased metabolism and blood flow at the focus of the seizures, therefore:
- Positron emission tomography scan (PET scan) can be used to detect changes in glucose uptake and metabolism.

- Single photon emission computed tomography (SPECT) may be used to detect changes in blood flow.

Classification of seizures

There are several kinds of epilepsy and over 40 different types of seizure that may affect individuals in different ways, therefore, the common seizures are discussed below. Boss and Huether (2017) state that there are different classifications of seizures which are based on clinical manifestations, site of origin, EEG readings or response to therapy. In a slightly different way, Cooper and Gosnell (2015) classified seizures based on incidence, characteristics and clinical signs. According to VanMeter and Hubert (2014), a commonly acceptable classification divides seizures into two broad categories ofpartial and generalised seizures(table 2). All these different classifications share some similarities and are based on International League Against Epilepsy (ILAE) classification.

Table 2 CLASSIFICATION OF SEIZURE

1.Partial seizures (focal)
a. Simple
i. Sensory activity
ii. Autonomic activity
iii. Psychic activity
iv. Motor activity (includes jacksonian)
b. Complex
i. Temporal lobe or psychomotor
2. Generalised
i. Tonic-clonic (grand mal)
ii. Absence (petit mal)
iii. Myoclonic
iv. Infatile spasms
v. Atonic (akinetic)
vi. Lennox-Gastaut syndrome (febrile seizures)

Partial seizure

This is usually unilateral, involving a localized or focal area of the brain. Partial or focal seizures involve neurone in an area of the cerebral hemisphere and the signs and symptoms reflect the

location of the seizure activity (Blundell 2006). Partial seizures are divided into Simple and Complex Partial Seizure.

Simple partial seizure

The affected person remain conscious (Blundell 2006) and VanMeter and Hubert (2014) states that although awareness is reduced, consciousness remain intact. Thus, the clinical manifestations of the seizure depends on the part of the brain that is affected and this include:

- Sensory activity such as visual and auditory hallucinations.
- Autonomic activity such as epigastric sensation and pallor of the skin.
- Psychic activity such as disturbed cerebral function.
- Motor activity are present if the motor cortex is the source e.g. jacksonian seizure.

Jacksonian seizure – This is a seizure type with an identified focus and it occurs almost entirely in people with structural brain medical condition. The characteristics depend on its location and the clinical signs commonly start with numbness and tingling in the hand, foot or face. However, this may or may not progress to a tonic-clonic seizure.

Complex partial seizures

This involves impairment of consciousness and may take the form of 'automatisms' such as chewing and swallowing, pacing, repeatedly scratching the head or searching for an object, e.g. temporal lobe or psychomotor seizure.

Temporal lobe or Psychomotorseizure– This complex partial seizure usually arise from the temporal lobe of the brain but may involve the frontal lobe or limbic system (Vanmeter and Hubert 2014). It is a sudden change in awareness, distortion of feeling and thinking with a partially coordinated motor activity. It can occur at any age and last longer than absence seizures. It involves impairment of consciousness and may take the form of 'automatisms', which are bizarre behaviours such as

waiving and clapping of hands, lip smacking, inappropriate repetitive movements, may continue previous ongoing activity before the seizure e.g. picking items from a shop shelf without remembering the activity. The person often appears intoxicated, may carry out antisocial behaviour such as exposing self and may experience visual and auditory hallucinations or feeling of déjà vu (VanMeter and Hubert 2014, Copper and Gosnell 2015, Boss and Huether 2017).

2-*Generalised seizures*

This typical epileptic attack have multiple foci and therefore affect both cerebral hemispheres with loss of consciousness e.g. tonic-clonic, absence, myoclonic, atonic (akinetic), infantile spasms and Lennox-Gastaut syndrome (febrile seizures).

Generalized tonic - clonic seizure (GTC): This was formerly referred to as grand mal epilepsy. According to VanMeter and Hubert (2014), GTC are generalised seizures that may occur spontaneously or after simple seizures and it normally follows a pattern of semiology (symptoms) characterised by prodromal to postictal period (Table 4). This seizure reflects involvement of the entire brain and characterized by a sudden loss of consciousness and immediate bilateral symmetric motor activity.

