Communication disorders associated with childhood epilepsy

Essay
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By
Dina Ahmed El-Sayed El-Refaie
M.B.,B.CH
Resident of Phoniatrics-*Ain Shams University*

Under Supervision of

Prof. Dr. Safaa Refaat El-Sady
Professor of Phoniatrics
Faculty of Medicine
Ain Shams University

Prof. Dr. Noran Nagdy El-Assal
Assistant Professor of Phoniatrics
Faculty of Medicine
Ain Shams University

Prof. Dr. Amal Sayed Saber
Assistant Professor of Phoniatrics
Faculty of Medicine
Ain Shams University

Faculty of Medicine
Ain Shams University
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LIST OF ABBREVIATIONS

ABR	Auditory brainstem response
ADHD	Attention deficit hyperactivity disorders
AS	Absence seizures
BDMH	Brain damaged motory handicapped
BECT	Benign epilepsy with centro-temporal spikes
BFE	Benign frontal epilepsy
BPE	Benign psychomotor epilepsy
CAE	Childhood absence epilepsy
CSWS	Continuous spike wave of sleep
DLD	Delayed language development
EcoG	Electrocorticography
EEG	Electroencephalogram
EOP	Epilepsy with occipital paroxysms
ESES	Electrical status epilepticus of sleep
GTCS	Generalized tonic-clonic seizures
IPEC	Idiopathic partial epilepsies in children
LKS	Landau Kleffner syndrome
MEG	Magnetoencephalogram
MSTs	Multiple subpial transections
OAE	Otoacoustic emission
PET	Positron emission tomography
RS	Rolandic spikes
SL	Sign language
SPECT	Single photon emission computed tomography
STM	Short-term memory
TBI	Trumatic brain injury
TLE	Temporal lobe epilepsy
VNS	Vagal nerve stimulation

INTRODUCTION

Communication entails exchange of ideas and meaning between two or more persons. Failure to communicate accurately may lead to many of the problems experienced in families, in school, in social interactions and in employment. The main process of communication is language, spoken or written *(Svoboda, 2004).*

Language is an arbitrary symbolic system that pairs sounds and signs to meanings *(Kotby, 1980)*.

Speech is defined as acoustic vibrations resulting from patterns of movement of speech organs such as lips, tongue, jaw, palate and pharynx *(Lawrence and Raymond, 1982).*

Epilepsy is a disorder of the brain characterized by generation of seizures and by neurological, cognitive, psychological and social consequences of this condition *(Fisher et al., 2005).*

The brain is a dynamically changing and developing organ, especially in the early years of life. Seizures can inhibit or distort brain development as well as the related functions. Seizures interfere with brain functions by over activation, inhibition or destruction of vital brain functional pathways *(Bishop, 1981).*

It has been noted that epilepsy may be associated with one or more of the following disorders:

1- Language problems: The seizure discharge may disrupt the language processing, which

results in delayed language in early childhood or aphasic symptoms if it occurs after language development *(Gilmore and Heilman, 1981)*

2- Speech problems: Stuttering may be a characteristic of left complex partial seizures, as an interictal or postictal finding *(Baratz and Mesulam, 1982).* Also *Lecours and Joanette (1980)* reported that dysarthria can be seen with partial seizures involving the dominant hemisphere.

3- Behavior problems associated with childhood epilepsy include attention deficits, autism, anxiety, depression, conduct disorders and psychosis *(Onuma, 2000).*

4- Learning problems in the form of learning disabilities are more apt to be seen with partial seizures and medication reactions *(Dam, 1990)*.

Thus it is claimed that childhood epilepsy is very much correlated with different communication disorders. It is accused with language, speech, learning and behavioral disorders whether per se or due to the antiepileptic drugs used. Still details of this correlation are not known to phoniatricians and may be to neurologists.

AIM OF THE WORK

The aim of this work is to present a comprehensive review about communication disorders associated with childhood epilepsy, its incidence, etiology, assessment and different lines of management. This will help phoniatricians a lot in dealing with a child with communicative disorders associated with epilepsy.

Speech, Language and Communication

Man is distinguished from other creatures by acquisition and utilization of complex codes for the purpose of communication. The main process of communication is language *(Kotby, 1980).*

Language is a dynamic complicated system of symbols, spoken or written, used in various ways for thought and for communication. Spoken language is not the only process of communication, other means of communication include reading and writing, visual symbols and telegraphy. Cognition has a primary and vital role of language. The child has to reach a certain level of cognition to start language symbolization *(Svoboda, 2004).*

Language processing

Incoming auditory sensations are perceived and discriminated in the temporal lobe. Language stimuli are then transferred to the posterior temporo-parietal area on the dominant side to be understood. Connections to the limbic system and diffuse cortical memory areas draw upon related associations to form concepts, which are projected forwards either to the dominant posterior inferior frontal lobe to be expressed in spoken efforts or

just above that area to be expressed in a written or gestured manner *(Gordon, 1996).*

Organization and localization of language in the brain:

The brain is neither anatomically nor functionally symmetrical. The dominant hemisphere, that handles the majority of basic language functions, is usually the left side. The non dominant hemisphere, usually the right, not only process non language functions such as perceptual-motor skills but also contributes the emotional flavor to language, adding feelings and melodious features that prevent the speech from being a flat monotone *(Ojelmann, 1979).*

Language areas are distributed around the Sylvian fissure, the anterior language area is responsible for expression while the posterior language area is responsible for reception. *Gordon (1996)* reviewed the organization of language in the brain at the American epilepsy conference. The auditory discrimination, which is the distinguishing between various speech sounds, is performed in the posterior superior temporal lobe. On the left, in the temporal parietal occipital junction, is Wernicke's area, where the word and phrase sound clusters are given meaning. In the inferior temporal and pre-occipital area, interpretation of the meanings of sounds and of pictures occurs on the left and right sides respectively. The information is then sent forwards to be expressed. Production of sounds and language is in the

classic Broca's expressive area in the lower pre-motor frontal area. About 90% of individuals are naturally right handed, with language function strongly in the left hemisphere. Only about 10% of individuals are left handed, often there is a family history of left handiness. Those left handed individuals with a strong family history of left handiness are more apt to have a language function shared between the two brain halves or lateralized to the right side. Those with little or no family history of left handiness may have had their language shifted to the right hemisphere because of some early brain insult.

Language development:

At 30 weeks of pregnancy, the language areas of the brain are already prominent. Language is not fully developed until nearly seven years of age. In prenatal period, language potentials, originally bilateral, tend to develop predominantly on the left side. The portion of the brain over the posterior temporal lobe known as the plenum temporale is already becoming asymmetrically prominent on the left side in 90% of fetuses (right handed individuals). This area is destined to serve receptive language functions. This area enlarges more than the similar area on the right side of the brain. The asymmetries are less marked, absent or sometimes are reserved in left handed individuals *(Geschwind, 1972).*

In infancy, within days or weeks after birth, the newborn can distinguish different sounds. Within months,

the infant is identifying and categorizing sounds. The young child's receptive understanding precedes the expressive language skills in development. Receptive language is continually being shaped and developed in the first year of life. The first half year of life is associated with emotional vocalization and babbling. The infant practices through repetition. Then, over the ensuing half year, repetitive sounds are initiated and practiced. By around one year and certainly below two years, the infant begins to experience with single words. By two years of age, the infant is into phrases, mainly nouns and verbs *(Geffen, 1976).*

In early childhood (2-7 years), receptive language develops before expressive language. Girls tend to develop language skills more rapidly than boys. By the age of two and half years, children have the beginning of the adult language pattern, with words, syntax and grammar basically developed. By three years of age, the child uses pronouns, adjectives and adverbs *(Hardy, 1965).*

In late childhood (above 7 years), complex sentences and full grammar are almost developed as the language areas approaches maturity. The language areas become fully developed by around seven years of age, when the myelination is essentially completed. By the eighth year of age, articulation is fully developed. In children older than eight years, the configuration of language cortex is similar to adults *(Risse et al., 1999).*

Kotby (1980) categorized pre-requisites for language development into four main groups:

1-Normal sensory channels.

2-Intact brain functions.

3-Intact psychological state and desire of the child to communicate with others

4-Stimulating environment.

Damage versus plasticity:

The ability of the immature human brain to adjust for insults is called plasticity. Damage to established language areas may result in a loss of language abilities, as seen in adult and older children. Damage to undeveloped areas, as in younger children, may allow the shift of development of language to other capable areas of the brain, resulting in non classical localizations or unexpected lateralization for language. The younger child may be able to compensate, after a slight developmental delay, by developing alternative sites for language functions *(Bishop, 1981).*

Ross and Mesulam (1979) reported that the right hemisphere processes melodious tone variations, accents and inflections that give emotional feeling to language. It assists in some recognition of single words but not phrases. Damage to the right hemisphere renders a person unable to understand or to express the emotional aspects and inflections of language. The resultant language may seem featureless monotone. The language of the left hemisphere is the language of thought and logic, while the language of the right hemisphere is the language of emotions and feelings.

Up to three years of age, the child is often able to recover language fairly well after an insult to left hemisphere language areas. If the original insult has been overcome, the child usually will essentially have recovered a good command of the language, within about ten years of the insult. Left hemisphere damage after five years of age but before puberty, does not necessary result in irreversible loss of functions but incomplete recovery is still possible. Permanent speech and language problems become apparent around puberty *(Bishop, 1981).* However, *Thulborn et al. (1999)* noticed functional recovery from dysphasia after acute stroke. They concluded that, recovery of dysphasia in adults can occur rapidly and is concomitant with an activation pattern that changes from left to a homologous right hemispheric pattern.

Childhood epilepsy

Definition:

Epilepsy is a brief and usually unprovoked stereotyped disturbance of behavior, emotion, motor function or sensation which on clinical evidence results from cortical neuronal discharges. It is a recurrent transitory disturbance of brain function which develops suddenly and ceases spontaneously. It is a recurrent state of disturbance in the chemico-electrical activity of the brain characterized physiologically by an abnormal excessive neuronal discharge (dysrythmia). Single fit does not in general merit a diagnosis of epilepsy or treatment *(Kaufman, 1995).*

Epileptic syndrome is a disorder of the brain characterized by generation of seizures and by the neurological, cognitive, psychological and social consequences of this condition *(Fisher et al, 2005).*

Incidence:

The rates of epilepsy range between 20-70 per 100,000 populations per year. The incidence is age dependent, with a maximum in early childhood, and lowest rates in early adulthood. Incidence figures rise again in older age groups, probably because of the higher prevalence of cerebrovascular disease. The overall

risk of epilepsy is slightly higher in males than in females ***(Barry et al., 1999).***

The electroencephalogram (EEG):

Table (1): Normal EEG rhythms ***(Kaufman, 1995)***

Activity	Hz(cycles/sec)	Usual location
Alpha	8-13	Posterior
Beta	>13	Anterior
Theta	4-8	Generalized, may be focal
Delta	1-4	Generalized, may be focal

Alpha activity (8-13 Hz) is the regular activity overlying the occipital region. It is accentuated when individuals are relaxed with their eyes closed, but it disappears if they open their eyes, concentrates or become anxious.

Beta activity, frequencies faster than 13 Hz, usually has relatively low voltage and overlies the frontal region. Although present in normal persons, beta activity is accentuated when people are concentrating or anxious.

Theta (4-8 Hz) and delta (1-4 Hz) frequencies are normally detected in children and in all people as they enter deep sleep.

Normal EEG rhythms *(Kaufman, 1995)*

During a seizure (ictus), the EEG reveals paroxysmal activity that usually consists of bursts of spikes, slow waves or complexes of spikes and waves. After the seizure, in the postictal period, EEG usually shows only

low voltage activity. In the interictal period between seizures, EEG contains specific abnormalities that support a diagnosis in up to 80% of epileptic patients. In patients suspected of having epilepsy, an EEG is performed after sleep deprivation. In about 15% of epileptic patients, a sleep deprived EEG reveals abnormalities not apparent in routine studies *(Kaufman, 1995).*

Intensive EEG-video monitoring consists of several days of continuous split-screen videotaped clinical and EEG recordings of seizures, changes in behavior and effects of sleep. EEG-video monitoring is extremely useful in:

1-Diagnosing, classifying and determining the frequency of seizures.

2-Evaluating patients for epilepsy surgery.

3-Follow up of patients who seem to suffer from refractory seizures (frequent seizures that seem unaffected by anticonvulsant).

4-Identifying disorders that mimic seizures *(Kaufman, 1995).*

Causes:

A-Idiopathic epilepsy: no cause can be found, may have a genetic basis.

B-Symptomatic epilepsy:

A Cause can be detected due to disorders of the central nervous system.

1-Congenital: cerebral palsy.

2-Traumatic: cerebral contusion or laceration

3-Inflammatory: encephalitis or meningitis.

4-Vascular: cerebral hemorrhage, thrombosis or embolism.

5-Neoplastic: primary or metastatic tumors *(Barry et al., 1999).*

Classification of epilepsies and epileptic syndromes:

An epileptic syndrome is characterized by a cluster of symptoms and signs customarily occurring together. The symptoms and signs may include the type of seizures, etiology, precipitating factors, age of onset, severity, chronicity, specific EEG characteristics and sometimes prognosis *(Barry, 1999).*

International classification of epilepsies and epileptic syndromes (1989) (Barry, 1999):

I-Localization related (focal, local or partial) epilepsies and syndromes:

A-Idiopathic (not preceded by a cause, may have a genetic basis):

1- Benign childhood epilepsy with centro-temporal spikes.

2- Childhood epilepsy with occipital paroxysms.

3- Primary reading epilepsy.

B-Symptomatic (known or suspected disorders of the central nervous system):

1- Chronic progressive epilepsia partialis continua of childhood.

2- Temporal lobe epilepsy.

3- Frontal lobe epilepsy.

4- Occipital lobe epilepsy.

5- Parietal lobe epilepsy.

II-Generalized epilepsies and syndromes:

A-Idiopathic, with age related onset:

1- Benign neonatal familial convulsions.

2- Benign neonatal convulsions.

3- Benign myoclonic epilepsy in infancy (brief jerks in the arms or legs).

4- Childhood absence epilepsy (pyknolepsy).

5- Juvenile absence epilepsy.

6- Epilepsies with grand mal seizures (GTCS) on awakening.

7- Other generalized idiopathic epilepsies not defined above.

B-Cryptogenic:

1- West's syndrome (infantile spasm).

2- Lennox-Gastaut syndrome.

3- Epilepsy with myoclonic-tonic seizures.

4- Epilepsy with myoclonic absence.

C-Symptomatic:

a- Non specific etiology:

1- Early myoclonic encephalopathy.

2- Early infantile epileptic encephalopathy.

3- Other symptomatic generalized epilepsies.

b- Specific syndromes (Epileptic seizures may complicate many disease states).

III-Undetermined epilepsies and syndromes:

A- With both generalized and focal seizures:

1- Neonatal seizures.

2- Severe myoclonic epilepsy in infancy.

3- Acquired epileptic dysphasia (Landau Kleffner syndrome).

4-Epilepsy with continuous spike waves during slow-wave sleep.

B- Without unequivocal generalized or focal features.

IV- Special syndromes:

A- Febrile convulsions.

B- Isolated seizures or isolated status epilepticus.

C- Seizures occurring only when there is an acute

or toxic event due to factors such as alcohol, drugs.

In partial seizures, the clinical and EEG changes indicate initial activation of a system of neurons limited to part of one cerebral hemisphere. In generalized seizures, there is initial involvement of both hemispheres; consciousness may be impaired and this impairment may be the initial manifestation; motor manifestations are bilateral and the ictal EEG patterns initially are bilateral *(Barry, 1999).*

Idiopathic generalized epileptic syndromes in childhood:

A-Childhood absence epilepsy (CAE):

CAE is a relatively rare form of idiopathic generalized epilepsy. Absence seizures (AS) occur in several forms of generalized epilepsies and a multiplicity of conditions have been commonly referred to as petit mal. CAE should be a term restricted to epilepsy characterized as follows:

1-A form of epilepsy with an onset before puberty.

2-Occuring in previously normal children.

3-AS as the initial type of seizures.

4-Very frequent AS of any kind, except myoclonic absence.

