INTRODUCTION

Epilepsy has been known since antiquity. The word epilepsy has been derived from the Greek verb ‘epilamvanein’ which means to be seized, to be taken hold of, or be attacked. In ancient time, the epilepsy was considered as a kind of witchcraft insanity, supernatural force, hysteria, madness or possession by the devil which leads to the stigmatization of the individuals with epilepsy (Vanzan & Paladin, 1992). As a consequence, treatment of epilepsy at that time included exorcism and bloodletting, and a variety of substances e.g., brew of mistletoe, blood from a decapitated, man and pulverized craniums to be given to aid the sick person. Seizures are dramatic, public, and frightening as they occur with unpredictable frequency in unexpected places. The forced cry, the loss of consciousness, the fall, the twitching, and the foaming at the mouth suggest possession by a spirit. The earliest detailed account of epilepsy is given in a part of a Babylonian text on medicine, Sakikku (All diseases), written over 3000 years ago, i.e. before 1000 BC which is preserved in the British Museum London. The Babylonians were keen observers of clinical phenomena and have provided remarkable descriptions of many of the seizure types that we recognize today, including what we would call tonic-clonic seizures, absences, drop attacks, simple and complex partial seizures and even focal motor (Jacksonian) or gelastic attacks. They also understood some aspects of prognosis including death in status as well as post-ictal phenomena. The Babylonians had no concept of pathology, however, each seizure type was associated with invasion of the body by a particular named evil spirit. Thus, treatment was not medical but spiritual. This supernatural view dominated thinking about epilepsy until quite recently and even now remains a deeply rooted negative social influence in some parts of the world.

It was, however, unsuccessfully challenged by Hippocrates in 5th-century BC, who first suggested that the brain was the seat of this disorder, it was also the mediator of the intellect, behaviour, and the emotions.
Hippocrates, a great Greek physician (460 to 377 BC) in his book on epilepsy, ‘On Sacred Disease’ (which is not a medical text; it was apparently written for the layperson) attacked the prevailing belief that those afflicted with epilepsy were possessed by ‘God or demons’. He proposed that epilepsy was a brain disease caused by the blockage by phlegm of air carrying vessels to the brain. By the fifth century BC, the word had gradually acquired the specific and particular meaning associated with it today (Temkin, 1945). During 17th and 18th centuries epilepsy was one of several key areas of debate in the gradual identification and separation of “nervous disorders” from “mental disorders”, which led to the beginning of modern neurology in the 19th century. As the concept of a brain disorder gradually took hold between the 17th and 19th centuries, it was widely believed that epilepsy must have a vascular basis attributable to either acute anaemia or acute congestion of the brain. It is often thought that Jackson was the first to develop an electrical theory of epilepsy with his famous statement that “Epilepsy is the name for occasional, sudden, excessive, rapid and local discharges of grey matter” (Jackson, 1873). The modern era, the age of enlightenment for patients with epilepsy, began after Adrian and Mathews (1934) who at a meeting of the physiological society in Cambridge confirmed and made scientifically respectable Hans Berger’s human EEG which he had discovered more than 30 years before. Modern concepts of epilepsy originated in the work of mid 19th-century physicians and scientists, the most notable of them being John Hughlings Jackson, 1931. He established a scientific approach to the study of epileptic phenomena. Hughlings Jackson established the important concept that there were different categories of seizures each with its own physiology and semiology. Even in comparison with all the advances made during the last century more than at any other time in history consider how enormous and fundamental was that first step attributed to Hippocrates in about 400 BC, that epilepsy is a disease of the brain that must be treated by diet and drugs, not by religious incantations (Jones, 1967). In the second half of the 20th century remarkable
progress was made in diagnostic facilities and possibilities through structural and functional neuroimaging including CAT and MRI as well as video-telemetry and magneto encephalography. With the development of effective anticonvulsants and the introduction of EEG, physicians have come full circle to Hippocrates’ belief that epilepsy is rooted in organic brain disease. The modern era is also marked by an expansion of interest in basic mechanisms underlying seizures and epilepsies, stimulated by developments in genetics, molecular biology, neurophysiology, functional imaging and numerous neurochemical techniques for exploring the concepts of excitation, inhibition, modulation, neurotransmission and synchronization. Every advance seems to add to the enormous complexity of the nervous system and the probability that multiple elusive ‘genetic–molecular–metabolic mechanisms’ contribute to the wide range of epilepsies.

Epilepsy is, of course, not a specific disease, or even a single syndrome, but rather a broad category of symptom complexes arising from any number of disordered brain functions that themselves may be secondary to a variety of pathologic processes. The terms convulsive disorder, seizure disorder, and cerebral seizures have been used synonymously with epilepsy. They all refer to recurrent paroxysmal episodes of brain dysfunction manifested by stereotyped alterations in behavior. Recently, the International League Against Epilepsy (ILAE) has proposed new definition for epileptic seizure and epilepsy. Epilepsy is defined “as a condition characterized by recurrent seizures (two or more), a clinical manifestation presumed to result from an abnormal and excessive discharge of a set of neurons in the brain. The clinical manifestation consists of a sudden and transitory abnormal phenomenon which may include alterations of consciousness, motor, sensory, autonomic or psychic events, perceived by the patient or an observer” (ILAE, 1993). Epileptic seizures are usually brief, lasting from second to minutes, and they are marked by the sudden appearance of behavioural manifestation that may be purely motor or they may affect other brain functions. Epilepsy is a physical condition that can
affect anyone of any age, background, and race. It may be caused by anything that affects the brain, including a genetic defect, a malformation in the development of the brain, or may be the result of physical trauma or an illness such as stroke, meningitis or a tumor. In some people there will be no evident cause. People with epilepsy will have seizures that arise when there is an abnormal electrical discharge of the nerve cells in the brain. Whilst many people will have a single seizure at some time in their life, a person with epilepsy will have recurrent seizures. Epileptic seizures can take many forms. Long gone are the days when people were said to either have ‘grand mal’ or ‘petit mal’ seizures. These terms are now outdated, particularly as they do not, in any way, convey the complexity and multiplicity of the different seizure types. While most people will imagine the stereotypical falling to the ground, jerking, shaking and frothing at the mouth. Most seizure types do not involve convulsions. Some people experience an aura—a partial seizure preceding a generalized seizure— which is often seen as a warning sign. An ‘aura’ may be emotional (fear, anxiety), physical (dizziness, nausea), or sensory (tingling or crawling sensation on the skin, spots or colors before the eyes, strange taste or smell). Everyone has a seizure threshold. The lower the seizure threshold, the more likely the brain cells are to fire at a lower level of stimulus causing a seizure. Children tend to have a relatively low seizure threshold. This may help to explain why children often outgrow their epilepsy as their brain matures. An individual’s seizure threshold may be lowered by genetics, brain damage, sleep deprivation, missed medication, drug toxicity (too much medication), poor nutrition, caffeine, drug abuse, consumption of alcohol, full bladder, constipation, fever, colds, infection, menstrual cycle, heat, humidity, and emotional stress. In combination, some of these factors may lower one’s seizure threshold even further. Environmental conditions may provoke seizures in humans and animals suffering from epilepsy. Flashing lights, high sounds, sleep deprivation, stress etc., are known triggers of seizure activity in susceptible individuals. Epilepsy is not necessarily a life long
condition. Epilepsy has the potential to be self-limiting. In humans, epilepsy is now regarded as a condition that, for the majority of patients, will remit spontaneously or by drug induction (Placencia et al., 1994; and Sander and Sillanpaa, 1997). The period during which the seizure actually occurs is called the ictus or ictal period. The aura is the earliest portion of a seizure recognized, and this is the only part remembered by the patients that acts as a warning. The clinical signs of aura should not be confused with the signs of prodromes, since prodromes by definition are long-lasting (hours to days) events in the form of anxiety, irritability, withdrawal and other emotional aberrations, whereas the time frame in which the aura acts consists of a few seconds to a few minutes. The time immediately after a seizure is referred to as the ‘postictal’ period. The interval between seizures is the ‘interictal’ period. A seizure may have many different appearances, which can be difficult to recognize, but some of the following may be indication that a seizure is occurring, viz:

- Sudden falls for no reason;
- Lack of response for short period of time;
- Daydreaming or short attention blackouts;
- Unusual sleepiness and/or irritability when awakened from sleep;
- Rhythmic movements of the head (head nodding) or head dropping;
- Rapid blinking or upward eye rolling;
- Frequent unwarranted complaints from the child that things look, sound, taste, smell or feel funny or different than they actually are;
- Sudden stomach pain followed by sleepiness or confusion
- Repeated movements or jerking movements that look out of place or unnatural;
- Memory gaps;
- A blank stare followed by repetitive, meaningless movements;
- Dazed behaviour with the inability to communicate or talk for a brief period of time;
- Bed wetting or waking with a side bitten tongue for no apparent reason; and
- Lip smacking, chewing or swallowing;
Epilepsy is a group of neurological conditions, the fundamental characteristics of which are recurrent, usually unprovoked, epileptic seizures. A common operational definition of epilepsy is two or more unprovoked seizures occurring more than 24 hours apart (ILAE, 1993). However, the new ILAE proposal (Fisher et al., 2005) offers a more fundamental definition of epilepsy as a chronic condition of the brain characterized by an enduring propensity to generate epileptic seizures, and by the neurobiological, cognitive, psychological, and social consequences of this condition. The new ILAE definition also acknowledges importantly the psychological and social consequences of epilepsy. This change recognizes that to the affected patient, epilepsy is more than seizures, and that the condition in its entirety comprises many facets, different for each individual, that contributes to disability and impaired quality of life. Epilepsy accounts for 1% of the global burden of disease, determined by the number of productive life years lost as a result of disability or premature death. Among primary disorders of the brain, this burden ranks with depression and other affective disorders, Alzheimer’s disease and other dementias, and substance abuses. Among all medical conditions, it ranks with breast cancer in women and lung cancer in men. Eighty per cent of the burden of epilepsy is in the developing world, where 80–90% of people with epilepsy receive no treatment at all (Meinardi et al., 2001). It is also necessary to recognize that epilepsy consists of more than seizures for the affected individual; and effects on his or her family. Epilepsy leads to multiple interacting medical, psychological, economic and social repercussions, all of which need to be considered. Fear, misunderstanding and the resulting social stigma and discrimination surrounding epilepsy often force people with this disorder “into the shadows”. The social effects may vary from country to country and culture to culture, but it is clear that all over the world the social consequences of epilepsy are often more difficult to overcome than the seizures themselves. Significant problems are often experienced by people with epilepsy in the areas of personal relationships and, sometimes,
legislation. These problems may, in turn, undermine the treatment of epilepsy.

**Etiology and Risk Factors**

Etiology and risk factors for epilepsy vary with age and geographic location. Congenital, developmental, and genetic conditions are mostly associated with epilepsy in childhood, adolescence, and early adulthood. Head trauma, CNS infections, and tumors may occur at any age and may lead to epilepsy, although tumors are more likely over age 40. Cerebrovascular disease (CVD) is the most common risk factor for epilepsy in people over 60 years of age. Some studies from India also suggest that head injury, development delay, and family history of epilepsy are significant risk factors (Bharucha et al., 1988; Pal, 1999; and Sawhney et al., 1999).

(1). Prenatal and Perinatal Risk Factors: Prenatal and perinatal adverse events do not appear to be associated with the occurrence of childhood epilepsy when children with cerebral palsy and mental retardation are excluded. A study (Degen, 1978) indicated that higher age of the mother at birth, toxemia of pregnancy, premature birth, and heavy birth weight were associated with later epilepsy. Nelson and Ellenberg (1987) concluded that among the hundreds of prenatal and perinatal factors studied, the main predictors of childhood seizure disorders were congenital malformations of the fetus (cerebral and noncerebral), family history of certain neurologic disorders, and neonatal seizures.

(2). Postnatal Causes: Among the following postnatal causes, no single cause predominates:

(a). **Traumatic Brain Injury**: Jennett (1973) found that in about 5% of all patients admitted to the hospital, seizures occurred within a week of the head injury. The incidence in children <5 years of age was almost double this. In civilian population, case control studies (Rocca et al., 1987), and retrospective cohort studies
(Annegers et al., 1980) have reported an increased risk for unprovoked seizure after head trauma. The increased risk for subsequent unprovoked seizures is related to severity of the injury (Annegers et al., 1980).

(b). Vaccination: It is unlikely that a relationship exists between vaccination, in particular, pertussis immunization, and serious acute neurologic illness in children. However, diphtheria-tetanus-pertussis (DPT) vaccination appears to increase the risk for fever resulting in an earlier onset of febrile seizures among children predisposed to such seizures (Hirtz et al., 1983; Shields et al., 1988). Thus, it appears that the only adverse neurologic consequence significantly associated with DPT vaccination is febrile seizure in children who are already predisposed to such seizures.

(c). Attention Deficit Hyperactivity Disorder: Clinically there is a perception that attention deficit hyperactivity disorder (ADHD) is more common among children with epilepsy, due to the seizure disorder or its treatment (Austin et al., 2001; Dunn et al., 1997). When the occurrence of new-onset seizures is examined in selected samples with ADHD, the percentage of children who develop unprovoked seizures (0.2% to 2%) is greater than the expected rate (Hemmer et al., 2001; Holtmann et al., 2003; Jennett, 1973; and Williams et al., 2001).

(d). Other Neurological Problems: Mental retardation and cerebral palsy both predispose to the development of epilepsy. In Rochester, Minnesota, neurological deficits from birth, mental retardation, and/or cerebral palsy were important antecedents of epilepsy (Hauser, 1993). Thus, in the absence of associated disability or postnatal injury, the incidence of epilepsy in those with mental retardation alone was more than three times that of the general population of Rochester, Minnesota. Additionally,
there appears to be an interaction between mental retardation and cerebral palsy on the risk for epilepsy

(3). Other Risk Factors for Epilepsy: Several established risk factors for epilepsy are as following-

(a). Head Injury: Head injury increases the risk for later unprovoked seizure, with the greatest risk occurring among survivors of severe injury. In civilian populations, case-control studies (Rocca et al., 1987) and retrospective cohort studies (Annegers et al., 1980) have reported an increased risk for unprovoked seizure after head trauma. The increased risk for subsequent unprovoked seizure was related to the severity of the injury (Annegers et al., 1980).

(b). Central Nervous System Infections: Infections of the CNS i.e. encephalitis and meningitis are associated with an increased risk for subsequent unprovoked seizures. Among children with complex partial seizures, there is a 31-fold increased risk associated with viral encephalitis (Rocca et al., 1987). CNS infections increased seizure risk 11-fold (Annegers et al., 1988), 16-fold increased risk was associated with encephalitis, a fourfold increased risk with bacterial meningitis, and a twofold increased risk with aseptic meningitis.

(c). Central Nervous System Malignancies: Case series have been used to evaluate the risk for epilepsy among patients with brain tumors or the tumors in newly diagnosed cases of epilepsy. Case series of the former type report that 28% of patients undergoing surgery for brain tumors have seizures (Foy et al., 1981; Franceschetti et al., 1988). Series of the latter type have revealed that 12% to 16.3% of adults with newly diagnosed epilepsy have brain tumors. Indeed, seizures are often the first signs of brain tumors (Roberts et al., 1982).
(d). **Occlusive Cerebrovascular Disease**: A chronic epileptogenic lesion at the stroke site may account for unprovoked seizures that occur more than 1 to 2 weeks after a clinical stroke (De Carolis et al., 1984). Such unprovoked seizures occur after a clinically detected stroke in 2.7% to 35% of patients (Ohman, 1990; Kotila and Waltimo, 1992). This variability can be attributed to several factors in study design such as variation in length of follow-up, inclusion of only patients with computed tomographic confirmation of stroke, or inclusion of patients with subarachnoid hemorrhage only if they survived long enough to have surgery. Generalized seizures are not uncommon after stroke, although some investigators include secondary generalized seizures in this category (Gupta et al., 1981). Researchers who distinguish primary from secondary generalized seizures find that primary generalized seizures account for 4% to 69% of all unprovoked seizures after stroke (Kotila and Waltimo, 1992; Sung, 1990; Ohman, 1990). Data from three studies suggest that if hypertension, measured by a physician's diagnosis or blood pressure reading, increases seizure risk, the magnitude of the increased risk is small ranging from 1.0 to 1.7 (Ng et al., 1993; Hesdorffer et al., 1996).