Table 4 – Pattern of tonic-clonic seizures

Stages	Semiology
Prodromal signs	This occur in some people e.g. nausea, irritability or muscle twitching some hours before the seizure.
Aura	Visual or auditory sensation precedes the loss of consciousness in many people. There is loss of consciousness and the person falls down.
Tonic	Strong muscle contraction resulting in a brief extension, flexion and rigidity in the trunk (ictal phase). The patient may cry due to the contraction of the abdominal and thoracic muscles forcing air out of the lungs. There is clenching of the jaws and respiration ceases.
Clonic	There is contraction and relaxation of the muscles resulting in forceful jerky movements involving the whole body. There is increased salivation (foaming) and individual may have bladder and bowel incontinence. This is followed by spontaneous relaxation of the muscles, the body becomes limb and there is gradual return of consciousness.
Postical	At this stage, the person is confused, tired with muscle aches and falls into sleep.

Absence– This type of seizure, which was formerly referred to as petit mal, occurs during childhood and adolescence and rarely continues above adolescence age. It is characterised by a brief loss of awareness and sometimes transient facial movements (VanMeter and Hubert 2014).Although the patient usually stares into a space for a moment before resuming the previous activity, the clinical manifestations may include occasional slight twitching of the eyelids or lip smacking (Copper and

Gosnell 2015, Van Meter and Hubert 2014).The seizure lasts for 5 – 10 seconds and it may occur several times in a day. It is prone to happen a few hours after waking up or when the individual is quiet and there is no recollection of the episode.

Myoclonic –This brief seizure that may occur in clusters is characterised by jerking (myoclonic movement) but without loss of consciousness. It is a type of seizure which may proceed tonic-clonic by months or years.

Infantile spasm – The onset is between 3 months and 1 year and it may be idiopathic, genetic or due to central nervous system trauma. This type of seizure is characterised by episodes of sudden flexion or extension of the neck, trunk and extremities. The spasms occur in clusters of 5 to 150 times in a day (Kerr, Huether and Narayanana 2017).

Atonic(**Akinetic**) – This is an uncommon seizure with peculiar generalised lack of muscle tone resulting in falls in a flaccid state. It is accompanied with unconsciousness for a minute or two and followed with a rapid recovery without a postictal period (Copper and Gosnell 2015).

Lennox-Gastaut syndrome (febrile seizures)– The onset is early childhood from 1 – 5 years and the clinical manifestations include various generalised seizures such as tonic-clonic, atonic (drop attacks), absence, and myoclonic seizures. This type of epilepsy can cause delayed psychomotor developments and mental retardation (Kerr, Huether and Narayanan 2017).

Revised ILAE operational classification of seizure

Classification of seizure is important to guide further testing, to group patients for treatment, for prognosis and descriptions of seizures by practitioner's and patient's, and to aid researchers to better focus their investigations on mechanisms of seizure types. Fisher et al. (2017) opine that insufficient current knowledge of the classification of seizures led to the recent International League Against Epilepsy (ILAE) revised classification. The new ILAE operational classification of seizures (Fisher et al. 2017) classified seizures into three main groups with further sub-groups (Figure 2). The

classifications that were based on three criteria recognise whether the seizure has a focal or generalised onset, the degree of awareness and other unclassified manifestations of seizures (Table 5). Fisher, Schafer and D'Souza (2016) state that changing terms has its challenges and it may take some time for it to be fully accepted.

Type of seizures

Fisher, Shafer and D'Souza (2016) see classification of seizures as part of the seizure description, therefore, there are other types of seizures within epilepsy syndromes. Menstruation and environmental stimuli such as blinking lights, a poorly adjusted television screen, loud noises, certain music, and certain odours have been associated with initiation of seizures (Van Meter and Hubert 2014).

Status epilepticus (SE) – The duration of a seizure varies and it can last for a few seconds or minutes but normally less than five minutes, however, all types of seizures can develop into status epilepticus. SE is regarded as a cluster of repeated seizures without regaining consciousness in between or a continuous seizure lasting up to half an hour or more (Kerr, Huether and Narayanan 2017, Bautista 2013). This is a life-threatening condition requires urgent attention because seizure activity that lasts longer than 30 minutes can cause damage to the brain or death if not promptly treated. Therefore, it is important to secure the airway, administer oxygen, assess cardio-respiratory function and secure intravenous access immediately.