5-AS associated in the EEG with bilateral,

symmetrical and synchronous discharge of regular 3 c/s spike and wave complexes on a normal background activity *(Roger et al., 1992).*

Incidence:

The annual incidence of CAE has been estimated at 6.3/100,000, it represent 8% of epilepsy in school-age children. Sixty to 76% of affected children are girls. A positive family history is found in 15% of cases *(Loiseau et al., 1990).*

Clinical features:

AS begin usually between 3 and 12 years of age. AS are characterized as follows: 1 to 3 seconds staring spells during which he becomes glassy-eyed and mute. Typically rolls his eyes up and blinks, though he loses his consciousness, he maintains bodily tone, the onset of AS is sudden, the attack ends abruptly. The child is very often unaware of his attack. A stimulus (call or pain) can shorten the absence seizure. The attacks are very frequent throughout the day *(Schwab, 1974).*

AS occur spontaneously but are influenced by environmental factors. AS are often triggered by many factors as: emotional (fear, anger), intellectual (lack of interest, meal-time for some children, school time for others), nycthermeral (evening or awakening) or metabolic (hypoglycemia, hyperventilation) *(Loiseau et al., 1990).*

Electroencephalography:

AS is associated with bilateral synchronous and symmetrical discharges of rhythmic spike and wave complexes. The spike-wave has the same shape and amplitude at homologous points in the two hemispheres. They have their highest amplitude under the fronto-central leads. The frequency of the spike-wave complex is 3 c/s at the beginning of the discharge and slow to 2.5-2 c/s towards the end. Interictal paroxysmal activity consists of single or brief discharges of bilateral spike-waves. These paroxysmal abnormalities are more numerous during non-REM sleep *(Kaufman, 1995).*

Development of tonic-clonic seizures:

Subsequent to the onset of AS, generalized tonic-clonic seizures (GTCS) occur in about 40% of patients. Development of GTCS is not in itself of grave significance for the patient. GTCS are infrequent and usually controlled by antiepileptic drugs. They begin in most cases between 10-15 years of age. Predisposing factors for GTCS are:

1-AS occurs after 8 years of age. The latter the onset of attacks, the more likely it is that the patient will develop GTCS.

2-Boys are more often affected than girls.

3-Prescribed drugs: in-patients treated early and correctly, GTCS appeared in 30% of patients, and in 68% if incorrectly treated *(Loiseau et al., 1990).*

Absence seizures with unusual patterns (Atypical absence):

In patients with atypical absences, both ictal and interictal EEG abnormalities are very different from the ones recorded in AS. Atypical absences are associated with consistently atypical spike-waves: irregularity of the rhythm, frequency lower than 3 or 2.5 c/s, and asymmetry in the discharge. Other parameters are of diagnostic value: early age of onset, prevalence in male patients, brain damage and mental deficiency, GTCS preceding AS, and drug resistance ***(Lugaresi et al., 1973).***

B-Epilepsy with generalized tonic-clonic seizures in childhood:

Epilepsy with GTCS in childhood is characterized by the following features:

1-The rate of incidence is lower than in adolescence.

2-The onset involves febrile, non-recurrent seizures.

3-The GTCS are associated with absence in which a slight tendency to affect girls in childhood and adolescence.

4-Generally, minor status epilepticus is frequent.

5-Typical; generalized EEG abnormalities can be

observed.

6-Mental disorders are rare.

7-The prognosis is favorable.

8-There is less likelihood of relapse after withdrawal of medication than in other age groups.

GTCS occurs in almost half of all the recorded cases of epilepsy (46%) *(Oller-Daurella and Oller, 1992).*

Many of GTCS are the result of an autosomal dominant trait. In the initial tonic phase, the patients are conscious, roll their eyes upward and extend their neck, trunks and limbs as if to form an arch. Subsequently, they undergo dramatic clonic phase in which their limbs, neck and trunk are wracked by violent jerks. During the tonic phase, the EEG shows repetitive increasingly higher amplitude spikes with increasing frequency in all channels. In the clonic phase, the spikes which become less frequent but greater in amplitude, are interrupted by slow waves *(Kaufman, 1995).*

Idiopathic partial epilepsies in children (IPEC):

Partial epilepsies range between 37-66% of childhood epilepsies. There are many types of idiopathic partial epilepsies of childhood:

A-Benign childhood epilepsy with rolandic or centro-temporal spikes (BECT) or benign rolandic epilepsy.

B-Benign psychomotor epilepsy (BPE) or temporal

lobe epilepsy (TLE) characterized by seizures with affective symptomatology.

C-Epilepsy of childhood with occipital paroxysms (EOP).

D-Benign frontal epilepsy (BFE) ***(Roger et al., 1992).***

Clinical features:

1-Absence of neurologic deficit; this condition represents not only a parameter of good prognosis, common to all forms of epilepsy, but it constitutes a specific part of the definition of all IPEC.

2-Absence of intellectual deficit.

3-High incidence of familial antecedents of epilepsy in their population: 32% of BECT, 38% of BPE, 19.3% of EOP.

4-The mean age of onset in all types of IPEC, ranges between 4 and 8 years.

5-In the majority of cases, The seizures are simple partial motor (BECT) or sensory (EOP) symptomatology. But in many cases, the seizures are of complex partial type.

6-The seizures are usually rare during the latter evolution of IPEC, but early in the course, there can be a high frequency of several seizures a day and this frequency can remain high for some days. Even when the seizures occur many times a day at onset, they are quickly controlled by treatment ***(Dalla Bernardina et***

al., 1992).

EEG criteria:

1-The peculiar EEG findings of IPEC is the presence of focal paroxysmal abnormalities, often changing in frequency during the evolution. The typical paroxysmal abnormalities are a focal slow spike followed by a slow wave, like the rolandic spike (RS) characterizing BECT. Most frequently is located on the centro-temporal, the parieto-temporal or the parieto-occipital areas.

2-The most striking finding is their significant increase in frequency during drowsiness and throughout all the stages of sleep. The amplitude of the paroxysmal abnormalities in BECT increases during non-REM sleep but decreases during REM sleep.

3-During evolution, brief and rare discharges of generalized spike waves appear in 1/3 or 1/2 of the patients **(***Dalla Bernardina et l., 1992).*

A-Benign partial epilepsy with centro-temporal spikes (BECT):

The most characteristic features are as follows:

1- The child usually aged 5 to 10 years.

2- A somatosensory onset with unilateral paresthesias involving the tongue, lips, gums, and inner cheeks.

3-Unilateral tonic, clonic or tonic-clonic convulsions involving the face, lips, tongue as well as pharyngeal

muscles.

4- Anarthria or speech arrest.

5- Drooling due to sialorrhea and saliva pooling.

6- Preservation of consciousness.

The seizures respond well to anticonvulsant therapy. The prognosis is excellent and recovery is the rule *(Loiseau and Duche, 1989).*

EEG criteria:

Interictal EEG records show centro-temporal spikes, either unifocal or multifocal. Typically, slow high voltage spikes, at times followed by a slow wave. When unilateral, they are always synchronous in the central (rolandic) and midtemporal areas. When bilaterally asynchronous spikes occur, both the rate and amplitude varies from side to side. In addition to the centro-temporal focus, some of the records show generalized spike-wave discharges. In approximately 30% of children with BECT, spikes appear only during sleep *(Blom and Heijbel, 1972).*

B-Benign psychomotor epilepsy (BPE):

Clinical features:

Th predominant features of the seizures were in all cases sudden fright or terror. This feature was sometimes associated with either chewing or swallowing movements, arrest of speech with glottal noises,

salivation, automatism (simple repetitive and purposeless movement), or some kind of autonomic manifestation (pallor, sweating or abdominal pain). These phenomena were associated with changes in awareness that did not amount to complete unconsciousness. The mean duration of the attack was between 1 and 2 minutes. No postictal deficit was ever observed, but the child could be temporarily sleepy or tired **(***Dalla Bernardina et al., 1992).*

EEG criteria:

The more frequent interictal abnormalities were characterized by ample slow spike/slow waves involving the fronto-temporal or parieto-temporal areas of one or both hemispheres. These abnormalities were always activated by sleep. It was observed the appearance of brief bursts of generalized spike wave, alone or in association with focal abnormalities **(***Dalla Bernardina et al., 1992).*

C-Epilepsy of childhood with occipital paroxysms (EOP):

Clinical features:

1-Visual ictal symptoms are represented by:

i- Amaurosis, i.e. partial or complete visual loss in the entire visual field.

ii- Elementary visual hallucinations, i.e. moving

flashing spots occupying the half or the entire visual field.

iii- Visual illusions, including micropsia, metamorphopsia.

2-Non-visual ictal symptoms may follow the visual symptoms and are represented by:

i-Hemiclonic seizures.

ii-Complex partial seizures with automatism.

iii-Other different ictal manifestations as dysphagia.

3-Postictal symptoms are represented by diffuse headache or migraine-like nausea and vomiting *(Gastaut, 1992).*

EEG criteria:

Slow wave in 80% of cases, sharp waves in 20%, high amplitude, over the occipital as well as postro-temporal regions of one or both hemispheres. Prompt disappearance with opening of the eyes in 94% of cases. Reappearance at eye closure and no significant effects of hyperventilation or intermittent photic stimulation *(Gastaut, 1992).*

D- Benign frontal lobe epilepsy:

Clinical features:

1- Simple partial seizures may be manifested by rhythmic jerky clonic movements of body part, which may be limited to one finger or extensive to affect an

entire side.

2- Complex partial seizures may lead to impairment of consciousness with behavior and thought disorders.

3- Left frontal lobe seizures may lead to dysphasic symptoms *(Kaufman, 1995).*

EEG criteria:

EEG typically shows paroxysms of spikes, slow waves or polyspikes and waves in channels overlying the frontal region *(Kaufman, 1995).*

Status epilepticus:

Successive tonic-clonic seizures, without regaining of consciousness between the attacks, may lead to circulatory collapse, respiratory failure, increased reflexes, inhalation pneumonia and lung abscess. It usually follows head injury or sudden antiepileptic drug withdrawal *(Barry et al., 1999).*

Speech and language problems in childhood epilepsy

The brain is a dynamically changing and developing organ, especially in the early years of life. Seizures can inhibit or distort brain development as well as the related functions. Seizures interfere with brain functions either by over-activation, inhibition or destruction of vital brain functional pathways. Long or frequent attacks may alter neuronal circuits and neurotransmitter balance *(Bishop, 1981).*

A communication disorder is impairment in the ability to understand and/or process spoken or written language. The problem may be in handling of the sounds and words themselves or their meanings. The problem may be difficulties in transmitting and using the sounds and words. A speech disorder is an impairment of articulation, resonance and/or fluency, while language disorder is the impairment of understanding and/or use of a spoken, written and/or other symbolic system *(Gilmore and Heilman, 1981).*

There is much controversy regarding the incidence of speech and language problems in childhood epilepsy. *Williams et al. (1992)* reported that speech-language problems occur in 24% of children with epilepsy. The relationship between the presence of language disorder and type of epilepsy in childhood was examined by *Parkinson (2002)* in 109 children with epilepsy, aged between 5 and 17 years. Evidence of association between focal epilepsy and language disorder was found.

Of the 46 (42.2%) children with language disorders in the research sample, 30 had focal epilepsy. Later, *Svoboda (2004)* noted that speech-language problems are more common than suspected, yet often overlooked. Language problems may be seen with nearly all types of seizures involving the frontal-temporal lobes and adjacent areas.

A-Speech problems in childhood epilepsy

1-Dysarthria:

Dysarthria is a combination of disorders of respiration, phonation, resonation, articulation and prosody that result from neuromuscular disorders *(Kotby et al., 1992).*

Dysarthria can be seen with elementary partial seizures in the form of spastic dysarthria (suprabulbar) manifested as strained strangled voice, imprecision of consonants and distorted vowels and disorders in prosody *(Lecours and Joanette, 1980).*

Prosody refers to the affect or emotional aspect of speech, i.e. the melodiousness of speech. The person who lacks prosody speaks in emotionless monotone. Prosody tends to be primarily a right (non dominant hemisphere) brain function *(Ross and Mesulam, 1979). Deonna et al. (1987)* reported that some

children with complex partial seizures seem to lack this prosody, speaking in a monotone style. The ictal speech disturbances may be limited to only the prosodic features of speech, i.e. intonation, rhythm and pause, resulting in a speech effort characterized by slow rate, monoloudness, monopitch and reduced stresses.

Boyce et al. (2003) suggested that patients with right hemisphere foci may show speech deficits that resemble those of patients with traumatic right hemisphere damage or stroke. Patients with right temporal lobe foci are frequently described as showing reduced or disturbed prosodic skills. The aberrant behaviors include both receptive skills (such as inability to interpret intonation as happy or sad) and expressive skills (such as lack of expression, sounding angry when sadness is more appropriate).

A temporary articulation problem may be a postictal symptom of oral damage occurred during a generalized tonic-clonic seizures with clinching of the teeth and tongue biting *(Lecours and Joanette, 1980).*

2-Stuttering:

Stuttering is a speech event that contains part word repetition, monosyllabic whole word repetition, prolongation or blockage *(ASHA, 1999).*

Stuttering may be a characteristic of left complex partial seizures, as an interictal or postictal finding. Paroxysmal epileptiform discharges from left temporal

lobe region have been associated with stuttering in the form of repetitions of initial word sounds and syllables and occasional prolongation of the initial word sounds and syllables *(Baratz and Mesulam, 1982).*

Stuttering in the form of blocks may be seen with absence seizures, myoclonic or atonic seizures, and complex or partial seizures. It may be an ictal or postictal finding *(Baratz and Mesulam, 1982).*

Chung et al (2004) reported a patient who presented with both acquired stuttering and long-lasting gait disturbance after supplementary motor area seizure.

Michel et al. (2004) reported a case of reflex epilepsy in which seizures were triggered by reading aloud or stressful conversation. Each paroxysmal event in left frontal region was associated clinically with a speech disorder mimicking stuttering. They suggested that reflex frontal focal epilepsy could be a cause for acquired stuttering.

3-Apraxia of speech:

Apraxia is the inability to form speech or even speech sounds, although the patient knows what he wants to say, in the absence of paralysis. A speech apraxia implies a problem in the transforming of a language concept into the motor act of expressing the thought through speaking. Seizures may interrupt such language processing, resulting in apraxia of speech *(Gilmore and Heilman, 1981).*

B-Language problems in childhood epilepsy

Gilmore and Heilman (1981) noted that epilepsy involving the left hemisphere is most likely associated with language problems. Such problems may precede, occur simultaneously with or follow seizures. The problems may be episodic or ongoing, if ongoing, they may result in a gradual deterioration of language abilities or even delayed language development from the start. The manifestations of such problems depend on the location of the seizure discharge. The seizure discharge may disrupt the language processing, which results in delayed language development in early childhood or dysphasic symptoms if it occurs after language development.

1-Agnosia:

Auditory agnosia is the state of being confused by and unable to recognize speech sounds. Recognition of various sound combinations is an important early stage to understand what is said. Agnosia is rarely recognized as a separate entity, more often, they are a part of or are confused with dysphasia syndrome or specific language impairment. Children with an epilepsy-associated Agnosia may seem deaf despite normal audiometric and brainstem evoked potentials. This has been referred to as central word deafness. The EEG shows epileptiform

discharges in the dominant temporal lobe. Antiepileptic drugs may control clinical seizures and may help the Agnosia *(Borkowski and Lotz, 1986).*

2-Dysphasia:

Dysphasia in childhood is an impairment of language that result from some form of cerebral insult after language acquisition has already commenced. The cerebral insult can result from a variety of causes including head trauma, infections and convulsive disorders (intractable epilepsy). Typically, these children have begun to develop language normally and were acquiring developmental milestones at an appropriate rate prior to injury. In the majority of cases, acquired epileptic childhood dysphasia is predominantly expressive. The symptoms most reported include initial mutism (suppression of spontaneous speech) followed by a period of reduced speech initiation, simplified syntax (telegraphic expression) associated with impaired auditory comprehension abilities, word finding difficulties, dysarthria and disturbances in reading and writing. Fluent dysphasia and receptive disorders of oral speech such as literal and verbal paraphasic errors and perseveration are rarely found in children with acquired epileptic dysphasia *(Aram et al., 1986).*

Dysphasia is known to occur as part of a seizure or in the immediate postictal period. Rarely, dysphasia may be the only symptom of the seizure. All types of receptive and expressive dysphasia may occur *(Gilmore and*

Heilman, 1981).