(e). **Dementia**: The underlying pathology of Alzheimer disease may be associated with an increased susceptibility for seizures. A population-based case-control study (Hesdorffer et al., 2000) found a six fold increased risk for unprovoked seizure associated with Alzheimer disease and dementia increased the risk for unprovoked seizure eightfold overall for both generalized- and partial-onset seizure.

(f). **Multiple Sclerosis**: Several studies now suggest that multiple sclerosis is associated with an increased risk for epilepsy suggesting an epileptogenic role for white matter lesions
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(Kinnunen and Wikstrom, 1986; Nicoletti et al., 2003; Olafsson et al., 1999; and Striano et al., 2003). People with multiple sclerosis, 1.8% to 4.8% had seizures at the time of diagnosis or developed seizures after diagnosis. Across studies, this risk is more than three times the risk for unprovoked seizures over a similar time period.

(g). Alcohol and Illicit Drugs: Alcohol intake appears to be associated with seizure occurrence. The association between heroin and marijuana use and seizures was investigated in case control study (Ng et al., 1993). Controlling for age, history of head trauma, history of stroke, and alcohol use, these investigators found that heroin increased the risk for unprovoked seizures threefold and the risk for provoked seizures fourfold. These interesting results require confirmation in other studies.

(h). Socioeconomic Status: Indices of low socioeconomic status (SES) are associated with many established risk factors for epilepsy, including cerebrovascular disease, head trauma, congenital malformations, central nervous system infection (meningitis, encephalitis), alcohol intake or abuse, brain neoplasm, and Alzheimer disease. Studies now show that low socioeconomic status is associated with an increased risk for developing epilepsy. A prospective study in England (Heaney et al., 2002) using a composite measure of SES concluded that low SES is a risk factor for the development of epilepsy. A case-control study in Iceland (Hesdorffer et al., 2005) found that low education increased the risk for unprovoked seizure twofold.

(i). Genetics: The concept of a genetics predisposition to epilepsy was proposed over 52 years ago (Lennox and Lennox, 1960). Twin studies have shown that genetic factors are particularly important in the generalized epilepsies but also play a role in the partial epilepsies (Berkovic et al., 1998). Human genetic
epilepsies can be categorized by mechanism of inheritance, whether they are idiopathic or symptomatic, generalized or partial and where it is known that which class of gene is involved. The mechanism of inheritance identifies three major group: Mendelian epilepsies, mutations in a single gene can account for segregation of the disease trait, Non-mendelian or complex epilepsy, several loci interact with environmental factors to produce the pattern of familial clustering. These include epilepsies which exhibit a maternal inheritance pattern due to mutations in mitochondrial DNA and Chromosomal disorders, in which the epilepsy results from a gross cytogenetic abnormality. The effects of some genotypes on epilepsy may involve gene-environment interaction, that is, the joint influence of genetic and environmental factors in a causal pathway leading to disease (Ottman, 1996; Rothman and greeland, 1998). Some genotypes might not affect risk directly, but instead might increase susceptibility to the effects of environmental factors. In this case, individuals who inherit the risk-raising genotype would not develop epilepsy unless they were also exposed to the environmental factor. Risk is higher in the relatives of patients with generalized epilepsy than in relatives of those with localization-related epilepsy (Ottman, 2001). Studies suggest that clinical characteristics of epilepsy tend to cluster in families. Both Tsuboi (1980) and Beck-Mannagetta et al (1991) found that the distribution of seizure types in affected relatives was skewed toward the same types of seizures, although different seizure types were also seen. Concordance rates for epilepsy are consistently higher in monozygotic twins than in dizygotic twins (Kjeldsen et al 2003; Kjeldsen et al., 2001;Vadlamudi et al., 2004; Lennox,1960), although the concordance rates vary across studies because of differences in methodology such as twin ascertainment methods,
numbers of pairs included, approaches for diagnosis and classification, and methods used to calculate concordance. In the classic twin study by Lennox (1955) concordance rates were higher in MZ than DZ pairs only for twins with intact brains, illustrating the greater importance of genetic factors in idiopathic or cryptogenic epilepsies than in symptomatic epilepsies.

Classification of Epileptic Seizures and Types of Epilepsy or Epileptic Syndromes:

Two types of classifications are used for epilepsy (1) classification of the epileptic seizures, and (2) classification of the epilepsies. Classifications of the epileptic seizures are concerned with classifying each individual seizure as a single event based on clinical and EEG information. Classifications of the epilepsies are designed to classify syndromes in which the type or types of seizures are one, but not the only feature of the syndrome. Specific epileptic syndromes have been identified by their characteristic seizure types, pattern of seizure recurrence, age of onset, associated neurologic and other clinical signs, electroencephalographic (EEG) findings, presence or absence of familial occurrence, and prognosis.

1981 ILAE Classification of Epileptic Seizures: Despite persistent controversy, the 1981 Classification of Epileptic Seizures has gained general acceptance and is widely used. An ILAE Task Force has been reviewing this classification since 1997, but no new classification has yet been proposed (Engel, 1998; 2001; 2006). Although flawed, it has been agreed that useful constructs should not be abandoned until a clearly better version is devised. The 1981 Classification of Epileptic Seizures was based on expert consensus, analyzing video-recorded seizures and considering their semiology (observable manifestations of the seizures) as a sequence of ictal events developing in time. The International Classification of Epileptic Seizure as follows:

1 Partial Seizures: In general, partial seizures are those in which the first clinical and electroencephalographic changes indicate initial activation
of a system of neurons limited to part of one cerebral hemisphere. These seizures are also called focal or local seizures. Partial seizure can be classified on the basis of whether or not consciousness is impaired during the attack into one of the following three fundamental groups:

a. **Simple Partial Seizures**: In a simple partial seizure consciousness is not impaired during the seizure but person is unable to control various body movements. He or she may experience unusual feelings. For some people, the first symptom of a seizure may be an aura. Simple partial seizures are caused by a local cortical discharge, which results in seizure symptoms appropriate to the function of the discharging area of the brain without impairment of consciousness. There is considerable evidence that simple partial seizures usually have unilateral hemispheric involvement. A simple partial seizure usually lasts for 2 to 10 seconds, although they may last longer.

b. **Complex Partial Seizures**: In complex partial seizures, the consciousness is impaired. Impaired consciousness is defined as the inability to respond normally to exogenous stimuli by virtue of altered awareness and responsiveness. Complex partial seizures frequently have bilateral hemispheric involvement. The attack characteristically ends gradually with a period of postictal drowsiness or confusion. Complex partial seizures usually last for 2 to 4 minutes. A complex partial seizure may take place (i) with impairment at onset and (ii) simple partial onset followed by impairment of consciousness.

c. **Partial Seizures evolving to Generalized Tonic-clonic Convulsions (GTC)**: A partial seizure may not terminate, but instead, progresses to a generalized motor seizure. A partial seizure may take place as a (i) simple evolving to GTC, and (ii) complex evolving to GTC, including those with simple partial onset.

(2) **Generalized Seizures**: Generalized seizures are those in which the first
clinical changes indicate initial involvement of both hemispheres and affect the entire body. Consciousness may be impaired and this impairment may be initial manifestation. Motor manifestations are bilateral. The ictal electroencephalographic patterns initially are bilateral and presumably reflect neuronal discharge which is widespread in both hemispheres. Generalized seizures can be categorized as followings:

a. Absence Seizure (*petit mal*): The hallmark of the absence seizure is a sudden onset, interruption of ongoing activities, a blank stare, possibly a brief upward rotation of the eyes. If the patient is speaking, speech is slowed or interrupted; and if walking, he or she stands transfixed. Usually the patient will be unresponsive when spoken to. The attack lasts from a few second to half a minute and evaporates as rapidly as it commenced. No aura is present and little or no postictal symptomatology occurs. Generally, absence seizures are the most tolerated socially, as the attacks are brief and involve little physical change. Absence seizures usually respond well to medication.

b. Myoclonic Seizures: Myoclonic jerks that can be single or multiple are sudden, brief shock-like contraction which may be generalized or confined to the face and trunk or to one or extremities or even to individual muscles or groups of muscles. They may occur predominantly around the hours of going to sleep or awakening from sleep. No loss of consciousness is usually detectable. Myoclonic jerks may be rapidly repetitive or relatively isolated.

c. Tonic Seizures: A tonic seizure consists of a sudden increase in muscle tone in the axial or extremity muscles, or both, producing a number of characteristic postures. A tonic seizure is a rigid, violent muscular contraction, fixing the limbs in some strained position. There is usually deviation of the eyes and of the head toward one side and this may amount to rotation involving the whole body. Consciousness is usually lost partially or completely. Prominent autonomic phenomena occur.
Tonic seizures are relatively rare and usually begin between 1 to 7 years of age.

d. **Atonic Seizures:** Atonic seizures consist of sudden loss of muscle tone. The loss of muscle tone may be confined to a group of muscles, such as the neck, resulting in a head drop. Alternatively, atonic seizures may involve all trunk muscles leading to a fall to the ground. Atonic seizures are known as drop attacks. If consciousness is lost, it is extremely brief.

e. **Clonic Seizures:** The clonic seizures begin with loss or impairment of consciousness associated with sudden hypotonia or a brief generalized tonic spasm. This is followed by 1 minute to several minutes of bilateral jerks, which are often asymmetric and may appear predominantly in one limb. During the attack, the amplitude, frequency and spatial distribution of these jerks may vary greatly from moment to moment. Clonic seizures occur almost exclusively in early childhood.

f. **Tonic-Clonic Seizure:** Tonic Clonic Seizures are also called grand mal seizures. Most frequently encountered of the generalized seizures are the generalized tonic-clonic seizures. In these seizures, there is a sudden loss of consciousness and a sudden sharp tonic contraction of muscles and the person falls to the ground in the tonic stage, occasionally injuring himself or herself in falling, the tongue may be bitten and urine may be passed involuntarily. This tonic stage then gives way to clonic convulsive movements by bilateral rhythmic jerks lasting for a variable period of time. The patient awakens by passing through stages of coma, confusional state, and drowsiness.

3. **Unclassified Epileptic Seizures:** These include all seizures that cannot be classified because of inadequate or incomplete data, and some defy classification in hitherto described categories. This includes some neonatal seizures, for example, rhythmic eye movements, chewing and swimming movements.

4. **Addendum (think to be added):** Prolonged or repetitive seizures that are called *status epilepticus*. The term status epilepticus is used whenever a
 seizure persists for sufficient length of time or repeated frequently enough that recovery between attacks does not occur. Repeated epileptic seizures occur under a variety of circumstances (i) as fortuitous attacks that can take place unexpectedly and without any apparent provocation, (ii) as cyclic attacks at more or less regular interval, for example, in relation to the menstrual cycle or to the sleep waking cycle, and (iii) as attacks provoked by nonsensory factors (fatigue, alcohol, emotion etc) and sensory factors, that are referred to as reflex seizures.

Classification of Epilepsies and Epileptic Syndromes:

ILAE made its first attempt to organize a coherent classification in 1985 (ILAE, 1985). The International Classification of Epilepsies and Epileptic Syndromes were again revised in 1989 and this is the classification in current use (ILAE, 1989).

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<thead>
<tr>
<th>International Classification of Epilepsies and Epileptic Syndromes</th>
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<tbody>
<tr>
<td><strong>1. Localization-related (focal, local, partial)</strong></td>
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<tr>
<td><strong>1.1 Idiopathic (with age-related onset)</strong></td>
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<td>- At present, the following syndromes are established, but more may be identified in the future:</td>
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<tr>
<td>- Benign childhood epilepsy with centrotemporal spikes</td>
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<td>- Childhood epilepsy with occipital paroxysms</td>
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<td>- Primary reading epilepsy</td>
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<td><strong>1.2 Symptomatic</strong></td>
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<td>- Chronic progressive epilepsia partialis continua of childhood</td>
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<td><strong>1.3 Cryptogenic (presumed to be symptomatic but with unknown etiology)</strong></td>
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<tr>
<td>- Anatomic localization</td>
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<tr>
<td>- Temporal lobe epilepsies</td>
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<td>- Frontal lobe epilepsies</td>
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- Parietal lobe epilepsies
- Occipital lobe epilepsies
- Bi- and multilobar epilepsies
- Etiology (in symptomatic epilepsies)
- Specific modes of precipitation

2. **Generalized**

2.1 Idiopathic (with age-related onset, in order of age)

- Benign neonatal familial convulsions
- Benign neonatal convulsions
- Benign myoclonic epilepsy in infancy
- Childhood absence epilepsy (pyknolepsy)
- Juvenile absence epilepsy
- Juvenile myoclonic epilepsy (impulsive petit mal)
- Epilepsy with grand mal (GTC) seizures on awaking
- Other idiopathic generalized epilepsies not defined above
- Epilepsies with seizures precipitated by specific modes of activation

2.2 Cryptogenic or symptomatic (in order of age)

- West syndrome (infantile spasms, Blitz-Nick-Salaam-Krämpfe)
- Lennox-Gastaut syndrome
- Epilepsy with myoclonic-astatic seizures
- Epilepsy with myoclonic absences

2.3 Symptomatic

2.3.1 Nonspecific etiology

- Early myoclonic encephalopathy
- Early infantile epileptic encephalopathy with suppression-burst
- Other symptomatic generalized epilepsies not defined above
2.3.2 Specific syndromes

3. Epilepsies and syndromes undetermined as to whether focal or generalized

3.1 With both generalized and focal seizures
- Neonatal seizures
- Severe myoclonic epilepsy in infancy
- Epilepsy with continuous spike-waves during sleep
- Acquired epileptic aphasia (Landau-Kleffner syndrome)
- Other undetermined epilepsies not defined above

3.2 Without unequivocal generalized or focal features (e.g., many cases of sleep-grand mal)

4. Special syndromes

4.1 Situation-related seizures (Gelegenheitsanfälle)
- Febrile convulsions
- Isolated seizures or isolated status epilepticus
- Seizures due to acute metabolic or toxic factors such as alcohol, drugs, eclampsia, nonketotic hyperglycemia, and so on


In the 1989 proposal for classification of the epilepsies, two key axes were identified, one for generalized versus localization related conditions; and second for etiology that includes idiopathic, symptomatic and cryptogenic (ILAE, 1989). Currently these are well accepted terms for organizing syndromes and epilepsy.

Generalized versus localization related condition: The international classifications of epilepsies begin by dividing epilepsies according to overall
seizure types. Generalized epilepsies involve seizures with initial activation of neurons in both cerebral hemispheres are involved simultaneously and symmetrically during the actual seizures whereas focal epilepsies involve seizures with initial activation of a group of neurons within one hemisphere, arise from a more discrete, lateralized region, usually in the cortex, regardless of later spread. Localization related (Focal, Local or Partial) and generalized epilepsy can be divided according to etiology as:

*Idiopathic, Symptomatic and Cryptogenic: Idiopathic* epilepsies are generally benign in the sense that they are not associated with brain lesions, neurologic abnormalities other than seizures, or mental impairment, and that they tend to be self-limited or respond readily to antiepileptic drugs. Genetic factors are important, and manifestations are typically age related. *Symptomatic* epilepsies are those in which seizures are the consequence of an identifiable lesion or other demonstrable physical or metabolic etiology. When epilepsies are presumably symptomatic but currently of unknown specific etiology, they have been termed as *cryptogenic* (ILAE, 1989), a term also used in epidemiologic studies to mean unknown as to whether idiopathic or symptomatic (ILAE, 1993). Because of its ambiguity, cryptogenic is a term that should be replaced by the more accurate probably symptomatic (ILAE, 2001). It is used in cases in which the epilepsy did not clearly fit the criteria of a defined idiopathic condition and there is no evidence by examination or history of a remote symptomatic cause. The use of the term reflects an absence of knowledge and relative uncertainty regarding the nature of the epilepsy. Symptomatic is increasingly difficult to justify. In reality, all epilepsy is symptomatic of something and idiopathic epilepsies have cause and consequences too. Mounting evidence demonstrates that idiopathic epilepsy as well as epilepsies that are considered cryptogenic are associated with a number of other neurological disorders and these disorders precede the onset of seizures.

