Chapter I

INTRODUCTION
In the medieval times, childhood was not regarded as a psychologically distinct period. Children were considered as underdeveloped adults at the disposition of the society in which they were born. It was only by the 18th and 19th century philosophers could identify that children are not underdeveloped adults, but they are unique and have unique psychological, educational and physical needs. By mid 19th century new approach to understand childhood evolved. More scientific studies were initiated. Great scientists and philosophers contributed to these new efforts. Charles Darwin was interested in the study of the developmental process, Stanely Hall tried to understand children in different age groups. Alfred Binet studied intellectual development in normal and subnormal children and Sigmund Freud from his clinical experiences suggested that early childhood experiences results in unusual behavior in adulthood.

In India, earliest document on child development was given in Ayurveda. It considered feeding practices during the early phase of development is the central factor for the formation of personality. According to them physical and mental disturbances were caused by the different positions of grahas. They recommended toys that stimulated cognitive functions like memory, intelligence and language.

In the present era we have new approaches and knowledge regarding child development. As children grow, from very early stages, brain development as well as changing social influences result in typical healthy cognitive, sensory-motor and socio-emotional functioning. Most of the children acquire adequate skills, thus becoming good citizens and achieving desired goals. Even though this is the situation for majority, a few have to face both biological and environmental risks which disrupt their normal
developmental course. This disruption can be at any level, from neural activity in central nervous system and peripheral nervous system, to modulation of visual attention, to control of cognitive or emotional events, to broad and easily observed behavior. To understand the nature and consequence of psychopathology we have to understand the variations that are taking place within and between the different levels of brain activities, cognitive, emotional and behavioral activities going on within the individual. These disruptions in normal development are responsible for the emergence of a wide range of symptoms resulting in mental and/or behavioral disorders or developmental psychopathologies.

1.1. DEVELOPMENTAL PSYCHOPATHOLOGY

The integration of child psychiatric and normative developmental research resulted in a new discipline—developmental psychopathology in 1980’s. The landmark papers by Cicchetti & Gardner in 1984, Rutter and Garmezy in 1983 and Sroufe and Rutter in 1984 introduced a new branch developmental psychopathology, as an integrative discipline seeking to unify, within a developmental, lifespan framework, contributions from multiple fields of studies including biology and developmental sciences.

Sroufe and Rutter in 1984 defined developmental psychopathology as “the study of the origin and course of individual patterns of behavioural maladaptations, what ever the age of onset, what ever the causes, whatever the transformations in behavioural manifestation, and however complex the course of developmental pathology may be.” In other words developmental psychopathology tries to understand psychopathology in relationship to normative adaptation, integrating knowledge across scientific disciplines
at multiple levels of analysis and multiple domains, rather than exposing a single theory that would account for all developmental phenomena.

The term developmental disorders of childhood refer to disorders of psychological development (WHO, 1992) that are usually first diagnosed in infancy, childhood or adolescence (APA, 1994). These disorders manifest with deficits in the traditional behavioural domain of cognition, language, visual-spatial functions, attention and socialization. Both DSM IV TR and ICD 10 contain categories for specific developmental disorders or developmental psychopathologies. The delay in development cannot be attributed to any other disorder or lack of opportunity to learn. In ICD 10, these developmental psychopathologies are divided into specific developmental disorders of

- Social skill
- Speech and language
- Motor functions.

In DSM IV this group of disorders are classified as

- Learning disorders
- Communication disorders
- Motor skill disorders

According to DSM IV Psychopathologies can be specific to children and same for adults.

*The disorders of Infancy, Childhood or adolescence*, includes disorders that are specific, like:

- Mental retardation
• Learning disorder
• Pervasive developmental disorder
• Attention deficit and disruptive disorder
• Elimination disorder
• Separation anxiety disorder
• Reactive attachment disorder of infancy or early childhood.

*The Disorders of both children and adults* manifests similar disorders for both, like:

• Substance abuse
• Schizophrenia
• Mood disorders
• Anxiety disorder
• Eating disorder

Causes of developmental psychopathology are multiple, as individuals grow in different contexts that exist together. The different contexts within which an individual develop are

• Intra personal context
• Inter personal context
• Super ordinate context
• Organic context

In intra personal context the major concern is variables within the person-personality characteristics, cognition, emotions etc. In inter personal context the concern is on the type of interaction among individuals within family or among friends or peer group. In super ordinate context the group, community, social class and culture within which the
individual grows are given importance. In organic context characteristics of human body that are relevant to the understanding of deviant development is emphasized. Some of the causal models share common features, some are complementary, and each has merits but is not fully satisfactory. Based on organic context there is a medical model. Medical model replaced the demonology of middle ages. Organic factors have been implicated in the causation of certain developmental psychopathologies like autism, hyperactivity and learning disabilities in children; but evidence varies in definitiveness. (Plante, et al. 1991, Monjauze, et al. 2005, Parkinson, 2002). The organic context interacts with the intra personal and inter personal context. To understand the causes of developmental psychopathologies we should have the knowledge regarding the interactions between the four contexts within which the person is living. Developmental psychopathology is not a theory but it is an approach to understand the emergence of psychopathology over the life span.

The literature provides ample evidence that psychopathology is more common in children with epilepsy than in general population. (Mc Dermott,1995, Rutter 1970, Cornaggia, & Gobbi, 2001, Rodenburg, 2005). Epilepsy is a syndrome encapsulating several diseases. It is the most prevalent neurological disorder. Complex partial seizures of temporal lobe epilepsy (TLE) have special neuropsychiatric significance, notably in relation to anxiety disorders, cognitive impairment and psychosis. (Rizzo, M. 2004).

1.2. EPILEPSY

Epilepsy is one of the oldest CNS disorders described, and is generally considered to be the most stigmatized brain disorder. In developing countries the stigma associated with this disorder is very high and so the patients do not take proper treatment. As a result
it becomes uncontrolled and affects cognitive, behavioural, social, educational, and occupational domains. Most of the cases with epilepsy are identified during childhood and it has been observed that there is a difference in the cognitive performance when compared to peers. Many studies have shown that cognitive and behavioural dysfunctions or impairment is found due to epilepsy. Epilepsy is characterized by seizures, the main finding being specific Electroencephalographic (EEG) abnormalities, and, in many patients with TLE, pathologic changes in the temporal lobes, namely mesal temporal lobe involvement indicates the role of limbic system. Because of this, many patients with epilepsy have behavioural and cognitive problems. The nature of epileptic attack depends on the area of brain affected and functions that area normally controls, as well as the extent of discharge to spread to other areas of the brain.

