INTRODUCTION

Epilepsy

Johan Walton (1985) defined epilepsy as "a paroxysmal and transitory disturbance of the function of the brain which develops suddenly, ceases spontaneously and exhibits a conspicuous tendency to recurrence".

Classification:

Epilepsy can be classified in several ways e.g. by clinical events (usually seizure type), electroencephalographic (EEG) changes, etiology, pathophysiology, anatomy or age. Since epilepsy is often best regarded as a symptom rather than as a condition per se, and since the essential pathophysiology is often obscure, classification is inevitably arbitrary. In 1969, the International League Against Epilepsy (ILAE) attempted to introduce a scheme for universal application. This scheme, revised in 1981 and widely adopted, is a classification of seizure type in which EEG data are taken into whereas etiology, age and anatomical site are ignored (*Shorvon 1990*).

ILAE classification of seizure type (1981):

I- Partial seizures:

A- Simple partial seizures:

(1) With motor signs.

(2) With somatosensory or special sensory hallucinations.

(3) With autonomic symptoms and signs.

(4) With psychic symptoms.

B- Complex partial seizures:

(1)Simple partial onset followed by impairment of consciousness.

(2)With impaired consciousness at onset.

C- Partial seizures evolving to secondary generalized seizures:

(1) Simple partial seizures evolving to generalized.

(2) Complex partial seizures evolving to generalized.

(3) Simple partial seizures evolving to complex partial seizures evolving to generalized.

II- Generalized seizures:

A- (1) Absence seizures

(2) Atypical absence.

B- Myoclonic seizures

C- Tonic seizures

D- Clonic seizures

E- Tonic-clonic seizures

F- Atonic seizures

III- Unclassified epileptic seizures:

Seizures which cannot be classified because of incomplete data.

ILAE classification of the epilepsies and epilepsy syndromes and related seizure disorders (1989):

This classification takes into account seizure type, EEG, and prognostic, pathophysiological and etiological data. It retains the division of epilepsy into generalized and partial (now called localization related) categories, with each category subdivided into symptomatic and idiopathic varieties. Two new categories are added, epilepsies and syndromes undetermined, whether, focal or generalized, and special syndromes. This scheme is complex and may well confuse non-taxonomists, but it is a serious attempt to incorporate more than simple seizure type data into comprehensive classification.

Etiologically, seizures disorders are often classified into 2 classes, the "idiopathic" (essential or primary) epilepsy and the "symptomatic" (acquired or secondary) epilepsy. In the "idiopathic" type the cause is known, but a significant genetic component is presumed. It is better termed as cryptogenic epilepsy. In the symptomatic "type a cause is identified or strongly suspected (*Kenpe et al., 1982*).

Etiology :

Role of genetic factors :

Genetic factors may be involved in all forms of epileptic seizure disorder, but their significance varies from one epileptic condition to another (*Doose et al., 1973*).

A Multifocal model of inheritance has been proposed by *Eva-Andermann (1980)*. This model permitted separate calculation of seizure heritability . In the focal epilepsy group, the heritability of total EEG abnormalities was calculated to be 45 % and that of seizures only 13%. In the primary generalized epilepsy group, the heritability of total EEG abnormalities was calculated to be 73% and the heritability of seizures was 62% . This concept of multifoctorial inheritance has been supported by work of *Eeg-Olofsson et al. (1982)* , who investigated the human leukocyte antigen complex in epilepsies .

Animal models for epilepsy present firm evidence that genetic factors influence the hypersensitivity of neurons, disturbe aminoacids, enzymes and hormones (*Anderson et al., 1990*).

Plomin et al., (1990) reported that dominant traits were less severe and had later onset than recessive traits, and added that, dominants affect cell surface phenomena (such as receptors), or structural proteins (such as collagen) rather than enzymes.

Degenerative diseases:

Most of neurodegenerative diseases and syndromes are usually accompanied by epileptic seizures (*Barolin and Karbowski, 1973*).

Neurodegenerative diseases and syndromes may be superimposed on well defined epileptic syndromes, especially with infantile spasms

(West's syndrome) and Lennox-Gastaut syndrome might be due to a special genetic predisposition (*Mckusick 1989*).

Craniocerebral trauma :

Convulsive seizures are an infrequent symptom of the acute phase of a head injury. (*Jennett, 1975*) , . This may occur immediately after or within the first few days of the injury. In these cases, the seizures are related to the acute brain damage or to the presence of Intracerebral hematomas or infection (*Feeney and Walker, 1979*). In most patients, seizures do not develop until several months after injury; 6-8 months is the most common interval. (*Jennett, 1975*). He also added that, the seizures that follow head injury may be of any type except the classic absence seizure , and they were more often generalized than focal.

The exact incidence of seizures following head injury vary from 2.5 - 40%, the high incidence reported when there has been penetration of the dura and laceration of the underlying cortex with the formation of a cerebromingeal scar. (*Freeney and Walker, 1979*).