Epilepsies and Syndromes undermined as to whether Focal or Generalized: The 1989 classification of the epilepsies includes a category
for “undetermined.” This contains two distinct subcategories. The first is for syndromes in which both focal and generalized features occur. Some of the better-known disorders in this category are Landau-Kleffner syndrome and Dravet syndrome (Roger et al., 2002). Use of the second subcategory under the label of “undetermined” reflects a diagnosis of ignorance and, by and large, is intended for unclassified forms of epilepsy. It is important, however, to distinguish between someone who has been appropriately and competently evaluated and for whom it is unclear whether the seizures and underlying epilepsy represent a generalized versus a more localized process and the case in which information is simply missing. For example, someone who has one or more generalized tonic–clonic seizures without evidence of partial onset and normal EEG, MRI study can reasonably be diagnosed as having an undetermined form of epilepsy without clear generalized or focal signs.

Special Syndromes: The ILAE task Force has defined an epilepsy syndrome as a complex of signs and symptoms that define a unique epilepsy condition. This must involve more than just the seizure type. Thus, frontal lobe seizures per se, for instance, do not constitute a syndrome. An epilepsy syndrome must be distinguished from an epilepsy disease that can be defined as a pathologic condition with a single specific well-defined etiology, for example, Progressive Myoclonus Epilepsy is a syndrome, but Unverricht-Lundborg is a disease (Engel, 2001). Seizures precipitated by special circumstances such as fever (fever convulsions) or seizures due to acute metabolic or toxic factors such as alcohol, drug or hyperglycemia etc can be explained as special syndromes.

Epidemiology

There are immense difficulties in establishing precise epidemiological statistics for a heterogeneous condition like epilepsy (Sander and Hart, 1999). The diagnosis of epilepsy is essentially clinical, based on an eyewitness account of the seizure. Neurological examination and investigations may be normal between attacks. Sometimes patients may not
be aware of the nature of attacks, seizures occurring at night may go unnoticed and hence, may not be reported.

Patients with infrequent or mild seizures may not receive ongoing medical care and so may be missed in epidemiological surveys. Patients may also tend to deny a history of epilepsy in view of the stigma attached to it or feign epilepsy (Sridharan, et al., 1986). Moreover, the lack of access to EEG or neuroimaging facilities in most community-based studies may lead to inaccurate diagnosis of epilepsy, its type or etiology. The diagnostic criteria for epilepsy may not be uniform in different studies. Persons with acute symptomatic (provoked) seizures, single unprovoked seizure, febrile seizures and inactive epilepsy may not be excluded in some studies, resulting in higher incidence/prevalence (Bharucha, 2000).

In 1993, the International Commission of Epidemiology and Prognosis of the International League Against Epilepsy (ILAE) published guidelines for epidemiologic studies (ILAE, 1993). These are particularly useful for field studies in developing countries, where facilities for investigation are unavailable. According to these guidelines, a person with active epilepsy is one who has had at least one epileptic seizure in the previous five years, regardless of antiepileptic drug treatment. Epilepsy in remission (no seizures for five or more years with or without treatment) should also be distinguished. Moreover, in community surveys, it may be difficult to exclude pseudoseizures and syncope. It is estimated that 10–20% of patients evaluated at centres for epilepsy surgery do not have epilepsy.

Epilepsy affects 20 to 40 million people worldwide and has prevalence of at least 63% and on annual incidence of approximately 0.50% (Mario and Ashla, 2005). Nearly 90% of epilepsy occurs in developing countries (WHO, 1999). Prevalence, cumulative incidence, and incidence are the measures used to describe the epidemiology. Prevalence is defined as the proportion of a specified population with the disease at a specified time (point prevalence) or during a defined time interval (period prevalence). Prevalence represents a complex interaction between several factors such as
incidence, death or remission of illness. Prevalence studies do not give much information regarding aetiology or prognosis, and are primarily helpful for health planning purposes. The prevalence of active epilepsy remains within the range of 4 to 10 per 1,000 populations throughout the world (ILAE, 1997; Radhakrishnan, 2000; Sridharan and Murthy, 1999). A higher prevalence of 35.1 per 1,000 has been reported from sub-Saharan African countries (Debrock et al., 2000) and the prevalence of 49 per 1,000 in Grand Bassa County in Liberia (Goudsmit et al., 1983). A rate of 20 per 1,000 was found among the Wapogoro in Tanzania (Jilek and Jilek, 1970), 50 in South and Central America, a rate of 57 per 1,000 has been found among the Guaymi Indians of Panama (Garcia et al., 1990). A large number of studies in India and China suggest a prevalence of active epilepsy between 4 and 6 per 1,000, similar to that in developed countries (Bharucha et al., 2005; Koul et al., 1988; Sridharan and Murthy, 1999; Wang et al., 2003). In areas with a high prevalence, there is often a strong family history of both epilepsy and consanguinity. Incidence is defined as the rate of occurrence of new cases in per 100,000 populations per unit time, usually one year. Incidence cohorts provide more valuable information than prevalence cohorts, since they include mild and severe persons with epilepsy; persons with epilepsy are identified at an earlier time in the course of their illness and hence more likely to provide reliable information regarding potential antecedents. However, incidence studies require ongoing surveillance of a population of adequate size over a sufficiently long time and hence are very expensive. In developed countries apart from recurrent unprovoked seizures (range from 28.9 to 53.1 per 1,00,000 person in a year), if single unprovoked seizure are included, that show higher figures upto 70 per 1,00,000 person in a year, are obtained. The few available studies in developing countries give a range from 35 to 190 per 1,00,000 persons in a year. The higher incidence in developing countries may be a consequence of the fact that populations in the developing world are younger and have poorer medical facilities, poorer general health, and a lower standard of living. Specifically, there are more
infections of the central nervous system (CNS), tuberculosis (TB), and human immunodeficiency virus (HIV), and head injuries. Incidence throughout the world is slightly higher in males than females, but many studies have found no gender difference or found a slight difference. The Minnesota study showed stable rates of overall incidence from 1935 to 1979. Differences in incidence rates in males and females are not statistically significant. There is no evidence of racial predilection in incidence, though the incidence is significantly higher in the lower socioeconomic classes. Cumulative incidence is the proportion of the population developing epilepsy over a given time. The risk of epilepsy from birth through age 20 is about 1% and reaches 3% by age 75. Overall, about 3% of population can be expected to have epilepsy at some time during their lives (Annegers, 2001). Unlike prevalence, cumulative incidence progressively increases with age because epilepsy may begin at any age. Studies in Ecuador, Pakistan, and Turkey showed the prevalence of epilepsy to be higher in rural areas, but the reverse was shown in the meta-analysis of the Indian studies (Aziz et al., 1994; Aziz et al., 1997; Placencia et al., 1992; and Sridharan and Murthy, 1999). In India, poverty is greater in rural areas where it is more difficult to conceal epilepsy. The whole relationship of socioeconomic factors to epilepsy needs further exploration. Prevalence, incidence, and cumulative incidence studies are useful for health service providers, especially in developing countries with few neurologists and 80% of the burden of epilepsy. Incidence studies give clues to risk factors and information about prognosis, but there are few studies from developing countries because they are difficult to carry out.

**Prognosis and Mortality**

For the majority of people with epilepsy, the condition is treatable and the term *prognosis* generally refers to the probability of attaining seizure freedom after treatment has been started, during treatment, and after drug withdrawal. The prognosis of epilepsy is defined by Hauser and Hesdorffer (1990) as the risk of recurrence following a first seizure or more
seizures; the probability of remission, both spontaneous and with treatment; the risk of relapse following drug withdrawal; and the mortality of epilepsy. In developed countries, prognosis for full seizure control is very good. More than 70% of patients achieve long-term remission within 5 years of diagnosis (Hauser et al., 1990; Lindsten et al., 2001; and Sillanpaa et al., 1998). In developing countries, after a first unprovoked seizure, the risk of recurrence is 33% to 37%, similar to that from developed countries (Carpio et al., 1999; Daoud and Adetunji, 1994; Kochen and Melcon, 2005; and Scotoni et al., 2004). Studies from developing countries show that patients with epilepsy secondary to underlying structural causes and with an abnormal EEG have the worst prognosis (Carpio et al., 1999; Kochen and Melcon, 2005; and Scotoni et al., 2004). The type of seizure and drug treatment used seems not to affect prognosis. Multivariate analysis showed no significant differences in recurrence risk due to sex, age, and family history of epilepsy, EEG results, or the type of seizure. The mortality rate associated with epilepsy is two to three times that of the general population (Cockerell et al., 1994; Hauser et al., 1990; Lhatoo et al., 2001; Lindsten et al., 2001; 1998; Loiseau et al.) and life expectancy in some of these patients is reduced (Gaitatzis et al., 2004). In developing countries, it is almost impossible to ascertain the number of deaths due to epilepsy because incidence studies are difficult, death certificates are unreliable, autopsies are not easy to obtain, and the cause of death is not usually known with certainty (Bharucha et al., 2005; and Carpio et al., 2005). Two Indian population studies of mortality among the parsis in Bombay and a semiurban community in vasai suggest that the poorer and rural population with epilepsy has a much higher mortality rate than the urban one. Mortality of epilepsy in developing countries is generally higher than the mortality reported in developed countries. Developing countries need more incidence studies on large cohorts with recent onset epilepsy to determine mortality.

**Diagnosis**

Diagnostic tasks in epilepsy management include establishing a seizure diagnosis, an etiologic diagnosis, and identification of precipitating factors. This is accomplished by a combination of history-taking, physical
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examination, electroencephalography (EEG), and laboratory examination. Seizure Diagnosis, the first step is establishing definitely whether or not the person has epilepsy because an erroneously diagnosis may subject to many inconveniences including medication side effects, expensive laboratory testing, loss of driver’s license, and possible loss of employment. Correctly diagnosed patients can be given correct therapy. The diagnosis of seizure types should be made according to the International League Against Epileptic Seizures. Etiologic Diagnosis, a seizure can be a symptom of old or recent cerebral trauma, a brain tumor, meningitis, metabolic disturbance, drug intoxication and many other disease processes. It is imperative that the underlying cause be identified and treated to facilitate seizure control. Precipitating Factor, anxiety, sleep deprivation and alcohol withdrawal in a given individual must be managed to reduce seizure frequency and the patient’s need for medication. History should include exact details of events before, during, and after the seizure, obtained from patients and observers. Questions regarding family history, head trauma, birth complication, and alcohol and drug abuse should be included. Electroencephalography (EEG) and laboratory examination are helpful diagnostic tool in the investigation of a seizure disorder as it conforms the presence of abnormal electrical activity that gives information regarding the seizure types and the location of the seizure focus. EEG recording in waking and sleep states, MRI, CT scan, toxic screen in case of alcohol or drug abuse and a lumber puncture can be performed if infection or malignancy is suspected. Magnetic Resonance Imaging (MRI) has increasing capabilities for identifying small epileptogenic lesions such as migration defects in patients previously believed to have cryptogenic epileptic disorders (Berkovich et al., 1998), functional Magnetic Resonance Imaging (fMRI) which has the spatial and temporal resolution to produce dynamic three-dimensional cerebral anatomic maps displaying ictal onset and propagation patterns(Jackson et al., 1994), PET and Single Photon Emission Computed Tomography (SPECT), which utilize a variety of tracers to localize biochemical abnormalities in the brain.
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and characterize presynaptic and postsynaptic neurotransmitter functions (Mazzotta and Gilman, 1992), Magnetic Resonance Spectroscopy (MRS), which can measure localized biochemical changes in the brain related to epilepsy (Petroff et al., 1995), and Magnetoencephalography (MEG) in association with EEG can reliably localize current sources of interictal and ictal epileptiform events occurring deep within the brain (Ebersole et al., 1995). These new imaging techniques may prove of extraordinary value in the analysis of epileptogenesis in human patients.

Differential Diagnosis

The common differential diagnostic problems associated with epilepsy at various ages are following:

(1) Epilepsy Vs Migraine: Some authors regard migraine as an important differential in the diagnosis of epilepsy (Gibbs and Appleton, 1992). Absent or less severe headache, bilateral and nonpulsatile, paroxysmal activity on interictal and ictal EEG and persistant slowing on interictal EEG are features suggest epilepsy but severe unilateral, pulsatile headache, nausea and vomiting, photophobia, family history of migraine and EEG slowing only during or immediately after attack are features suggest migraine.

(2) Epilepsy Vs Syncope: Syncope (fainting or blackout) is defined as a sudden decrease in cerebral perfusion of oxygenated blood, either from a reduction in cerebral blood flow itself or from a drop in the oxygen content or a combination of these (Stephenson, 2001; 1990). Syncope is the sudden loss of muscle tone, collapse of posture and loss of consciousness associated with a drop in systematic blood pressure. The patient recovers from a faint when the blood pressure is re-established.

(3) Epilepsy Vs Episodic Dyscontrol (rage attack): The episodic dyscontrol syndrome is characterized by recurrent attacks of controllable rage, aggression (verbal or physical), or both. Attacks typically have an identifiable precipitant. During the attacks the patients often appear
temporarily psychotic and after the attack, amnesia, fatigue and occasionally remorse may occur. Finally, for episodic dyscontrol a syndrome and real partial seizure to exist in the same patient is not uncommon because both are consequences of brain damage, especially in case of head injury.

(4) Epilepsy Vs Transient Ischemic Attacks: Onset of seizures at a younger age, presence of other signs or symptoms of partial seizures, alteration of consciousness, postictal confusion, duration less than 3 minutes, and focal spikes suggest epilepsy but duration longer than 3 minutes, monocular blindness, vertebral artery distribution deficit, decreased pulse and normal interictal EEG suggest diagnosis of a transient ischemic attacks.

(5) Epilepsy Vs Psychogenic Seizures: Psychogenic seizures are episodes of altered movement, emotion, sensation or experience that are similar to those caused by epilepsy but have purely emotional cause. Lesser (1996) has made a complete review of psychogenic seizures. Psychogenic seizures are gradual buildup and prolonged resolution. Tongue biting, self-injury, incontinence, postictal confusion rarely present and a secondary gain often identifiable.

(6) Epilepsy Vs Sleep Disturbances (Night terrors, sleepwalking): Onset between 4 and 12 years of age, typical spell beginning 1 to 2 hours after falling asleep, terrified scream, dilated pupils, fearful for upto 15 minutes before falling back to sleep without difficulty and normal EEG suggest a diagnosis of night terrors, but typical spell beginning 1 to 2 hours after falling asleep, walking about in a trance and carrying out purposeful activity such as dressing, opening doors and eating, and normal EEG suggest a diagnosis of sleepwalking. Automatic behaviour and amnesia are common features of epilepsy of sleep disturbance. Apart from these, transient global amnesia (prolonged spell for hours with normal behaviour except amnesia), panic attack (often with environmental trigger, palpitation, >5 minutes in duration), drop attacks
and hypoglycemia should be considered in suggesting a differential diagnosis of epilepsy.

**Psychosocial Outcome**

Epilepsy has many non-medical effects on the people with epilepsy, their family and community. Epilepsy can have far reaching psychological and social ramifications; and for some individuals these can be more debilitating than the seizures (Thompson et al., 2003; and Hermann and Jacoby, 2009). Published literature covers varieties of areas including psychiatric problems (e.g. mood disorders, anxiety, psychotic disorders); feeling of shame, fear, and worry; low self-esteem; problems related to education, employment, dating, marriage, child-bearing, poor quality of life; and stigma (Lai, 2007). Awareness of the psychosocial problems which may arise is essential for professionals working with people with epilepsy and their families. Such difficulties can have a profound impact on a person’s mental health and also upon seizure control. Problems in living with epilepsy related to social adjustment (e.g., driving and lack of employment opportunities) are also frequently reported in adults with epilepsy (Hayden et al., 1992; Jacoby, 1992). Long-distance travel can also be difficult for adults with epilepsy, especially for those with severe or frequent seizures. Social problems are important because they reduce quality of life and contribute to mental health problems such as depression, anxiety, and other psychopathologies in persons with epilepsy (Hermann, 1991)

(1). **Education:** Epilepsy has been found to negatively affect school attendance and academic performance (Ibekwe et al., 2007; and Adewuya et al., 2006). The adolescents with epilepsy are highly stigmatized and discriminated against in schools by fellow students (Matuja and Rwiza, 1994) and the teachers (Kabir et al., 2005 and Ojinnaka, 2002). Young people with epilepsy appear to do less well than their peers in terms of educational qualifications (Shackleton et al., 2003). Most attention has been focused upon seizure-related factors, with poor educational prognosis being associated with early age of
onset and a long seizure history, especially where seizure control has proved problematic. Other psychosocial variables have been implicated including teachers’ and parental expectations, misconceptions about epilepsy, absence from school, low self-esteem, and anxiety due to stresses at home.