One of the key challenges in neurology is that non-convulsive status epilepticus (NCSE) account for 25 – 50% of status epilepticus in adults. It is a common unrecognised condition, especially in comatose and critically ill patients. Although it is potentially fatal if untreated, there is no universal definition, classification and approaches to treatment (Maganti et al 2008, Fernadez-Torre 2012).Maganti et al 2008 and Fernadez- Torre (2012) emphasised the importance of further research on NCSE.

Catamenial epilepsy

This is an exacerbation of seizure during a menstrual cycle which is considered to be due to the effect of oestrogen and progesterone leading to altered seizure pattern in women during menarche, menstruation and menopause (Manford 2003). VanMeter and Hubert (2014) also state that the number of seizures may increase during pregnancy and the incidence of congenital abnormalities in children born to mothers with seizures is increased in some women, probably due to their drug treatment.

Photosensitive epilepsy –This is a form of epilepsy in which seizures are provoked by environmental stimuli such as strobe lighting (Van Meter and Hubert 2014). According to Hamandi (2017), seizure can be triggered in about 5% of people with epilepsy by flashing and flickering lights or certain geometric patterns, this is more common in girls than boys and between the age range of 12 and 16 years. Manford (2003) state that the visual stimuli need to occupy a large proportion of the visual field, therefore wearing glasses outdoors on a sunny day and avoiding sitting too close to the television are preventive measures for people who are susceptible to light induced seizures.

Treatment options

It is important to manage epilepsy due to its physiological, psychological and mortality due to status epilepticus or sudden unexplained death in epilepsy (SUDEP). The key goal is to control its cause if known and possible (Kerr, Huether and Narayanan 2017), otherwise, the goal of treatment will be to aid optimal control of seizure and thereby improve their quality of life.

Pharmacological treatment

The use of anti-epileptic drugs (AEDs) is the standard medical treatment for epilepsy and it is effective in controlling seizures in about two-thirds of people with epilepsy. AEDs act by suppressing the overexcitability of cortical neurons either by directly stabilising the nerve membrane, enhancing the activity of inhibitory transmitter Gamma aminobutyric acid (GABA) or a combination of both

mechanisms. Some drugs mimic, neutralise or prolong the effect of neurotransmitters such as GABA (Figure 3). Brown (2016) identified three ways by which AEDs exert their effects:

- By mainly targeting sodium and calcium channels to modulate the intrinsic membrane conducting activities to inhibit excessive firing of neurons.
- Inhibit GABA metabolism or block GABA transport.
- Inhibit the excitatory mechanism, mainly the glutamatergic system.

There is a wide range of effective AEDs which are available in tablet, capsule, injection or as a syrup for children. However, the strategy of treatment should be individualised based on the type and severity of symptoms, co-medication and co-mobidity, previous response to drug and patient's preference (NICE 2016). According to the British National Formulary (BNF) (2017), drugs used to control epilepsy include Oxcarbazepine, primidone, clobazam, retigabine, phenytoin, topiramate, valporic acid, vigabatrin, carbamazepine, ethosuximide, phenobarbital, and levetiracetam(Table 6). In the UK, initiation and subsequent drug management (first line treatment and adjunctive) is informed by NICE guidance.

All drugs have shortcomings such as a narrow therapeutic index, adverse effects and drug interactions (BNF 2017, Brown 2016). Therapeutic combinations that is sometimes inevitable due to a lack of response to frontline therapeutic agents may increase the risk of adverse effect; therefore, therapeutic drug monitoring is essential. NICE (2016) recommends monotherapy and indicated polytherapy for seizures that cannot be controlled by one drug. In addition, AEDs should be withdrawn gradually and under specialist supervision (BNF 2017) to avoid severe rebound seizures.VanMeterand Hubert (2014) state that compliance is crucial regardless of the side effects because sudden withdrawal may result in a more serious seizure or status epilepticus.

Although pharmacological approach is the most effective way of treating seizures, Manford (2003) acknowledges patients increasing interest in using complementary therapy. However, they should never be used to supplement conventional treatments without consulting with their doctor because

some natural remedies are strong convulsants. According to Manford (2003) this substances are contraindicated for epilepsy due to their high content of reactive monoterpene ketones, such as camphor, pinocamphone, thujone, cineole, pulegone, sabinylacetate and fenchone (table 7). Although pharmacologically neutral therapies, such as massage, reflexology, homeopathy, biofeedback and spiritual healing are not likely to be harmful in comparison to pharmacologically active therapies like herbs and aromatherapy, all complementary therapies should be provided by a qualified therapist (Manford 2003).