In left-handed individuals, language representation is often bilateral and thus all language function is organized more diffusely in either hemisphere than in the case of right-handed individuals. When an epileptic event occurs as the result of unilateral discharge, it is more likely to be associated with a paroxysmal dysphasia in left-handed individuals than in a right-handed person, irrespective of the side of the function. In right-handed, the side of the focus is much more related to the incidence of paroxysmal dysphasia than it is in left-handed. *(Hecaen and Piercy, 1956).*

Depasquet et al. (1976) reported that an episodic dysphasia may be lasting and in some patients, there is a history of seizures with episodic dysphasias. These episodes become worse, with the emergence of a persistent dysphasia. If this permanent dysphasia appears abruptly, the concern is that there may be an underlying lesion causing both the seizure and the dysphasia.

In some children, a transient dysphasia may occur following a seizure, although this is often overlooked as it is obscured by other phenomenon of the postictal state. Temporary dysphasias are known to occur in association with certain types of epilepsy, particularly temporal lobe complex partial seizures. Such dysphasia usually recovers, but it may recur after further seizures *(O'Donohoe, 1979).*

Dysphasia due to status epilepticus is rare, especially in the absence of any history of epilepsy. Dysphasia may be the only manifestation of a focal

epileptic status involving the left frontal lobe. In some patients, however, the seizure status concurrent dysphasia may last for days. This has been called dysphasic epileptic status. The use of anticonvulsant drugs improve the dysphasia by bringing about control of the seizure discharges *(Hamilton and Mathews, 1979).*

De Toledo et al. (2000) studied a patient with a 5 years history of recurrent episodes of inability to talk, without any other motor or cognitive impairment. Episodes lasted as long as 24 hours. During the episode, comprehension of complex verbal commands was preserved and she would make attempts to articulate words and correctly answered questions with head nodding or monosyllables, yes or no. Interictal EEG was normal, but EEG recordings during one of the episodes showed continuous discharge in the frontal and parasagittal areas demonstrating the ictal nature of the deficits. This case is unique for the isolated involvement of production of language during the seizure.

3-Anomia:

Anomia is a memory deficit in which an individual can not remember proper nouns, especially the names of people and items. It is associated primarily with complex partial seizures involving the dominant temporal lobe. This condition is not uncommon, But rarely recognized as an isolated item or even as a complication in childhood epilepsy. The individual may try to search about the

intended word or a similar word *(Ojemann, 1975).*

Fukatsu et al. (1999) reported a selective deficit in retrieving proper names after left temporal lobectomy. The patient showed anomia in conversation, in response to photographs and in verbal descriptions, despite being able to provide semantic information about people he was unable to name.

Bell et al. (2001) reported that object-naming impairment is common among temporal lobe epilepsy (TLE) patients. They examined object-naming ability and depth of semantic knowledge in healthy controls (n=29) and patients with early onset TLE (n=21). The TLE group demonstrated a significant deficit relative to controls in both object-naming ability and semantic knowledge for the target objects, even after controlling for IQ. Object-naming requires multiple information-processing levels, and anomia may be due to a deficit at or between one and more of these levels. The results suggest that a deficiency at the level of semantic knowledge can contribute to dysnomia in early onset TLE patients.

4-Delayed language development (DLD):

Childhood epilepsy from birth with DLD usually achieves other developmental milestones (motor development) normally. Often, a mixed developmental language deficit is found with defect in language form (syntax and phonology), content (semantic) and use (pragmatic), with receptive language more impaired, although in some children a pure expressive deficit that

improves with age has been identified. These children may have problems in adapting to a group, with later difficulties in social interactions *(Maccario et al., 1982).* Some children have delayed language from the beginning of language development, but others may have relatively normal language until after the onset of seizures. Yet another group of children may have significant DLD from birth, with a marked worsening noted at the time of seizure appearance *(Swarton, 1995).*

Children with DLD may display bilateral EEG abnormalities, Often bitemporal sharp spikes and waves, shifting from side to side. Epileptiform EEG abnormalities may be found on all night recordings *(Maccario et al., 1982).*

The term brain damaged motory handicapped child previously called cerebral palsy is defined as a persistent but not unchanging disorder of posture and movement due to dysfunction of the brain present before its growth and development are completed (before 3 years) *(Bax, M., 1964).*

Wojciech and Wojciech (2003) studied 198 children with brain damage. They reported that the overall epilepsy incidence was 41.4%. Epilepsy most commonly affected children with spastic tetraplegia was 65.6%. Low birth weight, neonatal seizures, seizures during the first year of life, family history of epilepsy, severity of brain damage and computer tomography findings were found to be related to significantly increased risk of epilepsy in children with brain damage.

Gururaj et al. (2003) studied the occurrence,

nature and prognosis of epileptic seizures in brain damaged motory handicapped child (BDMH). Fifty-six children with brain damage and seizures were studied. Two control groups of 35 children with brain damage without seizures and 50 children with seizures but no brain damage. They reported that spastic tetraplegia was the commonest type of brain damage associated with seizures, whereas spastic diplegia was the commonest variety of brain damage without seizures. Also, children with brain damage and seizures had a higher incidence of significant developmental delay, occurrence of significant abnormalities on brain imaging and a need for use of more than one antiepileptic drug. Over half of children in the study group presented with generalized tonic-clonic seizures. The overall outcome of seizures in children with brain damage was poor needing prolonged course of anticonvulsant medications, polytherapy and higher incidence of refractory seizures and admissions for status-epilepticus compared to the control group.

Dubois et al. (2004) reported that a 2 years-old boy presented with an early form of benign partial epilepsy with centro-temporal spikes and a severe language delay. Family video analysis revealed an early delay of babbling since the age of 12 months. Complete recovery occurred with antiepileptic treatment. They concluded that delayed development of language can be due to an epileptic dysfunction interfering with prelinguistic skills.

Types of seizures associated with speech and language disorders

Specific speech and language problems, such as dysarthria, stuttering, dysphasia and delayed language development, are more apt to occur with specific seizure types *(Henry and Brown, 1987).*

A-Partial seizures:

Vining (1996) noted that localization-related seizure syndrome might produce language dysfunction, such as with seizures originating in the lateral temporal lobe, the frontal lobe including the motor and opercular areas (Broca's expressive language area), and the posterior temporal parietal lobe (Wernicke's receptive area).

1-Simple partial seizures:

Simple partial seizures are usually brief, with the patient conscious at the onset. Seizures originating in the dominant hemisphere may disturb language. Dysphasia may occur during or after the seizure. When the speech and language areas are involved, stuttering, dysarthria and auditory hallucination may occur *(Lecours and Joanette, 1980).*

Simple partial seizures involving the left frontal lobe

may present with a pure expressive dysphasia, similar to that seen following partial frontal lobectomy for the control of epilepsy *(Alexander and Schmitt, 1980).*

Gilmore and Heilman (1981) reported that motor speech disturbances can originate from epileptic discharge in the supplementary motor area of either hemisphere. Partial stimulation of either supplementary motor area produces blocks in speech and/or bilateral mouth movement.

Benign Rolandic (centro-temporal) epilepsy:

Some children with benign Rolandic epilepsy, presenting with delayed language development and typical centro-temporal spikes, may show a more or less prolonged focal neurologic deficit referable to the Sylvian region. They may experience a transient impairment of oral movements of speech and swallowing, correlated with increased seizure frequency with EEG findings of an active left or bilateral centro-temporal typical Rolandic spike focus. There may be periods of dysarthria and anomia as well as associated drooling and difficulties in swallowing *(Croona et al. 1999).*

Although benign childhood epilepsy with centro-temporal spikes (BECTS) has a good prognosis, a few studies suggested the existence of language disorders relating to the interictal dysfunction of perisylvian language areas *(Monjauze et al., 2005).* They focused on language assessment in 16 children aged 6-15 years, affected by BECTS or in remission. An important

proportion of children showed moderate or more severe language impairment. The most affected domains were expressive grammar and literacy skills. They found linguistic deficits during the course of epilepsy but also persistent deficits in children in remission, suggesting long-term effects.

Northcott et al. (2005) demonstrated that children with benign rolandic epilepsy (BRE) have normal intelligence and language ability. However, a specific pattern of difficulties in memory and phonological awareness was found. EEG features were minimally associated with cognitive difficulties. Difficulties in phonologic awareness affect literacy and memory problems affect academic performance.

2-Complex partial seizures:

Children with complex partial seizures involving the dominant anterior temporal lobe are more apt to present with learning disabilities involving the auditory channel. Language performance also tends to be below normal. Speech may be slow in rate as if it is no longer automatic. More common complications include blocks, dysphasias and anomias ***(Mckeever et al. 1983).***

Caplan et al. (2001) reported that language deficits observed in children with complex partial seizures have included limited use of:

- Auxiliary and main verbs in children aged 3-5 years.
- Conjunctions and referential pronounces.
- Verbal reasoning such as making inferences and

determining cause and effect.

- Appropriate repair strategies.

Autosomal dominant lateral temporal lobe epilepsy previously has been linked to mutations in the LGI1 gene (Leucine-rich gene, Glioma Inactivated) have been found in some autosomal dominant lateral temporal lobe epilepsy families. They have identified a missense mutation affecting a conserved cysteine residue in the extra-cellular region of the LGI1 portion. The mutation is associated with autosomal dominant lateral temporal lobe epilepsy in a large Norwegian family showing unusual clinical features like short-lasting receptive dysphasia and auditory symptoms *(Gu et al., 2002).*

B-Generalized seizures:

With generalized seizures, the whole brain and all its functions are impaired.

1-Generalized tonic-clonic seizures

The patient is unconscious with the attack; therefore, speech and language functions are not in action. Postictal dysphasia or other speech or language problem may occasionally follow a generalized tonic-clonic seizure due to the seizure itself or due to the medication used to stop the attack. Status epilepticus may produce a reversible receptive dysphasia. The diagnosis is difficult, often; there are other neurologic

findings, usually with a non fluent expressive dysphasia. The EEG may show polyspike wave in the Wernicke's area that respond to diazepam, but language does not change *(Vining, 1996).*

2-Absence seizures

Absence seizures may be accompanied by various speech and language disturbances. Speech and language problems in absence seizures include speech blocks, slowing of speech, receptive and expressive dysphasia *(Mckeever et al., 1983).*

Cognitive and Learning problems in childhood epilepsy

Up to 50% of children with epilepsy have learning difficulties, with up to 30% at risk of developing serious learning problems. The main problems are mental retardation, intellectual regression, learning disabilities and underachievement. Some problems are potentially reversible, but others are permanent. There are multiple causes for learning problems in epilepsy, including the effects of seizures and seizure discharge, the seizure cause and the medications. Epileptic discharges may disrupt processing, thus interfering with attention, learning processing or storage or retrieval of information. Consequently, although the seizures may cease, the patient enters into the adult world unprepared and underemployed *(Dam, 1990).*

There are central brain sites related to specific types of learning. Vision prime sites are in the occipital lobe. Auditory receptive processes begin in the posterior area of the superior temporal gyrus. Learning input is processed primarily in the dominant hemisphere. Short-term memory utilizes the mesial-temporal hippocampal areas of the brain. The hippocampus and adjacent areas, so often involved in epilepsy, is a key site of learning *(Fedio et al., 1993).*

1-Intelligence and retardation:

The distribution of intelligence in children with epilepsy is directed towards a lower range, but it can vary from year to year in an individual. Retardation means that the IQ is below 70 which leads to poor scholastic achievement. In general population, 1-2% of people are mentally retarded. Around 20-29% of people with epilepsy are mentally retarded. Mental retardation with seizures is seen slightly more often in males than in females. An IQ below 80% will lead to poor scholastic achievement *(Besag, 1995)*

Children with absence seizures have an average IQ in the range 90-110. Children with partial seizures show higher intelligence than those with generalized tonic-clonic seizures. Children with mixed seizures have significantly lower IQ. Some of the generalized seizure syndrome of infancy and early childhood, including infantile spasm and some myoclonic and atonic mixed syndrome, often are associated with marked intellectual impairments. Factors contributing to lower IQ scores are early age of onset, longer duration, persistence of seizures and anticonvulsants effects. Also, the highest incidence of mental retardation (57%) is seen in children with very frequent seizures *(Cornaggia and Gobbi, 2001).*

Besag et al. (1991) reported that partial and generalized seizures are more associated with intellectual retardation than are absence or myoclonic seizures. The majority of cases of intellectual deterioration are seen in

children with more severe epileptic syndromes. In children with idiopathic epilepsy, deterioration is found in only 9.8%, whereas in symptomatic epilepsy, deterioration is found in 26-37%, especially in those with abnormal neurologic findings. Seizures may lead to a decrease in intelligence if they are frequent, prolonged or severe. Seizures interfere not only with neuronal development through the destruction of neurons by hypoxia, but also with the growth of nerve fibers and possibly RNA (ribo-nucleic acid) production in the brain. A deterioration of mental ability in children may be associated with high-dosage medication or polypharmacy.

2-Learning disabilities:

It is a generic term that refers to a heterogeneous group of disorders manifested by significant difficulties in the acquisition and use of listening, speaking, reading, writing and reasoning or mathematical abilities. A child with learning disability has an average IQ *(Cornaggia and Gobbi, 2001).*

Many children with epilepsy exhibit a higher incidence of learning disabilities associated with seizures, creating difficulties in academic achievement. This is especially apparent in their first years of schooling. Learning disabilities tend to be seen with partial seizures rather than with generalized seizures, especially involving the fronto-temporal brain areas, before age of six years, with seizures that are not controlled easily with

medications, especially if on phenytoin monotherapy *(Svoboda, 2004).*

The left hemisphere is usually dominant for language processing as well as functions including analysis, details notation, ordering of items, time notation and process of calculation in math. The right hemisphere relates to the processing of non-verbal skills, including the recognition and remembering of geometric shapes, spatial arrangements and relationships, directions, right-left differentiation and a sense of time. Children with left temporal lobe epilepsy tend toward problems in immediate memory and verbal attention, including verbal and to a lesser degree visual spatial memory, as well as problems in concept formation. Memory problems may be noted in understanding and in remembering words and names, math tables, prayers, addresses and phone numbers. In school, problems in reading and later in spelling may be noted. Children with right temporal lobe epilepsy may show a significantly poorer performance in visual spatial performance, visual spatial memory and non-verbal attention. The children tend to have problems in recognizing letters, numbers and familiar words. Individuals with a frontal epileptiform focus, especially right frontal disturbances, may have difficulties with organization, planning and independent work efforts *(Battaglia, 1998).*

Children with epilepsy underachieve especially in arithmetic, also in spelling, reading, and comprehension and word recognition. Children with generalized tonic-clonic or absence attacks have more achievement problems. Children with complex partial epilepsy, despite

having normal IQ, are often low in academic achievement, with poor school performance. Children with symptomatic epilepsy are more apt to underachieve than those with idiopathic seizures. With epilepsy, a significantly higher proportion of boys than girls experience educational difficulties. Under achievement is more common in children with early onset seizures, especially in the first year of life. Underachievement in academic subjects is associated with behavior disturbances, especially conduct disorders, although the behavior may then accentuate the learning difficulties *(Seidenberg et al., 1986).*

Giovagonoli and Avanzini (1999) studied the influence of epileptogenic lesions on learning and memory alterations in patients with temporal lobe epilepsy (TLE). They studied 131 patients (55 with left and 39 with right symptomatic TLE; 22 with left and 15 with right cryptogenic TLE) and 36 healthy subjects. They compared these groups by using a battery of tests to assess verbal and visual learning, delayed recall and recall after imposition of interfering activity. Compared with the controls and patients with right TLE, the patients with left TLE were significantly impaired in all verbal tests. In verbal tests, patients with right TLE were impaired compared with controls. They concluded that learning and memory abilities are impaired in patients with TLE irrespective of the presence of overt damage. This supports the theory that focal epileptic discharges, rather than the lesions themselves, affect these functions.

Breier et al. (2005) studied 83 children with

medically intractable complex partial seizures of either left or right hemisphere origin and were classified as having reading and/or spelling deficits (RS) or as not impaired (NI) by using standard achievement tests. All children had undergone noninvasive functional mapping of the receptive language cortex by using magneto encephalography (MEG). They found that reading and spelling achievement deficits in patients with complex partial seizures of left hemisphere origin are associated with atypical language organization, possibly secondary to reorganization of language function to right hemisphere areas.