(2) Employment: People with epilepsy are more likely to be unemployed than people in the general population with unemployment rate ranging from 24% to 36% (Sander and Sillanpaa, 1997). Existing evidence suggests that people with epilepsy experience high levels of unemployment and underemployment. Early employment studies showed that the situation for people with epilepsy was discouraging. Jacoby (1992) found that 50% of people with epilepsy were unemployed, and Scambler and Hopkins (1980) reported that 42% were unemployed. Young people with epilepsy especially are at a disadvantage when it comes to obtaining and retaining employment and may need special assistance and training to enable them to deal with difficulties they are likely to encounter. Even when the epilepsy is controlled, many find that their epilepsy is a barrier to employment. Employment problems are not simply due to seizures; rather they may be a result of personal and social factors, including discrimination, stigma, passive coping styles and low self-efficacy interacting with one another in a complex manner (Smeets et al., 2007; Clarke et al., 2006). Employed people with epilepsy experience fewer psychosocial problems than unemployed people with epilepsy (Jacoby and Baker, 2008).

(3) Family Life: People with epilepsy do not live in a vacuum; the attitudes and experiences of family members greatly influence how someone copes. Research studies have found lower parent-child relationship quality, higher rates of depression in mothers and problems with family functioning in association with epilepsy (Rodenburg et al., 2005). Focus by parents on the child with epilepsy can result in poor relationships
between the child with epilepsy and siblings and psychological difficulties among siblings. It can result in the people with epilepsy growing up to make a poor parent themselves (Devinsky, 2001). Parents may be overprotective through fear of injury or death. Families may harbour misconceptions about epilepsy and thus become socially isolated for fear of adverse public reactions. Emotional support is particularly important within the first few months following a diagnosis (Shore et al., 2009). As children grow older, parental vigilance may intensify as a watch is kept for any behaviour or physical sign that might herald the onset of epilepsy in their child. Older children may behave over protectively towards the parent with epilepsy and may exhibit worrying behaviours, such as non-attendance of school.

(4). Social Network: In a survey of young people with poorly controlled seizures were found with lack of social contacts, particularly friendships, and activities outside the parental home or outside parental supervision. Social isolation in people with poorly controlled seizures is, therefore, a major cause of concern. Many factors may underlie social isolation including anxiety, parental over-protectiveness, lack of employment, and a dearth of activities outside the home. Social isolation and poor social adaptation can result from perceived stigma or over-dependency caused by parental overprotection. Stigma can also have negative impact on the development of friendships. Factors predicting greatest risk of feeling stigmatized included having a first seizure before age 50 years, not being married or cohabiting, not being in paid employment, and living on a limited income. People with epilepsy may avoid socializing for fear of having seizures in front of others. The people with epilepsy also often fears embarrassment by a seizure causing reluctance to engage in social interaction, with concomitantly low self-esteem and academic under-achievement. These can result in a shrunken support network, fewer friends, a lower likelihood of marriage and greater likelihood of anti-social behavior
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(buchanan, 2002). a social withdrawal syndrome with low rates of employment and marriage may result from a combination of low educational attainment and self-esteem, legislative restrictions, perceived stigma and actual discrimination, and cognitive and behavioral deficits (impaired memory, hyposexuality).

(5). emotional adjustment: people react in different ways to the diagnosis of epilepsy. often there is a feeling of shock. others may react to the loss in independence and the restrictions imposed on their life such as having to surrender a driving licence. living with epilepsy means coping with an uncertain prognosis regarding seizure control. epilepsy carries increased risks of mortality and morbidity. diagnosis and prognosis aside, individuals have to cope with ongoing seizures. for some, these may be rare, short-lived episodes; but for others, epileptic attacks may involve bizarre behaviors, distorted awareness and perception, and embarrassing aspects such as incontinence. other emotional states which may recur include anxiety arising from the unpredictability of seizures and feelings of lack of control and helplessness. public misunderstandings and stigma cause additional stress. individuals also have to adjust to long-term drug treatment and accompanying side effects which may include weight gain, acne, unwanted facial hair, irritability and cognitive disturbances. these aspects may become more stressful at certain times of development with adolescence being a vulnerable period. many with poorly controlled seizures have to endure successive treatment failures and the accompanying emotional highs and lows as hopes are raised with the introduction of a new drug only to be dashed when seizures return. potentially more damaging psychologically is the failure of surgical treatment, particularly when this follows several years of freedom from seizures. anxiety and depression are over-represented in individuals with epilepsy but under-treated (ekinci et al., 2009). a recent pilot study combining cognitive therapy with mindfulness utilizing the
telephone and internet has shown some promise regarding mood improvements and in promoting the development of social support networks (Reisenger et al., 2010).

(6) Marriage and Sexual Relationship: Lower rates of marriage and fertility have also been reported in people with epilepsy when compared with the general population (Schupf and Ottman, 1994). Satisfactory relations with the other sex require self-esteem. Low self-esteem in a people with epilepsy can result in failure to establish good sexual relationships. Brain damage and/or antiepileptic drugs may also result in anhedonia. Head injury may result in reduced libido and erectile dysfunction. Inadequate sexual functioning may result in depression, marital distress or self-aversion (Gumnit, 1997). For many (young) people, marriage is one of the main goals in life. For a person with epilepsy, however, the chances of getting married can be particularly slim in the Third World. In some countries, a woman with epilepsy has virtually no chance of getting married at all, or when she does, epilepsy is a reason for an immediate divorce. In India, although the law forbidding people with epilepsy from getting married was repealed recently, such people still stand no chance of an arranged marriage, which is customary in that part of the world. Fundamentalist groups in many religious cultures, following ancient laws, still believe that epilepsy is inheritable, and of course these laws were originally intended to prevent the disease from spreading.

(7) Driving: For most people, driving is a surrogate for independence, and freedom to drive is therefore a major contributor to quality of life (Gilliam et al., 1997). Thus, it is not surprising that issues related to driving are among the most difficult routinely encountered in the care of patients, especially those with mild epilepsy. Recommendations must balance a physician's dual obligations to the patient and also to the society. Fortunately, fatal crashes due to seizures are rare. Between 1995 and 1997, only 0.2% of all driving fatalities in the United States
were attributed to seizures in mortality reports. Although patients with epilepsy had a greater rate of fatal crashes than those with other medical conditions, occurrence of fatal seizure-related crashes was 2.6 times lower than in the general population (Sheth et al., 2004).

(8). Sports, Exercise, Travel, and Alcohol: As with driving, it is hard to make generalizations regarding patients' involvement in sports and other activities. Recommendations have to be individualized, bearing in mind the frequency and type of seizures, presence of a reliable aura, the importance of sports to the patient, and the level of supervision required or available. Although physicians are appropriately concerned about their patients' safety, as are parents of children with epilepsy, it is also important that persons with epilepsy lead as normal lives as possible. Thus, individual decisions need to strike a balance between maximizing safety and minimizing the stigma that inevitably develops as a consequence of “being different” when activities are restricted. Nakken (1999) reported that over half of 204 patients with epilepsy had never reported having a seizure during exercise. About 10% of the patients, predominantly those with symptomatic localization-related epilepsy indicated that they often had seizures related to exercise. In general, sports and exercise are safe and healthy for persons with epilepsy, and they can generally be encouraged. Exceptions include such high-risk sports as scuba diving and sky diving, which should be avoided. Most fatal accidents in epilepsy occur in the water (60%). However, very few people with epilepsy drown during swimming. Most accidents occur in the bath, while fishing, or from falling into water. As noted, research shows that very few seizures occur during active sports. They do occur, however, in the rest period afterward (e.g., when resting alongside the swimming pool). Swimming must be supervised closely but can be permitted in most patients whose epilepsy is controlled. Having epilepsy does not usually stop people from being able to travel by air. However, some people find that their seizures are triggered by
extreme tiredness such as jetlag, excitement or anxiety, all of which can be caused by travelling or flying. Travel and leisure activities rarely need to be restricted, but it is helpful to advise patients about minimizing sleep deprivation during travel, emphasize the importance of medication compliance, and discuss plans for emergency care while away, should that be. Similarly, many patients consider it important to have a glass of wine at a dinner party or celebration. This poses only a minimal risk if seizures are controlled. In general, patients with epilepsy can tolerate alcohol in small amounts (e.g., one to two drinks per occasion or no more than three to six drinks per week). However, anyone with a history of alcohol or substance abuse, alcohol-related seizures, or noncompliance with AEDs should abstain completely. Adolescents and young adults who often find it difficult to control the amount of alcohol they consume, should also probably abstain but are not likely to do so (Gordon and Devinsky, 2001). Patients who drink moderately (three to four drinks per occasion) or heavy amounts (four or more drinks per occasion) should be warned that they are at increased risk of having seizures, with the greatest risk occurring 7 to 48 hours after the last drink (Hauser et al., 1988; and Ng et al., 1988). It follows that driving should be avoided the day or two after significant alcohol use.

(8). Impact on Women: Epilepsy causes unique problems for women. Seizure frequency and severity can be exacerbated by menstrual hormonal changes. Women with epilepsy often experience anxieties concerning children. Doubts about seizures being triggered by labour, their ability to care for their child, the possibility of inheritance of epilepsy and birth defects are compounded by fears about antiepileptic drug side effects and the mother’s ability to be a good role model during child-rearing (Gumnit, 1997).

(9). Economic Impact: Epilepsy has significant economic implication in terms of health care needs, premature death and loss of productivity.
Economic considerations are important when considering epilepsy treatments. Patients who are resistant to medical therapy suffer most from the detrimental economic and social impacts of the condition. Whereas the most refractory patients comprise only a small proportion of those with epilepsy, they account for a large share of the total costs imposed by epilepsy. One study estimated that the 15% of patients who are most refractory account for at least 50% of the total costs of the illness (Begley et al., 2000). Cost correlated with the severity of the illness, and that intractable patients cost eight times more that those with controlled epilepsy (Jensen, 1976). Indirect expenses, which account for up to 75% of total costs, include lost productivity from unemployment, underemployment, or lost work time; excess mortality; transfer payments (e.g., disability pension); and lost work by relatives or friends to care for the ill person (Begley et al., 2000; Jayakar et al., 1994). An Indian study calculates that the total cost per epilepsy case was US $344 per year (or 88% of the average income per capita). The total cost for an estimated five million cases in India was equivalent to 0.5% of gross national income.

Psychopathology in Epilepsy

The links between epilepsy and psychiatry have a long and respectable history, but for reasons best deliberated on by historians, the growing gap between neurology and psychiatry in the 20th century led to a considerable divergence of opinions as to the relationships between psychiatric disorders and epilepsy. By the mid of the 20th century, it was a strongly held view that people with epilepsy, if they had psychiatric difficulties, had them because of secondary factors. These included not only the stigmatization of having a terrible condition such as epilepsy, with such a poor quality of life and considerable social disadvantages, but also drug factors or cerebral trauma from head injuries following seizures. People with epilepsy can have a straightforward psychiatric problems. A number of these are classifiable in terms of standardized diagnostic schedules (for
example, DSM-VI or ICD-10) and, when present in people with epilepsy, should receive the same amount of attention for management as they would if the patient did not have epilepsy. However, the contention of the last 30 or so years has been that there are some psychiatric problems that are more intimately imbedded within the context of the underlying neurobiologic process of the epilepsy. Most neurologists now accept that psychopathology is an integral part of some epilepsy syndromes, whether it be memory disturbances and other cognitive difficulties or more florid psychopathologies as one may see, for example, in the psychoses. Epidemiologic studies found a significantly higher rate of psychiatric disorders in children with neurologic disorders compared with children chronic illnesses that do not involve the brain (Davies et al., 2003 and Rutter et al., 1970). Furthermore, more specific seizure-related factors, such as type of seizure disorder, seizure control, the age of onset of seizures, and the number and type of antiepileptic drugs (AEDs) can impact the behavior of child with epilepsy. In the developing child, chronic recurrent brief episodes of brain dysfunction might impact brain development and, therefore, the development of behavior, cognition, and language. The maturation of these three areas of functioning has important implications for the psychiatric and social functioning of the child. Several studies have established that there is a higher incidence of epilepsy (based on clinical or EEG evidences) in psychiatric hospitals (Court, 1965), in subnormality hospitals (Eyman et al., 1970), and in prison populations (Gunn and Fenton, 1969).

Epileptic activity within the brain has an effect on the behavior, mood, and cognitive functions of the patient; his behavior and mood may be affected by the attitudes of others to his disabilities; and the patient's social and psychological adaptations may modify his epileptic experience. Behavior of the person with epilepsy cannot be considered separately in terms of pathophysiology, psychology or sociology but rather as the resultant of the interaction of all these influences. Psychiatric or behavioral symptoms must be evaluated in patients with epilepsy in relation to ictus
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(including area and seizure proper), postical period, and interictal phase. Psychiatric symptoms may arise during any of these and diagnosis and treatment will depend on their relations to seizures. The scientific advancement in the 20th century have resulted in new diagnostic and treatment strategies for epilepsy and in studies of both ‘interictal’ and ‘ictal’ behaviours. Accelerated development of psychiatric methodologies, psychotropic medications, and neuroimaging techniques during the 1990s have resulted in identification of high rates of psychopathology and poor quality of life in adults and in children with epilepsy. As found in other chronic illnesses, coping with epilepsy involves a wide range of intrapersonal and interpersonal psychosocial factors that affect emotional and intellectual functioning (Caplan, 1998). Epidemiological studies from communities, psychiatric hospital and epilepsy clinics report a 20 to 60 parent prevalence of psychiatric problems among epilepsy patients. Epilepsy patients are prone to psychosis, depressions, personality disorders, hypossexuality and other behavioral disorders. Many studies found a special relationship to psychopathology in patients whose seizure emanated from mediobasal temporal lesions. Among epileptic patients, much of the psychopathology results from electrophysiological, structural or chemical changes in the temporal limbic system and possible the frontal lobes.

In epilepsy most of the seizures appear to occur spontaneously, they may be provoked or the occurrence modulated by a variety of endogenous and environmental phenomena. Seizure precipitants are those circumstances that precede the onset of an epileptic attack and are considered by both patient and neurologist to be a possible explanation for why the seizure happened when it did, and not earlier or later.

These precipitants include both seizure-inducing and seizure-triggering factors. Knowledge of seizure precipitants has practical implications in patient’s treatment and counseling. Hermann et al (1990) found that psychopathology was associated with poor adjustment to epilepsy, elevated number of stressful life events during the past year,
financial stress, vocational problems, external locus of control with increased perceived stigma, and an earlier onset of epilepsy. Multiple regression analyses identified three independent predictors of psychopathology—an increased number of stressful life events in the past year, poor adjustment to epilepsy, and financial stress. Emotional disturbance may lead to sleep deprivation, noncompliance, excessive drinking, and even hyperventilation.

In another study Denio (1989) examined the psychological factors confronting psychiatric patients with epilepsy before the onset of epilepsy. Many patients have seizures only at night. Sleep deprivation is the second most often reported seizure precipitant in four studies (Bauer et al., 2000; Da Silva Sousa, 2005; Nakken et al., 2005; Tan et al., 2005) As sleep deprivation usually occurs during periods of over activity or tension often associated with the use of stimulants and over hydration, its role is not always clinically clear. Patients with idiopathic epilepsies frequently reported sleep deprivation as a significant precipitant (Frucht et al., 2000). Alcoholic beverages are a frequent cause of seizures in adult patients with epilepsy. Alcohol consumption was the fourth most frequent precipitant (5.7%) reported in one study (Nakken et al., 2005). Alcohol abuse is often associated with poor compliance and sleep deprivation. Seizures occur during the period of rapidly falling alcohol blood levels, especially when excessive alcohol intake is associated with insufficient sleep (Nakken et al., 2005).

