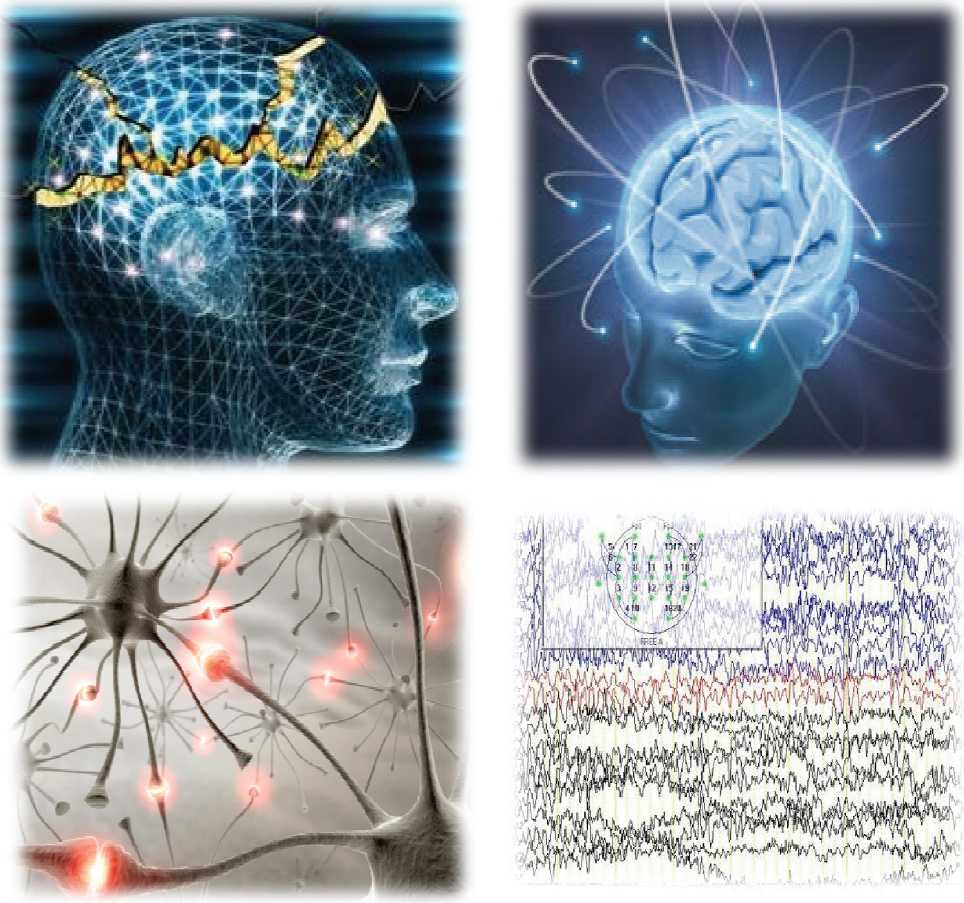
PREVALENCE AND NATURE OF SEIZURES IN CHILDREN WITH COMMUNICATION DISORDERS



Project funded by AIISH Research Fund (2007-08)

All India Institute of Speech and Hearing

Manasagangothri, Mysore-570006

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PROJECT REPORT

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PERSONNEL OF THE PROJECT

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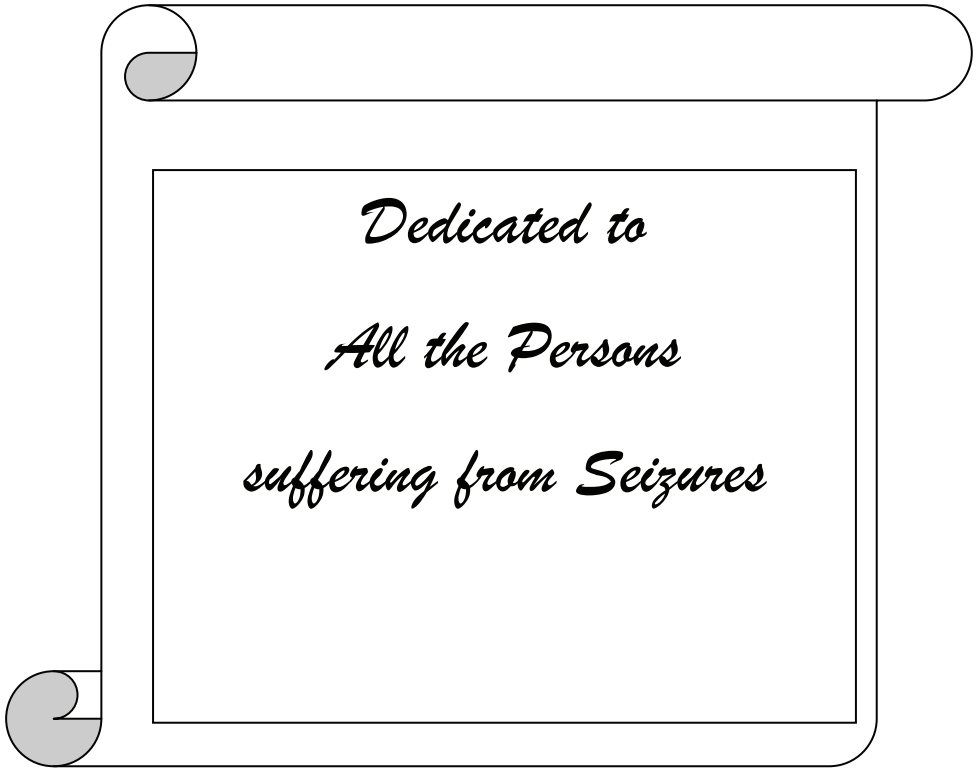
Dedicated to
All the Persons
suffering from Seizures

ACKNOWLEDGEMENTS

I take immense pleasure in thanking ***Dr Vijayalakshmi Basavaraj, Director, AIISH*** *for having permitted me to carry out this project work , extending project term and also for being supportive at all the times. I wish to express my deep sense of gratitude to my first Co- investigator,* ***Dr Y. V. Geetha, Prof & Head, Dept of Speech language sciences*** *for her able guidance and useful suggestions, which helped me in completing the project work.*

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Principal investigator

Sangeetha Mahesh

PREVALENCE AND NATURE OF SEIZURES IN CHILDREN WITH COMMUNICATION DISORDERS

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1. Initial sanction for a duration of one year from 2.05.2008 to 02.05.2009
2. Extended for three months duration from 02.05.2009 to 30.09.2009 (with one month break)

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1.31 Lakhs

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

PREVALENCE AND NATURE OF SEIZURES IN CHILDREN WITH COMMUNICATION DISORDERS

INTRODUCTION

Epilepsy is paroxysmal transient disturbances of brain function that may manifest as loss of consciousness and abnormal motor phenomena. Epilepsy affects approximately 1% of the population making it one of the most common neurological diseases (Genes and Disease by the National Centre for Biotechnology, 1998).

Epilepsy is an enduring condition; or rather a group of conditions, in which epileptic seizures occur repeatedly without a detectable extracerebral cause (Gastaut, 1973). According to the Epilepsy Foundation of America (2010), it is a physical condition that occurs when there is a sudden, brief change in how the brain works. When brain cells are not working properly, a person's consciousness, movement or actions may be altered for a short time. These physical changes are called epileptic seizures. Epilepsy is therefore sometimes called a seizure disorder. It affects people in all nations and of all races. About two million Americans have seizures. Of the 125,000 new cases that develop each year, up to 50% are in children and adolescents (Freeman, Vining, & Pillas, 1997).

Statistical information such as prevalence, incidence, deaths and other data is provided from numerous sources and is subject to numerous provisons. India has a large number of differentially abled population numbering about 100 million. The number is expected to increase substantially every year in view of the rapid changing demographic profiles and morbidity patterns. There are numerous statistics reported in articles and internet conducting the survey on the occurrence of epilepsy. Prevalence of active seizures (history of the disorder plus a seizure or use of antiepileptic medicine within the past 5 years) is estimated as almost 3 million in the United States. Prevalence tends to increase with age. 3,26,000 school children through age 15 had epilepsy (Epilepsy foundation of America, 2010). Data may be collected from different years, from various countries, states, areas, different age groups and different racial factors. It may not necessarily reflect the overall prevalence in the entire population. The estimate may not always be accurate and may not apply to Indian population. The prevalence of epilepsy (recurrent seizures)

in the general population with reference to Indian context has been studied since 1980's. The prevalence rates across the country ranged widely from as low as 1.28 in Baroda to 11.92 in rural Bangalore.

In general, the precipitation of a seizure probably requires a brain lesion, a genetic predisposition, or both, with its resulting hyperexcitability on the one hand and afferent stimuli of either cerebral or extra cerebral origin on the other. The state of the brain and its excitability is therefore influenced by both intrinsic factors, such as circadian rhythms, stress or the state of vigilance and extrinsic factors that may be neural or of another nature (Allen, 2008).

Children with developmental disabilities are at higher risk for seizures than the general population. Epilepsy is one of the most common neurological disorders occurring in children with developmental disabilities (Hauser, Annegers, & Kurland, 1991). Communication disorders include majority of children with developmental disabilities. They are borderline intelligence, cerebral palsy, mental retardation, developmental delay, pervasive developmental disorders, apraxia, attention deficit hyperactivity disorder and delay in speech and language. Epilepsy and communication disorder may occur together and both may result from same underlying brain disorder. However, the causal association between epilepsy and language impairment is poorly documented due to constraints in epidemiological methods. The distribution and nature of epileptic seizures in different subgroups of children with communication disorders (CWCDs) have not been studied in detail. It would be interesting to know the characteristic features of seizure disorders in different clinical population in order to take suitable preventive measures and to provide rehabilitation. Since there is only limited data about the prevalence of seizures in CWCDs with reference to Indian context the present study has been taken up to address the issue.

As speech language pathologists and audiologists we come across many children and adults with various communication disorders having a history of epilepsy or seizure disorders. It would be interesting and useful to know the prevalence and characteristic features of these in different clinical population in order to take suitable preventive measures. The current project was therefore proposed with this need in mind and with the primary objective to determine the

prevalence of epilepsy in children with communication disorders along with the following objectives:

Objectives of the study:

- To examine the prevalence of seizures in different subgroups of children with communication disorders
- To examine the age of onset, causes, type of seizure, frequency of occurrence of seizures
- To identify the pattern of inheritance if any
- To classify the seizure disorders causing communication disorders if possible
- To examine the co-morbidity of seizures and the nature of communication disorder
- To develop a high-risk register for children with seizures

REVIEW OF LITERATURE

1. Brain activity-normal and epileptic

The brain is the convoluted organ which fills the great cavity of the skull. It is composed of nerve fibers or expansions. Each living nerve cell is capable of developing energy which is propagated as electric current along its own expansions. The expansions are insulated except at their endings. At the endings of these synapses enough energy can be communicated by a chemical process to the body of another nerve cell to fire off energy in it. Thus, further conduction of electric potentials passes through expansions of the second cell and so by a succession of little activations a stream of impulses passes from one ganglion cell to another and another as determined, no doubt by complicated facilitations and inhibitions.

There are ten billion nerve cells (ganglion cells) within the human brain and probably has some capacity of generating energy within itself. The function of the brain is carried out by the passage of nervous impulses from one ganglion cell to another ganglion cell in an orderly and controlled manner. The impulses pass quickly along the insulated nerve fibers like an electrical current, while passage across the synapses to successive nerve cells is accomplished by a somewhat slower chemical process. The cell bodies are collected together forming gray matter and the nerve fibers which conduct the currents compose the white matter.

As long as the gray matter is normal, the energy of the nerve cells is employed only in the coordinated functional mechanisms of the brain. But if some area is injured by disease or pressure or lack of oxygen, the gray matter, although it may continue to function, may do so with abnormal additions of its own. However, defect in the regulating mechanisms normally limit excessive discharge. Thus sometimes, even years after injury, an abnormal area “ripens” slowly into a self discharging electrochemical unit. This is called an epileptogenic focus. Such an explosive discharge produces an epileptic fit. The fit is large or small depending upon the extent and intensity of discharge and the positions of the gray matter involved. The attack which begins as a small one, spread to other areas and increase in severity until there is a maximum discharge. Then the resultant condition is an epileptic fit, a grand mal falling unconscious with every muscle contracting, groaning and perhaps frothing at the mouth (Penfield & Jasper, 1954).

2. Epilepsy and seizures

The word “Epilepsy” is derived from a Greek word which means to seize. Jackson (1931) defines “Epilepsy as sudden excessive, rapid and local discharge of gray matter. Epilepsy is an enduring condition or rather a group of conditions, in which epileptic seizures occur repeatedly without a detectable extra cerebral cause (Gastaut, 1973). An individual’s tendency to recurrent paroxysmal dysfunction may result primarily from the presence of structural brain abnormalities or from an intrinsic constitutional propensity which is determined, at least in part, by genetic factors and neuronal excitability (Hauser & Anderson, 1986). Epileptic seizures are usually brief, lasting from second to minutes and are marked by the sudden appearance of behavioral manifestation that may be purely motor or that may affect other brain functions (Penfield & Jasper, 1954; Gastaut & Broughton, 1972). The abnormal and excessive neuronal activity during the epileptic seizures is inferred from both the clinical events and the electroencephalogram (EEG).

Epilepsy can also be defined clinically as a paroxysmal, recurrent, stereotyped disturbance of movement, feeling or behavior, almost always associated with a disturbance of consciousness, these being primarily cerebral in origin (Koul, Razdan, & Motta, 1988). An epileptic seizure results in a paroxysmal disorganization of one or several brain functions (Bancaud, Talairach, & Movel, 1974). This is always associated with an abnormal excessive neuronal discharge in the brain. The epileptic discharge is a complex phenomenon resulting from the interaction of excitatory and inhibitory influxes network formed by multiple diverse neuronal sources. The main characteristic of the epileptic discharge is its high amplitude and its rhythmicity, both caused by the excessive synchronization of an abnormal number of potentials in a neuronal aggregate (Fenwick, 1983; Aird, Masland, & Woodbury, 1989).

The terms epilepsy and seizures are often used synonymously but there is a slight difference between the two. Seizures constitute epilepsy and can also be called convulsions, fits or attacks. Seizures may appear as disturbances of consciousness only; they may be evidenced by sensory, visceral or motor signs or they may present as perseverations of ideation, emotion or mood. Some seizures may include a single symptom, while others may have a complex symptomatology (Gastaut & Broughton, 1972). All that shakes or trembles are not seizures and the latter is more

than a mere involuntary movement. Multiple seizures occurring over a 24- hour period have been considered as serial seizures or even status epilepticus (Das & Sanyal, 1996). Another way of defining epileptic seizures is as more than one attack of an afebrile seizure in any of its varied manifestations, therapeutic drugs and alcohol or drug abuse (Mani, 1997).

Childhood seizures are the result of abnormal electrical activity in the brain. Children who have recurrent seizures (those that occur more than once) may have epilepsy. In the epidemiologic studies (Sander & Sillanpaa, 1998) the occurrence of two seizures was accepted as the operational definition of seizures. This definition, though convenient, is obviously arbitrary. In other studies (eg, Todt, 1984) the threshold for a diagnosis of epilepsy was about three attacks.

Seizures occurring during the acute phase of meningitis, encephalitis, craniocerebral trauma or acute metabolic states, etc. are actually the sequelae of those conditions. The seizures when present with at least one afebrile seizure in the previous five years regardless of drug treatment are termed as active seizures. Inactive seizures refer to freedom from seizures for more than the previous five years. This is subdivided into seizures in remission with or without treatment depending on whether the patient is still on or off drugs on the day of ascertainment (International League Against Epilepsy, 1997).

3. Epidemiology of seizures

The Epidemiology is defined as “a science of epidemics prevalent among community at special time’ according to concise oxford dictionary, sixth edition (1981). Epidemiological studies on seizures suffer from certain methodological problems (Shorvan & Farmer, 1988). These include the all-important criteria of definition(s), survey methodology, and screening instrument, reliability of the observations and the problem of concealment of the illness. Following are the types of prevalence data available in the literature (Mani, 1997):

- Point prevalence refers to proportion of patients with seizures at a specific time (usually a specific day- prevalence day). Criteria for inclusion should be clearly specified and whether active or inactive and if the latter whether on treatment on that day.

- Period prevalence refers to defined period, usually one year inclusion criteria. Generally an only active seizure is included under prevalence figure.
- Crude prevalence rate (CPR) gives the total figure for all ages and both genders combined, while age-gender adjusted figures take these two factors into consideration.
- Lifetime prevalence refers to patients with a history of having seizures irrespective of active or inactive state or on treatment or not. This includes patients with active seizures, seizures in remission and individuals identified by history as having had seizures in the past. In general, prevalence rates are expressed as cases per 1000 population.
- Incidence refers to new or fresh cases of seizures occurring during point prevalence or prevalence period in the specified population. The figures are usually expressed as cases per 100,000 populations. The criteria should specify whether these are based on the date of diagnosis or date of onset. Generally, it is the latter which is in common use.
- Cumulative incidence refers to the risk of developing seizures by a certain time, generally by the time a specific age is reached. The figure is generally the same as lifetime prevalence, but with some differences. In cumulative incidence, the values are given in terms of gender and are specific rates adjusted to standardized census figures in a well-defined population for the last decade, e.g, 1970,1980, or equivalent is accepted as the standard.
- Standardized mortality rate (SMR) refers to the ratio of observed number of deaths in a population of subjects with seizures compared to that in the general population. The total figure is referred to as crude mortality rate, while age-gender adjusted rates are specific for these two parameters. If age at death is noted in the patients with seizures and the general population one can speak of mortality years, a more powerful statistical measure (Mani, 1997).

A. Prevalence of seizures in general population: Western studies

The estimate of the incidence of seizures (recurrent seizures) in the general population varies between 0.5-1.5 percent (Davis, 1977). The prevalence of seizures in Rochester, Minnesota was determined for a specific date in each of 5 decennia census years. Individuals with a diagnosis of seizures (Recurrent unprovoked

seizures) who were known to have experienced a seizure or who had received antiepileptic medication in the preceding 5 years were considered active prevalence cases by this definition. The adjusted prevalence per 1,000 populations increased steadily from 2.7 in 1940 to 6.8 in 1980. Prevalence rate was higher for males than females. After 1950, prevalence tended to increase with advancing age and was highest in the oldest age groups On the average, the 1980 prevalence cases had seizures <10 years and >50%, had their first diagnosis in the first 20 years of life (Hauser, Annegers, & Kurland, 1991). Details on prevalence of seizures in other countries are given in Table 1.

Table 1. *Prevalence of seizures in Other countries*

Author (year)	**Country**	**Prevalence (per 1000)**	**Remarks**
Pond (1961)	UK	6.2	Active seizures
Krohn (1961)	Norway	2.3	Active seizures
Bird, Heinz, and Klintwork (1962)	South Africa	3.7	-
Sato (1964)	Japan	1.5	Active seizures
Granieri et al. (1983)	Italy	6.2	Active seizures
Li et al.(1985)	China	4.4	Age adjusted
Sridharan, Radhakrishanan, Ashok, and Mousa (1986)	Libya	1.9	Age adjusted, age > 15 years
Hearer, Anderson, and Schoenberg (1986)	USA	6.7	Active seizures
Hearer et al.(1986)	USA	10.4	Lifetime prevalence
Osuntokun et al. (1987)	Nigeria	5.3	Active seizures
Hauser et al. (1991)	USA	6.8	Active seizures
Hauser et al. (1991)	USA	8.2	Lifetime
Aziz, Guvener, Aktar, and Hassan (1997)	Pakistan	9.98	Crude rates
Aziz et al. (1997)	Turkey	7.0	Crude rates

(Source: Sridharan (2002), Current science, 82 (6), March, 2002)

B. Prevalence of seizures in general population : Indian studies

There are many studies done on different subgroups of population in the Indian context. The estimates of the prevalence rates (per thousand) of seizures (recurrent seizures) in the population are as follows:

Gauribidnaur study (Gourie, Rao, & Prakashi, 1987)

An investigation from the National Institute for Mental Health and Neuro Sciences department of neurology (NIMHANS) during the period 1982-84 was conducted to determine the prevalence of seizures. The area of study was Gauribidanaur town (17,734) and adjacent rural parts (39.926) totaling 57,660. The figures for active seizures were 5.6 for rural and 2.5 per thousand for urban with an overall figure of 4.6 per population. Standardized population adjusted figure were not available. No incidence studies were carried out.

The urban Parsi study (Bharucha et al., 1988)

A study was carried out in Bombay (Mumbai) on Parsi community in the year 1985. The surveyed population of 14,010 were covered by a door-to-door survey. The prevalence of active seizures was 3.6, inactive seizures was 1.1, while for US census population for 1960 were 4.1 for active seizures and 4.8 for lifetime prevalence respectively. Incidence studies were not carried out.

Rural Kashmir study (Koul et al., 1988)

A study was conducted in Kuthar valley of Anantnag district in mountainous Kashmir in the year, 1986. The population made up mainly of poor illiterate Muslims and covered by a door to door survey. The population numbered 63,645 with a very high infant mortality rate of 104.8 per 1000 live births. The WHO protocol was used to determine the prevalence of seizures. Single febrile and acute symptomatic seizures were excluded. The prevalence figure for active seizures was a low of 2.47 per thousand, while that for inactive seizures was not mentioned. Standardized population rates and incidence studies were not included.

Rural West Bengal study (Das & Sanyal, 1996)

This was carried out in rural areas of Malda district, West Bengal with point prevalence on March 31, 1989. Trained health workers and volunteers carried out a door to door survey for major neurological disorders, including seizures. The population of 37,286 with a literacy of 21% were surveyed. The WHO questionnaire was used, which was found to have a sensitivity of 90% and specificity of 86% for all neurological disease combined.

Crude prevalence rate for seizures was 3.06. The age specific prevalence rate for males aged above 14 years was 3.75 and for 14 years and below were 2.60, corresponding figures for females being 3.53 and 1.94, respectively.

Rural Haryana study (Kokket & Verma, 1998)

This study was a commendable effort by two house officers from the department of community medicine, All India Institute of Medical sciences (AIIMS), New Delhi. It was based on a cross sectional study in 1993 in four adjoining villages in Haryana, 30 Kilometres from Delhi. The population involving 8595 were screened for seizures and paralysis, the latter mainly for sequelae of poliomyelitis. Seizures were defined as two or more afebrile seizures. Focal motor, complex partial and absence seizures were also categorized. Active seizures were defined as at least one seizure in the last five years. The crude prevalence rate was 4.74 for males, 6.57 for females and overall 5.58.

Yelandur study (Mani, Rangan, Srinivas, Kalyanasundaram, Narendran & Reddy, 1998)

This study was a rural–based in Yelandur taluk in the plains of Mysore district Karnataka state with a small tribal population in BR Hills. This was based on a door-to-door survey in a population of 64,963. They came from 13,562 families with an average of 4.79 members per family. The prevalence period taken up was from 1st April 1990 to March 31st 1991. The inclusive diagnosis of seizures and seizure types was not made on specialist opinion, but on criteria described in ICBERG studies (Placencia, Shorvon, Paredes, Bimos, Sander, Suarez, & Cascante, 1992). The protocol used was elaborate. Its sensitivity was 72.2%, specificity 99.9%, positive predictive value 95.9% and negative predictive value 99.3%. Single, febrile

acute symptomatic and drug/alcohol abuse related seizures were excluded. Active and inactive seizures were defined (Das & Sanyal, 1996) and validation resurvey was carried out on 4060 individuals from 662 families, enabling one to determine the maximum prevalence figures. The prevalence was available as a range of minimum and maximum figures rather than a single figure. The minimum and maximum crude prevalence rate was 3.91-4.63 for active seizures, 0.28-0.77 for inactive seizures and 4.19-5.41 for life-time prevalence. The minimum prevalence rate for active seizures was adjusted for US population, 1970 as per age and gender. They were 4.2 for men, 3.3 for women and 3.8 for both combined. The incidence figures for the same period i,e, for the first year only was 49.3 per 100,000 population. The problem of hot water seizures (HWE), peculiar to this part of the state was also noticed (Sander, 1993). In case of these reflex seizures, the minimum and maximum crude prevalence figures were 2.49-2.99 for active HWE, 0.35-0.85 for inactive HWE and 2.85-3.83 for lifetime prevalence. The figures for HWE are approximately two-third of that of the non-hot water seizures group, reflecting the high prevalence of this peculiar entity in the area studied.

Bangalore urban rural study (Sathishchandra, Gururaj, Gourie, Devi, & Subbakrishnan, 1996)

Bangalore urban-rural neurological study was conducted from the Department of neurology, NIMHANS, Bangalore. The methodology was very much akin to the Gauribidnur protocol. The population in such selected areas were subjected to a door to door survey and was made up of 51,502 from the urban and 51,055 from the rural areas, giving a total of 1, 02,557. The literacy rate was as high as 80%. The study yielded 905 individuals with seizures 296 in urban and 609 in rural areas. The crude prevalence rates for active seizures were 5.75 for urban and a very high 11.92 for rural Bangalore, giving a figure of 8.82 for the entire area. The prevalence rate for children was 4.42 for urban and 13.46 for rural areas, the latter thrice the former, though in contiguous area. HWE accounted for just 4 cases in Bangalore city (0.08 per 1000) and 58 (1.14 per 1000) in the rural areas giving an area figure of 62 cases (0.6 per 1000) only.

Kerala study (Radhakrishnan, Pandian, & Santosh, 2000)

An excellent and exhaustive survey was conducted in central Kerala. Survey was performed in 10 panchayats in three adjacent districts between Cochin and Calicut by the neurological team from Sree Chitra Tirunal Institute of Medical Sciences and Technology, Trivandrum. Seizures and other neurological services with EEG facilities were started in this institute before the survey. The population surveyed was a massive 2, 38,102 individuals from 43,681 households with an average of 5.45 members per household. Literacy rate was high (more than 95%) as expected for Kerala. The screening instrument was elaborate. This was subjected to a pilot study on 200 known patient of seizures, 200 healthy workers and another group with other neurological disorders. Sensitivity was 100% and specificity was 72%. Epilepsy was defined as more than one afebrile seizure, unrelated to acute metabolic derangements or withdrawal of alcohol or drugs. Active seizures were considered as at least one seizure in the last five years, irrespective of AED administration. The crude prevalence rate was 4.9 as the minimum and 5.4 as the maximum. The former was adjusted to the 1980 US census population, with a revised figure of 4.7.

The prevalence figures from several studies are summarized for active seizures and are depicted in table 2.

Table 2. *Prevalence of seizures in Indian population*

Author (year)	Region	Prevalence (per 1000)
Issac (1987)	Baroda	1.28
Issac (1987)	Calcutta	1.71
Issac (1987)	Patiala	3.17
Issac (1987)	Solur	6.95
Koul et al. (1988)	Rural Kashmir	2.47
Das and Sanyal, (1996)	Rural West Bengal	3.06
Mani et al. (1998)	Yelandur	3.91-4.63
Bharucha et al. (1988)	Parsi community, Bombay	3.6
Gourie et al. (1987)	Gauribidanur, Karnataka	4.6
Radhakrishnan et al. (2000)	Rural Kerala	4.9-5.4
Kokket and Verma, (1998)	Rural Haryana	5.58
Mathai, (1971)	Vellore, Tamil nadu	8.97
Satishchandra et al. (1996)	Urban Bangalore	8.82

(Source: Sridharan (2002), Current science, 82 (6), March, 2002)

The figures ranged widely from as low as 1.28 in Baroda to 11.92 in rural Bangalore. As India is a large country with diverse cultures and food habits, local figures for prevalence and incidence does not seem appropriate to extrapolate the results across the country.

To summarize, the data presented in literature varied widely across regions as uniform criteria of material and method were not adopted. The organizations and the funding available also needs to be considered. For example, SCTIMIST (Trivandrum), NIMHANS (Bangalore) and AIIMS (New Delhi) are government funded larger postgraduate medical colleges and hospitals, while the Yelandur study was carried out by a group of individuals from a nongovernmental organization. They were based on the knowledge and facility available then, and their validity today should be analyzed from that perspective. However, the inferences made then are still relevant for future reference.

4. Causes of epileptic seizures

The normal human brain under some circumstances may produce an epileptic seizure. Many alterations in homeostasis that originate outside the central nervous system (CNS) can provoke epileptic seizures, especially in children. A complete spectrum of epilepsies ranging from purely “lesional” to purely “functional” epilepsies has been encountered in previous research. These definitions however imply that a seizure is not an entity since it may result from many causes (Lebrun & Febbro, 2002).

Seizures have a genetic basis and runs in families. A positive family history of febrile seizures points to the importance of genetic factors and common environmental exposures. Saidulhaque (1981) and Farwell, Blackner, Sulzbacker, Adelman, and Voller (1994) have reported 20 percent and 29 percent of children with positive family history in their study, respectively. Genetic (or constitutional) factors and structural abnormalities are often found in various proportions in the same client. In other cases it can be caused by an illness such as meningitis or by damage to the brain that may have happened before, during or after birth. In general the production of a seizure probably requires a brain lesion and a genetic predisposition, both with its resulting hyper excitability on the one hand and afferent

stimuli of either cerebral or extra cerebral origin on the other. The state of the brain and its excitability is therefore influenced by both intrinsic factors such as circadian rhythms, stress or any other factors.