Vanasse et al. (2005) compared the reading skills and phonological awareness abilities in a set of 13 years old identical twins, one of whom is affected by temporal lobe epilepsy (TLE). They compared their performance to those of an age and IQ matched control group. Both siblings have an intellectual quotient above average as well as normal memory and linguistic abilities. They reported that the reading age of TLE (assessed by reading test) was more than two years behind expectations whereas that of her sister was above average. Further, in contrast to her sister and healthy control subjects, TLE exhibited specific deficits in elaborate phonological awareness abilities (non-word repetition, rhyme detection, phonemic segmentation and syllabic inversion). These could be linked to temporal lobe dysfunction, thus confirming the important role of temporal lobes in reading acquisition.

Dubois et al. (2003) studied a male child presenting with benign partial epilepsy with rolandic

spikes from the age of 7 years associated with a specific regression in writing skills. A longitudinal study over nearly 2 years showed improvement in handwriting under antiepileptic therapy. A detailed analysis with a computer monitored graphics table showed at first a rapid improvement of skills followed by protracted slower progress. They concluded that the initial rapid recovery of skills was directly linked to the improvement of his epilepsy. The slower late acquisition of motor programs that had never been fully established was due to long standing interference by his epilepsy.

Memory problems in childhood epilepsy

Memory is the cognitive ability to encode, store and retrieve information. Difficulties in memory and concentration may impede intelligence, leading to ineffective functioning. Memory functions are linked to the mesial portion of the temporal lobe, especially the hippocampus and connected structures. Memory is commonly divided into immediate (sensory), recent (short term) and remote (long term) types ***(Pergin et al., 1993).***

1-Immediate (sensory) memory:

Sensory memory involves the very brief storage of large amounts of information. Visual memory begins to fade at about one second and auditory memory in four seconds.

2-Recent (short term) memory:

Short-term memory declines over hours and fades over days. Without reinforcement, non verbal short term memory fades within 15-30 seconds; verbal memory fades even faster, depending on the nature of the information.

3-Remote (long term) memory:

Long-term memory contains information in the memory stores for a long time, perhaps a life time. It appears to be of unlimited capacity and existing as bits of information scattered diffusely throughout the brain.

Epilepsy in children may impair the development of memory skills. Impaired memory is associated with partial rather than generalized seizures. Children with complex partial seizures, especially those due to lesions, are especially prone to impaired memory functions, due to several problems in the verbal memory system. Verbal functions and related memory are more often affected, especially with dominant hemisphere involvement. The hippocampus is involved in the transferring of short term memory to long term memory. Hippocampal memory problems may be temporary or permanent. Both temporary and permanent amnesia may follow a simple seizure or a flurry of seizures *(Binnie et al., 1990).*

Children with generalized seizure disorders may show problems with visual sequential memory than auditory memory deficits. Children with generalized tonic-clonic seizures often have a below average memory. With absence attacks, memory may be impaired primarily during rather than between the attacks. The degree of

memory loss for events occurring during a seizure varies from a complete amnesia to total retention of memory. Memory recall may be impaired for a stimulus that precedes the discharge by up to four seconds *(Freeman et al., 1973).*

Behavior problems in childhood epilepsy

About 30% of patients with epilepsy have some kind of psychiatric symptoms. Epilepsy in children results in an increased risk for behavioral, emotional, psychiatric and social impairments, occurring at a higher frequency than in people with other chronic illness, than in people who are visually or hearing handicapped, and much higher than in healthy children.

Behavior problems in epilepsy include:

A-Behavior problems associated with childhood epilepsy:

1- Attention deficits hyperactivity disorder.

2- Autism.

B-Behavior problems caused by childhood epilepsy:

1- Anxiety.

2- Depression.

3- Conduct disorder.

4- Psychosis.

In looking at dysfunctions with seizure disorder, the problem may relate to the seizure discharge itself, the nature of the brain insult causing the seizure, if present, the effect of medication used to treat the seizures and the management of the person's condition *(Onuma, 2000).*

Behavioral problems may be related to both intrinsic and extrinsic factors. Intrinsic factors may result from the nature and location-lateralization of epilepsy. Extrinsic factors include the attitudes of the parents and siblings, the teachers and peers and the resultant self-concept of the individual, which often reflects the experiences with other individuals *(Austin, 1993).*

Batzel et al. (1984) reported that children are less prepared to cope with the stresses of epilepsy. Children with epilepsy are more dependent on others, whose attitudes shape personality development and social interactions. The growing child with epilepsy may be treated differently from siblings in the family and may often feel neglected or rejected. The uncontrolled limb movements or loss of consciousness may lead to feelings of a lack of control and dependence. A seizure may be fearsome to a young child. There may be a fear of returning to where the first seizure occurred, in a classroom or in bed. The fear of dying during the seizure may increase anxiety. School-aged children worry about the seizure, medical procedures, medications and the responses of others. Feeling of inferiority and insecurity may lead to withdrawal from social interactions, thus decreasing the chances of gaining social skills. Society is more apt to reject the patient because of personality than because of epilepsy.

Boel (2004) studied the associated behavioral and neuropsychological problems in 573 children with refractory epilepsy, during the period 1984-2000. He reported that the most frequent behavioral problems in different epilepsy categories were pervasive

developmental disorders such as autism (48/573=8%), attention problems (43/573=7.5%), loss of self-esteem (9%). Pervasive developmental disorders were significantly more frequent in secondary generalized epilepsies. In 86 children (15%), mental decline due to epileptic process itself was observed. As expected this was seen in all patients with Lennox-Gastaut syndrome, West syndrome and in severe myoclonic epilepsy in infancy.

Schacter (1996) noted that the postictal period may be one of confusion, depression, difficulty in talking, exhaustion, fear, frustration, headache, memory loss, loneliness, sleep, thirst and weakness. Some of these factors are direct consequences of the seizure event and some are the results of the psychosocial reactions. The postictal state is often more handicapping than the seizure itself, especially if a dysfunction is present. Patients cannot function for up to 12-24 hours.

A-Behavior problems associated with childhood epilepsy:

I-Attention deficit hyperactivity disorders in childhood epilepsy (ADHD):

Attention deficit disorders, with or without hyperactivity are a collection of similar behaviors caused by a wide variety of problems. Recognition of the cause is necessary to treat the problems effectively. There are

three subtypes of ADHD:

1- Predominantly inattentive.

2- Predominantly hyperactive-impulsive.

3- Combined type.

ADHD comprises marked inattentiveness and a lack of inhibition (impulsivity), often accompanied by restlessness (hyperactivity) present in two or more settings, of at least six months duration, with onset before seven years *(Wolraich, 2001).*

Diagnostic criteria for ADHD include:

1) Inattentive:

a- Fails to give close attention to details.

b- Difficulty sustaining attention in tasks.

c- Does not seem to listen when spoken to someone directly.

d- Does not follow instructions and fails to finish school work.

e- Easily distracted by external stimuli.

2) *Hyperactivity*:

a- Fidgets with hands and feet.

b- Leave seat in classroom or in other situation.

c- Difficulty in engaging in activities.

3) Impulsivity:

a- Blurts out answers before questions have been completed.

b- Has difficulty waiting turn.

Dunn et al. (2000) reported that ADHD is seen in 28-37% of children with epilepsy, 75% of these patients being boys. The inattentive subtype is more frequent than inattentive-hyperactive subtype. Impulsive behavior is seen in 39% of children with present or past epilepsy, compared with 11% of normal children. Children may exhibit ADHD as a coincidental disorder but more commonly due to one of many factors associated with epilepsy. Children with epilepsy are at risk for symptoms of ADHD. Children with epilepsy have poorer concentration and mental processing and are less alert than age-matched non-epileptic children. A combination of genetic and neuropsychological factors may cause ADHD. In the seizure patient, the environment, the early learning stages and the intelligence may all contribute to hyperkinetic behavior, or the cause may be a mixture of problems, including environmental stress.

Hughes et al. (2000) reported that generalized seizures, especially those with generalized spike-wave bursts on the EEG (in absence, atonic, and myoclonic seizures) are often seen in children who seem inattentive and impulsive. Abnormal spike-wave bursts interrupt attention. Increase in drug dosage may normalize the EEG and thus increase the attention abilities. Poor seizure control has been associated with hyperactivity in girls with incompletely controlled absence epilepsy.

The incidence of ADHD in children with complex partial seizures reaches up to 25%. In preschool children with complex partial epilepsy, up to two of every three may show some hyperactivity. The common syndrome may present with persistent hyperactivity, short attention span, and a lack of normal inhibitions or fears. Aggressiveness and destructibility may often be seen. This occurs most often in children with seizure onset before age of five years, especially in boys. Intellectual development may be slowed. The behavior appears with or soon after the seizure onset and may lessen or be outgrown in puberty. Children with left temporal seizures tend to be inattentive and overly active in noisy settings but children with right sided seizures tend to be virtually distractible. Children with frontal lobe seizures seem uninhibited and disorganized, with poor planning *(Sherman et al., 2000).*

Therapy:

1- Adjusting antiepileptic medications:

Attentional disorders can appear with antiepileptic drugs especially barbiturates and benzodiazepam. If attention deficits are due to antiepileptic therapy, it is more advisable to revise seizure therapies than add a stimulant drug *(Devinsky and Vazquez, 1993).*

2-Psychological intervention:

All ADHD children need behavioral approaches and all parents need counseling and guidance to change a

specific deviant behavior or develop a missing desirable behavior *(Dunn et al., 2000).*

3-Promoting language abilities and cognitive training:

These approaches seek to increase the child's ability to teach problem-solving strategies and to promote language abilities *(Dunn et al., 2000).*

4- Pharmacotherapy:

a- Stimulants

Stimulant medications are safe when used as prescribed, but they must be monitored carefully. They are the first drug of choice for the treatment of ADHD, although the incidence of seizure activation is around 1%. Stimulants such as methylphenidate (ritalin) work by increasing levels of dopamine in the brain *(Devinsky and Vazquez, 1993).*

b- Antidepressants:

The tricycles (such as imipramine) may be useful, especially in children with combined ADHD and depression or anxiety *(Dunn et al., 2000).*

II-Autism with childhood epilepsy:

Definition:

Autism is one of the pervasive developmental disorders of the brain function. Autism is characterized by early onset of deficits in verbal and non-verbal communication skills, failure to develop social relationship, and restricted range of activities and interests *(Onuma, 2000).*

Incidence of epilepsy and risk factors:

The increased incidence of epilepsy in the autism spectrum ranges from as low of 7% to a high of 42%, the frequency seeming to increase with age. The first peak occurs in early childhood before age of five and the second peak occurs in adolescence. The main risk factors for seizures in autism include low intelligence, especially if combined with a motor deficit. The prevalence of epilepsy with autism is more frequent in girls *(Onuma, 2000).*

Anand (2005) reported that there is an increased and variable association between autism and epilepsy. When autism associated with epilepsy, it places the child and the family in a very stressful situation. The prevalence of epilepsy among autistic is much higher than the normal population. About one in four autistic children develop seizures at puberty. In autistic children,

the risk of seizures increases if they have certain specific neurological disorders like neurofibromatosis, tuberous sclerosis or untreated phenylketonuria. Children with symptomatic infantile spasms (West's syndrome) tend to develop both epilepsy and autism. Also he reported a tendency for epileptic foci to occur in the temporal or frontal regions in autistic children and he suggested that temporal and frontal dysfunctions may be important in the mechanism of symptoms of autism.

Clinical features:

Three broad groups of symptoms were found in most children of autism *(Rapin, 1995):*

a- Social deficits:

1- Autistic infants may resist cuddling.

2- Autistic children look to other people as if they are looking through them.

3- They may fail to turn around when called

4- They do not know how to make friends.

b- Deficits in language and communication:

1- Late acquisition of language

2- Unintelligible speech in short sentences

3- Inability to express themselves.

4- Inability to receive information from others.

5- Deficits in non-verbal communication

6- Deficient ability to use prosody

c- Restricted range of activities and interest:

1- Repetitive stereotyped movements

2- They resist change in routines.

Tuchman et al. (1998) reported that no specific EEG findings are characteristic of the autism spectrum. Prolonged EEG studies, especially including overnight recordings, may reveal focal or generalized spikes or slow waves (60%). Even those patients without seizures have abnormal EEG (46%). In 65%, the spikes are localized to the temporal lobes, perhaps more on the left side.

Treatment:

1- Family counseling.

2- Language training therapy.

3- Behavior modification ***(Henry and Browne, 1987).***

B- Behavior problems caused by childhood epilepsy:

I-Anxiety disorders in childhood epilepsy:

Patients with anxiety disorders overestimate the degree of danger and the probability of harm in a given situation, while underestimating their abilities to cope with perceived threats. Anxiety disorders are reported in 5-32% of people with epilepsy, with an incidence of 13% in those with temporal lobe epilepsy *(Hermann et al., 2000).*

Goldstein and Harden (2000) reported that anxiety disorders may present as auras, ictal episodes, postictal states and interictal behaviors. Fear is the most common ictal emotion reported in epilepsy. Fear and anxiety are seen in simple partial seizures and anteromedial-temporal seizures. Postictal fear and anxiety may last hours to days, sometimes even up to seven days post-seizure. Interictal periods of anxiety may be seen with both generalized and complex partial seizures.

II-Childhood depression and epilepsy:

Depression, often unrecognized in children and adolescence, occurs in about 23-26% of children with epilepsy *(Kanner and Balabanov, 2002).*

Lambert and Robertson (1999) reported that depressive episodes may occur predominantly as preictal, ictal, postictal and interictal, but postoperative depressions may emerge after a temporal lobectomy when seizure control is gained. Depression presents as abrupt, out-of-context feelings ranging from mild fear or sadness to profound hopelessness. Symptoms include a dropping school performance, withdrawal from friends, and loss of pleasure from previous enjoyable activities. Postictal depression usually lasts a day or two, although it may last up to a week and is seen especially with complex partial seizures. Interictal depression appears to be endogenous, with sudden onset, marked fluctuations and abrupt cessation.

III-Conduct disorders in childhood epilepsy:

Conduct disorders is a more severe behavior problem, involving serious violations of the rules of society. Such acts include aggression (fighting, use of a weapon, cruelty to people or animals) and destroying property. The problem must be present for at least six months to a year. Children with epilepsy may show a higher incidence of conduct disorders, although such disorders are frequent in the non-epileptic population. Slightly more than 12% of children with epilepsy meet the criteria for a conduct disorder, compared with 5% in the non-epileptic population. Children with complex partial epilepsy have high rates of conduct disorders *(Weisbrot and Ettinger, 1998).*

Aggression has been reported to be one of the most common characteristics of patients with temporal lobe seizures and patients undergoing on anterior temporal lobectomy. Complex partial seizures suggest underlying brain dysfunction, often involving the limbic system, may contribute to behavioral disturbances, including violence. Children with temporal lobe foci are more aggressive than those with other seizure types. Aggressive behavior and rage outbursts may be seen in about 14% of children with complex partial seizures *(Gerad et al., 1998).*

IV-Psychosis in childhood epilepsy:

Psychosis is a condition characterized by delusions, hallucinations, and disorganized speech and behavior. The incidence of psychosis is very high in patients with temporal lobe epilepsy (14%) and is high in patients with generalized epilepsy (3.3%). Epileptic psychosis can be classified as peri-ictal (ictal and postictal), which are usually of short duration, and interictal, which are usually prolonged *(Torta and Keller, 1999).* An ictal psychosis is an expression of the seizure activity. Postictal psychosis occurs in about 6-11% of children with epilepsy. Within one to seven days of a cluster of generalized tonic-clonic seizures or a complex partial seizures, the child, most often a male, develops delusions with hallucinations, memory disturbances and fears. These appear to be related to complex partial epilepsy, especially involving the left temporal lobe.