The risk of early seizures is about 25% of those within brain contusion or hematoma (*Brooke, 1988*). In children, early seizures appeared in about 30% of those with cerebral edema (*Annegers et al., 1980*).

The risk factors for delayed seizures include Intracerebral hematoma (14 - 35% of adults, 7-17% of children) (*Brooke, 1988*) and in

depressed skull fractures, the rate was 17% (*Annegers et al., 1980, Walker, 1962*).

CNS Infections :

CNS infections may cause epileptic seizures at any age. Hemiplegia-Hemiconvulsion-Epilepsy (HHE) syndrome is mostly of infections origin during the first 3 years of life (*Pinsard et al., 1985*). Hemophilus influenza meningoencephalitis is a particularly common cause of seizures in the first years of life. Moreover, infections encephalitis associated with seizures or followed by an epileptic seizure disorder may also occur in measles, chickenpox, whooping cough and mumps (*Johnson, 1982*).

Tuberculous meningitis of early childhood can lead to subsequent chronic seizure disorders (*Walker, 1962*) *Pinsard and Co.-workers (1985)* demonstrated that fungus diseases of CNS (blastomycosis, histoplasmosis) may occasionally give rise to seizures .

AIDS is capable of attacking the brain directly resulting in sub-acute AIDS encephalitis but, epileptic seizures are rather uncommon in the neurological symptomatology of AIDS. (*Niedermeyer, 1991*).

Cerebrovascular disorders :

- *Cerebrovascular accidents* : Seizures of chiefly focal motor character may preceed a hemiplegic stroke by weeks or even by months or

years. Strokes are complicated in seizures in 12.5%-20% of cases (*Lesser et al., 1985*).

- *Chronic Cerebrovascular disorder:* In patients with chronic arteriosclerotic changes, epileptic seizures have been reported to occur in 4% of cases, grand mal attacks are most often noted, followed by focal motor seizures (*Lesser et al., 1985*).

- *Sub-arachnid hemorrhage due to intracranial aneurysms:* Epileptic seizures have been reported in 12.5% of these patients during the acute stage (*Walton, 1953*). Following surgery the prevalence of seizures may be high (*Scott and Cabral, 1975*).

Malignant hypertension :

This disorder is not seldom associated with grand mal seizures, it may be a contributory factor in epileptic seizures disorder (*Pinsard, et al., 1985*).

Metabolic and toxic CNS disorders :

Hypoglycemia induced convulsions of the grand mal type are fairly common in hyper-insulinism (functional or neoplastic with pancreatic islet cell tumor) and also in insulin-induced hypoglycemic coma in patients with diabetes mellitus (*Niedermeyer, 1991*), but myoclonus was found in 90% and grand mal in 3% of hypoglycemic

coma states when blood sugar level is about 1.5-2.5 mmoL/L or less. (*Shucard et al., 1985*)

Hyperglycemia is less epileptogenic than hypoglycemia, epileptic activity may be quite prominent in nonketotic hyperglycemia and a status-like type of focal motor seizures is most often found when blood sugar level exceeds 1000 mg/100mL. *(Maccario, 1968*).

Severe hypocalcemia is highly epileptogenic and may cause grand mal seizures when serum calcium level of 5.0-6.0 mg/100 ml in humans, in addition hypomagnesemia might play a contributory role in epileptogenesis, especially in patients with chronic alcoholism (*Delorenzo, 1986*) but in *hypercalcemia*, grand mal with an associated occipital lobe spike focus in the EEG has been reported at calcium level of 16 mg/100 ml (*Barolin and Karbowski, 1973*).

Hyponatremia may cause convulsions in neonates, and occasionally in children and adults. *Severe states of adrenocortical insufficiency* (Addison's disease) are associated with encephalopathies and occasional convulsions, status epilepticus grand mal has been reported in a case of compulsive water intake (*Zwang and Cohn, 1981*).

In renal failure, it was found that one third of patients develop seizures (*Loke et al., 1961*), but in hepatic encephalopathy epileptic manifestations are much less common than in renal failure (*Niedermeyer, 1991*).

Incidence :

Most studies have found incidence rates of 26-70 per 100,000 per year, and point prevalence rates of 4-10 per 1000 in general population (*Juul - Jenson and Fldspang, 1983*).

Sex incidence:

According to the older literatures, epilepsy was more common in males than in female sex. This was due to the inclusion of post-traumatic epilepsy , since males are more prone to head injury. *Benna et al., (1984)* demonstrated that males were affected slightly more frequently than females (13:12).

Lennox and lennox (1960), found that, under the age of 5 years there were 105 females for every 100 affected males, and over the age of 20 years male: female incidence was 100:59.

Age of onset :

Seizures may begin at any age, but most frequently start in the first two years of life and during adolescence. (*Van den Berg and Yerushalamy 1969*). Age specific rates are consistently lowest during the adult years, and several recent studies show an increasing incidence in the elderly (*Forsgren, 1990*).