Acute or no recurrent seizures are extremely varied. The convulsion is merely a symptom of another disease which has somehow triggered the cells of the brain to produce an irregular electrical discharge. Some causes of acute seizures are:

a. Fever accompanying a routine illness in a child under six
b. Infections (for example, meningitis [or infection of the tissues covering the brain],
   Encephalitis [or infection of the brain itself], brain abscess, tetanus, malaria, Dysentery)
c. Brain hemorrhage from birth injury or other injuries or from blood disorders such
   as hemophilia or sickle cell disease
d. Poisons such as lead and camphor
e. Sudden lack of oxygen
f. Sudden swelling of the brain as a result of water retention
g. Brain tumor
h. Idiopathic- Recurrent convulsions for which no causes can be found are called idiopathic seizures. Most recurrent convulsions fall into this category. These are easily recognized, but in many instances, clinical observation for several years may be necessary to arrive at a firm diagnosis.

Some of the more common causes are listed below:

a. permanent damage to the brain following hemorrhage from birth injury or accidental injury
b. permanent damage following infectious disease, lack of oxygen or poisons
c. Degeneration of brain tissue caused by chronic disease of the central nervous system
d. Congenital malformations of the brain
e. Parasitic brain infections such as syphilis
f. Chronic disorders of body chemistry, for example, low blood sugar, abnormal calcium metabolism, untreated phenylketonuria and kidney failure.

Triggering mechanism as a causative factor

Seizures induced by acute cerebral pathology or by extracerebral disturbances are called occasional seizures because they occur only in response to provoking circumstances. Many seizures do seem to occur under the influence of a triggering factor. These must be distinguished from seizures. This is a spontaneously recurring condition. The classic example of occasional epileptic seizures is that of febrile convulsions, which occur only in response to a rise in body temperature. In the induced seizures the provoking agents encompass a diversity of factors and mechanisms. These may be divided into no sensory and sensory; among the no sensory group are hyperthermia, hyperventilation, recurrent hypoglycemia, metabolic disorders, physical stress, sleep deprivation, emotional disturbances, and so forth. Specific entity of Hot Water Epilepsy (HWE) was found more frequently in Mysore, Mandya and Hassan districts than in Bangalore, Tumkur and Kolar Districts (Mani, 1996). Researchers speculated that the greater prevalence of active seizures, especially in rural Bangalore, could possibly be due to higher frequency of cysticercosis resulting from deteriorating environmental sanitation. The sensory triggers may be visual (e.g., photogenic television, reading), auditory (musicogenic), vegetative, tactile, and proprioceptive (Aird, 1988; Friis, 1990). They may be spontaneous normal sensory stimuli of daily life or unexpected sensory experiences (startle reaction), and at times self-induced.

Most seizures likely have a multifactorial origin. Regardless of etiology and pathology of the cerebral lesions, the pathophysiology of seizure discharge may be identical. The basic mechanism appears to be prolonged depolarization with consequent hyperactive and hyper synchronous discharge from abnormal neurons. Such epileptic discharge may be circumscribed or may spread, and thus activate distant neurons causing them secondarily or in become instances independently epileptogenic.

The biochemical basis of epileptic discharge remains incompletely understood, Experimental studies with epileptogenic cortex from animals and human patients suggest that the production and maintenance of such discharge involves (1) an accumulation of excess acetylcholine or other excitatory substance, (2) increased membrane permeability with increase in intracellular sodium ions and (3) depletion

of intracellular potassium ions. Systemic metabolic disturbances such as hypoglycemia, hypocalcemia, anoxia, hypocapnia, and pyridoxine deficiency are believed to exert their influence upon these basic factors and may become secondarily responsible for initiation of the epileptogenic discharge (Aird, 1988).

5. Types of epileptic seizures

Seizures syndromes are clusters of signs and symptoms customarily occurring together (Commission on Classification and Terminology of the International League Against Epilepsy, 1989). The signs and symptoms depend on the type of seizures, the mode of seizure recurrence, neurologic findings and neuro radiologic or other findings of special investigations. A syndrome can have more than one cause and consequently it may have different out-comes (eg, West syndrome with both cryptogenic and symptomatic types).

The International League Against Epilepsy (ILAE) classification uses two sets of criteria concurrently. One set is topographic, which leads to a dichotomy between generalized and partial epilepsies, the latter were renamed as localization-related epilepsies. The other set of criteria is based on the etiology of the disorder. One of its forms is symptomatic or secondary epilepsies which are probably the result of some undetermined brain disorder. Another type is cryptogenic seizures, those that are not due to any brain lesion or diseases but a possibility of genetic propensity to generate seizures is present. Last type is termed idiopathic indicating that in such cases, the seizures is a disease that is not secondary to any other condition. Classification of epileptic seizures as per the ILAE (1989) are provided in Table 3.

Table 3: *Proposed classification of epilepsies and seizure syndromes, ILAE, 1989*

1. Localization – related (focal, local, partial) epilepsies and epileptic syndromes	1. Idiopathic (with age-related onset)	1. Benign childhood seizures with centro temporal spikes 2. Childhood seizures with occipital paroxysms 3. Primary reading seizures
	2. Symptomatic	1. Chronic progressive epilepsia partials continua of childhood 2. Syndromes characterized by seizures with specific modes of precipitation 3. Temporal lobe epilepsies 4. Frontal lobe epilepsies 5. Parietal lobe epilepsies 6. Occipital epilepsies
	3. Cryptogenic	
2. Generalized epilepsies and syndromes	1. Idiopathic (with age-related onset)	1. Benign neonatal familial convulsions 2. Benign neonatal convulsions 3. Benign myoclonic seizures in infancy 4. Childhood absence seizures (pyknolepsy) 5. Juvenile absence seizures 6. Juvenile myoclonic seizures (impulsive petit mal) 7. Epilepsies with grand mal seizures on awakening 8. Other generalized epilepsies 9. Epilepsies with seizures precipitated by specific modes of activation
	2. Cryptogenic or symptomatic	1. West syndrome 2. Lennox-Gastaut syndrome 3. Seizures with myoclonic astatic seizures 4. Seizures with myoclonic absences
	3. Symptomatic	1. Nonspecific etiology 2. Early myoclonic encephalopathy 3. Early infantile epileptic encephalopathy with suppression- burst electroencephalogram 4. Other symptomatic generalized epilepsies not defined above 5. Specific syndrome (including diseases in which seizures are a presenting or predominant feature)
3. Epilepsies and epileptic syndromes undetermined whether focal or generalized	1. With both generalized and focal seizures	1.Neonatal seizures 2. Severe myoclonic seizures in infancy 3. Seizures with continuous spike waves during slow- wave sleep 4. Acquired epileptic aphasia (Landau-Kleffner syndrome). 5. Other undermined epilepsies not defined above
	2. Without unequivocal generalized or focal features	
4.Spécial syndromes: situation-related seizures	1. Febrile convulsions 2. Isolated seizures or isolated status epilepticus 3. Seizures occurring only when there is an acute metabolic or toxic event	

A. Generalized seizures

About a third of childhood seizures are generalized.

1) Tonic-clonic seizures

This is also called a grandmal seizure and is the most common form of generalized seizure. The child loses consciousness and falls to the ground. The arms and legs stiffen. This phase usually lasts for only a few seconds and, as breathing may be shallower than usual, the child's lips may turn blue. This is followed by a rhythmic jerking of the arms, legs and often the entire body - this can be violent and quite alarming to the observers. These are known as involuntary or clonic movements. This stage normally lasts less than five minutes. It may be associated with tongue biting and incontinence of urine or feces. The child is later confused, drowsy and may have a headache and doesnot remember what happened. The core of the attack consists of a stereotyped series of motor autonomic manifestations that are associated with an immediate loss of consciousness (Gastuat & Broughton, 1972). The tonic phase comprises a sharp, sustained contraction of muscles that produces a fall to the ground that is often injurious when the patient is standing or sitting. The patient lies rigidly in an extensor posture that is often preceded by a transient stage of fluxion. The tonic contractions of the diaphragm and intercostal muscles inhibit respiration and cyanosis occurs. After 10 to 30 seconds, the tonic phase gives way to clonic jerks, which often follow a brief inter- way to clonic jerks which often follow a brief intermediate period of vibratory tremor. The jerks are bilateral and symmetric. The jerks may be accompanied by brief expiratory grunts, as diaphragmatic contractions force air against the closed glottis, froth appears at the mouth and the tongue may be bitten during this stage. After 30 to 60 seconds muscular relaxation occurs (Gastuat, Broughton, & Roger, 1974a).

2) Tonic Seizures

This is a type of seizure in which the patient’s body stiffens with increased tonicity of the entire musculature, and consciousness is lost. Such attacks arise when the midbrain is primarily involved in the seizure discharge.

3) Absence seizures

Absence seizures are also known as petit-mal attacks. They are episodes of loss of consciousness without falling or involuntary movements. The child stops whatever he or she is doing, looks vacant for 5 to 20 seconds and then continues what he or she was doing as if nothing had happened. These attacks occur after the age of two years and are most common between five and nine years of age. Most children grow out of them by their teenage years but, rarely they continue into adult life.

4) Juvenile myoclonic seizures

Juvenile myoclonic seizures have a genetic basis and runs in families. It causes episodes of jerking of the hands, arms or entire body. The jerks occur most frequently in the early morning. It usually begins in late childhood and the affected child may also suffer from absence attacks or tonic-clonic seizures.

5) Infantile spasms (ISs)

ISs (West syndrome) are a remarkable age-dependent seizures syndrome. The syndrome occurs almost exclusively during the first year of life. In almost all patients, the spasms are associated with mental retardation or deterioration, and in a majority, with a striking EEG pattern called hypsarhythmia.

6) Lennox Gastaut syndrome (LGS)

LGS is one of the most severe forms of childhood seizures. Its is defined by the occurrence or multiple seizure types, the most characteristic of which are tonic and atonic seizures, atypical absences, myoclonic or myclonic astatic seizures and episodes of nonconvulsive status epilepticus. The syndrome may be cryptogenic in 25% to 30% of cases or it may be symptomatic of congenital or acquired brain anomalies. The outcome of the syndrome is gloomy, as more than 90% of patients are left with mental retardation or behavioral disturbances. In infancy and early childhood, myoclonic seizures are often associated with other types of brief seizures, as well as with cognitive and behavioral abnormalities even when no obvious brain lesion is present to account

for it and their prognosis is often guarded. They should be distinguished from other syndromes with frequent brief attacks and repeated falls that result from the tonic and atonic seizures observed in LGS. During the first two years of life convulsions from any causes are more common than at any other period.

7) Status epilepticus

Grand Mal seizures may occur in series without consciousness being regained between attacks. This condition is known as "status epilepticus". It may be induced by sudden withdrawal of anticonvulsant medication. If not treated promptly and effectively, the status may persist for many hours or days with development of serious sequelae of even death. Transient postictal signs and symptoms include aphasia, ataxia and mental sluggishness. Persistent symptoms and signs may develop indicating that irreversible brain damage has occurred as a result of prolonged cellular hypoxia.

B. Partial seizures

About two thirds of childhood seizures are partial seizures. Symptoms vary depending on part of the brain affected.

1) ***Simple partial seizures (SPS)***

In SPS the child does not loose consciousness. Symptoms can include twitching, numbness, dizziness, nausea, disturbances to hearing, vision, smell or taste or a strong sense.

2) ***Complex partial seizures (CPS)***

In CPS the child's consciousness is affected and he or she will have no memory of the episode. Symptoms can include the child making strange faces, swallowing, lip smacking, chewing and muttering while being apparently awake, but not aware of what is going on around the person.

6. Age factor and epileptic seizures

It is well known that seizures are much more common in children than in adults. In genetic seizures the appearance and disappearance of the clinical seizures and the electrical dysrhythmia are age dependent. Seizures that occur before 2 years of age suggest a metabolic disorder or an underlying structural defect of the brain. Newborn infants are more likely to have poorly organized movements, such as twitching, trembling or shaking rather than a bonafide tonic-clonic convulsion. They often have apneic or blue spells, limp or stiff spells, vasomotor changes with flushing, pallor and clamminess. Such seizure discharges probably originate mostly in subcortical structures and as the cortex develops more differentiated types of seizure patterns become evident.

Seventy five percent of structural growth of the brain occurs in the first 2 years of life. Generally speaking, the younger the child and the more immature the brain is, the more susceptible it is to injury and the more far-reaching the resultant functional disorganization. During the first two years of life convulsions from any cause are more common than at any other period. During six months until five or six years of age convulsions are most commonly caused by infections within and outside of the central nervous system. After six years of age the first convulsion in a child is probably idiopathic. Early adolescence is a common time for the first idiopathic seizure to occur. Deterioration in children with epileptic disorders can be due to several causes. Drug toxicity, school absences, low expectations (Long & Moorwe, 1979) and other sociopsychologic effects of status epilepticus are well recognized (Aicardi & Chevrie, 1983).

7. Assessment of epileptic seizure

A diagnosis of seizures has potentially serious consequences for health, psychosocial well-being and economics and therefore it should be made with a high level of certainty (Fisher & Leppik, 2008). Diagnosing seizures and the type of seizures is like putting the pieces of a puzzle together and includes information from many people and different tests. The first question is to find out if the person had a seizure and then to know the type of seizure or seizure syndrome that best explains the event. To do

this, more information will be needed, including details of the medical history, blood tests, EEG tests and brain imaging tests such as CT and MRI scans (Arzimanoglou, Guerrini, & Aicardi, 2004).

The high-voltage slow waves (HVS) on EEG associated with epileptic spasms can be used to clarify their characteristics and their relation to the pathophysiology of spasms in West syndrome and related disorders. The patterns of distribution of the ictal HVS may be related to the abnormal activation of the brain in the generation of spasms (Kobayashi, Oka, Inoue, Ogino, Yoshinaga, & Ohtsuka, 2005).

Neonatal seizures may be present at any age. Concurrent video-EEG recording during seizures (Binnie, 1991; Mizrahi, 1984) is particularly useful in these difficult cases as it allows the detection of subtle clinical seizure phenomena and a better interpretation of the EEG phenomena, which can then be precisely correlated with the clinical changes. Computerized motion analysis of videotaped seizures in neonates with seizures provides novel quantitative information regarding the behavioral characteristics of neonatal seizures (Karayiannias, Tao, Xiong, Sami, Varughese, Frost, Wise, & Mizrahi, 2005). It is not easy to determine the location of the cerebral generators and the other brain regions that may be involved at the time of an epileptic spike in the scalp EEG. The possibility to combine EEG recording with functional MRI scanning (fMRI) opens the opportunity to uncover the regions of the brain showing changes in metabolism and blood flow in response to epileptic spikes seen in the EEG (Gotman, 2008).

Radhakrishnan et al. (2000) used a questionnaire to detect the type of epileptic seizures. It was made up of six independent questions. The questions were, loss of consciousness, sudden fall, uncontrollable shaking of arms and legs, loss of bladder/bowel control (Generalized tonic clonic seizures- GTCS), blank spells with awake, brief episodes of sudden jerking of arms or legs (myoclonic jerks). It is notable that none of the screening instruments, simple to elaborate, cover very rare seizure types like atypical absence or myoclonic absence. These two along with atonic seizures are often part of a mixed seizure pattern and practically never a sole seizure manifestation. According to Janz (1969) the diagnosis depends on the occurrence of five GTCSs on

awakening without other types of schedules of attacks. However, the occurrence of some seizures during sleep or in the evening when the patient is relaxing is possible (Janz, 1991; Tsuboi, 1977b). The epilepsies of childhood that are exclusively or predominantly by GTCSs are much more heterogeneous than the corresponding epilepsies of adolescence and they do not represent a single syndrome but rather a collection of various seizure disorders with different causes and courses.

The subjective and objective information include the electrical activity of the brain, what the brain looks like, how the individual is feeling, how the seizures may be affecting the individual and possible causes of seizures. All these information put together would provide complete picture about the epileptic seizure (Schachter, 2005).

8. Epileptic seizures in communication disorders

Children with developmental disabilities are at higher risk for seizures than the general population. Epilepsy is one of the most common neurological disorders occurring in children with communication disorder. It includes borderline intelligence, speech regression, hearing loss, cerebral palsy, mental retardation, developmental delay, pervasive developmental disorders, apraxia, attention deficit hyperactivity disorder, delay in speech and language, learning disability, cleft lip and palate, dysarthria, misarticulation and stuttering. Epilepsy and communication disorder may occur together and both may result from same underlying brain disorder. However, the causal association between epilepsy and language impairment is poorly documented due to constraints in epidemiological methods.

A. Acquired injury to brain

Arzimanoglou, Guerrini, and Aicardi, (2004) studied 122 children who suffered a sudden onset of hemiplegia. Eighty-nine patients (Group 1) presented with hemiplegia following a seizure lasting more than 1 hour; 31 of these were seizures longer than 24 hours. Thirty-three patients (Group 2) developed an acute hemiplegia unassociated with seizures. In group 1, 90% of the cases had an age of onset before 2 years, while in group 2 patients ranged from 7 to 13 years. In 76% of the children, fever

was present at the onset of the seizures. Thirty-two children had a history of antecedent infection. The children in group 1 tended to have a history suggestive of previous neurologic dysfunction, low birth weight, previous seizures, or transient neurologic signs. In contrast, only one of the children in group 2 had a history of antecedent convulsion and only one had a history of abnormal mental development prior to the seizure.

B. Aphasia and Seizures

Most aphasic patients have damage to the left cerebral hemisphere from a stroke. Trauma to the head and occasional cerebral diseases (such as a brain tumor) are few causes. An important physical concomitant of brain injury is the traumatic convulsive seizure. Petit mal seizures (they produce only temporary spasms or loss of attentiveness and grand mal seizures (which produce major muscle contractions and often a loss of consciousness) are usually seen only in patients who have had cortical damage caused by an external injury to the brain.

If cerebral damage causes permanent aphasia, and if this damage also brings on epileptic seizures, the severity of the chronic aphasia may increase during attacks. During an epileptic attack a patient may also evidence a language impairment resembling chronic sensory aphasia. In such a case verbal comprehension is severely disturbed while speech production is copious but garbled. There may even be jargon aphasia, i, e, a verbal output replete with paraphasias (involuntary word substitutions), perseverations (unintentional replacements of adequate words by words used a little earlier), and neologisms (non-existent words produced unintentionally) by the patient. However, the patient may be able to recite overlearned series or prayers (nearly) correctly. Jargon aphasia may be accompanied by logorrhea, the patient being prolix in addition to producing deviant speech. Presumably the frequency of the bioelectrical paroxysms is such that it prevents the restoration of a normal verbal situation inter-ictally. This severe condition is called status epilepticus with aphasia or aphasic status epilepticus. The severity of the aphasia may fluctuate during the prolonged spell. If aphasia is the only clinical manifestation of the status epilepticus, it may be mistaken

for a language disorder of vascular origin and the patient is suspected of having suffered a stroke. Only electroencephalography can reveal the epileptic nature of the verbal deficit. Ictal aphasia regularly coincides with abnormal bioelectrical activity in the EEG. It may also happen that the patient presents not with constant aphasia but with a quick alternation of (near) normal and aphasic periods. In such a case the successive aphasic episodes generally correlate with electroencephalographic disturbances.

Hecaen and Piercy (1956) noted that there are proportionally more left handed than right handed patients with seizures who evidence ictal motor aphasia. From observation they concluded that in left handed individuals, language tends to be represented bilaterally in the brain, i.e. in left handed the two hemispheres are more or less equally involved in language processing. This conclusion receives support from the finding that following acquired unilateral brain damage aphasia is significantly more frequent in left handed than in right handed patients (Satz & Bates, 1981). During seizures, clients may perform inadequate linguistic functions. The excessive or disorderly firing of neurons may entail the suppression, activation or alteration of linguistic functions. Following are the ictal, pre ictal and post ictal disturbances reported by Lebrun and Fabbro (2002):

a. ***Confabulation***

Patients with partial seizures originating from the right hemisphere may at times produce confabulatory speech during attacks. Their discourse is syntactically correct and relatively coherent but it does not fit the situation, or contains obvious inaccuracies. This type of disorder may be considered a disturbance of pragmatics. An instance of this peculiar condition has been reported by Guard, Fournet, Sauetreaux, and Dumas (1983).

b. Vocalizations

Some patients produce a protracted or iterative vowel sound during fits. These involuntary vocalizations can be associated with an epileptic focus in either hemisphere. Not infrequently the focus lies in or near the supplementary motor area in the superior part of the medial aspect of the frontal lobes.

c. Speech automatisms

During partial seizures patients may also unintentionally utter words. These involuntary verbal productions are called speech automatisms. The utterances consist at times of mumbled words or of expletives. The latter are called paroxysmal coprolalias. For this rare clinical manifestation the phrase cursing and cursive seizures has been coined.

d. Palilalias

During seizures, patients may involuntarily repeat the same utterance a great number of times. The palilalic utterance may comprise a single syllable or a single word or, on the contrary, consist of a well-formed sentence. At times, spontaneous palilalias consist of reiterated neologistic strings of phonemes. Now and then, slight to moderate phonemic differences are observed between successive repetitions, the neologisms are phonemically akin but not strictly identical.

e. Pre-and post-ictal verbal disturbances

Pre-ictal motor aphasia may be associated with a strong urge to communicate verbally. The patients want to speak or write but find themselves unable to do so (Kapur, 1997). Generally patients are aware of their pre-ictal linguistic difficulties. Indeed, they often learn to recognize them as antecedent signs of a seizure. Post-ictally, there may also be transient aphasia of the anomic type. The patient does not make language errors but has word-finding difficulties. An access to their mental lexicon is temporarily impaired. Post-ictal aphasia is generally observed only after a seizure originating from the left hemisphere.

The aphasic patients develop a marked withdrawal from society and from therapy as a result of the seizure. At times some convulsive seizure occurring even once causes the patient to be away from all social contacts. Following such an experience, patient would exhibit great changes in his attitude toward himself and his family. Occasionally he may indulge in uncontrollable spells of laughter. Self-control may be recovered by avoiding conversational situations momentarily. An understanding of

one's own limitations is rarely achieved easily even by normal individuals and it is far more difficult for an individual with aphasia to achieve such insight because his mental apparatus has been damaged and his associational connections are impaired. The effects of the appearance of seizures upon the patient, his attitude towards recovery, must however, be recognized and adequately covered during therapy. The therapist must be extremely patient and keep encouraging the client to help himself (Lebrun & Febbro, 2002).

C. Acquired childhood aphasia

The Landau- Kleffner syndrome (LKS) is defined as an acquired childhood aphasia with mainly bitemporal paroxysmal EEG abnormalities. It is a seizure disorder without demonstrable focal brain lesions and a regression or stabilization of the disease after a variable time (Tassinari, Rubbolli, & Vopi, 2002). The children have normal motor and intellectual development and the language disturbances develop over a relatively brief period. The loss of acquired language is usually total or profound but nonverbal skills are generally completely or at least relatively preserved. Behavioral disturbances of various types can be observed in up to two thirds of patients. Clinical seizures are experienced by 75% to 85% of patients at some time, although they do not necessarily appear at the very beginning of the disorder. The course is initially progressive but fluctuations, stabilization or improvement of speech may occur over the years, although long follow-up is available for only a few cases.

Lee, Schottler**,** Collins, Lanzino, Couture, Rao**,** and Eisenman (1997) proposed a classification of epileptic aphasias that identified a number of potential mechanisms. The convulsive group could include children who lose language comprehension by a number of different mechanisms as a consequence of

1. convulsive status
2. post-ictal phenomenon (Todd's paralysis)
3. Primary pathology (e.g., temporal lobe inflammatory or malignant disease)
4. Minor epileptic status
5. Psychological reaction to seizures
6. Organic event occurring with seizures but not fulfilling the criteria 1-5

In this condition, EEG abnormalities are seen especially during sleep in temporal areas, where verbal language and acoustic information is processed. The typical language disturbance in epileptic aphasia is a marked deficit of auditory comprehension, which differs from the usual features of the more common aphasias of childhood. The defect is an inability to decode sounds which is a necessary step in language understanding (Rapin, 1999). The language disorder evolves in a systematized manner beginning with sensory aphasia followed by auditory agnosia and finally by word deafness. The full deficit evolves over weeks or even months although an abrupt onset or a sudden aggravation following the occurrence of a seizure has been reported in some cases (Dugas, Masson, & Leheuzey, 1982).

Aphasic arrest is a phenomenon in which speech production is stopped when certain areas of the cerebral cortex are stimulated electrically. It was described by Penfield and Rassmussen (1950) in their neurosurgical work for intractable seizures. Lesser et al. (1986) identified other aspects of aphasic arrest including the interference of comprehension by the electrical stimulation of Wernicke's area. These observations make it clear that language comprehension and production can be interfered with by artificial stimulation. They are the basis of the hypothesis that abnormal electrical discharges of the cerebral cortex that occur in seizure disorders can produce aphasias.

Behavioral disturbances commonly accompany the LKS and they may be prominent. Most commonly, they consist of hyperkinesias and outbursts of rage with aggressiveness and opposition (Roulet, Deonna, & Gaillard, 1991). In some patients anxiety, gestural stereotypes, avoidance of interpersonal contact or bizarre behaviour may suggest the presence of a psychotic component (Rapin, 1995; Deonna, 1991; Sawheney, Suresh, Dhand, & Chopra, 1988). Researchers suggested that the LKS and the behavioral disturbances observed in patients with "electrical status epilepticus of slow sleep" represent the two poles of a continuum, in which the latter is characterized by predominant psychiatric disturbances and the former, by predominant language difficulties. In some children, dysapraxia components may be evident (Hirsch, Maquet, & Metz-Lutz, 1995) and motor disturbances, including abnormalities of tonus, abnormal movements, ataxia and marked clumsiness (Neville, Burch, & Cass, 1998).

Course and Relationship between Seizures and Language Disturbances

The relationship between the activity of clinical seizures, the intensity of EEG disturbances and the degree of aphasia in the LKS probably differs from cases to cases. Indeed, continuous paroxysmal EEG activity may appear long before the language disturbances and its intensity may fluctuate independently of the severity of language problems (Hirsch et al., 1995). Some investigators (e.g., Nanda, Johnson, & Keogh, 1977) have found a relationship between predominantly temporal activity and language difficulties, although this may be difficult to ascertain by surface recording. Several authors have suggested that the evolution of aphasia may depend on the occurrence of continuous spike- wave activity during slow sleep (Deonna, 1991; Paetau, Kajola, & Korkman, 1991; Shinnar, Berk, & Moshe, 1990; Hankey & Gubbay, 1988) because the individual's language appears to improve during the period when the sleep EEG is less abnormal. In some patients, the severity of aphasia clearly fluctuates with the frequency and/or severity of the clinical seizures and these cases may represent one subgroup of the syndrome of epileptic aphasia. Beaumanoir and Dravet (1992) in a review of 77 cases found that the aphasia remained stable in 40 patients, whereas it was more or less variable in the remaining 37 cases. Sudden increase in the severity of aphasia may even occur relatively late in the syndrome; they have been up to 7 years after onset (Dugas et al., 1982). The prognosis is quite unpredictable. It tends to be more favorable in patients with a relatively late onset, whereas onset before the age of 5 years is associated with a more severe outlook (Bishop, 1985; Dugas et al., 1982). The persistent localization of EEG anomalies to the speech cortex and the duration of children with slow wave spikes (CSWS) activity are also associated with an unfavorable outcome (Tassinari et al., 2002).

D. Benign Rolandic Seizures and language disturbances

The widely held distinction between benign and malignant epilepsies in childhood has been clinically useful in both clinical management and research (Sridharan, 2002). However, recent studies of conditions previously considered benign in respect of its effect on language development, Rolandic seizures (Staden, Issacs, & Boyd, 1998), have shown that this is far from the whole picture. When developmental

arrest and disorders do occur in association with seizures, particularly in respect of language, the long-term consequences are increasingly reported and command attention. Benign rolandic seizures (BRE) usually begin between 4 and 10 years of age. It is characterized by partial sensori-motor seizures that produce mid-temporal spikes of EEG. These seizures often occur at night and are generally considered easy to confirm a range of neuropsychological deficits including speech and language impairments such as auditory processing, discrimination difficulties and problems with written language. Sometimes general learning difficulties and oro-motor dysfunction are reported. Of the 20 children with BRE reported by Staden, Isaacs, and Boyd (1998), 12 completed a follow-up program three years later, when they were aged 9 to 16 years. Using the 'form, content, use' model of Bloom and Lahey (1978) and based on the assessments used in the first battery, a number of language assessments were carried out. A questionnaire about school performance and further EEG were also used. The main findings of the follow-up cases are as follows:

- 7 out of 10 children classified as having language dysfunction in the original study were no longer classified as language impaired at the follow-up
- 2 of the 5 children not found to be language impaired in the first study did demonstrate language impairment at the follow-up
- only 2 children scored within normal limits on all measures
- all 7 children still taking AEDs at the time of follow-up had language difficulties
- There were no specific connections between such variables as age at first seizure, or time since onset of BRE, but there was a tendency to improvement of language function over time.
- auditory-verbal learning, one of the five specific areas of language impairment identified in the original study, showed the most significant improvement between the two studies
- While the language impairments detected in the follow-up were subtle in nature, they have important educational and social implications. They were largely confined to complex language skills such as defining words, formulating sentences, recalling narrative and basic literacy skills.

These difficulties had educational implications being a high risk for literacy difficulties. Similar high-level language problems have also been reported in the long-term follow-up of children with other types of acquired language problems (Lee et al., 1997).