There are few careful epidemiological studies of the psychopathology of patients with epilepsy. Pond and Bidwell (1959) in a general practice survey noted that 29% had psychological difficulties, 7% having been in a psychiatric hospital before or during the survey year. A temporal lobe group had a higher rate of hospitalizations to psychiatric hospital and higher rates of severe personality changes and psychosis. Gudmundson (1966) compared the prevalence rates for psychiatric illness in epilepsy with those without epilepsy and found 25% of the epileptics showed neurotic symptoms, 50%
had some type of abnormal personality, and 8% were psychotic. Again the frequency of psychopathology was much greater (50%) in those with temporal lobe epilepsy compared to those without (25%).

(1). Anxiety and Anxiety Related Disorders: Anxiety can be quite significant in the life of a person with epilepsy. Anxiety is related to epilepsy in more specific ways (Stagno, 2001). It can occur not only as a reaction to the diagnosis, but also as a symptom of the epilepsy, in some cases, as a side effect of seizure medicines. The knowledge that a seizure can occur at anytime and place without very much warning is a major point of anxiety for many people. It also can occur as a direct result of neurobiological factors like abnormal brain functions and seizures. Both psychological and biological components of anxiety are particularly apparent in people with seizures. Therefore, although the exact relationship between brain abnormalities and anxiety is extremely complex and not very well understood, it is clear that there is a relationship. Anxiety disorders are common in people with chronic medical disorders, including people with epilepsy (PWE). While population-based studies have suggested prevalence rates of 25%, equivalent to almost twice that of the general population, the actual incidence and prevalence rates of anxiety disorders in PWE is yet to be established (Gaitatzis et al., 2004; and Kessler et al., 1994). Various studies have estimated the prevalence of anxiety disorders to range from 10% and 25%, with the higher prevalence rates found among patients with intractable epilepsy. In a review, Torta and Keller (1999) found anxiety symptoms in up to 66% of patients with epilepsy. Using the Hospital Anxiety and Depression Scale in a study of 201 PWE, Cramer et al. (2005) found that 48% reported symptoms of anxiety; in 25% of these patients they were rated as mild, moderate in 16%, and severe in 7%. In this study, anxiety symptoms were more prevalent than depressive symptoms, which were reported in 38% of subjects. Symptoms of OCD have been found to be more frequent among PWE
than healthy controls in small case series (Monaco et al., 2005). Panic disorder is significantly more frequent among PWE than the general population. In a review of the literature, Beyenburg et al. (2005) estimated that PWE were six times more likely to suffer from PD than the general population, with point prevalence rates ranging between 5% and 30%, compared with 3.5% in the general population (Kessler et al., 1994). In PWE, agoraphobia or social phobias are among the more frequent types, resulting from fear of injury or social embarrassment should a seizure occur in public. Kanner et al. (2004) found postictal symptoms of agoraphobia in 29 patients out of 100 patients, 18 of these patients (62%) attributed these symptoms to the fear of seizure recurrence, but none of these patients experienced seizures in clusters to explain the agoraphobic symptoms. Nonetheless, none of these patients developed full-blown interictal agoraphobia.

(2). Depression and Mania: Depression is the most frequent psychiatric disturbances in adults with epilepsy. The prevalence of depression varies and may range from 7.5 to 34 percent of patients with epilepsy (Mario and Ashla, 2005). Psychological studies also suggest a greater incidence of ideational orientation, self-criticism, and depression among epilepsy patients with a left-hemisphere focus. Patients with complex partial seizures of temporal limbic origin have a higher incidence of depression than do patients with other types of seizure disorder. Some studies find depression in 25% to 80% of adults with epilepsy (Glosser et al., 2000; Paradiso et al., 2001; Perini et al., 1996; and Mendez et al., 1986) and 23% to 25% of children and adolescents with epilepsy (Caplan et al., 1998; Ott et al., 2000; Dunn et al., 1999 and Ettinger et al., 1998). The severity of depression is also related to poor vs good seizure control, seizure type and age range of patients. Thus biological factors clearly contribute to the depression of adults with epilepsy. Little mention is made in the literature of mania, hypomania or classic biopolar affective disorders in patients with
epilepsy and most authors describe these condition as rare. Not uncommonly, however, patients who appear classically manic may have a time-limited postictal psychiatric disturbance. Barczak et al (1988) reported three cases of mood disturbances following seizure that he labeled hypomania. In addition to elevated mood, these included hallucination or delusions. Their appearance postictally strongly suggests that the patients were experiencing a manic like mood disorders as a postictal psychosis. Mania may be seen less frequently than expected because a number of anticonvulsants such as carbamazepine, valproic acid and possibly phenytoin are effective in both short and long term treatment of biopolar mood disorders.

(3). Personality Disorders: At the turn of the 20th century, most of the lay and professional communities believed that people with epilepsy had pathologic personality traits and psychopathologic disorders such as aggression, sociopathy, and psychosis. Early in the 20th century, the term “epileptic personality” was used by psychoanalytic theorists to describe a set of character traits associated with epilepsy that focused on impulsivity, egocentricity, and affective viscosity (Delay et al., 1958). Personality disorders and other less pathologic personality changes are common and often unrecognized in epilepsy patients. These disorders likely result from biologic factors such as structural and physiologic abnormalities as well as social and emotional factors. Among epileptic patients there is a high prevalence of personality disorders like borderline, atypical or mixed, histrionic and dependent disorders (Mario and Ashla, 2005). The most common personality disorder in epilepsy is a borderline personality. Not surprisingly, epileptic patients frequently lack a stable character structure and can be immature and impulsive. Personality disorders and other less pathologic personality changes are common and often unrecognized in epilepsy patients.

(4). Psychoses: Psychoses is the specific psychiatric condition most clearly
associated with epilepsy. There is a long history of the relationship between epilepsy and psychoses, particularly schizophrenia. Hallucinations and delusions are the hallmark of psychoses and in epilepsy hallucinations and delusions may be experienced in certain settings for which patients have clear insight, in the most majority of cases insight is lacking and the condition is truly psychotic. The psychopathology of postictal psychosis is polymorphic, but most patients present with abnormal mood and paranoid delusions (Logsdail, 1988). The lifelong prevalence of all psychotic disorders among epileptic patients ranges from 7 to 12 percent. The rate of non-affective, schizophrenialike, interictal psychosis in adults with epilepsy is significantly higher than in general population (Bredkjaer, 1998). This type of psychosis is thought to occur in TLE (Temporal Lobe Epilepsy) rather than in other types of seizure disorders in both adults (Bredkjaer, 1998) and children (Caplan et al., 1988). The occurrence of interictal psychosis is generally regarded to be an adult-onset disorder. Several investigations have shown that seizures, especially of temporal lobe origin, must be present for at least 10 years before the onset of psychotic symptoms (Sachdev, 1998). Qin et al (2005) in a study confirmed the increased risk of schizophrenia and schizophrenialike psychosis in epilepsy, and a family history of psychoses and a family history of epilepsy were significant risk factor for psychosis.

(5). Aggression: Epilepsy and aggressive comorbidity is a particularly controversial issue (Geschwind, 1975). The precise prevalence of aggressive and violent behavior in the context of epilepsy is very difficult to assess. In patients with episodic affective aggression, a history of epilepsy is reported to be more common (Bach –Y-Rita et al., 1971). Ictal aggression is very rare (Gunn, 1971; and Saver et al., 1996). The patients are generally amnesic for these aggressive episodes and often express remorse or feelings of shame for their behavior after the event (Devinsky and Bear, 1984; and Fenwick, 1986). Postictal
aggression is more common than ictal aggression and, although it is still believed to be rare, it may be under-recognized and unreported (Treiman, 1991). Postictal aggressive behavior usually follows a cluster of complex partial seizures or secondary generalized seizures in patients in whom such episodes are not the usually expression of their epilepsy. Kanemoto (1999) made the important observation that well-directed and self-destructive behavior might even be a hallmark of postictal psychosis. Patients often feel angry and aroused, although they may appear calm and concentrated to the observer. Interictal aggressions are the most common, but generally less dramatic forms of aggressive behaviors in patients with epilepsy. In patients with epilepsy and mental handicap, interictal aggression is a common management problem. In these patients, the aggressive behavior is often the result of poor social and communication competence in expressing personal needs and rarely results in severe violence (Gunn, 1977). An interictal syndrome of episodic affective aggression, independent of observable ictal activity, major psychiatric disorder, or antisocial personality disorder, is well described and has been referred to as episodic dyscontrol. (Elliott, 1984; Leicester, 1982; Maletzky, 1973; Ratner and Shapiro, 1979). Finally, interictal aggression in the context of epilepsy can be a side effect of antiepileptic medication.

(6). Suicide: Suicide has been found higher in patients with epilepsy than in the general population, and some AEDs (Anti Epileptic Drugs i.e. phenobarbital) seem to contribute to this increase (Brent et al., 1987). The suicide rate in PWE is five times higher than the expected rate in the general population. However, among patients with temporal lob epilepsy the suicide rate can be 25 times higher (Gilliam and Kanner, 2002). Jallon (2004) reviewed the significantly higher mortality of patients with epilepsy by status epilepticus, sudden unexplained death and suicide compared with the general population. The generally difficult psychosocial circumstances of patients with chronic epilepsy
have often been considered the leading factor responsible for their elevated suicide rate, more important than the presence of psychiatric illness or the availability of drugs. Mendez et al. (1989) studied the causative factors for suicide attempts by overdose in 22 patients with epilepsy (from 711 patients hospitalized for a suicide attempt) and concluded that interictal psychopathologic factors were of primary importance. Suicide among patients with epilepsy clearly is not the result of psychosocial difficulties caused by having seizures, but rather occurs in the presence of significant interictal and at times postictal psychopathology. Suicide is only the most striking problem among the sizable number of patients with chronic epilepsy who suffer from dysphoric and psychotic disorders. Sudden unexpected death may occur in various situations. Some data suggest that the risk is significant only for remote symptomatic epilepsy (Hauser et al., 1980). Harvey et al (1993) found that mortality was not increased in idiopathic epilepsy compared with the general population. However, they found that in symptomatic epilepsy, the risk of death was 50-fold higher.

(7). Alcohol and Drug Abuse: Alcohol can make seizures more likely because of the effect of the alcohol on the brain. It can also interact with anti-epileptic drugs making them less effective. Although moderate alcohol consumption is not associated with increased seizure activity and alcohol withdrawal can cause seizures. A drink or two now and then do not increase seizure activity, nor does it alter the amounts of seizure medicines in blood or change findings on EEG studies. Studies suggested that alcohol withdrawal seizures must occur after 7 or 8 hours after heavy or prolonged drinking has stopped. Seizure medicines can seriously lower your tolerance for alcohol, so the immediate effects of alcohol consumption are greater. Alcoholism or chronic abuse of alcohol has been found in recent studies to be associated with the development of epilepsy in some people (National Drug Control Policy, 2003). The findings of these investigations
suggest that repeated alcohol withdrawal seizures might make the brain more excitable. Cocaine can cause seizures within seconds, minutes, or hours after its use. Seizures caused by cocaine are uniquely dangerous and may be associated with heart attacks, interruption of the heart’s normal rhythm (cardiac arrhythmia), and death. They can even occur in someone who has never had a seizure before. Amphetamines are brain stimulants. Amphetamines or other stimulants do not appear to increase the likelihood of seizures in people with epilepsy. With these effects, patients forget to take seizure medicine and much more likely to have seizures. Some research has also found long lasting damage to the brain from the use of some types of stimulants. Very high doses of amphetamines can cause severe tonic-clonic seizure, heart attacks and death. Marijuana has some antiseizure properties as well as seizure provoking effects. Taking large amounts of narcotics can cause serious oxygen deprivation to the brain, which can lead to seizures. Cigarette smoking presents a more likely danger than the effects of nicotine for people with seizure: the loss of consciousness or control of movement can cause the drop of a lighted cigarette and start a fire.

(8). Sleep Disorders: Sleep disorders are common and treatable conditions that frequently co-exists with epilepsy. A good night’s sleep plays a key role in the overall well being and health of all people yet it is even more vital in people with epilepsy. One reason why it is because a lack of sleep or poor quality of sleep can in turn increase frequency of seizures. Sleep-wake cycle is associated with prominent change in brain electrical activity, so seizures and the sleep-wake cycle are often clearly related. Most types of seizures are affected by sleep, although the degree varies greatly from type to type and patient to patient. Further, there are hormonal changes during sleep that could possibly be related to seizures (Stagno, 2001). Animal studies show that chronic seizures are associated with disruptions of sleep, particularly a decrease in rapid-eye-movement (REM) sleep (Engeletal, 1991).
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(9). Sexuality: Patients with epilepsy have been documented with hyposexuality, impaired sexual performance, reduced potency, and reduced sexual desire (Toone, 1987). Men and women experience disturbances of sexual arousal and a lower sexual drive. Some patients have a disinterest in all the usual libidinous aspects of life, including loss of erotic fantasies or dreams, and may experience impotence or frigidity. Disturbances in reproductive health, including reduced fertility often accompany epilepsy (Mario and Ashla, 2005). Women experience an increased incidence of endocrine disorders, menstrual cycle irregularities and ovulatory dysfunctions. Men have an increased risk of erectile dysfunction. Epilepsy appears to produce a higher incidence of sexual dysfunction than other chronic neurologic illnesses manifesting primarily as diminished sexual attitude and behavior with noted sexual dysfunction in 30% to 66% of epileptic men and 14% to 50% of women (Wyllie, 2001). Most studies of sexual functions have demonstrated that age of onset of epilepsy influences sexual adjustment; patients with early onset exhibited more hyposexuality, whereas those with onset after puberty were better adjusted. Seizure cessation, either with medication or with surgery, often results in increased sexual interest.

(10). Stress: Many reports provide evidence of an association between stressful life events or tension states and epileptic seizures (Frucht et al., 2000; Nakken et al., 2005; and Tan et al., 2005). Patients with generalized seizures and those with partial seizures seemed to be equally sensitive to emotional stress (Nakken et al., 2005). Frucht et al. (2000) in a study found that patients with temporal lobe epilepsy were the most likely to identify stress (46%) and patients with cryptogenic epilepsies were the least (15%). Stress can trigger an increase in the breathing rate, known as hyperventilation. Hyperventilation is taken to provoke seizures in certain patients, especially those with absence seizures. Stress can cause hormonal changes such as an increase in the
steroid hormone cortisol, which also may influence seizure activity. Negative emotions related to stress, such as worry or fright, may cause seizures. This happens because the limbic system, the portion of the brain that regulates emotion, is one of the most common places for seizures to begin. Living with epilepsy can be stressful for some people. The form their seizures take causes additional worry. Many seizures, particularly complex partial attacks, involve bizarre behaviours that would not be readily be identified by the general public as epilepsy and may be mislabeled as drunkenness, madness or a provocative outburst. The risk of injury may be high for some seizure types, such as drop attacks or complex partial seizures that involve episodes of wandering. This too can cause stress. Stress of having seizure may be greater when others are potentially placed in danger such as young children of parents who have epilepsy. Although difficult to quantify, emotional stress is the most common factor (30% to 66%) identified by patients, mostly by women (Nakken et al., 2005; Da Silva Sousa, 2005). Since the time of Hughlings Jackson (1873), attention has been paid to the role of the emotions in the precipitation of epileptic seizures. Many reports provide evidence of an association between stressful life events or tension states and seizures (Frucht et al., 2000; Nakken et al., 2005; Tan et al., 2005). In one study Mattson (1991) 58% of patients reported emotional stress, such as worry, anxiety, frustration, and anger, as the second most frequent precipitating or modulating factor for seizures.