Surgical management

This is excision of an identifiedepileptogenic focus following lack of response to medical treatment. Surgical interventions such as lobectomy, corpus callosotomy or hemispherectomy may be suitable where there is an underlying condition such as brain tumour or an identified focus for the seizures. Fisher et al. (2017) state that it may be possible to remove or disconnect areas of the brain where epileptic activity starts in focal onset epilepsy, if it can be identified. Likewise, WHO (2017) and Kerr, Huether and Narayanan (2017) state that surgical intervention may be helpful in patients who respond poorly to drug treatment. However, excision of an epileptogenic focus is always preceded by extensive diagnostic assessment and collaborative medical decision (Blundell 2006).

Vagal nerve stimulation (VNS)

The updated NICE (2016) guidance recommends VNS as an adjunctive therapy in managing seizures which are refractory to AEDs and for those that are unsuitable for surgery. This is the process of surgically connecting electrodes via a lead to left vagus nerve in the neck and connected to a stimulator located in left upper chest area for the purpose of sending electrical messages to slow down the irregular activity that lead to seizures. However, majority of people will continue on their AEDs, try new drugs when available as they will continue to experience seizure activity.

Ketogenic diet

Blundell (2006) states that a high fat, low carbohydrates and protein (ketogenic) diet has been found to reduce seizures in children, however, this requires nutritional monitoring because it can lead to nutritional deficiencies.Manford (2003) also reported that ketogenic diet may be beneficial in children and some adults but it has potential side effects of thirst, hunger, weight loss and toxicity in polypharmacy.

Medical and Nursing management

The primary aims of care are to stop seizure, prevent injury and ensure adequate cerebral oxygenation. The nurse has a key role to play to minimise the symptoms of epilepsy and help to reduce the risk of mortality during seizure. During the seizure, the nurse should remain calm; record the sequence, time and signs of the seizure. It is important to remove dangerous objects and not to restrain the person or put anything in their mouth. Similarly, it is crucial to maintain privacy and administer prescribed AEDif the patient is conscious and it is safe to swallowor through rectal or buccal. The nurse's role includes observing seizure precautions such as putting a soft object under the head, pad side rails of the bed and maintain the bed in a low position. The nurse should stay with the patient throughout, and if the patient becomes agitated after a seizure (postical), speak calmly and reassure the patient. The nurse is also expected to give appropriate explanations, and offer both physical and psychological support to the patient. Summary of the nursing care during seizure is presented in table 8.

Table 8: Nursing care during seizure

- Provide a safe private place away from curious onlookers
- If possible, ease the patient to the floor and if on the bed, remove pillows and raise side rails
- Protect the head with a pad
- Loosen tight clothing and remove eye glasses if worn
- Do not attempt to open the mouth
- Remove dangerous objects
- Never restrain the person or put anything in their mouth
- Position the patient in a recovery position
- Maintain privacy, remain calm and time the seizure
- Administer prescribed AED if safe
- Stay with them and give appropriate support and explanation.

In the case of status epilepticus, ABC (Airway, Breathing, and Circulation) assessment is carried out to maintain a clear airway and adequate oxygenation. The patient is positioned on the side to prevent aspiration of gastric content and an airway is inserted to avoid obstructed airway. Oxygen is administered as prescribed via nasal cannula and the oxygen saturation rate is monitored. Intubation and suctioning of the airway may also be required depending on the patient's condition (Bautista 2013). Immediate treatment for this prolonged seizure includes intravenous diazepam (Valium), oxygen and fluids to correct electrolyte imbalance (Van Meter and Hubert 2014). Intravenous lorazepan and fosphenytoinmay also be administered to stop seizures as soon as possible. Other medications such as phenytoin, Phenobarbital are also given at a later stage to maintain a seizure-free situation.Additionally, it is important to monitor the intravenous line because it can easily be dislodged during seizures (Bautista 2013).Most acute areas / NHS Trusts in the UK have an agreed SE protocol to guide medical management.