1.2.1. Definition

The word Epilepsy came from the Greek word Epilepsia, it means “taking hold of something seizing the subject as though that something were outside himself”. Epilepsy is the liability to recurrent epileptic attack, the outward effects of transient disturbances of brain function taking many different forms, often but not invariably with loss of consciousness. Epilepsy is the tendency to recurrent motor, sensory or behavioural seizures caused by a discrete electrical brain abnormality.

1.2.2. Prevalence

About 5% of the population experience seizure, but only 0.5% has epilepsy. To be specific 4-10/1000. In India the prevalence rate per 1000 population and with 95% confidence interval is:
In a hospital based study conducted in South India by Murthy et al (1999), showed that idiopathic localization related epileptic syndromes accounted for 1.8%, of child hood epilepsies and 0.7% of the study population. The figure is little higher (3.9%) in a study by Shah et al conducted among pediatric patients. In US epilepsy prevalence rates per thousand people ranges from 3.1 to more than 6. And it is the 4th most common neurological disorder. (Lazak, 1995)

Recent reports of prevalence of active epilepsy in India are 3.6 to 8 in 1000 (cited in Chopra and Sawhney, 1999).

Epilepsy, also called seizure disorders, is characterized by recurrent seizures. It is associated with structural or biochemical brain abnormalities. It is estimated that 1% of the general population has epilepsy. This disorder occurs more commonly in boys than girls. About 40% of individuals with epilepsy between the ages of 4 and 15 have one or more additional neurological disorders. The most common ones are mental retardation, speech-language disabilities, and specific learning disabilities. In fact, learning disabilities are more prevalent in individuals with epilepsy (approaching 50%) than in the general population.
1.2.3. Comorbidity

In children with new onset idiopathic epilepsy, Hermann et al (2006, 2007) identified two groups of children who presented with comorbid conditions that significantly affected cognitive status, children with ADHD and children with academic problems.

1.2.4. History

History of epilepsy is as long as history of mankind. The magicians of ancient Greece recognized different seizure types and attributed it to individual gods. Ancient Babylonians have written about epilepsy more than 4000 years ago. Ancient Indian Medicine (Ayurveda, Charaka Samhita) also has given an account of symptomatology, causes, diagnosis and treatment of epilepsy. Hippocrates wrote the first monograph on Epilepsy entitled, "On the Sacred Disease". He believed it can be treated by controlling diet and drugs and not by magic and sorcery. Galen was the person who mentioned pathophysiology of brain, spinal cord and nerves in the causation of epilepsy, but was not able to make any conclusion regarding it. He was the person who divided epilepsy into idiopathic epilepsy and symptomatic epilepsy. The terms persisted into 19th century. Aretaens gave a classical account of aura of epilepsy. After the period of Hippocrates there was the Dark Age in the field of medicine and psychopathology. Later during the Renaissance period new ideas and scientific inquest were made. Jean Fernal rejuvenated the interest in studying epilepsy. After him Paracelsus, Erastus etc were interested. Erastus identified and reported that aura is the commencement of a fit and not the cause, but this finding was accepted only in 19th century. In 15th century, Thomas Willis
introduced the role of Central Nervous system as the seat and source of all types of epilepsy.

The modern period of epilepsy can be dated back to 1827 when Bravis presented a paper on Partial and Generalized Epilepsy. After this till 1870’s no effective contributions were made, even though many studies and theories on epilepsy were put forward. By this time neurology emerged as a separate field and epilepsy was included as a brain disorder. Hughlings Jackson carefully analyzed cases and from his observations he formulated the modern definition of epilepsy as “an occasional, excessive, and disorderly discharge of nerve tissue.” He also emphasized the role of location in the brain. In 1920 EEG was developed by Hans Berger which led to a new mode of research in this area. As far as the history of treatment is considered the landmark event was in 1857. Bromide was introduced and was the first effective drug. From 1912 Phenytoin and Phenobarbitone were used. None of the drugs were found effective in eradicating epilepsy.

Many famous people have suffered from epilepsy, to name a few; Vincent van Gogh (Dutch Painter), Julius Caesar(Roman Statesman), Dostoyevsky(Writer), Alfred Nobel(Scientist), Lord Byron(English Poet), Alexander the Great, Hermann von Helmholtz (German Physicist), Lenin….etc

The International League against Epilepsy (ILAE) is an international organization or task force that is committed to fight against Epilepsy.
1.2.5. *Types*

In 1989, ILAE has given a new classification system of epilepsies. In 1981, seizures were classified as General and Partial. In 1989, epilepsy was classified into Generalized and Localized.

![Classification given by International League against epilepsy in 1981.](image)

**Figure: 1.1:** Classification given by International League against epilepsy in 1981.
In Generalized epilepsies, epileptic activity involving wide areas of cerebral hemispheres simultaneously form the onset of the attack with no evidence of an anatomic or functional focus. In partial epilepsies the seizure activity commences in one part of the cerebrum. There are a number of types of generalized seizures. Primary generalized seizures manifest themselves immediately and there is a simultaneous spread of the seizure throughout the cortex.

In absence seizures, formerly known as petit mal, there is an abrupt onset with cessation of ongoing activity and the person demonstrates vacant appearance accompanied by or without upward movement of eyes. It may last up to 30 seconds, and suddenly stops. Automatism like, lip smacking or chewing may occur. 80% of patient with this type of seizure fall in the age group 2.6yrs and 9 yrs. Incidence rate in children and adolescents range from 0.7-4.6/100,000.

Generalized Tonic Clonic seizure (Grand mal) is rarely preceded by a warning or aura. There may be a prodromal change in mood. There is a sudden contraction of muscles with tongue biting, urinary incontinence and absence of breathing, and this is then followed by clonic convulsive movements which are symmetrical, rhythmic and which decrease in amplitude over time. Seizures last no more than 2-3 minutes. The patient remains unconscious for a period after the attack and some may go to deep sleep. Tonic as well as clonic seizures may occur in the same individual.

Myoclonic seizures takes the forms of single / multiple jerking movements which may be generalized to the face, trunk or one or more limbs or muscle groups. These contractions occur especially on falling asleep or walking and occur as part of an
idiopathic generalized epilepsy or as part of a mixed seizure disorder such as the Lennox Gastant Syndrome.

Atonic seizures are characterized by a decrease in muscle tone such that there will be head drop, jaw slacking and the person will fall to the ground. These are often known as drop attack but need to be distinguished from drop attacks that occur in narcolepsy/cataplexy.

In partial seizures electrographic activity is initiated in a part of the cerebrum. The clinical characteristic of the seizure reflect the part of the brain affected and a wide variety of symptoms occur.