Common epilepsy syndromes and its specific associated communication disorders

I-Landau-Kleffner syndrome (LKS)

Definition:

Landau-Kleffner syndrome of acquired dysphasia with epilepsy is a childhood syndrome characterized by the sudden or gradual loss of receptive and expressive language, the usual emergence of a variety of seizures with EEG abnormalities and a resultant deterioration of behavior *(Bishop, 1985).*

Epidemiology:

The family history is often negative for epilepsy or significant language delay. In children who develop seizure, the family history for epilepsy is 12%, in those who don't develop seizures; the family history is 5%. The prenatal, natal, and postnatal history are normal. The syndrome is two to four times more likely to occur in boys than girls possibly related to an increased language lateralization difference in males or sex linked inherited predisposition *(Roger et al., 1992).*

Pathogenesis:

The etiology of LKS remains unknown. The nature of the disorder has been a matter of speculation. Possible causes include a tumour or vascular problem, a genetic or developmental disorder, low grade encephalitis, an infectious or immune problem and epileptic disruptions, with the later being the most popular theory. LKS may be a final common pathway with multiple potential etiologies, acquired or genetic, most likely insulting the tempro-parietal areas of the developmental brain *(Gascon et al., 2000).*

Clinical picture:

LKS occurs in normally developing children who are between 3 and 7 years of age. The child experiences the progressive loss of receptive and then expressive language over weeks to months without comparable intellectual deterioration. The disorder usually begins as an auditory verbal agnosia. The child ceases to comprehend and respond to speech and speech sounds, with difficulties discriminating between familiar words, non speech sounds, and even environmental sounds. A receptive dysphasia with a gradual loss in comprehension of spoken language usually follows the agnosia, although the two may merge to the degree that the agnosia is not recognized or is over looked. Often, the child has problems in understanding even when visual cues and lip-reading are possible. A rapid reduction of oral

expression then appears. Initially, the child makes little effort to speak spontaneously. What is said tends to be repetitive, with perseveration and paraphasia. The child may become mute, or exhibit inappropriate substitution of words and anomia or resort to gestures. Eventually, a telegraphic speech or a fluent jargon may be heard. Even the ability to use sign language can be lost. The language disorder can be progressive or characterized by remissions and exacerbations *(Wioland et al, .2001).*

Preceding, co-occurring with or following the language deterioration there may be a series of seizures *(Deonna, 1991).* He reported that of those cases that exhibit seizures, 43% experience the seizures before language regression, 16% display co-occurrence of seizures and language regression, 41% experience seizures within a few months to years after the language problems. The seizures begin as a single seizure or as status, especially at the onset. One or several types of seizures emerge (80%), often with atypical absence (16%) or complex partial seizures (16%) with psychomotor automatisms, although eventually a generalized tonic-clonic attacks (33%) are noted. Nocturnal simple partial motor seizers resembling benign central-temporal epilepsy, sometimes with a transient postictal facial weakness, may be seen. Regardless of whether or not there are clinically observable seizures, however, all patients with the syndrome exhibit epiletiform discharges in their EEGs.

Gascon et al. (2000) reported that behavioral disturbances are common (50-70%), especially at the onset of the disorder. The behavior deterioration usually

parallels the language deterioration and does not relate to intelligence. The behavior problems may relate to an acute anxiety of the child with impaired capability of understanding what is going on. Behavior problems include hyperactivity and attention deficits, aggressiveness, anger, withdrawal, social deficits, frustration outbursts, and if severe, autistic and psychotic-like presentations, all known to be reactions to a loss of communication skills.

In the recovery period, the condition improves gradually. The seizures cease and the EEG often normalizes, usually before age 10 (80%) of patients, and always before 15 years of age. Then over several years, the dysphasia improves with gradual partial recovery of language functions. About 33-42% are left with a severe residual deficit, 24-25% remains with a mild to moderate residual deficit, and 35-40% make a relatively good recovery. The degree of recovery of language depends on the age of onset, with the best outlook after 7 years of age. The younger the onset, the poorer the outlook (*Ballaban-Gil and Tuchman, 2000).*

Majerus et al. (2003) noted that the outcome of language abilities is variable and a residual impairment in verbal short-term memory (STM) is frequent. This STM deficit might be related to the persistent dysfunction of temporal lobe regions where epileptic discharges were observed during the active phase of the disorder.

Diagnosis:

1- Electroencephalography (EEG):

LKS appears to be partial seizures originating in the temporal cortex most often on the left posterior region. The seizures are associated with bilateral asymmetrical paroxysmal EEG abnormalities. Three types of EEG abnormalities are noted: generalized, often slow- spike or polyspike wave bursts or sharp waves, posterior temporal spikes, more prominently left-sided, or a combination of both. The posterior temporal spikes, which may be unilateral, bilateral or multifocal, spread forward to the mid-temporal region and to the motor areas in the frontal lobe or occasionally to the parietal or occipital areas. The EEG background activity between the discharges is often normal or borderline, but it may be abnormal. The abnormal EEG is usually found in the first ten years of life, especially around age 3-5 years *(Gomez and Klass, 1990)*

Gascon et al.(2000) reported that EEG findings vary in severity, lateralization and location, especially with sleep. There is significant activation of generally continuous or near continuous bilateral spike-waves during deeper stages of sleep called continuous spike-waves of sleep (CSWS), which may resemble sleep status called electrical status epilepticus of sleep (ESES).

2-Video-electro encephalography monitoring:

Ming et al (1996) noted that with recording of a seizure, there is a left temporal mono-rhythmic theta build-up, with rapid discharging of spike-waves

discharges maximum from the left side.

3-Radiological brain imaging:

CT scans, MRI scans and Angiograms obtained to exclude treatable lesions are usually normal. Occasionally, the abnormalities found are felt to be secondary to the chronic epileptic process *(Morrell et al., 1995).*

4-Functional Imaging studies:

Single photon emission computed tomography (SPECT) and positron emission tomography (PET) studies have shown focal abnormalities, predominantly in temporal or infratemporal locations, in brain perfusion and glucose metabolism, with increased blood flow ictally and decreased blood flow interictally. PET scans with a variety of chemicals have shown focal cerebral hypometabolism at the epileptic focus in one or both temporal lobes *(Da Silva et al., 1997).*

5-Psychlogical evaluation:

Although the patients are often thought to be retarded or psychotic, if tested by non-language tests, intelligence is found to be basically not impaired *(Gascon et al., 1973).*

6-Speech and language evaluation:

Attempts to elicit verbal communication and to improve receptive skills are unsuccessful in 42% of patients. An early language evaluation for both diagnostic and rehabilitative purposes is important (*Cooper and Ferry, 1978).*

Treatment:

1-Anticonvulsants:

The goal of treatment is to control the seizures and seizure discharges, although usually this does not lead to improvement in language, even if there is EEG normalization. In some patients, the language worsens with seizure control. The seizures may cease without medications. The older antiepileptics are often ineffective and may aggravate the situation. Phenytoin and carbamazepine may increase the duration of the spike-wave activity in sleep. The valproate, ethosuximide and clonazepame may be partially helpful for the seizures, at least for a brief time, but will not help the dysphasia, even if given intravenously. Vigabatrin and felbamate may be effective *(Glauser et al., 1995).*

2-Immunologic therapy:

Based on the theory that LKS is an autoimmune disorder, a trial of two to three months on corticosteroids, monitored closely for objective improvements, and has been reported to help both clinical and EEG abnormalities *(Marescaux et al., 1990).*There may be re- emergence of an epileptiform EEG and dysphasic relapse with the tapering of the steroids *(Morrel et al., 1995).*

Mikata and Saab (2000) reported that intravenous immunoglobulin may be useful in LKS both clinically and by EEG findings, with deterioration of both about two months post-infusion.

3-Ketogenic diet:

Bergqvist et al. (1999) described three patients with LKS refractory to traditional treatment who were successfully treated with ketogenic diet (high protein diet). All three patients had lasting improvement of their language, behavior and seizures for 26,24and 12months respectively,

4-Neurosurgical approaches:

Surgical approaches include temporal lobectomy, resulting in improvements in language and seizures control ***(Nass et al., 1999).***

Morrell et al. (1995) reported that multiple subpial transections result in recovery (28-50%), improvement (28-44%), or no change (12-22%). The improvements may be temporary. Some deficits in attention and expressive language remain. In follow-up to multiple transpial transections for LKS, the behavior ratings by parents were normal and the children were back in regular school classes.

5- Treatment of speech and language problems:

This will be discussed later.

6-Remediative support:

Children with LKS do best in special schools dealing with dysphasic patients. A school or class for deaf children is not appropriate. The children can be taught to read and to write. If the dysphasia onset occurs after the child has acquired handwriting, re-education is more successful ***(Deonna et al., 1989).***

Using sign language, lip-reading, writing language and communication boards, may help the child with a severe communication problem. Some individuals may remain dependent on sign language into adulthood *(Tharpe et al., 1991).*

Perez and Davidoff (2001) noted that children With LKS can benefit from visual forms of language, mainly sign language (SL). They reported a case of a boy with LKS who lost speech comprehension and expression from 3 years and was educated in SL from the age of 6 years. His SL was evaluated at the age of 13 years and compared with a control child with congenital sensorineural deafness. It was found that: (1) patients achieved the same proficiency in SL as the control child with deafness. (2) SL learning did not compete with, but perhaps even hastened, the recovery of oral language. Intact ability to learn a new linguistic code such as SL suggests that higher-order language areas were preserved and received input from a separate visual route.

Prognosis:

There are two types of LKS, transient and chronic, although they vary in severity and duration. Some children experience a brief, self-limited episode with good recovery. Those with chronic form may have an earlier onset and continue after the EEG recovery with a lack of speech, with language impairments inhibiting academics. Behavior remains infantile and dependent. The prognosis

may be influenced by several factors, including age of onset, pattern of language deficit, frequency and topography of EEG discharges, duration of epilepsy and efficacy of treatment as well as side effects of antiepileptic drugs. Bad prognosis in case of onset before 4 years of age, duration of dysphasia greater than one year and the duration and continuity of sleep spike-wave epileptic status *(Deonna et al., 1989).*

II-Electrical status epilepticus In sleep (ESES)

Definition:

Electrical status epilepticus during sleep, also known as continuous spike-waves of sleep (CSWS), presents with regression in language, cognition and behavior. This is a mixed partial and generalized seizure syndrome. Any child who loses language skills for no other apparent reason should have overnight EEG monitoring *(Besag, 2001).*

Epidemiology:

This is a rare disorder seen in up to 1% of children with epilepsy, more often in boys (58%) than in girls, without any evidence of inheritance *(Stores, 1990).*

Cause:

The pathophysiology is not known. ***Ballabn-Gil and Tuchman (2000)*** found that 50% of children might have a pre-existing brain insult, but no consistent cause has been found.

Course:

The seizures often emerge at about eight years of age and the ESES appear about one to two years later. Seizures are self limited, disappearing by the age 10-15 years ***(Roger et al., 1985).***

Clinical features:

The child, often of previously normal development, begins to exhibit a widespread regression of intelligence, behavior and language. The child may manifest word finding difficulties, pardysphasia or an expressive dysphasia but no oral apraxia. Behavior changes include attention deficits as well as hyperactivity and occasionally a psychotic state during the ESES ***(Yamashita et al., 2000).***

Ballaban-Gil and Tuchman (2000) noted that seizures occur in a majority of cases and often precede the ESES. The seizure may be nocturnal initially in sleep (50%) and then occur upon awakening. At the onset, the

initial seizures may be partial, often unilateral involving the face or generalized as clonic attacks. Atypical absence seizures are seen especially at the onset of the ESES.

Diagnosis:

Possible tests include EEG and imaging studies as CT, SPECT or MRI, the latter to exclude the possibility of a brain lesion **(***Swarton, 1995).*

Ballaban-Gil and Tuchman (2000) reported that an overnight EEG study may be needed to observe all stages of sleep. At the onset of ESES, the syndrome presents with sleep activation, with diffuse complexes or spikes and wave activity at a rate of 1.5-2 per second occurring in more than 85% of EEG tracings in non-REM slow sleep, which occurs every time the child sleeps. Less common are continuous focal discharges with frontal predominance. REM sleep halts the generalized spike-wave status, although rare bursts of diffuse spike-wave fragments and focal frontal-central rhythmic discharges may be seen. After the end of ESES, both the seizures and the EEG improve in adolescence.

Treatment:

Treated patients tend to show some improvement in cognition,language and seizures. Combinations of

carbamazepine, valproate, ethosuximide and clonazepam seem to control the seizures, stop the nocturnal status and lead to a decrease in signs and symptoms of ESES. When the seizures are controlled, there is an improvement in emotional stability *(yasuhara et al., 1991).*

Yamashita et al. (2000) reported that ACTH may suppress the ESES, improve language and cognitive functions, but only while the patient is on steroid. Several months after discontinuation of ACTH, moderate difficulties and word-problems findings return and the EEG deteriorates.

Prognosis:

The seizures usually respond to therapy and disappear, but cognitive and behavioral deficits may persist *(Roger et al., 1985).*

III-Rasmussen's syndrome

Definition:

Rasmussen's syndrome is a progressive disease affecting primarily one hemisphere, accompanied by intractable epilepsy and leading to slowly progressive hemispheric dysfunction and mental impairment, with a progressive atrophy of the affected hemisphere *(So and*

Andermann, 1997).

Epidemiology:

This is a relatively uncommon condition, occurring worldwide without any seasonal or epidemic pattern and seemingly non-familial *(So and Andermann, 1997).*

Cause:

The exact cause and mechanism is not known. The etiology of Rasmussen's encephalitis has been postulated to be a chronic focal viral infection, an immune reaction following an acute viral infection, or a focal immune reaction *(Pardo et al., 2000).*

Clinical features:

The disorder usually begins in childhood between the ages of one and 15 years. The seizures usually appear before age ten, and persist, being resistant to anticonvulsant therapy. Progressive partial seizures, with or without secondary generalization, emerge. Simple partial motor seizures are seen in all patients, with some patients also developing complex partial seizures. In some patients (20%), the onset may be an episode of status epilepticus. The seizures occur more frequently in

many patients, occurring at least daily *(So and Andermann, 1997).*

Capovilla et al. (1997) reported that a spastic hemiparesis ipsilateral to the focal seizures develops gradually over three months to ten years, although occasionally abrupt deficits may occur. There is often deterioration in intellectual function, as well as other neurological deficits. Mild to severe mental retardation usually emerges (85%). Other cortical deficits may emerge, including DLD or progressive dysphasia (if the dominant hemisphere is involved), cortical sensory loss, and hemianopia. Behavior disorders and psychological deficits may also be seen.

Diagnosis:

1-Electroencephalography:

Low voltage polymorphic slow waves appear in all patients in the affected hemisphere, between one day and four months after the onset of the seizures. Bilateral epileptic discharges may be found in one-third of patients, but asymmetry is usually present sufficiently to permit lateralization to one hemisphere *(Granata et al., 2000).*

2-Radiology:

Neuroimaging confirms the diagnosis. CT scan can demonstrate a progressive lateralized cerebral atrophy with localized functional abnormalities, while MRI can reveal focal abnormalities within the first month

(Graanta et al., 2000).

Treatment:

Medical treatment, including antiepileptic drugs, corticosteroids, and gamma globulins, are of little value in Rasmussen's syndrome, but they can delay the need for radical surgery *(Capovilla et al., 1997)*.

Knight et al. (1999) reported that an early total or subtotal functional hemispherectomy is followed by complete seizure remission, stabilization of the neurologic deterioration, and general improvement in function. Recovery of language processes appear to be related to the age of onset and duration of the illness. In the preschool years, children improve significantly. Even in adolescence, useful recovery can be obtained. General cognitive functions improve due to removal of the diseased interfering hemisphere.

Telfeian et al. (2002) noted that hemispherectomy is a very effective surgical treatment for intractable seizures. The decision of when to perform a dominant hemispherectomy depends greatly on how late the surgeon believes some shift in language to the non dominant hemisphere can occur. They reported a right-handed girl with Rasmussen's syndrome who underwent a left hemispherectomy at the age of 15 and has had excellent control of her seizures and remarkable language recovery.