The vital signs, neurological signs, seizure activity and response of the patient to care are monitored continuously and documented. Blood samples are taken to monitor electrolyte, glucose, blood cell count and drug levels. Symptomatic treatment may be initiated, for example, intravenous glucose is administered if the seizure is caused by hypoglycaemia. In the event of unsuccessful treatment, general anaesthetic such as short acting barbiturate may be administered with cardio-respiratory management (Bautista 2013). Seizures may result in complications such as laboured breathing or prolong seizure and injuries may occur during seizures. Depending on the outcome of the progress of the treatment pathway, NICE (2016) recommends involving the expertise of the anaesthetist and possible transfer to intensive care unit as part of the treatment protocol for prolonged or repeated seizures.

After the seizure, the nurse should prepare a patient centred care plans, help the client and family to understand epilepsy and explain how to live a full life and where to seek help, for example, epilepsy organisations offer a range of services to their members (table 9).Whilst the patient may gain knowledge of the subject through research on the internet, some of the information may be inaccurate, therefore, practitioners need to assess, dispel the myths and diplomatically re-orientate the patient. Providing health education is an integral part of the disease management and this should cover topics such as trigger factors, drug compliance, use of complimentary therapy, restrictions concerning driving, contraception and risk of teratogenicity associated with antiepileptic drugs during pregnancy. A detailed history and assessment coupled with telling them or a loved one or carer to keep a seizure diary, i.e. dates, time of day or night and circumstances is important.

NICE (2016) recommends a patient centred approach to decision making by involving the patients and families.The patient can play a central role in the identification and avoidance of the trigger factors and this includes the use of illicit drugs and alcohol. Anyone taking drugs such as antiepleptic

drugs which act on the brain are more susceptible to the effects of alcohol and illicit drugs. Manford (2003) states that the use of heroin, opiates, high dose of amphetamines and consumption of over 50 grams of alcohol daily increases the risk of seizure.Although it is optional, inform the patient about wearing identification tags such as medical alert bracelets, necklace and identification cards.In addition, epilepsy specialist should be a key part of the network of care, particularly, to ensure access to community and multi-agency services and to provide follow-up care and adequate support (NICE 2016).

Table 9: Support services

Epilepsy Action - www.epilepsy.org.uk/
Joint epilepsy Council - http://www.jointepilepsycouncil.org.uk/
Epilepsy Society - https://www.epilepsysociety.org.uk/
Epilepsy action Scotland - http://www.epilepsyscotland.org.uk/
Wales Epilepsy association cyf - https://beta.companieshouse.gov.uk/company/03262144
Brainwave The Irish Epilepsy association - http://www.janssen.com/ireland/ourgiving/36
Sudep Action - https://sudep.org/

Prognosis

Diagnosis of a long-term illness such as epilepsy can be unpleasant and painful for individuals affected and every patient copes with their illness differently (Lawal 2016). Although, 70% of patients can be successfully managed with anti-epileptic drugs, there is an associated risk of morbidity and mortality with epilepsy from causes not linked to epilepsy e.g. intracranial tumour, metastatic lesions to the brain and injuries resulting from falls (Manford 2003). Nonetheless, some deaths appear to be directly linked to epilepsy itself and this is termed sudden unexplained death in epilepsy (SUDEP).

Epilepsy Research UK (2017) states that SUDEP is an unwanted outcome for approximately 500 – 600 people in the UK each year. This occur mostly during sleep or when they are alone due to the impact of seizure on the body's vital functions such as breathing and cardiac rhythm or due to accidents

such as drowning and falls. Other possible contributory factors to SUDEP identified by Epilepsy Research UK are early onset of diagnosis (before age 16), having uncontrolled generalised seizures, drug non-compliance, young adults particularly males and having seizures while alone or at night. In a nine-year small-scale study in England, Shankar et al. (2014), found that SUDEP is underestimated in the country. The epidemiological study identified some epilepsy-related death risk factors and these include increase in seizure intensity and frequency within the last six months and a lack of epilepsy review within the last one year. Although death is an inevitable debt owed by everyone (Lawal 2016), the NHS Outcomes Framework (DH 2016) identified five domains geared towards improving patient's care in the UK, and domain 1 focuses on preventing avoidable deaths (Department of Health 2016). Therefore, there is a need to address the issue of potentially preventable death to help patients with neurological problems live independent lives.