Simple partial seizures (SPS) are caused by a local discharge that results in seizure symptoms appropriate to the function of the discharging area of the brain, without impairment of consciousness. The same symptoms can occur in both simple and complex partial seizures. Abnormal inter ictal EEG are found in 80%-90% of patient with SPS. Focal spikes or sharp discharges, slowing or suppression of the normal background are the usual abnormalities. There is an absence of focal findings in many patients as spikes are intermittent phenomenon and if originating from small areas of the cortex it may not be coded.

Complex partial seizure (CPS) has the central feature, “impairment of consciousness”. Also one can find types of simple partial onset and automatism. The sudden loss of consciousness can be accompanied by the loss of positional tone and so it is called “drop attack”. Inter ictal manifestation of CPS includes focal spikes, sharp waves and slowing. These abnormalities are found most often in the anterior temporal region. Abnormalities may be localized or bilateral. The hallmark of the partial seizure is a specific motor,
sensory or neuro psychiatric symptoms that help to pinpoint the onset of the seizure to one area of the brain. The origin can be from temporal lobe, frontal, parietal or occipital lobe. Most commonly found partial seizure is temporal lobe epilepsy.

**Temporal Lobe Epilepsy (TLE):**

In TLE, seizure and aura will involve simple or complex motor and sensory symptoms like speech defects, perceptual abnormalities, and temporary cognitive impairment with emotional involvement and forced thinking or thought insertion. Symptoms of TLE overlap with those generated by amygdale and the central autonomic network (Hypothalamus, mid brain, periaqueductal gray matter, pons and medulla). This network is thought to be responsible for automatic features of epilepsy like hyperventilation, sweating and flushing. Approximately 60% of complex partial seizures have their origin in the temporal lobe. TLE had two subclasses in terms of the anatomical structures involved, they are mesio-basal(MLS) and lateral neo cortical(LTNS) types but the symptomatology overlaps.

Meso basal Limbic Temporal Lobe Epilepsy (MLS) involves the hippocampus and is also known as hippocampal seizures (HS). Most common aura is that of a rising epigastric sensation, abelching and automatism of MLS. Vocalizations are common and recognizable words suggest a focus in the non dominant temporal lobe. Post-ictal confusion is typically longer and also headaches occur.

In Lateral temporal neo cortex temporal lobe epilepsy (LTNS), consciousness is usually preserved. Aura involves structured visual, auditory, gustatory and olfactory hallucinations and illusions for size. There is a rapid spread of seizure activity to neighboring areas. In majority of cases there is a structural pathology.
**Other than temporal lobe epilepsy (O-TLE):** Origin of the seizure other than TL is known as O-TLE (FLE, PLE and OLE).

*Frontal lobe epilepsy (FLE)* or Jacksonian seizure (simple type) have the origin from primary motor cortex. Complex seizures indicate the involvement of supplemental motor area. Agitation is a very specific symptom of seizures from orbito- frontal and fronto-polar regions. There is an acute onset of seizure of short duration. It is characterized by short tonic or dystonic movements and vocalizations and automatisms. Tendency to occur in night is high, about 30% of adults with partial seizures have FLE.

*Parietal Lobe epilepsy (PLE)* is seen only among 5% of all people with partial epilepsy. Sensory auras are common and at times even painful sensations are reported. Some of the interesting sensations are moving of limb, absence of limb, pain headaches etc. Spread occurs from the superior lobule to the frontal lobe and from inferior parietal lobule to the temporal lobe.

*Occipital lobe epilepsy (OLE)* makes about 8% of partial epilepsy. They have visual hallucinations or blindness. Multicolour circular patterns are typical of occipital lobe epilepsy. Rapid blinking and eyelid flutters have been reported to be reliable indicators of OLE. Progression to TL or FL is very common.

According to experts, only about 40-50% of children with epilepsy can be put into epilepsy syndrome. When these children cannot be put into or classified into an epilepsy syndrome, their epilepsy must be classified according to the seizure type or types that the child is experiencing, and this is used as the best basis for prognostic judgment.
1.2.6. **Etiology**

A seizure can arise from any condition that heightens the excitability of brain. The underlying cause of epilepsy can be brain damage from birth trauma, head injury, tumor, infections, metabolic disorders, cerebro-vascular accidents, a deteriorating brain disease or a host of other conditions (cited in Lezak, 1995). In many cases a clear physiological or anatomical abnormalities are rarely identified and such cases where there is unknown etiology are called idiopathic epilepsy. If there is a known cause then it can be called as symptomatic. In some cases intake of alcohol, physical debilitations like illness, and lack of sleep, physical exhaustion and emotional stress can trigger epilepsy.

Epilepsy developing in childhood after the 1st year of life may be due to any of the congenital or acquired lesions just mentioned. In addition, constitutional or idopathic epilepsy accounts for a proportion of cases and the attacks are sometimes associated with lesions of one temporal lobe which can be localized only by electro encephalograph (EEG). (Bannister, 1973). EEG is the most frequently used method to identify the seizure onset and propagation. Now, more effective video EEG, MRI, PET scan techniques are used. Children with intractable epilepsy are at considerable risk for cognitive impairment, school failure, behavior and mental health problems and overall compromised quality of life. It influences the development of cognitive functions during the period of brain plasticity.

1.2.7. **Risk factors and vulnerabilities**

**Genetic Predisposition**: Epilepsy tends to run in families, in those children who begin epilepsy in infancy or childhood.
**Cerebral insults**: Children who have penetrating head wounds develop epilepsy with varying intensity depending on the severity of the insult. A decrease in the brain volume is found in people with epilepsy.

**Precipitating conditions**: Epileptic seizure can occur without apparent provocation; but intake of alcohol, lack of sleep, physical exhaustion etc can result in seizure.

1.2.8. **Cognitive impairment in epilepsy**

The association between cognitive impairment and epilepsy is reported as early as in the literature of 19th century. Among the institutionalized population, an association between intellectual decline and epilepsy was found. Even now it is not clear whether epilepsy itself or the antiepileptic drugs are contributing to the cognitive impairment.