**Cognitive functions in Epilepsy Patients**

Epilepsy with cognitive symptomatology can be defined as those which manifest their effects mainly or exclusively in the cognitive sphere; the cognitive disturbance is itself the seizure, with no other visible manifestations besides the altered mental state. In many forms of epilepsy, cognitive functions are altered during the seizures itself, the postictal state and sometimes during what is thought to be the normal interictal period. However, the cognitive dysfunction is only one among other epileptic
manifestation and is not recognized or considered the main symptoms. Neither psychological consequences of the diagnosis nor side-effects of treatment can be blamed, it is suggested that direct cognitive effects of epilepsy exist more often than is usually acknowledged (Austin et al., 2001). In addition, any acquired disruption of cognitive function will have negative consequences on emotional or social behavior. The episodic nature of symptoms, considered characteristic of epilepsy, is often not immediately apparent. The clinical disturbances must sometimes be measured over days or weeks; and in special situations possibly, months or years. Gradual loss of cognitive functions, arrest or regression in development or new behavior disorders may thus be the presenting problems, with few or no hints of the epileptic origin. Temporary disturbances of cognitive functions can be due either to focal epileptic seizures originating in brain areas which mediate particular cognitive functions or to generalized discharges which interfere with more global aspects of mental functions such as vigilance or execution. The observed deficit corresponds either to the ictal or to the postictal phase. Various forms of aphasias, apraxia, frontal lobe dysfunctions, visuospatial disability or selective memory deficits have been reported, although precise details of these observations are rare in children (Deonna et al., 1982; Deonna et al., 1987; Jambaque and Dulac, 1989; Deonna, 1993; and Deonna et al., 1993). More prolonged cognitive epileptic deficits are probably consequences of recurrent seizures with repeated postictal deficits and incomplete recovery of functions between episodes. Logic suggests that frequent recurrent epileptic discharges in areas involved with complex developing mental functions can lead to more lasting consequences on cognition and behavior than those involving more elementary sensory and motor functions. People with epilepsy must be tested individually during and after episodes suspected of being cognitive ictal or postictal manifestations and direct correlations with EEG changes and treatment shown. To ascertain which aspects of mental functions are affected during a suspected cognitive seizures interactions as well as observation is necessary and it is difficult to
plan systematic studies (Gloor, 1991). Frequent mood changes, fluctuating attention, forgetfulness, uneven memory skills, variable school results, uneven speed of performance, and transient failures in specific domains have been noted in the history of epilepsy. Although none of these is specific, their combination and usually unexplained sudden occurrence and recurrence can be very suggestive that unrecognized cognitive seizures are interfering with vigilance, attention, with more specific functions such as language or memory. Sometimes, the changes resemble those already seen in postictal states of previous more clear-cut episodes.

In school children, failure to encode, store, consolidate newly formed memories during or after seizures is probably an under-recognized cause of learning problems. Transient memory impairment as the sole manifestation of complex partial seizures known in adults as epileptic amnesic attack (Galassi et al., 1993) could easily go unrecognized in children. Cognitive seizures must also be considered in brain-damaged children with epilepsy and chronic learning and behavioral disorders. Epilepsy can be responsible for, or aggravate the chronic cognitive disturbances usually attributed to the brain focal lesion in congenital hemiplegia (Varga-Khadom et al., 1992). The onset of epilepsy occurs before the age of 20 years in 60 percent of patients and one third has their first seizure when in junior school. The appearances of epilepsy in childhood often interfere with normal cognitive development and with academic achievement (Sillanpaa et al., 1998). Several studies have shown that children with epilepsy perform less well at school than their healthy peers. Neuropsychological impairment consisting of memory disturbances, visuo spatial and verbal deficits has been demonstrated in children even with very well controlled epilepsy (Pavone et al., 2001; and Deonna et al., 2000). Estimates of prevalence rate of learning problems range from 5 to 50 percent (Thompson, 1987). There is often uncertainty about the relationship between the learning disorders and epilepsy. Clinicians and researchers have identified several variables that influence learning and cognitive functions, such as age of onset of seizures,
duration of epilepsy, types and frequency of seizures and subclinical epileptiform EEG discharges. Differences in etiology such as a progressive brain disease, localized or generalized brain damage after a trauma or a meningoencephalitis may also influence learning problems. However, in at least 50 percent of children with epilepsy no etiologic factors can be determined (Sillanpaa et al., 1998). In addition to the influence of the epileptic disorders itself, antiepileptic treatment (AEDs) can also affect cognition. Another negative influence is the impact of epilepsy on social functioning, which in turn decreases school performance (Oostrom et al., 2000). Finally all of the above mentioned factors, such as epileptic clinical seizures, underlying brain dysfunction with cognitive deficits, AEDs, social stigma and low self-esteem can lead to behavioral changes in the child, thus worsening cognitive performance and learning. It is therefore not surprising that many questions concerning this topic still have to be answered (Cornaggia and Gobbi, 2001). Various studies have demonstrated that IQ levels of children with epilepsy tend to be below the population mean (Farwell et al., 1985; and Bourgeois et al., 1983). Rutter et al (1970) found a distribution of intelligence in children with epilepsy closely resembling the normal distribution values. However, it should be noted that the children investigated all attended normal school and showed a mild form of epilepsy. The IQ score of the epileptic child is influenced by the type, frequency and severity of epileptic seizures as well as by the etiology. Analysis of sub-tests scores reveals below-average scores especially in vocabulary (fluency, verbal language skills), coding or digit-symbol (attention, sensory-motor coordination), and information (verbal language skills).

It is known that seizure activity and many anticonvulsants depress cognitive functions (Dodrill 1975; and Schmidt, 1986) and following surgical control of seizures selected cognitive functions improve. The improvements are due to the elimination of ictal disturbances and from reduction in medications. The most likely improvements to occur are those related to general measures of cognition such as IQ and learning or to
selective functions associated with brain regions outside the resected area (Hermann et al., 1989; Nadig and Wieser, 1987; Novelly et al., 1984; Rausch and Crandall, 1982; and Wieser, 1986). Therefore, there is growing evidence that a limited anterior temporal resection in either hemisphere does not produce significant long-term adverse neurologic or neuropsychological sequel, and the result of a seizure-free status may even improve the patient’s neuropsychological baseline. Persons undergoing surgery before the age of 30 or as students tend to have significantly better outcomes, particularly, vocational. Patients with poor cognitive functioning impairing neuropsychological deficits—specifically of memory—and severe preoperative or postoperative psychiatric conditions can do less well. Early data from children undergoing temporal lobectomy suggested little overall risk to cognitive function (Westerveld et al., 2000).

Cognitive impairment is the most common comorbid disorder in epilepsy (Aldenkamp and Dodson, 1990; and Dodson and Pellock, 1993). Memory impairments, mental slowing, and attentional deficits are the most frequently reported cognitive disorders (Aldenkamp et al., 1995; and Donati et al., 2006). Such consequences may be more debilitating for a patient than the seizures, thus, it is worthwhile to explore the factors that lead to cognitive impairment. The exact causes of cognitive impairment in epilepsy could not be explored fully, but three factors clearly involved are: etiology, the seizures, and the “central” side effects of drug treatment (Aldenkamp, 2002). When evaluating the unwanted effects of antiepileptic medication on cognitive function separately, it is imperative to realize that in clinical practice most cognitive problems have a multifactorial origin and that, for the most part, the three aforementioned factors in combination are responsible for the makeup of a cognitive problem in an individual patient. Moreover, the factors are inter-related which cause therapeutic dilemmas in some patients when seizure control can only be achieved with treatments that are associated with cognitive side effects. A general conclusion that may be derived from meta-analyses is that polypharmacy shows a relatively
severe impact on cognitive functions when compared with monotherapy irrespective of the type of AEDs included (Vermeulen and Aldenkamp, 1995). Two drugs that individually have mild cognitive effects may induce serious cognitive impairment when used together, possibly because of potentiation of tolerability problems (Trimble, 1987). Regarding later onset of epilepsy, two of the most common types of pediatric epilepsy, complex partial seizure disorder and primary generalized epilepsy, typically begin in middle childhood. During middle childhood, acceleration occurs in the development of children's emotional, social, cognitive, and linguistic skills and continued maturation through adolescence. Ongoing seizures, high doses of multiple AEDs, and prolonged seizures might interrupt the functioning of the neural circuits involved in these maturational processes. In children with onset of epilepsy in middle childhood, although the presence of psychopathology does not appear to be associated with age of onset (Caplan et al., 2005). Earlier studies though have identified an association between early-onset and impaired cognitive functioning (Addy, 1987; Bourgeois et al., 1983; Ellenberg et al., 1984; and O’Leary et al., 1983). Yet these findings might also reflect the confounding effects of poor seizure control as well as AED polytherapy and high blood levels of treating drugs (Bourgeois et al., 1983). More recent studies have also found that cognition and language (Schoenfeld et al., 1999), verbal learning, and certain aspects of discourse skills (Caplan et al., 2001; and Caplan et al., 2002) are associated with age of onset in children with complex partial seizures. For example, unrelated to lateralization of EEG findings, children with earlier onset of complex partial seizures have significantly lower scores on neuropsychological and linguistic tasks, poor performance on long delay cued recall, and reduced monitoring and self-correction of errors in the organization of ideas during speech than do those with later onset (Caplan et al., 2001). The age of onset of epilepsy might be an indicator of the severity of the underlying pathology. Thus, early-onset epilepsy is often associated with mental retardation, and children with mental retardation have an earlier
onset of epilepsy (Rutter et al., 1970). These age relationships imply that epilepsy and mental retardation in children with early onset are epiphenomena of underlying pathologic processes in the brain. In terms of psychopathology, two epidemiologic studies have demonstrated significantly higher rates of psychopathology in children with epilepsy who have mental retardation or complicated epilepsy including learning disabilities than those with normal intelligence or uncomplicated epilepsy (Davies et al., 2003). A population study of children with both mental retardation and epilepsy also revealed high rates of severe behavior problems (Steffenburg et al., 1996). Fedio and Mirsky (1969) found specific disorders of attention and constructional apraxia in children with generalized tonic-clonic convulsions compared to those with partial seizures. Giordani et al (1985) also reported similar findings. Attention and concentration deficits are often recognized as a particular problem in children with epilepsy (Stores, 1978). Memory impairment has also been found during the occurrence of epileptiform-EEG discharges using short term memory tasks (Kasteleijin et al., 1990). Anti-epileptic drugs such as phenytoin has also been shown to influence memory negatively (Thompson et al., 1981). It is well recognized that children with epilepsy have a higher risk of deficits in cognitive functioning. In several studies over the past 25 years, it has been shown that children with epilepsy perform worse at school than their healthy peers and also performs worse than children with other chronic diseases such as asthma or migraine. Furthermore their academic achievement seems to be dependent on age of onset of the schooling period. Many different factors contribute to cognitive impairment and most of these are interrelated.

Coping in Epilepsy

Epilepsy exposes its patients to a wide range of unique psycho-social consequence which sometimes can be more damaging than the medical difficulties they deal with. The causes of such problems are considered multi-factorial encompassing neurological, psychological, social variables and their mutual interdependence (Hermann and Whitman, 1984). Life
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stressors and the coping strategies used to prevent or reduce high levels of stress are receiving considerable attention in today’s world. Persons with chronic illnesses face additional stressors. Treatment costs, adjustments in lifestyle, pain, and side-effects of medications are a few of the associated major stressors. Assisting persons to find strategies for handling these stressors is critical, as studies have shown the adverse effect that high levels of stress can have on symptomatology and progression of an illness.

Health professionals play an instrumental role in assisting persons in learning effective coping strategies. However, until more is known about the stressors associated with specific illnesses and the effectiveness of particular coping strategies, health professionals cannot intervene in a knowledgeable manner (Lazarus, 1983; and Moos and Tsu, 1977). Coping strategies are commonly defined as “efforts to manage environmental and internal demands and conflicts among them, which can tax or exceed a person’s resources” (Lazarus and Launier, 1978). Coping processes have been gradually recognized as distinct from defense mechanisms in the early 1960s. Ten years later, these processes have been used largely in psychological literature (Livneh et al., 2001). In adjusting to this chronic circumstance, the method of coping with epilepsy seems to be a significant determinant of the self-perceived severity of seizures (Oosterhuis, 1999). In addition, studies have shown that coping strategies have a marked influence on the psychosocial adjustment and well-being of patients with epilepsy (Goldstein et al., 2005). Adjustment resources and coping strategies can play a mediating role between stressors and the illness. In fact, these mediators can either decrease the effect of stressors causing the patient to move toward positive psychological adjustment, or increase the effects of stressors causing the patient to resort to negative psychological adjustment (Roesch and Weiner, 2001). Lazarus recognized two main coping categories: problem focused, based on specific strategies to find a direct resolution for the situation; and emotion focused, which deal chiefly with the emotional responses to stressors (Piazzini et al., 2007). More recently, some authors
have begun to analyze the coping modes in a variety of chronic disorders such as Parkinson’s disease (MacCarthy and Brown, 1989; Neelam and Singh, 2008) rheumatoid arthritis (Zatura and Manne, 1992), diabetes (Frenzel et al., 1988; Kaur and Singh, 2012), cancer (Helm et al., 1993) and finally, epilepsy (Rosenbaum and Palmon, 1984). Regarding this pathology, research findings highlight that one of the most adaptive coping patterns is the problem-focused strategy, which is generally linked to increased mental health/psychological well-being (Synder, 1990) and better psycho-social adaptation, (Murray, 1993; Krakow et al., 1999) while disengagement styles such as avoidance and denial of personal clinical conditions seem to cause greater distress (Upton and Thompson, 1992; and Oosterhuis, 1999). In this regard patients with epilepsy who adopted problem-focused and cognitive restructuring coping modes have been reported to have better psychosocial outcomes including better mental health, increased psychological well-being, decreased psychological distress, and lower levels of reported depression and anxiety (Livneh et al., 2001).

Epilepsy is one chronic condition for which it has been hypothesized that high levels of stress affect symptomatology by increasing the occurrence of seizures. A number of stressors associated with epilepsy have been suggested in the literature such as prejudice, social rejection, over-protectiveness, lack of understanding, difficulty in getting jobs and insurance, and lack of control over when seizures will occur (Anies, 1982; Minter, 1979; and Temkin and Devis, 1984). These stressors have, however, been identified from the perspective of health professionals, and their presence and stressfulness have not been verified by persons with epilepsy (Ferrari, 1983; and Schneider and Conrad, 1981).

Most persons develop a repertoire of mechanisms for coping with stressors they experience. Mattlin, Wethington, and Kessler (1990) noted that minimal knowledge exists on the nature of strategies in a person’s repertoire, and even less about the relative effectiveness of specific strategies. Persons who are able to mobilize adequate and or appropriate
coping strategies tend to have a higher level of health and well-being than those who lack adequate resources (Erickson and Swain, 1982; Devi and Singh, 2012). Meager information from the perspective of the patient has been found on epilepsy-specific stressors and the coping strategies used in handling these stressors. Sometimes, psychosocial consequences are more debilitating and damaging than the seizures and related physical problems (Lau et al., 2001). Such problems and challenges cause patients to resort to coping strategies.

**Treatment/Management of Epilepsy**

Epilepsy is usually treated with medication prescribed by physicians, primary caregivers, neurologists, and neurosurgeons. However, it has been emphasized that accurate differentiation between generalized and partial seizures is especially important in determining the appropriate treatment (Trost et al., 2005). In some cases, the implantation of a stimulator of the vagus nerve, or a special diet can be helpful. Neurosurgical operations for epilepsy can be palliative reducing the frequency or severity of seizures; or, in some patients, an operation can be curative.