E. Specific language impairment and seizures

Seizures are known to co-exist or develop along with a range of childhood conditions in which there is central nervous system (CNS) involvement. Corbett (1991) reports incidence of 6% of seizures in children with specific language difficulties. Robinson, Baird, Robinson, and Simonoff (2001) reported 21% with definite history of seizures and a further 11% where the history was 'questionable' in respect to seizures. He proposed three hypotheses to account for this. Firstly, 'the seizures themselves might cause the language disorder by interfering with brain function'. Against this hypothesis he said that the 'great majority of children with seizures do not have specific language disorders'. Secondly he proposed that 'genetic factors that predispose to specific developmental language disorder might also lead independently to seizures'. Against this his data on children with a family history of language disorder showed a negative trend towards the development of seizures. His third hypothesis was 'that seizures may indicate abnormal brain development or damage' and in favour of this his data demonstrated a correlation between a history of seizures and antecedent abnormalities which might cause language disorder. This hypothesis implies that language disorders in childhood are not isolated abnormalities but 'associated with other kinds of cerebral abnormality or dysfunction'.

F. Dysarthria and seizures

In some cases speech remains possible during ictal episodes but articulation is impaired. It may be concomitant with drooling and swallowing difficulties. Post-ictally, there may also be transient dysarthria.

a. Paroxysmal dysarthria in multiple sclerosis

In patients with multiple sclerosis, active or expanding stages of the disease may be accompanied by short spells causing various transient cerebellar symptoms,

including dysarthria. The latter may be so severe as to render speech unintelligible. Consciousness is not altered during the fleeting episodes, which usually last from a few seconds to a minute or two. Patients whose speech becomes difficult to understand may deliberately refrain from speaking. This voluntary silence should not be mistaken for a speech arrest. The episodes may be ushered in by some dizziness or a sensation resembling a short aura. They occur spontaneously or when the patient is under emotional stress. They can generally be triggered off by hyperventilation. Paroxysmal dysarthria in patients with multiple sclerosis does not correlate with anomalies in the EEG. No abnormal bioelectrical discharges can be detected during the episodes.

b. Speech arrests in Parkinson's disease

Parkinson patients may exhibit non-epileptic speech arrests. From time to time they suddenly become unable to activate their speech organs. They would like to communicate orally but are temporarily deprived of motor speech. During these episodes they may be unable to open their mouths on request, to chew and to swallow their saliva, which then drips out of their mouths (sialorrbea). These episodes of mutism do not correlate with EEG anomalies. Consciousness is preserved. The patients do not fall but they may be hypo-kinetic, i.e. move slowly and restrictedly.

c. Post encephalitic Parkinsonism

In patients with post encephalitic Parkinsonism which is a specific form of epidemic viral infection of the brain, spasms may occur in conjugate ocular muscles causing involuntary eye deviations. The attacks may range from a few seconds to hours. During these occulogyric seizures patients may unintentionally produce spontaneous palilalias or, if other people are present and if the patients attempt to communicate with them, they may repeat each of their sentences a great many times in sequence (Van Bogaert, 1934). They may also repeat the interlocutor's questions instead of answering them, so they may produce echolalias. Patients may also unintentionally utter profanities or let out yells. They are generally aware of this compulsive verbal behaviour and may repeatedly try to apologize (Wohlfart, Ingvar, & Helleberg, 1961).

d. Paroxysmal dysarthria or mutism caused by neuroleptics

Patients treated with neuroleptics, i.e. drugs that modify the biochemistry of the brain, particularly of the centrencephalic structures may have severe dysarthria or mutism, especially at the beginning of treatment. In patients who have become comatose following an overdose of sedative or anti depressant medication, temporary dysarthria is sometimes observed when consciousness is regained (Lebrun & Febbro, 2002).

G. Autism & Seizures

Autism and seizures frequently co-exist (Creak, 1963; Kolvin, Ounsted, & Roth**,** 1971). One investigation has demonstrated that two electroencephalograms with sleep tracings are required to evaluate this possibility (Ritvo et al., 1970). A high incidence of seizures in autism has been reported in earlier studies (Gillberg 1992). Seizures occur in 30 to 40% of autistic patients before 30 years of age (Gillberg, 1998). Slightly less than half of these experience various seizures types, including infantile spasm, with onset in early childhood. Such cases raise difficult therapeutic issues because the effects of the various deficits generally are multiplicative rather than additive. Steffenburg (1991) found that almost 9% of her sample (35 autistic and 17 autistic-like children) showed some evidence of brain damage or dysfunction. There have been several indications of a biogenic origin. The fact that autism is present so early in life is in itself suggestive of a biological problem. Autism has been linked to conditions which produce CNS impairment. Although perinatal complications have not been clearly linked to autism, Rubella (German measles) during pregnancy does cause a higher incidence of autism in the offspring (Chess, 1971). Other conditions in children which affect the CNS such as meningitis, encephalitis, tuberous sclerosis and phenyl-ketonuria have been reported to be associated with autistic patterns of behavior. Most of the autistic children have normal EEGs and neurological examinations earlier in life. Seizure disorders are more likely to clinically manifest in them as they become older (Rutter, Bartak, & Newman, 1970).

H. Stuttering and Seizures

Normalcy appears to hang on a very slender thread. A disorder with any great excitement or emotional tension may be sufficient to break that thread. In physiological terms the patient never deviates far from his convulsive threshold. In out parallel between stuttering and seizures both are similar only in symptoms. Both are more common in childhood than after puberty and are more frequent in males than in females. Idiopathic seizures are like familial stuttering. The spasms in both seizures and stuttering will aggravate the condition and so the fear of a convulsion may precipitate one. The most intriguing parallel, however, between stuttering and seizures is in the area of the physiological reactions, particularly with respect to sugar metabolism. The aspect of seizures to which attention is drawn is of course, its manifestation of convulsions-clonic twitching, tonic postural distortion, and squirming (Blumel, 1957).

West (1958) has proposed a parallel between stuttering and epileptiform seizures. West contends that stutterers are convulsion-prone and that they are linked to seizures and diabetes mellitus through the role of blood sugar. The diabetic is thought not to be convulsion prone because insulin and blood sugar are inversely related and the diabetic tends toward hyperglycemia whereas the epileptic tends toward hypoglycemia. The evidence related to this formulation is varied. Palasek and Curtis (1960) administered lactose placebos to nine persons with stuttering (PWS) and found a statistically nonsignificant tendency for stuttering to be reduced. Then, too, Glaser (1936) reported the responses of nineteen endocrinologists to a questionnaire inquiry into the relationship stuttering associated with hyperinsulinism and thyroid insufficiency. Kopp (1934), on the other hand, compared 49 PWS with 23 PWNS and found significantly higher blood sugar content in the PWS. Johnson, Stearns, and Warweg (1933) observed that blood samples were not indicative of disease. In fact, More (1959) hypnotized 12 PWS, asked them to talk about pleasant and unpleasant memories and then took blood samples. With one exception, blood sugar decreased rather than increased with unpleasant memories even though the entire subject stuttered more. Although PWS may be convulsion-prone, blood sugar level has not been clearly demonstrated as the link between their stuttering and any proclivities they may have to seizures.

A greater incidence of stuttering among epileptics has also been reported but the evidence is not impressive. Berry (1937a) compared the medical records of 430 children who stuttered with those of 462 who did not and found that 36 PWS as against 12 PWNS had suffered from seizures or convulsions prior to the age of five. Harrison's (1947) study of sixty epileptics also supported this position, in that stuttering was found 36 times as frequently as in the general population, disturbed laterality five times and twinning three times. He reported a large number of PWS having seizures. In contrast, Streifler and Gumpertz (1955) found only 1% of stuttering incidence among the epileptics respectively in their neurological clinic.

The mutism during the event of stuttering is comparable to the momentary lapse of consciousness that occurs in petitmal or minor seizures. In these attacks the patient is unable to think or talk and seems for a moment to be frozen with inhibition. He stares blankly ahead of him, may display blinking of the eyelids and a little jerking of the jaw, but stops the activity in which he is engaged. At the termination of the attack, he may resume his conversation or he may be confused and be unable to proceed. These are described as spells, lapses, teasers and tingles, etc. West (1958) of Brooklyn College has noted the similarity in the facial appearance of the child in a speech block and the child who has an attack of pynknolepsy. Pynknolepsy is a condition akin to petit mal perhaps identical with it. In any event the parallel to stammering is sometimes striking. There is an apparent relationship between stammering and seizures in the abnormality of the brain wave tracings. Interestingly enough the tracings suggest that "stutterers may be functioning in a state of reduced consciousness." The petit mal attack is cited to illustrate a biological pattern in which inhibition occurs. The struggles, which are relevant to stammering, are sometimes called hyperkinesias. They are so called spasms of the speech organs, the squeezing of lips, the thrusting of the tongue and the herculean effort to articulate. The parakinesias are the irrelevant reactions such as jerking the head, wringing the hands or stamping the foot found in stammering (Blumel, 1957).

Guil-lame and Mazars (1957) described a study in which three stuttering cases were having epileptic or convulsive symptoms. Surgery for the first case excised the focal cortical areas responsible for the epileptic discharges and the stuttering stopped. Anticonvulsant medication reduced the stuttering almost completely in the other two

cases. In four stutterers, Jones (1966) found that a similar remission of stuttering occurred following brain surgery to remove brain tumors or other brain pathologies.

I. Mental Retardation associated with seizure disorders

Monod, Pajot, and Guidasci (1972) observed that infants below 36 weeks of gestational age rarely had epileptic discharges due to the immaturity of the nervous system. Approximately 10% of children with seizures acquire mental retardation (Whylllie et al., 1986). Myoclonus seizures usually associated with mental retardation, do not represent a single disease entity but is rather a manifestation of a number of encephalopathic processes. Wortis (1970) reported 7 cases of myoclonus seizures associated with a variety of etiologies. Two young children with myoclonic encephalopathy presenting with acute onset often preceded by mild myoclonus, exacerbations with subsequent infections and improvement on steroids were reported. Both of these children were mentally retarded. Ricci and Vigevano (1993) studied 9 cases of infantile myoclonus with hypsarhythmia over a long period of time and noted that they developed into the Lennox–Gastaut syndrome (LGS). It is also known as *Lennox syndrome*, a difficult-to-treat form of childhood-onset seizures that most often appears between the second and sixth year of life and is characterized by frequent seizures and different seizure types. It is often accompanied by mental retardation, psychological and behavioral problems.

Infantile myoclonic spasms in association with hypsarhythmia were reviewed in a large series of patients followed for a long period of time by Jeavons, Harper, and Bower (1970). This type of seizure is usually associated with mental retardation (MR). Out of 80 cases only 13 were of normal intelligence. The use of ACTH modified the seizures but did not influence the associated MR. The prognosis was best in those cases in which the etiology was not clear. Hambert and Petersen (1970) reported 7 patients with myoclonus seizures. A variety of etiologies were represented ranging from lipidosis to Lafora's body. It was concluded that they consisted mostly of polyglucosans (glucose polymers), with varying amounts of proteins and sulfates.

McInerny and Schubert (1969) reported on the 70 patients with neonatal seizures who were available for follow-up at the age of 1-2 years. Forty percent were defined as "normal" (either by direct observation or via questionnaires answered by parents or physicians). 24% died and 34% had signs of significant "CNS morbidity" (usually mental retardation). The etiology was significant in determining the outcome. Hypocalcemia implicated in 30% of the neonatal seizures had a relatively good prognosis. Obstetrical complications (accounting for 31%) had a poor prognosis. Hypoglycemia in small for date infants was relatively benign.

Convulsions in the neonatal period carry an ominous prognosis. Amiel –Teson (1969) studied infants who had sustained severe CNS damage in the neonatal period. Out of the 41 severely involved infants, 4 had died in the neonatal period and 28 were available for follow-up (but 3 were only 18 months old and were excluded). Fifteen of the 25 children were felt to be completely normal. Of the children who had suffered status epilepticus and were available for follow up, 5 were severely retarded, 2 were questionably normal and 1 was definitely normal. The poor prognosis of status epilepticus in the neonatal period is also supported by Rose and Lombroso (1970). He reported on 137 FT newborns followed for an average of 4 years. At the conclusion of the study 51.8% of the patients were normal, 19.7% had died. Infants with seizures and a normal EEG had an 86% chance of normal development whereas the prognosis for those with abnormal EEGs was much less.

Van den Berg and Yershalmy (1969) conducted a most interesting study on febrile and nonfebrile convulsions in 18,500 children. The results indicated that nonfebrile seizures occur in an excessive number of children who were low birthweight infants (1500 to 2500 grams) and are most prevalent among those inappropriately small for gestational age. This is further evidence that the small for date babies (SFD) are potentially at high risk for CNS impairment.

Developmental retardation or deterioration may exist before the onset of the spasm in 68% to 85% of the patients (Rikkeonen, 1984). Associated neurologic abnormalities are often present (Aicardi & Chevrie, 1978). However, identifying mild degrees of cognitive delay retrospectively is difficult and even in patients who

apparently developed normally before the onset spasms, mild neurologic antecedent or subtle motor deficit has been found in upto 20% of cases (Lombroso, 1983a).

J. Cerebral Palsy & Seizures

Seizure disorders are seen more frequently in Cerebral Palsy (CP) than the general population. Some children with CP may be quite distractible but whether such distractibility is due to physiological condition must be thoroughly explored (Shames, Wiigh, & Secord, 1998). 25-35% of children with CP have seizures and those with spastic hemiparesis and quadriparesis have the highest frequency (Thompson & Robert, 1985)**.** The prevalence in patients with CP may range from 55-72% in cases of spastic hemiplegia to about 23% in the choreoathetotic or ataxic forms (Davis, 1977). Incidence of seizures among CP in (Sl No.s 1 - 4) New Jersey (Hopkins, Bice, & Colton, 1954) and Crothers and Paine (1959), Sl No.s 5 - 8 are provided in the Table 4.

Table 4. *Incidence of seizures among cerebral palsy*

Sl No.	Type of CP	Number reported in seizure study	Number with seizure history	% with seizures
1.	Spastic	643	179	28.3
2.	Athetoid	313	65	20.4
3.	Rigidity	172	72	41.9
4.	Ataxia	146	53	36.3
Total		1265	369	29.2
5.	Hemiplegic (Congenital)			55
6.	Hemiplegic (Acquired)			72
7.	Quadriplegic or triplegic			33
8.	Extrapyramidal			23

Cerebral palsy with mental retardation and seizure disorders

Although CP is considered to be non progressive, the presence of seizures in many cases could cause progressive, organic and/or motor complications. About 33% of persons with CP have convulsive seizures of various degrees (Towbin, 1960). Cerebral palsy frequently accompanies seizures and it is often associated with cognitive problems as well. A total 20 to 30% of them do have some degree of mental retardation, usually as a result of the cerebral lesion that is also responsible for their seizures.

Perlstein (1961) reported a series of 173 hemiplegic children. The mean IQ of the 76 children with seizures was 70.2, while the mean IQ of those without seizures was 83. The relative lowering of intellectual function is caused by the same neurologic dysfunction as that which caused the seizures and CP or is related to the treatment of the seizures remains unknown.

9. Management of the epileptic seizures in communication disorders

The management of the epilepsies of childhood has a wider scope involving the prevention of both seizures and the other consequences of paroxysmal neurophysiologic dysfunction. The recurrence risk must be assessed individually for each affected client. The goal of treatment is also to achieve the best possible balance between effectiveness and unpleasant side effects.

Epileptic syndromes with continuous spikes and waves during slow sleep (CSWS) refer to childhood epilepsies characterized by strong activation of seizure-like activity during sleep, occupying at least 85% of non-dreaming sleep time, coupled with cognitive dysfunction. There is strong evidence that cognitive functioning may improve if epileptic activity is reduced using anti-epileptic drugs (Aeby, Verheulpen, Wetzburger, & Bogaert, 2005)

A. Medication

Drug therapy is often a matter of experimentation in finding the appropriate drug for the particular child and his problem. Anticonvulsant medication is usually prescribed for the epileptic disorders. A wide variety of anticonvulsant drugs is now available. Phenytion, Phenobarbital, Carbamazepine, and Primidone are the drugs most commonly employed to treat GTCSs. Valproate is the most effective drug for the treatment of idiopathic forms (Tan & Urich, 1984). It is essential that these be administered only as directed by the physician. Millicahap and Aymat (1967) reported that control of Grand mal seizures is possible in about 60% of the cases. In Petit mal seizures, complete control with drug therapy was successful in 39%. Consistent treatment will suppress most manifestations and therefore relieve the social restrictions associated with this disease. Torres (1969) warns that anti-convulsive drug treatment

should be used only when a diagnosis of seizures has been made. The effects of their use should be checked regularly because hypersensitivity and anemia may develop as side effects. Failure to respond to drug treatment may indicate some organic lesion.

Wilson (1970) recommended that although anticonvulsant medication can usually be discontinued after two or more seizure free years, children with seizures should be urged to continue taking medication into the late teens because of the tendency for seizure control to relapse during adolescence. The long term effects on learning and behavior from medications given to control seizures are unknown. However, since the advent of tranquilizing drugs, aphasic children as well as others with behavioral problems have received much benefit. Increased attention span, more selective reaction to stimuli and diminution of emotional aspects are frequently evident following the inception of drug therapy. Increased learning occurs with the added ability to attend with the use of antiepileptic drugs (Hoare & Russel, 1995).

In many developing countries, people with seizures do not receive appropriate treatment for their condition, a phenomenon called the treatment gap (TG). The TG was mainly attributed to inadequate skilled manpower, cost of treatment, cultural beliefs and unavailability of antiepileptic drugs (AEDs). These factors have been addressed using different intervention strategies such as education and supply of AEDs (Mbuba, Ngugi, Newton, & Carter 2008).

Side effects of medication

Studies (Trimble, 1990a; Trimble & Cull, 1988; McLachlan, 1987; Zaret & Cohen 1986; Thompson & Trimble, 1983) have shown that antiepileptic drugs (AEDs) did have a measurable, generally detrimental effect on test performance. However, differences between the drugs were apparent. Phenytoin and to a lesser extent, the Benzodiazepines and Valproate do affect result on some tests of memory, mental processing and speed. Carbamazepine affected only motor but not mental speed. Phenobarbital and Primidone may also have detrimental effects on cognitive function in addition to their influence on behaviour, although remarkably few studies on these drugs are available (Thompson & Trimble, 1983; Reynoldas, 1975). The decrease in IQ

may not result from treatment but may simply be associated with mental deterioration, especially in more severe cases in which the patients require higher drug dosage. Topiramate may produce language and thought disturbances in some children (Reife, 1996; Aldenkamp, Alpherts, Dekker, & Overweg, 1990) encountered different types or problems in children with seizures. Mental slowness was sometimes marked and attention deficits also played a role. These deficits were most prominent in children taking polytherapy (more than two medicines).

B. Dietary approach

Cerebral metabolism of glucose has been shown to be altered after head injury and increasing cerebral metabolism of alternative substrates (ketones) has been shown to be neuroprotective in several models of traumatic brain injury. The ketogenic diet (KD) is an alternative treatment for medically refractory seizures. The KD is a high-fat, low-carbohydrate diet with proven efficacy in the treatment of intractable epilepsies (Allen, 2008). Neuroprotective strategies such as the KD, if implemented early, might exert an antiepileptogenic effect and could prevent associated learning and memory deficits (Noh, Kim & Choi, 2008). This altered dietary approach may have tremendous therapeutic potential for both the pediatric and adult head-injured populations (Prins, 2008).

C. *Treatment using EEG activity*

Markus et al. (1994) compared children with focal spikes in different areas of the brain. Focal spikes found in the language area in the left (temporal hemisphere) had significantly more language problems compared to the others. Children with focal spikes in the visual cortex (occipital region) had difficulties in complex visual transformation tasks, i.e., construction of triangles and arrangement of photo series. Hence, these finding indicate that focal spikes in the EEG may interfere with complex cognitive functions. Treatment strategy should not only concentrate on the overt seizures but also on the spike activity in the EEG in order to prevent selective deficits.

D. Surgical treatment

In young children with seizures that are not improving with medication, identification of localized areas of abnormally developed cortex (focal cortical dysplasia) on magnetic resonance (MR) brain scans is important. MR brain scans performed early within the first year of life in children with seizures are important to identify areas of abnormally developed cortex. Surgical removal of abnormal cortex may offer improvement of seizure control and subsequent developmental progress. However, appearances of abnormal cortex on MR images may change when the brain matures as children become older. The appearances on later scans can be very subtle, escaping recognition such that conclusions may be misleading with respect to diagnosis and appropriateness of surgical treatment (Eltze, Chong, Bhate, Harding, Neville, & Cross, 2005).

E. Complementary therapy

There is little evidence to show that homoeopathic or herbal remedies help. However, older children who have not responded to treatment may benefit from learning relaxation techniques or from cognitive behavioral therapy (Devinsky, Shachter, & Pacia, 2005).

F. Patient and parent education

Management is concerned not only with the rational aspects of the disease but also has to take into account the misunderstandings, erroneous opinions and prejudice that are deeply rooted even in modern societies (Eadie & Bladin, 2001). The parents of children with seizures must understand the nature of the unpredictable attacks and they should be fully informed about the nature of the disorder, its general mechanisms and its possible causes.

The origin of seizures should be emphasized and the parents should be brought to understand that epileptic seizures are just a symptom of many brain dysfunctions, some of which are quite benign and often transient. A full explanation of the aims and shortcomings of therapy, information on the possibility and the significance of the side

effects of the treatment and an indication of the probable duration of therapy and the problems that may be encountered upon discontinuation are imperative. The parents and/or patients should be encouraged to keep a detailed calendar of seizures, drug modifications, and eventual adverse events, particularly during periods of major changes in treatment.

The evaluation and care of a child with seizures is based not only upon laboratory tests and diagnostic studies but upon a fairly thorough knowledge of that child's personality, school performance, medical history and home environment. Providing education and support for parents is essential. In addition, they should be given counseling and help with educational and school problems and the management of behavioral difficulties. In one study (Mittan, Wasterlain, & Locke, 1982) 53% of patients considered seizures as their primary personal problem, 55% thought seizures could cause brain damage and 66% believed that putting something in their mouth was necessary to prevent "tongue swallowing", one-third of the adult patients felt that sports, strenuous physical activity, dancing, loud music or movies could be dangerous for them. Such misconceptions may be responsible for the social stigma as the low level of education (Rodin, 1989) and employment (Sillanpaa, 1990) and they may have restricted life style (Nakken, 1991). Every effort should be made to help the patients have lives that are as normal as possible and to support their integration as full members of the community. The understanding and cooperation of patients and families is clearly decisive for the success of therapy.

10. Prognosis

The probability of a favorable outcome is increased significantly when all or some of the following factors are present (Seidenberg , Beck, & Geisser, 1986),

1. absence of abnormalities at neurologic examination
2. normal intellectual function
3. absence of a demonstrable brain lesion
4. occurrence of only one type of seizure
5. relatively brief duration of uncontrolled seizures

6. late onset of seizure (i. e, after 3 or 4 years of age)
7. low frequency of seizures
8. absence of certain types of attacks, especially tonic and /or atonic seizures, and of episodes of status epilepticus
9. normality of the EEG at the start of treatment and disappearance of EEG abnormalities with therapy
10. rapid response to therapy
11. regular compliance with antiepileptic treatment

Need for the study

Epilepsy is the second most common chronic neurological condition seen by neurologist. It is estimated that there are 55, 00,000 persons with seizures in India, 20, 00,000 in USA and 3, 00, 00 in UK. 3-5% of the general population have active seizures. Prevalence of active seizures is in the range of 5-10/1000 in most locations, although it might be higher in some isolates. The incidence of seizures in developing countries is nearly double than that found in developed countries. Common causes of seizures include brain damage at birth, congenital or metabolic disorders, drug or alcohol abuse, severe head injury, stroke, brain infection and brain tumours. The higher incidence of seizures in developing countries could be attributed to the higher risk of infections and pre and post-natal complications causing brain damage which may increase the risk of seizures. India is a developing country and epileptic seizure with reference to Indian context needs future investigation.

Seizures occur more frequently in children with communication disorders (CWCDs) than in the general population. Epileptic seizures and few types of communication disorders – it is axiomatic that their occurrence together is usually not coincidental, but that both result from some underlying brain disorder. However, the prevalence of epileptic seizures in CWCDs is not clearly understood. The long term effects of epileptic seizures in CWCDs are unknown. The communication disorders and seizures whether are caused by the same neurologic dysfunction or the presence of recurrent seizures had an effect on communication is not clear.

As Speech language pathologists and Audiologists we come across many children and adults with various communication disorders having a history of seizures or seizure disorders consisting of various types of clinical seizures and epileptic syndromes. The appearance rate or distribution of various types of epilepsies and epileptic syndromes in communication disorders has not been explored much. Therefore, epidemiological findings are important not only for the public health but also for clinical practice. It would also be interesting to know the characteristic features of seizure disorders in different clinical population in order to take suitable preventive measures and to provide rehabilitation. Since there is only limited data about the prevalence of seizures in children with communication disorders in India, the present study was planned.

Objectives of the study: The primary aim of this project is to examine the prevalence of seizures in CWCDs in an Indian context. The other objectives include:

- To examine the prevalence of seizures in different subgroups of children with communication disorders.
- To examine the age of onset (both seizures and communication disorder), causes, type of seizure, frequency of occurrence of seizures and associated problems.
- To examine the co-morbidity of seizures and the nature of communication disorder.
- To classify the seizure disorders causing communication disorders if possible.
- To develop a high-risk register for children with seizures

METHOD

Epilepsy is one of the most common neurological disorders occurring in children with developmental disabilities compared to general population. The distribution and nature of epileptic seizures in different subgroups of children with communication disorders (CWCDs) has not been studied in detail. It would be interesting to know the characteristic features of seizure disorders in different clinical population. Since there is only limited data about the prevalence of seizures in CWCDs in India future epidemiological research needs to be addressed. The current project was therefore proposed with this need in mind and with the primary objective to determine the prevalence of epilepsy in children with communication disorders along with the objectives. The Objectives of the study were to determine the prevalence of seizures in different subgroups of children with communication disorders, to determine the age of onset, causes, type of seizure and frequency of occurrence of seizures, to identify the pattern of inheritance if any, to classify the seizure disorders causing communication disorders, to determine the co-morbidity of seizures and the nature of communication disorder and to develop a high-risk register for CWCDs with history of seizures.

Participants: Children below the age of 12 years, who visited AIISH with the complaint of speech, language and hearing problems over a period of two years (Jan 2007- Dec 2008) were considered in the present study. All the participants had history of communication disorder.

Procedure: The present study was conducted in three phases.

Phase 1: Case files of children below the age of 12 years, who visited AIISH with the complaint of speech, language and hearing problems over a period of two years (Jan 2007- Dec 2008) was reviewed for the presence or absence of seizures. A total number of 6,052 children had registered during the period of two years. 6,052 case files were reviewed for the presence or absence of seizures. Out of 6,052 children, 718 of them were identified as having positive history of seizures.

Phase 2: A questionnaire was developed to get further information on the variables related to seizures (Appendix 1). A total of 29 questions were included in the questionnaire. 16 questions were related to characteristic features, 2 each on causative factors & assessment and 9 questions on treatment factors. The variables included age of onset, gender, associated problems, family history of seizures, causes, type of seizure, frequency of occurrence, type and duration of medication, whether the seizure is under control with medications and overall prognosis.

Phase 3: 718 children with positive history of seizures were selected for further analysis. The developed questionnaire was administered to 135 children who were attending therapy and diagnostic services at AIISH over a period of two years (Jan 2007- Dec 2008). The questionnaires were also despatched to 595 clients with positive history of seizures identified in Phase I, with self addressed envelope to receive the filled up questionnaire. The response was received by post from 181 clients out of 595. The percentage of response for questionnaire was about 33% which was fairly good. The data was compiled from the filled up questionnaires. During analysis it was noted that the EEG reports and the type of seizure was not mentioned in most of the questionnaires. Hence, the neurologist, one of the co- investigators decided on the type based on signs and symptoms noted in the questionnaire.

The data was analyzed using appropriate statistical procedures for the above mentioned parameters to study the research objectives.

During all the phases of research ethical considerations/practices was rigorously respected and scrupulously followed. Information gathered from all the files was kept confidential.

RESULTS AND DISCUSSION

The purpose of the project was to determine the prevalence and nature of seizures in CWCDs in the Indian context. The results obtained in the present study have been discussed in two parts, with reference to each of the objectives of the study.

I. Prevalence of seizures in CWCDs

A. Prevalence of seizures in different subgroups of CWCDs

Children below the age of 12 years who visited AIISH over a period of two years (Jan 2007- Dec 2008) with the compliant of speech, language and hearing problems were found to be 6,052.

Table 5. *Prevalence of seizures in different subgroups of CWCDs*

Disorders	Clients registered at AIISH	Clients with + ve h/o seizures	Percentage
BI	34	25	73.53%
Speech Regression	30	20	66.6%
HLCPMR	29	17	58.62%
MRCP	332	170	51.20%
DD	78	24	30.76%
MR	1276	271	21.23%
PDD	162	32	19.75%
Apraxia	11	2	18.18%
MRHL	133	23	17.29%
ADHD	69	8	11.69%
HLCP	65	9	13.48%
CP	210	28	13.33%
DSL	867	31	3.53%
HL	1595	42	2.63%
LD	195	5	2.56%
CLP	104	2	1.92%
Dys	134	2	1.49%
MA	302	3	0.99%
Sttg	426	4	0.09%
Total	6052	718	11.86%

(Note: Hearing loss with Cerebral palsy and Mental Retardation (HLCPMR), Mental Retardation and Cerebral palsy (MRCP), Pervasive Developmental Disorder (PDD), Mental Retardation and Hearing Loss (MRHL), Attention Deficit Hyperactivity disorder (ADHD), Hearing loss and Cerebral palsy (HLCP), Border line Intelligence (BI), Development Delay (DD), Mental retardation (MR), Cerebral palsy (CP), Delay in speech and language (DSL), Hearing loss (HL), Learning Disability (LD), Cleft lip and Palate (CLP), Dysarthria (Dys), Misarticulation (MA), Stuttering (Sttg).