Epilepsy is associated with a mild to moderate deterioration in cognition in those with previously normal performance, but defining the exact nature of the deficits has proven elusive. One reason for this is that these deficits are multifactor; it may depend on the site and severity of neuronal damage, developmental effects and effects of medicines. Some people develop cognitive changes even with the mildest forms of seizure disorder. Some studies have shown that poor attention, memory and poor processing speed as a result of carbamazepine treatment in partial seizures (Engelberts, 2002)

The causes of cognitive impairment are three fold, they are:

1. Underlying disease process
2. Brain damage due to seizure
3. Anti epileptic drug.

At the other end of the epilepsy spectrum, patients with recurrent refractory seizures certainly suffer cognitive deficits that may persist after successful surgical treatment
Duration of illness and frequency of seizures are related to deterioration in cognitive function, but not all deficits nearly correlate with seizure burden. Age of onset if early is associated with more severe impairment, particularly in IQ rather than memory alone. In TLE, there is impairment in declarative memory, showing difficulty in naming things; these children are spared of problems with attention, concentration and executive functions. Comparisons of cognitive functions in TLE and other types of focal seizures are rare. A study by Hernandez,(2002) showed that children with FLE had deficits in planning and impulse control not seen in those with TLE. In cases with medial temporal atrophy, cognitive impairment particularly memory defects are reported. How far TLE patients are affected with memory impairment is an area yet to be explored. Dam(1990) concludes that children with epilepsy have poorer concentration and mental processing.

Some tendencies for cognitive dysfunction in patients with Epilepsy noted are:

a) **Overall levels of cognitive functioning**: With brain wave abnormalities it is reported that scores on cognitive tests to decline based on the severity of damage. As the rate of seizure increases the damage also increases there by the scores on neuropsychological tests also decreases. Generalized cognitive deficit is seen in children with generalized seizure than those children with focal seizure. Even when there is no structural damage, low blood flow negatively affects cognitive functioning. (Lezak, 1995)

b) **Focal seizures and cognitive dysfunctions**: Focal seizure involves one side of the brain and so will have similar conditions seen in patients who have lesions to the same
foci but have no seizures. Therefore people who have left hemisphere epileptic foci tend to have impaired verbal functioning including verbal memory, abstract reasoning, slow right hand finger tapping, visuo-perceptual defect, constructional disabilities etc. With right hemispheric damage however the general deficit of preservative errors, problems with concept formation and flexibility can be observed.

c) **Attention and memory disorders**: Lezak (1995) reports that some epileptic patients show lower scores on digit span, Arithmetic and digit symbol than comprehension, block design and object assembling test of WAIS. These tests asses the attention, memory and learning of the patients. It is found that people with TLE have problems in learning and memory.

Carmody *et al* (2006) reports that, greater activation of left superior temporal and left supra marginal gyri is associated with attention task. The fMRI study also sheds light to the type of risk and its relation to the pattern of brain activation. They report that medical risk is related to parietal cortex activation while environmental risk is related to temporal activation. Early risk is related to less mature patterns of brain activation, including reduced efficiency of processing and responding to stimuli.

One of the most notable effects of cognitive functioning in children with epilepsy is memory impairment. This impairment can range from poor concentration and minor forgetfulness to gross clouding of consciousness and disorientation.

Epilepsy might impact on learning in other ways. Daytime seizures can affect learning by reducing alertness and by interfering with short-term information storage and abstraction. Frequent and uncontrolled seizures impair learning new information due to
the amount of time that the individual is unaware of the environment. Night-time seizures can disrupt the consolidation of memory and affect language functions.

In the present day, cognitive impairment among children with epilepsy is more significant as it affects all areas of functioning including academic performance, occupation, interpersonal relations and also recreational activity. In children it will affect the learning process thereby affecting the academic performance and school adjustment. Stores (1973) have reported the negative effects of inattention on school success of children with epilepsy. It can also negatively influence other cognitive skills like short term memory and information processing. Children with epilepsy have been reported to be poor in learning verbal materials in addition to memory impairment, poor academic performance and embarrassment in social situations. (Quadfasel et al, 1955, Mayeux, et al. 1980) Problems are seen in speech and language (Geschwind, 1984).

The study by Reminger, (2004) used quantitative volume estimates of the hippocampus based on structural magnetic resonance imaging (MRI) to predict memory performance of individuals with epilepsy of temporal lobe origin (TLE). Twenty individuals with TLE completed standardized neuropsychological tests and a quality of life inventory, and participated in a brain MRI protocol designed to obtain high-resolution images of the hippocampus. The combined volume of the left and right hippocampi was found to be the best predictor of objective verbal memory performance. This finding is consistent with the functional adequacy model of hippocampal function. In contrast, the asymmetry between right and left hippocampal volume was the best predictor of subjective ratings of cognitive functioning, which is consistent with the
functional reserve model. The collective and complementary functions of the left and right hippocampi merit further exploration in prospective studies of memory function and TLE.

In unilateral left and right temporal lobotomy there is decreased ability to learn and recall verbal and visuo spatial material respectively, however this situation is not always replicable in patients with TLE. In a study by Hermann et al, 1992, Left TLE patients scored significantly lower than right TLE patients on visual naming, sentence repetition, reading and aural comprehension and on the token test, but not on verbal fluency and spelling. Performance was poor on verbal naming which was associated with verbal memory in both right and left TLE groups.

Fall in IQ has been associated with increasing number of seizures and occurrence of status epilepticus (Dordill, 1986). Early onset, long duration, high frequency complex partial seizures etc all contribute to low intelligence. When children with epilepsy are compared with normal controls, they are found to have lower IQ than normal children. Farwell et al (1985) has reported that children with seizure was significantly lower than controls group on Wechsler Full Scale Intelligence, neuropsychological impairment and errors on language related tasks.

Gathercole et al (2004) found that impairment of STM and working memory are associated with learning difficulties that can be substantial and that it can be minimized by appropriate methods of remedial support.

Scores on cognitive tests tend to decline with increased brain wave abnormalities reflecting the degree of underlying brain damage. Patients with generalized seizure activity tend to show greater and more generalized cognitive deficits than those with
focal seizures, and cognitive functioning with both kinds of seizure patterns worsen as the rate of seizures increases. (Cited in Lezak, 1995). Even if there is no structural damage it is found that the blood flow to these regions is less which negatively affects the cognitive functions. Similar studies are also reported by Mahapatra(1990), Rao, et al (1992), Pratibha, et al (1992).

1.2.9. Behavioral aspects of epilepsy

Rutter et al (1970) in his work has found behavioral problems to be high among children with both neurological damage and seizure (58.3%) when compared to general population (6.6%). 28% had behavioral problems with idiopathic epilepsy and 37.8% children with neurological brain damage had behavioral problem.