IV-Perisylvian-opercular syndromes

The perisylvian-opercular syndromes consist of a congenital type and acquired type, both presenting with pseudobulbar palsy and epilepsy

A-Congenital perisylvian-opercular syndrome

Cause:

The syndrome is caused by a bilateral structural defect in the anterior opercular area. Other causes include congenital cytomegalovirus infection, in utero ischemia, and chromosomal mutations *(Kuzniecky et al., 1994).*

Clinical features:

The syndrome becomes apparent in infancy or early childhood. Characteristic of this syndrome is that severe voluntary oromotor problems are noted, with preserved non-voluntary emotional movements. The child demonstrates developmental delays and mild to moderate mental retardation. Dysarthria with preserved comprehension is seen. Generalized seizures are common (80%). Absence seizures may be seen. Partial epilepsy

may also be noted *(Kuzniecky et al., 1994).*

Examination:

All patients with the congenital pseudobulbar palsy manifest as dysarthria in the form of poor palate function, impaired tongue movement, exaggerated jaw reflex and affection of swallowing *(Kuzniecky et al., 1994).*

Diagnosis:

EEG abnormalities present interictally with generalized bilateral slow spike-wave discharges, involving especially the central-temporal parietal regions. CT and MRI scans show symmetrical bilateral perisylvian abnormalities with increased cortical thickness *(Belousova et al., 1998).*

Treatment:

Multiple anticonvulsants have been given, but if the seizures are intractable, surgery may be helpful. A corpus callosotomy, either anterior or complete, is the most common approach *(Kuzniecky et al., 1994).*

B-Acquired perisylvian-opercular syndrome

Cause:

Bilateral neurological dysfunction of perisylvian region may be caused by disruption of normal connections or by an excessive inhibitory reaction to the epileptiform discharges *(Engel and Wilson, 1986).*

Clinical features:

The children mostly girls, are often preschoolers. The child develops recurrent prolonged episodes of severe oral apraxia, dysarthria, and drooling (due to swallowing difficulties). Each episode lasts weeks to months. There is a peculiar unawareness of the presence of food on the mouth (oral sensory agnosia). Twitching of the angle of the mouth and tongue, lasting up to hours, may be noted. Within a year, infrequent, brief, partial seizures may emerge *(Shafrir and Prensky, 1995).*

Diagnosis:

Shafrir and Prensky (1995) noted that EEG shows spike-wave discharges over the centrotemporal areas with variable speed. Video-EEG studies show

generalized spike-wave discharges maximal over the involved temporal area. The MRI is normal initially, but later may show a mild diffuse atrophy in the perisylvian regions.

Treatment:

Various antiepileptic drugs have been tried without significant response. A response is seen when felbamate or clonazepam is added. Some children may require oral surgery to help the drooling, resulting in transient improvement. This does not help the chewing and swallowing component. Tube feeding may be needed *(Shafrir and Prensky, 1995).*

V-West syndrome (infantile spasm)

Clinical features:

West syndrome usually starts during the first 3-5 months of life and characterized by the emergence of myoclonic spasm, deterioration of developmental skills in infancy, mental retardation and severe abnormalities on an EEG (hypsarrhythmia). When the spasm starts, development ceases and often regresses. The spasm manifest as abrupt contraction, lasting less than two seconds, followed by less intense but sustained tonic contractions lasting 2-10 seconds. Spasms frequently

occur in a series of 20-40 jerks and usually ablate after 10-30 minutes. When the hypsarrhythmiic EEG emerges, neurologic function deteriorates. The continuous slow-wave activity may interfere with cognitive function *(Dam, 1990).*

Treatment:

Askalan et al. (2003) compared the efficacy of vigabatrin (antiepileptic drug) versus corticotropin (ACTH) in treating infantile spasms. They concluded that vigabatrin may be effective for patients with symptomatic infantile spasms and patients with idiopathic infantile spasms showed improved cognitive function following treatment.

Prognosis:

Rener-Primec et al. (1999) reported that long term prognosis of children with symptomatic infantile spasm is poor, with cognitive and neurologic development being affected in over 90% of cases. Cognitive impairment can range from selective cognitive disorders to major mental retardation. Children with idiopathic infantile spasms have normal IQ and many have no cognitive impairment, but almost have behavior problems .

VI-Lennox-Gastaut syndrome

Epidemiology and cause:

Lennox Gastaut syndrome is one of the most severe childhood epilepsy syndromes. It accounts for about 3% of all childhood epilepsies and has a peak onset age of 3-5 years. About 20% of all cases are following West syndrome (symptomatic). Most of patients has no definite cause (idiopathic) *(Dam, 1990).*

Clinical features:

Lennox Gastaut syndrome presents in childhood with a mixed seizure disorder. The three main seizures seen are:

1- Tonic seizures: where there is a general stiffening of muscles lasting several seconds to a minute.

2- Atonic seizures: where there is sudden loss of muscle tone lasting 1-4 seconds.

3- Atypical absence: where there is a brief period of immobility with a stare lasting 3-30 seconds.

It usually has a slow spike-wave generalized EEG picture and mental retardation. Children often are not as retarded as those with infantile spasms, but they have more severe behavior problems *(dam, 1990).*

Treatment:

It is often resistant to old antiepileptic drugs. New drugs as felbamate is a drug of choice *(Svoboda, 2004).*

Prognosis:

Dravet (1999) reported that good prognostic factors of Lennox-Gastaut syndrome include later onset in childhood, previously normal development and an idiopathic form. Poor prognostic indicators include early onset, symptomatic nature (especially following West syndrome), frequent seizures, recurrent status epilepticus and persistence of slow-wave activity on the EEG.

VII-Inflicted traumatic brain injury in infancy

Head injuries are a common cause of death in childhood, and an important cause of traumatic brain injury (TBI). Between 24-33% of TBI admissions in children < 14 years of age are from inflicted (non accidental) TBI, and up to 42% require admission to the intensive care unit (ICU). The various forms of inflicted TBI (including shaken infant syndrome) are now well recognized and diagnosed in most countries. The

incidence in Scotland is 24.6 infants per 100000persons-year *(Barlow and Minnes, 2000).*

Clinical features:

There is very limited research regarding the outcome of inflicted TBI in childhood, including shaken infant syndrome. ***Barlow et al. (2005)*** described the long-term neurologic, behavioral and cognitive sequelae seen in 25 children with inflicted TBI in Scotland between 1980 and 1999. The mean length of follow-up was 59 months. A total of 68% of survivors were abnormal on follow-up, 36% had severe difficulties and were totally dependant, 16% had moderate difficulties, and 16% had mild difficulties on follow-up. A wide range of neurologic sequelae were seen, including motor deficits (60%), visual deficits (48%), epilepsy (20%), speech and language abnormalities (64%), and behavioral problems (52%). As regard epilepsy, children had symptomatic partial seizures with secondary generalization and one child developed infantile spasms at 8 months age. Speech and language problems include marked dysarthria and apraxia. Some children had delayed language development or expressive dysphasia all associated with cognitive impairment and marked behavioral abnormalities. Behavioral problems are hyperactivity, impulsivity, problems with attention, rage reactions; sleep disturbances and one patient had an autistic spectrum disorder.

Prognosis:

Barlow et al. (2005) concluded that inflicted TBI has a very poor prognosis and correlates with severity of injury. Extended follow-up is necessary so as not to underestimate problems such as specific learning difficulties and attentional and memory problems that may become apparent only once the child is in school. Behavioral problems begin to manifest clinically between the second and third years of life, although the consequences of frontal lobe injury may be underestimated unless follow-up is extended into adolescence and early adulthood.

Evaluation of communication disorders with childhood epilepsy

Assessment protocol passes by 3 phases:

1-Preliminary diagnostic measures.

2-Clinical diagnostic aids.

3-Additional instrumental measures.

For simplification, and to be specific, the special clinical diagnostic aids for each communication disorder will be presented in brief.

I-Screening of high risk group:

Communication disorders in children with epilepsy occur frequently enough, such that all children with epilepsy should be screened for possible speech and language problems. This is particularly true if the patient has complex partial seizures, especially if from the left hemisphere.(Talking with the child, the parents and the school personnel can identify areas of possible problems) *(Bradford, 1980).*

II- Language assessment:

Children who have epilepsy are at risk of developing language problems and should be evaluated to diagnose

delayed language development and dysphasia *(Bradford, 1980).*

1- Tests for delayed language development:

a- Evaluation of cognitive and perceptual abilities

- Snijders-Oomen non verbal Intelligence Scale; age from 4y, 11m to 15y, 11m, it is non verbal test, it can be performed for deaf and normal hearing children.
- Stanford Binnet Intelligence Scale; age from 2y to 16y, it measures verbal performance.
- Ruth Griffiths; age from birth to 2 y
- Vinland Social Maturity Scale; age from birth to 15y.

b- Specific language functions test (standardized Arabic Language Test). It gives a language age between 2-8 years. Before 2 years of age, quosiobjective way of evaluation is done through play situation with the child to get an idea about:

-Inside language:

-Object permanence.

-Object constancy.

-Reversibility.

-Causality.

-Passive vocabulary: giving a score out of 5 in each semantic category.

-Active vocabulary:

-babbling: 6 months.

-First word : 1 year.

-Twenty words ± starting 2 words sentence: 1.5 years.

-Two hundred words + 2 words sentence: 2 years.

Standardized Arabic Language Test entails the following items *(Kotby, 1989):*

1-Attention of the child by observation and ability to imitate.

2-Receptive part of the semantics:

- Ability to recognize different semantic groups: body parts-clothes- fruit- vegetables- food utincles- colors- transportation- money.

- Ability to categorize things into semantic groups.

- Ability to match / make pairs.

- Ability to understand opposites.

- Ability to recognize time concepts.

3-Expressive part of the semantics:

-Ability to name different semantic groups.

-Ability to say opposites.

4-Receptive part of the syntax:

Testing the ability to understand:

2 words sentence, Spatial indicators and prepositions, Time indicator, 3 words sentence, Verb tenses, Pronouns, Adjective, Adverb, Negation form,

Comparative, Numbers, Orders increasing in length, Action- agent use and Passive voices sentences.

5-The expressive part of the syntax: testing the ability to utter the following

His name and his mummy, Respond to questions whose answers are one word, Verb tenses, Prepositions, 3 words sentences, Pronouns, Adjective, Adverb, Conjunctions, Counting, Negation, Singular and pleural, Action- agent, Time indicators, Repetitions and Sample of spontaneous speech.

6-Pragmatics: test the ability to initiate dialogue, continue it and end it.

7-Prosody.

8-Phonology: by articulation test.

2- Tests for dysphasia (Kotby et al., 1985):

a- Evaluation of language status using dysphasia test, the test items include:

1-Presentation and orientation to time, person and place.

2-Auditory memory span.

3-Speech:

- Automatic speech.
- Spontaneous speech.

4-Understanding speech.

5-Understanding written text.

6-Reading.

7-Writing.

8-Color and form perception.

9-Calculation.

b-Psychometric evaluation using non-verbal psychometric tests:

-Progressive Matrices Test.

-Snidjers- Oomen Non-Verbal Intelligence Test.

-Marrian Frostig Test of Visual Perception.

-Bender-Visual Motor Gestalt Test.

-Taylor Test of Anxiety.

3- Test for learning disabilities: Hornsby (1984) described tests that are usually used at its assessment

a-Language test

b-Psychometric evaluation using:

-Wechsler intelligence scale (does not involve reading or writing)

-Stanford Binnet intelligence scale

-Marriane Frostige Developmental test of visual perception.

c-Illinois test of psycholinguistic abilities: it includes the following

-At the represential level (auditory reception- visual reception- auditory association- visual association- verbal expression- manual expression).

-At the automatic level (grammatic closure- visual closure- auditory sequential memory- visual sequential memory- auditory closure- sound blending).

d-Reading and writing tests: The child is asked to read aloud from a set text and then asked to give verbal answers to several comprehension questions, it gives a reading age. It may be presented in a more simplified form for younger age group.

e-Tests for central auditory function.

-Auditory attention.

-Auditory figure ground.

-Auditory discrimination.

-Auditory closure.

-Auditory blending.

-Auditory analysis.

III- Speech assessment:

Children who have epilepsy are at risk of developing speech problems and should be evaluated to diagnose stuttering and dysarthria.

1-Assessment for dysarthria (Kotby et al., 1992):

a-Auditory perceptual assessment:

-Articulation: consonant imprecision- vowel distortion- compensatory articulatory mechanism.

-Resonance: nasality- audible nasal emission of air.

-Respiration.

-Prosody: rate- stress- pauses.

-Phonation: degree of dysphonia- character- loudness- pitch.

-Overall intelligibility of speech.

b-Visual assessment of the vocal tract.

c-Dysphasia test: done to exclude associated dysphasia.

2- Assessment of stuttering:

It appears in the form of core behavior (intraphonemic disruptions) and reactions to the core behavior (prolongation- blocks- struggle- avoidance of speech situation) ***(Stromsta, 1986).***

IV- Hearing evaluation:

A proper speech and language evaluation begins with an evaluation of the child's hearing ability ***(Morales et al., 1992).***

The evaluation is performed according to the age:

I- Birth to 3 years:

1- Free field evaluation: Giving rough idea about hearing status of the child in both ears together at one time.

2- Immitancemetry: includes

a- Tympanometry to measure the middle ear

pressure.

b- Acoustic reflex to measure reflex contraction of both stapedius muscles in response to loud sound.

3- Auditory brainstem response (A.B.R.): The most valid objective method in uncooperative children.

4- Otoacoustic emission (O.A.E.): Objective reliable method for testing cochlear function.*(Morales et al., 1992).*

II- 3 years to 6 years:

- Play audiometry: The child is given a cube in his hand and asked to put the cube in a bucket when hearing the sound using pure tone stimuli (250-4000 Hz) to each ear separately
- Speech audiometry: Using phonetically balanced words to know the discrimination score
- Immitancemetry.
- A.B.R.
- O.A.E.

III- Above 6 years:

- Pure tone audiometry
- Immitancemetry

V- Neurodiagnostics:

1-Electroencephalography:

If a child is exposed to sudden or progressive change or loss of language, then the evaluation should include a wake and sleep EEG, with special attention being paid to the temporal lobe areas for possible seizure discharges or localized slowing that might suggest a seizure or other underlying neurological problem *(Gascon et al., 1973).*

If the history indicates delayed language development, the child may require an all-night EEG to exclude the possibility of nocturnal activation of spike-wave status in sleep seen with some of the language regression syndromes (see page 77). Such children usually require an all-night sleep recording to document the disorder *(Gascon et al., 2000).*

2- Video-EEG telemetry:

In hospital, evaluation with video-EEG telemetry to properly evaluate the sleep state and may be supplemented with a bedside speech and language evaluation while being monitored, which may be helpful in noting any relationship between the seizure discharges and the language abnormalities *(Gascon et al., 2000).*

3-Magnetoencephalography (MEG):

Multifactorial MEG has been introduced in the presurgical assessment as a supplement to EEG. In

contrast to EEG, MEG is not influenced by intervening tissues. Disadvantages of MEG include high costs and the susceptibility to movement artifacts *(Barry et al., 1999).*

4- CT scans and MRI scans:

It is indicated especially for resistant partial seizures, for complex partial seizures and for those seizures associated with speech and language deficits. These scans may reveal structural lesions, including temporal lobe atrophy, tuberous sclerosis nodules or minute vascular malformations *(Morrell, 1995).*

5- Single photon emission computed tomography and positron emission tomography:

SPECT and PET studies have been used to study cerebral metabolism in epilepsy and to plan surgery in epileptic patients *(Da Silva et al., 1997).*

Treatment of childhood epilepsy

I-antiepileptic drugs:

Pharmacological principles:

An antiepileptic drug does not affect only a specific brain site and a specific brain function. Rather, it affects the entire brain and many functions. Some seizures are due to excessive discharges of parts of the brain, as with partial seizures. Some seizures are due to bursts of inhibition of deeper portions of the brain, as with absence, myoclonic and atonic seizures. Anticonvulsants act by inhibiting excess discharges, by provoking excessive inhibition or by stopping the discharge spread. The actions of the brain are mediated by neurotransmitters. There are inhibitory neurotransmitters and excitatory neurotransmitters. Antiepileptic drugs tend to mimic the neurotransmitters, block the neurotransmitter action, facilitate the production of neurotransmitters or inhibit the breakdown and removal of them *(Mervaala et al., 1993).*

Antiepileptic action may be to inhibit a focus from firing, to limit the focus from becoming large enough to produce a seizure or to limit the spread from the focus to other parts of the brain. If the discharging focus inhibits or distorts a cognitive, linguistic or behavior signal traveling the involved neural pathway, then, the antiepileptic drug that inhibits the focus may improve the

related brain functions. Drugs that inhibit only the enlargement of the focus are less apt to benefit the associated dysfunctions. Drugs that primarily prevent the spread may have little benefit to the function of that area. Drugs that inhibit undesired excessive brain discharges tend to also inhibit desired brain functions, such as alertness, rate of thinking and language flow ***(Svoboda, 2004).***

Table (2): Neurotransmitters and epilepsy ***(Henry and Browne, 1987):***

Transmitter	Relation to epilepsy
1-Norepinephrine	Limit spread of seizures and its duration, its depletion decreases threshold for convulsants to produce seizures.
2-Dopamine	Induces a marked reduction in Hippocampal epileptiform activity.
3-Gamma aminobutyric acid	The brain's primary inhibitory neurotransmitter, maintain a reactive inhibitory tone that counterbalances neuronal excitation, inhibit release of glutamate, and its reduction might cause inadequate inhibition to stop a seizure.
4-Acetylcholine	Through its pathways, contribute to initiation and perpetuation of seizures.