Out of 6,052 children with communication disorders, 718 children had positive history of seizures. The total percentage of communication disorder having positive history of seizures was 11.86%. The prevalence of epileptic seizures in different subgroups of CWCDs is depicted in the following table 5 and figures 1 & 2. As seen in table 5 higher prevalence rate (73.53%) of seizures was noticed in clients with borderline intelligence and lowest prevalence rate was noticed in clients with stuttering (0.09%). 20 children (66.6%) had speech regression consequent to epileptic sequelae. 17 children (58.62%) with multiple disability i.e., hearing loss with cerebral palsy and mental retardation had positive history of seizures. In general, the percentage of seizures in children with borderline intelligence was 73.53%, followed by speech regression (66.6%), hearing loss with cerebral palsy and mental retardation (58.62%), mental retardation with cerebral palsy (51.20%) and least in stuttering (0.09%). Figures 1 & 2 represents the distribution of epileptic seizures in different subgroups of children with communication disorders.

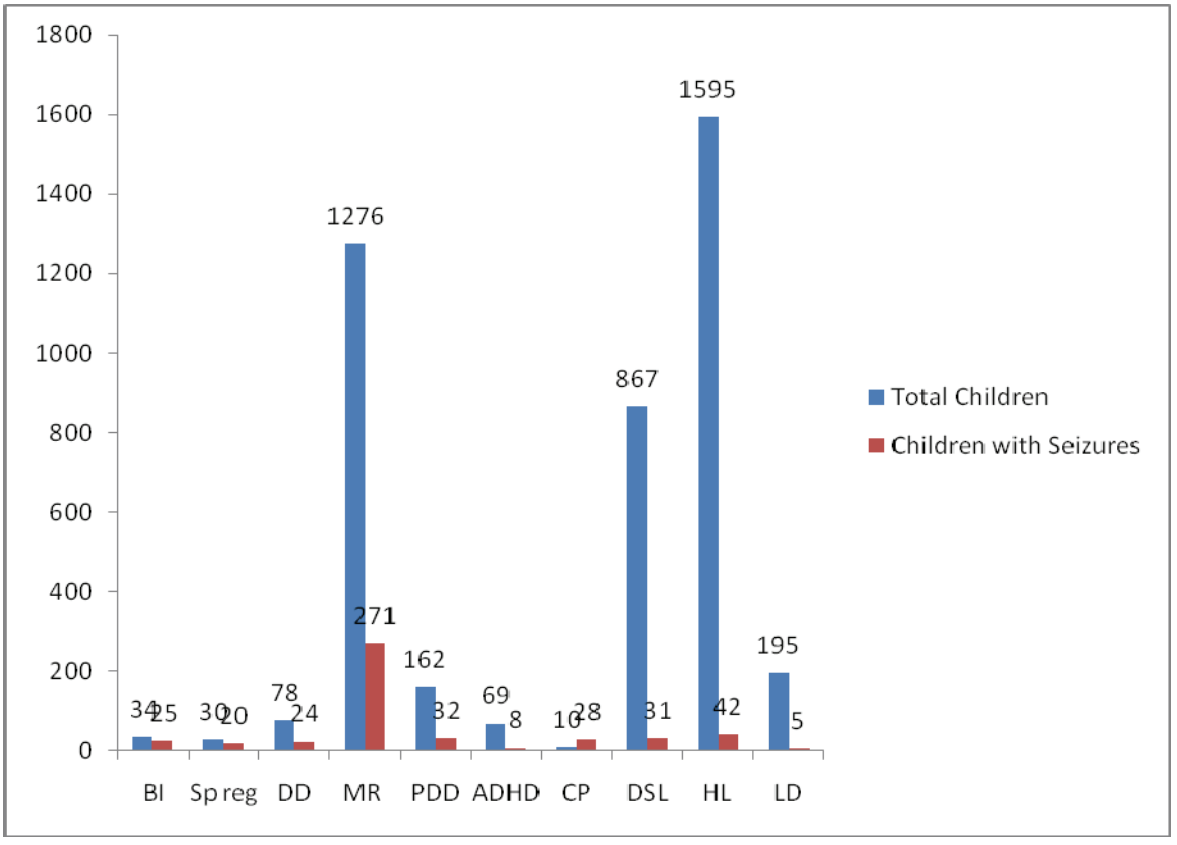

Figure 1. *Distribution of seizures in language subgroups of CWCDs*

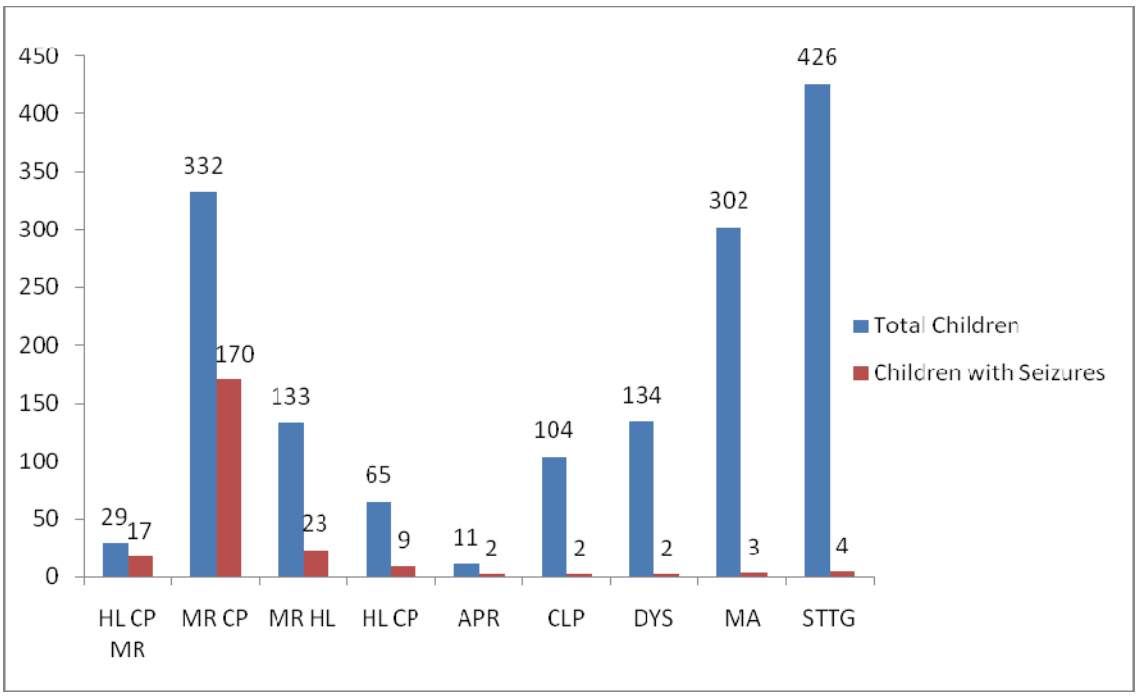

Figure 2. *Distribution of seizures in speech and multiple subgroups of CWCDs*

In the present study it was noted that the percentage of epilepsies ranged from as high as 73.53% in children with mental retardation to as low as 0.09% in stuttering group. Findings suggest that the prevalence rate of seizures was more in children with brain damage as in borderline intelligence, cerebral palsy, mental retardation, developmental delay and pervasive developmental disorders.

1) Seizures in children with mental retardation

In the present study, the maximum percentage of seizures ranged from 73.56% (BI) to 21.23% (MR) in children with mental retardation indicating brain damage as the major cause for seizures. Children with developmental disabilities are at higher risk for seizures than the general population (Walsh, 1999). The frequency and the severity of the epileptic syndrome are related more to the primary cause of mental retardation than to the severity of mental retardation. However, there is a direct relationship between severity of intellectual disability, frequency and severity of chronic epileptic seizures. In United States, the prevalence of mental retardation is approximately 0.3-0.8%, but 20-30% of children with mental retardation have seizures. Approximately 35-40% of children with seizures also have mental retardation (Alvarez, 1998). Rodin (1968) found

a low intelligence in children with brain lesions whereas, in “pure” seizures, the IQ was within normal limits, although the curve of IQ was shifted to the left. In the whole group, a mild irregular downward trend was present and significant loss was seen in only some cases. Sillanpaa (2000) found some deterioration in IQ among 11% of the children. The results of the present study indicate a much higher percentage of seizures in children with mental retardation compared to other studies.

2) Seizures in children with speech regression

History of seizures in speech regression indicated that around 66.6% of children had language deficits due to onset of seizures in the present study. This indicates that there might be greater amount of detrimental effects in children due to presence of seizures. Acquired epileptic aphasia (AEA) typically develops in healthy children who acutely or progressively lose receptive and expressive language ability coincident with the appearance of paroxysmal EEG changes. Population-based epidemiologic data related to AEA are limited. Many reports describe no correlation between EEG abnormality and language dysfunction (Menezes, 2010).

Those with neurological involvement and hence at risk for developing seizures constitute the tip of the iceberg (Singh & Prabhakar, 2008). Frequent causes of seizures in young children are central nervous system (CNS) malformation, CNS injury from infection or accident and CNS malfunction caused by a metabolic error (Riikkeonen, 2001). Seizures and speech regression may occur together and both may result from same underlying brain disorder. However, the causal association between seizures and language impairment is poorly documented due to constraints in epidemiological methods. The present study is a retrospective analysis and hence most of the information was considered from the case files was considered. Majority of the time information collected depend on the type of understanding the parents have regarding the condition. The first epileptic seizure that involves abnormal body movements for a short duration may have greater significance for parents. Hence, there is higher probability of parents to report as seizures being the causative factor for communication disorder.

3) Seizures in children with Pervasive developmental disorders (PDD)

In the present study the frequency of overt seizures among patients with Pervasive developmental disorders **(**PDD) was 19.75%. Seizures are common among male patients, and the seizures start in the first year of life in more than 80% of the patients. Tuchman and Rapin (2002) found that seizures was present in 14% of autistic children after they excluded patients with Rett's syndrome. Al-Salehi, Al-Hifthy, and Ghaziuddin (2009) examined a sample of 49 children (37 males and 12 females) diagnosed with an autistic spectrum disorder at a tertiary referral center in Saudi Arabia. They found eleven clients with a history of seizure disorder and one had a chromosome abnormality. Evald, Petur, and Rafnsson (2007) described autistic spectrum disorders (ASDs) in a cohort of children with history of unprovoked seizures other than infantile spasms in the first year of life. Eighty-four children (82.4%), 28 boys and 56 girls, participated in the study and 36.9% (31/84) were investigated for possible ASD. Twenty-four (28.6%) had at least one neuro-developmental disorder, 14.3% had mental retardation (MR) and six (7.1%) were diagnosed with ASD, all of whom also had MR and three of whom had congenital brain abnormalities. The results suggested that the estimated prevalence of ASD is higher in children with history of seizure in the first year of life than it is in the general population. The results of the present study thus indicate the presence of seizures in PDD as suggested in literature.

4) Seizures in children with multiple disability

The present study found children with multiple disabilities i.e., HLMRCP, MRCP, MRHL and HLCP had prevalence rate of seizures of 58.62%, 51.20%, 17.29% and 13.84% respectively. This indicates that, probably the children with coexistence of other disabilities were more prone to the occurrence of epileptic seizures. However, it is not so common with hearing loss compared to cerebral palsy and mental retardation. This finding is in agreement with findings in many other studies. An association of mental retardation with cerebral palsy is also common in those with seizures. Earlier studies have investigated the prevalence rate of seizures in children with cerebral palsy and mental retardation. In a group of non institutionalized individuals, the prevalence of

seizures was 20%, seizures and mental retardation was 43% and seizures associated with CP were 33% (Hauser et al., 1991).

5) Seizures in children with Cerebral Palsy (CP)

The present study found that children with cerebral palsy (CP) had 13.33% and Hearing loss with cerebral palsy (HLCP) had 13.84% prevalence rate of seizures. Thompson and Robert (1985) found 25-35% of children with cerebral palsy having seizures and those with spastic hemiparesis and quadriparesis had the highest frequency which supports the results of the present findings**.** The dual handicap of seizures and cerebral palsy occurs in 1 in 1,000 live births. When the two disorders coexist, it is reasonable to assume that the etiology also is related—that is, that the same brain injury responsible for causing cerebral palsy also has caused the seizures. Davis (1977) reported seizures rates as high as 55-72% in cases of spastic hemiplegia to about 23% in the choreoathetotic or ataxic forms. The population-based study in Sweden by Hagberg, Hagberg, Olow, and von Wendt (1996) found that 28% of people with CP also had seizures. An update of this study published in 2003 reported that 38% (55/146) developed seizures by 6 to 14 years of age (Carlsson, Hagberg, & Olsson, 2003). Similarly, the Danish Cerebral Palsy Register reported that 27.1% of patients born with cerebral palsy between 1979 and 1986 also had seizures (Topp, Uldall, Longhoff, & Roos, 1997). Data from Western Australia (Stanley, Alberman, & Blair, 2000) from 1975 through 1994 record the incidence rate of seizures to be 37% (618 of 1,664). The rate of combined seizures and CP of 0.8 per 1,000 live births has remained constant over 25 years. With improvements in neonatal care during the last 20 years, the number of surviving premature and low-birth-weight babies has increased, with a concomitant increase in the rate of CP (Camfield, Camfield & Watson, 2001).

The study by Kudrjveev, Schoenberg, Kurland, and Groover (1985) in Minnesota between 1950 and 1976, found 52% of those with severe cerebral palsy developed seizures. Conversely, seizures was present in 23% of those with mild to moderate CP. Watson and Stanley (as cited in Camfield et al., 2001) found that 65% of children with severe cerebral palsy born between 1975 and 1994 developed seizures, as

compared with 24% of children with minimal, mild or moderate cases of CP. Hence it is concluded that children severely affected by cerebral palsy seem to be more likely to develop seizures. However, the present study did not attempt to correlate the prevalence with severity.

6) Seizures in children with other subtypes of communication disorders

The lower prevalence rate of seizures was found in clients with speech and language delay (3.53%), hearing loss (2.63%), learning disability (2.56%), cleft lip and palate (1.92%), dysarthria (1.49%), misarticulation (0.99%) and stuttering (0.09%). Findings are probably because these communication disorders are not caused due to serious brain damage.

Learning disabilities can be caused by damage to the brain. This damaged part of the brain can then become irritable and provide a focus for epileptic seizures. The resulting seizures, however, may not appear until many years after the damage occurred. Usually seizures do not cause learning disabilities. However, having many and/or severe seizures over a length of time can cause damage to the brain. This in turn can lead to learning disabilities. Learning difficulties of variable severity are present in 5% to 50 % of children with seizures (Sawheney, Suresh, Dhand, & Chopra, 1988).

Oromotor Apraxia and speech problems may be congenital or they may develop or worsen with episodes of sustained spike and wave discharges during sleep (Deonna, Roulet, Fontan, & Marcoz, 1993).

Harrison (1947) and Van Riper (1971) found greater prevalence of stuttering among patients with seizures. These findings suggest a link between stuttering and seizures but do not enable one to specify the nature of the link. There is an apparent relationship between stammering and seizures in the abnormality of the brain-wave tracings. Interestingly enough the tracings suggest that persons with stuttering may be functioning in a state of reduced consciousness (Lebrun & Fabbro, 2002).

B. Prevalence of seizures across gender in CWCDs

The percentage of seizures across gender was analyzed in children with communication disorders. Out of 730 children with positive history of seizures, 541 (74%) were boys and 189 (26%) were girls. The prevalence of epileptic seizures across gender in different subgroups of communication disorders are depicted in the following table 6. In normal children, the gender ratio was 2.60:1.94 per thousand population in one of the earlier study (Das & Sanyal, 1996). Literature revealed a nearing double the time of occurrence in boys compared to girls. The present study is in agreement with literature that the occurrence was more in boys but with the increased ratio of 74:26.

In general, the percentage of seizures among boys was maximum of 206 (38.07%) in children with mental retardation and minimum of 2 (0.36%) in cleft lip & palate and Apraxia. Similarly, the percentage of seizures among girls was maximum of 65 (34.39%) in children with mental retardation and minimum of 1 (0.52%) in children with attention deficit hyperactivity disorder. The present study revealed no history of seizures among girls in subgroup of communication disorders like learning disability, stuttering, misarticulation, dysarthria, cleft lip & palate, Apraxia and in boys also it was <1%. However, as it is a retrospective analysis generalization of these results should be considered with caution.

The prevalence of seizures in Rochester, Minnesota was higher for males than females (Hauser et al., 1991). Among the Indian studies, Das and Sanyal (1996) found crude prevalence rate for seizures per thousand population was 3.06 in rural areas of Malda district, West Bengal. The age specific prevalence rate for males aged above 14 years was 3.75 and for 14 years and below was 2.60, corresponding figures for females being 3.53 and 1.94, respectively. Kaiser, Shong, and Kulkarni, (2009) made a prospective and retrospective observational, cross sectional study involving data collection on a specially designed proforma with respect to seizure diagnosis, duration, co-existing medical conditions, precipitating factors if any, along with details of drug treatment. Data from a total of 336 patients with the diagnosis of seizures was collected and recorded over a period of one year of the study. Analysis of the data for genderwise distribution showed a distinct predominance of males (62%) over females (38%). The

results of the present study suggested greater seizure occurrence in boys compared to girls as indicated in earlier studies, however it should be addressed with caution.

Table 6. *Prevalence of seizures across gender in CWCDs*

Disorders	Boys with + ve h/o seizures		Girls with + ve h/o seizures	
	No. of children	Percentage	No. of children	Percentage
BI	22	4.06	3	1.58
Speech regression	15	2.77	5	2.64
HLCPMR	11	2.03	6	3.17
MRCP	122	22.5	48	25.39
DD	15	2.77	9	4.76
MR	206	38.07	65	34.39
PDD	22	4.06	10	5.29
Apraxia	2	0.36	0	0
MRHL	15	2.77	8	4.23
ADHD	7	0.29	1	0.52
HLCP	5	0.92	4	2.11
CP	17	3.14	11	5.82
DSL	24	4.43	7	3.70
HL	30	5.54	12	6.34
LD	5	0.92	0	0
CLP	2	0.36	0	0
Dys	2	0.36	0	0
MA	3	0.55	0	0
Sttg	4	0.73	0	0
Total	529	73.67	189	26.32

(Note: Hearing loss with Cerebral palsy and Mental Retardation (HLCPMR), Mental Retardation and Cerebral palsy (MRCP), Pervasive Developmental Disorder (PDD), Mental Retardation and Hearing Loss (MRHL), Attention Deficit Hyperactivity disorder (ADHD), Hearing loss and Cerebral palsy (HLCP), Border line Intelligence (BI), Development Delay (DD), Mental retardation (MR), Cerebral palsy (CP), Delay in speech and language (DSL), Hearing loss (HL), Learning Disability (LD), Cleft lip and Palate (CLP), Dysarthria (Dys), Misarticulation (MA), Stuttering (Sttg).

The most profound difference between girls and boys is not in any brain structure per se, but rather in the sequence of development of the various brain regions. The different regions of the brain develop in a different sequence in girls compared with boys. This is the key insight from the past five years of neuroscience research in brain development. A most comprehensive study demonstrated that there is no overlap in the trajectories of brain development in girls and boys (Lenroot, Gogtay, & Greenstein, 2007).

II. To determine the nature of seizures in CWCDs

The second objective of the present study was to focus on the nature of seizures in children with communication disorders. It was a retrospective study hence included referring to case files for the presence of seizures. Detailed information about the nature of seizures could not be obtained only by referring to the case files. Hence a questionnaire was developed to get further information for the variables related to seizures. The nature of seizures was determined by analyzing the questionnaire data of 316 children (135 administered directly and 181 received by post) with communication disorders. The characteristic features of seizures in CWCDs have been discussed under different headings.

1. Age of onset of seizures

Seizures can strike anyone at any age. 50% of all cases develop before 10 years of age. The Epilepsy foundation of America (2010) publication showed some age groups to be more susceptible than others. First seizures occurring at different age groups are as follows. Children at the age of 0-9years had 47% of occurrence followed by 10-19years (30%), 20-29 years (13%), 30-39 years (6%) and 40+years (4%). Most people who develop seizures during their earlier years tend to experience a reduction in the intensity and frequency of their seizures as they grow older. In many cases the seizures will disappear completely in the general population. Hauser et al. (1991) commented that the highest incidence occurs during the first few years of life. However, the earlier studies considered the general population as their subjects. Though the present study considered CWCDs majority of them had their first attack during the beginning years which is in consensus with studies related to general population.

Although communication disorders can occur at any age, seizures are far more common in children than adults. In the present study the age of onset of first attack of seizures was obtained from 304 children. Out of 304, maximum no. of children 133 (43.8%) had their first attack before 6 months. 51 (16.8%) and 57 (18.8%) children had their first attack during age 6-12 months and 1.1- 2 years respectively. The first attack

of seizures significantly reduced after 2 years of age. A comparison of first attack of seizures was determined across subgroups of communication disorders and is depicted in table 7. Multiple disorders (MD) included Hearing loss with Cerebral palsy and Mental Retardation (HLCPMR), Mental Retardation and Cerebral palsy (MRCP), Mental Retardation and Hearing Loss (MRHL), Hearing loss and Cerebral palsy (HLCP). It can be noted from the below table that nearing 60% of the children had their first attack of seizures by one year of age.

The age at the first epileptic seizure relates to the cause of mental retardation. One study (Alvarez, Carvajal, Begaud, Moride, Vega, & Arias, 1998) which included 98 children with mental retardation aged 6-13 years, found that the average age at the time of the first seizure was 1.3 years for the whole group, 0.8 years in children with severe mental retardation, and 3.1 years in those with mild mental retardation. Another study including adults with mental retardation found that, of 63 individuals, 41% had a first seizure before the second year of life and 30% had a first seizure between ages 2 and 20 years.

Table 7. *First attack of seizures across subgroups of CWCDs*

Disorders	Total children	< 6 months (%)	6-12 months (%)	1.1-2 years (%)	2.1 - 4 years (%)	> 4 years (%)
BI	11	5 (45.5)	2 (18.2)	3 (27.3)	-	1 (100)
DD	8	7 (87.5)	-	1 (12.5)	-	-
Speech regression	1	-	-			1 (100)
MR	116	38 (32.8)	20 (17.2)	22 (20)	20 (17.2)	16 (13.8)
PDD	4	2 (50)	-	2 (50)	-	-
ADHD	3	2 (66.6)	1 (33.3)	-	-	-
CP	25	14 (56)	4 (16.0)	6 (24)	1(4)	-
DSL	40	13 (45)	6 (15.0)	8 (20)	5 (13)	3 (8)
HL	28	11 (39.3)	4 (14.3)	8 (28.6)	4 (14.3)	1 (3.6)
LD	2	2 (100)	-	-	-	-
MA	1	-	-	-	1 (100)	-
Multiple	65	34 (55.7)	13 (21.3)	7(11)	5 (6)	6 (9)
Total	304	133 (43.8)	51 (16.8)	57 (18.8)	36 (11.8)	27 (8.9)

(Note: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Learning Disability (LD), Hearing loss (HL), Mental retardation (MR), Misarticulation (MA), Mutiple- HLCP, MRHL, MRCP, HLCPMR).

A history of neonatal seizures is frequently found in children with the dual pathology of both seizures and cerebral palsy. Kwong, Wong, and Kwan (1998) found this association in 19% whereas Carlsson et al. (2003) found a slight increase of 24%. Most of the children's seizures were thought to be of prenatal origin. Age of onset of the seizures had a close relationship with the type of cerebral palsy. Kwong et al. (1998) found age of onset of seizures to be approximately 6 months in quadriplegics, 18 months in hemiplegic and 24 months in diplegics. Similar results were described from a Greek referral center (Zafeiriou, Kontopoulos, & Tsikoulas, 1999) who followed up 178 children with seizures and cerebral palsy. The prevalence of seizures in this clinic was 36%, and the onset of seizures occurred in the first year of life in 73% of the children. Carlsson, Hagberg, and Olsson (2003) in Sweden found that 91% had developed seizures by 6 years of age. It appears that earlier age of onset of seizures is correlated with an increased severity of cerebral palsy. The present study is in consensus with Zafeiriou et al. (1999) study with the prevalence rate of 60.6% at the age of onset before one year. Other studies reported lower prevalence rate of 19% and 24%, the difference could be due to methodological issues.

Holmes, Harvey, Coull, Huntington, Shahram, Khoshbin, Hayes, and Ryan (2001) reported that seizures during development can be harmful are myriad. The developing brain is highly plastic and seizures during early development could have pronounced effects on brain development. An early age at onset was significantly associated with cognitive difficulties. The combination of a severely chaotic EEG and spasms occurring at a critical developmental stage may be responsible. Children considered in the present study had communication disorders, majority had their first attack within one year of age and as reported in literature, seizures during early development might have led to pronounced effects on brain development.

One child with Landau Kleffner syndrome or acquired epileptic aphasia (AEA) in the present study had an epileptic attack after 4 years. The findings of the study are in consensus with Bishop (1985) who reported that Aphasia in AEA usually appears at 4-7 years of age. However, symptom onset has also been described in patients as young as 18 months and in those as old as 13 years. Some authors have included cases of

developmental dysphasia associated with seizure-related fluctuations in speech performance, whereas others have not.

In children, the factors of age, growth and development are of primary importance in determining not only whether seizures is developing but also what the clinical and electrical manifestations of the seizures are and the type of seizure disorders that will be encountered. Age of seizures onset was found to be positively correlated with achievement ($r=42$, $p < .05$). Children who were older when they had their first seizure performed better on the achievement domain than those who were younger at seizure onset. Individuals with early seizure onset are more likely to demonstrate atypical (right or bilateral) language representation than those with later injury. However, it is unclear whether pathology related to early seizures plays a role in determining language dominance (O' Donohoe, 1985).

Age is generally recognized as an important determinant of the prognosis for the disorder (Sillanpaa, 2000). Even within the same seizures syndrome (e. g., infantile spasms), a young age at onset compared with the average for the syndrome is an unfavorable predictor of prognosis. In some syndromes (e.g., absence seizures), cases of early onset (before 3 years of age) or of late onset (after 8 to 9 years of age) have a substantially poorer prognosis than do those with an onset between 4 and 8 years of age. Age has a strong influence on the outcome of status epilepticus. Sequelae are more common in those younger than 2 to 3 years. Supporting the literature, even in the present study most of the CWCDs had first attack of seizures as early as birth to 12 months. It can be concluded from the above discussion that earlier the occurrence of seizures more detriment would be the condition in children with communication disorders. Hence, it is evident that the age of onset of seizures should be considered during assessment of children with communication disorders.

2. Frequency of occurrence of seizures

The frequency of occurrence of seizure since the first attack was only once in 59 (22%) children, 2 to 3 times in 120 children (45%), 1 to 3 times per week in 25 children (9%), > 3 times per week in 18 children (7%), and 1 to 2 times per month in 47 children

(17%) with communication disorders. In the present study the frequency of occurrence varied from only once to greater than 3 times per week. The occurrence of only once and about 2 to 3 times only is not a serious condition compared to other conditions like 1 to 3 times per week and 1 to 2 times per month.

The results indicate that the majority of the children with the frequency of occurrence of seizure ranged about 2 to 3 times in childhood. Table 8 indicates the frequency of occurrence of seizure in children with communication disorders. 50% of children with LD, DD and ADHD had only one attack indicative of less risk factor due to seizures. About 100% of children with speech regression, 70% with cerebral palsy and 57% with mental retardation had only 2-3 times of occurrence which is also indicative of reduced risk factor due to seizures. Maximum no. of times (1-3 and > 3 times per week) would lead to greater risk factors and was noticed in 45% of children with BI and 50% of children with LD.

Researchers followed up persons in general population for an average of 8 years. They found that only 33% of subjects had a second seizure within 4 years after an initial seizure. Those who did not have a second seizure within that time remained seizure-free for the rest of the study. For subjects who did have a second seizure, the risk of a third seizure was about 73% on average by the end of 4 years (Hauser, Rich, Lee, Annegers, & Anderson, 1998). However, the present study is not in agreement with earlier mentioned study as there was greater frequency of occurrence about 90 (61.1%) children. This finding is attributed to the subjects considered in the study. The current study considered CWCDs whereas the earlier research focussed only on general population.

Table 8. *Frequency of occurrence of seizures*

Disorders	Total children	Only once (%)	2 to 3 times only (%)	1-3 times/ week (%)	>3 times/ week (%)	1- 2 times/ month (%)
BI	11	3 (27.3)	1(9.1)	2 (18.2)	3 (27.3)	2 (18.2)
DD	6	3 (50)	2 (33.3)	-	-	1 (16.7)
Speech regression	1	-	1(100)	-	-	-
MR	100	11(11)	57 (57)	12 (12)	6 (6)	14 (14)
PDD	3	-	1(33.3)	-	-	2 (66.7)
ADHD	4	2 (50)	2 (50)	-	-	-
CP	23	3 (13)	16 (69.6)	-	1 (4.3)	3 (13)
DSL	35	10 (28.6)	12 (34.3)	2 (5.7)	3 (8.6)	8 (22.9)
HL	23	6 (26.1)	9 (39.1)	2 (5.7)	1 (4.3)	5 (21.7)
LD	2	1(50)	-	-	1 (50)	-
MA	1	-	-	-	-	-
Multiple	60	20 (3)	19 (15)	7 (11.6)	3(5)	11 (18.3)
Total	269	59 (22)	120 (45)	25 (9)	18 (7)	47(17)

(Note: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Learning Disability (LD), Hearing loss (HL), Mental retardation (MR), Misarticulation (MA), Mutiple- HLCP, MRHL, MRCP, HLCPMR).