Children with epilepsy are known to have high rates of mental health problems such as anxiety, depression, attention problems and behavioral disruptions. They are five times more prone to have mental health problems than children from the general population (Gelder et al, 2006, Sutuh, 2002., Trimble, 1997). Personality change was reported in the past but recent studies do not support this notion. (Trimble, 1997). Medial temporal lesions are also found to be associated with behavioral abnormalities. (Gelder et al. 2006)

1.2.10. Social aspects of epilepsy

When the duration and frequency of epilepsy increases the quality of life of the patient is increasingly affected. Due to the unpredictability of epilepsy it has diverse implications and stigma related to the disorder. Lay persons have groundless fears and due to several misconceptions these people are segregated. Often these people with epilepsy are given extra privileges that will reduce the self esteem. Also in Western
countries they cannot lead a normal life because they are not given license to drive. (Gelder et al, 2006)

There are social implications to associate epilepsy with neurodevelopmental psychopathology and it is vital to ensure research in this area does not result in increased stigma for parents with epilepsy. (Johnson, 2011)

In a study by Poddar et al (1999) it was found that duration of epilepsy was significantly correlated with cognitive and behavioral functions and also with quality of life in children with idiopathic epilepsy. Quality of life in children with epilepsy is determined by seizure control and adequate cognitive and behavioral functioning. This shows that all the three domains, i.e., Cognitive, behavioral and social domains are equally important when we deal a child with epilepsy.

1.2.11. Treatment

When epilepsy is diagnosed, the physician must try to eradicate the irrational fears about epilepsy and help the patient to adjust to his disabilities. Epilepsy has a lot of stigma around it, which has to be handled effectively. If the case is a child, parents should be given awareness regarding the same. They have to be explained about the risk of every day life, to guard them. They should not be allowed to go near machineries, water, heights etc…They should avoid long periods without food and sleep. Thus by providing counseling they have to be prepared to face the new challenges of life. Other than this treatment with drugs are advisable. Certain drugs have been found to diminish the severity and frequency of epileptic attacks, and in favorable cases to abolish them completely. The objective of drug treatment is to secure an abolition of the attacks for a sufficient length of time to enable the patient to loose the epileptic habit. The most
frequently used drugs are Barbiturates, Phenobarbitone, Phenytoin sodium, Ethosuximide, The Diones, Troxidone, Sulthiame, Tegretol etc…

1.2.12. Prognosis

Improvement or prognosis depends on the response of the patient towards different modes of treatment. The term recovery means stopping of attacks, but even when this has been achieved and the patient remains free from attacks without treatment, there is usually some risk of recurrence at some future date. In most cases they continue taking drugs at least three years after the attacks have ceased. In some cases the treatment continues indefinitely in small dosages. If the treatment is started just after the first attack the prognosis is good. In idiopathic epilepsy if the onset is after an age of 20, the prognosis is found to be good. Those people who have frequent severe attacks are rarely cured.

The risk of death during an epileptic fit is slight, except in status epilepticus, in which condition the patient’s life is always threatened until consciousness returns, and death may occur even after recovery of consciousness. When death occurs as the result of a fit it is usually the accidental result of the loss of consciousness.

1.3. COGNITIVE MODELS

New scientists do not believe memory deficits as inevitable part of epilepsy but it is accepted as one of the most frequent cognitive problems. Other cognitive problems, including language difficulties and organizational problems, may underline or contribute to some of the reported difficulties. The memory problems are one of the important referral for neuropsychological assessment.
Memory is an amazing phenomenon. Memory is the means by which we draw our past experiences in order to use this information in the present. Memory refers to the dynamic mechanisms associated with retraining and retrieving information about past experience. The three operations of memory are encoding (transforms sensory data into mental representations), storage (keep the encoded information in memory) and retrieval (use the stored information).

1.3.1. Memory models:

Through the studies conducted in different contexts psychologists have reached many theoretical formulations. Waugh,N and Norman,D in 1965 proposed a model of memory in which they identified two structures of memory they are

1) Primary memory, which holds temporary information currently in use.

2) Secondary memory, which holds information for a long time.

In 1960s Atkinson, R and Shiffrin, R proposed an alternative conceptualization of memory in terms of three levels or stores.

1) A Sensory store, capable of storing limited amount of information for very brief period of time.

2) A short term store called as Short term memory(STM), capable of storing information for some what longer periods of time but also of relatively limited capacity.

3) A long term store also known as Long term memory (LTM), having very large capacity capable of storing information for long periods of time.
Baddeley and Hitch (1974) proposed a model for working memory (WM). WM has both storage and processing of information. The processing element of complex memory span tasks is supported by the central executive, a limited capacity system involved in regulatory control of working memory. The storage demands of verbal complex span tasks are met by a separate STM system, the phonological loop.

The phonological loop component of the WM model consists of two components: a short term store that maintains phonological representations and is subject to rapid delay, and a sub vocal rehearsal process that acts to refresh decaying phonological representations in the store. The spontaneous use of rehearsal does not emerge typically until about eight years of age. Any information that is verbalizable can be stored in the phonological loop. Development of phonological loop is steady and systematic and it levels off towards 15 years of age.
In 1972 Craik, F and Lockhart proposed another model to explain memory. They proposed that memory does not comprise of three or even any specific stores. Storage of information is based on depth of encoding. According to this theory there are theoretically an infinite number of levels of processing (LOP) at which items can be encoded, with no distinct boundaries between one level and the next. The level at which the information is stored depends on how it is encoded. If the level of processing is deeper then the possibility to retrieve is also high.

Baddeley & Hitch (1974) also proposed a model for working memory. The processing element of complex memory span tasks is supported by the central executive, a limited capacity system involved in regulatory control of working memory. The storage demands of verbal complex span tasks are met by a separate STM system, the phonological loop (Baddeley, 2000).

Figure 1.3: The detailed structure of current working memory model by Baddely.
The phonological loop component of the working memory model consists of two components, a short term store that maintains phonological representations and is subject to rapid delay, and a sub vocal rehearsal process that acts to refresh decaying phonological representations in the store. The spontaneous use of rehearsal does not emerge typically until about eight years of age. The capacity of the phonological loop to store verbal materials undergoes steady increase from early childhood through adolescence, leveling off at 15 years of age. Schacter, (2001) proposed an adaptive explanation for why we forget things. In this theory he says that natural selection has an important role. He lists the following *seven sins* along with the adaptive role of these seven sins.

*Transience:* There is a gradual weakening of memory over time. Its adaptive function is such that, not all experiences or knowledge have any future benefit. The information that memory throws out will be judged and the person will not be aware of its absence.

*Absent Mindedness:* It is an issue of inadequate storage. It is adaptive because we cannot attend to all stimuli in and around us. Attention is a selective process.

*Blocking:* Memory has an inhibitory process that stops activation from spreading uncontrollably by reducing activation of some memories. Thus one can use only a handful of memory which is of use immediately.