Older versus newer drugs:

The older antiepileptic drugs are based on the original barbiturate structure. They tend to be inhibitory drugs, causing sedation and slowed functions. This effect extends beyond the brain, calming down excessive reactions in other organs. The barbiturates and their derivatives produce both good effects and side effects in the central nervous system and in other organs. If more than one or two drugs are prescribed together, the drugs are more apt to augment adverse aspects than to improve seizure control. The newly designed antiepileptic drugs are often targeted to specific neurotransmitter systems involved in seizure production and thus can be selected in additive combinations without being additive in side effects. The newer drugs being designed to mimic neurotransmitters are more limited in action to the CNS *(Henry and Brown, 1987).*

Antiepileptic drug side effects on speech and language:

Anticonvulsants can affect speech and language. At a high dose or in patients especially sensitive to the drug, the speech may be affected and the patient's overall thinking and processing, including the processing of language, may be impaired. Pre-existing language-processing problems can be worsened. The use of multiple anticonvulsants or combinations of anticonvulsants with other drugs affecting the brain, such

as tranquilizers, antidepressants, stimulants, etc., is more apt to produce the same problems as strong doses of a single medication *(Mervaala et al., 1993).*

Anticonvulsant drugs rarely improve speech or language problems, unless underlying epileptiform activity interferes with the language process. Even in the latter case, improvement is often incomplete and occurs only in some patients. When a child has a seizure disorder and a speech or language problem, the child needs appropriate anticonvulsant medications selected and monitored carefully, along with speech and language therapy *(Svoboda, 2004).*

Drugs for partial and generalized tonic-clonic seizures:

Drugs that are used most often to treat partial seizures and generalized tonic-clonic seizures include the barbiturates, carbamazepine, the hydantoins, and valproate, which is a broad spectrum drug *(Svoboda, 2004).*

1-Barbiturates:

The barbiturates include phenobarbital, mephobabarbital, metharbital and a related drug, primidone. Barbiturates can impair verbal learning and depress auditory processing, especially auditory discrimination and auditory memory (especially sequential memory), and impair language comprehension

and verbal expression. These effects are seen at higher therapeutic levels. If a focal seizure discharge interferes with speech or language, occasionally phenobarbital may help to overcome this. Otherwise, there is no direct benefit with speech and language problems ***(Svoboda, 2004).***

Phenobarbital is the most widely used antiepileptic drug in the world, but its possible deleterious cognitive and behavioral side effects remain an important concern among physician and patients. ***Hassan Tonekaponi et al. (2003)*** investigated whether discontinuation of phenobarbital in children with epilepsy is accompanied by improvement in cognitive function. The case group comprised 24 patients who discontinued phenobarbital and the control group was comprised of 21 children who continued to take phenobarbital. They reported that discontinuation of phenobarbital improved total IQ in the case group compared to the control group. This increase was mostly in performance (non verbal) items but verbal items remain almost unchanged.

2-Carbamazepine (tegretol):

No speech or language impairments have been noted with carbamazepine, but periods of dysfluency may occur if the drug levels get high, especially if the patient takes the medicine on an empty stomach ***(Mervaala et al., 1993).***

Tzitiridou et al. (2004) studied 70 patients (aged 5-11 years) newly diagnosed with benign childhood epilepsy with centrotemporal spikes (BECTS) who were

assigned to oxcarbazepine monotherapy. All of them underwent clinical and EEG examination of baseline and at 3-6 months intervals during the study. They suggested that oxcarbazepine is effective in preventing seizures and normalizing EEG and seems to preserve cognitive functions and behavioral abilities in children with BECTS.

3-Hydantoins (phenytoin):

Rarely, phenytoin has been claimed to benefit children with speech or language problems who have a temporal lobe epileptiform EEG. If phenytoin levels are high or if the patient is especially sensitive to the drug, dysfluency, often with a slowed speaking rate, is seen as early symptoms of intoxication *(Henry and Brown, 1987).*

The majority of patients with epilepsy become seizure-free with antiepileptic drug therapy. However, seizures in approximately one-third of patients with epilepsy are difficult to treat with antiepileptic drugs and require high doses or polytherapy. *Jokeit et al. (2005)* investigated 162 patients with refractory temporal lobe epilepsy to determine whether the antiepileptic drugs carbamazepine, phenobarbital and phenytoin affect the acquisition and retention of verbal and visual information. They found that patients with high serum levels of these antiepileptic drugs were selectively impaired in the retention but not acquisition of new information. They suggested that patients with refractory epilepsy with high serum levels of the antiepileptic drugs are at higher risk of accelerated forgetting.

4-Valproate:

Valproate is a useful drug for nearly all types of seizures. When used in combination with other drugs, the protein-binding potential of valproate may drive more of the unbound form of other protein-binding drugs into the brain, resulting in signs of intoxication (dysfluency and memory problems) despite normal drug levels on testing. Rarely, valproate may induce hearing, motor, and cognitive impairments *(Morales et al., 1992).*

Drugs for absence, atonic and myoclonic seizures:

Drugs that are used for absence, atonic, and myoclonic seizures include the benzodiazepines, the succinimides, and valproate *(Svoboda, 2004).*

1-Benzodiazepines:

These drugs may cause dysfluency. The spoken efforts may appear to be blocked, and sentences and phrases may be incomplete. This is seen when such drugs are used at high therapeutic doses or if the child is especially sensitive to the drugs. Diazepam has been associated with dysfluency and dysarthria *(Wilson et al., 1983).*

Clonazepam is likely to impair intellectual efficiency and to provoke behavioral disturbances. Attention, concentration, and cognitive functioning may deteriorate *(Trimble and Cull, 1990).*

2-Succinimides:

These drugs are relatively free of language impairments. Ethosuximide has been reported to improve verbal learning. Speech disturbances and lowered IQ have been suggested *(Henry and Browne, 1987).*

New anticonvulsants:

These are available for partial seizures, especially complex partial and secondarily generalized seizures, but some of these new drugs have shown widespread action on many seizure types *(Svoboda, 2004).*

1-Felbamate:

This drug is indicated for partial epilepsies and Lennox-Gastaut syndrome. Potentially, it may offer a broader spectrum of activity. There is no specific language impairmens *(Svoboda, 2004).*

2-Lamotrigine:

Another new drug released for partial epilepsies but with the potential to be another broad-spectrum antiepileptic medication. Rarely, speech disorders and dysarthria have been reported *(Svoboda, 2004).*

3-Tiagabine:

It may produce speech difficulties if the dose is built

up too rapidly or too high level. Thus, the medication is begun at low dosage and increased slowly. A very small percentage of patients on this drug may experience speech problems, such as dysarthria, or language problems *(Ojemann et al., 1999).*

4-Topiramate:

A newer potent anticonvulsant released for partial and primary generalized seizures. If it is started at too strong dose or increased too rapidly, the patient may become drowsy and confused, with problems in memory and thinking. There may be problems in speech and language, with dysphasia, dysnomia and dysfluency. Such problems, although usually transient, may recur with increasing dosages *(Ojemann et al., 1999).*

5-Zonisamide:

It is another new drug for complex partial seizures. Zonisamide may produce anomia, memory difficulties and slowness of thought, and this can be reflected on language aptitude, and learning abilities *(Mandelbaum et al., 2001).*

6-Vigabatrin:

It is a very effective drug in patients with complex partial seizures and patient with West's syndrome, but it was found to produce peripheral vision loss. Some patients experienced memory problems and word-finding difficulties *(Mandelbaum et al., 2001).*

II-Seizure surgery and speech and language functions:

1-Temporal lobectomy:

Children with epileptic seizures not responding to tolerated anticonvulsants, and especially those who may be developing language or learning problems, may be considered for a temporal lobectomy not only for seizure control but also to avoid the adverse effects on functioning and socialization. Early surgery may avoid the risk of language and memory decline and take the advantage of the young brain's plasticity to allow greater recovery of function *(Davies et al., 1995).*

Preoperative workup:

The dominant temporal lobe is more limited in the amount of brain tissue that can be resected. Reorganization of language function after focal brain injury shows that contralateral hemisphere contributions can emerge, even in adulthood *(Speer et al., 2001).*

Devinsky et al. (2000) reported that hemisphere dominance for language should be assessed before approaching any temporal surgery. There are a variety of approaches used in determining the lateralization and localization of language in individuals, including handiness and family history, the Wada test, and regional cerebral activation of blood flow (PET and functional magnetic resonance imaging).

1-Intracarotid amytal (Wada) test:

The Wada test, in which a fast-acting barbiturate is injected into the carotid arteries carrying blood to the brain, one side at a time is most useful. This results in half of the brain being anesthetized briefly. During this time, specific psychologic and language tests are performed to see which skills are lost when one side of the brain is put to sleep *(Devinsky et al., 2000).*

2-Fucntional imaging:

Hemispheric dominance for language can be assessed by function MRI (fMRI), a non-invasive technique for demonstrating cerebral activation associated with language tasks, which correlates well with the Wada test *(Devinsky et al., 2000).*

3-Cortical mapping:

Loring and Meador (2001) noted that cortical mapping of language functions in patients before a standard temporal lobectomy or lateral resections may reduce the incidence of postoperative language impairments, such as anomia. Mapping of the cortex may be done by electrocorticography (EcoG) before surgery.

4-Lateralizing signs before surgery:

Loddenkemper and Kotagal (2005) reviewed lateralizing signs during epileptic seizures with respect to prediction of the side of epileptogenic zone and

therefore, presurgical diagnostic value. The lateralizing significance of signs and symptoms can frequently be concluded from knowledge of the cortical representation. Visual, auditory, painful and autonomic auras, as well as ictal motor manifestations (clonic and tonic activity, unilateral epileptic spasms, unilateral automatism, ictal spitting and vomiting, emotional facial asymmetry, unilateral eye blinking and ictal nystagmus), have been shown to have lateralizing value. Furthermore, ictal language manifestations and postictal features (postictal dysphasia, postictal memory dysfunction and headache) are reviewed. They concluded that knowledge and recognition of lateralizing signs during seizures is an important component of the presurgical evaluation of epilepsy surgery.

Surgical approaches:

The extent of temporal neocortical resection varies. Resection of the superior temporal gyrus may be done. The goal of functional surgery is to remove cellular centers or to interrupt neural pathways of seizure spread. The most common seizure surgery is the removal of all or, more often, part of the temporal lobe for control of complex partial seizures resistant to medical therapy. The major concern is that surgery should not remove any vital language area. To optimize the results, as much hippocampus as possible should be resected. The risk of taking more of the hippocampus posteriorly is damage to vital arteries, so these vessels need to be left intact. Memories already stored are not affected because the storage is more diffuse throughout the brain ***(Davies et***

al., 1995).

Results:

The temporal lobe is involved intimately with higher-level perceptual processing of sounds. A number of studies have focused on identifying possible postoperative changes in hearing function. ***Cranford et al. (1996)*** suggested that very little, if any, improvement in central auditory functioning is expected following temporal lobe surgery.

Postoperative complications:

Loring and meador (2001) reported that the complication rate is not higher than 1%. Conservative surgery on the dominant temporal lobe (i.e. language-related) of severely handicapped seizure patients may result in an anomia for the names of people and things more than verbs. Fluency is not affected. Some patients experience dysphasic symptoms that improve or normalize over time with dominant temporal lobe surgery. Both left and right temporal lobectomy patients show variable decreases in verbal memory, but those with a right temporal lobectomy show no language problem and may show improvement instead.

B-Corpus callosum sectioning:

In patients with seizure resistant to medication who

are not suitable for resection of a seizure site, a corpus callosotomy may be considered to prevent the spread of the discharge. A corpus callosum sectioning allows about 71% of patients to have a significant and valuable reduction in seizure frequency and/or intensity, but none become seizure-free ***(Rayport et al., 1991).***

Expressive language deficits after a callosotomy have been reported in patients with mixed cerebral dominance. There is a marked decrease in spontaneous speech. No deficit in receptive language function is observed. The deficit occurs only after sectioning of the posterior portion of corpus callosum ***(Crone et al., 1992).***

C-Subpial transections:

Multiple subpial transections (MSTs) for control of intractable seizures are based on the concept of horizontal epileptic discharges and vertical functional organization of the cerebral cortex. The MSY disconnects horizontal connections to decrease seizure propagation, preserving columnar functional organization. This is a surgical procedure designed to eliminate the capacity of cortical cells to generate epileptiform discharge without interfering with normal function. This has been performed in Broca's and Werniche's areas ***(Morell et al., 1995).***

MST is developed especially for those with Landau-Kleffner epileptic syndrome. It also has been used in focal motor or sensory seizures as well as in Rasmussen's

syndrome. Following surgery, one-third of patients recover completely, the remainder improves dramatically and is able to speak in sentences, but they still require special language training programs. Patients are able to return to school or work (*Loring and Meador, 2001)*.

D-Hemidecortication and hemispherectomy:

Hemispherectomies, as used in patients with Rasmussen's syndrome, result in a spastic hemiparesis and loss of related other contralateral sensory, visual, and motor functions, including language if the dominant hemisphere is involved. A surprising amount of recovery ensues, including language, as the other hemisphere takes over. This is seen especially when the surgery is performed in a younger child *(De Bode et al., 2000).*

III-Vagal nerve stimulation (VNS):

The mechanism of action of VNS remains unknown, although it is clearly different from that of pharmacotherapy which involves effects on neuronal membrane ionic conductance or their neurotransmitters and their receptors. VNS is mainly an option for non-surgical cases, offering chances of a significant reduction in seizure frequency by 25-30%, and in some up to 50% after one year. Partial seizures are helped the most, but GTCS and atypical absence as well as the atonic and tonic attacks of Lennox-Gastaut syndrome may benefit.

Improvement in alertness, verbal skills, memory, mood, and school performance emerge at three months and are even better at 15 months. The approach is effective, safe, and reversible, but the stimulus itself may produce changes of voice. Rarely, the voice may change in pitch during the firing of the stimulus. Other symptoms noted, especially when the stimulator emits an electrical charge, include a gagging sensation, cough, or exertional shortness of breath. Adverse effects are apt to affect speech rather than produce any language problems *(Gates, 1999).*

Hallbook et al. (2004) reported that VNS is a neurophysiological treatment for patients with refractory epilepsy. They investigated the effects of VNS on seizure frequency and severity and how these changes are related to cognitive abilities, behavior and mood. They concluded that the study has shown a good anti-seizure effect of VNS, an improvement in seizure severity with reduction in frequency, and a tendency to improvement regarding behavior, mood and depressive parameters.

Management of speech and language problems in childhood epilepsy

It is primarily directed towards treatment of the cause. Prescribing the suitable antiepileptic drug is mandatory, putting into consideration those drugs with side effects on communication to be avoided. It is rather a compromise between the necessity of the drug according to type of epilepsy and the dose and the associated communication disorder.

I- Treatment of language problems:

1- Treatment of D.L.D.:

a- Family counseling:

The parents and family need to be involved in the therapy *(Henry and Browne, 1987).*

It includes showing the parents how to communicate with the child and how to bring language learning into daily life activities.

1- Avoid commands, correction, question and frustration.

2- Using simple statements on each activity done by the child.

3- Rewarding is important.

4- Follow up every 6 weeks.

b- Direct training:

- Attention control: helping the child to focus and hold his attention according to the attention stage he is in.
- Performance abilities: help the child to use language (reception of adult‘ language) in planning and monitoring his activities.
- Concept formation: it involves the ability to move beyond the immediate perception towards the abstraction.
- Symbolic understanding: the aim is to get the child to understand that each symbol must be translated into concept.
- Verbal comprehension: the child can follow directions relating to 3 or more objects and can understand beyond the concept of “here” and “now” using the past and future tenses.
- Expressive language: it is stimulated by

  - Focusing on content.

  - Reinforcement by corrective feedback.