Fastenau, Johnson, Perkins, Byars, DeGrauw, Austin and Dunn (2009) indicated that in a intellectually normal cohort, 27% with just one seizure and up to 40% of those with risk factors exhibited neuropsychological deficits at or near onset. Risk factors associated with neuropsychological deficits included multiple seizures (i.e., second unprovoked seizure; odds ratio [OR] = 1.96), use of antiepileptic drugs (OR = 2.27), symptomatic/cryptogenic etiology (OR = 2.15), and epileptiform activity on the initial EEG (OR = 1.90); a child with all 4 risks was 3.00 times more likely than healthy siblings to experience neuropsychological deficits. It can be concluded from Fastenau et

al.'s study that a normal child with seizures can exhibit few neuropsychological deficits. It further suggests that if the impact of seizures is present in a normal child, serious effects can also be noticed in a subgroup of CWCDs.

Genton and Dravet (1988) noted that the frequency of seizures is a predictor of behavioral problems. Substantial evidence now supports the view that, in some situations, frequent seizures or epileptiform discharges result in substantial cognitive decline in children. The intractable infantile spasms may lead to changes in connectivity that permit the seizure type to continue. The recurrent seizures may be responsible to alter the developing brain. Liu, Mikati, and Holmes (1995) reported that recurrent seizures alter the mossy fiber plasticity and also result in alterations of neuronal pathways activated during seizures. Holmes et al. (1998) stated that learning proved to be directly related to the extent of mossy fiber projections to the pyramidal layers. Recurrent neonatal seizures are associated with impairment in cognition and behavior. The percentage of seizures is less at the higher than at the lower levels of intelligence as shown particularly in Hansen's (1960) large series from Denmark that covered all cerebral palsies. The risk of recurrence following a single seizure varied from 16-81% as summarized from various studies conducted in India by Sridharan (2002). The risk is least in persons with acute symptomatic seizures and maximum in those with pre-existing cerebral pathology. The risk factors for seizure recurrence include antecedent brain insult (most important), age <16 or > 65 years, seizure type (partial) abnormal EEG (spike wave pattern), family history of seizures, prior acute seizures, including febrile seizures, occurrence of status epilepticus or Todd's paralysis. McDonald et al. (2000) also found that the single most important prognostic factor for remission was the number of seizures in the initial period of the disease. It can be concluded from the earlier findings that more the frequency of occurrence greater is its ill effects on the developing child. The present study report higher frequency of seizure occurrence in children with communication disorders compared to other studies. This finding is a great caution to the clinician dealing with children with communication disorders.

The present study showed the maximum frequency of occurrence of seizure as 1-3 times and > 3 times per week in 43 (16%) children and 1 to 2 times per month in 47

(17%) children. These results are in agreement with Sridharan's study. Majority of children with mental retardation and cerebral palsy had increased frequency of occurrence. Supporting the literature even in the present study few children had recurrent seizures and this may be attributed to the pre-existing cerebral pathology in children. However, considering the risk factors identified with increased frequency of occurrence of seizures it warrants swift referral for neuropsychological evaluation and management in the children with communication disorders.

3. Family history of seizures

Positive family history of seizures was confirmed in 55 (19.2%) children out of 286 children with communication disorders. The relationship with the client was a close relative in 33 clients and far relative in 21 cases. Table 9 provides details on family history of seizures across communication disorders. Positive family history ranged from 12.5% to 50%. Maximum (50%) percentage was reported in LD and borderline intelligence (30%) and a minimum of 12.5% was found in child with DD.

All children with Speech regression, PDD, ADHD and MA had negative family history of seizures. Maximum no. of children with DD (87.5%), MD (88%) and DSL (87%) also had no family history.

Siddiqui (2002) found positive family history of febrile convulsions in 30% children. A positive family history of febrile seizures points to the importance of genetic factors and common environmental exposures. Farwel et al. (1994) also reported positive family history in 29 percent of the cases. Literature with reference to the family history reported siblings of persons with seizures to have 2.5 times increased risk for seizures. There is higher concordance for seizures among monozygotic compared to dizygotic twin pairs (Sridharan, 2002). Menezes (2010) reported 12% of patients with acquired epileptic aphasia (AEA) to have a family history of seizures. The present study is in consensus with earlier studies as the findings reveal positive family history of seizures. It was confirmed in 55 (19.2%) children which is slightly lower than that of the results of Siddiqui (2002) and Farwel et al. (1994). This difference could be due to considering febrile seizures too among the type of seizure in earlier studies.

Table 9. *Family history of seizures*

Disorders	Family history of seizures		
	Total children	Negative (%)	Positive (%)
BI	10	7 (70)	3 (30)
DD	8	7 (87.5)	1 (12.5)
Speech regression	1	1 (100)	-
MR	104	80 (76.9)	24 (23.1)
PDD	4	4 (100)	-
ADHD	4	4 (100)	-
CP	24	19 (79.2)	5 (20.8)
DSL	39	34 (87.2)	5 (12.8)
HL	26	20 (76.9)	6 (23.1)
LD	2	1 (50)	1 (50)
MA	1	1 (100)	-
Multiple	81	71 (88)	10 (12)
Total	286	231 (80.8)	55 (19.2)

(Note: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Learning Disability (LD), Hearing loss (HL), Mental retardation (MR), Misarticulation (MA), Mutiple- HLCP, MRHL, MRCP, HLCPMR).

A study (Science daily, 2008) found genetic abnormalities of the gene encoding alpha 1 subunit of the sodium channel (SCN1A) in more than 60% of patients with severe myoclonic seizures in infancy or its borderline phenotypes. The frequency of seizures is elevated in first-degree relatives of children with cerebral palsy and mental handicap, implying that genetic factors play an important part in both of these chronic disabilities. Curatolo, Seris, Verdecchia & Bombardieri (2001) reported that seizures were 17 times more frequent in those children than in normal controls. Asku (1990) described a cohort of children with cerebral palsy and seizures in whom 16% of first-degree relatives also had seizures, as compared to 8% of first-degree relatives of the normal controls. The results of the present study are in agreement with earlier studies i.e even in this study 33 clients had close relatives with positive history of seizures.

Majority of children (231, 80.8%) had negative family history in the present study. This may be attributed to other causative factors playing a major role in children with communication disorders. Seizures are a disorder with many possible causes. Anything that disturbs the normal pattern of neuron activity from illness to brain

damage to abnormal brain development can lead to seizures (Mulas, Hernández, Mattos, Abad-Mas, & Etchepareborda, 2006). The developing brain is susceptible to many kinds of injury. Injury to the brain may certainly cause seizures. This includes deprivation of oxygen at birth, trauma to the head at any time of life, and stroke (injury to part of the brain caused by blockage or haemorrhage of one of its blood vessels). Maternal infections, poor nutrition, and oxygen deficiencies are just some of the conditions that may take a toll on the brain of a developing baby. These conditions may lead to cerebral palsy, which often is associated with seizures, or they may cause seizures that are unrelated to any other disorders. About 20% of seizures in children are due to cerebral palsy or other neurological abnormalities (National Institute of Neurological Disorders and Stroke, 2009). Supporting studies does suggest that there are many other multifactorial events responsible for the occurrence of seizures apart from family history.

4. Ictal period of seizures

Approximate duration of the seizure attack was <5 mins in 131 (45.5%) children followed by 5-10 mins in 46 (16%) children, 10-20 mins in 22 (7.6%) children, 20- 30 mins in 25 (8.7%) and > 30 mins in 64 (22.2%) children with communication disorders.

45.5% of the children had seizure attack for <5 mins i.e, lesser duration and around 54.50% of them had an attack for more than 5 mins. The duration of the seizure attack ranged from minimum of <5 mins to maximum of >30 mins across communication disorders is depicted. Looking at the subgroup of communication disorders duration of the seizure attack was minimum (< 5 mins) in 86% of children with DD, >60% of children with BI, PDD and CP. Details are depicted in table 10. These children probably will have lesser risk factor compared to other conditions. Greater than 10 mins duration of the seizure attack was found in all children with speech regression, MA, 50% in ADHD, 43% in MR, 38% in DSL & BI, 30 % with PDD, 25% with CP & multiple disorders and 29% in HL.

Seizures can vary from the briefest lapses of attention or muscle jerks, to severe and prolonged convulsions (i.e. violent and involuntary contractions, or a series of

contractions of the muscles). The duration of seizures depend on the type of seizure. It can last anywhere from a few seconds to several minutes. In rare cases, seizures can last many hours. For example, a tonic-clonic seizure typically lasts 1-7 minutes. Absence seizures may only last a few seconds, while complex partial seizures range from 30 seconds to 2-3 minutes. "Status Epilepticus" refers to prolonged seizures that can last for many hours and this can be a serious medical condition (Theodore, Kelley, Toczek, & Gaillard, 2004).

Table 10. *Duration of seizure attack across subgroups of CWCDs*

Disorders	Total children	<5 mins (%)	5-10 mins (%)	10-20 mins (%)	20-30 mins (%)	>30 mins (%)
BI	11	7 (63.6)	-	1 (9.1)	2 (18.2)	1 (9.1)
DD	7	6 (85.7)	1 (14.3)	-	-	-
Speech regression	1		-	-	1 (100)	-
MR	109	35 (32.1)	27 (24.8)	10 (9.2)	11 (10.1)	26 (23.9)
PDD	3	2 (66.7)	-	-	-	1 (33.3)
ADHD	4	2 (50)	-	-	1 (25)	1 (25)
CP	24	15 (62.5)	3 (12.5)	-	1 (4.2)	5 (20.8)
DSL	39	19 (48.7)	3 (7.7)	2 (5.1)	4 (10.3)	11 (28.2)
HL	24	12 (50)	3 (12.5)	2 (8.3)	1 (4.2)	6 (25)
LD	2	1 (50)	1 (50)	-	-	-
MA	1	-	-	-	-	1 (100)
Multiple	63	32 (51)	8 (13)	7 (11)	4 (6)	12 (19)
Total	288	131 (45.5)	46 (16)	22 (7.6)	25 (8.7)	64 (22.2)

(Note: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Learning Disability (LD), Hearing loss (HL), Mental retardation (MR), Misarticulation (MA), Mutiple- HLCP, MRHL, MRCP, HLCPMR).

According to Sackeim, Deciena, Prohovinik, and Malitz (1987), first seizures commonly last more than 10 minutes, and may be as long as 30 minutes in children and adolescents. Researchers found that the average first seizure lasted 12.2 minutes, around half of the first seizures lasted more than 5 minutes, around 29% of first seizures lasted over 10 minutes and 12 per cent lasted over half an hour. Researchers from the Seizures Management Center at the Montefiore Medical Center in New York studied over 400 children in the age range 1 month to 19 years. It was found that 76% of the cases had average seizure duration of 3.6 minutes while the remaining 24% of cases had a average

duration of 31 minutes. Janelle, Smith, Pamela, and Braxton (2009) suggested that "once a seizure has lasted for 7 minutes, there is a greater than 95 per cent probability that it belongs to the group that will last much longer", concluding that once a seizure has lasted 5 to 10 minutes, the need for intervention is indicated. From the present study it is evident that 42.4% children had < 5 mins and 58% of children had >5 mins duration of attack. Findings indicate that more than 50% of CWCDs had >5 mins duration of attack. An increased seizure duration suggest longer time to recover as noted by authors and also there's a great need for intervention in more than 50% of the children considered in the study.

Duration of the seizure disorder was found to be negatively correlated with achievement. Janelle et al. (2009) found that children who had seizures for a longer period of time performed more poorly on the achievement domain than those who had the disorder for a shorter duration. It was noted a sufficiently long; a seizure probably can result in damage at any age. Albala, Moshe, and Okada (1984) reported that in addition to the cell loss, prolonged seizures can cause synaptic reorganization with aberrant growth (sprouting) of granule cell axons (the so-called mossy fibers) in the supragranular zone of the fascia dentata and the infrapyramidal region of hippocampus. Seizures also activate the trk subtype of neurotrophin receptor in the mossy fiber pathway and alter the expression of certain glutamate subreceptors (Represa, Tremblay, & Ben-Ari, 1990). Freeman & Vining (1992) reported that as the mossy fibers develop primarily in the early postnatal period, the development of these axons may be particularly prone to seizure-induced changes. Janelle et al. (2009) indicated that a longer duration of seizure influence how severe a caregiver judges seizures. The usual elements such as type of seizures, frequency of seizures, and most recent seizure were not significantly related to severity rating. In the present study it was found that majority of the children (157, 54.50%) had >5 minutes seizure duration. In the present study 50% of children had prolonged seizure. This further suggests that the axons may be particularly more prone to seizure-induced changes resulting in permanent damage in the neural system. However, considering the risk factors identified with increased

duration of seizures warrants swift referral for neuropsychological evaluation and management in children with communication disorders.

5. Fever

Seizure occurred only in the presence of fever in 122 children (44.2%), without the presence of fever in 70 children (24.2%) and there was no definite role of fever in 97 children (33.6%) with communication disorders.

Looking at the subgroup of communication disorders > 30% of children with BI, MR, DSL, HL, LD, CP, MD, PDD and ADHD had seizures only in the presence of fever, whereas >30 % of children with HL and LD had seizures without the presence of fever. There was no definite role of fever in > 30% of children with MR, ADHD, CP, DSL, MA and multiple disorders. Details regarding the presence and absence of fever in CWCDs are presented in the following table 11.

Table 11. *Fever during seizures in CWCDs*

Disorders	Total children	Fever during seizures		
		Present (%)	Absent (%)	Not always (%)
BI	11	5 (45.4)	3 (27.2)	3 (27.2)
Speech regression	1	-	1 (100)	-
DD	8	2 (25)	2 (25)	2 (25)
MR	109	48 (44)	24 (22)	37 (33.9)
PDD	4	1 (33.3)	3 (100)	-
ADHD	3	1 (33.3)	-	2 (66.7)
CP	24	9 (37.5)	5 (20.8)	10 (41.7)
DSL	38	17 (44.7)	8 (21.1)	13 (34.2)
HL	25	12 (48)	9 (36)	4 (16)
LD	2	1 (50)	1(50)	-
MA	1	-	-	1 (100)
Mutiple	63	26 (41)	14 (22)	25 (40)
Total	289	122 (44.2)	70 (24.2)	97 (33.6)

(Note: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Learning Disability (LD), Hearing loss (HL), Mental retardation (MR), Misarticulation (MA), Mutiple- HLCP, MRHL, MRCP, HLCPMR).

In children under the age of 5 years, fever from any cause may sometimes initiate a generalised seizure, causing great alarm. These "febrile convulsions" are similar to tonic-clonic seizures, but are much briefer. The tendency seems to run in families. In infants and in small children under the age of 18 months, febrile seizures

must be investigated especially with a view to excluding meningitis. In older children (after 5 years), seizures always linked to fever are unlikely to have a serious underlying cause unless abnormal physical signs are present. A child who suffers from febrile convulsions generally does not have a strong risk of developing seizures in later life. Overall, the risk of developing seizures in a child who has no neurological abnormality other than febrile convulsions is not greater than 3%. Febrile seizures are confined to infancy and early childhood, epileptic attacks which occur in children over the age of 5 cannot be accepted as being febrile convulsions, even if they do occur in a setting of fever. The risk of a young child suffering a further seizure during another feverish illness is about 30% (Maher & McLachlan, 1995).

Siddiqui (2002) found positive family history of febrile convulsions in 30% children. Initial febrile convulsion of simple type is more common in children with positive family history of convulsions, in whom first febrile convulsions tend to occur at earlier age. Further, complex febrile convulsions are more common when age at presentation is less than 12 months. As a whole 44% of children had first febrile convulsions below 12 months of age and 56% above 12 months of age. It was concluded that majority of febrile convulsions occurred in first two years of life. The febrile seizures can be very alarming to the parents and other caregivers. The risk of subsequent non-febrile seizures is only 2 to 3 percent unless one of the factors like a positive family history of seizures, signs of nervous system impairment prior to the seizure, or a relatively prolonged or complicated seizure is present. Researchers have now identified several different genes that influence the risk of febrile seizures in certain families. Studying these genes may lead to new understanding of how febrile seizures occur and perhaps point to ways of preventing them (Farwell et al., 1994).

The results of the present study are in partial agreement with earlier studies as it suggests presence of fever during seizure in 122 children (44.2 %) with communication disorders. Though children suffered from convulsions during fever, it was accounted for as the occurrence was during early childhood and the risk of developing seizures had to be determined. A previous study indicated the risk of a young child suffering a further seizure during another feverish illness is about 30% but in the current study almost all

children had another attack. A minimum number of one seizure attack was in 59 (22%), 2-3 times seizure attacks was present in 120 children (44.6%) and 33% of them had more than 3 times before consultation. The risk of subsequent seizures noted by the researchers is only 2 to 3 percent but in the current study it is nearing 30%. Hence, factors like signs of nervous system impairment prior to the seizure, or a relatively prolonged or complicated seizure may be present in children with communication disorders.

Nonfebrile seizures occur at all ages. The term "seizure" designates a clinical event that represents dysfunction of the central nervous system (CNS) and may signal a serious underlying abnormality. However, more often in children the seizures result from a transient disturbance of brain function. The ages of greatest risk for nonfebrile seizures are during infancy, childhood and adolescence. The annual incidence rate of seizures is 0.56 per 1000 in general population from birth to 20 years of age (Haslam, 1997).

Hirtz, Ashwal, Berg, Bettis, Camfield, and Shinnar (2000) analyzed first non febrile seizure in children aged 1 month to 21 years. They suggested that in the child with a first nonfebrile seizure, diagnostic evaluations influence therapeutic decisions, how families are counseled and the need for hospital admission and/or specific follow-up plans. An electroencephalogram should be obtained in all children in whom a nonfebrile seizure has been diagnosed to predict the risk of recurrence and to classify the seizure type and seizures syndrome. The majority of evidence from studies confirmed that an electroencephalogram helped in determination of seizure type, seizures syndrome and risk for recurrence, and therefore may affect further management decisions. Experts commonly recommend that an electroencephalogram be performed after first nonfebrile seizures. But, in the present study only limited clients had undergone EEG, probably due to cost factor.

Iannetti, Fiorilli, Sirianni, Paná, and Aiuti (2009) studied twelve hundred children with convulsions when feverish during a period of five years. Among them 52 subjects (4.33%) developed nonfebrile seizures after a period of eight months to five years from the first febrile convulsion (group A). Twenty-three children had neither

afebrile seizures nor EEG abnormalities during the period of observation (group B). The data suggested a correlation between persistence of neurologic abnormalities and cytomegalo virus (CMV) infection in children with nonfebrile seizures. Seki, Yamawaki, and Suzuki (2008) analyzed the frequency of development of nonfebrile seizures in 116 children (4.3%) who had experienced at least one febrile convulsion and were followed for more than five to eight years. Of these, three cases had prolonged generalized convulsions of the clonic or tonic-clonic type and two had brief generalized fits of the tonic-clonic type. The risk factors identified as nonfebrile seizures after febrile convulsions were the preexisting neurological abnormality or developmental retardation, focal features and more than 10-minute duration of the first febrile convulsions, and abnormal paroxysmal discharges at the initial interictal EEG recordings.

A variety of researchers documented afebrile seizures in 0.56, 4.3 and 5.3 cases per 1000 population with reference to general population. However, in the present study seizure occurred without the presence of fever in 70 (24.2%) children and there was no definite role of the presence of fever in 97 (33.6%) children with communication disorders. Findings revealed that the majority of CWCDs had afebrile seizures compared to general population. Thus data suggest a correlation between the persistence of neurologic abnormalities in majority of children. The findings also suggest importance of the selected follow up of difficult cases with non febrile seizures, for planning long term management and referral for specific problems. It is recommended that specialized seizures clinics could be organized which provide facilities for initial diagnosis and assessment. Such a system would improve the long term care of clients with seizures.

6. Time of occurrence of seizures

The epileptic seizure occurred in 102 children (42%) during the day, 60 children (25%) during night and in 80 (33%) CWCDs it occurred at any time. The occurrence of seizures in subgroup of communication disorders is depicted in the following table 12. Maximum number of children with mental retardation had occurrence during day, night and any time. It comprised of 20 (18.3%), 13 (11.9%) and 76 (69.72%) children during

day, night and any time respectively. On comparing the epileptic seizure among subgroup of communication disorders >50% of children with PDD, ADHD, DSL, HL, LD & MA had seizures during the day whereas only children with LD had seizures during night. Seizure occurred any time in >50% of children with Speech regression.

Table 12. *Time of occurrence of seizures in CWCDs*

Disorders	Occurrence of seizures			
	Total cases	Day (%)	Night (%)	Any time (%)
BI	7	3 (42.9)	1 (14.3)	3 (42.9)
Speech regression	1	-	-	1 (100)
DD	4	-	2 (50)	2 (50)
MR	96	35 (36)	25 (26)	36 (38)
PDD	3	2 (67)	1 (33.3)	-
ADHD	4	3 (75)	1 (25)	-
CP	21	10 (48)	4 (19)	7 (33)
DSL	33	19 (58)	7 (21.2)	7 (21.2)
HL	22	11 (50)	5 (23)	6 (27)
LD	2	1 (50)	1 (50)	-
MA	1	1 (100)	-	-
Multiple	49	17 (35)	13 (27)	18 (37)
Total	242	102 (42)	60 (25)	80 (33)

(Note: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Learning Disability (LD), Hearing loss (HL), Mental retardation (MR), Misarticulation (MA), Mutiple- HLCP, MRHL, MRCP, HLCPMR).

Durazzo, Spencer, Duckrow, Novotny, and Zaveri (2008) determined seizure occurrence in partial seizures under the influence of circadian rhythms and rhythmic exogenous factors, and how this influence varies according to cortical brain region. For these ends, they determined and analyzed detailed temporal distributions of seizures arising from the frontal, parietal, occipital, neocortical temporal and mesial temporal lobes. The results suggested that the seizure distribution was dependent on brain region ($p < 10^{-9}$). Nonuniform seizure distributions were observed in the parietal ($p < 10^{-4}$), occipital ($p < 10^{-7}$), mesial temporal ($p < 0.02$), and neocortical temporal lobes ($p < 0.04$). Occipital and parietal seizures occurred in strong gaussian-like distributions, 180° out of phase relative to each other; occipital seizure occurrence peaked between 16:00 and 19:00, whereas parietal seizures peaked between 4:00 and 7:00. Frontal lobe seizures followed a unimodal distribution, peaking between 4:00 and 7:00. Seizures

from the mesial temporal lobe were distributed bimodally, with the primary peak in the late afternoon between 16:00 and 19:00 and secondary peak in the morning between 7:00 and 10:00. Neocortical temporal seizures peaked slightly before the primary peak observed in the mesial temporal lobe; however, these distributions did not differ significantly. It was concluded that seizure occurrence in partial seizures is not random. Endogenous circadian rhythms and rhythmic exogenous factors likely play substantial roles in seizure occurrence. These roles vary considerably according to brain region. Frontal and parietal lobe seizures seem most likely to occur nocturnally, whereas occipital and temporal lobe seizures seem to have strong afternoon preferences.

Any form of seizures may occur during sleep, but some types of seizures are more likely to be restricted exclusively to sleep. These are sometimes called nocturnal seizures. Some people have seizures occur only during sleep while others have both daytime and nocturnal seizures. Studies have shown that 10 - 45% of people with seizures have seizures that occur predominantly or exclusively during sleep or occur with sleep deprivation. The majority of nocturnal seizures occur in light sleep - soon after falling asleep, before waking or around arousal during the night. This is especially true with temporal lobe seizures, myoclonic seizures, and atypical absence spells (William, 1979).

Although the mechanism is poorly understood, there is evidence that sleep activity may influence seizures. The results of the present study are in partial agreement with the earlier research indicating occurrence of seizure during night in 60 (25%) children. Though literature emphasizes on specific regions and nocturnal seizures (during sleep), the present study suggested mixed results and majority had no restriction on the seizure occurrence. The epileptic seizure occurred in 102 (42%) during the day, 60 (25%) during night and 80 (33%) CWCDs at any time. The data suggests no focal distributions of seizures arising from the lobes in the brain region and majority had generalized seizures. It further suggests that the abnormal neuronal activity would probably occur in most part of the brain.

7. Correlation of epileptic seizures and communication disorders

The speech, language, motor and cognitive skills were normal till the onset of seizures in 72 (19.2%) children with communication disorders as per the parent reports. Details on the parental opinion regarding epileptic seizures as a cause of communication disorders among subgroups are depicted in table 13.

Table 13. *Parental opinion regarding epileptic seizures*

Disorders	Total cases	Seizures as a causative factor (%)	Seizures as not a causative factor (%)	Not sure (%)
BI	9	3 (33.3)	5 (55.6)	1 (11.1)
Speech regression	1	1 (100)	-	-
DD	5	1 (20)	2 (40)	2 (40)
MR	106	30 (28.3)	37 (34.9)	39 (36.8)
PDD	3	1 (33.3)	2 (66.7)	-
ADHD	4	-	4 (100)	-
CP	21	6 (28.6)	8 (38.1)	7 (33.3)
DSL	32	11 (34.4)	16 (50)	5 (15.6)
HL	24	5 (20.8)	12 (50)	7 (29.2)
LD	2	-	-	2 (100)
MA	1	-	1 (100)	-
Multiple	60	14 (23)	25 (42)	21(35)
Total	268	72 (19.2)	112 (38.9)	84 (31.3)

(Note: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Learning Disability (LD), Hearing loss (HL), Mental retardation (MR), Misarticulation (MA), Mutiple- HLCP, MRHL, MRCP, HLCPMR).

Findings suggest that few parents assumed that the epileptic seizure is probably the cause for deficits seen in the children which ranged from 11.9%-100%. It was found that >30% of parents of children with BI, Speech regression, PDD and DSL assumed that epileptic seizure is probably the cause. One parent of child with speech regression was very sure that seizures were the cause for the client's condition as client was normal before the onset of seizures.

Acquired epileptic aphasia (AEA) typically develops in healthy children who acutely or progressively lose receptive and expressive language ability coincident with

the appearance of paroxysmal EEG changes. Few authors use AEA as a synonym for Landau-Kleffner syndrome (LKS). In most cases a clearly normal period of motor and language development occurs before AEA symptoms appear. However, in the last 20 years, several reported cases have been difficult to classify because the patients' presenting symptoms appear to have been variants of those originally described. In one case, expressive language deteriorated instead of receptive language, whereas in another case, a brief period of normal language development (single words) was followed by language regression with abnormal EEG findings. The pathophysiology of seizures and epileptiform discharges leading to language dysfunction in AEA is disputed. Aphasia and EEG abnormalities might have a common cause, for example, a left temporal brain astrocytoma or head injury. Some speculate that synaptogenesis mediates the neurologic deficits in AEA and that epileptiform discharges during a critical period of synaptic reinforcement or pruning in turn mediate the reinforcement of synaptogenesis. Some patients with AEA appear to have worsened language skills during periods of increased epileptiform activity (Menezes, 2010). The continuous spikes and waves during slow sleep during interictal EEG activity in Landau-Kleffner syndrome may be the primary cause of cognitive impairment. Hypsarrhythmia is a severely abnormal pattern, and this dysfunctional state probably prevents normal development (Bishop, 1985).

A majority of infants who develop infantile spasms have developmental delay prior to the onset of the seizures, with both the developmental delay and spasms resulting from the same symptomatic etiology. However, Bednarek et al. (1998) found infants who were previously normal were at substantial risk for developing delays with the onset of the spasms. They may regress with decline in attention, alertness, eye contact and activity level.

Thierry and Eliane (2006) reported that the onset of seizures in brain systems involved in social communication and/or recognition of emotions can occasionally be the cause of autistic symptoms or may aggravate preexisting autistic symptoms. Seizures and/or epileptic EEG abnormalities are frequently associated with autistic disorders in children but this does not necessarily imply that they are the cause. Great caution needs to be exercised before drawing any such conclusions. Nevertheless, there

are several early epilepsies (late infantile spasms, partial complex epilepsies, epilepsies in children with slow wave activity (CSWS), early forms of Landau-Kleffner syndrome) and with different etiologies (tuberous sclerosis) in which a direct relationship between seizures and some features of autism may be suspected. In young children who primarily have language regression (and who may have autistic features) without evident cause, and in whom paroxysmal focal EEG abnormalities are also found, the possible direct role of seizures can only be evaluated in longitudinal studies. On a group basis, both the cognitive level and the adaptive behaviour level were lower in the seizures group than in the non-seizures group.