Conclusion

A lot of changes in epilepsy knowledge and understanding have been driven by research, policies, new technologies, increases in the understanding of the physiology and classification of the disease, and drug discovery to improve the care of people with epilepsy in the UK and worldwide. Despite these considerable improvements in the treatment of epilepsy, the medical condition still represents a significant healthcare burden in terms of physical, social, emotional and financial costs. Regardless of the technological advancement, misdiagnosis remains an issue and classifying epilepsy is challenging. In addition, status epilepticus and SUDEP are challenging outcomes of epilepsy and there is no preventive measure for epilepsy. Therefore, there is a need to improve the current knowledge base through ongoing research to aid better care and reduce avoidable complications and death.

Key words

Epilepsy, Nervous system, Neurological disorders, seizure, Long-term conditions.

Key points

Epilepsy can affect anyone regardless of age, sex, race or social status.

The health impact lead to physical, economic and social consequences.

Seizures are recently categorised into three types with subcategories of motor or non-motor with or without impaired consciousness.

The duration of a seizure varies and it can last for a few seconds or minutes.

Although most patients respond to pharmacotherapy, surgical interventions is useful for drug-resistant epilepsy.

Table 1 – Clinical manifestations

- Temporary symptoms such as loss of awareness, disturbances in movement, sensation (vision, hearing and taste), mood or cognitive abilities.
- Physical problems such as bruising resulting from seizure related injuries
- Risk of premature death.

(WHO 2017)

Table 2 – Diagnostic techniques

- Detailed medical history
- Physical examination
- Laboratory tests such as electrolyte and urea
- EEG

- Magnetic Resonance Imaging
- Computerised axial tomography (CT or CAT scan)
- Video telemetry
- PET scan
- SPECT

Table 5: Criteria for the revised classification

- Origin of the seizure in the brain
- Degree of awareness during the seizure
- Other clinical features of seizures

Table 7: Natural remedies contraindicated in epilepsy

-Eucalyptus

Fennel

Hyssop

Pennyroyal

Rosemary

Sage

Tansy

Turpentine

Wormwood

Source: Manford (2003)

Table 6: AED options

AEDs	Indications	Daily adult dose	Common adverse reactions and comments
Carbamazepine (Tegretol)	Focal and secondary GTC seizures	0.8 – 1.2 g in divided doses (Maximum 1.6-2g)	Oedema, rash, fatigue, blood disorder and drowsiness. Blood cell counts and haematocrit should be done at least monthly.
Ethosuximide (Zarontin)	Absence seizures	500 mg daily with gradual increase to usual dose of 1-1.5g	Gastro-intestinal irritation, drowsiness, weight loss and headache.
Phenobarbitone (Luminal)	All forms of epilepsy	10mg/kg (Maximum 1g)	Rashes, drowsiness, mental slowing, aggressiveness, depression. Withdrawal seizures may occur if stopped abruptly after prolonged use.
Phenytoin (Epanutin)	Focal seizures. Tonic-clonic seizures	3-4mg/kg daily as a single or in two divided doses increased gradually as necessary. Usual dose 200-500mg.	Drowsiness, headaches, nausea, ataxia, diplopia or nystagmus, slurring of speech, gingival hyperplasia, rash and hirsutism. Monitor plasma concentration and ensure good oral hygiene.
Sodium Valproate	All forms of epilepsy	Initially 600mg daily. Maximum 30mg/kg daily.	Aggression, anaemia, confusion, diarrhoea, deafness. Associated with risk of congenital malformations and developmental delay if used during pregfnancy.
Primidone (Mysoline)	All forms of epilepsy except absence seizures	125mg daily (Maintenance 0.75-1.5)	Depression, irritability, dizziness, ataxia and rarely impotence.
Topamax (Topiramate)	Given alone or as adjunctive treatment in GTC or focal seizures with or without secondary generalisation. Adjunctive treatment for seizures associated with Lennox-Gastaut syndrome.	Initial 25mg at night for 1 week and increased gradually to usual 100-200mg daily in 2 divided doses.	Include nausea, diarrhoea, vomiting, constipation, dyspepsia, abdominal pain, dry mouth, taste disturbance, gastritis, impaired attention, cognitive impairment, movement disorders, seizures, tremor, malaise, speech disorder, drowsiness, sleep disturbance, anxiety and confusion.
Vigabatrin (Sabril)	Adjunctive treatment of focal epilepsy with or without secondary generalisation.	1g daily in single or 2 divided doses. (Maximum 3g daily)	Side effects include nausea, vomiting, drowsiness, mood change, agitation, confusion, impaired memory and weight gain. Prescribed and supervised by an appropriate specialist when other appropriate drug combinations are ineffective or have not been tolerated.
Clobazam (Frisium)	Adjunct in epilepsy	20-30mg daily (Maximum 60mg)	Drowsiness, dizziness, impaired co-ordination.