  - Expanding the utterance by extended feedback.
- Articulation
- Intellectual use of language

2- Treatment of dysphasia:

a- Environmental language intervention:

includes family counseling in the form of practical guidelines (*Scheull et al., 1964).*

- Slow down the rate of speech.
- Allow pauses between topics.
- Use short sentences.
- Encourage the patient attempts to communicate.

b- Direct intervention programs:

- Schuell stimulation approach: it is the stimulation of disrupted processes to function normally, through the use of suitable, isolated, repeated, augmented stimuli presented via multisensory channels in order to elicit a response (*Scheull et al., 1964).*

- Programmed instruction approach: in which the therapist define the language behavior to be taught. The program then moves in small, controlled steps toward closer approximation of the desired behavior. The program does not move to the following step without eliciting a calculated response from the patient *(Holland, 1972).*

3- Treatment of learning disability:

a- The Orton Gillingham remediation technique (Williston, 1980):

It is an alphabetic phonic approach to teach language skills using multisensory involvement. It concentrates on fusing smaller units (letters and syllables) into more complex whole words.

b- Fernald approach (Fernald, 1943):

It is a multisensory approach of learning whole word forms followed by analytic approach to form new combinations of new words even if non meaningful. The child gets the ability to recognize new words from their similarity to words or parts of words already learned.

c- Computer assisted instruction:

The computer provides a high degree of individual attention as well as multisensory experience in word construction; the words are typed into the computer. The children see the words and then repeat the phonemes along with the computer voice *(Rotenberg, 1984).*

II- Treatment of speech problems:

1- Treatment of stuttering:

a- Family counseling: (Curlee, 1989) it includes

- Parents should listen carefully when their children talk
- Limit the number of questions.
- Try to avoid verbal interruption.
- Change from negative behavior as annoyance to elicit behavior to rewarding and encouraging.
- Parents are instructed to identify dysfluency.

b- Van Riper method (Van Riper, 1973): it consists of:

1- Motivation: the clinician establishes a warm relationship with the child.

2- Identification: the child's attention is brought to involuntary movements.

3- Desensitization: reduction of frustration.

4-Variation: help the stutterers to realize that they can change their abnormal behavior and they can stutter fluently.

5-Alteration: the child is taught to manage the stuttering situation.

Stabilization: stabilize the new behavior.

c- Lidocombe Program: (Onslow and Packman, 1997):

It is based on weekly visits to the clinic by the child and parents in the same time, the parents carry out the treatment procedure at home.

Parents are instructed to record a once daily speech sample at different times and speaking situations,

Two measurements are done:

1- Severity rating which is made of 10 points scale, 1 means no stuttering, 10 means extremely severe stuttering

2- Calculation of the child's stuttering per minute time.

Parents learn to focus on rewarding the stutter free speech.

The parents can wait until the child has finished the stuttered utterance and then repeat it without stuttering

or ask the child to repeat it without stuttering

d-Modified coarticulation technique:

It is based on the idea of coarticulation which is evident in normal and fluent speech, while its absence may result in stuttering. It is done by preparing for the second sound of the syllable before uttering it *(Stromsta, 1986).*

2- Treatment of dysarthria (Kotby et al., 1992):

It is done by controlling of the following

a- Articulation:

- Syllable by syllable attack.
- Consonant exaggeration.

b- Phonation:

- Adjustment of optimum pitch.
- Adjustment of optimum loudness.
- Smith Accent method for dysphonia in elder children if cooperable.

c- Resonance: treatment of hypernasalty.

d- Respiration: by breathing exercise and physiotherapy.

SUMMARY

Communication is the exchange of ideas and meaning between two or more persons. Failure to communicate accurately may lead to many of the problems experienced in families, in school, in social interactions and in employment.

Language is an arbitrary symbolic system that pairs sounds and signs to meaning. Speech is defined as acoustic vibrations resulting from patterns of movement of speech organs such as lips, tongue, jaw, palate and pharynx.

Epilepsy is a disorder of the brain characterized by generation of seizures and by neurological, cognitive, psychological and social consequences of this condition. The rates of epilepsy range between 20-70 per 100,000 populations per year.

The two major categories of epileptic seizures are known as partial (focal) seizures and generalized seizures. Most partial seizures are classified either as partial with elementary symptoms (motor or sensory) or partial with complex symptoms (disturbed consciousness). Generalized seizures may be tonic-clonic seizures or absence seizures.

The brain is a dynamically changing and developing organ, especially in the early years of life. Seizures can inhibit or distort brain development as well as the related functions. Seizures and seizure therapy interfere with brain functions by over activation, inhibition or

destruction of vital brain functional pathways.

The incidence of speech and language problems was reported to be 24% of children with epilepsy. Dysarthria can be seen with elementary partial seizures, usually associated with more obvious ictal symptoms. A temporary articulation problem may be a postictal symptom of oral damage occurred during a generalized tonic-clonic seizure. Dysfluency may be a characteristic of left complex partial seizures, as an interictal or postictal finding. Epilepsy involving the left hemisphere is most likely associated with language problems. Such problems may precede, occur simultaneously with, or follow seizures. The problems may be episodic or ongoing, if ongoing; they may result in a gradual deterioration of language abilities or delayed language development from the start. The manifestations of such problems depend on the location of the seizure disturbance. The seizure discharge may disrupt the language processing, which results in delayed language in early childhood or dysphasic symptoms if it occurs after language development. All types of expressive and receptive dysphasias may occur.

Disorders of communication may also underlie learning difficulties and behavior problems. Up to 50% of children with epilepsy have learning difficulties, with up to 30% at risk of developing serious learning problems. Learning difficulties may present as retardation or learning disabilities. Retardation is most apt to appear in individuals with generalized seizures. Learning disabilities are more apt to be seen with partial seizures and medication reactions. Epileptic discharges may disrupt

processing, thus interfering with attention, learning processing, storage or retrieval of information.

About 30% of patients with epilepsy have some kind of psychiatric symptoms. Behavioral problems may be related to both extrinsic and intrinsic factors. Extrinsic factors include the attitudes of the parents and siblings and the resultant self-concept of the individual. Intrinsic factors may result from the nature and location-lateralization of epilepsy. Behavior problems associated with childhood epilepsy include attention deficits, autism, anxiety, depression, conduct disorders, and psychosis.

Landau-Kleffner syndrome of acquired dysphasia with epilepsy is a childhood syndrome consisting of loss of language functions, the usual emergence of a variety of seizures with EEG abnormalities and a resultant deterioration of behavior. The condition stabilizes for years and then the seizures cease and the EEG often normalizes, usually before ten years of age. In adolescence, the dysphasia improves, with gradual partial recovery of language functions.

Children who have epilepsy are at risk of developing speech and language problems and should be evaluated by:

1-Preliminary diagnostic measures.

2-Clinical diagnostic aids including:

a-Language assessment by tests for DLD, tests for dysphasia and tests for learning disabilities.

b-Speech evaluation by assessment of dysarthria and stuttering.

c-Hearing evaluation according to age of the child.

3-Additional instrumental measures.

If a child is having an evident of progressive change or loss of language, then the evaluation should include awake and sleep EEG, with special attention being paid to the temporal lobe areas for possible seizure discharges or localized slowing that might suggest a seizure or other underlying neurological problem.

An antiepileptic drug does not affect only a specific brain site and a specific function, but it affects the entire brain and many functions. At a high dose, or in patients especially sensitive to the drug, the patient’s overall thinking and processing, including the processing of language, may be impaired. The use of multiple anticonvulsants or combinations of anticonvulsants with other drugs affecting the brain is more apt to produce the same problems as strong doses of a single medication.

It is advised that children with seizures not responding to tolerated antiepileptic medications, and especially those who may be developing language or learning problems, may be considered for a temporal lobectomy not only for seizure's control but also to avoid the adverse effects on functioning and socialization. Early surgery may avoid the risk of language and memory decline and take advantage of the young’s brain plasticity to allow greater recovery of functions.

Treatment of speech and language problems should be done by:

1-Treatment of DLD by family counseling and

language therapy program.

2-Treatment of dysphasia by environmental language intervention and direct intervention programs.

3-Treatment of learning disabilities by Orton Gillingham remediation technique, Fernald approach or others.

4-Treatment of stuttering by family counseling, Van Riper method, Lidocomb program or modified coarticulation technique of Stromsta.

5-Treatment of dysarthria through syllable by syllable attack, consonant exaggeration and respiratory control.

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الاعتلالات التخاطبية المصاحبة لحالات صرع الأطفال

بروتوكول رسالة

توطئة للحصول على درجة الماجستير في أمراض التخاطب

مقدمة من

الطبيبة / دينا أحمد السيد الرفاعى

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الأستاذ الدكتور / نوران نجدى العسال

أستاذ مساعد أمراض التخاطب

كلية الطب – جامعة عين شمس

الأستاذ الدكتور / أمل سيد صابر

أستاذ مساعد أمراض التخاطب

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2005

الملخص العربى

إن التخاطب هو تبادل للأفكار و المعاني ما بين شخصين أو أكثر. و قد يؤدي الإخفاق في التخاطب على نحو صحيح إلى كثير من المشاكل في الأسرة أو المدرسة أو في التفاعلات الاجتماعية أو في العمل .
أما اللغة فهي نظام رمزي افتراضي يقترن فيه الصوت و الإشارات بالمعاني . و يعّرف الكلام على أنه الذبذبات الصوتية الناتجة عن حركة أعضاء الكلام مثل الشفاه , و اللسان , و الفك و سقف الحلق و البلعوم.
و الصرع هو اضطراب دماغي يتميز بحدوث نوبات مرضية و بتبعات عصبية و ادراكية و نفسية و اجتماعية لتلك الحالة. و تتراوح معدلات الصرع ما بين 20 إلى 70 لكل مائة ألف شخص سنويا .
و تقسم نوبات الصرع إلى نوعين رئيسيين و هما نوبات الصرع الجزئية (البؤرية) و النوبات الكلية .و يتم تصنيف نوبات الصرع الجزئية إما على أنها جزئية تصاحبها أعراض أولية (حركية أو حسية) أو جزئية مع أعراض معقدة (اعتلال الوعي) .و قد تكون نوبات الصرع الكلية توترية ارتجافية أو غير مصحوبة بنوبة .
و الدماغ هو عضو ديناميكي متغير و متطور خاصة في السنوات الأولى من عمر الطفل. و يمكن لنوبات الصرع أن تثبط أو تغير من تطور الدماغ و كذلك من الوظائف المرتبطة له. كما يمكن أن تتعارض النوبات و علاجها مع وظائف الدماغ عن طريق زيادة نشاط المسارات الوظيفية الدماغية الهامة أو تثبيطها أو تشتيتها مما قد ينعكس على القدرات التخاطبية .
و قد وجد أن 24 % من الأطفال المصابين بالصرع يعانون من مشاكل في الكلام أو اللغة , إذ يمكن أن تظهر الحبسة الكلامية مع نوبات الصرع الجزئية الأولية ., كما يمكن أن تكون مشاكل النطق المؤقتة عرضا لضرر أصاب اللسان أثناء نوبة كلية توترية ارتجافية . و يمكن أن يكون التلعثم صفة مميزة لنوبات الصرع الجزية المركبة التي تصيب الجانب الأيسر من الدماغ حيث يحدث

ما بين النوبات أو أثنائها . و عادة ما تكون مشاكل اللغة مصاحبة لصرع في النصف المخي الأيسر .و قد تسبق هذه المشاكل نوبات الصرع أو تصاحبها أو تكون لاحقة عليها . كما أنها يمكن أن تكون عارضة أو مستمرة و إذا كانت مستمرة فيمكن أن تؤدي إلى تدهور تدريجي في القدرات اللغوية أو إلى تأخر في نمو اللغة من البداية.
و تعتمد مظاهر تلك المشاكل على مواقع نوبات الصرع بالدماغ .
كما يمكن أن تعطل نوبات الصرع من نشوء اللغة مما قد يؤدي إلى تأخر نمو اللغة في السنوات الأولى من عمر الطفل أو إلى أعراض العي إذا ما حدثت بعد اكتمال اللغة . و بالتالي يمكن أن تتسبب في حدوث أي من أنواع العي التعبيري أو الاستقبالي .
كما يمكن أن تتضمن اعتلالات التخاطب صعوبات التعلم و المشاكل السلوكية . حيث وجد أن حوالي 50 % من الأطفال المصابين بالصرع يعانون من صعوبات في التعلم و أن حوالي 30% منهم عرضة لمشاكل شديدة في التعلم . و قد تظهر صعوبات التعلم كتأخر أو عجز في التعلم . و عادة ما يميل التأخر إلى الظهور بين الأطفال المصابين بالنوبات الكلية . بينما يميل العجز في التعلم إلى الظهور مع نوبات الصرع الجزئية أو كرد فعل للعلاج بأدوية الصرع. .و قد تعطل الانطلاقات الكهربية الصرعية من عمل الدماغ و بالتالي تؤثر على الانتباه و التعلم و تخرين المعلومات و استرجاعها .
و يعاني حوالي 30 % من الأطفال المصابين بالصرع من عرض ما من الأعراض النفسية.
و قد ترتبط المشاكل السلوكية بعوامل خارجية أو داخلية . و تتضمن العوامل الخارجية مواقف الوالدين و الأخوة و الشكل الذي ينظر به الطفل إلى نفسه نتيجة لذلك . و قد تنتج العوامل الداخلية من موقع و طبيعة الإصابة بالصرع . و تتضمن المشاكل السلوكية المصاحبة لصرع الأطفال نقص الانتباه و القلق و الاكتئاب و الاضطراب النفسي و مرض التوحدية .
و قد تصيب متلازمة لاندو كليفنر للعي المكتسب للأطفال و تتكون من فقدان وظائف اللغة , و ظهور نوبات صرعية متنوعة مع تغيرات في رسم الدماغ الكهربائي ينتج عنه تدهور في السلوك . و

عادة ما تستمر هذه الحالة إلى أن تتوقف النوبات الصرعية و يعود الرسم الكهربي للدماغ إلى شكله الطبيعي ليتحسن العي مع شفاء جزئي تدريجي للوظائف اللغوية .

إن الأطفال المصابين بالصرع معرضين لمشاكل في اللغة و الكلام و يتم تقييمهم عن طريق ما يلي :

1- مقاييس التشخيص الابتدائي

2- مساعدات التشخيص الإكلينيكية و التي تتضمن :

- تقييم للغة بواسطة اختبارات نمو اللغة أو اختبارات العي و اختبارات صعوبات التعلم
- تقييم الكلام باختبارات الحبسة الكلامية أو التلعثم
- تقييم للسمع تبعا لعمر الطفل

3- المقاييس التشخيصية الإضافية

إذا ما كان هناك دليل على أن الطفل يعاني من تغير متقدم أو فقدان للغة , فيجب أن يتضمن التقييم رسم دماغ كهربي أثناء الاستيقاظ و أثناء النوم مع اهتمام خاص بمناطق الفص المخي الصدغي لاحتمالات وجود إشارة صرعية أو تباطؤ يفيد باحتمال حدوث نوبة صرعية أو وجود أي مشكلة عصبية أخرى .

و لا تؤثر أدوية علاج الصرع على منطقة معينة من الدماغ فقط أو على وظيفة معينة فحسب و إنما على الدماغ ككل و العديد من وظائفه . و من المتوقع عند إعطاء هذه الأدوية بجرعات كبيرة أو للأشخاص المصابين بحساسية لهذه الأدوية أن يتأثر تفكير و عمل الدماغ و يتضمن ذلك حدوث مشاكل في اللغة كما يمكن أن تحدث نفس المشاكل عند استخدام أكثر من دواء من أدوية الصرع أو مع أدوية أخرى تؤثر على المخ .

و ينصح للأطفال غير المستجيبين لأدوية الصرع و بالأخص هؤلاء ممن يعانون من مشاكل لغوية أو صعوبات في التعلم بأن يتم النظر في إمكانية أجراء عملية جراحية لهم لاستئصال جزء الفص الصدغي المخي و ليس ذلك فقط من أجل التحكم في الصرع و إنما أيضا من أجل تجنب التأثيرات العكسية على علاقات الشخص الاجتماعية و عمله . و يمكن للجراحة المبكرة أن تتجنب مخاطر

التدهور في اللغة و الذاكرة و أن تستفيد من ليونة دماغ الأطفال و ذلك للسماح بأكبر قدر من استعادة وظائف الدماغ .

و يتم علاج مشاكل الكلام و اللغة كالآتي :

1- علاج تأخر نمو اللغة عن طريق الاستشارة العائلية و برامج التدريبات اللغوية
2- علاج العي عن طريق تدخل لغوي بيئي و برامج التدخل المباشرة .
3- علاج صعوبة التعلم بطريقة أورتون جيلنجهام و طريقة فيرنالد و غيرها
4- علاج التلعثم بالاستشارة العائلية و برامج ليدكوكب و كذلك طرق فان ريبر و سترومستا .
5- علاج الحبسة الكلامية عن طريق استهلال المقطع تلو المقطع و تضخيم السواكن و التحكم في التنفس .