Summarizing the results of present study parents reported speech, language, motor and cognitive skills as normal till the onset of seizures in 72 (19.2%) children, not normal in 112 (38.9%) and in 84 (31.3%) children the parents were not sure whether epileptic seizure had any effect on their child's condition. Seizures and communication disorder may occur together and both may result from same underlying brain disorder. The developing brain is susceptible to many kinds of injury. Injury to the brain may certainly cause seizures. This includes deprivation of oxygen at birth, trauma to the head at any time of life, and stroke (injury to part of the brain caused by blockage or haemorrhage of one of its blood vessels). These conditions may lead to cerebral palsy, which often is associated with seizures, or they may cause seizures that are unrelated to any other disorders. About 20 percent of seizures in children are due to cerebral palsy or other neurological abnormalities. Mental retardation, seizures, and other sensory and motor deficiencies frequently are associated in one particular syndrome. Several instances of partial epilepsies are associated with these cortical malformations. It is characterized by local neuronal abnormalities, lack of normal lamination in the cortex, abnormal giant neurons, and abnormalities in dendrites and axons. The epileptic disorder also varies in severity depending upon the degree of the cortical disorganization (Camfield et al., 2001). However, the causal association between seizures and language impairment is poorly documented due to constraints in epidemiological methods. The onset of seizures in clients with brain damage can occasionally be the cause of language disorder or may aggravate preexisting language

disorder. Nevertheless, there are several early epilepsies, occurring at very young age even before speech development starts. But this does not necessarily imply that they are the cause, great caution needs to be exercised before drawing any such conclusions. The possible direct role of seizures can only be evaluated in longitudinal studies. The present study is a retrospective analysis and hence most of the information was as reported by the parents considered from the casefiles. Majority of the time information collected depend on the type of understanding the parents have regarding the condition. High fever and infections might be unnoticed or even if noticed will have less significance. The first epileptic seizure that involves an abnormal body movement for a short duration may have greater significance for parents. Hence, there is a probability of parents to report as seizures being the causative factor for communication disorder. A group of parents reported that the speech, language, motor and cognitive skills were not normal since childhood. It was found that 50% of parents of children with BI, PDD, ADHD, MA, HL and DSL assumed that epileptic seizure is not the cause. 31.3% of the parents i.e majority of them was sure that seizures was not the cause for the condition in their child. In majority of the children communication problems are due to congenital problems. They might have observed a global developmental delay even before the occurrence of seizures hence they might have concluded, it as not a causative factor. Neurological handicaps at birth such as cerebral palsy and mental retardation are associated with an increase risk for seizures, probably due to common antecedents. They should be considered as markers for underlying brain abnormalities, which are responsible for the neurological handicap and for seizures. Clearly, the cause of seizures is the major factor, but the seizures itself is important because it multiplies the effect of the existing disabilities rather than simply adding to them.

8. Clinical manifestations of epileptic seizure

The involuntary muscle contractions exhibited by the children during the seizure included bilateral, symmetrical and repetitive muscle movements in 209 children (66.2%). Unilateral movements were exhibited in only 66 children (20.8%). Loss of consciousness was found in 101 children (30.7%), abnormal eye movements in 121 children (38.2%), froth in the mouth in 114 children (29.4%), letting out a yell in 36

children (29.4%) and repeated eye blinks in 41 children (12.7%). Different kind of combination symptoms mentioned above was found in 180 children (33.8%). Among the children in different subgroups of communication disorders majority of children with mental retardation had bilateral, symmetrical and repetitive muscle movements in 39 children (36%), Unilateral movements in 29 children (27%), Loss of consciousness in 49 children (50%), abnormal eye movements in 56 children (55%).

Researchers indicated that seizures vary and depend on where in the brain the disturbance first starts, and how far it spreads. Temporary symptoms can occur, such as loss of awareness or consciousness, and disturbances of movement, sensation (including vision, hearing and taste), mood or mental function. A person having a generalized tonic-clonic seizure may cry out, fall to the floor unconscious, twitch or move uncontrollably, drool, or even lose bladder control. Within minutes, the attack is over, and the person regains consciousness but is exhausted and dazed. Generalized seizures are a result of abnormal neuronal activity on both sides of the brain (Freeman, Vining, & Pillas, 2002). DeToledo and Ramsay (1996) recorded the patterns of involvement of the orbicularis oculi and other facial muscles during 654 events of seizures in 257 patients undergoing telemetry evaluation. Four hundred fifty-seven episodes represented epileptic seizures and 197 represented psychogenic seizures. Eyes were wide open in more than 90% of patients during the tonic phase of a generalized tonic clonic seizure. Lowering of the lid with partial closure of the eye, without contraction of the orbicularis oculi, was the predominant form of eye closure observed. Eye closure in any form was uncommon during the ictal stage of epileptic seizures with motor accompaniment and occurred in 21 of 408 cases and in 2 of 49 simple partial seizures somatosensory type. The mouth is usually wide open during the tonic phase of a generalized convulsion. The presence of a clenched mouth during a "tonic spell" should raise the possibility of psychogenic seizures. Injuries to the tongue due to biting during the epileptic seizures usually affect the side of the tongue. Biting of the lip or tip of the tongue was not seen with epileptic attacks and is also suggestive of psychogenic seizures.

In the present study majority of the children (209, 66.2%) exhibited involuntary muscle contractions during the seizure which included bilateral, symmetrical and

repetitive muscle movements. Findings suggest that the majority presented with generalized seizures which is a result of abnormal neuronal activity on both sides of the brain. It may be concluded that due to abnormal discharges in the whole brain a subgroup of CWCDs present with global delay in language. However to validate this findings further research is required on this dimension.

9. Triggering factors

Most seizures happen completely out of the blue. For some people, however, seizures are triggered by certain stimuli. These often differ from one person to another and many of the people interviewed noted that more than one factor was involved in setting off their seizures. Many participants reported that stress, anxiety or excitement triggered their seizures. Tiredness and lack of sleep were also common triggers. In some people caffeine can trigger seizures while others are susceptible to having seizures if they miss meals and have a low blood sugar level. Children are particularly likely to have more seizures when they develop infections such as tonsillitis and earache. This is possibly due to high temperature and usually eases within a few days. Allergies may provoke seizures in some people with seizures. Diarrhoea and vomiting can trigger seizures because they can prevent one's body from absorbing antiepileptic medication. There are other possible triggers with some unique to certain people. For example some unusual stimuli which have been known to trigger seizures include the colour yellow, the smell of glue and sounds such as the telephone ringing or a siren. Photosensitive seizures are triggered by sensory stimuli such as flickering sunlight, strobe lights and flickering television. In some people it may be triggered when the weather becomes very warm or rooms are overheated (Epilepsy foundation of Victoria, 2006). A seizure diary for a period of time will help in getting to know more about triggering factors and prevention of exposure to such factors is essential for seizure free condition.

However, the above reports were found with reference to general population. The present study considered communication disorders and only 36 children (15.9%) had seizures triggered by specific type of stimulus. The triggering stimulus type was auditory among 10 children, visual among 9 children and other triggering factors (not

specified by parents) in 17 children. Details regarding positive and negative triggering factors are depicted in the table 14. On comparing children in subgroups of communication disorders, seizures were triggered by specific type of stimulus that ranged from 8%- 67% of children, majority comprising of ADHD. All children with Speech regression, PDD, LD and MA had no triggering factors.

Rantala, Uhari, and Hietala (1995) analysed the symptomatology of infections as well as immunological and virological findings in 58 children experiencing their first febrile seizures. High temperature was the only variable to explain the occurrence of febrile seizures in the logistic model after adjusting for duration of symptoms.

Table 14. *Triggering factors*

Disorders	Total children	Triggering factors	
		Negative	Positive
BI	9	8 (88.9)	1 (11.1)
Speech regression	1	1 (100)	-
DD	4	3 (75)	1 (25)
MR	85	70 (82.4)	15 (17.6)
PDD	2	2 (100)	-
ADHD	3	1 (33.3)	2 (66.7)
CP	17	13 (76.5)	4 (23.5)
DSL	33	28 (84.8)	5 (15.2)
HL	20	18 (90)	2 (10)
LD	1	1 (100)	-
MA	1	1 (100)	-
Multiple	50	44 (88)	6 (12)
Total	226	190 (84.1)	36 (15.9)

(Note: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Learning Disability (LD), Hearing loss (HL), Mental retardation (MR), Misarticulation (MA), Mutiple- HLCP, MRHL, MRCP, HLCPMR).

Patients in the seizure group had a significantly higher temperature at home than controls before hospitalization (39.4 versus 38.8^{o}C). The finding of higher temperatures in children with febrile seizures supports its importance as the most important triggering factor in febrile convulsions. However, even in the present study seizure occurred only in the presence of fever in 70 children (24.2 %) with communication disorders. It may

be concluded that higher temperatures in children is a most important triggering factor in febrile convulsions.

Seizure may also be triggered by a transient disruption of cortical neuronal function such as a disturbed metabolic state (high fever, hypocalcemia, hyponutremia), temporary cortical disturbance after minor head trauma (concussion), ischemia, chemical/inflammatory excitation caused by infection (meningitis, encephalitis, sepsis), or bleeding (intraparenchymal or subarachnoid hemorrhage). A seizure can also be the manifestation of a chronic disturbance of neuronal function caused by a remote event such as perinatal asphyxia or in-utero stroke, the expression of a genetic syndrome, or a progressive neurologic disorder such as a tumor or a neurodegenerative or neurometabolic disease. Seizures may recur over days or weeks and disappear, never to recur (eg, in viral meningoencephalitis), disappear for months or years (eg, after perinatal asphyxia), and then recur, or continue unabated from the initial inciting event (eg, in infants with brain malformations). Majority of children (226, 79.2%) considered in the present study had no triggering factors. To support our findings, more than 2 million people in the United States, about 1 in 100 has experienced an unprovoked seizure or has been diagnosed with seizures (Kelley & Swierzewski, 2002). Many times a seizure can be a transient disruption or a chronic disturbance of neuronal function. These disruptions are one time cause leading to permanent ill effects. In majority of communication disorders such disturbances are more evident this suggests that they were at greater risk during prenatal period.

10. Reaction towards epileptic seizure

Children's reaction towards epileptic seizure before and after the attack was analyzed. Reaction before the attack of seizures was tiredness in 108 children (34.1%), stiffness in 123 children (38.9%), restlessness in 98 children (24.7%) and combination of these reactions in 77 children (24.3%). Among the different subgroups of children with communication disorders, majority of children with mental retardation presented with tiredness, stiffness and restlessness followed by cerebral palsy, borderline intelligence, mental retardation with hearing loss and autism.

Reaction after the attack of seizures was tiredness noticed in 203 children (64.2%), stiffness in 103 children (29.7%), restlessness in 76 children (34%) and combination of these reactions in 122 children (38.7%). Majority of CWCDs reacted with tiredness after the attack of seizures.

Donat (1992) reported a cry at the time of or just after the infantile spasm. It is often followed by a brief episode of akinesia and diminished responsiveness. After a series of spasms, the infant may be exhausted and lethargic. Conversely a brief period of increased alertness that appears to correlate with brief periods of improved background activity in EEG may also be observed (Lombroso, 1983a). Vignatelli et al. (2006) administered a questionnaire on daytime sleepiness–related symptoms and subjective sleep quality to 33 patients with Nocturnal Frontal Lobe Epilepsy (NFLE) and 20 controls. Tiredness and spontaneous sleep awakening were significantly more frequent in seizures patients than controls (36.4% vs. 11.1% and 50% vs. 22%, respectively). Our study is in consensus with Donat (1992) and Vignatelli et al. (2006) findings as majority of CWCDs reacted with tiredness after the attack of seizures. The reactions were constrained to tiredness, stiffness, restlessness and combination of these reactions. These were noticed probably due to temporary inhibition of all the systems associated with abnormal movements leading to reduced strength in the systems.

11. Seizure control

Seizure was completely under control with medication in 163 children (58.2%). However, the duration to reach seizure free condition varied across different children. Seizure was under control in 43 children for 6 months, 11 children for 6-12 months, 24 children for 12months- 2 years and 85 children for > 2 years. Controlled and uncontrolled seizures among subgroups of CWCDs are depicted in table 15. Among which seizures ranged from 33% - 100%. All children with LD & PDD , >50% of children with BI, MR, PDD, CP, DSL, HL and LD had seizures under control suggestive of improvement in seizure control in half of the children.

Table 15. *Controlled and Uncontrolled seizures*

Disorders	Total children	Uncontrolled Seizure (%)	Controlled Seizure (%)
BI	9	1 (11.1)	8 (88.9)
Speech regression	1	1 (100)	-
DD	6	4 (66.7)	2 (33.3)
MR	109	50 (45.9)	59 (54.1)
PDD	3	-	3 (100)
ADHD	4	2 (50)	2 (50)
CP	24	10 (41.7)	14 (58.3)
DSL	35	8 (22.9)	27 (77.1)
HL	25	6 (24)	19 (76)
LD	1	-	1 (100)
MA	1	1 (100)	-
Mutiple	62	34 (55)	28 (45)
Total	280	117 (41.8)	163 (58.2)

(Note: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Learning Disability (LD), Hearing loss (HL), Mental retardation (MR), Misarticulation (MA), Mutiple- HLCP, MRHL, MRCP, HLCPMR).

Millicahap and Aymat (1967) reported that control of grand mal seizures is possible in about 60% of the cases with medication. In petit mal seizures, complete control with drug therapy was successful in 39%. Sakauchi, Oguni, Mukahira, and Fukuyama (1995) investigated the clinical condition and followed up status of epileptic patients. The results revealed that out of 40 persons, 30 still had seizures. They concluded that epileptic patients should receive a long-term treatment plan. In series of childhood seizures, more than half the patients were seizure free after a 10-year or longer follow up (Sillanpaa, 2000). In a long (12 years), prospective, follow-up study of 119 children, Brorson and Wranne (1987) found an over all terminal remission rate of 64. The 3-years terminal remission rate was 89% if the child's neurologic status was of normal neurology. In a group of children with normal neurology remission rate was reported to be 89%. However, in the present study only 163 (58.2%) children had controlled seizures. This may be attributed to the fact that majority of CWCDs had neurologic status that may not be normal.

Huttenlocher and Hapke (1990) followed up children (61% had mild to moderate retardation, and 39% were of borderline intelligence) and found good seizure control which increased by roughly 4% yearly starting about 4 years after onset of seizures and only one-fourth of these followed for 18 years or more continued to have more than one seizure per year. The present study considered 109 children with mental retardation. Out of 109 children, 59 (54.1%) children had seizure control. This suggests that 50% of children with mental retardation had seizure control after taking medication. It further indicates that 50% of children with mental retardation showed satisfactory results regarding seizures free condition.

About 80 percent of those diagnosed with seizures, seizures can be controlled with modern medicines and surgical techniques. However, about 25 to 30 percent of people with seizures will continue to experience seizures even with the best available treatment. Such condition is termed as intractable seizures (American Academy of Neurology, 2006). According to an earlier study by Martino and Tuchman (2001), 36% of patients with seizures have inadequate control of seizures with antiepileptic drugs (AEDs); resulting in substantial deleterious effects on individual health and quality of life and a heavy burden on society. The study concludes that after two failed drug attempts, the likelihood of another drug succeeding is only four per cent. Yet another study followed up 613 children with newly diagnosed seizures seen by physicians in Connecticut for at least 18 months and an average of nearly five years. Of those, 10% met the criteria for difficult-to-treat or intractable seizures. It was defined as the failure of two or more first-line seizures drugs to control seizures; children had one or more seizures a month for 18 months. The researchers identified three factors that increase the risk of having intractable seizures. Those with a type of seizures called cryptogenic/symptomatic generalized seizures have the greatest risk. Children with this type of seizures are usually young when they have their first seizure, often less than a year old. It can occur in children who were previously neurologically normal, called cryptogenic. More often it occurs in children who were known to have neurological abnormalities, called symptomatic. In their study, 35% of the children with this type of seizures met the criteria for intractable seizures, compared to 8% of those with other forms of seizures (Lizana et al., 2009). A study by Berg, Shinnar, Levy and Testa

(1999) found an increasing percentage of seizure frequency which reached a maximum of nearly 27% in those with 200 or more seizures per month. Ellenberg, Hirtz, and Nelson (1984) found that 344 (67%) of their 513 children with seizures, excluding those with febrile convulsions and seizures associated with acute systemic disorders, still had seizures at the age of 7 years. Sridharan (2002) indicated that the factors during the course for those who come under control relatively late and have numerous seizures before being controlled as well as those who require multiple drugs to control their seizures have a worse prognosis for eventual remission.

Summarizing the literature researchers found controlled seizures ranged from 39%- 90% and uncontrolled seizures ranged from 25%- 70% in general population. One study considered children with reduced IQ and also found good seizure control which increased to 4% yearly only after 4 years of age. The results of the present study are in agreement with literature which revealed half the CWCDs had seizures completely under control. Though seizures were under control, children did present with one of the communication disorders. It suggests that the problems identified in any of the three domains behavioral, cognitive, and neurologic, were more influential than were only problems related to seizures. Remaining 41.8% had repeated seizures and not improving with medication. Seizures not under control indicate the impact of neurologic chronic disease in children with communication disorders. Children with slow activity in an area of the brain were also more likely to have intractable seizures than children with normal EEGs. Eltze et al. (2005) reported in young children with reduced control of seizures, identification of localized areas of abnormally developed cortex (focal cortical dysphasia, FCD) on magnetic resonance (MR) brain scans is important. Surgical removal of abnormal cortex may offer improvement of seizure control and subsequent developmental progress. The impact on the family of a child with seizures and communication disorders is significant, particularly when the epileptic seizures are uncontrolled. In children with both seizures and neurologic motor deficits, the neurologic deficit will have a greater impact on family and on the child's life than did seizures alone. Repeated and long seizures are most often associated with long postictal dysfunction, which, at some point, may not recover completely. The current data

suggest that few CWCDs should receive a long-term treatment plan. The neurologists should observe and follow closely the children who have a high seizure frequency. A careful analysis of recurrence of seizures should be taken up as a rule. The data also suggest that communication between primary care physicians and neurologists should be maintained, as this would promote better understanding of seizures and foster good relations among institutions.

12. Co-morbidities of seizures and the nature of communication disorders

Epilepsy is a chronic disorder that significantly affects learning and behavior. Children with seizures are particularly vulnerable to educational problems and resultant academic underachievement. Co-morbidities like cognitive and behavioral problems contribute significantly for the problems at school. Both seizures and neuropsychological deficits may occur as different clinical manifestations of a common etiological process (Vinayan, 2006). Reports in the literature have suggested that persons with seizures may display various cognitive impairments, including impoverished memory, reduced attentional capacity (Stores, 1981) and mental slowness (Trimble & Corbett, 1980). Mulas et al. (2006) analyzed the association between neuropsychological disorders and seizures in infancy. They determined the existence of electrical discharges, both in the presence and the absence of clinical seizures. It is linked to problems involving attention, recent memory, limitations in the linguistic, visuospatial and executive capabilities, with slowed psychomotor functioning and this leads to learning difficulties.

The information regarding co-morbidities of seizures (associated problems) were considered in the present study among subgroup of communication disorders. It was found that as a group they had drooling in 17 children (6%), behavioural problems in 3 children (1%), teeth grinding in 25 children (8.8%) and combination of these problems in 226 children (80.4%). The results revealed that in majority of children, the problems occurred in combination. Looking at the subgroup of communication disorders all children with BI (11), Speech regression (1), DD (6) and PDD (3) had problems in combination. Details regarding these are depicted in the table 16.

Table 16. *Co-morbid behaviors in CWCDs with seizures*

Disorders	Total children	Drooling (%)	Behaviour problems (%)	Teeth grinding (%)	Combination (%)
BI	11	-	-	-	11 (100)
Speech regression	01	-	-	-	1 (100)
DD	06	-	-	-	6 (100)
MR	113	8 (7.1)	1 (9)	11 (9.7)	93 (82.3)
PDD	03	-	-	-	3 (100)
ADHD	04	-	-	1 (25)	3 (75)
CP	24	1 (4.2)	1 (4.2)	2 (8.3)	20 (83)
DSL	33	1 (3)	-	5 (15.2)	27 (81)
HL	23	3 (1.3)	-	3 (1.3)	17 (73.9)
Multiple	63	4 (6.3)	1 (1.5)	3 (4.7)	55 (87.3)
Total	281	17 (6)	3 (1.0)	25 (8.8)	226 (80.4)

(Note: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Hearing loss (HL), Mental retardation (MR), Multiple- HLCP, CPMR, MRHL, HLCPMR).

Eyman, Caples, Moore, and Zachofsky (1969) in a study found that hyperactivity was more common in mentally retarded patients with seizures. Aggressive behavior, speech problems and difficulties in eating and dressing were also more common, but this may well have been a reflection of the more severe degree of retardation of the patients with seizures who are likely to be selectively admitted for these reasons. All the patients were institutionalized and drugs were thought to play an important part in causing behavior disturbance. In the present study 109 children with mental retardation had seizures. The results of the present study are in agreement with Eyman et al's study as all children with mental retardation presented with hyperactivity, cognitive problems and speech problems.

Northcott et al. (2005) demonstrated that children with benign rolandic seizures (BRE) had normal intelligence and language ability. However, a specific pattern of difficulties in memory and phonological awareness was found. Fastenau, Jianzhao Shen, Dunn, and Austin (2008) assessed rates of learning disabilities (LD) in general population. Using an IQ-achievement discrepancy definition, 48% exceeded the cutoff

for LD in at least one academic area. Younger children with generalized nonabsence seizures were at increased risk for math LD. Age of seizure onset and attention-deficit/hyperactivity disorder (ADHD) were risk factors for reading and math LD. Writing was the most common domain affected, but neither ADHD nor seizure variables reliably identified children at risk for writing LD. In the present study 2 children with LD had seizures. The results are in agreement with earlier research as both the children presented with reading and writing difficulties. However, finer details on the difficulties exhibited by these children need further investigation.

Oslejskova, Dusek, Makovska, and Rektor (2007) categorized speech problems in autistic children in a manner allowing recognition of associated risk factors. Epileptic seizures, epileptiform EEG abnormalities, non-right-handedness, hypotonia and severe decreased IQ score were found to be the most important mutually independent factors contributing to the increased risk of speech-related problems in patients with ASD.

Jeremy et al. (2009) compared developmental and psychological functioning in two groups of children with Autism Spectrum Disorder (ASD) with and without seizures. ASD with seizures were more likely to have received later ASD diagnoses and additional medical diagnoses. They also showed more motor difficulties, developmental delays and challenging behaviours, but were no more likely to be aloof and passive. The ASD only group experienced more abnormal fascinations with objects and used brief glances as a means of eye contact more than the ASD+e group. In the present study a comparison of ASD with and without presence of seizures was not made. However, considering only ASd with seizures, children did manifest learning difficulties especially language.

Caplan et al. (2009) examined the severity and range of linguistic impairments in young, intermediate, and adolescent youth with seizures. The results indicated that significantly more seizures subjects had language scores 1 standard deviation (SD) below average than the age-matched control groups did. The intermediate and adolescent seizures groups also had significantly lower mean language scores compared to their matched controls. The older compared to the younger seizures groups had more language impairment and a wider range of linguistic deficits. Longer duration of illness,

childhood absence seizures, psychiatric diagnosis, and socioeconomic status were associated with linguistic deficits in the young group. Prolonged seizures lower Performance IQ, and minority status predicted low language scores in the intermediate seizures group. In the adolescent group, language impairment was associated with poor seizure control, decreased performance IQ, and lower socioeconomic status. Linguistic and reading deficits were significantly related in each seizures group. In the present study 24 children with delay in speech and language had seizures. The results are in agreement with earlier research as findings suggest slow in development of language (linguistic impairment) in children with positive history of seizures.

Research suggests that some children with seizures experience learning difficulties in specific subject areas, most notably mathematics and reading. Some may experience behavioural problems and/or learning difficulties. It is important to remember that simply because a child has seizures it does not follow that every other problem is also related to their seizures. Behavioural problems and learning difficulties can have many different causes. They are any type of damage to the brain, area of the brain in which epileptic activity is occurring, severity of the seizures, type of seizure, sub-clinical seizure activity (refers to continuous epileptic activity taking place in the brain without any obvious outward signs), duration of seizures, anti-epileptic medication, psychological and social factors, such as family and peer attitudes as well as self-image (Keeley & Swierzewski, 2002).

Our findings suggested the presence of comorbidities (associated problems) in children with communication disorders. This could be due to the coexistence of neurological deficit, communication deficit, frequency, duration of seizures and anti-epileptic medication. However, direct correlation of severity of seizures and associated problems requires further investigation.

13. Type of seizures

The diagnosis of seizures presents unique difficulties in persons with communication disorders. Mulas et al. (2006) reported that the type of seizure or seizures syndrome is determined by taking complete medical history, blood tests, EEG

tests and brain-imaging tests such as CT and MRI scans. This gives information about the electrical activity of the brain, what the brain looks like and possible causes of seizures. This information when put together with how the individual was feeling pre, during and post attack would best explain the event. The parents generally are not able to describe the epileptic events, and the physician or someone trained in seizures observe the events only rarely. The clinical manifestations often are observed by people (eg, parents, family members, and teachers) who are not familiar with epileptic disorders. However, in the current study based on the signs and symptoms reported type of seizures was determined. EEG reports were not available for most of the children and hence the neurologist one of the co- investigators decided on the type based on signs and symptoms. The three types of seizures found among CWCDs are depicted in figure 3. They are primary generalized seizures (PGS) in 41 (13%) children, secondary generalized seizures (SGS) in 236 (74.7%) children and idiopathic seizures (IS) in 37 (11.7%) children.

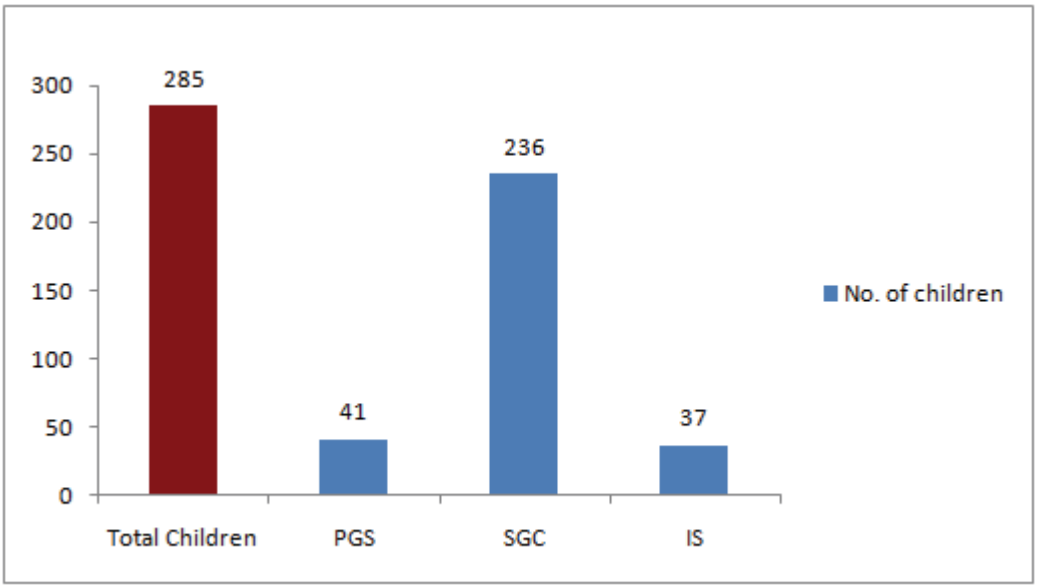

Figure 3. *Type of seizures among CWCDs*

As mentioned in the above figure majority of the children presented with secondary generalized seizures compared to primary generalized seizures and idiopathic seizures. Sridharan (2002) found the prevalence rate of some of the seizures syndromes

in general population. They were, West Syndrome: 1.4-1.9 per 10,000 children, 1-9 per 10,000 populations; Lennox-Gaustaut Syndrome: 1.3-3 per 10.000 children; 2-6 per 100,000 population; juvenile myclonic seizures; 3 per 10,000 (2.5% of prevalent persons with seizures); idiopathic localization-related seizures; (4-8% of prevalent persons with seizures) 2.4 -4 per 10.000 populations. In the new cases of seizures 50% have seizures of partial origin and 50% of generalized origin before the age of 40. Berg, Shinnar, Levy, and Testa (1999) accounted for 45% generalized seizures whereas focal seizures with secondary generalized seizures were present in 55% of cases. In most large studies of people with seizures only, 20% of seizures are considered to be symptomatic and the remainder either idiopathic (30%) or cryptogenic (50%). Durá-Travé, Yoldi-Petri, Hualde-Olascoaga, and Etayo-Etayo (2008) indicated that the aetiology was symptomatic in 40 cases (66.7%), cryptogenic in 16 (26.7%) and idiopathic in four cases (6.7%). Unfortunately, these studies note the presence of only "neurologic abnormality" rather than the specific type of problem (e.g., cerebral palsy, visual impairment, deafness). These studies were with reference to general population. However, the present findings were with reference to communication disorders. Though the groups varied across studies similar results have been obtained regarding the type of seizures. Hence it may be concluded that, whether the group is general or clinical population majority of children exhibit secondary generalized type of seizures.

Camfield et al. (2001) reported generalized tonic and tonic-clonic seizures and complex partial seizures with or without secondary generalization most frequently, myoclonic seizures and atonic seizures are also common. Typical absence seizures were observed less frequently in children with cerebral palsy (CP). The present study is in agreement with Camfield et al's (2001) study. It was found that majority had secondary generalized seizures in 236 (74.7%) children with communication disorders. Seizures with a known cause are called symptomatic seizures. The cause could be brain damage from a loss of oxygen or trauma during pre/ perinatal period, a trauma to the head, an infection of the brain such as meningitis, or a brain tumor etc., developmental anomalies and neurological abnormalities.

12. EEG findings

EEG findings was reported to be normal in 51 (29%), abnormal in 56 (31.8%) and the remaining parents did not report on EEG findings. The details regarding EGG abnormalities were not mentioned in the questionnaire. EEG is a cost effective procedure and in the country like India everyone cannot afford EGG evaluation. Following table 17 depict EEG patterns as reported by parents. It was found that only >40% of children with HL had normal EEG pattern and others had lesser than 40%. Abnormal EEG pattern was found in >40% of children with BI, DD, ADHD and MD suggestive of neurological involvement.