Clonazepam (Rivotril)	All forms of epilepsy	4-8mg daily in divided doses.	Drowsiness, mental slowing, dizziness, confusion.
Acetazolamide (Diamox)	Second-line drug for both tonic-clonic and partial seizures.	0.25-1.0g daily in divided doses.	Headache, lethargy, nausea, pins and needles.
Lamotrigine (Lamictal)	Monotherapy and adjunctive treatment for focal seizures, GTC seizures	100-200mg daily as a single or two divided doses.	Rash, headache, dizziness, insomnia, aggression blood disorders may occur.
Gabapentin (Neurontin)	Focal seizures with or without secondary generalisations. Neuropathic pain	Initially 300mg once daily (0.9-3.6g daily in 3 divided doses)	Drowsiness, dizziness, abdominal pain, ataxia, confusion, cough, diarrhoea.
Tiagabine (Gabitril)	Adjunctive treatment for partial seizures with or without secondary generalisations.	5-10mg daily, maintenance 30-45mg.	Dizziness, drowsiness, headache, tremor, confusion, speech impairment, diarrhoea.
Levetiracetam	Monotherapy and adjunctive therapy in the treatment of focal onset seizures with or without secondary generalisation.	Initially 1g daily in two divided doses. (Maximum 2.5g daily)	Drowsiness, dizziness, headache.
Oxcarbazepine (Trileptal)	Monotherapy and adjunctive treatment of focal seizures with or without GTC seizures.	0.6-2.4g daily in divided doses.	Rash, drowsiness, dizziness, headache, nausea, diarrhoea.
Thiopental Sodium	Status epilepticus (if other measures fail)	75-125mg for 1 dose (2.5% solution)	Arrhythmias, cough, headache, hypersensitivity reactions, hypotension, laryngeal spasm.
Brivacetam	Adjunctive therapy of partial onset seizures with or without secondary generalisation	25 – 50mg twice daily adjusted to 25 – 100mg twice daily	Anxiety, constipation, decreased appetite, depression, dizziness, insomnia, vertigo, vomiting.
Eslicarbamazepine acetate	Adjunctive treatment for adults with focal seizures with or without secondary generalisation	400mg once daily Increased gradually to a maximum dose of 1.2g daily.	Dizziness, fatigue, drowsiness, gastro-intestinal disturbances, headache, impaired coordination, rash, visual disturbances.
Lacosamie	Adjunctive treatment for adults with focal seizures with or without secondary generalisation	50mg twice daily with gradual increase to maximum dose of 200mg 2ce daily.	Abnormal gait, blurred vision, cognitive disorder, constipation, depression, dizziness, flatulence, nystagmus, pruritus, tremor, vomiting.
Perampanel	Adjunctive treatment for adults with focal seizures with or without secondary	2mg once daily with gradual increase to maintenance dose	Aggression, anxiety, ataxia, backpain, blurred vision, changes in appetite, suicidalideation, vertigo, weight increase.

	generalisation	of 4 – 8mg once daily. (Maximum 12mg/day)	
Piracetam	Adjunctive treatment of cortical myoclonus	7.2g daily in 2 to 3 divided doses (Maximum 24g/day)	Hyperkinesia, nervousness, weight gain.
Pregablin	Adjunctive treatment for adults with focal seizures with or without secondary generalisation. Peripheral and central neuropathic pain.	150mg daily in 2 to 3 divided doses (Maximum 600mg daily in 2-3 divided doses)	Appetite changes, blurred vision, confusion, constipation, diplopia, dizziness, drowsiness, impaired memory, insomnia, oedema, visual field defects, weight gain.
Zonismade	Monotherapy for treatment of focal seizures with or without secondary generalisation in adult with newly diagnosed epilepsy	100mg once daily Stepwise increase to maintenance dose of 300mg once daily (Maximum 500mg per day)	Abdominal pain, agitation, alopecia, anorexia, ataxia, confusion, constipation, impaired memory, insomnia, nausea, speech disorder, weight loss.

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