(1). Biological Management

    *a. Medications:* The mainstay of treatment of epilepsy is anticonvulsant medications. Often, anticonvulsant medication treatment will be lifelong and can have major effects on quality of life. The choice among anticonvulsants and their effectiveness differs by epilepsy syndrome. Mechanisms, effectiveness for particular epilepsy syndromes, and side-effects differ among the individual anticonvulsant medications. Currently there are 20 medications approved by the Food and Drug Administration to be used in the treatment of epileptic seizures in the US namely, Carbamazepine (common US brand name Tegretol), Clorazepate (Tranxene), Clonazepam (Klonopin), Ethosuximide (Zarontin), Felbamate (Felbatol), Fosphenytoin (Cerebyx), Gabapentin (Neurontin), Lacosamide (Vimpat), Lamotrigine (Lamictal), Levetiracetam (Keppra), Oxcarbazepine (Trileptal), Phenobarbital (Luminal), Phenytoin (Dilantin), Pregabalin
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(Lyrica), Primidone (Mysoline), Tiagabine (Gabitril), Topiramate (Topamax), Valproate Semisodium (Depakote), Valproic Acid (Depakene), and Zonisamide (Zonegran). Most of these have appeared after 1990. The goal for individual patients is no seizures and minimal side-effects; and the job of the physician is to aid the patient to find the best balance between the two during the prescription of anticonvulsants. Most patients can achieve this balance best with monotherapy, the use of a single anticonvulsant medication. Some patients, however, require polypharmacy i.e. the use of two or more anticonvulsants. If a person's epilepsy cannot be brought under control after adequate trials of two or three (experts vary here) different drugs, that person's epilepsy is generally said to be medically refractory. A study of patients with previously untreated epilepsy demonstrated that 47% achieved control of seizures with the use of their first single drug. 14% became seizure free during treatment with a second or third drug. An additional 3% became seizure-free with the use of two drugs simultaneously (Kwan and Brodie, 2000). Other treatments, in addition to or instead of, anticonvulsant medications may be considered by those people with continuing seizures.

b. Surgery: Epilepsy surgery usually involves the resection of the damaged or abnormal part of the brain. This may be a small amount of tissues such as a lesion on the brain or in extreme cases when there is a lot of damage to one half of the brain, may involve the removal of the complete hemisphere. The other strategy for surgical treatment is palliative, either to interrupt pathways of seizure spread by disconnecting the area of the brain that is causing seizures, or vagal nerve stimulation. Success rates are high and some procedures, such as the removal of part of the brain known as the hippocampus, located in the temporal lobe, can result in complete seizure freedom in more than 70 per cent of cases. But even in those patients who do not achieve complete seizure freedom, the surgery can result in far fewer seizures and a greatly improved quality of life. In children, surgery can also lead to improved developmental outcomes. Before the decision to go for
surgery is finally made, the patient will undergo many tests to pinpoint precisely the epileptogenic tissue and any potential adverse effects, and to establish the individual’s suitability for surgery. A multi-disciplinary approach should be undertaken in this pre-surgical assessment including the neurologist, neurosurgeon, psychologist, psychiatrist, neurophysiologist and radiologist. Epilepsy surgery is an option for people with focal seizures that remain resistant to treatment (Duncan et al., 2006). The goal for these procedures is total control of epileptic seizures (Birbeck et al., 2002), although anticonvulsant medications may still be required (Berg et al., 2007). The evaluation for epilepsy surgery is designed to locate the "epileptic focus" (the location of the epileptic abnormality) and to determine if respective surgery will affect normal brain functions. Physicians will also confirm the diagnosis of epilepsy to make sure that spells arise from epilepsy (as opposed to non-epileptic seizures). The evaluation typically includes neurological examination, routine EEG, Long term video-EEG monitoring, neuropsychological evaluation, and neuroimaging such as MRI, Single Photon Emission Computed Tomography (SPECT), Positron Emission Tomography (PET). Some epilepsy centers use Intracarotid Sodium Amobarbital Test (Wada test), functional MRI or Magnetoencephalography (MEG) as supplementary tests. Certain lesions require Long-term video-EEG monitoring with the use of intracranial electrodes if noninvasive testing was inadequate to identify the epileptic focus or distinguish the surgical target from normal brain tissues and functions. Brain mapping by the technique of Cortical Electrical Stimulation or Electrocorticography are other procedures used in the process of invasive testing in some patients. The most common surgeries are the resection of lesions like tumors or arteriovenous malformations, which in the process of treating the underlying lesion, often result in control of epileptic seizures caused by these lesions. Other lesions are more subtle and feature epilepsy as the main or sole symptom. The most common form of intractable (hard to control) epilepsy in these disorders in adults is temporal lobe epilepsy with
hippocampal sclerosis, and the most common type of epilepsy surgery is the anterior temporal lobectomy, or the removal of the front portion of the temporal lobe including the amygdala and hippocampus. Despite the efficacy of epilepsy surgery, some patients decide not to undergo surgery owing to fear or the uncertainty of having a brain operation.

c. Electrical Stimulation: Electrical stimulation is also an approved method in treatment of epilepsy (Theodore and Fisher, 2004). Methods of anticonvulsant treatment are both currently approved for treatment and investigational uses. A currently approved device is Vagus Nerve Stimulation (VNS). Investigational devices include the responsive neurostimulation system (RNS) and deep brain stimulation (DBS).

Vagus Nerve Stimulation is a major communication link between the body and the brain. VNS Therapy works by sending mild electrical signals to the brain via the nerve, disrupting the abnormal electrical activity that can cause seizures. In epilepsy treatment, a stimulator, a little like a heart pacemaker, is implanted under the skin in the person’s upper chest. Electrodes are connected to the stimulator at one end and are coiled around the left vagus nerve in the neck at the other end. The device stimulates the vagus nerve at preset intervals and intensities of current. Efficacy has been tested in patients with localization-related epilepsies demonstrating 50% of patients experience a 50% improvement in seizure rate. Case studies have demonstrated similar efficacies in certain generalized epilepsies, such as Lennox-Gastaut syndrome. Although success rates are not usually equal to that of epilepsy surgery, it is a reasonable alternative when the patient is reluctant to proceed with any required invasive monitoring when appropriate presurgical evaluation fails to uncover the location of epileptic foci or when there are multiple epileptic foci.

Responsive Neurostimulator System consists of a computerized electrical device implanted in the skull with electrodes implanted in presumed epileptic foci within the brain. The brain electrodes send EEG signals to the device which contain seizure-detection software. When certain
EEG seizure criteria are met, the device delivers a small electrical charge to other electrodes near the epileptic focus, which disrupt the seizure. The efficacy of the RNS is under current investigation with the goal of FDA (Food and Drug Administration) approval.

Deep Brain Stimulations consists of a computerized electrical device implanted in the chest in a manner similar to the VNS, but electrical stimulation is delivered to deep brain structures through depth electrodes implanted through the skull. In epilepsy, the electrodes target is the anterior nucleus of the thalamus. The efficacy of the DBS in localization-related epilepsies is currently under investigation.

d. Bio-feedback (consciously controlling activity in the brain): Biofeedback is a technique which may be helpful for people who experience partial seizures or secondarily generalised seizures that begin with some kind of warning or aura. Over the time some people can learn skills to help to consciously control activity in the brain. In some people this may help stop the seizure spreading. Furthermore, the technique may increase a persons’ self-esteem by giving them a sense of control over their epilepsy. This therapy is not widely used, as it requires a lot of input from the therapist, and much practice and time on the part of the patient to achieve results. Biofeedback may be offered to adults and young people in clinical societies.

e. Ketogenic Diet: Ketogenic diet is rich in lipids (fats) and oils, but low in proteins and carbohydrates can be useful in the treatment of epilepsy. This unusually high intake of lipids and oils creates a condition in the body known as "ketosis". The metabolic shift that is created increases the seizure threshold for some patients. It was first tested in the 1920s but became less used with the advent of effective anticonvulsants. In the 1990s specialized diets again gained traction within the medical community (Simon, 2004). The mechanism of action is unknown. It is used mainly in the treatment of children with severe and medically intractable epilepsies. Some children’s seizures respond very quickly to the diet, others can take upto
three months, and some will not respond at all. How long a child stays on
the diet will depend on how much that seems to be benefiting. If the child is
seizure free for two years, most doctors would suggest a trial of slowly
returning to a normal diet.

(2). Psychological Management

As with all psychiatric problems, psychopharmacologic management
alone is not sufficient. Maintaining and bringing such support to the person
with epilepsy is important, sustaining them in the community and preventing
their recurrent admission to the hospital. Further, in a good family
environment with adequate medical facilities and follow-up care, patients’
compliance will tend to be good. Deterioration of an otherwise delicate
situation induced by poor compliance, leading to more seizures and
exacerbation of psychopathology with loss of control by the family and the
physician, may thereby be avoided. Some psychological interventions are as
under:

(a). Cognitive Behavioural Therapy: Epilepsy is traditionally treated
with drugs; and for pharmacoresistant cases there may be a possibility of
epilepsy surgery. But there is a third line of therapy that can be useful if
applied to the appropriate patients i.e. Behavior modification. Behavior
modification can be defined as any type of non-pharmacologic intervention
aimed at the prevention or abortion of seizures. Like all other epilepsy
therapies, the ultimate goal of behavior modification is complete seizure
control. If precipitating factors are identified, behavior modification can be
used alone or in conjunction with other therapies. It can be used to arrest
seizure activity after onset or to prevent seizures altogether. These
interventions comprise seizure prevention by avoidance, modification,
counteraction, or desensitization of nonspecific facilitating factors such as
irregular sleep, psychogenic factors, and specific precipitating factors. Some
people may be benefited from cognitive behavioural therapy (CBT) and
other interventions aimed at reducing social anxiety and developing social
skills. People with epilepsy should be encouraged to reap the benefits of
social networking sites, epilepsy forums, and support groups. These outlets can provide emotional support, contacts, and information; and may help to reduce isolation. There are several studies that have established the efficacy of cognitive behavior therapy (CBT) for the management of depressive and anxiety disorders either by themselves or in combination with pharmacotherapy. In general, CBT consists of short-term treatments of 16 to 20 sessions given during 12 weeks on average (Fink, 1999). The treatment options of anxiety disorders include pharmacotherapy, a variety of psychotherapeutic modalities (i.e., cognitive behavior therapy, desensitization behavioral therapy, supportive and psychodynamically oriented psychotherapies) and a combination of psychotherapy and pharmacotherapy can be used in all epilepsy clinics. Psychological therapies in the form of anger management or other variations of cognitive behavior therapy are an integral part of management in many patients. Different methods of anger management, cognitive behavior therapy, or skills training, that have been developed irrespective of the context of impulsive behavior, may be very helpful in the therapy of aggression (Stanley et al., 2001; Thomas, 2001; and Willner et al., 2005).

(b). Avoidance Therapy: It consists of minimizing or eliminating triggers in patients whose seizures are particularly susceptible to seizure precipitants. For example, sunglasses that counter exposure to particular light wavelengths can improve seizure control in certain photosensitive epilepsies (Takashashi and Tsukahara, 1992).

(c). Alternative or Complementary Therapies: Acupuncture (Cheuk and Wong, 2008), psychological interventions (Ramaratnam et al., 2008), vitamins supplements (Ranganathan and Ramaratnam, 2005), and yoga (Ramaratnam and Shridharan, 2000) that includes exercises with different postures (asanas), special respiratory techniques (pranayama), and mental relaxation and meditation (dhyana), have also been evaluated as treatments for epilepsy. Yoga and yogic mindfulness meditation have been used for stress reduction (Nespor, 1994) and, therefore, have a therapeutic role in
epilepsy, particularly for patients with stress-induced seizures. Exercise or other physical activity (Arida et al., 2009; and Arida et al., 2008) have also been proposed as efficacious strategies for preventing or treating epilepsy. However, while some people may find complementary therapies to be beneficial, others may find they interfere with seizure control. Relaxation therapies such as massage, acupuncture or reflexology can be helpful for those people who find they have more seizures at times of stress or anxiety, but great care needs to be taken with aromatherapy. Although certain aromatherapy oils such as jasmine, chamomile, and lavender have a calming effect and may be helpful in improving seizure control but some other oils such as hyssop, rosemary, sweet fennel and sage are thought to have an alerting effect on the brain, may trigger seizures in people with epilepsy.

(3). Social Management

There has been longstanding concern in the field of epilepsy in relation to medical management of the disability, including the development of new generations of antiepileptic medications but attention is less focused on the social adjustment of individuals with the disability. The field of epilepsy is at a crossroads relative to addressing the psychosocial needs of people with this disability and their significant others. Despite the fact that psychosocial gains are being made in a number of areas, certain issue areas deserve increasing attention. Although there is a dramatic increase in quality-of-life assessment and an effort to measure the impact of seizure severity on patients' lives. Buck et al (1996) found that patient's feel poorly educated not only about epilepsy, but also about medications and potential side effects. It can be very difficult for physicians to make the effort carefully to explain epilepsy and issues in medical management to a patient and family members. Somehow, however, this needs to be done because it directly relates to patient compliance. Westbrook et al (1992) found that more than half kept their condition a secret from others and almost three fourths said they rarely or never talked about it to others, even though denying that having epilepsy affected their friendships and perceived
likeability among their peers. Stigma was positively associated with impaired self-esteem, self-efficacy, sense of mastery, perceived helplessness, increased rates of anxiety and depression, increased somatic symptomatology, and reduced life satisfaction was studied by Arnston et al., 1986; Jacoby, 1994; Jacoby et al., 1996; Westbrook et al., 1992; Dilorio et al., 2003 and Baker et al., 2000. The epilepsy associations are moving rapidly from a view of their function as that of support and information to an increasingly political and campaigning role, agitating for better services and less discrimination for their members (Lee, 2000).

(a). Changing Negative Public Attitudes: With regard to improving public attitudes toward epilepsy, possible strategies include education and information provision, advocacy, inducing a greater degree of empathy toward epilepsy, and increasing the level of contact between people with epilepsy and people without epilepsy. According to Brown et al (2001) the most frequently used strategy appears to be that of education, and findings from reviews and single studies related both to other health conditions support it as an effective one (Schulze et al., 2003; Tanak et al., 2003; Crisp et al., 2004; Pinfold et al., 2005). Evaluation, however, is often limited to fairly short-term assessments, and different kinds of educational interventions show mixed effects on patterns of attitude and behavioral change and stigma reduction. Interventions targeted at specific population subgroups appear more promising and cost-effective than broad-based public educational campaigns (Stuart, 2003). The overarching message appears to be that the type and content of educational interventions needs to be shaped within peoples' traditional way of thinking and must take account of the context in which stigma operates. Advocacy focuses on provision of a supportive, enabling environment within which attempts can be made to influence legislative and policy change and is proposed as an important strategy for stigma reduction by several authors (Ablon, 2002; Angermeyer and Matschinger, 2005; Corrigan, 2007). Attitude and behavior change initiatives appear to be more effective when targeted, Inducing empathy for
a member of a stigmatized group can improve attitudes toward members of that group as a whole (Batson et al., 2002). The assumption here is that by inducing others to see the world from the perspective of a stigmatized group member, they can be led to feel for this person and these empathetic feelings will generalize, resulting in a more positive attitude toward the group as a whole. Similarly, interventions based on increasing contact have been proposed as a means of reducing social distance toward affected persons. Such interventions can be targeted at individuals, groups, or the wider community. Brown et al (2001) argued that a contact strategy in conjunction with education is one of the most promising approaches to reducing negative attitudes. Whatever strategy is adopted, the literature makes it clear that conducting stigma intervention programs requires diverse skills to engage and interact with targeted communities and community agencies. It also suggests that multistategy, multilevel approaches are more effective in improving knowledge and reducing stigmatizing attitudes than are single interventions. Family, local community, health and social care systems, educational institutions, legal systems, employment, and insurance were all identified as areas in which people with epilepsy might encounter stigma. Continuing to challenge stigma in all these areas must remain a priority.

(b). Services for Facilitating Social Adjustment: The increased prevalence of social problems in people with epilepsy emphasizes the need for a comprehensive approach to treating people with this condition. Although seizure control is a critical factor affecting social function, managing the consequences of seizures on one's daily life is often the most challenging component of epilepsy care. Health care professionals must be aware of the multiple causes of social problems, how to screen for their occurrence, and how to provide appropriate referrals, education, and treatment. In addition, recent research highlighting the increased incidence of cognitive, mood, and behavior problems in children and adults with epilepsy stresses the need to identify and treat these problems early before the psychosocial consequences become intractable. The ultimate goal of
support services must be to help persons with epilepsy and their families become as socially capable and competent as possible and live independent self-directed lives. Ideally, models of care and support systems will “foster empowerment and independence for people with epilepsy and support their efforts towards improved seizure control and a positive quality of life” (Epilepsy Foundation of America, 2003). Early referral to services is recommended to prevent and treat social difficulties. Support services should be matched to specific needs of the persons with epilepsy and their families.