Table 17. *EEG findings in CWCDs*

Disorders	Total children	Normal EEG (%)	Abnormal EEG (%)	Not Sure (%)
BI	6	1 (16.7)	4 (66.7)	1 (16.7)
Speech regression	1	-	-	1 (100)
DD	4	1 (25)	2 (50)	1 (25)
MR	72	22 (30.6)	19 (26.4)	30 (41.7)
PDD	3	1 (33.3)	1 (33.3)	1 (33.3)
ADHD	2	-	1 (50)	1 (50)
CP	18	6 (33.3)	5 (27.8)	7 (38.9)
DSL	24	5 (20.8)	8 (33.3)	11 (45.8)
HL	17	8 (47.1)	2 (11.8)	7 (41.2)
Multiple	29	7 (24)	14 (48)	8 (28)
Total	176	51 (29)	56 (31.8)	68 (38.6)

(Note: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Hearing loss (HL), Mental retardation (MR), Mutiple- HLCP, MRHL, MRCP, HLCPMR).

Recent studies focusing exclusively on children have shown that the risk vary with the type of seizure and the presence or absence of neurologic and electroencephalographic (EEG) abnormalities (Berg & Shinnar, 1991; Shinnar et al., 1990; Boullovche et al., 1989). EEG findings if abnormal then a variety of epileptiform discharges such as multifocal discharges, focal discharges with temporal lobe predominance, hypsarrhythmia, and generalized spike and wave discharges is present.

The prognosis in terms of seizure management is generally poor, and the epileptic disorder tends to remain active for many years in spite of medications.

Rose and Lombroso (1970) followed up 137 FT newborns for an average of 4 years. At the conclusion of the study, 51.8% of the patients were normal, 19.7% died infants with seizures and a normal EEG had an 86% chance of normal development, whereas the prognoses for those with abnormal EEGs was much less.

The relationship between autism, epileptiform EEG, seizures, and language regression is complex and only partially understood. In a study in which 60% of an autistic population underwent EEG, about 22% had epileptiform abnormalities. In approximately one half of the children in whom EEG demonstrated epileptiform discharges, the discharges were located over the centrotemporal region, regardless of whether the child was epileptic or had regression. Two explanations are possible: One is that patients have comorbidity of benign (rolandic) seizures with centrotemporal spike-EEG trait with autistic symptoms (eg, benign seizures with centrotemporal spikes, one of the most prevalent epileptic syndromes). The second explanation tries to attribute a cause-effect relationship between the epileptiform abnormalities and the autistic and language regression symptoms. The subgroup of patients with autism, language regression, and epileptiform EEGs has been described as having autistic epileptiform regression. Patients in this group also have regression of their social, nonverbal, and cognitive skills, but determining whether development was completely normal before the regression is often difficult. All of the studies were biased because of the lack of universal EEG performance in patients with autism or PDD or the lack of prolonged-sleep tracing in patients with PDD. Tuchman and Rapin (2002) also reported 42 clients having autism associated with seizures, of which 75% of the clients had abnormal EEG and 8 % of clients having autism had abnormal EEGs even without the presence of seizures. However, in the present study out of 4 children with autism one had normal EGG, one had abnormal EGG and the remaining 2 had no EGG reports.

Children with focal spikes in the language area (left temporal hemisphere) had significantly more language problems compared to others. Children with focal spikes in

the visual cortex (occipital region) had difficulties in complex visual transformation tasks, i. e construction of triangles, and arrangement of photo series. Hence, these finding indicate that focal spikes in the EEG may interfere with complex cognitive functions. Treatment strategy should not only concentrate on the overt seizures, but also on the spike activity in the EEG in order to prevent selective deficits (Markus et al., 1994). Billard, Fluss, and Pinton (2009) reported the occurrence of sleep electroencephalography (EEG) abnormalities in some children with specific language impairment (SLI) and Landau-Kleffner syndrome (LKS). It indicated a large spectrum of interactions between language and seizures. However, in the present study 24 children had delay in speech and language. Among them 5 had normal EGG and 8 had abnormal EGG. Reports on EGG were not mentioned in one client with speech regression.

Recurrence was seen in 87 (52%) of 168 children after an average of 32 months in one series. The risk was highest for patients with sylvian (rolandic) seizures (70% when the EEG was normal and 97% when it was abnormal) and lowest for patients with generalized tonic clonic seizures (30% when no EEG anomalies were found and 63% when both neurologic and EEG abnormalities were present (Camfield et al., 1985b). These findings suggest that the children considered in the present study with abnormal EGG are at risk of recurrence.

In practice, the repertoire of EEG discharges that electroencephalogra-phers and clinicians recognize as epileptic is limited (Blume, 1982). Theoretically, such discharges are a constant concomitant of epileptic seizures even though they are not necessarily detectable by conventional techniques of EEG recording in every case. Subjects who do not demonstrate any clinical evidence of paroxysmal brain dysfunction yet who have generalized EEG paroxysmal abnormalities showing the typical EEG characteristics are not uncommon (Olofsson, Petersen, & Sellden, 1971). These findings help us to conclude that children with normal EGG and presence of seizures require careful assessment and monitoring.

The present study revealed EEG findings to be normal in 51 children (29%) and abnormal in 56 children (31.8%) and the remaining parents did not report on EEG findings. Recent studies suggest the presence of electroencephalographic (EEG) abnormalities indicate the risk factor and poor seizure management in spite of medications. Focal spikes in the EEG may interfere with complex cognitive functions. Recurrence of seizures has been found in both the presence and absence of EEG abnormalities. However, the risk is highest for clients with abnormal EEG. Also, the clients with positive clinical symptoms and normal EEG should not be neglected. Treatment strategy should not only concentrate on the overt seizures, but also on the spike activity in the EEG in order to prevent selective deficits.

14. Type of treatment undertaken

1. First aid

During the attack of epileptic seizures first aid treatment were provided by 60% of the parents. Most of them 37 (17.3%) reported facing the child sideways and only 2 (0.93%) parents reported loosening clothes around neck.

Combination of this first aid treatment was reported in 64 children (30%). Few parents 81 (38%) reported none of the above mentioned first aid treatment. Table 18 depicts the first aid treatment provided during the attack of epileptic seizures.

The appropriate behavior for helping someone who has a seizure depends on the type of seizure. While a person experiencing a tonic-clonic seizure may require some first aid, in most cases there is little that can be done. Tonic-Clonic (Grand Mal) type of seizure is often the most dramatic and frightening, but it is important to realize that a person undergoing an epileptic seizure is usually unconscious and feels no pain. The seizure usually lasts only a few minutes, and the person does not need medical care. Researchers suggest simple procedures to be followed during the seizure. They include 1) to keep calm as one cannot stop a seizure once it has started, 2) to ease the person to the floor and loosen the tight clothing, 3) to remove any hard, sharp, or hot objects that might injure the person, 4) not to put anything in the person's mouth, 5) as the person

relaxes after a fit, to turn him gently onto his stomach with the face to one side and the head extended so that the saliva can flow from the mouth. Its important to make sure that he can breathe freely, 6) to contact the parent or guardian, 7) if the person undergoes a series of convulsions, with each successive one occurring before he or she has fully recovered consciousness, or a single seizure lasting longer than 10 minutes, one should immediately seek medical assistance. It is also suggested for people with stereotyped recurrent severe seizures that can be easily recognized by the person's family, the drug diazepam is now available as a gel that can be administered rectally by a family member (Schachter, 2004).

Table 18. *First aid treatment*

Disorders	Total	a (%)	b (%)	c (%)	d (%)	e (%)	f (%)	g (%)
BI	7	1 (14.3)	-	1 (14.3)	-	-	3 (42.8)	2 (28.6)
Speech regression	1	-	-	-	-	-	1 (100)	-
DD	3	-	1 (25)	-	-	-	-	2 (25)
MR	90	19 (21.1)	2 (2.2)	12 (13.3)	4 (4.4)	-	33 (36.6)	20 (22.2)
PDD	3	1 (33.3)	-	1 (33.3)	-	-	1 (33.3)	1 (33.3)
ADHD	4	1 (25)	-	1 (25)	-	-	1 (25)	1 (25)
CP	19	4 (21.1)	-	3 (15.8)	-	1 (5.3)	4 (21.1)	7 (36.8)
DSL	28	4 (14.3)	2 (7.1)	3 (10.7)	2 (7.1)	-	6 (21.4)	28 (39.3)
HL	15	3 (20)	1 (6.7)	1 (6.7)	1 (6.7)	-	3 (20)	6 (40)
Multiple Disorders	43	4 (9.3)	1 (2.3)	5 (11.6)	3 (6.9)	1 (2.3)	12 (27.9)	17 (39.5)
Total	213	37 (17.3)	7 (3.2)	27 (12.6)	10 (4.69)	2 (0.93)	64 (30)	81 (38)

(Note: On X-axis: a- Facing the child sideways, b- Insertion of diazepam through rectum, c- Wiping froth coming out from the mouth, d- Avoid potential dangers, e- Loosen clothing around neck, f- Combination, g- None. On Y axis: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Hearing loss (HL), Mental retardation (MR), Mutiple-HLCP, MRHL, MRCP, HLCPMR).

The results of the present study revealed that during the attack of epileptic seizures first aid treatment were provided by 60% of the parents and few parents 81

(38%) reported none of the above mentioned first aid treatment. Majority of the parents were aware of only few of the first aid measures. This suggests that they should be provided with adequate information regarding measures to be taken up during seizure. By definition, Seizures is an enduring condition, or rather a group of conditions, in which epileptic seizures occur repeatedly without a detectable extracerebral cause (Gastaut, 1973). The condition thus gets repeated in majority of the clients, hence first aid treatment should be known by the immediate family members, friends, teachers and public. This will further reduce the ill effects of the epileptic seizure. Prolonged seizures can be avoided with an appropriate treatment and further could cause less damage to the brain.

2. **Medication**

The aim of medical treatment is to control child's tendency to have seizures, so they can get on with life with as little disruption from seizures as possible. Avoiding the things which may trigger seizures and taking anti-epileptic drugs are the main ways to achieve seizure control. A child will usually need to stay on anti-epileptic drugs until they have been free from seizures for at least two years and in certain cases they may need to continue taking anti-epileptic drugs indefinitely.

a). Antiepileptic drugs

In the present study the antiepileptic drug used maximally by most of the children was sodium valproate & valproic acid 52 (21.9%) and limited children was on ethosuximide 2 (0.84%). Various combinations of antiepileptic drugs were used by 104 (43.8%) children. Table 19 depicts the various antiepileptic drugs administered to control seizures.

Epileptic attacks should be suppressed for several reasons. The only effective means of treating seizures currently available are medication. A more systematic search for new anti-epileptic drugs is now under way, based on research progress in understanding how neurons transmit impulses to each other and increasing knowledge of the structure and function of the membrane which surrounds each neuron. The development of newer medications, especially Tegretol (Ciba-Geigy) and Epilim

(Reckitt & Colman) has meant that seizures can be suppressed in most patients without serious or annoying side effects. Seizures must be prevented using anti-epileptic drugs. The properties of drugs influence in treating seizures are as follows.

Table 19. *Antiepileptic drugs*

Disorders	Total children	a (%)	b (%)	b (%)	d (%)	e (%)	f (%)	g (%)	h (%)
BI	10	1 (10)	3 (30)	-	3 (30)	-	-	-	3 (30)
Speech regression	1	-	-	-	-	-	-	-	1 (100)
DD	6	-	2 (33.3)	1 (16.8)	-	-	-	1 (16.8)	2 (33.3)
MR	88	2 (2.3)	20 (22.7)	9 (10.2)	13 (14.8)	-	3 (3.4)	1 (1.1)	40 (45.4)
PDD	4	1 (25)	-	-	-	-	-	1 (25)	2 (50)
ADHD	4	-	-	2 (66.7)	1 (33.3)	-	-	-	1 (33.3)
MA	1	-	-	-	1 (100)	-	-	-	-
LD	1	-	-	1 (100)	-	-	-	-	-
CP	20	-	5 (25)	-	6 (30)	-	1 (5)	1 (5)	7 (35)
DSL	36	3 (88.3)	4 (11.1)	1 (2.8)	10 (27.8)	2 (5.6)	-	3 (8.3)	13 (36.1)
HL	18	1 (5.6)	4 (22.2)	-	5 (27.8)	-	-	1 (5.6)	7 (38.8)
Multiple	48	1 (2)	9 (18.7)	5 (10.4)	13 (27)	-	2 (4.1)	-	18 (37.5)
Total	237	9 (3.79)	47 (19.83)	19 (8.01)	52 (21.9)	2 (0.84)	6 (2.53)	8 (3.37)	104 (43.8)

(Note: On X-axis: a- Phenytoin, b- Phenobarbitone, c- Carbamazepine, d- Sodium valproate & valproic acid, e- Ethosuximide, f- Lamotrigine, g- Benzodiazepine, h- Combination. On Y axis: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Learning Disability (LD), Hearing loss (HL), Mental retardation (MR), Misarticulation (MA), Multiple- HLCP, MRHL, CPMR, HLCPMR).

The messages that one neuron sends to the next, meditated by releasing neurotransmitter chemicals, can either excite the neuron next in line, or can inhibit its electrical activity. The choice of drug depends on the type of seizures. Some drugs such as Epilim are active in a wide range of seizures (tonic-clonic, absence attacks, etc.) while Zarontin, for example, is active only against absence seizures (Ochoa & Riche, 2009).

White (2002) carried out a long-term double blind study of the relative efficacy of Primidone, Phenobarbital sodium and diphenylhydantion in preventing seizures but were unable to demonstrate any clear difference, though there was a suggestion that diphenylhydation was superior. Kramer (2008) attempted to describe the clinical spectrum and to evaluate the efficacy of different therapeutic agents in children with electrical status epilepticus in sleep (ESES). The antiepileptic drugs that were found to be efficacious were levetiracetam (41%), clobazam (31%) and sulthiame (17%). Valproic acid, lamotrigine, topiramate, and ethosuximide showed no efficacy. Steriods were efficacious in 65%: immunoglobulins were efficacious in 33%. High-dose diazepam was efficacious in 37%, but all the children had temporary response. Seventeen patients (57%) had cognitive deterioration, whereas the rest presented with regression in attention, speech, communication, and behavior. Fourteen children had permanent cognitive deficit. There was a significant correlation ($p=0.029$) between the duration of ESES and residual intellectual deficit at follow-up.

Martino and Tuchman (2001) reviewed the data on the current use of antiepileptic drugs in the treatment of autism, and on the association of affective disorders with seizures and autism. They concluded that although the role of antiepileptic drugs at the present time is not established, there is evidence that autism, seizures, and affective disorders commonly co-occur, and that they may share a common neurochemical substrate, which is the common target of the psychotropic mechanism of action of different antiepileptic drugs.

Summarizing the literature on medication various AEDs have been tried to control seizures. In the present study the antiepileptic drug used maximally by most of the children was sodium valproate & valproic acid and minimum usage was the drug ethosuximide. The choice of drug depends on the type of seizures and its efficacy in controlling seizures. Researchers recommend sodium valproate & valproic acid as the first drug to control seizures with less side effects. Sodium valproate is extremely useful drug with a wide range of anti-epileptic activity. It is thought to act by increasing the brain's levels of the inhibitory neurotransmitter, GABA. It is in practice a widely used, effective, and well tolerated medication. Some drugs are used as an additional treatment

in patients with partial seizures with or without secondary generalization, where seizures have not been controlled by other anticonvulsant drugs. When a person starts a new seizures drug, it is important to tailor the dosage to achieve the best results. People's bodies react to medications in very different and sometimes unpredictable ways, so it may take some time to find the right drug at the right dose to provide optimal control of seizures while minimizing side effects.

b). No. of drugs administered

Antiepileptic drug was administered singly in 104 children (32.9%), two medicines in 48 children (15.2%) and more than two drugs in 46 children (14.6%). However, 70 children were not on medication probably indicative of seizure free condition. Figure 4 depicts the graphical presentation of antiepileptic drugs taken up by children. It is generally preferable to take one drug only, since there is a risk of interaction between anti-epileptic drugs. Indeed, some neurologists go as far as saying that any patient taking more than one such drug is being mismanaged. There are some patients who, despite excellent blood levels of their medication, continue to have frequent seizures. The addition of a second drug may bring control which any single drug has failed to achieve. On the other hand, one can't often justify the use of a third and even a fourth drug (Meldrum, 1996). The present study suggests that majority of children used single drugs which indicates good prognosis and more than two drugs indicated less prognosis.

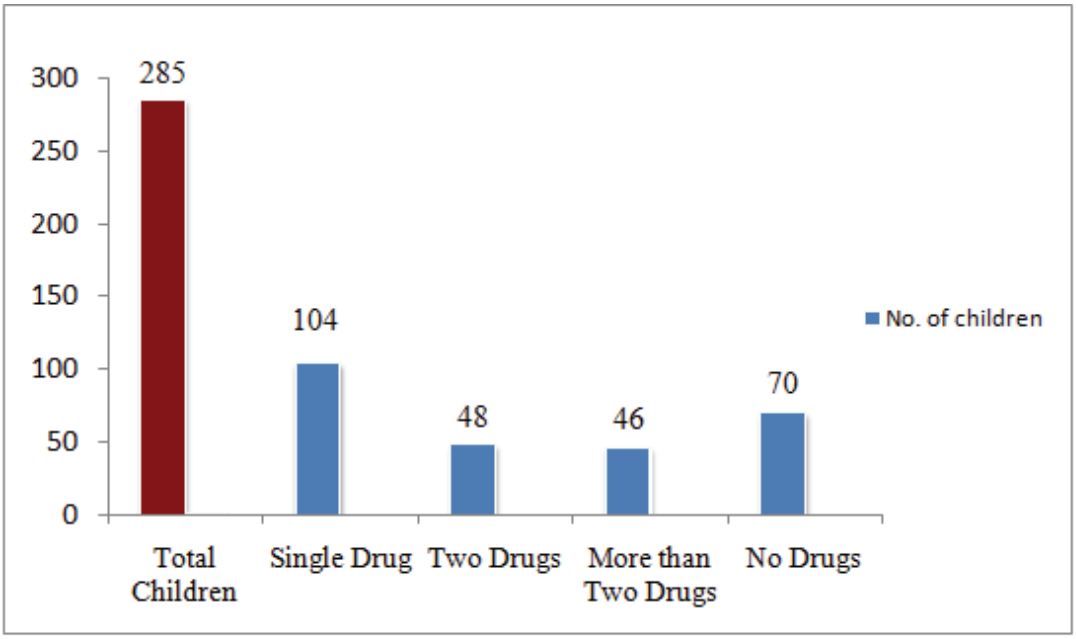

Figure 4. *No. of antiepileptic drugs*

The dosage and administration of drugs was in the morning in 63 children (11.4%), afternoon in 15 children (4.7%) and evening in 72 children (22.8%). Combination dosages were prescribed in 56 children (30.3%). A drug that has no effect or very bad side effects at one dose may work very well at another dose. Doctors will usually prescribe a low dose of the new drug initially and monitor blood levels of the drug to determine when the best possible dose has been reached. The metabolism of anticonvulsant medication also depends on the patient's age. The absorption, protein binding, clearance, and apparent half-life of antiepileptic drugs vary at different periods of life. In general, neonates metabolize anticonvulsant drugs slowly, and they become easily intoxicated. After a few weeks, the rate of metabolism of most drugs increases such that optimum blood levels are difficult to attain, even with the use of large doses. Thereafter, the dose requirements progressively diminish as the metabolism of drugs slowly becomes less rapid. These changes are a caution for the clients on anticonvulsant medication (Rho & Sankar, 1999). The present findings suggest that majority of children were taking greater dosage for seizure control.

c). Duration of administration

The duration of administration of drugs was <1 month in 28 children (8.9%), 1-3 months in 15 children (4.7%), 3-6 months in 20 children (6.3%) and greater than 6 months in 173 children (54.7%) as mentioned in the below figure 5. Majority of children were recommended to continue medication as seizures were not completely under control.

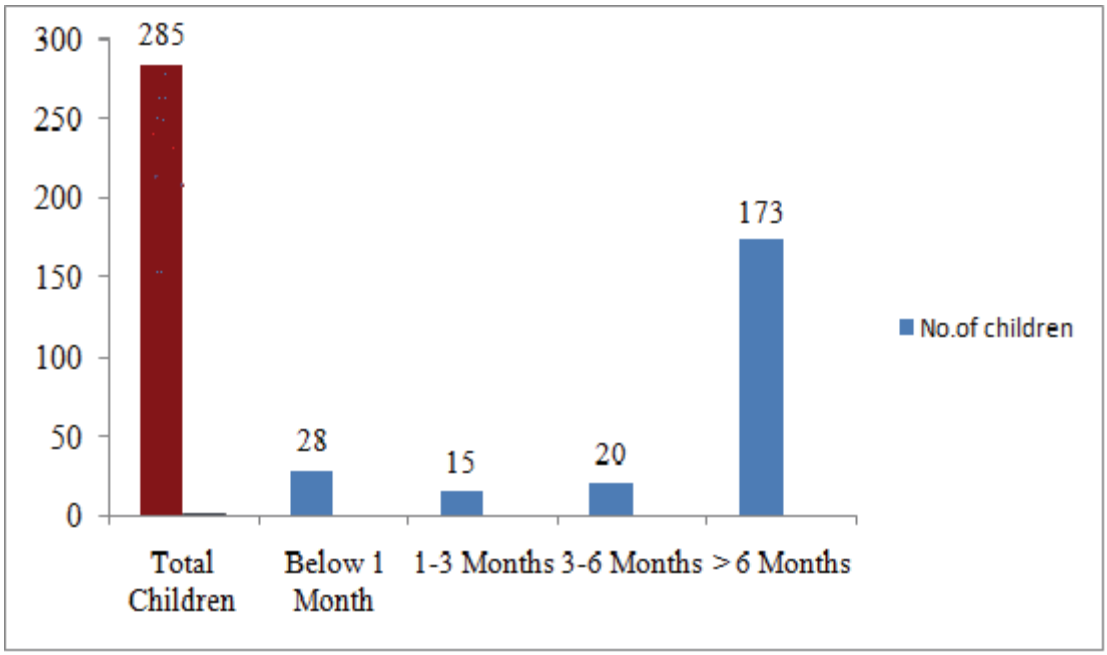

Figure 5. *Duration of administration of drugs*

Wilson (1970) recommended that although anticonvulsant medication can usually be discontinued after two or three seizure-free years, children with seizures should be urged to continue taking the pills into the late teens because of the tendency for seizure control to relapse during adolescence. He also suggests that parents need counseling about the drugs. They should be warned that success with drug therapy may not be immediate and that changes in treatment may be necessary without predicting a poor prognosis.

d). Side effects

The side effects reported by parents after administration of drugs was Teeth grinding and reduced sitting tolerance in majority of children. Various combination of

the side effects were reported by parents in 59 (36.6%) children. Table 20 depicts the various side effects reported by parents.

Table 20. *Side effects after administration of drugs*

Disorders	Total children	a (%)	b (%)	c (%)	d (%)	e (%)	f (%)	Combination
BI	4	1 (25)	-	-	-	1 (25)	-	2 (50)
Speech regression	1	-	-	-	-	-	-	1 (100)
DD	4	-	1 (25)	-	-	3 (75)	-	-
MR	75	6 (8)	17 (22.7)	4 (5.3)	2 (2.7)	5 (6.7)	15 (20)	26 (34.6)
PDD	3	1 (33.3)	-	-	-	-	1 (33.3)	1 (33.3)
ADHD	4	1 (25)	-	-	-	-	1 (25)	2 (50)
LD	1	-	-	-	-	-	1 (100)	-
CP	14	1 (7.1)	1 (7.1)	-	1 (7.1)	3 (21.4)	3 (21.4)	5 (35.7)
DSL	17	2 (11.8)	2 (11.8)	-	2 (11.8)	4 (23.4)	4 (23.4)	3 (17.6)
HL	9	2 (22.2)	1 (11.1)	-	-	-	1 (11.1)	5 (55.5)
Multiple	29	2 (6.8)	7 (24.1)	2 (6.8)	1 (3.4)	-	3 (10.3)	14 (48.2)
Total	161	16 (9.93)	29 (18)	6 (3.72)	6 (3.72)	16 (9.93)	29 (18)	59 (36.64)

(Note: On X axis: a- Screaming behavior, b- Teeth grinding, c- Self injurious behavior, d- Hyperactivity, e- Attention deficit, f- Reduced sitting tolerance, g- Combination). On Y axis: Border line Intelligence (BI), Pervasive Developmental Disorder (PDD), Attention Deficit Hyperactivity disorder (ADHD), Cerebral palsy (CP), Delayed Speech and Language (DSL), Development Delay (DD), Learning Disability (LD), Hearing loss (HL), Mental retardation (MR), Misarticulation (MA), Multiple- HLCP, MRHL, CPMR, HLCPMR).

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The effect of antiepileptic drugs is double edged in this setting. They may reduce the seizure burden and thus improve the cognitive function. However, these drugs also significantly affect the learning process. The treating pediatrician should be equipped to comprehensively address all these factors for an optimal outcome. Recent onset of educational problems in a child with seizures deserves immediate and aggressive evaluation and management (Vinayan, 2006).

All the effective drugs used as anticonvulsants are toxic in overdose. It is now generally agreed that are drawbacks to the use of phenobarbital sodium in that it may produce both irritation and impaired cerebration. There is an increasingly wide range of drugs available, among which may be mentioned intravenous diazepam (Valium) which may be alternative to the use of intramuscular paraldehyde in the control of status epilepticus. The new survey on quality of life discovered that 65 per cent of people with refractory seizures have tried between two and five medications, while an additional one-quarter of patients have tried between six and ten. Moreover, half currently experience side effects associated with their medications and find these side effects extremely frustrating. Managing seizures has to be a balance between controlling seizures and the number of medications each person is taking and their side effects. Three-quarters of the patients surveyed would be very interested in learning about new treatment options and finding ways to reduce the number of medications they take, according to this survey. Furthermore, approximately 80 per cent of the survey participants would consider changing their current treatment if their doctor told them about a new treatment that might maintain their current level of seizure control without the negative side effects (Martino, & Tuchman, 2001). Torres (1969) warns that anticonvulsive drug treatment should be used only when a diagnosis of seizures has been established, because the drugs may have some toxic effect, and that effects of their use should be checked regularly because hypersensitivity may develop, as well as side effects such as anemia may develop.

Szucs, Clemens, and Jakus (2008) found the risk of paradoxical levetiracetam effect to be increased in mentally retarded patients. A paradoxical effect was defined as an increased seizure frequency or the experience of more severe seizures including generalized tonic-clonic seizures (GTCS) within 1 month after starting levetiracetam (LEV). It was found that the paradoxical effect developed preferentially ($p< 0.001$) in mentally retarded patients. There was an increased risk of worsening seizures with levetiracetam treatment in epileptic patients with mentally retarded. Hence, there is a need for caution and close observation during the first weeks of therapy. The results of the present study are in agreement with earlier findings. Side effects were found in 161 children and more than one type of side effect was found in majority of the children.

Presnce of side effects in children with communication disorders could be due to multi factorial causes. The reasons could be prsence of neurological deficit, psycological problems and the effects of AEDs. But, there is a need for caution to cancould on the role of contributing factors with respect to side effects.

15). Improvement in language function

There was improvement in language function after treatment in 115 children (%) as reported by parents. However, improvement was not noticed in 29 children (57%) in the language function. Figure 6 depicts the parental opinion regarding improvement in language function in their children.

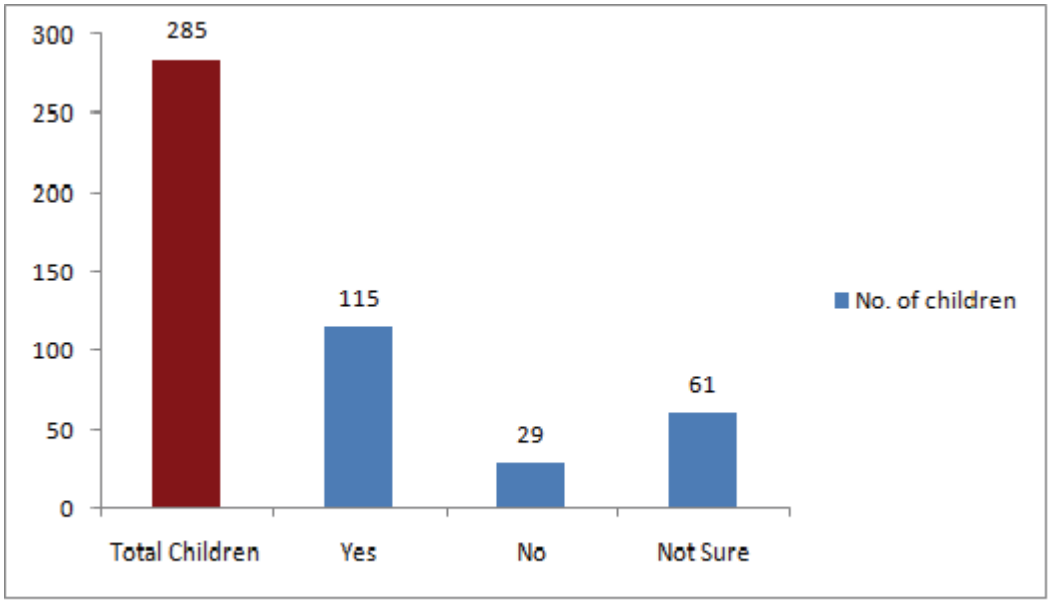

Figure 6. *Improvement in language function*

Epilepsies with onset in infancy and early childhood often have a severe course. The epileptic disorder is often severe and resistant to the treatments available, with a very low remission rate. Multiple seizure types, mental retardation, multifocal abnormal EEGs, and many cortical tubers are poor prognostic indicators. Overall, a combination of favorable factors, especially the absence of brain damage, normal intelligence and neurologic examinations, and a rapid response to therapy, predicts an absence of seizures in the long term with a probability of 90% to 95% (Sillanpaa, 2000).