*Misattribution and Suggestibility:* The adaptive perspective suggests that memory is not simply an archive of our experience, but, memory is designed to support future actions based on past experience. When much of the information are discarded, it can lead to misattribution and suggestibility due to inadequate ability for retrieval. Individuals forgets some parts of an event, but even if we forget the details we never lose the
processed information or abstraction, which is the useful thing in future, so this also has an adaptive function for memory.

**Bias:** Unconsciously rewriting the past events, we overestimate our contribution to successful events and underestimate our contributions to negative events.

**Persistence:** The repeated recall of unpleasant events has an adaptive function. By making the negative event prominent, it is the warning not to make the same mistakes.

The apparent weakness of our memory is the other side of some beneficial features.

Klein *et al* (2002) states that memory is evolved to support behavior by storing and searching information that enables us to make the choices we need to make in an ever changing world.

Tulving, 1972 proposed a distinction between semantic memory (general word knowledge) and episodic memory (personally experienced events). According to him we use episodic memory when we need to recall something that occurred to us at a particular time or in a particular context. The third discrete memory system for procedural knowledge is procedural memory.

Gathercole and Alloway in 2006 provide models of working memory and STM and try to link this to learning and diagnosis of impairment. The memory impairments are associated with a range of neurodevelopmental disorder. The researchers identified methods of alleviating the adverse consequences of working and STM impairment for learning. They concluded that impairment of STM and Working memory are associated with learning difficulties that can be substantial, and that can be minimized by appropriate methods of remedial support.
Deneman and Carpenter, 1980, also report that memory span is strongly associated with a range of measures of learning ability. Working Memory capacity has important consequences for high cognitive function. Gathercole & Alloway concluded that impairment in STM and working memory are associated with learning difficulties that can be substantial in remediation.

Similar reports are given in the study done by Jarvis and Gathercole (2003). They found that verbal as well as visuo spatial working memory scores are strongly linked with children’s attainment in national curriculum. Visuo spatial memory score is highly linked with mathematics and science scores.

It is clear that children with epilepsy are prone to have memory deficits and thereby it will affect their academic performance. Hence, we have to explore whether this memory impairment and poor performance is due to learning disability. Now a day’s learning disability is an area where ample research is being conducted, yet these areas are not probed effectively. In children with learning disability one or more of the basic psychological processes involved in understanding and using spoken or written language is involved. This will result in severe discrepancy between age and ability levels in one or more of the following areas of academic achievement: oral expression, listening comprehension, reading, writing or arithmetic. Learning disability is the most prevalent problem affecting children. The incidence rate of LD in India is not clearly known. Different studies give different statistical data. In all the studies gender difference is similar, Boys out number girls. (Lyon, 1996. Mangal, 2007)

There are also studies which suggest that frequency of academic difficulties are same for boys and girls, but boys are more likely to be referred for special education when they
have academic problems. This is because they have other problems like hyperactivity etc. which may irritate the teacher there by referring to other professionals. (Seewald&Zigmond, 1982; Shawitz, Fletche&Escobar, 1990).

**Comorbidity:** Around 20 to 25% of students with LD have Attention deficit hyperactivity disorder (ADHD). The reason for such a high rate is not yet known. One probable explanation, may be such that, problems with attention along with restlessness interfere with learning. Another reason can be, continued academic failure can lead to restlessness and inattention in the classroom. (Lyon G R 1996a; Kotkin, Forness & Kavale,2001., Forness & Kavale,2002., APA, 2000., Hallahan, Kauffman & Pullen, 2009).

**Academic problems:** Academic problems are the hallmark of learning disabilities, i.e., if there are no academic problems then there is no learning disability. The different areas affected in these children are:

1. Reading
2. Writing
3. Social usage of language
5. Perceptual motor and general coordination problems.

**Memory:** Early researchers in learning disability have identified memory deficits. Children with LD are reported to have memory problems in STM and working memory.(Swanson,2005) Children with LD perform poorly in memory tasks because they do not make use of strategies spontaneously which their normal peers do. To make new strategies the child should have the ability to recognize task requirement, ability to
select and implement appropriate strategies and ability to monitor or adjust performance which these children with LD are not capable. Memory problems are more reported in learning verbal materials than nonverbal materials. (Quadfasel & Pruyser, 1955, Kibby et al, 2004).

Charles, Mackenzie and Susie (1992) reported that working memory deficits seen in people with severe learning difficulties may contribute to their difficulties on cognitive tasks. Children with ADHD have documented with working memory deficit, response inhibition and dual tasks. Same cognitive processes are also closely associated with reading acquisition. (Savage et al., 2006)

1.3.2. Conceptual models of Learning Disability.

**Verbal learning disability Model**

Reading and writing represent special forms of speech activity. Auditory-perceptual and language deficits historically have been linked to poor performance in these languages based academic domains.

**Wernickes - Geschwind model of dyslexia.**

The reading process begins with visual perception and analysis of grapheme which is recorded to its phonemic structure that is being comprehended later. During initial stages of reading all the fundamental neurologic operations are incorporated in a clear serial manner. In a later stage the graphemes will elicit direct comprehension of written words even by eliminating intermediate phonemic analysis and synthesis.

**Phonological core deficit model**

This is an evidence based model. It has been estimated that 80% of individuals have an underlying phonological deficit contributing to reading problem. Children with such
problems are affected by other aspects of language like, speech perception, speech production, and naming. Phonological processing is a critical aspect of reading. Reading requires learning of the relationship between graphemes (written letters) and phonemes (sounds). Children with reading problems fail to use the alphabetic principles accurately. Shayuritz et al (2006) reports that dyslexia represents a disorder within the language system and more specifically within a particular subcomponent of phonological processing.

**Nonverbal learning disability model**

This model states that Children with nonverbal LD are unable to comprehend the significance of many aspects of the environment like, gestures, facial expressions and other elements of emotions. A significant lesion in right hemisphere will produce a nonverbal learning disability. Their visual-spatial organization, nonverbal problem solving, and paralinguistic abilities are impaired. Even though they have a good language skill, graphomotor skills can be appropriate but are delayed early in development. Academic deficits tend to be manifested in mechanical arithmetic, reasoning (math) and reading comprehension.