(c). Role of Family: Jacoby (1994) stress the importance of family support and clear interfamily communication as being preventive of perceived stigma by offspring. Educational programs for both patients and the general public could be very helpful in improving the prognosis for epilepsy. Thompson and Upton (1992) indicated levels of stress and dissatisfaction within families that can be high because those with epilepsy often face difficulties in maintaining primary work careers or even receiving respite care for their children. Within the context of psychosocial research, interventions with families and their impact on medical and social adjustment have been largely overlooked. Labeling theorists maintain that the impact of a negative social label, for example, being “epileptic,” can be quite profound in as much as the label overrides other aspects of a person's identity. The process by which people with epilepsy come to acknowledge their deviant status and accept that they are not like everyone else has been the focus of attention of several authors (West, 1979; Schneider and Conrad, 1983).

Parents emerge as key figures in these analyses because their reactions to a diagnosis of epilepsy in their child seem effectively to set the stage for the child's subsequent interpretation of its significance. When parental reactions are negative, their affected child learns to think of epilepsy as something shameful; when parental assumptions are that epilepsy will inevitably attract hostile reactions from others, their affected
child learns to think of it as something to keep quiet about. Other key figures are likely to be teachers and health care professionals, both of which groups are known as sometimes having less-than-positive attitudes about the condition and limited knowledge about its implications.

(d) Management at School: Parents and teachers should avoid implementing rules for a regular and quiet life for children with epilepsy. Although fatigue and sleep deprivation may provoke seizures in some people with epilepsy, nothing should be left out of the lives of children with epilepsy unless it would be dangerous in that particular person. Education of parents, teachers, and children with epilepsy should decrease unsubstantiated fears about the condition and the implementation of unnecessary restrictions on their activities. Decisions must be made about participation in activities. The children should participate in this decision making about their participation in activities from a young age; otherwise, they might have difficulty in accepting real restriction or precautions. It is obvious that living a full, independent life between the seizures involves taking risks for everybody, and taking risks implies that, with statistical certainty, accidents will occur. Intervention during childhood and particularly adolescence is crucial within this population, most existing intervention efforts appear to be conducted within short-term summer camp structures (Raty et al., 2005; Cusher-Weinstein et al., 2005).

(e) Management at Workplaces: The vast majority of jobs are suitable for people with epilepsy. When medical advice is sought about the suitability of particular jobs for people with epilepsy, the guidance given should take into account the requirements of the job, known facts about epilepsy, and the nature of the individual's seizures. More specifically, decisions should take into account information concerning the individual's epilepsy. Simple precautions can be taken to reduce the risks in the workplace, such as providing a chair with armrests to prevent the person from falling when having a seizure. In jobs known to carry a high degree of physical risk to the individual worker or to others, the organization of work
practice should be examined to reduce this potential risk to an acceptable level. Only in those situations in which this cannot be achieved are restrictions on the employment of people with epilepsy justified (International Employment Commission of IBE, 1989).

(f) Management at Home: Most accidents happen at home and this generalization applies to everyone, simply because we spend so much time there. Still, for people with epilepsy, there are extra risks, some of which can easily be avoided. In a living room or kitchen an open fire or stove should be surrounded by a shield, furniture should not have sharp edges, electric tea- or coffeemakers are less risky than a hot water kettle. In the bathroom the door should be made to open from the outside, baths should not be taken when the person is alone in the house, showering is safer than bathing and any glass windows in the shower should be of unbreakable glass. In the bedroom, if a person tends to fall out of the bed following seizures, a mattress may be placed on the floor. This is less restricting than a shield around the bed and it is quite safe.

(g). Leisure Activities and Club Membership: Health professionals are often asked to give opinions about the risks for people with epilepsy engaging in leisure activities. A well recognized that leisure activities such as going out and joining a club are highly desirable, especially for young persons when growing up and learning social skills, whether they have epilepsy or not. It also adds to the quality of life for adults. Epilepsy presents no constraint to traveling and cycling, However, Individuals who have frequent seizures need to make sensible arrangements. Travelers need to take medication with them, preferably two sets, with one packed in the hand luggage and one in the checked bag. A letter from a doctor explaining the epilepsy and medication can be helpful. Travelers need to have appropriate travel insurance. Taking an epilepsy passport is useful. Cycling is not considered more dangerous to people whose seizures are well controlled than to anybody else, and normal precautions should be taken, which may include wearing a helmet. If seizures are active, busy roads must
be best avoided. Furthermore, the use of tandem or properly fitted three-wheeler bikes can enable people who have frequent and severe seizures to ride a bike, thus offering them the feeling of freedom that comes with this activity. Most accidents occur in the bath, while fishing, or from falling into water. Certain precautions need to be taken in or around water when a person is not seizure free. In case of swimming, supervision is necessary.

Rehabilitation

To achieve a favorable outcome with regard to psychosocial status, in addition to the neurosurgeon, neurologist, neuropsychologist, and nursing staff, several psychosocial team members are generally necessary. These may include a vocational rehabilitation counselor to assist in educational and vocational planning, a job site coach or mentor, an occupational therapist for training in independent living skills, a rehabilitation psychologist as a primary therapist for the patient and family members, an educational consultant or tutor, and occasionally an assistive technologist or rehabilitation engineer, who can recommend changes in work site procedures, physical modifications to a setting, or assistive equipment (e.g., dictating machines, palm-top computers, electronic toggle switches that cut off power) that will enable individuals to compensate for cognitive limitations and sometimes alleviate physical safety issues. In any case, referral to state or national departments of vocational rehabilitation or developmental disabilities can be very helpful in securing the services of personnel and funding that are critical to making functional gains in the community. These personnel and services are not available at most epilepsy surgery centers.

Programs and services for addressing social functioning may include various educational approaches, counseling, social skills training, cognitive rehabilitation, support networks, peer mentoring, vocational rehabilitation, and independent living programs. Many different health care providers and community-based rehabilitation and educational specialists may provide these services, depending on their areas of expertise and practice setting.
Introduction

(i.e., nurses, social workers, psychologists, psychiatrists, educators, vocational rehabilitation therapists, recreation therapists, and resource specialists).

(1). Social Skills and Cognitive Rehabilitation: Some people with epilepsy have impaired social skills and cognitive problems that may be due in part to underlying brain dysfunction complicated by the effects of seizures and medications on their brain function. Rehabilitation professionals with expertise in brain injury and epilepsy can teach social awareness and help patients to learn social skills and develop specific strategies to compensate for cognitive problems such as memory deficits. These services can be provided within many specialized epilepsy centers, outpatient rehabilitation settings, and community-based programs.

(2). Self-help and Support Groups: Self-help groups generally are groups of peers who have joined together to help each other with a common problem. Self-help groups can lead to improved social function by extending social networks, providing new opportunities for social learning, and changing cognitive perceptions about one's condition (Kurtz and Powell, 1987). Courses have been organized to standardize methods for self-help groups in Italy (Piazzini and Moretti, 1993). In the United States, support groups can be found in many communities, established by individuals, health care facilities, or nonprofit agencies. The Epilepsy Foundation and affiliates offer support groups and networks that are often targeted to specific groups such as parents, teens, and women. Increasingly, varied ways of providing support are being tried, such as phone networks, online forums, and individual mentoring.

(3). Vocational Rehabilitation: Because employment is a major problem for many people with epilepsy, all adolescents and adults should receive vocational counseling. Needs can include help in identifying medical and social factors that may affect employment, assisting people to
become job-ready, educating potential employers about epilepsy, assisting with job searches, teaching people to develop new vocational skills that will build on strengths, and teaching people new vocational skills and how to find reasonable accommodations.

(4). Independent Living Programs: Independent living programs aim at enhancing independence and self-determination for people with epilepsy. These programs may be provided in residential settings for people with significant difficulties living independently. Many people with less severe functional problems may still experience difficulties living on their own and can benefit from teaching and assistance in developing critical skills such as personal care, budgeting, home management, transportation, shopping, and other independent living skills. Funding for these services may be problematic, but some federal health or disability insurance or private agencies may help.

**Rational of the Present Study:**

Theoretical discussion and review of literature about epilepsy clearly reveals it to be one of the most common serious neurological disorders affecting about 50 million of people across the globe (WHO, 2005). In India, the prevalence rate is about 5 to 5.59 per 1,000 populations with no statistically different rates between men and women or urban and rural residence (Bharucha, 2003). Epilepsy accounts for 10% of the global burden in developing countries where in some areas as many as 80-90% of epileptics receive no treatment at all. Before the advent of modern neurology in 19th century, epilepsy, in general, was considered as a kind of witchcraft insanity or possession by devil or God. But now with the advancement in diagnostic facilities through structural and functional neuroimaging including CAT scan and MRI as well as video-telemetry and magneto encephalography and in treatment facilities, epilepsy has been established as a neurological disorder. Consequently, the modern era is marked by as expansion of interest in understanding the basic mechanisms underlaying seizures and epilepsies stimulated by developments in genetics, molecular
biology, neurophysiology, functional imaging and various neurochemical techniques for exploring the processes of excitation, inhibition, modulation, neurotransmission, and synchronization. These advancements have highlighted the enormous complexity of nervous system and probability that multiple elusive ‘genetic-molecular-metabolic mechanisms are contributing to wide range of epilepsy.

With the foundation of International League Against Epilepsy in 1909, the researches regarding the diagnostics classification started, and by now its 2010 version is operational. The task force group of ILAE also emphasized the relevance of comorbid psychopathology for diagnosis and classification of epilepsy and seizures. It opened the field for the investigation of psychopathology comorbid with epilepsy. Since then, epilepsy is taken as a disorder with both neurological and psychological complexities. The new ILAE definition acknowledges importantly the psychological and social consequences of epilepsy.

Epilepsy has many non-medical effects on the people with epilepsy, their family and community. It can have far reaching psychological and social ramifications and for some individuals these can be more debilitating than the seizures (Herman & Jacoby, 2009). Published literature covers variety of areas including psychopathological problems (e.g. mood disorders, anxiety, psychotic disorders); feeling of shame, fear and worry; low self esteem, problems related to education, employment, dating, marriage, child-bearing, poor quality of life, and stigma (Lai, 2007). Social problems are also important because they reduce a quality of life and contribute to mental health problems (Herman, 1991).

People with diagnosed epilepsy are a high risk group for variety of psychopathologies ranging from neurotic spectrum disorders to psychotic spectrum disorders as well as adjustment and psychosocial problems in daily life (Swinkels et al, 2001). Affective and anxiety disorders represent the most common comorbid disorders in epilepsy (Barry, 2003), depression and suicide are 4 to 5 time more common in patients with epilepsy than in
general population (McCagh et al, 2009; Gandy et al, 2012). Risk of suicide is greatest when epilepsy starts in adolescence and is also combined with history of psychiatric disturbance. There are also differences in psychopathological disorders in relation to the age of onset of epilepsy. The most commonly found psychopathological disorders in cognitively normal children and adolescents with epilepsy are ADHD, mood disorders, anxiety, obsession, and phobias (Beyenburg et al, 2005). Public stigma also determines the severity of psychopathological disorders. Poorly controlled and longevity of epilepsy have also been found associated with increased psychopathology (Plioply, 2003). Stress has also been reported to aggravate the magnitude of both the seizures and comorbid psychopathologies (Jones & O'Brien, 2012). Treatment of epilepsy with comorbid psychopathological disorders is a challenge because the specific aspects of both conditions have to be carefully managed for optimal outcome. Review of literature also reveals that most of the studies on psychopathology comorbid with epilepsy have been conducted with univariate approach i.e. epileptics have been compared with normal controls on either one or two disorders such as anxiety and/or depression. Whereas fact is that psychopathological disorders are not absolute rather have overlaps with each others. The present study is an attempt to have comprehensive understanding of comorbid psychopathological disorders including neurotic spectrum disorders (somatic complaints, anxiety, anxiety-related disorders, and depression); psychotic spectrum disorders (schizophrenia, mania, paranoia, borderline features); behavioral or impulsive disorders (antisocial personality, drug problems); interpersonal styles (dominance, warmth); and treatment consideration variables (stress, aggression, non-support, suicide ideation, treatment rejection/compliance) with epilepsy simultaneously adopting multivariate approach.

In many form of epilepsy, cognitive functions are altered during seizures or after seizures. However, cognitive dysfunction is only one among other dysfunction is only one among other epileptic manifestations; and is
not recognized or considered main symptoms. Cognitive dysfunctions may or may not comorbid epilepsy depending mainly on the age of onset and cortical damage. Neither psychological consequences of diagnosis nor side-effects of treatment can be blamed, it has to be suggested that direct cognitive effects of epilepsy exist more often than is usually acknowledged (Austin et al, 2001). On the other side, cognitive dysfunctions may have negative consequences contributing in the magnification of some psychopathologies. Cognitive dysfunctions in the form of memory impairments, mental slowing, and attentional deficits are most frequently reported cognitive disorders comorbing epilepsy (Donati et al, 2006; Rasche et al, 2010; Berg et al, 2012; Cerminara et al, 2013). The present study is also oriented to examine the cognitive functions/dysfunctions (memory and attention) combined with psychopathologies in epileptic patients. Another merit of the present study in this regard is that it will also examine the visuomotor coordination between left and right hemispheres among the epilepsy patients.

Epilepsy exposes its patients to a wide range of psychosocial consequences which sometimes can be more damaging than the medical difficulties they deal with. Person with chronic illness such as epilepsy face life stressors and some additional stressors such as treatment cost, family burden, adjustment problems, pain and side-effects of medications, adjustment resources and coping strategies can play a mediating role between stressors and the illness, (Roesch & Weiner, 2001). In this regard researchers have reported that patients with epilepsy who adopted problem-focused and cognitive restructuring coping processes have better outcomes pertaining to both epilepsy specific manifestations and psychosocial problems (Livneh et al, 2001; Hosseini et al, 2010; Testa et al, 2010). The present study also orients to study the psychopathology, cognitive functions, and coping among epileptic patients. Methodologically the present study is meritorious in terms of using multivariate techniques in understanding the relationship among psychopathology, cognitive functions, and coping.
Psychopathology, Cognitive Functions, and Coping among Epileptic Patients

(Factor Analysis), differentiation between epileptic patients and normal controls (Discriminant Function Analysis), and grouping of epileptic patients in terms of variables of the psychopathology, cognitive functions, and coping (Cluster Analysis).

Problem:

The present problem is stated as:

“PSYCHOPATHOLOGY, COGNITIVE FUNCTIONS, AND COPING AMONG EPILEPTIC PATIENTS.”

Objectives

Main objectives of the study are:

1. To examine the extent of psychopathology which comorbid with epilepsy.
2. To examine the cognitive functions among epileptic patients.
3. To examine the ways of coping, the epileptic patients use for coping with disease-specific and general life stress.
4. To compare the epileptic patients with normal subjects in terms of their mean scores on measures of psychopathology.
5. To compare the epileptic patients with normal matched subjects in terms of their mean scores on measures of cognitive functions.
6. To compare the epileptic patients with normal matched subjects in terms of their mean scores on measures of coping.
7. To examine the joint contribution of measures of psychopathology, cognitive functions and ways coping in discriminating the epileptic patients and normal subject’s group.
8. Discovering the overlapping factors among the measures of psychopathology, cognitive functions and coping in the data from epileptic patients.
9. To find out the clusters of epileptic patients on the basis of similarity on measured variables
Hypotheses:

Specific hypotheses are:

1. Epilepsy patients tend to score high on measures of neurotic spectrum disorders than their counterpart normal subjects.
2. Epilepsy patients tend to score high on measures of psychotic spectrum disorders than their counterpart normal subjects.
3. Epilepsy patients tend to score low on measures of cognitive functions than their counterpart normal subjects.
4. Epilepsy patients tend to score low on problem-focused coping and high on emotion-focused coping measures than their counterpart normal subjects.
5. Some measures of psychopathology, cognitive functions and coping are likely to contribute jointly in discriminating the epilepsy patients from their counterpart normal subjects.
6. Some measures of psychopathology, cognitive functions and coping are likely to yield some overlapping factors in epilepsy data.
7. It being an exploratory study, it was hypothesized how many clusters of epileptic patients will emerge.