The risk for relapses is higher in patients with a structural brain lesion, abnormal neurologic signs, learning difficulties, a long history of seizures before remission, the occurrence of more than one seizure type, and some seizures syndromes. The relapse rate tends to be lower in children than in adults (Sander, 1993). Most relapses usually are controlled by the relative risk being 1.45 (Shinnar et al., 1994). The prognosis may require to be estimated at the time of first seizure, the time of first diagnoses of seizures, the time of achieving seizure control an attempt at medication withdrawal agree seizure control (Sridharan, 2002).

The standardized mortality rates compared with the general population on an age adjusted bases in seizures are 2 to 4 times higher than normal age. It is highest in the first 10 years. Factor associate with a higher mortality include male gender and seizures symptomatic of diffuse or focal cerebral diseases. Much of excess mortality is related to the underlying causes of the seizures. The mortality is more with generalized tonic clonic seizures but not in those with absence seizures (Sridharan, 2002). All the clinical and animal data support the caution that clinicians should make every attempt to diagnose quickly and aggressively to treat epileptic seizures. Delays in the recognition of infantile spasms or the use of inappropriate therapies may be very detrimental to the child.

Summarizing the results of the present study improvement in language function was reported in 115 children (40.3%). Prognosis could be seen due to early treatment, appropriate medication and regular speech and language therapy. However, improvement was not noticed in 29 children (10.1%) in the language function. This may be attributed to the condition with major structural involvement leading to associated problems.

Seizures are one of the world's oldest recognized conditions. Medical and research advances in the past two decades have led to a better understanding of seizures and seizures than ever before. Advanced brain scans and other techniques allow greater accuracy in diagnosing seizures and determining when a patient may be helped by surgery. More than 20 different medications and a variety of surgical techniques are

now available and provide good control of seizures for most people with seizures. Research on the underlying causes of seizures, including identification of genes for some forms of seizures and febrile seizures, has led to a greatly improved understanding of seizures that may lead to more effective treatments or even new ways of preventing seizures in the future.

Research has led to many advances in understanding and treating seizures but still there are many unanswered questions about how and why seizures develop, how they can best be treated or prevented, and how they influence other brain activity and brain development in children with communication disorders. The valuable information may allow the professionals to prevent seizures or to predict most beneficial intervention stratergy.

The primary aim of this project was to determine the prevalence of seizures in CWCDs in an Indian context. It also aimed to determine the nature of seizures such as age of onset (both seizures and communication disorder), causes, type of seizure, frequency of occurrence, associated problems, co-morbidity of seizures and the nature of communication disorder. Results of the present study indicated that epileptic seizures were one of the most common neurological disorders occurring in children with communication disorders. The present findings will help the professionals for better understanding of seizure disorder in a subgroup of children with communication disorder.

A high-risk register (appendix 2) for CWCDs with seizures was developed as a clinical utilization measure after the present study. It has been proposed based on the various characteristic features of seizures found in the present study and also summarizing the review of literature. The proposed high-risk register will help to identify the children at risk for seizures and its recurrence. It would also guide the persons in determining the possible causative factor, early identification, to determine the effects of seizure and also to prevent its ill effects.

Prevention is inhibiting or interrupting the progression of disease, disorder or disability. It is even more essential in children with communication disorders with

reference to occurrence to seizures. Based on the review of literature and the current study tips for prevention and control of seizures and communication disorder in high risk children have been proposed (appendix 3). It would guide the persons in all the three stages of prevention, i.e primary, secondary and tertiary prevention.

The present trend is for the clinician dealing with communication disorders is to work as a member of a multidisciplinary team including the neurologist, pediatrician, psychologist, special educator, social worker, and others. It further suggests at educating the parents of children with seizures about the condition and its management.

SUMMARY AND CONCLUSIONS

Epilepsy is a physical condition that occurs when there is a sudden, brief change in how the brain works. These physical changes are called epileptic seizures. Epilepsy is therefore sometimes called a seizure disorder. Seizures affect people in all nations and of all races. Epilepsy consists of various types of clinical seizures and epileptic syndromes. The appearance rate or distribution of various types of epilepsies and epileptic syndromes in various subgroups of communication disorders has not been explored much and more so in the Indian context. It would be interesting to know the characteristic features of seizure disorders in different clinical populations in order to take suitable preventive measures and to provide rehabilitation. Since there is only limited data about the prevalence of seizures in CWCDs, future epidemiological research needs to be addressed. Hence the present study was planned.

The primary aim of this project was to examine the prevalence of seizures in CWCDs in the Indian context. It also aimed to examine the nature of seizures such as age of onset (both seizures and communication disorder), causes, type of seizure, frequency of occurrence, associated problems, co-morbidity of seizures and the nature of communication disorder. Yet another aim was to develop a high-risk register for children with seizures as a clinical utilization measure after the study. The present study was conducted in three phases. Phase 1 included reviewing of case files of children below the age of 12 years who visited AIISH for the presence or absence of seizures. In Phase 2 a questionnaire was developed to get further information on the variables related to seizures. In Phase 3 children with positive history of seizures were followed up for further analysis. The developed questionnaire was administered on children who were attending therapy and diagnostics at AIISH during the of two years period (Jan 2007- Dec 2008).

The results of the present study are as follows:

- Epileptic seizures were one of the most common neurological disorders occurring in children with communication disorders.

- The percentage of seizures ranged from as high as 73.53% in children with mental retardation to as low as 0.09% in stuttering group.
- The prevalence rate of seizures was more in children with brain damage as in borderline intelligence, cerebral palsy, mental retardation, developmental delay and pervasive developmental disorders.
- Majority of the CWCDs had first attack of seizures as early as 1-12 months, maximum frequency of occurrence of seizure as >3 times per week, negative family history of seizures, seizure attack for <5 minutes, afebrile seizures, no such triggering factors, exhibited involuntary bilateral and symmetrical muscle contractions during the seizure in children with communication disorders.
- Majority of the children presented with secondary generalized seizures compared to primary generalized seizures and idiopathic seizures.
- Most of the parents reported speech, language, motor and cognitive skills as not normal since childhood and seizures may not be the only cause of their child's condition.
- The associated problems found in children were drooling, behavioral problems, teeth grinding and combination of these problems.
- 50% of CWCDs had seizures completely under control.
- Various antiepileptic drugs was administered singly, two or more at different dosages.
- Though seizures were under control, children did present with subtype of communication disorder. It suggests that the problems identified in any of the three domains behavioral, cognitive, and neurologic were more influential than were problems related to seizures.
- There was improvement reported by parents in language function in most of their children.

The results of the present study reveal that the clinical manifestation of seizures in CWCDs depend on the type of damage to the brain, area of the brain in which epileptic activity is occurring, severity of seizures, type of seizure, sub-clinical seizure activity, duration of seizures, anti-epileptic medication and the associated problems. Any type of seizure should be prevented with an appropriate treatment or it would further cause more damage to the brain.

Clinical implications of the study

- The present study is a first attempt to determine the prevalence of epileptic seizures in subgroup of CWCDs with reference to Indian context.
- It will help the professionals for better understanding of seizure disorder, (prevalence and nature) in a subgroup of children with communication disorder.
- It aims at educating the parents of children with seizures about the condition and its management by using the proposed high risk register as the project outcome.
- It further suggests the clinician dealing with communication disorders to work as a member of a multidisciplinary team including the neurologist, pediatrician, psychologist, special educator, social worker and others.

Limitations of the study

- Though various subgroups of CWCDs were considered, number of children in each group was limited.
- Out of 595, the filled up questionnaires were received by only 181 parents.
- The present study was a retrospective analysis, rather if the information was collected directly from the persons more qualitative and quantitative information would be obtained.
- The questionnaire requires further simplication of the terms.
- Information regarding detailed neurological evaluation was not available in majority of the children.

Recommendations for future research

- A larger study in the Indian context in each subgroup of CWCDs would be important to validate the findings.
- Future studies are recommended with complete neurological investigations and the same objectives.
- A longitudinal study to investigate the long term effects of epileptic seizures in CWCDs.
- Reliability and Validity of the proposed high risk register
- Role of seizures in each subgroup of adults with communication disorders

APPENDIX 1

Questionnaire on Prevalence and nature of seizures in children with Communication Disorders

Client name: Age / Name:

Client No: P.D:

Characteristic Features:

1. When was the attack of seizures first noticed?

a) < 6 months; b) 2 months; c) 1-2 years; d) 2-4 years; e) > 4 years

2. a) What is the frequency of occurrence of seizures since the first attack?

a) 1; b) 1-2 weeks; c) 3-5 weeks; d) 6-10/weeks; e) > week

b) What is the frequency of occurrence of seizure in general?

a) 1; b) Occasional (2-3); c) 1 or 2 / week; d) > 3/week; e) 1 or 2/month

3. **Do you have a positive history of seizures in the family? If yes, what is the relationship with the client?**

a) 0- No; b) 1- Yes;

If yes, b1) 0- Far relative; b2) 1-Close relative

4. Does the client remain unconscious after an attack if yes, how long?

a) 0-No; b) 1-Yes;

If yes, b1) < 5 mins; b2) < 5 mins

5. What is the approximate duration of the seizure attack?

a) < 5 mins; b) 5-10 mins; c) 10-20 mins; d) 20-30 mins; e) > 30 mins

6) Does seizure occur only in the presence of fever?

a) No; b) Yes; c) Not always

7) When does attack of seizures occur?

a) During the day; b) During the night; c) Awake; d) On sleep; e) Any time

8) Whether the speech & language / motor / cognitive skills were normal till the onset of seizures?

a) no; b) Yes; c) Not sure;

9) Is seizure triggered of by any kind of specific stimulus?

a) No; b) Yes;

If yes, b1) Auditory; b2) Visual; b3) Others (specify)

10. What are the involuntary muscle contractions exhibited by the child during the seizure? (Tick all options that are appropriate)

a) Abnormal movements; b) Only one side of body; c) Symmetrical; d) Fairly large movements; e) Repetitive muscle contractions

11. During the attack what are the behaviors exhibited by the client? (Tonic- clonic seizure) (Tick all options that are appropriate)

a) Abnormal motor movements; b) Loss of consciousness; c) Abnormal eye movements; D) Froth in the mouth; e) Fall to the ground; f) Let out a yell; g) Eye blink repeatedly; h) Any other (specify)

12. What are the other characteristics of seizure noted in the child?(Lennox-Gestatut Syndrome)

a) Stare; b) Jerk; c) Fall; d) Sudden dropping of head (Atonic); e) Others (specify)

13. How does the client react before the attack of seizures (Aura)?

a) Tired; b) Stiff; c) Restless; d) Others (specify)

14. How does the client react after the attack of seizures? (Post ictal behavior)

a) Tired; b) Stiff; c) Restless; d) Others (specify)

15. What are the associated problems with seizures in your child?

a) Drooling; b) Motor weakness; c) Mental retardation; d) Behaviour problem; e) Teeth grinding; f) Speech and language deficit; g) Hearing problem; h) Visual impairment; i) Any other (specify)

16. Is the seizure under control now? If yes specify?

a) No; b) Yes;

If yes, b1) since 6 months; b2) 6-12 months; b3) 12 months – 2 years; b4) > 2 years

17. Causative factors:

A. Do you think seizures are the direct cause of language deficit?

a) No; b) Yes; c) Not sure

B. What do you think is the possible cause for seizures in your child?

a) Organic; b) Inflammatory; c) Circulatory; d) Degenerative; e) Metabolic; f) Toxic affliction; g) Not sure; h) Any other (specify)

18. Assessment

A. What is the type of seizures according to neurological findings?

a) Primary generalized seizures; b) Secondary generalized seizures; c) Simple Partial seizures; d) Complex partial seizures; e) Others; f) Not sure

OR

a) Grandmal; b) Petitmal; c) Myoclonic ; d) Idiopathic ; e) Clonic; f) Tonioc; g) Atonic; h) Any other specify

B. What are the EEG findings? (Electroencephalography)

a) Normal; b) Abnormal EEG (Specify); c) Not sure/applicable

19. Treatment

A. During the attack of seizures what is the first aid treatment provided? (Tick all options that are appropriate)

a) Lay the child sideways; b) Insertion of diazepam through rectum; c) Wiping froth coming out from mouth; d) Avoid potential dangers; e) Loosen clothing around neck; f) None of the above; g) Any other (Specify)

B. Which one of the following antiepileptic drug (Medication) is provided to your child?

a) Phenytion; b) Phenobarbitone; c) Carbamazepine; d) Sodium valporate and valproic acid; e) Ethosuximide; f) Primidone; g) Primidone; h) Lamotrigine; i) Benzodiazepine; j) Any other specify; k) None/Not applicable

C. What is the dosage and administration of drugs?

a) 1-0-0; b) 0-1-0; c) 0-0-1; d) Any other dosage (Specify)

D. Are the drugs administered singly or combined?

a) Only one drug; b) Two Drugs; c) More than two drugs; d) Not applicable

E. What is the duration of administration of drugs?

a) < 1 month; b) 1-3 months; c) 3-6 months; d) > 6 months

F. What are the side effects noticed in the client after administration of drugs?

a) Screaming behaviour; b) Teeth grinding; c) Self injurious behaviour; d) Hyper activity; e) Attention deficit; f) Reduced sitting tolerance

G. Whether the medication was successful in reducing the number or severity of seizures?

a) No; b) Slightly; c) Fairly well; d) Completely; e) Not applicable

H. Whether there was improvement in language function after treatment?

a) Yes; b) No

I. How is the present condition of the child?

a) Improving; b) Static; c) Deteriorating

APPENDIX 2

HIGH RISK REGISTER FOR CHILDREN WITH SEIZURE DISORDER

Instructions: Read each question carefully and answer the following as "Yes" or "No"

1) Does anyone in the family have history of seizures?

2) Does the mother have any prenatal (during pregnancy) complications?

3) Does the child get fever accompanying a routine illness under six most of the time?

4) Did the child suffer from brain infections like meningitis **(**inflammation of the protective membranes covering the brain and spinal cord), encephalitis **(**inflammation of the brain caused by a virus) , brain abscess **(**inflammation and collection of infected material within the brain tissue) , syphilis (sexually transmitted disease caused by the bacteria) etc?

5) Did the child suffer from other severe infections like tetanus (prolonged contraction of skeletal muscle fibers) , malaria (mosquito borne infectious disease), dysentery (severe diarrhoea) or any degeneration (deterioration) of brain tissue?

6) Did the child suffer from sudden lack of oxygen any time during childhood?

7) Did the child suffer from brain hemorrhage (bleeding) from birth injury or from blood disorders such as hemophilia (impaired ability to control blood clotting) or brain tumor (abnormal growth) any time during childhood?

8) Did the child have excessive exposure to poisons such as lead and camphor?

9) Does the child have sudden swelling of the brain as a result of water retention?

10) Does the child have chronic disorders of body chemistry, for e.g, low blood sugar, abnormal calcium metabolism, untreated phenylketonuria (genetic disorder characterized by a deficiency in the hepatic enzyme) and kidney failure?

11) Does the child present with hyperventilation **(**faster breathing), metabolic disorders (accumulation of substances which are toxic or interfere with normal function**),** sleep deprivation and so forth?

12) Does the child present with physical stress, emotional disturbances and so forth?

Note: If the answer to any of the above questions is “Yes”, a detailed neurological and language evaluation and a follow up with neurologist is recommended. Such children may be at risk for the occurrence of seizures.

APPENDIX 3

TIPS FOR PREVENTION AND CONTROL OF SEIZURES AND COMMUNICATION DISORDER IN HIGH RISK CHILDREN

Prevention is inhibiting or interrupting the progression of disease, disorder or disability. It is even more essential in children with communication disorders with reference to occurrence to seizures. Based on the review of literature and the current study following are the tips for prevention and control of seizures in children.

- The developing brain is susceptible to many kinds of injury. Injury to the brain may certainly cause seizures. This includes deprivation of oxygen at birth, trauma to the head at any time of life, stroke, maternal infections, poor nutrition etc. Such prenatal, perinatal and post natal causative factors should be immediately attended to by the consultant physician to avoid further consequences.
- The diagnosis of seizures most of the time depend on observation of occurrence of seizure. Hence keen observation by the parents is a must and they should provide details regarding the nature of seizure to the neurologist. This would suggest appropriate management in CWCDs.
- Delays in the recognition of seizures may be detrimental to the overall development in children. Hence early identification and management of the problem is a must.
- Epileptic attacks should be suppressed for several reasons. The only effective means of treating seizures currently available are medication. People's bodies react to medications in very different and sometimes unpredictable ways. It may take some time to find the right drug at the right dose to provide optimal control of seizures while minimizing side effects. Hence medications should be continued to achieve the best results. One should follow the treatment plan. **Taking too little or too much of medicine, abruptly stopping medicine, or changing medicine schedule can aggravate seizures.**

- Sometimes if seizure is not under control some drugs are used as an additional treatment. It is also important that the both the anticonvulsant drugs suggested are continued to prevent the occurrence of seizures.
- An increased duration and greater frequency of occurrence of seizure may suggest longer time to recover from the neurologic abnormalities. Hence there is a great need to prevent the longer duration and frequency of seizure. In case of stereotyped recurrent severe seizures, the drug diazepam, a gel can be administered rectally by a family member which would reduce the duration of occurrence of seizure. However, any medication should be strictly under the guidance of a physician.
- An electroencephalogram (EEG) should be obtained in all children in whom a seizure has occurred. EEG would help in predicting the risk of recurrence and to classify the seizure type and seizure syndrome. Abnormalities in EEG findings indicate a thorough seizure management to prevent further consequences.
- During the occurrence of seizures, few simple procedures must be followed to prevent further complications due to seizures. They include,

1) To keep calm as one cannot stop a seizure once it has started

2) To ease the person to the floor and loosen the tight clothing, if any

3) To remove any hard, sharp, or hot objects in close vicinity that might injure the person

4) Not to put anything in the person's mouth

5) As the person relaxes after a fit, to turn him gently onto his stomach with the face to one side and the head extended so that the saliva can flow from the mouth

6) It is important to make sure that he can breathe freely

7) To contact the parent or guardian

8) If the person undergoes a series of convulsions, with each successive one occurring before he or she has fully recovered consciousness, or a single seizure lasting longer than 10 minutes, one should immediately seek medical assistance.

- In terms of cognitive outcome, individuals with seizure onset after the age of 2.5 years have a better cognitive outcome than those whose seizures started before that age. Hence preventive measures should be taken up to avoid occurrence of seizures during early childhood.
- Activities that might trigger a seizure, such as playing video games that have flashing or flickering lights, continuous music etc should be avoided.
- A complete nutritional coverage of vitamins, minerals and amino acids (vitamin E, manganese, taurine, and other nutrients) that empower the nervous and immune system etc. is essential to control seizures in children.

Note: Prevention is always better than cure. These preventive measures should be considered to avoid further consequences due to seizures in children in general and CWCDs in particular.

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APPENDIX 1

Questionnaire on Prevalence and nature of seizures in children with Communication Disorders

Client name: Age / Name:

Client No: P.D:

Characteristic Features:

1. When was the attack of seizures first noticed?

a) < 6 months; b) 2 months; c) 1-2 years; d) 2-4 years; e) > 4 years

2. a) What is the frequency of occurrence of seizures since the first attack?

a) 1; b) 1-2 weeks; c) 3-5 weeks; d) 6-10/weeks; e) > week

b) What is the frequency of occurrence of seizure in general?

a) 1; b) Occasional (2-3); c) 1 or 2 / week; d) > 3/week; e) 1 or 2/month

3. **Do you have a positive history of seizures in the family? If yes, what is the relationship with the client?**

a) 0- No; b) 1- Yes;

If yes, b1) 0- Far relative; b2) 1-Close relative

4. Does the client remain unconscious after an attack if yes, how long?

a) 0-No; b) 1-Yes;

If yes, b1) < 5 mins; b2) < 5 mins

5. What is the approximate duration of the seizure attack?

a) < 5 mins; b) 5-10 mins; c) 10-20 mins; d) 20-30 mins; e) > 30 mins

6) Does seizure occur only in the presence of fever?

a) No; b) Yes; c) Not always

7) When does attack of seizures occur?

a) During the day; b) During the night; c) Awake; d) On sleep; e) Any time

8) Whether the speech & language / motor / cognitive skills were normal till the onset of seizures?

a) no; b) Yes; c) Not sure;

9) Is seizure triggered of by any kind of specific stimulus?

a) No; b) Yes;

If yes, b1) Auditory; b2) Visual; b3) Others (specify)

10. What are the involuntary muscle contractions exhibited by the child during the seizure? (Tick all options that are appropriate)

a) Abnormal movements; b) Only one side of body; c) Symmetrical; d) Fairly large movements; e) Repetitive muscle contractions

11. During the attack what are the behaviors exhibited by the client? (Tonic- clonic seizure) (Tick all options that are appropriate)

a) Abnormal motor movements; b) Loss of consciousness; c) Abnormal eye movements; D) Froth in the mouth; e) Fall to the ground; f) Let out a yell; g) Eye blink repeatedly; h) Any other (specify)

12. What are the other characteristics of seizure noted in the child?(Lennox-Gestatut Syndrome)

a) Stare; b) Jerk; c) Fall; d) Sudden dropping of head (Atonic); e) Others (specify)

13. How does the client react before the attack of seizures (Aura)?

a) Tired; b) Stiff; c) Restless; d) Others (specify)

14. How does the client react after the attack of seizures? (Post ictal behavior)

a) Tired; b) Stiff; c) Restless; d) Others (specify)

15. What are the associated problems with seizures in your child?

a) Drooling; b) Motor weakness; c) Mental retardation; d) Behaviour problem; e) Teeth grinding; f) Speech and language deficit; g) Hearing problem; h) Visual impairment; i) Any other (specify)

16. Is the seizure under control now? If yes specify?

a) No; b) Yes;

If yes, b1) since 6 months; b2) 6-12 months; b3) 12 months – 2 years; b4) > 2 years

17. Causative factors:

A. Do you think seizures are the direct cause of language deficit?

a) No; b) Yes; c) Not sure

B. What do you think is the possible cause for seizures in your child?

a) Organic; b) Inflammatory; c) Circulatory; d) Degenerative; e) Metabolic; f) Toxic affliction; g) Not sure; h) Any other (specify)

18. Assessment

A. What is the type of seizures according to neurological findings?

a) Primary generalized seizures; b) Secondary generalized seizures; c) Simple Partial seizures; d) Complex partial seizures; e) Others; f) Not sure (**OR)**

a) Grandmal; b) Petitmal; c) Myoclonic ; d) Idiopathic ; e) Clonic; f) Tonioc; g) Atonic; h) Any other specify

B. What are the EEG findings? (Electroencephalography)

a) Normal; b) Abnormal EEG (Specify); c) Not sure/applicable

19. Treatment

A. During the attack of seizures what is the first aid treatment provided? (Tick all options that are appropriate)

a) Lay the child sideways; b) Insertion of diazepam through rectum; c) Wiping froth coming out from mouth; d) Avoid potential dangers; e) Loosen clothing around neck; f) None of the above; g) Any other (Specify)

B. Which one of the following antiepileptic drug (Medication) is provided to your child?

a) Phenytion; b) Phenobarbitone; c) Carbamazepine; d) Sodium valporate and valproic acid; e) Ethosuximide; f) Primidone; g) Primidone; h) Lamotrigine; i) Benzodiazepine; j) Any other specify; k) None/Not applicable

C. What is the dosage and administration of drugs?

a) 1-0-0; b) 0-1-0; c) 0-0-1; d) Any other dosage (Specify)

D. Are the drugs administered singly or combined?

a) Only one drug; b) Two Drugs; c) More than two drugs; d) Not applicable

E. What is the duration of administration of drugs?

a) < 1 month; b) 1-3 months; c) 3-6 months; d) > 6 months

F. What are the side effects noticed in the client after administration of drugs?

a) Screaming behaviour; b) Teeth grinding; c) Self injurious behaviour; d) Hyper activity; e) Attention deficit; f) Reduced sitting tolerance

G. Whether the medication was successful in reducing the number or severity of seizures?

a) No; b) Slightly; c) Fairly well; d) Completely; e) Not applicable

H. Whether there was improvement in language function after treatment?

a) Yes; b) No

I. How is the present condition of the child?

a) Improving; b) Static; c) Deteriorating

APPENDIX 2

HIGH RISK REGISTER FOR CHILDREN WITH SEIZURE DISORDER

Yes/No

1) Does anyone in the family have history of seizures?

2) Does the mother have any prenatal complications?

3) Does the child get fever accompanying a routine illness under six most of the time?

4) Did the child suffer from brain infections like meningitis, encephalitis, brain abscess, syphilis etc?

5) Did the child suffer from other severe infections like tetanus, malaria, dysentery or any degeneration of brain tissue?

6) Did the child suffer from sudden lack of oxygen any time during childhood?

7) Did the child suffer from brain hemorrhage from birth injury or from blood disorders such as hemophilia or sickle cell disease or brain tumor any time during childhood?

8) Did the child have excessive exposure to poisons such as lead and camphor?

9) Does the child have sudden swelling of the brain as a result of water retention ?

10) Does the child have chronic disorders of body chemistry, for e.g, low blood sugar, abnormal calcium metabolism, untreated phenylketonuria and kidney failure?

11) Does the child present with hyperventilation, metabolic disorders, sleep deprivation and so forth?

12) Does the child present with physical stress, emotional disturbances and so forth?

Note: If the answer to any of the above questions is "Yes", a detailed neurological and language evaluation and a follow up with neurologist is recommended. Such children may be at risk for the occurrence of seizures.

APPENDIX 3

TIPS FOR PREVENTION AND CONTROL OF SEIZURES AND COMMUNICATION DISORDER IN HIGH RISK CHILDREN

Prevention is inhibiting or interrupting the progression of disease, disorder or disability. It is even more essential in children with communication disorders with reference to occurrence to seizures. Based on the review of literature and the current study following are the tips for prevention and control of seizures in children.

- The developing brain is susceptible to many kinds of injury. Injury to the brain may certainly cause seizures. This includes deprivation of oxygen at birth, trauma to the head at any time of life, stroke, maternal infections, poor nutrition etc. Such prenatal, perinatal and post natal causative factors should be immediately attended to by the consultant physician to avoid further consequences.
- The diagnosis of seizures most of the time depend on observation of occurrence of seizure. Hence keen observation by the parents is a must and they should provide details regarding the nature of seizure to the neurologist. This would suggest appropriate management in CWCDs.
- Delays in the recognition of seizures may be detrimental to the overall development in children. Hence early identification and management of the problem is a must.
- Epileptic attacks should be suppressed for several reasons. The only effective means of treating seizures currently available are medication. People's bodies react to medications in very different and sometimes unpredictable ways. It may take some time to find the right drug at the right dose to provide optimal control of seizures while minimizing side effects. Hence medications should be continued to achieve the best results. One should follow the treatment plan. **Taking too little or too much of medicine, abruptly stopping medicine, or changing medicine schedule can aggravate seizures.**
- Sometimes if seizure is not under control some drugs are used as an additional treatment. It is also important that the both the anticonvulsant drugs suggested are continued to prevent the occurrence of seizures.
- An increased duration and greater frequency of occurrence of seizure may suggest longer time to recover from the neurologic abnormalities. Hence there is a great need to prevent the longer duration and frequency of seizure. In case of stereotyped recurrent severe seizures, the drug diazepam, a gel can be administered rectally by a family

member which would reduce the duration of occurrence of seizure. However, any medication should be strictly under the guidance of a physician.

- An electroencephalogram (EEG) should be obtained in all children in whom a seizure has occurred. EEG would help in predicting the risk of recurrence and to classify the seizure type and seizure syndrome. Abnormalities in EEG findings indicate a thorough seizure management to prevent further consequences.
- During the occurrence of seizures, few simple procedures must be followed to prevent further complications due to seizures. They include,

1) To keep calm as one cannot stop a seizure once it has started

2) To ease the person to the floor and loosen the tight clothing, if any

3) To remove any hard, sharp, or hot objects in close vicinity that might injure the person

4) Not to put anything in the person's mouth

5) As the person relaxes after a fit, to turn him gently onto his stomach with the face to one side and the head extended so that the saliva can flow from the mouth

6) Its important to make sure that he can breathe freely

7) To contact the parent or guardian

8) If the person undergoes a series of convulsions, with each successive one occurring before he or she has fully recovered consciousness, or a single seizure lasting longer than 10 minutes, one should immediately seek medical assistance.

- In terms of cognitive outcome, individuals with seizure onset after the age of 2.5 years have a better cognitive outcome than those whose seizures started before that age. Hence preventive measures should be taken up to avoid occurrence of seizures during early childhood.
- Activities that might trigger a seizure, such as playing video games that have flashing or flickering lights, continuous music etc should be avoided.
- A complete nutritional coverage of vitamins, minerals and amino acids (vitamin E, manganese, taurine, and other nutrients) that empower the nervous and immune system etc. is essential to control seizures in children.

Note: Prevention is always better than cure. These preventive measures should be considered to avoid further consequences due to seizures in children in general and CWCDs in particular.