**The dual route hypothesis of reading and writing**

The evidence from patients with acquired dyslexia supports the notion that there are two parallel systems for reading and writing; one utilizes sound-based route for reading and writing, and the other uses the more direct meaning route. These systems can break down independently, to produce different types of dyslexia and dysgraphia.
The lexical (Semantic) system

It uses whole word retrieval by consulting internal memory store of known spelling. This system is important for spelling familiar but orthographically irregular words and homophones (words with same pronunciation but different spelling—for example: ATE, EIGHT). Damage in this system produces specific problems in spelling irregular words and production of error which are phonologically plausible. This syndrome has been described in patients with lesions in rather diverse sites in the left temporal-parietal regions. Studies reports consistent finding in patients with focal temporal lobe atrophy.
Those children with dysgraphia suffer extensive left hemisphere damage. A disturbance in the ability to comprehend and write number is called acalculia and the angular gyrus region in the left hemisphere appears important for numbers. (Hodges, 1996).

**Nonverbal Learning disability Model**

This model states that children with such conditions (nonverbal problems) are unable to comprehend important aspects of environment. They fail to learn and interpret the actions such as gestures, facial expression and other elements of emotions. Studies show that patients with right brain damage have such problems. (Rourke, 1989) He found that damage to white matter of the right hemisphere is responsible for nonverbal learning disabilities.

Neuropsychologically, they have a distinct profile. Strength includes auditory perception, simple motor function and intact rote verbal learning. Selective auditory attention, phonological skills and auditory verbal memory also appear intact. They have problems with visuo-spatial organization problems, nonverbal problem solving abilities. Marked academic deficits tend to be manifested in mechanical, mathematical reasoning and reading comprehension. The model appears to have strong relationship with socio-emotional and adaptive behavior deficits. (Poor social perception will result in isolation.)

In conclusion, the models to explain learning disabilities are as complex as the diverse nature of the disorder itself. No two conditions can be similar, for some it is as simple as handwriting problems but for some all the different areas discussed may be affected. Students with reading problems also can manifest associated language disorders and writing problem with the co-occurrence of mathematic disability.
Neurobiological aspects of Learning disability:

Recent neurobiological studies have conceptualized reading primarily as a set of functions that are served by specific neurolinguistic abilities and associated neuroanatomical structure and neurologic processes. Neurolinguistic and neurocognitive studies are also very commonly studied. Findings suggest that children with reading disability will manifest phonological processing deficit and morphologic deficit (neurolinguistic study) (Stanovich et al, 1997). Molfese and colleagues (2002) have used auditory evoked potentials in newborns to predict language based problems in preschool and early school aged children even at the age of 8 years. Recently autopsy studies and imaging studies have revealed a lot regarding dyslexia. In most individuals (normal) the planum temporale, a posterior region of the sylvian fissur in the left hemisphere tend to be larger than the right hemisphere.(Geschwind & Levitsky, 1968) this normal asymmetry is not found in children with dyslexia. They have symmetrical plana or reversed asymmetry.(Larsen, Hoien, Lundberg & Odegaard, 1990., Leonard et al,1993.,Hynd & Hiemenz, 1997., Eckert & Leonard, 2000., Kibby et al,2004) Asymmetry is also reported in the parietal-occipital and prefrontal regions(Jernigan etal,1991., Kibby et al,2004)

In a fMRI study Shaywitz et al(2002) found semantic processing in individuals with dyslexia to be associated with wide spread brain activity. Over activity was located in the bilateral inferior frontal regions and under activity in the left posterior temporal region, when compared to normal readers.

It has been found that regions of temporal lobe of left hemisphere are involved in language comprehension and Broca’s area in speech production. Reading skills can be
impaired if visual cortex is damaged as information should pass to regions of temporal lobe to make language processing required for phonological decoding and reading comprehension. In some studies it is reported that compensatory activity in premotor regions of Broca’s area is found in cases with LD which is a compensatory strategy while reading. Under activity is also reported in Wernicke’s area, angular gyrus and striate cortex. Over activity of left frontal and right posterior region was found, which suggests anomalous brain function as a compensatory mechanism. (Shayuwitz, 1998, Snowling, 2000, Rumsey, 1992, Brunswick et al 1999). In another study by Paulesue et al, 1996 it is reported that a region called insula which connects Wernicke’s area and Broca’s area receive less stimulation. (Cited in Snowling, 2000).

Summing up it is highly evident that neurobiological factors play an important role in developmental aspects of reading acquisition. Variants may evolve based on their differential manifestations at specific developmental periods.

**Other specific learning disabilities**

When writing disorders are considered as a part of *dysgraphia*, they have poor spelling, punctuations, grammar or poor communication clarity. The influence of neuropsychological factors like memory and attention on writing is less documented. Levine et al (1993) suggested that memory, attention, graphomotor output, sequential processing higher order cognition, language and visuo-spatial functions are important. The first empirical study by Abbott & Berninger (1993) reports that sentence memory, word finding, phonological processing and reading contribute to composition fluency. Writing problems can be due to poor fine motor control, lag in visual memory, and lack
of attention, working memory deficit, executive functions, self-monitoring, planning, problem solving and cognitive flexibility.

According to Kellog(1999,1996), Working memory which is the cognitive work place is important to written expression as it is the fun underlining the activity that is maintaining multiple ideas, retrieval of grammatical rules from long term memory and the recursive self monitoring that is required during the act of writing. He proposed a model for writing that was based on working memory. It included three major components and they are verbal formulation, execution and monitoring. Planning and translating are the subcomponents of verbal formulation, programming and executing functions are the sub components of execution process and reading and editing are the subcomponents of monitoring in written expression. Working memory model of Baddeley(1986) hinted visuospatial sketchpad to formulation, phonological loop to monitoring and central executive to execution process.

Problems with mathematics (dyscalculia) were reported in 1900s, an overlap between reading disability was also found during this period. Dykman(1995) reports that there is no difference between children with reading problems and children with mathematics problem on phonological processing, memory, naming speed or picture vocabulary. Hammill(1975) reviewed more than 600 studies and found that visual discrimination, memory for visual sequences, visual motor coordination corrected highest with arithmetic success.

Gross-Tur et al(1996) found that 26% of children diagnosed with dyscalculia had symptoms of ADHD. It is reported by others like Rourke,(1989)and also by Zentall et al, (1994) that specific neuropsychological pattern of deficit is found among these children.
They have visual perceptual deficit, poor visual attention, poor visual memory and left sided sensory problems. In the neurological perspective, Luria(1980) reported that damage to temporal- parietal- occipital association areas in the right hemisphere contribute to problems with calculations and arithmetic operations. Contemporary neurological models suggest math skills are mediated by both the hemispheres.(Kahn and Whitaker, 1991)

Neurological and neuropsychological deficiencies and dysfunctions have been linked to a variety of learning components and problems, and these linkages will continue to require studies with well marked samples from various age ranges across life span.

*Neurobiological basis of LD*

It has been established that regions in temporal lobe of the left hemisphere are involved in language comprehension and Broca’s area in speech production. With regard to reading process, it is clear that visual stimulation is responsible. From the occipital lobe the information should pass to regions of temporal lobe to make language processing which requires phonological decoding and reading comprehension. The first kind of evidence about the brain structure and function of dyslexic individuals came from a series of postmortem studies of dyslexic brains carried out by Galaburde and his colleagues. They studied perisylvian region of left hemisphere and planum temporale, a posterior region of sylvian fissure which is asymmetrical usually. In dyslexic brains planum temporale was symmetrical. This finding suggests that difference in brain structure found in dyslexia is due to prenatal brain differentiation, a process that is controlled by genetic factors. (Hynd&Hiemens, 1997., Larsen, Hoien, Lundberg &Odegaard, 1990). Study by Monastra et al (1987) reported that there is an association between LD and
unilateral delay in cortical development. 43% of the sample showed significant neuropsychological defects in addition to psychopathology, academic and behavioral problems.

The effects of brain lesions are more complex in childhood than in adult life because the brain is developing. This has two consequences; they are greater capacity to compensate and delayed effect. A child’s brain is more able to compensate for localized damage. Early damage may not manifest until a later stage of development as the damaged area takes up some key function. It is well established that brain injury at birth may not result in seizures until many years later. In childhood there is a strong association between recurrent seizures and psychiatric disorders. Causal relationship can be of four kinds

1. The brain lesion causing the epilepsy may also cause the psychiatric disorders.

2. The psychological and social consequences of recurrent seizures may cause the disorder.

3. The effect of epilepsy on school performance may cause disorder.

4. The drugs used to treat epilepsy may cause the disorder through their side effects.

The site and type of epilepsy seems generally less important, with the exception that TLE seems more likely to be associated with psychopathology. (Gelder et al, 2006., Yuan et al, 2006)

When the temporal regions of animals are damaged, expected social behavior is lost and restlessness and repetitive motor behavior and limited behavioral repertories are seen. And when the epileptic focus is in the left temporal region then the language related
functions are affected. Studies have shown that children with repeated left TLE have difficulty with learning language or recall of learned task.

The literature review shows that behavior problems and cognitive deficits are well studied. The reviews show mainly comparative studies of generalized seizures and partial seizures were made. But partial seizures as such have not been studied separately.

1.4. **Need and significance of the study**

This study tries to explore the role of epilepsy specifically to TLE in developmental psychopathology. Epilepsy remains as one of the most stigmatized brain disorder. Due to illiteracy in countries like India and other developing countries the stigma associated to this disorder is high with in developing countries and because of this they do not seek treatment. As a result of this, it becomes uncontrolled affecting cognitive behavioral, social, educational and occupational domains. The present study proposes to evaluate cognitive and behavioral dysfunctions of children with epilepsy with focal seizures (TLE and OTLE), Children with Epilepsy with a co morbidity of ADHD and LD and children with learning disability alone will be compared with normal controls. Hence it could be a very significant study since research in this area has been seldom conducted. In countries like India and other third world countries, epilepsy manifests with several types of seizures, differing in age of onset, response to treatment, prognosis, EEG correlates, and risk factors. Having a single term that encompasses all of these conditions makes it possible to calculate the burden of disease from both economic and social perspective, to estimate the demand for health services planning and to meet other public health objectives. Children in these countries are treated with traditional medicines with
devastating consequences. Even when proper treatment with medicines is provided; parents do not recognize the significance of long term adherence to medication. To effectively address the needs of individuals with epilepsy and reduce the widespread stigma attached to the condition, national and local governments and health authorities of developing countries should support public education campaigns focused on the causation of epilepsy, the impact of the disease on the cognitive and behavioral patterns of the person, and the availability of safe and effective treatments.

This study is undertaken with a genuine interest in bringing out the cognitive and behavioral aspects of children with partial seizures as well as the link between developmental psychopathology and epilepsy. In the absence of knowledge about the genetic, behavioral, environmental contributions to developmental psychopathology as well as to epilepsy our understanding of both the conditions is incomplete. For some rare conditions genetics may provide the only route to understand, but for the more common psychopathologies genetic predisposition may combine other factors in a complex manner. Hence in the present study the mission is to identify causal link if any between type of seizure and the type of developmental psychopathology. No single study can hope to resolve all the problems of developmental psychopathology, but the value and challenges of research has to be taken up.

1.5. STATEMENT OF THE PROBLEM:

DEVELOPMENTAL PSYCHOPATHOLOGY: THE ROLE OF EPILEPSY WITH SPECIAL REFERENCE TO TEMPORAL LOBE EPILEPSY.
**Key terms used**

**Epilepsy**: Epilepsy is the tendency to recurrent motor, sensory or behavioral seizures caused by a discrete electrical brain abnormality.

**Temporal lobe epilepsy**: Simple partial seizures results from discrete electrical activity that arise from the Temporal Lobe.

**Other than Temporal lobe epilepsy** (OTLE): Simple partial seizures that originate from Frontal lobe, Parietal Lobe and Occipital lobe.

**Developmental Psychopathology**: Developmental psychopathology tries to understand disorders manifest with deficits in the traditional behavioral domain of cognition, language, visual-spatial functions, attention and socialization among children.

**Memory**: Memory refers to the dynamic mechanisms associated with retraining and retrieving information about past experience.

**Intelligence**: A person’s capacity for clear thinking and accurate intellectual work.

The key terms explains the meaning of the terms used and the operational definitions. The role of epilepsy in developmental psychopathology is explored in the present study. How epilepsy affects cognitive functions like, intelligence and memory. Following objectives were formulated to study the problem followed by major hypothesis of the present study.

**OBJECTIVES:**

1. To find whether there is any difference between children with epilepsy, children with epilepsy and co morbidities like LD and ADHD and children with LD and normal
children with regard to their memory intelligence and different areas of developmental psychopathology (DPCL).

2. To find whether there is any difference between the four groups in prenatal factors, birth weight, developmental milestones etc.

On the basis of the above objectives the following hypotheses were drawn.

1.7. HYPOTHESES:

- There will be no significant difference between the three clinical groups and normal children in developmental Psychopathology
- There will be no significant difference between the three clinical groups and normal children in memory.
- There will be no significant difference between TLE and O TLE children in the different components of developmental Psychopathology (DPCL).
- There will be no significant difference between TLE and OTLE children in the different components of memory tests for children.
- There will be no significant difference between the three clinical groups and normal children in their intelligence.